
FACTS ON: MEDICAL CONSEQUENCES OF ALCOHOL

BY JOHN BRICK, PH.D.

OVERVIEW

Alcohol affects virtually every organ system in the body. Acute and chronic intoxication have their unique consequences on physiology, affecting both the quality of life and longevity. The number of biological systems affected by alcohol is staggering, both in the scope of medical consequences, and in terms of the economics of medical treatment of alcohol-related disorders. Despite these undesirable effects, not all of the medical consequences of alcohol use are deleterious. The most significant and well-known medical consequences of alcohol use and abuse are presented in this overview.

ALCOHOL AND ACCIDENTAL INJURIES

Accidental injuries produce obvious direct medical consequences. Alcohol increases the risk for such injuries through the impairment of cognitive (mental) and psychomotor (coordinating movement with mental tasks) functioning, while performing or engaging in a variety of behaviors such as driving a car, riding a motorcycle or bicycle, operating a boat, swimming, and pedestrian activity. Alcohol increases reaction time, impairs sensory processing, motor control, attention, and the use of seat-belt devices. In young men, especially, increased risk-taking and impulsivity also occur. All of these factors significantly increase the risk for a serious or fatal injury. Drunken driving accidents alone kill about 16,000 people per year, with many more times that amount injured. Alcohol also increases the risk for injury or death due to suicide and fire.

Intoxication and Injury Outcome

Whereas the medical consequences from accidental injuries due to intoxication are obvious, less well known is the fact that alcohol intoxication also affects injury outcome. For example, motorcyclists with head injuries are about twice as likely to die from those injuries if they are intoxicated, than sober injury-matched controls. Contrary to popular misconception, intoxicated drivers are more likely to be seriously or fatally injured in comparison to sober drivers, and intoxicated victims with central nervous system injuries are more than twice as likely to die sooner from those injuries than sober people. The exact mechanism of this effect is not known, but may be related to alcohol inhibition of free radicals, alcohol induced cerebral edema (swelling from excessive fluids), or increased hemorrhagic shock due to alcohol-induced changes in blood chemistry (e.g., acidosis). Excessive alcohol intake causes generalized skeletal fragility and decreased bone density.

As a result, bones break more readily when exposed to alcohol. Microscopic examination of bone tissue from alcoholics suggests that the normal cycle of bone growth is disrupted by alcohol, but this effect is reversible. There is some evidence that modest alcohol intake, of less than 1 drink per day, increases bone density.

CANCERS

In most cases, the first tissues that alcohol contacts are within the gastrointestinal system. Because relatively high concentrations of alcohol come in contact with these cells, alcohol can cause inflammation of the esophagus (which connects the mouth to the stomach), and cause gastric acid reflux that will result in symptoms ranging from heartburn to severe esophagitis (inflammation of the esophagus). People who consume more than 3 drinks per day (21 drinks per week) have an almost tenfold higher risk of esophageal cancer than do those who drink less than 1 drink per day. Alcohol also causes gastritis and other stomach disorders, but not stomach cancer, as once believed. There is a weak association between alcohol use and cancers of the colon and rectum. Recent studies indicate that smoking tobacco coupled with drinking alcohol may serve as a triggering mechanism for colon cancer.

Despite decades of research suggesting that alcohol increases the risk for breast cancer, recent reviews of this relationship suggest that the evidence for this is not as compelling as once believed.

ALCOHOL-INDUCED LIVER INJURY

After alcohol is absorbed from the gastrointestinal tract into the blood, the first organ it reaches is the liver. The liver is the largest organ in the body and the primary site of alcohol metabolism. Metabolism is the process by which toxins are transformed or broken-down into metabolites. In some cases, the metabolites are also toxic. High concentrations of alcohol reaching the liver and the formation by the liver of toxic alcohol metabolites, such as acetaldehyde, make the liver a particularly vulnerable target and one of the most serious and deadly consequences of alcohol abuse. Epidemiological data clearly reveals that alcohol abuse is the leading cause of liver-related mortality in the United States. Excessive alcohol consumption leads to three serious types of liver injury: fatty liver, in which lipids (fats) infiltrate the liver cells, cause liver enlargement and cell damage (a reversible process); hepatic

inflammation (alcoholic hepatitis), characterized by prolific inflammation and tissue damage (significant recovery following abstinence), and progressive liver scarring (fibrosis or cirrhosis), which is often fatal. Cirrhosis is characterized by scarring and cell death. It is estimated that about 900,000 people have cirrhosis, and of the 26,000 who die each year, about 40-90 percent have a history of alcohol abuse. Cirrhosis is irreversible. Studies suggest that depending on whether you are a man or a woman, between about 3-12 drinks per day for about ten years will result in reliable signs of liver injury. Generally, women have a lower threshold for injury than men. Moreover, hereditary, environmental and other factors, not just alcohol, contribute to the pathogenesis of liver.

CARDIOVASCULAR DISEASES

Cardiovascular diseases (diseases of the heart and blood vessels) are the leading causes of death among Americans, followed by cancer and stroke. The results from many studies are clear: alcohol has both deleterious and beneficial effects on cardiovascular diseases, but the mechanisms and conditions under which these effects occur are complex.

The deterioration of heart muscle (alcoholic cardiomyopathy) is one of the most serious consequences of chronic heavy drinking. Alcoholic cardiomyopathy is common in Western societies and is a major source of heart failure and death.

Similarly, there is a well-documented association between heavy alcohol consumption and increased blood pressure or hypertension. As little as one drink a day can chronically increase blood pressure 1 millimeter of mercury in middle-aged individuals, and even more in the elderly and in people with pre-existing hypertension.

Another consequence of hypertension is an increased risk of stroke. A stroke results from the blockage or the rupture of a blood vessel in the brain. Strokes can result in permanent loss of some psychomotor functions, or fatal. Evidence is reasonably consistent that heavy drinking is associated with increased stroke risk in both sexes, but perhaps more so in women. There is some evidence of a protective effect of low doses of alcohol against stroke.

POSSIBLE BENEFICIAL EFFECTS OF ALCOHOL

Several large prospective studies have reported a reduced risk of death from coronary heart disease (CHD) across a wide range of alcohol consumption. Overall, beneficial effects of alcohol are typically detected when alcohol consumption was relatively low (from 1-2 drinks per day to 1-2 drinks per week). An association between moderate drinking and lower risk for CHD does not necessarily mean that alcohol itself is the protective agent. For example, the higher mortality risk among abstainers may be due to other traits – socioeconomic and employment status, mental health, overall health, and health habits such as smoking – rather than participants' non-use of alcohol. Finally, any apparent benefits of moderate drinking on CHD mortality or stroke may be offset

at higher drinking levels by an increased risk of death from other types of heart disease, cancer, liver cirrhosis, and trauma.

Controversy remains as to whether moderate alcohol consumption has any beneficial effects on blood pressure, but reducing alcohol intake may be one means of reducing blood pressure in people with hypertension. Alcohol is not a recommended preventative medicine or treatment for CHD or stroke.

REFERENCES

- Brick, J., Medical Consequences of Alcohol Abuse. In: Brick, J. (ed). Handbook of Medical Consequences of Alcohol and Drug Abuse. Haworth Medical Press (2004) pp. 7-31.
- Dufour, M.C.; Stinson, F.S. and Caces, M.F. (1993). Trends in cirrhosis morbidity and mortality: United States, 1979-1988. *Semin Liver Dis* 13(2):109-125.
- Gray, M.R.; Donnelly, R.I.; and Kingsnorth, A.N. (1993). The role of smoking and alcohol in metaplasia and cancer risk in Barrett's columnar lined oesophagus. *Gut* 34(6):727-731.
- Luna, G.K.; Maier, R.V.; Sowder, L.; Copass, M.K. and Oreskovich, M.R. (1984) The Influence of Ethanol Intoxication on Outcome of Injured Motorcyclists. *J. Trauma*, 24(8):695-700.
- National Institute on Alcohol Abuse and Alcoholism, Tenth Special Report to the U.S. Congress on Alcohol and Health, U.S. Department of Health and Human Services, 2000.
- National Institute on Alcohol Abuse and Alcoholism. *Ninth Special Report to the U.S. Congress on Alcohol and Health*. NIH Publication No. 97-4017. Bethesda, MD: National Institute on Alcohol Abuse and Alcoholism, 1997.
- Waller, P.; Steward, J.; Hansen, A.; Stutts, J.; Popkin, C. and Rodgman, E. (1986). The potentiating effects of alcohol on driver injury. *JAMA* 256(11):1461-1466.
- Yamada, K.; Araki, S.; Tamura, M.; Sakai, I.; Takahashi, Y.; Kashiwara, H. and Kono, S. (1997). Case-control study of colorectal carcinoma in situ and cancer in relation to cigarette smoking and alcohol use. *Cancer Causes Control* 8(5):780-785.
- Zink, B.; Maoi, R and Chen, B. (1996). Alcohol, central nervous system injury, and time to death in fatal motor vehicle crashes. *Alcoholism: Clinical and Experimental Research* 20(9):1518-1522.
- Zink, B.J.; Schultz, C.H.; Stern, S.A.; Mertz, M.; Wang, X.; Johnston, P.; Keep, R.F. (2001). Effects of Ethanol and Naltrexone in a Model of Traumatic Brain Injury with Hemorrhagic Shock. *J Clin Exper Res* 25(6):916-923.

John Brick, PhD, MA, FAPA, is a biological psychologist and Executive Director of Intoxikon International, 1006 Floral Vale, Yardley, PA 19067



Center of Alcohol Studies
Smithers Hall
607 Allison Road
Piscataway, NJ 08854
Tel. (732) 445-4442
Fax (732) 445-xxxx