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AHA SCIENTIFIC STATEMENT

Alcohol Use and Cardiovascular Disease: A Scientific Statement From the American Heart Association

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ABSTRACT: Alcohol is one of the most commonly consumed substances in the world, exhibiting complex relationships with multiple aspects of cardiovascular health and disease. The majority of the research on the topic is observational and therefore prone to bias and confounding. The available evidence suggests no risk to possible risk reduction when alcohol is consumed in low amounts (such as no more than 1 to 2 drinks a day) in regard to coronary artery disease, stroke, sudden death, and possibly heart failure. The risk associated with consuming 1 to 2 drinks a day on atrial fibrillation remains unknown. More randomized trials of low to moderate alcohol consumption are needed for more definitive conclusions. In stark contrast, heavier alcohol consumption such as binge drinking or consuming on average ≥3 drinks/d is consistently associated with worse outcomes in every cardiovascular disease entity studied. Considering the level of evidence, it remains unknown whether drinking is part of a healthy lifestyle and therefore clinicians should reinforce healthy lifestyle behaviors such as regularly engaging in physical activity, avoiding tobacco use, and maintaining healthy body weight.

Key Words: AHA Scientific Statements ■ alcohol consumption ■ atrial fibrillation ■ cardiovascular diseases ■ coronary artery disease ■ myocardial infarction ■ hypertension ■ risk reduction behavior

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The association between alcohol consumption and cardiovascular disease (CVD) is complex and controversial. Decades of research has led to inconsistent recommendations and mixed messages about alcohol use and cardiovascular conditions such as hypertension, myocardial infarction (MI), stroke, heart failure (HF), and cardiac arrhythmias. Although excessive alcohol use is a leading preventable cause of chronic medical conditions, low to moderate alcohol consumption (eg, no more than 1 to 2 drinks a day) has been hypothesized to confer a cardioprotective effect by reducing the risk of several forms of CVD. Data from recent studies using new methodologies (eg, individual participant-level data meta-analysis and mendelian randomization [MR]) have challenged the idea that any level of alcohol consumption has positive health effects. This document reviews, evaluates, and summarizes data on the association of alcohol use as it pertains to CVD to guide health care professionals, patients, and the lay public and highlights limitations of current studies and lingering uncertainties that warrant further scientific investigation.

TRENDS IN ALCOHOL USE

Alcohol consumption remains extremely common in the United States. Eighty-five percent of US adults report consuming alcohol at some point in their lives,[1] with per capita alcohol consumption estimated at 2.5 gallons/y.[2,3] A nationally representative study suggests that the prevalence of any alcohol use, including heavy alcohol use (≥5/4 [men/women] drinks on any given day or ≥15/8 [men/women] drinks/wk), increased during the COVID-19 pandemic (2018 versus 2020). The absolute increase for any alcohol use was 2.7% (relative increase, 4.0%) and for heavy alcohol use was 1.0% (relative increase, 20%), with these increases sustained in 2022.[4]

Initiation of alcohol use rapidly escalates through the teenage years, and prevalence rates peak in the early 20s.[1]

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Recent (past-month) drinking is reported by >50% of US adults >18 years of age and 44% of those >65 years of age,[1] indicating that alcohol use often persists across multiple decades of life. In addition, 61.4 million people ≥12 years of age (22% in this age group) reported binge drinking in the past month, defined as ≥5 alcoholic drinks on the same occasion for male individuals or 4 or more alcoholic drinks on the same occasion for female individuals. Although most alcohol users do not develop alcohol use disorders (≥1 in 10[1]), annual deaths potentially related to alcohol top 175 000 in the United States[5]; thus, risks from alcohol use extend far beyond alcohol use disorder.[2]

DEFINITIONS OF STANDARD DRINKS AND PATTERNS AND CURRENT GUIDELINES FOR DRINKING

In the United States, a standard drink includes about 0.6 oz (14 g) of alcohol. Globally, however, the definitions of a standard drink differ, ranging from 8 to 20 g. Alcohol by volume varies among beverage types; therefore, a standard drink equivalent differs by beverage type (Figure 1).

There is general agreement about harmful levels of drinking (Table 1), but mixed messages exist concerning health risks associated with lower levels of alcohol use such as 1 to 2 standard drinks/d (Figure 1).[6–12] The World Health Organization has stated that "no level of alcohol consumption is safe for health."[7] Canada's guidance on alcohol and health has somewhat followed suit, endorsing "drinking less is better" and delineating risks associated with different weekly levels of alcohol use.[6] Meanwhile, the 2020 to 2025 Dietary Guidelines for Americans (US Department of Agriculture) reflect ongoing uncertainty and equipoise derived from available evidence and refrain from definitive claims on health among low-level consumers.[12]

Recommendations and guidelines about alcohol use have been sex specific. The separate recommendations are based on the supposition that women develop higher

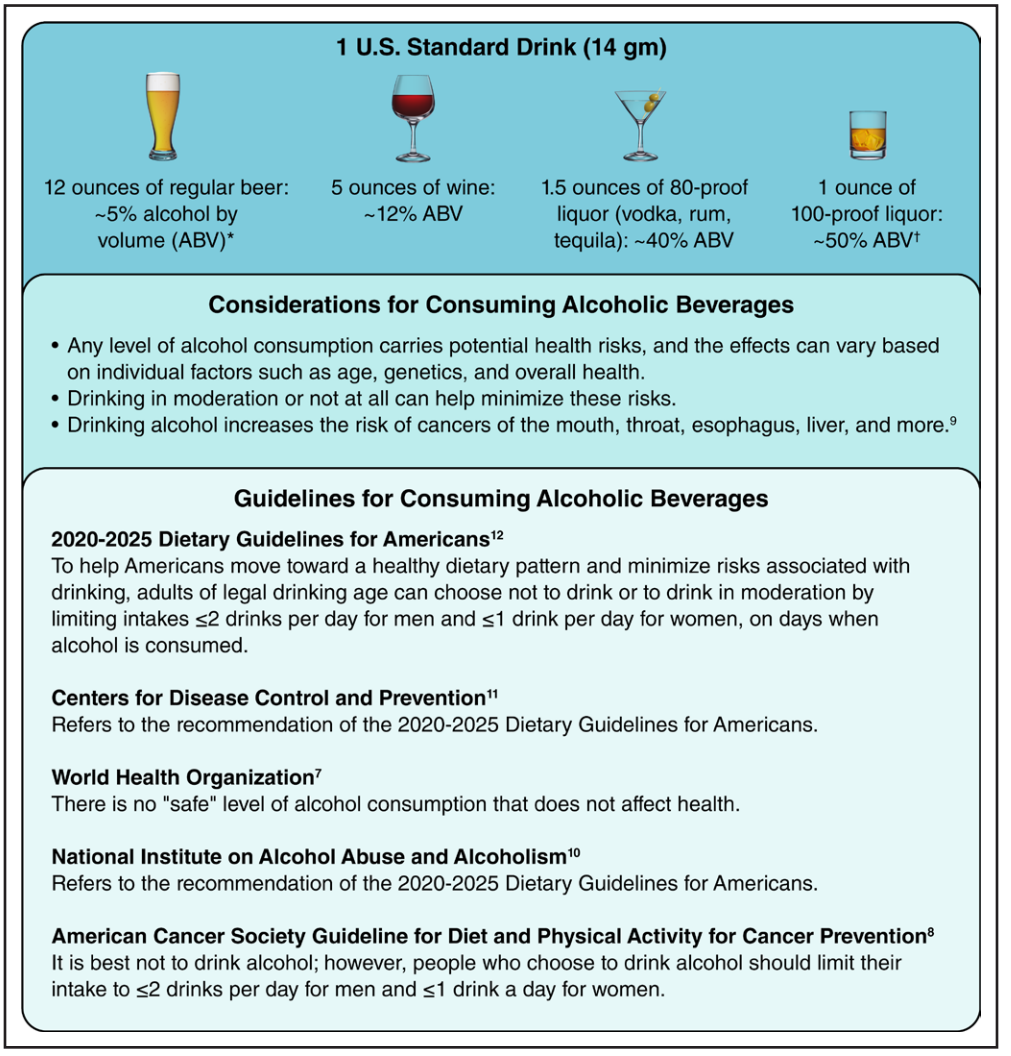

Figure 1. Considerations and guidelines for consuming alcoholic beverages.
*Some microbrews or India pale ales may have higher (7.5%) alcohol by volume (ABV). †Specialty crafted cocktails may also exceed 50% ABV.

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Table 1. Definition of a Standard Drink and Drinking Patterns

Each beverage represents 1 US standard drink (14 g)			
12 ounces of regular beer: ≈5% ABV*	5 ounces of wine: ≈12% ABV	1.5 ounces of 80-proof liquor (vodka, rum, tequila, etc): ≈40% ABV	1 ounce of 100-proof liquor: ≈50% ABV†
Current definitions of patterns of drinking			
Term	Definition		
Low-risk alcohol use	The NIAAA defines low-risk alcohol use for men up to 65 y of age as ≤4 drinks on any single day and ≤14 drinks/wk. For women and men ≥66 y of age, low-risk alcohol use is ≤3 drinks on any single day and ≤7 drinks/wk. Research shows that few people who drink within these limits have alcohol-related health conditions. In addition, some people should not drink at all (eg, those taking medications that interact with alcohol, patients with a medical condition that can be made worse by drinking, those under the legal drinking age, anyone planning to drive a vehicle or operate machinery, and women who are pregnant or trying to become pregnant).[10] ≤1 drink/d and ≤7 drinks/wk, ≤3 drinks at any one time, and no alcohol intake at least 2 d/wk (SAMSHA)[5a]		
Low to moderate drinking	Among studies, the terms low, moderate, and heavy have been used, and these terms have been defined differently. There is a greater lack of consensus in defining low and moderate levels. The exception is heavy drinking, which is usually defined as noted below. Low and moderate drinking has most often been defined as no more than 1 drink/d for women and no more than 2 drinks/d for men. Sometimes moderate drinking has been defined as ≥20 to 40 g/d, slightly more than 2 drinks/d.		
Hazardous drinking	Hazardous alcohol use is a pattern of drinking that increases the risk of future alcohol-related problems, although it may not currently be causing such problems.		
Harmful alcohol use	Harmful alcohol use is a pattern of drinking that is damaging to the physical or mental health of the user (WHO).[7]		
Binge drinking	Binge drinking is a pattern of alcohol use that on a single occasion brings blood alcohol concentration levels to ≥0.08 g/dL. This typically occurs after 4 drinks for women and ≈5 drinks for men, if consumed in ≈2 h (NIAAA).[10] Consuming ≥5 drinks for men and ≥4 drinks for women on an occasion in the last 30 d (CDC).[11] ≥5 drinks on the same occasion on at least 1 d in the past 30 d (SAMSHA and National Survey on Drug Use and Health).[1]		
Heavy drinking	Engaging in binge drinking on ≥5 d in the past 30 d (SAMHSA).[1]		
Alcohol dependence and alcohol use disorder	Alcohol dependence usually develops after years of regular drinking and periodic episodes of binge drinking. It involves a cluster of symptoms that may include a strong desire to drink, impaired control over alcohol use, persistent drinking even when it is causing harm, increased tolerance to the psychomotor effects of alcohol such that high doses can be consumed without apparent intoxication, and a withdrawal reaction when drinking is stopped or reduced. Alcohol dependence is the preferred term in *ICD-10*.		

ABV indicates alcohol by volume; CDC, Centers for Disease Control and Prevention; *ICD-10*, *International Classification of Diseases, 10th Revision*; NIAAA, National Institute on Alcohol Abuse and Alcoholism; SAMHSA, Substance Abuse and Mental Health Services Administration; and WHO, World Health Organization.

*Some microbrews or India pale ales may have higher (7.5%) ABV.

†Speciality crafted cocktails may also exceed 50% ABV.

blood alcohol levels compared with men when consuming the same amount of alcohol. This occurs because of differences between the sexes in the body water compartment (affects the distribution of alcohol) and first-pass gastric metabolism (higher in men) and higher progression of certain alcohol-induced diseases such as alcohol-related liver disease.[13]

ALCOHOL PHARMACOLOGY AND PHARMACOKINETICS

Alcohol (which, for human consumption, is the focus of this document and refers specifically to ethanol) presents a wide array of unique pharmacological qualities that can lead to unexpected interactions with numerous prescription and recreational substances.[14] On absorption mainly from the small intestine and into the bloodstream, alcohol readily diffuses into body organs with high blood flow and water content such as the myocardium and brain.[15,16] Its broad physiological distribution is further driven by its small molecular size. Alcohol is eliminated mainly (>90%) through metabolism in the liver by alcohol dehydrogenase; however, metabolism by the cytochrome P450 system and direct excretion through breath, sweat, and urine are also involved.[17]

Unlike other substances, the cognitive, behavioral, and subjective profiles (ie, how individuals experience the intoxication "high versus tired") of alcohol differ as blood alcohol concentration ascends to its peak compared with as it descends, being classified as both a stimulant (ascending limb) and sedative (descending limb).[18] The half-life of alcohol in the human body is generally between 4 and 5 hours; however, factors such as body composition, liver function, and genetics (variants in the alcohol and acetaldehyde enzymes) can affect alcohol metabolism and therefore the half-life of alcohol.[19] The complex profile of alcohol actions is also likely driven by its lack of a primary site of action, thus affecting various neurotransmitter systems (eg, GABA, glutamate, and dopamine) and neuromodulators (eg, adenosine, endocannabinoid), as well as neural and nonneural cell signaling proteins, second messenger systems, ion channels, and organelles.[20,21] This, in combination with its small size, broad distribution, and hepatic-focused elimination characteristic, places alcohol drinkers at risk for numerous pharmacokinetic and pharmacodynamic drug-drug interactions.[14,22] For example, alcohol can disrupt absorption and interfere with the metabolism of cardiovascular drugs and alter physiological reactions to these drugs through its actions on vascular tone, blood flow, cardiac output, and blood pressure (BP).[22] In sum, alcohol can be viewed as a whole-body drug,[21] with complex effects on multiple organs, overall physiology, and drug interactions. In addition, alcohol use, especially heavy or binge drinking, is associated with lower medication adherence

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across a range of medical conditions and includes adherence to medications for chronic diseases such as hypertension, diabetes, and HIV/AIDS.[23]

METHODOLOGIES AND DESIGNS IN ALCOHOL AND CVD RESEARCH

In most studies, alcohol use has been self-reported, which appears to be less accurate for heavier alcohol consumption.[24,25] Alcohol consumption, absorption, and metabolism vary across multiple domains, including beverage type, volume, frequency, duration, concomitant meals, and features inherent to the individual. These vagaries make the measurement of alcohol consumption and the characterization of dose-response relationships difficult, hindering universally applicable determinants of risks versus harms. In addition, the way in which alcohol consumption has been measured and categorized continues to vary among studies, making it challenging to compare data.

New methodologies, research designs, and statistical analysis techniques and more reliable methods of alcohol assessment are being used in studies to analyze the association between alcohol use and CVD. More often, objective measures of alcohol consumption are being used, including transdermal ethanol sensors,[26] home-based urine kits,[27] and biomarkers of alcohol consumption such as phosphatidylethanol.[28] Smartphones are also used to track real-time use of alcohol and to enhance research participation.[29,30]

Recent observational studies have implemented strategies to lessen lifetime selection bias, reverse causality, and confounding by including younger cohorts with baseline drinking assessed earlier in the life course, excluding outcomes such as deaths occurring during the first 2 years of follow-up,[31,32] or confining the study population to current drinkers.[33] With regard to study population and, in particular, the type of nondrinking reference group (eg, lifetime abstainers/never drinkers, former drinkers, occasional drinkers), Stockwell and colleagues[32] reported that the reference group used could be an important source of discrepancy among studies reporting no risk compared with risk with certain levels of alcohol consumption. These are examples of inherent limitations in observational studies that can be addressed by randomized study group assignment.

Another methodological strategy includes trial emulation, which uses observational data to mimic the design and analysis of a hypothetical randomized controlled trial (RCT). This approach allows researchers to leverage real-world data to evaluate the potential effects of an intervention without conducting a traditional RCT.[34] MR techniques have also been advocated as a method to mitigate against conventional confounding by using genetic variants associated with the exposure (eg, alcohol consumption) as instrumental variables (proxies); however, biases remain such as pleiotropy, linkage disequilibrium, weak instrument bias, and dynastic effects (ie, when the expression of the parental genotype in the parental phenotype directly affects the offspring genotype).[35,36] In addition, collider bias and insufficient statistical power may compromise the reliability of MR studies, particularly in the investigation of nonlinear associations. Although conventional studies certainly are not without problems, it remains to be seen whether MR and trial emulation studies can effectively remedy these shortcomings.

ALCOHOL USE AND BP

Within the initial hours of the consumption of 1 to 2 drinks of alcohol, BP levels are generally not affected. However, the consumption of ≥3 drinks has shown a biphasic effect on BP in which levels are decreased up to 12 hours after consumption and increased for 12 to 24 hours after consumption (systolic BP [SBP] increase, 3.7 mm Hg [95% CI, 2.3–5.1]).[37]

A meta-analysis of 36 RCTs evaluating different levels of alcohol use in short- to medium-term crossover and parallel-group designs (2865 participants with and without hypertension) showed that consumption of <2 drinks/d compared with no alcohol use was not associated with a significant change in BP.[38] However, drinking ≥3 drinks/d resulted in significantly higher (increasing with higher alcohol consumption) SBP and diastolic BP (DBP) compared with no or less drinking. In participants who consumed ≥6 drinks/d and reduced their intake on average by ≈50%, strong reductions in SBP and DBP were observed (mean SBP and DBP differences, −5.5 mm Hg [95% CI, −6.70 to −4.30] and −3.97 mm Hg [95% CI, −4.70 to −3.25], respectively). These observational findings demonstrate the benefit of abstinence or a significant reduction in alcohol intake on increased BP. Because the number of women in such trials ranged from only 5 to 48 and only 3 trials presented data specific to women, resulting in uncertain pooled-effect estimates, a need to include more women in such research clearly exists.[38]

A recent meta-analysis of cohort studies found a dose-response association between baseline levels of alcohol consumption and BP levels after 5.3 years of follow-up, with no threshold regardless of sex and geographic location.[39] People who drank on average 1 drink/d exhibited a 1.25–mm Hg higher SBP (95% CI, 0.5–2.0), whereas those who consumed a median of 3 drinks/d experienced a 4.9–mm Hg (95% CI, 3.7–6.1) higher SBP.[39] The increase in SBP was less for women compared with men.

In a systematic review and dose-response meta-analysis of nonexperimental cohort studies (>600 000 participants), there was a linear positive association between alcohol intake and incidence of new-onset hypertension above an alcohol intake of 1 drink/d (12 g/d).[40] In most studies, hypertension was defined as a SBP/DBP ≥140/90 mm Hg, and the follow-up period ranged from 2 to 22 years. Men demonstrated a linear positive association between alcohol consumption and the risk of hypertension compared with no alcohol intake, but the risk flattened at intakes of >3 to 4 drinks/d.[40] In women, however, an increase in the risk of hypertension was found at an alcohol intake exceeding 1 drink/d, with a steeper slope at increasing levels of alcohol consumption.[40]

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ALCOHOL USE AND CORONARY ARTERY DISEASE

Many studies have examined the relationship between alcohol use and the risk of different subtypes of ischemic heart disease, including MI. The effects of alcohol consumption on the risk and incidence of ischemic heart disease (hereafter referred to as coronary artery disease [CAD]) are complex and vary depending on the amount and pattern of alcohol intake and study design. In addition, how CAD was defined or coded (ie, health records such as using *International Classification of Diseases* codes versus self-report) and which CAD outcomes were evaluated, for example, MI alone or grouped (eg, *International Classification of Diseases, 10th Revision* codes that include angina, MI, complications after an MI), may demonstrate different risk relationships with alcohol use. For example, a meta-analysis using individual participant data from 83 observational studies found that compared with the reference group (those consuming between 0 and 25 g/wk, ≈1.78 drinks/wk), any consumption above this level (up to 21 drinks/wk) was associated with a lower risk of MI incidence.[33] However, in evaluations of CAD that excluded MI, there was a positive and linear association with ≥7 drinks/wk.[33] One of the earliest meta-analyses conducted to examine a dose-response relationship reported that alcohol consumption (≤1 drink/d for women, ≤2 drinks/d for men) compared with no alcohol consumption was associated with a lower (14%–25%) risk of incident CAD.[41] In a recent MR study using traditional (linear) and nonlinear MR techniques and using a single-nucleotide variant associated with alcohol use disorder (as a means to examine the association between a genetically predicted level of alcohol consumption), Biddinger et al[42] reported that levels of alcohol intake (3–6 drinks/wk) were associated with no to minimal increases in CAD risk but that risk began to rise at levels exceeding 7 drinks/wk.

In a recent burden-of-proof approach, Carr et al[43] examined the relationship between alcohol consumption and CAD risk from case-control, cohort, and MR studies. This type of analysis incorporates a 6-step framework for conducting a meta-analysis and allows the generation of conservative estimates and interpretations of risk-outcome relationships.[44] The main findings from this analysis indicated that data pooled from case-control studies (n=27) were associated with a 13% average reduction in CAD risk across average consumption levels of 0 to 3 drinks/d, whereas the risk reduction found from cohort studies (n=95) was lower (5%) across average consumption levels of 0 to 3.5 drinks/d. For case and cohort studies reporting only MI and analyzed together, the lower relative risk across 0 to 50 g/d was ≈0.40 to 0.80. This analysis revealed no statistically significant associations within the included MR studies.[43] These authors concluded that "data pooled from cohort and case-control studies showed weak association between average levels of alcohol consumption (≤50 g/day) and reduced CAD risk relative to no alcohol intake, while data pooled from MR studies showed no association between genetically predicted alcohol consumption and CAD risk."[43]

Drinking patterns, in particular binge and heavy episodic drinking, may modify the relationship between alcohol consumption and CAD risk. For example, moderate drinkers (<30 g/d) without a history of heavy drinking occasions had a pooled relative risk for CAD incidence of 0.64 (95% CI, 0.53–0.71) compared with lifetime abstainers. However, for moderate drinkers engaging in heavy episodic drinking occasions, the pooled relative risk was 1.12 (95% CI, 0.91–1.37), demonstrating that consuming <30 g/d was not associated with reduced CAD risk when accompanied by occasional heavy or binge drinking.[45]

To summarize, observational studies generally suggest lower risk of incident MI among individuals who consume alcohol consumption within the recommended US Dietary Guidelines for Americans limits compared with lifetime abstainers, but methodological challenges in the interpretation of these studies sharply limit the certainty of a causal relationship.[9] A recently initiated clinical trial of abstention compared with continued drinking among Spanish adults includes MI in its composite end point; if the trial is successful in its recruitment, retention, and adherence efforts, it will provide important evidence about the potential causality of this relationship.[46]

ALCOHOL USE AND STROKE

A meta-analysis of 27 prospective epidemiological studies found that compared with no alcohol use or occasional drinking, low to moderate drinking (≤2 drinks/d) was associated with a modestly (8%–10%) decreased risk of ischemic stroke, whereas high (2–4 drinks/d) or heavy (>4 drinks/d) drinking was associated with an increased risk of all stroke types.[47] Similar findings were reported in an earlier meta-analysis[48] and in later large-scale epidemiological studies.[42,49–51] Analyses confined to current drinkers have revealed a positive dose-response relationship between alcohol consumption and stroke risk,[33,50] with consistent findings regardless of stroke types, for nonfatal and fatal stroke and for men and women.[33] For example, in a combined analysis of 3 large-scale data sources in 19 high-income countries, a 100–g/wk (≈1 standard drink/d) increase in alcohol consumption among ≈600 000 current drinkers increased the risk of ischemic stroke by 13%, intracerebral hemorrhage by 17%, and subarachnoid hemorrhage by 9%.[33] The overall stroke risk increased by 15% in men and 9% in women per 100–g/wk increase in alcohol consumption.[33] Furthermore, MR studies in populations of European and Asian ancestries have reported a positive association between genetically predicted alcohol consumption and

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risk of total stroke,[42,52] ischemic stroke,[50,52,53] intracerebral hemorrhage,[50,52] and subarachnoid hemorrhage.[52]

Overall, evidence from both observational and MR studies indicates that heavy alcohol consumption (>4 drinks/d) is a risk factor for all stroke types. However, the evidence is currently insufficient to draw definitive conclusions about the relationship between moderate alcohol consumption (≤2 drinks/d) and ischemic stroke.

ALCOHOL USE AND ARRHYTHMIAS

Atrial Fibrillation

Meta-analyses have concluded that heavier alcohol consumption predicts a heightened risk of developing atrial fibrillation (AF).[54,55] Instrumental variable analyses have suggested causal relationships between alcohol exposure and AF in large populations.[56–58] No clear threshold effect has been identified, nor has any one type of alcoholic drink been differentially implicated; the relationship appears to be fairly linear.[55] Data remain conflicting as to whether 1 drink/d on average influences AF risk.[59,60] Alcohol abstainers appear to be at a lower risk of AF than those who continue to drink,[61] and a prospective, randomized trial of Australians consuming at least 10 drinks/wk demonstrated a substantial reduction in AF burden among those instructed to abstain.[62] Patients with paroxysmal AF fitted with continuous alcohol sensors demonstrated a heightened odds of AF occurring within hours of a drinking event,[63] and a per-protocol analysis of randomized N-of-1 trials of AF triggers found alcohol to be the only prespecified trigger associated with AF.[30]

Alcohol consumption has been associated with left atrial enlargement and fibrosis, both potential mediators of the alcohol-AF relationship.[64–66] More often, those with alcohol-related AF may also have vagally mediated episodes.[67] A randomized, double-blind, placebo-controlled study in humans demonstrated that alcohol acutely shortened pulmonary vein refractory periods.[68]

Supraventricular Tachycardia

There is no evidence that alcohol substantially influences the risk of supraventricular tachycardia.[58,67,69]

Premature Ventricular Contractions, Ventricular Tachycardia, and Ventricular Fibrillation

There is no consistent evidence that alcohol directly influences the risk of premature ventricular contractions,[70,71] ventricular tachycardia, or ventricular fibrillation.[72,73]

Sudden Death

Long-term consumption of ≈1 drink/d has been associated with the lowest risk of sudden death, whereas heavy alcohol consumption may be associated with a heightened risk of sudden death.[49,72,74,75] Because sudden deaths are now known to have multiple causes,[76] the underlying mechanisms may be related to alcoholic cardiomyopathies, MI, or other noncardiac causes of death.[77]

Bradycardias

Longitudinal cohort studies have not found a relationship between alcohol consumption and a higher risk of sinus node disease.[78,79] However, moderate alcohol consumption has been associated with a lower risk of developing sinus node disease.[78] Longitudinal cohort studies also suggest that alcohol either has no effect or has a protective effect on atrioventricular conduction disease.[69,78,80,81]

ALCOHOL USE, CARDIOMYOPATHY, AND HF

Long-term excessive alcohol use is associated with the development of a dilated left ventricle, normal or reduced left ventricular wall thickness and mass, and, in advanced stages, HF with reduced left ventricular ejection fraction.[16] Current cardiomyopathy schemas recognize excessive alcohol use as a nongenetic cause of dilated cardiomyopathy but also note that alcohol may act as an epigenetic trigger in the presence of underlying genetic variants.[82] In the United States, prevalence estimates are elusive and vary among reports. Data from the National Inpatient Sample show that among 352 million estimated hospitalizations of adults, 68 per 100 000 were associated with the diagnosis of alcoholic cardiomyopathy (ACM). This prevalence was higher among men than women.[83] The average age of hospitalization is similar for men and women (≈56 years); however, when stratified by race, both Hispanic men and women were hospitalized at a younger mean age (53 years).

The exact amount and duration of alcohol consumption associated with the development of ACM remain unknown. Data derived chiefly from case-control studies suggest that consuming ≈7 to 15 standard drinks/d over a 5- to 15-year period is associated with adverse changes in systolic or diastolic ventricular function.[84] Recently, however, in an observational cross-sectional study, Daka et al[85] reported that as few as 4 drinks/wk (in the past month) was associated with an increased odds of diastolic dysfunction.

The risk of ACM among those who consume alcohol is highly heterogeneous, suggesting important interactions with other environmental exposures or genetic factors. The presence of truncating variants in the gene encoding the giant sarcomeric protein titin (the most common cause of a dilated cardiomyopathy), may represent a genetic predisposition and increased

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vulnerability to ACM, particularly in individuals reporting a history of alcohol intake at ≈6 drinks/d over a 5-year period.[86] Women appear to be at risk of ACM when exposed to lower amounts and shorter durations of alcohol use compared with men.[87]

Controversy exists on the relationship between alcohol use and the risk of developing HF. Several meta-analyses and prospective cohort studies reported that <1 and 1 to 2 drinks/d were inversely associated with incident HF.[88–90] In contrast, an MR study reported both the absence of an association and an inferred increased risk of HF attributed to alcohol.[42] In the same MR study, when abstainers were excluded, exceeding 7 to 10 drinks/wk was associated with an increased risk of incident HF.[42] Similarly, studies of individual participant data from large-scale sources and 19 high-income countries revealed that usual alcohol consumption of <7 drinks/wk was not associated with increased HF risk, whereas levels of ≈21 drinks/wk were associated with an ≈50% increase in HF risk.[33]

Among studies and different populations, there appears to be a consensus that exceeding 21 drinks/wk is associated with an increased risk of HF. Recently, and similar to others, Wong and colleagues[91] reported that there was no protective or adverse association between alcohol consumption (≤5 drinks/wk) and the risk of developing HF. However, in patients with structural or functional cardiac abnormalities, alcohol use (≥5 drinks/wk) was associated with an increased risk of progression of asymptomatic left ventricular dysfunction and symptomatic HF (odds ratio, 5.0 [95% CI, 1.7–15.5]) over a mean follow-up of 5.4 years.[91]

ALCOHOL USE AND SPECIAL POPULATIONS

Some populations may be differentially affected by alcohol consumption related to their sex (women), age (older adults), predominant pattern of drinking (binge drinking), or presence of chronic diseases such as diabetes. For example, women appear to be at risk of ACM when exposed to lower amounts and shorter durations of alcohol use compared with men.[87] The reasons for these sex differences remain unresolved but as noted may be attributable to differences in the absorption, volume of distribution, and first-pass metabolism of alcohol between men and women, which give rise to differences in blood alcohol concentration after the consumption of similar amounts of alcohol. Populations of African and Asian ancestry may also experience different effects due to genetic polymorphisms in alcohol-metabolizing proteins that may increase the production of acetaldehyde, an aversive and mutagenic intermediate metabolic product.[92] Although studies are limited, follow-up of the National Health Interview Study suggested that the lowest mortality risk occurred at lower levels of consumption among Black individuals than White individuals.[93]

Older adults (50–80 years of age) have been included in many of the studies reviewed here; however, other factors such as health status and prescription drug use may affect the effects of alcohol on cardiovascular conditions. Older adults may be vulnerable to alcohol-medication interactions because of changes in metabolism and increased likelihood of taking multiple medications. This is important because among adults 50 to 80 years of age, 67% reported drinking occasionally in the past year.[94] Among those who drank, 27% (1 in 4) reported consuming ≥6 drinks on at least 1 occasion in the past year.[94]

Numerous reports indicate that young adults (18–30 years of age) engaging in binge drinking exhibit early signs of CVD such as endothelial dysfunction (a precursor to atherosclerosis),[95] coronary calcification,[96] and arterial stiffness.[97] Others have reported that binge drinking in young adults was associated with higher SBP,[98,99] and binge drinking in adolescence (12–18 years of age) was associated with a higher odds of high SP (SBP ≥130 mm Hg /DBP ≥80 mm Hg) in young adulthood (24–32 years of age).[100] In addition, several case-control studies have reported that binge drinking was associated with an increased risk of different stroke types in young adults.[46,101,102] In an analysis of INTERSTROKE data from 32 countries, binge drinking was associated with an odds ratio of 5.44 (95% CI, 1.81–16.4) of ischemic stroke among patients ≤45 years of age.[102] The potential for arrythmias among young adult binge drinkers was examined in the prospective MunichBREW II study.[103] Over a voluntary binge drinking episode (blood alcohol levels ≥120 mg/dL), young adults (N=202; mean age, 29.9 years) were monitored with a 48-hour electrocardiogram encompassing the time frame during and after a binge episode. The main findings were a significant increase in heart rate (maximum rate, 97±16 bpm 4 hours after consumption) and a significant increase in the percentage of atrial tachycardia beats. Only 1 participant developed AF, and 4 other participants experienced clinically relevant supraventricular and ventricular arrhythmias.[103] On the basis of these findings, there is a low occurrence of "holiday heart syndrome," a term used to refer to arrhythmias associated with binge-like drinking episodes in young adults.

There is a high rate of the co-occurrence of diabetes and CVD. In patients with diabetes, a few RCTs have examined the effects of alcohol intake on glycemic control and other cardiovascular parameters in patients. Cardiovascular Diabetes and Ethanol randomized patients with type 2 diabetes (N=22; age, 40–75 years) to a low-risk drinking group (1 drink/wk of either red or white wine) or mineral water with

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a Mediterranean diet/dinner for 2 years.[104] Findings indicated that this level of alcohol consumption was safe, and there were some small improvements in lipoproteins in the red wine group (a 2.0–mg/dL increase in high-density lipoprotein cholesterol) and a decrease in fasting glucose (−17.2 mg/dL) in the white wine group; no changes were found among groups in BP or measures of adiposity.[104] A crossover study reported that 4 weeks of red wine consumption (≈2 drinks/d) in men (n=19) and women (n=5) with type 2 diabetes significantly increased awake SBP (2.5±1.2 mm Hg) but had no effect on glycemic control, high-density lipoprotein cholesterol, fibrinogen, C-reactive protein, or plasma homocysteine.[105] In a 2-year trial among adults with type 2 diabetes, no significant progression was observed in carotid plaque volume among individuals randomized to water, white wine, or red wine (P=0.9 between groups).[106] In a post hoc analysis among individuals in the highest tertile of baseline carotid plaque volume, wine (red and white) significantly reduced plaque volume over 2 years compared with baseline, whereas no changes were found in the water group.[106]

Table 2. Cardiovascular Biomarkers and Changes With Moderate Alcohol Use*

Biomarker	Change reported in study
Lipid profiles	
Total cholesterol	No significant change[107,108]
High-density lipoprotein	↑ 3 mg/dL[107] ↑ 7%[108]
Low-density lipoprotein	↓ −3.09 mg/dL[107] No significant change[108]
Triglycerides	No significant change[107] ↓ −2.1%[108]
Lipoprotein(a)	↓ −0.04 g/L[107]
Apolipoprotein A1	↑ 0.04 g/L[107]
Hemostatic factors	
Plasminogen activator inhibitor 1	↑ 4.33 ng/mL
Fibrinogen	↓ −0.13 g/L[107]
E-selectin	No significant change[107]
Inflammation	
C-reactive protein	No significant change[107,108]
Interleukin 6	↓ −0.43 pg/mL[107]
Tumor necrosis factor-α	No significant change[107]
Adipokines	
Leptin	No significant change[107]
Adiponectin	↑ 0.06 mg/L[107] ↑ 4.3%[108]
Glycemic parameters	
Fasting glucose	No significant change[109]
Fasting insulin	No significant change[109]
Hemoglobin A_{1c}	No significant change[109]
Estimated insulin sensitivity	No significant change[109]

*Data were extracted from 1 meta-analysis[107] and another study using data from 3 prospective cohorts of US adults (Nurses' Health Study, Nurses' Health Study II, and Health Professionals Follow-up Study).[108] Huang et al[107] conducted a meta-analysis that included controlled short-term intervention studies (1–8 weeks) comparing moderate alcohol consumption (1–2 drinks/d) with no alcohol use. Values are median change values in designated biomarker.[107] Studies that included heavy drinking were excluded.[107] Li et al[108] compared individuals with moderate alcohol intake (average alcohol intake, 3.3 servings/wk) with nondrinkers or those who drank less than 1 serving/d. Values are percentage differences in log-transformed values.[108] Glycemic data are from Schrieks et al.[109]

Table 3. Patient Education About the Most Recent Evidence on Alcohol Use

BP	Alcohol consumption, even in moderation (1–2 drinks/d), can exacerbate high BP, and for some individuals, reducing or eliminating alcohol intake may be an important part of managing hypertension.
Stroke	Heavy (≥5/4 [men/women] drinks on any given day or ≥15/8 [men/women] drinks/wk) alcohol consumption increases the risk of all types of stroke. The impact of ≤2 drinks/d on stroke risk remains uncertain because of conflicting results. The association may partially be mediated by increased BP.[33]
CAD	Consuming alcohol at or below the 2020 to 2025 Dietary Guidelines for Americans (≤2 drinks/d for men and ≤1 drink/d for women) may provide some risk reduction for CAD, and heavy or binge drinking is associated with an elevated CAD risk.
Arrhythmias	Data remain conflicting as to whether 1 drink/d influences AF risk. However, alcohol abstainers appear to be at a lower risk of AF than those who continue to drink. A prospective trial of individuals with AF demonstrated a substantial reduction in AF burden with abstinence. Long-term consumption of ≈1 drink/d has been associated with the lowest risk of sudden death, whereas heavy (≥5/4 [men/women] drinks on any given day or ≥15/8 [men/women] drink/wk) alcohol consumption may be associated with a heightened risk of sudden death.
ACM	Long-term (5–15 y) excessive (≥7–15 drinks/d) drinking can be associated with the development of ACM. However environmental and genetic factors may modify the development of ACM. In patients who received guideline-concordant HF therapy and reduced their alcohol consumption to <80 g/wk (≈6 drinks), there was an improvement in ventricular function.[115,116] In addition, there was a better prognosis.[115,116]
HF	Low to moderate (1–2 drinks/d) amounts of alcohol consumption do not increase the risk for HF, but heavy or binge drinking may increase the risk. In patients with structural or functional cardiac abnormalities, alcohol use (≥5 drinks/wk) was associated with an increased risk of progression of asymptomatic left ventricular dysfunction and symptomatic HF.
DCM	The safe and harmful levels of alcohol consumption remain unknown for patients with DCM, especially women.*

ACM indicates alcoholic cardiomyopathy; AF, atrial fibrillation; BP, blood pressure; CAD, coronary artery disease; DCM, dilated cardiomyopathy; and HF, heart failure.

*In patients with DCM, Tayal et al[117] found that prior (baseline) moderate to excessive alcohol consumption of (≈1–5 drinks/d for women and ≈2–5 drinks/d for men) was not associated with adverse cardiovascular outcomes during the median follow-up of 3.9 years (hazard ratio, 1.29 [95% CI, 0.73–2.26] for the composite end point of cardiovascular mortality and HF and arrhythmic events). However, among patients who met this composite end point (n=78), only 15 had a history of moderate alcohol excess.[117]

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PHYSIOLOGICAL MECHANISMS AND OTHER MEDIATING FACTORS ASSOCIATED WITH ALCOHOL CONSUMPTION

The physiological mechanisms and other mediating factors associated with alcohol use and cardiovascular outcomes are complex and multifaceted and depend on the quantity and pattern of consumption. Some key mechanisms related to CVD may include effects on lipid profiles, influence on coagulation and thrombosis, and metabolic (ie, fasting glucose and insulin) alterations, as well as body weight. Other mediating factors might include socioeconomic status (SES), drinking with meals, and presence of other health factors.

Table 2 notes the directional (significant) changes in selected cardiometabolic parameters summarized from 1 meta-analysis[107] and another study using data from 3 cohorts of US adults.[108] Glycemic data were derived from a systematic review of moderate alcohol consumption (in most studies, defined as 1–2 drinks/d).[109] In addition, alcohol consumption (2 drinks/d) was found to reduce collagen-stimulated platelet aggregation[110] while heavier drinking (1.5 g/kg, ~ 7 drinks) was associated with acutely increased platelet aggregation.[111] Simultaneous intake of aspirin (325 mg) with alcohol (3–4 drinks) potentiates aspirin-induced bleeding time.[112]

Obesity is an important cardiovascular risk factor; several studies have examined the relationship between obesity and alcohol use, and findings are equivocal.[113] The most recent meta-analysis to date included 127 studies, and findings differed when cohort and cross-sectional studies were analyzed separately. Among the cohort studies analyzed, no association with alcohol intake was found for risk of overweight (body mass index ≥25–30 kg/m^2), obesity (body mass index ≥30 kg/m^2), and overweight/obesity (body mass index ≥25 kg/m^2). However, among the cross-sectional studies analyzed, alcohol intake was found to increase the odds for being overweight and overweight/obese, but no association was found for the odds of being obese. In this same study and in a dose-response analysis among cross-sectional studies, light alcohol use (<14 g/d) had no association with being overweight, obese, and overweight/obese. Similarly, alcohol use (14–28 g/d) had no association with being overweight or overweight/obese; however, there was 16% lower odds of being obese. Heavy (>28 g/d) alcohol use was positively associated with being overweight and overweight/obese, but no association

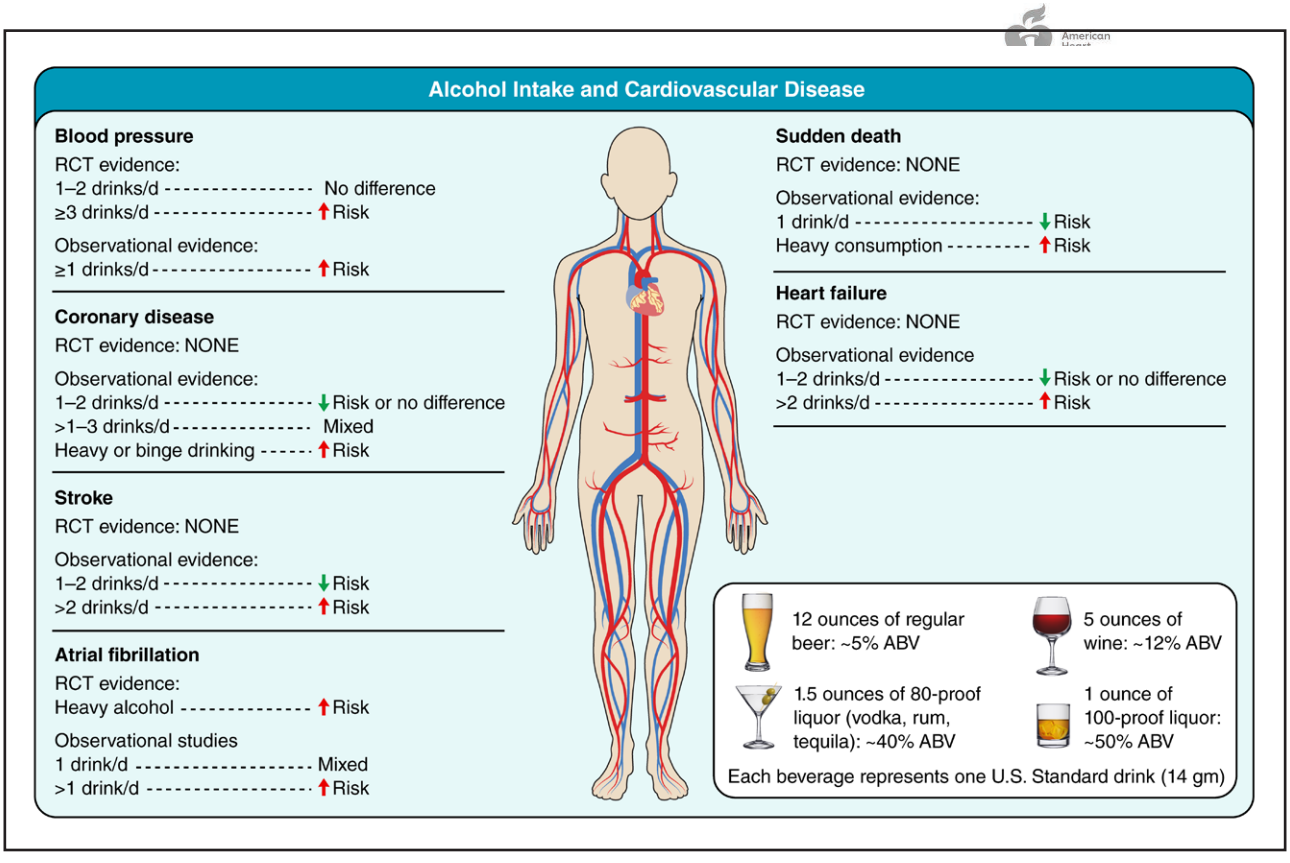

Figure 2. Alcohol intake and cardiovascular disease.
This figure summarizes data from studies reviewed herein and indicates the general direction of risk (with an up or down arrow) that has been observed with particular study designs. It is important to note that with most of these studies, especially those using observational study designs, fully accounting for unmeasured, unknown, residual confounding remains challenging. ABV indicates alcohol by volume; and RCT, randomized controlled trial.

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was found with obesity. As the authors of this study note, heterogeneity among studies was high, and examining the relationship between alcohol use and weight is complicated and mediated by many factors such as physical activity, dietary habits, genetics, and sex. Alcoholic beverages can be a large source of calories: 1 g of pure alcohol (ethanol) contains 7 calories. A standard glass of wine on average equals 158 calories, and some alcoholic beverages may contain ≥500 calories. Therefore, it seems that addressing problematic alcohol use should be an important component of obesity prevention and management strategies.

Findings from some studies suggest that SES and drinking during meals may be important mediating factors of alcohol use and cardiovascular risk association. For example, Ortolá et al[31] examined the association of different alcohol consumption levels with 12-year CVD mortality among older adults (≥60 years of age) with consideration of SES factors, beverage preference, and drinking with meals. Compared with occasional drinkers (≤2.8 g/d), low (≥20 g/wk–20 g/d for men and ≥20 g/wk–10 g/d for women) and moderate (≥20–40 g/d for men and >10–20 g/d for women) drinking was not associated with CVD mortality risk, whereas high-risk alcohol consumption (≥40 g/d for men and ≥20 g/d for women) was associated with higher CVD mortality (hazard ratio, 1.21 [95% CI, 1.04–1.41]). CVD mortality was higher in those with lower SES status and the presence of health-related factors; however, wine preference and drinking during meals modified and negated the association of mean alcohol intake with CVD mortality in all drinkers with lower SES and the presence of health-related factors but especially in high-risk drinkers.

Last, the way in which alcohol affects vascular, myocardial, and brain function may include many changes in cellular and molecular mechanisms. However, reviewing these mechanisms is beyond the scope of this scientific statement, but it would be interesting to explore them in future work. Several reviews have been published that include details related to cellular and molecular mechanisms potentially involved in mediating the effects of alcohol.[16,114]

PATIENT EDUCATION: WHAT DO WE TELL THE PATIENT?

Providing specific alcohol use recommendations for individuals with and without CVD conditions remains challenging because of the lack of data from well-conducted RCTs. If patients are currently consuming alcoholic beverages, Table 3 summarizes evidence that could be shared with patients.[115,116] In addition, it could be communicated to patients that consuming alcoholic beverages with meals will slow down the absorption of alcohol, as well as alternating alcoholic beverages with nonalcoholic beverages.

Some health care systems have begun to document alcohol use as a seventh vital sign. Screening for alcohol use and discussing what constitutes hazardous and harmful use (Table 1) are relevant to all patients. It is important to note that among patients surveyed in cardiology services (N=498), 66.3% were exposed to reports that indicated moderate drinking can be good for the heart.[118] It is important to deliver the right message because that study also reported that exposure to reports of healthy-heart effects or mixed messages about the cardiovascular effects of alcohol were associated with increased odds of hazardous alcohol use (odds ratio, 1.67 [95% CI, 1.02–2.74]).[118] Clinicians should reinforce healthy lifestyle behaviors such as regularly engaging in physical activity, avoiding tobacco use, and maintaining healthy body weight to achieve cardiovascular health and to prevent disease. Other considerations for consuming alcoholic beverages also can be shared with patients (Figure 1).

CONCLUSION, KNOWLEDGE GAPS, AND FUTURE DIRECTIONS

The overwhelming evidence demonstrates that heavy (generally >2 drinks/d) and binge alcohol consumption is harmful to cardiovascular health. Abstinence and reducing alcohol intake may lower the risk for certain cardiovascular conditions such as hypertension. Uncertainty remains about the true cardiovascular risk of drinking lightly such as 1 to 2 drinks/d (Figure 2 provides a graphical summary of data). Multiple unknowns persist, providing opportunities for important research into the effects of this commonly

Table 4. Research Gaps

More research is needed on women (across all age groups) because women have been excluded or underenrolled in alcohol use and CVD studies.
Differential effects by different types of alcoholic drinks, especially red wines, and potential interaction effects of consuming alcoholic beverages with meals require further investigation.
Interactions between alcohol use and cardiovascular medications should be examined.
The influence of changes in alcohol consumption in middle-aged and older adults on future CVD risk needs to be assessed.
Research is needed that incorporates biomarkers of alcohol use to corroborate self-report. For example, Domínquez-López et al reported correlations between urinary tartaric acid (a biomarker of wine consumption) and lower risk for CVD events (cardiovascular death, MI, stroke, or HF).[119]
Studies are needed of the influence of long-term consistent alcohol intake (considering changes in alcohol drinking over time) and changes in confounders.
Research is needed on the mechanisms by which alcohol consumption affects the risk of specific individual CVDs. For example, Wood et al[33] found that SBP only partially mediated the association between alcohol consumption and stroke risk.
The effects of alcohol consumption in patients with diabetes and specific CVDs need to be studied.
Randomized controlled trials are needed to determine the true health effects of light or moderate drinking (1–2 drinks per day or less).

CVD indicates cardiovascular disease; HF, heart failure; and MI, myocardial infarction.

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consumed substance. Although this document cannot review every knowledge gap, several are worth highlighting. More research into the following areas is needed: the heterogeneous effects within individuals, potentially driven by differences in SES status, other demographics, interactions with other environmental or dietary exposures, concomitant comorbidities, balancing overall health effects (eg, differential effects related to CVD versus cancer), and genetics, as well as effective strategies for behavior change to influence the healthiest patterns of alcohol consumption, for which evidence in populations without alcohol use disorders is especially lacking. In addition, to help establish biological plausibility, more research is needed on the cellular and molecular mechanisms that may underpin different patterns and amounts of alcohol consumption (more research gaps are noted in Table 4). Last, research using what has been established as the gold standard to provide the highest level of evidence and RCT designs is needed to better elucidate the true health effects of light to moderate alcohol consumption (1–2 drinks/d).

ARTICLE INFORMATION

The American Heart Association makes every effort to avoid any actual or potential conflicts of interest that may arise as a result of an outside relationship or a personal, professional, or business interest of a member of the writing panel. Specifically, all members of the writing group are required to complete and submit a Disclosure Questionnaire showing all such relationships that might be perceived as real or potential conflicts of interest.

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This table represents the relationships of writing group members that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all members of the writing group are required to complete and submit. A relationship is considered to be "significant" if (a) the person receives $5000 or more during any 12-month period, or 5% or more of the person's gross income; or (b) the person owns 5% or more of the voting stock or share of the entity, or owns $5000 or more of the fair market value of the entity. A relationship is considered to be "modest" if it is less than "significant" under the preceding definition.

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Peter M. Kistler	Baker Heart & Diabetes Institute, Alfred Hospital, University of Melbourne (Australia)	None	None	None	None	None	None	None
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*Significant.

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