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Zinc/lipid/autophagy-mediated plasma membrane integrity signaling pathway is a new target for developmental convulsive brain injury

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In pediatric clinics, about 75% of neonatal hypoxic-ischemic encephalopathy (HIE) have convulsions, and some children develop epilepsy during adulthood. On the one hand, many of the first-line anticonvulsant drugs in the clinic can effectively control convulsions caused by HIE. On the other hand, these drugs can also cause adverse effects such as white matter damage. Therefore, there is an urgent need to reveal the mechanism of epileptogenesis, thus providing clues for finding new targets for anti-epileptic treatment. Our research initially revealed that zinc (Zn) ion metabolism dyshomeostasis is involved in the developmental seizure-induced regenerative sprouting of hippocampal mossy fibers, especially zinc transporters 1, 3 and ZIP6. Secondly, lipid metabolism molecules and autophagy signals participate in the repair of long-term hippocampal and cortical plasma membrane damage, as well as neurobehavioral and cognitive function protection following developmental seizures. We have found that lipid metabolism control methods such as the ketogenic diet, leptin and melatonin, or intraperitoneal injection of autophagy inhibitors (3-MA, CBI, E-64d) immediately after seizures, can inhibit hippocampal sprouting and neurobehavioral damage. Our preliminary results indicate that the regulation of plasma membrane integrity mediated by zinc/lipid/autophagy signaling may be involved in the pathophysiological process of epileptogenesis, which may be a new target for repair of neuronal membrane damage after developmental seizures.

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