References

1. Marmot M, Brunner E. Alcohol and cardiovascular disease: the status of the U-shaped curve. *BMJ.* 1991;303:565.
2. Beaglehole R, Jackson R. Alcohol, cardiovascular diseases and all causes of mortality: a review of the epidemiological evidence. *Drug Alcohol Rev* 1992;II:275–290.
3. Wannamethee G, Shaper AG. Men who do not drink: a report from the British Regional Heart Study. *Int J Epidemiol.* 1988; 17:307–316.
4. Wannamethee G, Shaper AG. Changes in drinking habits in middle-aged British men. *J R Coll Gen Pract.* 1988;38:440–442.
5. Shaper AG, Wannamethee G, Walker M. Alcohol and mortality in British men: explaining the U-shaped curve. *Lancet.* 1988; ii:1267–1273.
6. Shaper AG. Alcohol and mortality: a review of prospective studies. *Br J Addict.* 1990;85:837–847.
7. Lazarus NB, Kaplan GA, Cohen RD, Leu D-J. Changes in alcohol consumption and risk of death from all causes and from ischaemic heart disease. *BMJ.* 1991;303: 553–556.
8. Coate D. Moderate drinking and coronary heart disease mortality: evidence from NHANES I and the NHANES I follow-up. *Am J Public Health.* 1993;83:888–890.
9. Linn S, Carroll M, Johnson C, Fulwood R, Kalsbeek W, Briefel R. High-density lipoprotein cholesterol and alcohol consumption in US White and Black adults: data from NHANES II. *Am J Public Health.* 1993;83:811–816.
10. Criqui MH, Cowan LD, Tyroler HA, et al. Lipoproteins as mediators for the effects of alcohol consumption and cigarette smoking on cardiovascular mortality: results from the Lipid Research Clinics Follow-up Study. *Am J Epidemiol.* 1987;126:629–637.
11. Diehl AK, Fuller JH, Mattock MB, Salter A, el-Gohari R, Keen H. The relationships of high density lipoprotein subfractions to alcohol consumption, other lifestyle factors, and coronary heart disease. *Atherosclerosis.* 1988;68:145–153.
12. Linn S, Fulwood R, Rifkind B, et al. High density lipoprotein cholesterol levels among U.S. adults by selected demographic and socioeconomic variables: the second National Health and Nutrition Examination Survey 1976–80. *Am J Epidemiol.* 1989;129:281–294.
13. Blackburn H, Wagenaar A, Jacobs DR Jr. Alcohol: good for your health? *Epidemiology.* 1991;2:230–231.
14. Peele S. The conflict between public health goals and the temperance mentality. *Am J Public Health.* 1993;83:805–810.
15. Boffetta P, Garfinkel L. Alcohol drinking and mortality among men enrolled in an American Cancer Society prospective study. *Epidemiology.* 1990;1:342–348.

Commentary: Alcohol, the Heart, and Public Policy

Meir J. Stampfer, MD, Eric B. Rimm, ScD, and Diana Chapman Walsh, PhD

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-

Meir J. Stampfer and Eric B. Rimm are with the Department of Epidemiology, and Diana Chapman Walsh is with the Department of Health and Social Behavior, Harvard School of Public Health, Cambridge, Mass. Meir J. Stampfer is also with the Channing Laboratory, Department of Medicine, Brigham and Women's Hospital and Harvard Medical School, Boston, Mass.

Requests for reprints should be sent to Meir J. Stampfer, MD, Channing Laboratory, 180 Longwood Ave, Boston, MA 02115.

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.

lation of approximately .9 between alcohol consumption estimated from the questionnaire and that measured from diet records.[5] Furthermore, we found highly significant correlations between reported alcohol intake and high-density lipoprotein cholesterol in both groups.

For the heaviest alcohol users, a questionnaire or interview may not provide valid information. However, alcohol abusers are far less likely than others to participate in epidemiological studies and therefore the purported presence of heavy drinkers in the nondrinker category is an untenable explanation for the inverse association.

In large and detailed studies, one also may compare moderate alcohol consumption with very light consumption. In our analyses of 87 000 nurses[3] and 51 000 male health professionals[4] we found a significant inverse association with increasing alcohol consumption even with total abstainers excluded.

In our two large cohorts we also tested the second, related, noncausal explanation, that men and women with preexisting disease abstain from alcohol. If true, this would tend to produce an artifactual association between abstinence and higher risk of coronary disease.

In most prospective studies, participants with diagnosed coronary disease are excluded at the start of follow-up. However, those with risk factors such as diabetes, hypertension, or hypercholesterolemia are usually not excluded. As expected, we did find a higher prevalence of these conditions among the abstainers in our cohorts. However, in alternative analyses excluding participants with those risk factors, we still found a strong inverse association between alcohol and coronary disease.

Other prospective studies have reported similar findings. Although Shaper[2] originally did not find a similar association after excluding men with preexisting disease in the British Regional Heart Study, with additional follow-up a reduction in ischemic heart disease was found even among men free of existing disease.[6] Overall mortality was not reduced, but this could be explained in part by the categorization of those reporting from one half up to six drinks per day as "moderate" drinkers. Further, in this population of 7735 men, the strong correlation between drinking and smoking makes it difficult to obtain precise estimates of the independent effect of drinking. The data from 276 802 men enrolled in the American Cancer Society prospective study provide much more convincing evidence. They show a maximal reduction in total mortality at one to two drinks per day among all participants, both before and after excluding those who were ill at baseline.[7]

In the Kaiser-Permanente study of over 120 000 persons, Klatsky and Armstrong[8] reported reduced coronary mortality among drinkers compared with lifelong nondrinkers. This important finding tends to refute the hypothesis that the protective effect is an artifact caused by the inclusion in the nondrinker group of moderate drinkers who quit because of disease. Similarly, in our cohorts, we excluded men and women with a marked decrease in alcohol intake over the previous 10 years; in those analyses the substantial reduction in risk among the moderate drinkers remained apparent.

Recently, attention has focused on the possible differences in the effect of different alcoholic beverages, particularly the purported special benefits of red wine. The epidemiological evidence suggests that all alcoholic beverages are similarly protective. Some studies find wine more protective; others, beer or spirits. For example, in the Health Professionals Follow-Up Study,[4] men consuming two drinks per day of spirits were at slightly lower risk than those who consumed alcohol from other sources. In the Nurses' Health Study,[3] wine was found to be a bit more protective, and in an earlier prospective study, Yano et al.[9] reported the lowest risk for moderate beer drinkers. Frankel et al.[10] reported on specific components of red wine that may act as antioxidants to reduce atherosclerosis. However, in a recent update from the large Kaiser cohort, Klatsky and Armstrong[8] found that white wine drinkers had a slight advantage over red wine drinkers, though both groups were at reduced risk compared with nondrinkers.

The best documented mechanism of the cardioprotective effect of alcohol is that it raises the concentration of high-density lipoprotein (HDL).[11] At one time, it was believed that alcohol raised only the HDL-3 subfraction and that only the HDL-2 subfraction was protective. Both of these beliefs are incorrect. Alcohol increases both subfractions, but it raises HDL-3 more than it does HDL-2. Both subfractions are associated with decreased risk, and fractionating high-density lipoprotein provides little or no additional information about risk beyond that derived from total high-density lipoprotein.[12]

Other mechanisms are likely. Alcohol intake decreases platelet aggregability and causes a marked short-term increase in tissue-type plasminogen activator.[13] Both effects point toward an acute reduction of clot formation and hence a decrease in risk. These mechanisms are consistent with a recent case-control study that found a short-term protective effect of alcohol consumption in addition to a benefit of habitual moderate intake.[14]

A protective effect of moderate alcohol consumption is well established from epidemiological data and plausible biological mechanisms. What are the public health implications of this finding? In a 1979 editorial, Castelli[15] concluded that although two drinks per day appear to be protective, "with 17 million alcoholics in this country we perhaps have a message for which this country is not yet ready."

Is this a message for which the country ought to ready itself? If the medical and health establishments were to advocate regular drinking of small amounts of alcohol, would the risk of increased problem drinking outweigh the benefit of healthier hearts? Whose risk would increase and who would benefit? Can clinicians correctly identify patients for whom such advice would be contraindicated?

People—and not only alcoholics—often experience unpleasantness, and occasionally very much worse, as a result of their drinking.[16] What we see far less clearly is how various factors combine to produce these bad outcomes—what the risk (and protective) factors are that explain why in some circumstances some people get into trouble with alcohol whereas others escape. Roughly half of American men who qualify as heavy drinkers never experience problems in connection with their alcohol use. Of those whose episodic abuse does lead to serious trouble, about half are not habitual heavy drinkers.[17]

Studies have identified markers of substance abuse in adolescents: early trial and initiation; strong peer influences; nonconformity and rebelliousness; low achievement in school; lack of family limit-setting, involvement, and support.[18] Among adults, being male, being younger than 30 years, having lower income, being in the working class, and coming from a family with a history of alcoholism increase the risk of heavy drinking, alcohol abuse, and alcoholism. Paradoxically, some of the same high-risk groups have higher proportions of abstainers and would, as a consequence, be particular targets of the prodrinking message. Nu-

merous theories—genetic, metabolic, psychological, social, cultural, and addiction-based—have been advanced to explain the onset and uneven course of problem drinking, alcohol abuse, or alcoholism. But no theory or combination of theories adequately explains what many scholars now believe are diverse phenomena. Meanwhile, numerous studies have demonstrated that physicians frequently miss the diagnosis even of severe alcoholism.[19]

In the United States, less than 10% of the population reports drinking more than two drinks per day, the cutoff for "heavy drinking" in national survey research. This means that "moderate" drinkers, because of their much greater numbers, probably account for well over half of all alcohol problems, a finding that led researchers at the Institute of Medicine to observe in a groundbreaking report that "if all the clinically diagnosed alcoholics were to stop drinking tomorrow, a substantial fraction of what we understand as alcohol problems would still remain."[20] The statement heralded a conceptual watershed in the way the world thinks about alcohol control, diverting the focus from treating alcoholics toward what was termed a new "public health" approach. Two key assumptions behind that approach are especially pertinent here.

First, public health thinking implies a systems approach, the object of which is to mobilize a range of change strategies—education, moral suasion, and formal rules and laws—in an integrated program of controls aimed at host, agent, and environment. In this approach, a united front and the absence of mixed messages become very important, because the hope is to create a constancy of messages and policies.[21] The possibility that a daily dose of alcohol might be cardioprotective is a perturbation that threatens to complicate or dilute messages designed to alert drinkers to risks.

The second important assumption behind the public health approach to alcohol control is that it seeks to move the whole consumption curve toward lower per capita consumption overall.[22] The hope is that alcohol problems will, as a consequence, abate. Again, the emergence of scientific evidence that alcohol may be salutary seems to fly in the face of this goal. It suggests that health risks increase at both tails of the consumption curve, so that wholesale shifting of the curve could put a subgroup of underconsumers at risk for heart disease.

Should we therefore promote the consumption of small amounts of alcohol? In theory, this would increase the "social availability" of alcohol—the perception among the public that drinking is normative.[23] We simply do not know what the effect might be on overall consumption rates and on alcohol-related problems. But we do have fairly robust evidence that problems decrease with reductions in the physical and economic availability of alcoholic beverages. Problem indicators decline when sales of alcohol are sharply curtailed or prohibited and increase again when restrictions on access are relaxed. Raising the taxes on alcohol reduces consumption, even among heavy drinkers, and at least some associated injuries and deaths.[24,25] Increasing the minimum legal drinking age seems to reduce highway crashes.[26]

The public health response to alcohol abuse is far from optimal. Both the reach and the range of alcohol treatment strategies need to be expanded. We need more inventive strategies to get people with nascent problems to notice them earlier and avail themselves of low-intensity interventions and supports, which must be made more diverse.

We also need to develop innovative programs to reach people where they study, live, and work and through the mass media. The focus of such efforts should be to change public awareness and behavior concerning alcohol-associated risk. The messages should promote norms that would presumably be protective against alcohol problems:

- It is always acceptable to decline a drink.
- It is never acceptable to become really intoxicated.
- It is never acceptable to drink in situations in which alcohol is associated with significant risk—during pregnancy, while taking medications, before driving a car or using other dangerous machinery, at work, or while engaged in other pursuits that demand coordination and full possession of one's faculties.

It is impossible to predict with confidence what the public health impact might be of an effort to promote the regular consumption of small amounts of alcohol. Large longitudinal studies would be required before we could safely say who might be at risk of progressing to heavier or hazardous drinking. Resources for such research have not been available. Comprehensive cost–benefit analyses are needed to sort out the benefits and risks to individuals and to society. Research is needed, too, to clarify whether the protective effect of alcohol is general or whether the message should apply only to a subgroup. Even with better risk-factor models, we would still be hard pressed to foresee situations, which unfortunately are not uncommon, in which episodes of alcohol abuse among usually moderate drinkers might result in the injury or death of the drinker or someone else.

If a prodrinking campaign were to be mounted, it should certainly seek to avoid communicating the message to certain groups: anyone with a family history of alcoholism, people younger than age 21, and pregnant women. It should also address *all* risk factors for cardiovascular disease, since the others—such as smoking and hypertension—can be reduced by individuals without putting the health of others at risk. But our society is so lacking in effective social controls on alcohol abuse and pays such a heavy price for its inadequate response that, although a policy opposing moderate alcohol consumption may be inadvisable, the thought of a public policy promoting alcohol consumption runs strongly against the grain, however much it might capture at least some hearts. □

References

1. Moore RD, Pearson TA. Moderate alcohol consumption and coronary artery disease. *Medicine.* 1986;65:242–267.
2. Shaper AG. Alcohol and mortality: a review of prospective studies. *Br J Addict.* 1990;85:837–847.
3. Stampfer MJ, Colditz GA, Willett WC, Speizer FE, Hennekens CH. A prospective study of moderate alcohol consumption and the risk of coronary disease and stroke in women. *N Engl J Med.* 1988;319: 267–273.
4. Rimm EB, Giovannucci EL, Willett WC, et al. Prospective study of alcohol consumption and coronary disease in men. *Lancet.* 1991;338:464–468.
5. Giovannucci EL, Colditz GA, Stampfer MJ, et al. The assessment of alcohol consumption by a simple self-administered questionnaire. *Am J Epidemiol.* 1991;133: 810–817.
6. Shaper AG. The British Regional Heart Study. *CVD Epidemiology Newsletter.* 1992/1993;48:27.
7. Boffetta P, Garfinkel L. Alcohol drinking and mortality among men enrolled in an American Cancer Society prospective study. *Epidemiology.* 1990;1:342–348.
8. Klatsky AL, Armstrong MA. Alcoholic beverage choice and coronary artery disease: do red wine drinkers fare best? *Circulation.* 1992;86(suppl 1):I–464. Abstract.
9. 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.

10. Frankel EN, Kanner J, German JB, Parks E, Kinsella JE. Inhibition of oxidation of human low-density lipoprotein by phenolic substances in red wine. *Lancet.* 1993;341: 454–456.
11. Veenstra J. Effects of alcohol on blood lipids and platelet function. In: Veenstra J, van der Heij DG, eds. *Alcohol and Cardiovascular Disease.* Wageningen, The Netherlands: Pudoc Press; 1992:65–69.
12. Stampfer MJ, Sacks FM, Salvini S, Willett WC, Hennekens CH. A prospective study of cholesterol, apolipoproteins, and the risk of myocardial infarction. *N Engl J Med.* 1991;325:373–381.
13. Kluft C, Jie AFH, Kooistra PK, et al. Alcohol and fibrinolysis. In: Veenstra J, van der Heij DG, eds. *Alcohol and Cardiovascular Disease.* Wageningen, The Netherlands: Pudoc Press; 1992:45–64.
14. Jackson R, Scragg R, Beaglehole R. Does recent alcohol consumption reduce the risk of acute myocardial infarction and coronary death in regular drinkers? *Am J Epidemiol.* 1992;136:819–824.
15. Castelli WP. How many drinks a day? *JAMA.* 1979;242:2000. Editorial.
16. *Seventh Special Report to Congress on Alcohol and Health.* Washington, DC: US Dept of Health and Human Services, National Institute on Alcohol Abuse and Alcoholism; 1990.
17. Fingarette H. *Heavy Drinking.* Berkeley, Calif: University of California Press; 1988:54.
18. Jessor R, Jessor S. *Problem Behavior and Psychosocial Development: A Longitudinal Study of Youth.* New York, NY: Academic Press; 1977.
19. Walsh DC, Hingson RW, Merrigan DM, et al. The impact of a physician's warning on recovery after alcohol treatment. *JAMA.* 1992;267:663–667.
20. Moore MI, Gerstein DR. *Alcohol and Public Policy: Beyond the Shadow of Prohibition.* Washington, DC: National Academy Press; 1981.
21. Mosher JI, Jernigan DH. New directions in alcohol policy. *Annu Rev Public Health.* 1989;10:245–279.
22. Bruun K, Edwards G, Lumio M, et al. *Alcohol Control Policies in Public Health Perspective.* Helsinki, Finland: The Finnish Foundation for Alcohol Studies; 1975.
23. Moskowitz JM. The primary prevention of alcohol problems: a critical review of the research literature. *J Stud Alcohol.* 1989; 50:54–88.
24. Farrell S. *Review of National Policy Measures to Prevent Alcohol-Related Problems.* Geneva, Switzerland: World Health Organization; 1985.
25. *Drinking Age Laws: An Evaluation Synthesis of Their Impact on Highway Safety.* Washington, DC: US Government Accounting Office; 1987.
26. Institute of Medicine. *Broadening the Base of Treatment for Alcohol Problems.* Washington, DC: National Academy Press; 1990.

Submissions Invited for Public Health Policy Forum

The Journal department "Public Health Policy Form" is intended to present divergent views on important public health policy issues in a more extensive format than the Journal usually allows. Three kinds of material appear in the Forum: articles, not exceeding 4500 words; commentaries, not exceeding 2500 words; and editorials. Customarily, commentaries and editorials are solicited by the Journal's Contributing Editor or Editor. Articles for the Forum are selected from those submitted generally to the Journal or specifically to the Forum. In general, peer review is sought.

Those wishing to submit articles directly to the Public Health Policy Forum should send them to the Journal office as follows:

George A. Silver, MD, MPH
Contributing Editor, *AJPH* Public Health Policy Forum
1015 Fifteenth Street, NW
Washington, DC 20005

Please send five copies of the manuscript and follow the Journal's guidelines, "What *AJPH* Authors Should Know," printed in each issue of the Journal.

The scholarly merit and the scientific accuracy of all submissions are considered. Additional criteria are relevance to an important policy issue, timeliness, and clarity and coherence of the policy argument. Brevity also helps considerably.