Sodium Selenite protects against 3-nitropropionic acid-induced toxicity in cultured primary cortical neurons


Introduction: Huntington’s disease (HD) is a progressive neurodegenerative disorder histologically characterized by death of striatal and cortical neurons. The neurodegeneration and cell death observed in HD patients have been related with mitochondrial dysfunction and oxidative damage. 3-Nitropropionic acid (3-NP), an inhibitor of the mitochondrial enzyme Succinate Dehydrogenase, induces deficit in energy metabolism, enhances reactive oxygen species (ROS) generation, oxidative stress, and neuronal death. Sodium selenite (Na₂SeO₃) has been used as dietary source of selenium, which display a crucial role in the active center of several antioxidant enzymes. **Objective:** The present study evaluated the potential protective effects of sodium selenite against the neurotoxicity induced by 3-NP in primary cultures of cortical neurons. **Material and Methods:** Primary neurons were prepared from cortex of Swiss mice brain at the gestation day 18. Neurons were pretreated with 30 nM Se for six days and/or 3-NP (0.1-10 mM) for 48 hours. After the incubation period, cell viability and oxidative stress-related parameters [ROS generation, reduced glutathione (GSH) and oxidized glutathione (GSSG) levels, and glutathione peroxidase (GPx) activity] were evaluated. **Results and Discussion:** 3-NP significantly decreased cell viability, as well as, increased ROS generation. Furthermore, 3-NP was able to induce a significant reduction in GSH levels and an increase in GSSG levels in the primary neurons. Sodium selenite protected the neurons against the reduction of cell viability, as well as decreased the ROS generation induce by 3-NP. Moreover, GPx activity was significantly increased by the treatment with sodium selenite. **Conclusion:** These findings show that sodium selenite exerts an in vitro protective action against the neurotoxicity induce by 3-NP. These data suggest that the protective effect of sodium selenite may be related to the ability of this compound to increase the GPx activity, a crucial enzyme for the detoxification of peroxides in the central neurons system.

**Keywords:** Huntington’s disease, 3-nitropropionic acid, oxidative stress, sodium selenite, primary culture.

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