against women as an important underlying factor in women's mental health problems.

The release of the report was timed to coincide with the International Campaign Against Violence Against Women, which began on Nov 25, the International Day for the Elimination of Violence Against Women, and ends on Dec 10, the International Human Rights Day.[9] This 16-day campaign links violence against women and human rights, emphasising that all forms of violence, whether perpetrated in the public or private sphere, are a violation of human rights.[10]

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I declare that I have no conflict of interest.

1 Brundtland GH. Preface. In: Krug EG, Dahlberg LL, Mercy JA, Zwi AB, Lozano R, eds. World report on violence and health. Geneva: WHO, 2002.
2 WHO. WHO multi-country study on women's health and domestic violence against women. Geneva: World Health Organisation, Nov 24, 2005.
3 Garcia Moreno C. WHO recommendations put women first. *Lancet* 1999; **354:** 2075–76.
4 World Bank. World development report: investing in health. Washington DC: Oxford University Press, 1993: 50.
5 Campbell JC. Health consequences of intimate partner violence. *Lancet* 2002; **359:** 1331–36.
6 Fathalla MF. Women's health: an overview. *Int J Gynecol Obstet* 1994; **46:** 105–18.
7 Garcia-Moreno C. Dilemmas and opportunities for an appropriate health service response to violence against women. *Lancet* 2002; **359:** 1509–14.
8 The Lancet. Zero tolerance for domestic violence. *Lancet* 2004; **364:** 1556.
9 United Nations Fund for Women (UNIFEM). Not a minute more—a call to end violence against women. http://www.unifem.org/campaigns/november25/ (accessed October 17, 2005).
10 Cook RJ, Dickens BD, Fathalla MF. Reproductive health and human rights-integrating medicine, ethics and law. Oxford: Oxford University Press, 2003: 385–92.

# Alcohol and ischaemic heart disease: probably no free lunch

A quarter of a century ago, *The Lancet* published an ecological observation by St Leger and colleagues of "a strong and specific negative association between ischaemic heart disease (IHD) deaths and alcohol consumption".[1] The authors attributed the association to wine consumption and concluded that: "If wine is ever found to contain a constituent protective against IHD then we consider it almost sacrilege that this constituent be isolated. The medicine is already in a highly palatable form."

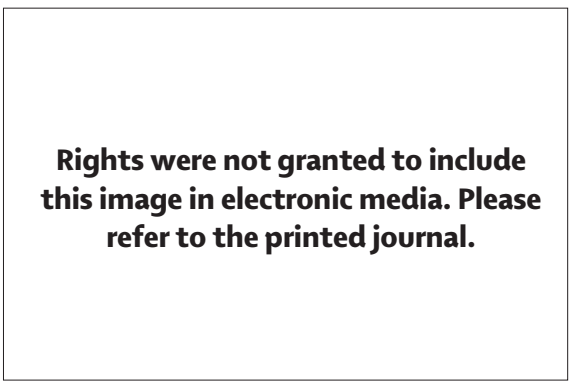
This study was among a number published during the 1970s and 1980s supporting a rare good-news public-health story. In 1990, Ellison's[2] provocative editorial entitled "Cheers", encapsulated what remains the dominant belief today that "small to moderate amounts of alcohol are good for your health". The benefit is attributed mainly to a protective effect of light to moderate drinking on IHD risk that is believed to outweigh adverse health effects in this window of modest consumption. A meta-analysis confirmed these earlier observations: self-reported consumption of between one and three standard alcoholic drinks (a standard drink included about 10 g of alcohol) a day is associated with a 20–25% reduction in the risk of IHD.[3]

We believe it timely to challenge this belief in a "window of protection" given the increasing evidence of uncontrollable confounding in non-randomised studies of IHD.[4,5]

The counter argument to the apparent coronary protection has attributed the observed protective association to misclassification and confounding. Shaper and colleagues proposed that ex-drinkers who stopped drinking because of cardiovascular-related illness (sick quitters) were often misclassified with never drinkers,[6] thus artifactually raising the coronary risk in non-drinkers. This hypothesis has now been discarded as new studies report a protective association after excluding ex-drinkers.[7]

The more likely explanation for an artifactual association—uncontrolled confounding—has been too readily dismissed by many researchers, including ourselves.[7,8] But this year, Timothy Naimi and colleagues have revived the confounding hypothesis using data on cardiovascular risk factors from a telephone survey of over 200 000 adults in the USA.[4] Of 30 cardiovascular-associated risk factors or groups of factors assessed, 27 (90%) were significantly more prevalent in non-drinkers than in light to moderate drinkers. The authors suggest residual confounding or unmeasured effect modification could account for some or all of the reported coronary protective associations.

The recent debacle over postmenopausal hormone

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therapy and IHD is another sobering reminder that non-randomised studies have their weaknesses. After adjusting for multiple potential confounders, the non-randomised Nurses' Health Study reported a halving of IHD risk associated with hormone therapy.[9] Randomised trials have now shown that hormone therapy does not reduce IHD risk and uncontrolled confounding is the most likely explanation for the non-randomised observations.[5] The Nurses' Health Study investigators reported a protective association of a similar size between light to moderate alcohol consumption and IHD as they did for hormone therapy and IHD.[10]

So why have we not been more critical of observations suggesting as little as one to three drinks a week is associated with a halving of IHD risk?[10] Perhaps we have been blinded by the plausible biological mechanisms. Alcohol raises HDL-cholesterol—estimated to account for about half the coronary protective effect—and also has "aspirin-like" thrombolytic effects.[11] However, in our opinion the answer lies more in the way the debate has been framed as a dichotomy; you are either a believer in IHD protection or a non-believer. We think the debate needs to be reframed to consider a middle ground that addresses the likelihood of bidirectional confounding.

Although less palatable, there is more compelling evidence for a coronary-protective effect of moderate to heavy drinking than for light to moderate drinking.[3,12] In heavy drinkers, confounding will obscure rather than exaggerate any coronary protection because of their heart-unhealthy behaviours.[8,12] The observations of relatively "clean" coronary arteries in autopsy studies of alcoholics are also consistent with a coronary-protective effect of heavy drinking.[13]

So if the debate is framed as coronary protection versus no coronary protection, we remain believers in protection. But the believers, perhaps convinced by the evidence of coronary protection in moderate to heavy drinkers, are overlooking the potential for confounding to account for much of the protective association in lighter drinkers. Similarly, the non-believers have underestimated the potential for confounding (in the opposite direction) to obscure a real coronary-protective effect of heavier drinking.

Any coronary protection from light to moderate drinking will be very small and unlikely to outweigh the harms. While moderate to heavy drinking is probably coronary-protective, any benefit will be overwhelmed by the known harms.[14] If so, the public-health message is clear. Do not assume there is a window in which the health benefits of alcohol are greater than the harms—there is probably no free lunch.

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1 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:** 1017–20.
2 Ellison RC. Cheers! *Epidemiology* 1990; **1:** 337–39.
3 Corrao G, Rubbiati L, Bagnardi V, Zambon A, Poikolainen K. Alcohol and coronary heart disease: a meta-analysis. *Addiction* 2000; **95:** 1505–23.
4 Naimi TS, Brown DW, Brewer RD, et al. Cardiovascular risk factors and confounders among nondrinking and moderate-drinking U.S. adults. *Am J Prev Med* 2005; **28:** 369–73.
5 Nelson HD, Humphrey LL, Nygren P, Teutsch SM, Allan JD. Postmenopausal hormone replacement therapy: scientific review. *JAMA* 2002; **288:** 872–81.
6 Shaper AG, Wannamethee G, Walker M. Alcohol and mortality in British men: explaining the U-shaped curve. *Lancet* 1988; **2:** 1267–73.
7 Fagrell B, De Faire U, Bondy S, et al. The effects of light to moderate drinking on cardiovascular diseases. *J Intern Med* 1999; **246:** 331–40.
8 Doll R, Peto R, Hall E, Wheatley K, Gray R. Alcohol and coronary heart disease reduction among British doctors: confounding or causality? *Eur Heart J* 1997; **18:** 23–25.
9 Stampfer MJ, Colditz GA, Willett WC, et al. Postmenopausal estrogen therapy and cardiovascular disease: ten-year follow-up from the Nurses' Health Study. *N Engl J Med* 1991; **325:** 756–62.
10 Fuchs CS, Stampfer MJ, Colditz GA, et al. Alcohol consumption and mortality among women. *N Engl J Med* 1995; **332:** 1245–50.
11 Rimm EB, Williams P, Fosher K, Criqui M, Stampfer MJ. Moderate alcohol intake and lower risk of coronary heart disease: meta-analysis of effects on lipids and haemostatic factors. *BMJ* 1999; **319:** 1523–28.
12 Rimm EB, Giovannucci EL, Willett WC, et al. Prospective study of alcohol consumption and risk of coronary disease in men. *Lancet* 1991; **338:** 464–68.
13 Moore RD, Pearson TA. Moderate alcohol consumption and coronary artery disease: a review. *Medicine (Baltimore)* 1986; **65:** 242–67.
14 Connor J, Broad J, Jackson R, Vander Hoorn S, Rehm J. The burden of death, disease and disability due to alcohol in New Zealand. Wellington: Alcohol Advisory Council of New Zealand occasional publication no 23, February, 2005: http://www.alac.org.nz/Publications.aspx (accessed Aug 15, 2005).