The Toxic Influence of Paraquat on Hippocampal Neurogenesis in Adult Mice

Kaikai Li¹, Xinran Cheng¹, Jinhua Jiang², Jiutao Wang¹,³, Jiongfang Xie¹, Xinde Hu¹, Yingxue Huang¹, Lingzhen Song¹, Mengmeng Liu¹, Leiming Cai², Liezhong Chen², Shanting Zhao¹,*

¹ College of Veterinary Medicine, Northwest A&F University, Yangling, Shaanxi, 712100, People’s Republic of China.

² Zhejiang Academy of Agricultural Science, Hangzhou, Zhejiang, 310021, People’s Republic of China.

³ China-US (Henan) Hormel Cancer Institute, Zhengzhou, Henan, 450003, People’s Republic of China.

*Corresponding authors

Dr. Shanting Zhao; Tel: +86-029-8708-0136; E-mail: shantingzhao@hotmail.com

Abstract: Objective Paraquat, a fast-acting non-selective contact herbicide, is considered an etiological factor related to Parkinson’s disease. This study investigated its effects on hippocampal neurogenesis and cognition in adult mice as well as possible mechanisms for the effects. Methods We administered paraquat (1.25 mg/kg, intraperitoneal injection, i.p.) and an equal volume of normal saline for 3 weeks to adult male C57BL/6J mice. Results The results showed that hippocampus-dependent spatial learning and memory was significantly impaired in paraquat-treated mice. Moreover, paraquat administration inhibited the proliferation of neural progenitor cells, and impaired the survival and altered the fate decision of newly generated cells in the hippocampus. The expression levels of caspase-3 and glial fibrillary acidic protein were significantly higher in paraquat-treated mice than in control mice. Interestingly, paraquat reduced the phosphorylation of Akt, but did not affect the total amount of Akt. Conclusion In conclusion, our findings suggest that paraquat negatively affected adult hippocampal neurogenesis and cognition function.

Key Words: paraquat; learning; memory; neurogenesis; phosphorylation