"Care for a drink?"

A Surprising Mix Of Medical Research On Alcohol

Complied by Verne Varona

"When I read about the evils of drinking, I gave up reading."

— Henny Youngman

"To your health!"
"A Votre Sante1!"
"Salaud!"
"Cheers!"

These common drink toasts offer a more positive side to the alcohol picture. In truth, the research picture is not as rosy. This brief article will present some current research and its sources about how alcohol can affect our health and well-being. As always, it's more about frequency than complete abstinence.

Below, three independent segments of valid research and references. To your health!

What Physical Problems Does Alcohol Cause?

1. Alcohol increases stomach acidity.
The irritating effects of alcohol may cause gastritis (inflammation of the lining of the stomach), pancreatitis (inflammation of the pancreas), or a peptic ulcer (a raw area in the lining of the stomach or intestines). Alcohol can also damage your small intestine and keep it from absorbing nutrients.

1. Alcohol negatively influences sleep.
A night's drinking an cause interrupted sleep. Drinking has proved to aggravate insomnia. Initially, while it may help you to get to sleep, as the drink wears off you are likely to wake up earlier, especially to urinate. It can also influence the ability to awaken freshly and energetically.

3. Alcohol can cause gout.
Alcohol inhibits your body from getting rid of uric acid, the crystals that clump together and are then responsible for joint inflammation and toe pain, also known as gout.

4. Higher risk for cancer of the pancreas, liver, mouth, tongue and throat
This risk is even increased if you use tobacco products.
5. Higher risk of liver dysfunction and cancer.
This includes, fatty liver, hepatitis, cirrhosis, and other liver conditions.

6. Vitamin , mineral and protein loss,
Your nerves, muscles, heart and brain need thiamine (Vitamin B-1) in particular, to function normally. Lack of thiamine can cause problems with short-term memory, eye movement, walking, cramps, numbness, tingling and weakness in your legs and hands.

7. High blood pressure and strokes.
To much alcohol may cause high blood pressure and strokes. It can also lead to heart muscle disease or heart failure.

8. Increased blood sugar levels
Of particular danger to diabetics and pre-diabetics, alcohol worsens diabetes, interacts poorly with medications and causes heart, kidney and circulation problems that compromise systemic well being.

— Compiled from University of Michigan Health System.

Alcohol and the Liver: Research Update *

Alcohol-induced liver disease (ALD) is a major cause of illness and death in the United States. Fatty liver, the most common form of ALD, *is reversible with abstinence*. More serious ALD includes alcoholic hepatitis, characterized by persistent inflammation of the liver, and cirrhosis, characterized by progressive scarring of liver tissue. Either condition can be fatal, and treatment options are limited.

The Prevalence of ALD
Approximately 10 to 35 percent of heavy drinkers (1) develop alcoholic hepatitis, and 10 to 20 percent develop cirrhosis (1). In the United States, cirrhosis is the seventh leading cause of death among young and middle-age adults. Approximately 10,000 to 24,000 deaths from cirrhosis may be attributable to alcohol consumption each year (2).

How Does Alcohol Damage the Liver?
Normal liver function is essential to life. Alcohol-induced liver damage disrupts the body's metabolism, eventually impairing the function of other organs.

Alcohol Metabolism
Most of the alcohol a person drinks is eventually broken down by the liver. However, some products generated during alcohol metabolism (e.g., *acetaldehyde*) are more toxic

than alcohol itself. In addition, a group of metabolic products called *free radicals* can *damage liver cells and promote inflammation, impairing vital functions such as energy production*. The body's natural defenses against free radicals (e.g., antioxidants) can be inhibited by alcohol consumption, leading to increased liver damage (3).

The Inflammatory Response
Inflammation is the body's response to local tissue damage or infection. Inflammation prevents the spread of injury and mobilizes the defense mechanisms of the immune system. One such defense mechanism is the generation of free radicals that can destroy disease-causing microorganisms. *Long-term alcohol consumption prolongs the inflammatory process, leading to excessive production of free radicals, which can destroy healthy liver tissue*.

Bacterial Response And Cytokines
Bacteria that live in the human intestine play a key role in the initiation of ALD. Alcohol consumption increases the passage of a noxious bacterial product called endotoxin through the intestinal wall into the bloodstream. Upon reaching the liver, endotoxin activates specialized cells (i.e., Kupffer cells) that monitor the blood for signs of infection. These cells respond to the presence of endotoxin by releasing substances called cytokines that regulate the inflammatory process (4-6).

Cytokines are produced by cells of the liver and immune system in response to infection or cell damage. *Alcohol consumption increases cytokine levels, and cytokines in humans produce symptoms similar to those of alcoholic hepatitis* (7). Recent studies implicate cytokines in scar formation and in the depletion of oxygen within liver cells, processes that are associated with cirrhosis (7). *Each of the disease mechanisms described above contributes to the death of liver cells*. The presence of damaged cells triggers the body's defensive responses, including the release of additional cytokines, resulting in a vicious cycle of inflammation, cell death, and scarring.

Internal Scar Formation
Normal scar formation is part of the wound-healing process. Alcohol-induced cell death and inflammation *can result in scarring that distorts the liver's internal structure and impairs its function*. This scarring is the hallmark of cirrhosis. The process by which cirrhosis develops involves the interaction of certain cytokines and specialized liver cells (i.e., stellate cells). In the normal liver, stellate cells function as storage depots for vitamin A. Upon activation by cytokines, stellate cells proliferate, lose their vitamin A stores, and begin to produce scar tissue. In addition, activated stellate cells constrict blood vessels, impeding the delivery of oxygen to liver cells (6,8).

Acetaldehyde may activate stellate cells directly, promoting liver scarring in the absence of inflammation (9,10). This finding is consistent with the observation that heavy drinkers can develop cirrhosis insidiously, without preexisting hepatitis.

— National Institute on Alcohol Abuse and Alcoholism
(* See references at the end of this article)

Miscellaneous Alcohol Research

Alcohol is a tumor-promoter and a carcinogen. The use of **alcohol increases cancer risk** and has a profound effect. For the lowest possible cancer risk, alcohol should be avoided.

—International Agency for Research on Cancer (IARC), in the 1999 book, "Stopping cancer before it starts," by the American Institute for Cancer Research

Cancer risk may be increased by drinking any amount of alcohol. It doesn't matter if it's beer, wine or whiskey, and the best way to protect yourself from alcohol-related cancers is not to drink. Alcohol increases cancer-causing free radicals. **Cancers of the mouth, pharynx, esophagus and larynx develop when sensitive tissues are directly exposed to alcohol in beverages**.

— "Alcohol/cancer link is solid," American Institute for Cancer Research (AIRC) Newsletter -aicr.org - Oct. 2001

A Harvard University study found that one alcoholic drink daily led to an eighty percent higher melanoma risk, and in an Australian study, two or more drinks daily resulted in an increase of two to two and one-half times.

— From the book, "Skin deep," Carol Turkinton and Jeffrey Dover, MD, 1998

Alcohol consumption has a strong anti-folic acid effect, and deficiency of this nutrient enhances intestinal cancer.

—"Alcohol, colon cancer, and folic acid," "Nutrition Hints," by Betty Kamen, PhD, and Dr. Michael Rosenbaum, MD, Source: Journal of Nutrition, 2002

The risk of prostate cancer was found to be increased according to the amount of alcohol consumed.

—US TOO International, Inc., Prostate Cancer Survivor Support Groups, 1996

Like saturated fat, **alcohol appears to raise prostate cancer by increasing the circulating levels of sex hormones, and men who drink the most alcohol were the most likely to die from prostate cancer,** said a University of Massachusetts study.

—Self Healing newsletter, Andrew Weil, MD, Apr. 1999

Alcohol can interfere with the function of the male reproductive system, and can lead to tissue shrinkage, reduced testosterone production, and sperm abnormalities, in the testes. This can lead to male infertility and to other deleterious effects throughout the body, such as bone loss and decreased muscle function.

— "Alcohol's Effects On Male Reproduction," Alcohol Health & Research World, NIAAA, 1998

Stop drinking alcohol if you want **to preserve testosterone, help prevent impotence and improve your quality of life**.

— HealthSense newsletter, Stephen Sinatra, MD, Aug. 1999

Alcohol may **interfere with normal sperm structure and movement by inhibiting the metabolism of Vitamin A,** which is essential for sperm development.

—"Alcohol Impairs Reproductive Functions," Alcohol Alert, NIAAA, 2000}

Alcohol **slows sperm production**, and its breakdown product, **acetaldehyde, is toxic to sperm**.

— Srikanth V., et al., "Effects of ethanol ingestion on sperm monosaccharides and fertility, Biochem. Mol. Biol. Int. 1999. On VitalCast, Feb. 2000

Alcohol is directly **toxic to the testes, causing reduced testosterone** levels in men.

— "Alcohol Impairs Reproductive Functions," Alcohol Alert, NIAAA, 2001}

Testosterone production is depressed by alcohol's toxicity.

— "Alcohol's Effect on the Liver," Charles Lieber, MD, Alcohol Research and Treatment Center, Professor of Medicine and Pathology, Mt. Sinai School of Medicine of the City University of New York, Sept. 2002

Long term alcohol use has also been associated with testicular atrophy, which can lead to increased estrogen and decreased testosterone. **Studies have linked alcohol use to benign prostatic hypertrophy** (BPH), which can lead to prostate cancer.

— HeartSense, Jan. 2000, Stephen Sinatra, MD

Avoid alcohol. BPH (benign prostate hyperplasia) is a noncancerous enlargement of the prostate, marked by frequent urination, a need to get out of bed at night to urinate, and incomplete voiding. BPH affects more than half of men in their 60's.

—"Prostate Health", Medizine Guidebook, Jul. 2000

When an enlarged prostate is at the root of BPH symptoms, there is much you can do to avoid aggravating the discomfort. Reduce or eliminate your consumption of caffeine and alcohol, diuretics that can trigger the urge to urinate.

— Andrew Weil, MD, "Self Healing" newsletter, Oct. 2000

It is believed that alcohol is related to the early stages of prostate disease. No difference was found between men who had stopped drinking and men who still drank, leading them to suspect that alcohol is related to the early stages of the disease. **Alcohol may cause prostate cancer either indirectly, through its negative effect on the body's ability to absorb nutrients, or directly through alcohol's carcinogenic properties**.

— "Alcohol Use and Prostate Cancer," ustoo.com - Oct. 2001}

** References for: National Institute on Alcohol Abuse and Alcoholism*

(1) National Institute on Alcohol Abuse and Alcoholism. Alcohol Alert No. 19: Alcohol and the Liver. PH 329. Rockville, MD: the Institute, 1993. (2) DeBakey, S.F.; Stinson, F.S.; Grant, B.F.; and Dufour, M.C. Surveillance Report #41. Liver Cirrhosis Mortality in the United States, 1970-93. Bethesda, MD: National Institute on Alcohol Abuse and Alcoholism, 1996. (3) Kurose, I.; Higuchi, H.; Kato, S.; Miura, S.; and Ishii, H. Ethanol-induced oxidative stress in the liver. Alcoholism: Clinical and Experimental Research 20(1):77A-85A, 1996. (4) Nanji, A.A.; Khettry, U.; and Sadrzadeh, S.M.H. Lactobacillus feeding reduces endotoxemia and severity of experimental alcoholic liver (disease). Proceedings of the Society for Experimental Biology and Medicine 205(3):243-247, 1994. (5) Thurman, R.G.; Bradford, B.U.; Iimuro, Y.; Knecht, K.T.; Connor, H.D.; Adachi, Y.; Wall, C.; Arteel, G.E.; Raleigh, J.A.; Forman, D.T.; and Mason, R.P. Role of Kupffer cells, endotoxin and free radicals in hepatotoxicity due to prolonged alcohol consumption: Studies in female and male rats. Journal of Nutrition 127(S5):903S-906S, 1997. (6) Lands, W.E.M. Cellular signals in alcohol-induced liver injury: A review. Alcoholism: Clinical and Experimental Research 19(4):928-938, 1995. (7) McClain, C.J.; Shedlofsky, S.; Barve, S.; and Hill, D.B. Cytokines and alcoholic liver disease. Alcohol Health & Research World 21(4):317-320, 1997. (8) Maher, J.J., and Friedman, S.L. Pathogenesis of hepatic fibrosis. In: Hall, P., ed. Alcoholic Liver Disease: Pathology and Pathogenesis. 2d ed. London: Edward Arnold, 1995. pp. 71-88. (9) Lieber, C.S. Hepatic and other medical disorders of alcoholism: From pathogenesis to treatment. Journal of Studies on Alcohol 59(1):9-25, 1998. (10) Ma, X.; Svegliati-Baroni, G.; Poniachik, J.; Baraona, E.; and Lieber, C.S. Collagen synthesis by liver stellate cells is released from its normal feedback regulation by acetaldehyde-induced modification of the carboxyl-terminal propeptide of procollagen. Alcoholism: Clinical and Experimental Research 21(7):1204-1211, 1997.