

Preventing or Slowing Liver Damage in Drinkers: Use of Lecithin, SAM-e and Glutathione

By

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Alcohol-induced disease is fairly commonplace among individuals who abuse alcohol. Fibrosis and cirrhosis by-and-large head the list. In both cases, abnormal changes take place in liver tissue that compromise this vital organ's ability to function optimally. For many people who drink, a doctor's finding of liver pathology (disease) is sufficient to get them to either curtail their drinking or abstain altogether (Whether on a temporary or permanent basis).

For others, however, a diagnosis of cirrhosis or other alcohol-induced disease does not put "the fear of God in 'em" with sufficient force for them to overcome the craving to elbow bend. What follows is information concerning a "dose of prevention" that may just spare the heavy drinker some grief down the line (Albeit this is by no means an argument to continue excessive drinking!)

This said, I would urge consistent moderate drinkers to pay attention to what I am about to share as well, as they too have the potential to experience liver damage over time that may not be readily repaired (and which could set the stage for problems later on in life).

A Dose of Prevention

In at least two separate animal studies carried out during the past fifteen years, a natural compound called lecithin protected animals who consumed booze in great quantities. Indeed, the animals were protected from developing many of the pathologic abnormalities common when alcohol is abused. Here are the details of this very compelling body of research:

In a study involving rats, 28 male littermates were pair-fed liquid diets containing 36% of energy either as ethanol (alcohol) or as additional carbohydrates for 21 days. Half of these rodents were given polyenylphosphatidylcholine (A component of lecithin) at 3 grams per liter of their food substrate (Liquid meals). The other group was given safflower oil (3 grams/liter) and choline (A chemical part of lecithin) as a bitartrate salt. The polyenylphosphatidylcholine (PPC) did not influence diet intake or alcohol consumption, but the booze-induced liver enlargement and accumulation of specific fats (lipids - triglycerides and cholesterol esters) and proteins were about half those in rats not given PPC. In rats that consumed PPC, post-eating rise in serum lipids was lower than was true of their littermates who had no PPC. The researchers, who worked at the Alcohol Research and Treatment Center, Bronx Veteran Affairs Medical Center (New York City), concluded that "These beneficial effects of PPC at the initial stages of alcoholic liver injury may prevent or delay the progression to more advanced forms of alcoholic liver

disease" (1).

In a separate 10 year-long study involving baboons, also carried out at the Bronx Veteran Affairs Center (Section of Liver Disease and Nutrition), the suggested benefits of lecithin ingestion were even more encouraging.

In the study, twelve baboons (eight females, four males) were fed a liquid diet rich in alcohol supplemented with polyunsaturated lecithin (50% of total energy) or isocaloric carbohydrate. This group was compared with another group of eighteen baboons who were fed an equivalent diet (with or without alcohol), but without of lecithin. (2) Both groups developed increases in specific lipids (associated with alcohol use), but there were significant differences in the degree of liver injury (fibrosis) seen. For one thing, septal fibrosis (with cirrhosis in two animals) and transformation of their fat cells (lipocytes) into transitional cells developed in seven of the nine baboons fed the regular diet with alcohol. Septal fibrosis did not develop in any of the animals fed lecithin! In fact, they did not progress beyond the stage of perivenular (area around veins) fibrosis and had significantly lesser activation of fat cells to transitional cells. (3) The clincher came when the scientists took three of the lecithin-consuming animals off same, but maintained their customary diet and alcohol mix. They very rapidly progressed to cirrhosis, accompanied by an increased transformation of their fat cells to transitional cells!

The fact these researchers found that choline exerted no protective effect in animals ingesting large quantities of alcohol led them to conclude that the polyunsaturated phospholipids might be responsible for the protective effect. This is underscored by the rodent study cited above, in which choline did not protect the animals from alcohol-induced liver damage, whereas PPC (Lecithin component) did. (4)

Baboon livers are remarkably similar to human livers (This is one reason an attempt was made many years back to transplant baboon livers into humans whose livers had failed). Given this, it seems logical that lecithin should provide human drinkers at least some of the benefits seen in the baboons. Accordingly, for those who drink -- especially heavily -- lecithin may be an invaluable form of health insurance. It is also easy on the pocketbook, being sold "dirt cheap" in health food and grocery stores plus pharmacies across the land.

In addition to lecithin, there are other compounds that if taken by drinkers should help reduce the damage to their livers.

For example, in alcoholics the conversion of the amino acid methione to S-adenosylmethionine (SAM-e) is significantly reduced. In baboon models of alcoholism, the animals experienced alcoholic cirrhosis that was opposed by replenishing SAM-e. Other lines of research indicate that bolstering SAM-e levels in human alcoholics decreases mortality, and offsets oxidative stress resulting from alcohol and alcohol byproduct induction of a liver detoxification enzyme designated cytochrome P4502E1 (CYP2E1).

SAM-e can readily be replenished by taking an oral form that is bioavailable (Not all forms are!)

As the liver is a prime site for manufacture of one of the bodies most powerful antioxidants, glutathione, it logically follows that heavy use of alcohol would impact synthesis of this compound. And indeed, at least one animal study indicates this to be the case.

Fortunately, glutathione can be orally supplemented. However, not just any form of glutathione will work, as most forms are broken down in the gut and thus never reach the bloodstream intact. There is patented forms that resist breakdown until the glutathione has reached tissues throughout the body.

Of course, when it comes to drinking to excess – be it binge drinking or habitual heavy imbibing -- curtailing or quitting is ideal. Those caught up in this sort of drinking pattern should seek professional help. But for addicts, alcohol abusers, and just plain ole social drinkers, offsetting some of the injury boozing does to the body (liver especially) is a prudent measure. The judicious use of lecithin, SAM-e and the right form of glutathione should readily help in this regard.

If only a small fraction of those who imbibe heavily take lecithin, SAM-e and glutathione benefit in terms of staving off the many diseases linked to alcohol abuse, the savings in terms of payouts for medical care and lost time from work alone could prove very substantial! This is a blessing to both the individual drinker and society at large.

References

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