

Short-term exposure to nickel alters the adult rat brain antioxidant status and the activities of crucial membrane-bound enzymes: neuroprotection by L-cysteine.

[Liapi C¹](#), [Zarros A](#), [Theocharis S](#), [Voumvourakis K](#), [Anifantaki E](#), [Gkrouzman E](#), [Mellios Z](#), [Skandali N](#), [Al-Humadi H](#), [Tsakiris S](#).

Author information

- ¹Department of Pharmacology, Medical School, National and Kapodistrian University of Athens, Athens, Greece.

Abstract

Nickel (Ni) is an environmental pollutant towards which human exposure can be both occupational (mainly through inhalation) and dietary (through water and food chain-induced bioaccumulation). The aim of this study was to investigate the effects of short-term Ni-administration (as NiCl₂, 13 mg/kg) on the adult rat whole brain total antioxidant status (TAS) and the activities of acetylcholinesterase (AChE), Na(+),K(+)-ATPase, and Mg(2+)-ATPase; in addition, the potential effect of the co-administration of the antioxidant L-cysteine (Cys, 7 mg/kg) on the above parameters was studied. Twenty-eight male Wistar rats were divided into four groups: A (saline-treated control), B (Ni), C (Cys), and D (Ni and Cys). All rats were treated once daily with intraperitoneal injections of the tested compounds, for 1-week. Rats were sacrificed by decapitation and the above-mentioned parameters were measured spectrophotometrically. Rats treated with Ni exhibited a significant reduction in brain TAS (-47%, $p < 0.001$, BvsA) that was efficiently limited by the co-administration of Cys (-4%, $p > 0.05$, DvsA; +83%, $p < 0.001$, DvsB), while Cys (group C) had no effect on TAS. The rat brain AChE activity was found significantly increased by both Ni (+30%, $p < 0.001$, BvsA) and Cys (+62%, $p < 0.001$, CvsA), while it tended to adjust to control levels by the co-administration of Ni and Cys (+13%, $p < 0.001$, DvsA; -13%, $p < 0.001$, DvsB). The activity of rat brain Na(+),K(+)-ATPase was significantly decreased by Ni-administration (-49%, $p < 0.001$, BvsA), while Cys supplementation could not reverse this decrease (-44%, $p < 0.001$, DvsA). The activity of Mg(2+)-ATPase was not affected by Ni-administration (-3%, $p > 0.05$, BvsA), but was significantly reduced when combined with Cys administration (-17%, $p < 0.001$, DvsA). The above findings suggest that Ni short-term in vivo administration causes a statistically significant decrease in the rat brain TAS and an increase in AChE activity. Both effects can be, partially or totally, reversed to control levels by Cys co-administration; Cys could thus be considered (for future applications) as a potential neuroprotective agent against chronic exposure to Ni. The activity of Na(+),K(+)-ATPase that was inhibited by Ni, could not be reversed by Cys co-administration. The matter requires further investigation in order to fully elucidate the spectrum of the neurotoxic effects of Ni.