Editorial: Alcohol, the Heart, and Health

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There is considerable evidence that both nondrinkers (abstainers) and heavy drinkers have higher rates of cardiovascular disease in general and coronary heart disease in particular than do light or moderate drinkers. This U-shaped relationship has been interpreted as showing that light or moderate alcohol intake is protective against coronary heart disease and may in general be beneficial to health. Two recent reviews of the subject present the available information, and it seems clear that, despite inconsistencies within many of the individual investigations, there is a remarkable consistency of findings in populations that differ widely in their composition and characteristics.[1,2] "The weight of the evidence suggests a real protective effect," concludes the most recent and comprehensive review.[2] With this overwhelming compilation of evidence of the beneficial effects of alcohol on coronary heart disease, one might well ask on what grounds these conclusions can be challenged. The findings of the British Regional Heart Study, a large prospective study of cardiovascular disease in middle-aged men drawn from 24 towns, are the major source of the initiative to question the beneficial effects of alcohol and to suggest that there may have been an overenthusiastic acceptance of the protective role of moderate alcohol intake in coronary heart disease.

The British Regional Heart Study showed that, at least among middle-aged British men, a large proportion of abstainers (nondrinkers) are ex-drinkers.[3] This does not imply that they were previously heavy drinkers or alcoholics, but merely that they would at a younger age have been classified as drinkers. These ex-drinkers have many characteristics likely to increase their morbidity and mortality. They are older than drinkers and they include a higher percentage of manual workers and unmarried men. They have the same high percentage of current cigarette smoking as moderate or heavy drinkers and a similar prevalence of hypertension and obesity. They have the highest rate of coronary heart disease as measured by a standardized chest pain questionnaire, electrocardiogram, and recall of physician's diagnosis. They also have higher prevalence rates of high blood pressure, diabetes, gall bladder disease, and bronchitis, and the highest rates of regular medication.[3] There seems good reason not to use the general category of nondrinkers as a baseline against which to measure the effects of alcohol consumption. Few studies have examined closely the nature and characteristics of nondrinkers, and although it may well be that in some countries this group is predominantly made up of lifelong abstainers, it is more likely that this group will include a significant proportion of men who previously were drinkers. This is particularly true as the group under study increases in age.

The British Regional Heart Study went on to show that men who were heavier drinkers were more likely to reduce their alcohol consumption as they grew older.[4] Men who were told by their doctor that they had developed coronary heart disease were more likely to reduce their alcohol intake than were men who remained free of coronary heart disease. The study suggested a strong downward

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drift from heavy or moderate drinking to occasional drinking or abstinence under the influence of accumulating ill health not necessarily related to alcohol intake.

On follow-up of the men in the British Regional Heart Study, the data showed a U-shaped relationship between alcohol intake and total mortality and an inverse relationship with cardiovascular mortality, even after age, smoking, and socioeconomic class had been taken into account.[5] These mortality patterns were seen in all smoking categories. Ex-smokers who were also ex-drinkers had the highest mortality. The latter phenomenon strongly suggests that giving up both drinking and smoking might be related to increasing ill health. Most critically, in this study the alcohol–mortality relationships described were present only in men with cardiovascular or cardiovascular-related doctor-diagnosed illnesses present at initial examination. The data suggest that the observed alcohol–mortality relationships are produced by disease present at the time of screening, when the alcohol category is decided, and by the prior movement of men with such disease into occasional drinking or nondrinking categories.

If this is the case, the hypothesis of a protective effect of alcohol, which is strongly based on the differences between nondrinkers and drinkers and which ignores the dynamic relationship between ill health and drinking behavior, is poorly founded.[6] In the face of the considerable evidence regarding the protective effects of moderate alcohol consumption, it is clear that what matters is not the finding of U-shaped curves for total mortality or inverse relationships with cardiovascular or coronary heart disease, but the interpretation placed on these findings. Critical to the interpretation appears to be an understanding of the nature of nondrinkers and an awareness that some people may change their drinking habits after receiving a diagnosis or developing the symptoms of disease, particularly cardiovascular disease. This dynamic aspect of drinking behavior has been emphasized by the Alameda County Study in the United States.[7]

The issue of moderate drinking and coronary heart disease mortality is addressed in this Journal in an examination of data from the National Health and Nutrition Examination Survey, 1971 through 1974 (NHANES I), and the NHANES Follow-up, 1982 through 1984.[8] The age-adjusted mortality rate of moderate drinkers (those who consumed up to two drinks per day, as assessed at baseline) from coronary heart disease was 50% lower than in nondrinkers and was 60% lower in the heavy drinkers (more than two drinks per day). When subjects in poor health at baseline (about 15% of the White men) were excluded from the analysis, an accelerated time-to-failure (age at death) model showed that moderate drinking increased the time until death from any cause by about 3%, whereas heavy drinking reduced the time to death by 2%. It is presumed that the benefit associated with moderate drinking is due to a protective effect on coronary heart disease mortality. No benefit was seen for White women. The statistical modelling used in this study will not communicate itself readily to most readers, and the author cautions that drinking at baseline may not be an adequate measure of drinking during the subsequent decade; nor, one might add, is it an adequate proxy for drinking patterns prior to the baseline examination.

Another NHANES paper is based on the 1976 through 1980 data and examines the association between alcohol consumption and high-density lipoprotein cholesterol concentration in a representative sample of the United States adult population.[9] There is already a considerable literature on this subject,[10–12] because it is this lipoprotein moiety that is presumed to constitute the critical protective factor associated with alcohol intake. Linn et al.'s paper deals particularly with the findings in women and African Americans. Not surprisingly, high-density lipoprotein cholesterol levels were higher in drinkers than in nondrinkers in all sex–race strata, and mean levels increased consistently with increased frequency of alcohol consumption even after adjustment for confounding variables. Reviewing the issue of alcohol, high-density lipoprotein cholesterol, and coronary heart disease, the authors agree with earlier comments that it would be undesirable to recommend increased alcohol use as a means of reducing coronary heart disease risk.[1,13] They draw attention to the other effects of alcohol, cardiovascular and noncardiovascular, as well as the social costs of increased alcohol intake; they also point out that it is unclear whether manipulation of the high-density lipoprotein cholesterol level by alcohol consumption would alter the risk of coronary heart disease.

In a third paper, Peele expresses a strong opinion that although the fact that alcohol reduces the risk of coronary heart disease is well established, the public health field has not been able to accept, digest, and use this finding.[14] The author considers that a strong "cultural set of antialcohol moralism" is working against frank scientific discussions in the United States of the benefits for the cardiovascular system and other benefits from drinking. He regards this antialcohol moralism as "inconsistent with public health goals."

Although Peele's intention, viewed in the best light, is to uphold the occasional drinking of moderate amounts of alcohol, the overall effect of the paper is to encourage and recommend the taking of larger amounts, that is, three to five drinks per day. Such a recommendation fails to take into account the critical issue in the debate over alcohol and health, which is the role of alcohol in areas other than coronary heart disease. There is currently wide acceptance, even in the field of public health, that light or moderate drinking may be associated with a reduced rate of coronary heart disease, although there is still argument regarding the interpretation of the association in terms of causality. The concern that is being expressed, even by those who regard the relationship as causal, is not that such a finding should not be made public but that the other, and considerable, effects of alcohol should be properly taken into account. The work based on the American Cancer Society prospective study shows clearly that at the levels of alcohol intake associated with the lowest rates of mortality from coronary heart disease, mortality from all causes is already beginning to increase.[15] To this finding can be added alcohol-associated morbidity and the behavioral and social aspects of alcohol intake. It seems unreasonable to ignore completely the issue of overall health.

Those involved in public health medicine have good reason to be concerned by the patterns of alcohol intake in our society. Although alcohol may offer a limited degree of protection from coronary heart disease, it appears to do so at levels of consumption that range from moderate (three to five drinks per day) to heavy (six or more drinks per day). Any benefit with regard to coronary heart disease must be far outweighed by the consequences of regular alcohol use at these levels. There is no denying the considerable pleasure associated with occasional and light drinking in society, and this editorial is not concerned with total abstinence. However, to take a hard view of the problems associated with even moderate alcohol consumption on a regular basis seems no dereliction of public health duty. □

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Commentary: Alcohol, the Heart, and Public Policy

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Light to moderate drinkers have substantially lower rates of cardiovascular mortality and mortality from all causes than do nondrinkers or heavy drinkers. This finding has been observed repeatedly in several dozen epidemiologic studies using a variety of designs.[1] Recent research has added further persuasive evidence to support a causal interpretation of this association.

In epidemiological studies, classification of moderate alcohol consumption ranges from half a drink per day (or less) in some studies up to six drinks a day in others. A 5-oz glass of wine, a 12-oz can of beer, or a shot (1.5 oz) of spirits contains about 13 g to 15 g of alcohol. We consider moderate drinking to be one to two drinks per day for a man and perhaps somewhat less for a woman. For most individuals, this is a safe definition. However, tolerance to alcohol depends on age, sex, body size, and cultural situation; therefore, no single global definition of "moderate" can be made. History of past consumption, rate of consumption, and proximity to meals also alter metabolism of alcohol.

In widely disparate populations, from across Europe and North America to Australia and Thailand, a consistent 20% to 40% reduction in coronary disease has been reported among moderate drinkers. This association is not in dispute. Although a causal interpretation is most plausible, a few investigators have advocated the alternative explanation that the comparison group of nondrinkers is at higher risk of coronary disease because that category includes covert alcohol abusers and those who quit drinking because of ill health.[2]

Work from our group[3,4] and from others strongly refutes these theories. We compared estimated average alcohol intake from our questionnaire with actual intake from 14 days of diet records. We found, in both men and women, a corre-

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Editor's Note. This commentary was written independently, if inadvertently so, of the editorial by Shaper and the article by Peele in this Public Health Policy Forum.