1. Metab Brain Dis. 2009 Sep;24(3):441-51. doi: 10.1007/s11011-009-9147-4. Epub 2009 Aug 21.

Combined thirty-day exposure to thioacetamide and choline-deprivation alters serum antioxidant status and crucial brain enzyme activities in adult rats.

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. **Abstract**

Choline (Ch) is an essential nutrient that seems to be involved in a wide variety of metabolic reactions and functions that affect the nervous system, while thioacetamide (TAA) is a well-known hepatotoxic agent. The induction of prolonged Ch-deprivation (CD) in rats receiving TAA (through the drinking water) provides an experimental model of mild progressive hepatotoxicity that could simulate commonly-presented cases in clinical practice. In this respect, the aim of this study was to investigate the effects of a 30-day dietary CD and/or TAA administration (300 mg/L of drinking water) on the serum total antioxidant status (TAS) and the activities of brain acetylcholinesterase (AChE), Na(+),K(+)- ATPase and Mg(2+)-ATPase of adult rats. Twenty male Wistar rats were divided into four groups: A (control), B (CD), C (TAA), D (CD+TAA). Dietary CD was provoked through the administration of Ch-deficient diet. Rats were sacrificed by decapitation at the end of the 30-day experimental period and whole brain enzymes were determined spectrophotometrically. Serum TAS was found significantly lowered by CD (-11% vs Control, $p < 0.01$) and CD+TAA administration (-19% vs Control, $p < 0.001$), but was not significantly altered due to TAA administration. The rat brain AChE activity was found significantly increased by TAA administration (+11% vs Control, $p < 0.01$), as well as by CD+TAA administration (+14% vs Control, $p < 0.01$). However, AChE was not found to be significantly altered by the 30-day dietary CD. On the other hand, CD caused a significant increase in brain Na(+),K(+)-ATPase activity (+16% vs Control, $p < 0.05$) and had no significant effect on Mg(2+)-ATPase. Exposure to TAA had no significant effect on Na(+),K(+)-ATPase, but inhibited Mg(2+)-ATPase (-20% vs Control, $p < 0.05$). When administered to CD rats, TAA caused a significant decrease in Na(+),K(+)-ATPase activity (-41% vs Control, $p < 0.001$), but Mg(2+)-ATPase activity was maintained into control levels. Our data revealed that an adult-onset 30-day dietary-induced CD had no effect on AChE activity. Treatment with TAA not only reversed the stimulatory effect of CD on adult rat brain Na(+),K(+)-ATPase, but caused a dramatic decrease in its activity (-41%). Previous studies have linked this inhibition with metabolic phenomena related to TAA-induced fulminant hepatic failure and encephalopathy. Our data suggest that CD (at least under the examined 30-day period) is an unfavorable background for the effect of TAA-induced hepatic damage on

Na(+),K(+)-ATPase activity (an enzyme involved in neuronal excitability, metabolic energy production and neurotransmission).

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