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Protective effects of selenium on cadmium neurotoxicity

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Prolonged exposure to Cadmium may cause serious toxic effects due to its accumulation on both central and peripheral nervous systems. Cadmium could be uptaken from the nasal mucosa via the olfactory pathways and gain direct access into the nervous system circumventing the blood-brain barrier. However, mechanisms underlying the cadmium uptake and neurotoxicity remain not completely understood. Oxidative damage, interference with calcium-, copper- and zinc-dependent processes, dysregulation of cell repair mechanisms, estrogen-like effects, and epigenetic modifications may be considered mechanisms for cadmium-induced neurotoxicity [1].

Our previous data demonstrated that zinc chloride counteracts the toxic effects of cadmium chloride ($CdCl_2$) on human neurons in vitro [2]. Thus, in this study we evaluate the efficacy of Sodium Selenite (Na_2SeO_3) in preventing and/or counteracting the damages induced by exposure to cadmium chloride on a human neuronal cell line.

In this study we treated SH-SY5Y human neurons with different sub-toxic concentrations of $CdCl_2$ for 24 h with and without a 24 h pre-treatment with Na_2SeO_3. Cell viability, morphological modifications, and protein expression of specific neuronal plasticity and apoptosis (Gap43 and caspase 3) markers were evaluated.

Our results suggest that toxic effects of $CdCl_2$ can be prevented and reverted by Na_2SeO_3 suggesting a role for selenium compounds in protecting neuronal cells and rebuilding the complex network connections.

References

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Keywords

Selenium; cadmium; neurons.