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Protection of Vitexin against Cerebral Ischemia-reperfusion Injury in Mice and the Mechanism

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***SUMMARY*. In this study, the protection of vitexin against cerebral ischemia-reperfusion injury (CIRI) in mice and the mechanism were investigated. Ninety mice were randomly divided into control, model and 10, 20, and 40 mg/kg vitexin groups, 18 mice in each group. The latter three groups were administrated with 10, 20, and 40 mg/kg vitexin, respectively, for seven days. Then, the CIRI model was established in the latter four groups. After 24 h from the modeling, compared with model group, in 20 and 40 mg/kg vitexin groups, the neurological deficit score, cerebral water content, and cerebral infarction range were significantly decreased, the brain tissue superoxide dismutase and glutathione peroxidase levels were significantly increased, the brain tissue malondialdehyde level was significantly decreased, the brain tissue tumor necrosis factor α, interleukin 1β, and intercellular cell adhesion molecule-1 levels were significantly decreased, the brain tissue nuclear factor kappa-B (NF-κB) p65 protein expression level was significantly decreased, and the brain tissue inhibitor of nuclear factor kappa-B α (IκBα) protein expression level was significantly increased (all $P < 0.05$). Above findings demonstrate that, the vitexin treatment may mitigate the CIRI in mice by decreasing the oxidative stress, reducing the inflammatory response and regulating the NF-κB/IκBα signaling pathway.**

RESUMEN. En este estudio se investigó la protección de la vitexina contra la lesión cerebral por isquemia-reperfusión (CIRI) en ratones y su mecanismo. Noventa ratones se dividieron aleatoriamente en grupos control, modelo y 10, 20 y 40 mg/kg de vitexina, 18 ratones en cada grupo. Los últimos tres grupos fueron administrados con 10, 20 y 40 mg/kg de vitexina, respectivamente, durante siete días. Luego, el modelo CIRI se estableció en los últimos cuatro grupos. Después de 24 h del modelado, en comparación con el grupo modelo, en grupos de vitexina de 20 y 40 mg/kg, el puntaje de déficit neurológico, el contenido de agua cerebral y el rango de infarto cerebral disminuyeron significativamente, los niveles de superóxido dismutasa y glutatión peroxidasa del tejido cerebral aumentaron significativamente, el nivel de malondialdehído del tejido cerebral disminuyó significativamente, el factor de necrosis tumoral del tejido cerebral α, la interleucina 1β y los niveles de la molécula 1 de adhesión celular intercelular disminuyeron significativamente, el nivel de expresión de la proteína p65 del factor nuclear del tejido cerebral kappa-B (NF-κB) disminuyó significativamente y el inhibidor del tejido cerebral del factor de expresión de la proteína del factor nuclear kappa-B α (IκBα) aumentó significativamente (todos $P < 0.05$). Los hallazgos anteriores demuestran que el tratamiento con vitexina puede mitigar el CIRI en ratones al disminuir el estrés oxidativo, reducir la respuesta inflamatoria y regular la vía de señalización NF-κB/IκBα.

KEY WORDS: cerebral ischemia-reperfusion injury, inflammatory response, mice, NF-κB, oxidative stress, vitexin.

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