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Effects of short-term exposure to manganese on the adult rat brain antioxidant status and the activities of acetylcholinesterase, (Na,K)-ATPase and Mg-ATPase: modulation by L-cysteine.

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Abstract

Manganese (Mn) is an essential metalloenzyme component that in high doses can exert serious oxidative and neurotoxic effects. The aim of this study was to investigate the potential effect of the antioxidant L-cysteine (Cys, 7 mg/kg) on the adult rat brain total antioxidant status (TAS) and the activities of acetylcholinesterase (AChE), Na+,K+-ATPase and Mg2+-ATPase induced by short-term Mn administration (as Mn chloride, 50 mg/kg). Twenty-eight male Wistar rats were divided into four groups: A (saline-treated control), B (Mn), C (Cys) and D (Mn and Cys). All rats were treated once daily, for 1 week with intraperitoneal injections of the tested compounds. Rats were killed by decapitation and mentioned parameters were measured spectrophotometrically. Rats treated with Mn exhibited a significant reduction in brain TAS (-39%, $P < 0.001$, B versus A) that was partially reversed by Cys co-administration (-13%, $P < 0.01$, D versus A), while Cys (group C) had no effect on TAS. The rat brain AChE activity was found significantly increased by both Mn (+21%, $P < 0.001$, B versus A) and Cys (+61%, $P < 0.001$, C versus A), while it was adjusted into the control levels by the co-administration of Mn and Cys. The activity of rat brain Na+,K+-ATPase was not affected by Mn administration, while Mg2+-ATPase exhibited a slight but statistically significant reduction in its activity (-9%, $P < 0.01$, B versus A) due to Mn, which was further reduced by Cys co-administration. The above findings suggest that short-term Mn 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 the control levels by Cys co-administration (which could thus be considered for future applications as a neuroprotective agent against chronic exposure to Mn and the treatment of manganism). The activity of Na+,K+-ATPase is not affected by Mn, while Mg2+-ATPase activity is slightly (but significantly) inhibited by Mn, possibly due to Mg replacement.