

12 weeks after surgery belonged to clinical stage of heart failure, and 16 weeks after surgery belonged to refractory stage of heart failure.

GW26-e2500

Effect of Salidroside on NRF-1 and NRF-2 of Rats Myocardial after Acute Exhaustive Exercise at Different Time

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OBJECTIVES Nuclear respiratory factors NRF-1 and NRF-2 are the key factors of the regulation of mitochondrial biogenesis. Through the establishment of a single bout of exhausted swimming model, our work is to research the protein and gene expression of mitochondrial biogenesis related factors, NRF-1 and NRF-2, at different time after exhaustive exercise. In order to investigate the effects of exhaustive exercise on NRF-1 and NRF-2, and the effect of Salidroside (SAL) of rats after acute exhaustive.

METHODS A total of 80 health male Sprague Dawley rats (average weight $(120 \pm 20\text{g})$) were randomly divided into 10 groups ($n=8$ in each group), including sedentary control group, exhausted exercise groups (0, 6, 12 and 24 hours after exhausted exercise), SAL and exhausted exercise groups (0, 6, 12 and 24 hours after exhausted exercise). Sedentary control group and exhausted exercise groups were administered with saline (10ml/kg) intragastrically for 14 days. SAL and exhausted exercise groups were administered with SAL (300mg/kg) intragastrically for 14 days. Then exhausted exercise model was established. Exhaustive exercise model is a single bout of exhausted swimming model, according to Thomas standards. Gene expression levels and protein expression of NRF-1, NRF-2 protein were detected.

RESULTS 1. The gene expression of NRF-1 of other groups were significantly higher than that of control group ($P < 0.05$), and 6h groups was the highest. With the same time of exhaustive group, the levels of each SAL group NRF-1 mRNA increased significantly ($P < 0.05$).

2. In addition to exhaustive 24h group after exhausted exercise, the gene expression of NRF-2 of other groups were significantly higher than that of control group ($P < 0.05$), and 6h groups was the highest. With the same time of exhaustive group, the levels of each SAL group NRF-2 mRNA increased significantly ($P < 0.05$).

3. Compared with control group (0.8574 ± 0.0180), level of the protein expression of NRF-1 in rats myocardial of 0h (0.7131 ± 0.0343), 6h (0.7132 ± 0.0377) and 24h (0.5910 ± 0.0404) after exhausted exercise, SAL and 0h (0.6135 ± 0.0384), 12h (0.5646 ± 0.0217), 24h (0.5617 ± 0.0283) after exhausted exercise were Significantly lower ($P < 0.05$).

4. Compared with control group (0.7523 ± 0.0333), level of the protein expression of NRF-2 in rats myocardial of SAL and 6h (1.0295 ± 0.0506), 12h (1.1343 ± 0.0632), 24h (0.8346 ± 0.0371) after exhausted exercise were Significantly higher ($P < 0.05$). 0h (0.3624 ± 0.0532), 6h (0.6601 ± 0.0231), 24h (0.6752 ± 0.0337) after exhausted exercise, SAL and 0h (0.6020 ± 0.0173) after exhausted exercise were Significantly lower ($P < 0.05$).

CONCLUSIONS Salidroside can promote the expression of NRF-1, NRF-2 mRNA and the expression of NRF-2 protein of rats myocardial after exhausted exercise.

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Increased expression of mitochondrial calreticulin in a rat model of dilated cardiomyopathy

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OBJECTIVES Calreticulin (CRT) is involved in the progress of dilated cardiomyopathy (DCM), but the underlying mechanism is still unknown. Since mitochondria have a key role in the progress of DCM, the present study analyzed whether CRT is localized at mitochondria of cardiomyocytes and whether such localization is affected under DCM.

METHODS The DCM model was generated in rats by the daily oral administration of furazolidone for thirty weeks. Echocardiographic and hemodynamic studies were used to assess cardiac function. The location of CRT and its expression were studied by immuno-electron microscopy and western blot. The myocardial apoptosis and mitochondrial enzyme activities were further studied.

RESULTS Echocardiographic and hemodynamic studies demonstrated enlarged left ventricular dimensions and reduced systolic and

diastolic function in DCM rats. Immuno-electron microscopy and western blot showed that CRT was present in cardiomyocyte mitochondria and the mitochondrial content of CRT was increased in DCM hearts ($P < 0.05$). Morphometric analysis showed notable myocardial apoptosis and mitochondrial swelling with fractured or dissolved cristae in the DCM hearts. Compared with the control group, the mitochondrial membrane potential level of the freshly isolated cardiac mitochondria and the enzyme activities of cytochrome c oxidase and succinate dehydrogenase in the model group were significantly decreased ($P < 0.05$), and the myocardial apoptosis index and the caspase activities of caspase-9 and caspase-3 were significantly increased ($P < 0.05$). Pearson linear correlation analysis showed that the mitochondrial content of CRT had negative correlations with the mitochondrial function, and a positive correlation with myocardial apoptosis index ($P < 0.001$). The protein expression level of cytochrome c and the phosphorylation activity of STAT3 in the mitochondrial fraction were significantly decreased in the model group compared with the control group ($P < 0.05$).

CONCLUSIONS These data demonstrate that CRT is localized at cardiomyocyte mitochondria and its mitochondrial content is increased in DCM hearts.

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Continuous Angiotensin-(1-7) infusion improves myocardial calcium transient and calcium transient alternans in ischemia-induced cardiac dysfunction rats

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OBJECTIVES The effects of Ang-(1-7) on calcium homeostasis in dysfunctional cardiomyocytes have not been fully elucidated. The aim of this study was to evaluate the impact of Ang-(1-7) on calcium transient (CaT) in cardiomyocytes during the pathogenesis of heart failure.

METHODS Cardiac dysfunction was induced by ligation of left anterior descending (LAD) coronary artery in adult SD rats. Randomly selected rats were ligated and continuously infused with Ang-(1-7) [HF+Ang-(1-7) group] or saline (HF+saline group) via osmotic minipumps. Sham-operated group without ligation of LAD was also included. After 28 days, hemodynamic parameters were assessed and left ventricular myocytes were isolated. The CaT, the heart rate threshold of CaT alternans (CaT-Alt) and L-type Ca^{2+} channel current (I_{cat}) were recorded or measured.

RESULTS The results showed that continuous Ang-(1-7) treatment could attenuate the impairment of cardiac function following LAD ligation. Compared to the Sham-operated group, the HF+saline group showed a decreased CaT amplitude, and a prolonged 50% and 90% CaT recovery time (all $P < 0.05$). However, Ang-(1-7) significantly improved these abnormalities (all $P < 0.05$ vs HF+saline group). Moreover, Ang-(1-7) significantly improved these abnormalities (All $p < 0.05$ vs HF+saline group). Moreover, the heart rate threshold of CaT-Alt was significantly reduced in HF+saline group, and Ang-(1-7) partly restored it ($P < 0.05$ vs HF+saline group). Although the I_{cat} reduced in dysfunctional myocytes, Ang-(1-7) had no effects on it.

CONCLUSIONS Ang-(1-7) attenuates CaT disturbance and increases the heart rate threshold of CaT-Alt during the pathogenesis of HF. These effects contribute to its benefits on improving contractile dysfunction preventing the incidence of arrhythmia in dysfunctional myocytes.

GW26-e3512

Tong Xinluo regulated gene transcription in human umbilical vein endothelial cells injured by homocysteine

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OBJECTIVES To identify key genes differentially expression in the human umbilical vein endothelial cells (HUVECs) injured by homocysteine (HCY) and the protective effect of TXL.

METHODS Human umbilical vein endothelial cells cultivated *in vitro* were divided into three groups: normal group, HCY group (treated with HCY 5mmol/ml 48hours) and TXL group (treated with HCY 5mmol/ml and TXL supermicro powder 5mg/ml 48hours). The total RNA extracted from HUVECs was assessed for differential expression