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Effects of Formononetin on Oxidative Stress, Inflammatory Response and PI3K/Akt Pathway in Pulmonary Arterial Hypertension: An Animal Model Study

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***SUMMARY*. The work investigated the alleviating effects and mechanisms of formononetin on pulmonary arterial hypertension (PAH) in rats. Forty rats were divided into control, monocrotaline, and 20 and 40 mg/kg formononetin groups. The PAH model was made in the latter three groups. Then, the latter two groups were treated with 20 and 40 mg/kg formononetin, respectively, for two weeks. Compared with control group, in 40 mg/kg formononetin group the body weight was increased, the mean pulmonary arterial pressure, right ventricular systolic pressure and right ventricular hypertrophy index were decreased, the serum superoxide dismutase activity was increased, the serum malondialdehyde, tumor necrosis factor α, interleukin 1β and interleukin 6 levels were decreased, and the lung tissue phosphoinositide-3 kinase (PI3K) and protein kinase B (Akt) protein expression levels were decreased. Formononetin may alleviate the monocrotaline-induced PAH in rats via resisting oxidative stress and inflammatory response and regulating PI3K/Akt pathway in lung tissue.**

RESUMEN. El trabajo investigó los efectos y mecanismos de alivio de la formononetina sobre la hipertensión arterial pulmonar (HAP) en ratas. Se dividieron cuarenta ratas en grupos de control, monocrotalina y de 20 y 40 mg/kg de formononetina. El modelo de PAH se realizó en los últimos tres grupos. Luego, los dos últimos grupos fueron tratados con 20 y 40 mg/kg de formononetina, respectivamente, durante dos semanas. En comparación con el grupo de control, en el grupo de 40 mg/kg de formononetina el peso corporal aumentó, la presión arterial pulmonar media, la presión sistólica del ventrículo derecho y el índice de hipertrofia del ventrículo derecho disminuyeron, la actividad de superóxido dismutasa sérica aumentó, el malondialdehído sérico, la necrosis tumoral los niveles de factor α, interleucina 1β e interleucina 6 disminuyeron, y los niveles de expresión de la proteína fosfoinositido-3 quinasa (PI3K) y proteína quinasa B (Akt) en el tejido pulmonar disminuyeron. La formononetina puede aliviar la HAP inducida por monocrotalina en ratas mediante la resistencia al estrés oxidativo y la respuesta inflamatoria y regulando la vía PI3K/Akt en el tejido pulmonar.

KEY WORDS: Akt, formononetin, PI3K, pulmonary arterial hypertension.

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