Alcohol consumption and risk of ventricular arrhythmias and sudden cardiac death: An observational study of 408,712 individuals

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BACKGROUND Although previous studies have demonstrated a U-shaped relationship between alcohol and sudden cardiac death (SCD), there is a paucity of evidence on the role of alcohol specifically on incident ventricular arrhythmias (VAs).

OBJECTIVE The purpose of this study was to characterize associations of total and beverage-specific alcohol consumption with incident VA and SCD using data from the UK Biobank.

METHODS Alcohol consumption reported at baseline was calculated as UK standard drinks (8 g of alcohol) per week. Outcomes were assessed through hospitalization and death records. Alcohol consumption was modeled as restricted cubic splines in multivariate Cox regression models and corrected for regression dilution bias.

RESULTS We studied 408,712 middle-aged individuals (52.1% female) over a median follow-up time of 11.5 years. A total of 1733 incident VA events and 2044 SCDs occurred. For incident VA, no clear association was seen with total alcohol consumption. Although consumption of greater amounts of spirits was associated with increased VA risk, no other significant beverage-specific associations were observed. For SCD, a U-shaped association was seen for total alcohol consumption, such that consumption of <26 drinks per week was associated with lowest risk. Consumption of greater amounts of beer, cider, and spirits was potentially associated with increasing SCD risk, whereas increasing red and white wine intake was associated with reduced risk.

CONCLUSION In this predominantly white cohort, no association of total alcohol consumption was observed with VA, whereas a U-shaped association was present for SCD. Additional studies utilizing accurately defined VA and SCD events are required to provide further insights into these contrasting findings.

KEYWORDS Alcohol; Beer; Cardiac arrest; Spirits; Sudden death; Ventricular arrhythmia; Wine

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Introduction
The relationship between alcohol consumption and cardiovascular diseases continues to receive much attention due to the potential public health benefits of such information. Although high alcohol consumption is universally considered harmful, the implications of light or moderate alcohol consumption, and any impact of specific beverages, remain areas of ongoing investigation. Previous studies that have focused on sudden cardiac death (SCD) as an outcome have demonstrated no increase in risk,\(^1\,^2\) or even a decrease\(^3\) at moderate levels of consumption, implying that a U-shaped association with alcohol consumption may exist. At the other end of the consumption spectrum, binge and heavy drinking have been associated with an increase in SCD risk.\(^1,^2,^7^8\)

However, limited biological specificity may exist for diagnoses of SCD to represent fatal arrhythmic events.\(^9\) Few studies have examined the relationship between alcohol and specifically ventricular arrhythmias (VAs), and the evidence available in select patient populations has demonstrated inconsistent findings.\(^10\,^12\) There also are few data on the role of alcoholic beverage type on either VA or SCD risk, and whether thresholds of risk differ by sex.\(^4,^13\)

To provide further insights into the risks of alcohol intake and VA in the general population as well as contemporary evidence for SCD, we characterized associations of total and beverage-specific alcohol consumption with incident VA and SCD using prospective cohort data from the UK Biobank.

Methods
Study population
We retrospectively analyzed data from the UK Biobank, a prospective cohort study of approximately 500,000 community-dwelling individuals aged 40–69 years across the United Kingdom recruited from 1 of 22 assessment centers between 2006 and 2010. The UK Biobank has ethical approval from the North-West Multi-centre Research Ethics Committee, and the research was performed in accordance with the Declaration of Helsinki. At enrollment, participants completed a questionnaire that collected information on sociodemographic and lifestyle factors. Anthropometric measurements and verbal interview for comorbidities were recorded. Death and hospital records were available through linkage with national databases.

For our primary analyses, we focused on incident cases of VA, including ventricular tachycardia (VT) and ventricular fibrillation (VF) events, but excluded diagnoses of premature ventricular contractions and SCD, herein including cardiac arrest with resuscitation (Table 1). We excluded participants who (1) had previous diagnosis of a VA or cardiac arrest; (2) were ex-drinkers of alcohol (so as to reduce the effect of reverse causality as these participants may have abstained from alcohol due to poor health);\(^14\); and (3) were missing specific alcohol consumption data.

Assessment of alcohol consumption
Participants who indicated current consumption of alcohol on the enrollment questionnaire were asked to indicate how much of each alcoholic beverage (beer/cider, red wine, white wine, and spirits) they consumed in an average week or month (see Supplemental Methods). To standardize estimates, we reported the average standard drinks consumed per week of each beverage type. In this analysis, 1 standard drink was defined as 8 g of alcohol, the size of a standard drink in the United Kingdom (UK). Notably, definitions of a standard drink vary by country (eg, a standard drink in the United States contains 14 g of alcohol).\(^15\)

Assessment of covariates
The following covariates were self-reported at baseline: age, sex, race, education, assessment center attended, Townsend deprivation index, and smoking status. The Townsend deprivation index was based on each participant’s postal code and is a measure of material deprivation calculated using census data.\(^16\) Total metabolic equivalent of task (MET) minutes per week was calculated from a modified International Physical Activity Questionnaire.\(^17\) Body mass index (BMI) was measured according to a standard protocol. Comorbidities at baseline were identified from (1) self-report on the baseline questionnaire or standardized verbal interview at enrollment; (2) hospital inpatient diagnosis codes; or (3) hospital operation/procedure codes (Supplemental Table 1).

Assessment of outcomes
We studied the first occurrence of VA and SCD events separately, and time to first event was analyzed without right censoring at the occurrence of the other event. Incident cases of VA and SCD were identified by the first occurrence of a relevant (1) hospital inpatient diagnosis; (2) hospital procedure; or (3) arrhythmia-related death, as previously described (Table 1 and Supplemental Methods).\(^18\)

Statistical analysis
Baseline characteristics were presented by categories of total alcohol consumption and summarized as median (interquartile range) if continuous, and as frequency and proportion if categorical. Comparisons were assessed using the Kruskal-Wallis and \(\chi^2\) tests as appropriate.

Cox proportional hazard models were used to assess the association between alcohol consumption and the first occurrence of the outcomes. Individuals were considered at risk from the date of enrollment into the UK Biobank until the (1) date of outcome; (2) date of death not due to outcome; (3) date lost to follow-up; or (4) end of available follow-up, whichever came first.

For alcohol consumption, we examined the total consumption as well as consumption of each beverage type individually. To correct for regression dilution bias due to measurement error and within-person variability in consumption over time, we performed regression calibration utilizing resurvey measurements available from 12.1% of the
participants to estimate long-term average alcohol consumption (Supplemental Methods).19

Alcohol consumption was included in the model as restricted cubic splines with 4 knots placed at the 5th, 35th, 65th, and 95th percentiles, defined a priori. The model was stratified by sex and adjusted for age, race (white, other, unknown), education (college or university degree, vocational qualifications, optional national exams at ages 17–18 years, national exams at age 16 years, none of the above, unknown), assessment center attended at baseline (1 of 22 centers), Townsend deprivation index (in quintiles), BMI, total MET-minutes/week, smoking status (never, past, current, unknown), and the following comorbidities as time-updated covariates: hypertension, coronary artery disease, heart failure, valvular disease, diabetes mellitus, hyperlipidemia, peripheral artery disease, and chronic kidney disease. Hospital inpatient diagnoses/operations were used to update the information. Missing values for BMI and total MET-minutes/week were handled using the indicator variable method. Hazard ratio (HR) and 95% confidence interval (CI) were estimated by general contrasts of regression coefficients, using the median values of covariates and 0 drinks per week as the referent value.

A number of sensitivity analyses were conducted: (1) excluding participants with events that occurred in the first 1 and 2 years of follow-up so as to mitigate any potential effect of reverse causality; (2) excluding participants with coronary artery disease, valvular disease, and/or heart failure at baseline so as to mitigate any potential bias due to survivorship; (3) performing complete case analyses instead of the indicator variable method; (4) performing competing risks analyses under a Fine-Gray subdistribution hazard model; and (5) mutually adjusting for the consumption of other beverages in models for individual beverage consumption.

We also performed a separate analysis including ex-drinkers, who were excluded from the main study cohort. As exploratory analyses, we assessed for a differential effect of alcohol consumption by sex. An interaction term between alcohol consumption and sex was included in the model, and likelihood ratio tests were performed with the nested model to identify significant interactions.

The proportional hazards assumption was tested using Schoenfeld residuals and interaction with time, and no major violations were present after stratifying models by sex. A 2-tailed $P$ value was set at .05 for statistical significance. Analyses were performed using R (version 4.0.2, R Core Team).

### Results

The study population for the primary analyses consisted of 408,712 participants after stepwise exclusion of 975 UK Biobank participants who were previously diagnosed with a VA or had experienced a cardiac arrest, 18,030 who were ex-drinkers, and 74,772 who were current consumers but did not have specific alcohol consumption information. A total of 1733 incident VA events and 2044 SCD events occurred over 4,623,231 person-years of follow up, with median follow-up duration of 11.5 (10.8–12.3) years and median time-to-event of 7.2 (4.2–9.5) years. Baseline characteristics of the study population are detailed in Table 2. Median total alcohol consumption in the study population was 8.0 (3.5–15.5) drinks per week, and 22,324 (5.5%) reported having never consumed alcohol. Participants who consumed greater amounts of alcohol were younger, more likely male, white, and largely more comorbid. Ex-drinkers, who were not included in the primary analyses, were more likely older, female,
Table 2  Baseline characteristics of the study population

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Overall</th>
<th>&lt;7</th>
<th>7–14</th>
<th>15–28</th>
<th>&gt;28</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of participants</td>
<td>408,712 (100)</td>
<td>180,246 (44.1)</td>
<td>112,174 (27.4)</td>
<td>83,998 (20.6)</td>
<td>32,294 (7.9)</td>
</tr>
<tr>
<td>Female</td>
<td>212,887 (52.1)</td>
<td>123,298 (68.4)</td>
<td>58,395 (52.1)</td>
<td>26,833 (31.9)</td>
<td>4361 (13.5)</td>
</tr>
<tr>
<td>Age (y)</td>
<td>58.3 (50.6, 63.7)</td>
<td>58.6 (50.6, 63.9)</td>
<td>58.2 (50.6, 63.6)</td>
<td>58.2 (50.8, 63.5)</td>
<td>57.8 (50.6, 63.2)</td>
</tr>
<tr>
<td>White race</td>
<td>385,918 (94.4)</td>
<td>163,194 (90.5)</td>
<td>109,014 (97.2)</td>
<td>82,112 (97.8)</td>
<td>31,598 (97.8)</td>
</tr>
<tr>
<td>Body mass index (kg/m^2)†</td>
<td>26.6 (24.1, 29.6)</td>
<td>26.4 (23.8, 29.8)</td>
<td>26.3 (23.9, 29.1)</td>
<td>27.0 (24.6, 29.7)</td>
<td>27.7 (25.3, 30.5)</td>
</tr>
<tr>
<td>Current smokers</td>
<td>41,325 (10.1)</td>
<td>12,992 (7.2)</td>
<td>9715 (8.7)</td>
<td>11,184 (13.3)</td>
<td>7434 (23.0)</td>
</tr>
<tr>
<td>Physical activity (MET-minutes/week)†</td>
<td>1790 (819, 3550)</td>
<td>1710 (775, 3470)</td>
<td>1800 (858, 3490)</td>
<td>1870 (872, 3610)</td>
<td>1930 (834, 4160)</td>
</tr>
<tr>
<td>Comorbidities</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td>113,773 (27.8)</td>
<td>48,217 (26.8)</td>
<td>28,375 (25.3)</td>
<td>25,090 (29.9)</td>
<td>12,091 (37.4)</td>
</tr>
<tr>
<td>Coronary artery disease</td>
<td>21,111 (5.2)</td>
<td>8931 (5.0)</td>
<td>5303 (4.7)</td>
<td>4757 (5.7)</td>
<td>2120 (6.6)</td>
</tr>
<tr>
<td>Heart failure</td>
<td>2059 (0.5)</td>
<td>882 (0.5)</td>
<td>509 (0.5)</td>
<td>466 (0.6)</td>
<td>202 (0.6)</td>
</tr>
<tr>
<td>Valvular disease</td>
<td>4543 (1.1)</td>
<td>2112 (1.2)</td>
<td>1226 (1.1)</td>
<td>883 (1.1)</td>
<td>322 (1.0)</td>
</tr>
<tr>
<td>Peripheral artery disease</td>
<td>2400 (0.6)</td>
<td>876 (0.5)</td>
<td>584 (0.5)</td>
<td>587 (0.7)</td>
<td>353 (1.1)</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>20,079 (4.9)</td>
<td>10,357 (5.7)</td>
<td>4303 (3.8)</td>
<td>3688 (4.4)</td>
<td>1731 (5.4)</td>
</tr>
<tr>
<td>Hyperlipidemia</td>
<td>55,892 (13.7)</td>
<td>23,675 (13.1)</td>
<td>13,964 (12.4)</td>
<td>12,630 (15.0)</td>
<td>5623 (17.4)</td>
</tr>
<tr>
<td>Chronic kidney disease</td>
<td>1283 (0.3)</td>
<td>747 (0.4)</td>
<td>271 (0.2)</td>
<td>197 (0.2)</td>
<td>68 (0.2)</td>
</tr>
<tr>
<td>Liver disease</td>
<td>3454 (0.8)</td>
<td>1550 (0.9)</td>
<td>876 (0.8)</td>
<td>666 (0.8)</td>
<td>362 (1.1)</td>
</tr>
<tr>
<td>Alcohol consumption (UK standard drinks/week)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total alcohol</td>
<td>8.0 (3.5, 15.5)</td>
<td>3.0 (0.7, 4.9)</td>
<td>9.7 (8.2, 11.7)</td>
<td>18.7 (16.0, 22.3)</td>
<td>36.4 (31.2, 45.6)</td>
</tr>
<tr>
<td>Beer/cider</td>
<td>0 (0, 4.7)</td>
<td>0 (0, 0.7)</td>
<td>1.6 (0.4, 7.0)</td>
<td>4.7 (0.1, 12.5)</td>
<td>15.6 (3.1, 31.2)</td>
</tr>
<tr>
<td>Red wine</td>
<td>1.2 (0, 5.8)</td>
<td>0 (0, 2.2)</td>
<td>3.5 (0, 7.0)</td>
<td>7.0 (0, 11.7)</td>
<td>7.0 (0, 21.0)</td>
</tr>
<tr>
<td>White wine</td>
<td>0.2 (0, 2.7)</td>
<td>0 (0, 0.9)</td>
<td>0.9 (0, 3.6)</td>
<td>0.9 (0, 5.4)</td>
<td>0 (0, 5.4)</td>
</tr>
<tr>
<td>Spirits</td>
<td>0 (0, 0.7)</td>
<td>0 (0, 0.0)</td>
<td>0 (0, 1.5)</td>
<td>0 (0, 2.3)</td>
<td>0 (0, 4.5)</td>
</tr>
</tbody>
</table>

Categorical variables are reported as no. (%) and continuous variables are reported as median (Q1, Q3). All comparisons between quartiles were statistically significant.

MET-minutes = metabolic equivalent of task minutes; UK = United Kingdom.

*Missing 2113 (0.5).
†Missing 76,820 (18.8).
nonwhite, and comorbid, and were more likely to have a history of a VA. The distributions of total alcohol and individual beverage consumption in the study population are shown in Supplemental Figure 1. Participants whose alcohol consumption was predominantly beer/cider or spirits were generally more comorbid than participants whose alcohol consumption was predominantly red or white wine (Supplemental Table 2).

**Figure 1** Association of alcohol consumption and incident ventricular arrhythmias. One standard drink is defined as 8 g of alcohol, the size of a standard drink in the United Kingdom. *Shaded area* represents 95% confidence interval.

**Figure 2** Association of alcohol consumption and sudden cardiac death. One standard drink is defined as 8 g of alcohol, the size of a standard drink in the United Kingdom. *Shaded area* represents 95% confidence interval.
VA
For aggregate total alcohol consumption and risk of VA, no statistically significant association was seen (Figure 1). Increasing spirit intake was linearly associated with increased risk of VA, with statistically significant increased risk in those consuming >14 drinks per week. No other beverage-specific associations were observed. Sensitivity analyses did not materially change the results (Supplemental Table 3), and associations were similar when ex-drinkers were included in the analysis (Supplemental Figure 2). A possible interaction between spirit consumption and sex was observed, with greater risk seen at lower levels in women ($P = .01$) (Supplemental Figure 3), although no significant sex interactions were observed for total alcohol or other individual beverages.

SCD
For aggregate total alcohol consumption and risk of SCD, a U-shaped association was seen, with lower risk in those consuming <26 drinks per week and the nadir of risk seen at 7 drinks per week (Figure 2). Associations of specific beverages with SCD seemed to differ. For beer/cider consumption, there was a positive curvilinear association, with statistically significant increased risk in those consuming >26 drinks per week. For spirits, there was a possible positive linear association, with the lowest risk seen in those with no consumption. For red and white wine consumption, there were negative linear associations, with highest risk seen in those with no consumption. Sensitivity analyses did not materially change the results (Supplemental Table 4), and associations were similar when ex-drinkers were included in the analysis (Supplemental Figure 4). No significant sex interactions were observed for total alcohol or individual beverage consumption and the risk of SCD.

Discussion
In this study of 408,712 participants of the UK Biobank, we characterized associations of total and beverage-specific alcohol consumption with VA and SCD events. The principal findings of our study are as follows:

1. No clear association was seen with total alcohol, beer, cider, red wine, or white wine consumption and incident VA events; however, consumption of greater amounts of spirits was associated with increased VA risk.
2. A U-shaped association was seen for total alcohol consumption and SCD, such that consumption of <26 UK standard drinks per week was associated with the lowest risk of SCD. This is equivalent to approximately 15 US standard drinks or 208 g of alcohol per week.
3. Beverage-specific analyses for SCD demonstrated contrasting associations. Consumption of greater amounts of beer, cider, and spirits was potentially associated with increased risk, whereas increasing red and white wine intake was associated with lowered risk.
4. No substantial evidence of interaction between alcohol consumption and sex for VA or SCD was found.

Alcohol and VAs
There is a paucity of evidence investigating the relationship between long-term alcohol consumption and VA, and the studies that have been conducted are in select patient populations and have demonstrated inconsistent findings. In a case-control study of ST-segment elevation myocardial infarction patients, consumption of >96 g of alcohol per week was associated with an increased risk of VF, with no protective association seen at levels lower than this. In contrast, in a historic study of myocardial infarction patients not receiving thrombolytic therapy, frequency of alcohol use was not associated with risk of VA. Finally, in a cohort of men who underwent 24-hour ambulatory electrocardiogram, consumption of >250 g of alcohol per week was associated with a greater risk of frequent ventricular ectopics or VT in those with cardiovascular disease. The limited generalizability of these data indicate a need to further characterize the role of alcohol on VA risk in the general population.

In the present study, we sought to clarify this by leveraging the large UK Biobank cohort. With 1733 incident VA events, this represents the largest analysis to our knowledge. We observed no clear association of long-term total alcohol intake with VA across the spectrum of consumption. To our knowledge, we are the first to also observe beverage-specific associations. Our analyses suggest that a linear association of harm may be present with greater consumption of spirits; however, no clear association was present with beer, cider, red wine, or white wine consumption. Although effect modification was potentially present for sex and spirit consumption with regard to VA risk, this may be due to inclusion of few women with heavy spirit consumption. This finding was also significant only without adjustment for multiplicity and therefore must be interpreted with caution.

Alcohol and SCD
The U-shaped association of alcohol consumption and SCD observed in this study is consistent with previous reports. We also observed potentially harmful associations of beer, cider, and spirits on SCD with increasing levels of alcohol consumption; in contrast, red and white wine was associated with reduced risk across the spectrum of intake. These beverage-specific associations have not been reported previously, and previous reports have had limited numbers of events and subsequently power to undertake such analyses. In the large current analysis with balanced sexes, we also found no evidence of effect modification by sex in these findings after adjusting for body composition and other sex-specific confounders, consistent with previous findings.

Potential explanations for contrasting results
A central tenet of SCD epidemiology has been that coronary artery disease is the most common precipitant of sudden death in the western world. Previous studies have suggested that coronary artery disease accounts for approximately 70%–80% of cases, with <5% of cases due to a noncardiac cause. The mechanism underling the majority of SCD...
cases is therefore presumed to be a malignant VA triggered by ischemia, infarction, structural abnormalities, or other arrhythmogenic substrate. This has led to the presumption of equivalence between VA and SCD in some epidemiological studies; yet, in our study specifically identifying incident VA events, we demonstrated contrasting relationships of alcohol with the 2 outcomes.

Historic autopsy studies investigating the etiologies of SCD have potentially been limited by referral bias, with only a minority of deaths in these studies undergoing postmortem investigation. Decline in the incidence of SCD has also been noted in recent decades, thought in part due to improvements in coronary artery disease mortality.\(^{20,21}\) Disputing these figures, a recent postmortem study capturing nearly all out-of-hospital cardiac arrest deaths in San Francisco county between 2011 and 2014 demonstrated that only 56% of SCDs were confirmed as a sudden arrhythmic death, and 40% were considered noncardiac.\(^9\)

The contrasting relationships (U-shaped relationship for alcohol and SCD, and no association of alcohol with VA) could potentially be explained by the limited biological specificity of SCD diagnoses for a fatal arrhythmic event. Notably, the U-shaped relationship between alcohol and SCD seen in our and other studies mirrors that of alcohol and cardiovascular disease in general.\(^{22,23}\) These findings also differ from the previously reported relationship between alcohol and nonfatal coronary artery disease, which has been observed as L-shaped, or a flattening of lowered risk with consumption of >1–2 drinks per day.\(^{23}\) These conflicting shapes of association suggest that different mechanisms may be at play between these related outcomes and are findings that require further studies with accurately defined VA and SCD events.

Study strengths and limitations
To our knowledge, this is the largest study to characterize the relationship between alcohol and incident VA and SCD. Greater statistical power and beverage-specific data have allowed study of associations by beverage, and balanced sex proportions allowed investigation for effect modification between sex. Furthermore, adjustment for measurement error and long-term variability via regression calibration has not been previously undertaken. Observational studies relying on single point estimates of exposure variables measured with error suffer from regression dilution bias, where effect estimates are biased toward the null.\(^{15}\) Correction for such bias as in the present study allows for more accurate estimates of long-term alcohol exposure.

Several limitations warrant discussion. Despite a large number of events, we cannot exclude a more modest effect of alcohol that may have been detected with even greater power. The diagnostic codes used in our study to identify events have not been validated specifically in the databases linked to the UK Biobank; however, validation studies of these codes in other populations have demonstrated strong positive predictive values >80%.\(^{24,25}\) Additionally, specific details regarding whether deaths were unexpected, autopsies were performed, VT events were sustained or nonsustained, and cardiac arrests were due to a VA were not available through the linked records. Despite extensive multivariate adjustment and robust sensitivity analyses, we cannot exclude the possibility of residual confounding and reverse causality. Drinking patterns and lifestyle characteristics vary by race and population, so these findings require confirmation in different populations beyond our predominantly white British cohort. The representativeness of UK Biobank to general populations is limited by the “healthy volunteer” phenomenon; however, valid assessment of exposure–disease relationships are nonetheless widely generalizable and do not require participants to be representative of the population at large.

Conclusion
In this predominantly white British cohort, no association was observed with total alcohol, beer, cider, and red or white wine consumption with VA risk, although increasing spirit intake was associated with increasing risk. Consumption of up to 26 UK standard drinks (208 g of alcohol per week) was associated with the lowest risk of SCD. Beverage-specific analyses for SCD demonstrated contrasting associations. Consumption of greater amounts of beer, cider, and spirits was potentially associated with increasing risk, whereas increasing red and white wine intake was associated with reduced risk. These findings require clarification in further epidemiological and experimental studies with accurately defined VA and SCD events.

Appendix
Supplementary data
Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.hrthm.2021.09.040.

References


