Ethanol intake: Effect on liver and brain mitochondrial function and acetaldehyde oxidation

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The effect of a chronic ethanol consumption by forcing rats to drink a 20% v/v ethanol solution as sole drinking fluid, for 3 months, was evaluated on: liver and brain mitochondrial function, the capacity of isolated mitochondria to oxidize acetaldehyde, as well as on the low Km mitochondrial AIDH activity, in rats. The O2 uptake by liver and brain mitochondria in the presence of glutamate + malate, succinate or ascorbate + TMPD, was measured polarographically with a Clark electrode. Acetaldehyde oxidation was measured by the disappearance rate in presence of the intact or disrupted mitochondria (AIDH activity) by gas chromatography. Results indicate that an ethanol intake of 11 g/kg b.wt. per day produce a significant reduction of the liver mitochondrial respiration tested with all the substrates used, including acetaldehyde. In contrast, the activity of AIDH in disrupted mitochondria remained unchanged. These results are in accord with the idea that a progressive deterioration of liv