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Protective roles of selenium and zinc against postnatal protein-undernutrition-induced alterations in Ca(2+)-homeostasis leading to cognitive deficits in Wistar rats

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Abstract

Postnatal protein-undernutrition impacts on mental development and cognition in children and can lead to problem with attention and unresponsiveness which compromise children's ability to learn. These behavioral disorders might be due to alteration in calcium homeostasis as calcium plays critical roles in fundamental functions of neuron. The role of low protein diet as well as Se and Zn supplementation on intracellular calcium concentration ([Ca(2+)]i), Ca(2+)-ATPase, Na(+)-K(+)-ATPase, calpain and caspase-3 activities from rat cortex and cerebellum were investigated. Well-fed (WF) and low protein diet-fed (LPDF) rats were given diets containing 16% and 5% casein, respectively, for a period of 10 weeks. Then, the rats were supplemented with Se and Zn at a concentration of 0.15 mgL(-1) and 227 mgL(-1), respectively, in drinking water for 3 weeks. The results obtained from the study showed a significant increase

in [Ca(2+)]i; calpain and caspase-3 activities as well as increase transfer latency in water maze study and reductions in Ca(2+)-ATPase and Na(+)-K(+)-ATPase activities for LPDF rats compared to WF rats. Se and Zn supplementation to LPDF rats reversed the elevation in [Ca(2+)]i, calpain and caspase-3 activities and restored the cognitive deficits and the activities of Ca(2+)-ATPase and Na(+)-K(+)-ATPase. Conclusively, protein-undernutrition results in the accumulation of synaptosomal calcium and inhibition of calcium transporters presumably via free radical generations and results in cognitive impairment which also probably results from neuronal death in rats through calpain activation and the caspase cascade mechanisms. However, Se and Zn supplementations ameliorated the anomalies observed.

Keywords: Calcium; Low protein; Neurodegeneration; Oxidative stress; Synaptosome.