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The neuroprotective role of L-cysteine towards the effects of short-term exposure to lanthanum on the adult rat brain antioxidant status and the activities of acetylcholinesterase, (Na+,K+)- and Mg2+-ATPase.

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Abstract

Lanthanum (La) is a rare earth element that is widely used for industrial, medical and agricultural purposes. Its neurotoxic effects are linked to its physical and chemical properties and its interaction with certain trace elements and membrane-bound enzymes. The aim of this study was to investigate the effects of short-term La-administration (as LaCl(3), 53 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, as well as the potential effect of the co-administration of the antioxidant L: -cysteine (Cys, 7 mg/kg) on the above parameters. Twenty-eight male Wistar rats were divided into four groups: A (saline-treated control), B (La), C (Cys),and D (La 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 La exhibited a significant reduction in brain TAS (-36%, $P < 0.001$, BvsA), that was partially limited by the co-administration of Cys (-13%, $P < 0.01$, DvsA), while Cys (group C) had no effect on TAS. The rat brain AChE activity was found significantly increased by both La (+23%, $P < 0.001$, BvsA) and Cys (+59%, $P < 0.001$, CvsA), while it was adjusted to control levels by the co-administration of La and Cys. The activity of rat brain Na(+),K(+)-ATPase was significantly decreased by La-administration (-28%, $P < 0.001$, BvsA), while Cys supplementation could not reverse this decrease. The activity of Mg(2+)-ATPase exhibited a slight but statistically significant reduction due to La (-8%, $P < 0.01$, BvsA), that was further reduced by Cys co-administration (-25%, $P < 0.001$, DvsA). The above findings suggest that La 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 into control levels by Cys co-administration, which could thus be considered for future applications as a neuroprotective agent against chronic exposure to La. The activities of Na(+),K(+)- and Mg(2+)-ATPase that were inhibited by La, could not be reversed by Cys co-administration. A role for the already reported concentration-dependent interaction of La with Ca-binding sites (such as Ca(2+)-ATPase) might be considered for certain of the above phenomena.

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