Binge Drinking

Acute Consequences and Lasting Impacts

ALCOHOL AND SOCIETY 2026

A REVIEW OF INTERNATIONAL AND SWEDISH RESEARCH

by Harold Holder, Tim Naimi, Tim Stockwell, Sven Andréasson, Tanya Chikritzhs, Frida Dangardt

Organisations initiating this report are voluntary or academic organisations independent of commercial interests.

Views expressed in this report are those of the authors and do not necessarily reflect those of the organisations that initiate the work.

Suggested citation (authors in rotating alphabetic order): "Holder H, Naimi T, Stockwell T, Andréasson S, Chikritzhs T, Dangardt F. (2026) Binge Drinking: Acute Consequences and Lasting Impacts. Alcohol and Society 2026. Stockholm: Svensk Sjuksköterskeförening, SFAM, SAFF, CERA IOGT i Norge, Hjärnfonden, Movendi International, SLAN, KSAN, NSF, KF Länken, FAS-föreningen, Frisksport, Sveriges Blåbandsungdom, Junis, UNF & IOGT-NTO.

A Swedish language version of this report is also available from **https://alcoholandsociety.report/sv/home-swe/**
Graphic design: Petra Handin, Poppi Design
Printers: Fridholm & Partners AB, Göteborg

ISBN: 978-91-988422-9-6 (print version in Swedish)
ISBN: 978-91-991213-0-7 (pdf in Swedish)
ISBN: 978-91-991213-1-4 (pdf in English)
URN: urn:nbn:se:iogt-2026-aos-sv

Published with support from Stiftelsen Ansvar för Framtiden (SAFF).

Contents

Executive Summary 4

Authors 6

1 Introduction: What is binge drinking and why is it a problem? 9

2 Mechanisms 12

3 Drinking patterns and their prevalence 15

3.1 Binge drinking and its relationship to a population's total alcohol consumption 15

3.2 Binge drinking prevalence 16

3.3 Binge drinking among adolescents and young adults 18

4 Risks from Binge Drinking 20

4.1 Blood alcohol concentration 20

4.2 Age: younger and older drinkers 22

4.3 Sex and gender 23

4.4 Socioeconomic status (SES) 23

4.5 Maternal and paternal drinking 24

5 Acute harms 25

5.1 Road crash injuries 25

5.2 Unintentional injuries 26

5.3 Intentional injuries fromaggression, assault, and homicide 26

5.4 Sexual aggression and binge drinking 26

5.5 Harm to children 27

5.6 Alcohol poisoning 27

6 Chronic harms 28

6.1 All-cause mortality 28

6.2 Cardiovascular disease (CVD) and stroke 28

6.3 Dementia and cognitive impairment 29

6.4 Mental health 29

6.5 Cancers 30

6.6 Diabetes 30

6.7 Gastrointestinal disease, including liver disease 31

6.8 Foetal alcohol syndrome (FAS) and Foetal alcohol spectrum disorders (FASD) 32

7 Strategies/interventions for reducing harms from binge drinking 34

7.1 Public policies 34

7.2 Treatment and individual level interventions 36

8 Conclusions and recommendations 39

8.1 Why it matters 39

8.2 Health risks 39

8.3 Social and economic costs 40

8.4 Who is most at risk? 40

8.5 What works to reduce binge drinking? 41

8.6 Key takeaway and recommendations 41

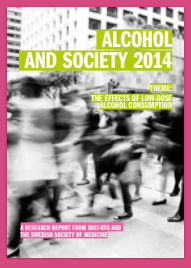
References 42

Executive Summary

- Binge drinking is typically defined as drinking 5 or more drinks per occasion. This is sometimes modified to 4 or more drinks per women or may be defined by grams of ethanol (e.g., 60 grams) given the variability in standard drink sizes internationally.
- In recent decades overall levels of binge drinking in Sweden, Norway and Denmark have been steady at between 30 and 35% but with some divergent trends by age and gender.
- While the major contribution to road crashes, violence and poisoning (i.e., acute effects) is widely understood, the enduring effects of binge drinking on dementia, alcohol dependence and chronic disease in later life are largely overlooked.
- Rapid alcohol use on a binge drinking occasion overwhelms the body's ability to process alcohol, leading to oxidative stress and to an increase in acetaldehyde, a dangerous metabolite and a major carcinogen.
- The body's increased exposure to high blood alcohol concentrations, to acetaldehyde concentrations and to oxidative stress leads to impairments in the short-term and risks enduring toxic and carcinogenic impacts on all organs.
- The extent of binge drinking is largely underestimated. Among people who drink within 'moderate' levels on average (e.g., up to one or to drinks per day), the majority also engage in occasional binge drinking.

THE RESEARCH REPORTS SERIES ALCOHOL AND SOCIETY

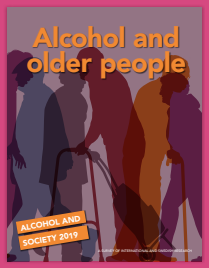






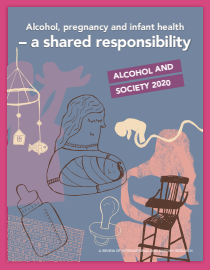




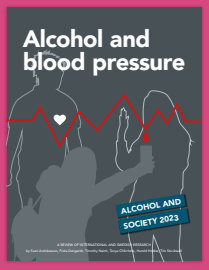


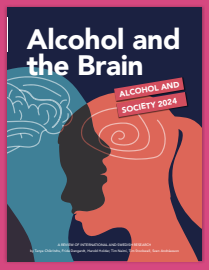
All previous research reports are available for download at **alcoholandsociety.report/the-research-report-series**

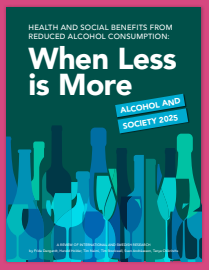
- Rates of binge drinking decline and fall with total alcohol consumption in a population. This is because 5+ drinking occasions account for a at least half of consumption in high income countries such as Sweden. As a result, policies that reduce total consumption also reduce rates of binge drinking and related harms.
- Fortunately, there are many effective strategies that reduce binge drinking and its consequences, including for those with established or early-stage alcohol use disorders. Examples of effective population-level policies include higher alcohol taxes, minimum unit pricing, restrictions on sales hours, reduced outlet density, marketing limits, minimum drinking age laws and , drink-driving enforcement, and liability laws for serving intoxicated patrons.
- Effective interventions for individuals include brief counselling in health care settings, medications, treatments for alcohol use disorder and use of devices such as ignition interlocks and alcohol detection systems in cars (that reduce drink driving).
- A cultural shift in societal norms and attitudes towards binge drinking throughout Sweden and other Nordic countries is in order. Future nutritional guidelines could include specific recommendations for avoiding binge drinking, in addition to recommended limits for total consumption.











Authors



Sven Andréasson is professor emeritus in social medicine at the Department of Global Health, Karolinska Institutet, Stockholm. He is also senior consultant at the health center Riddargatan 1 at the Stockholm Center for Dependency Disorders.

His research covers alcohol and drug epidemiology and studies on prevention and treatment of alcohol and drug problems.



Professor **Tanya Chikritzhs** leads the Alcohol Policy Research team at the National Drug Research Institute, Curtin University, Perth, Australia. She is Principal Investigator for high profile national projects such as the National Alcohol Indicators Project (NAIP) and the National Alcohol Sales Data project. The NAIP is Australia's central source of authoritative information on the epidemiology of alcohol in Australia and serves as a fundamental information base for the National Alcohol Strategies.

She has qualifications in epidemiology and biostatistics, some 20 years experience in alcohol research and a national profile as an expert in her field. Her research covers many areas of alcohol policy and alcohol epidemiology, such as alcohol consumption, alcohol related harms, alcohol taxation, liquor licensing, alcohol and heart disease, and alcohol and cancer.

She has received many awards including the prestigious Commonwealth Health Ministers Award for Excellence in Health and Medical Research and an NHMRC Achievement Award (1st ranked in Population Health).



Frida Dangardt is Associate Professor/Senior Lecturer and Senior Consultant, at Children's Heart Centre, Queen Silvia Children's Hospital, Gothenburg. Frida Dangardt received her medical degree 2005 and PhD degree 2008 at Sahlgrenska Academy at the University of Gothenburg. She was a Post-Doctoral Research Fellow at the National Centre for Cardiovascular Prevention and Outcomes, Vascular Physiology Unit, Institute of Cardiovascular Sciences, University College of London, UK, 2012 to 2014. Her research covers development and prevention of cardiovascular disease in children and youth, with focus on congenital heart disease, chronic disease, child obesity, mental stress and alcohol consumption.



Harold Holder, Ph.D., is a Senior Research Scientist Emeritus and the former Director of the Prevention Research Center (PRC) of the Pacific Institute for Research and Evaluation, a national center for prevention research, located in Berkeley, California, USA.

Dr. Holder holds a doctorate in communication science and mathematical sociology from Syracuse University. He has explored two major alcohol research areas: the prevention of substance abuse, and the cost and benefits of alcoholism and drug abuse treatment and published work on the impact of changes in retail sales of wine and spirits on drinking and alcohol-involved traffic crashes. His policy studies also include assessments of the prevention potential of alcohol server liability, mandated server training, and environmental strategies as part of comprehensive approaches to prevention. Dr. Holder has undertaken a series of collaborative

studies in the Nordic Countries to study the effects of public policies. These collaborations with researchers from Sweden, Norway, and Finland concern the role and changes in alcohol policy resulting from membership or association in the European Union. In addition, Dr. Holder has participated with prevention scientists from a dozen countries in international projects to document the effects of alcohol policy. The projects have produced three books in which he was a co-author, Alcohol Policy and the Public Good (1994), Alcohol: no ordinary commodity – Research and public policy (2003) and Alcohol: no ordinary commodity, second edition (2010). His most recent professional work has entailed working with a number of U.S. states and local communities on the application of prevention science to practice.

Recently Dr. Holder chaired an international research group in an evaluation of Swedish research on alcohol, narcotics, doping, tobacco and gambling for the Swedish Council for Working Life and Social Research. The evaluation report was published in 2012.

Dr. Holder has received the 1995 Jellinek Memorial Award, awarded for distinction gained by advancing knowledge about alcoholism or fostering its study, treatment, or prevention.



Timothy Naimi, MD, MPH is currently the Director at the Canadian Institute for Substance Use Research, University of Victoria, BC, Canada.He received his bachelor's degree from Harvard College, his M.D. degree from the University of Massachusetts, and his M.P.H degree from the Harvard School of Public Health. He completed a combined internal medicine-pediatrics residency program at Massachusetts General Hospital, the Epidemic Intelligence Service program with the Centers for Disease Control and Prevention (CDC), and a Preventive Medicine Residency with CDC. He has worked as a physician for the U.S. Indian Health Service, and as a senior epidemiologist with the Alcohol Team at CDC, and a professor in the Boston University Schools of Public Health and Medicine. His research interests include alcohol epidemiology, the health effects of substance use, and the impact of alcohol and cannabis policies.



Tim Stockwell is scientist at, and was Director from 2004 to 2020 of, the Canadian Institute for Substance Use Research (formerly the Centre for Addictions Research of BC), University of Victoria, BC, Canada. He was previously Director of Australia's National Drug Research Institute and Director of Australia's Alcohol Education and Research Foundation. He has served as a member of Canada's National Alcohol Strategy Advisory Committee and of WHO's Technical Advisory Groups on a) alcohol and drug epidemiology b) alcohol labelling.

Tim Stockwell holds degrees from Oxford University (MA Hons, Psychology and Philosophy), University of Surrey (MSc Clinical Psychology) and the University of London (PhD Institute of Psychiatry). His research has covered many aspects of substance use policy, prevention, treatment methods, liquor licensing issues, taxation and the measurement of drinking patterns and their consequences.

Tim Stockwel is a Fellow of the Royal Society of Canada and past recipient of the 2013 international E.M. Jellinek Memorial Award for Outstanding Research on Alcohol Policy.



1 Introduction: What is binge drinking and why is it a problem?

Binge drinking is defined as the fast consumption of large volumes of alcohol. Alcohol use in general and binge drinking in particular contribute to a variety of medical, social and economic outcomes. It is critically important to understand the relationship between alcohol use (i.e., the dose of ethanol) and these outcomes. Alcohol use is measured in several ways. The most common ways are to measure total use of alcohol at a population level per person (usually 15+ years) and average use per person, (e.g, number of drinks per day). Most studies of the impacts of alcohol consumption are based on total use measures or their derivatives.

However, patterns of use are also critically important, but are under-appreciated and are perhaps not coincidentally, under-studied. Patterns of use relate to how alcohol use is clustered over time – namely how much alcohol is used per day and/or per drinking occasion and also how frequently alcohol is consumed. The amount one consumes when actually drinking is referred to as "per-occasion" consumption. Even within a particular level of average consumption, there is great variability in terms of how different people spread out their drinks over time. For example, a person who drinks 7 drinks per week on average may have just one every day or all 7 on one drinking occasion. As will be shown later in this report, in most countries at least half of all alcohol purchased for consumption in a country is actually consumed in a binge drinking pattern.

Higher per-occasion consumption correlates closely with the degree of impairment from alcohol from a high blood alcohol concentration (BAC). Alcohol impairment refers to an acquired inability, or decrement in ability, to perform cognitive and physical tasks. Binge drinking (also referred to as 'heavy episodic drinking'), is a common threshold of per-occasion consumption used in research which typically results in BACs of 0.8‰ (0.08%) or greater. Such a BAC will be achieved by the average male after consuming 6 standard drinks (= 72g of ethanol in Sweden) and 5 standard drinks by a female of average weight. However, while this is a useful threshold, it is important to understand that decrements in impairment begin below BAC levels that typically are encountered at the binge drinking threshold.

In North American research, binge drinking is usually defined as the consumption of four or more drinks in one day for a woman and five or more for men with one "drink" defined as containing approximately 14g of pure alcohol. Some researchers also employ



In most countries at least half of all alcohol purchased for consumption in a country is actually consumed in a binge drinking pattern.



The term intoxication refers to situations in which there is visible (to others) or subjective (to the drinker him or herself) effects of alcohol. However, objective measures of impairment begin well before intoxication is apparent.

a stricter, more time-limited definition where these amounts are consumed within two hours, a time limit required to reach a BAC level of 0.8‰ (0.08%). The sex difference in the definition is intended to reflect the finding that females typically obtain higher blood alcohol concentrations in experimental studies from a fixed alcohol dose compared with males by virtue of lower bodyweight and slower metabolism.

Another tradition found in European and international research is to define binge drinking as the consumption of four or more standard drinks for both sexes. In Sweden this amounts to 48g of ethanol in a day, a threshold inspired by recent Australian drinking guidelines, which were even lower, at 4 or more 10 g standard drinks. It is reasoned variously that bodyweight rather than sex is the principle reason for individual variations in blood alcohol concentrations and, as well, females tend to drink alcohol at a slower pace than males.

In the present report we recognise that there is no risk-free level of alcohol use and that injury risk increases after consumption of just one or two drinks and proceeds with an exponential increase in risk at higher levels (e.g. [1]). The definition of exceeding 60g of ethanol during a drinking occasion will be our default in this report as it is both widely used and provides an effective contrast between higher and lower risk drinking patterns. We will, however, note exact definitions used in key studies we describe in this report. Again, we note all of these precise-sounding thresholds are somewhat arbitrary as there is an underlying continuum of risk that starts with any alcohol use and which then increases rapidly with increasing consumption and BAC level.

The term intoxication refers to situations in which there is visible (to others) or subjective (to the drinker him or herself) effects of alcohol. However, objective measures of impairment begin well before intoxication is apparent. In other words, everyone who appears intoxicated is impaired, but not everyone who is impaired is intoxicated.

High per-occasion consumption, particularly above the family of thresholds used to define binge drinking described above, is the key risk factor for "acute" outcomes such as unintentional injuries, interpersonal violence and social problems including child abuse and neglect, and crimes such as vandalism and assault. However, per-occasion consumption also increases the risk for a range of chronic diseases such as heart attacks, strokes, diabetes and dementia, and modifies relationships between average consumption and chronic disease outcomes. This contribution of binge drinking to chronic disease comes in two forms: first, binge drinking occasions constitute a large fraction of total consumption in the population; and second there may be BAC-related damage when high BACs overwhelm physiological protective mechanisms (see Section 2).

The risks from high per-occasion consumption varies by age. Youth and young adults have rapidly developing brains, higher levels of impulsivity and, usually, low tolerance and so are particularly vulnerable to alcohol's effects. In addition to being a risk factor for a variety of acute and chronic diseases, binge drinking in younger years is a very strong risk factor for the subsequent development of an alcohol use disorder (AUD). Virtually all persons with an AUD engage in binge drinking and/or have high BACs. Furthermore, it has been shown that an inability to stop drinking after two drinks is one of the earliest signs of a developing alcohol use disorder.[2]

There are also sex and gender considerations. Men generally have a higher prevalence of binge drinking compared to women, and drink more during binge drinking occasions. Women generally weigh less and metabolize alcohol more slowly, so a comparable amount of alcohol might lead to more impairment among women. Social expectancies and other factors may also alter the drinking context and risk – women may be at more risk of harms in terms of victimization in terms of interpersonal violence, including sexual aggression, or consequences related to, for example, unintended preg-

nancy. On the other hand, men may act more aggressively or impulsively leading to more adverse consequences for themselves and others, particularly with respect to violence and unintentional injuries.

In this report, the focus will be on describing patterns of per-occasion consumption and reviewing relationships between per occasion consumption and a variety of health and social outcomes. Because of its widespread use in the population, role as a powerful risk factor for multiple acute and chronic medical conditions, binge drinking might well be framed as perhaps the highest priority for prevention and public health interventions around alcohol and health. It is important to further emphasise the enduring impacts of binge drinking across the life-course as a result of more intense exposure to toxic and carcinogenic effects of high BAC drinking episodes, especially when drinking speed is above one drink per hour. We therefore conclude the report with evidence-based recommendations about how to prevent or reduce per-occasion consumption amongst individuals and within the general population.

BOX 1

This report, as with all others, is undertaken in two major steps. First, an extensive search is completed to identify relevant published science. Second, the search results are reviewed and summarized. This is done by in-person discussions and evaluation of the strength of scientific methods of studies by the group as well as opportunity to discuss each written draft in total as a group. In the end, all authors contribute writing, reviews, and edits to all sections of the report. The result is truly a group product extending over several months.

BOX 2 TYPES OF SCIENTIFIC EVIDENCE

Scientific studies considered in this report are of following types:

- **Mendelian randomization studies** are those that rely on genetic variants that are related to alcohol consumption in order to indirectly study effects of alcohol consumption.
- **Randomized controlled trials** "flip a coin" to randomly assign one group of participants to an intervention and another to not partaking of that intervention (e.g. experimental lab studies that administer alcohol to participants in the intervention group).
- **Observational studies** generally use data obtained from non-randomly "observing" participant self-reports of alcohol use to investigate whether drinking affects risk of mortality or morbidity from various diseases.
- **Neuroimaging studies** use non-invasive imaging technology (e.g. Magnetic Resonance Imagining [MRI]) to study the structure and function of the brain and central nervous system under certain conditions (e.g. presence or absence of alcohol).

Each type of study brings its own strengths and weaknesses. As discussed in previous reports, non-randomized observational studies are particularly prone to finding spurious protective effects from low-to-moderate alcohol intake for conditions that are more likely to occur in middle and older age.



2 Mechanisms

Binge drinking has long been recognised as a particularly harmful form of alcohol consumption. It is primarily this form of drinking, with its social and behavioural consequences, that has led to the regulation of drinking in most countries in the world. In more recent research, binge drinking is also found to exert a profound impact on human physiology and pathology.

Binge drinking is a pattern of drinking that involves the rapid consumption of large volumes of alcohol. The key difference between binge drinking and regular high consumption is the rate of consumption, where rapid consumption leads to a rapid rise in BAC, which the detoxification enzymes can't cope with.

Alcohol is a small molecule that easily reaches all tissues in the human body and readily crosses the blood brain barrier. When drinking, two types of reactions occur.[3] One is the intoxicating effect, where alcohol binds to several neurotransmitter systems (GABA, NMDA glutamate, dopamine). This effect often is perceived as pleasurable, with relaxation and lowered inhibition, but also generates impaired coordination, slowed reflexes, slurred speech, memory impairment, and, in severe cases, increased sedation and coma. This is entirely an effect of ethanol in itself on the brain. The other reaction, the unpleasant effects, including nausea, headache and organ harm, which are caused by acetaldehyde and oxidative stress.

The elimination of ethanol at lower concentrations increases linearly with rising alcohol concentration. However, this phase is limited because the primary metabolising enzyme, alcohol dehydrogenase (ADH), becomes saturated[4,5] at low BAC levels, roughly the level achieved after drinking one standard drink.

At low levels of drinking, ADH metabolises ethanol to acetaldehyde, which in turn normally is metabolised by aldehyde dehydrogenase (ALDH) to acetate. The key is whether acetaldehyde is efficiently detoxified by ALDH. If ALDH can keep up, blood acetaldehyde remains low. But with rapid drinking, ADH and ALDH both get saturated, resulting in continued and increasing high levels of ethanol and acetaldehyde respectively.

At low ethanol concentrations, acetaldehyde levels remain low but non-zero.

But even at these low levels, acetaldehyde has biological activity, and in some situations can lead to effects. In East Asian populations, up to ~40% carry a genetic variant of ALDH[6] which leads to reduced capacity to convert acetaldehyde to acetate. In these populations even one drink can lead to acetaldehyde spikes with flushing, headache, tachycardia and nausea and will increase cancer risk.

The more common ALDH variant, carried by most Europeans and Africans, results in quicker metabolism of acetaldehyde to acetate. Clinical symptoms of intoxication (disinhibition, slowed reflexes) are due to ethanol itself, not acetaldehyde. Acetaldehyde here likely contributes little, though subtle effects (mild vasodilation, warmth) can occur.

However, even if the systemic effects of low levels of acetaldehyde are negligible, acetaldehyde can accumulate locally in saliva and mucosa after small doses, due to oral bacteria metabolism. This is thought to explain the link between even light drinking and increased oral/oesophageal cancer risk.[7]

Furthermore, metabolism is increasingly shunted to a secondary system, the microsomal ethanol oxidizing system (MEOS), with the enzyme Cytochrome P-450 2E1 (CYP2E1). These metabolic shifts occur at increased consumption, especially in cases of chronic high consumption, when ADH/ALDH become saturated. The common definition of binge drinking is based on the behavioural effects of increasing ethanol. For the more long-term organ damage, based on MEOS and CYP2E1, an increase in harm is seen, with increased consumption.[8] Oxidative stress results from an imbalance between the production of reactive oxygen species (ROS) and the body's capability to cleanse these reactive intermediates. The more and the faster you drink, the more metabolism shifts toward CYP2E1 with escalating ROS and cumulative acetaldehyde exposure.

Acetaldehyde is a toxic intermediate from ethanol metabolism and appears when ALDH is unable to cope with high levels of acetaldehyde, resulting in a further buildup of acetaldehyde levels. Acetaldehyde crosses the blood–brain barrier, but it is normally metabolised rapidly by ALDH, so in most people it doesn't accumulate enough in the brain to drive the feeling of being drunk.

CYP2E1 metabolism produces reactive oxygen species (ROS) and contributes to cellular damage and inflammation.[9] This cellular damage in many organs (e.g., liver, pancreas and brain), leads to long-term harms, such as cirrhosis, pancreatitis, alcohol use disorder, and cancer, but is not a cause of immediate feelings of being drunk. Fast, high BAC peaks are metabolically more severe than moderate increases in BAC. Once ADH is maxed out, additional ethanol increasingly relies on pathways that generate more oxidative stress. Prolonged alcohol exposure enhances ROS generation, ultimately contributing to cellular damage and loss.

Consequences of binge drinking are not short-lived nor limited to the period of intoxication.[10] Imaging studies of binge drinking adolescents document long-lasting changes in the brain. Reports indicate structural changes in the prefrontal and parietal regions, as well as in regions known to mediate reward. These changes are thought to underpin long-lasting



The key difference between binge drinking and regular high consumption is the rate of consumption, where rapid consumption leads to a rapid rise in BAC, which the detoxification enzymes can't cope with.

effects of binge drinking on critical neurodevelopmental processes and, in particular, those involving executive functions. Impaired executive functioning, e.g., problem solving and the ability to exert self-control, are key components in the development of alcohol use disorder. Furthermore, brain research from the past ten years shows that even low or moderate levels of alcohol consumption are associated with brain atrophy, especially of the hippocampus,[11,12] where memories are formed and consolidated.

Binge drinking often occurs apart from meals, which may also contribute to its deleterious effects. Food consumed at the time of alcohol consumption influences not only alcohol absorption rate and blood alcohol concentration, but also the direct effect of alcohol on the gastrointestinal mucosa. Hence, binge drinking is more likely to contribute to organ injury than slower-paced consumption that is associated with a meal.

Some alcohol is metabolised in the brain. Although brain metabolism does not significantly contribute to inter-individual variation in BAC following alcohol ingestion, it is clinically significant due to its acute effects on behaviour and reward.[13]

In summary, some of the consequences of rapid drinking of large volumes of alcohol include:

- Toxic effects of ethanol itself;
- Acetaldehyde accumulation, leading to headache and other hangover symptoms;
- Oxidative stress leading to liver inflammation, fibrosis, cancer and other organ damage;
- Neurochemical dysregulation and increased addiction risk from increase in dopamine and reduced executive functioning;
- Loss of control over drinking and acute intoxication.



3 Drinking patterns and their prevalence

Consumption of large quantities of alcohol during a single occasion, i.e. binge drinking, is a highly prevalent behaviour throughout many countries[14] and carries considerable short-term and long-term risks to health and safety. For most countries, total alcohol consumption and rates of binge drinking in a population are closely related. For many, if not most high-income countries, at least 50% of total alcohol consumption can be attributed to episodic binge drinking occasions.[15] For lower income countries the contribution of binge drinking to total population consumption may be much higher. When under-reporting in surveys is accounted for, estimates of binge drinking's contribution to total alcohol consumption in a population may be even higher.

Approximately 30% of people living in OECD countries reported binge drinking at least once during the past month.[14] Prevalence of binge drinking behaviours can vary considerably between demographic groups (e.g. age, sex, socioeconomic status) and over time. In this context, the term 'prevalence', refers to an estimate of the proportion or percentage (e.g. half, 50%, 5 in 10) of a population said to have a specific characteristic during a specified time period. The term prevalence 'rate' is used interchangeably with population prevalence in this report. At the country level, changes in binge drinking prevalence have variously occurred over time. These changes have been particularly notable among youth, young adults and have pointed to stark differences in binge drinking trends between young females and males.

3.1 Binge drinking and its relationship to a population's total alcohol consumption

As noted, a strong and consistent association has been observed between a population's total alcohol consumption and binge drinking prevalence. This association is readily understandable given the high proportion of alcohol shown to be consumed on binge drinking occasions. In the US, for instance, per capita alcohol consumption and binge drinking are positively correlated at the state level with coefficients of between 0.45 and 0.61.[15] A high-quality study with minimal survey under-reporting found binge drinking occasions accounted for around 50% of all alcohol sales in high-income countries and closer to two-thirds in middle income countries.[16] Using the same instrument and a higher cut-off of 6+/8+ 12 g drinks of alcohol per day, one study[17] found that in many countries, most alcohol is consumed above these levels i.e., on binge drinking occasions. In New Zealand and England this proportion



Binge drinking occasions accounted for around 50% of all alcohol sales in high-income countries and closer to two-thirds in middle income countries.

was around 45%, in Vietnam, Peru, Scotland and Australia the proportion was 50% or just above, in Mongolia and Thailand 62%, and in South Africa 93%. Another study found that when the percentage contribution of binge drinking days to total population consumption was estimated with adjustment for under-reporting in surveys, there was a marked increase, i.e. from 37% of total consumption using unadjusted self-reports to 74% after adjustment.[18]

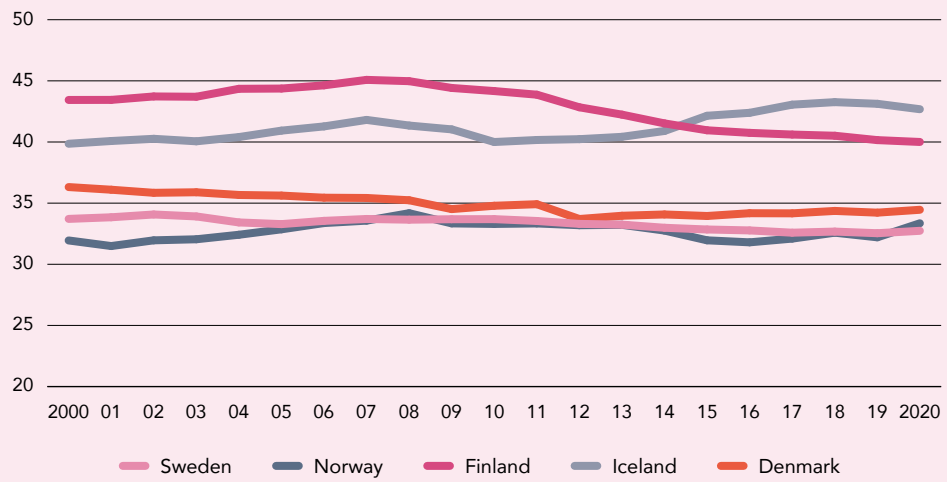
3.2 Binge drinking prevalence

The WHO Global Information System on Alcohol and Health (GISAH) database[19] contains estimates of total alcohol consumption by country. Total consumption includes both recorded use (from country specific records of production, imports, exports, sales data) and unrecorded consumption (i.e. untaxed).

According to a 2021 OECD report which used WHO's GISAH data on total alcohol to model estimates of binge drinking prevalence, about 30% of people living in OECD countries drank more than 60 grams of pure alcohol (equivalent to 5 Swedish standard drinks of 12g pure alcohol) during a single occasion at least once in the past month in 2016. Without exception, national binge drinking levels were markedly higher for males than females and in most countries (and overall), monthly binge drinking was most common among people under 45 years of age.[14]

Figure 1 shows trends in age-adjusted binge drinking prevalence among Nordic countries from 2000 to 2020. These estimates have been derived directly from GISAH data[19] and compiled specifically for this report. In keeping with Swedish reports[20], GISAH data indicated that Sweden experienced only marginal change in levels of binge drinking over time for people aged 15+ and that Swedish rates were on par with the OECD (as were Norwegian and Danish rates in 2020). Binge drinking prevalence in Iceland and Finland were comparatively higher than in Sweden and both showed changes over time, though in opposite directions, i.e., increasing trend for Iceland, decreasing trend for Finland.

FIGURE 1 Trends in estimated age-adjusted prevalence of binge drinking among 15+ year olds in Nordic countries, 2000 to 2020



Source: GISAH, https://www.who.int/data/gho/data/themes/global-information-system-on-alcohol-and-health.
Binge drinking defined as more than 60 grams of pure alcohol during a single occasion at least once in the past month.



Among Nordic countries, binge drinking rates reported by men were about twice that for women. In 2020, age standardised binge drinking rates for men and women aged 15+ years in Sweden were 44.3% and 20.5% respectively compared to 55.3% and 28.6% in Iceland, 52.6% and 26.7% in Finland and 45.2% and 20.7% in Norway.[19]

On average, among those who drink alcohol, people in higher income groups (21%) throughout OECD countries are more likely to binge drink than those in lower income (16%) groups.[21] However, in many countries, prevalence is high for both the lowest and highest income groups. In other words, binge drinking rates are often lowest among the middle socioeconomic groups.[14] For Sweden, corresponding estimates for binge drinking among high- and low-income groups in 2019 were 24% and 15%.[21]

Around 20% of EU residents with higher education report binge drinking at least once a month compared to 13% of people with lower education.[21] Women with higher educational attainment (14%) appear to binge drink at least once a month more often than their less educated (12%) counterparts.[14] It is important to note, however, that studies report substantial variability in peak BAC reached and/or quantity of drinks consumed during a binge drinking episode across socioeconomic groups. A US study found, for instance, that total binge drinks per binge drinker was much higher in low education and income groups compared to groups with high education and income.[22]

Binge drinking among drinkers (as opposed to the total population including non-drinkers) show a marked positive association with deprivation and disadvantage.

Furthermore, adjustment for under-reporting appears to further increase this disparity. One study[23] found that after an adjustment to reduce effects of under-reporting, respondents to a UK survey who were living in the most deprived areas became significantly more likely to binge drink than those in the least deprived areas.

3.3 Binge drinking among adolescents and young adults

During the past decade there has been considerable research interest around apparent changes in uptake of drinking and experience of binge drinking among adolescents and young adults. Declining prevalence of overall alcohol use and binge drinking among adolescents over the past 20 years has been reported for many developed countries.[14]



Although binge drinking (in the last 30 days) remains most prevalent among younger age groups, rates have diminished for those aged under 50 years while simultaneously increasing among the 50+ population.[20]

Across the population, the substantial majority of binge drinking episodes occur among those older than age 25.[24]

According to OECD analyses, proportions of 15-year-olds who experienced drunkenness in 2001–02 compared to 2017–18, decreased from 40% to 23% for boys and from 33% to 20% for girls.[14] In keeping with the overall OECD trend, from the beginning of the 2000s to 2024, fewer Swedish grade 9 students reported binge drinking at least once a month; around 23% for girls and 30% for boys. For 11th grade students, prevalence decreased from around 40% among girls and 50% among boys during 2005 (when surveys started), to 22% in 2024.[25] Interestingly, in Norway, despite declining binge drinking prevalence in mid-adolescence, one study[26] found that by the time cohorts reached their early 20s, no significant differences remained.

Various explanations for apparent declines in youth binge drinking have been canvassed, including: substantial rises in student debt diminishing disposable income; reduced alcohol affordability; social media increasing personal accountability for acts committed under the influence of alcohol; and reduction in time spent socialising in-person.[14,27]

A 2023 Gallup Poll[27] suggested that increasing racial/ethnic diversity and greater proportions of non-drinkers among non-whites in the population is a key reason for declining youth drinking. However, regardless of race/ethnicity the survey also found that young people were now much more likely hold the belief that alcohol, even at moderate levels, is bad for health. Whether this shift in health beliefs translates into actual and sustained behaviour change around alcohol use is not yet clear.

Not all research evidence points to reductions in binge drinking, however. In fact, several countries have reported increases in

binge drinking indicators specific to young females. This phenomenon appears to point to convergence in binge drinking between the sexes, whereby young females are rapidly 'catching up' with same-aged males.

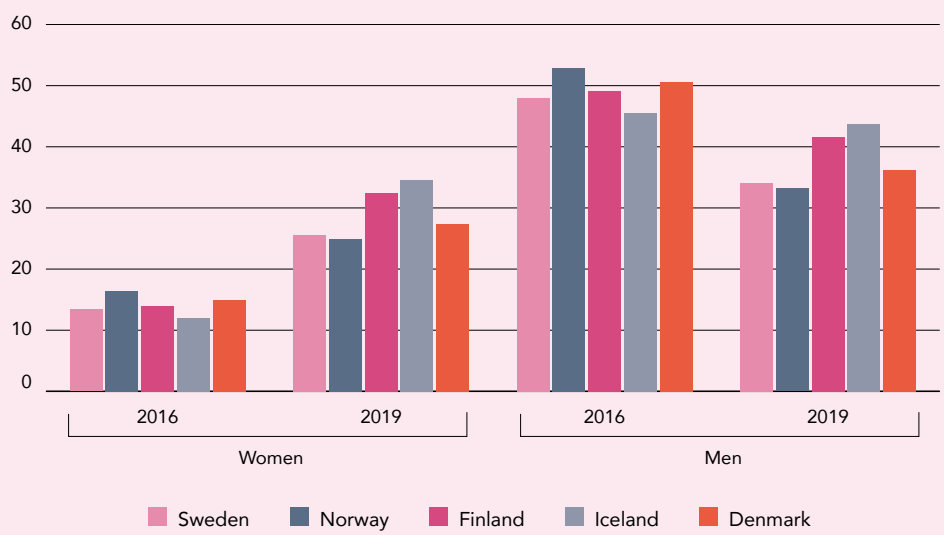
Using data drawn from WHO's GISAH database, Figure 2 shows binge drinking prevalence estimates for older teenagers aged 15 to 19 years by sex for Nordic countries. All five nations reported higher rates of 15–19 year-old female binge drinking in 2019 compared to 2016. These increases were substantial and occurred over a relatively short time period, ranging from 52% in Norway to 190% in Iceland. In contrast, binge drinking prevalence among 15–19 year-old males fell across the board with declines ranging from 4% (Iceland) to 37% (Norway). Despite the large increases in young female binge drinking rates, male rates were still around 30% higher on average.

It is important to note that due to the fact that young male binge drinking rates were so much higher than for females in 2016 and remained substantially higher than female rates in 2019, overall binge drinking prevalence did not necessarily increase and in fact, either declined or remained relatively stable in three countries (Sweden, Norway, Denmark). In Finland and Iceland, relatively modest declines in 15–19 year-old male binge drinking (15% and 4% respectively) were over-shadowed by very large (130% and 190% respectively) increases among females, resulting in net binge drinking increases of 18% and 36% respectively for those countries. In keeping with these trends, a review of US studies since 2008[28] found that binge drinking among adolescents and young adults has decreased and sex-specific rates have converged due to male binge drinking declining faster.



Several countries have reported increases in binge drinking indicators specific to young females. This phenomenon appears to point to convergence in binge drinking between the sexes, whereby young females are rapidly 'catching up' with same-aged males.

FIGURE 2 Estimated prevalence of binge drinking among 15-19 year-old males and females in Nordic countries, 2016 and 2019



Source: GISAH, https://www.who.int/data/gho/data/themes/global-information-system-on-alcohol-and-health. Binge drinking defined as more than 60 grams of pure alcohol during a single occasion at least once in the past month

4 Risks from Binge Drinking



One's blood alcohol concentration (BAC) is key to determining an individual's risk of alcohol-related harm.

A range of factors affect, or modify, a person's risk of harm from binge drinking. Key factors include level of Blood Alcohol Concentration (BAC) reached during the drinking occasion, young age, sex and gender, lower socioeconomic status, and maternal and paternal alcohol use.

4.1 Blood alcohol concentration

Alcohol is a central nervous system depressant.[29] One's blood alcohol concentration (BAC) is key to determining an individual's risk of alcohol-related harm. As BAC rises, risk of harm increases. This is particularly demonstrable for harms that occur during or within a short time of a drinking occasion (e.g. road crashes, falls). However, there is also increasing evidence for long lasting impacts arising from exposure to high level BACs during binge drinking that increase risk of degenerative disease (e.g. early onset dementia) and mental health problems (e.g. [12,30]).

When alcohol is consumed rapidly, the primary enzyme that normally breaks down the majority of alcohol (alcohol dehydrogenase) becomes overwhelmed. As a result, the body is forced to rely more heavily on alternative metabolism pathways (i.e. the microsomal ethanol oxidizing system) that produce higher levels of both acetaldehyde and organ-damaging free radicals.[31,32] These compounds circulate around the body, potentially damaging cells and organs, and leading to both short-term impairments and long-term damage that can present as degenerative disease many years later. It has been suggested that this biochemical process explains why intermittent binge drinking is more damaging to the human body than regular low-level use at any level of total consumption.[33–35]

Reviews of observational studies have found strong dose–response relationships between amount of alcohol consumed in a drinking occasion and risk of both motor vehicle and non-motor vehicle injury (e.g. [1,36–38]). For instance, odds of a fatal motor vehicle crash increase by 74% for every 0.2‰ (0.02%) increase in BAC. At 0.8‰ (0.08%) BAC – which is the legal driving limit in many countries – risk of a fatal crash is about 13 times higher than at zero BAC.[1,39,40] A comprehensive review of individual-level data showed that the dose-response relationship is non-linear such that risk increases exponentially as BAC increases. For example, compared to no alcohol consumption, drinking 24g of alcohol in a single drinking session approximately doubles the risk of injury, however, at levels of consumption approaching 120g, risk of motor vehicle injury is around 50 times higher.[1]

Even relatively modest elevations in BAC can cause problems. Risk of motor vehicle crash or other unintentional injuries starts to increase at 0.2‰ (0.02%) BAC, or approximately one drink.[38,40] In laboratory

experiments, impairment of reaction time and coordination begins around 0.4‰–0.5‰ (0.04–0.05%) BAC. At this level, people are already at increased risk of accidents and poor decision-making. Laboratory and experimental studies show that even at relatively low levels (e.g. 0.4‰–0.5‰ (0.04–0.05%) BAC), alcohol can impair balance and coordination, visual focus, reaction time, judgment, response inhibition, and change behaviour. A review of over 60 randomized controlled trials (RCTs) of alcohol's effect on neural circuits in the brain using electroencephalogram methods showed that alcohol's negative effects on attention, automatic auditory processing and performance monitoring increased as dose increased and, was detectable at doses as low as 0.26‰ (0.026%) BAC. [41]

When BAC is between 1.0‰ and 2.0‰ (0.10% and 0.20%), serious neurological impairment, including slurred speech, memory problems, and loss of coordination may occur. If BAC is high enough (e.g. >= 3.0‰ (0.30%)), it can result in loss of consciousness, coma, respiratory failure and death.[29,42–45]

However, BAC levels associated with alcohol poisoning deaths are variable, and influenced by factors such as the presence of other respiratory depressant drugs, hypothermia (which frequently accompanies alcohol poisoning, particularly in outdoor environments) and concomitant vomiting, which may cause aspiration and respiratory arrest.

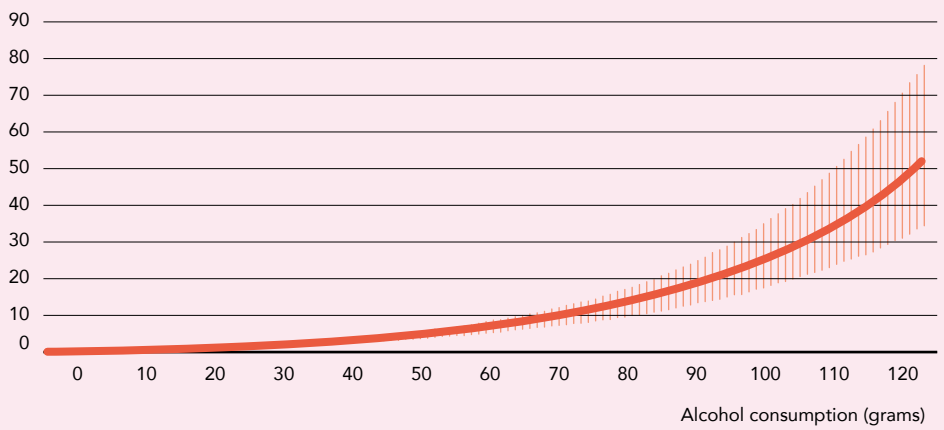
Tolerance to alcohol's effects

Drink-for-drink, alcohol's effects on the human body can vary widely among individuals. Much of this can be explained by individual variations in tolerance to alcohol. Tolerance is related to several metabolic processes.

Firstly, 'metabolic' tolerance arises due to increased efficiency in how the body metabolises alcohol. This results in lower BAC levels for a specific dose of alcohol and the need for higher levels of alcohol in order to achieve the same effects. Many people compensate by drinking more to attain a given BAC level and its desired intoxicating effects.

Secondly, 'functional' tolerance refers to how an individual can learn to perform tasks

FIGURE 3 Dose–response curve for the amount of alcohol consumed 3 h prior and the odds of motor vehicle accident injury



Source: Figure 5 in Taylor et al. (2010). The more you drink, the harder you fall: A systematic review and meta-analysis of how acute alcohol consumption and injury or collision risk increase together. Drug and Alcohol Dependence, 110(1–2), 108–116.



under the influence of alcohol at a specific BAC level i.e., with practice, deficits can sometimes be partially overcome. At the extreme end of the drinking spectrum, people with severe alcohol-induced brain and liver damage can experience a dramatic loss of tolerance so that they may exhibit impairment at low doses. Tolerance to alcohol's effects on mood, motor skills and behaviour develops over time with repeated experience of alcohol use, particularly with repeated episodes of binge drinking. Essentially, this means that, over time, someone who drinks frequently may need to consume more alcohol to achieve the same effect.

Drinkers with low tolerance for binge drinking are at risk of death at around 3,0‰ (0.30%) BAC, while individuals with alcohol dependence can sometimes survive at substantially higher BAC levels.[45] This wide variation illustrates how tolerance modifies BAC effect on risk but does not imply that tolerance eliminates risk. Tolerance affects wakefulness and the sensation of intoxication more than other aspects of impairment such as executive functioning.[46] Therefore, those with alcohol tolerance may be more likely to attempt complex tasks (due to feeling more awake and less drunk) while still being considerably impaired. This, paradoxically, can increase the likelihood of engaging in risky behaviour (e.g., driving).[47]

4.2 Age: younger and older drinkers

Age is another key factor that influences risks from binge drinking. Young people are especially vulnerable because their brains and bodies are still developing. Alcohol interferes with normal brain development, disrupting the balance between excitatory and inhibitory neurotransmitters (e.g. glutamate, GABA). Alcohol also retards production of new neurons in the hippocampus (crucial for memory and learning) and causes structural changes in brain areas responsible for decision-making and impulse control. These changes can last long after the drinking episode and when binge drinking begins in adolescence, long-term consequences can be especially damaging.[48–50]

When binge drinking begins early in life, it also brings significant risks of setting in place lifelong risky drinking behaviours. A 15-year prospective study, for example, found that 90% of males and 70% of females who started binge drinking in adolescence continued this pattern into young adulthood.[51]

Generally smaller body mass among young teenagers compared to fully grown adults means that drink-for-drink, young people will attain higher BACs. As described above, individuals who attain higher BACs are at greater risk of injuries and other short-term harms, as well as elevated risk of developing degenerative disease stemming from long-term damage.[12]

For older drinkers, factors affecting risks of harms arising from binge drinking are related to the process of ageing itself. As people age, metabolism slows down, the body contains less water leading to higher BACs from the same amount of drinking. For example, people over 60 years of age who binge drink at least once a month have been reported to have more than a tenfold increased risk of fall-related injuries. Older adults also tend to have lower muscle mass, decreased sensory perception, poorer balance.[12,52,53] Moreover, older people have a higher baseline risk for falls and injuries in general and are more likely to be taking one or more medications which may interact negatively with alcohol use.[54]

4.3 Sex and gender

Both biological sex differences (e.g. hormones, genetics) and gender-based sociocultural constructs (e.g. "masculinity", "femininity") significantly influence risks of harm from binge drinking.

Attributed to both biological (e.g. higher levels of testosterone) and sociocultural determinants, males have, overall, a greater propensity for risk-taking compared to females.[55] Differences in risk-taking propensity are likely to explain why males more often combine binge drinking with activities such as driving and violence than females. This leads to males having higher risks and greater morbidity/mortality from alcohol-related accidents and injuries, even at the same BAC.[56,57]

Drink-for-drink, the typically smaller body size and lower body water of females results in higher BACs compared to males. Studies show that the negative consequences of drinking increase more steeply for women. Between four and nine drinks per occasion, women experience sharp rises in hangovers, vomiting, and blackouts. For men, the increase in harms is more gradual across higher drink counts.[58] A Swiss study of self-reported data on consequences from binge drinking showed that for similar risk levels, consumption for women were about half of that for males.[59] Nonetheless, many national guidelines for reducing risk of harms from episodic drinking apply the same recommendations for males and females, e.g. Swedish clinical guidelines and Australian guidelines.[60,61]

Evidence is also emerging of gender-driven contrasting changes over time in alcohol use and alcohol-related harms for males and females.[28,62–64] In Sweden, binge drinking gender differences appear to have diminished somewhat but they remain more prevalent for men (38%) compared to women (23%).[20] Apparent convergence in alcohol use and harm levels between males and females is particularly stark for young people, among whom binge drinking is most common, with several jurisdictions reporting decreased or stabilized alcohol-related harms among teenage males while female rates have increased.[65] It is possible, however, that at least in part, gender gaps observed in self-report survey studies may be explained by differential under-reporting, e.g.[23].

4.4 Socioeconomic status (SES)

Socioeconomic status is a strong modifier of risks from binge drinking, it plays an important role in shaping how and why people binge drink. People in lower SES groups tend to binge drink less frequently but may



Differences in risk-taking propensity are likely to explain why males more often combine binge drinking with activities such as driving and violence than females.

drink larger amounts on each occasion which leads to higher peak BACs. For example, U.S. data show that total binge drinks per binge drinker are substantially higher among lower compared to higher SES groups.[21,66,67] Importantly, higher BACs result in more serious and frequent harms in the short-term, such as road traffic accidents, violent incidents and poisonings as well as more severe, long-lasting outcomes for the drinker and third parties.[22,68]

In addition to differences in consumption, at any particular level of consumption, lower SES groups are more vulnerable to harms compared to those who are more socially advantaged. This is referred to as the "harms paradox" which is driven by disadvantages in social, economic and underlying health factors, all of which can amplify alcohol-related risk.[67,69] For example, after adjusting for differences in alcohol consumption, rates of alcohol-attributable deaths differed four-fold among racial-ethnic groups in the U.S.[70]



In addition to differences in consumption, at any particular level of consumption, lower SES groups are more vulnerable to harms compared to those who are more socially advantaged.

Binge drinking in vulnerable populations rarely occurs in isolation. It often clusters with other individual risk factors such as (but not limited to) smoking, obesity, poor diet, lack of physical activity, as well as other social factors such as limited access to quality health care services, housing, education and employment opportunities. Together, these intersecting factors multiply health risks from binge drinking and may have particularly negative impacts on degenerative health concerns that typically appear in mid-life, e.g. lower quality of life, poorer outlook, greater risk of premature death.[71]

4.5 Maternal and paternal drinking

Binge drinking during pregnancy, especially when frequent, increases the likelihood that the foetus will be exposed to high BAC levels at critical developmental stages. High-dose exposures may lead to foetal alcohol spectrum disorders (FAS/FASD), which include a range of permanent physical, cognitive, and behavioural problems and other serious outcomes (e.g. spontaneous abortion, premature and still birth).[72–74]

Pregnancy is often not recognized until around five weeks gestation; this is driven in part by the high proportion of pregnancies that are miss-timed, unwanted or otherwise unexpected. Binge drinking among women of childbearing age can increase the likelihood of the fetus being exposed to high blood alcohol levels on one or more occasions. If the mother is unaware of the pregnancy and binge drinking continues during a critical stage of foetal development, risks of lifelong consequences to the foetus are significant. Binge drinking is also associated with unintended pregnancies and delays in recognizing pregnancy, both of which increase the likelihood of harmful foetal alcohol exposure.[72] As such, reductions in binge drinking among women of childbearing age should be a focus of prevention, not just a focus on binge drinking among those who are known to be pregnant.

Paternal drinking also contributes to risk, though in different ways. There is emerging scientific evidence for biological impacts of paternal alcohol use (even at low levels) on the foetus. Although not necessarily attributed to binge drinking per se, biological effects of paternal alcohol exposure prior to conception include reduced sperm health, spontaneous abortion, still birth, as well as foetal heart conditions and some cancers.[75–81]

Paternal binge drinking prior to and during pregnancy is also strongly linked to intimate partner violence, family conflict and household stress. These harms affect both mothers and infants by creating unsafe environments and increasing the likelihood of indirect alcohol-related harm.[72,82] Paternal drinking can also have a profound effect on whether women continue to drink during pregnancy; women who have a live-in male partner who drinks are more likely to continue drinking themselves, especially if the male partner is a heavy or risky drinker.[83]



5 Acute harms

Binge drinking can substantially increase the risk of a variety of immediate (”acute”) harms including injuries caused by violence, road crashes, drownings, burns, freezing, and injuries at the workplace. These harms will often be experienced by people other than the drinker e.g. victims of violence, child abuse or road crashes. A heavy drinking episode or binge can result in impaired judgment and ability to perform complex tasks (e.g. operating machinery, driving) and lower risk perception, (see e.g.[84]),

A more recent review of 100 published studies and publicly available data sets with BAC data for North American populations[85] estimated that alcohol was responsible for; 3% of injuries from plane crashes, 9% from child abuse, 31% of drownings, 37% from falls, 34% from burns, 24% from gunshots, 29% of homicides, 29% from hypothermia, 42% from road crashes, 21% from suicide and 27% from boating. It was further estimated that fully 27% of fatal and non-fatal injuries would not have occurred without alcohol use. These are findings echo those by earlier studies.[1,86]

5.1 Road crash injuries

Alcohol-impaired driving is a threat to others as well as to the driver, with evidence that even a low dose of alcohol (e.g. one drink) can significantly impair reaction time and ability to perform complex cognitive tasks frequently required when driving.[87] Degree of impairment increases and even accelerates with rising BAC as has been shown in a systematic review of simulated driving experiments[88] as well as in other reviews.[40,89]

These risks are of course more likely for binge drinkers than regular low level



Findings across a number of international research studies support a conclusion that alcohol's impairment effects exacerbate the likelihood of intentional injuries or violence due to alcohol's role in aggression and impaired decision-making.

drinkers, as demonstrated in a review of international data.[57] In Sweden, for example, men and women arrested for impaired driving had typically drunk more than five drinks. The odds of impaired driving are five times greater for binge drinkers than for other drinkers. One review of acute alcohol consumption and simulated driving[90] found that lane position and speed were significantly worse after alcohol consumption and that performance declined as higher BAC levels were reached. A Spanish study of over 12,000 survey respondents[91] found frequency of traffic crashes increased substantially for binge drinkers.

5.2 Unintentional injuries

Unintentional injuries associated with binge drinking typically include falls, burns, drownings, and workplace injuries arising due to impaired balance, vision, and risk perception. A recent Norwegian study[92] concluded that injury risk increased exponentially with rising BAC i.e. risk level accelerated with higher amounts consumed. Scandinavian studies from Sweden and Norway concur with international reviews[12,93] that heavier alcohol use in older age increases fall risk. In the Norwegian study, risk of fall injuries among those aged 60 years and older was over ten times greater for those who drank to intoxication at least once each month.[92] A review of epidemiological studies[94] found about half of all adult burn patients were intoxicated when admitted and, further, the presence of alcohol increased the risk of a fatal outcome and complications in the recovery of survivors.

5.3 Intentional injuries from aggression, assault, and homicide

Intentional injuries are those resulting from violence, assault, self-harm, suicide or aggression by another. The relationship between BAC and injury risk varies such that intentional injuries have a stronger association with alcohol use than do unintentional injuries. Findings across a number of international research studies support a conclusion that alcohol's impairment effects exacerbate the likelihood of intentional injuries or violence due to alcohol's role in aggression and impaired decision-making. A study of 19 European countries[95] on experiences of aggression (verbally abused, harmed physically, or having serious arguments) found that the risk of experiencing harm from others' alcohol-related aggression increased significantly with increasing population-wide prevalence of binge drinking. Survey studies find sizeable proportions reporting harm from others drinking. Examples are studies from Australia[68,96], from 9 countries[97] (Australia, India, Ireland, Lao People's Democratic Republic, Nigeria, Sri Lanka, Thailand, the United States, and Vietnam), Norway[98], and from Denmark, Finland, Iceland, Norway, Sweden, and Scotland.[99]

Homicide

Binge drinking can be a contributing cause to a violent death (homicide) through impairment of the perpetrator or the victim or both. Over 50% of offenders are intoxicated at the time of the homicide with alcohol being the most cited substance and the level of alcohol utilized is comparable for victims and perpetrators.[100] On average across studies 40–50% of homicide victims tested positive for alcohol, 30–35% met a BAC in excess of 0,8‰ (0.08%).[100,101]

5.4 Sexual aggression and binge drinking

Sexual aggression usually from males toward females is enhanced and increased by alcohol, especially binge drinking. One systematic review[57] also concluded that binge drinking among male college students made them more likely to engage in sexual aggression. The study also found that apart from incapacitation and rape, women who binge drink are also at greater general risk of sexual victimization for many possible reasons: men's misinterpretation of women's drinking as a sign of sexual availability, miscommunication of women's refusals, and women's underes-

timation of hazards from male companions. A longitudinal study[102] also found that adolescents who engaged in frequent binge drinking were more likely to perpetrate sexual harassment and stalking, and more likely to be victims of stalking and dating violence. At time points when adolescents had engaged in binge drinking in the past month, they were also more likely to perpetrate dating violence and more likely to be sexual harassment victims.

5.5 Harm to children

Children and youth can be harmed by the drinking of their caregivers. A Swedish National Public Health Institute survey found that while two thirds of respondents felt it was acceptable to get drunk at home when children were not present, less than 10% considered this acceptable when children were present.[103] A meta-analysis of 12 longitudinal studies from six high-income countries[104] found that caregiver alcohol-related diagnoses were associated with more than twice the rate of child maltreatment. One review of 19 articles[105] found that men's heavy drinking was associated with direct and indirect harms for children, including family conflict, maltreatment, and emotional distress.

A study of eight countries[106] (Australia, Chile, Ireland, Lao People's Democratic Republic, Nigeria, Sri Lanka, Thailand, and Vietnam) found harms to children were highly associated with drinking behaviour of their caregivers. Studies which have specifically examined effects of binge drinking on children suggest that: there is a negative effect from fathers binge drinking[107]; a doubling of odds of not completing high school with weekly or more binge drinking by fathers[108] and, that binge drinking by fathers was significantly associated with risk for substance-related disorders in (Swedish) children.[109] A Norwegian study of parental binge drinking found weekly or more frequent binge drinking in fathers negatively affected high school graduation prospects in their offspring.[108]

5.6 Alcohol poisoning

The mechanism for alcohol poisoning involves a combination of paralysis of respiratory and circulatory centers in the brain, potentially leading to a state of asphyxiation, i.e. the body is deprived of oxygen and vital organs begin to fail. Binge drinking can achieve a high BAC in the drinker in relatively short time. A binge drinker will experience alcohol poisoning at BACs of 3.5‰ (0.35%) or higher but even a BAC of 2.5‰ (0.25%) can put the drinker at risk of coma. Children and less experienced drinkers can be at risk at much lower BACs.

Alcohol poisoning mortality and alcohol-related psychosis hospital admissions can occur at BACs from 1‰ to 12‰ (0.1% to 1.2%). While the percentage of hospital patients admitted specifically for alcohol poisoning can be small, the broader impact of alcohol on hospital admissions is substantial.[110–112]





6 Chronic harms

6.1 All-cause mortality

Most deaths in developed countries are caused by chronic diseases. A US study[113] with survey data from 1997 to 2014 found that binge drinking one or more days a week was associated with a higher risk of mortality from all causes compared to life-time abstainers (1.15; 1.09 to 1.22). There was also an increased risk for cancer (1.22; 1.10 to 1.35), and accidents (unintentional injuries) (1.39; 1.11 to 1.74)).

Between ages 40 and 75, never-drinkers and moderate drinkers who did not binge drink had the longest disease-free life span (about 29–30 years). The shortest disease-free life span was seen in people with alcohol poisoning (about 23–24 years) and in heavy drinkers who binge drank (about 26–28 years). Overall, never-drinkers and moderate non-binge drinkers lived 5–6 more disease-free years than those with alcohol poisoning, and 2–3 more years than heavy binge drinkers.[114]

6.2 Cardiovascular disease (CVD) and stroke

Binge drinking is strongly implicated in cardiovascular morbidity and mortality. It increases the risk of hypertension, ischemic heart disease, atrial fibrillation, cardiomyopathy, and sudden cardiac death, elevates both ischemic and haemorrhagic stroke risk and increases coronary artery calcification (by 100% or 2 times with binge drinking).[115–117]

While some earlier work suggested that low-to-moderate drinking could have a protective effect, more recent reviews conclude that binge drinking patterns negate any cardioprotective benefit, instead driving a monotonic increase in CVD risk.[118–121]

Overall, binge drinkers have higher blood pressure and are more likely to develop hypertension compared to non-binge drinkers who consume alcohol weekly or more often but at low to moderate levels. Binge drinking in adolescence, young adulthood and middle age increases the risk of developing **high blood pressure** years later and during older age.[121]

For **ischemic heart disease** (IHD), many large reviews and meta-analyses show an increased risk for fatal ischemic heart disease (i.e., coronary heart disease). Most studies find a 1.5–2.0 × higher risk (50–100% increase). Some high-risk groups (e.g., Finnish Kuopio cohort, Russian men, or post-MI patients) show 2–3 times higher risk. Meta-analyses find around a 1.6–1.8 times increased risk for fatal IHD events associated with binge drinking. This means that binge drinkers have at least 60–100% higher risk of fatal IHD events, with some populations seeing up to 200–300% higher risk.[122–129]

Risk for **atrial fibrillation** is between 30% and 50% higher among populations that binge drink compared to populations with non-binge patterns. For regular heavy drinkers (e.g. at risk of alcohol use disorder), episodic binge drinking can double the risk of atrial fibrillation (i.e., a100% increase). Evidence for this effect is consistent across large cohorts, case-control studies, and experimental physiology.[116]

For **stroke**, a number of reviews and meta-analyses confirm that binge drinking significantly increases risks of both ischemic (blood clot) and haemorrhagic (bleeding) stroke.[130–136] For ischemic stroke, binge drinking increases risk by 1.2–1.6 times. Risk for haemorrhagic stroke is consistently higher, typically 1.5–2.5 times. Binge drinking doubles (or more) the risk of having a stroke in the immediate future i.e., within 24 hours. The main mechanism is through acute increases in blood pressure, but other factors such as inflammation and impact on the arterial walls such as endothelial dysfunction are likely contributors.

6.3 Dementia and cognitive impairment

There is strong evidence that binge drinking contributes to the development of dementia, particularly early-onset dementia. The likely mechanisms for this, such as direct toxic effects of ethanol and acetaldehyde, as well as increased inflammation through ROS and other inflammatory metabolites, are described earlier.

A large Swedish cohort of nearly 500,000 men found that adolescent binge drinking was the strongest predictor of early-onset dementia, while episodes of alcohol-induced unconsciousness more than doubled the risk of both early- and late-onset dementia, including Alzheimer's disease and vascular dementia.[137] Twin studies also demonstrate that binge drinking independently predicts cognitive decline and impairment, even after adjusting for total alcohol consumption, highlighting the role of drinking patterns over cumulative volume.[138,139] These findings are reinforced by meta-analyses and longitudinal cohorts showing that heavy or binge alcohol use increases long-term dementia risk, including Alzheimer's and vascular dementia. [11,140–142] Binge or heavy drinking is associated with 50–100% higher long-term dementia risk, with some studies showing more than two times greater risk for early-onset or vascular dementia.[143]

6.4 Mental health

Binge drinking is associated with a range of psychiatric outcomes, including depression, anxiety, and suicide. Overall, binge drinking increases depression risk by 50–80% and at least doubles suicide attempt risk.

Risks are particularly pronounced in socially disadvantaged groups.[144] Mendelian



Many large reviews and meta-analyses show an increased risk for fatal ischemic heart disease (i.e., coronary heart disease). Most studies find a 1.5–2.0 × higher risk (50–100% increase).



Binge drinking is associated with a range of psychiatric outcomes, including depression, anxiety, and suicide. Overall, binge drinking increases depression risk by 50–80% and at least doubles suicide attempt risk.

randomization studies suggest a bidirectional link, where binge drinking both contributes to and results from poor mental health.[66] In addition, binge drinking frequently co-occurs with other risk behaviours such as violence, self-harm, and risky sexual activity, which compound its psychiatric burden.[57] These findings are supported by systematic reviews and meta-analyses showing that binge drinking is independently associated with higher risk of major depression, anxiety disorders, and suicidal ideation.[145–149] Large-scale Mendelian randomization studies further highlight shared vulnerabilities linking binge drinking and psychiatric illness.[66]

These associations are reinforced by recent systematic reviews and meta-analyses showing that binge drinking significantly elevates risks of depression, anxiety, and suicidal behaviour.[144,150–154] Evidence indicates both acute and longer-term increases in risk, particularly in adolescents and socially disadvantaged groups.

6.5 Cancers

Alcohol is classified as a Group 1 carcinogen, is associated with 10 different types of cancer, of which 7 are causally related to alcohol consumption according to the International Agency for Research on Cancer or the World Health Organization. These cancers include breast cancer, colorectal cancer, liver cancer, oral cancer, pharyngeal cancer, laryngeal cancer and squamous cell cancer of the oesophagus. Both ethanol, acetaldehyde and inflammatory ROS species contribute to cancer development, by causing DNA damage and inflammation (see chapter 2. Mechanisms).

In addition to the contribution of binge drinking to the total consumption of alcohol, binge drinking may increase cancer risk, but this is an area with a relative lack of research. The most robust evidence is for female breast cancer, where both the number of drinks per occasion and the frequency of binge drinking increase risk, independent of average consumption.[155] A systematic review found that women who binge drank had a higher risk of breast cancer.[156] Large cohort studies support this: the Danish Nurse Cohort and the U.S. Nurses' Health Study both showed that weekend or monthly binge drinking increased breast cancer risk, even after adjusting for overall alcohol intake.[57] Case-control studies have also shown increased risk in Māori women (55% increase) and U.S. women with higher weekly ethanol intake.[57] More recent studies report similar findings: one study published 2020 found a 1.8-fold increase overall and a twofold increase for premenopausal breast cancer, while another study from 2017 showed that ever binge drinking raised risk by 30–40%.[157,158] A U.S. study further linked binge drinking to early-onset breast cancer.[159]

Binge drinking has been associated with higher mortality from all cancers in U.S. surveys.[113,160] Site-specific risks include head and neck cancer, where drinking frequency (but not daily amount) predicted risk[161–163], and lung cancer in Finland, where binge drinkers had nearly double the risk, particularly among smokers.[164] For men, the Finnish Twin Study found that binge drinking increased prostate cancer risk by 30%, while heavy drinkers had an even greater risk.[165]

6.6 Diabetes

Epidemiological studies of alcohol and diabetes show that binge drinking increases risk for type 2 diabetes (T2D) regardless of the level of average consumption.[166–168] Overall, large scale studies and meta-analyses show an increased risk for type 2 diabetes by 30–80% with binge drinking or heavy drinking. They also show worsened glycaemic control and increased risk for complications in people with diabetes. These findings suggest that episodic heavy drinking contributes to insulin resistance and long-term metabolic dysregulation.[169–173]

The Northern Swedish Cohort found that binge drinking during adolescence and midlife was associated with higher fasting glucose in women.[174] A large Mendelian



randomization study based on Danish and British cohorts investigated cardiovascular risk and found an increased risk for diabetes as a secondary outcome, especially for heavy alcohol consumption (based on high average consumption), but did not specifically examine binge drinking.[175]

Another Mendelian randomization study using a Korean cohort found higher alcohol intake in men particularly was associated with increased risk for type 2 diabetes, and that no protective effects were found at any level of consumption.[176] However, this study did not assess effects of binge drinking in particular, only regular heavy drinking.

6.7 Gastrointestinal disease, including liver disease

Binge drinking is a major contributor to development of alcohol-related liver disease (ALD), including fatty liver, alcoholic hepatitis, and cirrhosis. It also accelerates the progression of underlying liver disease, especially in those with Metabolic dysfunction-Associated Fatty Liver Disease (MAFLD).[177,178] MAFLD is now a leading cause of liver disease associated with metabolic factors that contribute to diabetes, overweight and high blood pressure. While higher total consumption is the key driver of ALD, binge drinking adds incremental risk through oxidative stress, immune dysregulation, and inflammatory mechanisms.[179,180] A global meta-analysis confirms that ALD accounts for a substantial share of chronic liver disease burden worldwide, with binge drinking accelerating its onset and severity.[181]

Binge drinking is also a major risk factor for inflammation in the pancreas. A series of meta-analyses show that binge drinking significantly raises risk for both acute (by 2.5 times) and chronic (by 2 times) pancre-



Systematic reviews confirm that even few binge episodes during early pregnancy can cause harm, particularly as many women binge drink before recognizing pregnancy.

atitis.[182–184] Moreover, binge drinking often coexists with chronic liver disease, further aggravating health outcomes.[185]

Binge drinking is also strongly related to peptic ulcer disease (PUD), and interacts with other PUD risk factors such as cigarette smoking and the use of non-steroidal anti-inflammatory medications such as ibuprofen. Drinking can also cause exacerbations of existing PUD including perforation or bleeding, and can exacerbate pancreatitis in the form of haemorrhage, pseudocyst or the development of chronic pancreatitis.[179,186,187] A review also notes that even a single episode of heavy drinking can cause duodenal erosions, jejunal mucosal injury, and gastrointestinal bleeding.[188]

One plausible mechanism for chronic disease development is that alcohol disrupts the gut microbiota, contributing to increased intestinal permeability and systemic inflammation.[189,190] Recent Mendelian randomization evidence links alcohol consumption causally to multiple GI diseases, including pancreatitis, reflux disease, and liver disorder.[190]

6.8 Foetal alcohol syndrome (FAS) and Foetal alcohol spectrum disorders (FASD)

Prenatal alcohol exposure, and especially binge drinking, is the direct cause of FAS and FASD, which are defined by neurodevelopmental delay, cognitive impairment, and behavioural disorders in the child. Systematic reviews confirm that even few binge episodes during early pregnancy can cause harm, particularly as many women binge drink before recognizing pregnancy.[73,191] The teratogenic risk is amplified by the high peak blood alcohol concentrations achieved during binge episodes, but no level of alcohol exposure is safe.

Around 10% of women use alcohol during pregnancy, and global prevalence estimates show that one in 13 women who drink during pregnancy gives birth to a child with some degree of foetal alcohol disorder, with binge drinking patterns driving the highest risks. One of every 23 heavy-drinking pregnant women will deliver a child with FAS, which is the most severe of the diagnoses under the FASD-umbrella.[192,193]

A Nature Reviews synthesis underscores that alcohol is the only cause of and a modifiable risk factor for FASD, with risk amplified when exposure occurs before pregnancy recognition and with increasing BAC.[194] A hierarchical meta-analysis of six prospective US cohorts showed that moderate-to-heavy prenatal alcohol exposure – including binge drinking – was consistently linked to cognitive and behavioural impairment in offspring, strengthening causal evidence.[195] A systematic review and meta-analysis of child neuropsychological outcomes found that binge drinking during pregnancy was associated with poorer executive function, attention, and memory.[196] Cohort evidence from Denmark and other Nordic countries confirms that heavy episodic drinking in pregnancy is associated with low birthweight, prematurity, and dysmorphic features, beyond total alcohol intake.[197,198]

It is important to note that there is no level below which alcohol use is risk-free, as even occasional drinking can be associated with congenital anomalies[75], adverse birthweight outcomes[199], and later mental health issues.[200] Mechanistic studies suggest effects on DNA methylation and immune system development.[201–205]

In conclusion, all prenatal alcohol exposure is a risk and no safe lower limit can be defined, but binge drinking, with its high blood alcohol peaks, produces the most harmful effects on foetal development.





7 Strategies/interventions for reducing harms from binge drinking

Binge drinking is the fundamental building block of most alcohol-caused medical, social and economic problems. Fortunately, there are a number of effective public policies that can reduce binge drinking and resulting harm in the general population. In addition to public policies, individually based interventions can also be effective and are an important complement to broader public health strategies.

Most people who engage in binge drinking, and the vast majority of binge drinking episodes, accrue to those whose average consumption might be considered 'moderate' from a social or research perspective. However, binge drinking is also a strong risk factor for subsequent alcohol use disorder, AUD. AUD is a medical condition in which treatment and individual intervention are essential. Thus, strategies to reduce binge drinking can operate at both the population level through public policy and at the individual level as an important complement to broader public strategies.

7.1 Public policies

Binge drinking is a risk behaviour which can be reduced through population-based prevention strategies. A number of effective public policies exist which can reduce binge drinking and resulting harms in the general population.[206]

Although not all policy studies evaluate impacts on binge drinking, international alcohol research has long demonstrated that changes in consumption are highly associated with changes in binge drinking.[207] That is, total alcohol use correlates with changes in other measures of consumption, including binge drinking.[15] For example, if a policy intervention reduces total consumption in the population then that policy intervention also reduces binge drinking. This makes sense since 5+ drinking occasions account for a majority of alcohol consumption in the population.[16,18] Thus, reductions in total or average daily alcohol consumption means that binge drinking is also lower.

Policies resulting in reductions in outcomes related to acute alcohol impairment (e.g., alcohol-involved traffic crashes, violence, unintentional injuries) also offer strong indirect evidence that those policies must be reducing high per-occasion consumption, including binge drinking. Thus reductions in drink driving crashes among under 21 age drivers means high volume per occasion drinking has been reduced.

Some alcohol policies target binge drinking specifically, either directly or indirectly. Examples of laws that directly target binge drinking include those that forbid serving alcohol to intoxicated patrons or minors and laws forbidding public intoxication. Policies indirectly targeting binge drinking include earlier closing hours for bars or liquor stores because of the recognition that large proportions of those drinking very late at night or early in the morning are already impaired. Minimum alcohol purchase age laws are effective in part because of their effects on binge drinking, i.e., the prevalence of binge drinking is high among underage youth as are incidences of impaired driving. A greater proportion of alcohol consumption among youth is accounted for by 5+ drinking occasions.[208] Minimum unit pricing is effective in part because the cheapest alcohol products are disproportionately consumed by those who drink the most on a per-occasion basis. In addition, liability laws hold alcohol retailers liable for damages incurred because of service to intoxicated patrons, and social host laws hold adults criminally liable for intoxication-related harms incurred or caused by underage youth to whom they served alcohol. Finally, most laws and policies to reduce drink driving are inherently designed to reduce the likelihood of driving among those who are impaired by alcohol (e.g., BAC limits for driving, late night driving restrictions for youth, sobriety checkpoints).

The most cost-effective alcohol policies affect alcohol's affordability (i.e., price), its physical availability, or its social acceptability. The most common pricing policy is alcohol taxation, which can be levied as excise taxes (a monetary amount per volume of alcoholic beverage) or ad valorem taxes (based on a percentage of price). In addition to the impact of tax on population-level consumption and harms, there is a large literature demonstrating that higher prices from tax increases reduces binge drinking[209,210] and harmful outcomes related to high per-occasion consumption[211] (e.g., impaired driving, violence). Minimum unit pricing imposes a floor price based on ethanol content. Research evidence indicates that raising minimum unit prices reduces overall consumption, reduces consumption in heavy drinking households, reduces cirrhosis mortality, and reduces mortality risks among those with an AUD (virtually all of whom binge drink).[212] In terms of consumption at bars and restaurants, discounted drink specials may be associated with binge drinking, driving under the influence, fighting, and unprotected sex.[213]

Examples of effective policies that restrict the physical availability in addition to minimum age of alcohol purchase include restrictions on hours or days of alcohol sales,[214,215] bans on sales, and limits on the number of alcohol outlets (either outlets where alcohol is consumed 'off-premises' such as liquor stores or supermarkets, or outlets where alcohol is consumed 'on premises' such as bars, restaurants or nightclubs). Especially helpful



The most cost-effective alcohol policies affect alcohol's affordability (i.e., price), its physical availability, or its social acceptability.



Government monopolies on alcohol sales, either at the wholesale or retail levels, tend to be associated with fewer alcohol-caused harms and may operate through several mechanisms including price, availability, and more responsible marketing practices.

are studies which have specifically evaluated their potential to reduce binge drinking or related outcomes such as violence,[216] including intimate partner violence.[217] Such studies are direct demonstrations of the potential of public policies to actually have population effects.

Policies pertaining to social acceptability include restrictions (or lack thereof) on alcohol marketing; more marketing is associated with increased binge drinking.[218] While studies have shown that exposure to alcohol marketing is a risk factor for youth drinking and youth binge drinking,[219] these are not intervention studies to assess the impact of restrictions in marketing (there have been few examples in this direction). Efforts to de-glamorize alcohol consumption through counter-marketing or informational messaging have not been well studied. Social acceptability also covers the issue of health warnings on alcohol labels, which may decrease consumption,[220] as well as increase support for labelling and other alcohol control measures.[221]

Government monopolies on alcohol sales, either at the wholesale or retail levels, tend to be associated with fewer alcohol-caused harms and may operate through several mechanisms including price, availability, and more responsible marketing practices. Increases in privatization are associated with increased excessive drinking (including binge drinking) and related harms.[222,223]

A number of studies have examined the impact of the aggregate alcohol policy environment or multiple policies on binge drinking or outcomes related to impairment. For example, a 10% increase in a scale measuring the restrictiveness of U.S. state alcohol policies was associated with a 9% decrease in binge drinking prevalence among adults224 and an 7% decrease in prevalence among underage youth.[225] Alcohol taxes and outlet density accounted for approximately half of the effect magnitude observed for all 29 policies.[226] The same policy scale found that stronger policy environments were protective against harms to others, particularly related to vandalism and impaired driving.[227] A study across 28 countries using the International Alcohol Policy and Injury Index concluded that more restrictive alcohol policies are associated with lower the rates of alcohol-related injury.[228]

Suicide is a complex phenomenon, but many suicides involve impairment level BACs which can trigger suicide through acute dysphoria and impulsivity. A review found that a number of policies are associated with a reduced risk of suicide.[229,230]

7.2 Treatment and individual level interventions

Population strategies reduce binge drinking for the total population including persons with alcohol use disorder because virtually all persons with AUD engage in binge drinking, often on a regular basis. There are also individually based interventions that can reduce binge drinking or the consequences of binge drinking. Those with AUD should be referred for treatment which may involve counselling, medications or both. The success of these treatments relies to a large extent on the severity of alcohol dependence. Although there is no specific treatment for binge drinking, binge drinking is related to loss of control, which is a core symptom in alcohol dependence.

Medications including naltrexone, acamprosate and baclofen reduce binge drinking episodes.[231] Disulfiram remains the most effective medication to reduce drinking, including binge drinking. Behavioral interventions including behavioral self-control training also reduce binge drinking frequency and severity. GLP-1 agonists, used mainly for diabetes and weight loss, are an exciting new class of medications with promising evidence of effectiveness for AUD.[232] Psychedelics are another promising therapeutic option which may reduce binge drinking days,[233–235] but more studies with less risk of bias are needed.



Brief counselling interventions, which are typically 5–15 minutes sessions that use motivational interviewing or structured advice, are for patients who are screened for risky drinking (including binge drinking) regardless of their meeting the criteria for AUD. Such interventions have the potential to reduce consumption and binge drinking.[236] The focus in these interventions is on the harmful effect of alcohol on the presenting disorder, rather than on a diagnosis of AUD.

Other individually-oriented interventions include ignition interlock devices to reduce impaired driving among those convicted of impaired driving,[237,238] and newer driver alcohol detection system for safety (DADSS) which is a passive sensor to detect elevated BACs in the driver which can block the car's ignition system that is being developed by the National Highway Traffic Safety Administration (NHTSA) and the Automotive Coalition for Traffic Safety in the U.S. DADSS technology could be mandatory equipment in all motor vehicles, similar to seatbelts or airbags.



8 Conclusions and recommendations

Binge drinking is a major contributor to serious alcohol-related harm that has been largely overlooked and underestimated. Binge drinking is a dangerous pattern of consumption that involves consuming a large quantity of alcohol in a short period, typically leading to blood alcohol levels high enough to cause significant impairment. While definitions vary, consuming 4–5 or more drinks in one occasion or sitting is generally considered binge drinking. This pattern of drinking is dangerous because it leads to sudden spikes in blood alcohol concentration, overwhelming the body's ability to process alcohol, leading to oxidative stress and to an increase in acetaldehyde, a dangerous metabolite and a major carcinogen.

8.1 Why it matters

Binge drinking is common worldwide. In most high-income countries, over half of all alcohol consumed happens during binge drinking occasions, but in many low- and middle-income countries it is even more common. Binge drinking can cause greater harm in disadvantaged groups or groups with structural vulnerabilities. Binge drinking is also responsive to an array of population-based preventive strategies, discussed above.

8.2 Health risks

Binge drinking carries both immediate ("acute") and or long-term ("chronic") risks to health. That binge drinking often results in immediate consequences for health may be readily apparent to many drinkers and the harms themselves often highly visible from a societal perspective. In contrast, longer-term harms to health from binge drinking are more easily overlooked or under-estimated by drinkers and society at large, even though for some, binge drinking impacts may endure throughout life. For example:

- **IMMEDIATE HARMS;** Accidents, falls, car crashes, violence, sexual assault, alcohol poisoning, and harm to children from impaired caregivers.
- **LONG-TERM HARMS;** Alcohol use disorder (i.e., alcohol dependence), heart disease, stroke, dementia (especially early onset), depression, cancers (especially breast, liver, and mouth cancers), liver disease, pancreatitis, diabetes, and gastrointestinal damage.
- **ENDURING IMPACTS;** For young people, binge drinking can disrupt brain development and increase the likelihood of developing an alcohol use disorder, chronic diseases or dementia later in life. Drinking during pregnancy can cause serious lifelong conditions such as foetal alcohol spectrum disorders (FASD).



In most high-income countries, over half of all alcohol consumed happens during binge drinking occasions, but in many low- and middle-income countries it is even more common.

8.3 Social and economic costs

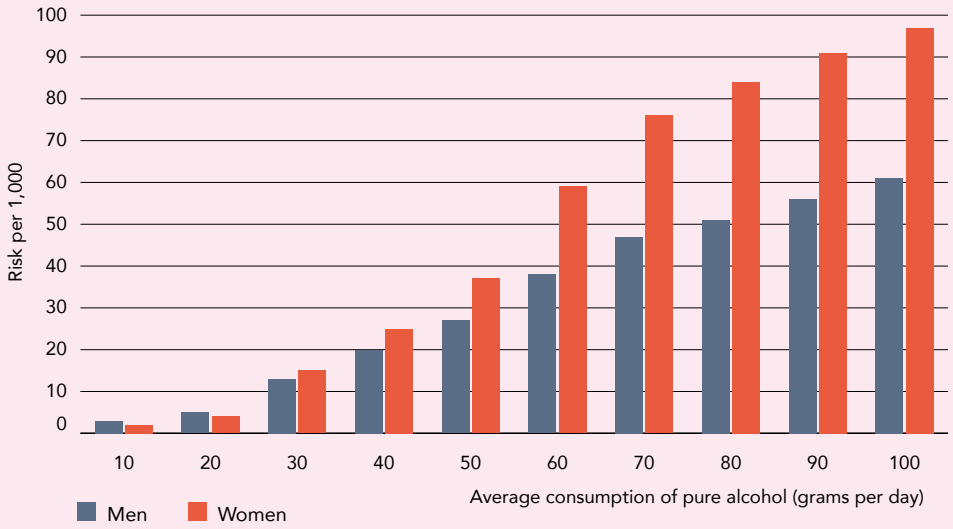
Consequences from binge drinking extend well beyond the individual drinker. Family violence, child neglect, lost productivity, crime, and health care costs are all closely tied to this drinking pattern. For Sweden, it is estimated that alcohol drinking reduces GDP by 2.3%[239] and costs around 103 billion SEK per year.[240]

8.4 Who is most at risk?

Those drinking more on a per occasion basis:

- **YOUNG PEOPLE:** More vulnerable due to brain development including poorly developed executive function.
- **WOMEN:** Risks of harms from binge drinking are greater for women than for men. Women are more affected physiologically by alcohol and (on average) weigh less, and they are also at higher risk of victimization.
- **MEN:** Men actually suffer more harms from binge drinking than women because they are more likely to binge drink, drink more per binge occasion, and may engage in more risk taking and aggression while impaired. As a result, men account for most impacts from binge drinking (e.g., those related to interpersonal violence and suicide)
- **LOWER-INCOME GROUPS**: Experience more severe harms even at similar or lower levels of drinking – the so-called "harms paradox."
- **FAMILIES:** Children of binge-drinking parents face emotional, developmental, and physical risks.

FIGURE 4 Lifetime risk per 1000 of overall chronic disease mortality by average daily alcohol consumption in grams per day of pure alcohol among men and women in Australia, 2002



Source: Figure 4 in Rehm et al. (2008). Method for moderation: measuring lifetime risk of alcohol-attributable mortality as a basis for drinking guidelines. Int J Methods Psychiatr Res. 2008;17(3):141-51

8.5 What works to reduce binge drinking?

Although binge drinking is common and dangerous, the good news is that there are many effective public policies that have been shown to reduce binge drinking and related consequences across the population. The main obstacle to public policy is political inaction abetted by efforts to undermine policy by vested interests. Ideally, effective public policies should be complemented with individual-level interventions.

- **POPULATION-LEVEL POLICIES**: Higher alcohol taxes, minimum unit pricing, restrictions on sales hours, reduced outlet density, and marketing limits.
- **LEGAL MEASURES:** Minimum drinking age laws, drink-driving enforcement, and liability laws for serving intoxicated patrons.
- **HEALTH INTERVENTIONS:** Brief counselling in health care settings, medications, and treatments for alcohol use disorder.
- **TECHNOLOGY:** Tools like ignition interlocks and alcohol detection systems in cars.

8.6 Key takeaway and recommendations

Binge drinking is a common and dangerous pattern of alcohol use. Binge drinking is not just a matter of "partying too hard" – it is one of the biggest contributors to alcohol-related disease, injury, and death, causing immediate harms and lasting health and social impacts. It harms not only the drinker but also families, communities, and health systems. Effective policies and interventions can reduce binge drinking and save lives, but they have not been adopted or implemented optimally.

Over the past 15-20 years, there has been a growing recognition of the adverse health effects at low average levels of alcohol consumption that might be considered 'moderate' from a social perspective. While this is important, high per-occasion consumption, with binge drinking (i.e., drinking to the point of acute impairment), remains ubiquitous, normalized and sometimes celebrated in Swedish society, as well as in other countries. In many cases it is considered a rite of passage, or as a means by which to spectate at a sporting event or to celebrate a family holiday. All of this is facilitated with massive expenditures on alcohol marketing, especially on social (digital) media, by companies profiting from the sale of alcohol and with a vested interest in maintaining the status quo.

Drinking patterns in Sweden and other Nordic countries are typically binge-drinking-oriented and have been so for a long time. In addition to a need for more and better public policies to support wellbeing with respect to alcohol, a cultural shift is in order. Adoption of effective policies can contribute to that cultural shift but also would be a by-product of a shift driven by political leaders and the public. This shift also needs to happen in health care.



In addition to a need for more and better public policies to support wellbeing with respect to alcohol, a cultural shift is in order

References

1. Taylor, B., Irving, H. M., Kanteres, F., Room, R., Borges, G., Cherpitel, C., Greenfield, T., & Rehm, J. (2010). The more you drink, the harder you fall: A systematic review and meta-analysis of how acute alcohol consumption and injury or collision risk increase together. *Drug and Alcohol Dependence*, 110(1–2), 108–116. https://doi.org/10.1016/j.drugalcdep.2010.02.011
2. Stockwell, T., Sitharthan, T., McGRATH, D., & Lang, E. (1994). The measurement of alcohol dependence and impaired control in community samples. *Addiction, 89*(2), 167–184. https://doi.org/10.1111/j.1360-0443.1994.tb00875.x
3. Wilson, D. F., & Matschinsky, F. M. (2020). Ethanol metabolism: The good, the bad, and the ugly. *Medical Hypotheses, 140*, 109638. https://doi.org/10.1016/j.mehy.2020.109638
4. Jones, A. W. (2019). Alcohol, its absorption, distribution, metabolism, and excretion in the body and pharmacokinetic calculations. *WIREs Forensic Science, 1*(5), e1340. https://doi.org/10.1002/wfs2.1340
5. Cederbaum, A. I. (2012). Alcohol metabolism. *Clinics in Liver Disease, 16*(4), 667–685. https://doi.org/10.1016/j.cld.2012.08.002
6. Brooks, P. J., Enoch, M.-A., Goldman, D., Li, T.-K., & Yokoyama, A. (2009). The Alcohol Flushing Response: An Unrecognized Risk Factor for Esophageal Cancer from Alcohol Consumption. *PLoS Medicine, 6*(3), e1000050. https://doi.org/10.1371/journal.pmed.1000050
7. Seitz, H. K., & Stickel, F. (2010). Acetaldehyde as an underestimated risk factor for cancer development: Role of genetics in ethanol metabolism. *Genes & Nutrition, 5*(2), 121–128. https://doi.org/10.1007/s12263-009-0154-1
8. Fiore, M. (2024). Oxidative Stress in Alcohol Abuse: An Unfortunately Still Open Question. *Antioxidants, 13*(8), 934. https://doi.org/10.3390/antiox13080934
9. Lu, Y., & Cederbaum, A. I. (2008). CYP2E1 and oxidative liver injury by alcohol. *Free Radical Biology and Medicine, 44*(5), 723–738. https://doi.org/10.1016/j.freeradbiomed.2007.11.004
10. Morris, L. S., Dowell, N. G., Cercignani, M., Harrison, N. A., & Voon, V. (2018). Binge drinking differentially affects cortical and subcortical microstructure. *Addiction Biology, 23*(1), 403–411. https://doi.org/10.1111/adb.12493
11. Topiwala, A., Allan, C. L., Valkanova, V., Zsoldos, E., Filippini, N., Sexton, C., Mahmood, A., Fooks, P., Singh-Manoux, A., Mackay, C. E., Kivimäki, M., & Ebmeier, K. P. (2017). Moderate alcohol consumption as risk factor for adverse brain outcomes and cognitive decline: Longitudinal cohort study. *BMJ*, j2353. https://doi.org/10.1136/bmj.j2353
12. Chikritzhs, T., Dangardt, F., Holder, H., Naimi, T., Stockwell, T., & Andreasson, S. (2024). *Alcohol and the brain* (Alcohol and Society). Swedish Society of Nursing, SFAM, SAFF, CERA, The Swedish Society of Addiction Medicine, Movendi International, Swedish Brain Council, SLAN, Junis, UNF & IOGT-NTO. https://alcoholandsociety.report/alcohol-and-the-brain/
13. Goldman, M. R., Molina-Castro, M., Etkins, J. C., Koide, T. L., Ramchandani, V. A., Plawecki, M. H., Mennella, J. A., & Pepino, M. Y. (2025). Recent advances in alcohol metabolism: From the gut to the brain. *Physiological Reviews, 105*(4), 2501–2535. https://doi.org/10.1152/physrev.00053.2024
14. OECD. (2021). *Preventing Harmful Alcohol Use* (OECD Health Policy Studies). OECD Publishing. https://www.oecd.org/en/publications/preventing-harmful-alcohol-use_6e4b4ffb-en.html
15. Nelson, D. E., Naimi, T. S., Brewer, R. D., & Roeber, J. (2010). US state alcohol sales compared to survey data, 1993-2006. *Addiction (Abingdon, England), 105*(9), 1589–1596. https://doi.org/10.1111/j.1360-0443.2010.03007.x
16. Casswell, S., Callinan, S., Chaiyasong, S., Cuong, P. V., Kazantseva, E., Bayandorj, T., Huckle, T., Parker, K., Railton, R., & Wall, M. (2016). How the alcohol industry relies on harmful use of alcohol and works to protect its profits. *Drug and Alcohol Review, 35*(6), 661–664. https://doi.org/10.1111/dar.12460
17. Viet Cuong, P., Casswell, S., Parker, K., Callinan, S., Chaiyasong, S., Kazantseva, E., Meier, P., MacKintosh, A.-M., Piazza, M., Gray-Phillip, G., & Parry, C. D. H. (2018). Cross-country comparison of proportion of alcohol consumed in harmful drinking occasions using the International Alcohol Control Study. *Drug and Alcohol Review, 37 Suppl 2*(Suppl Suppl 2), S45–S52. https://doi.org/10.1111/dar.12665
18. Stockwell, T., & Zhao, J. (2023). Estimates of compliance with Canada's guidelines for low and moderate risk alcohol consumption: The importance of adjustment for underreporting in self-report surveys. *Canadian Journal of Public Health = Revue Canadienne De Sante Publique, 114*(6), 967–972. https://doi.org/10.17269/s41997-023-00781-6
19. WHO. (n.d.). *Global Information System on Alcohol and Health*. Retrieved 1 December 2025, from https://www.who.int/data/gho/data/themes/global-information-system-on-alcohol-and-health
20. Guttormsson, U. (2024). *Självrapporterade alkoholvanor i Sverige 2004–2023* [Elektronisk resurs]. Centralförbundet för alkohol- och narkotikaupplysning, CAN.
21. Eurostat. (2021, July). *Alcohol consumption statistics*. https://ec.europa.eu/eurostat/statistics-explained/index.php?title=Alcohol_consumption_statistics
22. Kanny, D., Naimi, T. S., Liu, Y., Lu, H., & Brewer, R. D. (2018). Annual Total Binge Drinks Consumed by U.S. Adults, 2015. *American Journal of Preventive Medicine, 54*(4), 486–496. https://doi.org/10.1016/j.amepre.2017.12.021
23. Boniface, S., & Shelton, N. (2013). How is alcohol consumption affected if we account for under-reporting? A hypothetical scenario. *European Journal of Public Health, 23*(6), 1076–1081. https://doi.org/10.1093/eurpub/ckt016
24. Naimi, T. S., Brewer, R. D., Mokdad, A., Denny, C., Serdula, M. K., & Marks, J. S. (2003). Binge drinking among US adults. *JAMA, 289*(1), 70–75. https://doi.org/10.1001/jama.289.1.70
25. Selling, L. (2024). *CAN:s nationella skolundersökning 2024* [Elektronisk resurs]. Centralförbundet för alkohol- och narkotikaupplysning, CAN.
26. Rossow, I., Moan, I. S., & Bye, E. K. (2022). Declining Trend in Adolescent Alcohol Use: Does It Have Any Significance for Drinking Behaviour in Young Adulthood? *International Journal of Environmental Research and Public Health, 19*(13), 7887. https://doi.org/10.3390/ijerph19137887
27. Gallup Inc. (2023, August 22). *Young Adults in U.S. Drinking Less Than in Prior Decades*. Gallup.Com. https://news.gallup.com/poll/509690/young-adults-drinking-less-prior-decades.aspx
28. Keyes, K. M., Jager, J., Mal-Sarkar, T., Patrick, M. E., Rutherford, C., & Hasin, D. (2019). Is There a Recent Epidemic of Women's Drinking? A Critical Review of National Studies. *Alcoholism, Clinical and Experimental Research, 43*(7), 1344–1359. https://doi.org/10.1111/acer.14082

29. Davis, A. R., & Lipson, A. H. (1986). Central-nervous-system depression and high blood ethanol levels. *Lancet (London, England), 1*(8480), 566. https://doi.org/10.1016/s0140-6736(86)90928-1

30. Andreasson, S., Chikritzhs, T., Dangardt, F., Holder, H., Naimi, T., & Stockwell, T. (2015). *Second-hand effects of alcohol consumption* (Alcohol and Society). Swedish Society of Medicine & IOGT-NTO.

31. Lieber, C. S., & DeCarli, L. M. (1991). Hepatotoxicity of ethanol. *Journal of Hepatology, 12*(3), 394–401. https://doi.org/10.1016/0168-8278(91)90846-4

32. Frezza, M., Di Padova, C., Pozzato, G., Terpin, M., Baraona, E., & Lieber, C. S. (1990). High Blood Alcohol Levels in Women: The Role of Decreased Gastric Alcohol Dehydrogenase Activity and First-Pass Metabolism. *New England Journal of Medicine, 322*(2), 95–99. https://doi.org/10.1056/NEJM199001113220205

33. Hermens, D. F., & Lagopoulos, J. (2018). Binge Drinking and the Young Brain: A Mini Review of the Neurobiological Underpinnings of Alcohol-Induced Blackout. *Frontiers in Psychology, 9*, 12. https://doi.org/10.3389/fpsyg.2018.00012

34. Jacob, A., & Wang, P. (2020). Alcohol Intoxication and Cognition: Implications on Mechanisms and Therapeutic Strategies. *Frontiers in Neuroscience, 14*, 102. https://doi.org/10.3389/fnins.2020.00102

35. Lovinger, D. M. (2025). Alcohol effects on associative and sensorimotor cortico-thalamo-basal ganglia circuits alter decision making and alcohol intake. *Alcohol (Fayetteville, N.Y.), 127*, 21–46. https://doi.org/10.1016/j.alcohol.2025.05.005

36. Cherpitel, C. J., Witbrodt, J., Korcha, R. A., Ye, Y., Monteiro, M. G., & Chou, P. (2019). Dose-Response Relationship of Alcohol and Injury Cause: Effects of Country-Level Drinking Pattern and Alcohol Policy. *Alcoholism, Clinical and Experimental Research, 43*(5), 850–856. https://doi.org/10.1111/acer.13986

37. Zeisser, C., Stockwell, T. R., Chikritzhs, T., Cherpitel, C., Ye, Y., & Gardner, C. (2013). A systematic review and meta-analysis of alcohol consumption and injury risk as a function of study design and recall period. *Alcoholism, Clinical and Experimental Research, 37 Suppl 1*(Suppl 1), E1-8. https://doi.org/10.1111/j.1530-0277.2012.01919.x

38. Cherpitel, C. J., Ye, Y., Bond, J., Borges, G., & Monteiro, M. (2015). Relative risk of injury from acute alcohol consumption: Modeling the dose-response relationship in emergency department data from 18 countries. *Addiction (Abingdon, England), 110*(2), 279–288. https://doi.org/10.1111/add.12755

39. Eckardt, M. J., File, S. E., Gessa, G. L., Grant, K. A., Guerri, C., Hoffman, P. L., Kalant, H., Koob, G. F., Li, T. K., & Tabakoff, B. (1998). Effects of moderate alcohol consumption on the central nervous system. *Alcoholism, Clinical and Experimental Research, 22*(5), 998–1040. https://doi.org/10.1111/j.1530-0277.1998.tb03695.x

40. Taylor, B., & Rehm, J. (2012). The relationship between alcohol consumption and fatal motor vehicle injury: High risk at low alcohol levels. *Alcoholism, Clinical and Experimental Research, 36*(10), 1827–1834. https://doi.org/10.1111/j.1530-0277.2012.01785.x

41. Fairbairn, C. E., Kang, D., & Federmeier, K. D. (2021). Alcohol and Neural Dynamics: A Meta-analysis of Acute Alcohol Effects on Event-Related Brain Potentials. *Biological Psychiatry, 89*(10), 990–1000. https://doi.org/10.1016/j.biopsych.2020.11.024

42. Dry, M. J., Burns, N. R., Nettelbeck, T., Farquharson, A. L., & White, J. M. (2012). Dose-related effects of alcohol on cognitive functioning. *PloS One, 7*(11), e50977. https://doi.org/10.1371/journal.pone.0050977

43. Bjork, J. M., & Gilman, J. M. (2014). The effects of acute alcohol administration on the human brain: Insights from neuroimaging. *Neuropharmacology, 84*, 101–110. https://doi.org/10.1016/j.neuropharm.2013.07.039

44. Zoethout, R. W. M., Delgado, W. L., Ippel, A. E., Dahan, A., & van Gerven, J. M. A. (2011). Functional biomarkers for the acute effects of alcohol on the central nervous system in healthy volunteers. *British Journal of Clinical Pharmacology, 71*(3), 331–350. https://doi.org/10.1111/j.1365-2125.2010.03846.x

45. Mirijello, A., Sestito, L., Antonelli, M., Gasbarrini, A., & Addolorato, G. (2023). Identification and management of acute alcohol intoxication. *European Journal of Internal Medicine, 108*, 1–8. https://doi.org/10.1016/j.ejim.2022.08.013

46. Ostling, E. W., & Fillmore, M. T. (2010). Tolerance to the impairing effects of alcohol on the inhibition and activation of behavior. *Psychopharmacology, 212*(4), 465–473. https://doi.org/10.1007/s00213-010-1972-y

47. Weafer, J., & Fillmore, M. T. (2012). Acute tolerance to alcohol impairment of behavioral and cognitive mechanisms related to driving: Drinking and driving on the descending limb. *Psychopharmacology, 220*(4), 697–706. https://doi.org/10.1007/s00213-011-2519-6

48. Almeida-Antunes, N., Crego, A., Carbia, C., Sousa, S. S., Rodrigues, R., Sampaio, A., & López-Caneda, E. (2021). Electroencephalographic signatures of the binge drinking pattern during adolescence and young adulthood: A PRISMA-driven systematic review. *NeuroImage: Clinical, 29*, 102537. https://doi.org/10.1016/j.nicl.2020.102537

49. Crews, F. T., Robinson, D. L., Chandler, L. J., Ehlers, C. L., Mulholland, P. J., Pandey, S. C., Rodd, Z. A., Spear, L. P., Swartzwelder, H. S., & Vetreno, R. P. (2019). Mechanisms of Persistent Neurobiological Changes Following Adolescent Alcohol Exposure: NADIA Consortium Findings. *Alcoholism: Clinical and Experimental Research, 43*(9), 1806–1822. https://doi.org/10.1111/acer.14154

50. Crews, F. T., Vetreno, R. P., Broadwater, M. A., & Robinson, D. L. (2016). Adolescent Alcohol Exposure Persistently Impacts Adult Neurobiology and Behavior. *Pharmacological Reviews, 68*(4), 1074–1109. https://doi.org/10.1124/pr.115.012138

51. Degenhardt, L., O'Loughlin, C., Swift, W., Romaniuk, H., Carlin, J., Coffey, C., Hall, W., & Patton, G. (2013). The persistence of adolescent binge drinking into adulthood: Findings from a 15-year prospective cohort study. *BMJ Open, 3*(8), e003015. https://doi.org/10.1136/bmjopen-2013-003015

52. Bye, E. K., Bogstrand, S. T., & Rossow, I. (2022). The importance of alcohol in elderly's hospital admissions for fall injuries: A population case-control study. *Nordisk Alkohol- & Narkotikatidskrift: NAT, 39*(1), 38–49. https://doi.org/10.1177/14550725211015836

53. Andreasson, S., Chikritzhs, T., Dangardt, F., Holder, H., Naimi, T., & Stockwell, T. (2014). *Evidence About Health Effects of "Moderate" Alcohol Consumption: Reasons for Scepticism and Public Health Implications* (No. 2014 The Effects of Low-dose Alcohol Consumption; Alcohol and Society, pp. 6–23). IOGT-NTO, Swedish Society of Medicine. https://alcoholandsociety.report/effects-of-low-dose-alcohol-consumption-alcohol-and-society/

54. Aira, M., Hartikainen, S., & Sulkava, R. (2005). Community prevalence of alcohol use and concomitant use of medication – A source of possible risk in the elderly aged 75 and older? *International Journal of Geriatric Psychiatry, 20*(7), 680–685. https://doi.org/10.1002/gps.1340

55. Li, T., Beard, J. D., Orr, W. E., Kwo, P. Y., & Ramchandani, V. A. (1998). GENDER AND ETHNIC DIFFERENCES IN ALCOHOL METABOLISM. *Alcoholism: Clinical and Experimental Research, 22*(3), 771–772. https://doi.org/10.1111/j.1530-0277.1998.tb04345.x

56. Pelletti, G., Boscolo-Berto, R., Anniballi, L., Giorgetti, A., Pirani, F., Cavallaro, M., Giorgini, L., Fais, P., Pascali, J. P., & Pelotti, S. (2024). Prevalence of alcohol-impaired driving: A systematic review with a gender-driven approach and meta-analysis of gender differences. *International Journal of Legal Medicine, 138*(6), 2523–2540. https://doi.org/10.1007/s00414-024-03291-3

57. Wilsnack, R. W., Wilsnack, S. C., Gmel, G., & Kantor, L. W. (2018). Gender Differences in Binge Drinking. *Alcohol Research: Current Reviews, 39*(1), 57–76.

58. Evans-Polce, R. J., Maggs, J. L., Lanza, S. T., & Patrick, M. E. (2024). Negative consequences associated with daily alcohol use as a nonlinear function of number of drinks in a daily diary study. *Drug and Alcohol Dependence, 256*, 111089. https://doi.org/10.1016/j.drugalcdep.2024.111089

59. Labhart, F., Livingston, M., Engels, R., & Kuntsche, E. (2018). After how many drinks does someone experience acute consequences-determining thresholds for binge drinking based on two event-level studies. *Addiction (Abingdon, England), 113*(12), 2235–2244. https://doi.org/10.1111/add.14370

60. *Insatser vid riskbruk av alkohol*. (2024). Socialstyrelsen.

61. *Australian Guidelines to Reduce Health Risks from Drinking Alcohol*. (2020). National Health and Medical Research Council.

62. Pascal, R., Liang, W., Gilmore, W., & Chikritzhs, T. (2013). Risks of alcohol-attributable hospitalisation and death in Australia over time: Evidence of divergence by region, age and sex. *The Australasian Medical Journal, 6*(3), 134–151. https://doi.org/10.4066/AMJ.2013.1618

63. Adams, R. S., McKetta, S. C., Jager, J., Stewart, M. T., & Keyes, K. M. (2023). Cohort effects of women's mid-life binge drinking and alcohol use disorder symptoms in the United States: Impacts of changes in timing of parenthood. *Addiction, 118*(10), 1932–1941. https://doi.org/10.1111/add.16262

64. Shuey, B., Wen, H., Suda, K. J., Burnett, A., Wharam, J. F., Anderson, T. S., & Liebschutz, J. M. (2025). Sex-Based Differences in Binge and Heavy Drinking Among US Adults. *JAMA, 333*(20), 1831–1833. https://doi.org/10.1001/jama.2025.2726

65. Vieira, E., Taylor, N., Stevely, A., Pennay, A., Raninen, J., Holmes, J., Vashishtha, R., & Livingston, M. (2025). A systematic review of adolescent alcohol-related harm trends in high-income countries with declines in adolescent consumption. *Addiction (Abingdon, England), 120*(8), 1551–1570. https://doi.org/10.1111/add.70026

66. Treur, J. L., Munafò, M. R., Logtenberg, E., Wiers, R. W., & Verweij, K. J. H. (2021). Using Mendelian randomization analysis to better understand the relationship between mental health and substance use: A systematic review. *Psychological Medicine, 51*(10), 1593–1624. https://doi.org/10.1017/S003329172100180X

67. Bloomfield, K., Grittner, U., Kramer, S., & Gmel, G. (2006). Social inequalities in alcohol consumption and alcohol-related problems in the study countries of the EU concerted action 'Gender, Culture and Alcohol Problems: A Multi-national Study'. *Alcohol and Alcoholism (Oxford, Oxfordshire). Supplement, 41*(1), i26-36. https://doi.org/10.1093/alcalc/agl073

68. Laslett, A.-M., Room, R., Kuntsche, S., Anderson-Luxford, D., Willoughby, B., Doran, C., Jenkinson, R., Smit, K., Egerton-Warburton, D., & Jiang, H. (2023). Alcohol's harm to others in 2021: Who bears the burden? *Addiction (Abingdon, England), 118*(9), 1726–1738. https://doi.org/10.1111/add.16205

69. Boyd, J., Sexton, O., Angus, C., Meier, P., Purshouse, R. C., & Holmes, J. (2022). Causal mechanisms proposed for the alcohol harm paradox-a systematic review. *Addiction (Abingdon, England), 117*(1), 33–56. https://doi.org/10.1111/add.15567

70. Naimi, T. S., Sherk, A., Lawrence, K. W., Reece, J., & Esser, M. B. (2025). Racial and Ethnic Disparities in Alcohol Consumption and Mortality in the U.S. *American Journal of Preventive Medicine, 69*(6), 107968. https://doi.org/10.1016/j.amepre.2025.107968

71. Bellis, M. A., Hughes, K., Nicholls, J., Sheron, N., Gilmore, I., & Jones, L. (2016). The alcohol harm paradox: Using a national survey to explore how alcohol may disproportionately impact health in deprived individuals. *BMC Public Health, 16*, 111. https://doi.org/10.1186/s12889-016-2766-x

72. Andreasson, S., Chikritzhs, T., Dangardt, F., Holder, H., Naimi, T., & Stockwell, T. (2020). *Alcohol, pregnancy and infant health – a shared responsibility* (Alcohol and Society). Swedish Society of Nursing, SFAM, SAFF, CERA & IOGT-NTO. https://alcoholandsociety.report/alcohol-pregnancy-and-infant-health/

73. Lange, S., Probst, C., Rehm, J., & Popova, S. (2017). Prevalence of binge drinking during pregnancy by country and World Health Organization region: Systematic review and meta-analysis. *Reproductive Toxicology (Elmsford, N.Y.), 73*, 214–221. https://doi.org/10.1016/j.reprotox.2017.08.004

74. Volpicelli, J. R., & Menzies, P. (2022). Rethinking Unhealthy Alcohol Use in the United States: A Structured Review. *Substance Abuse: Research and Treatment, 16*, 11782218221111832. https://doi.org/10.1177/11782218221111832

75. Zhang, S., Wang, L., Yang, T., Chen, L., Zhao, L., Wang, T., Chen, L., Ye, Z., Zheng, Z., & Qin, J. (2020). Parental alcohol consumption and the risk of congenital heart diseases in offspring: An updated systematic review and meta-analysis. *European Journal of Preventive Cardiology, 27*(4), 410–421. https://doi.org/10.1177/2047487319874530

76. Bianchi, E., Boekelheide, K., Sigman, M., Braun, J. M., Eliot, M., Hall, S. J., Dere, E., & Hwang, K. (2019). Spermatozoal large RNA content is associated with semen characteristics, sociodemographic and lifestyle factors. *PloS One, 14*(5), e0216584. https://doi.org/10.1371/journal.pone.0216584

77. Li, Y., Lin, H., Li, Y., & Cao, J. (2011). Association between socio-psycho-behavioral factors and male semen quality: Systematic review and meta-analyses. *Fertility and Sterility, 95*(1), 116–123. https://doi.org/10.1016/j.fertnstert.2010.06.031

78. Borges, E., Braga, D. P. de A. F., Provenza, R. R., Figueira, R. de C. S., Iaconelli, A., & Setti, A. S. (2018). Paternal lifestyle factors in relation to semen quality and in vitro reproductive outcomes. *Andrologia, 50*(9), e13090. https://doi.org/10.1111/and.13090

79. Milne, E., Greenop, K. R., Scott, R. J., de Klerk, N. H., Bower, C., Ashton, L. J., Heath, J. A., & Armstrong, B. K. (2013). Parental alcohol consumption and risk of childhood acute lymphoblastic leukemia and brain tumors. *Cancer Causes & Control: CCC, 24*(2), 391–402. https://doi.org/10.1007/s10552-012-0125-5

80. Steinberger, E. K., Ferencz, C., & Loffredo, C. A. (2002). Infants with single ventricle: A population-based epidemiological study. *Teratology, 65*(3), 106–115. https://doi.org/10.1002/tera.10017

81. Landberg, J., Danielsson, A.-K., Falkstedt, D., & Hemmingsson, T. (2018). Fathers' Alcohol Consumption and Long-Term Risk for Mortality in Offspring. *Alcohol and Alcoholism (Oxford, Oxfordshire), 53*(6), 753–759. https://doi.org/10.1093/alcalc/agy058

82. Wilson, I. M., Eurenius, E., Lindkvist, M., Edin, K., & Edvardsson, K. (2019). Is there an association between pregnant women's experience of violence and their partner's drinking? A Swedish population-based study. *Midwifery, 69*, 84–91. https://doi.org/10.1016/j.midw.2018.10.019

83. McBride, N., & Johnson, S. (2016). Fathers' Role in Alcohol-Exposed Pregnancies: Systematic Review of Human Studies. *American Journal of Preventive Medicine, 51*(2), 240–248. https://doi.org/10.1016/j.amepre.2016.02.009

84. Townshend, J. M., & Duka, T. (2005). Binge drinking, cognitive performance and mood in a population of young social drinkers. *Alcoholism, Clinical and Experimental Research, 29*(3), 317–325. https://doi.org/10.1097/01.alc.0000156453.05028.f5

85. Alpert, H. R., Slater, M. E., Yoon, Y.-H., Chen, C. M., Winstanley, N., & Esser, M. B. (2022). Alcohol Consumption and 15 Causes of Fatal Injuries: A Systematic Review and Meta-Analysis. *American Journal of Preventive Medicine, 63*(2), 286–300. https://doi.org/10.1016/j.amepre.2022.03.025

86. Cherpitel, C. J., Ye, Y., Bond, J., Borges, G., Monteiro, M., Chou, P., & Hao, W. (2015). Alcohol Attributable Fraction for Injury Morbidity from the Dose-Response Relationship of Acute Alcohol Consumption: Emergency Department Data from 18 Countries. *Addiction, 110*(11), 1724–1732. https://doi.org/10.1111/add.13031

87. Shinozaki, J., Matsumoto, H., Saito, H., Murahara, T., Nagahama, H., Sakurai, Y., & Nagamine, T. (2024). Low blood concentration of alcohol enhances activity related to stopping failure in the right inferior frontal cortex. *Cerebral Cortex, 34*(3), bhae079. https://doi.org/10.1093/cercor/bhae079

88. Allen, A. J., Meda, S. A., Skudlarski, P., Calhoun, V. D., Astur, R., Ruopp, K. C., & Pearlson, G. D. (2009). Effects of alcohol on performance on a distraction task during simulated driving. Alcoholism, *Clinical and Experimental Research, 33*(4), 617–625. https://doi.org/10.1111/j.1530-0277.2008.00876.x

89. Høye, A. K., & Storesund Hesjevoll, I. (2023). Alcohol and driving-How bad is the combination? A meta-analysis. *Traffic Injury Prevention, 24*(5), 373–378. https://doi.org/10.1080/15389588.2023.2204984

90. Irwin, C., Iudakhina, E., Desbrow, B., & McCartney, D. (2017). Effects of acute alcohol consumption on measures of simulated driving: A systematic review and meta-analysis. *Accident Analysis & Prevention, 102*, 248–266. https://doi.org/10.1016/j.aap.2017.03.001

91. Valencia-Martín, J. L., Galán, I., & Rodríguez-Artalejo, F. (2008). The joint association of average volume of alcohol and binge drinking with hazardous driving behaviour and traffic crashes. *Addiction (Abingdon, England), 103*(5), 749–757. https://doi.org/10.1111/j.1360-0443.2008.02165.x

92. Bye, E. K., Bogstrand, S. T., & Rossow, I. (2022). The importance of alcohol in elderly's hospital admissions for fall injuries: A population case-control study. *Nordic Studies on Alcohol and Drugs, 39*(1), 38–49. https://doi.org/10.1177/14550725211015836

93. Andreasson, S., Chikritzhs, T., Dangardt, F., Holder, H., Naimi, T., & Stockwell, T. (2019). *Alcohol and the Elderly* (Alcohol and Society). Swedish Society of Nursing, Swedish Society of Medicine, SAFF, CERA & IOGT-NTO.

94. Chen, M. M., Carter, S. R., Curtis, B. J., O'Halloran, E. B., Gamelli, R. L., & Kovacs, E. J. (2017). Alcohol Modulation of the Postburn Hepatic Response: *Journal of Burn Care & Research, 38*(1), e144–e157. https://doi.org/10.1097/BCR.0000000000000279

95. Sundin, E., Landberg, J., Galanti, M. R., Room, R., & Ramstedt, M. (2022). Country-Level Heavy Episodic Drinking and Individual-Level Experiences of Harm from Others' Drinking-Related Aggression in 19 European Countries. *European Addiction Research, 28*(2), 134–142. https://doi.org/10.1159/000520079

96. Laslett, A.-M., Anderson-Luxford, D., Willoughby, B., Room, R., Doran, C., Egerton-Warburton, D., Jenkinson, R., Smit, K., & Jiang, H. (2024). Harm from the drinking of people you know: A range of effects from different relationships. *Addiction (Abingdon, England), 119*(8), 1460–1467. https://doi.org/10.1111/add.16509

97. Callinan, S., Rankin, G., Room, R., Stanesby, O., Rao, G., Waleewong, O., Greenfield, T. K., Hope, A., & Laslett, A.-M. (2019). Harms from a partner's drinking: An international study on adverse effects and reduced quality of life for women. *The American Journal of Drug and Alcohol Abuse, 45*(2), 170–178. https://doi.org/10.1080/00952990.2018.1540632

98. Storvoll, E. E., Moan, I. S., & Lund, I. O. (2016). Negative consequences of other people's drinking: Prevalence, perpetrators and locations: Harm from other people's drinking. *Drug and Alcohol Review, 35*(6), 755–762. https://doi.org/10.1111/dar.12376

99. Moan, I. S., Storvoll, E. E., Sundin, E., Lund, I. O., Bloomfield, K., Hope, A., Ramstedt, M., Huhtanen, P., & Kristjánsson, S. (2015). Experienced Harm from Other People's Drinking: A Comparison of Northern European Countries. *Substance Abuse: Research and Treatment, 9*(Suppl 2), 45–57. https://doi.org/10.4137/SART.S23504

100. Darke, S. (2010). The toxicology of homicide offenders and victims: A review. *Drug and Alcohol Review, 29*(2), 202–215. https://doi.org/10.1111/j.1465-3362.2009.00099.x

101. Kuhns, J. B., Wilson, D. B., Clodfelter, T. A., Maguire, E. R., & Ainsworth, S. A. (2011). A meta-analysis of alcohol toxicology study findings among homicide victims: Alcohol toxicology findings among homicide victims. *Addiction, 106*(1), 62–72. https://doi.org/10.1111/j.1360-0443.2010.03153.x

102. Waterman, E. A., Lee, K. D. M., & Edwards, K. M. (2019). Longitudinal Associations of Binge Drinking with Interpersonal Violence Among Adolescents. *Journal of Youth and Adolescence, 48*(7), 1342–1352. https://doi.org/10.1007/s10964-019-01035-w

103. Kvillemo P, Andréasson S, Bränström R, El-Khouri BM, & Karlsson L. (2008). E*ffekter av lokalt alkohol- och narkotikaförebyggande arbete. Utvärdering av det förebyggande arbetet i sex försökskommuner. (Effects of local alcohol and drug preventive work. Evaluation of the preventive work in six trial communities).* (No. R 2008:22). Swedish National Institute of Public Health.

104. Leung, J. Y. Y., Parker, K., Lin, E.-Y., & Huckle, T. (2025). The association of parental or caregiver alcohol use with child maltreatment: A systematic review and meta-analysis of longitudinal studies. *Addiction (Abingdon, England), 120*(9), 1724–1738. https://doi.org/10.1111/add.70055

105. Hopkins, C., Haugland, S. H., Greenfield, T. K., Tamutienė, I., Hettige, S., & Laslett, A.-M. (2024). Harms to children from men's heavy drinking: A scoping review. *International Journal of Alcohol and Drug Research, 12*(2), 85–100. https://doi.org/10.7895/ijadr.523

106. Laslett, A.-M., Rankin, G., Waleewong, O., Callinan, S., Hoang, H. T. M., Florenzano, R., Hettige, S., Obot, I., Siengsounthone, L., Ibanga, A., Hope, A., Landberg, J., Vu, H. T. M., Thamarangsi, T., Rekve, D., & Room, R. (2017). A Multi-Country Study of Harms to Children Because of Others' Drinking. *Journal of Studies on Alcohol and Drugs, 78*(2), 195–202. https://doi.org/10.15288/jsad.2017.78.195

107. Laslett, A., Kuntsche, S., Wilson, I. M., Taft, A., Fulu, E., Jewkes, R., & Graham, K. (2022). The relationship between fathers' heavy episodic drinking and fathering involvement in five Asia-Pacific countries: An individual participant data meta-analysis. *Alcoholism: Clinical and Experimental Research, 46*(12), 2137–2148. https://doi.org/10.1111/acer.14955

108. Burdzovic Andreas, J., Torvik, F. A., & Lund, I. O. (2022). Parental binge drinking and offspring's high school non-completion: A prospective HUNT survey and educational registry study. *Drug and Alcohol Dependence, 230*, 109189. https://doi.org/10.1016/j.drugalcdep.2021.109189

109. Thor, S., Hemmingsson, T., Danielsson, A.-K., & Landberg, J. (2022). Fathers' alcohol consumption and risk of substance-related disorders in offspring. *Drug and Alcohol Dependence, 233*, 109354. https://doi.org/10.1016/j.drugalcdep.2022.109354

110. Lindsay, A. R., Shearer, R. D., Bart, G., & Winkelman, T. N. A. (2022). Trends in Substance Use Disorder-related Admissions at a Safety-net Hospital, 2008 – 2020. *Journal of Addiction Medicine, 16*(3), 360–363. https://doi.org/10.1097/ADM.0000000000000896

111. Norstrom, T., & Razvodovsky, Y. (2010). Per capita alcohol consumption and alcohol-related harm in Belarus, 1970-2005. *The European Journal of Public Health, 20*(5), 564–568. https://doi.org/10.1093/eurpub/ckq011

112. Vardy, J., Keliher, T., Fisher, J., Ritchie, F., Bell, C., Chekroud, M., Clarey, F., Blackwood, L., Barry, L., Paton, E., Clark, A., & Connelly, R. (2016). Quantifying alcohol-related emergency admissions in a UK tertiary referral hospital: A cross-sectional study of chronic alcohol dependency and acute alcohol intoxication. *BMJ Open, 6*(6), e010005. https://doi.org/10.1136/bmjopen-2015-010005

113. Tian, Y., Liu, J., Zhao, Y., Jiang, N., Liu, X., Zhao, G., & Wang, X. (2023). Alcohol consumption and all-cause and cause-specific mortality among US adults: Prospective cohort study. *BMC Medicine, 21*(1), 208. https://doi.org/10.1186/s12916-023-02907-6

114. Nyberg, S. T., Batty, G. D., Pentti, J., Madsen, I. E. H., Alfredsson, L., Bjorner, J. B., Borritz, M., Burr, H., Ervasti, J., Goldberg, M., Jokela, M., Knutsson, A., Koskinen, A., Lallukka, T., Lindbohm, J. V., Nielsen, M. L., Oksanen, T., Pejtersen, J. H., Pietiläinen, O., … Kivimäki, M. (2022). Association of alcohol use with years lived without major chronic diseases: A multicohort study from the IPD-Work consortium and UK Biobank. *The Lancet Regional Health. Europe, 19*, 100417. https://doi.org/10.1016/j.lanepe.2022.100417

115. Correale, M., & Brunetti, N. D. (2022). Alcohol consumption and subclinical and clinical coronary heart disease: New insight into potential causal mechanisms. *European Journal of Preventive Cardiology, 29*(15), 2003–2005. https://doi.org/10.1093/eurjpc/zwac190

116. Alvarado, J. D., Zuniga, P., Stringer, I., Ramirez, A., Cortes, E., & Solano, J. (2025). Holiday Heart Syndrome: A Literature Review. *Cureus, 17*(2), e79816. https://doi.org/10.7759/cureus.79816

117. Pletcher, M. J., Varosy, P., Kiefe, C. I., Lewis, C. E., Sidney, S., & Hulley, S. B. (2005). Alcohol consumption, binge drinking, and early coronary calcification: Findings from the Coronary Artery Risk Development in Young Adults (CARDIA) Study. *American Journal of Epidemiology, 161*(5), 423–433. https://doi.org/10.1093/aje/kwi062

118. Rehm, J., Gmel, G. E., Gmel, G., Hasan, O. S. M., Imtiaz, S., Popova, S., Probst, C., Roerecke, M., Room, R., Samokhvalov, A. V., Shield, K. D., & Shuper, P. A. (2017). The relationship between different dimensions of alcohol use and the burden of disease – An update. *Addiction, 112*(6), 968–1001. https://doi.org/10.1111/add.13757

119. Dangardt, F., Holder, H., Naimi, T., Stockwell, T., Andreasson, S., & Chikritzhs, T. (2025). H*ealth and Social Benefits from Reduced Alcohol Consumption: When Less is More* (Alcohol and Society). Swedish Society of Nursing, SFAM, SAFF, CERA & IOGT-NTO. https://alcoholandsociety.report/wp-content/uploads/2025/03/When-less-is-more_Alcohol-and-society-2025_report_en.pdf

120. Rehm, J., Rovira, P., Llamosas-Falcón, L., & Shield, K. D. (2021). Dose-Response Relationships between Levels of Alcohol Use and Risks of Mortality or Disease, for All People, by Age, Sex, and Specific Risk Factors. *Nutrients, 13*(8), 2652. https://doi.org/10.3390/nu13082652

121. Andreasson, S., Chikritzhs, T., Dangardt, F., Holder, H., Naimi, T., & Stockwell, T. (2023). *Alcohol and blood pressure* (Alcohol and Society). Swedish Society of Nursing, SFAM, SAFF, CERA, The Swedish Society of Addiction Medicine, SIGHT, Movendi International, Swedish Heart and Lung Association, SLAN & IOGT-NTO. https://alcoholandsociety.report/alcohol-and-blood-pressure/

122. Roerecke, M., & Rehm, J. (2014). Chronic heavy drinking and ischaemic heart disease: A systematic review and meta-analysis. *Open Heart, 1*(1), e000135. https://doi.org/10.1136/openhrt-2014-000135

123. Degerud, E., Høiseth, G., Mørland, J., Ariansen, I., Graff-Iversen, S., Ystrom, E., Zuccolo, L., Tell, G. S., & Næss, Ø. (2021). Associations of Binge Drinking With the Risks of Ischemic Heart Disease and Stroke: A Study of Pooled Norwegian Health Surveys. *American Journal of Epidemiology, 19*0(8), 1592–1603. https://doi.org/10.1093/aje/kwab063

124. Skov-Ettrup, L. S., Eliasen, M., Ekholm, O., Grønbæk, M., & Tolstrup, J. S. (2011). Binge drinking, drinking frequency, and risk of ischaemic heart disease: A population-based cohort study. *Scandinavian Journal of Public Health, 39*(8), 880–887. https://doi.org/10.1177/1403494811425605

125. Roerecke, M., & Rehm, J. (2010). Irregular Heavy Drinking Occasions and Risk of Ischemic Heart Disease: A Systematic Review and Meta-Analysis. *American Journal of Epidemiology, 171*(6), 633–644. https://doi.org/10.1093/aje/kwp451

126. Ricci, C., Wood, A., Muller, D., Gunter, M. J., Agudo, A., Boeing, H., van der Schouw, Y. T., Warnakula, S., Saieva, C., Spijkerman, A., Sluijs, I., Tjønneland, A., Kyrø, C., Weiderpass, E., Kühn, T., Kaaks, R., Sánchez, M.-J., Panico, S., Agnoli, C., … Ferrari, P. (2018). Alcohol intake in relation to non-fatal and fatal coronary heart disease and stroke: EPIC-CVD case-cohort study. *BMJ (Clinical Research Ed.), 361*, k934. https://doi.org/10.1136/bmj.k934

127. Kauhanen, J., Kaplan, G. A., Goldberg, D. E., & Salonen, J. T. (1997). Beer binging and mortality: Results from the Kuopio ischaemic heart disease risk factor study, a prospective population based study. *BMJ (Clinical Research Ed.), 315*(7112), 846–851. https://doi.org/10.1136/bmj.315.7112.846

128. Malyutina, S., Bobak, M., Kurilovitch, S., Gafarov, V., Simonova, G., Nikitin, Y., & Marmot, M. (2002). Relation between heavy and binge drinking and all-cause and cardiovascular mortality in Novosibirsk, Russia: A prospective cohort study. *Lancet (London, England), 360*(9344), 1448–1454. https://doi.org/10.1016/S0140-6736(02)11470-X

129. Mukamal, K. J., Maclure, M., Muller, J. E., & Mittleman, M. A. (2005). Binge drinking and mortality after acute myocardial infarction. *Circulation, 112*(25), 3839–3845. https://doi.org/10.1161/CIRCULATIONAHA.105.574749

130. Smyth, A., O'Donnell, M., Rangarajan, S., Hankey, G. J., Oveisgharan, S., Canavan, M., McDermott, C., Xavier, D., Zhang, H., Damasceno, A., Avezum, A., Pogosova, N., Oguz, A., Ryglewicz, D., Iversen, H. K., Lanas, F., Rosengren, A., Yusuf, S., Langhorne, P., & INTERSTROKE Investigators. (2023). Alcohol Intake as a Risk Factor for Acute Stroke: The INTERSTROKE Study. *Neurology, 100*(2), e142–e153. https://doi.org/10.1212/WNL.0000000000201388

131. Larsson, S. C., Wallin, A., Wolk, A., & Markus, H. S. (2016). Differing association of alcohol consumption with different stroke types: A systematic review and meta-analysis. *BMC Medicine, 14*(1), 178. https://doi.org/10.1186/s12916-016-0721-4

132. Zhang, C., Qin, Y.-Y., Chen, Q., Jiang, H., Chen, X.-Z., Xu, C.-L., Mao, P.-J., He, J., & Zhou, Y.-H. (2014). Alcohol intake and risk of stroke: A dose-response meta-analysis of prospective studies. *International Journal of Cardiology, 174*(3), 669–677. https://doi.org/10.1016/j.ijcard.2014.04.225

133. Patra, J., Taylor, B., Irving, H., Roerecke, M., Baliunas, D., Mohapatra, S., & Rehm, J. (2010). Alcohol consumption and the risk of morbidity and mortality for different stroke types – A systematic review and meta-analysis. *BMC Public Health, 10*, 258. https://doi.org/10.1186/1471-2458-10-258

134. Reynolds, K., Lewis, B., Nolen, J. D. L., Kinney, G. L., Sathya, B., & He, J. (2003). Alcohol consumption and risk of stroke: A meta-analysis. *JAMA, 289*(5), 579–588. https://doi.org/10.1001/jama.289.5.579

135. Hillbom, M., Saloheimo, P., & Juvela, S. (2011). Alcohol consumption, blood pressure, and the risk of stroke. *Current Hypertension Reports, 13*(3), 208–213. https://doi.org/10.1007/s11906-011-0194-y

136. Mostofsky, E., Chahal, H. S., Mukamal, K. J., Rimm, E. B., & Mittleman, M. A. (2016). Alcohol and Immediate Risk of Cardiovascular Events: A Systematic Review and Dose-Response Meta-Analysis. *Circulation, 133*(10), 979–987. https://doi.org/10.1161/CIRCULATIONAHA.115.019743

137. Nordström, P., Nordström, A., Eriksson, M., Wahlund, L.-O., & Gustafson, Y. (2013). Risk Factors in Late Adolescence for Young-Onset Dementia in Men: A Nationwide Cohort Study. *JAMA Internal Medicine, 173*(17), 1612. https://doi.org/10.1001/jamainternmed.2013.9079

138. Virtaa, J. J., Järvenpää, T., Heikkilä, K., Perola, M., Koskenvuo, M., Räihä, I., Rinne, J. O., & Kaprio, J. (2010). Midlife alcohol consumption and later risk of cognitive impairment: A twin follow-up study. *Journal of Alzheimer's Disease: JAD, 22*(3), 939–948. https://doi.org/10.3233/JAD-2010-100870

139. Järvenpää, T., Rinne, J. O., Koskenvuo, M., Räihä, I., & Kaprio, J. (2005). Binge drinking in midlife and dementia risk. *Epidemiology (Cambridge, Mass.), 16*(6), 766–771. https://doi.org/10.1097/01.ede.0000181307.30826.6c

140. Xu, W., Wang, H., Wan, Y., Tan, C., Li, J., Tan, L., & Yu, J.-T. (2017). Alcohol consumption and dementia risk: A dose–response meta-analysis of prospective studies. *European Journal of Epidemiology, 32*(1), 31–42. https://doi.org/10.1007/s10654-017-0225-3

141. Sabia, S., Fayosse, A., Dumurgier, J., Dugravot, A., Akbaraly, T., Britton, A., Kivimäki, M., & Singh-Manoux, A. (2018). Alcohol consumption and risk of dementia: 23 year follow-up of Whitehall II cohort study. *BMJ*, k2927. https://doi.org/10.1136/bmj.k2927

142. Krueger, K. R., Hainselin, M., Kuspinar, A., Barbosa, B. J. A. P., & de Jager Loots, C. A. (2025). Lifestyle and healthy aging to prevent cognitive decline and dementia. *Frontiers in Dementia, 4*, 1694494. https://doi.org/10.3389/frdem.2025.1694494

143. Topiwala, A., Levey, D. F., Zhou, H., Deak, J. D., Adhikari, K., Ebmeier, K. P., Bell, S., Burgess, S., Nichols, T. E., Gaziano, M., Stein, M., & Gelernter, J. (2025). Alcohol use and risk of dementia in diverse populations: Evidence from cohort, case-control and Mendelian randomisation approaches. *BMJ Evidence-Based Medicine*, bmjebm-2025-113913. https://doi.org/10.1136/bmjebm-2025-113913

144. Puddephatt, J., Irizar, P., Jones, A., Gage, S. H., & Goodwin, L. (2022). Associations of common mental disorder with alcohol use in the adult general population: A systematic review and meta-analysis. *Addiction, 117*(6), 1543–1572. https://doi.org/10.1111/add.15735

145. Brière, F. N., Rohde, P., Seeley, J. R., Klein, D., & Lewinsohn, P. M. (2014). Comorbidity between major depression and alcohol use disorder from adolescence to adulthood. *Comprehensive Psychiatry, 55*(3), 526–533. https://doi.org/10.1016/j.comppsych.2013.10.007

146. Kuntsche, E., Kuntsche, S., Thrul, J., & Gmel, G. (2017). Binge drinking: Health impact, prevalence, correlates and interventions. *Psychology & Health, 32*(8), 976–1017. https://doi.org/10.1080/08870446.2017.1325889

147. Jennison, K. M. (2004). The Short-Term Effects and Unintended Long-Term Consequences of Binge Drinking in College: A 10-Year Follow-Up Study. *The American Journal of Drug and Alcohol Abuse, 30*(3), 659–684. https://doi.org/10.1081/ADA-200032331

148. Li, J., Wang, H., Li, M., Shen, Q., Li, X., Zhang, Y., Peng, J., Rong, X., & Peng, Y. (2020). Effect of alcohol use disorders and alcohol intake on the risk of subsequent depressive symptoms: A systematic review and meta-analysis of cohort studies. *Addiction (Abingdon, England), 115*(7), 1224–1243. https://doi.org/10.1111/add.14935

149. Borges, G., Bagge, C. L., Cherpitel, C. J., Conner, K. R., Orozco, R., & Rossow, I. (2017). A meta-analysis of acute use of alcohol and the risk of suicide attempt. *Psychological Medicine, 47*(5), 949–957. https://doi.org/10.1017/S0033291716002841

150. Isaacs, J. Y., Smith, M. M., Sherry, S. B., Seno, M., Moore, M. L., & Stewart, S. H. (2022). Alcohol use and death by suicide: A meta-analysis of 33 studies. *Suicide & Life-Threatening Behavior, 52*(4), 600–614. https://doi.org/10.1111/sltb.12846

151. Amiri, S., & Behnezhad, S. (2020). Alcohol use and risk of suicide: A systematic review and Meta-analysis. *Journal of Addictive Diseases, 38*(2), 200–213. https://doi.org/10.1080/10550887.2020.1736757

152. Guo, K., Jiang, H., Shield, K. D., Spithoff, S., & Lange, S. (2025). Alcohol Consumption Per Capita and Suicide: A Meta-Analysis. *JAMA Network Open, 8*(9), e2533129. https://doi.org/10.1001/jamanetworkopen.2025.33129

153. Ning, K., Gondek, D., Patalay, P., & Ploubidis, G. B. (2020). The association between early life mental health and alcohol use behaviours in adulthood: A systematic review. *PLOS ONE, 15*(2), e0228667. https://doi.org/10.1371/journal.pone.0228667

154. McCambridge, J., McAlaney, J., & Rowe, R. (2011). Adult Consequences of Late Adolescent Alcohol Consumption: A Systematic Review of Cohort Studies. *PLoS Medicine, 8*(2), e1000413. https://doi.org/10.1371/journal.pmed.1000413

155. Liu, Y., Nguyen, N., & Colditz, G. A. (2015). Links between alcohol consumption and breast cancer: A look at the evidence. *Women's Health (London, England), 11*(1), 65–77. https://doi.org/10.2217/whe.14.62

156. Shield, K., Keyes, K. M., Martinez, P., Milam, A. J., Naimi, T. S., & Rehm, J. (2025). *Draft Report: Scientific Findings of the Alcohol Intake & Health Study for Public Comment*. Interagency Coordinating Committee on the Prevention of Underage Drinking (ICCPUD). https://www.samhsa.gov/substance-use/prevention/iccpud/alcohol-intake-health-study

157. Sánchez-Bayona, R., Gea, A., Gardeazabal, I., Romanos-Nanclares, A., Martínez-González, M. Á., Bes-Rastrollo, M., Santisteban, M., & Toledo, E. (2020). Binge Drinking and Risk of Breast Cancer: Results from the SUN ('Seguimiento Universidad de Navarra') Project. *Nutrients, 12*(3), 731. https://doi.org/10.3390/nu12030731

158. White, A. J., DeRoo, L. A., Weinberg, C. R., & Sandler, D. P. (2017). Lifetime Alcohol Intake, Binge Drinking Behaviors, and Breast Cancer Risk. *American Journal of Epidemiology, 186*(5), 541–549. https://doi.org/10.1093/aje/kwx118

159. Chen, J., Kehm, R., Yang, W., & Terry, M. B. (2024). Increasing rates of early-onset Luminal A breast cancers correlate with binge drinking patterns. *Breast Cancer Research: BCR, 26*(1), 145. https://doi.org/10.1186/s13058-024-01894-7

160. Xi, B., Veeranki, S. P., Zhao, M., Ma, C., Yan, Y., & Mi, J. (2017). Relationship of Alcohol Consumption to All-Cause, Cardiovascular, and Cancer-Related Mortality in U.S. Adults. *Journal of the American College of Cardiology, 70*(8), 913–922. https://doi.org/10.1016/j.jacc.2017.06.054

161. Koo, H. Y., Han, K., Shin, D. W., Yoo, J. E., Cho, M. H., Jeon, K. H., Kim, D., Hong, S., & Jun, J. K. (2021). Alcohol Drinking Pattern and Risk of Head and Neck Cancer: A Nationwide Cohort Study. *International Journal of Environmental Research and Public Health, 18*(21), 11204. https://doi.org/10.3390/ijerph182111204

162. Yokoyama, A., & Omori, T. (2003). Genetic polymorphisms of alcohol and aldehyde dehydrogenases and risk for esophageal and head and neck cancers. *Japanese Journal of Clinical Oncology, 33*(3), 111–121. https://doi.org/10.1093/jjco/hyg026

163. Zakhari, S. (2006). Overview: How is alcohol metabolized by the body? *Alcohol Research & Health: The Journal of the National Institute on Alcohol Abuse and Alcoholism, 29*(4), 245–254.

164. Toriola, A. T., Kurl, S., Laukkanen, J. A., & Kauhanen, J. (2009). Does binge drinking increase the risk of lung cancer: Results from the Findrink study. *European Journal of Public Health, 19*(4), 389–393. https://doi.org/10.1093/eurpub/ckp049

165. Dickerman, B. A., Markt, S. C., Koskenvuo, M., Pukkala, E., Mucci, L. A., & Kaprio, J. (2016). Alcohol intake, drinking patterns, and prostate cancer risk and mortality: A 30-year prospective cohort study of Finnish twins. *Cancer Causes & Control: CCC, 27*(9), 1049–1058. https://doi.org/10.1007/s10552-016-0778-6

166. Llamosas-Falcón, L., Rehm, J., Bright, S., Buckley, C., Carr, T., Kilian, C., Lasserre, A. M., Lemp, J. M., Zhu, Y., & Probst, C. (2023). The Relationship Between Alcohol Consumption, BMI, and Type 2 Diabetes: A Systematic Review and Dose-Response Meta-analysis. *Diabetes Care, 46*(11), 2076–2083. https://doi.org/10.2337/dc23-1015

167. Huang, J., Wang, X., & Zhang, Y. (2017). Specific types of alcoholic beverage consumption and risk of type 2 diabetes: A systematic review and meta-analysis. *Journal of Diabetes Investigation, 8*(1), 56–68. https://doi.org/10.1111/jdi.12537

168. Knott, C., Bell, S., & Britton, A. (2015). 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, 38*(9), 1804–1812. https://doi.org/10.2337/dc15-0710

169. Baliunas, D. O., Taylor, B. J., Irving, H., Roerecke, M., Patra, J., Mohapatra, S., & Rehm, J. (2009). Alcohol as a risk factor for type 2 diabetes: A systematic review and meta-analysis. *Diabetes Care, 32*(11), 2123–2132. https://doi.org/10.2337/dc09-0227

170. Li, X.-H., Yu, F.-F., Zhou, Y.-H., & He, J. (2016). Association between alcohol consumption and the risk of incident type 2 diabetes: A systematic review and dose-response meta-analysis. *The American Journal of Clinical Nutrition, 103*(3), 818–829. https://doi.org/10.3945/ajcn.115.114389

171. Koppes, L. L. J., Dekker, J. M., Hendriks, H. F. J., Bouter, L. M., & Heine, R. J. (2005). Moderate alcohol consumption lowers the risk of type 2 diabetes: A meta-analysis of prospective observational studies. *Diabetes Care, 28*(3), 719–725. https://doi.org/10.2337/diacare.28.3.719

172. Polsky, S., & Akturk, H. K. (2017). Alcohol Consumption, Diabetes Risk, and Cardiovascular Disease Within Diabetes. *Current Diabetes Reports, 17*(12), 136. https://doi.org/10.1007/s11892-017-0950-8

173. Estruch, R., & Hendriks, H. F. J. (2022). Associations between Low to Moderate Consumption of Alcoholic Beverage Types and Health Outcomes: A Systematic Review. *Alcohol and Alcoholism (Oxford, Oxfordshire), 57*(2), 176–184. https://doi.org/10.1093/alcalc/agab082

174. Nygren, K., Hammarström, A., & Rolandsson, O. (2017). Binge drinking and total alcohol consumption from 16 to 43 years of age are associated with elevated fasting plasma glucose in women: Results from the northern Swedish cohort study. *BMC Public Health, 17*(1), 509. https://doi.org/10.1186/s12889-017-4437-y

175. Holmes, M. V., Dale, C. E., Zuccolo, L., Silverwood, R. J., Guo, Y., Ye, Z., Prieto-Merino, D., Dehghan, A., Trompet, S., Wong, A., Cavadino, A., Drogan, D., Padmanabhan, S., Li, S., Yesupriya, A., Leusink, M., Sundstrom, J., Hubacek, J. A., Pikhart, H., … on behalf of The InterAct Consortium. (2014). Association between alcohol and cardiovascular disease: Mendelian randomisation analysis based on individual participant data. *BMJ, 349*(jul10 6), g4164–g4164. https://doi.org/10.1136/bmj.g4164

176. Liu, M., & Park, S. (2022). A causal relationship between alcohol intake and type 2 diabetes mellitus: A two-sample Mendelian randomization study. *Nutrition, Metabolism, and Cardiovascular Diseases: NMCD, 32*(12), 2865–2876. https://doi.org/10.1016/j.numecd.2022.08.013

177. Bellentani, S., Saccoccio, G., Costa, G., Tiribelli, C., Manenti, F., Sodde, M., Saveria Crocè, L., Sasso, F., Pozzato, G., Cristianini, G., & Brandi, G. (1997). Drinking habits as cofactors of risk for alcohol induced liver damage. The Dionysos Study Group. *Gut, 41*(6), 845–850. https://doi.org/10.1136/gut.41.6.845

178. Roerecke, M., Vafaei, A., Hasan, O. S. M., Chrystoja, B. R., Cruz, M., Lee, R., Neuman, M. G., & Rehm, J. (2019). Alcohol Consumption and Risk of Liver Cirrhosis: A Systematic Review and Meta-Analysis. *The American Journal of Gastroenterology, 114*(10), 1574–1586. https://doi.org/10.14309/ajg.0000000000000340

179. Haber, P. S., & Kortt, N. C. (2021). Alcohol use disorder and the gut. *Addiction (Abingdon, England), 116*(3), 658–667. https://doi.org/10.1111/add.15147

180. Rehm, J., Room, R., Monteiro, M. G., Gmel, G., Graham, K., Rehn, N., Sempos, C. T., Frick, U., & Jernigan, D. H. (2004). Alcohol use. In *Comparative quantification of health risks: Global and regional burden of disease attributable to selected major risk factors* (Vol. 2004). World Health Organization.

181. Niu, X., Zhu, L., Xu, Y., Zhang, M., Hao, Y., Ma, L., Li, Y., & Xing, H. (2023). Global prevalence, incidence, and outcomes of alcohol related liver diseases: A systematic review and meta-analysis. *BMC Public Health, 23*(1), 859. https://doi.org/10.1186/s12889-023-15749-x

182. Samokhvalov, A. V., Rehm, J., & Roerecke, M. (2015). Alcohol Consumption as a Risk Factor for Acute and Chronic Pancreatitis: A Systematic Review and a Series of Meta-analyses. *EBioMedicine, 2*(12), 1996–2002. https://doi.org/10.1016/j.ebiom.2015.11.023

183. Irving, H. M., Samokhvalov, A. V., & Rehm, J. (2009). Alcohol as a risk factor for pancreatitis. A systematic review and meta-analysis. JOP: Journal of the Pancreas, 10(4), 387–392.

184. Alsamarrai, A., Das, S. L. M., Windsor, J. A., & Petrov, M. S. (2014). Factors that affect risk for pancreatic disease in the general population: A systematic review and meta-analysis of prospective cohort studies. *Clinical Gastroenterology and Hepatology: The Official Clinical Practice Journal of the American Gastroenterological Association, 12*(10), 1635-1644.e5; quiz e103. https://doi.org/10.1016/j.cgh.2014.01.038

185. Singhvi, A., Abromitis, R., Althouse, A. D., Bataller, R., Arteel, G. E., & Yadav, D. (2020). Coexistence of alcohol-related pancreatitis and alcohol-related liver disease: A systematic review and meta-analysis. *Pancreatology: Official Journal of the International Association of Pancreatology (IAP) ... [et Al.], 20*(6), 1069–1077. https://doi.org/10.1016/j.pan.2020.07.412

186. Bujanda, L. (2000). The Effects of Alcohol Consumption Upon The Gastrointestinal Tract. *American Journal of Gastroenterology, 95*(12), 3374–3382. https://doi.org/10.1111/j.1572-0241.2000.03347.x

187. Shimpi, T. R., Shikhare, S. N., Chung, R., Wu, P., & Peh, W. C. G. (2019). Imaging of Gastrointestinal and Abdominal Emergencies in Binge Drinking. *Canadian Association of Radiologists Journal = Journal l'Association Canadienne Des Radiologistes, 70*(1), 52–61. https://doi.org/10.1016/j.carj.2018.10.009

188. Bode, C., & Bode, J. C. (2003). Effect of alcohol consumption on the gut. *Best Practice & Research Clinical Gastroenterology, 17*(4), 575–592. https://doi.org/10.1016/S1521-6918(03)00034-9

189. Bajaj, J. S. (2019). Alcohol, liver disease and the gut microbiota. *Nature Reviews Gastroenterology & Hepatology, 16*(4), 235–246. https://doi.org/10.1038/s41575-018-0099-1

190. Yuan, S., Chen, J., Ruan, X., Sun, Y., Zhang, K., Wang, X., Li, X., Gill, D., Burgess, S., Giovannucci, E., & Larsson, S. C. (2023). Smoking, alcohol consumption, and 24 gastrointestinal diseases: Mendelian randomization analysis. *eLife, 12*, e84051. https://doi.org/10.7554/eLife.84051

191. Naimi, T. S., Lipscomb, L. E., Brewer, R. D., & Gilbert, B. C. (2003). Binge drinking in the preconception period and the risk of unintended pregnancy: Implications for women and their children. P*ediatrics, 111*(5 Pt 2), 1136–1141.

192. Popova, S., Lange, S., Probst, C., Gmel, G., & Rehm, J. (2017). Estimation of national, regional, and global prevalence of alcohol use during pregnancy and fetal alcohol syndrome: A systematic review and meta-analysis. *The Lancet. Global Health, 5*(3), e290–e299. https://doi.org/10.1016/S2214-109X(17)30021-9

193. Roozen, S., Peters, G.-J. Y., Kok, G., Townend, D., Nijhuis, J., & Curfs, L. (2016). Worldwide Prevalence of Fetal Alcohol Spectrum Disorders: A Systematic Literature Review Including Meta-Analysis. *Alcoholism, Clinical and Experimental Research, 40*(1), 18–32. https://doi.org/10.1111/acer.12939

194. Popova, S., Charness, M. E., Burd, L., Crawford, A., Hoyme, H. E., Mukherjee, R. A. S., Riley, E. P., & Elliott, E. J. (2023). Fetal alcohol spectrum disorders. *Nature Reviews. Disease Primers, 9*(1), 11. https://doi.org/10.1038/s41572-023-00420-x

195. Jacobson, J. L., Akkaya-Hocagil, T., Ryan, L. M., Dodge, N. C., Richardson, G. A., Olson, H. C., Coles, C. D., Day, N. L., Cook, R. J., & Jacobson, S. W. (2021). Effects of prenatal alcohol exposure on cognitive and behavioral development: Findings from a hierarchical meta-analysis of data from six prospective longitudinal U.S. cohorts. *Alcoholism, Clinical and Experimental Research, 45*(10), 2040–2058. https://doi.org/10.1111/acer.14686

196. Flak, A. L., Su, S., Bertrand, J., Denny, C. H., Kesmodel, U. S., & Cogswell, M. E. (2014). The Association of Mild, Moderate, and Binge Prenatal Alcohol Exposure and Child Neuropsychological Outcomes: A Meta-Analysis. *Alcoholism: Clinical and Experimental Research, 38*(1), 214–226. https://doi.org/10.1111/acer.12214

197. Broccia, M., Hansen, B. M., Winckler, J. M., Larsen, T., Strandberg-Larsen, K., Torp-Pedersen, C., & Kesmodel, U. S. (2023). Heavy prenatal alcohol exposure and obstetric and birth outcomes: A Danish nationwide cohort study from 1996 to 2018. *The Lancet. Public Health, 8*(1), e28–e35. https://doi.org/10.1016/S2468-2667(22)00263-8

198. Akison, L. K., Hayes, N., Vanderpeet, C., Logan, J., Munn, Z., Middleton, P., Moritz, K. M., Reid, N., & Australian FASD Guidelines Development Group, on behalf of the Australian FASD Guidelines Consortium. (2024). Prenatal alcohol exposure and associations with physical size, dysmorphology and neurodevelopment: A systematic review and meta-analysis. *BMC Medicine, 22*(1), 467. https://doi.org/10.1186/s12916-024-03656-w

199. Patra, J., Bakker, R., Irving, H., Jaddoe, V., Malini, S., & Rehm, J. (2011). Dose–response relationship between alcohol consumption before and during pregnancy and the risks of low birthweight, preterm birth and small for gestational age (SGA) – A systematic review and meta-analyses. BJOG: *An International Journal of Obstetrics & Gynaecology, 118*(12), 1411–1421. https://doi.org/10.1111/j.1471-0528.2011.03050.x

200. Easey, K. E., Dyer, M. L., Timpson, N. J., & Munafò, M. R. (2019). Prenatal alcohol exposure and offspring mental health: A systematic review. *Drug and Alcohol Dependence, 197*, 344–353. https://doi.org/10.1016/j.drugalcdep.2019.01.007

201. Laufer, B. I., Chater-Diehl, E. J., Kapalanga, J., & Singh, S. M. (2017). Long-term alterations to DNA methylation as a biomarker of prenatal alcohol exposure: From mouse models to human children with fetal alcohol spectrum disorders. *Alcohol (Fayetteville, N.Y.), 60*, 67–75. https://doi.org/10.1016/j.alcohol.2016.11.009

202. Mandal, C., Halder, D., Jung, K. H., & Chai, Y. G. (2017). Gestational Alcohol Exposure Altered DNA Methylation Status in the Developing Fetus. *International Journal of Molecular Sciences, 18*(7), 1386. https://doi.org/10.3390/ijms18071386

203. Gao, J., Liu, B., Chen, H., Xu, P., Guo, X., Yao, D., Li, X., Wang, T., Wang, Y., Yao, H., Qiao, S., Yuan, J., & Liu, Y. (2026). The role of DNA methylation in alcohol-mediated neurodevelopmental toxicity. *Toxicology, 519*, 154315. https://doi.org/10.1016/j.tox.2025.154315

204. Noor, S., & Milligan, E. D. (2018). Lifelong Impacts of Moderate Prenatal Alcohol Exposure on Neuroimmune Function. *Frontiers in Immunology, 9*, 1107. https://doi.org/10.3389/fimmu.2018.01107

205. Lussier, A. A., Bodnar, T. S., & Weinberg, J. (2021). Intersection of Epigenetic and Immune Alterations: Implications for Fetal Alcohol Spectrum Disorder and Mental Health. *Frontiers in Neuroscience, 15*, 788630. https://doi.org/10.3389/fnins.2021.788630

206. Babor, T. F., Casswell, S., Graham, K., Huckle, T., Livingston, M., Österberg, E., Rehm, J., Room, R., Rossow, I., & Sornpaisarn, B. (2023). *Alcohol: No Ordinary Commodity: Research and public policy* (3rd edn). Oxford University PressOxford. https://doi.org/10.1093/oso/9780192844484.001.0001

207. Edwards, G., Anderson, P., Babor, T. F., Casswell, S., Ferrence, R., Giesbrecht, N., Godfrey, C., Holder, H., Lemmens, P., Mäkelä, K., Midanik, L. T., Norström, T., Österberg, E., Romelsjö, A., Room, R., Simpura, J., & Skog, O.-J. (1994). *Alcohol policy and the public good*. Oxford University Press.

208. Toomey, T. L., Rosenfeld, C., & Wagenaar, A. C. (1996). The Minimum Legal Drinking Age: History, Effectiveness, and Ongoing Debate. *Alcohol Health and Research World, 20*(4), 213–218.

209. Wagenaar, A. C., Salois, M. J., & Komro, K. A. (2009). Effects of beverage alcohol price and tax levels on drinking: A meta-analysis of 1003 estimates from 112 studies. *Addiction, 104*(2), 179–190. https://doi.org/10.1111/j.1360-0443.2008.02438.x

210. Guindon, G. E., Zhao, K., Fatima, T., Garasia, S., Quinn, N., Baskerville, N. B., & Paraje, G. (2022). Prices, taxes and alcohol use: A systematic umbrella review. *Addiction, 117*(12), 3004–3023. https://doi.org/10.1111/add.15966

211. Wagenaar, A. C., Tobler, A. L., & Komro, K. A. (2010). Effects of alcohol tax and price policies on morbidity and mortality: A systematic review. *American Journal of Public Health, 100*(11), 2270–2278. https://doi.org/10.2105/AJPH.2009.186007

212. Patterson, C., Giles, L., Whitehead, R., Greci, S., Ferguson, K., Fraser, C., Chalmers, N., Wyper, G. M. A., Myers, F., Craig, N., & Beeston, C. (2023). Evaluating the impact of minimum unit pricing for alcohol in Scotland: A theory-based synthesis of the evidence. *The Lancet, 402*, S14. https://doi.org/10.1016/S0140-6736(23)02065-2

213. Puac-Polanco, V., Keyes, K. M., Mauro, P. M., & Branas, C. C. (2020). A Systematic Review of Drink Specials, Drink Special Laws, and Alcohol-Related Outcomes. *Current Epidemiology Reports, 7*(4), 300–314. https://doi.org/10.1007/s40471-020-00247-0

214. Marcus, J., & Siedler, T. (2015). Reducing binge drinking? The effect of a ban on late-night off-premise alcohol sales on alcohol-related hospital stays in Germany. *Journal of Public Economics, 123*, 55–77. https://doi.org/10.1016/j.jpubeco.2014.12.010

215. Nepal, S., Kypri, K., Tekelab, T., Hodder, R. K., Attia, J., Bagade, T., Chikritzhs, T., & Miller, P. (2020). Effects of Extensions and Restrictions in Alcohol Trading Hours on the Incidence of Assault and Unintentional Injury: Systematic Review. *Journal of Studies on Alcohol and Drugs, 81*(1), 5–23. https://doi.org/10.15288/jsad.2020.81.5

216. Baumann, F., Buchwald, A., Friehe, T., Hottenrott, H., & Mechtel, M. (2019). The effect of a ban on late-night off-premise alcohol sales on violent crime: Evidence from Germany. *International Review of Law and Economics, 60*, 105850. https://doi.org/10.1016/j.irle.2019.105850

217. Kearns, M. C., Reidy, D. E., & Valle, L. A. (2016). *The Role of Alcohol Policies in Preventing Intimate Partner Violence: A Review of the Literature.*

218. Jernigan, D., Noel, J., Landon, J., Thornton, N., & Lobstein, T. (2017). Alcohol marketing and youth alcohol consumption: A systematic review of longitudinal studies published since 2008. *Addiction (Abingdon, England), 112* Suppl 1, 7–20. https://doi.org/10.1111/add.13591

219. Tanski, S. E., McClure, A. C., Li, Z., Jackson, K., Morgenstern, M., Li, Z., & Sargent, J. D. (2015). Cued Recall of Alcohol Advertising on Television and Underage Drinking Behavior. *JAMA Pediatrics, 169*(3), 264. https://doi.org/10.1001/jamapediatrics.2014.3345

220. Zhao, J., Stockwell, T., Vallance, K., & Hobin, E. (2020). The Effects of Alcohol Warning Labels on Population Alcohol Consumption: An Interrupted Time Series Analysis of Alcohol Sales in Yukon, Canada. *Journal of Studies on Alcohol and Drugs, 81*(2), 225–237. https://doi.org/10.15288/jsad.2020.81.225

221. Zuckermann, A. M. E., Morissette, K., Boland, L., Garcia, A. J., Domingo, F. R., Stockwell, T., & Hobin, E. (2024). The effects of alcohol container labels on consumption behaviour, knowledge, and support for labelling: A systematic review. *The Lancet Public Health, 9*(7), e481–e494. https://doi.org/10.1016/S2468-2667(24)00097-5

222. Hahn, R. A., Middleton, J. C., Elder, R., Brewer, R., Fielding, J., Naimi, T. S., Toomey, T. L., Chattopadhyay, S., Lawrence, B., & Campbell, C. A. (2012). Effects of Alcohol Retail Privatization on Excessive Alcohol Consumption and Related Harms. *American Journal of Preventive Medicine, 42*(4), 418–427. https://doi.org/10.1016/j.amepre.2012.01.002

223. Lintonen, T., Ahtinen, S., & Konu, A. (2020). Alcoholic beverage preferences among teenagers in Finland before and after the 2018 alcohol law change. *Nordic Studies on Alcohol and Drugs, 37*(2), 141–152. https://doi.org/10.1177/1455072520910547

224. Silver, D., Macinko, J., Giorgio, M., & Bae, J. Y. (2019). Evaluating the relationship between binge drinking rates and a replicable measure of U.S. state alcohol policy environments. *PLOS ONE, 14*(6), e0218718. https://doi.org/10.1371/journal.pone.0218718

225. Xuan, Z., Blanchette, J. G., Nelson, T. F., Nguyen, T. H., Hadland, S. E., Oussayef, N. L., Heeren, T. C., & Naimi, T. S. (2015). Youth Drinking in the United States: Relationships With Alcohol Policies and Adult Drinking. *Pediatrics, 136*(1), 18–27. https://doi.org/10.1542/peds.2015-0537

226. Xuan, Z., Blanchette, J., Nelson, T. F., Heeren, T., Oussayef, N., & Naimi, T. S. (2015). The alcohol policy environment and policy subgroups as predictors of binge drinking measures among US adults. *American Journal of Public Health, 105*(4), 816–822. https://doi.org/10.2105/AJPH.2014.302112

227. Greenfield, T. K., Cook, W. K., Karriker-Jaffe, K. J., Patterson, D., Kerr, W. C., Xuan, Z., & Naimi, T. S. (2019). The Relationship Between the U.S. State Alcohol Policy Environment and Individuals' Experience of Secondhand Effects: Alcohol Harms Due to Others' Drinking. *Alcoholism, Clinical and Experimental Research, 43*(6), 1234–1243. https://doi.org/10.1111/acer.14054

228. Cherpitel, C. J., Witbrodt, J., Korcha, R. A., Ye, Y., Kool, B., & Monteiro, M. (2018). Multi-level analysis of alcohol-related injury, societal drinking pattern and alcohol control policy: Emergency department data from 28 countries. *Addiction, 113*(11), 2031–2040. https://doi.org/10.1111/add.14276

229. Xuan, Z., Naimi, T. S., Kaplan, M. S., Bagge, C. L., Few, L. R., Maisto, S., Saitz, R., & Freeman, R. (2016). Alcohol Policies and Suicide: A Review of the Literature. *Alcoholism: Clinical and Experimental Research, 40*(10), 2043–2055. https://doi.org/10.1111/acer.13203

230. Kõlves, K., Chitty, K. M., Wardhani, R., Värnik, A., De Leo, D., & Witt, K. (2020). Impact of Alcohol Policies on Suicidal Behavior: A Systematic Literature Review. *International Journal of Environmental Research and Public Health, 17*(19), 7030. https://doi.org/10.3390/ijerph17197030

231. Jonas, D. E., Amick, H. R., Feltner, C., Bobashev, G., Thomas, K., Wines, R., Kim, M. M., Shanahan, E., Gass, C. E., Rowe, C. J., & Garbutt, J. C. (2014). *Pharmacotherapy for Adults With Alcohol-Use Disorders in Outpatient Settings.* Agency for Healthcare Research and Quality (US). http://www.ncbi.nlm.nih.gov/books/NBK208590/

232. de Faria Moraes, B., André Pedral Diniz Leite, G., André Pedral Diniz Leite, G., Silveira, I. B., Lana, N. V., & Cançado, G. G. L. (2025). Impact of glucagon-like peptide-1 receptor agonists on alcohol consumption and liver-related outcomes: A systematic review and meta-analysis. *Drug and Alcohol Dependence, 275*, 112840. https://doi.org/10.1016/j.drugalcdep.2025.112840

233. van der Meer, P. B., Fuentes, J. J., Kaptein, A. A., Schoones, J. W., de Waal, M. M., Goudriaan, A. E., Kramers, K., Schellekens, A., Somers, M., Bossong, M. G., & Batalla, A. (2023). Therapeutic effect of psilocybin in addiction: A systematic review. *Frontiers in Psychiatry, 14*, 1134454. https://doi.org/10.3389/fpsyt.2023.1134454

234. Sicignano, D., Hernandez, A. V., Schiff, B., Elmahy, N., & White, C. M. (2024). The impact of psychedelics on patients with alcohol use disorder: A systematic review with meta-analysis. Current Medical Research and Opinion, 40(2), 293–302. https://doi.org/10.1080/03007995.2023.2296968

235. Bogenschutz, M. P., Ross, S., Bhatt, S., Baron, T., Forcehimes, A. A., Laska, E., Mennenga, S. E., O'Donnell, K., Owens, L. T., Podrebarac, S., Rotrosen, J., Tonigan, J. S., & Worth, L. (2022). Percentage of Heavy Drinking Days Following Psilocybin-Assisted Psychotherapy vs Placebo in the Treatment of Adult Patients With Alcohol Use Disorder: A Randomized Clinical Trial. *JAMA Psychiatry, 79*(10), 953–962. https://doi.org/10.1001/jamapsychiatry.2022.2096

236. Rehm, J., & Roerecke, M. (2013). Reduction of drinking in problem drinkers and all-cause mortality. *Alcohol and Alcoholism (Oxford, Oxfordshire), 48*(4), 509–513. https://doi.org/10.1093/alcalc/agt021

237. Elder, R. W., Voas, R., Beirness, D., Shults, R. A., Sleet, D. A., Nichols, J. L., Compton, R., & Task Force on Community Preventive Services. (2011). Effectiveness of ignition interlocks for preventing alcohol-impaired driving and alcohol-related crashes: A Community Guide systematic review. *American Journal of Preventive Medicine, 40*(3), 362–376. https://doi.org/10.1016/j.amepre.2010.11.012

238. Zeithammer, R., Macinko, J., & Silver, D. (2025). Assessing the Deterrent Effects of Ignition Interlock Devices. *American Journal of Preventive Medicine, 68*(1), 137–144. https://doi.org/10.1016/j.amepre.2024.09.009

239. *OECD Public Health Explorer.* (n.d.). Retrieved 4 December 2025, from http://oecdpublichealthexplorer.org/

240. Ramboll Management Consulting AB. (2019). *Alkoholens samhällsekonomiska konsekvenser*. https://www.omsystembolaget.se/globalassets/pdf/alkoholproblematik/ramboll---resultatrapport----alkoholens-samhallskonsekvenser---uppdaterad-oktober-2020.pdf

Addresses

Actis – Rusfeltets samarbeidsorgan
Torggata 1
0181 Oslo
Norway
www.actis.no

Alkohol & Samfund
Høffdingsvej 36, stuen
2500 Valby
Denmark
www.alkohologsamfund.dk

Centrum för Utbildning och forskning kring riskbruk, missbruk och beroende (CERA)
Göteborgs universitet
Box 500
405 30 Göteborg
Sweden
www.cera.gu.se

FAS-föreningen
Borgmästargatan 6
261 32 Landskrona
Sweden
www.fasportalen.se

Hela Människan
Box 14038
167 14 Bromma
Sweden
www.helamanniskan.se

Hjärnfonden
Box 2364
103 18 Stockholm
Sweden
www.hjarnfonden.se

IOGT-NTO
Box 12825
112 97 Stockholm
Sweden
www.iogt.se

IOGT i Norge
Torggata 1
0181 Oslo
Norway
www.iogt.no

Junis
Box 12825
112 97 Stockholm
Sweden
www.junis.se

Kvinnoorganisationernas Samarbetsråd i Alkohol- och Narkotikafrågor
c/o Solidaritetshuset
Tegelviksgatan 40
116 41 Stockholm
Sweden
www.ksan.se

Länkens Kamratförbund
Videhusvägen 3
441 72 Sollebrunn
Sweden
www.lankenskamratforbund.com

MA – Rusfri Trafikk
Postboks 752 Sentrum
0106 Oslo
Norway
www.marusfritrafikk.no

MHF
Nygårdsgatan 3
543 51 Tibro
Sweden
www.mhf.se

Movendi International
Gammelgårdsvägen 38
112 64 Stockholm
Sweden
www.movendi.ngo

Nykterhetsrörelsens Scoutförbund
Box 128 25
112 97 Stockholm
Sweden
www.nsf.scout.se

Stiftelsen Ansvar För Framtiden
Box 128 25
112 97 Stockholm
Sweden
www.ansvarforframtiden.se

Svenska Frisksportförbundet Frisksportens Ungdomsförbund
Stensund
619 91 Trosa
Sweden
www.frisksport.se

Svensk förening för allmänmedicin
Box 738
101 35 Stockholm
Sweden
www.sfam.se

Svensk sjuksköterskeförening
Baldersgatan 1
114 27 Stockholm
Sweden
www.swenurse.se

Sveriges Blåbandsförbund
Bangårdsgatan 13 bv
753 20 Uppsala
Sweden
www.blabandet.se

Sveriges Blåbandsungdom
Box 12825
112 97 Stockholm
Sweden
www.sverigesblabandsungdom.se

Sveriges Frikyrkosamråd
Box 14038
167 14 Bromma
Sweden
www.skr.org/kristen-tro/kyrkorna/frikyrkor/frikyrkosamrad

Sveriges Landsråd för alkohol- och narkotikafrågor S.L.A.N
Kasernvägen 6
504 31 Borås
Sweden
www.slan.se

UNF
Box 12825
112 97 Stockholm
Sweden
www.unf.se



© Published by: CERA, Svensk förening för allmänmedicin, Svensk sjuksköterskeförening, Stiftelsen Ansvar för Framtiden, Actis-Rusfeltets samarbeidsorgan, Alkohol & Samfund, FAS-föreningen, Hela människan, Hjärnfonden, IOGT-NTO, IOGT i Norge, Junis, KSAN, Länkens Kamratförbund, MA – Rusfri Trafikk, MHF Motorförarnas Helnykterhetsförbund, Movendi International, Nykterhetsrörelsens Scoutförbund, Svenska Frisksportförbundet och Frisksportens Ungdomsförbund, Sveriges Blåbandsförbund och Sveriges Blåbandsungdom, Sveriges Frikyrkosamråd, Sveriges Landsråd för Alkohol- och Narkotikafrågor, UNF 2026.