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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 C[1], Zarros A, Theocharis S, Voumvourakis K, Anifantaki F, Gkrouzman E, Mellios Z, Skandali N, Al-Humadi H, Tsakiris S.

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 NiCl(2), 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.

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