

## Alleviating Effect of Salidroside on Cognitive Impairment due to Post-Traumatic Stress Disorder in Rats and the Mechanism

Fangfang TIAN<sup>1,2</sup>, Lin GUAN<sup>2</sup>, Lili YE<sup>2</sup>, Shuai ZHANG<sup>2</sup> & Peiyan SHAN<sup>3\*</sup>

<sup>1</sup> Qilu Hospital, Shandong University, Jinan 250012, China

<sup>2</sup> Department of Special Examination, Affiliated Hospital of Shandong University of Traditional Chinese Medicine, Jinan 250011, China

<sup>3</sup> Department of Geriatric, Qilu Hospital, Shandong University, Jinan 250012, China

**SUMMARY.** The effect of salidroside on cognitive impairment due to post-traumatic stress disorder (PTSD) in rats and the mechanism were investigated. The rats were randomized into control, model, and 20, 40, and 80 mg/kg salidroside groups. PTSD model was established in later four groups, and then the later three groups were treated with 20, 40, and 80 mg/kg salidroside groups for 2 weeks, respectively. After treatment, compared with model groups, in 40 and 80 mg/kg salidroside groups the open-field test, elevated cross-maze test, and Morris water maze test outcomes were significantly improved ( $P < 0.05$ ), the serum superoxide dismutase level was significantly increased ( $P < 0.05$ ), the malondialdehyde level was significantly decreased ( $P < 0.05$ ), the hippocampus 5-hydroxytryptamine level was significantly increased ( $P < 0.05$ ), the c-fos protein level was significantly decreased ( $P < 0.05$ ), the B-cell lymphoma-2 protein expression level was significantly increased ( $P < 0.05$ ), and the B-cell lymphoma-2 associated X protein expression level was significantly decreased ( $P < 0.05$ ). Salidroside can alleviate the cognitive impairment due to PTSD in rats, and the mechanisms may be related to its reducing oxidative stress, regulating 5-hydroxytryptamine and c-fos protein levels and antagonizing hippocampus neuron apoptosis.

**RESUMEN.** Se investigó el efecto del salidroside sobre el deterioro cognitivo debido al trastorno de estrés post-traumático (TEPT) en ratas y su mecanismo. Las ratas se asignaron al azar a los grupos control, modelo y de salidroside de 20, 40 y 80 mg/kg. El modelo de TEPT se estableció en cuatro grupos posteriores y luego los tres grupos posteriores se trataron con salidroside de 20, 40 y 80 mg/kg durante 2 semanas, respectivamente. Después del tratamiento, en comparación con los grupos modelo, en los grupos de salidroside de 40 y 80 mg/kg la prueba de campo abierto, la prueba de laberinto cruzado elevado y los resultados de la prueba de laberinto de agua de Morris mejoraron significativamente ( $P < 0.05$ ), el nivel de superóxido dismutasa en suero aumentó significativamente ( $P < 0.05$ ), el nivel de malondialdehído disminuyó significativamente ( $P < 0.05$ ), el nivel de 5-hidroxitriptamina del hipocampo aumentó significativamente ( $P < 0.05$ ), el nivel de proteína c-fos disminuyó significativamente ( $P < 0.05$ ), el nivel de expresión de la proteína del linfoma-2 de células B aumentó significativamente ( $P < 0.05$ ), y el nivel de expresión de la proteína X asociada al linfoma-2 de células B disminuyó significativamente ( $P < 0.05$ ). El salidroside puede aliviar el deterioro cognitivo debido al TEPT en ratas, y los mecanismos pueden estar relacionados con la reducción del estrés oxidativo, la regulación de los niveles de 5-hidroxitriptamina y proteína c-fos y la antagonización de la apoptosis de las neuronas del hipocampo.

**KEY WORDS:** apoptosis, c-fos, 5-hydroxytryptamine, oxidative stress, post-traumatic stress disorder, salidroside.

\* Author to whom correspondence should be addressed. E-mail: peiyanshan@126.com