

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.

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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's² 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 uncon-

trollable 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



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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We are somewhat more tentative, and state in both our title and conclusion that there is probably no free lunch. They also ask for evidence about the potential harms of light-to-moderate drinking. In their Comment (Dec 3, p 1911) on alcohol and ischaemic heart disease, Rod Jackson and colleagues conclude that there is no window of benefit for light to moderate drinking. This conclusion is based on two statements: first, that coronary protection exists in light-to-moderate drinking, and second, that the harms related to such consumption outweigh the benefits. Jackson and colleagues provide scientific evidence that supports the coronary protective benefit of moderate alcohol consumption. However, no evidence is provided for the harms on which the main conclusion is based. Full-Text. PDF. Alcohol consumption, drinking patterns, and ischemic heart disease: A narrative review of meta-analyses and a systematic review and meta-analysis of the impact of heavy drinking occasions on risk for moderate drinkers. Article. Full-text available. Much discussion has revolved around the diverse findings on the complex relationship between alcohol consumption and the leading cause of death and disability, ischemic heart disease (IHD). We conducted a systematic search of the literature up to August 2014 using Preferred Reporting Items for Systematic Reviews and Meta-Analyses guidelines to identify meta-analyses and observational studies examining the relationship between alcohol drinking, drinking patterns, and IHD risk, in comparison to lifetime abstainers. Alcohol and coronary heart disease. Circulation 2005;112:1379-81. 21. Mukamal KJ, Conigrave KM, Mittleman MA, et al. Roles of drinking pattern and type of alcohol consumed in coronary heart disease in men. N Engl J Med 2003;348:109-18. 22. Greeneld JR, Samaras K, Hayward CS, Chisholm DJ, Campbell LV. JAMA 2007;297:842-57. 49. Jackson R, Broad J, Connor J, Wells S. Alcohol and ischaemic heart disease: probably no free lunch. Lancet 2005;366:1911-2. 50. Thun MJ, Peto R, Lopez AD, et al. Coronary heart disease and the protective role of alcohol has been the focus of most research and will be discussed in a separate point below. Most studies suggest that low-level consumption equally offers some protection against ischaemic stroke. 2 Part of this lack of an influence on patterns of cancer risk may be due to methodological reasons. Most epidemiological studies only measure volume of consumption and only model monotonically increasing trends and thus could not detect any influence of patterns of drinking even it were present.