The relationship between alcohol consumption and disease has been explored for centuries. Today, recent studies have linked alcohol consumption to a reduction in risk in coronary heart disease (CHD). CHD is the most common cause of death in America among both men and women. Therefore, the implications of these new findings are promising. However, one must be careful in interpreting the new data on alcohol consumption, since alcohol has been linked to numerous other health problems and alcohol-related events take the lives of thousand of peoples each year. Also, new studies have implied that the benefits of alcohol may be different for an individual who consumes 15 beers during a weekend binge, compared to an individual who has a glass of wine every night with dinner. Lastly, the precise mechanisms by which alcohol lowers the risk of CHD is still not completely understood however great progress in this area has recently occurred.

Women, alcohol and CHD

In 1976, the Nurses’ Health Study began. The study included 121,700 female nurses (aged 34 to 59) living in 11 states who completed a mailed questionnaire which included medical questions, including risk factors for coronary heart disease (CHD). In 1980, a semiquantitative food-frequency questionnaire was added which included questions on the intake of alcoholic beverages. The questionnaire listed beer (12oz), wine (4oz) and liquor (one standard drink) separately, and from these three drinks, total alcohol intake was obtained. Beer was assumed to contain 13.2g of alcohol, wine 10.8g, and liquor 15.1g. At the end of the year, high-density lipoproteins (HDLs) levels were measured. A total of 98,462 nurses returned the diet questionnaire, and after excluding those questionnaires with 10 or more answered questions and those with unrealistic total food scores, 87,526 subjects were left for analysis. Those subjects who reported a myocardial infarction by telephone interview or questionnaire in 1982 were considered as coronary heart disease patients in the study, after it was confirmed they met World Health Organization criteria (1).

The study showed that women with the highest alcohol intake had the lowest risk for coronary heart disease. Their relative risk of 0.4 was half that of women who consumed less than 1.5g per day (0.8). Among subjects consuming 5 to 14.9g per day of alcohol (about 1 drink per day), the risk of CHD decreased by half as compared to nondrinkers. A separate analysis compared women who had greatly decreased their intake of alcohol during the past 10 years with women who had never drank. The analysis showed the relative risk of those who had never drank was 1.1 (1). However, the above data may be skewed by several factors. First, not all risk factors such as diabetes and smoking were equally distributed among the groups studied. Also, heavy drinkers tend not to participate as volunteers in surveys and would be even less likely to complete detailed repeat questionnaires (2). Lastly, alcohol was self-reported on the questionnaire and could have been a major limitation of the study. However, a separate study was done which compared some of the nurses self-reported data with that of their medical records. According to this study, the nurses’ reporting was deemed "highly accurate"(3).
A 1995 12-year prospective study on over 85,000 women showed that all mortality was decreased at certain levels of alcohol intake. More specifically, women who consumed one to three drinks per week had a decreased mortality when compared to those who abstained from alcohol. Despite this, those women who drank more than two drinks per day had a higher mortality rate than all other groups, but in post-menopausal women their risk for CHD went down (4). There is some data that suggests the protective effect of alcohol is somewhat less in women when compared to men (21).

Men, alcohol and CHD

The Health Professionals Follow-up Study is an investigation of the correlation of dietary intakes and heart disease and cancer among health professionals. It is very similar to the Nurses’ Health Study for the exception that it studied 51,529 men, aged 40-75 years and not women. It also differs from the Nurses’ study in that it was controlled for diet and other risk factors, and excluded men with pre-existing cardiovascular disorders. It is similar because in 1986, the cohort members completed a food frequency questionnaire and provided information about medical history, heart disease risk factors, and dietary changes during the previous ten years. The study excluded men with abnormal daily food intakes, those with less than 70 food entries, and those who reported a diagnosis of cancer, myocardial infarction, angina, stroke, coronary artery bypass graft(CABG) or percutaneous transluminal coronary angioplasty(PTCA). The food frequency questionnaire asked about average daily consumption of beer, white wine, red wine, and distilled spirits. The response categories for alcohol consumption had nine possible choices ranging from never to 6 times or more per day. The standard alcohol portions and estimated alcohol contents were similar to those in the Nurses’ Study. Those who had fatal coronary heart disease, non-fatal myocardial infarction, CABG and PCTA between the 1986 questionnaire and January 31, 1988 were considered to have coronary heart disease. All CABGs and PTCAs were self-reported and myocardial infarctions were confirmed if they met World Health Organization criteria (5).

The results of the Study supported the notion that alcohol consumption is inversely related to coronary heart disease. In fact, alcohol drinkers were less likely to have non-fatal myocardial infarctions, fatal coronary heart disease, CABGs and PTCAs. For all total coronary events combined(n=350), there was a highly significant trend in risk with alcohol consumption(chi trend=-3.87,p=0.0001). Control for potential risk factors in multivariate analysis only slightly changed the relative risks. When comparing light drinkers to men who drank 30-50g/day(95% CI 0.37-0.90), the heavier drinkers(>30g/day) had a relative risk for total coronary disease of 0.58. Heavier drinkers compared to non-drinkers had a relative risk of 0.61(95% CI 0.32-1.17). Also, the number of days per week on which alcohol was consumed was also inversely related to risk of CHD. Men who reported drinking on average 3-4 days per week had a relative risk of 0.66((5% CI 0.46-0.96) compared with men who drank on less than 1 day a week (5).

The Helsinki Heart Study shows similar relationships between alcohol and CHD among men to the Health Professionals Study. The randomized, placebo-controlled study
cohort consisted of middle-aged men, 40-55 years. It showed a linear and positive association of alcohol consumption with HDLs and triglycerides. Also, a curvilinear association between alcohol consumption and CHD incidence was found (Fig.1). In addition, those men consuming more than 800g annually, subjects with more than 2-3 drinking occasions per week had a CHD rate of 5.6 per 1000 person years compared to 10.9 per 1000 in those who drank only once or less per week (6). Another study done in France, show a similar correlation with amount of alcohol consumed, in this case wine, to CHD mortality in men. In the French town of Toulouse, where the average diet consists of 383g of wine per day, there were only 78 CHD deaths per 100,000 men. This is in contrast to the town of Lille, where the average wine consumption consists of 267g per day and the CHD mortality is 105 per 100,000 men (7).

Type of alcohol and relation to risk

As to whether one type of alcoholic beverage is more protective than another, most studies in the US indicate that wine, beer and distilled spirits are equally inversely related to CHD (7, 8). However, in one study, beer and wine were associated with a greater reduction in CHD than spirits in non-smokers (9) while in the Health Professionals Follow-Up Study, spirits had the most pronounced inverse association to CHD when compared with beer and wine (5). In another study examining the correlation of alcohol and CHD in women, it was found that the strongest benefit obtained came among wine drinkers consuming 5g of alcohol or more per day, with a relative risk of 0.4 (95% confidence interval, 0.2 to 0.8), and beer drinkers consuming less than 5g per day, with a relative risk of 0.3 (confidence interval 0.2 to 0.7) (1).

The findings that wine drinkers are at lower risk for CHD than liquor drinkers must be interpreted very carefully, for there are many other factors besides alcohol that could influence these findings. With respect to CHD, the traits of wine drinkers are more favorable than those of liquor drinkers, corresponding to the relative risks of CHD death in a Klatsky, Armstrong and Kipp study (10). In this study, two aspects of the data showed a correlation between wine drinking and decreased risk of CHD. The first was that there was a lower risk in those who had never smoked. Secondly, there was an inverse relationship of CHD risk to frequency of wine use among heavier drinkers. There are also other traits, uncontrolled in these studies, such as diet, exercise, and psychological factors. It may be that the lifestyle of wine drinkers is conducive to the reduction of cardiac risk. Or it could be the fact that wine is usually taken with food, slowly and with regularity, thus having a prolonged protective effect (7, 11). However, some argue that the antioxidant value of phenolic substances or bioflavonoids in red wine render it more potent in reducing CHD risk. In both in vitro and in vivo studies, red wine has shown to protect against LDL oxidative damage (12). Yet, in another study, there was no difference in risk between those who drank red wine and those who drank white wine (11).

Mechanism of alcohol’s protective effect
There have been a number of mechanistic possibilities that have emerged from observational and experimental epidemiological studies in the past 20 years. However, it seems that the most plausible mechanism for the beneficial effect of alcohol consumption on CHD, deals with the increase in HDL cholesterol, a plasma lipoprotein. HDL cholesterol, which is produced primarily in the liver and intestines, is released into the blood stream. In the blood, HDL binds to cholesterol and brings it back to the liver for elimination or reprocessing, thereby lowering total cholesterol levels in the body tissues. Also, by reducing the cholesterol buildup on the arterial wall, it hinders the atherosclerotic process. Lastly, HDL may play a role in preventing the oxidation of LDL which is one of the steps in cholesterol plaque buildup in the arterial lumen (12).

There have been numerous studies that show that alcohol intake raises HDL cholesterol, however, there has been some argument as to which HDL component provides the beneficial effects. One experimental study shows that 39g/day of alcohol for 6 weeks, increased HDL by 17% (13). HDL cholesterol is composed of two subfractions: HDL2 and HDL3. Each fraction has a slightly different effect. A 1988 study by Diehl, Fuller, and Mattock, suggested that moderate alcohol consumption raised HDL3, but not HDL2 (14). Therefore concluding that only HDL2 has the protective effects. A study by Ballantyne supports this theory. The study found lower levels of HDL2 but not HDL3 among 31 survivors of myocardial infarction (15). In a contradicting case-matched control study, both HDL2 and HDL3 were elevated in relation to amounts of daily ethanol consumption up to three drinks per day. In addition, both subfractions were associated with a decreased risk of myocardial infarction (16). Other substances such as apoproteins A-1 and A-2, which are associated with HDL particles, have been shown to increase with alcohol consumption, but their direct influence on CHD have not been extensively studied (17).

Another potential mechanism that may contribute to the protective effect of light to moderate alcohol consumption include alterations in factors affecting blood clotting. Clot formation is an important step in the development of a myocardial infarction, since it is the formation of such a clot which eventually occludes the arterial lumen, limiting the oxygen perfusion of adjacent cardiac tissue. One study suggests that alcohol consumption increases the levels of a clot-dissolving enzyme, tissue plasminogen activator (TPA) (18). Another hemodynamic mechanism involves a decrease in platelet aggregability. One study done on rats showed that an addition of 4-6% ethanol to drinking fluid reduces platelet aggregation, an effect that occurs rapidly but is also lost rapidly when alcohol is degraded in the body (19). It has also been postulated that alcohol consumption lowers the level of fibrinogen, a potent risk factor for coronary heart disease (20).

Drinking to your health?

There is substantial current evidence showing the inverse relationship between CHD and alcohol. So does this mean we can drink our way to a healthier heart? Maybe. Yet, this does not mean alcohol is correlated with overall health. In fact, at levels of alcohol consumption which reduce the risk of CHD, there is an increase risk of hemorrhagic strokes, hypertension, cancer of the esophagus, breast cancer, colorectal adenoma and
liver cirrhosis (21). Being that alcohol disease relationships are so complex, alcohol consumption should not be viewed as a person’s main preventive strategy for fighting coronary heart disease. On the other hand, alcohol consumption should not necessarily be viewed as an unhealthy behavior.

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