Published in final edited form as:

Addiction. 2013 April ; 108(4): 723–724. doi:10.1111/add.12137.

Commentary on Dawson *et al.* (2013): Drink to your health? Maybe not

KATHERINE M. KEYES¹ and RICHARD MIECH²

¹Columbia University, New York, NY, USA

²Department of Health and Behavioral Sciences, University of Colorado Denver, Denver, CO, USA

Keywords

Alcohol consumption; cardiovascular disease; J-shaped curve; moderate alcohol intake; mortality; wine

Hundreds of observational, epidemiological studies have found that moderate alcohol intake is associated with a lower risk of a range of health outcomes compared to alcohol abstinence, but that risk increases with heavy drinking (a distribution commonly known as a ‘U-shaped’ or ‘J-shaped’ curve) [1]. Many researchers, clinicians and the lay public now accept as common knowledge that moderate drinking is beneficial to one’s health [2], and the association is hypothesized to be due to biological effects on several systems, including reduced blood pressure, increased blood lipoproteins and oxidation of low-density lipoprotein (LDL) cholesterol, among other pathways [3,4].

There are, however, methodological issues and data inconsistencies regarding the relation between alcohol intake and health that require further investigation before the case is closed on moderate drinking. Resolving these outstanding issues and inconsistencies is of substantial public health importance: many individuals are now consuming alcohol, typically red wine, as a prescriptive for better health. If the relation between moderate alcohol consumption and health is more artifactual than causal, then public health recommendations will need to be reconsidered.

The first issue raised by Dawson *et al.* [5], and documented in other studies as well, is the ‘sick quitter’ effect. Dawson *et al.* document that individuals with incident cardiovascular disease and those rating health as good/fair/poor (versus very good or excellent) at age 55+ were more likely to stop drinking, as well as individuals with liver disease at ages 18–34. Thus, when alcohol intake is measured at a single time-point, non-drinkers will constitute both former drinkers who quit due to poor health and life-time abstainers. Separating former drinkers from lifetime abstainers in studies of the effects of alcohol consumption on mortality is thus essential. Indeed, a recent analysis that did this showed that the J-shaped

Declarations of interest

None.

curve disappeared after taking into account former drinkers who quit due to poor health and that the association between alcohol consumption and mortality changed to a simple monotonic function: greater levels of alcohol consumption are associated with higher mortality [6].

A second issue is that uncontrolled confounding remains a threat to the validity of epidemiological studies on the relation between moderate alcohol consumption and health. Cross-cultural analyses and novel designs can shed light on this issue because they do not rely on traditional survey-based measures and regression-based control of confounders. The results of such studies are not promising for a causal effect of moderate intake. First, Rimm *et al.* found that the association between moderate intake and health varies by beverage type across countries [7]; the beverage type most associated with health benefits is consistently that which is most normative and prevalent within the country. If there were a biological effect of moderate wine or other beverage intake on improving health, we should observe this phenomenon consistently across countries. Secondly, a meta-analysis of Mendelian randomization studies focusing on genetic variants that regulate alcohol metabolism has demonstrated that blood pressure is increased even among those with genotypes associated with moderate drinking, in contrast to traditional observational epidemiological study design findings [8]. These findings call into question the extent to which the protective association of moderate consumption with health is truly causal.

A final and related issue is that the protective benefits of moderate alcohol consumption are not evident in all demographic groups. Accumulating evidence indicates that African Americans experience no or little protective effect [9–12] of moderate alcohol consumption on incident cardiovascular disease [10], cardiovascular mortality [12] or all-cause mortality [9,11]. The seminal studies documenting the protective health benefits of moderate alcohol consumption have been assessed predominately in samples of majority white, relatively affluent men in the developed world [1]. Studies on the health benefits of moderate drinking among women are limited, although data from the Nurses' Health Study indicate that middle-aged, majority white women also demonstrate the health benefits of moderate drinking [13]. If moderate drinking is beneficial for health through biological vascular or other mechanisms, why is it not apparent in all demographic subgroups? This inconsistency must be addressed before the full extent of the relation between moderate alcohol consumption and health is firmly established.

In conclusion, there has been substantial concern about the potential intended consequences of promoting a message that moderate alcohol consumption is beneficial for health [2], and the points outlined in this commentary are well known to those skeptical of a causal relation [14]. Converging evidence suggests that moderate drinking increases the risk of some cancers [15], thus the unintended consequences of promoting moderate drinking for cardiovascular health in the absence of a true causal link could have an overall detrimental effect on population health. However, the prevailing viewpoint with regard to moderate intake, that it is a health-promoting activity, remains. We have been proved wrong too often about the effects of dietary factors [16], medication and supplements [17,18], and even salt consumption [19], with regard to potential health benefits or adverse effects based on observational study designs. As illustrated by Dawson *et al.*, the case for moderate drinking

should not be closed. Investment in research is paramount at this critical time, before moderate drinking is subsumed under the category of known wisdom.

Acknowledgments

This research was supported in part by support from New York State Psychiatric Institute and the Department of Epidemiology at the Mailman School of Public Health (Keyes). The funding organizations had no role in the design and conduct of the study; collection, management, analysis and interpretation of the data; and preparation, review or approval of the manuscript.

References

1. Shaper AG. Alcohol and mortality: a review of prospective studies. *Br J Addict.* 1990; 85:837–47. discussion 849–61. [PubMed: 2204454]
2. Casswell S. Public discourse on the benefits of moderation: implications for alcohol policy development. *Addiction.* 1993; 88:459–65. [PubMed: 7993385]
3. Manttari M, Tenkanen L, Alikoski T, Manninen V. Alcohol and coronary heart disease: the roles of HDL-cholesterol and smoking. *J Intern Med.* 1997; 241:157–63. [PubMed: 9077373]
4. Savolainen MJ, Kesaniemi YA. Effects of alcohol on lipoproteins in relation to coronary heart disease. *Curr Opin Lipidol.* 1995; 6:243–50. [PubMed: 7670754]
5. Dawson DA, Goldstein RB, Grant BF. Prospective correlates of drinking cessation: variation across the life-course. *Addiction.* 2013; 108:712–22. [PubMed: 23216848]
6. Rogers RG, Krueger PM, Miech R, Lawrence EM, Kemp R. Nondrinker mortality risk in the United States. *Popul Res Policy Rev.* 2013 in press.
7. Rimm EB, Klatsky A, Grobbee D, Stampfer MJ. Review of moderate alcohol consumption and reduced risk of coronary heart disease: is the effect due to beer, wine, or spirits. *BMJ.* 1996; 312:731–6. [PubMed: 8605457]
8. Chen L, Davey Smith G, Harbord RM, Lewis SJ. Alcohol intake and blood pressure: a systematic review implementing a Mendelian randomization approach. *PLoS Med.* 2008; 5:e52. [PubMed: 18318597]
9. Sempos CT, Rehm J, Wu T, Crespo CJ, Trevisan M. Average volume of alcohol consumption and all-cause mortality in African Americans: the NHEFS cohort. *Alcohol Clin Exp Res.* 2003; 27:88–92. [PubMed: 12544011]
10. Fuchs FD, Chambless LE, Folsom AR, Eigenbrodt ML, Duncan BB, Gilbert A, et al. Association between alcoholic beverage consumption and incidence of coronary heart disease in whites and blacks: the Atherosclerosis Risk in Communities Study. *Am J Epidemiol.* 2004; 160:466–74. [PubMed: 15321844]
11. Kerr WC, Greenfield TK, Bond J, Ye Y, Rehm J. Racial and ethnic differences in all-cause mortality risk according to alcohol consumption patterns in the national alcohol surveys. *Am J Epidemiol.* 2011; 174:769–78. [PubMed: 21856649]
12. Mukamal KJ, Chen CM, Rao SR, Breslow RA. Alcohol consumption and cardiovascular mortality among U.S. adults, 1987 to 2002. *J Am Coll Cardiol.* 2010; 55:1328–35. [PubMed: 20338493]
13. 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; 319:267–73. [PubMed: 3393181]
14. Connor J. The life and times of the J-shaped curve. *Alcohol Alcohol.* 2006; 41:583–4. [PubMed: 17028307]
15. Boffetta P, Hashibe M. Alcohol and cancer. *Lancet Oncol.* 2006; 7:149–56. [PubMed: 16455479]
16. Davey Smith G, Ebrahim S. Folate supplementation and cardiovascular disease. *Lancet.* 2005; 366:1679–81. [PubMed: 16291049]
17. Rossouw JE, Anderson GL, Prentice RL, LaCroix AZ, Kooperberg C, Stefanick ML, et al. Risks and benefits of estrogen plus progestin in healthy postmenopausal women: principal results from the Women's Health Initiative randomized controlled trial. *JAMA.* 2002; 288:321–33. [PubMed: 12117397]

18. Rimm EB, Stampfer MJ, Ascherio A, Giovannucci E, Colditz GA, Willett WC. Vitamin E consumption and the risk of coronary heart disease in men. *N Engl J Med*. 1993; 328:1450–6. [PubMed: 8479464]
19. Bayer R, Johns DM, Galea S. Salt and public health: contested science and the challenge of evidence-based decision making. *Health Aff (Millwood)*. 2012; 31:2738–46. [PubMed: 23213158]