Does Moderate Alcohol Consumption Prolong Life?

Prepared for the American Council on Science and Health by

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Editor's Note

In reflecting on our publication, *Does Moderate Alcohol Consumption Prolong Life?*, some readers might ponder the questions, "Why is ACSH, an organization dedicated to promoting public health, publishing a position paper which reports that the moderate consumption of alcohol prolongs life? Given the disastrous consequences of alcohol abuse, why present data on moderate use? Might any benefits to the majority who read this report be outweighed by the harm done to individuals who, after reading it, proceed to abuse alcohol?"

We believe these questions deserve careful consideration, particularly since a small number of our peer reviewers raised these or similar queries.

In this publication ACSH's author, Dr. R. Curtis Ellison, stresses the dangers of alcohol misuse and abuse. He carefully defines "moderation." In a related ACSH publication, *Alcohol: Defining the Parameters of Moderation*, ACSH noted the dangers associated with driving after consuming any amount of alcohol and urged consumers never to drink alcoholic beverages in the hours before getting behind the wheel. Any alcohol consumption before driving is excessive and constitutes abuse. ACSH recognizes the substantial contribution this and other forms of alcohol abuse makes to total premature mortality in the United States. We have, in related publications, reported the estimate that approximately 100,000 Americans die prematurely each year due to the misuse of alcohol. We will continue to stress that when used to excess, alcohol is the cause of not only physical ill health, but also emotional ill health. Alcohol abuse not only affects the drinker but also the victims of alcohol-impaired driving and, indeed, any individual who must witness alcoholism's devastating effects on a loved one.

ACSH, however, with its commitment to differentiating between health hyperbole and health facts, cannot ignore the growing scientific consensus that small amounts of alcohol prolong life, primarily by reducing the risk of cardiovascular disease. The majority of ACSH scientists reject the hypothesis that such sound health information should be withheld because a minority of people may misuse or misinterpret the facts. In this report, ACSH does not recommend the consumption of alcohol. It merely presents the scientific evidence now at hand so that consumers may make their own, informed decisions.

Executive Summary

Through the ages, conventional wisdom has been that the moderate intake of alcoholic beverages is consistent with a long and healthy life. Data from epidemiologic studies within recent decades demonstrate that death rates from coronary heart disease are lower among consumers of small to moderate amounts of alcohol than among non-drinkers. Some of the proposed mechanisms for the beneficial health effects of moderate alcohol consumption are:

- alcohol lowers harmful LDL-cholesterol levels
- alcohol raises protective HDL-cholesterol levels
- alcohol decreases formation of blood clots in the arteries
- alcohol increases coronary blood flow
- alcohol increases estrogen levels.

The risk of cirrhosis, some cancers and certain other diseases increases with alcohol consumption. At low to moderate levels of intake, however, the adverse effects on mortality from such causes do not outweigh the beneficial effects on mortality of the drinker from coronary heart disease. Thus, the net effect of the reported consumption of small to moderate amounts of alcohol is a reduction in total mortality of the drinking population.

The results of this report should not be viewed as endorsement or justification of heavy drinking. Rather, the health effects of moderate alcohol consumption are presented so the general public can make informed decisions.

Introduction

Modern medicine became particularly interested in the effects of alcohol consumption on mortality in the 1950s and 1960s when coronary heart disease became a major cause of death in the United States and in most other industrialized countries. Primarily from epidemiologic studies designed to identify factors associated with high death rates from coronary heart disease, it became apparent that these rates were lower among drinkers of small to moderate amounts of alcohol than among non-drinkers.[1-8] However, these studies did not often explore effects of alcohol consumption on overall mortality rates. Specifically, it was not known whether increases in death from other diseases that are known to be associated with alcohol abuse, such as cirrhosis of the liver and certain cancers, offset the apparent beneficial effects of low to moderate alcohol intake on mortality from coronary heart disease.

The net effects of drinking alcoholic beverages on the risk of death of the drinker from any cause is the subject of this report. We will present data relating alcohol consumption to total mortality rates from a number of the major follow-up (cohort) studies in which drinkers of varying amounts were followed over a number of years. In this report we will attempt to distinguish between a level of alcohol use that, on average, is consistent with good health and the level at which alcohol consumption has predominantly adverse health effects.

What is a “Drink”?

Before discussing the effects of alcohol on mortality, it is necessary to consider how much alcohol is consumed. In epidemiologic studies, alcohol intake is reported in a confusing variety of measures, including grams, milliliters, ounces of alcohol or number of “drinks.” These measures are reported per day, per week or even per month. Furthermore, categories of intake in the major follow-up epidemiologic studies have varied, generally because the drinking habits of the populations being followed were different or the data were available only in a certain format.

We believe that most people can relate better to “drinks per day” than to grams of ethanol per day or week or month. Consequently, when possible, we will present data from different studies as drinks/day, using the following guidelines to define a “drink” – 10 to 12 grams of ethyl alcohol, which is approximately equivalent to one 12 ounce can or bottle of beer, 4 to 5 ounces of table wine or 1.25 ounces of 80-proof spirits.

Definition of “Non-drinker”

Some of the early epidemiologic studies that collected information on alcohol consumption simply

inquired about the current drinking habits of individuals. No information was obtained on previous drinking habits. Studies differ as to the percentage of ex-drinkers included in the non-drinking category. In some populations, a high proportion of non-drinkers may be lifetime abstainers, while in other populations a high proportion of non-drinkers may be ex-drinkers. Many of these ex-drinkers may have stopped drinking due to problems with alcohol abuse or serious medical conditions. Thus, their inclusion with lifetime abstainers in a single "non-drinking" category is inappropriate.

Most of the recent epidemiologic studies, as well as some of the earlier studies, separate lifetime abstainers from previous drinkers. Where such data are available, mortality rates will be presented separately for lifetime abstainers and for ex-drinkers.

Definition of "Moderate Drinking"

How does someone decide what is "moderate drinking"? In 1874, the English physician Anstie stated that a daily intake not exceeding 1.5 ounces of ethanol (the equivalent of about three ounces of 100 proof spirits or three "drinks") produced "no perceptible injurious effects" that he could determine at postmortem examination.[9] Anstie's assertion did not have the support of epidemiologic research, but today we have a considerable compendium of research results. One approach to determine "moderate" consumption is to choose the levels of intake from epidemiologic studies that, on average, are not associated with increases in deaths from diseases or other conditions that are known to relate to excess alcohol use.*

* (In this publication we discuss only deaths from diseases or injuries that occurred among drinking and non-drinking individuals who were participating in epidemiologic studies. We do not have data that would permit us to comment on deaths of other individuals that may have occurred because of drinking-related accidents by the participants or from the effects on fetuses from drinking by pregnant women who participated in these studies.)

We must remember that "moderate" drinking for one individual may be excessive for another, as there are marked differences among individuals in their tolerance to alcohol. Alcohol tolerance is based, in part, on an individual's level of alcohol dehydrogenase (the enzyme that helps metabolize alcohol). Tolerance level is also a function of an individual's gender, age, body size, health conditions, genetics and other factors. For some people, such as those with a history of alcoholism or any type of drug abuse, there is no "moderate" consumption level. These individuals should abstain from all alcohol consumption. Also, if taken prior to driving, even small amounts of alcohol can increase the risk of accidental death of the driver and others.

Nevertheless, for purposes of this publication, we will use the term "moderate drinkers" to refer to individuals who state that they consume an average of approximately 1 to 2 drinks per day. In most clinical and epidemiologic studies, this level is not associated with increased rates of diseases related to alcohol abuse nor, as we will show, to increases in total mortality rates.

Limitations of Data on Alcohol Consumption

The data available for judging the effects of alcohol on health are almost always based on self reports by participants in epidemiologic studies. Individuals may underestimate (or overestimate) their usual amounts of alcohol intake. Furthermore, in most epidemiologic studies, data are collected in terms of the "average number of drinks per week," and the drinking pattern (which may markedly affect the risk of disease) is not well

characterized.

It is perhaps even more important that data are usually not obtained on the circumstances under which alcohol is consumed, especially whether or not it is consumed with food. The presence of food in the stomach affects the amount of alcohol required for intoxication and may influence alcohol's effects on many diseases. Wine is usually consumed with food, whereas beer and spirits are more likely consumed without food. The concentration of ethanol in the beverage and the rapidity with which the beverage is consumed also influence alcohol's effects. These limitations make it difficult to use the results from epidemiologic studies as a basis for recommending specific amounts of alcohol consumption for individuals. However, associations between alcohol consumption and disease within populations can be obtained.

Does Alcohol Protect Against Coronary Heart Disease?

While searching for the risk factors for coronary heart disease in comparisons among countries[10] as well as among individuals[1-4], epidemiologists identified a potential benefit from alcohol consumption on mortality from heart disease. In the past three decades, almost every follow-up epidemiologic study has demonstrated that individuals who drink small to moderate amounts of alcohol have a lower risk than non-drinkers of dying from coronary heart disease.* (* Since drinkers are more likely than non-drinkers to smoke cigarettes (another factor linked to increased risk of coronary heart disease and other diseases), it is necessary to adjust for smoking habits and other risk factors when studying the relation between alcohol consumption and coronary heart disease.) Among drinkers, the categories with the lowest coronary heart disease rates vary across studies, ranging from less than one drink per day to 3 to 5 drinks per day. In most studies, individuals who state that they normally consume six or more drinks per day, or who admit to having problems with alcohol abuse, have rates of dying from heart disease higher than both non-drinkers and moderate drinkers. Such deaths may often be due to cardiomyopathy, from toxic effects of alcohol on the heart muscle, rather than from disease of the coronary arteries.

As stated previously, many of the early studies were criticized for including in the category of non-drinkers both lifetime abstainers and ex-drinkers. Many ex-drinkers may have given up drinking due to alcoholism or other health problems (including cancer and heart disease) and thus would be expected to have a greater risk of early death.[11] Other follow-up studies have demonstrated, however, that even when only lifetime abstainers [5,38,40,42] or healthy abstainers [6-8] make up the non-drinker category, the risk of coronary heart disease for non-drinkers is still higher than it is for moderate drinkers.

Mechanisms for Alcohol's Protection Against Coronary Heart Disease

Epidemiologic, clinical and experimental studies indicate that alcohol consumption is associated with milder degrees of atherosclerosis and/or with a lower risk of coronary heart disease. The primary mechanism by which alcohol reduces the risk of heart disease has traditionally been thought to be its effects on blood lipids.[12,13] Alcohol increases the level of HDL-cholesterol, the so-called "good cholesterol," that tends to protect against coronary heart disease.[14-20] In addition, alcohol is associated with lower levels of LDL-cholesterol, the "bad cholesterol," which is the primary blood lipid associated with increased heart disease risk.[21]

An additional mechanism by which alcohol prevents heart disease is through its effects on thrombosis (blood clotting). Alcohol makes platelets, a portion of the blood that contributes to clotting, less sticky so that

they aggregate less. Thus, clot formation within arteries is less likely.[22-26] Alcohol also affects thrombosis by decreasing blood levels of fibrinogen, another blood constituent that promotes clotting. Further, in some studies, alcohol has been shown to increase fibrinolysis, the process by which clots dissolve.[27] Other mechanisms related to moderate alcohol consumption may also decrease coronary heart disease risk. Biologic mechanisms that have been proposed to explain the protection against heart disease are summarized in Table 1. While some of these mechanisms (e.g., effects of alcohol on lipids and platelet aggregation) have been confirmed in multiple studies, others (e.g., effects of alcohol on blood insulin and estrogen levels) have been described more recently and require further investigation. Although questions still remain as to the exact mechanisms by which alcohol may exert its protective effects, the epidemiologic data show consistently that individuals who regularly consume small to moderate amounts of alcohol have a lower risk of coronary heart disease.

Table 1: Proposed Biologic Mechanisms by Which Alcohol May Protect Against Coronary Heart Disease

1. Alcohol improves blood lipid profile
    a. Increases HDL-cholesterol
    b. Reduces LDL-cholesterol
2. Alcohol decreases thrombosis
    a. Reduces platelet aggregation
    b. Reduces fibrinogen
    c. Increases fibrinolysis
3. Alcohol acts through other mechanisms
    a. Reduces coronary artery spasm in response to stress
    b. Increases coronary blood flow
    c. Reduces blood pressure (when used in moderation)
    d. Reduces blood insulin levels
    e. Increases estrogen levels

Are There Differences in Coronary Heart Disease Risk According to Type of Alcoholic Beverage Consumed?

Wine, especially red wine, has garnered considerable attention in the media recently as to whether it protects against coronary heart disease more than other types of alcoholic beverages. Scientific data are not yet sufficient to answer this question. Data from limited experimental studies in rabbits support a greater protection by wine than by other alcoholic beverages against atherosclerosis that develops when such animals are fed a high-cholesterol diet.[29] Another study recently showed that among healthy adult males, platelet adhesiveness decreased when the subjects consumed red wine but not when they consumed white wine or an ethanol solution.[25] Decreased platelet adhesiveness should lead to reduced thrombosis and a lower risk of heart disease.

In cross-cultural studies, populations that consume primarily wine have lower rates of coronary heart

disease than those that consume primarily beer or spirits.[10] However, in follow-up investigations, characteristics of the population other than which type of alcohol the majority of individuals consumed may play a role in determining alcohol's effects.[1,28,30] Some studies show that alcoholic beverages other than wine may offer more protection against coronary heart disease.[30] Other studies illustrate that white wine may offer as much protection against heart disease as does red wine. At this time, the data do not allow one to conclude that red wine definitely conveys more protection than does white wine.

However, if wine is ultimately found to offer more protection against coronary heart disease than other alcoholic beverages, it may be due to one of the many non-alcoholic organic compounds it contains. One such compound that has recently received attention is resveratrol,[31] a naturally occurring fungicide that is present in many plants, including wine grapes.[32] While the data from animal studies with this compound are provocative, [31] corresponding studies in humans have not yet been carried out. Furthermore, there are probably hundreds of other biologically active compounds in wine, beer and spirits that may also play a role in the effects of alcoholic beverages on health and disease. It is not yet possible to tell whether or not certain types of alcoholic beverages are more protective than others against coronary heart disease.

Effects of Alcohol on Other Diseases

Although there is little doubt that the moderate consumption of alcoholic beverages is associated with a lower risk of coronary heart disease, the risk of death from non-cardiac diseases or other causes depends on how much someone drinks. In discussing whether alcohol consumption increases or reduces the risk of non-cardiac diseases, we chose to first consider the net effects on health of consuming **moderate** amounts of alcohol. Then we consider the net health effects of consuming **large** amounts of alcohol.

Moderate Alcohol Intake

There are a number of diseases or conditions that are often attributed to the use of alcohol (e.g., cirrhosis of the liver, certain types of cancer, accidents). In general, cirrhosis and alcohol-related cancers are associated with alcohol abuse and not with the consumption of small to moderate amounts of alcohol. In case-control studies, however, in which the prior drinking experience of individuals with a given disease such as cancer ("cases") is compared with the drinking experience of individuals without the disease ("controls"), a frequent finding is that all levels of alcohol consumption are reported more commonly by the cases than by the controls. Some of the apparent relationship between the disease and moderate drinking may be due to what is referred to as recall bias: Individuals with a disease may be more prone to recall or admit to drinking a certain amount than individuals without the disease.

In cohort or follow-up studies, the data on alcohol consumption are recorded prior to the detection of disease and the potential for recall bias in reporting is much less. According to these follow-up studies, the risk of death in drinkers of small amounts of alcohol from certain diseases known to be related to alcohol use (such as cirrhosis) generally is not increased. The risk of death from such diseases usually increases only among people who consume moderate to large amounts of alcohol, suggesting a threshold effect. However, even moderate amounts of alcohol may be related to the risk of death from hemorrhagic stroke. On the other hand, the risk of thrombotic stroke, the much more common type of stroke in the U.S. and Europe, is reduced by moder-

ate amounts of alcohol. Therefore, the net effect of moderate alcohol intake in these parts of the world is a lowering of the risk of stroke.

Questions still remain on the apparent relation between moderate alcohol intake and breast cancer. Certain studies indicate that the risk of breast cancer in women may increase with only moderate levels of alcohol intake.[33,34] For example, data from the Nurses' Health Study indicate that in comparison with women who stated that they consumed no alcohol, those who stated that they consumed 3 to 9 drinks per week had a 30 percent increased risk of having breast cancer.[34] Those who drank more than 9 drinks had a 60 percent increased risk over non-drinkers.

Other studies indicate no such relation between moderate alcohol intake and increased risk of breast cancer. For example, the Framingham Study did not find an increase in breast cancer rates among women who consumed small to moderate amounts of alcohol.[35] In addition, in nations where wine consumption (as well as total alcohol consumption) is highest, breast cancer rates are not higher than those in countries with low alcohol consumption. Age-standardized death rates for breast cancer among women, as reported by the World Health Organization for 1987, were 28, 29 and 21 per 100,000 for three countries with the most wine consumption, France, Italy and Spain, respectively.[36] For three countries with very low wine consumption, the U.S., Great Britain and Ireland, they were 32, 41 and 38, respectively. However, these cultural comparisons do not provide enough evidence to conclude that alcohol does not affect breast cancer rates. Other lifestyle factors in the countries with a high intake of wine could ameliorate the effect of alcohol. It will require further research to clearly define what role, if any, alcohol plays in breast cancer.

There is always the possibility that a positive relationship between low or moderate levels of alcohol consumption and certain non-cardiac diseases may be spurious, due to under-reporting of actual intake by some individuals. For example, in some epidemiologic studies, the risk of death from cirrhosis of the liver begins to increase with the reported use of any alcohol. From physiologic studies, however, healthy individuals show liver abnormalities only when they consume fairly large amounts of alcohol. This suggests that some heavy drinkers under-report their intake and are classified as moderate drinkers. Nevertheless, epidemiologists must rely on self reporting of alcohol consumption when evaluating the effects of alcohol on disease.

Excessive Alcohol Intake

Alcoholics and other abusers of alcohol generally are at increased risk for a number of diseases and causes of death. Driving while intoxicated causes the most accidental alcohol-related deaths. Rates of suicide are also increased for alcohol abusers. Furthermore, excessive drinkers experience increased rates of oral, pharyngeal, esophageal and stomach cancer. This is presumably due to the direct toxic effect of alcohol, and probably other substances in alcoholic beverages, on the tissues lining the upper gastrointestinal tract. Frequently, rates of such cancers are much higher, or even seen almost exclusively, among drinkers who also smoke cigarettes. Indeed, the combination of heavy drinking and smoking is particularly harmful.

Effects of Alcohol Consumption on Total Mortality

One approach to determine the net effects of an exposure (such as alcohol consumption) on disease rates is to calculate the rates for all diseases that may be related to the exposure and then attempt to balance

the beneficial and harmful effects. When considering death as the outcome, however, we can simply look at total, or all-cause, mortality to judge the net effects of varying levels of alcohol consumption.

The best way to judge the net effects of alcohol consumption on a variety of causes of death is to look at all-cause mortality in follow-up studies, where data on alcohol use are collected prior to the development of disease or death. The net effects of alcohol consumption on all-cause mortality in nine recently reported follow-up cohort studies are listed in Table 2. The studies in the table include a follow-up study of Japanese physicians and an investigation of individuals in a small town in Australia (the Busselton Study). Also included in the table are results from a very large nationwide study in the U.S. by the American Cancer Society that followed more than 270,000 men for 12 years. Given in the table are the relative risks of dying during the follow-up period of each study according to the amount of alcohol consumption reported at the beginning of the study. A discussion of the term "relative risk" and of epidemiologic concepts of risk is provided in the Appendix and should be reviewed so as to fully comprehend the implications of the data.

Table 2: Relative Risk of Dying (All Causes),*
According to Level of Reported Usual Alcohol Intake
(with referent group being non-drinkers, given a value of 1.0)

Study	Follow Up	Year	Alcohol Category (# Drinks)	Relative Risk
Japanese Physician's Study (Kono, et al.) [38]	19 yr	1986	None	1.00
			Occasional	0.86
			1-4 /day	0.91
			5+ /day	1.28
			Ex-drinker	1.38
British Regional Heart Study (Shaper, et al.) [39]	7.5 yr	1988	Occasional	1.0
			0.1-2/day	0.7
			2-6 /day	0.7
			>6 /day	0.9
American Cancer Society (Boffetta & Garfinkel) [6]	12 yr	1990	Non-drinker	1.00
			Occasional	0.88
			1/day	0.84
			2/day	0.93
			3/day	1.02
			4/day	1.08
			5/day	1.22
			≥6/day	1.38
			Irregular	1.01
Kaiser Permanente Study (Klatsky, et al.) [5]	<7 yr	1990	Abstainers	1.0
			<1/mth	1.0
			>1/mth, <1/day	0.9
			1-2/day	0.9
			3-5/day	1.0
			≥6/day	1.4
			Ex-Drinkers	1.1

Study	Follow Up	Year	Alcohol Category (# Drinks)		Relative Risk
Busselton Study (Cullen) [40]	23 yr	1991	Men	None	1.00
				Mild	0.90
				Moderate	0.69
				Ex-drinkers	1.13
			Women	None	1.00
				Mild	1.01
			Moderate	0.35	
				Ex-drinkers	2.47
			Combined	None	1.00
				Mild	0.92
				Moderate	0.66
				Ex-drinkers	1.26
British Doctor's Study*** (Doll)[7]	12 yr	1991	0		1.00
			0.1-1.3/day		0.64
			1.4-2.7/day		0.78
			2.8-4.1/day		0.72
			4.2-5.6/day		0.73
			≥5.7/day		1.03
Normative Aging Study (De Labry, et al.)[8]	12 yr	1992	<49 yrs	None	1.00
				Moderate	0.76
				Heavier	1.35
			50-64 yrs	None	1.00
				Moderate	0.80
				Heavier	1.36
			>65 yrs	None	1.00
				Moderate	0.60
				Heavier	0.88
Studies in Elderly (Scherr, et al.)[41]	5 yr	1992	East Boston	0 in year	1.0
				Some in yr, 0 in month	1.1
				Some in mth, <1oz./day	0.6
				≥1oz./day	0.6
			Iowa	0 in year	1.0
				Some in yr, 0 in month	1.4
				Some in mth, <1oz./day	1.0

Study	Follow Up	Year	Alcohol Category (# Drinks)		Relative Risk	
			New Haven	≥1oz./day	1.6	
				0 in year	1.0	
				Some in yr, 0 in month	0.5	
				Some in mth, <1oz./day	0.5	
				≥1oz./day	0.6	
					White	Black
Kaiser Permanente Study (Klatsky, et al.) [42]	<10 yr	1992	Abstainers		1.0	1.0
			<1/mth		1.0	0.9
			≥1/mth, <1/day		0.8	0.9
			1-2/day		0.9	0.9
			3-5/day		1.0	1.1
			≥6/day		1.2	1.3
			Ex-Drinkers		1.2	1.0

* adjusted for smoking and other risk factors
** values estimated from graph in publication
*** men with no history of cardiovascular disease at baseline
**** for this study, an ounce of alcohol was calculated to be the equivalent of approximately one mixed drink or cocktail, 1.5 4-ounce glasses of wine, and 1.7 12-ounce bottles of beer

As evident from the table, all of the studies show that the relative risk of dying from any cause (overall mortality) is lower (less than 1.0) for individuals in at least one category of alcohol consumption than for individuals consuming no alcohol (or, for the the Iowa cohort in the Studies in Elderly, those who consumed no alcohol in the preceding month). While the changes were not always statistically significant for each category in each study, the general pattern is very clear. Usually, the lowest risk of death is found in the categories reflecting moderate consumption (1-2 drinks per day or individuals classified as “moderate drinkers”). In the American Cancer Society Study, for example, individuals stating at baseline that they consumed an average of one drink per day had a 16 percent lower death rate (relative risk of 0.84) than non-drinkers over the 12 year follow-up period.

Some of the studies included ex-drinkers and lifetime abstainers in the non-drinking category. The greater the proportion of ex-drinkers in the non-drinking category, the higher the death rate is expected to be for that category. In the British Regional Heart Study, for example, most (71 percent) of the non-drinkers were former drinkers. Therefore, in this study some of the apparent protection from moderate drinking may reflect the higher risk of dying among ex-drinkers who were included in the non-drinking category.

Fortunately, a number of the studies separated the non-drinking category into ex-drinkers and lifetime abstainers. These include the Japanese Physician’s Study, the Busselton Study and the studies from Kaiser Permanente. In each of these studies, moderate drinkers had lower death rates than even lifetime abstainers. Results are the same when only healthy abstainers are included in the non-drinking category.[6-8] Thus, these

studies do not support the contention that the inclusion of ex-drinkers or sick individuals in the non-drinking category is the reason that moderate drinkers have lower rates of cardiovascular and other diseases. Overall, the data indicate that, in comparison with non-drinkers (even when the category is limited to healthy, lifetime abstainers), the risk of dying of any cause is lower for individuals who consume moderate amounts of alcohol.

Does Alcohol Consumption Prolong Life?

In reviewing the results of all of the studies summarized above, two things become clear. First, individuals who reported that they consumed small to moderate amounts of alcohol had lower death rates than non-drinkers. Thus, on the average, the lives of moderate drinkers were prolonged. The second message from the epidemiologic studies is that those individuals who consumed large amounts of alcohol had higher death rates than non-drinkers (and usually much higher than moderate drinkers). Therefore, on the average, the lives of heavy drinkers were shortened.

How sure can we be that it was the consumption of alcoholic beverages that improved mortality rates in these studies? It is always difficult to prove scientifically that some factor that is associated with an effect causes that effect. It is still possible, though unlikely, that it is not alcohol consumption itself but some lifestyle factor associated with the moderate consumption of alcohol that tends to lead to a prolongation of life.

However, the accumulating evidence strongly suggests that it is the consumption of alcoholic beverages that is resulting in the lower risk of death among moderate drinkers in epidemiologic studies. The evidence includes the following factors:

- a marked consistency of the findings of lower total mortality among drinkers in both men and women, in different ethnic groups and in different geographic areas of the world;
- a consistency across different age groups;
- a reduction of coronary atherosclerosis following alcohol intake in animal studies;
- a large number of identified mechanisms (increase in HDL-cholesterol, decrease in platelet aggregation, etc.) by which alcohol would be expected to reduce the risk of heart disease;
- higher death rates among non-drinkers even when ex-drinkers or individuals with other diseases are excluded.

How does one take a reduced relative risk of death for moderate drinkers in epidemiologic studies and translate this into additional days, months or years of life for an individual? How much longer do moderate drinkers live? The calculation of the number of years of life gained by moderate drinkers, while mathematically possible, would involve many assumptions. Such a calculation could not be made with much precision. Further, the estimates would reflect only average effects and would have little meaning for an individual. For example, such estimates could be a combination of a marked prolongation of life for some individuals, no prolongation of life for other individuals and a shortening of life for some who die from alcohol-related accidents. In general, epidemiologic studies provide average risks; they do not allow one to provide an estimate specific to a single individual of the potential benefit (or harm) that might occur from the consumption of alcohol.

Many Lifestyle Factors Affect Longevity

Many factors other than alcohol consumption affect life span. Genetics probably plays the largest role in determining longevity. However, diet, smoking habits, physical activity, obesity, use of seat belts and many other lifestyle factors play a role as well. For example, it has been well demonstrated that certain religious groups such as Seventh-Day Adventists, who tend to be vegetarians and to avoid smoking or drinking alcohol, live longer than the general population.[42,43] Mormons have similar lifestyles and also tend to have longer than average life spans.[44] When thinking of alcohol and life span, it is important to remember that any potential prolongation of life associated with moderate drinking varies markedly according to individual characteristics, especially other health-related habits and medical conditions.

Implications for Individuals

Many factors should be taken into account when someone is considering whether or not to drink alcoholic beverages. Inappropriate use can lead to addiction, as well as to an increased risk of accidents, violence and a number of severe and even fatal diseases. The societal and personal costs of alcohol abuse are great. Women at increased risk of breast cancer may decide that it is preferable not to drink because of the possibility of a relation between alcohol and this disease. Thus, it is not possible to make blanket recommendations for everyone in the population.

Even physicians find it difficult to know whether it is safe (or advisable) for a given individual to drink. We must rely on the answers to questions such as, “Is there a family history of alcoholism?” “Has that individual ever abused alcohol or other substances?” “Does heart disease or certain cancers tend to run in the family?” “What is the individual’s age, sex, body size?” “Is the individual taking medications or ill with a disease?” Such information helps determine how alcohol affects an individual and, to some extent, the risk that the individual will become an abuser of alcohol. We know that, on the average, the consumption of small to moderate amounts of alcohol results in few adverse effects and large beneficial effects in terms of preventing heart disease. However, we can never be sure, *a priori*, that an individual who begins to drink will not become an alcoholic. The decision to drink alcohol must be an individual choice, based not only on average values from epidemiologic studies, but on individual characteristics and the recognition of all of the potential implications of the decision.

Closing Statements

In comparison with non-drinkers, people who consume small to moderate amounts of alcohol have, on average, lower death rates. For heavy drinkers and alcohol abusers, death rates are greater than they are for non-drinkers. Thus, while the consumption of large amounts of alcohol tends to shorten life, the consumption of small to moderate amounts of alcohol is associated with prolongation of life.

References

1. Yano K, Rhoads GG, Kagan A: Coffee, alcohol and risk of coronary heart disease among Japanese men living in Hawaii. *N Engl J Med* 1977; 297:405-409.
2. Dyer AR, Stamler J, Paul O, *et al*.: Alcohol consumption, cardiovascular risk factors, and mortality in two Chicago epidemiologic studies. *Circulation* 1977; 56: 1067-1074.
3. Marmot MG, Shipley MJ, Rose G, Thomas BJ: Alcohol and mortality: A U-shaped curve. *Lancet* 1981; 1: 580-583.
4. Gordon T, Kannel WB: Drinking and mortality. The Framingham Study. *Am J Epidemiol* 1984; 120: 97-107.
5. Klatsky AL, Armstrong MA, Friedman GD: Risk of cardiovascular mortality in alcohol drinkers, ex-drinkers and nondrinkers. *Am J Cardiol* 1990; 66: 1237-1242.
6. Boffetta P, Garfinkel L: Alcohol drinking and mortality among men enrolled in an American Cancer Society prospective study. *Epidemiology* 1990; 1:342-348.
7. Doll R: Alcohol and Health: An Overview. In: *Conference Proceedings, The Medicinal Virtues of Alcohol in Moderation*, Sydney, Australia, October 30-November 1, 1991, pp. 16-43.
8. de Labry LO, Glynn RJ, Levenson MR, *et al*.: Alcohol consumption and mortality in an American male population: Recovering the U-shaped curve - Findings from the Normative Aging Study. *J Studies on Alcohol* 1992;53:25-32.
9. Barbor T, Kranzler H, Louerman R: Social drinking as a health and psychosocial risk factor: Anstie's Limit Revisited. In *Alcoholism*, Marc Galanter, ed Vol. 5, Plenum Press, 1987.
10. St. Leger AS, Cochrane AL, Moore F: Factors associated with cardiac mortality in developed countries with particular reference to the consumption of wine. *Lancet* 1979;1:1017-1020.
11. Shaper AG: Alcohol and mortality: A Review of prospective studies. *Br J Addiction* 1990;85:837-847.
12. LaPorte RE, Cresanta JL, Kuller LH: The relationship of alcohol consumption to atherosclerotic heart disease. *Prev Med* 1980; 9:22-40.
13. Moore RD, Pearson TA: Moderate alcohol consumption and coronary artery disease. *Medicine* 1986; 65:242-267.
14. Ernst N, Fisher M, Smith W, *et al*.: The association of plasma high-density lipoprotein cholesterol with dietary intake and alcohol consumption. The Lipid Research Clinics program prevalence study. *Circulation* 62 (suppl IV) 1980:41-52.
15. Willett W, Hennekens CH, Siegel AJ, Adner MM, Castelli WP: Alcohol consumption and high density lipoprotein cholesterol in marathon runners. *N Engl J Med* 1980; 303:1159-1161.
16. Barrett-Connor E, Suarez L: A community study of alcohol and other factors associated with the distribution of high density lipoprotein cholesterol in older vs. younger men. *Am J Epidemiol* 1982; 115:888-893.
17. Phillips NR, Havel RJ, Kane JP: Serum apolipoprotein A-1 levels. Relationship to lipoprotein lipid levels and selected demographic variables. *Am J Epidemiol* 1982; 116:302-313.
18. Fraser GE, Anderson JT, Foster N, Goldberg R, Jacobs D, Blackburn H: The effect of alcohol on serum high density lipoprotein (HDL). A controlled experiment. *Atherosclerosis* 1983; 46:275-286.
19. Camargo CA Jr, Williams PT, Vranizan KM, Albers JJ, Wood PD: The effect of moderate alcohol intake on serum apolipoproteins A-1 and A-11: A controlled study. *JAMA* 1985; 253:2854-2857.

20. Valimaki M, Nikkila EA, Taskinen MR, Tlikahri R: Rapid decrease in high density lipoprotein subfraction and postheparin plasma lipase activities after cessation of chronic alcohol intake. *Atherosclerosis* 1986; 59:147-153.
21. Castelli WP, Gordon T, Hjortland MC, *et al*.: Alcohol and blood lipids. The cooperative lipoprotein phenotyping study. *Lancet* 1977; 2:153-155.
22.Meade TW, Vickers MV, Thompson SG, Stirling Y, Haines AP, Miller GJ: Epidemiologic characteristics of platelet aggregability. *Br Med J* 1985; 290:428-432.
23. Jakubowski JA, Vaillancourt R, Deykin D: Interaction of ethanol, prostacyclin, and aspirin in determining human platelet reactivity in vitro. *Atherosclerosis* 1988; 8:436-441.
24. Meade TW, Imeson J, Stirling Y: Effects of changes in smoking and other characteristics of clotting factors and the risk of ischaemic heart disease. *Lancet* 1987; 1:986-988.
25. Seigneur M, Bonnet J, Dorian B, *et al*.: Effect of the consumption of alcohol, white wine, and red wine on platelet function and serum lipids. *J Applied Cardiol* 1990; 5:215-222.
26. Renaud SC, Beswick AD, Fehily AM, Sharp DS, Elwood PC: Alcohol and platelet aggregation: The Caerphilly prospective heart disease study. *Am J Clin Nutr* 1992; 55:1012-1017.
27. Sumi H, Hamada H, Tsushima H, Mihara H: Urokinase-like plasminogen activator increased in plasma after alcohol drinking. *Alcohol & Alcoholism* 1988; 23:33-43.
28. Rimm EB, Giovannucci EL, Willett WC, *et al*.: Prospective study of alcohol consumption and risk of coronary heart disease in men. *Lancet* 1991; 338:464-468.
29. Klurfeld DM, Kritchevsky D: Differential effects of alcoholic beverages on experimental atherosclerosis in rabbits. *Exp Molec Pathol* 1981; 34:62-71.
30. Klatsky AL, Armstrong MA: Alcohol beverage choice and coronary artery disease: Do red wine drinkers fare best? *Circulation* 1992; 86:I-464 (abstract).
31. Kimura Y, Okuda H, Arichi S: Effects of stillbenes on arachidonate metabolism in leukocytes. *Biochem et Biophys Acta* 1985; 834:275-278.
32. Siemann E, Creasy L: Natural compound in wine can lower cholesterol levels. *Am J Enology Viticulture* 1992; 43:49-52.
33. Longnecker MP, Berlin JA, Orza M, *et al*.: A meta-analysis of alcohol consumption in relation to breast cancer. *JAMA* 1988; 260:652-656.
34. Willett WC, Stampfer MJ, Colditz GA, Rosner B, Hennekens CH, Speizer FE: Moderate alcohol consumption and risk of breast cancer. *N Engl J Med* 1987; 315:1174-1180.
35. Schatzkin A, Carter CL, Green SB, *et al*.: Is alcohol consumption related to breast cancer? Results from the Framingham Heart Study. *J Natl Cancer Inst* 1989; 81:31-35.
36. *World Health Statistics Annual*, World Health Organization, Geneva. 1989.
37. Friedman LA, Kimball AW: Coronary heart disease mortality and alcohol consumption in Framingham. *Am J Epidemiol* 1986; 124:481-489.
38. Kono S, Ikeda M, Tokudome S, Nishizumi M, Kuratsune M: Alcohol and mortality: A cohort study of male Japanese physicians. *Int J Epidemiol* 1986; 527-532.
39. Shaper AG, Wannamethee G, Walker M: Alcohol and mortality in British men: Explaining the U-shaped curve. *Lancet* 1988; 2:1267-1273.

40. Cullen K: The Busselton Population Studies. In: *Conference Proceedings, The Medicinal Virtues of Alcohol in Moderation*, Sydney, Australia, October 30-November 1, 1991, pp.110-119.
41. Scherr PA, LaCroix AZ, Wallace RB, *et al*.: Light to moderate alcohol consumption and mortality in the elderly. *J Am Geriatr Soc* 1992; 40:651-657.
42. Klatsky AL, Armstrong MA, Friedman GD: Alcohol and mortality. *Ann Intern Med* 1992; 117:646-654.
43. Phillips RL: Role of life-style and dietary habits in risk of cancer among Seventh-Day Adventists. *Cancer Research* 1975; 35:3513-3522.
44. Berkel J, de Waard F: Mortality pattern and life expectancy of Seventh-Day Adventists in the Netherlands. *Int J Epidemiol* 1983; 12:455-459.
45. Enstrom, JE : Health practices and cancer mortality among active California Mormons. *J Natl Cancer Inst* 1989; 81:1807-1814.

Glossary

All-cause mortality rate: the proportion of a population that dies of any cause during a defined period of time (frequently, one year), usually expressed as the number of deaths per year per 100,000 persons in the population. If one wishes to compare mortality rates between two populations, both rates are usually adjusted to a standard age distribution so that any age differences between the populations (such as one population containing a higher proportion of young people than the other) are taken into account.

Case-control study: an epidemiologic study in which a group of individuals with a disease ("cases") are compared with a group without that disease ("controls") in terms of an earlier exposure (e.g., a dietary constituent, alcohol use, cigarette smoking).

Follow-up or cohort study: an epidemiologic study that follows a group of individuals free of disease after obtaining information on certain baseline exposures, such as cigarette smoking or alcohol consumption. The individuals are observed over time for the development of certain diseases. Rates of disease in individuals who were exposed to a certain substance are compared with rates in those unexposed. The results are often expressed as relative risk (see below).

Relative risk: the risk of disease in exposed individuals divided by the risk in non-exposed individuals. If an exposure increases the risk of disease, the relative risk will be greater than 1.0; if it decreases the risk, the relative risk will be less than 1.0.

Risk, or absolute risk: the proportion of a population that gets a disease over a specified period of time.

Appendix: Epidemiologic Concepts of Risk

Relative Risk: When studying the effects of an exposure (such as cigarette smoking) on the risk of getting a certain disease (say, lung cancer), epidemiologists often evaluate the effects in terms of the chance that someone exposed to the agent (e.g., smokers) will get the disease relative to someone not exposed to that agent (e.g. non-smokers). When these two chances are expressed as a ratio, this is known as the relative risk. Studies have indicated that someone who smokes a pack of cigarettes per day for twenty years has at least ten times the risk of getting lung cancer as someone who has never smoked cigarettes. Thus, the relative risk of lung cancer for someone with such a smoking history would be 10.0 or more.

When the relative risk associated with a particular exposure is large (as in the smoking and lung cancer example), it is often described as five or ten times the risk of someone who never smokes, or as a relative risk of 5.0 or 10.0, respectively. Most exposures do not have such large effects, however, and an increased risk is often stated as a certain percent increase. For example, let us assume that consuming a high-fat diet increases the risk of a certain cancer by 30 percent; this is the equivalent of saying that the relative risk of the cancer for someone consuming a high-fat diet, in comparison with someone not consuming such a diet, is 1.3. Similarly,

if consuming a very low-fat diet decreases the risk of the cancer by 30 percent, the relative risk for someone consuming such a diet would be 0.7.

Implications When Considering the Entire Population: When interpreting the importance to the public of various lifestyle exposures that lead to certain diseases, one must take into account the frequency (or rarity) of those diseases in the population. For example, heart disease and other blood vessel diseases such as stroke, heart failure and hypertension, collectively known as cardiovascular disease, account for approximately one million deaths each year in the U.S. An exposure to the population that increases the relative risk of dying from cardiovascular disease by 10 percent (relative risk of 1.1) could theoretically result in 100,000 more deaths in the U.S.. In contrast, an exposure that decreases the relative risk of dying from cardiovascular disease by 10 percent, changing the relative risk to 0.9, could theoretically result in 100,000 fewer deaths. In other words, even small changes in the relative risk of dying from a very common disease could have profound public health implications, simply because of the absolute numbers of people who would be affected. In contrast, even large increases in relative risk for a rare condition would not affect very many people.

Sometimes, a particular exposure reduces the risk of one disease but increases the risk of another. Alcohol consumption is a good example, since it is known to lower the risk of cardiovascular disease but, at least in large amounts, is known to have many adverse effects on health. If we were to say that a substance would lead to a 10-20 percent decrease in the risk of cardiovascular death (a very conservative estimate for alcohol), it would not sound like a very impressive decrease in risk. However, as stated, given the very large number of people who die of cardiovascular disease, a small percentage reduction could mean many fewer deaths (perhaps 100,000 or more) each year in the U.S.

Let us assume that the same substance resulted in a 100 percent increase in the risk of a certain cancer. This would appear to be much a more dramatic effect than was seen for cardiovascular disease. However, if only a few people (say, 5,000) die of this cancer each year, even a 100 percent increase would mean only 5,000 more deaths. While this would be tragic for the individuals involved, the actual numbers would still be only a small proportion of the large number of people whose risk of dying from cardiovascular disease was reduced by the substance. Obviously, in such a case, the ideal would be to be able to identify the people who would develop cancer from the substance so that they could avoid it. Unfortunately, when considering adverse health effects of alcohol, we are usually not able to detect which individuals would suffer such effects.

Considering the Absolute Risk of a Certain Disease: While relative risks are important when studying the relation between different exposures and disease and when answering scientific questions about the causes of disease, an individual may be more interested in the absolute risk of a disease given a certain exposure. For example, if the risk of dying of a disease over a five-year period for a person not exposed to a substance is 100/1,000 (or 10 percent), and the relative risk following exposure is 3.0, the actual risk of dying during the next five years for a person exposed to the substance becomes 300/1,000 (or 30 percent), quite a considerable risk.

On the other hand, if the risk for another disease for a person not exposed to a substance is only 0.1/1,000, and the relative risk following exposure to the substance is 3.0, being exposed to the substance would increase the absolute risk to only 0.3/1,000. In this situation, even with the exposure, the absolute risk

would not be very great.

Implications of Other Factors on Risk: It should be pointed out that the absolute risk for most diseases depends on many factors, especially age. For example, the 5-year risk of death from heart disease for a 40 year old man with a certain set of risk factors may be 50/1,000 (5 percent), but for a 70 year old with the same risk factors it might be 200/1,000 (20 percent). Thus, an increase in the relative risk of any amount has greater importance in people already at increased risk because of age, a strong family history of disease or other factors.