Alcohol Intake and Health Study: No Protective Effect at Low Levels, With Mortality Increasing to 1 in 25 at 14 Drinks Per Week

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ABSTRACT. Objective: The purpose of this study was to estimate the lifetime risk of alcohol-attributable mortality and morbidity in the United States based on a person's average lifetime weekly alcohol consumption to assess the impact of per-occasion alcohol consumption on health. **Method:** Lifetime risks were estimated using a cause-specific modeling approach that combined exposure data from national health surveys, relative risks, population data from the U.S. Census Bureau, mortality data from the Centers for Disease Control and Prevention, and morbidity data from the Institute for Health Metrics and Evaluation. A narrative review assessed the health impact of per-occasion alcohol consumption on health. **Results:** At low levels of consumption, no protective net effect of alcohol consumption on health was observed. Elevated mortality and morbidity risks were associated with alcohol consumption starting at relatively low levels. Males consuming >6.5 (95% CI [<1, 13.5]) and females consuming >7.0 (95% CI [<1, 11.5]) drinks per week had lifetime alcohol-attributable mortality risks >1:1,000. At >8.5 (95% CI [2.5,

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13]) drinks per week for both males and females, these risks increased to >1:100. At 14 drinks per week for males (the upper limit of the former Dietary Guidelines for males), the risk of an alcohol-caused death was 1:25 (4%). Drinking patterns also impacted risk. Above 1 drink per occasion, higher consumption was associated with progressively increased risks of breast cancer, cardiovascular disease, and injury. **Conclusions:** Alcohol consumption, including at what may be perceived as "moderate" levels, is associated with increased mortality and morbidity risks. These results support tightening alcohol use guidance in the United States, for both males and females, to no more than 1 drink per day. (*J. Stud. Alcohol Drugs, 87,* 621–638, 2026)

Public health significance statement: The Alcohol Intake and Health Study shows that for Americans, even what is socially considered "moderate drinking" increases the risk of dying or developing health problems, helping people better understand the net health impact of alcohol. Furthermore, by identifying the levels of alcohol use that raise the risk of cancer, cardiovascular disease, and injury, these findings can guide individuals, families, and communities in making safer choices about drinking patterns. The results also support changing the U.S. Dietary Guidelines on alcohol to recommend that current adult drinkers consume 1 drink or less in a day.

ALCOHOL REMAINS the most widely used psychoactive substance in the United States. In 2024, 134.3 million individuals age 12 and older, 46.6% of the population, reported past-month drinking, and 57.9 million adults reported binge drinking (≥5 drinks within 2 hours for males, ≥4 for females; National Institute on Alcohol Abuse and Alcoholism [NIAAA], 2025a). The overall prevalence of past-year drinking has remained relatively stable in recent years, with binge drinking showing a 1.6% decrease between 2021 and 2024 (Substance Abuse and Mental Health Services Administration [SAMHSA], 2025). Alcohol consumption, even at what is perceived as "moderate" levels, is causally linked to more than 200 health conditions (measured by three-digit ICD-10 codes), underscoring its role in preventable disease, death, and economic loss (Rehm et al., 2017). As a result of alcohol consumption, the annual number of alcohol-attributable deaths in 2020–2021 was estimated to be 178,000, making alcohol a leading preventable cause of death and accounting for 12.9% of all deaths among working-age adults (Esser et al., 2022; Slater & Alpert, 2021).

Despite alcohol's substantial health burden, national estimates of lifetime-attributable mortality remain limited. All-cause mortality studies (i.e., studies relating alcohol consumption levels to deaths from any cause) include deaths from many conditions that are not causally related to alcohol. Furthermore, these studies combine studies that are often nonrepresentative (e.g., underrepresent marginalized groups, are outdated, and are conducted in locations whose mortality patterns do not reflect those in the United States; Shield et al., 2025a). These challenges limit the generalizability and relevance of the studies to U.S. public health. Rather, to effectively inform prevention strategies and drinking guidelines, it is important to estimate alcohol-attributable mortality using a cause-specific approach that includes conditions that have evidence of being causally related to alcohol use (including both harmful and protective risk estimates), as we do in the present study. The cumulative net risk from alcohol in a population can then be estimated by combining risk functions for all conditions causally related to alcohol consumption, weighting each risk function by its contribution to mortality in the population being studied.

The 2020–2025 U.S. Dietary Guidelines recommended limiting consumption to 2 drinks a day or less for males and 1 drink or less for females on days when alcohol is consumed (U.S. Department of Agriculture and U.S. Department of Health and Human Services, 2020). As new research emerges on the health effects of alcohol consumption, the guidance should be regularly reviewed and updated to reflect the most current scientific evidence and ensure that public health recommendations remain accurate and relevant. Furthermore, there is a need to update the guidance to include recommendations on low-risk alcohol consumption limits over longer periods, such as weekly intake, to better support public understanding of the cumulative health risks of alcohol. Accordingly, to inform an update to the guidelines that reflects new scientific evidence on the health impacts of alcohol at various levels of consumption, the Interagency Coordinating Committee on the Prevention of Underage Drinking (ICCPUD) convened an expert Scientific Review Panel to integrate the best available evidence (SAMHSA, 2025). This effort resulted in the present Alcohol Intake and Health study, which quantifies alcohol-related risks across consumption levels, drinking patterns, sex, and age.

Method

The full study protocol, detailing the analytical framework, data sources, and modeling procedures, has been previously published to ensure methodological transparency and facilitate reproducibility of the findings (Shield et al., 2025b). This study used a multi-method, cause-specific modeling approach to (a) estimate the lifetime risk and burden of alcohol-specific mortality and morbidity in the United States, and (b) perform a narrative review on the impact of per-occasion drinking on health.

The estimation of alcohol-specific risk involved three steps. First, a scoping review of systematic reviews and meta-analyses on alcohol-related diseases and injuries was performed (see supplemental material for details on the methods). (Supplemental material appears as an online-only addendum to this article on the journal's website.) Experts used this evidence to identify the most appropriate risk relationship for

each condition. Second, we applied these relationships to U.S. data on alcohol use, disease, and injury, comparing risks at specific consumption levels (e.g., 1, 2, or 3 drinks per week or per day) to those among lifetime abstainers (i.e., individuals who never consumed alcohol). Lifetime abstainers were used as the reference group to minimize bias from including former drinkers who may have quit due to health issues (i.e., sick-quitter bias; Stockwell et al., 2024).

Absolute risk curves were developed for conditions with established causal relationships to alcohol consumption (see Supplemental Table S5 for a list of conditions and corresponding ICD-10 codes), stratified by age, sex, and level of alcohol use. These condition-specific risks were aggregated to estimate the total alcohol-specific mortality risk. This framework followed established methodologies from the Centers for Disease Control and Prevention (CDC), the World Health Organization (WHO), and the Institute for Health Metrics and Evaluation (IHME; GBD 2016 Alcohol Collaborators, 2018; Rumgay et al., 2021; WHO, 2018a, 2018b), and with other recently released guidance documents on alcohol and health (Alcohol Guidelines Project Team, 2020; Canadian Centre on Substance Use and Addiction, 2023; Department of Health [United Kingdom], 2016; Santé publique France, 2019).

We used separate models to assess risk from any alcohol consumption and specific average daily intake levels, defined according to the U.S. definition of a standard drink (i.e., 13.6 g of ethanol; NIAAA, 2025b). Models included only diseases and injuries that (a) are causally linked to alcohol use (determined based on epidemiological, animal, and mechanistic evidence; Alldredge & Lowenstein, 1993; Cherpitel et al., 2018a, 2018b; Connor, 2017; Morojele et al., 2021; Roerecke, 2021), (b) have an available dose-response risk function based on daily alcohol intake (grams), and (c) have mortality or morbidity data specific to alcohol-related cases. Alcohol-related cancer types were treated separately because of varying alcohol associations by site. The modeling addressed both fatal and nonfatal outcomes: deaths, years of potential life lost (YPLL), and disability-adjusted life years (DALY; Murray, 1994). Mortality data are detailed in this report, and methods and results for YPLLs and DALYs are presented in the supplemental material.

To ensure relevance for U.S. public health policy and practice, the study avoided reliance on international all-cause mortality data and instead modeled cause-specific risks using nationally representative U.S. data sets. As the analysis relied solely on publicly available, de-identified data, research ethics approval was not required.

Data sources

Alcohol consumption data were obtained from multiple national population-based surveys, including the National Alcohol Survey, the National Survey on Drug Use and Health, the National Health Interview Survey (Alcohol Research Group, 2025), the National Epidemiologic Survey on Alcohol and Related Conditions–III (NIAAA, 2012), and the Behavioral Risk Factor Surveillance System (CDC, 2025; see Supplemental Table S1 for details on the surveys used).

U.S. mortality data from 2022 (the latest year available at the time of the study), including death counts by age, sex, and cause, were sourced from the National Vital Statistics System (Ahmad et al., 2023). The 2022 age and sex demographic data were obtained from the U.S. Census Bureau (2023). YPLL were estimated based on average life expectancy at death (estimated using a period life expectancy at birth methodology), using mortality and life table data from the National Vital Statistics System (CDC, 2025). The 2022 injury data, including blood alcohol concentration (BAC) levels associated with injuries, were obtained from the Fatality Analysis Reporting System (FARS)[1], CDC's National Violent Death Reporting System (NVDRS)[2], and a systematic review and meta-analysis by Alpert et al. (2022).

We modeled past-year drinking prevalence using these survey data (Supplemental Table S1); however, survey data account for only 40%–60% of alcohol sold (WHO, 2024). Since risk estimates rely on accurate consumption data, we corrected survey data for undercoverage by using data on adult per capita sales from the Alcohol Epidemiologic Data System as a proxy for per capita consumption levels (APC; Midanik, 1988; Slater & Alpert, 2021) and WHO data to account for unreported APC (e.g., moonshine; WHO, 2024). We upshifted the mean consumption from survey data, stratified by age and sex, to 80% of the APC data. Eighty percent was chosen rather than 100% to account for alcohol sold but not consumed, wasted, spilled, and the underreporting of consumption in survey data from which relative risk (RR) estimates were obtained. Following the methods of Kehoe et al. (2012) and Rehm et al. (2010), we modeled consumption as a gamma distribution, with the distribution's mean used to predict the standard deviation.

This method provides a population-level approximation of alcohol consumption that aligns with survey and APC data. It assumes that underreporting and nonconsumed alcohol are relatively consistent across age and sex groups and over time, and generally estimates a value for these competing factors. As a result, although this approach improves alignment between survey and sales data, there remains uncertainty about true consumption levels, which should be considered when interpreting the estimated risks and burdens.

Risk relationship estimation

Alcohol-related risk relationships were identified through a systematic review of meta-analyses focusing on condi-

[1]https://www.nhtsa.gov/research-data/fatality-analysis-reporting-system-fars

[2]https://www.cdc.gov/nvdrs/about/index.html

tions causally linked to alcohol consumption. Panels of experts from relevant fields (cancer, cardiovascular diseases, digestive conditions, neurological disorders, and infectious diseases) evaluated the evidence. They selected the highest quality meta-analytic RR between alcohol use at various levels and health conditions. These experts were selected based on their publications over the past decade, identified through a systematic PubMed search. Each expert completed a questionnaire selecting their top three RRs within their field of expertise. For each field, the RR with the highest total ranking was selected for modeling. When sex-specific RRs were not selected through this process, generalized estimates were applied to both males and females.

Lifetime risk calculations

To estimate the lifetime risk of alcohol-attributable mortality and morbidity, we constructed risk curves that incorporated age-, sex-at-birth-, and alcohol-consumption-level-specific mortality and morbidity risks, applying the same alcohol-attributable fractions to both. Cause-specific risk curves were generated by calculating age- and sex-specific alcohol-attributable mortality risks for each condition and then summing them across causes. These risks were weighted by the probability of surviving to each age from the U.S. Vital Statistics data, which ensured that competing causes of death, including non–alcohol-related mortality, were incorporated. Exact formulas for these calculations are provided in the supplemental material. For RRs derived from epidemiological studies, we applied a correction assuming approximately 10% underreporting of alcohol intake due to self-report, consistent with the exposure definitions used in the underlying RR studies (WHO, 2024).

The life-year specific alcohol-attributable mortality risks were combined with the YPLL for each cause to account for total years of life lost. The final lifetime risk curves incorporated both the calculated alcohol-attributable mortality and morbidity risks and the baseline risks for lifetime abstainers. This approach allowed us to model the absolute risks associated with alcohol consumption while accounting for demographic factors and varying consumption patterns.

The risk of alcohol-attributable cancer for a given year was calculated by summing the incidence risks for each cancer type that is causally associated with alcohol use (according to the International Agency for Research on Cancer), adjusted for age, sex, and daily alcohol consumption. Survival probabilities were incorporated to accurately reflect the likelihood of surviving to the next year, accounting for alcohol intake.

Population-attributable fraction calculation and estimation of alcohol-attributable burden

Population-attributable fractions (PAFs) were calculated to quantify the proportion of risk attributable to alcohol consumption, using the Levin-based method that integrated alcohol exposure data with identified RR estimates (Levin, 1953). This approach is based on the theoretical minimal risk exposure level (TMREL), defined as lifetime abstention from alcohol use. The resulting health burden estimates reflect the alcohol consumption prevalence data from 2022 for all diseases causally associated with alcohol use, except for cancer, where data from 2012 were used to account for disease latency. For other diseases and injuries, PAFs were calculated using age- and sex-specific mortality data. For conditions 100% attributable to alcohol (e.g., alcoholic cardiomyopathy), the PAF was set to 1.0.

Risk estimation and adjustment for injury

When estimating the burden of injuries attributable to alcohol, we focused on the proportion of injuries associated with a BAC of 0.10 g/dl or higher (Alpert et al., 2022; Naimi et al., 2023). This threshold was chosen based on RR functions from case-crossover studies, which indicate that injuries at or above this BAC level are attributable to alcohol use (Cherpitel et al., 2018a, 2018b). For motor vehicle crashes, however, we used a BAC threshold of .08% to define alcohol attribution, as per FARS protocol. This does not account for injuries at a lower BAC, where alcohol can play a role (e.g., motor vehicle injuries at .05%, at which level alcohol is shown to significantly impact driving skills in closed road studies; Moskowitz & Florentino, 2000; Schnabel, 2012; Verster & Ramaekers, 2009).

We then followed a two-step process to estimate injury-related RRs. First, we determined the shape of the risk curve between alcohol use and injury. Second, we calculated the RRs for individuals who consumed alcohol in the past year, based on their average daily alcohol intake. To estimate these RRs, we used data on PAFs of injuries derived from toxicology reports on BAC and self-reported alcohol use.

Risk thresholds

The concept of risk acceptability provides important context for interpreting these findings. Acceptable levels of risk from alcohol consumption vary across individuals and populations, with societal norms often permitting higher tolerance for voluntary risks (e.g., alcohol use) compared with involuntary risks (e.g., environmental exposures). Historical analyses, like those by Starr, suggest that society may accept risks up to 1 in 1,000 alcohol-attributable deaths as reasonable (Rehm et al., 2014; Starr, 1969). However, more recent international standards, such as those used in Australia and the United Kingdom, suggest a threshold of 1 in 100 lifetime alcohol-attributable deaths as an acceptable risk level (Alcohol Guidelines Project Team, 2020; UK Chief Medical Officer, 2016). However, these thresholds are subjective choices based on expert opinion

and have not been verified for acceptability among the general population. For PYLL and DALYs, a threshold of 15 years per 1,000 people (corresponding to 1:1,000 risk of death) and 100 people (1:100 risk of death) was used. This value reflects average years of life lost across all deaths in the U.S. population and was used to translate mortality risk into PYLL and DALY benchmarks.

Risk thresholds were identified based on point estimates crossing these values rather than the associated confidence intervals (CIs). Further discussion of the implications of this choice is available in the limitations section.

Narrative review of per-occasion drinking patterns

To comprehensively assess the risks associated with per-occasion drinking patterns, we conducted a narrative review using multiple data sources. These included (a) systematic reviews and meta-analyses that examine the association between drinking patterns and disease/injury risk, (b) re-analyses of U.S. emergency department case-crossover studies exploring the link between alcohol use and injury occurrence, and (c) roadside survey studies comparing BAC levels of road injury decedents with those of randomly selected drivers at the same location and time. This approach ensured a robust evaluation of both chronic and acute impacts of alcohol use.

On the use of significance tests

As explained above, the main objective of this report was to describe the mortality risks associated with different levels of alcohol consumption and the precision of these estimates. No hypotheses were tested in a Popperian or other scientific framework; thus, significance tests do not play a major role in this report (Popper, 1959). When describing words such as *significant,* we mainly refer to the precision of our analyses relative to a common value and provide CIs.

All calculations were conducted using R software (R Core Team, 2013).

Results

Systematic review of alcohol use and the risk of alcohol-related conditions

The search strategy yielded 7,294 publications after duplicates were excluded. After completion of the full-text assessments, a total of 56 unique systematic reviews were included in the current review (Figure 1), of which 16 were selected by topic-area experts and used for the analyses. Despite a previously established causal relationship, no systematic reviews on the direct relationship between alcohol consumption and the risk of HIV/AIDS (Rehm et al., 2017), other sexually transmitted diseases (Llamosas-Falcón et al., 2023), cervical cancer (Oh et al., 2015; Schiffman et al., 1993), or depression (Bahorik et al., 2016) were found, and those health outcomes were thus excluded from risk estimation. Supplemental Table S6 describes the study characteristics of the systematic reviews selected by the experts.

Lifetime alcohol-attributable mortality risk based on all conditions causally linked to alcohol use

Table 1 outlines the absolute lifetime risk of alcohol-attributable death at levels of average alcohol consumption, stratified by age at death and sex. Although there were small protective associations at up to 3 drinks per week for females and males, they were not statistically significant. Males who consumed more than 6.5 drinks per week (95% CI [<1, 13.5] drinks per week) exhibited a lifetime alcohol-attributable mortality risk of >1 in 1,000, whereas females reached this same risk threshold at 7.0 standard drinks per week (95% CI [<1, 11.5] drinks per week). The mortality risk increased to >1 in 100 for both sexes when weekly consumption exceeded 8.5 drinks per week (95% CI [2.5, 13] drinks per week). At 14 drinks per week for males, the risk of an alcohol-attributable death was approximately 4% (39.3, 95% CI [9.6, 69.6] deaths per 1,000 people). For females consuming 7 drinks per week, the risk was 0.1 (95% CI [-19.4, 21.9]) alcohol-attributable deaths per 1,000 people (i.e., a 0.01% chance of an alcohol-caused death).

Sex and age differences

Overall, total mortality risks were similar by sex, up to a consumption of approximately 14 drinks per week (i.e., 2 standard drinks per day), at 39.34 per 1,000 (95% CI [9.65, 69.62]) for males and 40.53 per 1,000 (95% CI [18.49, 63.28]) for females (Table 1, Figure 2). Beyond this, for example, at 3 standard drinks per day, the risk for males (68.92 per 1,000, 95% CI [37.51, 101.23]) is lower than for females at the same level of consumption (82.43 per 1,000, 95% CI [57.78, 108.45]; Figure 2).

Results indicated that younger adults demonstrated a greater vulnerability to alcohol-related deaths compared with older adults (Table 1). Among people less than 40 years of age, no protective net effect of alcohol on health was observed even at lower levels of alcohol consumption. Furthermore, within this age group (<40 years of age), most alcohol-attributable deaths are caused by road traffic crashes, other unintentional injuries, and intentional injuries, compared with older age groups, which have a further spread across causes of death.

Figure 3 shows the risk of an alcohol-attributable death for different levels of alcohol consumption, by disease. In contrast, Table 2 and Supplemental Figures S1–S4 show the relative risks for different levels of alcohol consumption, by disease. A similar pattern was observed for cause-specific

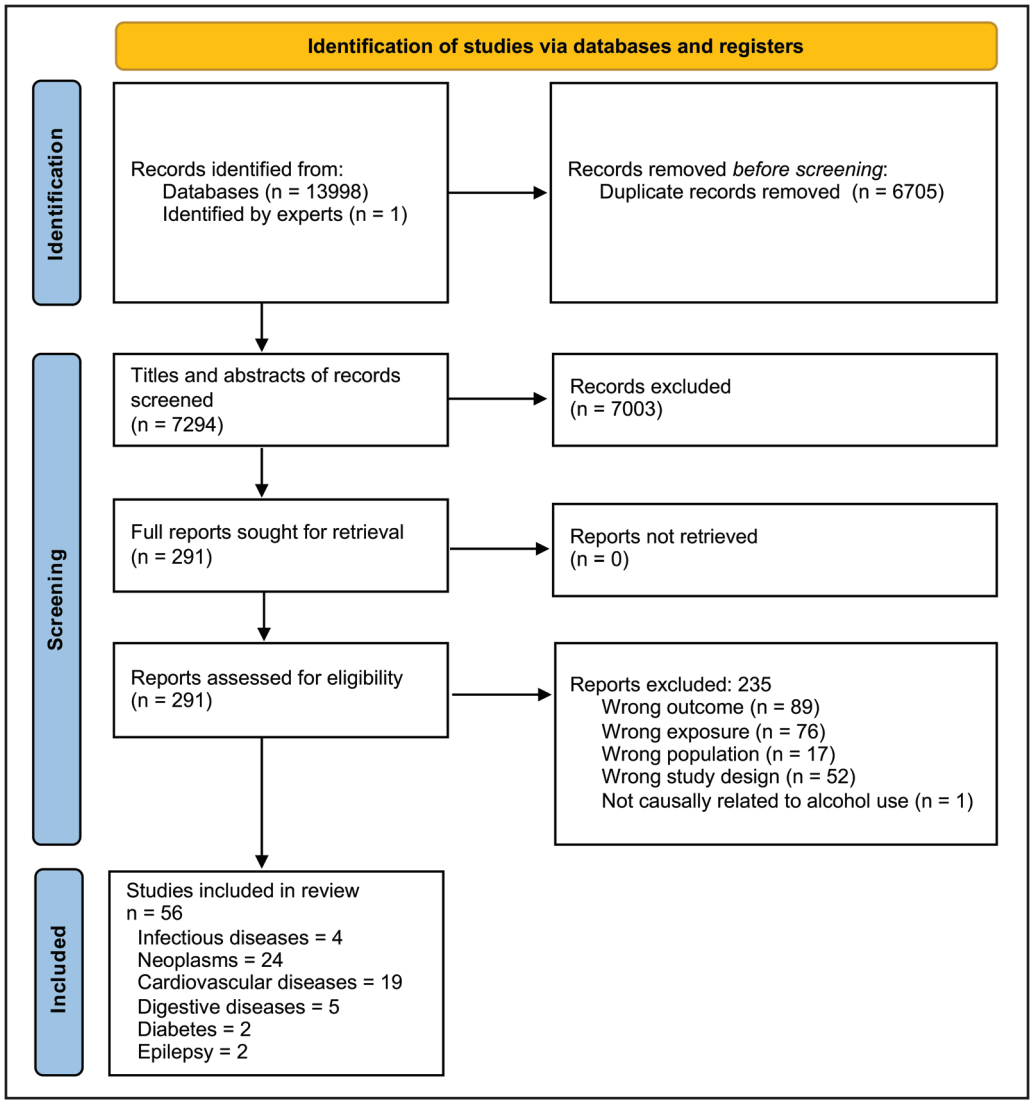

FIGURE 1. Systematic search results for systematic reviews and meta-analyses on the risk relationship between alcohol consumption and disease occurrence

relative risks, which were generally similar for males and females up to approximately 14 drinks per week. Beyond this level, differences by sex became more pronounced, with females at higher risk. For example, at 14 drinks per week, the relative risk of death due to cirrhosis and other chronic liver diseases was 2.10 (95% CI [1.68, 2.65]) for males, compared with 5.38 (95% CI [3.81, 7.73]) for females. These differences increased further at 21 drinks per week, with relative risks of 3.58 (95% CI [2.90, 4.48]) for males and 10.67 (95% CI [7.78, 14.63]) for females.

Impact of alcohol use on disability-adjusted years of life lost

Among males, the risk of 15 lifetime alcohol-attributable DALYs lost per 1,000 people occurred at a consumption level above 2.5 (95% CI [<1, 6.5]) drinks per week. In contrast, the risk of 15 alcohol-attributable DALYs lost per 100 people occurred above 4.5 (95% CI [<1, 8]; Figure 4). Among females, the risk of 15 lifetime alcohol-attributable DALYs lost per 1,000 people occurred at a consumption level above 6.5 (95% CI [<1, 9.5]) drinks per week. In comparison, the risk of 15 alcohol-attributable DALYs lost per 100 people occurred above 8 (95% CI [5, 11]) drinks per week.

The health impacts of per-occasion alcohol consumption (i.e., drinking patterns) on disease risk

Patterns of alcohol use at the occasion level influence the likelihood of infectious disease, noncommunicable disease, and injury. Many studies reviewed use thresholds of ≥5 drinks for males and ≥4 drinks for females to define binge or heavy episodic drinking. Consumption at or above these

Table 1. Lifetime risk [95% CI] of an alcohol-attributable death per 1,000 people, overall and stratified by age at death

Sex	Age, in years	Drinks per week					
		1	2	3	7	14	21
Male	Total	-16.30 [-46.57, 11.41]	-13.46 [-43.99, 13.80]	-10.56 [-40.92, 16.42]	1.70 [-28.77, 29.08]	39.34 [9.65, 69.62]	68.92 [37.51, 101.23]
	15–19	0.05 [0.04, 0.06]	0.10 [0.09, 0.13]	0.16 [0.13, 0.19]	0.38 [0.32, 0.47]	0.82 [0.68, 1.04]	1.33 [1.08, 1.71]
	20–24	0.08 [0.06, 0.10]	0.17 [0.13, 0.21]	0.25 [0.20, 0.32]	0.62 [0.50, 0.79]	1.35 [1.07, 1.75]	2.18 [1.71, 2.88]
	25–29	0.10 [0.06, 0.13]	0.21 [0.15, 0.28]	0.32 [0.24, 0.42]	0.80 [0.61, 1.05]	1.75 [1.34, 2.34]	2.84 [2.15, 3.87]
	30–39	0.17 [0.00, 0.34]	0.44 [0.23, 0.65]	0.70 [0.44, 1.00]	1.83 [1.30, 2.50]	4.18 [3.10, 5.78]	6.88 [5.09, 9.65]
	40–49	-0.10 [-0.77, 0.56]	0.19 [-0.49, 0.84]	0.48 [-0.22, 1.18]	1.71 [0.86, 2.62]	4.61 [3.27, 6.30]	7.75 [5.81, 10.58]
	50–59	-0.82 [-2.82, 1.10]	-0.47 [-2.47, 1.42]	-0.12 [-2.13, 1.79]	1.36 [-0.71, 3.31]	5.46 [3.14, 8.14]	9.52 [6.75, 12.94]
	60–69	-2.13 [-6.37, 1.86]	-1.72 [-5.95, 2.23]	-1.30 [-5.57, 2.61]	0.46 [-3.83, 4.46]	6.12 [1.87, 10.85]	11.12 [6.55, 16.37]
	≥70	-13.66 [-36.71, 7.46]	-12.37 [-35.56, 8.44]	-11.06 [-34.26, 9.41]	-5.47 [-28.59, 14.57]	15.04 [-6.71, 35.66]	27.30 [5.58, 48.33]
Female	Total	-14.79 [-34.02, 5.57]	-12.93 [-32.42, 8.00]	-10.88 [-30.25, 10.57]	0.15 [-19.37, 21.90]	40.53 [18.49, 63.28]	82.43 [57.78, 108.45]
	15–19	0.02 [0.02, 0.03]	0.04 [0.03, 0.06]	0.06 [0.05, 0.09]	0.16 [0.13, 0.21]	0.35 [0.27, 0.48]	0.58 [0.44, 0.81]
	20–24	0.03 [0.02, 0.04]	0.06 [0.05, 0.08]	0.09 [0.07, 0.13]	0.24 [0.18, 0.32]	0.53 [0.40, 0.72]	0.87 [0.65, 1.22]
	25–29	0.03 [0.02, 0.05]	0.08 [0.05, 0.11]	0.12 [0.09, 0.16]	0.31 [0.23, 0.42]	0.71 [0.54, 0.99]	1.21 [0.91, 1.72]
	30–39	0.07 [0.00, 0.14]	0.18 [0.10, 0.28]	0.30 [0.19, 0.44]	0.85 [0.61, 1.18]	2.17 [1.65, 2.98]	3.91 [3.01, 5.42]
	40–49	-0.05 [-0.29, 0.21]	0.09 [-0.16, 0.38]	0.25 [-0.02, 0.56]	0.99 [0.58, 1.52]	3.08 [2.36, 4.17]	5.87 [4.66, 7.93]
	50–59	-0.34 [-1.02, 0.41]	-0.12 [-0.80, 0.66]	0.12 [-0.58, 0.93]	1.32 [0.46, 2.39]	5.02 [3.78, 6.77]	9.92 [7.96, 12.76]
	60–69	-1.08 [-2.70, 0.69]	-0.80 [-2.43, 1.00]	-0.49 [-2.09, 1.31]	1.16 [-0.57, 3.25]	6.72 [4.48, 9.55]	13.79 [10.87, 17.66]
	≥70	-13.48 [-30.26, 4.23]	-12.47 [-29.19, 5.30]	-11.34 [-28.08, 6.52]	-4.87 [-22.22, 13.88]	21.95 [3.61, 40.93]	46.28 [25.55, 65.84]

Note: CI = confidence interval.

levels typically results in BAC levels of .08% or higher for most individuals.

Impact of drinking patterns on cancer risk

In their systematic review, Sohi et al. identified two studies demonstrating that females who reported binge drinking within the past year had a higher risk of breast cancer than those who did not engage in binge drinking (Sánchez-Bayona et al., 2020; Sohi et al., 2024; White et al., 2017). In addition, the same review noted evidence from two studies indicating that increasing the number of drinks consumed on a single occasion was associated with elevated breast cancer risk (Chen et al., 2011; Mørch et al., 2007; Sohi et al., 2024). At present, there is limited empirical support suggesting that per-occasion drinking volumes substantially influence the incidence of other cancer types.

Impact of drinking patterns on cardiovascular disease risk

Roerecke and Rehm's (2014) systematic review examined seven studies evaluating ischemic heart disease risk associated with binge-level consumption (60 g or 4.3 standard drinks per occasion) among individuals whose average intake remained low to moderate (<30 g, or roughly 2.1 standard drinks per day). When compared with lifetime abstainers, moderate drinkers without binge episodes exhibited a pooled relative risk (RR) of 0.64 (95% CI [0.53, 0.71]) for ischemic heart disease incidence, whereas individuals with the same average intake who engaged in binge drinking had a pooled RR of 1.12 (95% CI [0.91, 1.37]). These findings indicate that heavy episodic drinking may offset or reverse the protective associations often observed with moderate alcohol consumption.

Complementary evidence from Mostofsky et al. shows that high-intensity drinking episodes markedly increase short-term cardiovascular risk, including myocardial infarction and both ischemic and hemorrhagic stroke, with elevated risk observed both the following day (6–9 drinks: RR 1.3 to 2.3) and over the subsequent week (19–30 drinks: RR 2.3 to 6.2; Mostofsky et al., 2016).

Impact of drinking patterns on liver disease risk

A systematic review by Llamosas-Falcón and colleagues examined drinking patterns in relation to liver cirrhosis and found that daily alcohol use was associated with a substantially higher risk of cirrhosis than nondaily consumption, even when total weekly or monthly drinking volume was held constant. However, because the included analyses did not adjust for the quantity consumed per occasion, the implications for understanding per-occasion effects remain unclear (Llamosas-Falcón et al., 2023).

Impact of drinking patterns on injury risk

Research consistently shows a strong, dose-responsive relationship between BAC, the amount of alcohol consumed, and the likelihood of sustaining an injury. As BAC rises, injury risk increases markedly. In their systematic review, Taylor and Rehm (2012) reported that each .02% increase in BAC, roughly equivalent to a single standard drink, raises the odds of a fatal motor vehicle collision by about 74%. At the commonly enforced legal limit of .08% BAC, the estimated odds ratio (OR) for a fatal crash was 13.0 (95% CI [11.1, 15.2]). Earlier work by Taylor et al. demonstrated that this relationship is nonlinear, with particularly sharp escalations at higher consumption levels; for instance, drinking

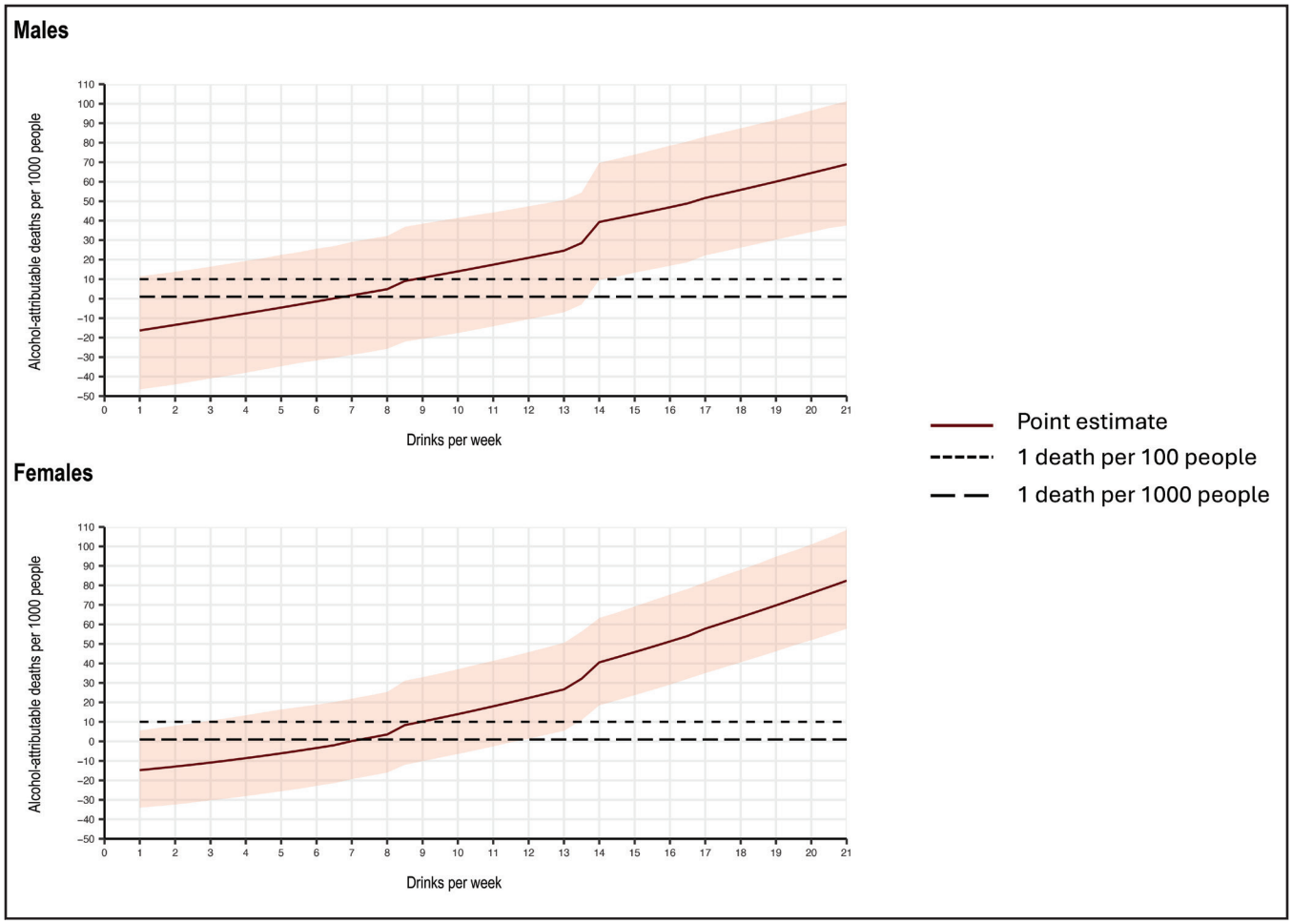

FIGURE 2. Lifetime risk of an alcohol-attributable death among males and females for differing levels of alcohol consumption

roughly 120 g of alcohol (about 8.5 drinks) within 3 hours before driving was linked to an OR of 52.0 (95% CI [34.5, 78.3]). Even moderate alcohol consumption carries meaningful risk: Estimated ORs were 1.79 (95% CI [1.59, 2.00]) for nontraffic injuries and 2.20 (95% CI [2.03, 2.09]) for motor vehicle–related injuries associated with alcohol consumption of around 24 g per day, roughly 1.7 standard drinks (Taylor et al., 2010).

Motor vehicle collisions remain the most prominent adverse outcome associated with elevated BAC. According to data from the 2013–2014 National Roadside Survey, the proportion of drivers testing positive for alcohol has decreased substantially over time, particularly for drivers at or above the legal BAC threshold (Ramirez et al., 2016). However, these improvements have not been observed uniformly across all groups; drivers younger than 20 years have not shown meaningful reductions in alcohol-related crash risk. A large review by Høye and Storesund Hesjevoll, synthesizing findings from 60 studies, found that crash and injury risk increase steeply, and nearly exponentially, with rising BAC levels, particularly for severe collisions, across a BAC range from .01% to .20% (Høye & Storesund Hesjevoll, 2023).

Alcohol also plays a role in intentional injuries. Meta-analytic evidence indicates that acute alcohol consumption is strongly associated with suicide attempts. In the most comprehensive review to date, Borges et al. reported that any acute alcohol use, measured as a detectable BAC in emergency or postmortem samples, or self-reported drinking before the attempt, was linked to an OR of 6.97 (95% CI [4.77, 10.17]). Risk varied with dose: "Low-level" acute drinking was tied to an OR of 2.71 (95% CI [1.56, 4.71]), whereas "high-level" drinking was associated with an OR of 37.18 (95% CI [17.38, 79.53]; Borges et al., 2017). In addition, a U.S. study spanning 2003–2018 found significant annual increases in the proportion of alcohol-positive suicides (BAC ≥ .08 g/dl), particularly among females of all ages and middle-aged males; increases in pre-suicide drinking were especially pronounced among females (Lange et al., 2022).

Patterns of alcohol use also shape risks of interpersonal violence. The evidence shows that perpetrators typically present with higher BAC levels than victims (Kilian et al.,

TABLE 2. Relative risks of disease-specific mortality for different levels of alcohol consumption, compared with lifetime abstainers

Cause of death	Source	Sex	Drinks per week					
			1	2	3	7	14	21
Communicable, maternal, neonatal, and nutritional diseases								
Tuberculosis	(Simou et al., 2018b)	Both	1.02 [1.01, 1.02]	1.03 [1.03, 1.03]	1.05 [1.04, 1.05]	1.11 [1.09, 1.12]	1.24 [1.19, 1.26]	1.37 [1.31, 1.41]
Pneumonia	(Simou et al., 2018a)	Both	1.01 [1.01, 1.01]	1.02 [1.02, 1.02]	1.03 [1.02, 1.04]	1.07 [1.06, 1.08]	1.15 [1.11, 1.17]	1.24 [1.18, 1.27]
HIV/AIDS	–	–	–	–	–	–	–	–
Other sexually transmitted diseases	–	–	–	–	–	–	–	–
Noncommunicable diseases								
Neoplasms								
Oral cavity	(World Cancer Research Fund/ American Institute for Cancer Research, 2018a)	Both	1.03 [1.02, 1.04]	1.06 [1.04, 1.08]	1.09 [1.05, 1.13]	1.22 [1.13, 1.32]	1.48 [1.27, 1.75]	1.80 [1.44, 2.31]
Pharyngeal cancer (excluding nasopharynx)	(World Cancer Research Fund/ American Institute for Cancer Research, 2018a)	Males	1.02 [1.01, 1.04]	1.04 [1.01, 1.08]	1.06 [1.02, 1.12]	1.16 [1.04, 1.31]	1.34 [1.09, 1.71]	1.55 [1.13, 2.23]
		Females	1.05 [1.00, 1.10]	1.09 [1.00, 1.20]	1.14 [0.99, 1.32]	1.37 [0.99, 1.90]	1.87 [0.97, 3.60]	2.55 [0.96, 6.83]
Laryngeal cancer	(World Cancer Research Fund/ American Institute for Cancer Research, 2018a)	Males	1.02 [1.01, 1.02]	1.04 [1.02, 1.05]	1.05 [1.03, 1.07]	1.13 [1.07, 1.17]	1.27 [1.15, 1.37]	1.44 [1.23, 1.61]
		Females	1.04 [1.01, 1.08]	1.08 [1.01, 1.16]	1.13 [1.02, 1.25]	1.32 [1.04, 1.68]	1.75 [1.09, 2.83]	2.31 [1.13, 4.76]
Esophageal cancer	(World Cancer Research Fund/ American Institute for Cancer Research, 2018b)	Males	1.06 [1.04, 1.08]	1.12 [1.08, 1.17]	1.19 [1.13, 1.26]	1.51 [1.32, 1.71]	2.27 [1.75, 2.94]	3.42 [2.31, 5.04]
		Females	1.05 [1.03, 1.06]	1.09 [1.05, 1.13]	1.14 [1.08, 1.21]	1.37 [1.20, 1.55]	1.87 [1.44, 2.41]	2.55 [1.73, 3.75]
Colon and rectum cancer	(Jun et al., 2023)	Males	1.16 [1.04, 1.28]	1.16 [1.04, 1.28]	1.16 [1.04, 1.28]	1.14 [1.09, 1.20]	1.29 [1.23, 1.34]	1.29 [1.23, 1.34]
		Females	1.00 [0.97, 1.04]	1.00 [0.97, 1.04]	1.00 [0.97, 1.04]	1.01 [0.78, 1.30]	1.07 [1.00, 1.14]	1.07 [1.00, 1.14]
Liver cancer	(World Cancer Research Fund/ American Institute for Cancer Research, 2015)	Males	1.01 [1.00, 1.01]	1.01 [1.00, 1.02]	1.02 [1.01, 1.03]	1.04 [1.01, 1.07]	1.09 [1.03, 1.15]	1.13 [1.04, 1.23]
		Females	1.04 [1.01, 1.06]	1.07 [1.02, 1.13]	1.11 [1.02, 1.20]	1.28 [1.06, 1.52]	1.63 [1.12, 2.32]	2.08 [1.18, 3.53]
Breast cancer								
Premenopausal	(Sohi et al., 2024)	Females	1.01 [1.00, 1.01]	1.02 [1.00, 1.03]	1.02 [1.01, 1.04]	1.06 [1.01, 1.10]	1.12 [1.03, 1.21]	1.18 [1.04, 1.33]
Postmenopausal		Females	1.02 [1.02, 1.03]	1.05 [1.04, 1.05]	1.07 [1.06, 1.08]	1.17 [1.14, 1.20]	1.37 [1.31, 1.44]	1.61 [1.49, 1.73]
Cervical cancer	–	Females	–	–	–	–	–	–
Cardiovascular diseases								
Ischemic heart disease	(Zhao et al., 2017)	Both	0.87 [0.71, 1.06]	0.87 [0.71, 1.06]	0.87 [0.71, 1.06]	0.87 [0.71, 1.06]	0.92 [0.75, 1.14]	0.92 [0.75, 1.14]
Ischemic stroke	(Larsson et al., 2016)	Both	0.90 [0.85, 0.95]	0.90 [0.85, 0.95]	0.90 [0.85, 0.95]	0.92 [0.87, 0.97]	1.08 [1.01, 1.15]	1.08 [1.01, 1.15]
Intracerebral hemorrhage and subarachnoid hemorrhage	(Zhang et al., 2014)	Both	0.96 [0.74, 1.24]	0.96 [0.74, 1.24]	0.96 [0.74, 1.24]	0.96 [0.74, 1.24]	1.21 [0.85, 1.73]	1.29 [0.98, 1.71]
Hypertensive heart disease	(Cecchini et al., 2024)	Males	1.03 [1.02, 1.03]	1.06 [1.04, 1.09]	1.08 [1.05, 1.10]	1.18 [1.13, 1.24]	1.34 [1.26, 1.42]	1.44 [1.34, 1.55]

Table continued

TABLE 2. *Continued*

Cause of death	Source	Sex	Drinks per week					
			1	2	3	7	14	21
		Females	1.00 [0.97, 1.03]	1.00 [0.93, 1.09]	1.01 [0.91, 1.11]	1.03 [0.85, 1.26]	1.23 [0.95, 1.59]	1.54 [1.02, 2.34]
Atrial fibrillation and flutter	(Jiang et al., 2022)	Males	1.01 [1.01, 1.02]	1.03 [1.02, 1.04]	1.04 [1.02, 1.05]	1.09 [1.06, 1.13]	1.20 [1.12, 1.28]	1.31 [1.19, 1.44]
		Females	1.01 [0.99, 1.02]	1.02 [0.99, 1.04]	1.02 [0.98, 1.07]	1.06 [0.95, 1.17]	1.12 [0.91, 1.36]	1.19 [0.87, 1.58]
Digestive diseases								
Cirrhosis and other chronic liver diseases	(Llamosas-Falcón et al., 2022)	Males	1.04 [1.02, 1.07]	1.09 [1.04, 1.15]	1.14 [1.06, 1.23]	1.37 [1.18, 1.62]	2.10 [1.68, 2.65]	3.58 [2.90, 4.48]
		Females	1.13 [1.07, 1.19]	1.27 [1.15, 1.41]	1.43 [1.24, 1.67]	2.33 [1.74, 3.17]	5.38 [3.81, 7.73]	10.67 [7.78, 14.63]
Pancreatitis	(Samokhvalov et al., 2015)	Both	1.01 [1.00, 1.02]	1.02 [1.00, 1.04]	1.03 [1.01, 1.05]	1.07 [1.01, 1.13]	1.14 [1.02, 1.27]	1.22 [1.04, 1.44]
Diabetes mellitus	(Llamosas-Falcón et al., 2023)	Males	1.00 [1.00, 1.00]	1.00 [1.00, 1.01]	1.01 [1.00, 1.01]	1.02 [1.00, 1.03]	1.03 [1.00, 1.07]	1.05 [0.99, 1.10]
		Females	0.93 [0.91, 0.94]	0.86 [0.82, 0.89]	0.80 [0.75, 0.84]	0.70 [0.65, 0.75]	0.74 [0.67, 0.81]	0.79 [0.69, 0.90]
Epilepsy	(Woo et al., 2022)	Both	1.02 [1.01, 1.03]	1.04 [1.02, 1.06]	1.06 [1.02, 1.09]	1.13 [1.06, 1.21]	1.29 [1.12, 1.48]	1.46 [1.18, 1.79]
Depression	–	–	–	–	–	–	–	–
Injuries								
Unintentional injuries								
Road injuries	*	Males	1.03 [1.02, 1.03]	1.05 [1.05, 1.07]	1.08 [1.07, 1.10]	1.20 [1.17, 1.25]	1.43 [1.36, 1.56]	1.71 [1.59, 1.94]
		Females	1.03 [1.02, 1.04]	1.05 [1.04, 1.07]	1.08 [1.07, 1.11]	1.20 [1.16, 1.28]	1.45 [1.34, 1.63]	1.74 [1.56, 2.09]
Poisonings (other than alcohol)	*	Both	1.02 [1.01, 1.03]	1.04 [1.03, 1.07]	1.07 [1.04, 1.10]	1.17 [1.10, 1.25]	1.36 [1.20, 1.56]	1.58 [1.32, 1.94]
Other unintentional injuries (excluding poisonings)	*	Both	1.04 [1.01, 1.08]	1.08 [1.01, 1.17]	1.12 [1.02, 1.27]	1.29 [1.04, 1.74]	1.68 [1.08, 3.04]	2.17 [1.12, 5.29]
Intentional injuries								
Self-harm	*	Males	1.02 [1.01, 1.02]	1.04 [1.03, 1.04]	1.05 [1.05, 1.07]	1.13 [1.11, 1.16]	1.28 [1.23, 1.35]	1.44 [1.37, 1.57]
		Females	1.03 [1.02, 1.03]	1.05 [1.04, 1.07]	1.08 [1.06, 1.11]	1.20 [1.15, 1.27]	1.43 [1.33, 1.61]	1.71 [1.54, 2.04]
Interpersonal violence	*	Males	1.01 [1.01, 1.02]	1.03 [1.03, 1.04]	1.04 [1.04, 1.06]	1.11 [1.09, 1.14]	1.23 [1.19, 1.29]	1.36 [1.30, 1.47]

Notes: CI = confidence interval. *Relative risks for injuries were estimated using data from the Fatality Analysis Reporting System, the National Highway Traffic Safety Administration, the U.S. Centers for Disease Control and Prevention's National Violent Death Reporting System (NVDRS), and a systematic review and meta-analysis by Alpert et al. (2022).

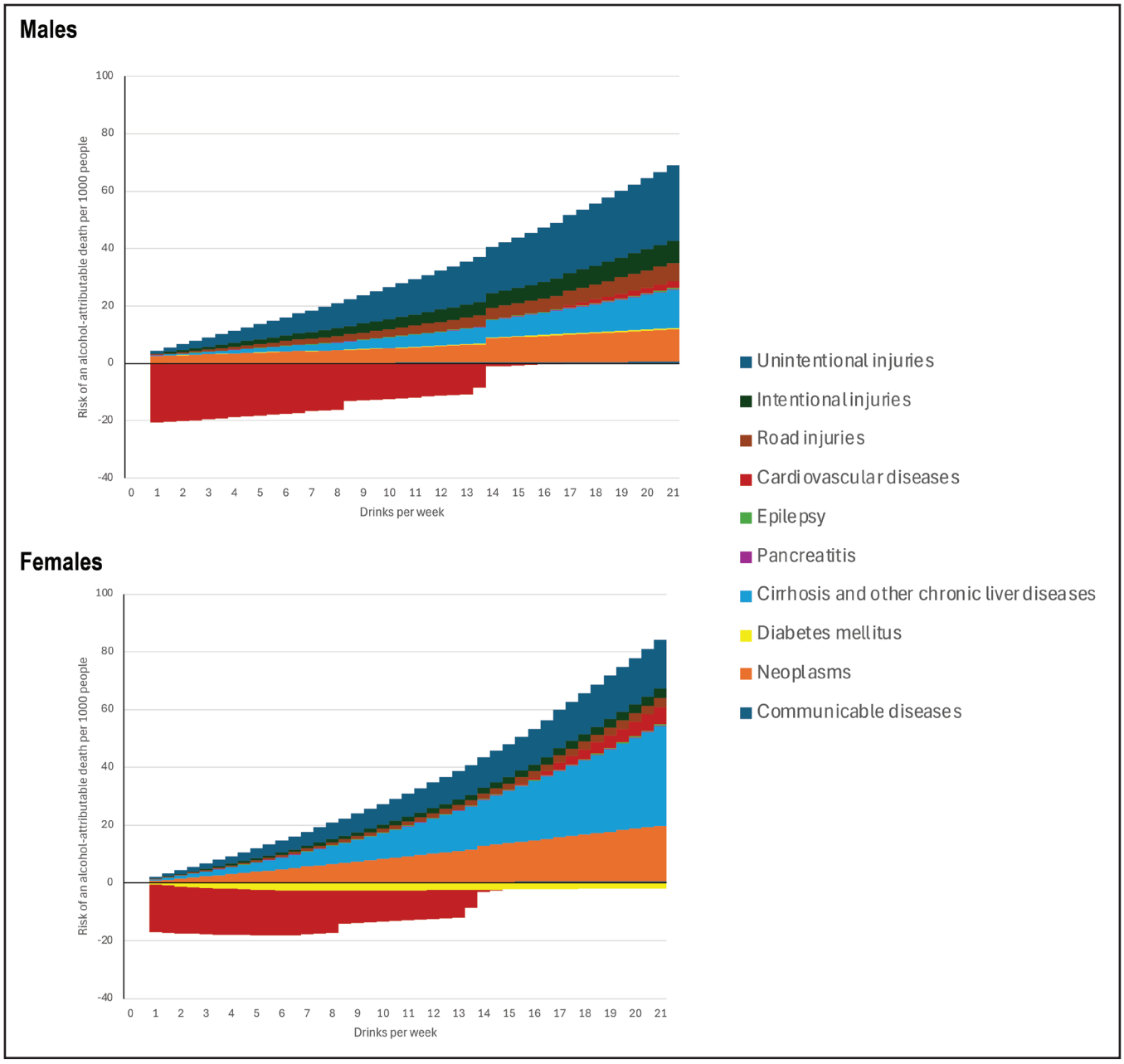

FIGURE 3. Lifetime risk of an alcohol-attributable death among males and females by cause for differing levels of alcohol consumption

2024). A systematic review by Kilian et al. estimated that in the United States, 5.5% (95% CI [4.4%, 6.7%]) of males and 3.4% (95% CI [2.6%, 4.2%]) of females had experienced physical violence attributable to someone else's drinking. Findings from the 2010–2012 National Intimate Partner and Sexual Violence Survey further underscore the broader harms associated with others' alcohol use: Sexual violence linked to another person's drinking affected an estimated 4.5% (95% CI [4.2%, 4.8%]) of males and 10.1% (95% CI [9.7%, 10.4%]) of females, whereas intimate partner violence resulting from another person's drinking was reported by 3.6% (95% CI [3.4%, 2.8%]) of respondents (Basile et al., 2021).

Discussion

The Alcohol Intake and Health Study quantifies the health risks associated with varying levels of alcohol consumption in the United States. Applying condition-specific relative risks to U.S. mortality data, it provides a precise, up-to-date estimate of alcohol-attributable harm in the United States, compared with all-cause mortality studies. There was no

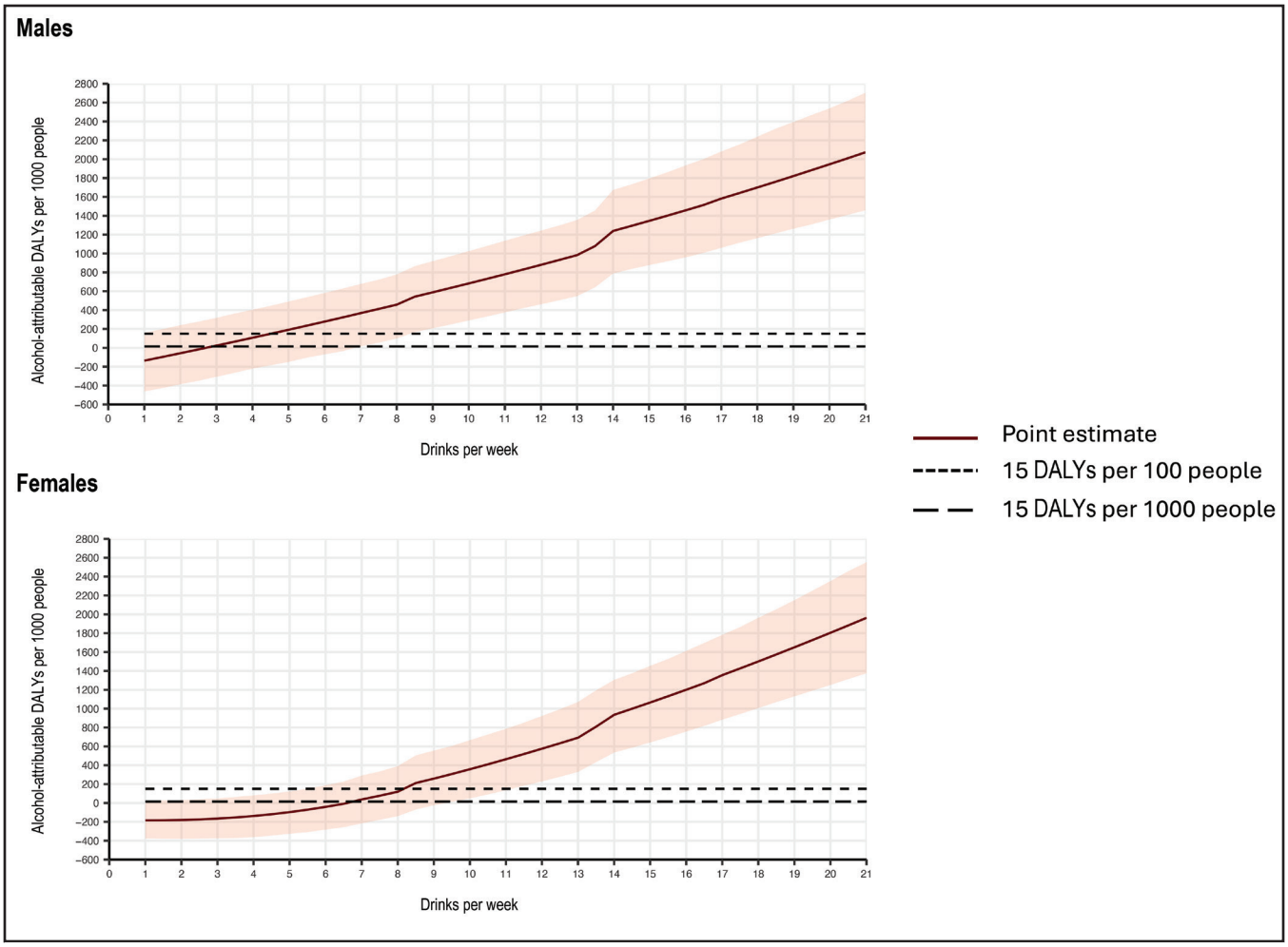

FIGURE 4. Lifetime risk of alcohol-attributable disability-adjusted life years (DALYs) lost among males and females for differing levels of alcohol consumption

protective net effect of alcohol observed at any level of alcohol consumption. Our findings demonstrate that both males and females who consume more than 7 standard drinks per week face a lifetime risk of alcohol-attributable death of at least 1 in 1,000. This risk rises sharply to 1 in 100 when consumption exceeds 8.5 drinks per week. For males who drink 14 drinks weekly, equivalent to the upper limit in the 2020–2025 U.S. Dietary Guidelines, the risk of an alcohol-related death is approximately 1 in 25. Even moderate levels of alcohol use (e.g., 1 drink per day) are associated with elevated risks of death from liver cirrhosis, esophageal and oral cancers, and injury-related deaths. Among females, these risks extend to liver cancer, although some protective effect against diabetes mellitus was observed. However, these risks are not evenly distributed across individuals, as biological and contextual factors can substantially modify outcomes.

Although these thresholds provide context, they do not inherently justify the health risks associated with alcohol use, particularly for guidance intended to optimize health in the general population (Rehm et al., 2014). They also fail to consider the interaction with other hazards. For example, alcohol also contributes substantially to drug overdose deaths, with alcohol–opioid poisonings rising 4.6-fold from 2000 to 2019 (Buckley et al., 2022). As drug use continues to increase in the United States, ignoring the compounding risks of alcohol when used in combination with other substances presents a crucial gap in evaluating what constitutes acceptable risk.

In addition to the effects of average consumption, the narrative review highlights that drinking patterns also affect outcomes. Although some studies suggest a protective effect of low-level alcohol consumption on ischemic stroke, this potential benefit could be negated by episodes of high per-occasion drinking, even if infrequent (Roerecke & Rehm, 2014). Although older adults have increased sensitivity to alcohol and vulnerability to serious medical conditions and falls (Satre et al., 2025), our results indicate that younger adults are particularly at risk due to more frequent binge drinking episodes, which contribute to elevated rates of alcohol-related injuries in this age group (Shuey et al., 2025). The observation that drinking patterns, in addition to total

alcohol consumption, influence mortality risk underscores the importance of addressing high-intensity drinking behaviors, especially in contexts where diminished judgment or coordination increases the likelihood of injury.

Importantly, the BAC thresholds used to attribute injuries in our burden estimates (≥.08% for motor vehicle crashes and ≥.10% for other injuries) do not capture the meaningful impairment or injury risk that can occur at levels below these thresholds. For instance, evidence from controlled laboratory, simulator, and closed-course studies shows that skills relevant to safe driving can be impaired at BACs between .02% and .04% (Moskowitz & Florentino, 2000), and that poorer driving performance is observed in simulated driving studies and closed-course road studies around .05% (Schnabel, 2012; Verster & Ramaekers, 2009). Further, evidence indicates that consuming alcohol more slowly and with food is associated with lower peak BACs (Forney & Hughes, 1963; Jones & Jönsson, 1994). Accordingly, prevention messaging should emphasize that injury risk increases along a continuum and can be elevated even below legal intoxication thresholds, particularly with faster drinking, drinking on an empty stomach, and higher per-occasion intake.

Limitations

This study did not identify systematic reviews on the impacts of alcohol consumption on an increased risk of conditions such as HIV or other sexually transmitted diseases, cervical cancer, depression, or alcohol use disorder, and consequently, does not quantify alcohol's impact on these outcomes. In addition, the analysis was limited to conditions with established causal links to alcohol consumption. As scientific knowledge evolves, some conditions previously thought to be causally linked to alcohol may be reassessed, as seen with gastric cancer. Similarly, new evidence may establish causal relationships for conditions not currently considered alcohol attributable.

Alcohol consumption in this study was estimated using population surveys that were adjusted to match alcohol sales data, and adjusted RRs drawn from epidemiologic studies that rely on self-reported intake. As a result, the estimates reflect average alcohol consumption patterns at the population level rather than the unique variability associated with individual drinking behaviors. As such, the risk estimates produced in this study should be interpreted with the understanding that they are derived from population-level data and are not individual risk estimates.

There are also limitations associated with the use of lifetime abstainers as the reference group in alcohol risk estimation. Evidence suggests that individuals who abstain from alcohol from early adulthood tend to have poorer baseline health profiles than their drinking peers, which may bias this group toward ill health independent of alcohol exposure (Stockwell et al., 2024). In addition, although many epidemiologic studies report excluding former drinkers, definitions of lifetime abstention often rely on self-report. They may inadvertently include "sick quitters" as a result of loose or inconsistent criteria. Since this study relied on RR estimates from the existing literature, it inherits any reference-group bias present in the original studies, including variation in how lifetime abstainers were defined (e.g., including individuals who reported minimal past drinking or infrequent lifetime use). These biases may lead to inflated baseline risks among abstainers, which in turn could attenuate estimated RRs associated with alcohol consumption at all levels. As a result, alcohol-attributable risk estimates presented here may be conservative.

This study modeled alcohol-attributable risk using cause-specific mortality and morbidity rather than all-cause mortality. Although this approach allows risks to be estimated using condition-specific evidence and avoids some well-documented biases of all-cause mortality analyses, it also has limitations. Cause-specific estimates depend on the accurate classification of causes of death and may not fully capture indirect or interacting effects of alcohol across conditions. Further, results are sensitive to assumptions regarding exposure distributions and RR functions. As noted by Rehm (2019) and the National Academies of Sciences, Engineering, and Medicine (2025), both cause-specific and all-cause mortality approaches involve trade-offs, and neither provides a definitive estimate of alcohol-related risk. Accordingly, the findings should be interpreted in light of these methodological constraints (National Academies of Sciences, Engineering, and Medicine, 2025; Rehm, 2019).

Multiple individual and contextual factors modulate the health risks associated with alcohol, including smoking, diet, physical activity, obesity, hepatitis and other infections, and genetic predispositions (e.g., ALDH2 variants). Although some factors were controlled for in the systematic reviews used, others remained unaddressed, introducing variability in alcohol-related outcomes. For instance, individuals with hepatitis C infection may face elevated risks from even low levels of alcohol consumption (Llamosas-Falcón et al., 2021). In addition, this study limited its comparison to biological sex and did not explore differences by gender expression.

A further limitation is that, whereas this review examined average alcohol consumption and per-occasion drinking, it did not comprehensively synthesize other drinking pattern dimensions, such as frequency, beverage type (e.g., beer, wine, spirits, or other alcohol), consumption with food, or speed of drinking. These factors may independently modify alcohol-related health risks, but their systematic evaluation was beyond the scope of this review.

This study is also limited based on the sources used for modeling the risk relationships between alcohol consumption and disease and injury risk. The RRs were derived from meta-analyses of observational studies, which combine results from studies with differing designs, exposure

and outcome definitions, statistical analyses, and controlled confounders. This heterogeneity, along with potential biases inherent to observational research, may introduce bias and limit analyses. In addition, several risk estimates in this study are based on meta-analyses of observational studies that report protective associations between low levels of alcohol consumption and outcomes such as ischemic heart disease, ischemic stroke, and diabetes, particularly among females. The magnitude of these protective effects has varied substantially across studies and over time, ranging from no protection to protection at relatively high levels of consumption. Because ischemic heart disease is highly prevalent and a leading cause of death, assumptions regarding the presence and magnitude of protective effects have a substantial influence on both lifetime risk estimates and estimates of alcohol-attributable burden. These protective associations have been questioned in the literature, including by Mendelian randomization studies, which generally do not support a clear protective effect of low-level alcohol consumption (Biddinger et al., 2022; Millwood et al., 2023). However, systematic reviews of Mendelian randomization studies conclude that this body of evidence has limitations due to substantial heterogeneity in study design and reliance on genetic instruments associated with multiple dimensions of alcohol use that may confer opposing risks (Bouajila et al., 2024; Rehm, 2019; van de Luitgaarden et al., 2022).

Furthermore, observational studies may underestimate alcohol-related risk because of residual confounding, selection bias, and misclassification of former drinkers (Naimi & Chikritzhs, 2025). Taken together with residual underestimation of alcohol consumption and the possibility that some alcohol-attributable conditions remain unidentified, these factors suggest that the risk and burden estimates presented here are likely conservative. In addition, observational cohort studies often include healthier and more socioeconomically advantaged participants, which may further limit generalizability to more diverse populations.

For injury outcomes, risk estimates vary across studies and are influenced by heterogeneity in study designs and injury definitions (e.g., some studies use different BAC thresholds). Injury-related mortality estimates may also be affected by how causes of death are recorded, particularly for deaths involving multiple contributing factors. In addition, many injury studies rely on observational data of varying quality, which limits precision in estimating the size of alcohol-attributable injury burden, even though the overall relationship between alcohol use and injury risk is well established.

Finally, the interpretation of lifetime risk estimates also depends on how uncertainty around these estimates is handled. Risk thresholds (e.g., 1 in 1,000 or 1 in 100 lifetime risk) may be crossed either by the point estimate or only when the associated 95% CIs are considered. In this study, we primarily discuss results based on point estimates that cross these thresholds, which provide a conservative and consistent basis for interpretation. However, this approach may identify elevated risk at consumption levels where statistical uncertainty is present. Alternatively, focusing only on thresholds crossed by the 95% CIs would yield more conservative estimates but may miss meaningful increases in risk at lower consumption levels. Readers should therefore consider both the point estimates and their associated CIs when interpreting the risk thresholds presented in this study.

Public health implications

These findings have important implications for public health policy and prevention. This study's evidence indicates that many health risks increase at a consumption level of 1 drink per day. Importantly, health risks are not uniform and vary substantially by drinking patterns, individual characteristics, and context, meaning that some people may experience harm at levels of consumption lower than those reported in this study. Public health guidance should aim to capture this nuance by emphasizing graduated risk and informed choice alongside a potential population-level threshold of 1 drink per day.

Some harms from alcohol consumption at such low levels are not well known by the U.S. public. For example, a 2025 survey of U.S. adults found that 56% were aware that alcohol is a cause of cancer (Annenberg School for Communication, 2025). Accordingly, large-scale education and information-based interventions (e.g., alcohol health warning labeling) are recommended strategies for raising public awareness of alcohol's health risks and of alcohol guidance (Schoueri-Mychasiw et al., 2020; Zuckermann et al., 2024).

The reduction in alcohol-related harms requires population-level reductions in consumption, which are best achieved by implementing effective alcohol control policies, particularly in the areas of pricing and physical availability (e.g., alcohol taxes, limits on hours of sale). However, effective prevention efforts must also account for demographic variability, the social and cultural contexts of drinking, and high-risk patterns of use. Accordingly, public health policy and programming should prioritize the risks associated with drinking patterns, particularly binge drinking, rather than focusing exclusively on average intake. Possible policies for addressing drinking patterns in addition to average intake include routine alcohol screening in community health settings (Derges et al., 2017). Furthermore, strengthening surveillance of average alcohol intake and consumption patterns, especially across demographic groups and contexts, will support the development of more targeted and equitable public health policies.

Conclusions

The Alcohol Intake and Health Study provides the most comprehensive U.S. estimates to date of lifetime risks of

alcohol-attributable mortality and morbidity, showing that what might be considered socially moderate levels of consumption increase the risk of premature death and disability. No protective effect of drinking was observed even at low levels, and a lifetime risk of 1 alcohol-attributable death per 1,000 people occurred at roughly 7 drinks per week for both males and females, with risks rising sharply beyond this level. Drinking patterns also shape health risks. High per-occasion consumption further magnified risks of breast cancer, cardiovascular diseases, and injuries. These findings underscore the need to revise the U.S. Drinking Guidelines to reflect cumulative and per-occasion risks and promote awareness of alcohol's health impacts. The upper limit of the 2020–2025 U.S. Dietary Guidelines for males (up to 2 drinks per day) is associated with a considerably elevated risk of alcohol-caused mortality. As such, the current results support changing the 2025–2030 U.S. Dietary Guidelines on alcohol to recommend that current adult drinkers consume no more than 1 drink per day.

Conflict-of-Interest Statement

The authors declare no competing interests.

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