Reviews in Cardiovascular Medicine

*Rev. Cardiovasc. Med.* **2023**; 24(10): 292
https://doi.org/10.31083/j.rcm2410292

*Short Communication*

# Detrimental Effects of Alcohol on the Heart: Hypertension and Cardiomyopathy

Vincent M. Figueredo[1,*], Akash Patel[1]

[1]Department of Cardiology, St Mary Medical Center, Langhorne, PA 19047, USA
*Correspondence: vincent.figueredo@stmaryhealthcare.org (Vincent M. Figueredo)

Academic Editors: Carl J. Lavie and Guido Grassi
Submitted: 26 December 2022 Revised: 6 May 2023 Accepted: 18 May 2023 Published: 17 October 2023

**Abstract**

**Background**: Epidemiological evidence suggests a J-shaped association between alcohol consumption and cardiovascular mortality, with higher cardiovascular event rates occurring among abstainers and heavy drinkers compared to moderate consumers. However, this hypothesis has been challenged by more recent studies. Furthermore, ethnicity, gender, type of alcoholic beverage, and pattern of alcohol intake, influence the relationship between alcohol and heart health. **Methods**: We undertook a review of the relavent literature utilizing PubMed. **Results**: Heavy alcohol consumption causes resistant hypertension, cardiomyopathy, arrhythmias, hemorrhagic strokes, as well as hepatic cirrhosis and pancreatitis. Excessive drinking is the third most preventable cause of death worldwide behind hypertension and smoking. **Conclusions**: In this review, we discuss the effects of alcohol abuse on hypertension (a major cause of myocardial infarction and stroke) and alcoholic cardiomyopathy. Another article in this Special Issue "Alcohol and Heart Health" discusses the problem with alcohol and arrhythmias sudden cardiac death.

**Keywords:** alcoholic; alcohol abuse; cardiomyopathy; hypertension; heart failure

## 1. Introduction

Alcohol, a psychoactive substance, has been a significant part of human culture for millennia. Throughout history it has been recognized for its harmful potential to individuals, as well as to society, due to its dependence-producing properties. Harmful alcohol consumption contributes to health, social, and economic burdens globally.

Significant alcohol consumption remains highly prevalent globally. In 2018, alcohol consumption averaged 6.4 liters of pure alcohol per person worldwide [1]. In the United States, 85% of individuals 18 years-old or older have consumed alcohol in their lifetime [2]. Furthermore, binge drinking, a major reason for alcohol related problems, is widespread. According to a United States governmental survey in 2019, 26% of individuals aged 18 and older reported binge drinking habits within the last month [3].

The abusive use of alcohol costs billions of dollars globally. In 2010, excessive alcohol consumption cost the healthcare sector approximately 28 billion dollars [4]. If damages related to workplace productivity, crime, and accidents were included, the costs is more than 250 billion dollars a year [4]. The World Health Organization (WHO) reported that excessive alcohol use is a risk factor for more than 200 diseases, accounting for more than 5% of the global burden of diseases and injuries [1]. Furthermore, it acts as a common risk factor for mental disorders and abnormal behavioral conditions [1]. The Organization for Economic Cooperation and Development Strategic Public Health Planning for Non-Communicable Diseases (OECD SPHeP-NCDs) model predicts that consuming more than 1 drink per day for women and 1.5 drinks per day for men will cause 24 million new cases of cardiovascular diseases, 10 million cancer cases,12 million new diabetes cases, and 37 million injury cases between 2020–2050 in 52 countries [5]. Consistent excessive drinking can lead to alcohol use disorder (AUD). Individuals with this condition consume too much alcohol despite the mental and physical harms. In USA, approximately 15 million individuals had been diagnosed with AUD [6].

Excessive alcohol consumption causes 3 million deaths yearly, and it accounts for 5% of all deaths worldwide. It was the 7th leading cause of early death globally in 2016 [7]. In addition, 14% of all deaths of individuals between age 20 and 39 in 2016 were alcohol consumption related [1]. Most alcohol attributable deaths (28.7%) in 2016 were due to injuries. Other complications were cardiovascular diseases (19%), liver cirrhosis and pancreatitis (21.3%), and cancer (12.6%) [1]. Due to increased alcohol consumption seen in adults worldwide, an increased focus needs to be placed on the cardiovascular harms of heavy drinking.

Heavy alcohol use is defined as greater than 4 drink per day or 14 drinks per week for men and greater than 3 drinks per day or 7 drinks per week for women [8]. While numerous studies suggest a cardioprotective effect of light to moderate alcohol consumption, especially on coronary artery disease, heavy alcohol consumption, abuse and binging, can negatively affect the cardiovascular system. Potential cardiovascular detrimental effects include congestive

**Table 1. Study characteristics comparing alcohol use and hypertension.**

| Study | Study design | Country | Number of participants | % Men and women | Average age (years) | Alcohol consumption | HR/OR/RR (95% Confidence Interval) |
|---|---|---|---|---|---|---|---|
| Bae *et al*., 2002 [18] | Nested Case Control | South Korea | 988 | 100% Men | 40–59 | 71–280 g/week | OR:<br>1.84 (1.31–2.56) |
| Sesso *et al*., 2008 [19] | Prospective Cohort | United States of America | 42,303 | 32% Men<br>68% Women | Men: 52<br>Women: 53.8 | 5–6 drinks/week | RR:<br>Men: 1.15 (1.04–1.27)<br>Women: 0.90 (0.80–1.00) |
| Forman *et al*., 2009 [20] | Prospective Cohort | United States of America | 83,882 | 100% Women | 27–44 | 5.1–10.0 g/day<br>10.1–15.0 g/day<br>15.1–29.9 g/day<br>≥30 g/day | HR:<br>0.84 (0.78–0.90)<br>0.98 (0.91–1.07)<br>1.11 (1.01–1.23)<br>1.61 (1.42–1.82) |
| Ascherio *et al*., 1996 [21] | Prospective Cohort | United States of America | 41,541 | 100% Women | 38–63 | 20–29.9 g/day<br>≥30 g/day | RR:<br>1.46 (1.21–1.76)<br>1.32 (1.11–1.57) |
| Halanych *et al*., 2010 [22] | Prospective Cohort | United States of America | 659 | 56.6% Men<br>43.4% Women | 18–30 | 7–14+ drinks/week | HR:<br>1.33 (0.76–2.32) African-American Men<br>1.47 (0.44–4.96) European-American Men |
| Peng *et al*., 2013 [23] | Prospective Cohort | China | 32,389 | 74.2% Men<br>25.8% Women | 49.9 | 50–99 g/day<br>100–149 g/day<br>≥150 g/day | RR:<br>1.80 (1.63–2.00)<br>2.06 (1.83–2.31)<br>2.28 (1.99–2.61) |
| Okubo *et al*., 2014 [24] | Prospective Cohort | Japan | 45,428 | 35.6% Men<br>64.4% Women | 40–79 | 20–39.9 g/day<br>20–59.9 g/day<br>≥60 g/day | HR:<br>Men:<br>1.29 (1.24–1.35)<br>1.45 (1.39–1.52)<br>1.57 (1.46–1.70)<br>Women:<br>1.10 (1.03–1.17)<br>1.14 (0.93–1.40)<br>1.29 (0.89–1.87) |
| Diederichs *et al*., 2017 [25] | Prospective Cohort | Germany | 2231 | 47.5% Men<br>52.5% Women | 18–79 | ≥10–20 g/day | OR:<br>Men: 2.88, *p*-value 0.014<br>Women: 2.13, *p*-value 0.092 |

**Table 1. Continued.**

| Study | Study design | Country | Number of participants | % Men and women | Average age (years) | Alcohol consumption | HR/OR/RR (95% Confidence Interval) |
|---|---|---|---|---|---|---|---|
| Saremi *et al*., 2004 [26] | Prospective Cross Sectional | United States of America | 3789 | 34.2% Men<br>65.8% Women | 36.3 | 1–2 drinks/day | HR:<br>Men: 1.20 (0.95–1.53)<br>Women: 1.48 (1.24–1.78) |
| Yoo *et al*., 2019 [27] | Prospective Cohort | South Korea | 6259 | 34.5% Men<br>65.5% Women | 40–69 | ≥5 and <30 g/day | HR:<br>Men: 1.292 (1.033–1.617)<br>Women: 1.128 (0.652–1.952) |
| Fuchs *et al*., 2001 [28] | Prospective Cohort | United States of America | 8334 | 33.2% White Men<br>40% White Women<br>10.0% Black Men<br>16.8% Black Women | 45–64 years | 1 to 209 g/week | OR:<br>White men: 0.88 (0.71–1.08)<br>White women: 0.89 (0.73–1.09)<br>Black men: 1.71 (1.11–2.64)<br>Black women: 0.88 (0.59–1.33) |
| Ohmori *et al*., 2002 [29] | Prospective Cohort | Japan | 267 | 39% Men<br>61% Women | 40+ | 23–45 g/week | RR:<br>Men: 2.60 (1.50–4.49) |

Table notes: OR, odds ratio; HR, hazard ratio; RR, relative risk.

heart failure, dilated cardiomyopathy, sudden cardiac death and hemorrhagic stroke. Some of these cardiovascular complications are the result of alcohol-induced hypertension. In this review, we discuss the effects of alcohol abuse on hypertension (a major cause of myocardial infarction and stroke) and alcoholic cardiomyopathy. Another article in this Special Issue "Alcohol and Heart Health" discusses the problem with alcohol and arrhythmias and sudden cardiac death.

## 2. Alcohol and Hypertension

Hypertension is the most common medical condition in the world, with 1.28 billion cases in 2021 [9]. Hypertension is a major cause of premature death worldwide [10]. Moreover, many individuals with hypertension are unaware that they have it (~46%) [9]. It is a risk factor for stroke, cardiovascular and kidney diseases [9,10]. According to WHO, increased alcohol consumption is a major risk factor for developing hypertension [9]. The correlation between alcohol consumption and hypertension had been investigated in cross sectional, cohort and epidemiological studies across various populations. Meta-analyses of these studies have also been performed and published in the last decade which provide the strongest evidence for an association between alcohol and hypertension.

Roerecke *et al*. [11] conducted a meta-analysis investigating the incidence of hypertension in moderate drinking individuals. Their analysis included 361,254 participants from 20 original cohort studies. They found the relationship between alcohol and hypertension is dependent on gender and dose. Any amount of drinking was shown to be associated with more hypertension in men. In women, no risk was found at 1 to 2 drinks per day, but greater risk was found at higher levels of consumption [11]. Similar results were observed by Aladin and colleagues [12]. They found that moderate drinkers (7–13 drinks/week) were at increased risk for stage 1 and stage 2 hypertension, when compared to never drinkers, in 17,059 subjects from NHANES III (Third National Health and Nutrition Examination Survey) [12].

Another meta-analysis by Roerecke *et al*. [13], investigating whether reducing the intake of alcohol had any effect on blood pressure, studied data from 36 trials. They demonstrated that individuals who had $\leq$2 drinks per day had no effect on their blood pressure when alcohol consumption was reduced, while individuals consuming $>$2 drinks per day had significant reductions in their blood pressure with reduced alcohol intake [13].

McFadden and colleagues [14], studying the effects of daily alcohol consumption on blood pressure, found that there is a statistically significant increase in blood pressure immediately after alcohol intake.

Chen *et al*. [15] applied a Mendelian randomization approach to study the effects of alcohol on blood pressure in subjects with the alcohol dehydrogenase 2 (ALDH2) genotype (aldehyde dehydrogenase 2 is a major enzyme in alcohol metabolism). They concluded that alcohol intake had a significant immediate effect on blood pressure, as well as it increasing the risk for hypertension [15].

Jung *et al*. [16] recently conducted a regional analysis of the relationship between alcohol and hypertension. They found that the risk of hypertension is dose dependent in both Asian and western populations [16]. Further, hypertension risk is evident even at lower levels of consumption compared to recommended guidelines. Asian populations showed a higher risk of hypertension at low doses of alcohol compared to a western population [16]. Another meta-analysis showed that even at low to moderate doses of alcohol, men had an increased trend towards development of hypertension, when compared to women [17]. Table 1 (Ref. [18–29]) shows study characteristics for studies included in the above reviews and meta-analyses examining alcohol use and hypertension.

Thus, the risk of hypertension is dose dependent with higher use of alcohol contributing to higher hypertension risk. Other factors which contribute include gender, timing of alcohol consumption (e.g., binging), and genetics.

## 3. Dilated Cardiomyopathy

Alcoholic cardiomyopathy is a type of dilated cardiomyopathy (DCM) demonstrating increased left ventricular (LV) mass and decreased ventricular function. Alcoholic cardiomyopathy is similar to other dilated non-ischemic cardiomyopathies. The majority of alcohol abusers are asymptomatic for years. Most never develop clinical manifestations of congestive heart failure. However, most alcoholics do demonstrate preclinical heart muscle disease. Autopsies reveal dilated cardiomyopathic in alcoholics not experiencing symptomatic heart failure [30]. It has been estimated that 2% of heavy alcohol users ultimately develop symptomatic alcoholic cardiomyopathy. And because there are so many alcoholics, approximately 35% of all non-ischemic cardiomyopathies are caused by excessive alcohol use [31–33].

Data suggest that most alcoholics develop significant changes in cardiac function and myocyte structure after consuming on average greater than 90 grams of alcohol daily for at greater than 5 years [34–37]. Cardiac damage due to longstanding heavy alcohol consumption is not beverage or quantity specific and varies depending on the population studied. Genetic and environmental factors play a role, as well as the specific beverage type used by a culture or individual.

Studies have examined the association between alcohol abuse and cardiomyopathy [31–33]. In one case control study researchers found that 40% of DCM cases were attributable to excessive alcohol use history [38]. In another case control study by Komajda *et al*. [39], abnormal use of alcohol was found to be strong predictor for cardiomyopathy cases irrespective of the type of beverage used. Gillet *et al*. [40] found that the higher use of alcohol ($>$82 g/day)

**Table 2. Study Characteristics Comparing Alcohol Use and Congestive Heart Failure.**

| Study | Study design | Country | Number of participants | % Men and women | Average age (years) | Alcohol consumption | HR/OR/RR (95% Confidence Interval) |
|---|---|---|---|---|---|---|---|
| Klatsky *et al*., 2005 [42] | Prospective Cohort | United States of America | 1035 | 44.4% Men<br>55.6% Women | 74 | 1–2 drinks/day<br>3–5 drinks/day<br>≥6 drinks/day | RR:<br>1.0 (0.8–1.3)<br>1.2 (0.9–1.6)<br>1.7 (1.1–2.6) |
| Park *et al*., 2018 [43] | Prospective Cohort | South Korea | 49,714 | 72.2% Men<br>27.8% Women | 49.1 | 15–30 g/day<br>30–60 g/day<br>>60 g/day | OR:<br>1.25 (1.08–1.44)<br>1.33 (1.15–1.54)<br>1.32 (1.11–1.57) |
| Larsson *et al*., 2015 [44] | Meta analysis | United States of America Canada Sweden Finland | 6211 | Variable among studies | 21–85 | 14 drinks/week | RR:<br>1.07 (0.77–1.48) |
| Whitman *et al*., 2017 [41] | Prospective Cohort | United States (California) | 268,084 | 68.6% Men<br>31.4% Women | 48.8 | 7–14+ drinks/week | HR:<br>2.34 (2.29–2.39) |
| Sidorenkov *et al*., 2011 [45] | Cohort Autopsy Study | Russia (Arkhangelsk) | 318 | 46.9% Male<br>53.1% Female | 30–70 years | 7–14+ drinks/week | OR:<br>1.36 (0.74–2.48) |
| Aguilar *et al*., 2004 [46] | Prospective Cohort | Canada United States of America | 2228 | 82.5% Men<br>17.5% Women | 58 | 1–10 drinks/week<br>>10 drinks/week | HR:<br>0.93 (0.75–1.17)<br>1.25 (0.91–1.72) |
| Li *et al*., 2016 [47] | Prospective Cohort | China | 10,824 | 46.1% Men<br>53.9% Women | 54 | 1–2 drinks/day<br>3+ drinks/day | OR:<br>1.183 (0.774–1.808)<br>1.482 (1.117–1.965) |
| Yousaf *et al*., 2014 [48] | Prospective Cohort | United States (Minnesota) | 2042 | 52.3% Men<br>47.7% Women | 63.1 | <1 drinks/day<br>1–2 drinks/day<br>>2 drinks/day | OR:<br>0.14 (0.04–0.43)<br>1.56 (0.39–5.20)<br>4.75 (1.18–15.98) |

Table notes: OR, odds ratio; HR, hazard ratio; RR, relative risk.

was seen in DCM group compared to a control group (32 g/day) in a French population. Whitman and colleagues [41] performed a prospective cohort study on 268,084 alcoholics in California, United States, using the Healthcare Cost and Utilization Project database. They found a significant increase in congestive heart failure cases compared to the non-alcoholic population, with a hazard ratio of 2.34. Table 2 (Ref. [41–48]) shows study characteristics for studies included in the above reviews and meta-analyses examining alcohol use and congestive heart failure.

The toxic effects on alcohol on muscle cells are well recognized [49,50]. In 1989, researchers demonstrated that long term alcohol use produces toxic effects on striated muscles, including heart and skeletal muscles [51]. Investigators also found that acetaldehyde, a metabolite of alcohol metabolism, negatively affected cardiac and skeletal muscle [52]. *In vitro* studies conducted on cardiomyocytes found that ethanol interferes with a number of muscle cell functions. For example, Guarnieri and Lakatta [53] demonstrated that ethanol inhibits the calcium-myofilament interaction, interfering with electromechanical coupling of muscle cell contractile filaments. Das and Harris [54] found that mitochondrial adenosine triphosphate (ATP) synthase becomes defective in the presence of alcohol leading to further loss of function in rat cardiomyocytes. Other studies found that ethanol has apoptotic effects on cardiac myocytes damaging overall function of heart [55,56].

Research has shown alcohol consumption affects all areas of cell protein metabolism, from its synthesis to degradation. Human and mouse tissue studies demonstrated alcohol is a myocardial toxin and causes ultrastructural damage. Alcohol damage can include sarcoplasmic reticulum edema, contractile element fragmentation, intercalated disc expansion, and fatty deposition [57]. Alcohol produces dose dependent depression of contractile function in rat cardiomyocytes due sarcoplasmic calcium depletion [58] and decreased myofilament calcium sensitivity [59]. Studies have found an inverse relationship between use of alcohol and protein synthesis [60,61]. Vary and colleagues [62] showed that chronic alcohol feeding in rats produced lower heart weights due to a 25% loss of cardiac proteins and a 30% reduction in the rate of protein synthesis. Potential mechanisms of alcoholic cardiomyopathy are shown in Fig. 1. Of note, Steiner and colleagues [60] reported that only chronic heavy consumption interferes with protein synthesis while moderate use has little to no effect.

Asymptomatic impairment of echocardiographic systolic and diastolic function parameters is found in most alcoholics [51]. Alcoholics have lower left ventricular ejection fraction, increased end diastolic volume, decreased mean fractional shortening and a larger mean left ventricular mass in a dose-dependent fashion [51]. Only a small percentage go on to develop overt manifestations of heart failure.

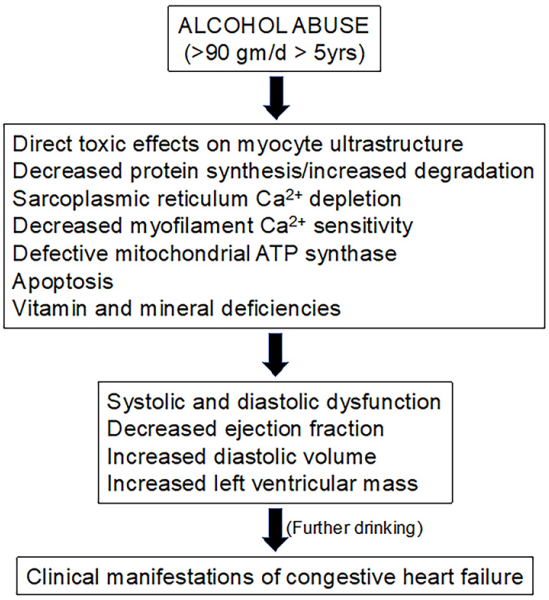


**Fig. 1. Potential mechanisms of alcoholic cardiomyopathy.** ATP, adenosine triphosphate.

Symptomatic alcoholic cardiomyopathy is similar to other dilated cardiomyopathies. Diagnosis can be complicated by the presence of other risk factors. Taking a good history is important to rule out other risk factors, including prescribed and non-prescribed drugs (e.g., doxorubicin and cocaine), diabetes and coronary artery disease. Alcoholic cardiomyopathy is a diagnosis of exclusion.

Clinical findings of heart failure, including a S3 gallop, jugular venous dilation, cardiomegaly, and rales can be present. Co-existing alcoholic cirrhosis can lead to diagnostic confusion. Supraventricular arrhythmias (holiday heart syndrome) and sudden cardiac death are complications of alcohol abuse. Causes of death in alcoholic cardiomyopathy are similar to those with idiopathic cardiomyopathy, progressive chronic heart failure and sudden cardiac death [34]. Coexisting alcoholic cardiomyopathy and cirrhosis carries a worse prognosis [63].

Studies have not found an association between drinking in moderation and the development of cardiomyopathy. In fact, studies have reported a beneficial effect of moderate drinking in reducing mortality for patients with heart failure. For example, the Framingham heart study reported a lower incidence of heart failure with moderate alcohol consumption compared to patients who drank less than 1 drink per week [64,65]. Another study reported a statistically significant reduction in the incidence of heart failure in individuals consuming moderate alcohol among older adults compared to those abstaining from alcohol [66]. In the Physicians Heart Study I, they observed a 56% decreased risk of heart failure in association with moderate consumption of alcohol [64].

The treatment of alcoholic cardiomyopathy is similar to that of other non-ischemic cardiomyopathies. Com-

plete abstinence from alcohol is the mainstay of treatment, though even moderation may help. Four-year mortality for those who continue to abuse alcohol is near 50%. Treatment centers around heart failure guidelines from the European Society of Cardiology or the American College of Cardiology. Guideline-directed therapy begins with a combination of beta-blockers and an angiotensin-converting enzyme inhibitor, angiotensin receptor blocker, or angiotensin blocker-neprilysin inhibitor, and an aldosterone receptor antagonist [31]. Symptomatic management may require diuretic therapy. Co-existing nutritional deficiencies (vitamins, minerals such as magnesium, selenium, or zinc) should be supplemented. If concurrent atrial fibrillation is present, digitalis and anticoagulants may help.

Abstinence-maintaining medications, such as naltrexone, acamprosate, disulfiram, and nalmefen have shown some success in cardiomyopathy patients [67]. Spironolactone has recently been evaluated as a potential therapy in alcoholic cardiomyopathy patients, demonstrating not only therapeutic effects on treatment of cardiomyopathy, but an effect of reducing alcohol craving [68]. Little data is published for heart transplantation in alcoholic cardiomyopathy patients, and relapse would be a concern. Relapse rates in a study of liver transplants was 5.6 cases per 100 patients per year for any alcohol use and 2.5 cases per 100 patients per year for heavy alcohol use [69]. Likelihood of myocyte and contractile recovery depend first on amount and duration of alcohol consumption. Irreversible changes with cell death and fibrosis prevent recovery. For viable myocytes to recover, abstinence and compliance with guideline-directed medical therapy are necessary, as with any dilated cardiomyopathy.

## 4. Conclusions

Current guidelines and most physicians today will recommend to their patients limiting alcohol consumption to one drink per day for women, and two drinks per day for men. While epidemiological data suggests that light to moderate alcohol consumption can have advantageous cardiovascular effects, newer studies have shown that these recommendations should be tailored for each individual patient. In patients with or at higher risk for hypertension, newer studies show that even moderate drinking can lead to the progression of hypertension.

Abusing alcohol can cause a cardiomyopathy similar to other dilated cardiomyopathies. While the majority of alcoholics are clinically asymptomatic (mild systolic and diastolic dysfunction) and may never develop clinical manifestations of heart failure, a small percentage go on to develop symptomatic dilated cardiomyopathy. Because there are so many heavy drinkers, alcoholic cardiomyopathy may be the most common nonischemic dilated cardiomyopathy.

## Availability of Data and Materials

Not applicable.

## Author Contributions

AP and VF reviewed the literature and prepared the manuscript. AP and VF contributed to editorial changes in the manuscript. All authors read and approved the final manuscript. All authors have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

## Ethics Approval and Consent to Participate

Not applicable.


## Acknowledgment

Not applicable.

## Funding

This research received no external funding.


## Conflict of Interest

The author declares no conflict of interest. Vincent Figueredo is serving as one of the Editorial Board members and Guest editors of this journal. We declare that Vincent Figueredo had no involvement in the peer review of this article and has no access to information regarding its peer review. Full responsibility for the editorial process for this article was delegated to Carl J. Lavie and Guido Grassi.

## References


[1] WHO. Global Status Report on Alcohol and Health 2018. 2018. Available at: https://www.who.int/publications/i/item/9789241565639 (Accessed: 6 March 2023).

[2] Administration SA and MHS. Alcohol Use in Lifetime, Past Year, and Past Month among Persons Aged 12 or Older, by Detailed Age Category: Percentages, 2018 and 2019. 2018. Available at: https://www.samhsa.gov/data/sites/default/files/reports/rpt29394/NSDUHDetailedTabs2019/NSDUHDetTabsSect2pe2019.htm (Accessed: 6 March 2023).

[3] Administration SA and MHS. Binge Alcohol Use in Past Month among Persons Aged 12 or Older, by Age Group and Demographic Characteristics: Percentages, 2018 and 2019. 2018. Available at: https://www.samhsa.gov/data/sites/default/files/reports/rpt29394/NSDUHDetailedTabs2019/NSDUHDetTabsSect2pe2019.htm (Accessed: 6 March 2023).

[4] CDC. The Cost of Excessive Alcohol Use. 2019. Available at: https://www.cdc.gov/alcohol/onlinemedia/infographics/cost-excessive-alcohol-use.html (Accessed: 6 March 2023).

[5] OECD. SPHeP-NCD. Organisation for Economic Co-operation and Development. 2019. Available at: http://oecdpublichealthexplorer.org/ncd-doc/ (Accessed: 6 March 2023).

[6] Administration SA and MHS. Alcohol Use Disorder in Past Year among Persons Aged 12 or Older, by Age Group and Demographic Characteristics: Numbers in Thousands, 2018 and 2019. 2018. Available at: https://www.samhsa.gov/data/sites/default/files/reports/rpt29394/NSDUHDetailedTabs2019/NSDUHDetTabsSect2pe2019.htm (Accessed: 6 March 2023).

[7] Griswold MG, Fullman N, Hawley C, Arian N, Zimsen SRM, Tymeson HD, *et al*. Alcohol use and burden for 195 countries

and territories, 1990-2016: a systematic analysis for the Global Burden of Disease Study 2016. Lancet. 2018; 392: 1015–1035.
[8] United States Department of Agriculture. Dietary Guidelines For Americants 2020–2025. 2020. Available at: https://www.dietaryguidelines.gov/sites/default/files/2020-12/Dietary_Guidelines_for_Americans_2020-2025.pdf (Accessed: 6 March 2023).
[9] WHO. Hypertension. 2021. Available at: https://www.who.int/news-room/fact-sheets/detail/hypertension (Accessed: 6 March 2023).
[10] Xu J, Murphy SL, Kochanek KD, Arias E. Mortality in the United States 2018. 2020. Available at: https://www.cdc.gov/nchs/data/databriefs/db355-h.pdf (Accessed: 6 March 2023).
[11] Roerecke M, Tobe SW, Kaczorowski J, Bacon SL, Vafaei A, Hasan OSM, *et al*. Sex-Specific Associations Between Alcohol Consumption and Incidence of Hypertension: A Systematic Review and Meta-Analysis of Cohort Studies. Journal of the American Heart Association. 2018; 7: e008202.
[12] Aladin A, Chevli P, Ahmad MI, Rasool S, Herrington D. Alcohol Consumption and Risk of Hypertension. Journal of the American College of Cardiology. 2019; 73: 12–12.
[13] Roerecke M, Kaczorowski J, Tobe SW, Gmel G, Hasan OSM, Rehm J. The effect of a reduction in alcohol consumption on blood pressure: a systematic review and meta-analysis. The Lancet Public Health. 2017; 2: e108–e120.
[14] McFadden CB, Brensinger CM, Berlin JA, Townsend RR. Systematic review of the effect of daily alcohol intake on blood pressure. American Journal of Hypertension. 2005; 18: 276–286.
[15] Chen L, Smith GD, Harbord RM, Lewis SJ. Alcohol intake and blood pressure: a systematic review implementing a Mendelian randomization approach. PLoS Medicine. 2008; 5: e52.
[16] Jung MH, Shin ES, Ihm SH, Jung JG, Lee HY, Kim CH. The effect of alcohol dose on the development of hypertension in Asian and Western men: systematic review and meta-analysis. The Korean Journal of Internal Medicine. 2020; 35: 906–916.
[17] Briasoulis A, Agarwal V, Messerli FH. Alcohol consumption and the risk of hypertension in men and women: a systematic review and meta-analysis. Journal of Clinical Hypertension. 2012; 14: 792–798.
[18] Bae JM, Ahn YO. A nested case-control study on the high-normal blood pressure as a risk factor of hypertension in Korean middle-aged men. Journal of Korean Medical Science. 2002; 17: 328–336.
[19] Sesso HD, Cook NR, Buring JE, Manson JE, Gaziano JM. Alcohol consumption and the risk of hypertension in women and men. Hypertension. 2008; 51: 1080–1087.
[20] Forman JP, Stampfer MJ, Curhan GC. Diet and lifestyle risk factors associated with incident hypertension in women. The Journal of the American Medical Association. 2009; 302: 401–411.
[21] Ascherio A, Hennekens C, Willett WC, Sacks F, Rosner B, Manson J, *et al*. Prospective study of nutritional factors, blood pressure, and hypertension among US women. Hypertension. 1996; 27: 1065–1072.
[22] Halanych JH, Safford MM, Kertesz SG, Pletcher MJ, Kim YI, Person SD, *et al*. Alcohol consumption in young adults and incident hypertension: 20-year follow-up from the Coronary Artery Risk Development in Young Adults Study. American Journal of Epidemiology. 2010; 171: 532–539.
[23] Peng M, Wu S, Jiang X, Jin C, Zhang W, Kailuan Cardiovascular Survey Group. Long-term alcohol consumption is an independent risk factor of hypertension development in northern China: evidence from Kailuan study. Journal of Hypertension. 2013; 31: 2342–2347.
[24] Okubo Y, Sairenchi T, Irie F, Yamagishi K, Iso H, Watanabe H, *et al*. Association of alcohol consumption with incident hypertension among middle-aged and older Japanese population: the Ibarakai Prefectural Health Study (IPHS). Hypertension. 2014; 63: 41–47.
[25] Diederichs C, Neuhauser H. The incidence of hypertension and its risk factors in the German adult population: results from the German National Health Interview and Examination Survey 1998 and the German Health Interview and Examination Survey for Adults 2008-2011. Journal of Hypertension. 2017; 35: 250–258.
[26] Saremi A, Hanson RL, Tulloch-Reid M, Williams DE, Knowler WC. Alcohol consumption predicts hypertension but not diabetes. Journal of Studies on Alcohol. 2004; 65: 184–190.
[27] Yoo MG, Park KJ, Kim HJ, Jang HB, Lee HJ, Park SI. Association between alcohol intake and incident hypertension in the Korean population. Alcohol. 2019; 77: 19–25.
[28] Fuchs FD, Chambless LE, Whelton PK, Nieto FJ, Heiss G. Alcohol consumption and the incidence of hypertension: The Atherosclerosis Risk in Communities Study. Hypertension. 2001; 37: 1242–1250.
[29] Ohmori S, Kiyohara Y, Kato I, Kubo M, Tanizaki Y, Iwamoto H, *et al*. Alcohol intake and future incidence of hypertension in a general Japanese population: the Hisayama study. Alcoholism: Clinical and Experimental Research. 2002; 26: 1010–1016.
[30] Davidson DM. Cardiovascular effects of alcohol. The Western Journal of Medicine. 1989; 151: 430–439.
[31] Shaaban A, Gangwani MK, Pendela VS, Vindhyal MR. Alcoholic Cardiomyopathy. In StatPearls [Internet]. StatPearls Publishing: Treasure Island (FL). 2022.
[32] Elliott P, Andersson B, Arbustini E, Bilinska Z, Cecchi F, Charron P, *et al*. Classification of the cardiomyopathies: a position statement from the European Society Of Cardiology Working Group on Myocardial and Pericardial Diseases. European Heart Journal. 2008; 29: 270–276.
[33] Movva R, Figueredo VM. Alcohol and the heart: To abstain or not to abstain? International Journal of Cardiology. 2013; 164: 267–276.
[34] Fauchier L, Babuty D, Poret P, Casset-Senon D, Autret ML, Cosnay P, *et al*. Comparison of long-term outcome of alcoholic and idiopathic dilated cardiomyopathy. European Heart Journal. 2000; 21: 306–314.
[35] Kupari M, Koskinen P, Suokas A, Ventilä M. Left ventricular filling impairment in asymptomatic chronic alcoholics. The American Journal of Cardiology. 1990; 66: 1473–1477.
[36] Lazarević AM, Nakatani S, Nesković AN, Marinković J, Yasumura Y, Stojicić D, *et al*. Early changes in left ventricular function in chronic asymptomatic alcoholics: relation to the duration of heavy drinking. Journal of the American College of Cardiology. 2000; 35: 1599–1606.
[37] Kupari M, Koskinen P, Suokas A. Left ventricular size, mass and function in relation to the duration and quantity of heavy drinking in alcoholics. The American Journal of Cardiology. 1991; 67: 274–279.
[38] McKenna CJ, Codd MB, McCann HA, Sugrue DD. Alcohol consumption and idiopathic dilated cardiomyopathy: a case control study. American Heart Journal. 1998; 135: 833–837.
[39] Komajda M, Richard JL, Bouhour JB, Sacrez A, Bourdonnec C, Gerbaux A, *et al*. Dilated cardiomyopathy and the level of alcohol consumption: a planned multicentre case-control study. European Heart Journal. 1986; 7: 512–519.
[40] Gillet C, Juilliere Y, Pirollet P, Aubin HJ, Thouvenin A, Danchin N, *et al*. Alcohol consumption and biological markers for alcoholism in idiopathic dilated cardiomyopathy: a case-controlled study. Alcohol and Alcoholism. 1992; 27: 353–358.
[41] Whitman IR, Agarwal V, Nah G, Dukes JW, Vittinghoff E, Dewland TA, *et al*. Alcohol Abuse and Cardiac Disease. Journal of the American College of Cardiology. 2017; 69: 13–24.
[42] Klatsky AL, Chartier D, Udaltsova N, Gronningen S, Brar S,

Friedman GD, *et al*. Alcohol drinking and risk of hospitalization for heart failure with and without associated coronary artery disease. The American Journal of Cardiology. 2005; 96: 346–351.
[43] Park SK, Moon K, Ryoo JH, Oh CM, Choi JM, Kang JG, *et al*. The association between alcohol consumption and left ventricular diastolic function and geometry change in general Korean population. European Heart Journal-Cardiovascular Imaging. 2018; 19: 271–278.
[44] Larsson SC, Orsini N, Wolk A. Alcohol consumption and risk of heart failure: a dose-response meta-analysis of prospective studies. European Journal of Heart Failure. 2015; 17: 367–373.
[45] Sidorenkov O, Nilssen O, Nieboer E, Kleshchinov N, Grjibovski AM. Premature cardiovascular mortality and alcohol consumption before death in Arkhangelsk, Russia: an analysis of a consecutive series of forensic autopsies. International Journal of Epidemiology. 2011; 40: 1519–1529.
[46] Aguilar D, Skali H, Moyé LA, Lewis EF, Gaziano JM, Rutherford JD, *et al*. Alcohol consumption and prognosis in patients with left ventricular systolic dysfunction after a myocardial infarction. Journal of the American College of Cardiology. 2004; 43: 2015–2021.
[47] Li Z, Guo X, Bai Y, Sun G, Guan Y, Sun Y, *et al*. The Association Between Alcohol Consumption and Left Ventricular Ejection Fraction: An Observational Study on a General Population. Medicine. 2016; 95: e3763.
[48] Yousaf H, Rodeheffer RJ, Paterick TE, Ashary Z, Ahmad MN, Ammar KA. Association between alcohol consumption and systolic ventricular function: a population-based study. American Heart Journal. 2014; 167: 861–868.
[49] McDonald CD, Burch GE, Walsh JJ. Alcoholic cardiomyopathy managed with prolonged bed rest. Annals of Internal Medicine. 1971; 74: 681–691.
[50] Regan TJ, Levinson GE, Oldewurtel HA, Frank MJ, Weisse AB, Moschos CB. Ventricular function in noncardiacs with alcoholic fatty liver: role of ethanol in the production of cardiomyopathy. The Journal of Clinical Investigation. 1969; 48: 397–407.
[51] Urbano-Marquez A, Estruch R, Navarro-Lopez F, Grau JM, Mont L, Rubin E. The effects of alcoholism on skeletal and cardiac muscle. The New England Journal of Medicine. 1989; 320: 409–415.
[52] Fernandez-Sola J, Estruch R, Grau JM, Pare JC, Rubin E, Urbano-Marquez A. The relation of alcoholic myopathy to cardiomyopathy. Annals of Internal Medicine. 1994; 120: 529–536.
[53] Guarnieri T, Lakatta EG. Mechanism of myocardial contractile depression by clinical concentrations of ethanol. A study in ferret papillary muscles. The Journal of Clinical Investigation. 1990; 85: 1462–1467.
[54] Das AM, Harris DA. Regulation of the mitochondrial ATP synthase is defective in rat heart during alcohol-induced cardiomyopathy. Biochimica et Biophysica Acta - Molecular Basis of Disease. 1993; 1181: 295–299.
[55] Hajnóczky G, Buzas CJ, Pacher P, Hoek JB, Rubin E. Alcohol and mitochondria in cardiac apoptosis: mechanisms and visualization. Alcoholism, Clinical and Experimental Research. 2005; 29: 693–701.
[56] Steiner JL, Lang CH. Dysregulation of skeletal muscle protein metabolism by alcohol. American Journal of Physiology-Endocrinology and Metabolism. 2015; 308: E699–E712.
[57] Burch GE, Colcolough HL, Harb JM, Tsui CY. The effect of ingestion of ethyl alcohol, wine and beer on the myocardium of mice. The American Journal of Cardiology. 1971; 27: 522–528.
[58] Danziger RS, Sakai M, Capogrossi MC, Spurgeon HA, Hansford RG, Lakatta EG. Ethanol acutely and reversibly suppresses excitation-contraction coupling in cardiac myocytes. Circulation Research. 1991; 68: 1660–1668.
[59] Figueredo VM, Chang KC, Baker AJ, Camacho SA. Chronic alcohol-induced changes in cardiac contractility are not due to changes in the cytosolic Ca2+ transient. The American Journal of Physiology. 1998; 275: H122–H130.
[60] Steiner JL, Gordon BS, Lang CH. Moderate alcohol consumption does not impair overload-induced muscle hypertrophy and protein synthesis. Physiological Reports. 2015; 3: e12333.
[61] Cunningham CC, Preedy VR, Paice AG, Hesketh JE, Peters TJ, Patel VB, *et al*. Ethanol and protein metabolism. Alcoholism: Clinical and Experimental Research. 2001; 25: 262S–268S.
[62] Vary TC, Lynch CJ, Lang CH. Effects of chronic alcohol consumption on regulation of myocardial protein synthesis. American Journal of Physiology-Heart and Circulatory Physiology. 2001; 281: H1242–H1251.
[63] Henriksen JH, Møller S. Cardiac and systemic haemodynamic complications of liver cirrhosis. Scandinavian Cardiovascular Journal. 2009; 43: 218–225.
[64] Djoussé L, Gaziano JM. Alcohol consumption and risk of heart failure in the Physicians' Health Study I. Circulation. 2007; 115: 34–39.
[65] Walsh CR, Larson MG, Evans JC, Djousse L, Ellison RC, Vasan RS, *et al*. Alcohol consumption and risk for congestive heart failure in the Framingham Heart Study. Annals of Internal Medicine. 2002; 136: 181–191.
[66] Bryson CL, Mukamal KJ, Mittleman MA, Fried LP, Hirsch CH, Kitzman DW, *et al*. The association of alcohol consumption and incident heart failure: the Cardiovascular Health Study. Journal of the American College of Cardiology. 2006; 48: 305–311.
[67] Maisch B. Alcoholic cardiomyopathy: The result of dosage and individual predisposition. Herz. 2016; 41: 484–493.
[68] Farokhnia M, Rentsch CT, Chuong V, McGinn MA, Elvig SK, Douglass EA, *et al*. Spironolactone as a potential new pharmacotherapy for alcohol use disorder: convergent evidence from rodent and human studies. Molecular Psychiatry. 2022; 27: 4642–4652.
[69] Dew MA, DiMartini AF, Steel J, De Vito Dabbs A, Myaskovsky L, Unruh M, *et al*. Meta-analysis of risk for relapse to substance use after transplantation of the liver or other solid organs. Liver Transplantation. 2008; 14: 159–172.