The effects of alcohol on metabolism of liver - lab report example

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The effects of alcohol on metabolism of liver It is an established fact that the liver of a chronic alcoholic is under much more metabolic stress as compared to a person practicing total abstinence and affects both body weight and energy metabolism. Alcoholism claims 100, 000 lives annually in the United States alone (1, 2).

Alcohol is exclusively consumed as ethanol which has a substantial energy value (7. 1 kcal/g) and represents 50% of the total dietary energy intake in a heavy drinker which results in displacement of normal nutrients in diet, impairs their utilization leading to secondary malnutrition (4).

The primary metabolic pathway of alcohol in the liver is by oxidation through the alcohol dehydrogenase pathway with the resultant production of acetaldehyde. Redox homeostasis is compromised resulting in metabolic disorders like hyperuricemia, hyperlipemia and a rise in HDL (High Density Lipoprotein) An increase in NADH levels consequent to oxidation promotes fatty acid synthesis with the resultant hepatic fat accumulation.

Acetaldehyde produced itself has detrimental effects by decreasing the activity of key enzymes and reducing oxygen utilization in mitochondria (2, 4).

Experiments in the past have demonstrated increase in the rate of oxygen consumption in hepatocytes and decrease in ATP as a result of increases sodium pump activity (8, 6).

Experiments in rats and sub human primates suggest that chronic alcohol exposure to the liver results in an increase in ethanol metabolism involving the alcohol dehydrogenase pathway resulting in increased oxygen uptake (3)

and results in a fatty liver despite an adequate nutritional intake exclusively due to the action of alcohol (5, 9). Synthesis of ATP is suppressed due to due to decrease in both mitochondrial as well as glycolytic activities as a result of oxygen deficiency (6).

Cirrhosis is a common sequel to chronic alcohol consumption with abundance of fibrous tissue in the liver, whose chief component is collagen indicating a direct effect of alcohol consumption on hepatic collagen metabolism. Increased synthesis of collagen triggered by the oxidation of alcohol in the hepatocytes which results in elevated lactate concentration which enhances the activity of collagen proline hydroxylase enzyme. (7).

Experimental and clinical studies over the last four decades have indicated the alcohol induced oxidative stress and the direct effects of acetaldehyde as the main culprits for liver disease (4). Strategies to counter them have been experimentally proven to reduce liver damage.

The objective of this experiment was to estimate the effect on metabolic rate in liver by measuring the oxygen consumption.

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