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MINI REVIEW

Effect of Alcohol Consumption on the Risk of Erectile Dysfunction

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Alcohol consumption is a common behavior in social circumstances worldwide. Epidemiological studies have suggested that moderate alcohol consumption reduces cardiovascular morbidity and mortality. The cardiovascular protective effects of alcohol may be attributed to its antioxidant, vasorelaxant, and antithrombotic properties, elevation of high-density lipoprotein or increase of nitric oxide production. Erectile dysfunction (ED) is a harbinger of cardiovascular diseases. Most epidemiological studies have also found that alcohol consumption, like its relationship with coronary artery disease, is related to ED in a J-shaped manner, with moderate consumption conferring the highest protection and higher consumption less benefits. In epidemiological studies, it is difficult to distinguish the ethanol effects from those of associated confounding factors. Meanwhile, long-term alcohol users, especially in those with alcohol liver disease, are highly associated with ED. More research is needed to investigate the true effects of alcohol consumption on cardiovascular diseases or ED.

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There are 2 CME questions based on this article

1. Introduction

Alcohol beverages (ethyl alcohol or ethanol), tobacco and caffeine are the most widely used psychoactive substances since time immemorial. People consume alcohol for a wide range of reasons in different social and cultural contexts. It may be consumed for sociability, cultural participation, religious observance or celebratory occasions and may serve a symbolic purpose in a round of toasting or as a sacrament. People may drink alcohol for pleasure, relaxation, mood alteration, enhanced creativity, intoxication, addiction, forgetting sorrows or for thirst-quenching. Alcohol is always taken orally in the forms of beer, wine and distilled spirits.

The use of alcohol also carries with it the potential for harm, whether in the short term or long term. Alcohol consumption is a major factor in much of the injury resulting from road crashes, accidents and in social problems. The consumption of alcoholic beverages is a common feature of daily living that affects blood pressure, uric acid, and thrombogenesis, and also appears to influence the risk of cardiovascular disease, both favorably and adversely. Excessive ethanol consumption is associated with many medical disorders, including liver cirrhosis, cardiomyopathy, poor general health and malnutrition.[1] On the other hand, epidemiological studies have shown that daily light to moderate consumption of alcohol confers cardioprotective benefits and decreases cardiovascular disease morbidity and mortality.[2,3] The combination of protective and harmful influences of alcohol consumption results in a U-shaped mortality curve.

Erectile dysfunction (ED) is a highly prevalent medical disorder with significant impact on quality of life and interpersonal relationships. Meanwhile, ED is regarded as an early marker of systemic atherosclerosis, a predictor for coronary artery disease (CAD) and cardiac events.[4] An epidemiological study has demonstrated that ED shares the same risk factors as vascular disease, including

smoking, hypertension, diabetes, dyslipidemia, obesity and metabolic syndrome.[5] The pathophysiology of ED involves the production of nitric oxide (NO), smooth muscle relaxation and endothelial dysfunction in the corpus cavernosum.[6] It is very interesting to understand the potential effects of alcohol consumption on the risk of ED.

2. Effect on Behavior

The blood alcohol concentration reaches its peak about 30–45 minutes after the consumption of one standard drink (about 10 g alcohol) (Table 1).[7] It generally takes about an hour for the body to clear one standard drink, although this varies from person to person. Eating when drinking alcohol slows the increase in alcohol concentration as food in the stomach reduces the speed of its absorption into the bloodstream. The most obvious and immediate effects of alcohol, usually within about 5 minutes of being swallowed, are on the brain, beginning with feelings of relaxation, wellbeing and loss of inhibitions. As the intake of alcohol increases, these effects are counterbalanced by less pleasant effects, such as drowsiness, loss of balance, nausea and vomiting, as well as other harmful effects. The acute behavioral effects of ethanol vary between individuals according to many factors such as dose, rate of drinking, sex, body weight, blood alcohol level and the time since the previous dose.[8] Very small amounts of ethanol may be excreted unchanged in urine, sweat and the breath while most of it is metabolized to acetaldehyde by alcohol dehydrogenase in the liver.[9] Ethanol induces diverse types of tolerance. Metabolic tolerance occurs and is a function of the upregulation of metabolic enzymes, with the result that an increased dose or more frequent use of alcohol is required to obtain the desired psychopharmacological effects.

Ethanol increases the inhibitory activity mediated by gamma-aminobutyric acid-A receptors that involves motor incoordination, anxiolysis and sedation in humans.[10] Alcohol also decreases the excitatory activity mediated by glutamate receptors, especially the NMDA (N-methyl D-aspartate) receptors.[11] These two mechanisms together contribute to the general sedative effect of alcohol and impairment of memory during periods of intoxication.[11] In addition to the effects on central nervous system receptors, alcohol suppresses the release of gonadotropin and antidiuretic hormone from the pituitary that may be associated with hypogonadism and hypovolemic shock in alcohol abusers. The chronic abuse of alcohol may cause testicular atrophy, inhibition of testosterone production and impairment of spermatogenesis, apart from its direct oxidative toxicity.[12]

Table 1 Number of standard drinks in common containers of various alcoholic beverages*

Category	Standard drink(s)
Can, low-strength (2.7% alcohol) beer	0.8
Can, mid-strength (3.5% alcohol) beer	1
Can, full-strength (4.9% alcohol) beer	1.4
100 mL wine (9.5–13.5% alcohol)	1
30 mL nip spirits (37–40% alcohol)	1
700 mL bottle spirits (37–40% alcohol)	22

*Adapted from the Australian National Health and Medical Research Council.[7]

3. Cardioprotective Effects

Both animal and human epidemiological studies have demonstrated beneficial effects of alcohol consumption. Monkeys on a high cholesterol diet were found to have less arterial obstruction in the group given alcohol compared to placebo.[13] There is a considerable body of rather consistent data that show a protective effect of light to moderate drinking of alcohol against CAD in population-based studies. A cross-sectional investigation involving 2500 people by angiographic examination exhibited an inverse relationship between the extent of coronary artery atherosclerosis and alcohol consumption.[14] The Framingham Cohort Study based on 24 years of follow-up found that alcohol consumption is significantly associated with a decreased risk of coronary heart disease in both men and women after taking other cardiovascular risk factors into account (Table 2).[15] A meta-analysis of case-control and cohort studies including 116,702 subjects from 156 qualified articles performed by Conway showed a J-shaped relationship between alcohol consumption and CAD risk.[16] He reported that the minimum relative risk for CAD was 0.80 at 20 g ethanol/day, a significant protective effect occurred at up to 72 g ethanol/day and a significantly increased risk was present at 90 g ethanol/day. Based on 52,364 cases from a meta-analysis, Maclure suggested that the full preventive effect could be achieved by as little as half a drink per day, and that there is no further benefit with increasing intake beyond this.[17]

A balanced view of alcohol drinking and health should consider not only harmful and beneficial effects, but also the amount of alcohol consumption, beverage choice, and drinking patterns. Alcohol, particularly red wine, has been found to contain powerful antioxidants that favorably affect many aspects of coagulation and thrombolysis.[18] Klatsky et al.[19] collected demographic and history data from 128,934 adults and concluded that frequency of wine drinking but not of liquor or beer drinking was independently related to lower mortality risk, especially for CAD and respiratory deaths. The relative risks of myocardial infarction were almost identical for consumers of beer, wine and spirits in a meta-analysis.[17] Frequent small amounts (consumed several times per week, perhaps daily) appear to be more protective than the same amount taken at one sitting (binge drinking).[20] Coronary

Table 2 Coronary heart disease mortality by alcohol consumption in the Framingham Study 24-year follow-up after adjusting for age*

Men			Women		
Alcohol consumption (oz/wk)†	Non-smokers	Heavy smokers	Alcohol consumption (oz/wk)†	Non-smokers	Heavy smokers
None	16.3	28.3	None	5.5	6.3
1	14.8	16.0	1	5.4	3.7
2–3	14.6	14.4	2–3	1.9	3.0
4–7	7.8	14.0	4–9	6.5	4.6
8–19	5.7	13.1	≥10	5.9	5.0
≥20	7.4	12.5			

*Data presented as %; †1 ounce of alcohol is equal to 2.0–2.5 standard drinks, with a drink being defined as 12 ounces of beer, 4–5 ounces of wine, or 1.25 ounces of 80 proof spirits. (Adapted from Reference 15.)

artery occlusions have also been found to be more extensive among binge drinkers than daily drinkers of the same amount of alcohol per week.[20]

However, some studies did not find any significant cardioprotective effect from alcohol consumption or all-cause associations with it. A systematic review[21] and some other studies[22] suggested that the cardioprotective or all-cause effect may have been overestimated. The prevalence and severity of alcoholic myopathy and cadiomyopathy is directly related to the lifetime dose of ethanol consumed. The cardioprotective effects of ethanol pertain to its influence on CAD, but otherwise alcohol consumption damages the heart, especially at intakes >60–80 g/day.[23]

The best documented mechanism of cardioprotection from moderate consumption of alcohol was attributed to the elevation of high-density lipoprotein (HDL) subfractions (HDL_2 and HDL_3).[24] In addition, alcohol consumption was associated with an increase in the prostacycline/thromboxane ration, decreased platelet aggregability, increased release of plasminogen activator, and lower fibrinogen.[25] Animal study of rats fed with alcohol and endothelial NO synthase (eNOS) protein for 8 weeks demonstrated that moderate alcohol intake improved postischemic myocardial function and increased NO production by the vascular endothelium.[26] Another animal study also supported the protective effect of NO through alcohol consumption.[27] On the contrary, some animal studies found endothelial oxidative injury and downregulation of the NO generating system that might impair erectile function.[28,29] Alcohol may also increase energy intake and fat storage further by increasing appetite and displacing fat and carbohydrate oxidation that are harmful to health.

4. Impact on the Risk of ED

The relationship between alcohol consumption and ED is complex. The consumption of alcohol often precedes sexual activity and alcohol is commonly believed to be a powerful sexual facilitator and aphrodisiac due to its disinhibition properties. Meanwhile, alcohol has long been regarded as a risk factor for ED, whether in textbooks, review articles or in clinical teaching.

Nevertheless, most population-based epidemiological studies have constantly demonstrated that alcohol consumption, like its relationship with CAD, is related to erectile function in a J-shaped manner, with moderate consumption conferring the highest protection and greater consumption fewer benefits (Table 3).[30–41] Chew et al.[30] conducted a population-based cross-sectional study on 1770 Australian men and found that compared with never-drinkers, the age-adjusted odds of ED were lower among current, weekend and binge drinkers and higher among ex-drinkers. Meta-analysis of population-based cross-sectional studies found significantly decreased odds of ED among alcohol drinkers in 7 of the 11 studies cited.[42] Similar results have been reported in longitudinal studies. However, the prospective Massachusetts Male Aging Study enrolled 513 men without ED at baseline and the results showed that there was no significant difference in the incidence of ED among different degrees of alcohol consumption, after adjusting for confounding factors.[31] The Boston Area Community Health Survey showed significantly decreased odds of moderate to severe ED with alcohol use of one to three drinks a day.[32]

Considering that ED and heart disease share similar cardiovascular risk factors, and taking into account the well-known chronic cytotoxic effects of alcohol on hepatic function and immune function, general health might be a mediator between the association of high alcohol consumption and ED. This finding of a J-shaped relationship might explain why studies have shown harmful effects of heavy alcoholism on erectile function.

Chronic and long-term alcohol use directly or indirectly affects nearly every organ system in the body. O'Farrell et al.[43] found that alcoholic men had more than three times the prevalence of serious ED of demographically

Table 3 Summary of population-based studies of alcohol consumption on erectile dysfunction

Authors	Sample	Category of alcohol consumption	OR (95% CI) for having ED	Study design
Chew et al.[30]	1580 Australian men aged 20–80^{+} yr	Never-drinkers <1 drink/wk 1–20 drinks/wk 21–30 drinks/wk >30 drinks/wk	1 0.78 (0.31–2.00) 0.54 (0.24–1.24) 0.56 (0.22–1.42) 0.59 (0.23–1.53)	Population-based cross-sectional study; ED assessed by IIEF-5
Feldman et al.[31]	513 American men aged 40–70 yr	<1 drink/d 1–3 drinks/d ≥4 drinks/d	1 0.95 (0.54–1.67) 0.87 (0.41–1.86)	Prospective cohort study from MMAS; ED assessed by a single question
Bacon et al.[32]	22,086 American men aged 40–75 yr	0 g/d 0.1–4.9 g/d 5.0–14.9 g/d 15.0–29.9 g/d ≥30 g/d	1 1.0 (0.9–1.1) 1.0 (0.9–1.1) 1.0 (0.9–1.1) 1.1 (1.0–1.2)	Prospective cohort study from HPFS; ED assessed by a single question
Millett et al.[33]	8367 Australian men aged 16–59 yr	Non-drinker 1–4 drinks/d >4 drinks/d	1 0.36 (0.28–0.45) 0.89 (0.81–1.34)	Cross-sectional random study; ED assessed by a single question
Terai et al.[34]	2084 Japanese men aged 40–60^{+} yr	Daily/not daily	1.70 (0.96–2.99)	Cross-sectional study of noninstitutionalized men; ED assessed by IIEF-5
Cho et al.[35]	3501 Korean men aged 20–60^{+} yr	None or light (<3/wk) Moderate (3–4/wk) Heavy (>4/wk)	1 0.91 (0.70–1.18) 1.36 (0.95–1.96)	Cross-sectional hospital-based sample; ED assessed by IIEF-5
Bai et al.[36]	2203 Chinese men aged 20–86 yr	Yes/No	0.69 (0.56–0.84)	Cross-sectional population-based study; ED assessed by IIEF-5
Shaeer et al.[37]	1814 Pakistani, Egyptian or Nigerian men aged 35–70 yr	Yes/No	0.53 (0.39–0.74)	Random hospital-based sample; ED assessed by a single question
Nicolosi et al.[38]	2417 Brazilian, Italian, Japanese or Malay men aged 40–70 yr	None 1–7 drinks/wk ≥8 drinks/wk	1 0.74 (0.53–1.02) 0.73 (0.53–0.99)	Community-based cross-sectional study; ED assessed by a single question
Moreira et al.[39]	602 Brazilian men aged 40–70 yr	Yes/No	0.54 (0.37–0.87)	Cross-sectional population study; ED assessed by a single question
Akkus et al.[40]	1982 Turkish men aged ≥40 yr	Yes/No	0.55 (0.36–0.85)	Stratified random sample; ED assessed by a single question
Mak et al.[41]	799 Belgian men aged 40–70 yr	<20 g/d 20–40 g/d >40 g/d	1 0.56 (0.37–0.84) 1.29 (0.88–1.88)	Aged-stratified random population study; ED assessed by IIEF

OR = odds ratio; CI = confidence interval; ED = erectile dysfunction; IIEF = International Index of Erectile Function; MMAS = Massachusetts Male Aging Study; HPFS = Health Professionals Follow-up Study.

similar nonalcoholic men. Mandell and Miller[44] interviewed 44 alcoholic men and found that 59% experienced ED during periods of heavy drinking and 84% reported some sexual dysfunction related to alcohol abuse. Snyder and Karacan[45] measured nocturnal penile tumescence in a sample of 26 alcoholic men going through detoxification, and found that alcoholic men were more likely to have fewer, slower and less rigid nocturnal erections than a nonalcoholic comparison group. The research from Lemere and Smith[46] suggested that many alcoholics suffered from ED even after years of sobriety, possibly due to the permanent neurological damage caused by long-term alcohol use. Some long-term male alcoholics may experience feminization syndrome associated with advanced liver cirrhosis and characterized by ED, testicular atrophy and gynecomastia.[47]

5. Limitations

ED is a mutifactorial disorder, and the association between alcohol and ED could be confounded by other factors. In epidemiologic studies, it is difficult to distinguish the effects of ethanol from those of associated socioeconomic influences, tobacco, drug abuse, or diet. Poor health habits are common in alcohol abusers. Causal inference from the cross-sectional design is weak. The alternatives for better causal inference are either a cohort study or a randomized controlled trial, but in either case, few studies have been done. The focus of relevant studies is generally on health risks, without consideration of alcohol-attributable social harm. It should be noted that the potential cardiovascular or erectile function benefits form alcohol consumption can also be gained from other means, such as exercise or diet modification.[48]

In addition to amount of consumption, drinking behaviors and the above confounders, there are a number of additional factors that influence the effect of alcohol consumption. Klatsky et al.[19] reported that individuals who prefer wine tend to have amore favorable lifestyles, better cardiovascular risk profiles, smoke less and are better educated. More research needs to be done to assess the association between acute and chronic alcohol consumption and the development of ED, particularly using large-scale cohorts since randomized controlled trials may be unethical.

6. Conclusion

ED and CAD share similar risk factors. Most epidemiological studies have found that alcohol consumption, like its relationship with CAD, is related to erectile function in a J-shaped manner, with moderate consumption conferring the highest protection and greater consumption fewer benefits. The beneficial effects of alcohol are attributed to its antioxidant, vasorelaxant and antithrombotic properties, as well as elevation of HDL and increase of NO production. Nevertheless, alcoholism is reported to be highly associated with cardiomyopathy and ED. More studies are needed to assess the effect of alcohol consumption on cardiovascular disease and erectile function.

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