Alcohol: Training Pack

A reference resource for Drug & Alcohol Service Providers, Health Professionals, Frontline Workers, and Allied Professionals. Developed by Tony D'Agostino, TD Consultancy — Updated Edition 2025–2026.

TD CONSULTANCY

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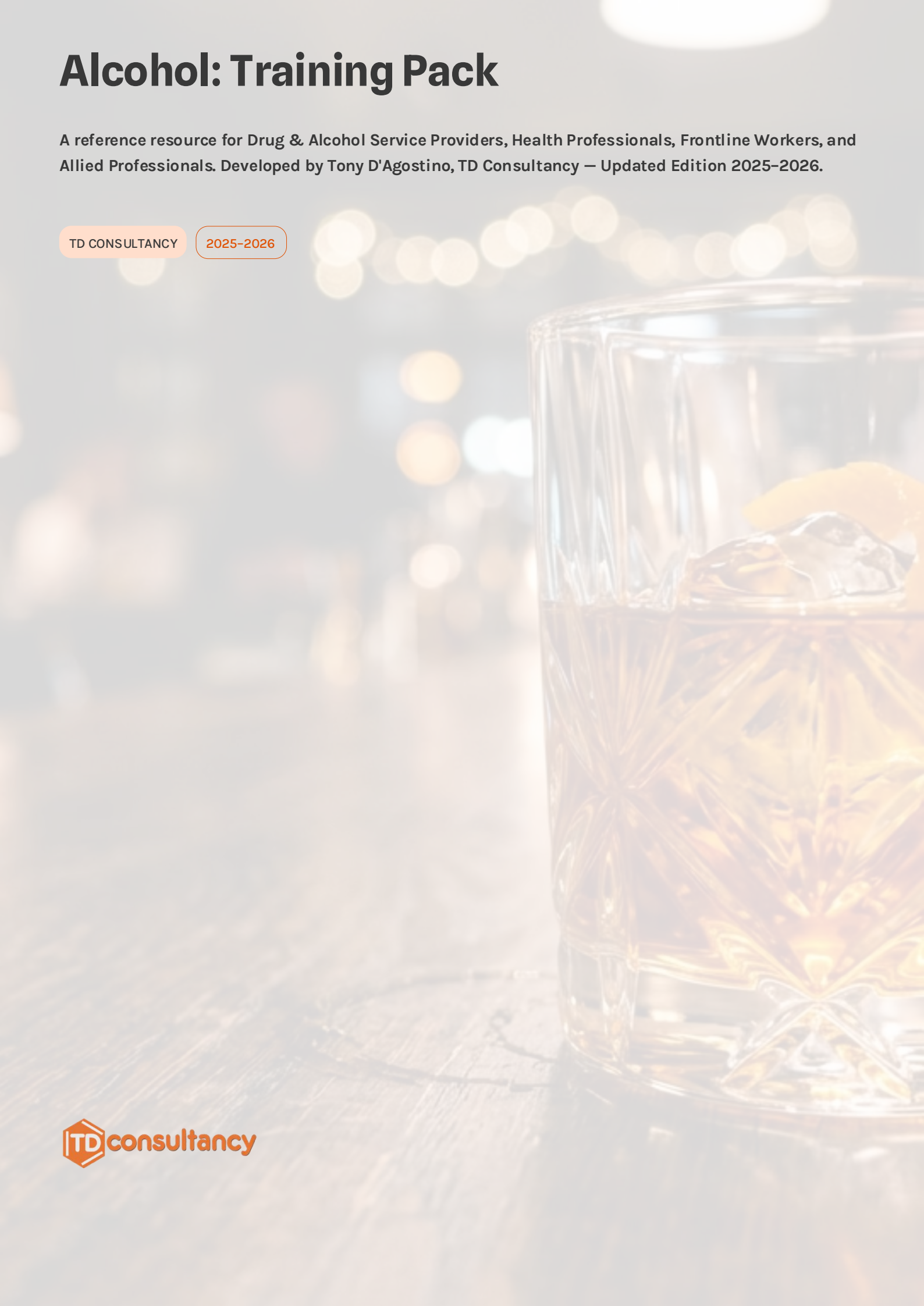

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How to Use This Pack

This pack is intended for use **in conjunction with training — not in isolation**. It is a reference resource to be used before, during, and after training sessions. Not all material will be relevant to all services. The type of service, the stage of the client, and the regional context of use will all influence which sections are most relevant.

Before Training

Familiarise yourself with key sections relevant to your service tier and client group.

During Training

Use as a reference guide alongside facilitated sessions and group exercises.

After Training

Return to specific sections for clinical reference, screening tools, and appendix resources.

CHAPTER 1

History of Alcohol: The Ancient World

Alcohol is one of the oldest psychoactive substances known to humanity. Its history is inseparable from the history of human civilisation — woven into agriculture, religion, medicine, trade, and social life. Understanding this history helps contextualise why alcohol occupies such a unique and contested position in contemporary public health.

1

7000 BC — Earliest Production

Archaeological evidence from the Jiahu site, Henan Province, China, reveals fermented beverages made from rice, honey, and fruit — the earliest confirmed alcohol production in human history (McGovern et al., 2004).

2

5000 BC — Beer in Mesopotamia

Beer brewing is documented in ancient Sumeria (modern Iraq). Barley-based fermented drinks are consumed widely, associated with ritual, trade, and daily sustenance. Ancient Sumerian texts include hymns to Ninkasi, the goddess of beer, with embedded brewing recipes.

3

3500 BC — Wine in the Near East

Wine production is documented in the Zagros Mountains region (modern Iran/Georgia). Grape cultivation expands across the Mediterranean, establishing wine as a cornerstone of Greek and Roman culture.

4

3000 BC — Egypt

Beer and wine are integral to daily life, religious practice, and medicine in ancient Egypt. Workers constructing the pyramids receive daily beer rations of approximately 4–5 litres — understood as a caloric supplement and safer alternative to contaminated water.

5

400 BC — Hippocrates

The Greek physician Hippocrates documents both the medicinal uses and harmful effects of wine — one of the earliest recorded recognitions of alcohol-related harm in the medical literature.

6

55 BC — Romans in Britain

Roman legions introduce viticulture to Britain, establishing the foundations of British wine and drinking culture.

History of Alcohol: Medieval to Early Modern Period

700s–1400s — Islamic Distillation & Monastic Brewing

Islamic scholars — particularly Jabir ibn Hayyan (Geber) — significantly advance distillation technology, refining the alembic still. This development eventually enables the production of spirits far stronger than anything achievable through fermentation alone. Simultaneously, monasteries across Europe become centres of brewing and winemaking. In medieval England, ale is the staple daily drink — water sources were frequently unsafe, and low-alcohol beer provided safer hydration. The pub (or alehouse) becomes the primary social institution.

1400s–1516 — Whisky & Early Regulation

Spirits ("aqua vitae" — water of life) are distilled in Scotland and Ireland, initiating a tradition that will define Scottish and Irish cultural identity. In 1516, the Reinheitsgebot — one of the world's first alcohol regulations — is introduced in Bavaria, requiring beer to be made only from water, barley, and hops. It represents an early attempt to regulate alcohol quality.

1600s–1750s — The Gin Craze

Gin is introduced to England from the Netherlands following the Glorious Revolution. Initially valued for its medicinal properties, it becomes widely available and affordable in the early 18th century. The "Gin Craze" produces one of the UK's first modern public health crises — widespread cheap gin consumption leads to social disorder, increased mortality, and neglect of children. William Hogarth's contrasting engravings *Beer Street* and *Gin Lane* (1751) capture the moral panic. The Gin Acts of 1736 and 1751 attempt regulation, with mixed success.

> "Benjamin Rush (1784) was among the first to describe habitual drunkenness as a **disease rather than a moral failing** — a conceptual foundation for the modern disease model of alcohol use disorder."

History of Alcohol: 19th & 20th Century

19th Century Milestones

1 1830s–1840s — Temperance Movement

The Temperance Movement grows rapidly in the UK and USA, advocating for abstinence. The Band of Hope, founded in Leeds in 1847, becomes a major force for abstinence education, particularly among working-class communities.

2 1838 — "Alcoholism" Coined

Swedish physician Magnus Huss coins the term "alcoholism" to describe a chronic disease pattern. Note: this terminology is now clinically discouraged — preferred terms are "alcohol use disorder" (AUD) and "alcohol dependence."

3 1872 & 1898 — Legislation & Treatment

The Licensing Act 1872 introduces the first significant UK regulation of pub opening hours. By 1898, the first specialist "inebriates" treatment institutions open in the UK, marking the beginnings of a formal treatment response to dependent drinking.

20th Century Milestones

1 1914–1918 — WWI Licensing Restrictions

Prime Minister David Lloyd George introduces sweeping licensing restrictions to improve wartime industrial productivity. The 11pm closing time becomes standard and remains in force for most of the 20th century.

2 1920–1933 — US Prohibition

The United States prohibits the production, sale, and transportation of alcohol. While associated with organised crime and speakeasies, public health data suggest alcohol-related illness fell significantly — evidence cited in debates about minimum unit pricing.

3 1935 — Alcoholics Anonymous Founded

AA is founded in Akron, Ohio, by Bill Wilson ("Bill W.") and Dr. Bob Smith ("Dr. Bob"). The 12-Step model of recovery is born. AA spreads globally, establishing in the UK in 1952, and remains one of the most widely accessed recovery resources in the UK.

1960s–70s

Growing medical recognition of alcohol dependence. First specialist NHS detoxification units open. Dedicated NHS funding for alcohol treatment begins in 1976.

1979

Royal College of Psychiatrists publishes *Alcohol and Alcoholism* — a landmark report establishing safe drinking guidelines and calling for a comprehensive public health approach.

1980s–90s

Evidence base for Brief Interventions (BI) developed. The AUDIT screening tool is developed by WHO in 1989. Harm reduction approaches begin to be applied to alcohol.

CHAPTER 2

Types of Alcohol & How It Is Produced

What Is Alcohol?

The term "alcohol" in everyday use refers specifically to **ethanol** (ethyl alcohol; CH_3CH_2OH) — the psychoactive compound present in all alcoholic beverages. It is important to distinguish ethanol from other toxic alcohols:

- **Methanol** (wood alcohol, CH_3OH) — toxic; causes blindness and death even in small quantities; produced in illicit or poorly distilled spirits
- **Isopropyl alcohol** (rubbing alcohol) — used as a disinfectant; toxic if ingested
- **Ethanol** (drinking alcohol) — psychoactive; metabolised by the liver; safely metabolised in moderate amounts by healthy individuals

Only ethanol is present in regulated alcoholic beverages. Practitioners working with people who drink non-beverage alcohol (methylated spirits, hand sanitiser, screen wash) — typically in the context of severe dependence or homelessness — should be aware of the additional toxicity risks from methanol and additives.

What Is a Unit?

In the UK, **one unit of alcohol equals 10ml (8g) of pure ethanol.** This standardised measure enables consistent communication about consumption levels regardless of the type of drink.

Calculating units:

Units = ABV% × Volume (ml) ÷ 1000

Example: A pint (568ml) of 5% lager = 5 × 568 ÷ 1000 = **2.84 units.**

Most people significantly underestimate their unit consumption. A standard 175ml glass of 13% wine contains **2.3 units** — not "one glass = one unit" as is commonly assumed.

How Alcohol Is Produced

- **Fermentation:** Sugars fermented by yeast produce ethanol and CO_2. Produces beverages up to ~15% ABV. Includes beer, wine, and cider.
- **Distillation:** Fermented liquid is heated; ethanol evaporates and is collected, concentrating alcohol to 40% ABV or higher. Includes whisky, gin, vodka, rum, and brandy.
- **Fortification:** Spirits added to wine halt fermentation, raising ABV to 17–22%. Includes port, sherry, and vermouth.

UK Alcoholic Beverages: Units at a Glance

The table below provides a reference guide to ABV ranges, typical serving sizes, and unit content across common UK alcoholic beverages. Most consumers significantly underestimate their consumption — particularly with craft beers, large wine pours, and stronger lagers.

Type	ABV Range	Typical Serving	Units per Serving	Notes
Regular beer/lager (pint)	4–5%	568ml	2.3–2.8	Commonly underestimated
Strong beer/lager (pint)	7–9%	568ml	4.0–5.1	"Super strength" cans up to 9%
Craft beer (pint)	5–10%	568ml	2.8–5.7	ABV often higher than traditional ales
Wine (175ml glass)	12–14%	175ml	2.1–2.5	Standard restaurant pour
Wine (large glass 250ml)	13%	250ml	3.25	Three large glasses = a bottle
Spirits (25ml measure)	37.5–40%	25ml	0.94–1.0	35ml in Scotland (1.3 units)
Prosecco/Champagne (125ml)	12%	125ml	1.5	Frequently underestimated
Alcopops/RTDs (275ml)	4–5%	275ml	1.1–1.4	Popular with younger drinkers
Cider (pint)	4–8.5%	568ml	2.3–4.8	Significant ABV variation
Fortified wine (50ml)	20%	50ml	1.0	Port, sherry

Congeners

Non-ethanol compounds produced during fermentation and distillation — including fusel alcohols, aldehydes, esters, and tannins. They contribute to flavour, aroma, and colour, and are implicated in hangover severity. Dark spirits (whisky, brandy, dark rum) and red wine contain significantly higher congener levels than clear spirits (vodka, gin).

The Craft Alcohol Market

The craft beer and artisan spirits market has expanded significantly in the UK since 2010. Many craft beers now have ABVs of 6–10% — substantially stronger than traditional ales (3.5–4%). Packaging often emphasises artisanal quality rather than alcohol content. Practitioners should be alert to this pattern when taking drinking histories.

No/Low Alcohol Options

The no/low alcohol market has grown substantially between 2020 and 2025. Alcohol-free beverages (below 0.5% ABV) may be appropriate in harm reduction contexts. However, practitioners should exercise clinical judgement: for some people in recovery — particularly those using AA/12-step frameworks — alcohol-free alternatives that replicate the sensory experience of drinking may not be appropriate.

Further Reading: UK CMO Low Risk Drinking Guidelines (2016) — gov.uk | Drinkaware Unit Calculator — drinkaware.co.uk | NHS Alcohol Units — nhs.uk

CHAPTER 3

How Alcohol Works: Pharmacology & Neuroscience

Alcohol (ethanol) is rapidly absorbed following ingestion. Unlike most drugs, it requires no active transport mechanism — it crosses biological membranes by simple diffusion. Approximately 20% is absorbed in the stomach and 80% in the small intestine. Food in the stomach slows gastric emptying and therefore slows absorption — eating before or during drinking significantly reduces peak blood alcohol concentration (BAC). Carbonated drinks speed absorption, as CO_2 accelerates gastric emptying. Alcohol enters the bloodstream directly and reaches the brain within approximately **five minutes** of consumption.

Alcohol is water-soluble and distributes throughout total body water. Women typically reach a higher BAC than men consuming an equivalent dose, due to lower total body water percentage, higher body fat percentage, and lower levels of gastric alcohol dehydrogenase (ADH) — meaning more alcohol reaches the bloodstream unmetabolised. Older adults have reduced total body water and often lower liver enzyme activity, increasing vulnerability to harm at equivalent doses.

Blood Alcohol Concentration: Effects by Level

BAC (g/100ml)	Approximate Effects	Clinical Notes
0.02–0.05	Relaxation, mild euphoria, reduced inhibition, slight impairment of judgement	Driving impairment begins below legal limit
0.05–0.08	Impaired judgement, coordination, and reaction time; reduced risk perception	Legal driving limit in England/Wales is 0.08
0.10–0.15	Slurred speech, pronounced ataxia, emotional lability, nausea in some	Clearly visible intoxication to others
0.15–0.20	Disorientation, significant motor impairment, aggression or emotional distress	High risk of falls and injury
0.20–0.25	Severe impairment; vomiting; blackouts (en bloc or fragmentary amnesia)	Medical attention may be required
0.30–0.40	Loss of consciousness; respiratory depression; hypothermia risk	Medical emergency — call 999
0.40+	Potentially fatal coma; respiratory arrest; death	**FATAL DOSE RANGE — emergency resuscitation**

⚠ **CLINICAL WARNING — Alcohol Poisoning:** A person who is unconscious, cannot be woken, has slow or irregular breathing, is blue around the lips, or is vomiting while unconscious is experiencing acute alcohol poisoning. Call 999 immediately. Place in the recovery position if unconscious and breathing. Do NOT leave them alone. BAC above 0.30g/100ml is a medical emergency. Do not assume someone is "just drunk" and will sleep it off.

Metabolism and Mechanism of Action

Metabolism

The liver metabolises approximately **90–95%** of ingested alcohol at a relatively fixed rate of approximately one unit per hour — this cannot be meaningfully accelerated by coffee, cold showers, food, or exercise.

The primary metabolic pathway is: Ethanol → **Acetaldehyde** (via ADH) → Acetate (via ALDH) → Water + CO_2. Acetaldehyde is the primary toxic intermediate — it causes DNA damage, protein adduct formation, and is responsible for much of alcohol's carcinogenic and organ-damaging effects. It is also the target of disulfiram (Antabuse).

Genetic variation in ALDH2 activity — common in East Asian populations — causes intense flushing, nausea, and tachycardia even from small amounts of alcohol. Chronic heavy drinking induces the MEOS pathway, generating reactive oxygen species and contributing to liver damage and increased cancer risk.

Brain Mechanisms

Alcohol is a CNS depressant with multiple sites of action:

- **GABA-A agonism:** Enhances inhibitory signalling — producing sedation, anxiolysis, and motor impairment
- **NMDA antagonism:** Reduces excitatory signalling — contributing to sedation, memory impairment (blackouts), and dissociative effects
- **Dopamine release:** Triggers reward pathway activation — the key neurobiological driver of addiction
- **Endogenous opioid activation:** Releases endorphins — the mechanism targeted by naltrexone
- **Serotonin modulation:** Explains relationship with mood, anxiety, and impulsive behaviour

Tolerance, Neuroadaptation & The Hangover

With repeated heavy drinking, the brain adapts to alcohol's effects — a process of **neuroadaptation** that is the biological foundation of physical dependence. GABA-A receptors downregulate (fewer receptors, reduced sensitivity) while NMDA receptors upregulate (more receptors, increased sensitivity). When alcohol is removed abruptly in a physically dependent drinker, the brain is left in a state of **CNS hyperexcitability** — too little GABA inhibition and too much glutamate excitation. This produces the alcohol withdrawal syndrome, which can cause seizures, delirium tremens, and death.

i The Dopamine "Credit Card": Alcohol temporarily "borrows" from the brain's dopamine reward system. Over time, the brain compensates by reducing baseline dopamine function — "paying back the debt." The result is that the person eventually needs to drink simply to feel normal, not to feel good. This explains why willpower alone is rarely sufficient for recovery from physical dependence.

A **hangover** is the constellation of symptoms following heavy drinking as BAC falls: headache, nausea, fatigue, sensitivity to light and sound, anxiety, and cognitive impairment. Contributing factors include dehydration, electrolyte imbalance, disrupted sleep architecture, acetaldehyde toxicity, congener effects, immune system activation (cytokine release), and rebound excitatory activity. The anxiety often experienced the morning after — sometimes called **"hangxiety"** — reflects CNS rebound excitation as alcohol's GABA-enhancing effect wears off.

CHAPTER 4

Who Is Drinking Problematically? UK Epidemiology

24%	600K	10,473	£21bn
Adults at Risk	**Alcohol Dependent**	**Deaths in 2023**	**Annual Cost**
Adults in England drinking at levels that increase health risk (more than 14 units/week) (OHID, 2024)	Estimated people in England with alcohol dependence — yet only around **18%** are in treatment (OHID NDTMS, 2023–24)	Alcohol-specific deaths registered in the UK in 2023 — the highest figure since records began (ONS, 2024)	Alcohol-related harm costs England per year in public services, NHS costs, crime, and lost productivity

Approximately 76,000 hospital admissions per year in England have alcohol as the primary cause. Alcohol is a contributory factor in approximately **1 in 4 emergency department attendances** and a factor in approximately **40% of all violent crime** in England. Highest rates of alcohol-related harm are concentrated in North East England, North West England, and Yorkshire and the Humber.

Drinker Typology: A Framework for Practitioners

Not everyone who drinks problematically is the same. Understanding where a client sits within a typology framework helps practitioners select appropriate interventions — from brief advice for increasing risk drinkers to medically supervised detoxification for severely dependent drinkers.

Type	Key Characteristics	Typical Intervention
Low Risk	Drinks within 14 units/week; spread over 3+ days; no adverse consequences	Education; reinforce positive behaviour
Increasing Risk	Regularly exceeds 14 units/week; pattern establishing; may not identify drinking as problematic	Brief intervention (AUDIT-C); motivational conversation; self-monitoring tools
Higher Risk	Consistently drinks at higher-risk levels; value systems beginning to shift; relationships and work affected	Extended brief intervention; motivational interviewing; GP referral; nalmefene may be appropriate
Dependent (Moderate)	Daily or near-daily drinking to manage withdrawal; physical dependence established; drinking to function	Full assessment; CIWA-Ar; medical review; community detox if appropriate; acamprosate/naltrexone post-detox
Dependent (Severe)	Severe physical dependence; high seizure and DTs risk; life organised around alcohol; complex comorbidities	Inpatient/residential detox; CIWA-Ar guided prescribing; specialist medical input; intensive psychosocial support

⚠ **CLINICAL WARNING:** NEVER advise a physically dependent drinker to stop drinking suddenly without medical assessment. For a severely dependent drinker, abrupt cessation without medical support can cause withdrawal seizures and delirium tremens — both potentially fatal. Always assess the level of dependence before advising any change. If in doubt, refer to medical services urgently.

Key Population Groups

Older Adults

Significantly under-identified in treatment services despite high rates of AUD. Age-related physiological changes mean harm occurs at lower consumption levels. Cognitive impairment, falls, and medication interactions are particular risks.

Women

Rates of alcohol-related harm have risen disproportionately in women over the past two decades. Women have higher rates of alcohol-related liver disease at equivalent consumption levels. Approximately **1 in 5 women** in England drink at increasing or higher-risk levels (OHID, 2024). Barriers include shame, stigma, caring responsibilities, and domestic abuse.

LGBTQ+ Populations

Higher rates of AUD are consistently documented. The minority stress model (Meyer, 2003) provides the theoretical framework: discrimination, stigma, and rejection increase psychological distress, increasing vulnerability to alcohol use as a coping mechanism. Services should be explicitly LGBTQ+ inclusive.

Veterans

Significantly elevated rates of AUD driven by military culture, trauma exposure, difficulties transitioning to civilian life, and underuse of mainstream services. Reference: Veterans' Gateway and NHS Veterans' Complex Treatment Service.

People Experiencing Homelessness

Very high rates of dependent drinking, often alongside complex comorbidities including mental health disorders, drug use, physical illness, and significant trauma histories. Outreach-based harm reduction, wet houses, and low-threshold services are the evidence-based response.

Black, Asian & Minoritised Ethnic Communities

Those who drink problematically within these communities may face additional barriers including language barriers, cultural stigma, lack of culturally specific services, and concerns about confidentiality. Reference: Race Equality Foundation alcohol and ethnicity guidance (2022).

CHAPTER 5

Health Implications: Overview

Alcohol affects virtually every organ system in the body. This section provides an overview of both the acute and long-term physical and mental health consequences of harmful and dependent drinking. Practitioners do not need to be clinicians to recognise these harms, discuss them sensitively with clients, and make appropriate referrals.

Short-Term Harms

Injury, acute alcohol poisoning, blackouts, risky sexual behaviour, self-harm and suicide

Liver Disease

Fatty liver, alcoholic hepatitis, cirrhosis — the most common serious physical consequence of dependent drinking

Cardiovascular

Cardiomyopathy, atrial fibrillation, hypertension, stroke — multiple mechanisms of harm

Cancer

Group 1 carcinogen — causally associated with 7 cancer types; ~12,000 cases per year in England attributable to alcohol

Neurological

Wernicke-Korsakoff syndrome, peripheral neuropathy, cerebellar degeneration, alcoholic dementia

Mental Health

Depression, anxiety, PTSD, psychosis, and significantly elevated suicide risk — bidirectional and complex

Short-Term Harms

Injury and Acute Poisoning

Alcohol is a factor in approximately **40% of all violent crime** in England. It significantly increases the risk of falls, road traffic accidents, industrial injuries, and accidental drowning. The combination of impaired judgement, coordination, reaction time, and risk perception makes injury the most common acute consequence of heavy single-episode drinking.

At BAC above approximately 0.30g/100ml, life-threatening acute alcohol poisoning may occur. Risks include asphyxiation from vomiting while unconscious, respiratory depression and arrest, hypothermia, and cardiac arrhythmias.

Blackouts

Alcohol-induced amnesia is caused by NMDA receptor blockade impairing hippocampal memory consolidation. In **fragmentary blackouts**, the person has patchy memory of events. In **en bloc blackouts**, there is complete amnesia for a period despite the person appearing conscious and functional. Regular blackouts are a significant risk indicator and a red flag for dependent drinking.

Self-Harm and Suicide

Acute alcohol intoxication significantly increases impulsivity and lowers the threshold for self-harm and suicide attempts. Alcohol is a factor in approximately **20–30% of suicides** in England (ONS; OHID). People presenting in crisis following heavy drinking should receive appropriate mental health assessment — not simply be discharged when sober.

Risky Sexual Behaviour

Alcohol-impaired judgement is associated with reduced condom use, increased number of sexual partners, and higher STI and HIV transmission risk. Alcohol is also associated with a high proportion of sexual assaults. Practitioners should address sexual health using non-judgemental, sensitive, trauma-informed language.

⚠ **CRITICAL ALERT — Acute Alcohol Poisoning:** Call 999 immediately if someone is unconscious, cannot be roused, has slow or irregular breathing, is blue around the lips, or is vomiting while unconscious. Place in the recovery position if unconscious but breathing. Never leave a heavily intoxicated person alone. Do not give coffee, food, or cold water — these do not speed up alcohol metabolism.

Alcohol-Related Liver Disease (ArLD)

Alcohol-related liver disease (ArLD) is the most common serious physical consequence of dependent drinking and the leading cause of liver-related mortality in the UK. Liver disease deaths have increased by over **400%** since the 1970s. Approximately 60% of liver disease in England is alcohol-related, and England has significantly worse liver disease outcomes than comparable European countries.

Stage 1 — Fatty Liver (Steatosis)

Fat accumulates in liver cells due to metabolic disruption. Usually asymptomatic. **Reversible with abstinence.**

Stage 2 — Alcoholic Hepatitis

Liver inflammation from ongoing alcohol use. Severe acute alcoholic hepatitis carries a **30-day mortality of up to 30–50%.** Symptoms include jaundice, abdominal pain, and fatigue. Urgent medical assessment required.

Stage 3 — Cirrhosis

Irreversible scarring of the liver. Progressive liver failure, portal hypertension, and risk of hepatocellular carcinoma. Abstinence can stabilise but not reverse damage. Liver transplantation may be required.

⚠ **ArLD Clinical Alert:** Any client with a significant history of alcohol use presenting with jaundice (yellow skin/eyes), abdominal swelling (ascites), confusion, or blood in vomit must be referred for urgent medical assessment. Jaundice + heavy drinking = possible acute alcoholic hepatitis. Do not delay. Reference: NHS England Liver Disease Strategy (2023).

Cardiovascular Disease & Cancer

Cardiovascular Effects

- **Cardiomyopathy:** Heavy chronic drinking causes weakening of the heart muscle, potentially leading to heart failure
- **Atrial Fibrillation:** "Holiday heart syndrome" — AF triggered by binge drinking even in otherwise healthy individuals. AF increases stroke risk fivefold
- **Hypertension:** Even modest regular alcohol consumption elevates blood pressure in a dose-dependent fashion
- **Stroke:** Alcohol increases risk of both ischaemic and haemorrhagic stroke

i The J-Curve Controversy: The claim that low-level alcohol is cardioprotective is now considered largely an artefact of research methodology. Mendelian randomisation studies (2018–2024) consistently fail to find a cardiovascular protective effect. Current UK CMO guidance states there is no completely safe level of alcohol.

Cancer Risk

Alcohol is classified as a **Group 1 carcinogen** by the IARC — meaning the evidence for causation is conclusive. Alcohol is causally associated with cancers of the:

- Mouth and oropharynx
- Larynx and pharynx
- Oesophagus (squamous cell carcinoma)
- Stomach and liver
- Colorectum (bowel)
- Breast (in women — even at low consumption levels)

In England, approximately **12,000 cancer cases per year** are attributable to alcohol (Cancer Research UK, 2024). The risk begins at the lowest levels of consumption — there is no threshold below which alcohol is definitively safe for cancer risk. Raising awareness of this connection is a priority in brief interventions.

Neurological Complications

⚠ **CRITICAL ALERT — Wernicke's Encephalopathy — MEDICAL EMERGENCY:** Caused by severe thiamine (Vitamin B1) deficiency. Classic triad: confusion/altered consciousness + ataxia (unsteady gait) + ophthalmoplegia (eye movement abnormalities). If untreated, it WILL progress to Korsakoff's syndrome — severe, often PERMANENT memory impairment. Treatment: HIGH-DOSE IV PABRINEX URGENTLY — oral thiamine alone is INSUFFICIENT. Do NOT wait for all three features — the full triad is seen in fewer than 20% of cases. Treat on suspicion. Reference: NICE CG141.

Wernicke-Korsakoff Syndrome

Wernicke's is an acute, potentially reversible neurological emergency caused by thiamine deficiency. Korsakoff's syndrome is the chronic, largely irreversible amnestic state resulting from untreated Wernicke's — characterised by profound anterograde amnesia, retrograde amnesia, and confabulation. Patients typically require long-term residential or nursing care.

Peripheral Neuropathy

Chronic heavy drinking causes damage to peripheral nerves — producing numbness, tingling, pain, and weakness, typically beginning in the hands and feet. Mediated by thiamine deficiency and direct ethanol toxicity. Symptoms may partially recover with abstinence and thiamine supplementation.

Cerebellar Degeneration

Alcohol causes degeneration of the cerebellum, producing progressive ataxia (unsteady gait, poor balance and coordination). Cerebellar damage may be partially reversible with abstinence.

Alcoholic Dementia

Distinct from Korsakoff's syndrome — refers to diffuse brain damage from chronic heavy drinking, characterised by impairments in memory, executive function, and social cognition. In severe cases, the clinical picture resembles other dementias.

Gastrointestinal, Immune & Endocrine Effects

Gastrointestinal Complications

- **Gastritis and peptic ulcer disease:** Alcohol damages the gastric mucosa, increasing acid production and impairing mucosal defence
- **Pancreatitis:** Acute pancreatitis (severe abdominal pain — medical emergency) and chronic pancreatitis leading to diabetes
- **Oesophageal varices:** In cirrhosis, portal hypertension causes dilation of veins in the oesophagus. Rupture causes massive haemorrhage — potentially fatal; emergency 999 response required
- **Mallory-Weiss tears:** Oesophageal mucosal tears caused by repeated retching, producing blood in vomit — medical review required

Immune System

Chronic heavy alcohol consumption suppresses immune function, increasing susceptibility to serious infections including tuberculosis, pneumonia (particularly aspiration pneumonia), and post-COVID complications. Wound healing is impaired. Immune dysregulation contributes to alcohol-related inflammation across organ systems.

Endocrine Effects

Alcohol disrupts hormonal balance: it suppresses testosterone production in men (causing hypogonadism, reduced libido, testicular atrophy) and disrupts oestrogen balance in women. It significantly disrupts sleep architecture, suppressing REM sleep and causing fragmented, non-restorative sleep. Elevated cortisol from chronic alcohol use contributes to anxiety, immunosuppression, hypertension, and metabolic dysregulation.

Pregnancy and Foetal Alcohol Spectrum Disorders (FASD)

⚠ **CRITICAL ALERT — FASD:** There is no safe level of alcohol in pregnancy. Alcohol crosses the placenta freely and enters foetal circulation. FASD is the **leading preventable cause of neurodevelopmental disability** in the UK. Estimated 6,000–7,000 births per year in England are affected (BMA, 2022). UK CMO guidance: women who are pregnant or planning pregnancy should not drink any alcohol. There is no known safe level, safe timing, or safe type of alcohol during pregnancy.

FASD encompasses a spectrum of presentations ranging from Foetal Alcohol Syndrome (FAS) — characterised by specific facial features, growth restriction, and severe CNS abnormalities — to milder learning and behavioural difficulties without the full FAS phenotype. Features include: attention and memory difficulties, learning disabilities, executive function impairment, language and communication difficulties, adaptive behaviour challenges, and social/emotional dysregulation.

FASD is lifelong and there is no cure, but early diagnosis and appropriate educational and social support significantly improve outcomes. Reference: NICE antenatal care guidelines (NG201, updated 2021); BMA report on FASD (2022); FASD Trust (www.fasdtrust.co.uk).

Mental Health: A Bidirectional Relationship

The relationship between alcohol use and mental health is bidirectional, complex, and often clinically underrecognised. Heavy drinking causes and exacerbates depression through direct neurotoxic effects, disruption of sleep, nutritional deficiencies, and social consequences. Importantly, **depressive symptoms in heavy drinkers often substantially improve within four to six weeks of abstinence** — practitioners and GPs should be cautious about prescribing antidepressants to people who are actively drinking heavily without first establishing a period of abstinence and reassessment.

Anxiety

Alcohol temporarily reduces anxiety via GABA enhancement — why many people with anxiety disorders drink to manage symptoms (self-medication model). However, as BAC falls, rebound CNS excitation produces worsened anxiety. Repeated cycles of relief and rebound create and maintain anxiety disorders. Practitioners should explore whether anxiety preceded or followed problematic drinking.

PTSD

Very high rates of AUD are documented in trauma survivors. Alcohol's sedating and dissociative properties provide temporary relief from intrusive memories and hyperarousal. However, heavy drinking impairs PTSD recovery and makes trauma-focused therapies less effective. Integrated trauma and alcohol treatment produces better outcomes than sequential treatment. Reference: NICE NG116 (2018).

Psychosis

Alcohol can trigger psychotic episodes, particularly in vulnerable individuals. Delirium tremens involves psychotic features including visual, auditory, and tactile hallucinations. Alcohol-induced psychotic disorder (ICD-11: 6C41) may also occur in chronic heavy drinkers independently of withdrawal.

Suicide

Alcohol is a factor in approximately **20–30% of suicides** in England. Acute intoxication dramatically increases impulsivity and lowers the threshold for acting on suicidal thoughts. All practitioners should conduct routine suicide risk assessment, particularly during or following heavy drinking episodes and during early abstinence.

CHAPTER 6

Alcohol Dependence and Withdrawal

Alcohol dependence is a recognised medical condition, not a moral failing. Accurate diagnosis guides appropriate treatment — particularly the level of medical support required for safe withdrawal.

ICD-11 Diagnostic Criteria (Code 6C40.2)

1. Strong internal urge (craving) to use alcohol
2. Impaired ability to control the onset, frequency, amount, or cessation of drinking
3. Alcohol use takes priority over other activities and obligations
4. Tolerance: need for increasing amounts to achieve the same effect
5. Withdrawal symptoms or use of alcohol to avoid withdrawal
6. Continued drinking despite evidence of significant harm

Diagnosis requires **three or more features** present over a 12-month period.

DSM-5-TR — Alcohol Use Disorder (AUD)

11 criteria assessed over a 12-month period. Severity:

- **Mild AUD:** 2–3 criteria met
- **Moderate AUD:** 4–5 criteria met
- **Severe AUD:** 6 or more criteria met

Criteria include: craving; impaired control; social impairment; risky use; tolerance; withdrawal.

Screening Tools: AUDIT, AUDIT-C & CAGE

AUDIT (Full — 10 Items)

The gold standard screening tool (Babor et al., WHO, 1989). Covers: frequency of drinking; amount consumed; frequency of heavy sessions; loss of control; failure of expectations; morning drinking; guilt; blackouts; injury; and concern expressed by others.

- **0–7:** Lower risk — routine education
- **8–15:** Increasing risk — brief advice
- **16–19:** Higher risk — extended brief intervention; consider GP referral
- **20+:** Possible dependence — full assessment; specialist referral

AUDIT-C (3-Item Brief Version)

Items 1, 2, and 3 only — scored out of 12. Score of **5 or more in men, 4 or more in women**, suggests harmful or hazardous drinking and warrants fuller assessment. Ideal for opportunistic screening in primary care, A&E, and antenatal settings.

CAGE Questionnaire

A simple 4-item screening tool widely used in primary care:

- (C) Have you ever felt you should **Cut down** on your drinking?
- (A) Have people **Annoyed** you by criticising your drinking?
- (G) Have you ever felt **Guilty** about your drinking?
- (E) Have you ever needed a drink first thing in the morning **(Eye-opener)**?

Two or more positive responses suggest possible dependence — warrants AUDIT and further assessment.

Alcohol Withdrawal: Why It Can Kill

Alcohol withdrawal is potentially fatal. As described in the pharmacology section, chronic heavy drinking causes neuroadaptation: GABA-A receptors downregulate and NMDA receptors upregulate. When alcohol is removed, the brain is suddenly unbalanced — too little inhibition and too much excitation. The result is **CNS hyperexcitability**, which can produce seizures, delirium tremens, cardiovascular collapse, and death.

Time After Last Drink	Symptoms	Clinical Priority
6–12 hours	Tremor, sweating, anxiety, nausea/vomiting, tachycardia, hypertension, insomnia	Begin CIWA-Ar assessment; medical review if high risk
12–24 hours	Above worsening; possible hallucinations (visual, auditory, or tactile) — "alcoholic hallucinosis"	Medical review; consider prescribing if not already started
24–48 hours	PEAK RISK: grand mal (tonic-clonic) withdrawal seizures	⚠ HIGH RISK — medical review essential; hospital admission if seizure occurs
48–72 hours	Risk of Delirium Tremens (DTs): confusion, severe agitation, fever, vivid hallucinations, cardiovascular collapse	🚨 LIFE-THREATENING — inpatient management essential
5–7 days	Acute withdrawal resolving in most cases; Post-Acute Withdrawal Syndrome (PAWS) may follow	Ongoing psychosocial support; relapse prevention planning

⚠ **CRITICAL ALERT — Delirium Tremens:** DTs occur in approximately 5% of people undergoing alcohol withdrawal and carry a mortality of **5–10% if untreated.** Features: severe agitation, confusion, fever (>38°C), intense visual/tactile hallucinations, tachycardia, hypertension, sweating. Anyone presenting with features of DTs MUST be admitted to hospital IMMEDIATELY — call 999. Do NOT attempt community management of DTs.

CIWA-Ar: Clinical Withdrawal Assessment

The CIWA-Ar is the validated clinical tool for measuring withdrawal severity and guiding prescribing decisions. It assesses ten domains, each scored 0–7 (except one scored 0–4), giving a maximum score of 67.

CIWA-Ar Domain	What Is Assessed
Nausea/vomiting	Presence and severity of nausea; retching or vomiting
Tremor	Outstretched arms; severity of hand tremor
Sweating	Observation of diaphoresis
Anxiety	Self-reported and observed anxiety
Agitation	Observed psychomotor agitation
Perceptual disturbances	Visual, auditory, or tactile hallucinations/misperceptions
Headache/fullness in head	Self-reported
Clouding of sensorium	Orientation to time, place, date; ability to do serial additions
Paroxysmal sweats	Sudden episodes of sweating

Score <8

Mild — monitor; may not require pharmacotherapy; community detox may be appropriate

Score 8–14

Moderate — prescribing indicated; review medical suitability for community detox

Score 15–19

Moderately severe — medical review essential; consider inpatient management

Score 20+

Severe — inpatient management required; high seizure/DTs risk

⚑ **PRACTITIONER NOTE:** CIWA-Ar is a clinical tool requiring trained administration. Non-clinical practitioners should use the scale to understand risk categories and escalation thresholds, and ensure appropriate medical referrals for all clients assessed as moderate-severe.

The Kindling Effect & Post-Acute Withdrawal Syndrome

The Kindling Effect

Each episode of alcohol withdrawal **sensitises the brain to subsequent withdrawals** — a phenomenon called "kindling." People who have previously experienced withdrawal seizures or DTs are at significantly higher risk of more severe withdrawal on subsequent attempts.

Always obtain a full withdrawal history: Has the person ever had fits or seizures related to stopping drinking? Have they ever experienced DTs? This information is essential for risk stratification.

Post-Acute Withdrawal Syndrome (PAWS)

Beyond the acute withdrawal period (days 1–7), many people experience a protracted withdrawal syndrome lasting weeks to months. PAWS symptoms include:

- Anxiety, irritability, and emotional dysregulation
- Depression and anhedonia (inability to feel pleasure)
- Insomnia and disturbed sleep
- Poor concentration and cognitive fog
- Intense cravings
- Fatigue and reduced stress tolerance

PAWS is a major factor in relapse. Clients should be informed that these symptoms are a normal part of recovery — not evidence that they cannot get better. Symptoms generally diminish over weeks to months with sustained abstinence.

CHAPTER 7

Harm Reduction

The UK Chief Medical Officers' Low Risk Drinking Guidelines (CMO, 2016) define low risk alcohol consumption as: no more than **14 units per week** for both men and women; spread over three or more days — not "saved up" for one or two heavy sessions; having several alcohol-free days each week. Women who are pregnant or planning pregnancy should not drink any alcohol. Importantly, "low risk" does not mean "no risk" — the CMO guidance explicitly states there is no completely safe level of alcohol consumption.

⚠ **CLINICAL WARNING — Do Not Advise Abrupt Cessation Without Assessment:** For physically dependent drinkers, sudden cessation can cause withdrawal seizures and delirium tremens — both potentially fatal. ALWAYS assess the level of physical dependence before advising any reduction or cessation. If a client reports morning drinking, drinking to stop shaking, or a history of withdrawal symptoms, urgent medical assessment is required before any reduction advice is given.

Sensible Drinking Strategies

For clients who are not yet dependent and not ready for formal treatment, the following strategies can support harm reduction:

→ Track Your Units

Keep a drinking diary — track units consumed each day using the MyDrinkaware app or NHS unit calculator. Awareness is the first step to change.

→ Pace and Alternate

Alternate alcoholic and non-alcoholic drinks throughout a session. Eat before and during drinking — food slows absorption and reduces peak BAC. Hydrate with water alongside and after drinking.

→ Plan Alcohol-Free Days

Plan alcohol-free days and activities to fill the time that drinking previously occupied. Set a weekly budget for alcohol spend — financial cost is often a powerful motivator.

→ Avoid Triggers

Avoid drinking when emotionally distressed, lonely, or under pressure — use the HALT check (Hungry, Angry, Lonely, Tired). Identify triggers and plan for high-risk situations.

→ Switch to Lower ABV

Switch to lower ABV alternatives where possible — e.g., session beers (3–4%) rather than premium lager (5–6%). Avoid the "rounds" culture when trying to reduce — feel empowered to decline or buy your own.

Harm Reduction for Dependent Drinkers & Thiamine

Medical Harm Reduction Approaches

- **Supervised reduction:** Medically supervised tapering regime using chlordiazepoxide or diazepam reduces withdrawal risk
- **Nalmefene (Selincro):** Taken as needed before anticipated drinking — for people not aiming for immediate abstinence but wanting to reduce consumption
- **Hydration and nutrition:** Dependent drinkers are often severely malnourished and dehydrated; nutritional support is a harm reduction priority
- **Wet housing and managed alcohol programmes:** For people with severe dependence unable to achieve abstinence, these models reduce chaotic use and have been associated with improved health and social outcomes

Thiamine: A Critical Priority

All alcohol-dependent patients must be prescribed **oral thiamine 100mg three times daily**.

Anyone presenting acutely with symptoms of Wernicke's encephalopathy, those who are malnourished, or those admitted for detoxification MUST receive **IV Pabrinex (parenteral thiamine)** — oral thiamine is insufficient as absorption is severely impaired in heavy drinkers.

Do NOT wait for signs of Wernicke's before prescribing parenteral thiamine — by that point irreversible brain damage may already have occurred. Reference: NICE CG141.

Alcohol, Driving & Dangerous Medication Interactions

Driving Limits

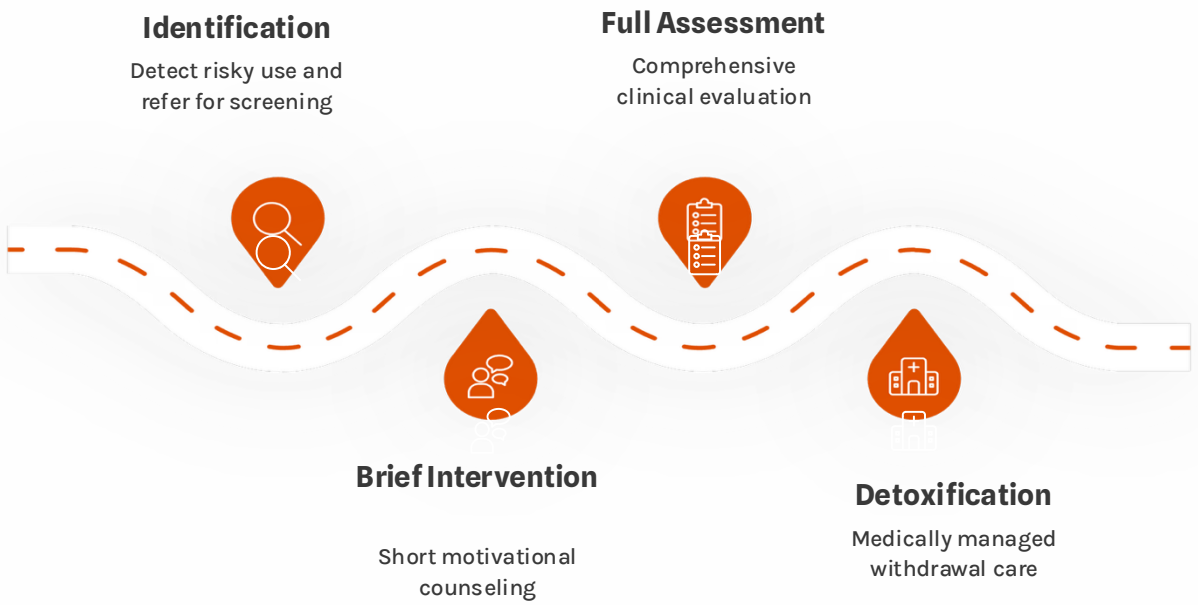
England & Wales: 80mg per 100ml blood (0.08g/100ml). **Scotland:** 50mg per 100ml blood (0.05g/100ml) — introduced December 2014. Approximately 2 units consumed in one hour raises most people in England/Wales to approximately the legal limit. There is NO reliable way to calculate when you are below the limit after drinking the previous night — the only safe choice is not to drive after drinking.

Medication	Interaction with Alcohol	Risk Level
Benzodiazepines (diazepam, etc.)	Synergistic CNS/respiratory depression	**VERY HIGH**
Opioids (codeine, morphine, tramadol)	Synergistic respiratory depression; risk of fatal overdose	**VERY HIGH**
Disulfiram (Antabuse)	Severe acetaldehyde accumulation reaction — nausea, flushing, collapse	**VERY HIGH**
Metronidazole (antibiotic)	Disulfiram-like reaction — severe nausea, vomiting	**HIGH**
Warfarin	Increased bleeding risk; erratic anticoagulant effect	**HIGH**
Paracetamol (heavy drinking)	Hepatotoxicity risk significantly increased; lower toxic dose threshold	**HIGH**
Antidepressants (SSRIs, TCAs)	Enhanced CNS depression; impaired antidepressant efficacy	MODERATE-HIGH
Pregabalin/gabapentin	Enhanced CNS depression; respiratory depression risk	MODERATE-HIGH
NSAIDs (ibuprofen, naproxen)	Increased GI bleeding risk; gastric ulceration	MODERATE

CHAPTER 8

Treatment Approaches: Overview

Effective treatment for alcohol use disorder combines evidence-based psychosocial interventions with pharmacological support where indicated. Treatment should be tailored to the individual's level of dependence, readiness to change, physical and mental health status, social circumstances, and personal goals — which may include abstinence or controlled drinking, depending on the level of dependence.



This pathway — from opportunistic screening through to sustained recovery support — represents the full continuum of care for alcohol use disorder. Each stage has distinct tools, evidence-based approaches, and referral thresholds. The sections that follow detail each component in depth.

Screening and Brief Interventions

Brief Intervention (BI) following AUDIT or AUDIT-C screening is the most cost-effective intervention in the alcohol harm reduction toolkit. NICE recommends BI in primary care, A&E, antenatal services, and other opportunistic settings (NICE PH24).

The 5-As Framework

- **Ask:** Screen using AUDIT or AUDIT-C; ask routinely about alcohol use
- **Advise:** Give personalised feedback based on screening results; link to guidelines
- **Assess:** Determine readiness to change; assess dependence level
- **Assist:** Provide brief advice, motivational conversation, and self-help resources
- **Arrange:** Follow-up; refer to specialist services where indicated

The FRAMES Framework

- **Feedback:** Personalised feedback about current use and risk
- **Responsibility:** The choice and responsibility for change belongs to the individual
- **Advice:** Clear, non-judgemental advice about reducing harm
- **Menu of options:** Provide a range of options for change
- **Empathy:** Warm, empathetic, non-confrontational style
- **Self-efficacy:** Support the individual's belief in their ability to change

Psychosocial Interventions

Motivational Interviewing (MI)

First-line approach; particularly effective in precontemplation and contemplation stages; collaborative, evocative, autonomy-supportive. Miller & Rollnick (4th ed., 2023). NICE-endorsed.

Cognitive Behavioural Therapy (CBT)

Evidence-based for relapse prevention; addresses thoughts and behaviours that maintain drinking; relapse prevention planning. Reference: NICE CG115.

Community Reinforcement Approach (CRA)

Comprehensive evidence-based treatment; addresses social, environmental, and behavioural maintaining factors; restructures reinforcement to favour sobriety.

Behavioural Couples Therapy

Strong evidence base for people in relationships; addresses relationship dysfunction as a maintaining factor in drinking; involves the partner directly in treatment.

SMART Recovery

Secular, cognitive-behavioural alternative to 12-step; based on REBT and CBT principles; growing UK network; online and in-person. www.smartrecovery.org.uk

Mindfulness-Based Relapse Prevention (MBRP)

Growing evidence base (Bowen et al., 2014–2023); combines mindfulness practices with CBT-based relapse prevention; reduces automatic craving responses.

Detoxification: Settings and Criteria

Community Detox

Appropriate for mild-moderate dependence; no history of seizures or DTs; adequate social support; CIWA-Ar <15. Requires daily medical review and prescribing of chlordiazepoxide or diazepam. Reference: NICE CG100.

Residential/Inpatient Detox

Required for: severe dependence; CIWA-Ar ≥15; history of withdrawal seizures or DTs; polydrug use; complex comorbidities; inadequate social support. CIWA-Ar-guided prescribing; 24-hour medical cover. Reference: NICE CG100, CG141.

Medical detoxification is required for all physically dependent drinkers. It cannot be safely managed without medical oversight. The choice of setting is determined by clinical risk assessment — primarily CIWA-Ar score, withdrawal history, and social circumstances. All detoxification regimes should include thiamine supplementation as standard.

Pharmacological Treatments

Chlordiazepoxide (Librium)

First-line benzodiazepine for alcohol detoxification. Long-acting; reduces seizure risk. Standard community regime: 30mg QDS days 1–2; 20mg QDS days 3–4; 10mg TDS days 5–6; 5mg BD day 7; then stop. Must be prescribed by a medical professional. Individual doses vary by clinical assessment.

Acamprosate (Campral)

NICE-recommended first-line relapse prevention (CG115). Reduces cravings by modulating the glutamate/GABA balance disrupted by dependence. Start after detoxification is complete; taken three times daily; suitable for patients with liver disease (renally excreted); no interaction with alcohol.

Naltrexone

Opioid receptor antagonist that blocks the euphoric and reinforcing effects of alcohol and reduces craving. NICE-recommended (CG115). Contraindicated in active hepatitis or liver failure, and in people currently using opioids (will precipitate immediate withdrawal). Also available as injectable extended-release formulation (Vivitrol).

Disulfiram (Antabuse)

Aversive agent: inhibits ALDH, causing acetaldehyde accumulation if alcohol is consumed — producing intense flushing, nausea, vomiting, tachycardia, and potentially severe cardiovascular effects. Reserved for highly motivated patients. Supervised consumption strongly recommended. Contraindications: cardiovascular disease, severe liver disease, psychosis, pregnancy. Reference: NICE CG115.

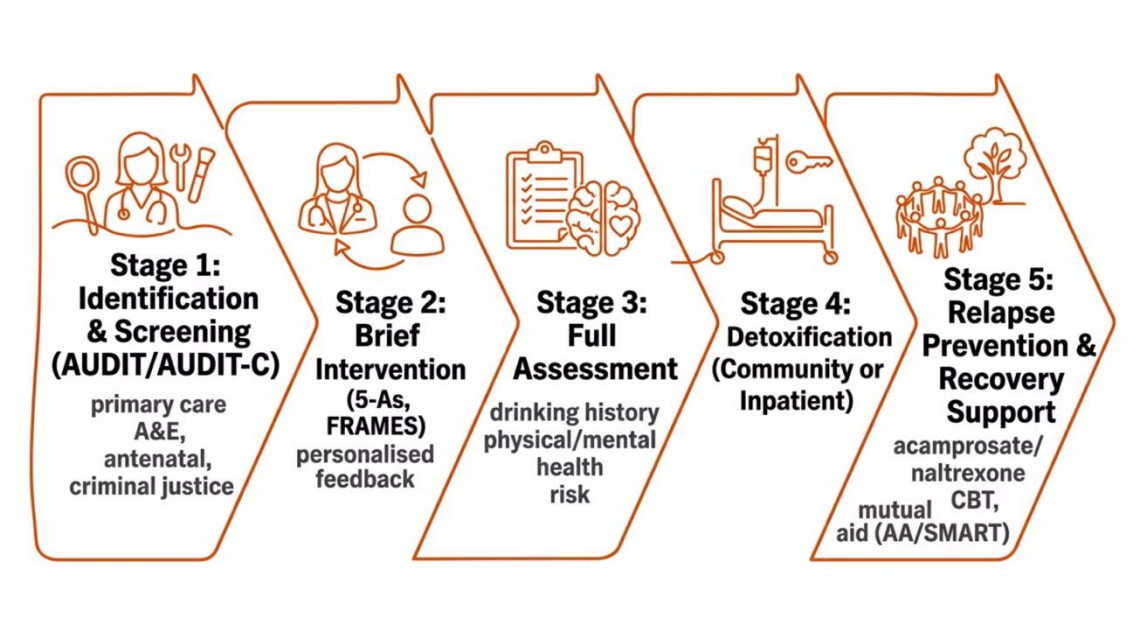
Nalmefene (Selincro)

Licensed in the UK (NICE TA325, 2014) for reducing alcohol consumption in adults not requiring immediate detoxification. Taken "as needed" before anticipated drinking — a harm reduction pharmacological approach. Specifically indicated for consumption reduction rather than abstinence. Appropriate for people not yet aiming for immediate abstinence.

Baclofen

GABA-B receptor agonist increasingly used off-label for alcohol dependence in the UK, and licensed in France (Bacloville trial, 2017). Some evidence for reducing cravings, particularly in patients with liver disease for whom naltrexone is contraindicated. Requires careful dose titration; NICE does not currently recommend routinely — flagged for specialist prescriber consideration.

Treatment Pathway Overview



Recovery capital — housing, employment, social networks, identity, and purpose — is as important as treatment in sustaining long-term recovery. Treatment services should actively support the development of recovery capital alongside clinical interventions. Mutual aid resources include Alcoholics Anonymous (www.alcoholics-anonymous.org.uk) and SMART Recovery (www.smartrecovery.org.uk).

CHAPTER 9

Attracting People to Services

The treatment gap in alcohol services is enormous. Around **600,000 people** in England are estimated to be alcohol dependent, yet only around **18%** are in formal treatment. Stigma, shame, denial, and a lack of awareness of available services are the primary barriers. Services must actively work to reduce barriers and increase accessibility.

Reducing Stigma and Shame

Use non-stigmatising language consistently ("person with alcohol dependence" not "alcoholic"). Explicitly challenge the moral failing narrative — alcohol use disorder is a recognised medical condition with a neurobiological basis. Share recovery narratives — with consent — from people who have been through treatment successfully. Ensure physical environments are welcoming, discreet, and free from institutional stigma.

Digital Engagement

Digital channels increasingly provide the first point of contact for people ambivalent about accessing services. Key resources include: NHS alcohol support pages (nhs.uk/live-well/alcohol-advice); Drinkaware (www.drinkaware.co.uk) — unit calculator, self-assessment tools, MyDrinkaware app; Drinkline: **0300 123 1110** — free, confidential national alcohol helpline; online AA and SMART Recovery meetings — particularly valuable for people not yet ready for face-to-face contact.

Opportunistic Screening

The most effective routes to early identification are opportunistic settings: primary care (GPs and practice nurses); emergency departments (alcohol specialist nurses in A&E are highly cost-effective); workplace EAPs; criminal justice settings; and antenatal services where routine alcohol enquiry is recommended (NICE NG201).

Lived Experience, Peer Mentors & Criminal Justice

Lived Experience and Peer Mentors

Peer mentors and people with lived experience of alcohol use disorder are powerful in reducing stigma, building therapeutic alliance, and inspiring hope. OHID's co-production framework supports the integration of lived experience voices into service design and delivery. Where possible, services should employ peer support workers in recovery and provide them with appropriate training and supervision.

Criminal Justice Pathways

Alcohol-related offending accounts for a significant proportion of all crime in England and Wales. Practitioners working in criminal justice settings should:

- Use AUDIT screening routinely at every contact point
- Be aware that Drug Rehabilitation Requirements (DRRs) under probation supervision can include alcohol treatment conditions
- Understand that court-mandated treatment, while complex ethically, can be effective when combined with genuine therapeutic engagement

Reference: Alcohol Change UK and OHID Criminal Justice Alcohol Data (2023–24).

Working with Families and Carers

Families and carers of people with alcohol use disorder are often profoundly affected — experiencing anxiety, depression, financial hardship, and the strain of managing unpredictable behaviour. They should be **assessed and supported in their own right**, not merely as a route to engaging the person who drinks.

i Care Act 2014 — Carer Assessment Duty: Under the Care Act 2014, local authorities have a duty to assess carers who may have support needs. This duty applies to carers of people with substance use disorders. Practitioners should ensure carers are offered a carer assessment and informed of their rights under the Act.

CRAFT

Community Reinforcement and Family Training — equips family members with skills to reduce enabling behaviours, improve their own wellbeing, and increase the likelihood of the person with AUD entering treatment. Stronger evidence than Al-Anon for increasing treatment uptake.

The 5-Step Method

Developed in the UK (Orford et al.); practical framework for supporting family members in five steps: information, stress identification, coping response analysis, social support, and wellbeing enhancement.

ADFAM UK

National charity for families affected by drug and alcohol use; provides guidance, resources, and a service directory for practitioners and families. www.adfam.org.uk

Children of Dependent Drinkers & Safeguarding

An estimated **700,000 children in England** live with a dependent drinker (OHID). Growing up in a household where alcohol use disorder is present significantly increases risk of adverse childhood experiences (ACEs), mental health difficulties, educational disruption, and later development of AUD.

The Hidden Harm agenda — originating from drug services but equally applicable to alcohol — requires practitioners to:

1 Ask Routinely

Ask routinely about dependent children whenever working with a person with alcohol use disorder.

2 Follow Safeguarding Protocols

Follow safeguarding protocols where there is concern about a child's welfare. Refer to children's social care as required under *Working Together to Safeguard Children* (2023).

3 Connect with Support Services

Connect families with support services for children — e.g., Nacoa (National Association for Children of Alcoholics): www.nacoa.org.uk

⚑ **PRACTITIONER NOTE — Trauma-Informed Practice with Families:** Many carers and family members have their own significant trauma histories — including domestic abuse, bereavement, or their own childhood experiences of parental alcohol use. Practitioners should approach family work with the same trauma-informed lens applied to direct work with clients. Avoid reinforcing shame, blame, or the idea that families have "caused" AUD.

Emergency Contacts & Key Resources

Drinkline

0300 123 1110 Free, confidential national alcohol helpline
9am–8pm weekdays, 11am–4pm weekends

NHS 111

11124/7 — mental health and medical support

Samaritans

116 123 Free, 24/7 emotional support

FRANK

0300 123 6600www.talktofrank.com
Drug and alcohol information and support

Emergency Services

999 For acute alcohol poisoning, withdrawal seizures, delirium tremens, or any life-threatening emergency

Your Key Worker

Record your key worker's contact details here for quick reference during and after training.

This training pack was developed by **Tony D'Agostino, TD Consultancy**

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For further information visit **www.tonydagostino.co.uk.**