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Dehydrocostunolide Attenuates Lipopolysaccharide-induced Hippocampal Inflammatory Injury in Mice

Niuniu WANG [1], Xinlin ZHAO [1], Lanqing MENG [1],
Jonnea Japhet TIBENDA [1], Xiaobo WANG [2] * & Qipeng ZHAO [1,3] *

[1] *School of Pharmacy, Ningxia Medical University, Yinchuan, China*

[2] *Meishan Hospital of Chengdu University of Traditional Chinese Medicine, Meishan, China*

[3] *Key Laboratory of Ningxia Ethnomedicine Modernization, Ministry of Education (Ningxia Medical University), Yinchuan, China*

SUMMARY. The aim was to study the protective effect of dehydroandrolactone on lipopolysaccharide-induced hippocampal neuronal inflammatory injury in mice. ICR mice were randomly divided into six groups: control group, model group (LPS), LPS+DHL high-dose (2mg/kg), LPS+DHL low-dose group (1mg/kg) and LPS+donepezil group (0.65mg/kg). The drug was administered by intraperitoneal injection. After dissecting and weighing the hippocampus, hematoxylin-eosin (HE) staining was used to observe the pathological changes of the hippocampus in the brain section; The expression of activation markers Iba-1 and TLR4 of microglia in the hippocampus was detected by RT-PCR, and the expression level of various inflammatory factors and proteins in the hippocampus was detected by Western blot. The results showed that LPS could activate microglia and release pro-inflammatory factors IL-1 β, IL-6, and TNF-a, which lead to inflammatory damage of the hippocampus and then lead to neurodegenerative disease in animals. However, dehydroandrolactone can inhibit the activation of microglia induced by LPS, reduce the release of pro-inflammatory factors, and improve the neuroinflammation of LPS mice.

RESUMEN. El objetivo era estudiar el efecto protector de la deshidroandrolactona sobre la lesión inflamatoria neuronal del hipocampo inducida por lipopolisacáridos en ratones. Los ratones ICR se dividieron aleatoriamente en seis grupos: grupo control, grupo modelo (LPS), grupo LPS+DHL en dosis alta (2 mg/kg), grupo LPS+DHL en dosis baja (1 mg/kg) y grupo LPS+donepezilo (0,65 mg). /kg). El fármaco se administró mediante inyección intraperitoneal. Después de diseccionar y pesar el hipocampo, se utilizó tinción con hematoxilina-eosina (HE) para observar los cambios patológicos del hipocampo en la sección del cerebro; La expresión de los marcadores de activación Iba-1 y TLR4 de la microglía en el hipocampo se detectó mediante RT-PCR, y el nivel de expresión de diversos factores inflamatorios y proteínas en el hipocampo se detectó mediante Western blot. Los resultados mostraron que el LPS podría activar la microglía y liberar factores proinflamatorios IL-1 β, IL-6 y TNF-a, que provocan daño inflamatorio del hipocampo y luego conducen a enfermedades neurodegenerativas en animales. Sin embargo, la dehidroandrolactona puede inhibir la activación de la microglía inducida por LPS, reducir la liberación de factores proinflamatorios y mejorar la neuroinflamación de ratones con LPS.

KEY WORDS: dehydroandrolactone, lipopolysaccharide neuritis, neurodegenerative disease

* Authors to whom correspondence should be addressed. *E-mails:* zhqp623@126.com (Q. Zhao), VitaDrwang@cdutcm.edu.cn (X. Wang)

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