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ALCOHOL, WHY SOME PEOPLE CAN DRINK MORE THAN OTHERS

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The effects of alcohol ingestion vary considerably from one individual to the next. Some people become somewhat tipsy after drinking only an ounce or less while others can consume several ounces with few apparent effects.

A study published in the January 11th issue of the ***New England Journal of Medicine*** discussed the reasons for the different effects of alcohol consumption between men and women. In the past the explanation of why women could not drink as much as men was simply that women were physically smaller than men. However researchers have now found that the difference is due to the greater absorption of alcohol from the stomach in women compared to men. Women were found to absorb about one-third more alcohol into their blood stream than men of the same weight and height who consumed an equal amount of alcohol.

The liver is a major organ that degrades or oxidizes alcohol from the blood stream. The enzyme alcohol dehydrogenase, present in the liver in large amounts, is the principle enzyme responsible for this detoxifying process. This enzyme has also been found to be actively present in the lining of the stomach, which can therefore act as a protective barrier to alcohol absorption. In volunteer subjects researchers found the activity of alcohol dehydrogenase to be 70-80% higher in the stomach lining of men as compared to the stomach lining of women subjects.

The enzymes alcohol dehydrogenase, the catalase system, and the microsomal ethanol-oxidizing systems are involved in the oxidation of alcohol. The major enzyme is alcohol dehydrogenase, which has an absolute requirement for the mineral zinc. Without adequate zinc these enzyme functions are lessened, and their activity not only becomes less active in the stomach lining but would also decrease the ability of the liver to degrade alcohol. Our studies indicate that the probable mechanism for the decreased effect of this enzyme in women is that generally speaking, women have a lowered tissue zinc-to-copper ratio. However, anyone with this type of trace element imbalance would experience a similar sensitivity to alcohol.

Often individuals may notice that they cannot handle alcohol as well following an infection such as mononucleosis or viral hepatitis since these conditions can cause an increase in copper retention relative to zinc. Any factor that adversely affects zinc status such as a vitamin deficiency or toxic metal accumulation affects the body's ability to degrade alcohol.

Dr. J.J. Smith proposed in the "***New York State Journal of Medicine"*** in 1950 that adrenal cortical insufficiency produces a physiological and psychological basis for the craving of alcohol. However, alcoholism is not always associated with increased sensitivity. Past hair tissue mineral analysis studies have shown that fast metabolic types with hyperadrenia can

handle alcohol much better than slow metabolic types. Their rapid metabolic rate stimulates many of the cellular enzyme processes thereby clearing alcohol more readily from the blood stream. It is common to see alcohol addiction in fast metabolic types who apparently crave the alcohol in order to help maintain their high metabolic rate and energy levels yet they do not necessarily become intoxicated. Hypoadrenia on the other hand can certainly contribute to the desire for alcohol to gain quick energy and to relieve depression, etc. However, individuals with hypoadrenia, or slow metabolic types, are generally more susceptible to the effects of alcohol. This is largely due to the various cellular enzymes that are linked to the metabolism of alcohol.

Hormones will affect the cellular ratio of linking enzymes to alcohol dehydrogenase, particularly the NAD/NADPH ratio. The high adrenal and thyroid activity in the fast metabolizer stimulates liver function, while liver function is more lethargic in the slow metabolizer.

Adequate nutritional status can also protect the individual from the adverse effects of chronic alcohol ingestion, such as cirrhosis, fatty degeneration of the liver, and iron toxicity.