

Characteristics of Acetaldehyde Oxidation in Rat Liver Mitochondria*

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Rat liver mitochondria oxidized acetaldehyde (180 μ M) at the rate of approximately 1 nmol/min/mg of protein. Indeed, our group reported before that alcoholics display a 60% higher blood acetaldehyde level than non-alcoholics given the same dose of ethanol (28). High levels of acetaldehyde in the The ADP/O ratio (2.6) was similar to that of intact mitochondria. Also stimulated the acetaldehyde metabolism was virtually abolished by cyanide and antimycin A. Acetaldehyde oxidation is linked to the mitochondrial respiratory chain and coupled with mitochondrial oxidative phosphorylation. Indeed, little acetaldehyde was metabolized by disrupted mitochondria. In the disrupted mitochondria, addition of NAD⁺, suggesting that acetaldehyde oxidation in intact mitochondria is coupled with NAD⁺ reduction. acetaldehyde levels even further. Since recent observations suggest that acetaldehyde may contribute to the development of ethanol dependence (29) as well as heart (30) and liver (22) damage commonly found in chronic alcoholics, the reduced capacity of liver mitochondria of ethanol-fed rats to oxidize acetaldehyde observed in this study may result in an exacerbation of the toxicity of ethanol in the alcoholic. Chronic feeding of ethanol significantly increased blood acetaldehyde. This was associated with a significant reduction of the mitochondrial respiration. By contrast, the activity of aldehyde dehydrogenase in disrupted mitochondria remained unchanged.