Calcium influx into neurons triggers neuronal death during cerebral ischemia/reperfusion injury. Various calcium channels are involved in cerebral ischemia/reperfusion injury. Cav3.2 channel is a main subtype of T-type calcium channels. T-type calcium channel blockers, such as pimozide and mibefradil, have been shown to prevent cerebral ischemia/reperfusion injury-induced brain injury. However, the role of Cav3.2 channels in cerebral ischemia/reperfusion injury remains unclear. In vitro and in vivo models of cerebral ischemia/reperfusion injury were established using middle cerebral artery occlusion in mice and high glucose hypoxia/reoxygenation exposure in primary hippocampal neurons. The results showed that Cav3.2 expression was significantly upregulated in injured hippocampal tissue and primary hippocampal neurons. We further established a Cav3.2 gene-knockout mouse model of cerebral ischemia/reperfusion injury. Cav3.2 knockout markedly reduced infarct volume and brain water content, and alleviated neurological dysfunction after cerebral ischemia/reperfusion injury. Additionally, Cav3.2 knockout attenuated cerebral ischemia/reperfusion injury-induced oxidative stress, inflammatory response, and neuronal apoptosis. In the hippocampus of Cav3.2-knockout mice, calcineurin overexpression offset the beneficial effect of Cav3.2 knockout after cerebral ischemia/reperfusion injury. These findings suggest that the neuroprotective function of Cav3.2 knockout is mediated by calcineurin/nuclear factor of activated T cells 3 signaling. Findings from this study suggest that Cav3.2 could be a promising target for treatment of cerebral ischemia/reperfusion injury [1)]

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Dai F, Hu C, Li X, Zhang Z, Wang H, Zhou W, Wang J, Geng Q, Dong Y, Tang C. Cav3.2 channel regulates cerebral ischemia/reperfusion injury: a promising target for intervention. Neural Regen Res. 2024 Nov 1;19(11):2480-2487. doi: 10.4103/1673-5374.390966. Epub 2023 Dec 15. PMID: 38526284.