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The Protection of Salidroside on Cardiac Function of Repeated Exhaustive Rat Via Anti-Oxidative Stress and MAPKs Signal Transduction

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***SUMMARY*. The study is to investigate the cardioprotective effect of salidroside against repeated exhaustive injury and explore the potential involvement of anti-oxidative stress and MAPKs signal transduction. In this experiment, we found that salidroside reduced the myocardium ultrastructure injury caused by exhaustive swimming, decreased the contents of mitogen-activated protein kinases (CK), kinase isoenzyme (CK-MB), lactate dehydrogenase (LDH) in repeated exhaustive rats significantly, and improved the LVDP, ± LVdp/dt$_{max}$ of repeated exhaustive rats effectively. Furthermore, salidroside also reduced the content of malondialdehyde (MDA) and the phosphorylation degree of N-terminal kinase (JNK), p38 mitogen-activated protein kinase (p38 MARK), increased the content of superoxide dismutase (SOD) and the phosphorylation degree of extra cellular signal-regulated kinase (ERK) of repeated exhaustive rats, so we conclude that salidroside has the protection on the heart against repeated exhaustive injury and the mechanism is related to anti-oxidative stress and MAPKs signal transduction.**

RESUMEN. El propósito de este estudio es investigar el efecto cardioprotector del salidrósido contra lesiones exhaustivas y repetidas y explorar la posible participación del estrés anti-oxidante y transducción de señales MAPK. En este experimento se encontró que el salidrósido reduce la lesión ultraestructural del miocardio causada por la natación exhaustiva, disminuye significativamente los contenidos de las proteín-quinasas activadas por mitógenos (CK), la isoenzima quinasa (CK-MB) y la lactato deshidrogenasa (LDH) y mejora la LVDP, ± LVdP/dtmax de ratas sometidas a natación exhaustiva. Además, el salidrósido también redujo el contenido de malondialdehído (MDA) y el grado de fosforilación de la quinasa N-terminal (JNK), la proteín-quinasa p38 mitógeno-activada (MARCA p38), aumentó el contenido de la superóxido dismutasa (SOD) y el grado de fosforilación de la quinasa extracelular regulada por señal (ERK) de ratas sometidas a natación exhaustiva, por lo que concluimos que el salidrósido ejerce una protección del corazón contra lesiones exhaustivas repetidas y el mecanismo estaría relacionado con el estrés oxidativo y la transducción de señales anti-MAPK.

KEY WORDS: Anti-oxidative stress, Heart protection, MAPKs Signal transduction, Repeated exhaustion, Salidroside.

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