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REVIEW

Alcohol use disorder: an Australian perspective on screening, diagnosis, treatment and prevention

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Abstract

Alcohol is one of Australia's most harmful recreational drugs, contributing more to death, disease and economic harm than all illicit drugs combined. Though it accounts for 4.1% of the national disease burden, it remains under-prioritised in health policy, prevention and treatment. Most of this harm is downstream of hazardous alcohol consumption and untreated alcohol use disorder (AUD), a common but stigmatised condition that is under-recognised and rarely treated with evidence-based therapies. Alcohol-related harm falls disproportionately on First Nations peoples, rural communities and socioeconomically disadvantaged Australians. Epidemiological trends are alarming: hospitalisations for alcohol-related liver disease are rising, and emerging data show a causal link between alcohol and early-onset colorectal cancer. These findings underscore alcohol's role as both a hepatotoxin and a multisystem carcinogen, with harms manifesting at younger ages and lower levels of consumption than previously recognised. Despite clear evidence that no level of alcohol use is safe, misconceptions persist, fuelled by industry influence and inconsistent public health messaging. Evidence-based treatments exist but are underused: only 2.9% of Australians with AUD receive approved pharmacotherapy, and delays to treatment average 18 years. Population-level strategies such as taxation, pricing and marketing restrictions remain the most effective levers, but progress is weak, as illustrated by the repeal of minimum unit pricing in the Northern Territory. Clinicians play a central role in reframing hazardous drinking and AUD as the root cause of alcohol-related disease and in advocating for evidence-based, patient-centred care and policy. Addressing AUD directly offers the clearest path to reducing Australia's alcohol burden.

Introduction

Alcohol is one of the most harmful drugs in Australia.[1] The damage caused by alcohol exceeds that of all other illicit drugs combined, causing more deaths, harm and economic loss (Table 1).[7] Only tobacco surpasses it as the primary cause of drug-related death, with 6500 premature Australian deaths attributed to alcohol each year.[7] Harmful drinking affects adults in the prime of their lives more than others, with alcohol use disorder (AUD) being a leading cause of premature morbidity and mortality.[8] On average, individuals with AUD lose >20 years of quality-adjusted life-years compared to the population average.[9] For individuals who do seek treatment, societal stigma results in an average delay of 18 years from the first onset of the alcohol-related problem.[10] Health and social impacts are compounded by chronic underfunding for research and services for this vulnerable population. This review reframes hazardous alcohol consumption and AUD as the primary driver of Australia's alcohol-related disease burden, a treatable condition too often overlooked by policy and practice, by critically appraising current approaches to screening, diagnosis, treatment and prevention, and consolidating a rapidly emerging body of evidence, including early-onset harms, new cancer data, updated epidemiology and shifts in treatment and policy, to

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provide a contemporary synthesis of alcohol-related harm and AUD management in Australia. Figure 1 summarises this conceptual framework, illustrating how population-level alcohol consumption leads to hazardous drinking and AUD, and how structural determinants and barriers to care shape exposure, recognition and outcomes.

Alcohol causes high rates of injury and harm in our communities

Eighty percent of Australians older than 13 years regularly drink alcohol, and one in four Australians engage in drinking behaviours that pose risk of morbidity and mortality.[2] Drink driving remains pervasive, with 30% of accidents and 20% of deaths on Australian roads involving alcohol.[2,11] Excessive alcohol consumption not only leads to major organ disease (liver disease, cardiovascular disease) but also increases the risk of developing solid organ cancers (including breast, mouth, throat, oesophagus, liver and colorectal cancer), cognitive impairment, peripheral neuropathy, sleep apnoea, depression and other mental health issues, as well as hindering care of other existing medical conditions.[12,13] Furthermore, one in five Australians reports harm because of someone else's alcohol intake.[11] Taken together, harms to individuals, others (e.g. families and loved ones, road users) and society represent a significant accumulated impact.

Table 1 The impact of alcohol and other drug use in Australia

Impact of alcohol and other drug use in Australia	
Alcohol	Other drug use
Past-year use (number of persons aged ≥14 years)	
14 million	3.4 million
Hazardous use and/or substance use disorder (number of persons aged ≥14 years)	
3.8 million	0.5 million
Number of deaths	
6512	1732
Economic cost per year	
$82.8 billion	$6.1 billion

Based on data from previous studies.[2–7]

Burden of alcohol: economic costs and health costs

The economic and health burden of alcohol use in Australia is substantial. Conservative estimates of alcohol-related harms place the per-person cost at $1203 annually.[14] In 2018–2019, our hospitals spent $2.1 billion treating AUD, injuries, cancers and cardiovascular diseases.[3] Broader social and economic consequences, including lost productivity and criminal justice costs, brought the total estimated burden to $66.8 billion in 2017–2018.[4] When the estimated $16 billion annual

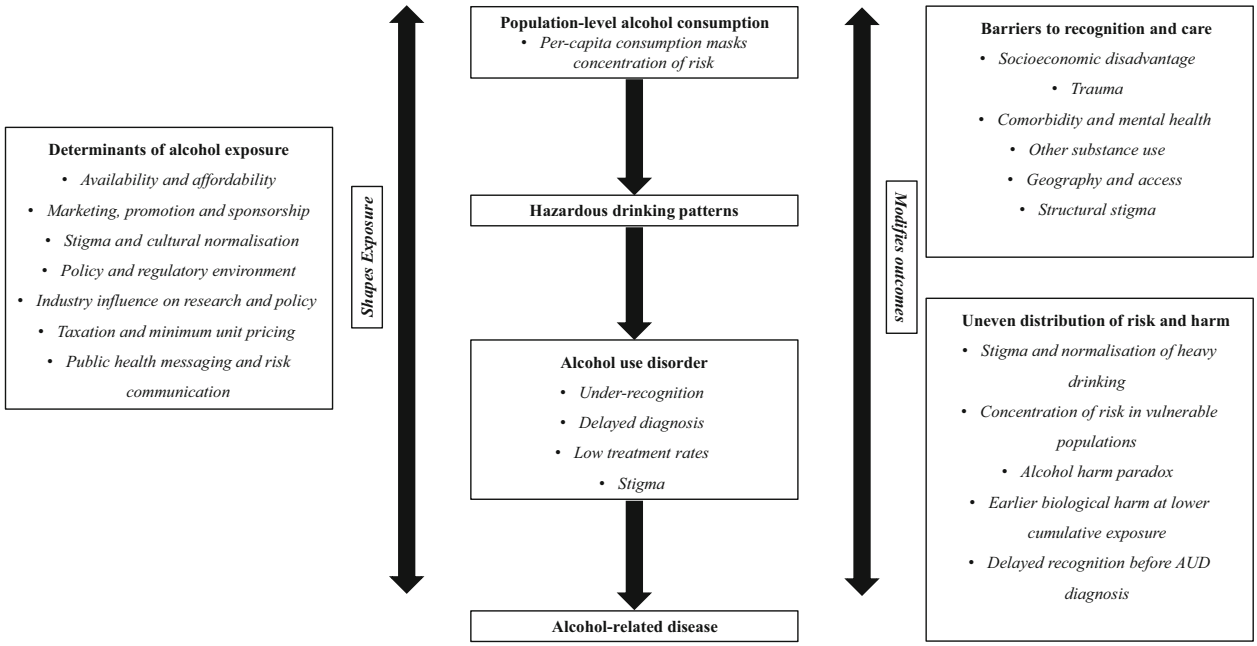

Figure 1 A conceptual model illustrating how population-level alcohol consumption leads to hazardous drinking and AUD, with outcomes shaped by structural determinants and barriers to care. AUD, alcohol use disorder.

impact of foetal alcohol spectrum disorder is included, the total cost reaches approximately $82.8 billion per year: a figure comparable to the entire JobKeeper wage subsidy program during the COVID-19 pandemic ($88.9 billion).[5,15] In contrast, alcohol excise taxes generated $6.9 billion in 2019, with only a fraction of this reinvested into alcohol-related research, prevention or treatment, underscoring a stark imbalance between the revenue raised and the societal costs.[7]

This burden is not evenly distributed. Alcohol-related harm is disproportionately experienced by Australia's First Nations peoples. While a higher proportion of First Nations Australians abstain from alcohol compared to non-Indigenous Australians, those who do drink are more likely to do so at hazardous levels.[5] First Nations peoples experience more than twice the total disease burden attributable to alcohol (8%–10%), and alcohol-attributable mortality is approximately four times higher than in non-Indigenous Australians.[15] Social disadvantage, structural inequities and intergenerational trauma all contribute to the harm experienced by Australia's First Nations peoples, highlighting the need for culturally safe, community-led and healing-informed responses.[6]

Recent work demonstrates the effectiveness of such approaches. In a cluster randomised trial conducted in Aboriginal Community Controlled Health Services, a co-designed 24-month support model led to a substantial increase in recorded verbal alcohol interventions, showing that tailored, community-led strategies can strengthen alcohol screening and early intervention in primary care settings. Although gains in relapse-prevention pharmacotherapy were modest, the study provides real-world evidence that community-controlled services can enhance alcohol care when supported with appropriate resources and culturally informed models.[16]

Geographic variation also shapes patterns of risk and access to care. People in rural and remote communities are significantly more likely to consume alcohol at hazardous levels.[17] Moreover, these communities often have significant barriers to care, with over a quarter of rural patients needing to travel more than an hour to access treatment.[18] Emerging digital and tele-health-delivered behavioural interventions offer additional pathways for engagement, particularly in rural areas, and structured relapse-prevention follow-up within general practice remains essential for sustaining abstinence.[19]

While historically and culturally, hazardous alcohol consumption and AUD have been more common in men, the gender gap is narrowing.[20] Women often face unique barriers to seeking care and are more likely to have experienced significant trauma, including sexual abuse and domestic violence.[21] Similarly, youth-onset AUD and alcohol use among older adults present distinct clinical and social challenges.[7] Among Australian men aged 15 to 49 years, alcohol has become the leading risk factor for disease and injury. This underscores the need for age-appropriate, tailored interventions across the lifespan.[22,23]

Epidemiological trends: emerging harms in younger populations and misconception of risk

While official *per capita* alcohol consumption in Australia may suggest a decline, this metric fails to capture the uneven social distribution of alcohol intake and the similarly discrepant ancillary harms across different populations.[24] This pattern reflects the well-described 'alcohol-harm paradox', wherein socioeconomically disadvantaged groups experience greater alcohol-related morbidity and mortality despite consuming the same or less alcohol than more advantaged groups.[25] The paradox arises from an accumulation of structural and contextual vulnerabilities including higher rates of comorbidity, unstable housing, food insecurity, trauma exposure, limited access to preventive healthcare, differential patterns of drinking such as heavier episodic use and reduced social and economic resources.[26] These factors amplify the biological and social consequences of alcohol consumption, meaning that equivalent drinking levels generate disproportionately greater harm in lower socioeconomic groups.

Against this backdrop, reductions in spending and consumption have occurred primarily among lower-risk drinkers, while high-risk consumption has remained stable. Notably, 3.8 million Australians, around 16% of drinkers, account for 75% of alcohol sales. This subgroup of hazardous drinkers disproportionately bears the burden of alcohol-related physical, mental and social harm, while simultaneously driving industry profits.[27]

Recent trends indicate that national *per capita* consumption is now increasing following the COVID-19 pandemic.[24] This has coincided with a rising burden of alcohol-related harm, particularly in the healthcare system. Hospitalisations for alcohol-related liver disease (ArLD) have steadily increased over the past decade.[28] Internationally, an alarming trend has also emerged: rising rates of ArLD, alcohol-related hepatitis and pancreatitis in individuals younger than 30 years.[29–31] These individuals are first coming to medical attention with advanced disease, despite having had little prior engagement with healthcare: a reflection of both the silent progression of AUD and the apparent missed opportunities for earlier intervention.

Importantly, alcohol has also been implicated in the sharp rise of early-onset colorectal cancer (EOCRC) in adults younger than 50 years. A 2025 systematic review and meta-analysis confirmed a dose–response relationship between alcohol intake and EOCRC, while a 2024 Mendelian randomisation study reported genetic evidence supporting a causal relationship between alcohol and EOCRC.[32,33] Taken together, these data underscore that alcohol acts as both a hepatotoxin and a multisystem carcinogen, with harms now manifesting at younger ages and at levels of consumption once thought to be low risk.

The increasing incidence of alcohol-related harms at both ends of the age spectrum illustrates the limitations of using *per capita* alcohol consumption as a public health indicator. While average consumption may appear stable, the most vulnerable populations are experiencing worsening health outcomes. This paradox highlights the need for more refined, stratified data to enhance our understanding of at-risk populations and inform more effective and equitable public health responses.

No safe threshold: current perspectives on alcohol harm

While the harm of high-risk, high-volume alcohol intake is increasingly apparent, the community narrative around low-moderate alcohol intake persistently enables the understating of harm. The International Agency for Research on Cancer classifies alcohol as a Group 1 carcinogen because of its toxic properties.[34] Historical claims that low-to-moderate alcohol consumption conferred cardiovascular benefits, popularised through the concept of a 'J-shaped curve', have been increasingly discredited.[8,35–40] Emerging evidence suggests that there is no reduction in health risk at any level of consumption. Apparent protective effects from earlier studies are now understood to result from methodological flaws, including biased abstainer comparison groups and confounded by the alcohol-harm paradox.

Re-evaluation of the cardiovascular literature demonstrates that once these limitations are addressed, any protective signal attenuates substantially, with more recent analyses showing no meaningful reduction in all-cause or cardiovascular mortality at low levels of consumption.[37,40] Even studies that continue to report benefit emphasise that such effects are restricted to individuals with consistent low-level intake without any pattern of heavy episodic drinking: a pattern rarely observed in real-world drinking behaviour.[41] When episodic or chronic heavy drinking is present, even intermittently, any cardioprotective effect disappears entirely.

In contrast to the diminishing cardiovascular narrative, the evidence supporting a carcinogenic effect of alcohol, even at low levels of consumption, is consistent, biologically plausible and well demonstrated. Recent population-based analyses across multiple regions show that cancers attributable to alcohol arise in individuals consuming amounts historically considered low risk.[42–44] These findings align with mechanistic evidence demonstrating that acetaldehyde-mediated DNA damage, oxidative stress and hormonal perturbation occur at low exposures, with no identifiable threshold below which cancer risk disappears.[40]

Taken together, this body of evidence demonstrates that serious health consequences among so-called moderate drinkers rarely arise from innocuous use. Rather, they reflect cumulative carcinogenic and cardiometabolic harm that persists unchallenged, often enabled by inconsistent public health messaging and longstanding misconceptions about risk. Evolving data therefore support the conclusion that there is no clear safe level of alcohol consumption. The term 'safe' implies negligible or absent risk; for alcohol, as for tobacco, measurable harm occurs even at low levels of exposure. Public health guidance should therefore avoid reassurance-based terminology and instead clearly communicate that all levels of alcohol intake carry some degree of risk.

Efforts to clarify the risk have been confounded by the alcohol industry's involvement in scientific research. The MACH (Moderate Alcohol and Cardiovascular Health) trial was terminated after revelations that alcohol industry funding, including global producers with significant Australian operations such as Diageo (Bundaberg Rum, Johnnie Walker), had influenced its design and objectives.[45,46] This episode remains largely unknown to many Australian clinicians despite its implications for trust in alcohol research. In Australia, concerns have been raised about the influence of industry submissions on Australian alcohol and health policy.[47]

Considering robust evidence links even low levels of alcohol to increased risks of cancer, cardiovascular disease and cognitive decline, health professionals need to reframe alcohol use to patients as a modifiable risk factor. Australian guidelines define low-risk alcohol use as ≤10 standard drinks per week and ≤4 per occasion.[48,49] The World Health Organisation (WHO) and *Lancet Public Health* now recognise that there is no 'safe' level of alcohol consumption.[40,50] In 2023, Canada released updated guidelines recommending a limit of just two standard drinks per week, with even one to two drinks acknowledged as carrying measurable risk.[51] Their approach prioritised transparency and reflected the evidence on alcohol related harm, offering graded risk levels to support informed decision-making by consumers.

Australia's use of the 10-drink threshold and continued use of the term 'low risk' reflect a missed opportunity to more proactively restate harm. Public submissions from various sectors, including political, community and the alcohol industry, many with strong ideological and economic interests, contributed to the 2020 NHMRC alcohol consumption guideline process.[47,52] Further scientific health evidence has accumulated in the 5 years since this publication. The next iteration of these guidelines would benefit from these new data, international health advice and limiting non-empirical influences.

Public policy needs to communicate clearly that even low-moderate consumption of alcohol can be associated with health risk. Current Australian guidelines have created a situation where public health messaging is misaligned with the evidence, and in contrast to the Canadian guidelines, consumers are unable to make informed choices. Replacing the language of relative safety with clarity about absolute risk is essential to ensure that individuals can make genuinely informed choices about their alcohol use. Consistent messaging from health professionals is vital to counteract decades of conflicting public health narratives and reduce community ambivalence.

Alcohol use disorder

AUD involves frequent or heavy alcohol drinking that becomes difficult to control and leads to problems in relationships, work, school, family or other areas. AUD is diagnosed when ≥2 of 11 symptoms occur within a 12-month period. The full criteria are summarised in Table 2.

Risk factors for AUD

A variety of factors affect the levels and patterns of alcohol consumption at individual and societal levels. Societal factors include level of economic development, culture, social norms, availability of alcohol and implementation and enforcement of alcohol policies.[54] The adverse health impacts and social harm from the level and pattern of drinking are concentrated in lower socioeconomic groups.[26,55]

Individual factors include age, male gender and family history of AUD. Although there is no single dominant risk factor, these risks are additive and synergistic, with the more vulnerabilities a person has, the greater the risk of developing AUD.[56] Twin studies have shown heritability to account for approximately 50% of the risk for the development of AUD.[57]

Stigma is a major barrier to AUD diagnosis and care

The societal stigma surrounding AUD provides a barrier for Australians with this condition to seek treatment, which delays or even prevents treatment-seeking behaviour.[10] Stigma is often reinforced through language, and clinicians should be aware that even well-intentioned language can reinforce stigma.[58] Table 3 outlines recommended language for reducing stigma in AUD. Community-based stigma, self-stigma and structural stigma all contribute to failure to seek help or delays in seeking help, inferior healthcare and negative health outcomes.[60] These factors contribute to the low treatment rates for AUD in Australia. In 2022–2023, 50 106 patients sought help for their AUD and received 92 417 episodes of care: fewer than two episodes per patient.[61] With 3.8 million Australians consuming alcohol hazardously, this indicates that as little as 1.3% of patients are being provided with the help and support they need. Stigma-reducing interventions are needed to address this issue.[62]

Screening for AUD

A comprehensive history is the key for diagnosing AUD, but it can be inaccurate if the patient has reason to not disclose the severity of their alcohol problems – for example, perceived personal or family embarrassment, occupational risk and forensic assessments. However, structured questionnaires can facilitate the process by providing additional data to inform an AUD diagnosis. The 10-item Alcohol Use Disorders Identification Test (AUDIT) was created as a collaborative effort by the WHO[63] to screen for alcohol dependence. AUDIT-Consumption (AUDIT-C), an abbreviated version that incorporates the first three items, is a well-validated brief alcohol screening instrument.[64] Table 4 shows the questions, scoring and interpretation for males and females. Rates of screening for AUD in Australia have historically been low.[65] Although there is a paucity of contemporary data, low rates of treatment suggest that lack of recognition of AUD remains a problem, one that screening tools can help to address, and stands in contrast to the routine screening of cancer or cardiovascular risk. Given the ubiquity of heavy alcohol consumption in Australian society and its related harms, screening for AUD should be a routine part of any medical encounter.

Routine biomarker monitoring can supplement patient self-reports. Phosphatidylethanol (PEth) testing is an extremely sensitive and specific blood biomarker produced only in the presence of alcohol, although not yet widely available outside of public hospital laboratories.

Table 2 DSM- diagnostic criteria for AUD and interpretation

1	Alcohol is taken in larger amounts or over a longer period than was intended
2	There is a persistent desire or unsuccessful efforts to cut down or control alcohol use
3	A great deal of time is spent in activities necessary to get alcohol, use alcohol or recover from its effects
4	Craving, or a strong desire or urge to use alcohol
5	Recurrent alcohol use resulting in a failure to fulfil role obligations at work, school or home
6	Continued alcohol use despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of alcohol
7	Important social, occupational or recreational activities are given up or reduced because of alcohol use
8	Recurrent alcohol use in situations in which it is physically hazardous
9	Alcohol use is continued despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by alcohol
10	Tolerance
11	Alcohol withdrawal.

The severity classification for AUD ranges from mild (with 2 or 3 criteria), moderate (with 4 or 5 criteria) and severe (with ≥6 criteria). Adapted from the American Psychiatric Association.[53] AUD, alcohol use disorder; DSM-5, Diagnostic and Statistical Manual of Mental Disorders, 5th Edition.

PEth level reflects consumption over the past 4 to 6 weeks and can help facilitate discussions where there are concerns for inaccurate self-reporting, helping identify AUD before the development of alcohol-related harms manifests and facilitate prevention strategies.[66]

MET-ALD framework

The recent shift of terminology in liver disease to the metabolic dysfunction–alcohol-related liver disease (Met-ALD) spectrum recognises the importance of alcohol as an (often unrecognised) cofactor and underscores the need to assess alcohol exposure with much greater precision.[67] Given that Met-ALD diagnostic thresholds depend on accurately estimating cumulative alcohol intake, reliable assessment of lifetime alcohol exposure, rather than short-term recall alone, is increasingly essential for distinguishing metabolic from alcohol-predominant disease. Structured lifetime alcohol exposure tools demonstrate moderate to strong reliability and validity and provide a more accurate understanding of cumulative exposure and its contribution to Met-ALD presentations.[68] For patients across the Met-ALD spectrum, early identification and management of hazardous or unhealthy drinking, including AUD, remain central to disease modification and should be integrated with metabolic risk management across hepatology and primary care settings.

Table 3 Recommended language for reducing stigma in AUD

Stigmatising or ambiguous term	Recommended language/framing	Rational
Alcoholic or alcohol abuse	Person with AUD Person who uses alcohol	Person-first; avoids defining identity by condition Diagnostic and respectful; avoids moral judgement
Alcoholic liver disease	AUD complicated by chronic liver disease Alcohol-related liver disease	Accurate, non-labelling clinical terminology
Binge drinker	Person who engages in episodic heavy drinking	Focuses on behaviour, not identity
Relapse	Return to use/ recurrence/lapse	Neutral, non-moralising; aligns with chronic disease framework
Slip (*when acknowledged by the patient*)	Recognised lapse with renewed commitment to goals	Reflects individual agency and avoids framing as failure
Failure	Limited treatment response/continuing use	Shifts focus from individual blame to systemic or clinical factors
Non-compliant	Facing barriers to care	Encourages understanding of structural and psychosocial barriers
Recovery (if used rigidly)	Individual's goals or journey toward improved health and functioning	Encourages person-centred, non-binary understanding of recovery

Adapted from the National Institute on Drug Abuse, National Institute of Health.[59] AUD, alcohol use disorder.

Treatment of AUD

Patients with AUD require application of evidence-based treatment combined with a non-judgemental, empathic and compassionate approach. Shared decision-making is essential for engaging these people in treatment.[48,56] Treatment of AUD includes managing acute withdrawal, initiating psychosocial intervention and providing ongoing support.

Table 4 The AUDIT-C questions, scoring and interpretation

Points	0	1	2	3	4
How often do you have a drink containing alcohol?	Never	Monthly or less	2–4 times a month	2–3 times a week	4+ times a week
How many standard drinks containing alcohol do you have on a typical day?	1–2	3–4	5–6	7–9	10 or more
How often do you have six or more standard drinks on a single occasion?	Never	Less than monthly	Monthly	Weekly	Daily or almost daily

In men, a score of ≥4 is considered positive, optimal for identifying hazardous drinking or active AUD. In women, a score of ≥3 is considered positive, optimal for identifying hazardous drinking or active AUD. Adapted from Bush *et al.*[64] AUD, alcohol use disorder; AUDIT-C, Alcohol Use Disorders Identification Test-Consumption.

Alcohol withdrawal

Alcohol effects multiple neurotransmitters including dopamine, serotonin, glutamate and γ-aminobutyric acid (GABA). GABA-ergic pathways are key to alcohol's sedative effects. While alcohol increases GABA initially, long-term use causes receptor adaptation and heightened excitatory activity, resulting in withdrawal when alcohol is abruptly ceased. Administration of benzodiazepines helps stabilise GABAergic function during alcohol withdrawal and avoid issues including seizures. Despite the potentially life-threatening nature of alcohol withdrawal, most cases are treatable outside of hospitals given sufficient support. Table 5 provides an example schedule for alcohol as an option to help manage withdrawal. Ambulatory alcohol withdrawal is not recommended for individuals with a history of severe withdrawal, seizures, unstable health conditions (such as liver disease), active mental illnesses, polysubstance use, lack of social support, those experiencing severe withdrawal, poor adherence to treatment, pregnancy or unsafe housing. In these cases, inpatient and/or specialist care is recommended to ensure safety and prevent complications. All patients should be commenced on 300 mg oral thiamine daily to prevent the development of Wernicke encephalopathy.[48]

Brief intervention

Most patients who screen positive for high-risk drinking will not receive a diagnosis of AUD. However, hazardous drinkers should be offered a brief intervention as this may reduce alcohol use in these individuals and those with mild AUD.[69] The core components of a brief intervention comprise engaging empathically with the patient, investigating their motivations for decreasing alcohol consumption, psychoeducation around the effects of drinking, goal setting, encouraging patient autonomy, increasing self-efficacy and self-confidence and providing some simple behavioural strategies to reduce or abstain from alcohol use. The initial brief intervention may result in immediate change or establish a rapport/groundwork for more meaningful interventions to occur later. Trust and perseverance are key, as it may take multiple encounters for the patient to become self-motivated and committed to change. Despite their proliferation in hospital settings, the evidence for brief intervention is strongest in the primary care setting.[69] For people with moderate to severe AUD, a brief intervention is likely to be insufficient, and more specialist and intensive treatment and therapy are typically indicated.

Psychosocial intervention

Psychosocial interventions are the mainstay of treatment for moderate–severe AUD.[70] The *Guidelines for the Treatment of Alcohol Problems* recommends cognitive behaviour therapy (CBT) as a first-line treatment for AUD, although the choice of psychosocial treatment should be guided by the principles of patient-centred care.[71,72] A well-structured CBT program with a mental health clinician typically includes motivational interviewing to strengthen commitment to change; psychoeducation about substance use disorders, cravings and what to expect when becoming abstinent; craving management strategies and identifying triggers; engaging in alternative behaviours; changing thinking patterns; challenging positive expectations of substance use; improving self-efficacy to refuse or reduce use; adjusting unhelpful views of self/others/world; improving coping skills to manage difficult emotions; managing impulsivity; and relapse prevention planning. Consideration of assessment and treatment of concurrent co-existing mental health disorders is also recommended, e.g., anxiety, depression and trauma, and linking with other supports and services.[72] Patients are expected to see increased rates of abstinence and better

Table 5 Example ambulatory diazepam schedule for outpatient detoxification

Days since last alcohol-containing drink	Suggested diazepam dose†
1–2	5–10 mg three to four times a day
3	5–10 mg twice a day
4–5	5–10 mg at night

Adapted from Haber *et al*.[48] †Dose should be adjusted and tailored to the individual patient.

social functioning with effective psychosocial intervention.[48,56,70]

Pharmacological treatment

Concurrent adjunctive pharmacotherapy shows additional, though modest benefit. Acamprosate and naltrexone are available for AUD treatment in Australia and subsidised under Australia's universal health system (Pharmaceutical Benefits Scheme (PBS)).

Acamprosate targets the glutamatergic NMDA receptor system, which is affected by prolonged alcohol consumption. Administering acamprosate to patients with AUD may reduce alcohol cravings and increase rates of abstinence. Naltrexone is a potent μ-opioid receptor blocker that diminishes the pleasurable effects of alcohol by decreasing dopamine levels in the nucleus accumbens. In AUD, naltrexone is thought to aid in maintaining abstinence by lessening the intensity and duration of alcohol relapse. Both acamprosate and naltrexone have a number needed to treat of approximately 12 and are usually taken for 3 to 6 months.[73–75] Topiramate and baclofen have evidence for data to support treatment of AUD but are not subsidised by the PBS for this indication and their use remains off-label and should only be prescribed by addiction specialists.[76,77] Emerging evidence suggests a future role for GLP-1 agonists.[78] Despite the proven efficacy of pharmacotherapy for AUD, only 2.9% patients with an AUD are receiving PBS listed pharmacological treatment and only 5.1% complete a minimum of 3 months of treatment.[79] In practice, combining psychosocial therapy with pharmacotherapy offers the most effective approach, with structured behavioural treatment forming the foundation and medication providing supportive benefit.

Peer support

In Australia, the main two forms of peer support are 12-step programs such as Alcoholics Anonymous (AA) and SMART Recovery (Self-Management and Recovery Training). Both promote dialogue about experiences, offer guidance and present a free, widely available form of care that may be beneficial alongside professional treatment.[80–82]

Population-focused interventions

The WHO 'Best Buys' lists the most cost-effective way to tackle the harms associated with excessive alcohol consumption through population-based interventions.[83,84] Factors such as acceptability, availability and affordability influence alcohol consumption. From the acceptability perspective, public awareness of alcohol-related harms should be promoted through clear labelling and industry-independent health campaigns. Most alcohol industry advertising seeks to increase market by increasing acceptability and attractiveness of alcohol. Regarding availability, policy reforms may include restrictions on pervasiveness of alcohol marketing, advertising, sponsorship and promotion, as well as improved regulation of licensing, including outlet density, location and online sales and delivery services. From the affordability perspective, increasing prices, excise taxes and moderating other fiscal measures, such as reducing and ending financial incentives and subsidies, have all demonstrably reduced alcohol consumption at the population level.[85] The positive experience of Scotland with minimum unit pricing (MUP) demonstrates the benefit of such policies in a society that shares many of Australia's cultural attachments to problematic alcohol use. In Scotland, MUP legislation led to notable decreases in deaths and hospitalisations solely linked to alcohol consumption.[86] The most significant progress occurred in the bottom four socioeconomic groups, demonstrating that the policy effectively addresses inequalities related to alcohol-related disease. The Northern Territory had implemented a form of MUP with positive results.[87–90] Concerns about political feasibility, perceived economic impacts on small retailers and competing local business interests were prominent in public debate. These arguments sat alongside organised alcohol industry lobbying, with commercial stakeholders exerting considerable influence over pricing and regulatory settings. The episode illustrates how reforms can be challenging to sustain in shifting political contexts and highlights the importance of professional advocacy in supporting lasting, health-focused policies.

Conclusion

AUD remains under-recognised, under-diagnosed, under-resourced and under-treated. For many Australians, there is more stigma associated with not drinking than with drinking to excess. Harmful alcohol use can diminish motivation, self-esteem and confidence, undermining an individual's health-seeking behaviours and delaying care.

This results in alcohol-related disease, the predictable outcome of persistent gaps in identifying and treating AUD. Shifting upstream to address the root cause, i.e., hazardous alcohol consumption and AUD, offers the clearest path to reducing harm. The current treatment gap for AUD reflects a broader failure to align clinical care, policy and public health responses with the scale of harm. Meaningful progress will require sustained leadership, consistent clinical practice and coordinated investment across services, systems and sectors. Progress requires increasing the clinical recognition of AUD as a preventable and treatable disease. Reducing alcohol-related harm starts with tackling the problem that causes it.

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