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Vesicular zinc depletion by acamprosate reduces traumatic brain injury-induced hippocampal neuron death

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Acamprosate or N-acetyl homotaurine is a N-methyl-D-aspartate (NMDA) receptor antagonist that is used as a pharmacological means of treatment for chronic alcohol dependence. Although the exact mechanism of acamprosate has not been clearly established, it appears to work by promoting a balance between the excitatory and inhibitory neurotransmitters, glutamate and gamma-aminobutyric acid (GABA), respectively. Several studies have demonstrated that acamprosate provides neuroprotection against cerebral ischemia-induced brain injury. However, there are no studies investigating the role of acamprosate on traumatic brain injury (TBI) induced neuronal death. In the present study, we sought to analyze the therapeutic potential of acamprosate to protect against neuronal death and other underlying pathogenic mechanisms that arise following TBI. Rats were given acamprosate (200 mg/kg) orally once per day for two weeks. Two week later, rats were subjected to a controlled cortical impact (CCI; 5 m/sec, 500 ms duration, 5 mm deformation) injury over the right parietal cortex. Histological analysis was performed at 3 or 24 hours, or 7 days after TBI. We found that acamprosate treatment for 2 weeks reduces levels of vesicular glutamate and zinc in the hippocampus. Consequently, this reduced vesicular glutamate and zinc level resulted in reduction of ROS production at 3 hours after TBI. When evaluated at 24 hours after TBI, acamprosate administration reduced the number of degenerating neurons, blood-brain barrier (BBB) disruption, leukocyte infiltration and dendritic loss. Acamprosate also reduced glial activation and neuronal loss at 7 days after TBI. In addition, acamprosate rescued TBI-induced cognitive dysfunction. The present study demonstrates that acamprosate attenuates TBI-induced brain damage by depletion of vesicular glutamate and zinc levels. Therefore, the present study suggests that acamprosate may have a high therapeutic potential for prevention of TBI-induced neuronal death.

Biography

Sang Won Suh has completed his PhD in 2001 from University of Texas, Medical Branch, Galveston and Postdoctoral studies from University of California, San Francisco, School of Medicine. He is a Professor and an Associate Dean of Hallym University, College of Medicine. He has published more than 90 papers in reputed journals and has been serving as an Editorial Board Member of MDPI journal.

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