

EFFECT OF CHRONIC ALCOHOL CONSUMPTION ON ETHANOL AND ACETALDEHYDE METABOLISM*

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CHRONIC ALCOHOL CONSUMPTION AND METABOLISM

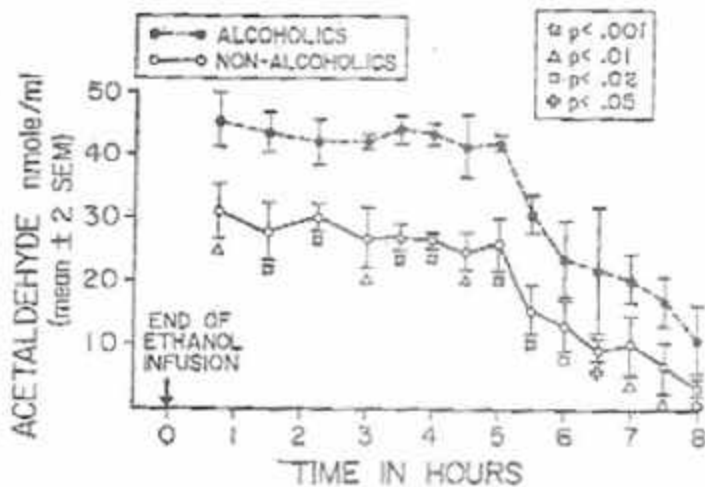


Figure 7.
Comparison of blood acetaldehyde levels of alcoholic and non-alcoholic subjects following i.v. alcohol infusion.

Moreover, the plateau level of acetaldehyde was significantly higher in alcoholics (42.7 ± 1.2 nmole/ml) than in non-alcoholics (26.5 ± 1.5) (Figure 7).

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faster than that of non-alcoholics. This "induction" may be the source of greater production of the toxic metabolite (acetaldehyde) resulting in a "vicious cycle" (Figure 8): acetaldehyde causes mitochondrial dysfunction which in turn promotes higher acetaldehyde levels; this circular process could result in progressive liver damage.

generated in vivo. Finally, myocardial protein synthesis was impaired by acetaldehyde (Schreiber et al., 1972, 1974) at concentrations comparable to those found in our study; this effect might contribute to the development of alcoholic cardiomyopathy. Thus, the high levels of blood acetaldehyde found at high alcohol concentrations may have fundamental pathogenetic consequences in the alcoholic since he has significantly higher blood acetaldehyde levels than the non-alcoholic. It is conceivable that the decreased capacity of injured mitochondria to dispose of the toxic acetaldehyde may initiate a self sustaining mechanism of liver injury by promoting the accumulation of acetaldehyde which aggravates the mitochondrial dysfunction.

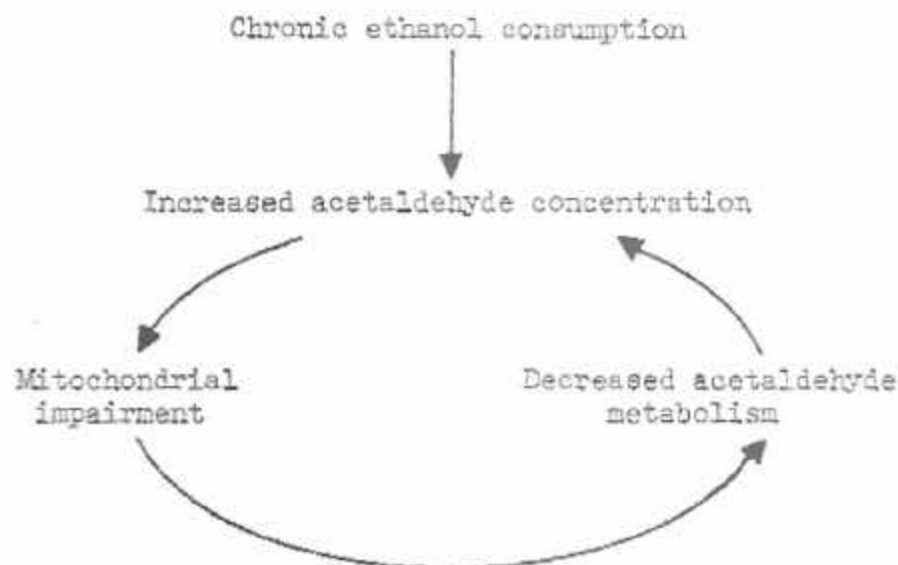


Figure 8.
Possible relationship between ethanol metabolism, altered hepatic acetaldehyde levels and mitochondrial impairment.