

Acetaldehyde metabolism by liver mitochondrial ALDH from UChA and UChB rats: Effect of inhibitors

Tampier, Lutske

Sánchez, Eliana

Quintanilla, María Elena

We have observed that blood acetaldehyde (AcH) levels after an ethanol dose were significantly higher in disulfiram-pre-treated UChA (low ethanol consumer) than in UChB (high ethanol consumer) rats. In order to explore these results further, we studied the effect of disulfiram (300 mg/kg i.p.) and chlorpropamide (80 mg/kg i.p.) pre-treatment on blood AcH levels after oral ethanol (60 mmol/kg) and on AcH metabolism by liver mitochondrial aldehyde(s) dehydrogenase(s) from UChA and UChB rats. AcH metabolism by liver mitochondrial aldehyde dehydrogenase (ALDH) was studied by following AcH disappearance rate and the formation of NADH at 340 nm in the incubation medium. The results showed that chlorpropamide, like disulfiram, produced a higher blood AcH level consistent with a greater inhibition of the low-K_m mitochondrial ALDH in the UChA rats than in the UChB rats. These drugs did not inhibit the high K_m mitochondrial ALDH. Kinetic studies of mitochondrial ALDH show that low-K_m mitochondr