ETHANOL AS METABOLIC PRECURSOR AND FUEL FOR THE BRAIN
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Background: Previous studies in humans and rats have shown ethanol (Etoh) consumption to increase brain glucose utilization, and ethanol, which is converted to acetate (Ac), increases brain acetate uptake. We have seen that in humans, Ac transport and oxidation are faster in heavy drinkers than in light drinkers. It is unknown if Ac only or if Etoh also provides energy to the brain.

Aims: To examine the effects of acetate on the subjective response to alcohol and energy metabolism of acetate by examining the differences of blood acetate levels between subjects with active and inactive ALDH2. Moreover, if acetate levels differ between ALDH2 genotypes, it should also have effects on acetate levels.

Method: We examined blood acetate levels and subjective response to alcohol and compared them between subjects with and without ALDH2*2 by employing the alcoholic clamp method to administer alcohol.

Results: Subjects with active ALDH2 had significantly higher levels of acetate than those with inactive ALDH2. During maintaining BAC at 50 ± 5 mg/dL, while blood acetate levels were positively correlated with sedative effects on BAC, acetate was negatively correlated with stimulant effects on BAC even after controlling for blood acetate level.

Conclusion: Subjects with active ALDH2 had significantly higher acetate levels compared to those with inactive ALDH2. Acetate was suggested to have depressive effects on subjective response to alcohol and it is supposed that higher levels of subjective response to alcohol observed in subjects with active ALDH2 are not only due to acetate but might be partially mediated by lower levels of alcohol but further studies were warranted to draw a firm conclusion.