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Jia Wei Kai Xin San Could Reduce the Deposition of Amyloid in the Brain Tissue of AD Rats

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Alzheimer's Disease (AD) is a group of illnesses with a relationship to aging and particular neuropathology that eventually causes mortality via cognitive decline. One of the key biomarkers in the development of AD is amyloid. Additionally, it has been demonstrated that amyloid deposition plays a significant role in the activation of NF-B signalling pathways and the subsequent mediating of the inflammatory response. Our team has been concentrating on Jiawei Kaixin San (JKS) for a long time. Based on the literature support and the findings of this investigation,observing the clinical effectiveness of JKS, understanding the mechanism ofJKS on enhancing brain health and reducing inflammation inAD rats were discussed.

The Liaoning University of Traditional Chinese Medicine in Shenyang, China, was the site of this investigation. Liaoning Changsheng Biotech Co., Ltd. donated 62 SPF-grade Sprague-Dawley rats, half of which were male and half of which were female (6-8 weeks old, weighing 20020 g). The rats were randomly assigned to one of four groups based on the model's evaluation results: model control group (MC group), high-dose group (HD group), medium-dose group (MD group), and low-dose group (LD group). The JKS was purchased from the Affiliated Hospital of Liaoning University of Traditional Chinese Medicine's Outpatient Pharmacy. Biyuntian Biotechnology Research Institute supplied the protein extraction kits and secondary antibodies for fluorescence labelling. The experimental data were examined using GraphPad Prism 5 and the SPSS 22.0 program.

Following the training of rats in each group, the outcomes of the space exploration experiment, location navigation experiment, and water maze training are displayed. Intrinsic immune cells in the central nervous system are called microglia, which are derived from monocytes in the bone marrow and have a high degree of flexibility and biological characteristics that are strikingly similar to those of macrophages. It has been noted that persistently activated microglia exacerbates the neuronal lesions of neurodegenerative disease and that the development of AD is closely related to the absence of beta-amyloid clearance, which in turn permits microglia to exert neurotoxicity and promote the progression of neurodegenerative disease. According to the findings of brain histopathology, the model group's neurons had damage.

Anti-inflammatory medication is one of the key treatment options for AD since chronic inflammation

has been linked to practically the entire course of the disease.The amount of TNF- was examined to ascertain JKS's ability to reduce inflammation in AD "Rat brain tissue and blood were examined for levels of IL-1$, IL-6, and IFN-. The findings revealed that the amounts of TNF- "Rats in the MC group had considerably greater levels of IL-1$, IL-6, and IFN- in their brain tissue and blood. These findings indicated that JKS significantly inhibits the abnormally activated TLR4/MyD88/NF-B signalling pathway in AD rats. This is likely related to JKS's inhibitory effect on the activation of microglia from the M0 type to the M1 type and its promotion of the polarization of microglia from the M0 type to the M2 type.

JOURNAL REFERENCE

Liu, Y., S.Y. Chen, J.J. Ma, Y.Q. Jiang, F. Gu and H.X. Zheng, 2022. Improving cognitive function through inhibiting the activation of microglia by Jia Wei Kai Xin San on Alzheimer's disease rats. Int. J. Pharmacol., 18: 947-961.

KEYWORDS

Jia Wei Kai Xin San, Alzheimer's disease, Microglia, TLR4/MYD88/NF-κB, signalling pathway