CARDIOLOGY Rounds®

As presented in the Rounds of the Division of Cardiology, St. Michael's Hospital, University of Toronto

Alcohol and Cardioprotection – Reality or Observational Mistake?

By AKSHAY BAGAI, MD, FRCPC, AND BETH ABRAMSON, MD, MSc, FRCPC, FACC

Excessive chronic consumption of alcohol is a significant cause of morbidity and premature mortality. In contrast, an extensive body of scientific evidence indicates that drinking alcohol in moderation on a daily basis appears to offer protection against disease of the heart and the vascular system. This U-shaped alcohol-mortality relationship, in which mortality risk increases among abstainers and heavy drinkers relative to light or moderate drinkers has been observed since the 1980s.[1] Although reproduced in numerous observational studies over the last 25 years, this association has not been studied in a randomized, controlled-outcome clinical trial. Therefore, the question still remains: Are the cardioprotective effects of moderate levels of alcohol intake seen in observational studies real or a flaw in the nature of these studies?

The purpose of this issue of *Cardiology Rounds* is to:

- **highlight the association between alcohol intake and cardiovascular outcomes**
- **describe both the risks and benefits of alcohol consumption in relation to the cardiovascular system**
- **discuss the threshold of intake at which drinking has detrimental effects**
- **describe the mechanisms through which alcohol may be cardioprotective**
- **elucidate the beverage types, drinking patterns, ideal quantities, and the individuals who are most likely to benefit.**

Alcohol and health outcomes: the J-shaped curve

The health effects of ethanol are dependent on the amount of alcohol consumed and the pattern of drinking. The relationship between alcohol intake and the relative risk of developing coronary heart disease (CHD) is a J- or U-shaped dose-response; ie, the risk is lower when alcohol consumption is light to moderate and higher when alcohol consumption is high or absent altogether. "Moderate drinking" is defined as the consumption of no more than 1 drink/day for women and 2 drinks/day for men. One standard drink is 15 g of ethanol, which is equivalent to 355 mL (12 oz) of beer, 148 mL (5 oz) of wine, or 44 mL (1.5 oz) of spirits. A recent meta-analysis of more than 1 million individuals revealed that moderate consumption of alcohol was associated with a reduction in total mortality of 18%.[2] On the other hand, higher intakes were associated with increased mortality in a dose-dependent fashion (Figure 1).

Several studies have found that moderate consumption of alcohol reduces cardiovascular (CV) mortality.[3-5] In a prospective study of 490,000 men and women in the United States (US), the relative risk of death from CV disease in moderate drinkers compared with nondrinkers was 0.7 for men and 0.6 for women (Figure 2).[4] This benefit has been evident in studies of individuals with[6] and without[3] known CHD.

Although reductions in CV mortality are likely mediated by several mechanisms, the majority of benefit appears to be from a reduction in CHD. In the National Health and Nutrition Survey I and the Epidemiologic Follow-up Survey, people who consumed 2 to 7 drinks/week had the lowest risk of CHD;[7] for women, the relative risks for CHD incidence and mortality were

St. Michael's Hospital
30 Bond St.,
Suite 7049, Queen Wing
Toronto, Ont. M5B 1W8
Fax: (416) 864-5941

Figure 1: Alcohol consumption and all-cause mortality[2]

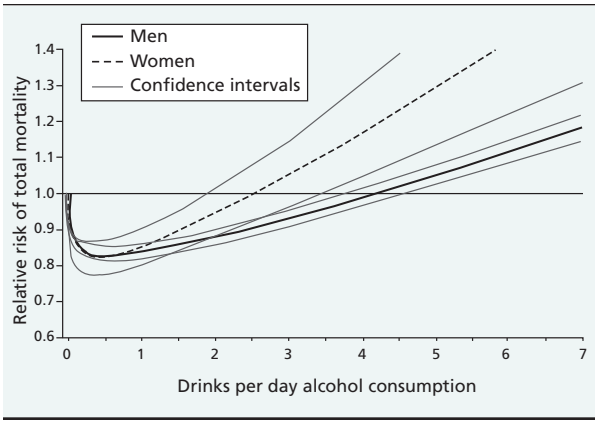
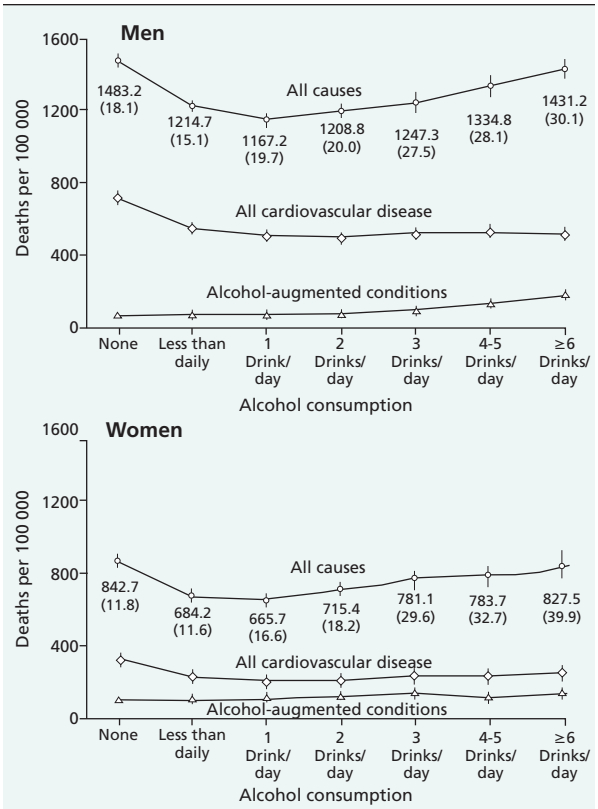


Figure 2: Alcohol consumption and cardiovascular mortality[4]



0.51 (95% confidence interval [CI], 0.37–0.70) and 0.55 (95% CI, 0.30–1.01), respectively, while for men, the relative risks were 0.62 (95% CI, 0.48–0.79) and 0.73 (95% CI, 0.49–1.08), respectively. Similarly, in the INTERHEART study,[8] involving 27,000 patients from 52 countries, regular alcohol consumption was associated with a reduced incidence of myocardial infarction (MI) in both males and females. This association was found even among a group of 8,867 men at the lowest risk for MI (nonsmokers, exercising at least 30 minutes daily, healthy diets, body mass index [BMI] <25 kg/m^2). Consumption of 1 or 2 drinks per day was associated with a 40%–50% decreased risk of MI (Figure 3).[9]

Excessive alcohol intake is one of the most common reversible risk factors for hypertension; conversely, it appears that patients with hypertension also benefit from moderate alcohol consumption. Among 11,711 men with hypertension followed for 16 years in the Health Professionals Follow-Up Study, consumption of 1 drink/day was associated with a 30% decrease in the incidence of MI.[10] Although chronic excessive consumption may lead to cardiomyopathy, light-to-moderate drinking may be protective against the development of heart failure, especially for those with CHD.[11]

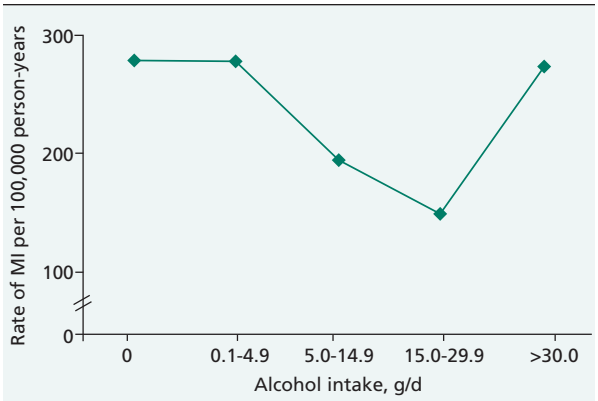
Similarly, consistent J-shaped curve associations are seen with alcohol consumption and stroke outcomes. A meta-analysis of 19 cohort and 16 case-control studies demonstrated that heavy alcohol use, >60 g/day, increased the risk of both ischemic and hemorrhagic strokes; however, light or moderate consumption decreased the risk of ischemic stroke, but did not affect the risk of hemorrhagic stroke.[12] Alcohol appears to reduce the risk of peripheral vascular disease among apparently healthy people. In the Physician's Health study of 22,071 male physicians who were followed for 11 years, daily drinkers (≥7 drinks/week) had a relative risk of 0.68 for peripheral vascular disease compared with the reference group (<1 drink/week).[13]

Possible mechanisms for cardioprotection from alcohol

Multiple population studies and experimental animal studies indicate that alcohol may have antiatherosclerotic effects. Moderate alcohol use has been associated with decreased atherosclerotic burden as assessed by coronary angiography,[14] computerized tomography-detected coronary calcium (Figure 4),[15] and carotid ultrasound.[16] Mechanisms for antiatherosclerosis may be related to alcohol-induced changes in serum lipids, lipoproteins, blood-clotting proteins, platelets, inflammatory cytokines, and insulin resistance. Moderate alcohol consumption has been shown to increase high-density lipoprotein cholesterol (HDL-C) by approximately 30% and is believed to account for approximately 50% of the reduced risk for developing CHD.[17] Genetic factors may influence the effect of alcohol intake. In one report, men and women who were homozygous for the slow-oxidizing allele of alcohol dehydrogenase type 3 (ADH3) had the greatest increase in HDL-C with moderate alcohol intake and the greatest reduction in the risk of MI.[18]

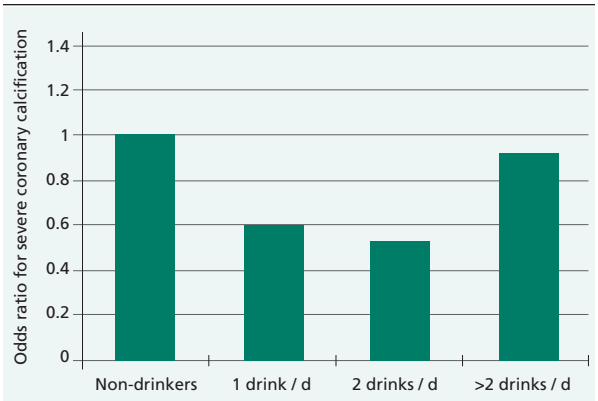
Some proportion of the reduced risk that is not accounted for by increased HDL-C may be attributable to alcohol-induced insulin sensitivity. Consuming moderate amounts of alcohol, like exercising aerobically, will

Figure 3: Alcohol intake and risk of MI in men following healthy lifestyle[9]



Moderate alcohol intake (1 to 2 drinks per day) reduced the rate of MI in this group of 8,867 middle-aged males already following healthy lifestyle recommendations.
MI = myocardial infarction

Figure 4: Alcohol consumption and risk of coronary calcification on CT scanning[15]



CT = computerized tomography

increase insulin sensitivity and glucose metabolism for the ensuing 12 to 24 hours.[19,20] Ethanol, when consumed by diabetic patients in small-to-moderate quantities with or immediately before the evening meal, has been demonstrated to substantially reduce glucose excursion following the meal. The biological mechanism through which alcohol improves insulin sensitivity appears to involve suppression of fatty acid release from adipose tissue.[21] This reduction in fatty acids decreases substrate competition in the Krebs cycle of skeletal muscles, thereby facilitating glucose metabolism. In addition, light-to-moderate alcohol intake is associated with reductions in both the prevalence and incidence of diabetes. A large meta-analysis of 370,000 individuals followed for 12 years revealed a 30% reduction in new diabetes for those who consumed 1 to 2 drinks per day (Figure 5).[22]

Alcohol has antithrombotic activities that may contribute to the observed reduction in CHD. These include the modification of platelet function via reducing plasma fibrinogen levels and elevating plasma prostacyclin concentrations and, possibly, improving fibrinolysis via reductions in the levels of plasminogen activator inhibitor activity, von Willebrand factor, and factor VII, and an increase in concentrations of tissue-type plasminogen activator (tPA). Moderate alcohol ingestion may also have antioxidant properties. Phenolic compounds and flavonoids in red wine inhibit the formation of more atherogenic, oxidized forms of low-density lipoproteins (LDLs).

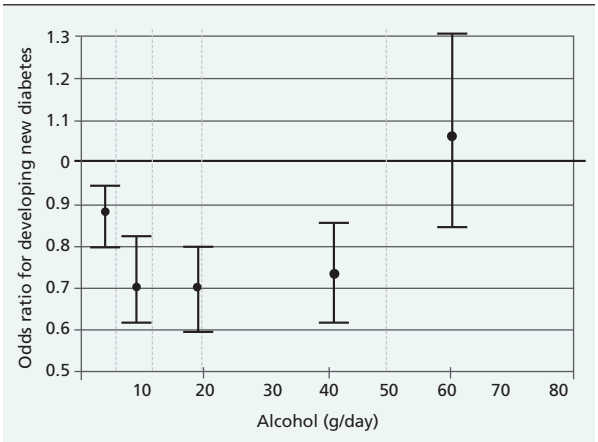
Finally, anti-inflammatory effects of alcohol intake have been suggested by observations of reductions in C-reactive protein (CRP), tumour necrosis factor alpha, interleukin-6, and fibrinogen.[23,24] In the Pravastatin Inflammation/CRP Evaluation (PRINCE) study,[25] CRP levels were correlated with self-reported alcohol intake in 2,833 subjects, 41% of whom had a history of CHD. There was a progressive decline in CRP levels with increasing alcohol intake from 2.6 mg/L for those consuming <1 drink per month to 1.6 mg/L for those consuming 5 to 7 drinks per week.

Beverage type and drinking patterns

Red wine consumption was thought to be important in explaining the "French paradox;" this refers to the low mortality rate from ischemic heart disease and CV diseases displayed by French men despite a high level of risk factors (high cholesterol, diabetes, hypertension, and a high intake of saturated fat). The hypothesis that red wine was more beneficial than liquor or beer was proposed for 2 primary reasons.

- Several international comparisons indicated a lower incidence of CHD mortality in wine-drinking countries than in countries where beer or distilled spirits predominate the type of alcohol consumed.

Figure 5: Alcohol consumption and risk of new type 2 diabetes mellitus[22]



- Potential benefits could be due to the nonalcoholic antioxidant phenolic compounds and antithrombotic substances in wine.

Clinical trial data have varied on this issue; some studies suggest that all alcoholic beverage categories confer CV health benefits,[26] while others report that the cardioprotection is greatest for wine.[27] The developing consensus suggests that the specific alcoholic beverage is less important than the quantity and pattern of the alcohol intake.[28]

Data suggest that daily alcohol intake provides greater cardioprotection compared with less frequent consumption.[29] This may be due to alcohol-induced favourable changes in insulin sensitivity, HDL-C, and inflammation that are transient, reverting to baseline within 24 hours.[30]

Detrimental effects of alcohol

In contrast with to light-to-moderate drinking, which may be cardioprotective, excessive alcohol consumption and binge drinking are toxic both to the heart and to overall health. Excessive alcohol intake is associated with an increased incidence of a number of medical conditions, such as liver cirrhosis, breast and gastrointestinal cancer, sleep apnea, fetal alcohol syndrome, dementia, and hypertension. It is believed that approximately 10% of the American population abuse alcohol. A survey of mental health and substance abuse disorders in nearly 20,000 American adults found a 13.5% lifetime prevalence of alcohol abuse or dependence.[31]

Long-term excess consumption is the leading cause of secondary, nonischemic, dilated cardiomyopathy. Most people who develop alcoholic cardiomyopathy have been drinking >80–90 g of ethanol per day for >5 years (some studies quote an average of 15 years). Although the exact pathogenesis is not fully understood, several mechanisms have been proposed: direct myocardial toxicity from alcohol leading to myocardial cell apoptosis; myocardial depression from acetaldehyde, a metabolite of alcohol; nutritional deficiencies, particularly of thiamine; and possible toxicity from alcohol additives such as cobalt. Genetic predispositions for this condition may be present in individuals with a DD polymorphism of the angiotensin-converting enzyme (ACE) gene that results in higher plasma and cardiac levels of ACE.[32] The exact prevalence of alcoholic cardiomyopathy is not well defined. In one series of 50 asymptomatic alcoholic women and 100 asymptomatic alcoholic men, approximately one-third had evidence of left-ventricular dysfunction.[33]

Arrhythmias occur in a significant proportion (up to 60%) of binge drinkers with or without underlying myocardial damage. This phenomenon of "holiday heart syndrome" refers to the onset of atrial fibrillation related to binge drinking over weekends or holidays. Although not conclusive, an increased risk of atrial fibrillation has been reported among men with chronic heavy alcohol consumption.[34]

Limitations of the current literature

To date, no long-term randomized trials of alcohol administration exist. Knowledge of the effects of moderate alcohol consumption in humans is derived primarily from two sources: short-term trials analyzing the effect of alcohol upon physiological measures and observational studies comparing moderate drinkers with abstainers. Both of these sources have limitations; short-term studies do little to inform the debate about the balance between the long-term risks of alcohol consumption and the benefits, since alcohol use for most populations extends over decades. Furthermore, these trials tend to be performed using healthy, young, white male subjects; consequently, the results cannot necessarily be extrapolated to other populations.

The primary limitation of observational studies, comparing abstainers with alcohol users, is that alcohol use is not distributed randomly among individuals; people who abstain are different from those who drink in more ways than just how much alcohol they consume. Simply adjusting for known, measurable differences between abstainers and drinkers may be inadequate to make the two groups comparable.

Conclusion and recommendations

Cumulative evidence from observational studies suggests that moderate alcohol intake is associated with beneficial cardioprotective effects. However, caution is necessary when promoting the beneficial effects of alcohol on coronary risk, since there is little agreement regarding the "ideal dosage" and the upper safe limit of alcohol. In addition, good data do not exist about which group of individuals may benefit from different levels of alcohol use with no additional risk. It may also be difficult to titrate the dose of alcohol within a safe range; ie, a level of consumption that would not risk a loss of the cardioprotective effects and, as well, not increase the risk for alcohol-related conditions such as hepatic cirrhosis.

Health organizations have made variable recommendations in this regard. Guidelines for sensible drinking developed in the United Kingdom state that "middle-aged or elderly men and postmenopausal women who drink infrequently or not at all may wish to consider the possibility that light drinking may benefit their health."[28] The latest American Heart

Association (AHA) guidelines in 2001, state that "Moderate intake of alcoholic beverages (one to two drinks per day) is associated with a reduced risk of CHD in populations. … Despite the biologic plausibility and observational data in this regard, it should be kept in mind that these are insufficient to prove causality." … Without a large-scale, randomized, clinical endpoint trial of wine intake, there is little current justification to recommend alcohol (or wine specifically) as a cardioprotective strategy. The AHA maintains its recommendation that alcohol use should be an item of discussion between physician and patient."[35] The Heart and Stroke Foundation of Ontario does not recommend drinking alcohol for the purpose of reducing the risk of heart disease and stroke.

Despite convincing observational data and randomized trials using surrogate endpoints suggesting that, in women, hormone replacement therapy and antioxidant vitamins improved CV outcomes, subsequent large randomized trials demonstrated the opposite. Therefore, until we have more randomized, clinical-outcome data and tools for predicting susceptibility to problem drinking, light-to-moderate drinking cannot be universally recommended to the general public or even to patients with CVD.

Information for patients

Educational materials on this topic are available for patients on the Heart and Stroke Foundation website at http://www.heartandstroke.com.

References

1. Marmot MG, Rose G, Shipley MJ, Thomas BJ. Alcohol and mortality: a U-shaped curve. *Lancet.* 1981;1:580-583.
2. Di Castelnuovo A, Costanzo S, Bagnardi V, Donati MB, Iacoviello L, de Gaetano G. Alcohol dosing and total mortality in men and women: an updated meta-analysis of 34 prospective studies. *Arch Intern Med.* 2006;166:2437-2445.
3. Fuchs CS, Stampfer MJ, Colditz GA, et al. Alcohol consumption and mortality among women. *N Engl J Med.* 1995;332:1245-1250.
4. Thun MJ, Peto R, Lopez AD, et al. Alcohol consumption and mortality among middle-aged and elderly U.S. adults. *N Engl J Med.* 1997;337:1705-1714.
5. Mukamal KJ, Maclure M, Muller JE, Sherwood JB, Mittleman MA. Prior alcohol consumption and mortality following acute myocardial infarction. *JAMA.* 2001;285:1965-1970.
6. Iestra JA, Kromhout D, van der Schouw YT, Grobbee DE, Boshuizen HC, van Staveren WA. Effect size estimates of lifestyle and dietary changes on all-cause mortality in coronary artery disease patients: a systematic review. *Circulation.* 2005;112:924-934.
7. Rehm JT, Bondy SJ, Sempos CT, Vuong CV. Alcohol consumption and coronary heart disease morbidity and mortality. *Am J Epidemiol.* 1997;146:495-501.
8. Yusuf S, Hawken S, Ounpuu S, et al. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. *Lancet.* 2004;364:937-952.
9. Mukamal KJ, Chiuve SE, Rimm EB. Alcohol consumption and risk for coronary heart disease in men with healthy lifestyles. *Arch Intern Med.* 2006;166:2145-2150.
10. Beulens JW, Rimm EB, Ascherio A, Spiegelman D, Hendriks HF, Mukamal KJ. Alcohol consumption and risk for coronary heart disease among men with hypertension. *Ann Intern Med.* 2007;146:10-19.
11. Djousse L, Gaziano JM. Alcohol consumption and risk of heart failure in the Physicians' Health Study I. *Circulation.* 2007;115:34-39.
12. Reynolds K, Lewis B, Nolen JD, Kinney GL, Sathya B, He J. Alcohol consumption and risk of stroke: a meta-analysis. *JAMA.* 2003;289:579-588.
13. Camargo CA Jr, Stampfer MJ, Glynn RJ, et al. Prospective study of moderate alcohol consumption and risk of peripheral arterial disease in US male physicians. *Circulation.* 1997;95:577-580.
14. Femia R, Natali A, L'Abbate A, Ferrannini E. Coronary atherosclerosis and alcohol consumption: angiographic and mortality data. *Arterioscler Thromb Vasc Biol.* 2006;26:1607-1612.
15. Vliegenthart R, Oei HH, van den Elzen AP, et al. Alcohol consumption and coronary calcification in a general population. *Arch Intern Med.* 2004;164:2355-2360.
16. Schminke U, Luedemann J, Berger K, et al. Association between alcohol consumption and subclinical carotid atherosclerosis: the Study of Health in Pomerania. *Stroke.* 2005;36:1746-1752.
17. Gaziano JM, Buring JE, Breslow JL, et al. Moderate alcohol intake, increased levels of high-density lipoprotein and its subfractions, and decreased risk of myocardial infarction. *N Engl J Med.* 1993;329:1829-1834.
18. Hines LM, Stampfer MJ, Ma J, et al. Genetic variation in alcohol dehydrogenase and the beneficial effect of moderate alcohol consumption on myocardial infarction. *N Engl J Med.* 2001;344:549-555.
19. Greenfield JR, Samaras K, Hayward CS, Chisholm DJ, Campbell LV. Beneficial postprandial effect of a small amount of alcohol on diabetes and cardiovascular risk factors: modification by insulin resistance. *J Clin Endocrinol Metab.* 2005;90:661-672.
20. Turner BC, Jenkins E, Kerr D, Sherwin RS, Cavan DA. The effect of evening alcohol consumption on next-morning glucose control in type 1 diabetes. *Diabetes Care.* 2001;24:1888-1893.
21. Greenfield JR, Samaras K, Jenkins AB, Kelly PJ, Spector TD, Campbell LV. Moderate alcohol consumption, estrogen replacement therapy, and physical activity are associated with increased insulin sensitivity: is abdominal adiposity the mediator? *Diabetes Care.* 2003;26:2734-2740.
22. Koppes LL, Dekker JM, Hendriks HF, Bouter LM, Heine RJ. Moderate alcohol consumption lowers the risk of type 2 diabetes: a meta-analysis of prospective observational studies. *Diabetes Care.* 2005;28:719-725.
23. Zairis MN, Ambrose JA, Lyras AG, et al. C-reactive protein, moderate alcohol consumption, and long-term prognosis after successful coronary stenting: four year results from the GENERATION study. *Heart.* 2004;90:419-424.
24. Lucas DL, Brown RA, Wassef M, Giles TD. Alcohol and the cardiovascular system research challenges and opportunities. *J Am Coll Cardiol.* 2005;45:1916-1924.
25. Albert MA, Glynn RJ, Ridker PM. Alcohol consumption and plasma concentration of C-reactive protein. *Circulation.* 2003;107:443-447.
26. 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;348:109-118.
27. Klatsky AL, Armstrong MA. Alcoholic beverage choice and risk of coronary artery disease mortality: do red wine drinkers fare best? *Am J Cardiol.* 1993;71:467-469.
28. Ellison RC. Importance of pattern of alcohol consumption. *Circulation.* 2005;112:3818-3819.
29. Mukamal KJ, Jensen MK, Gronbaek M, et al. Drinking frequency, mediating biomarkers, and risk of myocardial infarction in women and men. *Circulation.* 2005;112:1406-1413.
30. Veenstra J, Ockhuizen T, van de Pol H, Wedel M, Schaafsma G. Effects of a moderate dose of alcohol on blood lipids and lipoproteins postprandially and in the fasting state. *Alcohol Alcohol.* 1990;25:371-377.
31. Regier DA, Farmer ME, Rae DS, et al. Comorbidity of mental disorders with alcohol and other drug abuse. Results from the Epidemiologic Catchment Area (ECA) Study. *JAMA.* 1990;264:2511-2518.
32. Kaplan NM. Alcohol and hypertension. *Lancet.* 1995;345:1588-1589.
33. Urbano-Marquez A, Estruch R, Fernandez-Sola J, Nicolas JM, Pare JC, Rubin E. The greater risk of alcoholic cardiomyopathy and myopathy in women compared with men. *JAMA.* 1995;274:149-154.
34. Cooper HA, Exner DV, Domanski MJ. Light-to-moderate alcohol consumption and prognosis in patients with left ventricular systolic dysfunction. *J Am Coll Cardiol.* 2000;35:1753-1759.
35. Goldberg IJ, Mosca L, Piano MR, Fisher EA. AHA Science Advisory: Wine and your heart: a science advisory for healthcare professionals from the Nutrition Committee, Council on Epidemiology and Prevention, and Council on Cardiovascular Nursing of the American Heart Association. *Circulation.* 2001;103:472-475.

Abstracts of Interest

Dose-related effects of red wine and alcohol on hemodynamics, sympathetic nerve activity, and arterial diameter.

Spaak J, Merlocco AC, Soleas GJ, Tomlinson G, Morris BL, Picton P, Notarius CF, Chan CT, Floras JS. Stockholm, Sweden.

The cardiovascular benefits of light to moderate red wine consumption often have been attributed to its polyphenol constituents. However, the acute dose-related hemodynamic, vasodilator, and sympathetic neural effects of ethanol and red wine have not been characterized and compared in the same individual. We sought to test the hypotheses that responses to one and two alcoholic drinks differ and that red wine with high polyphenol content elicits a greater effect than ethanol alone. Thirteen volunteers (24-47 yr; 7 men, 6 women) drank wine, ethanol, and water in a randomized, single-blind trial on three occasions 2 wk apart. One drink of wine and ethanol increased blood alcohol to 38 +/- 2 and 39 +/- 2 mg/dl, respectively, and two drinks to 72 +/- 4 and 83 +/- 3 mg/dl, respectively. Wine quadrupled plasma resveratrol ($P<0.001$) and increased catechin ($P<0.03$). No intervention affected blood pressure. One drink had no heart rate effect, but two drinks of wine increased heart rate by 5.7 +/- 1.6 beats/min; $P<0.001$). Cardiac output fell 0.8 +/- 0.3 l/min after one drink of ethanol and wine (both $P<0.02$) but increased after two drinks of ethanol (+0.8 +/- 0.3 l/min) and wine (+1.2 +/- 0.3 l/min) ($P<0.01$). One alcoholic drink did not alter muscle sympathetic nerve activity (MSNA), while two drinks increased MSNA by 9-10 bursts/min ($P<0.001$). Brachial artery diameter increased after both one and two alcoholic drinks ($P<0.001$). No beverage augmented, and the second wine dose attenuated ($P=0.02$), flow-mediated vasodilation. One drink of ethanol dilates the brachial artery without activating sympathetic outflow, whereas two drinks increase MSNA, heart rate, and cardiac output. These acute effects, which exhibit a narrow dose response, are not modified by red wine polyphenols.

Am J Physiol Heart Circ Physiol. 2008;294(2):H605-612.

Alcohol consumption and cardiovascular mortality accounting for possible misclassification of intake: 11-year follow-up of the Melbourne Collaborative Cohort Study.

Harriss LR, English DR, Hopper JL, Powles J, Simpson JA, O'Dea K, Giles GG, Tonkin AM. Melbourne, Australia.

AIMS: To investigate the relationship between usual daily alcohol intake, beverage type and drinking frequency on cardiovascular (CVD) and coronary heart disease (CHD) mortality, accounting for systematic misclassification of intake.

DESIGN: Prospective cohort study with mean follow-up of 11.4 years.

SETTING: The Melbourne Collaborative Cohort Study, Australia.

PARTICIPANTS: A total of 38 200 volunteers (23 044 women) aged 40-69 years at baseline (1990-1994).

MEASUREMENTS: Self-reported alcohol intake using beverage-specific quantity-frequency questions (usual intake) and drinking diary for previous week.

FINDINGS: Compared with life-time abstention, usual daily alcohol intake was associated with lower CVD and CHD mortality risk for women but not men. For women, the hazard ratio [HR (95% CI)] for CVD for those drinking > 20 g/day alcohol was 0.43 (0.19-0.95; P trend = 0.18), and for CHD, 0.19 (0.05-0.82; P trend = 0.24). Male former drinkers had over twice the mortality risk for CVD [HR = 2.58 (1.51-4.41)] and CHD [HR = 2.91 (1.59-5.33)]. Wine was the only beverage associated inversely with mortality for women. Compared with drinkers who consumed no alcohol in the week before baseline, drinking frequency was associated inversely with CVD and CHD mortality risk for men but not women. HR for men drinking 6-7 days/week was 0.49 (0.29-0.81; P trend = 0.02) for CVD, and 0.49 (0.26-0.92: P trend = 0.23) for CHD.

CONCLUSIONS: Usual daily alcohol intake was associated with reduced CVD and CHD mortality for women but not men. This benefit appeared to be mainly from wine, although comparison of beverages was not possible. Drinking frequency was associated inversely with CVD and CHD death for men but not women.

Addiction. 2007;102(10):1574-1585.

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