Health Information and Quality Authority

An tÚdarás Um Fhaisnéis agus Cáilíocht Sláinte

Modelling Alcohol-Attributable Mortality Risks and Hospital Admission Rates in Ireland

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Organisations and individuals that assisted HIQA in providing information, in writing or through meetings, included:

- The Central Statistics Office, who facilitated access to mortality data.
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Conflicts of interest

None reported.

Table of contents

List of tables

List of figures

Foreword

Research has shown that alcohol consumption is a leading risk factor for death and disability worldwide. In Ireland in 2024, 73% of people aged 15 years or older reported consuming alcohol in the preceding 12 months. The World Health Organization has identified alcohol consumption as contributing to a wide range of health risks, with higher levels of consumption generally associated with greater risk of harm. Reducing alcohol-related harm remains an important public health priority.

Low-risk alcohol guidelines provide information on levels of alcohol consumption associated with a lower risk to health outcomes, and on circumstances in which alcohol should be avoided. The current Irish guidelines were last revised in 2015. Since then, new evidence has emerged and international practice has advanced in using quantitative modelling to inform guideline development.

At the request of the Department of Health, HIQA undertook this analysis to estimate the lifetime risk of alcohol-attributable mortality and years of life lost, and the relative rate of alcohol-attributable hospital admissions, in Ireland. This report was conducted alongside a systematic review of associations between alcohol consumption and mental health outcomes. Together, these reports provide evidence to support the update of the Irish low-risk alcohol guidelines.

Work on this report was undertaken by an Evaluation Team from HIQA's HTA Directorate, supported by a multidisciplinary Expert Advisory Group. HIQA would like to thank the members of the Evaluation Team, the Expert Advisory Group and all others who contributed to the preparation of this report.

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Deputy Chief Executive & Director of Health Technology Assessment

Health Information and Quality Authority

Key findings and advice to the Minister for Health

Following a request from the Department of Health, the Health Information and Quality Authority (HIQA) agreed to model the lifetime risk of alcohol-attributable mortality and the relative rate of hospital admissions in Ireland. This report uses Irish mortality, hospital admission, and alcohol consumption data to characterise the risks associated with different levels and patterns of alcohol consumption. The findings are intended to provide a technical evidence base to inform an update to the Irish low-risk alcohol guidelines.

- Between 2022 and 2024, alcohol was estimated to account for an average of 1,420 deaths per year, representing 4.0% of all deaths (6.0% in males and 1.9% in females). Alcohol was also estimated to account for 27,066 hospital admissions per year. For both sexes, harm is not restricted to the heaviest drinkers as over half of alcohol-attributable deaths occurred among the 90% of drinkers with the lowest levels of consumption (59% in males and 54% in females). These alcohol-attributable deaths are population-level modelled estimates of deaths that would not be expected to occur in the absence of alcohol consumption.
- The lifetime risk of alcohol-attributable premature death (that is, death occurring before 75 years), overall death, and years of life lost increased with higher average daily consumption for both males and females.
- Small non-significant reductions in lifetime risk of premature death were observed at the lowest levels of alcohol consumption among males, although these effects are not consistent across outcomes or sexes.
- The relative rate of alcohol-attributable hospital admissions due to acute and chronic alcohol-attributable causes also increased with higher levels of alcohol consumption. Patterns were similar for males and females.
- Results were interpreted using lifetime risk thresholds of 1 in 1,000 and 1 in 100 for mortality outcomes, consistent with thresholds used in comparable international analyses. For years of life lost (YLL), results are expressed relative to risk thresholds of 17.5 YLL attributable to alcohol per 1,000 and per 100 lifetimes, reflecting an average of 17.5 YLL per alcohol-attributable death in Ireland, based on 2023 estimates from the Institute for Health Metrics and Evaluation Global Burden of Disease study.
- The estimated levels of average daily alcohol consumption associated with these thresholds are shown below. Note that, in Ireland, there are 10 grams of pure alcohol in a standard drink, which is approximately half a pint of

normal-strength beer, stout or cider, or a small glass of wine (around 100 ml).

- Premature deaths
  - 1 in 1,000 risk of alcohol causing premature death
    - Males: 5 g/day (95% Uncertainty intervals (UI): <1-9)
    - Females: 4 g/day (95% UI: 1-5)
  - 1 in 100 risk
    - Males: 18 g/day (95% UI: 10-23)
    - Females: 16 g/day (95% UI: 13-18)
- Deaths
  - 1 in 1,000 risk
    - Males: 6 g/day (95% UI: <1-14)
    - Females: 6 g/day (95% UI: <1-11)
  - 1 in 100 risk
    - Males: 13 g/day (95% UI: 1-20)
    - Females: 12 g/day (95% UI: 1-18)
- Years of life lost
  - 17.5 per 1,000 lifetimes
    - Males: 3 g/day (95% UI: <1-7)
    - Females: 3 g/day (95% UI: <1-4)
  - 17.5 per 100 lifetimes
    - Males: 11 g/day (95% UI: 6-15)
    - Females: 11 g/day (95% UI: 9-12)

- Based on Healthy Ireland Survey data, 88% of current drinkers drink on two or fewer days (mean=1.2; median=1) a week. A scenario analysis examined how alternative assumptions about the distribution of weekly alcohol consumption across the number of weekly drinking days affected the prevalence of chronic binge drinking and the associated risk of deaths due to injury. Assuming that the same weekly volume of alcohol was consumed over fewer drinking days increased the proportion of drinkers exceeding the chronic binge drinking threshold and, in turn, increased population-level annual relative risks for deaths due to injury.

- The analysis is restricted to health outcomes with established causal relationships and available dose-response functions from meta-analyses that met the required quality criteria. Harms to others and broader social or economic impacts were not quantified or included within the model. Consequently, these estimates do not fully represent the total potential burden of alcohol use in Ireland.

- Developing low-risk alcohol guidelines requires translating a continuous relationship between alcohol consumption and health outcomes into discrete consumption categories defined by specified risk thresholds. Selecting these thresholds requires judgement regarding how population-level risk is summarised and communicated. This modelling provides a quantitative evidence base to inform that judgement.

Arising from the findings of this assessment, HIQA advises the Minister for Health as follows:

- This analysis provides quantitative estimates of alcohol-attributable mortality risk and hospital admission rates in Ireland to inform an update to the national low-risk alcohol guidelines.
- Lifetime risk of alcohol-attributable mortality generally increased with increasing average alcohol consumption. This overall pattern was broadly consistent across outcomes, sexes, and sensitivity analyses.
- Lifetime risk of alcohol-attributable premature mortality was estimated to exceed benchmarks commonly used in international guideline development at lower consumption levels than those implied by the current Irish low-risk alcohol guidelines. If these benchmarks are used, the consumption levels currently described as low risk in Irish guidance may warrant reconsideration.
- Small, non-significant reductions in lifetime risk of premature death were observed at the lowest levels of alcohol consumption among males only. As these reductions were not consistent across outcomes or sexes, the modelling did not identify any level of alcohol consumption above zero at which alcohol-attributable mortality risk was consistently absent. In updating the guidelines, presenting alcohol-related risk as a continuum rather than as a binary threshold should be considered, this would be more consistent with the underlying evidence.
- Although higher levels of consumption were associated with the highest individual risk, most estimated alcohol-attributable deaths occurred among the 90% of drinkers with lower levels of consumption. This supports guidance that communicates this risk to the wider drinking population, not only among high-risk drinkers.

- Estimated differences in risk between males and females were small, with substantial overlap in uncertainty intervals. The modelling therefore does not provide a strong basis for different sex-specific thresholds.
- Scenario analyses indicated that drinking pattern influences risk independently of total volume consumed. Consuming the same weekly volume over fewer drinking occasions was associated with higher estimated injury mortality risk. Guidance based solely on weekly volume may therefore not fully capture alcohol-related risk.
- The modelling does not consider harms to others from alcohol consumption, as well as several potentially alcohol-related conditions for which suitable risk functions were not identified. The estimates are therefore conservative in relation to the overall health and societal burden associated with alcohol use. The modelling also does not capture individual-level variation in risk and should therefore be interpreted as providing population-level rather than individual-level estimates.
- The selection of specific consumption limits or benchmark risk levels for updated guidelines cannot be determined by modelling alone. These decisions require judgement by guideline developers, informed by the evidence presented in this analysis, international practice, remaining uncertainty, and the broader societal impacts of alcohol consumption. In this context, lifetime risk thresholds commonly used in international guideline development, including 1 in 1,000 and 1 in 100, may provide useful reference points for informing where different levels of risk might reasonably be communicated.

Executive Summary

Background and Purpose

Research has shown that alcohol consumption is a leading risk factor for death and disability globally. In 2024, 73% of people aged 15 years or older in Ireland reported consuming alcohol in the previous year. In order to make informed decisions, individuals should have access to accurate information on the health risks associated with alcohol consumption.

The current Irish low-risk alcohol guidelines were last revised in 2015. Since then, new evidence has emerged and quantitative modelling methods have enabled more quantitative estimation of the health risks associated with different levels of alcohol consumption.

At the request of the Department of Health, HIQA undertook a modelling study to estimate:

- the lifetime risk of alcohol-attributable mortality, including premature mortality (that is, death occurring at an age of up to 75 years).
- the lifetime risk of alcohol-attributable years of life lost.
- the relative rate of alcohol-attributable hospital admissions.

The objective of this work was to provide a technical evidence base to inform an update to the low-risk alcohol guidelines.

Methods

A comparative risk assessment modelling framework was used to estimate alcohol-attributable mortality and hospital admissions in Ireland. The model combined Irish data on alcohol consumption, mortality, hospital admissions and population structure with condition-specific dose-response risk functions derived from published meta-analyses.

Alcohol consumption patterns were estimated using the 2024 Healthy Ireland Survey and calibrated to recorded alcohol sales data from the Office of the Revenue Commissioners. Mortality data (2022 to 2024) were obtained from the Central Statistics Office, and hospital admission data (2022 to 2024) from the Hospital In-Patient Enquiry (HIPE) system. Population estimates were sourced from the Central Statistics Office (2024). Dose-response risk functions used in the modelling were drawn from published meta-analyses identified through a systematic review conducted by HIQA, which updated a prior review by the Canadian Centre on Substance Use and Addiction. Where a more recent meta-analysis that met the stated inclusion and quality criteria was available for a condition, it replaced the one used in the Canadian Centre on Substance Use and Addiction model.

Alcohol-attributable conditions included both wholly and partially attributable acute and chronic diseases and injuries with causal relationships to alcohol consumption as determined by the World Health Organization's *Global Status Report on Alcohol and Health and Treatment of Substance Use Disorders*.

The modelling was conducted in four stages. First, age- and sex-specific distributions of alcohol consumption were estimated. Second, baseline mortality risk was derived under a scenario in which alcohol consumption was set to zero. Third, cause-specific relative risks were applied to estimate alcohol-attributable increases in annual mortality risk at different levels of consumption. Finally, a life-course model simulated survival over time to estimate the cumulative lifetime risk of alcohol-attributable premature death, death, and years of life lost.

For hospital admissions, the same framework was applied. However, results are expressed as annual relative rates of admission for acute and chronic alcohol-related conditions rather than lifetime probability, reflecting that hospital data capture discharge events rather than unique individuals.

Uncertainty intervals were estimated using Monte Carlo simulation, drawing relative risks from statistical distributions based on published confidence intervals. Sensitivity and scenario analyses examined the impact of alternative assumptions regarding alcohol consumption levels and drinking patterns.

Key Findings

Key findings are presented in four sections. First, estimated alcohol-attributable mortality and hospital admissions are presented to describe the estimated burden of alcohol use in Ireland. Second, the primary analyses describe how alcohol-attributable risk varies across levels of average alcohol consumption and identify consumption levels associated with selected lifetime risk thresholds. Third, scenario analyses examine the influence of drinking pattern on injury mortality risk. Fourth, sensitivity analyses examine the influence of selected modelling assumptions on the estimated lifetime risk of alcohol-attributable mortality.

Estimated alcohol-attributable mortality and hospital admissions, 2022-2024

Between 2022 and 2024, alcohol was estimated to account for an average of 1,420 deaths per year, representing 4.0% of all deaths (6.0% in males and 1.9% in females). Alcohol was also estimated to account for 27,066 hospital admissions per year.

Most (78%) alcohol-attributable deaths were due to chronic conditions, while acute conditions accounted for most (51%) alcohol-attributable hospital admissions.

Among males, the leading causes of alcohol-attributable deaths were cancers (29%), injuries (24%), digestive diseases (20%) and cardiovascular diseases (17%). Among females, the leading causes were cancers (37%), digestive diseases (35%), injuries (14%), and cardiovascular diseases (7%).

For both sexes, more than half of alcohol-attributable deaths (59% in males and 54% in females) occurred among the 90% of drinkers with the lowest levels of consumption. This reflects the prevention paradox whereby people at lower levels of individual risk collectively account for more harm than the smaller group of heavier drinkers.

Primary analyses

Across the range of alcohol consumption examined, the lifetime risk of alcohol-attributable premature death, overall death, and years of life lost increased with higher average daily consumption for both males and females.

Small reductions in risk were observed at the lowest levels of alcohol consumption for certain outcomes, although these effects are not consistent across all outcomes or sexes.

The relative rate of alcohol-attributable hospital admissions due to acute and chronic alcohol-attributable causes also increased with higher levels of alcohol consumption.

Overall, risk patterns were similar for males and females.

To support interpretation of these risk curves, estimated consumption levels were identified at which selected lifetime risk thresholds were met or exceeded. Results were interpreted using lifetime risk thresholds of 1 in 1,000 and 1 in 100 for mortality outcomes, as these provide illustrative reference points spanning levels of lifetime risk that have been used in comparable international analyses.

The estimated levels of average daily alcohol consumption associated with these thresholds are shown below. Note that, in Ireland, there are ten grams of pure alcohol in approximately half a pint of normal-strength beer, stout or cider, or a small glass of wine (around 100 ml).

Outcome	Risk threshold	Males, g/day (95% uncertainty interval)	Females, g/day (95% uncertainty interval)
Premature deaths	1 in 1,000	5 (<1 to 9)	4 (1 to 5)
	1 in 100	18 (10 to 23)	16 (13 to 18)

Deaths	1 in 1,000	6 (<1 to 14)	6 (<1 to 11)
	1 in 100	13 (1 to 20)	12 (1 to 18)
Years of life lost	17.5 per 1,000 lifetimes	3 (<1 to 7)	3 (<1 to 4)
	17.5 per 100 lifetimes	11 (6 to 15)	11 (9 to 12)

Note: Point estimates are the lowest whole-gram consumption level at which the modelled risk met or exceeded the risk threshold. Uncertainty intervals are simulation-derived percentiles and are not necessarily symmetric around the point estimate. Interval limits were rounded outwards to the nearest whole gram.

Scenario analyses

Based on Healthy Ireland Survey data, 88% of the population drinks alcohol on two or fewer days (mean=1.2; median=1) a week. A scenario analysis examined how alternative assumptions about the distribution of weekly alcohol consumption across drinking days affected the prevalence of chronic binge drinking and the associated risk of injury mortality.

Assuming that the same weekly volume of alcohol was consumed over fewer drinking days increased the proportion of drinkers exceeding the chronic binge drinking threshold and, in turn, increased population-level annual relative risks for deaths due to injury. For example, in a scenario that assumed two drinking days per week, at 50g/day, injury mortality risk increased by 53.4% for females and 20.5% for males compared to the base-case scenario (which assumed consumption spread over seven days). This pattern reflects the structure of the acute risk model, in which injury risks are influenced by the prevalence of binge drinking even when total alcohol consumption remains unchanged.

Sensitivity analyses

In a sensitivity analysis that applied thresholds below which wholly alcohol-attributable conditions were assumed to confer no risk, the estimated lifetime risk of premature mortality at low consumption levels was reduced. This indicates that the lower end of the lifetime-risk curve depends on whether these conditions are assumed to contribute risk at low consumption levels.

When survey alcohol consumption estimates were calibrated to 60% and 90% of recorded per-capita alcohol sales, compared with 80% in the primary analysis, the estimated lifetime risk of premature mortality decreased as coverage increased, reflecting the model structure rather than any protective effect of higher alcohol consumption coverage. This sensitivity analysis reflects sensitivity to the alcohol consumption adjustment factor due to the modelled decomposition of observed mortality into baseline and alcohol-attributable components.

Interpretation

This analysis provides a quantitative estimate of lifetime alcohol-attributable mortality risk and hospital admission rates in Ireland using nationally representative data within an established comparative risk assessment framework.

The analysis demonstrates a clear increase in lifetime mortality risk and hospital admission rates as average daily alcohol consumption increases. Reductions in risk at the lowest levels of alcohol consumption were small and inconsistent across outcomes and sexes.

Uncertainty intervals varied by outcome and threshold; however, lower bounds below 1 g/day indicate that, within the uncertainty range, even very low consumption may carry risk. Intervals for males and females overlapped substantially, so small differences in point estimates should be interpreted cautiously; intervals are plausible ranges rather than precise cut-offs.

The estimated locations of selected lifetime risk thresholds are broadly consistent with those reported in comparable international studies, with differences plausibly explained by variation in national consumption patterns, baseline health profiles, and methodological approaches.

The analysis is restricted to health outcomes with established causal relationships and available dose-response functions. Harms to others and broader social or economic impacts were not quantified. Consequently, the total potential burden of alcohol use in Ireland is not fully represented in these estimates.

Developing low-risk alcohol guidelines requires translating a continuous relationship between alcohol consumption and health outcomes into discrete consumption categories defined by specified risk thresholds. Selecting these thresholds requires a policy judgement regarding how population-level risk is summarised and communicated. The modelling provides a quantitative evidence base to inform that judgement.

Conclusion

This study estimated the lifetime risk of alcohol-attributable mortality and the relative rates of hospital admission in Ireland to inform an update of the low-risk alcohol guidelines.

Across the range of alcohol consumption examined, mortality risk and hospital admission rates increase with higher average daily consumption. Small risk reductions are observed at the lowest alcohol consumption levels for certain mortality outcomes, though these effects are not consistent across all outcomes or sexes. Concentrating a fixed weekly amount of alcohol consumption into fewer drinking days increased the estimated acute risk for both males and females.

For premature mortality, the estimated lifetime risk is approximately 1 in 1,000 at around 5 g/day and 1 in 100 at around 18 g/day for men, and approximately 1 in 1,000 at around 4 g/day and 1 in 100 at around 16 g/day for women.

Although subject to uncertainty and restricted to health outcomes experienced by the drinker, these estimates provide a transparent quantitative assessment of alcohol-attributable risk in Ireland.

Plain language summary

Research has shown that drinking alcohol increases the risk of health problems, including injuries, cancers, digestive diseases, heart disease, and other serious conditions that can lead to death or hospital admission. At the request of the Department of Health, HIQA carried out this work to help inform an update of the national low-risk alcohol guidelines, which were last revised in 2015.

In Ireland, there are 10 grams of pure alcohol in approximately half a pint of normal-strength beer, stout or cider, or in a small glass of wine of around 100ml.

We combined information on alcohol consumption, deaths, hospital admissions, and how alcohol affects the risk of particular health conditions. We got this data from the Healthy Ireland Survey, alcohol sales data from the Office of the Revenue Commissioners, death records from the Central Statistics Office, and hospital admission data from the Hospital In-Patient Enquiry system.

In this study, we estimated the lifetime risk of dying because of alcohol, including premature death (that is, dying before age 75). We also estimated how hospital admission rates change with different levels of drinking.

Between 2022 and 2024, alcohol was estimated to account for an average of 1,420 deaths every year in Ireland. This represented 4% of all deaths, including 6% of deaths in males and 1.9% of deaths in females. Alcohol was also estimated to account for 27,066 hospital admissions every year.

Most deaths caused by alcohol were due to long-term conditions. Among men, the leading causes of death were cancers, injuries, digestive diseases, and heart disease. Among women, the leading causes of death were cancers, digestive diseases, injuries, and heart disease. Short-term conditions, including injuries, made up a large share of alcohol-related hospital admissions.

Overall, alcohol-related risk generally increased as people drank more. The lifetime risk of death before age 75, death at any age, years of life lost, and the rate of hospital admissions all increased when people drank more alcohol per day.

We also examined alcohol consumption levels linked to lifetime risk levels of 1 in 1,000 and 1 in 100. A lifetime risk of 1 in 1,000 means that if 1,000 people drank at that average level throughout their lives, about one extra person would be expected to die before age 75 because of alcohol. A lifetime risk of 1 in 100 means that if 100 people drank at that average level throughout their lives, about one extra person would be expected to die before age 75 because of alcohol. These lifetime risk levels have been used in similar international studies.

For death before age 75, the 1 in 1,000 risk level was reached at about five grams of alcohol per day for men and four grams per day for women. The 1 in 100 risk level was reached at about 18 grams of alcohol per day for men and 16 grams per day for women.

A small reduction in the risk of death before age 75 was seen for men with the lowest levels of alcohol consumption. However, this reduction in the risk of death was not seen across all outcomes or in women. Overall, we did not find a level of drinking where there was no consistent risk of death because of alcohol.

A large share of deaths caused by alcohol happened among people drinking at lower individual levels, because this is a much larger group. This means that it is not only people who drink heavily or who are dependent on alcohol that are harmed by alcohol.

How alcohol is consumed also matters. People who drink the same amount each week may have different risks depending on whether they spread drinking across the week or whether they drink a lot in short amounts of time. In additional analyses, drinking the same weekly amount over fewer days increased the estimated risk of dying from injuries.

The results are estimates, not exact cut-offs. The estimates for men and women were close enough that small differences between them should be interpreted cautiously.

The estimates are likely to underestimate the full impact of alcohol on health and society. The analysis only included health conditions where there is strong evidence that alcohol can be a cause and where enough information was available to estimate the risk. It did not include all alcohol-related conditions, harms to others, or wider harms to families, communities, and society.

Despite these limitations, the findings provide important evidence to inform an update of the low-risk alcohol guidelines. This report is one part of the wider evidence base that will be considered as part of that process. It provides evidence to help inform how alcohol-related risk should be described and communicated. Decisions about guideline limits involve policy judgement, including how much risk is considered acceptable and how this should be explained to the public.

List of abbreviations used in this report

AAF	alcohol attributable fraction
CCSA	Canadian Centre on Substance Use and Addiction
CSO	Central Statistics Office
HIPE	Hospital In-Patient Enquiry
HIQA	Health Information and Quality Authority
HSE	Health Service Executive
ICD-10	International Classification of Diseases, 10th Revision
UI	uncertainty interval
RoB	risk of bias
YLL	years of life lost

1 Background

Alcohol consumption is a leading risk factor for death and disability worldwide, with higher levels of alcohol consumption associated with a greater burden of disease.(1) In Ireland in 2024, 73% of people aged 15 years or older reported having drunk alcohol in the preceding 12 months.(2) In order to make informed decisions, individuals should have access to accurate information on the health risks associated with alcohol consumption.(3, 4) Low-risk alcohol guidelines can provide this information, in addition to providing guidance on levels of alcohol consumption that are associated with lower levels of risk and when it is not safe to drink any alcohol. In providing this information, low-risk alcohol guidelines can be viewed as one tool in the portfolio of measures to reduce alcohol-related harm.(4)

The current Irish low-risk alcohol guidelines were last revised in 2015.(5) They recommend:

- consuming no more than 11 standard drinks (one standard drink contains 10g or 12.5ml of pure alcohol) per week for women, and 17 standard drinks per week for men
- spreading drinks out over the week
- having two to three alcohol-free days per week
- drinking no more than six standard drinks on any one occasion.

Guidance is also provided on when it is not safe to drink any alcohol, or when alcohol consumption is not recommended, such as:

- if aged under 18 years
- if pregnant or thinking about becoming pregnant
- if taking certain medicines or having a condition made worse by drinking
- before operating machinery
- before or while driving
- before doing anything risky or that requires skill
- before or during swimming
- before or while you're looking after children.

Since these guidelines were last updated, new evidence has emerged and international practice has advanced in terms of the methods used to inform low-risk alcohol guidelines. Historically, expert groups reviewed and summarised epidemiological evidence on the health outcomes associated with drinking different amounts of alcohol. Defining 'low risk' and formulating guidelines required informed but ultimately subjective decisions on methodological and technical factors. In practice, determining what constituted a low-risk level of drinking was a matter of expert opinion.(6)

It has been suggested that low-risk alcohol guidelines require a scientific basis that extends beyond individual or group judgements of risk.[7] Indeed, it has become increasingly common to use mathematical modelling to estimate the risk of death and hospitalisation from various causes that are associated with specific levels of alcohol consumption, using defined risk thresholds to inform what constitutes a low-risk level of drinking.[8-10] While expert judgment is still required with this approach, not least in defining these risk thresholds, the quantitative analysis provides valuable information and transparency to inform and justify these judgments.

Following a request from the Department of Health, the Health Information and Quality Authority (HIQA) agreed to model the lifetime risk of alcohol-attributable mortality and the relative rate of hospital admissions in Ireland. This report uses Irish mortality, hospital admission, and alcohol consumption data to characterise the risks associated with different levels and patterns of alcohol consumption. The findings are intended to provide a technical evidence base to inform an update to the low-risk alcohol guidelines.

2 Methods

This section describes the methods used to estimate the alcohol-attributable mortality risk and rates of hospital admission in Ireland at different levels of alcohol consumption. The analysis uses a comparative risk assessment modelling framework that combines Irish data on alcohol exposure, mortality, and hospital admissions with condition-specific risk relationships from the international literature. Data sources used to characterise alcohol consumption, health outcomes, and population demographics are described in Section 2.1. The analytical approach used to estimate alcohol-attributable risk is described in Section 2.2, including the life-course modelling of mortality, the estimation of hospital admission rates, and the treatment of uncertainty through scenario analyses and sensitivity analyses. Further detail is available in Appendix 1 and the protocol which was published on the HIQA website on 13 August 2025.(11)

One adaptation was made from the published protocol. The protocol specified that acute and chronic risks would be modelled separately, with acute risks estimated using self-reported data on the maximum number of drinks consumed on a single occasion in the past 12 months. Instead, a single integrated model was implemented in which both acute and chronic risks were estimated as functions of average weekly alcohol consumption, expressed in grams per day. This reflected both data availability and the objective of informing a single, internally consistent set of low-risk alcohol drinking guidelines. Within this framework, the influence of concentrated drinking patterns on acute risks was considered through a scenario analysis, in which a fixed weekly volume of alcohol was redistributed across different numbers of drinking days.

2.1 Data sources

2.1.1 Alcohol-attributable conditions

The inclusion of diseases and injuries in the modelling was based on three criteria: 1) they had to be causally related to alcohol use as determined by the World Health Organization for the recently published *Global Status Report on Alcohol and Health and Treatment of Substance Use Disorders*;(12) 2) a suitable dose-response risk curve had to be available (that is, a mathematical model describing how the risk of a disease or injury changes with different levels of alcohol consumption); and 3) alcohol-attributable death or hospitalisation had to be specifically measurable for the condition. The included health conditions and the sources of their associated risk estimates for calculating alcohol-attributable fractions (AAFs) are listed in Table 2.1. They are categorised into four groups, according to whether the condition is wholly or partially attributable to alcohol, and whether the relationship is primarily driven by chronic or acute alcohol consumption:

- **Partially alcohol-attributable chronic conditions**: Conditions that may occur without alcohol consumption but for which the risk increases with long-term (chronic) alcohol use.
- **Partially alcohol-attributable acute conditions:** Conditions that may occur without alcohol consumption but for which the risk increases with short-term (acute) alcohol use.
- **Wholly alcohol-attributable chronic conditions:** Conditions that occur only as a result of chronic alcohol use.
- **Wholly alcohol-attributable acute conditions:** Conditions that occur only as a result of acute alcohol use.

In some cases, conditions with acute clinical presentations are classified as chronic where the associated alcohol-attributable risk is primarily driven by long-term (chronic) alcohol consumption, for example acute pancreatitis.

Table 2.1 Alcohol-attributable health conditions included in the mathematical modelling

Health condition	ICD-10 codes	Risk function source[a]
Partially alcohol-attributable chronic conditions		
Atrial fibrillation and flutter	I48	Zhang et al. 2022[13]
Breast cancer	C50	Sohi et al. 2024[14]
Cirrhosis of the liver	K73, K74	Llamosas-Falcón et al. 2024;[15] Llamosas-Falcón et al. 2022[16]
Colorectal cancer	C18-C21	Vieira et al. 2017[17]
Diabetes mellitus	E11	Llamosas-Falcón et al. 2023[18]
Epilepsy	G40, G41	Samokhvalov et al. 2010[19]
Gout	M10	Wang et al. 2016[20]
Hypertensive heart disease	I10-I15	Liu et al. 2020[21]
Intracerebral haemorrhage	I61, I62, I62.9, I69.1-I69.298	Larsson et al. 2016[22]
Ischaemic heart disease	I20-I25	Zhao et al. 2017[23]
Ischaemic stroke	G45, G46, I63, I65, I66, I67.2-I67.8, I69.3, I69.4	Larsson et al. 2016[22]
Larynx cancer	C32	Bagnardi et al. 2015[24]
Liver cancer	C22	World Cancer Research Fund & American Institute for Cancer Research 2018[25]
Lower respiratory infections (pneumonia)	J09-J22, P23, U04	Samokhvalov et al. 2010[26]
Oropharyngeal cancer	C00-06, C09-10, C12-C14	Bagnardi et al. 2015[24]
Oesophageal cancer (only squamous cell carcinoma)	C15	Bagnardi et al. 2015[24]

Health condition	ICD-10 codes	Risk function source[a]
Pancreatitis (acute)	K85 (excl. K85.2)	Samokhvalov et al. 2015[27]
Pancreatitis (chronic)	K86 (excl. K86.0)	Samokhvalov et al. 2015[27]
Subarachnoid haemorrhage	I60, I67.0, I67.1, I69.0	Larsson et al. 2016[22]
Tuberculosis	A15-A19, B90	Imtiaz et al. 2017[28]
Partially alcohol-attributable acute conditions		
Accidental poisoning by exposure to noxious substances	T36-T50, T52-T65, X40-X44, X46-X49, Y10-Y14, Y16-Y19	Shield et al. 2020[29] based on World Health Organization, 2018[30]
Assault	X85-Y09, Y87.1	Shield et al. 2020[29] based on World Health Organization, 2018[30]
Drowning	W65-W74, Y21	Shield et al. 2020[29] based on World Health Organization, 2018[30]
Exposure to mechanical forces (including machinery injuries)	W20-W52	Shield et al. 2020[29] based on World Health Organization, 2018[30]
Fall injuries	W00-W19	Shield et al. 2020[29] based on World Health Organization, 2018[30]
Fire injuries	X00-X09, Y26	Shield et al. 2020[29] based on World Health Organization, 2018[30]
Intentional self-harm	X60-X64, X66-X84, Y87.0	Shield et al. 2020[29] based on World Health Organization, 2018[30]
Other intentional injuries	Y35	Shield et al. 2020[29] based on World Health Organization, 2018[30]

Health condition	ICD-10 codes	Risk function source[a]
Other unintentional injuries	W75-W99, X10-X33, Y20, Y22-Y25, Y27-Y29, Y31-Y34	Shield et al. 2020[29] based on World Health Organization, 2018[30]
Transport injuries (including road traffic injuries)	V01-V98, Y85.0	Shield et al. 2020[29] based on World Health Organization, 2018[30]
Wholly alcohol-attributable chronic conditions		
Alcohol use disorders	F10 (excl. F10.0)	Calibrated
Alcoholic cardiomyopathy	I42.6	Calibrated
Alcoholic gastritis	K29.2	Calibrated
Alcoholic liver diseases	K70	Calibrated
Alcoholic myopathy	G72.1	Calibrated
Alcoholic polyneuropathy	G62.1	Calibrated
Alcohol-induced acute pancreatitis	K85.2	Calibrated
Alcohol-induced chronic pancreatitis	K86.0	Calibrated
Degeneration of nervous system due to alcohol	G31.2	Calibrated
Wholly alcohol-attributable acute conditions		
Alcohol Poisoning (accidental)	T51 (X45, Y15)	Calibrated
Alcohol Poisoning (intentional)	T51 (X65)	Calibrated
Alcohol use disorders (acute intoxication)	F10.0	Calibrated
Alcohol-induced pseudo-Cushing's syndrome	E24.4	Calibrated
Excessive Blood Level of Alcohol	R78.0, Y90.0-Y90.9, Y91.0-Y91.9	Calibrated

Key: ICD-10 – International Classification of Diseases, 10th Revision.
[a]Absolute risk functions were calibrated for wholly alcohol-attributable conditions using the method described by Churchill et al.[31] to estimate population-level risk among drinkers in the absence of a comparison group.

2.1.2 Mortality and hospitalisation

Mortality and hospital admission data for the included alcohol-attributable health conditions were sourced for the years 2022 to 2024. The mean annual number of deaths and hospital admissions for each condition were calculated over this period.

Mortality data were obtained from the General Mortality Register via the Central Statistics Office (CSO).[32] These data include the total number of deaths registered in Ireland by age, sex, year, and cause of death, coded using the International Classification of Diseases, 10th Revision (ICD-10). Premature death was defined as a death occurring at an age of up to 75 years.[33] Years of life lost (YLL) attributable to alcohol were estimated using remaining life expectancy at each age, derived from CSO life tables.[34]

Hospital admission data were obtained from the Hospital In-Patient Enquiry (HIPE) scheme, which collects information on all inpatient and day-case discharges from public acute hospitals in Ireland.[35] For each discharge, HIPE records include demographic details and up to 30 clinical codes (ICD-10-Australian Modification), including both primary and secondary diagnoses. Each HIPE record corresponds to a single completed episode of care. Because HIPE records discharge events rather than unique individuals, multiple admissions for the same person appear as separate records. As a result, analyses based on these data estimate rates of hospital admission events in the population, rather than the probability that an individual will experience at least one admission. As HIPE captures discharges from public acute hospitals, it does not include emergency department presentations that do not result in admission and may not capture inpatient activity occurring outside the public acute hospital setting, such as some psychiatric inpatient admissions. The HIPE database is maintained by the Healthcare Pricing Office of the Health Service Executive (HSE).

2.1.3 Alcohol consumption

Survey data on the levels and patterns of alcohol consumption were obtained from the 10th wave of the Healthy Ireland Survey in 2024.[3] This is an interviewer-administered, nationally representative survey of people aged 15 years and older, commissioned by the Department of Health and conducted by Ipsos B&A. In 2024, 7,411 people participated in the survey, corresponding to a participation rate of 54%. Relevant variables included:

- age (years)
- sex (male or female)
- the average number of standard drinks (10g of pure alcohol) consumed per drinking day

- the average number of drinking days per week over the past 12 months
- the frequency of consuming six or more standard drinks on a single occasion in the past 12 months.

Respondents who reported drinking alcohol within the past 12 months were considered current drinkers. For each current drinker, average alcohol consumption in grams per day was estimated by multiplying the reported average number of standard drinks consumed per drinking day by the reported number of drinking days per week and by 10 grams, and dividing by seven. Episodic binge drinkers were defined as those reporting drinking six or more standard drinks on a single occasion at least once per month in the past 12 months. Chronic binge drinkers were those with a mean consumption of six or more standard drinks per day, equivalent to at least 42 standard drinks per week. In the analyses, survey responses were weighted using calibration weights provided with the dataset to adjust for differences between the survey sample and the population by sex, education, employment status and region.

Alcohol Products Tax data were obtained from the Office of the Revenue Commissioners for the same year (2024).[36] Alcohol Products Tax is a tax charged on alcohol and alcohol products when they are released for consumption in Ireland. These data reflect the volume of alcohol released for sale on the domestic market and serve as a proxy for alcohol consumption in Ireland. Figures are reported by beverage type and subcategories within these, defined by alcohol-by-volume ranges. Where volumes were reported in litres of pure alcohol (for example, certain beer and spirits products), these values were used directly. Where volumes were reported in litres of product (for example, most cider, perry, wine, intermediate beverages, and some other fermented products), they were converted to litres of pure alcohol using assumed average alcohol content for each subcategory. Although considered a reliable indicator of overall alcohol availability, these data exclude unrecorded consumption (for example, some home production or cross-border purchases) and may be affected by stockpiling or tourism, including both non-residents drinking in Ireland and Irish residents drinking abroad. Total recorded litres of pure alcohol were converted to grams of pure alcohol by multiplying litres by 1,000 to convert to millilitres and by 0.789 to convert millilitres of ethanol to grams.

2.1.4 Population demographics

Data on the age and sex structure of the Irish population in 2024 were obtained from the Central Statistics Office.[37] They were used to define the population at risk in each age-sex group. Age was grouped into seven categories: 15-24, 25-34, 35-44, 45-54, 55-64, 65-74, and 75 years and older. Sex was classified as male or female.

2.1.5 Risk estimates

Sources of condition-specific risk functions for calculating AAFs are listed in Table 2.1. Risk functions are mathematical relationships that estimate how the risk of developing a particular health condition changes at different levels of alcohol consumption. Separate functions for mortality and hospital admission, and for males and females, were used where available. These functions were applied across alcohol consumption levels by age and sex to estimate the association between alcohol consumption and each condition. Different methodological approaches were required, as described below:

- Partially alcohol-attributable chronic conditions: Relative risk functions were obtained from published meta-analyses of epidemiological studies identified in a systematic review which updated the 2022 Canadian Centre on Substance Use and Addiction (CCSA) review, *Update of Canada's Low-Risk Alcohol Drinking Guidelines: Evidence Review Technical Report* [38] (see the protocol for review methods and Section 3.1 for included reviews).[11]
- Partially alcohol-attributable acute conditions: Relative risk functions were obtained from Shield et al.,[29] based on functions from the World Health Organization's 2018 *Global status report on alcohol and health*.[30]
- Wholly alcohol-attributable acute and chronic conditions: Absolute risk functions were calibrated using the method described by Churchill et al.[31] to estimate population-level risk among drinkers in the absence of a comparison group.

2.2 Mathematical modelling approach

2.2.1 Primary analyses

A high-level overview of the mathematical modelling approach is provided here. The approach was adapted from comparative risk assessment methods for estimating alcohol-attributable harm that have been applied in modelling studies in Canada, the US, France, and across European countries as part of the Joint Action on Reducing Alcohol Related Harm project.[9, 39-41] Further technical detail, including relevant equations and life-course calculations, is provided in Appendix 1. The modelling was used to estimate both the overall alcohol-attributable burden of mortality and hospital admissions in Ireland and the lifetime risk of alcohol-attributable mortality across levels of alcohol consumption. The description below focuses on mortality, and the same modelling framework was applied to hospital admissions. However, for hospital admissions, annual relative rates of admission, rather than lifetime risk of admission, were estimated, reflecting that hospital admission data capture episodes of care rather than unique individuals.

Lifetime risk of alcohol-attributable mortality was estimated using a comparative risk assessment framework. The modelling combines information on alcohol consumption, mortality under a counterfactual in which alcohol consumption is set to zero, alcohol-related increases in mortality risk, and a life-course calculation that translates annual risks into estimates of lifetime alcohol-attributable mortality risk and YLL. Lifetime abstention from alcohol was used as the theoretical minimum risk exposure level, consistent with established comparative risk assessment practice.(9, 39-41) This choice defines the counterfactual reference against which alcohol-attributable risk is estimated and does not imply an assumption that lifetime abstention represents the level of alcohol consumption associated with the lowest overall risk of health loss. Evidence at low levels of consumption is uncertain, subject to bias and does not provide a sufficiently robust basis for defining a non-zero minimum risk exposure level.(42) The analysis was conducted in four main steps.

Step 1. Modelling alcohol consumption among people who drink

The first step characterises alcohol consumption among people who drink and how consumption varies within the population. Survey-based estimates of drinking prevalence by age and sex were combined with per-capita alcohol consumption data to construct age- and sex-specific distributions of average daily alcohol consumption among people who drink. Survey estimates were adjusted so that total estimated consumption corresponded to 80% of recorded alcohol sales, consistent with standard practice.(12, 29, 43, 44) For each age- and sex-specific group, a gamma distribution was used to represent variation in consumption.(43, 45) These distributions form the exposure inputs for subsequent estimation of alcohol-attributable risk.

Step 2. Estimating baseline mortality under a counterfactual in which alcohol consumption is set to zero

The second step estimates mortality risk under a counterfactual in which alcohol consumption is set to zero. This was done by estimating the proportion of observed mortality attributable to alcohol and removing this component from observed mortality counts, with the remaining deaths used to estimate cause-specific baseline mortality risk by age and sex. These estimates represent mortality risk under the zero-consumption counterfactual and serve as the reference risk within the modelling framework against which alcohol-related increases in risk are assessed.

Step 3. Estimating alcohol-attributable mortality risk

In the third step, alcohol-attributable increases in mortality risk were estimated by applying cause-specific relative risk relationships to baseline mortality risks. For each age, sex, and cause of death, the increase in mortality risk associated with alcohol consumption was estimated based on the level of alcohol consumption. Cause-specific alcohol-attributable risks were then summed to obtain total alcohol-attributable mortality risk by age and sex. These estimates represent annual alcohol-attributable mortality risks among individuals alive at the start of each age interval.

Step 4. Life-course estimation of lifetime mortality risk

The final step translates age-specific annual alcohol-attributable risks into life-course measures of mortality impact. A hypothetical birth cohort was followed forward in one-year age intervals, with survival updated at each age based on combined mortality risks from alcohol-attributable and other causes. Lifetime risk of alcohol-attributable death was estimated by summing age-specific alcohol-attributable mortality risks, conditional on survival to each age. Results were expressed as the risk of alcohol-attributable deaths per 1,000 people over the life course.

Premature mortality was defined as death occurring before age 75, in line with the OECD definition of premature death.(33) YLL attributable to alcohol were estimated using remaining life expectancy at each age, derived from CSO life tables.(34) Lifetime risks are presented numerically per 1,000 individuals to facilitate interpretation and comparison across outcomes. For descriptive purposes, these estimates are interpreted with reference to lifetime risk thresholds of 1 in 1,000 and 1 in 100 for both mortality and premature mortality, as these thresholds are commonly reported in comparable international analyses.(9, 39-41) For YLL, results are expressed relative to risk thresholds of 17.5 YLL attributable to alcohol per 1,000 and per 100 lifetimes, reflecting an average of 17.5 YLL per alcohol-attributable death in Ireland, based on 2023 estimates from the Institute for Health Metrics and Evaluation Global Burden of Disease study.(46) As estimates were reported in whole grams per day, threshold values were defined as the lowest whole-gram consumption level at which the modelled risk met or exceeded the benchmark. This avoided implying greater precision than supported by the analysis, while ensuring that the reported value corresponded to a consumption level at or above the benchmark.

2.2.2 Scenario analyses

Scenario analyses were conducted to assess how alternative assumptions about drinking patterns affected estimated injury mortality risk. Specifically, they examined whether the same total weekly alcohol consumption produced different estimates when consumed over fewer or more drinking days.

In the primary analysis, AAFs for acute causes of death were estimated using pattern-specific relative risk functions from Shield et al.,(29) which distinguish regular drinking from heavy episodic drinking. Age- and sex-specific AAFs were calculated by combining these relative risks with Healthy Ireland Survey estimates of alcohol consumption and heavy episodic drinking prevalence.

In the scenario analyses, relative risk functions, total weekly alcohol consumption, and baseline mortality rates were held constant. For each respondent, weekly alcohol consumption was redistributed across one to seven assumed drinking days. For each assumption, grams consumed per drinking day were recalculated, and respondents whose per-drinking-day consumption met or exceeded 60 g were classified as heavy episodic drinkers for that scenario.

The resulting age- and sex-specific prevalence estimates replaced the corresponding values from the primary analysis in the acute AAF calculation. Age- and sex-specific AAFs and population-level annual relative risks for injury mortality were then re-estimated for each scenario.

2.2.3 Sensitivity analyses

Two sensitivity analyses were conducted to assess the extent to which the primary analyses were sensitive to key modelling assumptions.

Sensitivity analysis 1: Alternative thresholds for wholly alcohol-attributable conditions

The level of alcohol consumption, if any, below which wholly alcohol-attributable conditions should be assumed to confer no alcohol-attributable mortality risk is uncertain. In the primary analyses, no lower threshold was assumed for these conditions (that is, risk increases with any consumption). This assumption may influence estimated risks at low levels of consumption. In this sensitivity analysis, thresholds of 32 g/week for males and 24 g/week for females were imposed, consistent with prior analyses.(47) Absolute risk was set to zero for exposures below the threshold. Above the threshold, risks are calibrated as in the base model. This analysis was intended to assess sensitivity to the application of a lower exposure threshold, rather than to identify empirically established thresholds below which alcohol-attributable risk is absent. Lifetime risk of premature mortality was then re-estimated.

Sensitivity analysis 2: Alternative consumption coverage rates

In the primary analyses, aggregate survey consumption was adjusted so that total estimated consumption equaled 80% of recorded per-capita alcohol sales. This adjustment was applied because alcohol consumption is typically under-reported in survey data, while calibrating to 100% of recorded sales could overstate exposure relative to the self-reported consumption data used in the epidemiological studies from which relative risk functions were derived.(44) However, the appropriate coverage value is subject to uncertainty. To assess model sensitivity to this assumption, lifetime risk of premature mortality was re-estimated after aligning survey totals to 60% and 90% of recorded per-capita sales. These values were selected as illustrative lower and higher assumptions around the primary 80% value and should not be interpreted as alternative best estimates.

2.2.4 Uncertainty estimations

Uncertainty intervals were estimated using Monte-Carlo simulation. Uncertainty was propagated for the relative risk functions only, with relative risk estimates drawn from distributions based on the published confidence intervals from the underlying meta-analyses. Lifetime alcohol-attributable outcomes were recalculated repeatedly across 5,000 simulation runs. Other model inputs were treated as fixed. Ninety-five per cent uncertainty intervals were defined using the 2.5th and 97.5th percentiles of the resulting distributions. Interval limits were rounded outwards to the nearest whole gram, so that the reported interval fully contained the underlying decimal-value intervals.

3 Results

This chapter presents the results from:

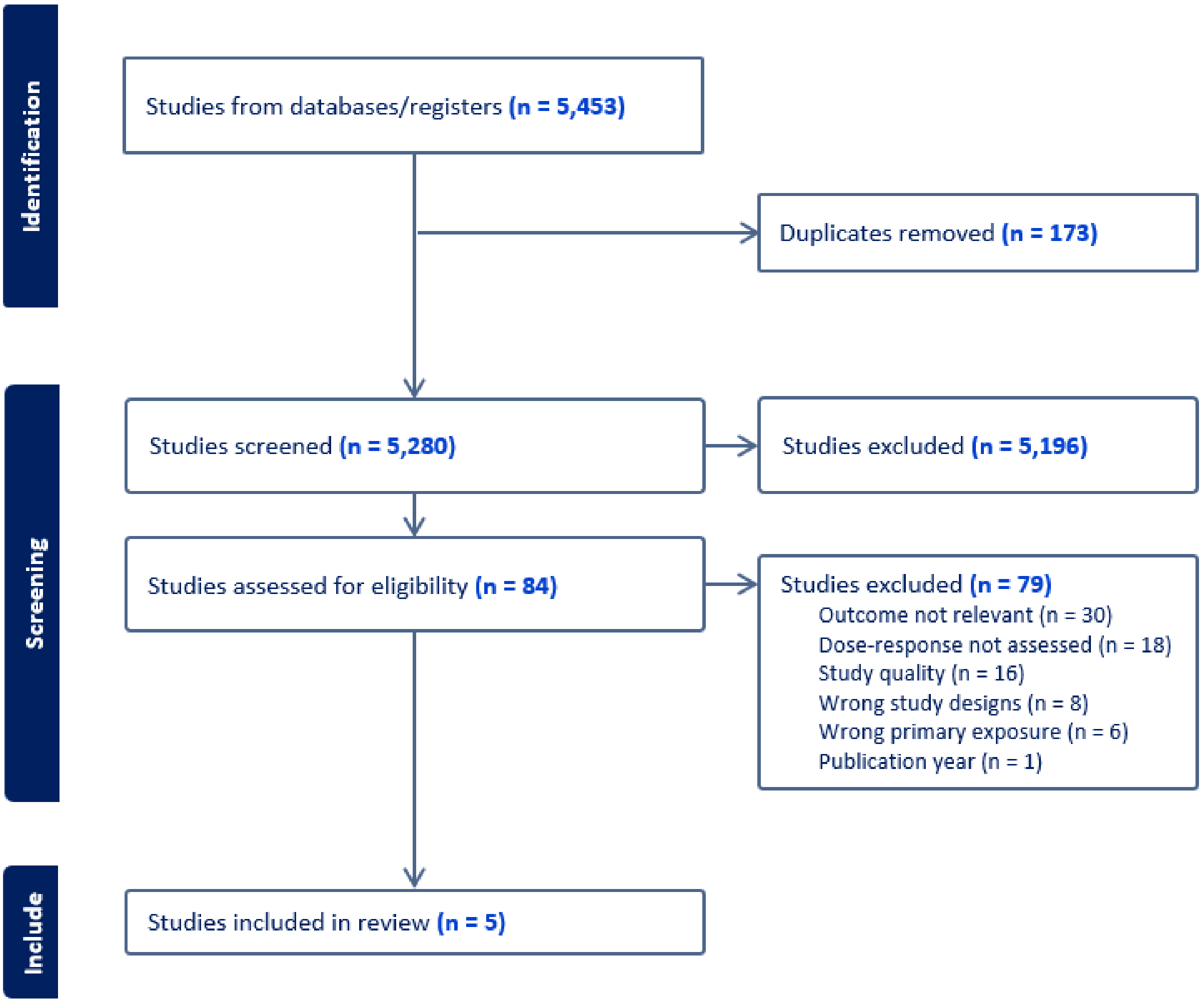
- the systematic review to identify risk estimates for chronic health outcomes that are partially attributable to alcohol consumption, for inclusion in the modelling (Section 3.1)
- estimated alcohol-attributable mortality and hospital admissions for 2022 to 2024 (Section 3.2)
- primary modelling analyses estimating lifetime risk of alcohol-attributable premature mortality, mortality and years of life lost, and relative rates of hospital admissions (Section 3.3)
- scenario analyses examining the impact of alternative drinking patterns (Section 3.4)
- sensitivity analyses (Section 3.5).

3.1 Systematic review of relative risk estimates

The relative risk estimates used in the modelling were drawn from published meta-analyses identified through a systematic review conducted by HIQA, which updated the 2022 CCSA review, *Update of Canada's Low-Risk Alcohol Drinking Guidelines: Evidence Review Technical Report*.[38, 48] Where a more recent meta-analysis that met stated inclusion and quality criteria was available for a condition, it replaced the one used in the CCSA model. This section summarises the updated review findings. Note that in standard GRADE assessments, evidence from observational studies typically starts as low certainty. However, consistent with the approach used in the review being updated, evidence from prospective observational studies was initially rated as moderate certainty for this review. This reflects the view that prospective cohort studies are often the most appropriate and feasible study design for public health questions of this type. Certainty was then downgraded or upgraded, as detailed in each assessment. Full methods of the review are detailed in the protocol.[11]

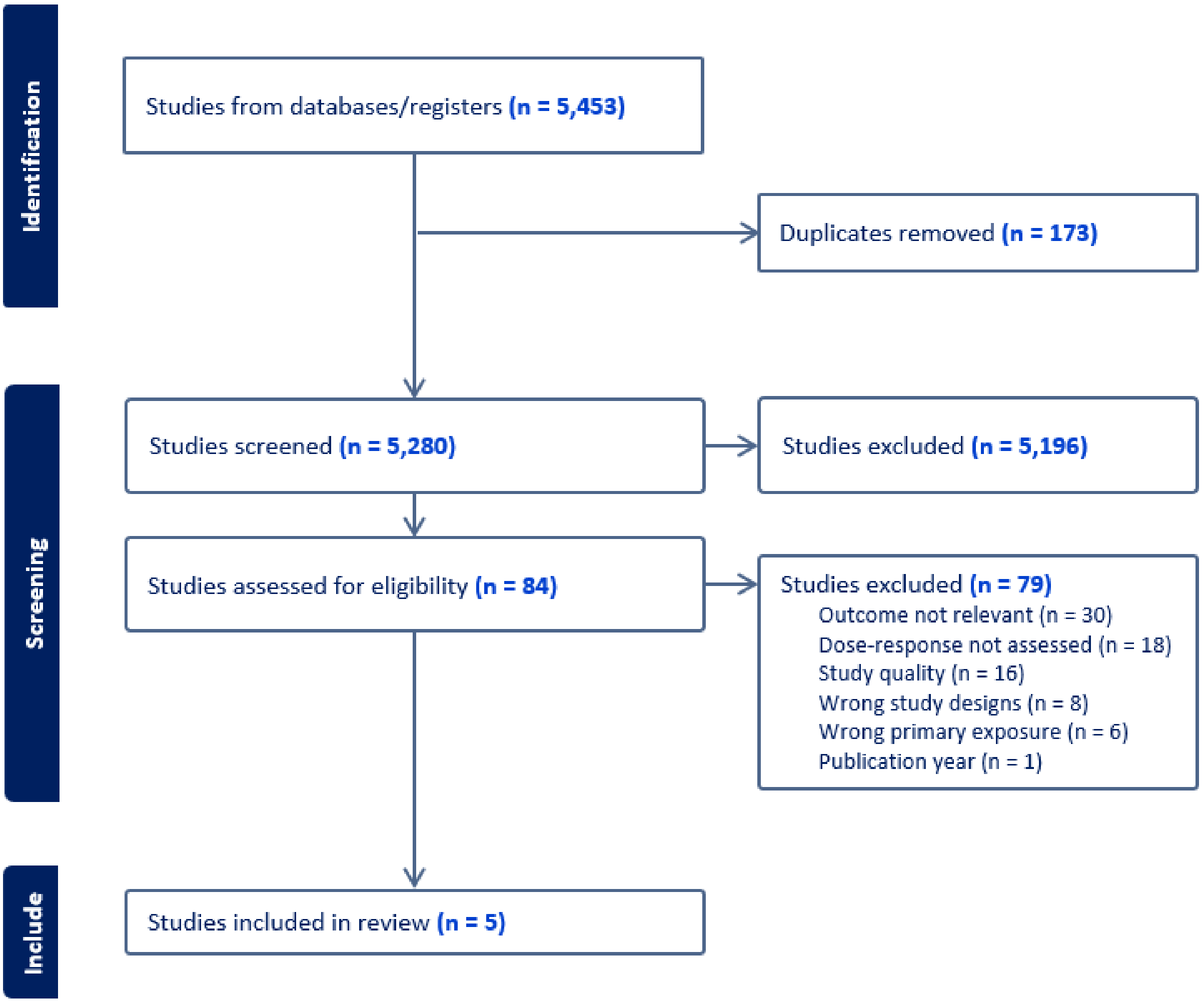
3.1.1 Search results

Figure 3.1 PRISMA flow diagram of the article screening process



presents the flow diagram of the screening process. The search, conducted up to 16 May 2025, identified 5,453 citations. After removing duplicates, 5,280 records remained. Of these, 5,196 were excluded based on title and abstract screening. The full texts of 84 articles were assessed for eligibility, with 79 excluded (see

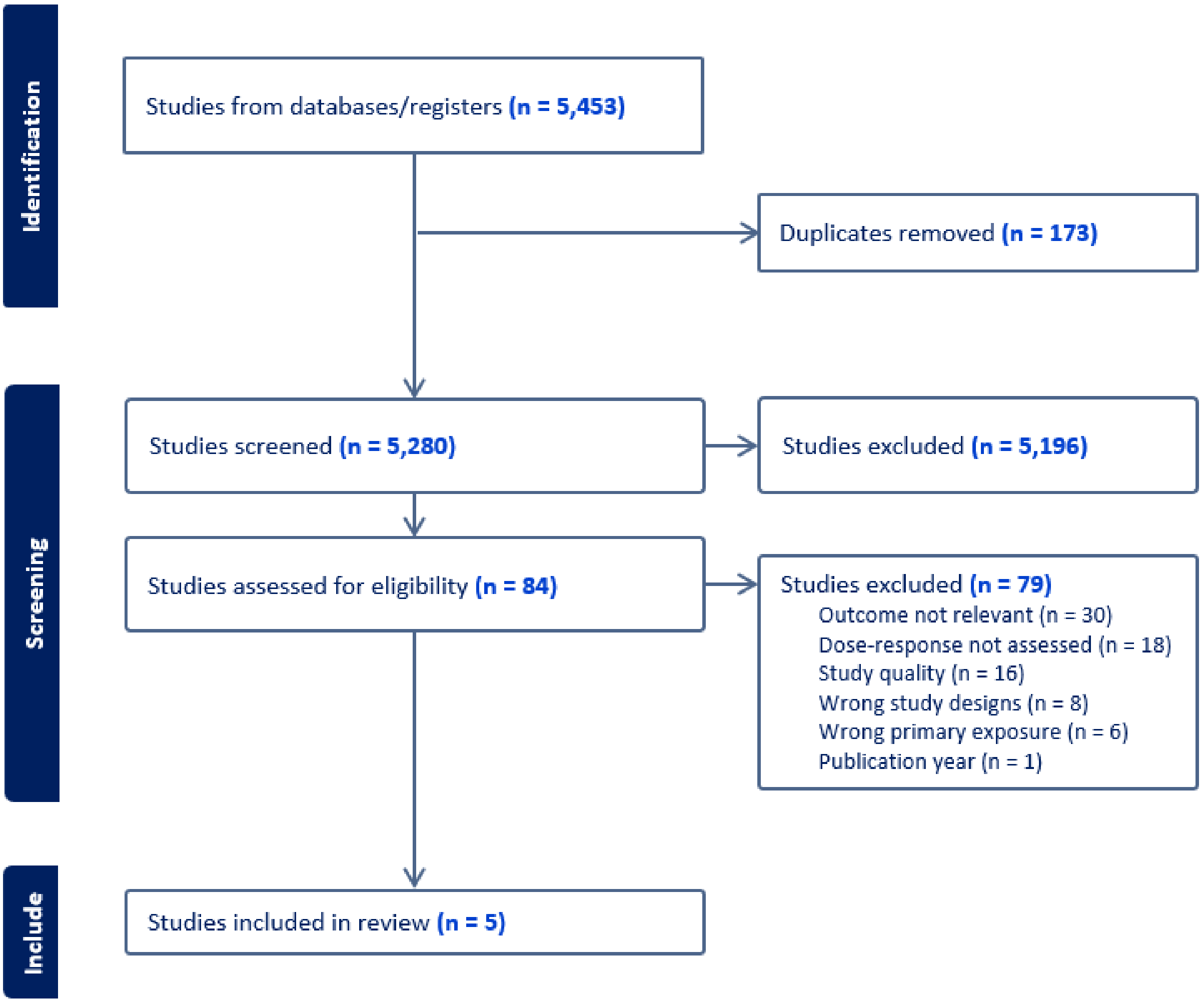
Figure 3.1 PRISMA flow diagram of the article screening process



for reasons). Five systematic reviews were included, covering the following conditions: liver cirrhosis,[15, 16] type 2 diabetes,[18] atrial fibrillation,[13] and breast cancer.[14]

For each condition, a single meta-analysis was included, except for liver cirrhosis, where two from the same author group were identified. One reported sex-specific risk estimates,[16] while the other incorporated a secondary analysis conducted by the authors and provided separate estimates for morbidity and mortality.[15] For the purposes of the current modelling, sex-specific risk estimates for both morbidity and mortality, derived from the meta-analysis including this secondary analysis, were provided by the study authors.

Figure 3.1 PRISMA flow diagram of the article screening process



3.1.2 Included reviews

Liver cirrhosis

Two new systematic reviews relevant to liver cirrhosis were identified for inclusion in the mathematical modelling: Llamosas-Falcón et al.[15] (2024) and Llamosas-Falcón et al.[16] (2022). The 2022 review reported sex-specific risk estimates, whereas the 2024 review incorporated a secondary analysis and provided separate morbidity and mortality estimates. For modelling purposes, sex-specific relative risk estimates for morbidity and mortality from the 2024 review, provided by the study authors, were used in the modelling. These reviews replace the previous review used in the CCSA model.[38, 49] Study characteristics are presented in Table 3.1. The GRADE assessment, including a summary of findings, is presented in Table 3.2. The AMSTAR 2 assessment is presented in Appendix 2.

Relative to lifetime abstainers, alcohol consumption was significantly associated with an increased risk of liver cirrhosis mortality and morbidity amongst males and females. Risk increased with higher levels of consumption. The evidence was rated as moderate certainty, as included studies were at high risk of bias and publication bias was not assessed, but dose-responses were detected, and effect sizes were large. No rating changes were applied for the other domains.

Table 3.1 Study characteristics for Llamosas-Falcón et al. (2024) and Llamosas-Falcón et al. (2022)

Reference	Population	Exposure	Outcome	Study type (n)	Meets PEO/study type criteria	Search dates	Criteria 1: Compre-hensive literature search?	Criteria 2: Character-istics of included studies in systematic review?	Criteria 3: Quality assessment of included studies in systematic review?	Criteria 4: Inclusion/ exclusion criteria?
Llamosas-Falcón et al.[15] (2024)	General population	Alcohol consumption (g/day)	Liver cirrhosis morbidity (incidence of liver cirrhosis or decompensated liver cirrhosis) and mortality	Cohort and case-control studies (n= 44 studies plus one secondary data analysis)	Yes	Database inception to 08/03/2023	Partial yes	Partial yes	Yes	Yes
Llamosas-Falcón et al.[16] (2022)	General population	Alcohol consumption (g/day)	Liver cirrhosis morbidity (incidence of liver cirrhosis or decompensated liver cirrhosis) and mortality	Longitudinal and case-control studies (n=24; 13 female and 19 male)	Yes	Database inception to 14/01/2022	Partial yes	Partial yes	Yes	Yes

Key: PEO – Population, Exposure, and Outcome.

Table 3.2 GRADE assessment for Llamosas-Falcón et al. (2024) and Llamosas-Falcón et al. (2022)

Reference	Numbers of unique studies and participants	Summary of results	GRADE	GRADE reasons for downgrading or upgrading	Certainty of evidence
Llamosas-Falcón et al.[15] (2024)	44 included studies and one included secondary data source (5,122,534 included participants)	**Liver cirrhosis morbidity risk ratio (95% confidence interval)** Lifetime abstainers: reference 23 g/day: 1.81 (1.68-1.94) 50 g/day: 3.54 (3.29-3.81) 100 g/day: 8.15 (7.46-8.91) **Liver cirrhosis mortality risk ratio (95% confidence interval)** Lifetime abstainers: reference 23 g/day: 2.65 (2.22-3.16) 50 g/day: 6.83 (5.84-7.97) 100 g/day: 16.38 (13.81-19.42)	Risk of bias: -2 Inconsistency: 0 Indirectness: 0 Imprecision: 0 Publication bias: -1 Dose response: 1 Effect size: 2	Risk of bias: All included studies at moderate, serious or critical risk of bias. Over one-third of included studies had a case-control study design. Publication bias: Not assessed. Dose response: Detected. Effect size: Large and very large.	⊕⊕⊕◯ Moderate
Llamosas-Falcón et al.[16] (2022)	24 included studies 3,037,329 included participants (2,112,476 females and 924,853 males)	**Female risk ratio (95% confidence interval)** Lifetime abstainers: reference 20 g/day: 3.34 (2.81-3.97) 40 g/day: 9.35 (7.64-11.45) 60 g/day: 17.54 (13.80-22.91) 80 g/day: 23.32 (18.24-29.82) 100 g/day: 25.33 (19.09-33.60) 120 g/day: 25.77 (17.62-37.68)	Risk of bias: -2 Inconsistency: 0 Indirectness: 0 Imprecision: 0 Publication bias: -1 Dose response: 1 Effect size: 2	Risk of bias: All included studies at moderate, serious or critical risk of bias. One-third of included studies had a case-control study design. Publication bias: Not assessed.	⊕⊕⊕◯ Moderate

Reference	Numbers of unique studies and participants	Summary of results	GRADE	GRADE reasons for downgrading or upgrading	Certainty of evidence
		Male risk ratio (95% confidence interval) Lifetime abstainers: reference 20 g/day: 1.60 (1.46-1.77) 40 g/day: 2.82 (2.53-3.14) 60 g/day: 5.09 (4.85-5.65) 80 g/day: 7.93 (7.12-8.83) 100 g/day: 10.76 (9.69-11.94) 120 g/day: 13.40 (11.30-15.88)		Dose response: Detected. Effect size: Very large.	

Type 2 diabetes

One new systematic review relevant to type 2 diabetes was identified for inclusion in the mathematical modelling: Llamosas-Falcón et al.[18] (2023). This review replaces the previous review used in the CCSA model.[38, 50] Study characteristics are presented in Table 3.3. The GRADE assessment, including a summary of findings, is presented in Table 3.4. The AMSTAR 2 assessment is presented in Appendix 2.

Relative to lifetime abstainers, alcohol consumption was significantly associated with a decreased risk of type 2 diabetes at 20 g/day and 40 g/day of alcohol consumption among females. There were no significant associations at higher levels of alcohol consumption among females or at any level of alcohol consumption among males. The evidence was rated as low certainty, as publication bias was not assessed. No rating changes were applied for the other domains.

Table 3.3 Study characteristics for Llamosas-Falcón et al. (2023)

Population	Exposure	Outcome	Study type (n)	Meets PEO/study type criteria	Search dates	Criteria 1: Compre-hensive literature search?	Criteria 2: Character-istics of included studies in systematic review?	Criteria 3: Quality assessment of included studies in systematic review?	Criteria 4: Inclusion/ exclusion criteria?
General population	Alcohol consumption (g/day)	Type 2 diabetes, including incident and/or mortality cases, as determined by an objective assessment (for example, laboratory findings or medical records) or self-report	Cohort and case-control studies (n= 55 studies plus one secondary data analysis; 29 female, 40 male, and 13 both sexes combined)	Yes	Database inception to 01/07/2022	Partial yes	Yes	Yes	Yes

Key: PEO – Population, Exposure, and Outcome.

Table 3.4 GRADE assessment for Llamosas-Falcón et al. (2023)

Numbers of unique studies and participants	Summary of results	GRADE	GRADE reasons for downgrading or upgrading	Certainty of evidence

55 included studies plus one secondary data source 2,653,983 included participants (1,290,628 females and 1,363,355 males)	**Males risk ratio (95% confidence interval)** Lifetime abstainers: reference 20 g/day: 1.02 (0.99-1.04) 40 g/day: 1.04 (0.99-1.09) 60 g/day: 1.06 (0.98-1.14) 80 g/day: 1.08 (0.98-1.19) 100 g/day: 1.10 (0.97-1.24) **Females risk ratio (95% confidence interval)** Lifetime abstainers: reference 20 g/day: 0.70 (0.65-0.75) 40 g/day: 0.78 (0.70-0.88) 60 g/day: 0.86 (0.63-1.17) 80 g/day: 0.94 (0.56-1.59) 100 g/day: 1.03 (0.48-2.19)	Risk of bias: 0 Inconsistency: 0 Indirectness: 0 Imprecision: 0 Publication bias: -1 Dose response: 0 Effect size: 0	Publication bias: Not assessed.	⊕⊕◯◯ Low

Atrial fibrillation

One new systematic review relevant to atrial fibrillation was identified for inclusion in the mathematical modelling: Zhang et al.[13] This review replaces the previous review used in the CCSA model.[38, 51] Study characteristics are presented in Table 3.5. The GRADE assessment, including a summary of findings, is presented in Table 3.6. The AMSTAR 2 assessment is presented in Appendix 2.

Relative to non-drinkers, alcohol consumption was significantly associated with an increased risk of atrial fibrillation among males and females. Risk was greater at higher levels of consumption. The evidence was rated as moderate certainty as both publication bias and a dose-response were detected. No rating changes were applied for the other domains.

Table 3.5 Study characteristics for Zhang et al.

Population	Exposure	Outcome	Study type (n)	Meets PEO/study type criteria	Search dates	Criteria 1: Compre-hensive literature search?	Criteria 2: Character-istics of included studies in systematic review?	Criteria 3: Quality assessment of included studies in systematic review?	Criteria 4: Inclusion/ exclusion criteria?
Participants without atrial fibrillation or atrial flutter at baseline	Alcohol consumption (g/day)	Atrial fibrillation or atrial flutter	Prospective cohort studies (n=13; 7 in dose response analyses)	Yes	Database inception to 01/05/2021	Partial yes	Yes	Yes	Yes

Key: PEO – Population, Exposure, and Outcome.

Table 3.6 GRADE assessment for Zhang et al.

Numbers of unique studies and participants	Summary of results	GRADE	GRADE reasons for downgrading or upgrading	Certainty of evidence
7 included studies 222,357 included participants	**Females hazard ratio (95% confidence interval)** 0 g/day: reference <12 g/day: 0.97 (0.90-1.04) 12-24 g/day: 1.02 (0.91-1.14)	Risk of bias: 0 Inconsistency: 0 Indirectness: 0	Publication bias: Detected. Dose response: Detected.	⊕⊕⊕◯ Moderate

	>24 g/day: 1.32 (1.10-1.60) **Males hazard ratio (95% confidence interval)** 0 g/day: reference <12 g/day: 1.04 (0.97-1.11) 12-24 g/day: 1.21 (1.10-1.33) >24 g/day: 1.54 (1.26-1.89)	Imprecision: 0 Publication bias: -1 Dose response: 1 Effect size: 0		

Breast cancer

One new systematic review relevant to breast cancer among females was identified for inclusion in the mathematical modelling: Sohi et al.[14] This review replaces the previous review used in the CCSA model.[38, 52] Study characteristics are presented in Table 3.7. The GRADE assessment, including a summary of findings, is presented in Table 3.8. The AMSTAR 2 assessment is presented in Appendix 2.

Relative to current or lifetime abstainers, alcohol consumption was associated with an increased risk of breast cancer at all levels of consumption. Risk increased with higher levels of consumption. The evidence was rated as moderate certainty, as results from publication bias analyses were not reported and a dose-response was detected. No rating changes were applied for the other domains.

Table 3.7 Study characteristics for Sohi et al.

Population	Exposure	Outcome	Study type (n)	Meets PEO/study type criteria	Search dates	Criteria 1: Compre-hensive literature search?	Criteria 2: Character-istics of included studies in systematic review?	Criteria 3: Quality assessment of included studies in systematic review?	Criteria 4: Inclusion/ exclusion criteria?
General population	Alcohol consumption (g/day)	Breast cancer incidence	Prospective cohort studies	Yes	Database inception to 15/11/2023	Partial yes	Yes	Yes	Yes

Key: PEO – Population, Exposure, and Outcome.

Table 3.8 GRADE assessment for Sohi et al.

Numbers of unique studies and participants	Summary of results	GRADE	GRADE reasons for downgrading or upgrading	Certainty of evidence
23 included studies (8,526,261 included participants)*	**Total population risk ratio (95% confidence interval)** Current or lifetime abstainers: reference 3 g/day: 1.05 (1.04-1.06) 10 g/day: 1.10 (1.08-1.12) 20 g/day: 1.18 (1.15-1.21) 30 g/day: 1.22 (1.19-1.25) 40 g/day: 1.24 (1.20-1.28) 50 g/day: 1.26 (1.21-1.31) 60 g/day: 1.28 (1.21-1.35)	Risk of bias: 0 Inconsistency: 0 Indirectness: 0 Imprecision: 0 Publication bias: -1 Dose response: 1 Effect size: 0	Publication bias: Assessed but results not presented. Dose response: Detected.	⊕⊕⊕◯ Moderate

3.2 Estimated alcohol-attributable mortality and hospital admissions, 2022-2024

Alcohol-attributable mortality and hospital admission estimates were generated as part of the modelling and are summarised here to contextualise the lifetime risk analyses. These outputs describe the scale and distribution of estimated alcohol-attributable harm in Ireland, including variation by outcome, sex, cause, and consumption level.

Table 3.9 shows the estimated annual number of alcohol-attributable deaths and hospital admissions by cause and sex for 2022 to 2024. Alcohol was estimated to account for 1,420 deaths and 27,066 hospital admissions per year. The majority of alcohol-attributable deaths (77%) and hospital admissions (75%) occurred in males. Chronic conditions accounted for most alcohol-attributable deaths (78%) and hospital admissions (51%).

For both sexes, more than half of all alcohol-attributable deaths (59% in males and 54% in females) occurred in the 90% of the drinking population with the lowest consumption. This reflects the population-level distribution of alcohol-attributable harm, whereby a large share of mortality arises from lower levels of consumption due to the greater number of individuals in these groups. Among males, the leading causes of alcohol-attributable mortality were cancers (29%), injuries (24%), digestive diseases (20%) and cardiovascular diseases (17%). Among females, the leading causes were cancers (37%), digestive diseases (35%) and injuries (14%) and cardiovascular diseases (7%).

Table 3.9 Estimated annual alcohol-attributable mortality and hospital admissions by cause and sex

Outcome	Cause	Males	Females	Total
Mortality	Acute alcohol attributable	264	47	311
	Chronic alcohol attributable	832	277	1,109
	Total alcohol attributable	1,096	324	1,420
	% of all-cause deaths	6.0%	1.9%	4.0%
Hospital admissions	Acute alcohol attributable	9,493	3,642	13,135
	Chronic alcohol attributable	10,680	3,251	13,931
	Total alcohol attributable	20,173	6,893	27,066

Note: Percentages represent the estimated proportion of all deaths in 2022-2024 that were attributable to alcohol within each sex.

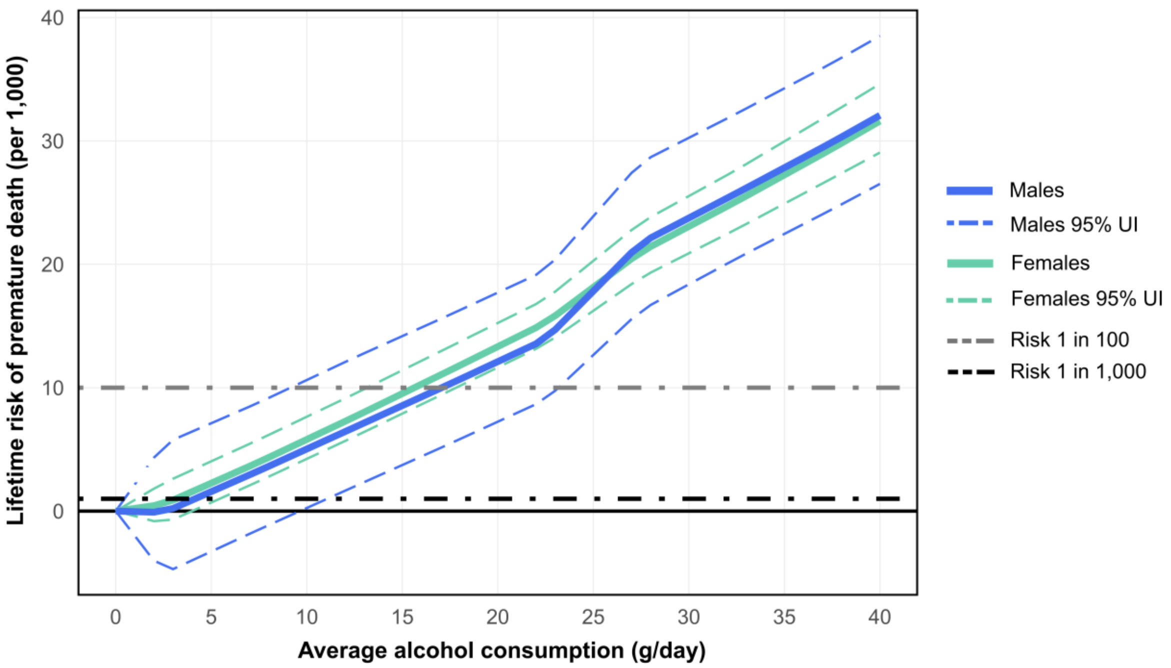
3.3 Primary analyses

The primary analyses estimated lifetime risk of alcohol-attributable mortality and annual relative rates of hospital admissions across levels of alcohol consumption in Ireland. Results are presented here for premature mortality, mortality, years of life lost (YLL) and hospital admissions, by sex. Additionally, Appendix 2 (females) and Appendix 4 (males) present summaries of cause-specific relative risks for premature mortality by average daily consumption.

3.3.1 Lifetime risk of alcohol-attributable premature death by sex

The absolute risk of an alcohol-attributable premature death increased with higher levels of alcohol consumption (Figure 3.2), apart from a small reduction in risk among males at consumption levels up to 2 g/day. Full tabulated estimates by sex and average daily alcohol consumption corresponding to the estimates shown in Figure 3.2, are provided in Appendix 5. The level of alcohol consumption corresponding to a 1 in 100 risk of alcohol-attributable premature death was 18 g/day (95% uncertainty interval [UI]: 10-23) for males and 16 g/day (95% UI: 13-18) for females. The level corresponding to a 1 in 1,000 risk of premature death was 5 g/day (95% UI: <1-9) for males and 4 g/day (95% UI: 1-5) for females.

Figure 3.2 Lifetime risk of a premature death attributable to alcohol use at varying levels of average alcohol consumption

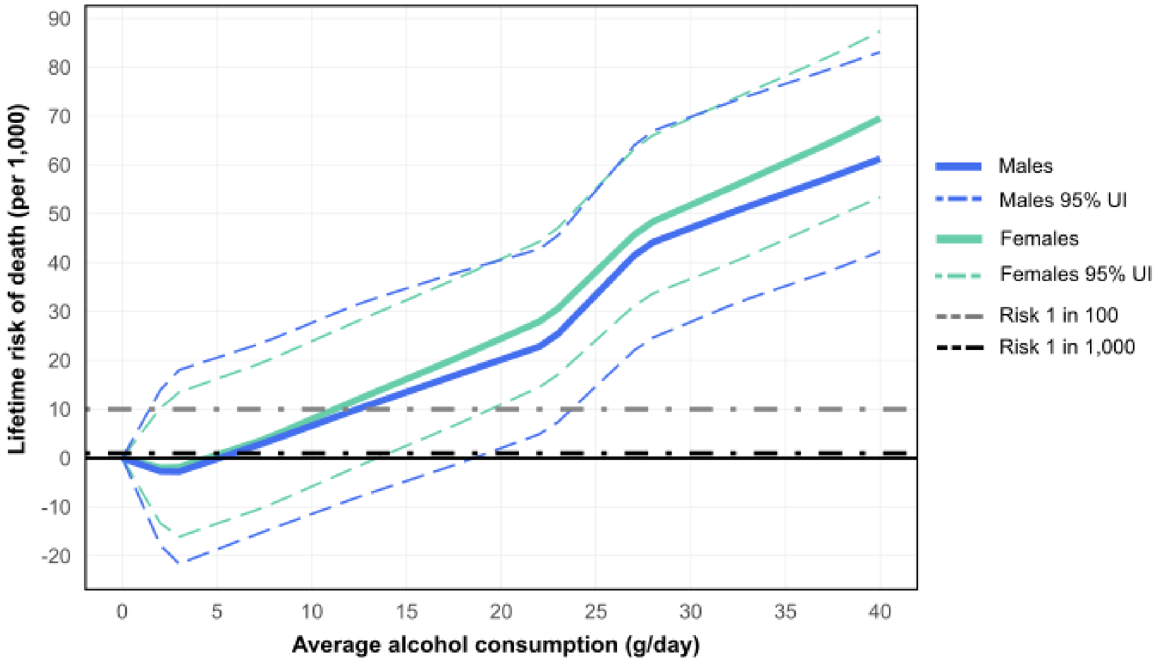


3.3.2 Lifetime risk of alcohol-attributable death by sex

The absolute risk of an alcohol-attributable death increased with higher levels of alcohol consumption (Figure 3.3), apart from a small reduction in risk among males and females at consumption levels up to 5 g/day and 4 g/day, respectively. The level of alcohol consumption corresponding to a 1-in-100 risk of alcohol-attributable death was 13 g/day (95% UI: 1-20) for males and 12 g/day (95% UI: 1-18) for females.

The level corresponding to a 1-in-1,000 risk of death was 6 g/day (95% UI: <1-14) for males and 6 g/day (95% UI: <1-11) for females.

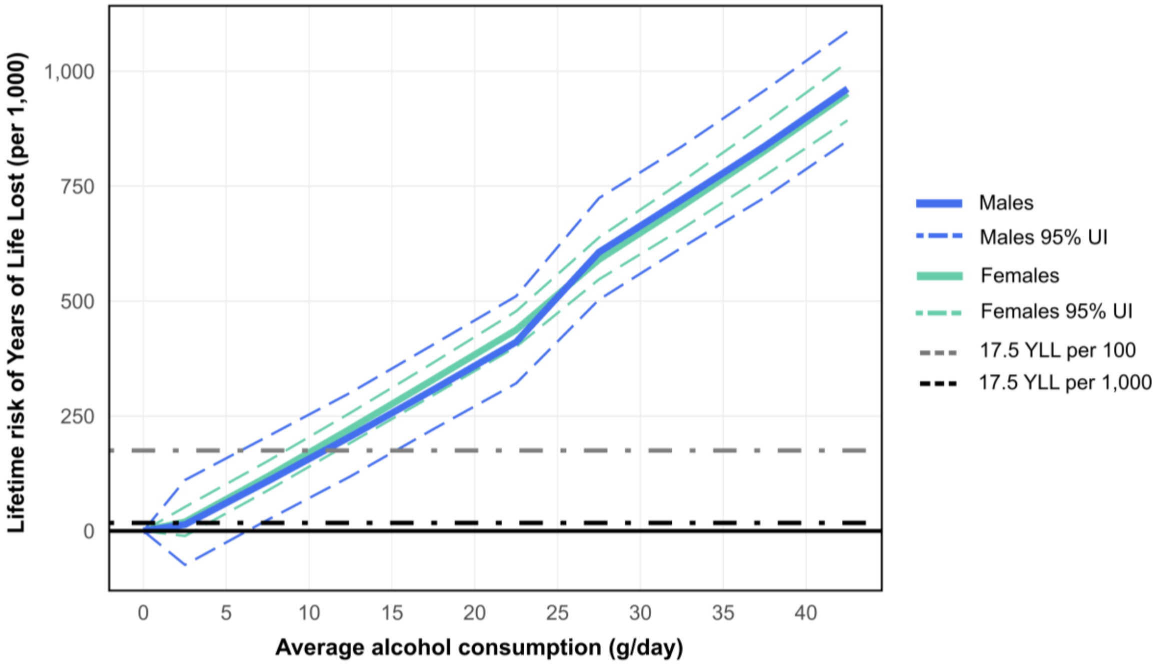
Figure 3.3 Lifetime risk of a death attributable to alcohol use at varying levels of average alcohol consumption



3.3.3 Lifetime risk of alcohol-attributable years of life lost by sex

The absolute risk of alcohol-attributable YLL increased with higher levels of alcohol consumption (Figure 3.4). The level of alcohol consumption corresponding to a risk of 17.5 YLL in 100 lifetimes was 11 g/day (95% UI: 6-15) for males and 11 g/day (95% UI: 9-12) for females. The level corresponding to a risk of 17.5 YLL in 1,000 lifetimes was 3 g/day (95% UI: <1-7) for males and 3 g/day (95% UI: <1-4) for females.

Figure 3.4 Lifetime risk of a year of life lost (YLL) attributable to alcohol use at varying levels of average alcohol consumption



3.3.4 Relative rate of hospital admission by sex

The relative rate of alcohol-attributable hospital admissions increased with higher levels of alcohol consumption for both acute and chronic alcohol-related causes (Figure 3.5 and Figure 3.6). For acute causes, the association was specified as linear on the logarithmic scale and the increase was steeper than for chronic causes. For acute conditions, the slope was slightly steeper among females than males, although uncertainty intervals overlapped across the range of alcohol consumption. For chronic conditions, admission rates increased more gradually and were similar between males and females. At 10 g/day (that is, one standard drink), the relative rate of hospital admission for acute injury was 1.23 (95% UI: 1.16-1.30) in females and 1.19 in males (95% UI: 1.14-1.24), increasing to 1.49 (95% UI: 1.35-1.64) in females and 1.40 in males (95% UI: 1.30-1.51) at 20 g/day. For chronic conditions, the relative rates at 10 g/day were 1.04 for females (UI 95%: 1.02-1.07) and 1.04 for males (95% UI: 1-1.10), increasing to 1.10 in both females (95% UI: 1.06-1.13) and males (95% UI: 1.05-1.17) at 20 g/day.

Figure 3.5 Relative rate of alcohol-attributable hospital admission events at varying levels of average alcohol consumption for acute alcohol-related causes

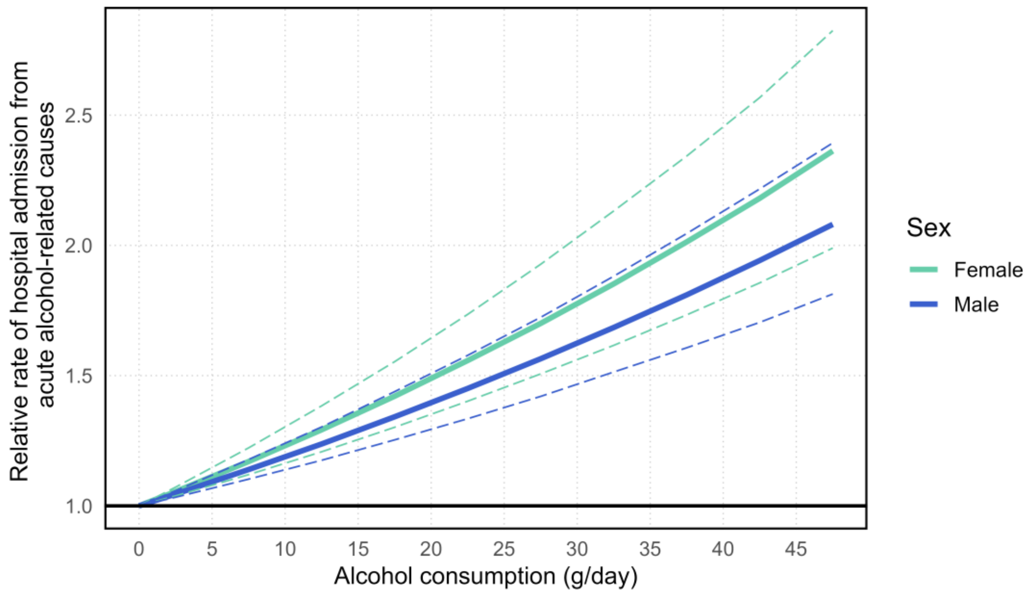
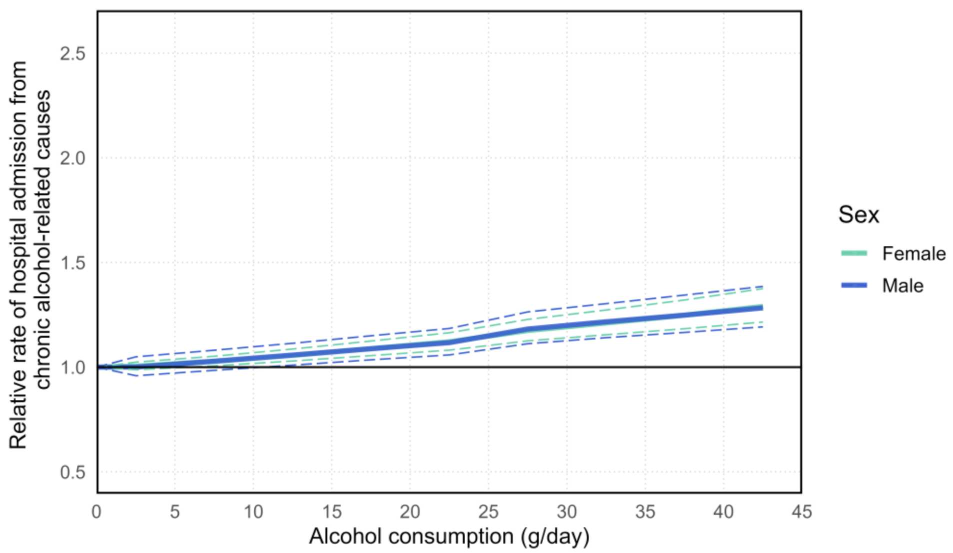


Figure 3.6 Relative rate of alcohol-attributable hospital admission events at varying levels of average alcohol consumption for chronic alcohol-related causes



3.4 Scenario analyses

Scenario analyses examined how alternative assumptions about binge drinking prevalence affected the modelled relative risk of injury mortality. Table 3.10 shows the distribution of binge drinking prevalence by sex and scenario. In females, prevalence of binge drinking ranges from 22% in the base-case scenario to 42% in Scenario 1, which assumes one drinking day per week. In males, prevalence ranges from 45% to 64% across the same scenarios. Based on the Healthy Ireland Survey, 88% of the population drink on two or fewer days (mean=1.2; median=1).

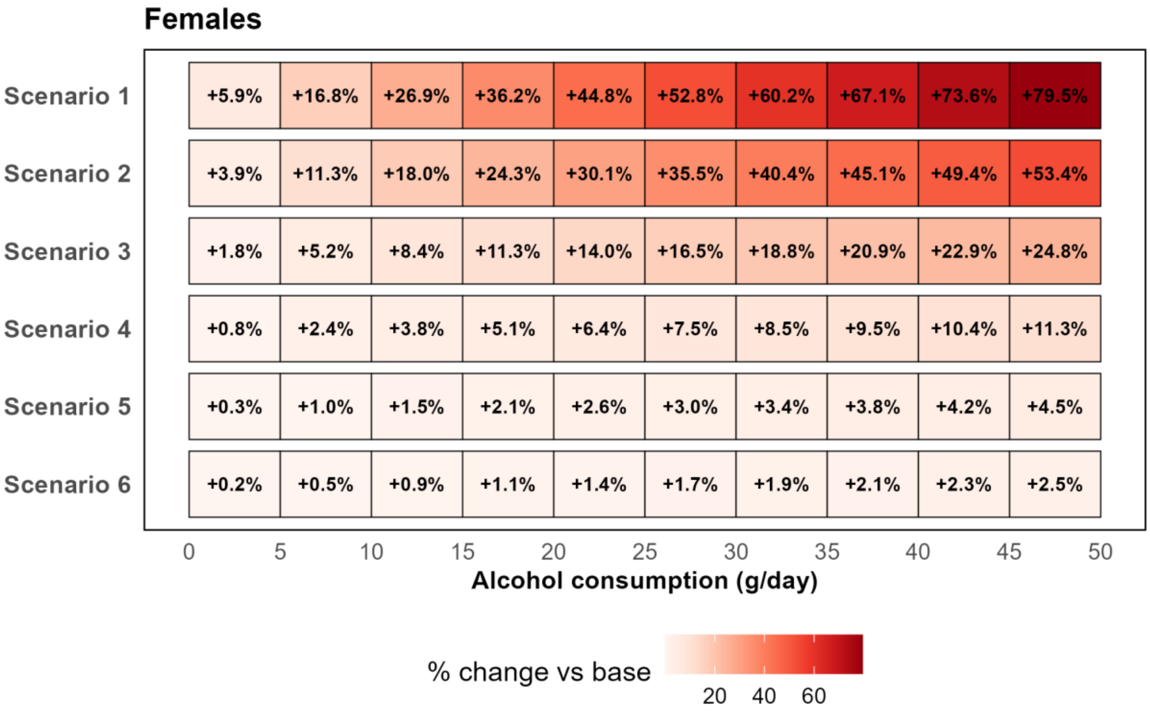
Table 3.10 Binge drinking prevalence by sex across scenario assumptions on drinking frequency

Scenario	Drinking Frequency	Females (%)	Males (%)
Scenario 1	Once/week	41.7	63.7
Scenario 2	Twice/week	35.8	60.0
Scenario 3	3 days/week	29.0	54.4
Scenario 4	4 days/week	25.4	50.5
Scenario 5	5 days/week	23.4	47.6
Scenario 6	6 days/week	22.8	46.5
Base-case scenario	7 days/week	22.0	44.6

Note: Scenarios reflect alternative assumptions regarding drinking frequency used to derive binge drinking prevalence distributions.

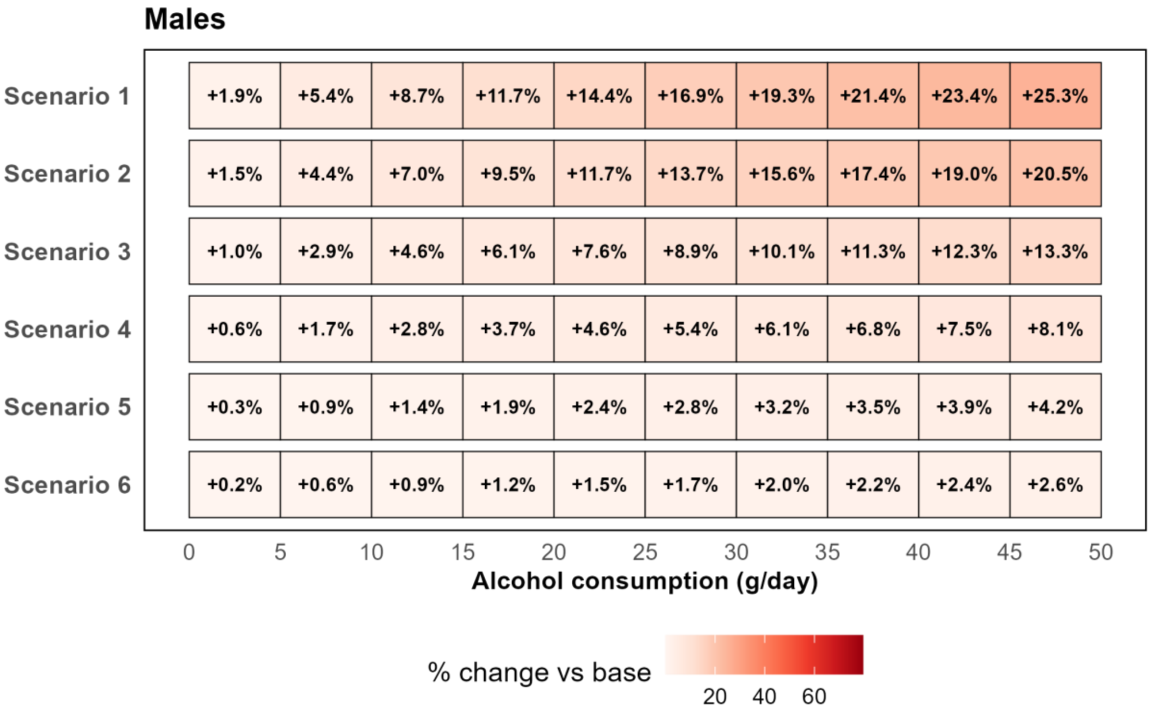
Figure 3.7 and Figure 3.8 show the percentage increase in population-level annual relative risk of injury mortality relative to the primary analysis assumption for females and males, respectively. In both sexes, higher assumed prevalences of chronic binge drinking was associated with progressively higher estimated injury mortality risk. For example, in Scenario 2 at 50g/day, injury mortality risk increased by 53.4% for females and 20.5% for males. The overall pattern was similar for males and females, although larger proportional increases were observed among females likely driven by the greater proportional increase in the implied prevalence of chronic binge drinking across scenarios. This reflects the structure of the acute risk model, in which a higher prevalence of chronic binge drinking increases the alcohol-attributable fraction for injury outcomes while total alcohol consumption remains unchanged.

Figure 3.7 Percentage increase in population-level annual relative risk of injury mortality under alternative assumptions about drinking frequency among females



Note: Scenario numbers correspond to assumptions about the number of drinking days per week over which weekly alcohol consumption is distributed, for example, Scenario 2 assumes drinking on two days per week. Under each assumption, the implied prevalence of chronic binge drinking (≥60 g per drinking day) was recalculated and used in the alcohol-attributable fraction estimates.

Figure 3.8 Percentage increase in population-level annual relative risk of injury mortality under alternative assumptions about drinking frequency among males



Note: Scenario numbers correspond to assumptions about the number of drinking days per week over which weekly alcohol consumption is distributed, for example, Scenario 2 assumes drinking on two days per week. Under each assumption, the implied prevalence of chronic binge drinking (≥60 g per drinking day) was recalculated and used in the alcohol-attributable fraction estimates.

3.5 Sensitivity analyses

This section reports the results of two sensitivity analyses conducted to assess how key modelling assumptions influence the estimated lifetime risk thresholds.

3.5.1 Sensitivity analysis 1: Alternative thresholds for wholly alcohol-attributable conditions

In sensitivity analysis 1, consumption thresholds below which wholly alcohol-attributable conditions were assumed to confer no additional risk were applied (32 g/week for males and 24 g/week for females). Applying these thresholds shifted the estimated lifetime risk thresholds to higher consumption levels. As shown in Table 3.11, the effect was modest at the 1 in 1,000 risk threshold and larger at the 1 in 100 risk threshold, and uncertainty intervals overlapped substantially with those from the primary analysis.

Table 3.11 Alcohol consumption levels associated with selected risk thresholds for lifetime risk of premature mortality for sensitivity analysis 1

Risk threshold	Analysis	Males, g/day (95% uncertainty interval)	Females, g/day (95% uncertainty interval)
1 in 1,000	Sensitivity	7 (<1 to 13)	5 (1 to 8)
	Primary	5 (<1 to 9)	4 (1 to 5)
1 in 100	Sensitivity	24 (15 to 28)	24 (21 to 25)
	Primary	18 (10 to 23)	16 (13 to 18)

3.5.2 Sensitivity analysis 2: Alternative alcohol consumption coverage rates

In sensitivity analysis 2, survey alcohol consumption was adjusted to 60% and 90% of recorded per-capita alcohol sales, compared with 80% in the primary analysis. As shown in Table 3.12, estimated lifetime risk of premature mortality decreased as coverage increased, meaning that risk thresholds were crossed at higher grams/day under 90% than 80%, and higher under 80% than 60% for both sexes. Differences are small and uncertainty intervals overlap but ordering is consistent, reflecting the model structure as explained in Section 4.3.

Table 3.12 Alcohol consumption levels associated with selected risk thresholds for lifetime risk of premature mortality for sensitivity analysis 2

Risk threshold	Adjustment level	Males, g/day (95% uncertainty interval)	Females, g/day (95% uncertainty interval)
	60%	4 (<1 to 8)	3 (1 to 4)
1 in 1,000	80% (base case)	5 (<1 to 9)	4 (1 to 5)
	90%	5 (<1 to 10)	4 (1 to 5)
	60%	14 (8 to 19)	13 (11 to 14)
1 in 100	80% (base case)	18 (10 to 23)	16 (13 to 18)
	90%	19 (11 to 23)	17 (14 to 19)

4 Discussion

4.1 Purpose and overview of findings

This analysis was undertaken to model the lifetime risk of alcohol-attributable mortality and relative rate of hospital admission in Ireland, using Irish mortality, hospital admission and alcohol consumption data, to inform an update of the low-risk alcohol guidelines.

Alcohol was estimated to account for 1,420 deaths and 27,066 hospital admissions per year between 2022 to 2024 in Ireland. The leading contributors to alcohol-attributable mortality were cancers. Most alcohol-attributable deaths (78%) and hospital admissions (51%) were due to chronic conditions (for example, cancers). For both sexes, more than half of alcohol-attributable deaths occurred among the 90% of the drinking population with the lowest consumption. This reflects the prevention paradox, as observed elsewhere, whereby people at lower levels of individual risk collectively account for more harm than the smaller group of heavier drinkers.[53, 54] From a policy perspective, this indicates that strategies focused solely on the highest-risk drinkers are unlikely to address most alcohol-related mortality, and that population-level approaches are therefore required.

Across the range of alcohol consumption examined, the risk of alcohol-attributable death, premature death and YLL and relative rate of hospital admission increased with higher average daily consumption, apart from small reductions in the risk of premature death among males at consumption levels up to 2 g/day and in the risk of death among both males and females up to 5 g/day and 4 g/day, respectively. Overall, risk patterns were broadly similar for males and females. The alcohol consumption levels corresponding to the selected risk thresholds for the three mortality outcomes were:

- Premature deaths
    - 1 in 1,000 risk
        - Males: 5 g/day (95% UI: <1-9)
        - Females: 4 g/day (95% UI: 1-5)
    - 1 in 100 risk
        - Males: 18 g/day (95% UI: 10-23)
        - Females: 16 g/day (95% UI: 13-18)
- Deaths
    - 1 in 1,000 risk
        - Males: 6 g/day (95% UI: <1-14)
        - Females: 6 g/day (95% UI: <1-11)
    - 1 in 100 risk

        - Males: 13 g/day (95% UI: 1-20)
        - Females: 12 g/day (95% UI: 1-18)
- Years of life lost
    - 17.5 per 1,000 lifetimes
        - Males: 3 g/day (95% UI: <1-7)
        - Females: 3 g/day (95% UI: <1-4)
    - 17.5 per 100 lifetimes
        - Males: 11 g/day (95% UI: 6-15)
        - Females: 11 g/day (95% UI: 9-12)

As shown, uncertainty intervals varied by outcome and threshold; however, lower bounds below 1 g/day indicate that, within the uncertainty range, even very low consumption may carry risk. Intervals for males and females overlapped substantially, so small differences in point estimates should be interpreted cautiously.

Relative admission rates were estimated for acute and chronic alcohol-attributable causes. As the underlying hospital data record admission events rather than unique individuals, the analyses estimate population rates of hospital admission events rather than the probability that an individual will experience at least one admission. Higher alcohol consumption was associated with higher rates of alcohol-attributable hospital admissions for both acute and chronic causes, with greater increases observed for acute conditions.

In the scenario analyses, assuming weekly alcohol consumption is concentrated into fewer drinking days increased the proportion of drinkers exceeding the chronic binge drinking threshold and, in turn, increased population-level annual relative risks for injury mortality. This relationship was consistent across the full range of frequency assumptions examined and reflects the structure of the acute risk model, in which injury AAFs are directly influenced by the prevalence of binge drinking. Within the current modelling framework, holding total alcohol consumption constant, scenarios with more concentrated drinking patterns are associated with a higher injury mortality risk.

These estimates should be considered alongside the assumptions and limitations described in Section 4.4. The following sections of the Discussion explore how the findings compare with prior evidence, examine sources of uncertainty, and outline considerations for translating these results into practical guidance for the Irish population.

4.2 Comparison with prior evidence

Modelling studies from the US, Canada, France and seven European countries have estimated alcohol consumption risks using methods consistent with the current analysis.[9, 39-41] While methodologies differ in aspects such as condition inclusion, relative risk function selection and certain model assumptions, their outputs provide a relevant reference point for interpreting the current findings.

Focusing on the primary outcome, the lifetime alcohol-attributable risk of premature mortality increased with consumption in a pattern broadly consistent with international modelling studies. The estimated locations of the 1 in 1,000 and 1 in 100 premature mortality risk thresholds are comparable with international findings, occurring at the lower end of the range reported across prior studies: 4-10 g/day in females and 5-15 g/day in males for the 1 in 1,000 threshold, and 15-22 g/day in females and 16-26 g/day in males for the 1 in 100 threshold.[9, 39-41] Consistent with the French and European modelling studies, thresholds occur at lower average consumption levels for women than for men, indicating a slightly higher premature mortality risk at equivalent consumption.[9, 41] However, sex differences observed in the current analysis are small and fall within the 95% uncertainty intervals.

Findings for the secondary mortality outcomes were also broadly consistent with international evidence. For lifetime alcohol-attributable mortality, the 1 in 1,000 and 1 in 100 risk thresholds occurred at similar consumption levels to those reported in Canada but at lower levels than those reported in the US, Australia, and the UK while for YLL the corresponding risk thresholds were similar to those reported in Canada and the US.[39, 40, 47, 55]

Differences between the current findings and international estimates likely reflect several factors. First, national alcohol consumption data differ in source and collection methods, which may affect the estimated consumption distribution. Second, the relative risk functions applied to specific conditions vary between studies, reflecting differences in the underlying meta-analyses used. Third, outcomes data, including mortality and hospital admissions, differ in source, coverage and coding practices. Sociodemographic factors and baseline health profiles may also vary between countries and contribute to observed differences. While the overall modelling approaches are largely comparable, these methodological differences can influence the study results.

In addition to international comparisons, findings from previous Irish studies allow comparison with elements of the current analysis. The estimated 1,420 (77% male) alcohol-attributable deaths per year (2022-2024) is 31% higher than the 1,080 (74% male) alcohol-attributable deaths recorded annually by the National Drug-Related Deaths Index (2008-2017).[56] This comparison should be interpreted cautiously because the two approaches differ in how alcohol-attributable deaths were identified and in the time periods examined. The NDRDI records drug- and alcohol-related deaths, including deaths among people who are alcohol dependent, whereas the current study uses General Mortality Register data and applies AAFs to selected wholly and partially attributable causes of death. A higher estimate in the current report is therefore expected, as the AAF-based approach estimates the contribution of alcohol to conditions where alcohol may not be explicitly recorded as a contributing factor. However, the General Mortality Register analysis is limited by reliance on the ICD-10 code assigned to the primary underlying cause of death, rather than on all conditions contributing to death. The current estimate aligns more closely with the Global Burden of Disease study, which attributed 1,543 deaths to alcohol in Ireland in 2019.(57)

A 2021 Health Research Board study estimated that there were 40,090 alcohol-attributable admissions in 2017, compared to the current estimate of 27,066.(56) The two estimates should be compared cautiously because the analyses differed in purpose, methods, and years examined. The HRB study aimed to describe the overall burden placed on Irish hospitals by alcohol-related admissions, whereas the current analysis focuses on quantifying the risk of alcohol-attributable mortality and rate of hospital admissions associated with different consumption levels. The difference in estimates is likely to be explained primarily by resulting methodological differences. One feature of the current analysis would be expected to produce lower estimates than those seen in the HRB study. This is the use of a narrower definition of alcohol-related hospital admission, restricted to cases where the primary reason for admission was attributable to alcohol rather than also including those in which an alcohol-related condition appeared in additional diagnosis fields. Conversely, two features of the current analysis would be expected to produce higher estimates. These are the adjustment of survey-based consumption data for under-reporting and inclusion of a broader range of conditions in the modelling. Finally, differences in the consumption and outcome years analysed may also account for some variation in estimates.

Overall, comparisons with international modelling studies and Irish data sources indicate that the present estimates are consistent with the broader evidence, while also reflecting differences that are explained in part by variations in data inputs and methodological choices.

4.3 Sensitivity analyses

The extent to which the primary analyses were sensitive to key modelling assumptions was examined using uncertainty and sensitivity analyses. In sensitivity analysis 1, imposing minimum thresholds for wholly alcohol-attributable conditions reduced estimated lifetime risk of premature mortality at low levels of alcohol consumption with the largest effects seen around the threshold values. Differences from the primary analysis diminished at higher consumption levels. This suggests that estimates at the lower end of the lifetime-risk curve are sensitive to assumptions about whether wholly alcohol-attributable conditions contribute risk at very low levels of consumption.

In sensitivity analysis 2, survey consumption was calibrated to 60% and 90% of recorded per-capita alcohol sales, compared with 80% in the primary analysis. Estimated lifetime risk of premature mortality decreased as coverage increased, reflecting the model structure rather than any protective effect of higher consumption coverage. Under lower coverage assumptions, alcohol-attributable fractions are smaller, so fewer deaths are removed when constructing the lifetime abstinence baseline. This results in a higher baseline mortality risk. Because alcohol-attributable risk is estimated relative to this baseline, the lifetime risk curve shifts upward across all consumption levels, and the 1 in 1,000 and 1 in 100 risk thresholds are reached at lower levels of consumption. This sensitivity analysis should therefore be interpreted as reflecting sensitivity to the under-reporting adjustment and in the modelled decomposition of observed mortality into baseline and alcohol-attributable components.

Overall, these analyses suggest that the main pattern of results is stable, while the precise location of individual thresholds remains sensitive to selected modelling assumptions.

4.4 Strengths and limitations

4.4.1 Strengths

The current analysis uses Irish data for consumption patterns, cause-specific mortality, hospital admissions, and demographic profiles. Aside from the relative risk functions derived from international meta-analyses, all model inputs reflect drinking patterns and baseline health profiles in Ireland. The methodology follows established protocols used to inform low-risk alcohol guidelines internationally, facilitating comparison with findings from other countries. By incorporating mortality, hospital morbidity, and YLL, the analysis provides complementary measures of alcohol-attributable risks. To our knowledge, this represents the most detailed quantification of lifetime alcohol-attributable risk in Ireland to date.

4.4.2 Limitations of the model inputs

Alcohol consumption

The consumption data are derived from self-reported surveys, which systematically underestimate actual consumption. While the data were adjusted to account for 80% of recorded alcohol sales, as is common practice, this approach assumes under-reporting is uniform across all demographic subgroups.(12, 29, 43, 44) It is plausible that misreporting varies by age, sex, drinking frequency, and socioeconomic status, which would distort the estimated consumption distribution.(58-61) For example, if higher-consuming drinkers under-report their consumption more than lower consumers, the adjustment would artificially compress the consumption distribution toward the mean. This would lead to an underestimation of the AAF for conditions with a steep dose-response curve (for example, liver cirrhosis), as the distribution would fail to capture the greater risk concentrated in the under-represented high-consumption tail. Furthermore, recorded sales do not account for unrecorded consumption, such as home-brewed or smuggled alcohol, nor do they adjust for the net difference between alcohol consumed abroad by Irish residents and that consumed in Ireland by tourists.(62) While unrecorded and consumption abroad likely represent a small proportion of total consumption, their exclusion may contribute to further underestimation of alcohol exposure.

Health outcomes

The scope of outcomes is restricted to conditions with established causal relationships and available dose-response risk functions. As a result, several conditions with a substantial health burden, such as dementia and depression, are excluded, meaning that mental health and cognitive harms are not fully captured.(12, 63) The morbidity analysis is limited to hospital admissions and does not capture treatment in other settings. Notably, it omits injuries resolved in emergency departments, which likely constitute the majority of cases,(64) as well as psychiatric inpatient activity not captured within the HIPE dataset. Additionally, the model focuses exclusively on harms experienced by the drinker and does not quantify the broader societal impacts or harms to others.

A protective effect for certain cardiovascular outcomes is included, reflecting recent meta-analyses.(23, 65) However, this may not account for specific consumption patterns; for example, individuals engaging in heavy episodic drinking may not experience protective effects against ischaemic cardiovascular disease.(66, 67) Furthermore, the existence of any protective effect remains a subject of debate in alcohol epidemiology.(68-70) Potential biases in the underlying observational studies, including the misclassification of former drinkers as abstainers and residual confounding, suggest that the presence and magnitude of any protective effects should be interpreted with caution.

Risk estimates

The dose-response relationships are derived from international meta-analyses of observational studies. While these represent the current evidence base, they are subject to limitations inherent in the underlying research, including abstainer misclassification, publication bias, and heterogeneity in population characteristics and health systems.[42, 71-73] These factors may affect the direct applicability of international risk relationships to the Irish population and should be considered when interpreting the results.

4.4.3 Limitations of the model

Simplified modelling of drinking patterns

The model estimates risk based on average daily alcohol consumption but does not explicitly account for variability in heavy episodic or binge drinking. Individuals with a similar average weekly consumption may differ substantially in their risk profiles depending on how that alcohol is consumed, such as whether it is spread evenly across the week or concentrated in fewer sessions. Such risk variation is difficult to quantify because the available dose-response functions are generally based on average consumption and do not provide condition-specific estimates for different drinking patterns. As a result, the model may underestimate the risk for individuals who engage in heavy episodic drinking or overestimate it for those who consume alcohol more evenly.

Uncertainty in model inputs

While limitations of individual model inputs are detailed in Section 4.4.2, the combined effect of these uncertainties is not fully quantifiable. Sensitivity analyses tested how specific assumptions influence the results, but do not capture all sources of uncertainty. The uncertainty intervals reflect uncertainty in the relative risk functions, which is likely to represent a major source of quantified parameter uncertainty, but do not capture uncertainty in other model inputs or wider structural uncertainties. These include how model inputs interact, the selection of specific functional forms for dose-response curves, and whether key assumptions hold across different contexts. As a result, the uncertainty intervals should not be interpreted as representing total uncertainty around the modelled estimates.

Stable dose-response relationships assumed

The modelling performed assumes that the relationships between alcohol consumption and mortality and hospital admission remain constant over time and across different populations. As a result, the model treats the health risk associated with a specific level of consumption as invariant across time, location and population groups. For example, the risk associated with drinking 20g of alcohol per day is treated as being the same in 2024 as it would be in future years, whether it is consumed in a location with better or worse healthcare access and across all population groups within an age-sex category. In practice, real-world differences such as changes in healthcare, behaviours or baseline health may affect the actual level of risk. This simplification is necessary due to the lack of data on how these relationships may vary over time or across settings. However, it introduces uncertainty when applying population-level estimates to diverse and changing contexts.

Lack of individual-level and subgroup variation

The model assumes uniform risk for all individuals within a specific age-sex-consumption cohort. It does not account for intra-group variation driven by socioeconomic status, comorbid conditions, genetics, or ethnicity. Notably, the model does not adjust for the alcohol harm paradox, in which lower socioeconomic groups often experience disproportionately higher rates of harm at similar consumption levels.(56, 74) The absence of high-resolution data on how relative risks vary across these subgroups means these potentially significant differences are not reflected in the estimates. These limitations should be borne in mind when interpreting population-level risk thresholds derived from the model

4.5 Harms to others from alcohol consumption

The outcomes modelled in this report are limited to harms experienced by the drinker. However, alcohol consumption results in significant harm to others which remains difficult to quantify within a dose-response framework. Between 2010 and 2026, expert panels in the United Kingdom, the European Union, Australia, New Zealand and Canada evaluated the relative harms of various substances.(75-79) In each instance except Canada, alcohol was ranked as causing greater aggregate harm to others than to the consumer. These assessments considered a range of harms, including economic costs, family adversities and injury.

In Ireland, a 2015 HSE survey found that 51% of respondents reported experiencing harm due to a stranger's drinking in the previous 12 months.(80) Reported harms included threats to personal safety, such as street harassment (23%) and feeling unsafe in public places (19%). Additionally, 44% of respondents reported harm caused by known drinkers, with 38% reporting psychological harms, such as stress or anxiety, and 24% reporting tangible harms, including physical injury or being a passenger with an intoxicated driver.(80) More recently, the 2019/2020 National Drug and Alcohol Survey reported that 17.0% of respondents had experienced harm from others' drinking, the lower estimate reflecting a narrower range of assessed harms.[81] In that study, the most frequent impacts were harm to family (10.8%), being a passenger with an intoxicated driver (6.0%), and physical assault (4.5%).

Adult alcohol consumption can also affect children, both directly and indirectly. While much of the evidence focuses on parental alcohol misuse, some research indicates that lower levels of consumption may also result in adverse effects.[82-84] Documented harms include child neglect, disrupted emotional development, and foetal alcohol spectrum disorder.[85-87] Furthermore, the 2015 HSE survey found that 16% of carers reported that children in their care had been harmed by someone else's drinking.[80] The most prevalent harms included verbal abuse (9%) and witnessing domestic violence (4%). In 64% of these cases where children were negatively affected, the carer identified a parent as the source of harm.

Alcohol consumption is associated with aggression and the perpetration of violence. A 2022 review by the CCSA examined the link between alcohol and intimate partner violence, sexual violence, and general aggression.[88] Experimental data indicate that acute alcohol consumption can contribute directly to aggressive behaviour, though the effect magnitude varies based on situational provocation and social pressure. Event-level studies in naturalistic settings find that intoxication is consistently associated with the perpetration of violence, particularly among younger cohorts and male perpetrators.

In summary, the harms caused by alcohol to individuals other than the drinker are substantial and have been assessed by several expert panels as causing greater aggregate harm to others than to the consumer. Although a formal dose-response relationship between consumption levels and harms to others cannot be quantified currently, it is likely that these harms are reduced by lower population-level alcohol consumption and reduced prevalence of intoxication.

4.6 Considerations for using the results to inform the development of low-risk alcohol guidelines

Developing low-risk alcohol guidelines from these results requires specific decisions on risk tolerance and the consideration of harms that are not currently quantified. This report identifies the estimated health risks for the drinker but does not set consumption limits. The following sections address how to use these findings in a policy context:

- Section 4.6.1 examines how the current results should be interpreted.
- Section 4.6.2 details considerations for setting guideline thresholds.

4.6.1 Interpreting the current report

Population-level risk estimates

The risk estimates in this report represent average expected outcomes for the population rather than a prediction for any specific individual. For example, a 1 in 100 risk of premature death at an average consumption of 16 g/day indicates that if a large cohort consumed alcohol at this level over their lifetimes, 1% of that cohort would be expected to die from an alcohol-attributable cause before the age of 75 years. These estimates do not reflect how an individual's risk would change following a shift in consumption. Real-world risk is influenced by a range of biological, behavioural, and situational factors (such as genetics, drinking history and baseline health) which are not captured in population-level modelling.

Uncertainty in the estimates

The results are subject to uncertainty arising from limitations in the underlying data and the modelling assumptions. Detailed in Section 4.4, these limitations warrant caution when interpreting the risk associated with specific consumption levels. Given the exclusion of several alcohol-related conditions and unrecorded consumption, these figures likely represent a conservative estimate of the actual risk profile in Ireland.

Scope of the outcomes considered

The analysis is restricted to health outcomes with a confirmed causal link to alcohol and established dose-response functions. This captures only a subset of the total impact of alcohol consumption. Excluded factors include harms to people other than the drinker (see Section 4.3) and non-health outcomes such as financial loss or family disruption. Consequently, the total potential burden of alcohol use in Ireland is not fully represented in this report.

Vulnerable populations, co-exposures and situational risks

The model estimates average risk within broad age and sex categories and does not account for individual-level factors that may modify alcohol-related risk. These include pregnant women, adolescents, pre-existing medical conditions, medication use, nutrition, poly-drug use, and socioeconomic context. The model also excludes situational risks where any alcohol consumption is unsafe, such as before driving or operating machinery. Furthermore, the model does not adjust for the higher mortality and morbidity rates often observed in lower socioeconomic groups at equivalent consumption levels.[56, 74] For some populations and circumstances, the report may therefore underestimate the actual risk of harm.

4.6.2 Guideline development

Defining risk thresholds for guideline development

Developing low-risk alcohol guidelines requires translating a continuous relationship between alcohol consumption and the risk of various health outcomes into discrete consumption categories defined by specified risk thresholds. Because the evidence does not identify a single alcohol consumption threshold at which alcohol-related risk is consistently absent across outcomes, thresholds used in guideline development should be understood as evidence-informed reference points rather than biological cut-points. Consequently, guidelines are commonly framed in terms of absolute lifetime risk to support transparent communication and individual decision-making.

As detailed in Section 4.6.1, the mortality estimates presented in this report describe absolute population-level risks of alcohol-attributable death over the life course. Premature mortality, defined as deaths occurring before the age of 75 years, was selected as the primary outcome for risk assessment. This outcome reduces the influence of high background mortality from other causes at older ages and diagnostic uncertainty in attributing cause-of-death that is common later in life.[41] Premature mortality is also commonly used as the basis for defining population-level risk thresholds in alcohol guideline development.[9, 41, 89] Overall mortality, YLL, and hospital admissions are also examined to provide a broader perspective on alcohol-related health effects.

Internationally, countries have adopted a range of quantified lifetime risk thresholds when developing low-risk alcohol guidelines. Recent guidelines have applied acceptable lifetime risk thresholds for death or premature death ranging from 0.1% to 2% (that is, a 1 in 1,000 to 1 in 50 lifetime risk). Australia, France, and the United Kingdom currently apply a 1% threshold;[9, 10, 90] Denmark applies a threshold between 1% and 2%;[91] and an analysis of seven European countries as part of the Joint Action on Reducing Alcohol Related Harm project recommended a threshold of 0.1%.[41] Recent Canadian guidelines from 2023 present multiple risk zones using 0.1% and 1% thresholds,[39] while previous Canadian guidance from 2011 adopted a 0% threshold, reflecting a position that acceptable alcohol consumption should not increase mortality risk above that of non-drinkers.[92] While the rationale for specific thresholds is not always explicit, these approaches illustrate how different jurisdictions operationalise the concept of acceptable risk.

The weekly consumption thresholds derived from these and other approaches vary substantially across countries. Most low-risk alcohol guidelines across Europe are clustered around 100-170 g/week for men and 70-110 g/week for women.[93] In many countries, weekly limits are accompanied by daily or per-occasion thresholds and or specified alcohol-free days. These differences should be interpreted cautiously, as guideline thresholds may reflect differences in methods, baseline risks, drinking patterns, and judgements about how alcohol-related risk should be defined and communicated. OECD data indicate that Ireland's average recorded alcohol consumption is broadly similar to the OECD average, but that Ireland is among the countries with the highest prevalence of monthly heavy episodic drinking.[94] This is relevant to guideline development as weekly average consumption does not fully capture how alcohol is consumed. In many countries, weekly limits are accompanied by daily or per-occasion thresholds, specified alcohol-free days, or both. The current analysis does not estimate a separate threshold for per-occasion or per-day limits. However, drawing on WHO evidence used in the scenario analyses, heavy episodic drinking, defined as 60g of pure alcohol on a single occasion or day, is associated with increased acute mortality risk.[30] Revised guidelines may therefore need to consider whether weekly consumption limits should be accompanied by guidance on single-occasion consumption and drinking frequency.

Ireland has not previously applied a quantified lifetime risk threshold in the development of its low-risk alcohol guidelines. The findings in this report provide an evidence base to support doing so. Selecting a threshold is not a purely technical determination and cannot be resolved by modelling alone. It requires evidence-informed judgement by guideline developers about how population-level risk should be summarised and communicated, informed by the modelled estimates, international practice, and the need for credible, transparent and evidence-based guidance. Presenting more than one threshold may support a graded or risk-zone approach, as adopted in Canada, and may assist in communicating that alcohol-related harm varies across levels of consumption.

Addressing variation in risk across the population

The estimates in this report reflect average risks categorised by sex. Individual risk varies due to a range of other factors not examined in these analyses, including socioeconomic status, health status, and other behavioural or biological characteristics.[7, 95, 96] This variation presents a challenge for guideline development, as reflecting differences in risk across population subgroups may be desirable but is often constrained by data limitations and the need for guidance that remains clear and usable at a population level.

Consideration of harms to others

This report does not quantify harms to others within the modelling framework, although these impacts constitute a substantial component of the total alcohol-attributable burden (Section 4.3). Such outcomes cannot currently be modelled using dose-response functions but represent an important part of alcohol's overall impact. In developing or communicating guidelines, it may therefore be useful to consider these broader harms.

Communication

Public understanding and uptake of low-risk alcohol guidelines likely depends on clarity, consistency with other health messaging, and the use of language that supports informed decision-making.[97, 98] The recent Canadian approach of presenting several risk zones, or a continuum of risk, rather than a single low-risk threshold, provides a novel way to communicate the risks associated with alcohol consumption. This approach reflects that alcohol-related harm increases across the full range of consumption, allows people to situate their own drinking within graded levels of risk and supports communication of the benefits of reducing consumption even when low-risk levels are not reached.[99] Given the limited empirical evidence comparing the behavioural impact of different communication approaches, decisions about how to present low-risk alcohol guidance should be grounded in the epidemiological evidence and expert judgment.

5 Conclusion

This study estimated the lifetime risk of alcohol-attributable mortality and the relative rates of hospital admission in Ireland to inform an update of the low-risk alcohol guidelines. These findings must be interpreted alongside the assumptions and limitations inherent in the modelling approach.

Across the range of alcohol consumption examined, mortality risk and hospital admission rates increase with higher average daily consumption. Concentrating a fixed amount of alcohol consumption into fewer drinking days increased estimated acute risk for both males and females. Small risk reductions are observed at the lowest consumption levels for certain mortality outcomes, though these effects are not consistent across all outcomes or sexes. For premature mortality (that is, death occurring at an age of up to 75 years), the estimated lifetime risk is approximately 1 in 1,000 at around 5 g/day and 1 in 100 at around 18 g/day for men, and approximately 1 in 1,000 at around 4 g/day and 1 in 100 at around 16 g/day for women.

These estimates are likely conservative, as outcomes were limited to those conditions with well-established causal evidence and sufficient data for inclusion in the model. Wider social and economic impacts and harms caused to people other than the drinker are also not captured.

References

1. Griswold MG, Fullman N, Hawley C, Arian N, Zimsen SRM, Tymeson HD, et al. Alcohol use and burden for 195 countries and territories, 1990–2016: a systematic analysis for the Global Burden of Disease Study 2016. The Lancet. 2018;392(10152):1015-35.
2. Healthy Ireland. Healthy Ireland Survey 2024 Summary Report: 2024 [Available from: https://assets.gov.ie/static/documents/healthy-ireland-survey-summary-report-2024.pdf.
3. Department of Health. Healthy Ireland Survey [Internet]. Irish Social Science Data Archive: Department of Health; 2024 [cited 2025 May 26]. Available from: https://www.ucd.ie/issda/data/healthyireland/.
4. Broholm K, Galluzzo L, Gandin C, Ghirini S, Ghiselli A, Jones L, et al. Good practice principles for low risk drinking guidelines. National Institute for Health and Welfare (THL). 2016.
5. Health Service Executive. Weekly low-risk alcohol guidelines: 2025 [Available from: https://www2.hse.ie/living-well/alcohol/health/improve-your-health/weekly-low-risk-alcohol-guidelines/.
6. Room R, Rehm J. Clear criteria based on absolute risk: Reforming the basis of guidelines on low - risk drinking. Drug and alcohol review. 2012;31(2):135-40.
7. Shield KD, Gmel G, Gmel G, Mäkelä P, Probst C, Room R, et al. Life - time risk of mortality due to different levels of alcohol consumption in seven European countries: implications for low - risk drinking guidelines. Addiction. 2017;112(9):1535-44.
8. Canadian Center on Substance Use and Addiction. Canada's Guidance on Alcohol and Health: Final Report [Internet]. Canada: Canadian Center on Substance Use and Addiction; 2023 [cited 2025 May 26]. Available from: https://www.ccsa.ca/canadas-guidance-alcohol-and-health-final-report.
9. Santé publique France. Avis d'experts relatif à l'évolution du discours public en matière de consommation d'alcool en France organisé par Santé publique France et l'Institut national du cancer [Internet]. France: Santé publique France; 2017 [cited 2025 May 26]. Available from: https://www.santepubliquefrance.fr/les-actualites/2017/avis-d-experts-relatif-a-l-evolution-du-discours-public-en-matiere-de-consommation-d-alcool-en-france-organise-par-sante-publique-france-et-l-insti.
10. UK Chief Medical Officers. UK Chief Medical Officers' Low Risk Drinking Guidelines [Internet]. UK: UK Chief Medical Officers; 2016 [cited 2025 May 26]. Available from: https://assets.publishing.service.gov.uk/media/5a80b7ed40f0b623026951db/UK_CMOs__report.pdf.
11. Health Information and Quality Authority. Protocol for modelling the risk of alcohol-attributable mortality and hospital admission in Ireland. Dublin: 2025 2025/08/13. Report No.
12. World Health Organization. Global status report on alcohol and health and treatment of substance use disorders [Internet]. Ireland: World Health

Organization; 2024 [cited 2025 May 26]. Available from: https://www.who.int/publications/i/item/9789240096745.
13. Zhang H-Z, Shao B, Wang Q-Y, Wang Y-H, Cao Z-Z, Chen L-L, et al. Alcohol consumption and risk of atrial fibrillation: a dose-response meta-analysis of prospective studies. Frontiers in Cardiovascular Medicine. 2022;9:802163.
14. Sohi I, Rehm J, Saab M, Virmani L, Franklin A, Sánchez G, et al. Alcoholic beverage consumption and female breast cancer risk: A systematic review and meta-analysis of prospective cohort studies. Alcohol Clin Exp Res (Hoboken). 2024;48(12):2222-41.
15. Llamosas-Falcon L, Probst C, Buckley C, Jiang H, Lasserre AM, Puka K, et al. How does alcohol use impact morbidity and mortality of liver cirrhosis? A systematic review and dose–response meta-analysis. Hepatology International. 2024;18(1):216-24.
16. Llamosas-Falcón L, Probst C, Buckley C, Jiang H, Lasserre AM, Puka K, et al. Sex-specific association between alcohol consumption and liver cirrhosis: An updated systematic review and meta-analysis. Frontiers in Gastroenterology. 2022;1:1005729.
17. Vieira AR, Abar L, Chan DSM, Vingeliene S, Polemiti E, Stevens C, et al. Foods and beverages and colorectal cancer risk: a systematic review and meta-analysis of cohort studies, an update of the evidence of the WCRF-AICR Continuous Update Project. Annals of Oncology. 2017;28(8):1788-802.
18. Llamosas-Falcón L, Rehm J, Bright S, Buckley C, Carr T, Kilian C, et al. The Relationship Between Alcohol Consumption, BMI, and Type 2 Diabetes: A Systematic Review and Dose-Response Meta-analysis. Diabetes Care. 2023;46(11):2076-83.
19. Samokhvalov AV, Irving H, Mohapatra S, Rehm J. Alcohol consumption, unprovoked seizures, and epilepsy: a systematic review and meta-analysis. Epilepsia. 2010;51(7):1177-84.
20. Wang M, Jiang X, Wu W, Zhang D. A meta-analysis of alcohol consumption and the risk of gout. Clinical rheumatology. 2013;32(11):1641-8.
21. Liu F, Liu Y, Sun X, Yin Z, Li H, Deng K, et al. Race-and sex-specific association between alcohol consumption and hypertension in 22 cohort studies: a systematic review and meta-analysis. Nutrition, Metabolism and Cardiovascular Diseases. 2020;30(8):1249-59.
22. Larsson SC, Wallin A, Wolk A, Markus HS. Differing association of alcohol consumption with different stroke types: a systematic review and meta-analysis. BMC Medicine. 2016;14(1):178.
23. Zhao J, Stockwell T, Roemer A, Naimi T, Chikritzhs T. Alcohol Consumption and Mortality From Coronary Heart Disease: An Updated Meta-Analysis of Cohort Studies. Journal of Studies on Alcohol and Drugs. 2017;78(3):375-86.
24. Bagnardi V, Rota M, Botteri E, Tramacere I, Islami F, Fedirko V, et al. Alcohol consumption and site-specific cancer risk: a comprehensive dose–response meta-analysis. British Journal of Cancer. 2015;112(3):580-93.
25. World Cancer Research Fund & American Institute for Cancer Research. Diet, nutrition, physical activity and cancer: A global perspective London, UK: World Cancer Research Fund International; 2018 [Available from:

https://www.wcrf.org/wp-content/uploads/2021/02/Summary-of-Third-Expert-Report-2018.pdf.
26. Samokhvalov AV, Irving HM, Rehm J. Alcohol consumption as a risk factor for pneumonia: a systematic review and meta-analysis. Epidemiology and Infection. 2010;138(12):1789-95.
27. Samokhvalov AV, Rehm J, Roerecke M. Alcohol Consumption as a Risk Factor for Acute and Chronic Pancreatitis: A Systematic Review and a Series of Meta-analyses. EBioMedicine. 2015;2(12):1996-2002.
28. Imtiaz S, Shield KD, Roerecke M, Samokhvalov AV, Lönnroth K, Rehm J. Alcohol consumption as a risk factor for tuberculosis: meta-analyses and burden of disease. Eur Respir J. 2017;50(1).
29. Shield K, Manthey J, Rylett M, Probst C, Wettlaufer A, Parry CD, et al. National, regional, and global burdens of disease from 2000 to 2016 attributable to alcohol use: a comparative risk assessment study. The Lancet Public Health. 2020;5(1):e51-e61.
30. World Health Organization. Global status report on alcohol and health 2018. 2018.
31. Churchill S, Angus C, Purshouse R, Brennan A, Sherk A. Expanding attributable fraction applications to outcomes wholly attributable to a risk factor. Statistical methods in medical research. 2020;29(9):2637-46.
32. Central Statistics Office. Deaths Occuring (2007-2018) [Internet]. Ireland: CSO; 2021 [cited 2025 May 26]. Available from: https://data.cso.ie/table/VSA08.
33. OECD/Eurostat. Avoidable mortality: OECD/Eurostat lists of preventable and treatable causes of death (January 2022 version) [Internet]. OECD/Eurostat; 2022 [cited 2025 May 26]. [21]. Available from: https://www.oecd.org/health/health-systems/Avoidable-mortality-2019-Joint-OECD-Eurostat-List-preventable-treatable-causes-of-death.pdf
34. Central Statistics O. VSA32: Period Life Expectancy. PxStat Open Data Platform. Ireland: Central Statistics Office; 2020.
35. Health Service Executive (HSE). Hospital In-Patient Enquiry (HIPE) [Internet]. Ireland: Healthcare Pricing Office, National Finance Division, HSE; 2024 [cited 2025 April 22]. Available from: https://hpowp.com/embed_stats_reporter_frame/.
36. Revenue Commissioners. Excise Receipts by Commodity. Dublin: Office of the Revenue Commissioners [Internet]. Ireland: Revenue Commissioners; 2024 [cited 2025 May 26]. Available from: Available from: https://www.revenue.ie/en/corporate/ information-about-revenue/statistics/excise/receipts-volume-and-price/excise-receipts-commodity.aspx
37. Central Statistics Office. 2024 [Available from: https://www.cso.ie/en/index.html.
38. Canadian Centre on Substance Use and Addiction. Update of Canada's Low-Risk Alcohol Drinking Guidelines: Evidence Review Technical Report [Internet]. Canada: Canadian Centre on Substance Use and Addiction; 2022 [cited 2025 May 26]. Available from: https://www.ccsa.ca/update-canadas-low-risk-alcohol-drinking-guidelines-evidence-review-technical-report-report.

39. Canadian Centre on Substance Use and Addiction. Lifetime Risk of Alcohol-Attributable Death and Disability: Canadian Centre on Substance Use and Addiction; 2023 [Available from: https://www.ccsa.ca/lifetime-risk-alcohol-attributable-death-and-disability.
40. Shield K, Keyes KM, Martinez P, Milam AJ, Naimi TS, Rehm J. Draft report: scientific findings of the alcohol intake & health study for public comment. 2025.
41. Rehm J, Gmel G, Probst C, Shield K. Lifetime-risk of alcohol-attributable mortality based on different levels of alcohol consumption in seven European countries: Implications for low-risk drinking guidelines. Toronto, On, Canada: 2015.
42. Zhao J, Stockwell T, Naimi T, Churchill S, Clay J, Sherk A. Association between daily alcohol intake and risk of all-cause mortality: a systematic review and meta-analyses. JAMA network open. 2023;6(3):e236185-e.
43. Rehm J, Kehoe T, Gmel G, Stinson F, Grant B, Gmel G. Statistical modeling of volume of alcohol exposure for epidemiological studies of population health: the US example. Population Health Metrics. 2010;8:1-12.
44. Stockwell T, Zhao J, Sherk A, Rehm J, Shield K, Naimi T. Underestimation of alcohol consumption in cohort studies and implications for alcohol's contribution to the global burden of disease. Addiction. 2018;113(12):2245-9.
45. Kehoe T, Gmel G, Shield KD, Gmel G, Rehm J. Determining the best population-level alcohol consumption model and its impact on estimates of alcohol-attributable harms. Population Health Metrics. 2012;10(1):6.
46. Institute for Health Metrics and Evaluation. GBD Compare | Global Burden of Disease Results [Data set]: 2023 [Available from: https://vizhub.healthdata.org/gbd-results/.
47. Angus C HM, Meier P, Brennan A, Holmes J. Mortality and morbidity risks from alcohol consumption in Australia: Analyses using an Australian adaptation of the Sheffield Alcohol Policy Model (v2. 7) to inform the development of new alcohol guidelines. ScHARR UoS, 2019.
48. Levesque C, Sanger N, Edalati H, Sohi I, Shield KD, Sherk A, et al. A systematic review of relative risks for the relationship between chronic alcohol use and the occurrence of disease. Alcohol: Clinical and Experimental Research. 2023;47(7):1238-55.
49. Roerecke M, Vafaei A, Hasan OSM, Chrystoja BR, Cruz M, Lee R, et al. Alcohol Consumption and Risk of Liver Cirrhosis: A Systematic Review and Meta-Analysis. Official journal of the American College of Gastroenterology | ACG. 2019;114(10).
50. Knott C, Bell S, Britton A. Alcohol Consumption and the Risk of Type 2 Diabetes: A Systematic Review and Dose-Response Meta-analysis of More Than 1.9 Million Individuals From 38 Observational Studies. Diabetes Care. 2015;38(9):1804-12.
51. Larsson SC, Drca N, Wolk A. Alcohol Consumption and Risk of Atrial Fibrillation: A Prospective Study and Dose-Response Meta-Analysis. Journal of the American College of Cardiology. 2014;64(3):281-9.

52. Sun Q, Xie W, Wang Y, Chong F, Song M, Li T, et al. Alcohol Consumption by Beverage Type and Risk of Breast Cancer: A Dose-Response Meta-Analysis of Prospective Cohort Studies. Alcohol and Alcoholism. 2020;55(3):246-53.
53. Sherk A, Churchill S, Cukier S, Grant SC, Shield K, Stockwell T. Distributions of alcohol use and alcohol - caused death and disability in Canada: Defining alcohol harm density functions and new perspectives on the prevention paradox. Addiction. 2024;119(4):696-705.
54. Poikolainen K, Paljärvi T, Mäkelä P. Alcohol and the preventive paradox: serious harms and drinking patterns. Addiction. 2007;102(4):571-8.
55. Holmes J, Angus C, Buykx P, Ally A, Stone T, Meier P, et al. Mortality and morbidity risks from alcohol consumption in the UK: Analyses using the Sheffield Alcohol Policy Model (v.2.7) to inform the UK Chief Medical Officers' review of the UK lower risk drinking guidelines. Final report.: 2016 [Available from: https://www.drugsandalcohol.ie/25023/1/Sheffield_Drinking_Guidelines_Final.pdf.
56. O'Dwyer C, Mongan D, Doyle A, Galvin B. Alcohol consumption, alcohol-related harm and alcohol policy in Ireland Dublin: 2021.
57. Kabir Z, Gilheany S, McKinney E. Global burden of disease: estimates of alcohol use and attributable burden in Ireland. What the data tell us and what we need to do to address the burden of alcohol. Dublin: Alcohol Action Ireland; UCC School of Public Health 2022. 2022.
58. Boniface S, Kneale J, Shelton N. Drinking pattern is more strongly associated with under-reporting of alcohol consumption than socio-demographic factors: evidence from a mixed-methods study. BMC public health. 2014;14(1):1297.
59. Devaux M, Sassi F. Social disparities in hazardous alcohol use: self-report bias may lead to incorrect estimates. The European Journal of Public Health. 2016;26(1):129-34.
60. Meier PS, Meng Y, Holmes J, Baumberg B, Purshouse R, Hill-McManus D, et al. Adjusting for unrecorded consumption in survey and per capita sales data: quantification of impact on gender-and age-specific alcohol-attributable fractions for oral and pharyngeal cancers in Great Britain. Alcohol and Alcoholism. 2013;48(2):241-9.
61. Stockwell T, Zhao J, Macdonald S. Who under - reports their alcohol consumption in telephone surveys and by how much? An application of the ' yesterday method' in a national C anadian substance use survey. Addiction. 2014;109(10):1657-66.
62. World Health Organization. Global health observatory data repository: Alcohol levels of consumption. 2025.
63. Rehm J, Hasan OS, Black SE, Shield KD, Schwarzinger M. Alcohol use and dementia: a systematic scoping review. Alzheimer's research & therapy. 2019;11(1):1.
64. Cherpitel CJ. Alcohol and injuries: a review of international emergency room studies since 1995. Drug and alcohol review. 2007;26(2):201-14.
65. Carr T, Kilian C, Llamosas-Falcón L, Zhu Y, Lasserre AM, Puka K, et al. The risk relationships between alcohol consumption, alcohol use disorder and

alcohol use disorder mortality: A systematic review and meta-analysis. Addiction. 2024;119(7):1174-87.
66. Roerecke M, Rehm J. Irregular heavy drinking occasions and risk of ischemic heart disease: a systematic review and meta-analysis. Am J Epidemiol. 2010;171(6):633-44.
67. Sundell L, Salomaa V, Vartiainen E, Poikolainen K, Laatikainen T. Increased stroke risk is related to a binge drinking habit. Stroke. 2008;39(12):3179-84.
68. Stockwell T, Zhao J, Panwar S, Roemer A, Naimi T, Chikritzhs T. Do "moderate" drinkers have reduced mortality risk? A systematic review and meta-analysis of alcohol consumption and all-cause mortality. Journal of studies on alcohol and drugs. 2016;77(2):185-98.
69. Naimi TS, Stockwell T, Zhao J, Xuan Z, Dangardt F, Saitz R, et al. Selection biases in observational studies affect associations between 'moderate'alcohol consumption and mortality. Addiction. 2017;112(2):207-14.
70. Fekjær HO. Alcohol—a universal preventive agent? A critical analysis. Addiction. 2013;108(12):2051-7.
71. Stockwell T, Zhao J, Panwar S, Roemer A, Naimi T, Chikritzhs T. Do "Moderate" Drinkers Have Reduced Mortality Risk? A Systematic Review and Meta-Analysis of Alcohol Consumption and All-Cause Mortality. J Stud Alcohol Drugs. 2016;77(2):185-98.
72. Probst C, Roerecke M, Behrendt S, Rehm J. Socioeconomic differences in alcohol-attributable mortality compared with all-cause mortality: a systematic review and meta-analysis. International journal of epidemiology. 2014;43(4):1314-27.
73. Rehm J, Baliunas D, Borges GL, Graham K, Irving H, Kehoe T, et al. The relation between different dimensions of alcohol consumption and burden of disease: an overview. Addiction. 2010;105(5):817-43.
74. Katikireddi SV, Whitley E, Lewsey J, Gray L, Leyland AH. Socioeconomic status as an effect modifier of alcohol consumption and harm: analysis of linked cohort data. The Lancet Public Health. 2017;2(6):e267-e76.
75. Nutt DJ, King LA, Phillips LD. Drug harms in the UK: a multicriteria decision analysis. The Lancet. 2010;376(9752):1558-65.
76. van Amsterdam J, Nutt D, Phillips L, van den Brink W. European rating of drug harms. Journal of psychopharmacology. 2015;29(6):655-60.
77. Bonomo Y, Norman A, Biondo S, Bruno R, Daglish M, Dawe S, et al. The Australian drug harms ranking study. Journal of Psychopharmacology. 2019;33(7):759-68.
78. Crossin R, Cleland L, Wilkins C, Rychert M, Adamson S, Potiki T, et al. The New Zealand drug harms ranking study: A multi-criteria decision analysis. Journal of Psychopharmacology. 2023;37(9):891-903.
79. Crépault J-F, Russell C, Asbridge M, Bonn M, Chaiton M, Darnay K, et al. Drug harms in Canada: A multi-criteria decision analysis. Journal of Psychopharmacology. 2026:02698811251409147.
80. Hope A, Barry J, Byrne S. The untold story: Harms experienced in the Irish population due to others' drinking: Health Service Executive (HSE); 2018.
81. Mongan D, Millar SR, Galvin B. The 2019–20 Irish National Drug and Alcohol Survey: Main findings [Internet]. Ireland: Health Research Board; 2021 [cited

2025 May 26]. Available from: https://www.hrb.ie/publication/the-2019-20-irish-national-drug-and-alcohol-survey-main-findings/.

82. Wahlström J, Magnusson C, Låftman SB, Svensson J. Parents' drinking, childhood hangover? Parental alcohol use, subjective health complaints and perceived stress among Swedish adolescents aged 10–18 years. BMC public health. 2023;23(1):162.
83. Kim S, Chae W, Min SH, Kim Y, Jang S-I. Alcohol consumption frequency of parents and stress status of their children: Korea National Health and Nutrition Examination Survey (2007–2016). International journal of environmental research and public health. 2020;17(1):257.
84. Brolin Låftman S, Östberg V, Wahlström J, Ramstedt M, Raninen J. Exposure to parental problem drinking during adolescence and symptoms of depression and anxiety in young adulthood: A Swedish national cohort study. Drug and alcohol review. 2024;43(6):1461-72.
85. Kelley ML, Klostermann K, Doane AN, Mignone T, Lam WK, Fals-Stewart W, et al. The case for examining and treating the combined effects of parental drug use and interparental violence on children in their homes. Aggression and violent behavior. 2010;15(1):76-82.
86. Wells K. Substance abuse and child maltreatment. Pediatric Clinics of North America. 2009;56(2):345-62.
87. Health Service Executive. HSE Position on Prevention of Fetal Alcohol Spectrum Disorders (FASD) [Internet]. Ireland: Health Service Executive; 2022 [cited 2025 May 26]. Available from: https://www.hse.ie/eng/about/who/healthwellbeing/our-priority-programmes/alcohol-programme/hse-position-on-prevention-of-fasd.pdf.
88. Canadian Centre on Substance Use and Addiction. Update of Canada's Low-Risk Alcohol Drinking Guidelines: Overview of Reviews of the Association Between Alcohol Use and Aggression and Violence [Internet]. Canada: Canadian Centre on Substance Use and Addiction; 2022 [cited 2025 May 26]. Available from: https://www.ccsa.ca/update-canadas-low-risk-alcohol-drinking-guidelines-overview-reviews-association-between-alcohol.
89. Canadian Centre on Substance Use and Addiction. Canada's Guidance on Alcohol and Health, Public Summary: Drinking Less Is Better (Infographic) [Internet]. Canada: Canadian Centre on Substance Use and Addiction; 2023 [cited 2025 May 27]. Available from: https://www.ccsa.ca/sites/default/files/2023-05/CGAH-Drinking-Less-is-Better-en.pdf.
90. National Health and Medical Research Council. Australian guidelines to reduce health risks from drinking alcohol [Internet]. Australia: National Health and Medical Research Council; 2020 [cited 2025 May 26]. Available from: https://www.nhmrc.gov.au/about-us/publications/australian-guidelines-reduce-health-risks-drinking-alcohol.
91. Sundhedsstyrelsen. Teknisk notat om risiko ved alkoholforbrug. Technical report. Copenhagen: 2022.
92. Butt P, Beirness D, Gliksman L, Paradis C, Stockwell T. Alcohol and health in Canada: a summary of evidence and guidelines for low-risk drinking. Ottawa, ON: Canadian Centre on Substance Abuse. 2011.

93. European Commission Joint Research Centre. National low-risk drinking recommendations (or drinking guidelines) and standard units: 2024 [updated 2024/10/17. Available from: https://knowledge4policy.ec.europa.eu/health-promotion-knowledge-gateway/national-low-risk-drinking-recommendations-drinking-guidelines_en.
94. OECD. Health at a Glance 2025: OECD Indicators. Paris: 2025 2025/11/13. Report No.
95. Mäkelä P, Paljärvi T. Do consequences of a given pattern of drinking vary by socioeconomic status? A mortality and hospitalisation follow-up for alcohol-related causes of the Finnish Drinking Habits Surveys. Journal of Epidemiology & Community Health. 2008;62(8):728-33.
96. Bellis MA, Hughes K, Nicholls J, Sheron N, Gilmore I, Jones L. The alcohol harm paradox: using a national survey to explore how alcohol may disproportionately impact health in deprived individuals. BMC public health. 2016;16:1-10.
97. Shield K, Paradis C, Butt P, Naimi T, Sherk A, Asbridge M, et al. New perspectives on how to formulate alcohol drinking guidelines. Addiction. 2024;119(1):9-19.
98. Holmes J, Angus C, Meier PS, Buykx P, Brennan A. How should we set consumption thresholds for low risk drinking guidelines? Achieving objectivity and transparency using evidence, expert judgement and pragmatism. Addiction. 2019;114(4):590-600.
99. Shield KD, Paradis C, Butt PR, Naimi T, Sherk A, Asbridge M, et al. New perspectives on how to formulate alcohol drinking guidelines: response to commentaries. Addiction. 2024;119(1):26-7.
100. Gmel G, Shield KD, Kehoe-Chan TA, Rehm J. The effects of capping the alcohol consumption distribution and relative risk functions on the estimated number of deaths attributable to alcohol consumption in the European Union in 2004. BMC medical research methodology. 2013;13:1-9.

Appendix 1. Technical specification of the mathematical model

Lifetime risk of alcohol-attributable mortality was estimated using a comparative risk assessment framework. The approach builds on methods previously applied in international alcohol risk modelling studies, including those developed to inform national low-risk drinking guideline updates.[9, 39-41] The analysis was conducted in four sequential steps, described below.

Lifetime abstention from alcohol was used as the theoretical minimum risk exposure level, consistent with established comparative risk assessment practice.[9, 39-41] This choice defines the counterfactual reference against which alcohol-attributable risk is estimated and does not imply an assumption that lifetime abstention represents the level of alcohol consumption associated with the lowest overall risk of health loss. However, evidence at low levels of consumption is uncertain, subject to bias and does not provide a sufficiently robust basis for defining a non-zero minimum risk exposure level.[42]

In addition to mortality, alcohol-attributable hospital admissions were examined. Hospital admission data were obtained from the HIPE system and represent counts of admissions rather than person-level follow-up. Accordingly, results for hospital admissions are expressed as annual alcohol-attributable admission rates. To do this, the same formulas as in the mortality analysis were followed up to, but not including, the life-course component used to estimate cumulative lifetime risk. Results for acute and chronic conditions were reported separately.

Step 1. Modelling alcohol consumption among people who drink

This step estimates age- and sex-specific distributions of average daily alcohol consumption among people who drink. Estimates of drinking prevalence and mean alcohol consumption by age and sex were obtained from the 2024 Healthy Ireland Survey.[3] Estimated mean consumption among people who drink was adjusted such that implied per-capita alcohol consumption corresponded to 80% of recorded alcohol sales for the same year, consistent with standard practice.[12, 29, 43, 44]

For each age- and sex-specific subgroup, the distribution of average daily alcohol consumption among people who drink was modelled using a Gamma distribution. Under this formulation, the Gamma distribution represents a continuous prevalence density over levels of average daily alcohol consumption such that, when scaled by the prevalence of people who drink, it describes the proportion of the population consuming alcohol at each consumption level.

The Gamma distribution was parameterised using the mean daily alcohol consumption (μ) among people who drink and an empirically derived relationship between the mean and standard deviation (σ). Following Rehm et al.[43] and Kehoe et al.,[45] the standard deviation was specified as a function of the mean and sex (Formula 1), which fully determines the Gamma distribution for each subgroup.

Average daily alcohol consumption was modelled over the range 0.027 g/day to 150 g/day. The lower bound corresponds to an annual consumption of one standard drink (10g of pure alcohol), reflecting the minimum level used to define current drinking. The upper bound of 150 g/day reflects a modelling choice, recognising that sustained average consumption above this level may be uncommon even among very heavy drinkers.[100] Because the Gamma distribution is defined on (0, ∞), the distribution was normalised over this range so that integration of the prevalence density corresponds to the observed prevalence of people who drink in each subgroup.

Formula 1

$$\hat{\sigma} = (1.171 + 0.087 * sex) * \hat{\mu}$$

Where:

- *σ* is the standard deviation of daily alcohol consumption (g/day) among people who drink
- *μ* is the mean daily alcohol consumption (g/day) among people who drink
- *sex* is coded as 1 for females and 0 for males

Step 2. Estimating baseline mortality under a counterfactual in which alcohol consumption is set to zero

The second step estimates age- and sex-specific baseline mortality risk under a counterfactual scenario in which alcohol consumption is absent.

Age-, sex- and cause-specific AAFs were estimated by combining age- and sex-specific alcohol consumption distributions with the corresponding cause-specific relative risk functions, using a Levin-based formulation (Formula 2). Ex-drinkers were treated as a separate exposure category with an associated relative risk.

For acute causes of death, AAFs were derived using relative risk functions obtained from Shield et al.,[29] based on functions from the World Health Organization's 2018 *Global status report on alcohol and health*.[30] These functions distinguish between regular drinking and heavy episodic drinking patterns. Using prevalence estimates of episodic and chronic binge drinking derived from the Healthy Ireland Survey, an AAF was calculated for each age-sex group that incorporated both average volume of consumption and binge-pattern prevalence.

Alcohol-attributable deaths were estimated by multiplying the AAF by the observed number of deaths for each cause, age group and sex (Formula 3). Baseline mortality was then derived by removing alcohol-attributable deaths from observed mortality counts and dividing the remaining deaths by the corresponding population (Formula 4).

Formula 2

$$AAF = \frac{P_{EXD}(RR_{EXD} - 1) + \int_{0.027\ \text{g/day}}^{150\ \text{g/day}} P_D(x)(RR_D(x) - 1)dx}{1 + [P_{EXD}(RR_{EXD} - 1) + \int_{0.027\ \text{g/day}}^{150\ \text{g/day}} P_D(x)(RR_D(x) - 1)dx]}$$

Where:

- P_{EXD} is the prevalence of ex-drinkers
- RR_{EXD} is the relative risk for ex-drinkers
- $P_D(x)$ is the population share of current drinkers at *x* g/day
- $RR_D(x)$ is the condition-specific relative risk at *x* g/day
- *x* is grams of pure alcohol per day

Formula 3

$$AA\_Deaths_{a,s,c} = AAF_{a,s,c} \times Deaths_{a,s,c}$$

Where:

- *AA_Deaths* is the number of deaths attributable to alcohol for cause *c* in age group *a* and sex *s*
- *AAF* is the alcohol-attributable fraction for cause *c* in age group *a* and sex *s*
- *Deaths* is the total number of deaths recorded for cause *c* in age group *a* and sex *s*

Formula 4

$$Risk\_Death\_LA_{a,s,c} = \frac{[Deaths_{a,s,c} - AA\_Deaths_{a,s,c}]}{Pop_{a,s}}$$

Where:

- *Risk_Death_LA* is the estimated baseline risk of death for cause *c* in age group *a* and sex *s* under lifetime abstention
- *Deaths* is the total number of deaths recorded for cause *c* in age group *a* and sex *s*

- *AA_Deaths* is the number of deaths attributable to alcohol for cause *c* in age group *a* and sex *s*
- $Pop_{a,s}$ is the population size for the specified age group *a* and sex *s*

Step 3. Estimating alcohol-attributable mortality risk

The third step estimates age- and sex-specific alcohol-attributable increases in mortality risk by cause, based on baseline mortality in the absence of alcohol and alcohol consumption levels.

For acute causes of death, drinker-specific relative risks were derived by calibration to ensure consistency between observed population mortality and the observed distribution of alcohol consumption among drinkers. This calibration was implemented by numerically solving the population risk identity for the drinker-specific relative risk parameter, with observed population mortality held fixed (Formula 5).

Cause-specific alcohol-attributable mortality risks were then estimated by applying the relevant cause-specific relative risk relationships to baseline mortality risks, expressed as excess risk associated with alcohol consumption (Formula 6). For acute causes, alcohol-attributable risk was estimated using drinker-specific relative risks calibrated in Formula 5, while for chronic causes, relative risks were taken directly from published dose-response meta-analyses. For wholly alcohol-attributable causes, the observed cause-specific mortality among drinkers was redistributed across consumption levels using a calibrated linear risk function. Within each age-sex group, the function was parameterised so that the average modelled risk reproduced the observed cause-specific mortality rate. Deaths were then allocated across consumption categories according to their relative risk and population size, with final rescaling to ensure that totals matched observed deaths.

Finally, cause-specific alcohol-attributable mortality risks were summed across all alcohol-related causes to obtain total alcohol-attributable mortality risk by age, sex and level of alcohol consumption (Formula 7).

These estimates represent one-year alcohol-attributable mortality risks among individuals alive at the start of each age interval and form the inputs to the life-course risk calculations in the subsequent step.

Formula 5

$$P_{LA_{a,s}} + P_{EXD_{a,s}} + \sum_{x=1}^{xn} \left(P_{D_{a,s}}(x) RR_{D_c}(x) \right) - RR_{POP_{a,s}} = 0$$

Where:

- P_{LA} is the prevalence of lifetime abstainers in age group *a* and sex *s*
- P_{EXD} is the prevalence of ex-drinkers (for whom the relative risk is assumed to be 1) in age group *a* and sex *s*
- $P_D(x)$ is the population share at *x* g/day among current drinkers in age group *a* and sex *s*
- $RR_D(x)$ is the condition-specific relative risk at *x* g/day
- RR_{POP} is the observed population relative risk for the acute outcome compared with a counterfactual of lifetime abstention in age group *a* and sex *s*.

Formula 6

$$Risk\_D\_AA_{a,s,c}(x) = Risk\_D\_LA_{a,s,c}.\left(RR_{a,s,c}(x) - 1\right)$$

Where:

- $Risk\_D\_AA_{a,s,c}(x)$ is the one-year alcohol-attributable mortality risk at *x* g/day for cause *c* in age group *a* and sex *s*
- $Risk\_D\_LA_{a,s,c}$ is the baseline risk of death for cause *c* in age group *a* and sex *s* under lifetime abstention
- $RR_{a,s,c}(x)$ is the relative risk of death at *x* g/day for condition *c*, by age and sex

Formula 7

$$Risk\_D\_AA_{a,s}(x) = \sum_{c=ci}^{cn} Risk\_D\_AA_{a,s,c}(x) + \sum_{c=ci}^{cn} Risk\_D\_WAA_{a,s,c}(x)$$

Where:

- $Risk\_D\_AA_{a,s}(x)$ is the overall one-year alcohol-attributable mortality risk at x g/day in age group a and sex s
- $Risk\_D\_AA_{a,s,c}(x)$ is the one-year alcohol-attributable mortality risk at x g/day for partially attributable cause c in age group a and sex s
- $Risk\_D\_WAA_{a,s,c}(x)$ is the one-year mortality risk at x g/day for wholly-attributable cause c in age group a and sex s

Step 4. Life-course estimation of lifetime mortality risk

To derive lifetime risks, a hypothetical birth cohort was constructed and followed across ages using age- and sex-specific one-year mortality risks observed in the population. Lifetime mortality risk was defined as one minus the cumulative survival probability across the life course.

For each age, sex, and level of average daily alcohol consumption, the proportion of the cohort alive at the end of each age interval was estimated recursively based on the proportion alive at the end of the previous age interval and the corresponding one-year mortality risks (Formula 8). These risks include both baseline all-cause mortality in the absence of alcohol and alcohol-attributable mortality risk. Alcohol-attributable mortality risk was set to zero below age 15. However, survival through earlier ages influences the number of individuals at risk of alcohol-related mortality later in life and was therefore incorporated in the life-course recursion.

The lifetime risk of an alcohol-attributable death was then estimated by summing age- and sex-specific alcohol-attributable annual mortality risks weighted by the proportion of the cohort alive at the end of the previous age interval (Formula 9).

Lifetime YLL attributable to alcohol were estimated by summing age- and sex-specific alcohol-attributable annual mortality risks weighted by the remaining life expectancy at each age, derived from CSO life tables,[34] and the proportion of the cohort alive at the end of the previous age interval (Formula 10).

These life-course estimates represent the cumulative risk of alcohol-attributable mortality and premature mortality associated with a given level of average daily alcohol consumption.

Formula 8

$$Alive_{a,s,x} = Alive_{a-1,s,x} \cdot \left[1 - \left(Risk\_D\_AA(x)_{a,s} + Risk\_D\_LA_{a,s}\right)\right]$$

Where:

- $Alive_{a,s,x}$ is the proportion of the cohort alive at the end of age interval a, given sex s and consumption level x

- *Risk_D_AA* is the one-year alcohol-attributable mortality risk at *x* g/day in age group *a* and sex *s*
- *Risk_D_LA* is the total risk of death among lifetime abstainers in age group *a* and sex *s*

Formula 9

$$Lifetime\_R\_Death(x)_s = \left[\sum_{a=0}^{n} Alive_{a,s,x} \cdot Risk\_D\_AA(x)_{a,s}\right] \cdot 1{,}000\ people$$

Where:

- *Lifetime_R_Death(x)* is the lifetime risk of death attributable to alcohol at consumption level *x*, per 1,000 people
- $Alive_{a,s,x}$ is the proportion of the cohort alive at the end of age interval *a*, given sex *s* and consumption level *x*
- *Risk_D_AA(x)* is the one-year alcohol-attributable mortality risk at *x* g/day in age group *a* and sex *s*

Formula 10

$$Lifetime\_R\_YLL(x)_s = \left[\sum_{a=0}^{n} Alive_{a,s,x} \cdot Risk\_D\_AA(x)_{a,s} \cdot YLL_{a,s}\right] \cdot 1{,}000\ people$$

Where:

- *Lifetime_R_YLL(x)* is the lifetime risk of YLL attributable to alcohol at consumption level *x*, per 1,000 people
- $Alive_{a,s,x}$ is the proportion of the cohort alive at the end of age interval a, given sex *s* and consumption level *x*
- *Risk_D_AA(x)* is the alcohol-attributable risk of death
- $YLL_{a,s}$ is the remaining life expectancy at age *a*, derived from CSO life tables

Appendix 2. AMSTAR 2 assessments of meta-analyses included in the systematic review

Table A.1 AMSTAR 2 assessment for Llamosas-Falcón et al. (2024) and Llamosas-Falcón et al. (2022)

AMSTAR 2 Question	Llamosas-Falcón et al.[15] (2024)	Llamosas-Falcón et al.[16] (2022)
1. Did the research questions and inclusion criteria for the review include the components of PICO (Population, Intervention, Comparison and Outcomes)?	Yes	Yes
2. Did the report of the review contain an explicit statement that the review methods were established prior to the conduct of the review and did the report justify any significant deviations from the protocol?	Yes	Yes
3. Did the review authors explain their selection of the study designs for inclusion in the review?	No	No
4. Did the review authors use a comprehensive literature search strategy?	Partial yes	Partial yes
5. Did the review authors perform study selection in duplicate?	No	Yes
6. Did the review authors perform data extraction in duplicate?	No	No
7. Did the review authors provide a list of excluded studies and justify the exclusions?	No	No
8. Did the review authors describe the included studies in adequate detail?	Partial yes	Partial yes
9. Did the review authors use a satisfactory technique for assessing the risk of bias (RoB) in individual studies that were included in the review?	Yes	Yes
10. Did the review authors report on the sources of funding for the studies included in the review?	No	No
11. If meta-analysis was performed did the review authors use appropriate methods for statistical combination of results?	Yes	Yes
12. If meta-analysis was performed, did the review authors assess the potential impact of RoB in individual studies on the results of the meta-analysis or other evidence synthesis?	Yes	Yes
13. Did the review authors account for RoB in individual studies when interpreting/ discussing the results of the review?	Yes	Yes

AMSTAR 2 Question	Llamosas-Falcón et al.[15] (2024)	Llamosas-Falcón et al.[16] (2022)
14. Did the review authors provide a satisfactory explanation for, and discussion of, any heterogeneity observed in the results of the review?	Yes	Yes
15. If they performed quantitative synthesis did the review authors carry out an adequate investigation of publication bias (small study bias) and discuss its likely impact on the results of the review?	No	No
16. Did the review authors report any potential sources of conflict of interest, including any funding they received for conducting the review?	Yes	Yes

Table A.2 AMSTAR 2 assessment for Llamosas-Falcón et al. (2023)

AMSTAR 2 Question	Answer
1. Did the research questions and inclusion criteria for the review include the components of PICO (Population, Intervention, Comparison and Outcomes)?	Yes
2. Did the report of the review contain an explicit statement that the review methods were established prior to the conduct of the review and did the report justify any significant deviations from the protocol?	Yes
3. Did the review authors explain their selection of the study designs for inclusion in the review?	No
4. Did the review authors use a comprehensive literature search strategy?	Partial yes
5. Did the review authors perform study selection in duplicate?	Yes
6. Did the review authors perform data extraction in duplicate?	Yes
7. Did the review authors provide a list of excluded studies and justify the exclusions?	No
8. Did the review authors describe the included studies in adequate detail?	Yes
9. Did the review authors use a satisfactory technique for assessing the risk of bias (RoB) in individual studies that were included in the review?	Yes
10. Did the review authors report on the sources of funding for the studies included in the review?	No
11. If meta-analysis was performed did the review authors use appropriate methods for statistical combination of results?	Yes
12. If meta-analysis was performed, did the review authors assess the potential impact of RoB in individual studies on the results of the meta-analysis or other evidence synthesis?	Yes

AMSTAR 2 Question	Answer
13. Did the review authors account for RoB in individual studies when interpreting/ discussing the results of the review?	Yes
14. Did the review authors provide a satisfactory explanation for, and discussion of, any heterogeneity observed in the results of the review?	Yes
15. If they performed quantitative synthesis did the review authors carry out an adequate investigation of publication bias (small study bias) and discuss its likely impact on the results of the review?	No
16. Did the review authors report any potential sources of conflict of interest, including any funding they received for conducting the review?	Yes

Table A.3 AMSTAR 2 assessment for Zhang et al.

AMSTAR 2 Question	Answer
1. Did the research questions and inclusion criteria for the review include the components of PICO (Population, Intervention, Comparison and Outcomes)?	Yes
2. Did the report of the review contain an explicit statement that the review methods were established prior to the conduct of the review and did the report justify any significant deviations from the protocol?	No
3. Did the review authors explain their selection of the study designs for inclusion in the review?	Yes
4. Did the review authors use a comprehensive literature search strategy?	Partial yes
5. Did the review authors perform study selection in duplicate?	No
6. Did the review authors perform data extraction in duplicate?	Yes
7. Did the review authors provide a list of excluded studies and justify the exclusions?	No
8. Did the review authors describe the included studies in adequate detail?	Yes
9. Did the review authors use a satisfactory technique for assessing the risk of bias (RoB) in individual studies that were included in the review?	Yes
10. Did the review authors report on the sources of funding for the studies included in the review?	No
11. If meta-analysis was performed did the review authors use appropriate methods for statistical combination of results?	Yes
12. If meta-analysis was performed, did the review authors assess the potential impact of RoB in individual studies on the results of the meta-analysis or other evidence synthesis?	Yes
13. Did the review authors account for RoB in individual studies when interpreting/ discussing the results of the review?	Yes
14. Did the review authors provide a satisfactory explanation for, and discussion of, any heterogeneity observed in the results of the review?	Yes

AMSTAR 2 Question	Answer
15. If they performed quantitative synthesis did the review authors carry out an adequate investigation of publication bias (small study bias) and discuss its likely impact on the results of the review?	Yes
16. Did the review authors report any potential sources of conflict of interest, including any funding they received for conducting the review?	Yes

Table A.4 AMSTAR 2 assessment for Sohi et al.

AMSTAR 2 Question	Answer
1. Did the research questions and inclusion criteria for the review include the components of PICO (Population, Intervention, Comparison and Outcomes)?	Yes
2. Did the report of the review contain an explicit statement that the review methods were established prior to the conduct of the review and did the report justify any significant deviations from the protocol?	Yes
3. Did the review authors explain their selection of the study designs for inclusion in the review?	No
4. Did the review authors use a comprehensive literature search strategy?	Partial yes
5. Did the review authors perform study selection in duplicate?	Yes
6. Did the review authors perform data extraction in duplicate?	Yes
7. Did the review authors provide a list of excluded studies and justify the exclusions?	No
8. Did the review authors describe the included studies in adequate detail?	Yes
9. Did the review authors use a satisfactory technique for assessing the risk of bias (RoB) in individual studies that were included in the review?	Yes
10. Did the review authors report on the sources of funding for the studies included in the review?	No
11. If meta-analysis was performed did the review authors use appropriate methods for statistical combination of results?	Yes
12. If meta-analysis was performed, did the review authors assess the potential impact of RoB in individual studies on the results of the meta-analysis or other evidence synthesis?	No
13. Did the review authors account for RoB in individual studies when interpreting/ discussing the results of the review?	No
14. Did the review authors provide a satisfactory explanation for, and discussion of, any heterogeneity observed in the results of the review?	Yes
15. If they performed quantitative synthesis did the review authors carry out an adequate investigation of publication bias (small study bias) and discuss its likely impact on the results of the review?	No

AMSTAR 2 Question	Answer
16. Did the review authors report any potential sources of conflict of interest, including any funding they received for conducting the review?	Yes

Appendix 3. Cause-specific relative risks for mortality by average weekly consumption among females

This appendix presents cause-specific relative risks for mortality among females by average daily alcohol consumption (g/day). Colours indicate the percentage change in risk compared with the reference category, lifetime abstention.

Atrial fibrillation	0%	2.5%	6.3%	8.6%	9.9%	9.8%	9.8%	9.8%
Breast cancer	2.4%	7.4%	12.2%	16.2%	19.1%	21%	22.3%	23.2%
Colorectal cancer	1%	3%	5%	7.1%	9.2%	11.4%	13.6%	15.8%
Diabetes	-9.5%	-23.9%	-30.6%	-31.2%	-29%	-26.7%	-24.5%	-22.5%
Drowning	3.9%	12.1%	20.6%	29.7%	39.1%	49.1%	59.5%	70.5%
Epilepsy	3.8%	10.3%	17.3%	24.8%	32.7%	41.1%	50%	59.5%
Exposure to mechanical forces	4.3%	13.3%	22.7%	32.6%	43%	53.9%	65.4%	77.5%
Falls	2.6%	7.9%	13.5%	19.5%	25.7%	32.2%	39.1%	46.3%
Fire	3.2%	9.8%	16.7%	24%	31.6%	39.7%	48.1%	57%
Hypertension	1.5%	4.5%	7.4%	10.3%	13.3%	16.4%	19.7%	23.1%
Interpersonal violence	3.6%	10.9%	18.7%	26.8%	35.4%	44.4%	53.8%	63.8%
Intracerebral haemorrhage	-8%	-8%	-1%	-1%	-1%	25%	25%	25%
Ischemic heart disease	-5%	-5%	-5%	-5%	-5%	4%	4%	4%
Ischemic stroke	-8%	-8%	-8%	-8%	-8%	8%	8%	8%
Larynx cancer	3.7%	11.5%	19.7%	28.4%	37.6%	47.2%	57.5%	68.2%
Liver cancer	4.5%	13.9%	24.3%	35.6%	47.9%	61.3%	76%	92%
Liver cirrhosis	16.3%	56.9%	112.4%	188.4%	290.6%	423.3%	587.9%	780.4%
Oesophageal cancer	3.4%	10.4%	17.9%	26%	34.5%	43.7%	53.4%	63.7%
Oropharyngeal cancer	6.4%	20.1%	35.4%	52.3%	71%	91.6%	114.2%	139%
Other poisoning	4.6%	14.2%	24.3%	34.9%	46%	57.8%	70.1%	83%
Other unintentional injuries	3.2%	9.8%	16.8%	24.1%	31.8%	39.8%	48.3%	57.3%
Pancreatitis (acute)	-6.6%	-18.2%	-26.1%	-29%	-26.7%	-20%	-9%	6%
Pancreatitis (chronic)	4.7%	14.7%	25.8%	37.8%	51.1%	65.6%	81.5%	98.9%
Pneumonia	1.2%	3.7%	6.2%	8.7%	11.3%	14%	16.8%	19.6%
Road/Transport injuries	6%	18.2%	31.2%	44.8%	59.1%	74.1%	89.9%	106.5%
Self-harm	3.8%	11.7%	20.1%	28.8%	38.1%	47.7%	57.9%	68.6%
Subarachnoid haemorrhage	21%	21%	11%	11%	11%	39%	39%	39%
Tuberculosis	4.6%	14.4%	25.2%	37%	49.8%	63.9%	79.3%	96.2%

0 5 10 15 20 25 30 35 40
Alcohol consumption (g/day)

% Change
>50%
41–50%
31–40%
21–30%
16–20%
11–15%
6–10%
0–5%
<0%

Appendix 4. Cause-specific relative risks for mortality by average weekly consumption among males

This appendix presents cause-specific relative risks for mortality among males by average daily alcohol consumption (g/day). Colours indicate the percentage change in risk compared with the reference category, lifetime abstention.

Atrial fibrillation	0%	2.5%	6.3%	8.6%	9.9%	9.8%	9.8%	9.8%
Colorectal cancer	2%	5.9%	10.1%	14.4%	18.9%	23.6%	28.4%	33.5%
Diabetes	0.2%	0.7%	1.1%	1.6%	2.1%	2.5%	3%	3.5%
Drowning	3.9%	12%	20.4%	29.4%	38.7%	48.6%	59%	69.8%
Epilepsy	3.8%	10.3%	17.3%	24.8%	32.7%	41.1%	50%	59.5%
Exposure to mechanical forces	3.7%	11.4%	19.4%	27.9%	36.8%	46.2%	56%	66.4%
Falls	3.1%	9.5%	16.2%	23.3%	30.7%	38.6%	46.8%	55.4%
Fire	3.3%	10%	17.1%	24.5%	32.3%	40.6%	49.2%	58.3%
Hypertension	3.6%	11.1%	17%	21.1%	25.3%	29.7%	33%	34.9%
Interpersonal violence	3.2%	9.9%	17%	24.4%	32.1%	40.3%	48.9%	57.9%
Intracerebral haemorrhage	-8%	-8%	-1%	-1%	-1%	25%	25%	25%
Ischemic heart disease	-5%	-5%	-5%	-5%	-5%	4%	4%	4%
Ischemic stroke	-8%	-8%	-8%	-8%	-8%	8%	8%	8%
Larynx cancer	3.7%	11.5%	19.7%	28.4%	37.6%	47.2%	57.5%	68.2%
Liver cancer	0.7%	2.2%	3.8%	5.3%	6.9%	8.5%	10.1%	11.7%
Liver cirrhosis	5.6%	17.7%	32.2%	50.2%	74%	106.1%	147.8%	200.4%
Oesophageal cancer	3.4%	10.4%	17.9%	26%	34.5%	43.7%	53.4%	63.7%
Oropharyngeal cancer	6.4%	20.1%	35.4%	52.3%	71%	91.6%	114.2%	139%
Other poisoning	4.2%	13%	22.1%	31.8%	42%	52.6%	63.9%	75.7%
Other unintentional injuries	3.4%	10.5%	17.9%	25.7%	33.9%	42.5%	51.6%	61.2%
Pancreatitis (acute)	4.5%	13.9%	24.2%	35.5%	47.7%	61.1%	75.7%	91.6%
Pancreatitis (chronic)	4.7%	14.7%	25.8%	37.8%	51.1%	65.6%	81.5%	98.9%
Pneumonia	1.2%	3.7%	6.2%	8.7%	11.3%	14%	16.8%	19.6%
Road/Transport injuries	6.6%	20.1%	34.4%	49.3%	65.1%	81.7%	99.1%	117.4%
Self-harm	3.2%	9.7%	16.6%	23.8%	31.4%	39.4%	47.8%	56.6%
Subarachnoid haemorrhage	21%	21%	11%	11%	11%	39%	39%	39%
Tuberculosis	4.6%	14.4%	25.2%	37%	49.8%	63.9%	79.3%	96.2%

0 5 10 15 20 25 30 35 40
Alcohol consumption (g/day)

% Change
>50%
41–50%
31–40%
21–30%
16–20%
11–15%
6–10%
0–5%
<0%

Appendix 5. Tabulated estimates of lifetime risk of alcohol-attributable premature death by sex and average daily alcohol consumption

This appendix presents the full tabulated estimates and 95% uncertainty intervals for lifetime risk of alcohol-attributable premature death per 1,000 lifetimes, by sex and average daily alcohol consumption, corresponding to Figure 3.2.

Alcohol consumption		Males	Females
g/day	Standard drinks/week	Lifetime risk per 1,000 lifetimes (95% uncertainty interval)	Lifetime risk per 1,000 lifetimes (95% uncertainty interval)
0	0	0.0 (0.0 to 0.0)	0.0 (0.0 to 0.0)
1	0.7	0.0 (-2.0 to 2.2)	0.2 (-0.4 to 0.9)
2	1.4	-0.1 (-4.0 to 4.3)	0.4 (-0.8 to 1.8)
3	2.1	0.2 (-4.7 to 5.8)	0.9 (-0.7 to 2.6)
4	2.8	0.9 (-4.0 to 6.4)	1.6 (0.0 to 3.3)
5	3.5	1.6 (-3.3 to 7.1)	2.3 (0.7 to 4.0)
6	4.2	2.3 (-2.6 to 7.8)	2.9 (1.4 to 4.7)
7	4.9	2.9 (-1.8 to 8.5)	3.6 (2.1 to 5.4)
8	5.6	3.6 (-1.1 to 9.2)	4.3 (2.8 to 6.2)
9	6.3	4.3 (-0.4 to 9.9)	5.1 (3.5 to 6.9)
10	7	5.0 (0.3 to 10.6)	5.8 (4.2 to 7.6)
11	7.7	5.7 (0.9 to 11.3)	6.5 (5.0 to 8.4)
12	8.4	6.4 (1.6 to 12.1)	7.3 (5.7 to 9.1)
13	9.1	7.1 (2.3 to 12.8)	8.0 (6.4 to 9.9)
14	9.8	7.8 (3.0 to 13.5)	8.8 (7.2 to 10.6)
15	10.5	8.5 (3.7 to 14.2)	9.5 (7.9 to 11.4)
16	11.2	9.3 (4.4 to 14.9)	10.3 (8.6 to 12.2)
17	11.9	10.0 (5.1 to 15.6)	11.0 (9.4 to 12.9)
18	12.6	10.7 (5.8 to 16.3)	11.8 (10.1 to 13.7)
19	13.3	11.4 (6.5 to 17.0)	12.6 (10.9 to 14.5)
20	14	12.1 (7.2 to 17.7)	13.3 (11.6 to 15.2)
21	14.7	12.8 (8.0 to 18.4)	14.1 (12.4 to 16.0)
22	15.4	13.6 (8.7 to 19.1)	14.9 (13.2 to 16.8)
23	16.1	14.7 (9.8 to 20.4)	15.8 (14.1 to 17.8)
24	16.8	16.3 (11.2 to 22.1)	17.0 (15.2 to 19.0)
25	17.5	17.8 (12.7 to 23.9)	18.1 (16.2 to 20.3)
26	18.2	19.4 (14.1 to 25.7)	19.3 (17.3 to 21.5)
27	18.9	21.0 (15.6 to 27.4)	20.5 (18.4 to 22.8)
28	19.6	22.1 (16.7 to 28.7)	21.4 (19.3 to 23.8)
29	20.3	22.9 (17.5 to 29.5)	22.3 (20.1 to 24.7)
30	21	23.8 (18.4 to 30.3)	23.1 (20.9 to 25.5)
31	21.7	24.6 (19.2 to 31.0)	23.9 (21.7 to 26.4)
32	22.4	25.4 (20.1 to 31.8)	24.7 (22.5 to 27.2)

Alcohol consumption		Males	Females
g/day	Standard drinks/week	Lifetime risk per 1,000 lifetimes (95% uncertainty interval)	Lifetime risk per 1,000 lifetimes (95% uncertainty interval)
33	23.1	26.2 (20.9 to 32.6)	25.5 (23.3 to 28.1)
34	23.8	27.0 (21.7 to 33.4)	26.4 (24.1 to 29.0)
35	24.5	27.8 (22.5 to 34.3)	27.2 (24.9 to 30.0)
36	25.2	28.7 (23.3 to 35.1)	28.1 (25.7 to 30.9)
37	25.9	29.5 (24.1 to 35.9)	29.0 (26.6 to 31.8)
38	26.6	30.3 (24.9 to 36.8)	29.8 (27.4 to 32.7)
39	27.3	31.2 (25.7 to 37.6)	30.7 (28.2 to 33.7)
40	28	32.1 (26.5 to 38.5)	31.6 (29.1 to 34.6)

Note: Cells are colour-coded to reflect the magnitude of the estimated lifetime risk: white indicates <1 per 1,000, yellow indicates 1 to <10 per 1,000, and red indicates ≥10 per 1,000. Standard drinks/week assumes 10 g of pure alcohol per standard drink.

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