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Overview of disorders of volatile inhalant use

Overview of disorders of volatile inhalant use

A wide variety of volatile inhalants is consumed in Australia; the most common are:

- nitrous oxide (eg sold in small canisters as a propellant for whipped cream) used to achieve brief intoxication
- amyl nitrite used as a party drug and to enhance sexual experience (chemsex).

Other volatile inhalants include:

- petrol
- solvents, such as toluene, found in glues, paints, paint thinners and correction fluids
- acetone in nail polish remover
- butane in deodorants and gas canisters.

The use of volatile inhalants is described by colloquial terms. Substances can be inhaled from:

- a soaked cloth on the nose and mouth—'huffing'
- a plastic bag—'bagging'
- a container—'sniffing'.

Use of volatile inhalants in Australia has increased from 0.4% of the population in 2001 (reporting use in the past year) to 1.7% in 2019. Use is more prevalent among some populations, such as young people (of whom 13% report use in the past year). Around one-third of use occurs monthly. Volatile inhalants are readily available, and it is rarely feasible to reduce access to them.

The spectrum of substance use in these guidelines is described by the terms 'hazardous use', 'harmful use' or 'substance dependence', outlined in [Table 22.1](#).

Harms result from acute intoxication and chronic consumption of volatile inhalants. Presentations of acute intoxication vary according to the substance; they are most frequently characterised by euphoria, disinhibition and excitation, but also feature drowsiness, disorientation, hallucinations and ataxia, particularly at high doses. Most patients experiencing inhalant intoxication can be treated with removal of the causal agent, and bed rest. Deaths are rare, but may arise through asphyxia, ventricular fibrillation and other cardiac arrhythmias. Advice on the management of volatile inhalant toxicity is available in [Hydrocarbon poisoning: Inhalation](#).

Chronic, high-level exposure to some volatile inhalants can result in diffuse neurotoxicity, with varying symptoms, most commonly peripheral neuropathy and encephalopathy. Use of nitrous oxide is associated with functional vitamin B₁₂ deficiency and subacute combined degeneration of the spinal cord. Other organ systems, including the kidney and liver, may also be affected by volatile inhalant use.

Chronic use of volatile inhalants can result in dependence. Although inhalant withdrawal is not well described, symptoms reported on abrupt stopping after dependent use include headaches, nausea, vomiting, hallucinations, rhinorrhoea, craving, tachycardia, depressed mood, agitation, insomnia and anxiety.

Screening and assessment of volatile inhalant use

Screening and assessment of volatile inhalant use

Ask all new patients routinely and others (adolescents and adults) opportunistically and periodically about volatile inhalant use, as part of a general screen for disorders of substance use and gambling; these disorders are common (and often co-exist) and people are reluctant to disclose them, often due to fear of stigma. [Screening and assessment of substance use and addictive behaviours](#) outlines history-taking (including use of the [ASSIST-Lite tool](#)), examination, and investigations that should be considered in a broad review of substance use and addictive behaviour. A sociopsychobiomedical assessment is critical as use in young people often reflects significant psychosocial disadvantage (eg poverty, an unstable home situation or major stressors at school). Inhalant use is associated with psychiatric comorbidity and patients identified as using inhalants should be screened for this.

Overview of management of disorders of volatile inhalant use

Overview of management of disorders of volatile inhalant use

[Overview of substance use and addictive behaviours](#) explains key [principles of care](#) for a person with a disorder of substance use. Establishing a therapeutic relationship that engages the person (and ideally those close to them) is central to the management of substance dependence.

The most important element of treatment for substance use is a therapeutic relationship.

Specialist advice on any aspects of care for patients with substance use is available and contact is encouraged; see [Clinical advisory services](#).

Specialist advice is available by phone on any aspect of the management of substance use; contact is encouraged.

Management strategies in disorders of volatile inhalant use include:

- [harm reduction](#)
- [withdrawal management](#)
- [long-term care](#).

Specific considerations for young people are discussed in this topic, but also consider factors that may apply for [other populations](#).

Harm reduction in volatile inhalant use

Harm reduction in volatile inhalant use

Harm reduction in disorders of volatile inhalant use can be helpful, such as teaching patients, particularly young people:

- to avoid putting plastic bags over their heads when ‘bagging’
- to avoid smoking when using inhalants
- how to provide first aid
- to call an ambulance if they are worried about themselves or a friend.

Consider offering a [printable patient information sheet](#) on ways to reduce harms from substance use.

See [Ensuring the safety of a person with a disorder of substance use or addictive behaviour](#) for further advice on immediate safety issues, such as managing agitation, as well as impacts of substance use on fitness to work and to drive. For other harm reduction measures to consider for any person with a disorder of substance use or addictive behaviour, see [Harm reduction](#); these include managing sexual health risks for people who have ‘chemsex’ [\[Note 1\]](#) (eg people who use amyl nitrite).

Note 1: 'Chemsex' refers to using recreational drugs to enhance sexual experience.

Withdrawal management in disorders of volatile inhalant use

Withdrawal management in disorders of volatile inhalant use

Evidence to guide management of dependent inhalant use is very limited. If significant symptoms of moderate agitation occur on withdrawal, use [verbal de-escalation and psychological interventions](#); seek specialist advice (eg from a [clinical advisory service](#)) if agitation persists. Offer patients who wish to stop using inhalants the supports outlined in [Long-term care in the management of disorders of volatile inhalant use](#).

Long-term care in the management of disorders of volatile inhalant use

Long-term care in the management of disorders of volatile inhalant use

Long-term treatment for disorders of volatile substance use includes:

- accessing psychosocial supports (eg from [services that are specific to young people](#)); this is critical to address the severe psychosocial disadvantage faced by many people using volatile inhalants
- [cognitive behavioural therapy](#)
- [family therapy](#)
- activity therapy—programs that engage young people in fun activities while also providing psychoeducation have been very effective in some communities
- [residential treatment](#)—this may be considered as a second-line treatment option.

For advice on long-term management relevant to any person with a disorder of substance use or addictive behaviour, see [Long-term care in disorders of substance use and addictive behaviours](#).

Further educational resources for healthcare professionals about volatile inhalant use

Further educational resources for healthcare professionals about volatile inhalant use

Further educational resources for healthcare professionals about volatile inhalant use include:

- the [Better Health Channel](#)
- videos and webinars about substance use in young people, available at the [Dovetail website](#).

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Overview of varenicline for tobacco smoking and nicotine dependence

Overview of varenicline for tobacco smoking and nicotine dependence

Varenicline is a high-affinity partial agonist at nicotinic acetylcholine receptors; by stimulating dopamine release (although to a lesser extent than nicotine does) it reduces nicotine cravings and withdrawal symptoms. By also blocking nicotine-induced dopamine release, varenicline reduces the reinforcing effects of nicotine from smoking. In a systematic review [\[Note 1\]](#), varenicline was found to have similar effectiveness to [combination nicotine replacement therapy](#) (NRT) and to be more effective than bupropion or NRT monotherapy.

Varenicline may be more effective in:

- females
- patients who have high alcohol consumption
- patients with schizophrenia.

Varenicline is not a first-line choice for [management of tobacco smoking in pregnancy](#) and is not recommended for use in people younger than 18 years. Because varenicline is renally excreted, dosing is dependent on renal function; it is contraindicated in patients with end-stage (dialysis-dependent) kidney disease. Use varenicline with caution in patients with a history of Stevens–Johnson syndrome / toxic epidermal necrolysis (SJS/TEN) and other severe skin hypersensitivity reactions.

Earlier concerns about varenicline and risk of psychiatric adverse effects such as increased suicide risk have not proved valid. Psychiatric comorbidities are not contraindications to varenicline, but follow-up is required after 2 weeks (as for all patients) to review symptoms, any adverse effects and advise on continuation; see [Starting and reviewing varenicline for tobacco smoking and nicotine dependence](#).

Note 1: Cahill K, Lindson-Hawley N, Thomas KH, Fanshawe TR, Lancaster T. Nicotine receptor partial agonists for smoking cessation. Cochrane Database of Systematic Reviews 2016, Issue 5.
<https://www.ncbi.nlm.nih.gov/pubmed/27158893>.

Starting and reviewing varenicline for tobacco smoking and nicotine dependence

Starting and reviewing varenicline for tobacco smoking and nicotine dependence

Varenicline dosing is dependent on renal function and contraindicated in dialysis-dependent kidney disease. If varenicline is preferred for smoking management, use:

varenicline

creatinine clearance 30 mL/minute or more: 0.5 mg orally, daily for 3 days, then 0.5 mg twice daily for 4 days, then 1 mg twice daily for the remainder of a 12-week course

creatinine clearance less than 30 mL/minute: 0.5 mg orally, daily for 3 days, then 1 mg orally, daily for the remainder of a 12-week course.

Most patients naturally stop smoking during the first 1 to 2 weeks of treatment.

Varenicline alone is usually sufficient to manage nicotine cravings, but medium- or fast-acting NRT can safely be added (usually after 2 weeks) if needed to control cue-driven cravings. Evidence is lacking for the impact of combining varenicline and bupropion.

The effectiveness and tolerability of varenicline depend on the patient's genetically determined nicotine receptor structure. Therapeutic and adverse effects become apparent within 4 to 7 days of starting treatment. For those with an incompatible nicotine receptor structure, varenicline can cause withdrawal symptoms that cannot be overcome by smoking [Note 2]. Advise patients about the potential for nicotine withdrawal symptoms within the first week of treatment; encourage them to stop varenicline and seek medical advice if withdrawal symptoms occur.

Independent of withdrawal symptoms, up to 30% of patients taking varenicline experience nausea, which can be reduced by taking it with food.

If insomnia occurs, it can be reduced by taking the second dose of the day in the mid-afternoon, rather than in the evening.

Review all patients 2 weeks after starting varenicline and again at 12 weeks (or earlier) to assess the effectiveness and tolerability of varenicline and provide encouragement.

Offer a repeat 12-week course to anyone who wishes to continue (regardless of whether their smoking has reduced with the current course) to increase the likelihood of prolonged abstinence.

Note 2: Varenicline binding results in partial agonism causing inadequate activation, leading to nicotine withdrawal symptoms, while partial antagonism remains in place, so nicotine from cigarettes is unable to overcome the withdrawal.

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Overview of bupropion for tobacco smoking and nicotine dependence

Overview of bupropion for tobacco smoking and nicotine dependence

Bupropion is a noradrenaline and dopamine reuptake inhibitor and a nicotinic receptor antagonist, reducing the severity of both nicotine withdrawal and cravings that persist beyond the withdrawal period. Although it is a first-line option for smoking management, bupropion is less commonly used than combination nicotine replacement therapy (NRT) or varenicline because it is less effective.

Consider bupropion for tobacco smoking and nicotine dependence for people who:

- cannot tolerate varenicline or combination NRT or found them ineffective
- require concomitant treatment for depression [\[Note 1\]](#)
- prefer the simplicity of taking a single drug.

Bupropion is contraindicated in patients with a history of seizures, eating disorders, those taking irreversible monoamine oxidase inhibitors, and during abrupt withdrawal from alcohol or benzodiazepines (because seizure risk may be increased). Like varenicline, bupropion is only considered for management of smoking during pregnancy when NRT is not suitable. Bupropion is not recommended in people younger than 18 years.

Use bupropion with caution in patients who:

- use other drugs that lower seizure threshold (eg antipsychotics, antidepressants). Doses of bupropion more than 300 mg daily are not recommended because of the risk of dose-dependent seizures
- have bipolar affective disorder, because of the risk of mania.

Bupropion is metabolised by cytochrome P450 enzymes; consider potential drug interactions before prescribing.

The most common adverse effect of bupropion is insomnia, followed by dizziness, anxiety, nausea and dry mouth. Seizures are a rare but serious adverse effect.

Note 1: Although not approved by the Australian Therapeutic Goods Administration (TGA) for this indication, bupropion is commonly used as an antidepressant in other countries.

Starting and reviewing bupropion for tobacco smoking and nicotine dependence

Starting and reviewing bupropion for tobacco smoking and nicotine dependence

If bupropion is preferred for smoking management, use:

bupropion 150 mg orally, in the morning for 3 days, then 150 mg orally, twice a day. Review symptom control and adverse effects after 14 days. If effective and tolerated, continue at this dose for the remainder of a 9-week course.

Patients usually stop smoking around the second week of therapy, but the full 9-week course should be completed. Review patients at the end of the second week of treatment to assess efficacy, tolerability and adjust smoking management advice.

Anecdotal evidence supports the addition of medium- or fast-acting NRT to bupropion after the first 2 weeks if required to manage cue-driven cravings. Evidence is lacking for the impact of combining bupropion and varenicline.

Review all patients 2 weeks after starting bupropion and again at 9 weeks (or earlier) to assess the effectiveness and tolerability of bupropion and provide encouragement.

Bupropion is subsidised by the Pharmaceutical Benefits Scheme (PBS) for one 9-week course per year. Some patients benefit from longer or repeated courses. Offer a further course to any patient who wishes to continue, regardless of whether their smoking has reduced with the current course.

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Nortriptyline for tobacco smoking and nicotine dependence

Nortriptyline for tobacco smoking and nicotine dependence

Nortriptyline, a tricyclic antidepressant, can be used without an approved indication for managing smoking and nicotine dependence if first-line therapies are contraindicated or adequate trials have not been effective. Its use is limited by adverse effects, drug interactions and the smaller body of evidence for its effect in smoking management. Nortriptyline has similar effectiveness to [bupropion](#) and can be beneficial for managing smoking even in patients without depression.

The most common adverse effects of nortriptyline are dry mouth, constipation, nausea, sedation, weight gain and headache. In those with cardiovascular disease, the risk of arrhythmia is increased.

Nortriptyline has clinically significant drug interactions with drugs used to treat depression, mania, epilepsy, thyroid disease and hypertension.

Nortriptyline is not recommended in pregnancy or in people younger than 18 years.

If nortriptyline is preferred for smoking management, use:

nortriptyline 25 mg orally, at night for 7 days, then 50 mg at night for 7 days, then 75 mg at night thereafter if tolerated. Review at 1 month; if beneficial, continue for a further 3 months [\[Note 1\]](#).

Note 1: At the time of writing, nortriptyline is not approved by the Australian Therapeutic Goods Administration (TGA) for smoking management. See the [TGA website](#) for current information.

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Overview of nicotine vaping products for tobacco smoking and nicotine dependence

Overview of nicotine vaping products for tobacco smoking and nicotine dependence

Nicotine vaping products are liquids used in devices that allow people to inhale aerosolised liquid nicotine. All nicotine vaping devices contain a heating element, battery, container for vaping liquid, vents for air entry and a mouthpiece. The vaping liquid is heated and aerosolised before being inhaled into the mouth and lungs where the nicotine is absorbed. Nicotine vaping devices are known by many names, most commonly 'e-cigarettes' and 'vapes'.

While nicotine vaping products have been commercially available for nearly 20 years, at the time of writing, the evidence for efficacy in smoking management is evolving and long-term safety is not certain. Nicotine vaping products can be considered as a second-line option for managing smoking and nicotine dependence if no first-line therapies are suitable or adequate trials of first-line therapies combined with behavioural interventions have not been effective. See [Figure 22.6](#) for an overview of drug therapy to manage tobacco smoking and nicotine dependence.

Discuss the risks of nicotine vaping products as part of shared decision-making before prescribing them; see the section on electronic cigarettes and nicotine vaping products in the [Royal Australian College of General Practitioners Guidelines on Smoking cessation](#). Advise patients to avoid concurrent use of nicotine vaping products and tobacco, to maximise possible benefit and minimise harm. Explain that use of nicotine vaping products is associated with rare instances of severe lung injury and that the long-term effects of use are not known; for more detail, see [E-cigarette and vaping -induced lung injury](#). If a patient has not had an adequate trial of first-line therapies for nicotine dependence; offer alternatives as outlined in [Overview of drug therapy for tobacco smoking and nicotine dependence](#).

At the time of writing, nicotine vaping products are classed as 'unapproved medicines' in Australia and require a prescription for purchase in Australian pharmacies or for importation into Australia. The following safety regulations from the Australian Therapeutic Goods Administration (TGA) are in place for nicotine vaping products sold in Australian pharmacies [\[Note 1\]](#):

- ingredients, nicotine concentration (in mg/mL) and a warning statement must be included on the label
- packaging must be child-resistant (liquid nicotine risks child poisoning through oral or transdermal absorption; see [Nicotine poisoning](#))
- the maximum nicotine concentration is capped at 100 mg/mL
- specific additives known to be toxic and active ingredients other than nicotine are prohibited.

Further information about the history and prescription of nicotine vaping products is available at:

- [TGA guidance on nicotine vaping products](#); this has a link to [TGA guidance for the use of nicotine vaping products for smoking cessation](#), which includes practical guidance and a [flowchart and dosage tables](#)
- [Royal Australian College of General Practitioners Guidelines on Smoking cessation](#)
- [Royal Australian and New Zealand College of Psychiatrists Mental Health Clinician Guidance for Managing People's Smoking Cessation](#).

The simplest way to prescribe nicotine vaping products is to become an [authorised prescriber](#). Other options are to apply under the Special Access Scheme for each patient or to provide a script for a patient who is using the Personal Importation Scheme.

Note 1: The maximum concentration and the prohibition of specific additives also apply to products imported under the Personal Importation Scheme.

Starting and reviewing nicotine vaping products for tobacco smoking and nicotine dependence

Starting and reviewing nicotine vaping products for tobacco smoking and nicotine dependence

Nicotine forms available in Australia for use in nicotine vaping products are freebase nicotine or nicotine salts.

Freebase nicotine is the original form of nicotine for vaping; it suits people who use nicotine vaping products in small inhalations because it can cause throat irritation at concentrations higher than 20 mg/mL.

Nicotine salts have been pH-balanced to reduce the risk of irritation; these newer products are suited to people who are highly nicotine dependent or those who cannot, or prefer not to, use small inhalations of nicotine vaping products regularly during the day and prefer to rely on larger intakes less frequently. These salts have a higher potential for nicotine dependence than freebase nicotine because the concentration of nicotine in each use may be higher.

For patients with low or moderate nicotine dependence (those who start smoking more than 30 minutes after waking), appropriate products are freebase nicotine of 6 to 12 mg/mL or nicotine salts of 20 to 30 mg/mL.

For highly nicotine-dependent patients (those who start smoking within 30 minutes of waking), appropriate concentrations are freebase nicotine of 18 to 20 mg/mL or nicotine salts of concentrations above 30 mg/mL.

If unsure of the appropriate dose, start at a low dose and increase if needed to the lowest effective dose.

Prescribers can specify a nicotine concentration, flavouring and brand if desired, and whether the device is prefilled or refillable. For patients without a preference, a prefilled unit, at a low nicotine concentration (eg 6 to 12 mg/mL) is recommended. Counsel patients that the long-term safety of flavourings in nicotine vaping products is unknown.

Review patients using nicotine vaping products at least 3-monthly. Discuss reducing the nicotine concentration in the nicotine vaping product or switching to [nicotine replacement therapy](#).

E-cigarette– and vaping-associated lung injury

E-cigarette– and vaping-associated lung injury

E-cigarette– and vaping-associated lung injury (EVALI) is a rare, but potentially fatal respiratory illness in which individuals develop pneumonia associated with lipid-laden macrophages in the lungs.

EVALI may occur with nicotine-only vaping liquid but more commonly occurs with vaping liquids containing tetrahydrocannabinol (THC), vitamin E acetate and oil-containing additives. Only products designed specifically for vaporising and inhalation should be used in vaping units. Cannabis oil should not be used in vaping units.



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Overview of management of tobacco smoking in specific populations

Overview of management of tobacco smoking in specific populations

Specific considerations apply in the management of tobacco use in patients who are pregnant or breastfeeding.

For other specific populations see [Considerations for specific populations in substance use and addictive behaviours](#).

Management of tobacco smoking during pregnancy

Management of tobacco smoking during pregnancy

Pregnancy is often the time of highest motivation for a person to make changes to tobacco use. It can also be an opportunity for a clinician to provide prospective parents (and others who will be involved with a newborn) with help in managing smoking.

Smoking during pregnancy exposes the fetus to nicotine and other harmful chemicals that increase the risks of spontaneous abortion, premature delivery, intrauterine growth restriction, sudden infant death syndrome and neonatal nicotine withdrawal syndrome. Neurocognitive and neurobehavioural deficits in older children are also associated with fetal exposure to smoking.

Behavioural interventions in pregnancy are safe and effective. These are first-line treatment in pregnancy. Nicotine replacement therapy (NRT) exposes the fetus to less nicotine than would heavy smoking; NRT also removes the risks from other chemicals in tobacco. Intermittent use of medium- and fast-acting NRT (eg gum, lozenges, inhalators) is preferable to using patches, which result in continuous nicotine replacement. If intermittent NRT is unsuccessful, the 16-hour NRT patch can be added; the patch should be removed before bed, with the goal of reducing total daily exposure of the fetus to nicotine and maximising placental blood flow overnight.

The use of varenicline or bupropion in pregnancy requires consideration of the individual balance of harms and benefits. The uncertainty of the effects of these medications in pregnancy and on long-term development needs to be balanced against the well-known risks of tobacco smoking. Use of varenicline or bupropion is only considered if NRT is ineffective or not tolerated (eg because of marked nausea in pregnancy).

Management of tobacco smoking during breastfeeding

Management of tobacco smoking during breastfeeding

Tobacco smoking during lactation reduces milk production, interferes with the let-down reflex and changes the taste of breastmilk. Nicotine in breastmilk and environmental tobacco smoke on parents' clothes, skin and furniture can cause colic, diarrhoea, tachycardia, irritability, apnoeic episodes and impairment of the immune system in the infant. These risks increase with the number of cigarettes smoked per day. Delaying smoking until after each episode of breastfeeding is completed minimises the amount of nicotine in the breastmilk.

Exposure to environmental tobacco smoke, both passive smoking and on objects and people in the environment, is a risk factor for sudden infant death syndrome.

If a breastfeeding patient continues smoking after a trial of behavioural interventions, combination NRT or bupropion use during breastfeeding is safer for their infant than further exposure to tobacco smoking.



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Terminology in alcohol use

Terminology in alcohol use

Alcohol use is quantified in standard drink measures. A standard drink in Australia contains 10 g of alcohol, the amount in:

- 285 mL of full-strength beer (4.9% alcohol)
- 100 mL of wine (13% alcohol)
- 60 mL of fortified wine (20% alcohol)
- 30 mL of spirits (40% alcohol).

For a visual representation of standard drinks in Australia, see the [National Health and Medical Research Council Standard drinks guide](#).

The [Australian Guidelines to Reduce Health Risks from Drinking Alcohol](#) advise that adults should drink no more than 10 standard drinks per week and no more than 4 drinks on any given day, regardless of gender. This limits the lifetime risk of alcohol-related death from disease or injury to less than 1%; drinking below this level is still associated with an increase in the risk of certain cancers [\[Note 1\]](#) and poorer brain health. Any intake during pregnancy risks fetal injury. Abstinence is recommended for adolescents and anyone planning to become pregnant.

No level of alcohol intake is safe. Australian guidelines advise that intake should not exceed 10 standard drinks per week or 4 drinks on any given day for adults.

The spectrum of substance use is described in these guidelines by the terms 'hazardous use', 'harmful use' or 'substance dependence', outlined in [Table 22.1](#). Almost 80% of Australians drink some alcohol, of whom around a quarter develop a disorder of alcohol use; 4 to 5% of Australians develop alcohol dependence.

An example of [hazardous alcohol use](#) is episodic heavy (binge) drinking, particularly if it is frequent. For most people, 8 or more drinks on one occasion are likely to be associated with harm, while smaller amounts (eg more than 4 drinks on one occasion) can be harmful for older people and those with comorbidities such as liver disease.

[Harmful drinking](#) is heavy drinking that results in injury, accidents, drink-driving convictions or illness. This pattern of use accounts for the largest category of alcohol-related harms; it is a significant contributor to injury and deaths from drowning or driving.

[Alcohol dependence](#) is responsible for one-third of alcohol-related disease burden in Australia; it causes more chronic tissue injury, and physical and mental disease than hazardous or harmful use, but fewer deaths because it is less prevalent. Drugs for the management of disorders of alcohol use (eg [naltrexone](#), [acamprosate](#)) require a diagnosis of alcohol dependence to be subsidised on the Pharmaceutical Benefits Scheme (PBS).

Note 1: Risks are increased for head and neck, gastrointestinal, breast and prostate cancers and non-Hodgkin lymphoma.

Overview of management of disorders of alcohol use

Overview of management of disorders of alcohol use

[Overview of substance use and addictive behaviours](#) explains key [principles of care](#) for a patient with a disorder of substance use. Many patients with alcohol dependence have denied their drinking or feared the consequences of confronting it for years; most present late. Establishing a therapeutic relationship that engages the person (and ideally those close to them) is central to the management of substance dependence. Develop an agreement for the first steps (eg to undertake short-term [planned withdrawal](#) or to [reduce intake gradually](#)) and review the plan as soon as possible.

The most important element of treatment for substance use is a therapeutic relationship.

Specialist advice on any aspects of care for patients with alcohol and other substance dependence is available and contact is encouraged; see [Contact details for substance use clinical advisory services for clinicians](#).

Specialist advice is available by phone on any aspect of the management of substance use; contact is encouraged.

Alcohol use can pose risk of acute harms to the person (such as self-harm, falls and other accidents, suicide) and those around them (through impacts on driving, childcare, fitness to work and acute behavioural disturbances). In any presentation, [ensuring the safety](#) of the person and those around them is a priority, especially if the person is highly agitated. Profound disinhibition in highly intoxicated people (eg young men who have a prior history of aggressive behaviour) can lead to serious acts of violence; most perpetrators and victims of fatal 'king hit' injuries (single blows to the head causing loss of consciousness, also called 'coward punches' or 'one-punch attacks') are highly intoxicated. Alcohol use is also associated with a broad range of acute medical conditions that may require stabilisation (eg liver failure, pancreatitis, gastrointestinal bleeding, head trauma, seizures, [Wernicke encephalopathy](#), aspiration, sepsis).

Further management options for disorders of alcohol use include:

- brief interventions
- short-term withdrawal management
- gradual reduction of alcohol use
- harm reduction strategies
- long-term management of alcohol dependence, including drug therapies.

Consider offering a [brief intervention](#) for anyone with excessive alcohol use. Brief interventions produce small reductions in hazardous use in research settings compared to standard treatment. The benefits in harmful or dependent alcohol use are less clear than in hazardous use, but motivational interviewing should be considered as part of a stepped approach to aid the development of a treatment plan.

Some patients require or choose to undergo intervention early, such as a [planned withdrawal](#) or a [gradual reduction](#), possibly with a view to abstinence.

Some patients will not be willing to engage with these options but will consider [harm reduction](#) strategies, which should be offered to all patients.

All patients should also be offered [long-term management](#) as part of a chronic illness management plan. Many aspects of long-term management involve [cognitive behavioural therapy](#). In addition, for those who are abstinent, maintenance drug therapy can be considered for relapse prevention. Other supports include residential rehabilitation, peer supports and self-help. Encourage social contact, as isolation increases the risk of poor outcomes.

Certain populations may benefit from specific consideration in the management of substance use—see discussion in [Overview of substance use and addictive behaviours](#). Management advice specific to [alcohol use in pregnancy](#) and [alcohol use while breastfeeding](#) is included in this topic.

References



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History-taking in assessing alcohol use

History-taking in assessing alcohol use

Ask all new patients routinely and others (adolescents and adults) opportunistically and periodically about alcohol use, as part of a general screen for disorders of substance use and gambling; these disorders are common (and often co-exist) and patients are reluctant to disclose them, often due to fear of stigma. Without routine screening, the diagnosis of a disorder of alcohol use is often missed.

Screening and assessment of substance use and addictive behaviours outlines history-taking (including use of the ASSIST-Lite tool), examination, and investigations that should be considered in a broad review of substance use and addictive behaviour.

If a person appears intoxicated or has signs of alcohol withdrawal, keep history-taking brief (focusing on asking about withdrawal symptoms as part of identifying dependent use); defer efforts to quantify use until the person is oriented, attentive and coherent.

If alcohol use is identified, ensure a full history is taken to assess:

- the types of drinks, and quantity and frequency of drinking; use familiar measures (eg cans of beer, glass size) and strengths (for a visual representation of standard drinks in Australia, see the National Health and Medical Research Council Standard drinks guide)
- the environments, social context and time of day when drinking occurs, and duration of drinking sessions
- when the last drink occurred
- measures of alcohol-related harm (for a graphic summarising potential organ damage, see the United States National Institute of Health website). In particular, ask about neurological harms (eg memory blackouts, seizures), features of liver disease or pancreatitis, cardiovascular harms [\[Note 1\]](#), drink-driving, and accidental injury
- social impact (eg impact of drinking on family, parental and occupational capacity) and the extent of the person's current support network
- the person's understanding of their triggers for relapse and remission
- previous attempts to cut down or stop drinking and the severity of any withdrawal symptoms
- previous treatment (including duration) for a disorder of alcohol use [\[Note 2\]](#)
- collateral history (eg from someone close to the person) of the drinking pattern, time course of change in drinking, landmark events (such as loss of alcohol tolerance, and personality changes [\[Note 3\]](#))
- the age at which excessive alcohol use began and any family history of a disorder of alcohol use [\[Note 4\]](#).

Alcohol-specific diagnostic questionnaires may be used as part of assessing the extent and impact of use; for a comprehensive review, see the Guidelines for the Treatment of Alcohol Problems. AUDIT-C is the shortest questionnaire, with only 3 items. The interactive AUDIT questionnaire has 10 items but is quick and easy to administer.

If a questionnaire is not used, it can be useful to ask 'In a typical week, how many days might you drink?' and 'On an average/lighter/heavier drinking day, how many drinks might you have?'

Ask the patient to describe what happens when they drink and explore any negative consequences (eg accidents, falls, injuries). Allow them to tell their story candidly and listen intently; the 3 goals of history-taking are to gather information, formulate key questions (to help the person make their own connections between their drinking, patterns of relapse and harms) and to build a therapeutic relationship.

A diagnosis of **alcohol dependence** [\[Note 5\]](#) requires recurrent (episodic or continuous) drinking with 2 or more of the following features:

- impaired control—for example, having more than 4 drinks daily, drinking more often or more intensely, drinking in new contexts or continuing to drink despite previous admissions for withdrawal management
- ongoing drinking despite evidence of harms—for example, drink-driving charges, alcohol-related organ damage and damage to relationships, work or other important life activities
- physiological features—tolerance (a reduced effect from drinking a constant amount of alcohol), withdrawal symptoms, or use to prevent or alleviate withdrawal (eg early-morning drinking).

If a person is reported to have had a **seizure** associated with alcohol use, assess whether the event was:

- a memory blackout [\[Note 6\]](#)
- syncope
- an episode of excessive shaking
- a panic attack
- a witnessed convulsion with loss of consciousness (a seizure).

If a seizure is diagnosed, it is important to know whether it was generalised or focal and whether it occurred at a time of peak intoxication (suggesting alcohol poisoning) or when blood alcohol concentration was falling (suggesting alcohol withdrawal). Causes of generalised seizures include alcohol toxicity, alcohol withdrawal and generalised epilepsy. Focal seizures suggest an intracranial cause such as head trauma causing posttraumatic epilepsy. Seizures that occur or recur late in the alcohol withdrawal period are not typical of withdrawal seizures and require investigation for epilepsy. Many patients with epilepsy who drink heavily have difficulties adhering to their antiepileptic medication regimens. Distinguishing the cause of a seizure is important in determining management, including assessing the impact on fitness to drive.

Note 1: Even moderate consumption of alcohol increases the risk of cardiovascular harms such as hypertension, cardiomyopathy, atrial fibrillation, atrial flutter and stroke.

Note 2: Most people with alcohol dependence are likely to have had years of excessive use and some will have had years of treatment for dependence.

Note 3: Personality change correlates strongly with the development of alcohol-related brain damage. Loss of a high level of alcohol tolerance (ie no longer being able to drink large amounts of alcohol without intoxication) correlates strongly with the combination of brain damage and cirrhosis.

Note 4: While onset of alcohol use in adolescence is associated with a family history and poor outcomes in adulthood, onset in older patients may indicate a comorbidity such as severe depression or early dementia.

Note 5: For detail on terminology in substance use, including comparison of DSM-5 and ICD-11 classification systems, see [Table 22.1](#).

Note 6: Causes of acute alcohol-related memory loss include alcohol intoxication after which memory function returns; chronic alcohol-related memory impairment is most commonly caused by thiamine deficiency.

Examination and investigations in assessing alcohol use

Examination and investigations in assessing alcohol use

A **mental state examination** should exclude delirium and target conditions associated with alcohol use such as anxiety, mood disorders, suicide risk and cognitive impairment. For patients with cognitive impairment,

identify the pattern of cognitive decline, and ascertain if the main feature is memory loss (in keeping with [Korsakoff syndrome](#)) or global impairment (in keeping with Alzheimer or post-traumatic dementias). In patients who developed alcohol dependence later in life and have cognitive problems associated with behaviour changes, consider frontotemporal dementia. For advice on assessing cognitive impairment, see [Cognitive testing](#).

Full assessment may not be possible until after the patient has undergone alcohol withdrawal; mild to moderate cognitive impairment can improve with abstinence. Identification of psychosis or marked paranoia is essential in assessing a person's decision-making capacity and the risk of serious harm to them and others. For advice on informed consent and assessing capacity, see [Informed consent and shared decision-making in a person with a psychiatric disorder](#).

A **physical examination** is important to identify evidence of recent drinking (eg the smell of alcohol on the breath), intoxication or withdrawal, and signs of organic disease, particularly gastrointestinal and neurological disease. See the [Guidelines for the Treatment of Alcohol Problems](#) for common features of harmful alcohol use. Look for signs of urgent medical complications of alcohol use, such as head trauma [[Note 7](#)], aspiration, peritonism, gastrointestinal bleeding and [Wernicke encephalopathy](#) .

Investigations to consider in evaluating alcohol use are listed in [Table 22.8](#). Breathalyser and blood alcohol concentration tests are the only investigations that can assess if the patient has recently used alcohol; other tests are indicated to assess the impact of known alcohol use. Blood and urine tests are not recommended first-line to screen for alcohol dependence because questionnaires such as [AUDIT-C](#) are more sensitive and specific and less expensive. For more detail on the limitations of blood and urine tests in screening for alcohol use, see the [Guidelines for the Treatment of Alcohol Problems](#).

Table 22.8 Investigations to consider in disorders of alcohol use

Test	Indication
	confirmation of recent alcohol use
breathalyser or blood alcohol concentration [NB1]	assessment of blood alcohol concentration (eg for use in assessing suspected intoxication or to guide timing of benzodiazepine therapy to manage withdrawal)
	assessment of tolerance
serum urea and electrolyte concentrations (including calcium, magnesium and phosphate)	assessment of electrolyte replacement needs (particularly magnesium and potassium) in alcohol-dependent patients [NB2]
liver biochemistry	assessment for potential organ damage in hazardous, harmful and dependent alcohol use [NB3]
full blood examination	assessment before starting drugs that may affect liver function
coagulation screen	
Hepascore test	
iron studies	
serum lipid concentrations	assessment of acute and/or chronic liver disease if signs or symptoms are present
tests for viral hepatitis	
tests for concurrent nonviral liver disorders; consider conditions listed in the Liver Disorders Guidelines	
abdominal ultrasound scan	
urine drug screen	assessment for other substance use, particularly benzodiazepines or other drugs with sedative effects

Test	Indication
blood glucose concentration	assessment for hypoglycaemia (eg if alcohol intoxication or withdrawal is suspected) [NB4]
brain imaging	assessment for features of dementia (eg atrophy) if suspected on clinical grounds (eg a low Mini Mental State Examination score, late-onset alcohol dependence, or behavioural change in a patient aged 50 years or older); imaging helps determine if specialist referral is needed.
neuropsychological testing	

NB1: A breathalyser reading is preferred to a blood alcohol reading as results are more rapid than from a blood alcohol concentration measurement. Skilled use and calibration are required. Delayed gastric emptying can cause breathalyser readings to continue to rise after intake stops.

NB2: Serum magnesium concentration is likely to be low in patients with low weight or other signs of malnourishment, or those taking a proton pump inhibitor. Check serum electrolyte concentrations daily in these patients because there may be risk of refeeding syndrome.

NB3: If signs of chronic liver disease are present, investigate to assess the extent of organ damage and to identify contributing causes.

NB4: Hypoglycaemia is a differential diagnosis in both suspected alcohol intoxication and alcohol withdrawal. Hypoglycaemia can also be induced by alcohol intoxication.

Note 7: Scalp bruising and haematoma are associated with risk of subdural haemorrhage. Neck stiffness can result from meningitis or subarachnoid haemorrhage. Bleeding around the orbits (raccoon eyes), behind the eardrums or over the mastoid process suggests a base of skull fracture. Leakage of clear fluid from the nose could suggest a cerebrospinal fluid leak; this is likely if a nasal fluid dipstick test is positive for glucose. Bleeding from the nose can also be a sign of base of skull fracture.

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Symptoms and signs of alcohol withdrawal

Symptoms and signs of alcohol withdrawal

Symptoms and signs of acute alcohol withdrawal usually appear within 6 to 24 hours of the last drink, peak by about 48 to 72 hours, and wane over up to 5 days. Symptoms that persist beyond 7 days after the last drink are not likely to be caused by acute alcohol withdrawal; they are likely to reflect comorbidities, such as anxiety disorders, which may be precipitating factors for drinking. For an illustration of the progression of alcohol withdrawal over time, see the [Guidelines for the Treatment of Alcohol Problems](#).

Most alcohol withdrawal is mild and does not require the use of medication.

Most people have only **mild to moderate** symptoms and signs of alcohol withdrawal such as anxiety, insomnia, tremor, mild sweating, headache, nausea and vomiting.

More severe symptoms and signs of alcohol withdrawal include agitation, marked sweating, [seizures](#), perceptual disturbances, and [delirium](#). [Delirium tremens](#) is rare; a patient may use the term 'DTs' incorrectly to describe withdrawal tremors experienced on waking (without delirium). Recognising that these are different entities is important to avoid undue alarm when assessing the risk of severe withdrawal. Predictors of severe alcohol withdrawal are listed in [Figure 22.7](#). A history of severe alcohol withdrawal is typically a good clinical predictor of risk during a new episode of alcohol withdrawal.

Withdrawal tremor without confusion in a person with alcohol dependence is not a sign of delirium tremens.

Rarely, withdrawal can be life threatening if severe features go untreated.

Wernicke encephalopathy is an important comorbidity and differential diagnosis to consider in alcohol withdrawal; see [Thiamine supplementation and Wernicke-Korsakoff syndrome in alcohol dependence](#) for discussion of diagnosis, management and prevention.

Figure 22.7 Predictors of severe or complicated alcohol withdrawal

a history of [moderate to severe alcohol withdrawal](#), especially multiple bouts

current acute medical or surgical illness

current acute mental illness, such as anxiety [NB1]

severe comorbidity that might be destabilised (eg chronic liver disease, chronic obstructive pulmonary disease), seizure disorder or suspected lowered seizure threshold

history of memory blackouts (acute memory loss during alcohol intoxication)

cognitive impairment

early-morning drinking to alleviate withdrawal symptoms (anxiety, nausea, tremor)

use of more than one substance (polysubstance use), particularly benzodiazepines, but also opioids, stimulants

in older patients or those with polysubstance use, an intake of more than 8 standard drinks of alcohol per day [NB2]

NB1: Alcohol withdrawal in a person with an anxiety disorder causes escalation in anxiety levels. Many of the signs monitored on an alcohol withdrawal scale are also exacerbated by anxiety (tremor, tachycardia, sweating). The usual duration of the acute alcohol withdrawal phase is 2 to 3 days. Anxiety that persists longer is often misinterpreted as persisting acute withdrawal, leading to prolonged use of the alcohol withdrawal scale and excessive administration of diazepam.

NB2: In other patients, the feature of early-morning drinking to alleviate withdrawal is more relevant than the number of drinks per day.

Choice of setting for planned alcohol withdrawal

Choice of setting for planned alcohol withdrawal

The safety of the setting for planned alcohol withdrawal (at home, in a community facility or in hospital) depends on the likelihood of severe withdrawal and the extent of the person's social supports. Seek specialist advice on choice of setting from a [clinical advisory service](#) if a patient has any [predictors of severe or complicated alcohol withdrawal](#) or if unsure about assessing these factors.

Planned alcohol withdrawal can be undertaken in a **home setting** if the person has **no predictors of severe withdrawal** and all of the [requirements for withdrawal in a home setting](#) are met. In some metropolitan centres, **community-based inpatient withdrawal management units** manage patients with [predictors of severe withdrawal](#), although patients with multiple predictors may require hospitalisation. **Hospital management** is required if the person has an acute illness (medical, surgical or mental) or a deterioration during community treatment.

Overview of alcohol withdrawal management

Overview of alcohol withdrawal management

Alcohol withdrawal can be unplanned or planned.

Unplanned withdrawal occurs when a person is suddenly unable to drink alcohol for example, when admitted to hospital for an acute medical or surgical illness. The risk of complications is increased, particularly with delayed diagnosis of withdrawal. Concurrent conditions, particularly severe liver disease, are [considerations](#) that affect benzodiazepine prescribing for withdrawal.

Planned withdrawal can take place in a variety of settings; see [Choice of setting for planned alcohol withdrawal](#). Involuntary planned withdrawal as an inpatient under urgent medical guardianship (or specific legislation on involuntary treatment in some states) is an intervention of last resort for a patient with dependence that poses serious risk to their life.

Mild alcohol withdrawal generally responds well to supportive care (eg calm low-stimulus environment, fluids, multivitamins, reassurance and symptomatic management of nausea and headache) and can be managed without benzodiazepines in the home setting (see [Choice of setting for planned alcohol withdrawal](#)), with support from someone close to the person (who does not have a disorder of substance use) and review by the clinician. Review may be undertaken by phone to provide support and assess progress; unless urgent issues arise (eg the patient or their support person feels they are not coping), in-person review can be undertaken after a few days when symptoms have improved. [Psychological and behavioural interventions for insomnia](#) may also be helpful; advice includes a printable patient handout on good sleep practices [\[Note 1\]](#).

Benzodiazepines are not required for all patients undergoing alcohol withdrawal. Evidence supports early use of benzodiazepines for alcohol withdrawal for people at risk of severe symptoms and complications; see [Figure 22.7](#) for predictors of severe withdrawal. Benzodiazepines may also be used if mild to moderate withdrawal is predicted, because it can be difficult to predict severe withdrawal. Treatment goals of benzodiazepine prescribing for managing planned or unplanned alcohol withdrawal are to reduce symptom severity and risk of complications, and improve outcomes, such as rates of withdrawal completion.

Thiamine supplementation is recommended for all patients undergoing withdrawal treatment to reduce risk of peripheral neuropathy and Wernicke encephalopathy.

Improvement in mood and re-establishment of normal physiological patterns (such as circadian rhythm) after alcohol withdrawal can take weeks. Because this takes time, some people relapse to drinking soon after withdrawal. To mitigate this risk, close follow-up in the weeks following withdrawal is essential; this comprises daily contact (eg by phone) and a plan for how to escalate care if needed.

Note 1: For further information on home withdrawal management, such as the use of a patient contract, see Davis, C. Home detoxification supporting patients to overcome alcohol addiction. Australia Prescriber 2018;41:180–182 [URL](#).

Benzodiazepine treatment of alcohol withdrawal

Benzodiazepine treatment of alcohol withdrawal

Considerations before starting benzodiazepines for alcohol withdrawal

Considerations before starting benzodiazepines for alcohol withdrawal

For discussion of the role of benzodiazepines in managing alcohol withdrawal, see [Overview of alcohol withdrawal management](#). Before starting a benzodiazepine regimen for alcohol withdrawal, review local guidelines; specialist advice is available from [clinical advisory services](#) to discuss queries.

Benzodiazepine treatment is contraindicated in [hepatic encephalopathy](#) because it can cause serious exacerbation. Hepatic encephalopathy results from severe liver disease and typically causes sedation (sometimes coma), obscuring the presentation of acute alcohol withdrawal; specialist advice is recommended for any patient with hepatic encephalopathy.

Benzodiazepines reduce symptoms and complications in alcohol withdrawal but are contraindicated in patients with hepatic encephalopathy.

Diazepam is usually preferred for managing alcohol withdrawal because it has a long duration of action. It should be given early because withdrawal escalates during the first 24 to 48 hrs, and peaks by 48 to 72 hours. Diazepam is not recommended for patients with chronic liver disease (Child–Pugh class B or C); it has active metabolites that accumulate in these patients. Lorazepam or oxazepam, can be used instead for inpatients to reduce the risk of sedation or delirium; seek specialist advice.

Midazolam (by intravenous injection or infusion) is preferred if rapid but easily reversible sedation is required during hospital management of alcohol withdrawal (eg for a patient in an emergency department with recent seizure and with suspected head injury).

Fixed-schedule diazepam regimen for planned outpatient alcohol withdrawal

Fixed-schedule diazepam regimen for planned outpatient alcohol withdrawal

If [requirements for planned alcohol withdrawal in a home environment](#) are met and no [predictors of severe withdrawal](#) are present, a fixed-schedule diazepam regimen can be considered. Before prescribing, see advice in [Safe prescribing and supply in substance withdrawal in a home setting](#) on measures to reduce risk of dependence on medications, overdose and harm from sedation.

An example fixed-schedule diazepam regimen is:

diazepam orally

day 1: 10 mg 6-hourly

day 2: 10 mg 8-hourly

day 3: 10 mg 12-hourly

day 4: 5 mg 12-hourly

day 5: 5 mg at night, then stop.

Review patient progress daily to assess symptom control and avoid oversedation.

Diazepam regimens for inpatient alcohol withdrawal

Diazepam regimens for inpatient alcohol withdrawal

In a community residential withdrawal unit or a hospital alcohol and drug unit (where skilled monitoring is available), diazepam for alcohol withdrawal can be administered for planned or unplanned withdrawal in the following ways:

- symptom-triggered method
- loading-dose method (which is sometimes followed by a gradual tapering dose).

The decision about when to start dosing with any of these regimens is ideally guided by blood alcohol concentration (BAC) in conjunction with withdrawal scales. Generally, benzodiazepine dosing is not started until the BAC falls below 0.05% (approximately 10 mmol/L) and is consistently falling and symptoms of withdrawal are present.

The **symptom-triggered method** is usually used in patients presenting in mild to moderate withdrawal or who are planning withdrawal and have no risk factors for severe withdrawal. This method requires staff skilled in monitoring to use a validated alcohol withdrawal scale (eg the Clinical Institute of Alcohol Withdrawal Scale [CIWA-Ar], available at the [Insight website](#)). In most individuals, treatment is only required for 24 to 48 hours because symptoms peak during this period. Use of diazepam in this method after 48 hours risks accumulation, oversedation, confusion or delirium. If using the symptom-triggered method to manage alcohol withdrawal, a common example that may be used with a withdrawal scale by staff skilled in monitoring is:

diazepam 5 to 10 mg orally, 4- to 6-hourly as required to control withdrawal symptoms. Seek specialist advice if scores on the withdrawal scale escalate despite treatment or symptoms persist beyond 24 hours [\[Note 2\]](#).

The **loading-dose method** is recommended for inpatient care for patients who present in severe withdrawal or have risk factors for it, such as a history of complications (eg seizures). This technique reduces withdrawal severity and incidence of seizures if started early. Seizures start as early as 6 hours after the last drink; start diazepam as soon as clinical signs of withdrawal are evident, but not before (to avoid oversedation). If using the loading-dose method to manage alcohol withdrawal, staff skilled in monitoring may use:

diazepam 20 mg orally, 1- to 2-hourly as required to control withdrawal symptoms, up to a maximum of 60 mg [\[Note 3\]](#).

Severe alcohol withdrawal may require loading doses higher than 60 mg (eg doses up to 100 mg in 24 hours). Specialist advice is recommended in this situation because risks of respiratory depression and delirium, and potential need for transfer to an intensive care unit increase at this dose threshold.

Occasionally, a short taper is needed (eg over 2 to 3 days) after use of the loading-dose method. However, if the patient has concurrent benzodiazepine dependence, a longer taper is required; a common approach is to stabilise the patient on a reduced benzodiazepine dose in hospital, followed by a slow taper as an outpatient.

Note 2: Higher doses are sometimes used under specialist advice.

Note 3: Higher doses are sometimes used under specialist advice especially if the patient is already on

benzodiazepines for other indications.

Seizures in alcohol withdrawal

Seizures in alcohol withdrawal

People undergoing planned or unplanned alcohol withdrawal are at risk of seizures caused by the withdrawal state, and are also at risk of a comorbid seizure disorder (eg posttraumatic epilepsy caused by head injury).

For people already taking antiepileptic drugs for a seizure disorder, consider nonadherence when assessing any seizure during alcohol withdrawal. In all patients, consider whether medications that reduce seizure threshold should be withheld during the withdrawal period.

Alcohol-withdrawal seizures are usually:

- brief (not longer than 30 to 60 seconds)
- generalised
- seen in people with alcohol dependence with a long history of high alcohol intake
- evident early in withdrawal.

In a person known to be dependent on alcohol, a witnessed brief seizure is likely to be caused by withdrawal if it is followed by features of adrenergic activity (eg tachycardia, agitation, sweating). In contrast, after a seizure, a person with epilepsy is usually calm, sleepy or drowsy, has normal vital signs and is not sweating. This distinction is useful to decide the extent of investigations, need for antiepileptics and duration of monitoring after a seizure.

Tachycardia, sweating and agitation after a seizure support a diagnosis of a withdrawal seizure; a calm, drowsy postictal patient is likely to have a comorbid seizure disorder.

Seizures in alcohol withdrawal can occur before the blood alcohol concentration falls to zero; they can recur in 10 to 20% patients within 6 to 12 hours. After one withdrawal seizure, the likelihood of another in future episodes of alcohol withdrawal is up to 50%. In patients with [risk factors for severe withdrawal](#) prophylactic benzodiazepines using the loading-dose method very early during [inpatient withdrawal](#) substantially reduce the risk for alcohol-withdrawal seizures. The use of antiepileptic drugs before or after alcohol-withdrawal seizures has not been shown to be effective; in contrast, antiepileptics are indicated if the person has an established comorbid seizure disorder.

Antiepileptics are indicated for comorbid seizure disorders in patients undergoing alcohol withdrawal but not to treat or prevent alcohol-withdrawal seizures.

Outcomes following a withdrawal seizure include [delirium](#) (more likely in people with alcohol dependence and acquired brain injury) and [status epilepticus](#) (more likely in people who are seizure-prone for other reasons). Exclude differential diagnoses such as head injury, subdural haematoma, central nervous system infection or other pathology and consider the need for neurological consultation. This approach is required for a first episode of seizure, patients with delirium after a seizure and patients known to have seizures whose presentation differs from their usual pattern. Free specialist advice is available from [clinical advisory services](#) around the country.

Specialist advice is available by phone on any aspect of the management of substance use; contact is encouraged.

Delirium in alcohol withdrawal

Delirium in alcohol withdrawal

Most delirium in hospitalised patients is multifactorial; see [Risk factors for delirium in adults](#). In patients with alcohol dependence, common risk factors for delirium are acquired brain injury, cognitive impairment

including dementia, and acute medical or surgical illness. Intoxication should be excluded. Delirium tremens is rarely seen, probably because of the widespread use of benzodiazepine treatment to manage alcohol withdrawal.

In assessing delirium in alcohol withdrawal, consider precipitating factors particularly infection, anaemia, head injury, electrolyte disturbances and metabolic disturbances such as hypoglycaemia, Hepatic encephalopathy and Wernicke encephalopathy. Some of these disturbances may be part of refeeding syndrome; refer patients to a dietitian to assist with refeeding.

Seek specialist review for all patients with delirium during alcohol withdrawal because risk of complications is high.

Hyperactive delirium can complicate delirium of any cause; it poses a serious risk for falls, absconding, or harm from the use of physical restraints. Use local hospital guidelines and seek specialist advice for all patients with delirium during alcohol withdrawal, because they are at high risk of complications. For general advice on managing delirium, see Principles of managing delirium. Drug therapy is preferred to physical restraint. Benzodiazepines are not a treatment for delirium (other than for delirium tremens) and will only exacerbate it. Antipsychotics are the drugs of choice in managing hyperactive delirium.

Benzodiazepines are not a treatment for delirium; they will exacerbate it.

Evaluate bleeding risk before giving any intramuscular injection; haemorrhage has occurred after intramuscular injection in patients with unrecognised severe thrombocytopenia or coagulopathy associated with liver disease.

Delirium tremens

Delirium tremens

Delirium tremens is sometimes referred to as ‘alcohol withdrawal delirium’; this can be confusing because delirium tremens is rare; many other causes of delirium are more common in a patient withdrawing from alcohol. Delirium tremens (also known as the ‘DTs’) is the most severe manifestation of alcohol withdrawal and is characterised by gross generalised tremulousness, sweating, fluctuating levels of agitation, hallucinations (usually tactile), disorientation and impaired attention [[Note 4](#)]. Low-grade fever, tachycardia and dehydration may also be present.

Delirium tremens is a medical emergency that always requires hospitalisation and, if inadequately treated, carries mortality risk, mainly from heart failure.

Delirium tremens is usually identified after 48 to 72 hours of more severe alcohol withdrawal in the setting of recent acute illness or surgery. It usually occurs if withdrawal has been:

- unrecognised (possibly ‘masked’ by other medications such as short-acting hypnotics)
- delayed by recent general anaesthesia
- undertreated.

The differential diagnosis of delirium tremens is hyperactive delirium of mixed aetiology.

Patients with delirium tremens need specialist treatment, and often need admission to an intensive care unit.

Delirium tremens is a medical emergency requiring specialist treatment and often intensive care unit admission.

Note 4: A patient may use the term ‘DTs’ incorrectly to describe withdrawal tremors experienced on waking (without delirium). Recognising that these are different entities is important to avoid undue alarm when assessing the risk of severe withdrawal.

References



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Gradual reduction of alcohol use

Gradual reduction of alcohol use

Gradual reduction of alcohol use may be a more realistic goal than abstinence for some patients; people with harmful or dependent use should also be offered psychosocial supports. For people with alcohol dependence, long-term management strategies are also indicated.

After reduction goals are agreed, the patient monitors their alcohol intake, circumstances of drinking and mood (eg with a drink diary or an app such as Daybreak), and uses techniques such as spacing drinks (interspersing with glasses of water) or eating while drinking. The clinician reviews the diary with the person regularly to identify high-risk situations for heavy drinking (eg pub sessions, stressful events) and opportunities to reduce drinking.

Other approaches, as outlined in long-term management can be combined with gradual reduction, particularly cognitive behavioural therapy to learn skills in drink refusal and managing urges. Any reduction in alcohol use is worthwhile.

Any reduction in drinking is worthwhile in the management of disorders of alcohol use.

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Overview of harm reduction in alcohol use

Overview of harm reduction in alcohol use

Offer harm reduction measures to everyone with a disorder of alcohol use. Measures to consider in any disorder of substance use are outlined in Figure 22.2. Issues specific to alcohol use include:

- thiamine supplementation
- managing organ damage—see Management of complications of alcohol use.

Table 22.5 is a printable patient information sheet on ways to get help and reduce harm from use of alcohol and other drugs.

In some states and territories, sobering-up centres (to supervise people who are intoxicated) are an additional harm reduction measure. Managed alcohol programs are being trialled; they provide regulated amounts of alcohol in a hostel setting to people with severe alcohol dependence who are homeless.

A nonjudgemental approach to harm reduction can help to maintain a therapeutic relationship and promote opportunities to reconsider gradual reduction of alcohol use, short-term interventions for withdrawal management and long-term management of alcohol dependence.

Thiamine supplementation and Wernicke-Korsakoff syndrome in alcohol dependence

Thiamine supplementation and Wernicke-Korsakoff syndrome in alcohol dependence

Thiamine deficiency occurs in alcohol dependence and other conditions that impair nutrition and absorption; clinical effects are listed in here Wernicke–Korsakoff syndrome has a spectrum of neurological features caused by thiamine deficiency, most commonly seen in alcohol dependence, but other causes include bariatric surgery, gastrointestinal cancer, hyperemesis gravidarum, poor diet and chronic vomiting. Wernicke encephalopathy is a severe manifestation of acute deficiency that requires immediate treatment with intravenous thiamine; without adequate treatment, brain damage can lead to severe loss of short-term memory and difficulty learning new information (Korsakoff syndrome). Korsakoff syndrome is sometimes known as alcohol amnestic disorder, although it can also evolve from nonalcohol-related Wernicke encephalopathy.

Wernicke encephalopathy in alcohol dependence

Wernicke encephalopathy in alcohol dependence

Wernicke encephalopathy is triggered by ingestion of carbohydrate (orally or intravenously) in patients with thiamine deficiency before thiamine has been adequately supplemented [Note 1]. In severe cases, brain injury may occur; brain magnetic resonance imaging (MRI) may identify oedema of the mammillary bodies, and sometimes bleeding in specific areas. Persisting brain damage can result in Korsakoff syndrome and, very rarely, death.

Wernicke encephalopathy should be suspected if any of the following features are present:

- delirium (approximately 80% of patients)
- ataxia (approximately 20 to 25% of patients)

- eye signs (nystagmus or ophthalmoplegia) (approximately 30% of patients).

Wernicke encephalopathy can occur independently or with other causes of delirium, including alcohol intoxication, withdrawal and hepatic encephalopathy.

Prompt treatment of Wernicke encephalopathy reduces the likelihood of permanent neurological damage. Give high doses of intravenous or intramuscular thiamine promptly if any clinical signs are present; see the Gastrointestinal guidelines for dosages. Exclude coagulopathy and severe thrombocytopenia before using the intramuscular route. Oral thiamine supplementation is not adequate in Wernicke encephalopathy.

Thiamine requires magnesium as a cofactor; even high thiamine doses may not have optimal effect if a patient has untreated hypomagnesaemia. See Identifying adults at risk of refeeding syndrome for a list of patients at high risk of electrolyte abnormalities (including hypomagnesaemia). Some patients may not be visibly wasted but may have low serum magnesium concentrations from use of medications such as proton pump inhibitors. Check serum electrolyte concentrations (calcium, magnesium and phosphate) or administer preventive magnesium concurrently with thiamine; see Hypomagnesaemia for intravenous regimens. Concurrent treatment of hypokalaemia is also important.

Treat suspected Wernicke encephalopathy promptly with intravenous or intramuscular thiamine; treat hypomagnesaemia and hypokalaemia concurrently.

Note 1: Thiamine is essential for carbohydrate metabolism; if thiamine is deficient, neurones exposed to carbohydrate experience metabolic injury.

Preventive thiamine supplementation in alcohol dependence

Preventive thiamine supplementation in alcohol dependence

Preventive thiamine supplementation is important for all patients with alcohol dependence and malnourishment to limit development of peripheral neuropathy and risk of Wernicke encephalopathy. Supplementation is also recommended for all patients undergoing alcohol withdrawal treatment even if their nutritional status and dietary intake are good; they may still lack micronutrients.

Preventive thiamine supplementation must be started before giving any carbohydrate load; the metabolism of carbohydrate (including intravenous glucose) can deplete thiamine further and precipitate Wernicke encephalopathy.

Intravenous or intramuscular thiamine is required initially for people with poor nutritional status or dietary intake. Doses are not as high as those required to treat existing Wernicke encephalopathy. Exclude coagulopathy and severe thrombocytopenia before using the intramuscular route. Oral supplementation is usually adequate after the first few days of parenteral treatment, unless the patient has no oral intake (eg patients with acute pancreatitis, preoperatively).

Oral supplementation is usually sufficient for patients who have good nutritional status and good oral intake.

For thiamine dosages, see recommendations for adults at high risk of thiamine deficiency.

Prevent Wernicke encephalopathy by replacing thiamine adequately in alcohol-dependent patients before oral or intravenous carbohydrate intake.

References

References: Harm reduction in alcohol use

- Haber PS, Riordan BC. Guidelines for the Treatment of Alcohol Problems. 4th ed. Sydney: Specialty of Addiction Medicine, Faculty of Medicine and Health, The University of Sydney; 2021.
<https://alcoholtreatmentguidelines.com.au>
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Overview of long-term management of alcohol dependence

Overview of long-term management of alcohol dependence

Long-term management of alcohol dependence involves regular review of the patient to maintain a therapeutic relationship, assess their physical, mental and social wellbeing, reconsider treatment choices and manage complications of use, as outlined in the [Overview of substance use](#). Modest improvements in treatment outcomes can be achieved by case management compared to usual care.

Many aspects of long-term management of alcohol dependence involve cognitive behavioural therapy, which is one of the most evidence-based approaches to relapse prevention. Patients learn about triggers of relapse, develop strategies to reduce risk of recurrence and shorten episodes. However, cognitive impairment affects most patients with alcohol dependence and can limit the learning of new skills, such as managing cravings, drink refusal skills and setting limits.

Mild to moderate cognitive impairment can improve with abstinence, improved nutrition and social engagement. Progressive deterioration in patients with more severe impairment (particularly dementia) may require an assessment of the patient's [mental capacity](#), usually after planned withdrawal. Some may require specific intervention, such as guardianship or placement in a suitable institution. Prognosis is worse for those with serious comorbidity (such as dementia or liver failure).

Supporting [gradual reduction of alcohol use](#) can be more effective than recommending abstinence as the only appropriate goal.

Abstinence is recommended for patients wishing to achieve this and for those with serious alcohol-related organ damage. Alcoholics Anonymous (AA) has substantial supporting evidence including a systemic review [\[Note 1\]](#) for its role in helping patients achieve abstinence. While patients self-refer to AA, practitioners can encourage this process by recommending the patient try at least 3 meetings in 3 different locations (because not all AA groups are the same). Attendance is anonymous; self-report of attendance, and monitoring of clinical progress and markers of alcohol use [\[Note 2\]](#) can assess the extent of a patient's engagement with AA. [Smart Recovery](#) groups are an alternative form of peer support with groups led by facilitators, although these are not free. [Moderation Management](#) is also an option. [Sober in the Country](#) provides peer support to people in rural areas.

Some drugs increase the duration of abstinence but have not been shown to promote moderation of drinking. All are best commenced after alcohol withdrawal, with strategies that enhance adherence (eg dose supervision at home) and should be provided together with [psychosocial interventions](#). Three drugs with different modes of action have most evidence for efficacy: naltrexone, acamprosate and disulfiram. The choice of drug needs to be individualised:

- [Naltrexone](#) is the most popular choice because it has once-daily dosing, but it is not suitable for patients with liver failure or acute hepatitis, or who are taking long-term opioid therapy.
- [Acamprosate](#) has less convenient dosing (3 times per day), so is often preferred for patients with significant liver impairment (which precludes other options). Patients require support to help adherence to the dosing regimen.
- [Disulfiram](#) is used infrequently, possibly because of the (intended) aversive effects if the person drinks while taking it. It is accessed via the [Special Access Scheme](#), and the prescriber and patient must understand its effects.

Note 1: Kelly JF, Humphreys K, Ferri M. Alcoholics Anonymous and other 12-step programs for alcohol use disorder. Cochrane Database of Systematic Reviews 2020;3:CD012880.

<https://www.ncbi.nlm.nih.gov/pubmed/32159228>

Note 2: Laboratory markers might include serum gamma glutamyl transferase (if it has previously correlated with their drinking patterns), breathalyser readings, urine screens for alcohol, mean corpuscular volume, ferritin. Clinical markers can include alcohol-induced flaring of psoriasis and eczema.

Naltrexone for long-term management of alcohol dependence

Naltrexone for long-term management of alcohol dependence

Naltrexone is an opioid antagonist; it is similar to naloxone but longer-acting and taken orally. Because naltrexone is taken once daily, it is often preferred for convenience as a first-line agent for long-term management of alcohol dependence. However, it is contraindicated in patients who require opioid therapy. Naltrexone blocks the effect of opioid analgesics and can cause opioid withdrawal. It is also contraindicated in patients with liver failure or acute hepatitis.

Naltrexone blocks endogenous opioid-mediated release of dopamine that activates a reward stimulus. In people with alcohol dependence, naltrexone attenuates the desire (craving) to drink and the pleasure response related to drinking; naltrexone reduces rates of relapse to heavy drinking and increases the number of abstinence days in these patients. It has no effect in patients with minimal or moderate alcohol intake. Alcohol-induced impairment is not affected by naltrexone.

Naltrexone is sometimes prescribed (with specialist advice) for use before an episode of drinking to reduce consumption, although it is not approved for this indication. When used in this situation, naltrexone is only taken pre-emptively (eg before entering a high-risk situation such as pub drinking) rather than every day.

Because naltrexone can cause liver toxicity, it is important to assess liver biochemistry before starting treatment, then monthly for the first 3 months, then (if normal) every 3 months.

Naltrexone may cause nausea, but a gradual dose increase and night-time dosing can reduce this.

If naltrexone is considered appropriate for long-term management of alcohol dependence, start after withdrawal symptoms have resolved (usually 3 to 7 days after the last drink); use:

naltrexone 25 mg orally, daily at bedtime; if tolerated, increase after 5 days to 50 mg daily.

Treatment is often continued for 3 to 6 months (less commonly 12 months) but duration should be determined individually, based on factors such as adverse effects, history of relapse and the patient's social situation.

Acamprosate for long-term management of alcohol dependence

Acamprosate for long-term management of alcohol dependence

Acamprosate reduces neuronal hyperexcitability, has a mildly anxiolytic effect, and attenuates the desire to drink. While not an effective treatment for acute alcohol withdrawal, acamprosate may reduce some post-acute withdrawal symptoms, such as anxiety, irritability and cravings. It increases the time to first drink, prolongs abstinence, and reduces the number of drinking days. Acamprosate combined with psychosocial treatment significantly improves treatment outcomes compared to psychosocial treatment alone.

Acamprosate is renally cleared; seek specialist advice before prescribing for patients with kidney impairment. Liver disease is generally not a limitation because acamprosate has minimal hepatic metabolism.

Acamprosate must be taken 3 times a day to maintain effect because of rapid renal clearance. Frequent dosing, together with the inconvenience of taking 2 large tablets at each dose, makes acamprosate less popular; it is generally only used as a first choice in patients whose options are limited by liver disease.

If acamprosate is considered appropriate for long-term management of alcohol dependence, start after withdrawal symptoms have resolved (usually 3 to 7 days after the last drink); use:

acamprosate

patient less than 60 kg: 666 mg orally, in the morning, 333 mg at midday and 333 mg at night

patient 60 kg or more: 666 mg orally, 3 times daily.

Treatment is usually for 6 to 12 months, occasionally longer.

Naltrexone plus acamprosate for long-term management of alcohol dependence

Naltrexone plus acamprosate for long-term management of alcohol dependence

Evidence of benefit is insufficient to recommend a combination of naltrexone and acamprosate as a standard treatment in preventing relapse, although evidence from rates of hospital admissions supports it being safe. Consider prescribing a trial of the combination when documented treatment with each drug alone has not achieved the desired result.

Disulfiram for long-term management of alcohol dependence

Disulfiram for long-term management of alcohol dependence

Disulfiram is a behavioural treatment used to deter patients from drinking by causing vomiting, facial flushing, pounding headache, palpitations, and a risk of elevated blood pressure if they drink (the ethanol–disulfiram reaction). This reaction also occurs if the patient is exposed to any form of alcohol (including aftershaves, antiseptics, mouthwashes) while taking disulfiram. Adherence needs to be enhanced by regular dose supervision (typically by someone close to the patient).

Disulfiram may not be effective in people unconcerned about the prospect of vomiting; effectiveness may also be reduced by regular use of antiemetics or other drugs with antiemetic effects (eg some antipsychotics). Contraindications include coronary heart disease, angina, hypertension, significant cognitive impairment or dementia [\[Note 3\]](#), suicidal ideation and psychosis.

Disulfiram is best started before discharge, after planned withdrawal in a hospital or community facility and discussion with a specialist. Dose titration may be needed, and there are a number of precautions and drug–drug interactions to consider. Liver biochemistry is recommended because disulfiram can be hepatotoxic. A breathalyser reading to confirm no recent alcohol use is also advised before starting.

Note 3: If a person has some cognitive difficulties but has capacity to consent to disulfiram treatment and a support person (eg partner) to supervise dosing, it may be appropriate to offer them a trial of disulfiram.

Other drugs and novel treatments for long-term management of alcohol dependence

Other drugs and novel treatments for long-term management of alcohol dependence

Drugs that may be considered by specialists when first-line drugs have been unsuccessful for treating alcohol dependence include baclofen, gabapentin, topiramate and varenicline. These drugs are not approved for this indication, have insufficient evidence to support use as first-line treatments and some (eg baclofen, gabapentin) have a risk of dependence.



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Overview of management of disorders of alcohol use in specific populations

Overview of management of disorders of alcohol use in specific populations

Specific considerations (including the need for specialist referral) apply in the [management of alcohol use during pregnancy](#) and [management of alcohol use during breastfeeding](#). For advice in other groups, see [considerations in other specific populations](#).

Management of alcohol use during pregnancy

Management of alcohol use during pregnancy

Heavy drinking before pregnancy may be associated with increased risk of impaired nutrition; micronutrient deficiencies (in particular, thiamine, folate and iron) may have an adverse effect on the pregnancy. Offer patients who use alcohol options for [contraception](#). For patients who are planning pregnancy, preventive micronutrient supplementation and planning for abstinence are recommended. For information on supplements, see [Folate supplementation for pregnant people](#), [Thiamine supplementation](#) and [oral iron supplementation](#).

Approximately 10 to 15% of people continue to consume alcohol during pregnancy. Many people who continue drinking during pregnancy have a history of a parent drinking while pregnant; this history may reduce a patient's perception of risk.

Any alcohol consumption during pregnancy is strongly discouraged because there is no known safe amount. Alcohol has potential adverse effects on the embryo, starting early in the first trimester, often at a time when a person is unaware of their pregnancy. The harm from alcohol does not appear to be confined to first trimester alcohol consumption, and it is possible that the pattern of drinking may be more important than the average number of drinks per week. Learning about the potential adverse effects of alcohol on a fetus can heighten anxiety and exacerbate drinking in pregnancy. Careful counselling is helpful for many, but some patients require specialist counselling about this risk. Specialist alcohol and other drug services to support pregnant patients are listed by state and territory on the [every moment matters website](#).

Refer pregnant patients with a disorder of alcohol use as early as possible to a specialist antenatal service with drug and alcohol support.

Alcohol withdrawal can be associated with increased risk for spontaneous abortion in the first trimester and premature labour in the third trimester. Planned alcohol withdrawal should be timed for the second trimester, preferably with support from a specialist alcohol and drug treatment service. After withdrawal and subsequent improvement in cognitive functioning, a decision about continuing the pregnancy may change. Referral to a specialist obstetric service with access to perinatal psychiatry is recommended to support decision-making.

Alcohol withdrawal management should be followed by counselling to support ongoing abstinence. The safety during pregnancy of drugs used to reduce alcohol cravings (eg naltrexone, acamprosate) has not been established. The safety of disulfiram is also unknown; while there are case reports of normal pregnancies, there are also isolated reports of congenital malformations including a case of limb reductions.

Infants with significant alcohol exposure *in utero* can develop a neonatal abstinence syndrome; this is characterised by irritability, poor feeding and impaired bonding, and may be associated with impaired

engagement in breastfeeding. Neonatal abstinence syndrome should be managed by a specialist neonatologist or in consultation with them.

Fetal Alcohol Spectrum Disorder (FASD) affects a substantial proportion of infants with significant alcohol exposure *in utero*. While some of these infants might be identified early by a paediatrician, many are not diagnosed in childhood and some may not receive a specific diagnosis during their lifetime. For information on diagnosis and management, see Fetal alcohol spectrum disorder.

Tobacco smoking is frequently associated with heavy drinking; this highlights the importance of providing support to a person to stop smoking and drinking in pregnancy. See Management of tobacco smoking in pregnancy.

Patient information on alcohol and pregnancy is available at the MotherSafe website.

Intervention for alcohol, smoking and other drug use is a priority during the postnatal period. Some patients may have already made significant changes during pregnancy that require consolidation, while others may still be precontemplative. Every effort should be made to capitalise on a person's motivation to engage with treatment during this time and thus provide the safest environment for their child.

Management of alcohol use during breastfeeding

Management of alcohol use during breastfeeding

Alcohol consumption during breastfeeding is often a continuation of consumption during pregnancy, making it difficult to separate adverse effects on the infant from each period. Confounders such as smoking also make interpretation difficult.

Alcohol use in breastfeeding has been associated with delayed let-down reflex for lactation and reduced breastmilk supply, probably related to inhibition of oxytocin release.

An infant's liver maturation is not complete until approximately 3 months; reduced capacity to metabolise alcohol could lead to accumulation. Excessive drowsiness and irritability have been described in infants exposed to alcohol in breastmilk. Both animal and human studies suggest potential adverse long-term effects in offspring. Significantly poorer development of motor skills has been reported in infants breastfed by individuals who consumed one standard drink per day. It is not clear whether exposure to alcohol in breastmilk leads to an earlier start to drinking.

The level of alcohol consumption during breastfeeding that is safe for an infant has not been defined. Consider the relative harms and benefits of breastfeeding for patients who regularly consume alcohol. The safety of prescribing drugs to reduce alcohol cravings (eg naltrexone, acamprosate) has not been established in breastfeeding patients. There are no reports of disulfiram use while breastfeeding; the potential effects on the infant are unknown. Educational and behavioural interventions should be the mainstay of treatment; specialist advice and engagement is recommended.

Specialist advice is recommended to guide decisions on whether to prescribe drugs to reduce alcohol cravings for patients who are breastfeeding.

Patient information on harm reduction measures while breastfeeding (such as timing of drinks and expressing milk) is available at the MotherSafe website. The FeedSafe app helps a patient plan safe timing for breastfeeding after drinking alcohol.

References

References: Management of disorders of alcohol use in specific populations

- Giglia R. Alcohol and lactation: An updated systematic review. *Nutrition & Dietetics* 2010;67(4):237-43. <https://onlinelibrary.wiley.com/doi/abs/10.1111/j.1747-0080.2010.01469.x>


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What is covered in this topic?

What is covered in this topic?

This topic describes the management of harms from:

- illicit opioid use, such as heroin
- nonmedical use of prescription opioids, such as oxycodone, codeine, fentanyl, tramadol, morphine and hydromorphone.

Nonmedical use of opioids describes use of prescription drugs in ways that do not align with the directed use; for example, use in order to become intoxicated or to treat a symptom other than the clinician intended.

The spectrum of substance use is described in these guidelines by the terms 'hazardous use', 'harmful use' and 'substance dependence', outlined in [Table 22.1](#).

Toxicity (overdose) from opioids is covered in the Toxicology and Toxinology Guidelines; see:

- [Opioid poisoning: advice for first responders in the community or primary healthcare setting](#)
- [Opioid poisoning: general management](#)
- [Buprenorphine poisoning](#)
- [Novel psychoactive substance poisoning](#), including novel synthetic opioids (eg fentanyl analogues), which are associated with an increased risk of overdose because of their high potency.

Heroin was once a leading source of opioid harm but has been supplanted by prescription opioids. Prescription opioids can be used by injection, orally or transdermally. Illicit opioids may be used by a range of routes; heroin is used intravenously, intranasally ('snorted') or by vapour inhalation ('chasing the dragon').

Prescription opioid use has increased over 10-fold in the last 2 decades in Australia; currently over one million Australians are prescribed repeat opioid prescriptions annually. In 2016, deaths and hospitalisations from prescription opioids exceeded those from illegal opioids in Australia. Some of these events are the result of nonmedical use (the focus of this topic), but [harms](#), including dependence, can also arise from use as prescribed, particularly if doses are high or prolonged (eg in some [patients with chronic pain](#)).

Screening for and assessment of opioid use

Screening for and assessment of opioid use

Ask all new patients routinely and others (adolescents and adults) opportunistically and periodically about prescribed and illicit opioid use, as part of a general screen for disorders of substance use and gambling; these disorders are common and often co-exist. Patients may be unaware that their use, particularly of prescribed opioids, is harmful. Others are reluctant to disclose disorders of use or addictive behaviour, often due to fear of stigma. [Screening and assessment of substance use and addictive behaviours](#) outlines history-taking (including use of the [ASSIST-Lite tool](#)), examination, and investigations that should be considered in a broad review of substance use and addictive behaviour.

Diagnostic questionnaires may be used as part of assessing the extent and impact of use. The [OWLS tool](#) can be considered to assess prescription opioid use; for illicit opioids, the [Severity of Substance Dependence tool](#), can be used.

Table 22.9 The OWLS screening for a disorder of prescribed opioid use

Question	Response	Score
	Not at all	0
	A little	1
	Quite a lot	1
	A great deal	1
	Not at all	0
	A little	1
	Quite a lot	1
	A great deal	1
	Not at all	0
	A little	1
	Quite a lot	1
	A great deal	1
	Not at all	0
	A little	1
	Quite a lot	1
	A great deal	1

Total score [NB1]

NB1: A score of 3 or more indicates the person is likely to meet criteria for a disorder of opioid use; further assessment is warranted.

Reproduced from Picco L, Middleton M, Bruno R, Kowalski M and Nielsen, S. Validation of the OWLS, a Screening Tool for Measuring Prescription Opioid Use Disorder in Primary Care. *Pain Medicine* 2020; 21(11):2757-2764 by permission of Oxford University Press. [URL](#).

After initial screening, assess the extent and impact of opioid use, by determining:

- how much, how often and by what route opioids are used [\[Note 1\]](#)
- patterns of use, including any past efforts to reduce
- withdrawal symptoms (current and past)
- [potential harms](#), including self-harm, accidental overdose, impacts on relationships, work and child protection concerns.

Opioid withdrawal [symptoms and signs](#) are likened to a 'bad case of the flu and gastro together'. Symptoms usually peak at around day 2 to day 3 and are largely resolved after 5 to 7 days. Subjective symptoms of opioid withdrawal are more sensitive indicators of a need for medical intervention than objective signs, which

may be modest. For additional details, see the Subjective Opiate Withdrawal Scale and the Clinical Opiate Withdrawal Scale at the [National Centre for Education and Training on Addiction website](#).

Assessment of the extent and impact of use can categorise opioid use as [hazardous](#), [harmful](#) or [dependent](#). Advice from or referral to pain or addiction specialists is recommended for assessment of complex issues such as [substance use in chronic pain](#).

Table 22.10 Symptoms and signs of opioid withdrawal

Symptoms [NB1]

agitation, anxiety

insomnia

sweating (diaphoresis)

musculoskeletal pain

abdominal cramps

diarrhoea

nausea

vomiting

hot flushes, cold flushes

cravings to use opioids

Signs [NB1]

elevated heart rate

enlarged pupils (mydriasis)

teary eyes (epiphora)

runny nose (rhinorrhoea)

sweating

tremor

restlessness

yawning

‘gooseflesh’ (piloerection)

NB1: Symptoms are more sensitive indicators of opioid withdrawal than signs.

Note 1: Although evaluating dose and frequency of opioid use are part of the assessment of whether use is controlled, they are not criteria in their own rights for determining [opioid dependence](#) and they do not determine dosing requirement in [medication-assisted treatment of opioid dependence](#).

Overview of management of disorders of opioid use

Overview of management of disorders of opioid use

[Overview of substance use and addictive behaviours](#) explains key [principles of care](#) for a person with a disorder of substance use. Establishing a therapeutic relationship that engages the person (and ideally those close to them) is central to the management of substance dependence.

The most important element of treatment for substance use is a therapeutic relationship.

Specialist advice on any aspect of care for people with opioid and other substance dependence is available and contact is encouraged; see [Contact details for substance use clinical advisory services for clinicians](#).

Specialist advice is available by phone on any aspect of the management of substance use; contact is encouraged.

The treatment of disorders of opioid use involves one or more of the following:

- prevention of [opioid overdose](#)
- harm reduction strategies including prevention of overdose
- management of dependence with [medication-assisted treatment of opioid dependence](#) (MATOD)
- [planned opioid withdrawal](#)
- weaning of prescribed opioids—see [Chronic pain in substance use and addictive behaviours](#)
- [psychosocial support](#)
- [long-term care](#).

[Harm reduction in substance use and addictive behaviours](#) is relevant for all patients; see [Table 22.5](#) for a printable patient information sheet. Measures to reduce opioid harms include the use of safer-injecting facilities, needle and syringe programs, screening for bloodborne viruses (including hepatitis B, hepatitis C and HIV), vaccination against hepatitis B, and provision of take-home naloxone to reduce risk of death from [opioid overdose](#). [Ensuring the safety of a person with a disorder of substance use or addictive behaviour](#) gives advice on managing behavioural disturbance, assessing fitness to drive, and occupational implications of substance use.

[MATOD therapy](#) is the mainstay of managing opioid dependence because it is the most effective treatment; it is much more successful than [planned withdrawal](#). Evidence of benefit is lacking for psychological interventions as standalone treatments of opioid dependence.

MATOD is the most effective treatment for opioid dependence.

Some patients (eg some using prescription opioids) may not be willing to consider MATOD therapy or it may not be clinically appropriate; consultation with a [clinical advisory service](#) is recommended for managing [substance use in patients with chronic pain](#). Long-term tapered [weaning \(deprescribing\)](#) of opioids may be an appropriate choice before considering MATOD therapy, provided the prescriber has sought specialist advice and reviewed the [legal considerations for prescribing in substance use](#).

[Long-term care](#) offers all patients an ongoing therapeutic relationship and broad consideration of their physical and mental health needs, including relapse prevention for those who achieve abstinence.

Certain populations may benefit from [specific considerations in the management of substance use](#). Advice specific to [management of opioid dependence during pregnancy](#), and [management of opioid dependence during breastfeeding](#) is included in this topic.

Opioid overdose

Opioid overdose

Opioid overdose is characterised by sedation, loss of consciousness, miosis (small pupils), shallow breathing, respiratory depression and cyanosis. Pulmonary oedema may also occur. Opioid overdose can occur with use of opioids by any route and in any formulation (including slow-release preparations). Risk of death is particularly increased in people who use an opioid together with another drug with sedative effects (eg [pregabalin](#) or [gabapentin](#), [alcohol](#), [benzodiazepines](#)), and in those who use novel opioids such as fentanyl analogues.

For advice on the management of opioid overdose (also referred to as opioid poisoning), see the following topics in the Toxicology and Toxinology Guidelines:

- [Opioid poisoning: advice for first responders in the community or primary healthcare setting](#)
- [Opioid poisoning: general management](#)
- [Buprenorphine poisoning](#).

For advice on overdose with novel psychoactive substances including novel opioids, see [Introduction to novel psychoactive substance poisoning](#).

To reduce risk of death from overdose, discuss **take-home naloxone** with all patients with [hazardous, harmful or dependent use](#) of opioids (and those close to them because they are likely to be the people administering the drug). Naloxone is a mu-receptor antagonist for immediate use in opioid overdose. Take-home naloxone is available as a nasal spray or preprepared injection. It can be obtained free of charge and over-the-counter through the [Take Home Naloxone program](#) in all Australian states and territories. Higher doses of naloxone may be required if the overdose involves a drug with a high affinity for the mu receptor, such as novel fentanyl analogues or buprenorphine. For more information on naloxone for overdose and take-home naloxone, see the [Pain and Analgesia guidelines](#).

Take-home naloxone is lifesaving and should be made available to all patients with hazardous, harmful or dependent use of opioids.

Overview of medication-assisted treatment of opioid dependence (MATOD)

Overview of medication-assisted treatment of opioid dependence (MATOD)

Medication-assisted treatment of opioid dependence (MATOD) is also known as chronic opioid therapy (COT), medication for opioid use disorder (MOUD), opioid agonist therapy (OAT), opioid replacement therapy (ORT), opioid substitution therapy (OST) and opioid treatment program (OTP). It involves the use of one of the following opioids to help patients reduce or stop using other opioids:

- [buprenorphine](#)—as a sublingual film or tablet taken daily (or on every 2 or 3 days), or a modified-release subcutaneous injection given weekly or monthly
- [methadone](#)—as an oral liquid taken daily.

Both buprenorphine and methadone are opioid receptor agonists. Buprenorphine is a partial mu-receptor agonist; methadone is a full mu-receptor agonist.

Naltrexone [\[Note 2\]](#) is generally not used for the treatment of opioid dependence, because of very poor adherence. If a patient requests oral naltrexone for opioid dependence, seek specialist advice. No long-acting naltrexone depot injections are registered for use in Australia. Naltrexone implants are not registered or recommended because of inadequate safety data on their use.

[Table 22.11](#) compares MATOD with [planned opioid withdrawal](#). Although planned withdrawal may initially appeal to a patient, encourage the patient to consider MATOD because it is more effective and safer.

Table 22.11 Comparison of medication-assisted treatment of opioid dependence (MATOD) with planned withdrawal to manage opioid dependence

	MATOD	Planned withdrawal
	decreases mortality	
	highly effective in reducing use of nonprescribed opioids	
Advantages	reduces risk of bloodborne viral infections	short-term commitment
	improves quality of life	access may be easier
	avoids withdrawal in patients who are ill or unstable	entry point to other treatments
	widely available (in metropolitan centres)	
	for sublingual buprenorphine or oral methadone: consider travel costs and dispensing fees for the patient, and restrictions of supervised dosing [NB1]	increased opioid overdose risk following withdrawal because of reduced tolerance
Considerations	long-term opioid adverse effects	poor long-term outcomes if used as a standalone treatment
	stigma	
	possibly prolonged withdrawal on stopping	can destabilise other conditions (eg chronic pain, mental health)

NB1: These issues are less relevant for injectable weekly or monthly buprenorphine treatment.

MATOD is more effective and safer than planned withdrawal in the treatment of opioid dependence.

Robust evidence with a high level of certainty supports the use of MATOD in combination with psychosocial interventions to:

- decrease the reinforcing euphoric effects of other opioids that may be used concurrently
- reduce use of other opioids and the risk of overdose
- reduce risk of bloodborne infections
- prevent opioid withdrawal symptoms
- reduce cravings
- improve health and social functioning, including ability to work and stay in stable accommodation.

MATOD therapy is the most effective treatment for opioid dependence; it can improve health and restore ability to work or study.

Use shared decision-making to help a patient decide the aim of MATOD for them. Some patients continue MATOD in the long term, while others have a period of maintenance before gradually reducing the dose to stop.

Factors to consider when deciding whether to stop MATOD therapy are discussed in [Table 22.15](#). Planning to stop MATOD requires support with relapse prevention strategies. It is common for patients to want to stop MATOD early; encourage them from the start of treatment to continue MATOD for a minimum of 12 months (although longer treatment is likely to offer more benefit); this allows time for the patient to develop strategies to reduce the risk of relapse. Discuss the fact that relapse can happen (particularly if a patient requires opioids for surgery) and recommend that patients seek treatment again early if relapse should occur.

Indications to seek specialist advice if a patient is considering starting MATOD therapy include:

- [pregnancy and breastfeeding](#)
- polysubstance use (use of more than one substance)
- acute medical illness
- comorbidities such as liver disease, respiratory or central nervous system depression or [chronic pain](#)
- [mental illness](#)
- [recent release from prison](#).

These issues are also discussed in more depth in Section A2.1 of the [National Guidelines for MATOD](#).

Monitoring is more intensive at the start of MATOD; sublingual buprenorphine and liquid methadone require each dose to be supervised (observation of the dose being taken at a pharmacy or clinic). Once the patient's condition stabilises, the frequency of reviews can be reduced and some of the doses can be unsupervised ('takeaways') [\[Note 3\]](#). Signs of stability include negative urine screens for illicit drugs; involvement in education, training, employment or childcare; punctual attendance at appointments; signs of self-care, and positive interactions with staff. The timing and frequency of takeaway doses varies with state and territory regulations.

Buprenorphine and methadone each have features that influence the choice of drug, as summarised in [Table 22.12](#).

Table 22.12 Comparison of buprenorphine and methadone in medication-assisted treatment of opioid dependence (MATOD)

	Buprenorphine	Methadone
Considerations when starting	therapeutic doses can be achieved more quickly with buprenorphine than when starting methadone	methadone is less likely to precipitate withdrawal than buprenorphine when starting [NB1]
Requirement for attendance for dosing	more flexible with buprenorphine; daily or alternate daily sublingual dosing can be used. Weekly and monthly injectable formulations free patients to focus on other life activities [NB2]	attendance for daily oral dosing is less convenient than for injectable buprenorphine and has more travel cost and stigma
Overdose risk	overdose less likely with buprenorphine than with methadone	higher risk of toxicity, especially when starting methadone
Drug interactions	fewer drug interactions occur with buprenorphine than with methadone	clinically relevant drug interactions increase or decrease the metabolism of methadone
Impact on cognitive function	buprenorphine may have less impact than methadone on cognition	methadone can increase the QTc interval distressed patients may prefer the sedative effects of methadone
Ease of stopping	may be easier to stop buprenorphine than methadone	symptomatic withdrawal is more prolonged after stopping methadone

NB1: Buprenorphine can precipitate withdrawal if started too soon after the last use of a more potent opioid; this is because buprenorphine can displace the other opioid from mu receptors but results in less stimulation of the receptor (partial agonism).

NB2: [Table 22.13](#) outlines characteristics of the available preparations of buprenorphine.

Note 2: Naltrexone is an opioid antagonist, not to be confused with another, naloxone. Naloxone is used for reversal of [opioid overdose](#).

Note 3: Most jurisdictions allow more unsupervised treatment with buprenorphine than with methadone because overdose risk is less with buprenorphine than methadone.

Patient evaluation before starting MATOD

Patient evaluation before starting MATOD

Consider [indications for specialist advice](#) on MATOD.

Before prescribing MATOD therapy, perform a thorough [assessment](#) of the patient's general physical and mental health over a number of extended appointments.

Ask about use of medications that induce or inhibit hepatic cytochrome P450 enzymes; these metabolise buprenorphine and methadone, and dose adjustment may be required (particularly for methadone).

Assess liver function and test for viral hepatitis. For patients with liver impairment (eg Child–Pugh class B or C cirrhosis), lower starting doses, slower rates of dose adjustments and closer monitoring for sedation may be required to reduce risk of fatal overdose. Particularly for methadone, also consider these 'start low, go slow' measures if the patient has respiratory or central nervous system depression, or if they are using alcohol, benzodiazepines, gabapentinoids or other sedatives.

An electrocardiogram (ECG) is recommended before starting methadone for patients who are at increased risk of QTc prolongation; see [Methadone for medication-assisted treatment of opioid dependence](#).

Legal and practical requirements before starting MATOD

Legal and practical requirements before starting MATOD

Observe the legal requirements for prescribing opioids to drug-dependent people. Most jurisdictions have constraints on the prescription of opioids, requiring permits (or authority) for prescribing. Some jurisdictions require clinicians to complete specific training before prescribing any form of MATOD; others require this only for prescribing methadone. Free online training regarding MATOD is available from the [Royal Australian College of General Practitioners \(RACGP\)](#) for general practitioners, specialists and nurse practitioners.

Before prescribing MATOD, see the state and territory educational resources, clinical guidelines and regulations governing prescribing MATOD therapy at the following websites:

The Australian Therapeutic Goods Administration (TGA) website has [contacts for state and territory medicines and poisons regulation units](#) that provide advice on interpretation of legal requirements for prescribing.

Before prescribing MATOD, see the state- and territory-based educational resources, clinical guidelines and regulations.

See [Figure 22.8](#) for practical steps required before prescribing MATOD.

Figure 22.8 Practical steps before prescribing medication-assisted treatment of opioid dependence (MATOD)

Review state and territory guidance on MATOD prescribing.

Consult a [clinical advisory service](#) for any clinical queries about dosing and monitoring.

Review real-time prescription monitoring sources to ascertain details of treatment provided by other prescribers.

Obtain a permit or authority from your state or territory Department of Health before writing a prescription.

Identify a pharmacy or clinic that will provide the buprenorphine or methadone.

Clarify the dosing arrangements (for sublingual or oral forms) and costs with the pharmacy and the patient.

Certify a photograph of the patient for identification (needed in some jurisdictions to collect the medication from the pharmacy).

Ensure the patient is not intoxicated at the time of their first dose. For buprenorphine, consider the need to assess for evidence of withdrawal before dosing, to avoid precipitated withdrawal.

Buprenorphine for medication-assisted treatment of opioid dependence (MATOD)

Buprenorphine for medication-assisted treatment of opioid dependence (MATOD)

Buprenorphine is a safe and effective treatment for medication-assisted treatment of [opioid dependence](#) (MATOD). It is used sublingually or subcutaneously in higher doses than those used for analgesia.

The characteristics of buprenorphine preparations are outlined in [Table 22.13](#). Therapy can be started using either a [sublingual preparation](#) or a [weekly modified-release subcutaneous preparation](#); maintenance therapy can be with either a sublingual or modified-release (weekly or monthly) subcutaneous injection.

For a comparison of buprenorphine with methadone, see [Table 22.12](#).

For considerations before starting buprenorphine, see [Patient evaluation before starting MATOD](#) and [Legal and practical requirements before starting MATOD](#). Contact with a [specialist advisory service](#) is encouraged.

Specialist advice on prescribing buprenorphine is available by phone and contact is encouraged. [Table 22.13 Preparations of buprenorphine for medication-assisted treatment of opioid dependence \(MATOD\)](#) [Printable table](#)

sublingual preparations (brand names):

- [buprenorphine+naloxone film \(Suboxone\)](#)
- [buprenorphine tablet \(Subutex\)](#)

modified-release subcutaneous preparations (brand names):

- [buprenorphine injection \(Buvidal Weekly\)](#)
- [buprenorphine injection \(Buvidal Monthly\)](#)
- [buprenorphine injection \(Sublocade\)](#)

sublingual buprenorphine+naloxone film (Suboxone)

Frequency of administration	daily or on alternate days
Available strengths	2+0.5 mg; 8+2 mg
Indication	initiation and maintenance of MATOD
Advantages	less risk of being injected than Subutex [NB1]

Considerations	dissolves faster than Subutex tablets
	naloxone is contraindicated in liver failure

sublingual buprenorphine tablet (Subutex)

Frequency of administration	daily or on alternate days
Available strengths	0.4 mg; 2 mg; 8 mg
Indication	initiation and maintenance of MATOD
Advantages	preferred over Suboxone in liver failure and in some pregnant patients
Considerations	not available in some states and territories unless patient is pregnant or has a specific additional indication (eg allergy to naloxone)
	fewer takeaway doses permitted for Subutex than for Suboxone

modified-release subcutaneous buprenorphine injection (Buvidal Weekly)

Frequency of administration	weekly
Available strengths	8 mg/0.16 mL; 16 mg/0.32 mL; 24 mg/0.48 mL; 32 mg/0.64 mL
Indication	initiation and maintenance of MATOD. Some state and territory guidelines suggest starting after a patient has had a short trial (eg 7 days) of sublingual buprenorphine
Advantages	less frequent attendance for dosing frees people to engage with other life activities
Considerations	strategies might be needed to minimise pain on injection [NB2]
	some patients have cosmetic concerns about the visible lumps resulting from the volume of the injection

modified-release subcutaneous buprenorphine injection (Buvidal Monthly)

Frequency of administration	monthly
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Available strengths	64 mg/0.18 mL; 96 mg/0.27 mL; 128 mg/0.36 mL; 160 mg/0.45 mL
Indication	maintenance of MATOD after initiating therapy with Buvidal Weekly or sublingual buprenorphine
Advantages	less frequent attendance for dosing frees people to engage with other life activities strategies may be needed to minimise pain on injection [NB2]
Considerations	some patients have cosmetic concerns about the visible lumps resulting from the volume of the injection
modified-release subcutaneous buprenorphine injection (Sublocade)	
Frequency of administration	monthly
Available strengths	100 mg/0.5 mL; 300 mg/1.5 mL
Indication	maintenance of MATOD after initiating therapy with sublingual or subcutaneous buprenorphine
Advantages	less frequent attendance for dosing frees people to engage with other life activities duration of action is the longest of the monthly formulations, which may add to its convenience strategies might be needed to minimise pain on injection [NB2]
Considerations	some patients have cosmetic concerns about the visible lumps resulting from the volume of the injection

NB1: Naloxone is added to discourage injection. Injected naloxone may precipitate opioid withdrawal, but sublingual naloxone is much less likely to do so because it is poorly absorbed.

NB2: Pain may be reduced with use of cold packs and local anaesthetic.

Buprenorphine can precipitate withdrawal if started too soon after the last use of a more potent opioid. This is because buprenorphine can displace the other opioid from mu receptors but results in less stimulation of the receptor (partial agonism). For patients who have been using heroin or other short-acting opioids (eg morphine, oxycodone), buprenorphine should be started after the effects of the previous opioid have subsided to the extent that mild to moderate withdrawal is evident (from signs such as sweating and dilated pupils). If patients are transferring from longer-acting opioids (eg methadone) to buprenorphine, seek specialist advice from a [clinical advisory service](#).

Buprenorphine can precipitate withdrawal if used too soon after the last opioid dose.

Starting sublingual buprenorphine MATOD therapy

Starting sublingual buprenorphine MATOD therapy

Sublingual buprenorphine is available as a film (in combination with naloxone) or tablet preparation; see [Table 22.13](#) for a comparison of preparations.

When starting sublingual buprenorphine MATOD therapy for patients who have been using a short-acting opioid (eg heroin, morphine, oxycodone), wait until signs of opioid withdrawal are evident; then use:

buprenorphine 4 mg sublingually, as a single dose. Review in 1 to 2 hours. If the first dose did not precipitate worsening of withdrawal, and cravings are present, repeat the dose. Increase the dose each day in 2 mg, 4 mg, or 8 mg increments to achieve a maintenance dose (usually 12 to 24 mg daily) within several days. Maximum daily dose is 32 mg.

During buprenorphine dose titration, monitor for signs of withdrawal and toxicity, and use of other opioids and nonprescribed drugs. If exacerbation of withdrawal occurs, seek specialist advice.

In patients with low opioid tolerance, consider using a lower starting dose of buprenorphine (eg 2 mg) and 2 mg increments for dose titration because this reduces the risk of respiratory suppression and precipitating marked withdrawal.

Starting Buvidal Weekly modified-release subcutaneous buprenorphine MATOD therapy

Starting Buvidal Weekly modified-release subcutaneous buprenorphine MATOD therapy

Buvidal Weekly modified-release subcutaneous buprenorphine injections are an alternative to sublingual preparations for starting MATOD therapy.

If starting Buvidal Weekly modified-release subcutaneous buprenorphine injections for a patient who is not already taking sublingual buprenorphine, use:

buprenorphine modified-release (Buvidal Weekly) 16 mg subcutaneously. If the first dose did not control symptoms of withdrawal and cravings, give an additional 8 mg dose once or twice, at least 1 day apart. Sum the doses given in the first week to determine the dose for the second week. Maximum weekly dose is 32 mg.

For patients who have never taken buprenorphine, consider a short trial of sublingual buprenorphine before starting subcutaneous injections to ensure a positive response (eg no precipitated withdrawal or other adverse events, and a good opioid effect). Some state and territory guidelines suggest that patients have an initial 7 days of sublingual buprenorphine. See [Starting sublingual buprenorphine MATOD therapy](#) for advice on sublingual dosage.

For patients already stabilised on a maintenance dose of sublingual buprenorphine who are switching to Buvidal Weekly, see [Switching from sublingual buprenorphine to Buvidal Weekly modified-release subcutaneous buprenorphine MATOD therapy](#) for advice on dosage.

Switching between different formulations of buprenorphine MATOD therapy

Switching between different formulations of buprenorphine MATOD therapy

Some patients may transfer from sublingual buprenorphine to modified-release subcutaneous injections. Modified-release buprenorphine injections are available as weekly (Buvidal Weekly) or monthly preparations (Buvidal Monthly or Sublocade); see [Table 22.13](#) for a comparison of preparations.

[Table 22.14](#) outlines usual starting doses of modified-release subcutaneous buprenorphine when switching between formulations. To switch from one MATOD formulation to another, see the following recommendations:

- from [sublingual buprenorphine to Buvidal Weekly](#)
- from Buvidal Weekly to Buvidal Monthly (or directly from sublingual buprenorphine to Buvidal Monthly)
- from [sublingual buprenorphine to Sublocade](#).

Table 22.14 Starting doses of modified-release subcutaneous buprenorphine preparations when switching between formulations

Dose of daily sublingual buprenorphine	Starting dose of Buvidal Weekly injection [NB1]	Starting dose of Buvidal Monthly injection [NB1]	Starting dose of Sublocade monthly injection [NB1]
2 to 6 mg	8 mg	n/a	n/a

8 to 10 mg	16 mg	64 mg	
12 to 16 mg	24 mg	96 mg	300 mg monthly for 2 months, then 100 mg monthly (no less than every 26 days) [NB2]
18 to 24 mg	32 mg	128 mg	
26 to 32 mg	n/a	160 mg	

n/a = not appropriate; use an alternative preparation

NB1: Starting doses in this table are not for standalone use; see text for information on titration.

NB2: If switching from Buvidal to Sublocade, seek expert advice. The initial loading doses are not generally needed.

Switching from sublingual buprenorphine to Buvidal Weekly modified-release subcutaneous buprenorphine MATOD therapy

Switching from sublingual buprenorphine to Buvidal Weekly modified-release subcutaneous buprenorphine MATOD therapy

For a patient switching from sublingual buprenorphine to Buvidal Weekly, [Table 22.14](#) provides a guide to initial dosing. To transfer from sublingual to weekly subcutaneous buprenorphine, use:

buprenorphine modified-release (Buvidal Weekly) subcutaneously, weekly; see [Table 22.14](#) for starting dose. Start on the day after the last sublingual buprenorphine dose. If required to manage withdrawal symptoms, give an additional 8 mg subcutaneous dose once or twice, at least 1 day apart. Sum the doses given in the first week to determine the dose for the second week. Maximum weekly dose is 32 mg.

Switching to Buvidal Monthly modified-release subcutaneous buprenorphine MATOD therapy

Switching to Buvidal Monthly modified-release subcutaneous buprenorphine MATOD therapy

Switching to Buvidal Monthly can be considered after starting Buvidal Weekly, or directly from sublingual buprenorphine.

To switch **from Buvidal Weekly to Buvidal Monthly** for MATOD therapy, use:

buprenorphine modified-release (Buvidal Monthly) subcutaneously, monthly; see [Table 22.14](#) for starting dose. Start 1 week after the last dose of Buvidal Weekly. If required to manage withdrawal symptoms, give one additional 8 mg dose of Buvidal Weekly, to a maximum total monthly dose of 160 mg. Adjust Buvidal Monthly dose as required to manage withdrawal symptoms and avoid toxicity.

To switch **from sublingual buprenorphine directly to Buvidal Monthly**, use [Table 22.14](#) to determine the starting dose. Start the day after the last sublingual dose and adjust the dose in the same manner as if switching from Buvidal Weekly to Buvidal Monthly (as above).

Switching to Sublocade monthly modified-release subcutaneous buprenorphine MATOD therapy

Switching to Sublocade monthly modified-release subcutaneous buprenorphine MATOD therapy

Sublocade is a monthly preparation of modified-release subcutaneous buprenorphine that has a longer duration of action than Buvidal Monthly.

Patients can transfer to Sublocade after 1 week of sublingual buprenorphine, provided the sublingual dose is 8 mg daily or more. If transferring from sublingual buprenorphine to Sublocade, use:

buprenorphine modified-release (Sublocade) 300 mg subcutaneously, monthly for 2 months, then reduce to 100 mg monthly. Minimum interval is 26 days.

Transfer to Sublocade may also be considered under expert guidance for patients using Buvidal Weekly or Buvidal Monthly. It may be useful for patients who have experienced repeated withdrawal towards the end of the dosing interval during treatment with Buvidal Weekly or Buvidal Monthly. Most patients will have adequate serum concentrations of buprenorphine, so will not require the 300 mg loading doses of Sublocade when switching from Buvidal Weekly or Monthly.

Methadone for medication-assisted treatment of opioid dependence (MATOD)

Methadone for medication-assisted treatment of opioid dependence (MATOD)

Long-term treatment with oral (liquid) methadone syrup or solution (methadone maintenance) for medication-assisted treatment of [opioid dependence](#) is effective in reducing illicit opioid use and nonmedical use of prescription opioids.

For a comparison of methadone and buprenorphine, see [Table 22.12](#).

For considerations before starting methadone, see [Patient evaluation before starting MATOD](#) and [Legal and practical requirements before starting MATOD](#). Approval for each patient from the state or territory Department of Health is required before starting treatment with methadone. Contact with a specialist advisory service is encouraged.

Methadone is associated with QTc interval prolongation; it is important to perform an electrocardiogram (ECG) to assess QTc interval before starting methadone for patients with:

- known QTc prolongation
- potential symptoms of QTc prolongation (syncope, palpitations, dizziness)
- other risk factors, such as congenital long QTc syndrome (family history), cardiac abnormalities or use of [other drugs that prolong the QTc interval](#).

Also perform an ECG periodically for patients taking more than 120 mg methadone daily.

Methadone doses (initial and maintenance) should be individualised; titrate the dose to suppress withdrawal, minimise ongoing use of other opioids, maximise the patient's function and avoid toxicity. Methadone has a long half-life (20 to 36 hours). During the start of treatment, methadone accumulates in the serum; serum concentrations may take 1 week to stabilise after each dose change. Fatal toxicity can occur (even with doses as low as 30 mg daily) if the dose is increased too rapidly, or if the patient has low opioid tolerance or is using other sedatives (eg alcohol, benzodiazepines). Risk of overdose is particularly increased in the first 2 weeks of methadone treatment. 'Start low, go slow, aim high' is a helpful maxim. Review patients regularly when starting them on methadone to observe for any signs of opioid toxicity. Seek specialist advice before dosing if the patient is intoxicated or continues to use nonprescribed opioids. If a patient is intoxicated, methadone should not be given until the cause is determined. Naloxone or transfer to hospital may be indicated. For information on management of methadone overdose, see [Management overview of opioid poisoning](#).

Avoid rapid increases in methadone dose, particularly in the first 2 weeks of treatment to reduce risk of fatal toxicity.

It may take several months to achieve a methadone dose at which a patient stops using nonprescribed opioids. Outcomes are usually better when higher maintenance doses are provided for longer periods; many patients are maintained in the range of 60 to 120 mg daily. Higher doses are sometimes required; seek [specialist advice](#)

in these situations.

Specialist advice on prescribing methadone is available by phone and contact is encouraged.

When starting methadone MATOD therapy for a patient who uses opioids daily and does not use other sedatives, a suitable regimen is:

methadone 20 to 30 mg orally, daily. Increase the dose in increments of 5 to 10 mg no more frequently than every 3 to 5 days. Assess for symptoms of withdrawal or features of intoxication before each dose increase. Aim to achieve a maintenance dose (usually 60 to 120 mg daily) within 4 to 8 weeks.

Considerations when stopping medication-assisted treatment of opioid dependence (MATOD)

Considerations when stopping medication-assisted treatment of opioid dependence (MATOD)

When planning to stop medication-assisted treatment of opioid dependence (MATOD), consider whether factors are present that may promote positive long-term outcomes, as outlined in [Table 22.15](#). The minimum duration of MATOD is ideally 12 months (although longer treatment is likely to offer more benefit); this allows time for the patient to address other life concerns with the aim of reducing risk of relapse. Discuss the fact that relapse can happen (particularly if a patient requires opioids for surgery) and recommend that patients seek treatment again early if relapse should occur.

Stopping MATOD requires support and relapse prevention strategies. [Specialist advice](#) is available and contact is encouraged when planning MATOD tapering. Most commonly, treatment is gradually tapered over months with careful monitoring.

Table 22.15 Factors to consider when planning to stop medication-assisted treatment of opioid dependence

Patient factors

minimal or stable use of nonprescribed substances including alcohol

good physical and mental health

positive social factors (housing, work, recreation, support from friends and family)

central role in decision-making and good understanding of the process of stopping MATOD

Treatment process factors

plan agreed to taper MATOD over months with regular review of progress

coping strategies and ways jointly devised to reduce risk of lapses and relapses

Opioid withdrawal

Opioid withdrawal

Overview of opioid withdrawal

Overview of opioid withdrawal

Opioid withdrawal is very rarely life threatening, but often very distressing for the patient and those close to them; avoiding withdrawal can be a principal cause of relapse. For symptoms and signs of withdrawal, see [Table 22.10](#).

[Planned opioid withdrawal](#) is rarely successful as a standalone treatment for opioid dependence; treatment with buprenorphine or methadone as part of [medication-assisted treatment of opioid dependence \(MATOD\)](#) is much more effective.

Managed (planned) opioid withdrawal as a sole treatment is rarely successful.

Pregnant individuals are advised not to undergo opioid withdrawal; refer to a drug and alcohol specialist service to discuss options. MATOD is recommended as it is safer for the patient and the fetus.

Withdrawal from opioids with unusual pharmacokinetics (eg fentanyl, methadone) is complex; seek specialist advice.

If a patient chooses managed withdrawal after discussion of the options, ensure that withdrawal takes place in a safe environment. For advice on how to assess safety of an environment for managed withdrawal, see [Choice of setting for planned withdrawal management](#). For advice on safe prescribing and supply during withdrawal in a home setting, see [Safe prescribing and supply during planned substance withdrawal](#).

Relapse after withdrawal can be life threatening because the patient's tolerance to opioids is decreased. All patients undergoing opioid withdrawal should be given [take-home naloxone](#), and significant others (partner, family, friends) should be instructed on how to use it and advised to call an ambulance if overdose occurs.

Planned opioid withdrawal

Planned opioid withdrawal

Treatment of opioid dependence by planned (managed) withdrawal is ineffective compared to longer-term therapy ([MATOD](#)); encourage patients to consider MATOD as an alternative because relapse after withdrawal is very common and associated with increased risk of overdose because of reduced opioid tolerance.

If a patient chooses planned opioid withdrawal, short-term prescribing of buprenorphine is preferred over [symptomatic treatment](#) because it is more effective in controlling symptoms, requires a shorter treatment time and increases the likelihood of withdrawal completion. During buprenorphine treatment, offer patients the option of switching from the buprenorphine withdrawal regimen to buprenorphine maintenance (MATOD) because MATOD has better long-term outcomes.

During a planned withdrawal, switching to long-term buprenorphine maintenance treatment is encouraged for relapse prevention.

Buprenorphine for planned opioid withdrawal

Buprenorphine for planned opioid withdrawal

The same [legal requirements before starting medication-assisted treatment of opioid dependence](#) apply when prescribing buprenorphine to manage opioid withdrawal.

Because it has partial agonist effects, buprenorphine may precipitate opioid withdrawal [[Note 4](#)] in patients taking another more potent opioid. The first dose of buprenorphine should not be given until objective signs of withdrawal and subjective withdrawal symptoms are present. The dose and duration of use should be titrated according to withdrawal severity using the Clinical Opiate Withdrawal Scale, available from the [National Centre for Education and Training on Addiction website](#). Buprenorphine can be used in either an inpatient or an outpatient setting.

A reasonable regimen for use of buprenorphine to manage planned withdrawal in an **outpatient** setting is:

buprenorphine 4 to 8 mg sublingually, as a single dose on the first day, increasing to a maximum of 16 mg as a single dose on the third day. For patients wishing to stop buprenorphine, taper the dose from the fourth day over the following 2 to 5 days. For patients who wish to switch to maintenance therapy, see [Overview of MATOD](#).

In an **inpatient** setting, the benefit of psychosocial engagement means a lower starting dose and smaller incremental dose increases may be sufficient. A reasonable regimen for use of buprenorphine to manage planned opioid withdrawal in an inpatient setting is:

buprenorphine 2 to 4 mg sublingually, on the first day, repeated as required every 2 hours to a maximum of 16 mg on the first day. For patients wishing to stop buprenorphine, taper the dose from the second day over the following 2 to 5 days. For patients who wish to switch to maintenance buprenorphine, see [Overview of MATOD](#).

Exercise caution when prescribing buprenorphine with other sedatives, including other opioids, benzodiazepines or gabapentinoids. Observe state and territory statutory health regulations on the prescription of buprenorphine.

Note 4: Buprenorphine can precipitate withdrawal if started too soon after the last use of a more potent opioid; this is because buprenorphine can displace the other opioid from mu receptors but results in less stimulation of the receptor (partial agonism).

Overview of symptomatic management of planned opioid withdrawal

Overview of symptomatic management of planned opioid withdrawal

If buprenorphine cannot be used for management of planned opioid withdrawal, the second-line alternative is to use a range of medications to manage withdrawal symptoms; see [Table 22.16](#).

Table 22.16 Medications to manage symptoms of opioid withdrawal

Symptom	Medication
anxiety and agitation	diazepam for a maximum of 7 days (10 days for inpatients)
nausea and vomiting	antiemetics (eg metoclopramide, prochlorperazine, olanzapine, ondansetron) for a maximum of 7 days
diarrhoea	loperamide (see Functional diarrhoea for dosage) for a maximum of 7 days
abdominal cramps	hyoscine (see Antispasmodics for pain in palliative care for dosage) for a maximum of 7 days
physical pain and headaches	paracetamol, NSAIDs (see Oral drugs for mild, nociceptive pain in adults for dosages) for a maximum of 7 days
insomnia	temazepam for a maximum of 5 days
sweating, tachycardia, hypertension, agitation and restless legs	clonidine
NSAIDs = nonsteroidal anti-inflammatory drugs	

Drug therapy for specific symptoms in planned opioid withdrawal

Drug therapy for specific symptoms in planned opioid withdrawal

For treatment of **anxiety and agitation** in opioid withdrawal in a **home setting** if buprenorphine cannot be used, consider:

diazepam 5 to 10 mg orally, 6-hourly as required. Review after 2 to 3 days to assess symptom control. Maximum duration 7 days.

For treatment of **anxiety and agitation** in opioid withdrawal in a **community residential unit or hospital setting**, patients may be reviewed using the Clinical Opiate Withdrawal Scale (available from the [National Centre for Education and Training on Addiction website](#)) or other opiate withdrawal scale, provided an appropriately trained person is available before each dose. If buprenorphine cannot be used, consider:

diazepam 5 to 10 mg orally, 6-hourly as required to control anxiety and agitation. Maximum duration 10 days.

For treatment of **insomnia** during planned opioid withdrawal, use:

temazepam 10 to 20 mg orally, at night as required. Maximum duration 5 days.

Benzodiazepines increase overdose risk if the person subsequently relapses; avoid prescribing more than one benzodiazepine or combinations of a benzodiazepine and other drugs with sedative effects such as opioids or gabapentinoids.

Diazepam could be collected daily by the patient or if supplying a few days' medication at a time, administration should be supervised by a reliable person. Only prescribe 2 to 3 days of diazepam supply at a time because of risk of overdose. Avoid ongoing prescribing so the patient does not become dependent on benzodiazepines. Advise patients not to drive or drink alcohol during treatment with benzodiazepines for opioid withdrawal.

Avoid prescribing benzodiazepines for opioid withdrawal unless the patient collects the supply daily or supervision by a reliable person is available. Only prescribe a few days of diazepam doses at a time because of risk of overdose.

Clonidine (an alpha-2 adrenergic agonist) may be used to manage **sweating, tachycardia, hypertension, agitation or restless legs** in an inpatient setting if buprenorphine cannot be used. Check heart rate and blood pressure before every dose, and ensure patients are hydrated to limit risk of hypotension.

A suggested regimen for the use of clonidine to manage opioid withdrawal is:

clonidine 50 to 75 micrograms orally, 6- to 8-hourly. Increase dose as required to control withdrawal symptoms. Withhold or reduce dose if bradycardia occurs or systolic blood pressure is less than 90 mmHg. A maximum daily dose of 600 micrograms can be given in hospital. Taper dose and aim to stop in 5 to 6 days.

For more information on opioid withdrawal management, see this quick reference withdrawal guide available at the [Insight website](#).

Sometimes inpatient admissions for planned opioid withdrawal end with the patient leaving early, before the withdrawal syndrome has ended. All patients should be offered [take-home naloxone](#) because of the risk of relapse following withdrawal.

Offer all patients take-home naloxone on discharge after planned opioid withdrawal.

Unplanned opioid withdrawal in inpatient settings

Unplanned opioid withdrawal in inpatient settings

Opioid withdrawal commonly presents covertly as withdrawal in a general hospital or mental health acute care setting in patients admitted for other reasons. An opioid may need to be given during admission to prevent further withdrawal symptoms.

Consider starting [medication-assisted treatment of opioid dependence \(MATOD\)](#) (with methadone or buprenorphine) during the admission for patients who develop unplanned withdrawal. Advice from a [clinical advisory service](#) is recommended. A comprehensive assessment of opioid and other drug use is essential before starting MATOD. Liaison with community treatment providers is needed during treatment planning if care will be transferred to them. Methadone or buprenorphine doses may sometimes be provided more than once daily ('splitting' the dose) in inpatient settings. Comprehensive discharge planning is important to ensure continuous care.

If the patient will only consider short-term treatment, buprenorphine can be used as for treatment of [planned opioid withdrawal](#).

Exercise caution when prescribing buprenorphine with other sedatives, including other opioids, benzodiazepines or gabapentinoids. Observe state and territory statutory health regulations on the prescription of buprenorphine.

Considerations for specific populations in management of disorders of opioid use

Considerations for specific populations in management of disorders of opioid use

Unmanaged opioid use during [pregnancy](#) or [breastfeeding](#) is associated with harms; specific considerations apply in managing opioid dependence in both patient groups, including the need for urgent specialist referral. For other groups, see [Considerations for specific populations in substance use and addictive behaviours](#).

Management of opioid dependence during pregnancy

Management of opioid dependence during pregnancy

Refer patients with opioid dependence who are considering pregnancy or are pregnant urgently to a specialist antenatal and drug and alcohol treatment service.

Patients with opioid dependence who are considering pregnancy or are pregnant should be urgently referred to a specialist antenatal and drug and alcohol treatment service to assist with management in planning pregnancy, during pregnancy and the postpartum.

Potential harms of unmanaged opioid use in pregnancy result from overdose, withdrawal and injection harms; they include intrauterine growth restriction, premature rupture of membranes, placental abruption, stillbirth, and maternal and neonatal infections. Additional risks include other drug use (eg alcohol, tobacco), partner violence, unstable accommodation, poverty, mental health problems and sex work that can increase pregnancy risks and result in poor antenatal attendance. Opioid withdrawal is not recommended because of concerns about relapse, poor antenatal attendance and the risk of premature labour and miscarriage.

[Medication-assisted treatment of opioid dependence \(MATOD\)](#) is the standard recommendation for opioid dependence during pregnancy. Benefits of MATOD include improved maternal health, attendance at antenatal care, decreased miscarriage and neonatal loss, higher birth weight babies, longer gestations and increased likelihood of the infant being discharged into the patient's care.

Most infants exposed to buprenorphine or methadone develop a withdrawal syndrome (neonatal opioid withdrawal syndrome [NOWS]). Reassure patients that severity of NOWS is not related to maternal buprenorphine or methadone dose. The prevalence, severity and duration of clinically significant NOWS appear to be less with buprenorphine compared to methadone.

Methadone and sublingual buprenorphine (both buprenorphine alone and the combination products with naloxone) can be prescribed during pregnancy and breastfeeding. For patients who are not already on MATOD, buprenorphine may be the preferred first-line treatment, particularly because the risk of NOWs is less with buprenorphine compared to methadone treatment. Modified-release buprenorphine (as a subcutaneous injection) can be prescribed provided a harm–benefit analysis is favourable for the patient and baby.

For pregnant patients who are not stable on sublingual buprenorphine, switching to modified-release buprenorphine or methadone may be considered. Switching from methadone to buprenorphine is generally not advised in pregnancy because of the risk of precipitated withdrawal. Seek specialist advice on switching treatments from a substance use in pregnancy service.

Metabolic changes in pregnancy may require increases in the dose of buprenorphine or methadone (more so for methadone than buprenorphine).

The onset of NOWS is usually 48 to 72 hours after birth, but may be up to 1 week after birth. Therefore, an extended stay in hospital for the patient and their baby is recommended for observation. Nonpharmacological support for babies with NOWS (cuddling, feeding, tight wrapping in a blanket, low stimulation) is important.

Management of opioid dependence during breastfeeding

Management of opioid dependence during breastfeeding

Methadone is considered relatively safe to use while breastfeeding. The concentration in breastmilk is low, regardless of maternal dose. Provided they have no medical contraindications to breastfeeding (illicit drug use, HIV), patients taking methadone should be strongly encouraged to breastfeed. Pharmacokinetics change after delivery; patients usually need to have their methadone dose incrementally reduced in the postpartum period to avoid oversedation, especially when caring for infants. Symptoms of withdrawal may occur in the first week in 60% of infants born to patients who were taking methadone during pregnancy; the concentration of methadone in breastmilk is insufficient to prevent withdrawal. Infants in withdrawal are often very difficult to breastfeed (irritable and hypertonic with poor suck and swallow coordination); involvement of a lactation consultant is desirable.

Buprenorphine has not been associated with adverse effects on the breastfed infant in a small number of reports, and transmission to breastmilk is low. Breastfeeding with buprenorphine is not contraindicated; however, data are lacking on long-term outcomes for exposed infants. No data are available to support the use of buprenorphine plus naloxone while breastfeeding.

Patients who are stable on methadone or buprenorphine should be supported if they choose to breastfeed.

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Overview of disorders of benzodiazepine, zolpidem and zopiclone use

Overview of disorders of benzodiazepine, zolpidem and zopiclone use

Benzodiazepines and the Z drugs, zolpidem and zopiclone, act on the benzodiazepine binding site of the GABA_A receptor: they are collectively called benzodiazepine receptor agonists. While most of these drugs are prescribed restricted substances (Schedule 4, as defined by the [Therapeutic Goods Administration \[TGA\]](#) alprazolam and flunitrazepam are controlled drugs (Schedule 8).

Potential harms of benzodiazepines, zolpidem or zopiclone use include toxicity (particularly respiratory depression, if used with other sedatives, including alcohol and opioids), falls, and impaired cognition, concentration and attention. These can lead to traffic accidents, poor self-care and impairment of work and parenting. Although early media reports described unusual behaviours with zolpidem and zopiclone use, behavioural changes following their ingestion do not differ from those seen with benzodiazepines. Newer illicit designer benzodiazepines such as etizolam are being detected in Australia through toxicovigilance screening.

The spectrum of substance use is described in these guidelines by the terms 'hazardous use', 'harmful use' and 'substance dependence', outlined in [Table 22.1](#). Within these categories, types of use of benzodiazepines, zolpidem or zopiclone range from use as prescribed (eg if prolonged doses are prescribed for anxiety or sleep disorders) to nonmedical use. Nonmedical use does not align with the directed use, for example, use to become intoxicated or to treat a symptom other than the clinician intended.

Patients may not be aware that continued use (even as prescribed) poses risk. With lower doses (eg less than 15 mg [oral daily diazepam equivalent](#)), patients may be stable for some time at that dose but can still experience harms. These harms include impacts on cognition and increased falls.

Patients on long-term benzodiazepines, zolpidem or zopiclone (even at stable lower doses) are at risk of harms.

A person with [substance dependence](#) may experience a strong drive to escalate their use, despite harms occurring. Current or past disorders of alcohol or other drug use increase the likelihood of dependence on benzodiazepines, zolpidem or zopiclone. Higher-dose, high-risk use of more than one substance (polysubstance use) can sometimes be chaotic.

Screening and assessment of disorders of benzodiazepine, zolpidem and zopiclone use

Screening and assessment of disorders of benzodiazepine, zolpidem and zopiclone use

Ask all new patients routinely and others (adolescents and adults) opportunistically and periodically about medication use, as part of a general screen for disorders of substance use and gambling; these disorders are common (and often co-exist) and people are reluctant to disclose them, often due to fear of stigma. [Screening and assessment of substance use and addictive behaviours](#) outlines history-taking (including use of the [ASSIST-Lite tool](#)), examination, and investigations that should be considered in a broad review of substance use and addictive behaviour.

An additional approach that is beneficial in addressing benzodiazepine use is to identify patients prescribed benzodiazepines by searching practice records, and sending them a tailored letter that encourages reduction. A template 'Practice letter to patients about benzodiazepine reduction' is available from the [Royal Australasian College of General Practitioners \(RACGP\) website](#), which also includes a fact sheet for patients. Sample responses to requests for benzodiazepines are also available from the [RACGP website](#).

In discussing benzodiazepine, zolpidem and zopiclone use, ask about trends in use, including any past efforts to reduce, withdrawal symptoms, reasons for starting and continuing (eg anxiety or sleep disorders) and [potential harms](#), including risk of overdose and child protection concerns.

Withdrawal symptoms include anxiety, insomnia, irritability, tremor, palpitations, poor concentration, distorted perception and hyperacusis. Signs of withdrawal include tachycardia, hypertension and myoclonic twitches. Abrupt discontinuation may be accompanied by seizures, particularly in patients taking higher doses for prolonged periods and those with a history of seizures. Delirium may also occur on abrupt discontinuation.

Withdrawal symptoms typically emerge within 1 to 5 days of stopping, peak around 7 days and usually abate over the next 2 to 3 weeks. Symptoms of anxiety and insomnia may persist for months after stopping. Withdrawal symptoms may emerge and abate earlier with use of short half-life benzodiazepines. More severe withdrawal may be seen with prolonged higher-dose use.

To **quantify use** of benzodiazepines, zolpidem or zopiclone, calculate the approximate oral daily diazepam equivalent for each drug using [Table 22.17](#). If several different drugs are being used concurrently, sum the diazepam equivalents. This will provide an estimate of the dose of diazepam on which the patient may be stabilised. Widely varying half-lives and receptor-binding characteristics make exact dose equivalents difficult to establish; a range incorporating values 50% above and below the estimates in [Table 22.17](#) is reasonable. Patients sometimes overestimate their consumption; seek corroboration of the doses they are being prescribed using [real-time prescription monitoring](#), although this will not identify any drugs acquired illicitly. Observation in hospital can help assess a patient's tolerance by establishing whether they require their stated dose to avoid withdrawal. If the oral daily diazepam equivalent exceeds 40 mg, seek specialist advice.

Table 22.17 Oral dose equivalents of benzodiazepines, zolpidem and zopiclone

This table is used to quantify total daily oral doses of benzodiazepines, zolpidem and zopiclone to estimate the equivalent dose of diazepam on which the patient should be stabilised; if several different drugs are being used concurrently, sum the diazepam equivalents.

Exact dose equivalents are difficult to establish; a range incorporating values 50% above and below these estimates is reasonable.

Drug	Approximate dose equivalent to 10 mg of oral diazepam [NB1]
alprazolam	0.5 to 1 mg
bromazepam	6 mg
clobazam	20 mg
clonazepam	0.5 mg
diazepam	10 mg
flunitrazepam	1 to 2 mg
lorazepam	1 to 2 mg [NB2]
nitrazepam	10 mg
oxazepam	20 to 60 mg
temazepam	20 mg
zolpidem	20 mg
zopiclone	15 mg

NB1: To calculate the oral daily diazepam equivalent for each drug, divide the daily dose by the approximate dose equivalent in this table and multiply by 10 mg. For example, if the patient is taking temazepam 40 mg daily, the calculation is: 40 mg divided by 20 mg multiplied by 10 mg = 20 mg oral daily diazepam equivalent.

NB2: Lorazepam may be relatively more potent at higher doses.

Overview of management of disorders of benzodiazepine, zolpidem and zopiclone use

Overview of management of disorders of benzodiazepine, zolpidem and zopiclone use

[Overview of substance use and addictive behaviours](#) explains key [principles of care](#) for a person with a disorder of substance use. Establishing a therapeutic relationship that engages the person (and ideally those close to them) is central to the management of substance dependence.

The most important element of treatment for substance use is a therapeutic relationship.

Specialist advice on any aspects of care for people with substance dependence is available and contact is encouraged; see [Contact details for substance use clinical advisory services for clinicians](#).

Specialist advice is available by phone on any aspect of the management of substance use; contact is encouraged.

Patients on higher doses of benzodiazepines, zolpidem or zopiclone may present with intoxication, particularly if using multiple substances. For general advice on managing their safety, see [Ensuring the safety of a person with a disorder of substance use or addictive behaviour](#). Isolated overdose of benzodiazepines, zolpidem or zopiclone can generally be managed with supportive care, but ingestion with other sedative hypnotics, such as opioids, can be fatal. For further information on management of overdose, see [Benzodiazepine poisoning](#). Do not use flumazenil to manage dependence on benzodiazepines, zolpidem or zopiclone because it can precipitate acute withdrawal and seizures.

Do not use flumazenil in managing dependence on benzodiazepines, zolpidem or zopiclone.

The main elements of managing benzodiazepine, zolpidem or zopiclone dependence are:

- [weaning schedules for lower-dose dependence](#) for patients with dependence on lower doses (estimated oral daily diazepam equivalent of 15 mg or less)
- [weaning schedules for higher-dose dependence](#), which often requires specialist input and may involve reducing the dose more quickly initially, followed by gradual dose reduction over months
- [management of unplanned withdrawal](#).

[Psychological interventions](#) add benefit to weaning in stopping use.

[Harm reduction strategies](#) are relevant for all patients. Provide advice on [driving](#), and avoiding intoxication while alone. Warn patients not to use other sedative drugs or alcohol concurrently with benzodiazepines, zolpidem or zopiclone. Discuss falls prevention, particularly for older patients.

[Considerations for management of specific populations](#) may apply. Seek specialist advice for any patient who is pregnant or breastfeeding and dependent on benzodiazepines, zolpidem or zopiclone.

Elements of [long-term management](#) are relevant to all disorders of substance use. Addressing the reasons why the patient has been using benzodiazepines, zolpidem or zopiclone (generally anxiety and/or sleep disturbance) is central to management.

Weaning benzodiazepines, zolpidem or zopiclone

Weaning benzodiazepines, zolpidem or zopiclone

Overview of weaning benzodiazepines, zolpidem or zopiclone

Overview of weaning benzodiazepines, zolpidem or zopiclone

Weaning regimens are generally only required for people who have been using benzodiazepines, zolpidem or zopiclone continuously for 4 weeks or more. Abrupt stopping after use for less than 4 weeks usually does not require weaning because it is unlikely to cause significant [withdrawal symptoms](#).

Management of [dependent use](#) of benzodiazepines, zolpidem or zopiclone is individualised. Establishing a supportive therapeutic relationship is key to engaging patients in the process of weaning these drugs, as some may be reluctant to reduce their dose or highly anxious about the process. Weaning may take months; the rate is influenced by the patient's age, general health, use of other substances and the estimated [oral daily diazepam equivalent](#) they have been taking.

These guidelines outline weaning schedules for:

- [lower-dose \(therapeutic dose\) dependence](#)—in these guidelines, this is considered to be use of an estimated oral daily diazepam equivalent of 15 mg or less; this may be seen in those taking long-term benzodiazepines, zopiclone or zolpidem as prescribed by their treating practitioner
- [higher-dose dependence](#)—in these guidelines, this is considered to be use of more than an estimated oral daily diazepam equivalent of 15 mg; this may be associated with attending one practitioner, seeing multiple practitioners and/or obtaining medications illegally.

Seek [specialist advice](#) for any queries on weaning, particularly for people with any features listed in [Figure 22.9](#).

Seek specialist advice for any queries on weaning, particularly for patients with any features outlined in [Figure 22.9](#). Figure 22.9 Features requiring specialist advice in weaning benzodiazepines, zolpidem or zopiclone

an estimated [oral daily diazepam equivalent](#) exceeding 40 mg

use of more than one substance (polysubstance use)

unstable mental health or unmanaged mental health comorbidities

history of severe [withdrawal symptoms](#)

complex physical comorbidities such as:

- frailty
- impaired liver function
- impaired respiratory function
- impaired cognitive function
- reduced seizure threshold

pregnancy or breastfeeding.

Before starting weaning of benzodiazepines or zolpidem or zopiclone, identify and develop a treatment plan for the conditions for which the patient was using the drug (generally sleep disturbance and/or anxiety). Consider the need for advice on good sleep practices or referral to a sleep psychologist; see [Principles of treating insomnia in adults](#). Consider psychological interventions and/or first-line drugs for anxiety, as outlined in [Overview of anxiety disorders](#). [Psychosocial interventions](#) add benefit to weaning in stopping use.

Develop a **treatment agreement** [\[Note 1\]](#) with the patient before starting weaning, identifying:

- that there will be only one benzodiazepine prescriber
- that drugs will be dispensed from one nominated pharmacy
- the frequency of supervised dosing or staged supply (eg daily or twice weekly) by the pharmacist
- the use of prescription monitoring programmes
- the frequency of clinical reviews—closer monitoring is required for patients with a history of unstable drug use (eg illicit use, dose escalation, overdoses, social or health harms)
- the importance of not driving if feeling sedated, at any time during [dose stabilisation](#), or at any time of unstable use; also discuss the need to review any impacts on driving at each visit. See [Fitness to drive](#) for further advice.

Consider also whether [urine drug screening](#) would be useful (eg to detect use of other drugs).

[Legal considerations for prescribing in substance use and addictive behaviour](#) includes links to prescription monitoring programmes, which can assist in corroborating patient reports of use and monitoring treatment effectiveness. Communication between healthcare professionals about who is prescribing which drugs is also important to avoid excess or undersupply, because not all drugs are included in monitoring programmes.

Note 1: A template agreement is available from the [Royal Australian College of General Practitioners website](#).

Weaning schedules for lower-dose benzodiazepine, zolpidem or zopiclone dependence

Weaning schedules for lower-dose benzodiazepine, zolpidem or zopiclone dependence

In these guidelines, lower-dose dependence is use of an estimated oral daily diazepam equivalent of 15 mg or less. For advice on quantifying use and estimating the oral daily diazepam equivalent, see [Screening and assessment](#). Seek [specialist advice](#) for any queries on weaning, particularly for patients with any of the features outlined in [Figure 22.9](#).

See [Overview of weaning of benzodiazepines, zolpidem or zopiclone](#) for other issues to consider before starting treatment. These include identifying and developing a treatment plan for the underlying reasons for use and specifies how dispensing and monitoring (including fitness to drive) will occur.

In patients using a benzodiazepine, zolpidem or zopiclone at an oral daily diazepam equivalent of less than 15 mg daily, wean the drug dose by 10 to 25% every 1 to 4 weeks, with a goal of completing the reduction in 12 weeks. The [New South Wales Therapeutic Advisory Group](#) has a deprescribing tool and patient information leaflets for use in weaning. Slower weaning may be needed if [withdrawal symptoms](#) are troublesome; sometimes a period of maintenance on a stable dose is required before weaning is resumed.

Psychological treatment improves outcomes in helping patients stop their use; offer psychosocial interventions such as [cognitive behavioural therapy](#) (CBT).

Weaning schedules for higher-dose dependent use of benzodiazepines, zolpidem or zopiclone

Weaning schedules for higher-dose dependent use of benzodiazepines, zolpidem or zopiclone

In these guidelines, higher-dose dependence on benzodiazepines, zolpidem or zopiclone is defined as use of an estimated oral daily diazepam equivalent of more than 15 mg. For advice on quantifying use and estimating the oral daily diazepam equivalent, see [Screening and assessment of benzodiazepines, zolpidem and zopiclone](#).

Limited evidence guides the management of patients with higher-dose dependence on benzodiazepines, zolpidem or zopiclone; advice in these guidelines is based on the consensus view of the Addiction Guideline group.

Stabilisation on oral daily diazepam is generally recommended before weaning because elimination of diazepam is slow, which may make weaning smoother; see [Table 22.17](#) for dose calculations. However, the stabilisation step may need to be omitted or modified in patients who are more likely to accumulate diazepam (those with severe liver disease) or more susceptible to neuropsychiatric adverse events (eg those with pre-existing cognitive impairment).

Seek [specialist advice](#) for any queries on weaning and for patients with any features outlined in [Figure 22.9](#), including an oral daily diazepam equivalent exceeding 40 mg.

An oral daily diazepam equivalent exceeding 40 mg requires specialist advice for weaning benzodiazepines, zolpidem or zopiclone.

Weaning schedules to be started in hospital or a specialist outpatient clinic

Weaning schedules to be started in hospital or a specialist outpatient clinic

People **with** [features that require specialist advice](#) can undergo dose stabilisation in hospital or a specialist outpatient clinic over 2 to 7 days, followed by dose reduction at a rate determined by symptom severity.

Regular clinical assessment of withdrawal, intoxication, substance use and general health is required. The Clinical Institute Withdrawal Assessment Scale for benzodiazepines (available at the [Insight website](#)) can be used in conjunction with clinical reviews to monitor the progress of withdrawal and the response to treatment.

After stabilisation, reductions of 10% of the oral daily diazepam equivalent per day can be achieved in inpatient settings. In general, patients should be discharged only when the oral daily diazepam equivalent has reached 20 mg or less. Slower reduction over months can then take place in the community, at a pace that avoids significant [withdrawal symptoms](#). As outlined in a [treatment agreement](#), this requires supervised dosing or staged supply, and close clinical monitoring (including fitness to drive). Risk level may fluctuate over the course of treatment. A higher-risk patient with a history of **unstable drug use** should have closer monitoring, with regular review of the monitoring interval. Features of unstable use include illicit use, dose escalation, overdoses, and social or health harms from drug use.

Weaning schedules that can be undertaken wholly in the community

Weaning schedules that can be undertaken wholly in the community

Patients **without** [features that require specialist advice](#) can undergo dose stabilisation and weaning in the community over about 2 to 4 weeks.

See [Overview of weaning of benzodiazepines, zolpidem or zopiclone](#) for considerations before starting treatment. These include identifying underlying reasons for use and agreeing a treatment plan that specifies how dispensing and monitoring (including fitness to drive) will occur.

During dose stabilisation in the community, the oral daily diazepam equivalent should be given in a single daily dose, with frequent oversight (supervised dosing or staged supply) from a community pharmacist. Review patients regularly for signs of withdrawal, intoxication, substance use and assessment of general health. The Clinical Institute Withdrawal Assessment Scale-Benzodiazepines (available at the [Insight website](#)) can be used in conjunction with clinical reviews to monitor the progress of withdrawal and the response to treatment. Risk of harms may fluctuate over the course of treatment and should be regularly reassessed; if features outlined in [Figure 22.9](#) develop, closer monitoring will be indicated. Dose reductions in the community often require months. Slower tapering may be needed if a patient develops intrusive [withdrawal symptoms](#).

Offer [psychosocial interventions](#) concurrently as part of [long-term care](#).

Management of unplanned withdrawal from benzodiazepines, zolpidem and zopiclone

Management of unplanned withdrawal from benzodiazepines, zolpidem and zopiclone

Limited evidence guides the management of patients with unplanned withdrawal from benzodiazepines, zolpidem or zopiclone; advice in these guidelines is based on the consensus view of the Addiction Guideline group.

Patients who develop unplanned withdrawal from benzodiazepines, zolpidem or zopiclone while in hospital for other reasons should have their use quantified if possible; determine their oral daily diazepam equivalent as described in [Screening and assessment of disorders of benzodiazepine, zolpidem and zopiclone use](#).

To treat inpatients with unplanned benzodiazepine, zolpidem or zopiclone withdrawal:

- confirm the calculated oral daily diazepam equivalent with community prescribers, pharmacists and real-time prescription monitoring
- if a patient has higher-dose dependence (an oral daily diazepam equivalent more than 15 mg), consider dose stabilisation on diazepam
- consider the need to reduce the oral daily diazepam equivalent based on the clinical scenario (eg frailty, liver failure, hypercapnic [type 2] respiratory failure, impaired cognitive function)
- consider whether long-term use can be modified after acute management.

These considerations can guide the management plan while in hospital. For indications to seek specialist advice, see [Figure 22.9](#).

Regular clinical assessment of withdrawal, intoxication, substance use and general health is required. The Clinical Institute Withdrawal Assessment Scale-Benzodiazepines (available at the [Insight website](#)) can be used in conjunction with clinical reviews to monitor the progress of withdrawal and the response to treatment.

If stabilisation is undertaken using a calculated oral daily diazepam equivalent (and there are no [indications for specialist advice](#)), divide the calculated diazepam dose into 4 daily doses. Supplemental as-required (prn) diazepam may be required to titrate the dose to control withdrawal symptoms, but care must be taken to avoid sedation. The total daily dose of diazepam (regular plus as-required doses) should not exceed 60 mg without specialist advice.

If the oral daily diazepam equivalent cannot be clarified after consultation with community prescribers, pharmacists and real-time prescription monitoring (and there are no [indications for specialist advice](#)), to manage unplanned withdrawal from benzodiazepines, zolpidem or zopiclone, use:

diazepam 5 to 20 mg orally, 2 hourly as required. Titrate to control withdrawal symptoms and monitor for accumulating sedation. Withhold dose if patient is sedated. Maximum dose is 60 mg in the first 24 hours.

The cumulative diazepam dose given in the first 24 hours of titration can be continued on subsequent days of stabilisation, given in divided daily doses. Seek specialist advice if the diazepam dosing requirement in the first 24 hours exceeds 60 mg.

For all patients who have undergone 1 to 2 days of stabilisation, consider whether the diazepam dose can be reduced, depending on the clinical scenario and treatment goals.

Psychosocial interventions for benzodiazepine, zolpidem or zopiclone dependence

Psychosocial interventions for benzodiazepine, zolpidem or zopiclone dependence

[Psychosocial interventions](#) have a key role in managing substance dependence. Lower-dose benzodiazepine use has been reduced by cognitive behavioural therapy in the short-term (at 3 months) when combined with a weaning schedule.

Addressing the underlying reasons for which the patient has been using benzodiazepines, zolpidem or zopiclone (generally anxiety and/or sleep disturbance) is central to case management that recognises the importance of [trauma-informed care](#).

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Overview of disorders of cannabis use

Overview of disorders of cannabis use

Cannabis is derived from the cannabis plant in the form of dried leaves and flowers (cannabis plant material, also called marijuana), resin (hashish) and oil (hashish oil) [\[Note 1\]](#). In Australia, cannabis is mainly smoked in cigarettes (joints) or in water pipes (bongs) or vaporised, although edible products are increasing in popularity. The main psychoactive ingredient is delta-9-tetrahydrocannabinol (THC), a partial agonist at cannabinoid receptors in the central nervous system.

Cannabis is one of the most widely and increasingly used psychoactive substances in Australia and the world. More than 1 in 3 Australian adults have used cannabis in their lifetime, and more than 1 in 10 have done so in the past 12 months. Many people also use other substances, especially tobacco; managing [nicotine dependence](#) is important.

Manage other substance dependence, especially nicotine dependence, in people who use cannabis.

In the last 25 years, a wide variety of synthetic cannabinoid-receptor agonists (SCRAs), a form of novel psychoactive substance, has appeared on the illicit drug market. Most SCRAs are full agonists at cannabinoid receptors, so are more potent than THC (a partial agonist) and have increased risk of harms, including cardiovascular effects. In contrast to the illicitly manufactured SCRAs, other synthetic cannabinoids (eg dronabinol and nabilone) have been approved overseas for therapeutic use. SCRAs, dronabinol and nabilone are expected to cause dependence similar to THC.

Prescribing advice for medicinal cannabinoids (synthetic or plant-derived) is outside the scope of these guidelines [\[Note 2\]](#).

Most people who use cannabis consume relatively small amounts without serious negative consequences, but even occasional use can cause harm. [Table 22.18](#) lists some common effects, including potential harms.

The spectrum of substance use is described in these guidelines by the terms 'hazardous use', 'harmful use' or 'substance dependence', outlined in [Table 22.1](#).

The risk of cannabis dependence is increased in people who start using at a young age and those who use frequently; 20 to 30% of people who use cannabis weekly develop dependence compared to 50% of people who use daily. Cannabis dependence can develop with medicinal use, but data on its prevalence are limited.

Table 22.18 Cannabis effects and potential harms

Short-term effects

mild euphoria, relaxation and disinhibition

altered perception (eg time perception, heightened experiences)

increased appetite

nausea, headache, red sclerae

elevated heart rate

dizziness, impaired balance and coordination

panic, confusion, paranoia

can unmask undiagnosed mental illness

impaired short-term memory and judgement

risk of accidental injury (eg motor vehicle accidents)

Long-term effects

cannabis dependence

cannabinoid hyperemesis syndrome

may increase risk of mental illness

cognitive impairment (largely reversible on stopping)

lower educational attainment

chronic bronchitis [NB1]

may increase risk of oropharyngeal and some lung cancers

cardiovascular damage [NB2]

NB1: Even in the absence of concurrent tobacco use, cannabis increases the risk of chronic bronchitis and may be a risk factor for oropharyngeal and some lung cancers

NB2: Cannabis can cause vasoconstriction, myocardial infarction and stroke, and may affect long-term myocardial function.

Note 1: For other names of cannabis, see the Australian Drug Foundation website

Note 2: Use of medicinal cannabinoids is increasing; see the Australian Therapeutic Goods Administration (TGA) website for advice on prescribing.

Screening and assessment of cannabis use

Screening and assessment of cannabis use

Ask all new patients routinely and others (adolescents and adults) opportunistically and periodically about cannabis use (including prescribed medicinal cannabinoids), as part of a general screen for disorders of substance use and gambling; these disorders are common (and often co-exist) and people are reluctant to disclose them, often due to fear of stigma. Screening and assessment of substance use and addictive behaviours outlines history-taking (including use of the ASSIST-Lite tool), examination, and investigations that should be considered in a broad review of substance use and addictive behaviour.

Useful information to gather on cannabis use includes:

- frequency of use (and of tobacco use, because co-dependence is common)
- factors that precipitate or perpetuate use
- previous efforts to cut down (including severity of any withdrawal effects)
- whether the person or others are concerned about their current use
- potential harms (including neglect of life activities and relationships).

Questionnaires that can be used to gauge the severity of cannabis use include the 5-item Severity of Dependence Scale (SDS), and the 8-item Cannabis Use Disorder Identification Test (CUDIT-R).

Common symptoms of cannabis withdrawal include insomnia, tremor, reduced appetite, irritability and anxiety. Intensity and time course vary considerably between individuals, but symptoms generally start within 1 day of stopping, peak in 2 to 3 days and resolve over 2 to 3 weeks. Although some individuals

experience anger, aggression seems most common in those with a previous history of this behaviour. Cravings and sleep disturbance can persist for months. Past experience of withdrawal symptoms, and severity of dependence are key determinants of the likely severity of a planned withdrawal, which can be explored by asking 'What happened when you last tried to cut down?'.

To identify people who require specialist referral or inpatient withdrawal, ask about the [factors that determine safety of the withdrawal setting](#) for the person and those around them.

Assessment of mental health is important, as psychiatric comorbidities are common among people who use cannabis; see [Mental illness considerations in substance use and addictive behaviours](#).

Overview of management of disorders of cannabis use

Overview of management of disorders of cannabis use

[Overview of substance use and addictive behaviours](#) explains key [principles of care](#). Establishing a therapeutic relationship that engages the person (and ideally those close to them) is central to the management of substance dependence and addictive behaviours.

The most important element of treatment for substance use is a therapeutic relationship.

Specialist advice on any aspects of care for patients with cannabis and other substance dependence is available and contact is encouraged; see [Contact details for substance use clinical advisory services for clinicians](#).

Specialist advice is available by phone on any aspect of the management of substance use; contact is encouraged.

Patients using large amounts of cannabis may present with intoxication, particularly those who use multiple substances. This can pose risk of acute harms to the person (such as self-harm, falls and other accidents) and those around them (through impacts on driving, childcare, fitness to work and acute behavioural disturbances). For advice on managing these risks, see [Ensuring the safety of a person with a disorder of substance use or addictive behaviour](#). For advice on the management of toxicity from synthetic cannabinoid-receptor agonists (SCRAs), see [Management overview for synthetic cannabinoid-receptor poisoning](#).

Management strategies for disorders of cannabis use include:

- [brief interventions](#)
- [gradual reduction of cannabis use](#)
- [management of cannabis withdrawal](#)
- [psychological interventions](#)
- [harm reduction](#).

No drugs have specific approval to treat cannabis dependence. At the time of writing, evidence for the use of drugs in cannabis withdrawal and relapse prevention is limited; however, research is ongoing.

Elements of long-term management relevant to all disorders of substance use are covered in [Long-term care in disorders of substance use and addictive behaviours](#). Management includes addressing comorbid mental health conditions that may precipitate or perpetuate use, such as [anxiety](#), [depression](#) and [insomnia](#) and difficulties with regulation of emotional distress (eg in [personality disorder](#)).

Considerations in managing cannabis use (including specialist referral) may be relevant for [specific populations](#).

Brief interventions for management of disorders of cannabis use

Brief interventions for management of disorders of cannabis use

Evidence is limited for [brief interventions](#) as a standalone approach to reduce cannabis use. However, brief interventions may increase motivation to make changes that support [gradual reduction of cannabis use](#), [planned management of cannabis withdrawal](#), or [psychological interventions](#) to reduce use or prevent relapse; evidence of efficacy is more certain for these treatment approaches. For information on how to approach motivational interviewing, see [Brief interventions for disorders of substance use and addictive behaviours](#). Brief advice can include [harm reduction measures](#).

Gradual reduction of cannabis use

Gradual reduction of cannabis use

Planned gradual reduction in cannabis use is usually straightforward and can often be managed with gradual changes to daily use (eg reducing the number of bongs or joints used each day, decreasing the amount of cannabis in each bong or joint, or increasing the interval between waking and use each day). Making the changes over 1 to 4 weeks may reduce the likelihood and severity of withdrawal and the need for medications to manage symptoms, compared to stopping abruptly. [Psychological interventions](#) have a key role in assisting gradual reduction.

Cannabis withdrawal

Cannabis withdrawal

Planned cannabis withdrawal over 7 to 10 days may be preferred to gradual reduction if a person is highly motivated to stop using completely, has a reason to stop suddenly (eg a court date, health event, upcoming inpatient admission) or if gradual reduction has not been successful. The safety of the setting for planned cannabis withdrawal (at home, in a community facility or in hospital) depends on the likelihood of severe withdrawal and the extent of the patient's social supports. For guidance on selecting a withdrawal setting, see [Choice of setting for planned withdrawal management in substance use](#). Seek specialist advice from a [clinical advisory service](#) if unsure about assessing these factors.

Tobacco smoking may predict poorer outcomes for planned cannabis withdrawal. Concurrent management of [nicotine dependence](#) is important.

Supportive management, education and counselling without medication are often enough to manage cannabis withdrawal. No drugs (including medicinal cannabinoids) are approved for the management of cannabis withdrawal. Cannabinoid agonists appear to have promise in the treatment of withdrawal; these include nabiximols (an extract of cannabis plants that contains tetrahydrocannabinol and cannabidiol), and the synthetic cannabinoids, dronabinol and nabilone.

Medications can be used to treat withdrawal symptoms if the patient is distressed, and may increase the likelihood of maintaining abstinence if this is the treatment goal. Evidence to guide choice of regimen is limited. Seek specialist advice from a [clinical advisory service](#) if symptoms are severe or the patient has a history of severe withdrawal.

[Table 22.19](#) summarises medications that can be used if severe or distressing symptoms of cannabis withdrawal occur or are predicted by previous episodes. Short-term use (for up to 7 days) of benzodiazepines or antipsychotics can reduce severe anxiety, restlessness or irritability, while minimising risks of dependence. For managing nausea, use of mirtazapine or olanzapine is as effective or superior to 5-HT₃-receptor antagonists (eg ondansetron).

Unplanned cannabis withdrawal can be managed with a similar approach to management of planned withdrawal, but is more likely to be severe and require medication.

Table 22.19 Medications for short-term use to manage symptoms of cannabis withdrawal

Symptom	Medications
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insomnia	benzodiazepines, zolpidem, zopiclone or promethazine see Medications to manage insomnia in cannabis withdrawal diazepam, mirtazapine or olanzapine
restlessness, anxiety, irritability	see Medications to manage restlessness, anxiety or irritability in cannabis withdrawal hyoscine
abdominal cramps	see Antispasmodics for pain in palliative care for doses paracetamol, NSAIDs
physical pain, headaches	see Oral drugs for mild, acute nociceptive pain in adults mirtazapine, olanzapine, promethazine or metoclopramide
nausea	see Antiemetic drugs
NSAIDs = nonsteroidal anti-inflammatory drugs	

Medications to manage restlessness, anxiety or irritability in cannabis withdrawal

Medications to manage restlessness, anxiety or irritability in cannabis withdrawal

Before prescribing, see advice in [Safe prescribing and supply in substance withdrawal](#) on measures to reduce risk of dependence, overdose and harm from sedation. A suitable regimen for outpatient management of **anxiety or agitation** in cannabis withdrawal is:

1diazepam 5 mg orally, 6-hourly as required. Review every 1 to 3 days to assess symptom control. Maximum duration 7 days

OR

1mirtazapine 7.5 to 15 mg orally, at night as required. Review every 1 to 3 days to assess symptom control. Maximum duration 7 days

OR

1olanzapine 2.5 mg orally, twice daily as required. Review every 1 to 3 days to assess symptom control. Maximum duration 7 days.

Managing insomnia in cannabis withdrawal

Managing insomnia in cannabis withdrawal

Temazepam, zolpidem or zopiclone may be used if the main symptom in cannabis withdrawal is severe **insomnia** but these should not be used together, nor with another benzodiazepine or antipsychotic, without specialist advice. Before prescribing, see advice in [Safe prescribing and supply in substance withdrawal](#) on measures to reduce risk of dependence, overdose and harm from sedation. Suitable regimens to manage insomnia in cannabis withdrawal are:

1temazepam 10 to 20 mg orally, at night as required. Maximum duration 7 days

OR

1zolpidem immediate-release 10 mg orally, at night as required. Maximum duration 7 days

OR

1zopiclone 7.5 mg orally, at night as required. Maximum duration 7 days.

Psychological and behavioural interventions for insomnia may also be helpful; see the [Psychotropic guidelines](#) for advice, including a printable patient handout on good sleep practices.

Psychosocial interventions for long-term management of disorders of cannabis use

Psychosocial interventions for long-term management of disorders of cannabis use

The most consistent evidence for psychosocial interventions to reduce cannabis use and the severity of dependence is for [psychological interventions](#) such as cognitive behavioural therapy (CBT) and motivational enhancement therapy (MET).

Internet and computer-based psychological interventions are effective adjuncts to psychological therapies in reducing regular (but nondependent) cannabis use and may make treatment more accessible. CBT also reduces risk of relapse after withdrawal.

Support may be accessed through free online and phone services such as [Counselling Online](#) and the [Alcohol and Drug Information Service hotline](#) in each state and territory.

Harm reduction in cannabis use

Harm reduction in cannabis use

Overview of harm reduction in cannabis use

Overview of harm reduction in cannabis use

Offer advice on harm reduction to people who use cannabis. Some people will not choose to stop using but will consider measures to limit risk and may be open to revisiting their options for behavioural change later, particularly if harm reduction information is provided in a nonjudgemental way. The [general principles of care](#) in chronic disease management are important, particularly maintaining an engaging therapeutic relationship and a sense of hope.

Strategies to consider for harm reduction include:

- avoiding cannabis use when experiencing symptoms of anxiety or paranoia (which can be exacerbated by use) and seeking [psychosocial support](#)
- avoiding cannabis use together with other drugs that heighten anxiety or paranoia (eg [stimulants](#), [lysergic acid diethylamide \[LSD\]](#))
- use of a dry-herb vaporiser approved by the Australian Therapeutic Goods Administration (TGA) in preference to smoking cannabis; these can be purchased at a pharmacy (advise patients not to use cannabis oil in a vaping unit)
- avoiding driving under the influence of cannabis, including during withdrawal
- being aware that oral forms of cannabis (also known as ‘edibles’) are absorbed more slowly than inhaled; if a person uses an oral form of cannabis, they should try a small amount and wait 1 to 2 hours before having more to avoid toxicity.

[Table 22.5](#) is a printable patient information sheet on ways to get help and reduce harm from the use of cannabis and other drugs.

Harm reduction also involves management of complications of cannabis use, such as:

- [cannabinoid hyperemesis syndrome](#)
- exacerbations of [mental illness](#)
- chronic bronchitis.

Management of disorders of cannabis use in specific populations

Management of disorders of cannabis use in specific populations

Specific considerations (including the need for specialist referral) apply in the management of cannabis use in patients who are pregnant or breastfeeding.

For other specific populations (including young people, those with mental illness and people with chronic pain), see Considerations for specific populations in substance use and addictive behaviours.

Management of cannabis use during pregnancy

Management of cannabis use during pregnancy

In most developed countries, approximately 10% of pregnant people smoke cannabis, most often in conjunction with tobacco. Animal and human research suggests that permanent neurobehavioural effects can result from cannabis exposure *in utero*, and the nature of these depends on the gestation at exposure. Cannabis use in pregnancy is associated with low birth weight and infants being small for gestational age. This may reflect the high prevalence of tobacco use in people who use cannabis. However, the association with low birth weight and small-for-gestational-age infants may be independent of concurrent tobacco use. Children of people who used cannabis while pregnant may have cognitive (especially visuospatial) deficits, greater impulsivity and hyperactivity and higher rates of depression than people without prenatal cannabis exposure. Interpretation of data on harms from cannabis exposure *in utero* requires caution owing to possible confounding factors.

Explore with pregnant patients their understanding of the risks that cannabis may pose to the fetus, and use motivational interviewing approaches and psychological interventions to support positive change. If they smoke tobacco as well as cannabis, intervention should focus on supporting the person to stop the tobacco use because this has the clearest evidence of causing fetal harm.

Medications to manage cannabis withdrawal symptoms should only be considered after specialist consultation.

Education and psychological interventions are key to helping pregnant patients stop using cannabis.

With the growing use of medicinal cannabinoids and decriminalisation of cannabis in many countries, there has been an incorrect perception in the general population that cannabis may be appropriate for the treatment of morning sickness. Cannabis and medicinal cannabinoids are not recommended to treat nausea and vomiting during pregnancy.

Cannabis is **not** recommended in any form (including medicinal cannabinoids) to treat nausea or vomiting in pregnancy.

Management of cannabis use during breastfeeding

Management of cannabis use during breastfeeding

Cannabis passes readily into breastmilk. Animal studies have demonstrated adverse effects on the rapidly developing infant brain. Human data are difficult to interpret because of confounding factors (including effects of second-hand smoke) but appear to support a detrimental neurocognitive impact. No amount of cannabis use is considered safe during breastfeeding.

No amount of cannabis is considered safe in breastfeeding.

Educational and psychological interventions such as cognitive behavioural therapy and motivational enhancement therapy are key to helping breastfeeding patients to stop using cannabis. Medication to manage cannabis withdrawal symptoms should only be considered after specialist consultation.


[\[X\] Close](#)

Overview of disorders of GHB use

Overview of disorders of GHB use

Gamma-hydroxybutyrate (GHB, also known as 'GBH', 'fantasy', 'G', 'Gina' or 'liquid ecstasy') is a sedative and anaesthetic drug usually ingested as a precursor drug, such as gamma-butyrolactone (GBL) or 1,4-butanediol, which the body rapidly converts to GHB. It is used therapeutically (as sodium oxybate) overseas but is not approved by the Australian Therapeutic Goods Administration (TGA).

GHB is usually distributed as a colourless liquid and is taken orally. Effects are very similar to those of alcohol or benzodiazepines and include sedation, euphoria, disinhibition and amnesia. Like amyl nitrate and stimulants, GHB is used to enhance sexual experience ('chemsex'). Because of its sedative effects, it is also sometimes used to mitigate the effects of [stimulants](#) such as metamfetamine. Prevalence of GHB use in the previous 12 months is estimated at 0.1% of the Australian population.

The spectrum of substance use in these guidelines is described by the terms 'hazardous use', 'harmful use' or 'substance dependence', outlined in [Table 22.1](#).

Potential harms from GHB and its precursors include:

- toxicity—overdose is common because the difference between the doses for desired and toxic effects is very small; vulnerability while intoxicated (including to physical and sexual assault) is also a risk
- withdrawal—severe withdrawal may require treatment in an intensive care unit
- local tissue damage—oral burns can occur from residual sodium hydroxide used in manufacturing.

GHB overdose is characterised by confusion, agitation, hallucinations, respiratory depression, hypotension, bradycardia, involuntary movements (sometimes interpreted as seizures) and coma [\[Note 1\]](#). Overdose typically lasts only a few hours with spontaneous (often abrupt) waking from coma. GHB overdoses are common; some are fatal. The risk of toxicity and respiratory depression is increased if GHB is taken with other sedatives such as alcohol. Concurrent alcohol use can cause delayed onset of GHB toxicity.

GHB dependence can present with withdrawal similar to severe alcohol withdrawal.

Note 1: Patients experiencing GHB overdose may have the drug in their possession because of their need to use it frequently; it is useful to be aware of this when seeking the cause of a person's intoxication.

Screening and assessment of GHB use

Screening and assessment of GHB use

Ask all new patients routinely and others (adolescents and adults) opportunistically and periodically about GHB use, as part of a general screen for disorders of substance use and gambling; these disorders are common (and often co-exist) and people are reluctant to disclose them, often due to fear of stigma. [Screening and assessment of substance use and addictive behaviours](#) outlines history-taking (including use of the [ASSIST-Lite tool](#)), examination, and investigations that should be considered in a broad review of substance use and addictive behaviour.

Overview of management of disorders of GHB use

Overview of management of disorders of GHB use

[Overview of substance use and addictive behaviours](#) explains key [principles of care](#) for a person with a disorder of substance use. Establishing a therapeutic relationship that engages the person (and ideally those close to them) is central to the management of substance dependence and addictive behaviours.

The most important element of treatment for substance use is a therapeutic relationship.

Specialist advice on any aspects of care for patients with GHB or other substance dependence is available and contact is encouraged; see [Contact details for substance use clinical advisory services for clinicians](#).

Specialist advice is available by phone on any aspect of the management of substance use; contact is encouraged.

Treatment of GHB overdose is supportive; see [Gamma-hydroxybutyrate \(GHB\) poisoning](#). Patients may display aggressive behaviour on waking from coma. For advice on ensuring the safety of patients with disorders of substance use, including during an acute behavioural disturbance, see [Ensuring the safety of a person with a disorder of substance use or addictive behaviour](#).

Offer all patients who use GHB [brief intervention](#); brief advice can include information on harm reduction strategies as outlined in [Figure 22.10](#). Seek specialist advice if the person uses more than one substance (polysubstance use) or has features of GHB [dependence](#), to minimise the risk of complications including withdrawal. For GHB dependence, [managed withdrawal](#) is recommended (rather than gradual dose reduction) because it is difficult to make small dose adjustments without risking unplanned withdrawal.

Figure 22.10 Safer use of gamma-hydroxybutyrate (GHB)—patient information sheet

[Printable figure](#)

Seek medical attention immediately if you or someone with you has taken too much GHB.

Signs of a GHB overdose include slow and shallow breathing, irregular heart rate, vomiting, sweating, being irritable or very anxious, fitting, losing consciousness.

Act in an emergency—call 000 for an ambulance if someone has collapsed or is unconscious.

Place an unconscious person on their side in the recovery position to keep their airway open.

Start CPR (cardiopulmonary resuscitation) if the unconscious person stops breathing or has abnormal breathing (eg they are gasping with gaps between gasps). The 000 operator will tell you what to do.

Do not try to reverse the effects of GHB with other drugs.

Prepare GHB safely

Never drink GHB without diluting it.

To avoid drinking GHB by mistake, add food colouring and label the container. Never keep GHB in drink bottles or leave it unattended.

Always measure GHB doses accurately using a standard device (eg syringe or pipette).

Use GHB responsibly

Use GHB in a safe place; have someone who has not taken drugs watch you for any signs of overdose.

Do not drive after using GHB.

Make sure the people with you know that you use GHB and how to give you first aid; it is common to become unconscious on GHB. You might want to write 'GHB' on the back of your hand to help alert first-aiders.

Avoid using GHB with other sedative drugs (eg ketamine) or alcohol; the risk of overdose is much higher.

Do not re-dose too soon. Wait for at least 2 hours after you feel the effects of GHB; this reduces the risk of overdose.

Sleep on your side after using any substances in case you are sick.

Avoid frequent use, especially daily use. GHB is addictive and dependence (the need to keep using) can occur quickly.

Manage long-term use of GHB

If you are dependent and you miss a dose of GHB or suddenly reduce your dose, you might experience severe withdrawal symptoms (eg severe anxiety, confusion, fitting). Go to an emergency department if you feel unwell after missing or reducing a dose.

If planning to stop GHB use, get medical advice. Do not try to quit GHB suddenly on your own. If you want to reduce your dose, do so in very small amounts until you can get medical advice.

Stabilise your GHB use. Keep a record of your doses and the times you use in your phone or a diary.

Management of GHB withdrawal

Management of GHB withdrawal

Heavy or regular (eg daily) use of GHB (or precursors) can lead to dependence. As GHB has a short half-life, people who use it very regularly may be taking multiple daily doses (some every few hours). Symptoms of GHB withdrawal in a dependent person can start within 1 to 4 hours of stopping. Advise patients not to attempt to reduce or stop their GHB use without medical advice because it is difficult to make even small dose adjustments without risking unplanned withdrawal.

Common symptoms of GHB withdrawal include anxiety, agitation, sweating, tremor, tachycardia, hypertension, hyperthermia, cravings and insomnia. Severe withdrawal can cause myoclonus, bradycardia, autonomic instability, perceptual disturbance, auditory or visual hallucinations, confusion, acute delirium, seizures, rhabdomyolysis and renal impairment.

Factors that predict severe GHB withdrawal include:

- frequent dosing (eg less than 4 hourly), including waking at night to dose
- higher doses (eg more than 15 mL GHB in 24 hours)
- previous severe withdrawal
- no days without use in the previous 6 weeks
- concurrent use of a stimulant (eg metamfetamine); this increases seizure risk on withdrawal from GHB.

Patients planning managed withdrawal who are at risk of severe withdrawal or those with significant concurrent medical or mental health problems should be managed in hospital. Severe GHB withdrawal may require treatment in an intensive care unit. See [Withdrawal management in disorders of substance use](#) for considerations in ensuring safety in choice of environment, prescribing and the supply of medications for substance withdrawal. Seek specialist advice regarding any concerns about evaluating these factors.

Benzodiazepines are the primary treatment for GHB withdrawal. Dosages depend on the severity of withdrawal.

Outpatient regimens for management of GHB withdrawal**Outpatient regimens for management of GHB withdrawal**

For **outpatient management** of withdrawal from GHB (with monitoring by a clinician skilled in withdrawal management), a suitable regimen is:

diazepam 10 mg orally, 2-hourly, starting within 2 hours of the last dose of GHB if the patient does not have signs of sedation. Continue until light sedation is evident and agitation is controlled. Do not exceed 40 mg daily. Once agitation is controlled, gradually reduce the dose over 5 to 7 days.

Inpatient regimens for management of GHB withdrawal**Inpatient regimens for management of GHB withdrawal**

For **inpatient management** of planned or unplanned withdrawal from GHB (with monitoring by staff skilled in withdrawal management), a suitable regimen is:

diazepam 10 mg orally, 2-hourly, starting within 2 hours of the last dose of GHB if the patient does not have signs of sedation. Continue until light sedation is evident and agitation is controlled. Seek specialist advice before exceeding 60 mg on the first day. Once agitation is controlled, gradually reduce the dose over 5 to 7 days.

Baclofen can also reduce the severity of GHB withdrawal and the dose of diazepam required. Baclofen can be used concurrently with diazepam to treat GHB withdrawal in an inpatient. An example regimen is:

baclofen 10 to 25 mg orally, 8-hourly. Gradually reduce the dose during the second week to complete treatment in 14 days. Seek expert advice if withdrawal symptoms are prolonged.

Warn patients there is an increased risk of overdose after GHB withdrawal because their tolerance will be reduced. [Long-term care](#) aims to reduce relapse and support the person to maintain good health and wellbeing.



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Overview of disorders of hallucinogen use

Overview of disorders of hallucinogen use

Hallucinogens include lysergic acid diethylamide (LSD), psilocybin (from 'magic' mushrooms) and certain analogues of amphetamine. They are usually taken orally. A wide range of novel drugs including phenethylamines and tryptamines can also induce hallucinogenic effects. Ketamine, an anaesthetic, also has hallucinogenic properties; for the management of ketamine use, see [Overview of disorders of ketamine use](#).

The spectrum of substance use in these guidelines is described by the terms 'hazardous use' and 'harmful use' as outlined in [Table 22.1](#).

Intoxicating effects of hallucinogens include hallucinations, depersonalisation, hypersensitivity to external stimuli and changes in perception of time; psychotic behaviour may be observed. Panic, feelings of loss of control and labile mood may be prominent. Sympathomimetic effects (eg hypertension, tremor) may be prominent in overdose. For advice on hallucinogenic drug toxicity, see [Sympathomimetic toxidrome](#) and [Novel hallucinogenic drug poisoning](#).

The duration of hallucinogen action depends on the drug consumed and the dose taken. In some instances, hallucinations and other effects may persist for several days.

Hallucinogenic drugs are normally used sporadically and withdrawal symptoms are not seen. Flashbacks may occur; these are recurrence of experiences simulating those of the previous intoxicated state but occurring after the intoxication episode.

Management of disorders of hallucinogen use

Management of disorders of hallucinogen use

Hallucinogen use may put a person at risk of acute harms (from self-harm, falls, other accidents, suicide) and risk the safety of others, particularly if the person is agitated or aggressive. For advice on managing these risks, including acute behavioural disturbances, see [Ensuring the safety of a person with a disorder of substance use or addictive behaviour](#). For general advice on harm reduction in substance use (including a printable patient information sheet), see [Harm reduction in substance use and addictive behaviour](#).

[Specialist advice](#) is recommended for managing psychosis, particularly because some hallucinogens have anticholinergic effects that may be enhanced by antipsychotic medications.

References

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Overview of disorders of ketamine use

Overview of disorders of ketamine use

Ketamine (also known as 'K', 'super K', 'vitamin K', 'special K') is an anaesthetic used in human and veterinary medicine. Nonmedical use also occurs by a variety of routes because ketamine has dissociative and hallucinogenic intoxicating properties. Nonmedical use is use that does not align with the directed use; examples include use to become intoxicated or to treat a symptom other than the clinician directed.

The spectrum of substance use in these guidelines is described by the terms 'hazardous use' and 'harmful use,' outlined in [Table 22.1](#).

Intoxicating effects of ketamine include relaxation, changed perceptions of time and space, dissociation, visual and auditory hallucinations, drowsiness, slurred speech, blurred vision and confusion. Higher doses can produce severe dissociation (the 'k-hole'), more intense sensory misperceptions and hallucinations and sedation.

Ketamine overdose typically lasts for 1 to 2 hours and is characterised by hallucinations, delirium, cardiovascular and respiratory stimulation, and hyperthermia. Respiratory depression can occur, particularly with intravenous administration, but is uncommon. Treatment of overdose is supportive; for advice on management, see [Resuscitation for poisonings](#).

Management of disorders of ketamine use

Management of disorders of ketamine use

The dissociative and analgesic effects of ketamine can put a person at risk of serious injury and death, and risk the safety of others. For advice on managing these risks, see [Ensuring the safety of a person with a disorder of substance use or addictive behaviour](#).

Heavy and regular use of ketamine can result in chronic harms such as hallucinatory flashbacks, headaches, abdominal cramping and painful ulcerative cystitis. Tolerance (a requirement to use more drug to achieve the desired effects) may occur; however, stopping ketamine does not appear to produce a classic withdrawal syndrome. Nevertheless, people trying to reduce or stop regular use of ketamine may experience cravings to use the drug and should be offered [harm reduction advice for substance use](#) (including a printable patient leaflet) and referral to a [specialist service](#).

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[\[X\] Close](#)

Overview of disorders of pregabalin and gabapentin use

Overview of disorders of pregabalin and gabapentin use

Pregabalin and gabapentin (gabapentinoids) are gamma amino butyric acid (GABA analogues) but do not interact significantly with GABA receptors; they act primarily by inhibiting voltage-gated calcium channels, indirectly reducing neuronal excitability and firing. Both are often overprescribed and used for unapproved indications, but nonmedical use is more common with pregabalin because gabapentin absorption becomes saturated at higher doses, reducing the effect of escalating doses. Nonmedical use describes use that does not align with the directed use. For example, a person may use pregabalin or gabapentin to treat a symptom other than the clinician intended, or to become intoxicated.

The spectrum of substance use in these guidelines is described by the terms 'hazardous use', 'harmful use' or 'substance dependence', outlined in [Table 22.1](#).

Nonmedical use of pregabalin (in particular) and gabapentin is increasing in Australia and overseas. In 2019, an estimated 4.3% of people taking any pain medication reported nonmedical use of pregabalin or gabapentin within the last 12 months.

Case reports describe dependence with chronic use of very high doses of pregabalin (up to 9 g per day). Other substance use is a risk factor for pregabalin or gabapentin dependence.

The harms of nonmedical use of pregabalin and gabapentin include:

- enhanced toxicity, including fatal sedation, especially when taken with opioids, benzodiazepines or alcohol
- seizures in acute withdrawal
- delirium following dose increases or in withdrawal.

Pregabalin and gabapentin lower the threshold at which opioid overdoses are fatal. While any combination of these drugs may be problematic, risk of toxicity is particularly high if pregabalin doses more than 300 mg daily or opioid doses more than the [oral morphine equivalent](#) of 100 mg daily are used. Risk of toxicity escalates markedly if pregabalin or gabapentin is used with 2 or more sedative drugs (eg opioids, benzodiazepines, alcohol). Risk of death is lower for patients using pregabalin or gabapentin with prescribed buprenorphine or methadone than for those who use other opioids. For advice on managing pregabalin or gabapentin toxicity, see [Pregabalin and gabapentin poisonings](#).

Combination of pregabalin or gabapentin and an opioid lowers the threshold at which opioid overdose is fatal.

Significant withdrawal symptoms, similar to benzodiazepine and selective serotonin reuptake inhibitor (SSRI) withdrawal syndromes, have been reported with both tapering and abruptly stopping pregabalin or gabapentin.

Screening and assessment of pregabalin and gabapentin use

Screening and assessment of pregabalin and gabapentin use

Ask all new patients routinely and others (adolescents and adults) opportunistically and periodically about medication use, as part of a general screen for disorders of substance use and gambling; these disorders are common (and often co-exist) and patients are reluctant to disclose them, often due to fear of stigma.

[Screening and assessment of substance use and addictive behaviours](#) outlines history-taking (including use of the [ASSIST-Lite tool](#)), examination, and investigations that should be considered in a broad review of substance use and addictive behaviour.

If pregabalin or gabapentin use is identified, specific questions to assess use in more detail include:

- duration and dosage—prolonged use increases the likelihood of withdrawal symptoms on stopping, but withdrawal can follow short-term use (for a few weeks) of higher doses (eg more than 150 mg twice daily of pregabalin)
- use of any other substances (alcohol, opioids, benzodiazepines, over-the-counter medications, complementary therapies, other prescribed or illicit drugs) to assess risk of fatal sedation or other dangerous interactions
- factors that precipitate or perpetuate use—assess for undertreated [anxiety](#), difficulties with regulation of emotional distress (eg as can occur in [personality disorder](#)), [insomnia](#), other [disorders of substance use and pain](#)
- withdrawal symptoms experienced on previous attempts to reduce usage.

Pregabalin and gabapentin withdrawal symptoms include agitation, anxiety, irritability, sweating, gastrointestinal symptoms, hypertension, tachycardia, insomnia, delirium including confusion and catatonia, and seizures, including status epilepticus. Previous withdrawal is the best **predictor of severe or complicated withdrawal**; other risk factors are use of higher doses of pregabalin or gabapentin, seizure disorders, psychosis, polysubstance use (use of more than one substance), and comorbid physical and mental health conditions, such as pre-existing anxiety. Anxiety that persists for more than 1 week after stopping pregabalin or gabapentin is likely to reflect an anxiety disorder or other illness rather than acute withdrawal.

Management of disorders of pregabalin and gabapentin use

Management of disorders of pregabalin and gabapentin use

[Overview of substance use and addictive behaviours](#) explains key [principles of care](#) for a person with a disorder of substance use. Establishing a therapeutic relationship that engages the person (and ideally those close to them) is central to the management of substance dependence.

The most important element of treatment for substance use is a therapeutic relationship.

Specialist advice on any aspects of care for patients with disorders of substance use is available and contact is encouraged; see [Contact details for substance use clinical advisory services for clinicians](#).

Specialist advice is available by phone on any aspect of the management of substance use; contact is encouraged.

Weaning of pregabalin or gabapentin can be undertaken in the [outpatient](#) setting for most people; [inpatient rapid withdrawal](#) can be considered for some patients. Consider specialist advice or referral to manage withdrawal of pregabalin or gabapentin if the patient has [predictors or evidence of severe or complex withdrawal](#).

Offer alternative strategies (which may include group or individual [psychosocial support](#)) for managing other factors that precipitate or perpetuate prolonged use of pregabalin or gabapentin, such as [insomnia](#), [anxiety](#) and chronic pain.

In patients using pregabalin or gabapentin for chronic pain, pain symptoms may be exacerbated as part of a withdrawal syndrome when stopping; seek advice on management as alternative analgesics may not be helpful in managing such conditions and continued use of pregabalin or gabapentin may be required with lower doses and closer supervision.

Substance use can pose risk of acute harms to the person (such as self-harm, falls and other accidents, suicide) and those around them (through impacts on driving, childcare, fitness to work and acute behavioural disturbances). For advice on managing these risks, see [Ensuring the safety of a person with a disorder of](#)

substance use or addictive behaviour. For harm reduction measures to consider for any person with a disorder of substance use or addictive behaviour, see Harm reduction.

Elements of long-term management relevant to all disorders of substance use are covered in Long-term care in disorders of substance use and addictive behaviours.

Considerations for specific populations may be relevant in managing disorders of pregabalin and gabapentin use.

Planned outpatient weaning of pregabalin and gabapentin

Planned outpatient weaning of pregabalin and gabapentin

Before planning outpatient weaning, seek specialist advice or refer if any predictors or evidence of severe or complex withdrawal are present. Patients sometimes overestimate their consumption; seek corroboration of the doses they are being prescribed using real-time prescription monitoring if available, although this will not identify any drugs acquired illicitly. Observation in a hospital or specialist outpatient setting can help assess a patient's tolerance by establishing whether they require their stated dose to avoid withdrawal; at least 24 hours of monitoring is recommended for patients who report that they take doses at the maximum end of the therapeutic range.

International guidelines recommend gradual outpatient weaning at a rate of 50 to 100 mg pregabalin every 7 days or 300 mg gabapentin every 4 days. Faster outpatient weaning can be considered if a patient has only used pregabalin or gabapentin for a short period.

Consider staged supply (dispensing small amounts of medication [eg daily, second daily or weekly]) and use of real-time prescription monitoring systems, if available.

Planned inpatient withdrawal of pregabalin and gabapentin

Planned inpatient withdrawal of pregabalin and gabapentin

A person may seek rapid inpatient withdrawal of pregabalin or gabapentin if a specific circumstance (eg the start of rehabilitation) requires abstinence. Rapid inpatient withdrawal may also be required for some patients using multiple substances (polysubstance use) as part of rationalising their use. Evidence is lacking to guide inpatient rapid withdrawal of high-dose pregabalin or gabapentin. Seek specialist advice and do not undertake without monitoring by clinicians experienced in withdrawal management.

For **inpatient rapid withdrawal of high-dose pregabalin**, expert opinion suggests a reasonable approach is:

pregabalin 900 mg (or usual total daily dose if it is less than 900 mg) orally, daily in 3 divided doses on the first day. Then reduce total daily dose by 75 mg each day. Use 2 divided doses for total daily doses lower than 600 mg.

If a benzodiazepine is required to manage withdrawal symptoms, add:

diazepam 5 to 10 mg orally, 8-hourly as required. Maximum duration for diazepam use is 48 hours after the last dose of pregabalin.

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[\[X\] Close](#)

Overview of disorders of quetiapine and other antipsychotic use

Overview of disorders of quetiapine and other antipsychotic use

Concern is growing about the use of some antipsychotics, particularly quetiapine, for indications that are not approved by the Australian Therapeutic Goods Administration (TGA). Quetiapine is being prescribed more often, using low doses for unapproved indications, because of its anxiolytic properties and the perception that it is safer than benzodiazepines. However, quetiapine and other antipsychotics have [long-term adverse effects](#) and the potential for nonmedical use. Nonmedical use of quetiapine describes use that does not align with the directed use. For example, a person may use quetiapine to treat a symptom other than the clinician directed, or to become intoxicated. Most nonmedical use of quetiapine is oral, but there are reports of intranasal use, especially in prisons, and of it being smoked. At the time of writing, there have also been limited reports of nonmedical use of the antipsychotic, olanzapine. Sedating properties increase the likelihood of nonmedical antipsychotic use.

Quetiapine toxicity can result in sedation, respiratory depression, seizures, cardiovascular effects and anticholinergic toxidrome. For advice on assessment and management of toxicity from quetiapine and other antipsychotics, see [Antipsychotic drug poisoning](#).

Check [real-time prescription monitoring programmes](#) before prescribing quetiapine [\[Note 1\]](#); olanzapine is not currently included in these programmes. Consider staged supply (dispensing small amounts of medication [eg daily, second daily or weekly]) if nonmedical use is considered likely.

The spectrum of substance use is described in these guidelines by the terms 'hazardous use', 'harmful use' or 'substance dependence', outlined in [Table 22.1](#). Quetiapine and other antipsychotic dependence can manifest as withdrawal after abruptly stopping the drug (following use as prescribed or nonmedical use).

Note 1: At the time of writing, real-time monitoring of quetiapine is required in some jurisdictions only.

Screening and assessment of quetiapine and other antipsychotic use

Screening and assessment of quetiapine and other antipsychotic use

Ask all new patients routinely and others (adolescents and adults) opportunistically and periodically about medication use, as part of a general screen for disorders of substance use and gambling; these disorders are common (and often co-exist) and patients are reluctant to disclose them, often due to fear of stigma. [Screening and assessment of substance use and addictive behaviours](#) outlines history-taking (including use of the [ASSIST-Lite tool](#)), examination, and investigations that should be considered in a broad review of substance use and addictive behaviour. Consider an electrocardiogram (ECG) to detect QTc prolongation in patients taking high doses of quetiapine.

If quetiapine or other antipsychotic use is identified, specific questions to assess use in more detail include:

- duration and dosage—prolonged use of recommended or higher doses can result in withdrawal symptoms on stopping
- use of any other substances (alcohol, over-the-counter medications, complementary therapies, other prescribed or illicit drugs) to assess risk of fatal sedation or other dangerous interactions; see [Quetiapine poisoning](#) for clinical presentation of central nervous system, cardiac and anticholinergic effects
- factors that precipitate or perpetuate use—assess for undertreated [anxiety](#) [\[Note 2\]](#), untreated or undertreated [psychosis](#), difficulties with regulation of emotional distress (eg in [personality disorder](#)), [insomnia](#) and other [disorders of substance use](#)
- withdrawal symptoms experienced on previous attempts to reduce usage.

Symptoms reported after abrupt stopping of quetiapine include dysphoria, anxiety and irritability, palpitations, tachycardia, dizziness, light-headedness, hypertension, nausea and vomiting, insomnia, sweating, movement disorders, craving, fatigue and headache.

Note 2: Pre-existing anxiety increases the likelihood of severe withdrawal symptoms on stopping; anxiety that persists for more than 1 week after stopping quetiapine or other antipsychotics is likely to reflect an anxiety disorder or other illness rather than persisting withdrawal.

Management of disorders of quetiapine and other antipsychotic use

Management of disorders of quetiapine and other antipsychotic use

Overview of substance use and addictive behaviours explains key principles of care for a person with a disorder of substance use. Establishing a therapeutic relationship that engages the person (and ideally those close to them) is central to the management of substance dependence.

The most important element of treatment for substance use is a therapeutic relationship.

Specialist advice on any aspects of care for patients with disorders of substance use is available and contact is encouraged; see Contact details for substance use clinical advisory services for clinicians.

Specialist advice is available by phone on any aspect of the management of substance use; contact is encouraged.

No studies have been published on managing quetiapine or other antipsychotic withdrawal with dependence. General advice is available in Stopping antipsychotics. Gradual weaning and symptomatic treatment are likely to be best tolerated. Other sedatives, anxiolytics or hypnotics should be used with caution for short periods of time due to the risk of a patient developing dependence on another medication.

Offer alternative strategies for managing factors that precipitate or perpetuate prolonged antipsychotic use, such as insomnia, difficulties with emotional regulation (eg as can occur with personality disorder), and anxiety.

Substance use can pose risk of acute harms to the person (such as self-harm, falls and other accidents, suicide) and those around them (through impacts on driving, childcare, fitness to work and acute behavioural disturbances). For advice on managing these risks, see Ensuring the safety of a person with a disorder of substance use or addictive behaviour. For harm reduction measures to consider for any person with a disorder of substance use or addictive behaviour, see Harm reduction.

Considerations for specific populations may be relevant in managing disorders of antipsychotic use.

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Overview of disorders of sedating antihistamine use

Overview of disorders of sedating antihistamine use

Oral sedating antihistamines are sometimes used in a way that does not align with the directed use (nonmedical use). For example, a person may use sedating antihistamines to treat a symptom other than the clinician intended, or to become intoxicated. Nonmedical use can apply to sedating antihistamines used alone, in compound preparations (eg cough and cold preparations), or as part of a disorder of polysubstance use (use of more than one substance). Sedating antihistamines available in Australia include alimemazine (trimeprazine), brompheniramine, chlorphenamine, cyclizine, cyproheptadine, dexchlorpheniramine, dimenhydrinate, diphenhydramine, doxylamine and promethazine.

The spectrum of substance use is described in these guidelines by the terms 'hazardous use', 'harmful use' or 'substance dependence', outlined in [Table 22.1](#). Hazardous or harmful use of sedating antihistamines occurs most often; dependent use is rare. Prolonged daily use of recommended doses is most common, and is seen in people seeking to manage conditions such as [anxiety](#), [insomnia](#), chronic itch or cravings for other substances.

The harmful effects of sedating antihistamine intoxication include fatal sedation, seizures, delirium and [anticholinergic toxidrome](#) and possibly [serotonergic toxidrome](#) with some antihistamines [\[Note 1\]](#). These effects are increased by concurrent use of other sedating substances (eg opioids, benzodiazepines, alcohol).

For advice on the management of sedating antihistamine toxicity, see [Antihistamine poisoning: sedating antihistamines](#).

Note 1: The antihistamine cyproheptadine is used in the treatment of serotonergic toxidrome.

Screening and assessment of sedating antihistamine use

Screening and assessment of sedating antihistamine use

Ask all new patients routinely and others (adolescents and adults) opportunistically and periodically about medication use, as part of a general screen for disorders of substance use and gambling; these disorders are common (and often co-exist) and patients are reluctant to disclose them, often due to fear of stigma. [Screening and assessment of substance use and addictive behaviours](#) outlines history-taking (including use of the [ASSIST-Lite tool](#)), examination, and investigations that should be considered in a broad review of substance use and addictive behaviour.

If sedating antihistamine use is identified, specific questions to assess use in more detail include:

- duration and dosage—prolonged use of recommended or higher doses can result in withdrawal symptoms on stopping
- use of any other substances (alcohol, over-the-counter medications, complementary therapies [\[Note 2\]](#), other prescribed or illicit drugs)—assess risk of fatal sedation or other dangerous interactions
- factors that precipitate or perpetuate use—assess for undertreated [anxiety](#), difficulties with regulating emotional distress (eg in [personality disorder](#)), [insomnia](#), other [disorders of substance use](#) and chronic itch
- withdrawal symptoms experienced on previous attempts to reduce usage.

Common **withdrawal symptoms** are anxiety, insomnia, irritability and cravings; they are similar to those the patient may have been seeking to relieve when starting the drug. Pre-existing anxiety increases the likelihood of severe withdrawal; anxiety that persists for more than 1 week after stopping sedating antihistamines is likely to reflect an anxiety disorder or difficulties with emotional regulation rather than persisting withdrawal. Other withdrawal symptoms include nausea, vomiting, headache, sweating and dizziness.

Note 2: St John's wort can increase the risk of [serotonergic toxidrome](#) when taken with large doses of sedating antihistamines.

Management of disorders of sedating antihistamine use

Management of disorders of sedating antihistamine use

[Overview of substance use and addictive behaviours](#) explains key [principles of care](#) for a person with a disorder of substance use. Establishing a therapeutic relationship that engages the person (and ideally those close to them) is central to the management of substance dependence.

The most important element of treatment for substance use is a therapeutic relationship.

Specialist advice on any aspects of care for patients with any disorder of substance use is available and contact is encouraged; see [Contact details for substance use clinical advisory services for clinicians](#).

Specialist advice is available by phone on any aspect of the management of substance use; contact is encouraged.

Consider specialist referral to manage withdrawal if a patient has severe withdrawal symptoms or comorbid conditions, especially psychosis or polysubstance use (use of more than one substance).

Weaning of sedating antihistamines can usually be performed in an outpatient setting. Abruptly stopping a sedating antihistamine after prolonged use (even at recommended doses) can cause unplanned withdrawal, but it is rarely severe. Patients dependent on very high doses of sedating antihistamines with anticholinergic properties (eg diphenhydramine) may require hospital admission for severe symptoms of cholinergic rebound, such as urinary urgency, tachycardia, orthostatic hypotension, severe anxiety or severe insomnia.

Evidence to guide weaning regimens for sedating antihistamines is limited; advice in these guidelines is based on the consensus view of the Addiction Guideline Group, extrapolating from approaches used for managing polypharmacy in older patients. The New South Wales Therapeutic

Advisory Group (TAG) deprescribing guide for sedating antihistamines can be downloaded at the [TAG website](#); this outlines weaning schedules, monitoring advice and alternative management for those who were using the sedating antihistamine to manage allergy or itch.

Offer alternative strategies (which may include group or individual [psychosocial support](#)) for managing other factors that precipitate or perpetuate prolonged use of sedating antihistamines, such as [insomnia](#) and [anxiety](#).

Substance use can pose a risk of acute harm to the person (such as self-harm, falls and other accidents, suicide) and others (through impacts on driving, childcare, fitness to work and acute behavioural disturbances). For advice on managing these risks, see [Ensuring the safety of a person with a disorder of substance use or addictive behaviour](#). For harm reduction measures to consider for any person with a disorder of substance use or addictive behaviour, see [Harm reduction in substance use and addictive behaviours](#).

Elements of long-term management relevant to all disorders of substance use are covered in [Long-term care in disorders of substance use and addictive behaviours](#).

[Considerations for specific populations](#) may be relevant in managing disorders of sedating antihistamine use.

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Overview of disorders of stimulant use

Overview of disorders of stimulant use

Central nervous system stimulants enhance dopaminergic, adrenergic and serotonergic neurotransmission. They produce desired effects, such as euphoria, a sense of wellbeing and confidence, alertness, increased sexual drive and appetite suppression, but are also associated with [harms](#).

Stimulants (also known as amphetamine-type substances) are used across the spectrum of society; they include:

- amphetamine and metamphetamine
- methylenedioxymetamfetamine (methylenedioxymethamphetamine, MDMA, 'ecstasy', 'Molly')
- cocaine
- prescription and over-the-counter amphetamines:
 - dexamfetamine, lisdexamfetamine, methylphenidate—to manage attention deficit hyperactivity disorder (ADHD), binge eating disorder and narcolepsy
 - phentermine—to manage obesity
 - ephedrine—intravenously to manage shock and hypotension in spinal anaesthesia, and intranasally to reduce nasal congestion
 - pseudoephedrine—to reduce nasal congestion
- novel psychoactive substances (discussed in the [Toxicology and Toxinology Guidelines](#))
 - [synthetic cannabinoid-receptor agonists](#) (SCRAs) which have stimulant as well as cannabinoid effects
 - [novel stimulant drugs](#) (eg cathinones)
- khat—a plant chewed for euphoric effect; for more information, see the [Australian Drug Foundation website](#).

Stimulants are produced as:

- powders or pills
- crystals, such as crystalline metamfetamine ('ice', which is usually inhaled ['smoked'] or injected) or 'crack' cocaine (which can be inhaled ['smoked'])
- pastes.

Routes of stimulant administration include oral, intravenous, intranasal ('snorting') and vapour inhalation ('smoking').

Unregulated drugs sold as stimulants may not actually contain the advertised substance and may have a range of ingredients of unknown identity and strength.

Prescribed and over-the-counter stimulants are sometimes used in a way that does not align with the directed use (nonmedical use). For example, a person may use them to treat a symptom other than the clinician intended, or to become intoxicated. The extent of nonmedical use of stimulants is unclear because data are not collected separately for each category of stimulant. Wider availability of stimulant medications in the last 50 years may be contributing to nonmedical use. Each state and territory has legislation, policies, and real-time prescription monitoring systems for dexamfetamine, lisdexamfetamine and methylphenidate.

Harms of stimulant use

Harms of stimulant use

Some people can use stimulants for long periods without developing adverse consequences, others develop severe harms. The spectrum of substance use is described in this guideline by the terms ‘hazardous use’, ‘harmful use’ and ‘substance dependence’, outlined in [Table 22.1](#).

Potential physical and mental harms from stimulant use are outlined in [Table 22.20](#). Other harms associated with dependent use include damage in other areas of life, including relationships, work and education.

Table 22.20 Potential harms of stimulant use

Cardiovascular harms

hypertension

arrhythmias

myocardial infarction

cardiomyopathy

heart failure

infective endocarditis related to injecting

Neurological harms

clonic seizures

haemorrhagic or ischaemic stroke

poor attention, memory, concentration, learning, cognitive impairment

Psychiatric harms [NB1]

misperceptions and delusions

depression

paranoia

anxiety

aggression, with risk of trauma to others (including children witnessing violence)

delirium

psychosis

Respiratory harms

nasal inflammation and mucosal and septal damage related to intranasal use (‘snorting’)

chronic bronchitis and restrictive-pattern lung fibrosis from inhaling vapour (‘smoking’)

Oral health harms

dental damage from bruxism, poor self-care, periodontal disease

mouth burns from pipe smoking

Sexual health harms

sexual risk-taking related to disinhibition

Reproductive health harms

spontaneous abortion, miscarriage, placental abruption, fetal harms, premature labour

Dermatological harms

infections and abscesses related to injecting or skin picking (eg when experiencing the stimulant-induced sensation of insects crawling under their skin [formication])

Other harms

weight loss caused by reduced appetite and accelerated metabolism

hyperthermia caused by toxicity; see [Stimulant drug poisoning](#)

bloodborne infections (eg from injecting, sharing pipes, unprotected sex)

NB1: Mood, anxiety and psychotic symptoms may be predisposing factors to or consequences of stimulant use; a pre-existing condition increases the likelihood that long-term mental health management will be required.

Screening and assessment of stimulant use

Screening and assessment of stimulant use

Ask all new patients routinely and others (adolescents and adults) opportunistically and periodically about stimulant use, as part of a general screen for disorders of substance use and gambling; these disorders are common (and often co-exist) and people are reluctant to disclose them, often due to fear of stigma. [Screening and assessment of substance use and addictive behaviours](#) outlines history-taking (including use of the [ASSIST-Lite tool](#)), examination, and investigations that should be considered in a broad review of substance use and addictive behaviour.

Core features of the assessment should identify:

- how much, how often and how stimulants are used
- features of toxicity; see [Risk assessment for stimulant drug poisoning](#)
- any other [harms](#) experienced
- severity of [dependence](#) and [withdrawal](#) on previous attempts to reduce or stop using
- psychiatric symptoms including psychosis, anxiety, depression; difficulties with emotional regulation (eg as can occur with [personality disorder](#)), suicide risk, and features of attention deficit hyperactivity disorder (and whether symptoms preceded use)
- potential drug interactions, which can complicate intoxication or withdrawal presentation (eg [serotonin toxicity](#))
- likelihood of pregnancy
- what changes the patient is interested in making to their use and how they prioritise them; see [Brief interventions](#) for discussion of motivational interviewing.

Withdrawal from stimulants is not usually complicated; however, the risk of agitation and violence is increased if a patient uses large amounts of stimulants, has pre-existing psychiatric symptoms or uses more than one substance (polysubstance use). [Table 22.21](#) illustrates 4 phases of withdrawal. Strong cravings and mood symptoms can persist for longer than with some other substances (eg alcohol or opioids).

Table 22.21 Time course of stimulant withdrawal symptoms

Phase	Physical symptoms	Psychological symptoms
Days 1 to 3 (comedown or crash phase)		depression, anxiety
	exhaustion, low energy	irritability
	increased sleep	paranoia
	increased appetite	amotivation
Days 2 to 10 (withdrawal phase)	restlessness	anhedonia
		suicidal ideas or behaviour
	strong cravings	strong urge to use

	sleep difficulties, nightmares	depression, anxiety
	aches, pains, stiffness	mood swings
	headaches	poor concentration, confusion
	increased appetite	paranoia
		easily upset
		strong urge to use
Days 7 to 28 (persisting symptoms)	strong cravings	mood swings
	sleep difficulties, nightmares	anxiety
		boredom
	cravings persist	
Months 1 to 3	sleep and activity levels return to normal	urge to use persists
		mood improves
	general health improves	

Specific questionnaires can be useful in assessing the extent of stimulant use, including the Amphetamine Withdrawal Questionnaire (AWQ) and the Severity of Dependence Scale (SDS); both are available at the [National Centre for Education and Training on Addiction website](#). The [S-check app](#) can be used by patients to track their use of crystalline metamfetamine ('ice').

Examination of a patient who uses stimulants should include assessment of the person's mental state, and a physical examination including a comprehensive systems review, to look for harms outlined in [Table 22.20](#). Consider the need for investigations to assess for:

- toxicity, as outlined in [Key investigations for stimulant drug poisoning](#)
- organ damage—assess liver and kidney function
- bloodborne and sexually transmitted infections
- pregnancy.

Overview of management of disorders of stimulant use

Overview of management of disorders of stimulant use

[Overview of substance use and addictive behaviours](#) explains [key principles of care](#). Establishing a therapeutic relationship that engages the person (and ideally those close to them) is central to the management of substance dependence.

The most important element of treatment for substance use is a therapeutic relationship.

Specialist advice on any aspects of care for patients with stimulant and other substance use is available and contact is encouraged; see [Contact details for substance use clinical advisory services for clinicians](#).

Specialist advice is available by phone on any aspect of the management of substance use; contact is encouraged.

Patients using stimulants may present with intoxication, particularly if using large amounts or more than one substance (polysubstance use). Issues to address include:

- [ensuring the safety of an intoxicated person](#) and those around them, particularly if the person is agitated
- [implications of substance use for driving](#)
- [occupational implications of substance use](#).

For advice on the medical management of stimulant toxicity, see [Stimulant poisoning](#).

[Brief interventions](#) can use motivational interviewing to explore a patient's view of risks associated with use, their goals for treatment, and harm reduction. Stimulants are commonly used for enhancing sexual experiences (as are [gamma-hydroxybutyrate \(GHB\)](#) and [amyl nitrite](#)), but use is associated with sexual risk-taking; offer measures for sexual health harm reduction. For broad advice on harm reduction, including a patient information sheet, see [Harm reduction in substance use and addictive behaviours](#).

Management of planned or unplanned [withdrawal](#) involves drug therapy to alleviate symptoms, and psychosocial interventions.

[Psychosocial interventions](#) are the main treatments to reduce stimulant use and prevent relapse. No drugs are approved to treat stimulant dependence. Maintenance drug treatment options are being investigated; although data on effectiveness are inconclusive, several drugs warrant further investigation.

[Long-term care](#) includes management of physical and mental comorbidities such as [anxiety](#), [depression](#) and [psychotic episodes](#). Residential rehabilitation and specialist outpatient services are appropriate options for those who have been more severely affected by their stimulant use.

Considerations for specific populations may be relevant in managing stimulant use. This topic considers [pregnant patients](#) and [breastfeeding patients](#). Other populations are discussed in [Considerations for specific populations in the management of substance use and addictive behaviours](#).

Management of stimulant withdrawal

Management of stimulant withdrawal

Withdrawal from stimulants requires planning to provide support for several [phases of withdrawal](#) that span 3 weeks or more. The safety of the setting for planned stimulant withdrawal (at home, in a community facility or in hospital) depends on the likelihood of severe withdrawal and the extent of the patient's social supports. For guidance on selecting a withdrawal setting, see [Choice of setting for planned withdrawal management in substance use](#). Seek specialist advice from a [clinical advisory service](#) if unsure about assessing these factors.

Management of stimulant withdrawal is mainly medication to alleviate symptoms, and psychosocial interventions.

Evidence to guide choice of medication for symptomatic management is limited, and most medications used are not approved for this indication. Limit the use of sedative medication for withdrawal (eg olanzapine, diazepam) to 1 week to reduce risk of overdose or dependence. For other considerations in managing medications during withdrawal management, see [Safe prescribing and supply during planned substance withdrawal in a home setting](#).

More details on symptomatic treatment are discussed in clinical guidance on withdrawal from alcohol and other drugs available on the [New South Wales Health website](#). Psychosocial interventions in the first 7 to 10 days of stimulant withdrawal focus on psychoeducation (education about the process of withdrawal and its treatment) and reassurance to alleviate anxiety and increase the patient's motivation to continue treatment. Once withdrawal symptoms are less severe, [psychological interventions](#) can be used to promote retention in treatment, abstinence and reduce mood symptoms in the short-term. [Psychological and behavioural interventions for insomnia](#) may also be helpful; advice includes a printable patient handout on good sleep practices.

Psychological interventions for long-term management of stimulant use are the mainstay of promoting abstinence and relapse prevention.

For further advice on managing stimulant withdrawal, consult local state or territory guidelines or contact a clinical advisory service.

Psychosocial interventions for long-term management of disorders of stimulant use

Psychosocial interventions for long-term management of disorders of stimulant use

All forms of psychosocial interventions increase short-term abstinence and retention in treatment for disorders of stimulant use. Relapse rates are high, however, and long-term benefits of any single intervention are not clear. A combination of therapies for at least 3 to 4 months may be more effective than single short-term approaches. Techniques include cognitive behavioural therapy, motivational interviewing, acceptance and commitment therapy and contingency management; these can be combined with other relapse prevention strategies, such as regular urine drug screening and access to mentors with lived experience.

Management of disorders of stimulant use in specific populations

Management of disorders of stimulant use in specific populations

Specific considerations (including the need for specialist referral) apply in the management of stimulant use for people who are pregnant or breastfeeding. For other groups, see Considerations for specific populations in substance use and addictive behaviours.

Management of disorders of stimulant use during pregnancy

Management of disorders of stimulant use during pregnancy

Refer pregnant patients with a disorder of stimulant use early to an obstetric service that has support from an alcohol and other drug service.

Stimulant use during pregnancy is associated with harms to the fetus and the pregnant patient. Fetal harms include distress, risk of placental abruption, low birth weight, prematurity and increased mortality. Pregnant patients are at increased risk of harms, outlined in Table 22.20.

Explore the patient's understanding of the risks that stimulants may pose to the pregnancy and fetus, and use motivational interviewing approaches and psychosocial interventions to support positive change.

Management of disorders of stimulant use during breastfeeding

Management of disorders of stimulant use during breastfeeding

Stimulants are found in breastmilk. Exposure to stimulants through breastfeeding is associated with harmful effects on the infant, including behavioural problems such as infant irritability and poor sleep.

If a patient uses stimulants and wishes to breastfeed, explore their understanding of the risks and support them to develop a management plan. Advise them of the benefits of abstinence for themselves and the infant.

If a breastfeeding patient uses stimulants rarely, or in binges, discuss harm prevention; this may include:

- recommending abstinence or (if abstinence is not likely) expressing and discarding the breast milk for 24 to 48 hours after the use of stimulants
- highlighting the value of a back-up feeding plan for events where unplanned use occurs.



[\[X\] Close](#)

Overview of disorders of volatile inhalant use

Overview of disorders of volatile inhalant use

A wide variety of volatile inhalants is consumed in Australia; the most common are:

- nitrous oxide (eg sold in small canisters as a propellant for whipped cream) used to achieve brief intoxication
- amyl nitrite used as a party drug and to enhance sexual experience (chemsex).

Other volatile inhalants include:

- petrol
- solvents, such as toluene, found in glues, paints, paint thinners and correction fluids
- acetone in nail polish remover
- butane in deodorants and gas canisters.

The use of volatile inhalants is described by colloquial terms. Substances can be inhaled from:

- a soaked cloth on the nose and mouth—'huffing'
- a plastic bag—'bagging'
- a container—'sniffing'.

Use of volatile inhalants in Australia has increased from 0.4% of the population in 2001 (reporting use in the past year) to 1.7% in 2019. Use is more prevalent among some populations, such as young people (of whom 13% report use in the past year). Around one-third of use occurs monthly. Volatile inhalants are readily available, and it is rarely feasible to reduce access to them.

The spectrum of substance use in these guidelines is described by the terms 'hazardous use', 'harmful use' or 'substance dependence', outlined in [Table 22.1](#).

Harms result from acute intoxication and chronic consumption of volatile inhalants. Presentations of acute intoxication vary according to the substance; they are most frequently characterised by euphoria, disinhibition and excitation, but also feature drowsiness, disorientation, hallucinations and ataxia, particularly at high doses. Most patients experiencing inhalant intoxication can be treated with removal of the causal agent, and bed rest. Deaths are rare, but may arise through asphyxia, ventricular fibrillation and other cardiac arrhythmias. Advice on the management of volatile inhalant toxicity is available in [Hydrocarbon poisoning: Inhalation](#).

Chronic, high-level exposure to some volatile inhalants can result in diffuse neurotoxicity, with varying symptoms, most commonly peripheral neuropathy and encephalopathy. Use of nitrous oxide is associated with functional vitamin B₁₂ deficiency and subacute combined degeneration of the spinal cord. Other organ systems, including the kidney and liver, may also be affected by volatile inhalant use.

Chronic use of volatile inhalants can result in dependence. Although inhalant withdrawal is not well described, symptoms reported on abrupt stopping after dependent use include headaches, nausea, vomiting, hallucinations, rhinorrhoea, craving, tachycardia, depressed mood, agitation, insomnia and anxiety.

Screening and assessment of volatile inhalant use

Screening and assessment of volatile inhalant use

Ask all new patients routinely and others (adolescents and adults) opportunistically and periodically about volatile inhalant use, as part of a general screen for disorders of substance use and gambling; these disorders are common (and often co-exist) and people are reluctant to disclose them, often due to fear of stigma. [Screening and assessment of substance use and addictive behaviours](#) outlines history-taking (including use of the [ASSIST-Lite tool](#)), examination, and investigations that should be considered in a broad review of substance use and addictive behaviour. A sociopsychobiomedical assessment is critical as use in young people often reflects significant psychosocial disadvantage (eg poverty, an unstable home situation or major stressors at school). Inhalant use is associated with psychiatric comorbidity and patients identified as using inhalants should be screened for this.

Overview of management of disorders of volatile inhalant use

Overview of management of disorders of volatile inhalant use

[Overview of substance use and addictive behaviours](#) explains key [principles of care](#) for a person with a disorder of substance use. Establishing a therapeutic relationship that engages the person (and ideally those close to them) is central to the management of substance dependence.

The most important element of treatment for substance use is a therapeutic relationship.

Specialist advice on any aspects of care for patients with substance use is available and contact is encouraged; see [Clinical advisory services](#).

Specialist advice is available by phone on any aspect of the management of substance use; contact is encouraged.

Management strategies in disorders of volatile inhalant use include:

- [harm reduction](#)
- [withdrawal management](#)
- [long-term care](#).

Specific considerations for young people are discussed in this topic, but also consider factors that may apply for [other populations](#).

Harm reduction in volatile inhalant use

Harm reduction in volatile inhalant use

Harm reduction in disorders of volatile inhalant use can be helpful, such as teaching patients, particularly young people:

- to avoid putting plastic bags over their heads when 'bagging'
- to avoid smoking when using inhalants
- how to provide first aid
- to call an ambulance if they are worried about themselves or a friend.

Consider offering a [printable patient information sheet](#) on ways to reduce harms from substance use.

See [Ensuring the safety of a person with a disorder of substance use or addictive behaviour](#) for further advice on immediate safety issues, such as managing agitation, as well as impacts of substance use on fitness to work and to drive. For other harm reduction measures to consider for any person with a disorder of substance use or addictive behaviour, see [Harm reduction](#); these include managing sexual health risks for people who have 'chemsex' [\[Note 1\]](#) (eg people who use amyl nitrite).

Note 1: 'Chemsex' refers to using recreational drugs to enhance sexual experience.

Withdrawal management in disorders of volatile inhalant use

Withdrawal management in disorders of volatile inhalant use

Evidence to guide management of dependent inhalant use is very limited. If significant symptoms of moderate agitation occur on withdrawal, use [verbal de-escalation and psychological interventions](#); seek specialist advice (eg from a [clinical advisory service](#)) if agitation persists. Offer patients who wish to stop using inhalants the supports outlined in [Long-term care in the management of disorders of volatile inhalant use](#).

Long-term care in the management of disorders of volatile inhalant use

Long-term care in the management of disorders of volatile inhalant use

Long-term treatment for disorders of volatile substance use includes:

- accessing psychosocial supports (eg from [services that are specific to young people](#)); this is critical to address the severe psychosocial disadvantage faced by many people using volatile inhalants
- [cognitive behavioural therapy](#)
- [family therapy](#)
- activity therapy—programs that engage young people in fun activities while also providing psychoeducation have been very effective in some communities
- [residential treatment](#)—this may be considered as a second-line treatment option.

For advice on long-term management relevant to any person with a disorder of substance use or addictive behaviour, see [Long-term care in disorders of substance use and addictive behaviours](#).

Further educational resources for healthcare professionals about volatile inhalant use

Further educational resources for healthcare professionals about volatile inhalant use

Further educational resources for healthcare professionals about volatile inhalant use include:

- the [Better Health Channel](#)
- videos and webinars about substance use in young people, available at the [Dovetail website](#).

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[\[X\] Close](#)

Overview of nicotine replacement therapy

Overview of nicotine replacement therapy

Nicotine replacement therapy (NRT) is available in several formulations, categorised by onset of effect:

- slow-acting NRT — [transdermal patch](#)
- medium-acting NRT — [gum](#), [lozenge](#) and [inhalator](#)
- fast-acting NRT — [mist spray](#).

Almost everyone who smokes tobacco is likely to benefit from slow-acting NRT with a patch; after stopping smoking, people who use nicotine patches show reduced brain activation in areas involved in craving compared to those who use placebo. However, those who only smoke in highly specific cue-driven situations may not require a patch (eg those who smoke after work with specific friends).

Combination NRT uses a slow-acting nicotine patch with an as-required medium-acting and/or a fast-acting formulation to control cravings; this is more effective than nicotine monotherapy. The pattern of nicotine delivery during combination NRT mimics that from tobacco smoking, with a constant baseline and bursts of nicotine during the day. Advise patients that they can choose to reduce their smoking at their own pace when they feel their cravings reduce.

Combination NRT using a patch plus a medium-acting and/or fast-acting formulation is more effective than nicotine monotherapy.

Individual requirements for NRT are highly variable and based on the severity of dependence. Some patients (eg those who smoke within 30 minutes of waking) require very large doses of NRT, which may involve the use of 2 or 3 patches along with medium- and fast-acting NRT. When NRT is not effective, the usual causes are inadequate dosage and incorrect use.

When NRT is not effective, the usual causes are inadequate dosage and incorrect use.

Patches, gum and lozenges are subsidised by the Pharmaceutical Benefits Scheme (PBS) as monotherapies; see the [PBS website](#) for current information. Inhalators and mist spray are not PBS-subsidised but are substantially cheaper than tobacco smoking. Prescribing only according to PBS funding will result in underdosing; to prescribe adequate treatment, combination NRT is needed using a mix of PBS-subsidised and nonsubsidised items.

The minimum course of NRT is 12 weeks, but longer treatment is more effective; see [Review of nicotine replacement therapy](#).

Precautions in nicotine replacement therapy

Precautions in nicotine replacement therapy

For advice on nicotine replacement therapy (NRT) in pregnancy and breastfeeding, see [Management of tobacco smoking in pregnancy](#) and [Management of tobacco smoking in breastfeeding](#).

There is no safe level of exposure to nicotine in adolescence; the developing prefrontal cortex is particularly sensitive to the effects of nicotine, affecting learning, memory and mood. Young people exposed to tobacco smoking are more likely to become nicotine dependent than those who start in adulthood. Nicotine in NRT also affects the adolescent brain; however, it remains a much safer alternative to tobacco smoking. No trial evidence supports the safety and efficacy of NRT in people younger than 12 years.

Although starting doses of NRT are not dependent on weight, patients who weigh less than 45 kg may be more likely to require lower maintenance doses of NRT to avoid toxicity.

Nicotine replacement can be used cautiously by patients who have had a recent coronary event or skin graft, balancing the risks from vasoconstriction from NRT against those from continued smoking.

Use NRT gum without artificial sweeteners in anyone with phenylketonuria.

Patients with nicotine hypersensitivity are unlikely to be nicotine dependent as a result of tobacco smoking. Seek specialist advice if a patient reports a history of hypersensitivity, which may be suggested by vomiting, skin reactions or anaphylaxis after exposure to nicotine.

Choice of formulation of nicotine replacement therapy

Choice of formulation of nicotine replacement therapy

Combination nicotine replacement therapy is recommended for most patients because it is more effective than nicotine monotherapy.

Nicotine patches are available as a 24-hour preparation (left on all day) or a 16-hour preparation (removed at night). The 24-hour patch provides continuous nicotine exposure to receptors when the person is not smoking (eg during sleep) and offers better control of morning cravings than the 16-hour patch. The 24-hour patch is preferred over the 16-hour patch except in:

- pregnancy.
- people who have been troubled by nightmares with the 24-hour patch.

The mist spray mimics the delivery of nicotine into the bloodstream in rapid 'hits', while the gum, lozenge and inhalator take slightly longer to work. Inhalators mimic the experience of holding a cigarette and inhaling. Gum is generally cheapest, but correct use is important ('chew and park', not continuous chewing).

Table 22.7 summarises the correct use, advantages and adverse effects of NRT formulations, and can be used to help patients choose a suitable formulation. The Quit website also has patient information on the correct use and advantages of each formulation and how to address adverse effects. The Quit Centre webpage has tools for health professionals including videos on how to demonstrate use of each formulation to patients.

Table 22.7 Correct use of nicotine replacement therapy — patient information

Printable table

slow-acting formulations

- patch

medium-acting formulations

- gum
- inhalator
- lozenge

fast-acting formulations

- mist spray

Patch

slow acting: takes 2 to 6 hours for peak effect

How much to use 21 mg in 24 hours OR 25 mg in 16 hours.

	Apply a patch to clean, dry, hairless skin.
How to use	<p>Rotate site daily to avoid adhesive build up, which can reduce nicotine absorption.</p> <p>Cost is subsidised by the PBS [NB1].</p>
Advantages	<p>Gives a constant supply of nicotine.</p> <p>Using the 24-hour patch can reduce morning cravings.</p> <p>If you have nightmares with the 24-hour patch, change to using the 16-hour patch.</p>
Adverse effects and other considerations	<p>Skin irritation from adhesive might require change of brand.</p> <p>Use the 16-hour patch if pregnant.</p>
Gum	
medium acting: takes 20 minutes for peak effect	
How much to use	<p>4 mg every 1 to 2 hours as required; to control strong urges, a second piece of gum can be chewed after 30 minutes.</p> <p>Usual maximum dose is 16 pieces in 24 hours.</p> <p>Chew gum to release flavour and produce a tingling sensation, then park in cheek pouch to absorb nicotine. When tingling stops, repeat the 'chew and park' cycles for up to 30 minutes or until all flavour is gone.</p>
How to use	<p>Try not to swallow saliva while chewing (swallowing saliva or the gum inactivates the nicotine).</p> <p>Chewing more often or harder doesn't release more nicotine.</p> <p>Cost is subsidised by the PBS [NB1].</p>
Advantages	<p>Keeps mouth busy and engaged.</p> <p>Can be used without others noticing.</p>
Adverse effects and other disadvantages	Can cause taste changes, throat irritation, hiccups and indigestion.
Inhalator	
medium acting: takes 20 to 30 minutes to peak effect	
How much to use	<p>Puff from a 15 mg cartridge as required.</p> <p>Usual maximum dose is 1 cartridge per hour and 6 cartridges in 24 hours.</p>
How to use	Place cartridge in tube to pierce it and access nicotine. Breathe normally or puff through the plastic tube. A cartridge that is being regularly used will last about 40 minutes.
Advantages	Allows for the familiar experience of smoking.
Adverse effects and other disadvantages	<p>Cost is not subsidised by the PBS.</p> <p>Can cause cough, taste changes and throat irritation.</p>
Lozenge	
medium acting: takes 20 minutes to peak effect	
How much to use	<p>4 mg every 1 to 2 hours as required. To control strong urges, a second lozenge can be sucked after 30 minutes.</p> <p>Usual maximum dose is 16 lozenges in 24 hours.</p>

Keep the lozenge in the cheek pouch (where absorption occurs) to get maximal absorption.

How to use Try not to swallow saliva while the lozenge is dissolving (swallowing saliva or the lozenge inactivates the nicotine).

Crunching or hard sucking of the lozenge doesn't release more nicotine.

Cost is subsidised by the PBS [NB1].

Advantages Keeps mouth busy and engaged.

Can be used without other people noticing.

Adverse effects and other disadvantages Can cause taste changes, throat irritation, hiccups, indigestion.

Mist spray

fast acting: takes 10 minutes to peak effect

1 to 2 sprays every 15 minutes as required.

How much to use

Usual maximum dose is 4 sprays per hour (or 64 sprays in 24 hours).

Spray under the tongue, avoiding the lips. Do not inhale or swallow while spraying.

How to use

Approximately 140 sprays are in each unit.

Advantages Rapid action, most closely mimicking nicotine effect from cigarette.

Cost is not subsidised by the PBS.

Adverse effects and other disadvantages

Can cause hiccups, increased salivation, throat irritation.

PBS = Pharmaceutical Benefits Scheme

NB1: The PBS subsidises the use of one form of nicotine replacement therapy (NRT) at a time; to use combination NRT will require addition of nonsubsidised form(s).

Adapted from the Albany Psychiatric Unit (Western Australia) patient information sheet on correct use of nicotine replacement, 2022.

Starting nicotine transdermal patches

Starting nicotine transdermal patches

Nicotine transdermal patches are best used as part of combination nicotine replacement therapy (NRT) with one or more medium- or fast-acting form of NRT ([gum](#), [lozenge](#), [mist spray](#) or [inhalator](#)). For advice on how to tailor treatment, see [Overview of nicotine replacement therapy](#) and [Choice of formulation of nicotine replacement therapy](#). The minimum course of NRT is 12 weeks, but longer treatment is more effective; see [Review of nicotine replacement therapy](#).

Nicotine patches are slow acting; time to peak effect is 2 to 6 hours. They are 24-hour or 16-hour preparations.

Higher-dose patches (21 mg/24 hours or 25 mg/16 hours) are preferred for NRT because they are more effective; lower doses do not generally provide adequate nicotine replacement, even after maximal absorption. Lower-dose patches may be useful for weaning, only if a patient is concerned about stopping abruptly after completing a course of NRT with the higher-dose patch.

Start a **24-hour patch** for smoking management unless the patient is pregnant. For nonpregnant patients, use:

nicotine 21 mg/24 hours transdermally, once daily applied for 24 hours.

The **16-hour patch** is removed at night; this reduces nocturnal nicotine exposure, which is important if a patch is required for [management of tobacco smoking in pregnancy](#) (after behavioural interventions and medium- to fast-acting forms of NRT have not been effective). The 16-hour patch is also beneficial for people who have nightmares when using the 24-hour patch. If a 16-hour patch is appropriate for smoking management, use:

nicotine 25 mg/16 hours transdermally, once daily applied for 16 hours during the day; remove at night.

Starting nicotine gum

Starting nicotine gum

Nicotine gum is a medium-acting formulation of nicotine replacement therapy (NRT); it takes 15 to 20 minutes to reach peak effect. Gum is best used as part of combination NRT with a [patch](#) and possibly also a [mist spray](#); for advice on how to tailor treatment, see [Overview of nicotine replacement therapy](#) and [Choice of formulation of nicotine replacement therapy](#).

The 4 mg gum is preferred over lower doses because it is more likely to control cravings and increases the likelihood of success in the management of tobacco smoking.

If nicotine gum is chosen for NRT, use:

nicotine 4 mg gum, chew 1 piece to release flavour and produce a tingling sensation, then park gum in cheek pouch to absorb nicotine. When tingling stops, repeat the 'chew and park' cycles for up to 30 minutes or until all flavour is gone. Repeat with a new piece of gum 1- to 2-hourly as required to control cravings; for strong cravings, repeat after 30 minutes at the earliest (to allow time to reach peak effect). Usual maximum dosage is 16 pieces in 24 hours.

If a patient has symptoms of withdrawal with gum use, ask about their gum-chewing technique; printable patient advice is available in [Table 22.7](#); see also [patient education videos](#) on the Quit website.

Starting nicotine lozenges

Starting nicotine lozenges

Nicotine lozenges are a medium-acting formulation of nicotine replacement therapy (NRT); they take 20 minutes to reach peak effect. Lozenges are best used as part of combination NRT with a [patch](#) and possibly also a [mist spray](#); for advice on how to tailor treatment, see [Overview of nicotine replacement therapy](#) and [Choice of formulation of nicotine replacement therapy](#).

The 4 mg lozenge is preferred over lower doses because it is more likely to control cravings and increases the likelihood of success in managing tobacco smoking.

If nicotine lozenges are chosen for NRT, use:

nicotine 4 mg lozenges, suck 1 lozenge 1- to 2-hourly as required to control cravings; for strong cravings, repeat after 30 minutes at the earliest (to allow time to reach peak effect). Usual maximum dosage is 16 lozenges in 24 hours.

If a patient has symptoms of withdrawal with lozenge use, ask about their technique; printable patient advice is available in [Table 22.7](#); see also [patient education videos](#) on the Quit website.

Starting nicotine inhalators

Starting nicotine inhalators

The nicotine inhalator is a cigarette-shaped device that delivers nicotine from a cartridge. It is a medium-acting formulation of nicotine replacement therapy (NRT); it takes 20 to 30 minutes to reach peak effect. It is best used as part of combination NRT with a [patch](#) and possibly also a [mist spray](#); for advice on how to tailor treatment, see [Overview of nicotine replacement therapy](#) and [Choice of formulation of nicotine replacement therapy](#).

If a nicotine inhalator is chosen for NRT, use:

nicotine cartridge, 15 mg inhaled via inhalator. Repeat as required to manage cravings. Replace cartridge after a total of 40 minutes of use. Usual maximum dosage is 1 cartridge per hour, and 6 cartridges in 24 hours.

If a patient has symptoms of withdrawal with inhalator use, ask about their technique; printable patient advice is available in [Table 22.7](#); see also [patient education videos](#) on the Quit website.

Starting nicotine mist sprays

Starting nicotine mist sprays

The nicotine mist spray is a fast-acting formulation of nicotine replacement therapy (NRT); it takes less than 10 minutes to reach peak effect. Nicotine spray is best used as part of combination NRT with a [patch](#) and possibly also a medium-acting formulation ([gum](#), [lozenge](#) or [inhalator](#)); for advice on how to tailor treatment, see [Overview of nicotine replacement therapy](#) and [Choice of formulation of nicotine replacement therapy](#).

If a nicotine mist spray is chosen for NRT, use:

nicotine 1 mg/spray, 1 to 2 sprays under the tongue. Repeat as required to manage cravings, up to a maximum of 4 mg (4 sprays) per hour. Usual maximum dosage is 64 mg (64 sprays) in 24 hours.

Lifting the tongue and spraying underneath is the easiest way to maximise absorption, while reducing the risk of accidentally inhaling or swallowing the spray. If a patient has symptoms of withdrawal with mist spray use, ask about their technique; printable patient advice is available in [Table 22.7](#); see also [patient education videos](#) on the Quit website.

Review of nicotine replacement therapy

Review of nicotine replacement therapy

Review tolerability and effectiveness of nicotine replacement therapy (NRT) 2 weeks after starting and at intervals during a minimum 12-week course.

NRT is generally well tolerated. Excess use may cause mild symptoms of nicotine toxicity, most commonly dizziness, nausea and palpitations. If the patient is still smoking, explain how this is contributing to their symptoms and encourage them to cut down or stop smoking. If toxicity symptoms occur while a patient is using combination NRT, advise them to reduce the fast- or medium-acting NRT before considering removal of the patch.

[Table 22.7](#) outlines management of other adverse effects specific to different formulations. [Varenicline](#) or [bupropion](#) can be added to combination NRT, but this is not generally required if NRT is used at adequate dosages with correct technique.

Offer a repeat course of combination NRT to anyone who wishes to continue beyond the first 12-week course, regardless of whether their smoking has reduced. No maximum duration of therapy is specified; longer durations up to 6 months are more effective for long-term smoking management. Some patients require indefinite NRT to prevent relapses. The impact of long-term replacement is not clear; possible risks of microvascular damage from vasoconstriction must be balanced against harms of continued smoking.



[\[X\] Close](#)

What is covered in the Addiction guidelines?

What is covered in the Addiction guidelines?

The Addiction guidelines include disorders due to substance use or addictive behaviours; these are mental and behavioural disorders that result from the use of psychoactive substances, including medications, or specific repetitive rewarding and reinforcing behaviours. [Terminology for substance use and addictive behaviours](#) used in these guidelines is based on the International Classification of Diseases 11th Revision (ICD-11).

Disorders of substance use in these guidelines include:

- licit or legal use of
 - [tobacco](#)
 - [alcohol](#)
 - pharmaceuticals (eg [opioids](#), [benzodiazepines](#), [zolpidem](#) and [zopiclone](#), [pregabalin](#) and [gabapentin](#), [quetiapine](#) and other [antipsychotics](#), [sedating antihistamines](#), [stimulants](#))
- illicit (illegal or unlicensed) use of
 - [cannabis](#) (other than prescribed [medicinal] forms)
 - [stimulants](#) (eg cocaine and amfetamines)
 - [opioids](#) (eg heroin)
 - [gamma-hydroxybutyrate \(GHB\)](#)
 - [ketamine](#) and other [hallucinogens](#)
 - [volatile inhalants](#).

Addictive behaviours in these guidelines include:

- [gambling](#)—examples include poker machines, card games, scratch cards, betting on sports, speculative online investment trading
- [gaming](#)—examples include fantasy games, first-person shooter games, e-sports.

For many, the experience of using substances, gambling or gaming is a pleasure, with few adverse consequences; however, for some, use leads to harm.

Not all substance use, gambling or gaming is a health concern.

The negative outcomes (harms) that can occur with substance use, gambling or gaming include:

- acute consequences—intoxication, overdose, assault, loss of reputation and money
- chronic consequences—precipitation or exacerbation of physical and mental health problems
- impairment in function—affecting relationships, parenting, work, finances
- harm to others, including family, friends, community.

Specialist advice is available on any aspects of care for people with a disorder of substance use or addictive behaviour. Contact is encouraged; see [Clinician resources](#).

Specialist advice is available by phone on the management of substance use or addictive behaviours; contact is encouraged.

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[X] Close

Terminology for substance use and addictive behaviours

Terminology for substance use and addictive behaviours

Disorders due to substance use and addictive behaviours are classified by the Diagnostic and Statistical Manual of Mental Disorders 5th edition (DSM-5) and the International Classification of Diseases 11th Revision (ICD-11) international categorisation systems. Terms used in these guidelines are based on ICD-11 because the criteria for prescribing in disorders of substance use under the Pharmaceutical Benefits Scheme (PBS) use ICD-11 terminology.

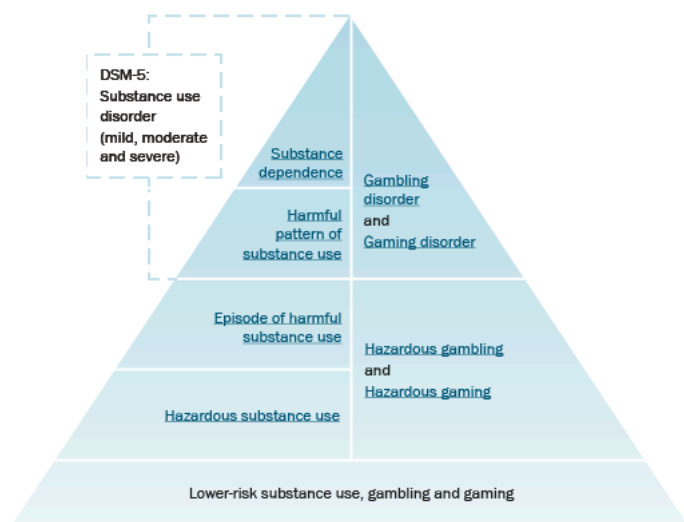
Both DSM-5 and ICD-11 describe a **spectrum of disorders of substance use**. DSM-5 uses the term 'substance use disorder', with criteria for classification into 'mild', 'moderate' and 'severe' disorders [Note 1]. ICD-11 describes a broader spectrum of severity, dividing the equivalent of DSM-5 'substance use disorder' into 'harmful pattern of substance use' and 'substance dependence'—for criteria, see [ICD-11 classification of substance use](#).

In describing the **spectrum of addictive behaviours**, both DSM-5 and ICD-11 recognise 'gambling disorder' as an addictive behaviour. DSM-5 criteria are available at the [American Psychiatric Association website](#). ICD-11 distinguishes between 'gambling disorder' and 'hazardous gambling'; it also includes 'hazardous gaming' and 'gaming disorder'—for criteria, see [ICD-11 classification of addictive behaviour](#).

The ICD-11 classifications of substance use, gambling and gaming are represented in [Figure 22.1](#).

Figure 22.1 ICD-11 classifications of substance use, gambling and gaming

[NB1]



DSM-5 = Diagnostic and Statistical Manual of Mental Disorders 5th edition; ICD-11 = International Disease Classification 11th Revision

NB1: Labels in the pyramid are ICD-11 diagnoses, other than the category 'lower-risk substance use, gambling and gaming'. The 'lower-risk' category is not a diagnostic term but is used here to illustrate the full spectrum of substance use and addictive behaviours. Proportions for each classification are not represented to scale. Corresponding DSM-5 terms for substance use are shown on the left of the diagram.

Note 1: DSM-5 criteria are outlined in Hasin DS, O'Brien CP, Auriacombe M, Borges G, Bucholz K, Budney A, Compton WM, Crowley T, Ling W, Petry NM, Schuckit M and Grant BF. DSM-5 Criteria for Substance Use Disorders: Recommendations and Rationale. Am J Psychiatry 2013; 170(8): 834–851 [URL](#).

ICD-11 classification of substance use

ICD-11 classification of substance use

The ICD-11 terms used in these guidelines to describe the spectrum of substance use are 'hazardous use', 'harmful use' and 'substance dependence'; for criteria, see [Table 22.1](#). The hierarchy of escalating use is represented in [Figure 22.1](#).

Table 22.1 Terminology describing the spectrum of substance use

Hazardous use

Substance use increases risk of physical or mental harm to the person or others through its frequency, the amount used, the method of ingestion, associated behaviours or a combination of these.

Harmful use

Substance use has damaged the physical or mental health of the person or others. This term is used in these guidelines to include a single episode or a pattern of harmful use.

Substance dependence

Substance use is recurrent and episodic or continuous, with 2 or more of the following features [NB1] [NB2]:

- impaired control of use (onset, frequency, intensity, duration, termination, context of use)
- use continues or escalates despite harms or negative consequences on other aspects of life (eg disruption in relationships, work, school, health)
- physiological features are present, including tolerance [NB3], withdrawal, or repeated use to prevent or alleviate withdrawal symptoms [NB4].

NB1: Features of substance dependence are usually evident over at least 12 months, but the diagnosis may be made if use is daily or almost daily for 3 months.

NB2: For comparison, the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) defines substance use disorder as the presence of at least 2 of a set of 11 criteria, as described in Hasin DS, O'Brien CP, Auriacombe M, Borges G, Bucholz K, Budney A, et al. DSM-5 criteria for substance use disorders: recommendations and rationale. *Am J Psychiatry* 2013;170(8):834-51 [URL](#).

NB3: Tolerance is a decreased response to a substance over repeated or prolonged exposure.

NB4: Withdrawal symptoms occur with stopping or reducing substance use.

Each of these diagnoses (hazardous use, harmful use or substance dependence) can apply to use of any psychoactive substance, regardless of its legal status. Each can occur with the use of medications as prescribed, or with nonmedical use. **Nonmedical use** describes the use of medications that does not align with the directed use; for example, use in order to become intoxicated or to treat a symptom other than the clinician intended. Nonmedical use describes the use of medications that does not align with the directions of the clinician.

ICD-11 classification of addictive behaviours

ICD-11 classification of addictive behaviours

At the time of writing, gambling and gaming are the only addictive behaviours considered to have sufficient evidence to be included in ICD-11; other addictive behaviours (eg excessive use of food or sex) are not included. For contrast, DSM-5 recognises 'gambling disorder' as a diagnosis but classifies 'internet gaming disorder' as a condition requiring further study. DSM-5 terms are not used in these guidelines, but DSM-5 criteria for diagnosis of gambling disorder are available at the [American Psychiatric Association website](#).

The ICD-11 terms used to describe addictive behaviours in these guidelines are:

- hazardous gambling or hazardous gaming—patterns of activity with increased risk of harm to the person or those close to them (eg because of the time or money spent, neglect of other activities or associated risky behaviours)
- gambling disorder or gaming disorder—impaired control over the activity, with increasing priority given to it despite escalating harm to the person or those around them.

Diagnostic criteria are outlined in the topics on [gambling](#) and [gaming](#). The hierarchy of escalating addictive behaviours is represented in [Figure 22.1](#).

Principles of care in substance use and addictive behaviours

Principles of care in substance use and addictive behaviours

Stigma in substance use and addictive behaviours

Stigma in substance use and addictive behaviours

People with a disorder of substance use or addictive behaviour often experience stigma and discrimination because these disorders or behaviours are often misperceived as a personal choice for which the individual is blameworthy. Institutions stigmatise people through policies, rules and practices that limit access and opportunities. Public and individual attitudes and stereotyping drive people to see themselves as less worthy or shameful.

The experience of stigma and consequent reduced access to health care are both associated with chronic ill health. Barriers deter disclosure of concerns, prevent people from seeking treatment and decrease adherence to treatment. This can lead to poorer outcomes, including lower quality of life.

To counter this stigma, it is important for clinicians to consider their own assumptions and how unconscious judgements might be enacted through [language](#) and actions that adversely impact care.

Using nonjudgemental language in substance use and addictive behaviours

Using nonjudgemental language in substance use and addictive behaviours

Welcoming, clear language is important when screening for, assessing and managing disorders of substance use and addictive behaviours. Use of nonjudgemental language that puts the person first (rather than labelling them as defined by their illness) is central to creating an environment where the patient feels less inhibited by [stigma](#), safe to talk about their concerns, able to participate in shared decision-making, clear about boundaries and hopeful that there are effective treatment options available.

Resources that give practical advice on the use of nonjudgemental language include:

- Wilson H. How stigmatising language affects people in Australia who use tobacco, alcohol and other drugs. *Aust J Gen Pract* 2020;49(3):155-8 [URL](#)
- [Language Matters](#) from the Network of Alcohol and Other Drug Agencies (NADA) website
- [Recovery Oriented Language Guide](#) from the Mental Health Co-ordinating Council.

Trauma-informed approach to care in substance use and addictive behaviour

Trauma-informed approach to care in substance use and addictive behaviour

Past trauma is any experience that is physically or emotionally harmful or life threatening (eg violence, physical or sexual abuse, neglect from childhood, natural disasters). Trauma is common; 90% of people who access specialist services for management of disorders of substance use have experienced at least one traumatic event. Enduring effects of trauma on a person's physical, mental, social and emotional functions alter their experience of care and limit treatment access, engagement and outcomes.

Past trauma contributes to substance use and addictive behaviours and affects a person's experience of and ability to participate in health care.

A trauma-informed approach to care involves an understanding of the impact trauma may have on a person's behaviour and complexity of need. This approach can improve the patient experience but organisational change and staff training is required to support trauma-informed care.

A trauma-informed approach is based on:

- creating a safe environment—elements include a welcoming waiting area, appropriate lighting, good signage, culturally sensitive décor and appropriate staff responses to challenging behaviours in the waiting room
- building trust—ensure patients are given clear information about their rights and responsibilities, what treatment services can offer and the limits of confidentiality. Ensure informed consent and articulate and maintain clear professional boundaries

- patient choice—provide patient-centred treatment that offers options and supports the person to make informed decisions. Ask the person what matters to them rather than what is the matter with them
- empowerment—improve the power imbalance between health staff and patients by supporting the patient to take an active role in treatment decisions, building on their strengths, promoting their sense of self-efficacy and giving messages of hope for positive health outcomes
- collaboration—work with the patient to achieve mutually agreed goals. Seek feedback on patients' experience of the service.

A trauma-informed approach does not mean condoning unsafe behaviours or contributing to this with reckless prescribing. It is being clear about professional boundaries and allowing the person to choose whether they concur and are happy to work with the treatment team.

Substance use and addictive behaviours as chronic illnesses

Substance use and addictive behaviours as chronic illnesses

Substance use, gambling and gaming disorders are chronic illnesses, of which relapse (including slips and lapses) is an integral feature.

Relapse is a feature of chronic illness, not a failure of the person being treated.

Despite the challenges, effective treatments are available for substance use and addictive behaviours. People can recover good health and wellbeing, but as in any chronic illness, ongoing care is needed to achieve the best outcomes. Explaining this to the patient can foster agreement of realistic goals and convey an important message of hope.

Treatment goals vary with the individual; a person can recover good health, despite the challenging nature of their illness.

Positive health outcomes will be different for different people. Treatment goals are established by collaborating with the person (and ideally, those close to them). Treatment goals in disorders of substance use and addictive behaviours include:

- stopping the use or behaviour completely
- reducing the use or behaviour, combined with [harm reduction measures](#)
- continuing the use or behaviour at current levels but taking [harm reduction measures](#).

Informed consent and capacity in substance use and addictive behaviours

Informed consent and capacity in substance use and addictive behaviours

Informed consent is central to consultations with people with disorders of substance use or addictive behaviours. It is important to assess a person's capacity to give informed consent at each consultation. Consider whether the person has capacity to give consent at that moment or whether they are cognitively impaired. Impairment may be transient (due to an acute problem, such as intoxication) or long-term (due to a chronic condition, such as acquired brain injury).

Detailed advice (including for urgent situations) is available in [Informed consent and capacity in a person with a psychiatric disorder](#).

Legal considerations for prescribing in substance use and addictive behaviours

Legal considerations for prescribing in substance use and addictive behaviours

Real-time prescription monitoring records prescribing and dispensing of drugs with potential for dependence and provides an alert in certain high-risk circumstances, such as prescription from multiple providers, high-risk combinations of drugs or when the opioid dose threshold has been exceeded. It is important to check the real-time prescription monitoring system in the relevant state or territory for details of the patient's history of prescriptions; see the links in [Table 22.2](#).

Real-time prescription monitoring does not monitor all drugs, and the range of drugs monitored varies between systems. Communication between prescribers (eg in a collaborative-care model) is important to co-ordinate prescribing and avoid excess or missed supply.

It is also essential to know the relevant state or territory permit requirements to prescribe any Schedule 8 drugs to a person with substance dependence. Specific requirements apply for prescribing [medication-assisted treatment of opioid dependence](#); each state and territory has different legislation guiding this.

Table 22.2 State- and territory-based real-time prescription monitoring systems

State or territory	Real-time prescription monitoring system
Australian Capital Territory and Tasmania	Drugs and Poisons Information System Online Remote Access
New South Wales	SafeScript NSW
Northern Territory	NTScript
Queensland	QScript
South Australia	ScriptCheckSA
Victoria	SafeScript
Western Australia	Real Time Prescription Monitoring

Screening for and assessment of substance use and addictive behaviours

Screening for and assessment of substance use and addictive behaviours

History-taking in screening and assessment of substance use and addictive behaviours

History-taking in screening and assessment of substance use and addictive behaviours

Ask all new patients routinely and others (adolescents and adults) opportunistically and periodically about substance use and gambling. Disorders of substance use and gambling are common (and often co-exist) and people are reluctant to disclose these, often due to fear of stigma. Patients are more likely to disclose associated conditions such as anxiety, headaches, depression or hypertension, which can act as prompts for opportunistic enquiries. Ask also about video gaming in anyone presenting with [potential harms of gaming](#).

Asking about substance use and gambling should be part of routine preventive health care. Framing a screening question as part of routine health screening can reduce [stigma](#) and increase the likelihood of disclosure. An approach to seeking permission for screening could be 'Can I ask you some routine questions that I ask all my patients about habits that could affect your health? This includes things like exercise, nutrition, stress, weight, smoking, alcohol and other drugs and gambling. Is that OK?'

An approach to asking about these issues at follow-up visits could be framed as 'We've spoken before about your drinking/smoking/gambling. How is that all going now?'

The [ASSIST-Lite tool](#) can be used to screen for use of multiple substances and takes 3 to 5 minutes to complete. This validated questionnaire categorises risk for each substance and gives recommendations. For moderate-risk use, the recommendation is to provide a brief intervention; for high-risk use, brief advice and referral are recommended. A printed version of the questionnaire is also available for download from the [ASSIST Portal](#). Questions included in the ASSIST-Lite tool are given in [Table 22.3](#).

Table 22.3 ASSIST-Lite alcohol, smoking and substance involvement screening tool

[NB1] [NB2]

1 In the past 3 months, did you smoke a cigarette containing tobacco?

1a Did you usually smoke more than 10 cigarettes each day?

1b Did you usually smoke within 30 minutes after waking?

Risk category (total 'yes' answers) [NB3]:

0 = low; 1 or 2 = moderate; 3 = high

2 In the past 3 months, did you have a drink containing alcohol?

2a On any occasion, did you drink more than 4 standard drinks of alcohol?

2b Have you tried and failed to control, cut down or stop drinking?

2c Has anyone expressed concern about your drinking?

Risk category (total 'yes' answers) [NB3]:

0 or 1 = low; 2 = moderate; 3 or 4 = high

3 In the past 3 months, did you use cannabis?

3a Have you had a strong desire or urge to use cannabis at least once a week or more often?

3b Has anyone expressed concern about your use of cannabis?

Risk category (total 'yes' answers) [NB3]:

0 = low; 1 or 2 = moderate; 3 = high

4 In the past 3 months, did you use an amphetamine-type stimulant, or cocaine, or a stimulant medication not as prescribed?

4a Did you use a stimulant at least once each week or more often?

4b Has anyone expressed concern about your use of a stimulant?

Risk category (total 'yes' answers) [NB3]:

0 = low; 1 or 2 = moderate; 3 = high

5 In the past 3 months, did you use a sedative or sleeping medication not as prescribed?

5a Have you had a strong desire or urge to use a sedative or sleeping medication at least once a week or more?

5b Has anyone expressed concern about your use of a sedative or sleeping medications?

Risk category (total 'yes' answers) [NB3]:

0 = low; 1 or 2 = moderate; 3 = high

6 In the past 3 months, did you use a street opioid (eg heroin) or an opioid-containing medication not as prescribed?

6a Have you tried and failed to control, cut down or stop using an opioid?

6b Has anyone expressed concern about your use of an opioid?

Risk category (total 'yes' answers) [NB3]:

0 = low; 1 or 2 = moderate; 3 = high

7 In the past 3 months, did you use any other psychoactive substances?

If yes, what did you take?

(Not scored, but prompts further assessment)

NB1: An interactive and printable ASSIST-Lite tool is available for download at the [AssistPortal](#).

NB2: Versions of the questionnaire validated in people younger than 18 years (ASSIST-Youth 10 to 14 years or 15 to 17 years) are available at the [ASSIST portal](#).

NB3: Provide a brief intervention relevant to the risk category.

Reproduced with permission from: Assist Portal. ASSIST-Lite: Alcohol, Smoking and Substance Involvement Screening Test. The University of Adelaide; Accessed September 2022. <https://assistportal.com.au/resources/>

As an alternative to the ASSIST-Lite tool, substances and addictive behaviours to consider on initial screening include:

- nicotine (cigarettes, vaping products, chewing tobacco)
- alcohol
- cannabis (nonprescribed and prescribed forms [\[Note 2\]](#))
- stimulants (metamfetamine, ecstasy, cocaine)
- heroin, gamma-hydroxybutyrate (GHB), ketamine or lysergic acid diethylamide (LSD)
- prescription and over-the-counter drugs (eg opioids, benzodiazepines, zolpidem, zopiclone, pregabalin, gabapentin, quetiapine and other antipsychotics, sedating antihistamines [\[Note 3\]](#))
- other substances such as volatile inhalants (eg nitrous oxide, petrol, solvents, acetone, butane)
- gambling.

Ask also about video gaming in anyone presenting with [potential harms of gaming](#).

Following identification of substance use, gambling or gaming, it is useful to further categorise use or addictive behaviours according to International Disease Classification criteria, see [Figure 22.1](#). Ask about:

- frequency of substance use (and method of ingestion) or addictive behaviours
- whether the person or anyone close to them is concerned about their use or behaviours
- motivations to use, gamble or game—see [Brief interventions](#) for helpful questions
- potential harms
- previous efforts to reduce substance use/gambling/gaming, including what did and did not work, withdrawal symptoms and their severity
- concurrent mental illness—for many people, their behaviour is driven by a wish to relieve symptoms or numb emotions; many also experience mental health harms

- possibility of pregnancy
- [suicide risk](#), past history of overdoses, history of self-harm
- domestic violence and child protection considerations.

Diagnostic questionnaires can contribute to gathering information for screening and assessment; individual topics in these guidelines provide links.

If a person appears intoxicated or in withdrawal, keep the history-taking brief. Ask about withdrawal symptoms to help identify dependent use. Defer efforts to quantify use until the person is oriented, attentive and coherent. Exclude differential diagnoses of suspected intoxication or withdrawal, including [precipitants of delirium](#), such as [diabetic ketoacidosis](#), hypoglycaemia, infection and intracranial events.

Note 2: Prescribed cannabis products (medicinal cannabinoids) are extracts of cannabis plant material or synthetic products.

Note 3: Prescription drugs include medicinal cannabinoids which form part of the earlier list item enquiring about cannabis use.

Examination and investigations in assessing substance use and addictive behaviours

Examination and investigations in assessing substance use and addictive behaviours

A **general physical and mental state examination** may identify signs of:

- intoxication, withdrawal or comorbid mental illness—identification of psychosis or marked paranoia is essential in assessing a person's decision-making capacity and the risk of serious harm to them and others. For advice on informed consent and assessing capacity, see [Informed consent and shared decision-making in a person with a psychiatric disorder](#)
- organ damage from substance use
- dental disease (caused by lack of oral care or drug effects such as dry mouth, tooth grinding and vascular damage to gums)
- malnutrition
- injecting-related harms (eg viral or bacterial bloodborne infections)
- pregnancy
- sexually transmitted infections.

Investigations can support treatment decisions but are generally not useful as screening tools.

Investigations to consider when assessing substance use include tests to detect:

- substance-specific organ damage—liver biochemistry is useful to assess the effects of alcohol use on the liver (but is insensitive as a screening test for alcohol use)
- harms associated with the route of use such as injecting harms, including site infections (eg cellulitis, fasciitis, abscesses) and bloodborne infections (eg [hepatitis B](#), [hepatitis C](#), [HIV](#), [bacterial endocarditis](#)); infections can also result from using pipes and bongs (water pipes) but this is less common
- consequences of other risky behaviours such as unprotected sexual intercourse (eg sexually transmitted infections, unplanned pregnancy).

For some disorders of substance use, specific investigations should be considered before starting drug therapy; these are discussed in the specific topics.

Routine **urine drug screening** is not generally required in primary care, but discussing its role in treatment decisions and safe prescribing can open conversations about substance use. It can be useful to ask 'If I did a urine drug screen today what would I find in your urine?'. Screening may be useful when transferring patient care, or may be required in specific situations (eg by child protection agencies, changing prescribers of [medication-assisted treatment of opioid dependence](#)). Testing specified by courts requires specific techniques and demonstration of a chain of custody of a sample. Interpretation of urine drug screens is complicated by factors such as false positives and negatives; advice from a pathologist is recommended before interpretation. Not all commonly prescribed drugs are part of a standard urine screen covered by the Medicare Benefits Schedule (MBS). Confirmatory urine drug testing is not reimbursed. Pathology costs may add financial burden for the patient; shared decision-making about testing is recommended. Before ordering a urine drug screen, determine the availability, utility and cost from local pathology providers because these factors vary with location and over time.

When to ask for help or refer a person with a disorder of substance use or addictive behaviour

When to ask for help or refer a person with a disorder of substance use or addictive behaviour

Seeking specialist advice is encouraged for any aspect of care for people experiencing harm or at risk of harm from substance use or addictive behaviours. Sources of expert advice include specialists in addiction medicine, addiction psychiatry or general psychiatry, nurse practitioners and clinical psychologists.

For sources of clinical advice by phone and service referral details for patients with **disorders of substance use**, see [Contact details for substance use clinical advisory services for clinicians](#).

For advice on managing or referring patients with **gambling disorder**, clinicians can contact [specialist gambling treatment services](#) in some areas. In all states and territories, [gambling support services](#) advise patients directly but also welcome contact from clinicians to source help for patients.

Advice on managing patients with **gaming disorder** is most likely to be available from an expert in adolescent mental health (eg psychiatrist, clinical psychologist, nurse practitioner); see [Support services and specialist treatment for gaming disorder](#).

Referral is encouraged for patients with complex needs, including complicated physical health concerns (eg severe liver disease) or any other problems that may impact adequate patient care. Indications for urgent referral include suicide risk and other urgent safety concerns (eg overdose).

Specialist advice is available by phone on the management of substance use or addictive behaviours; contact is encouraged.

People can benefit from their specialist(s) and usual clinician(s) working collaboratively to formulate a chronic disease management plan. Communication between prescribers is important to co-ordinate prescribing and avoid excess or missed supply; either one practitioner does all the prescribing or there is an agreement about which medications each practitioner will prescribe. [Real-time prescription monitoring systems](#) do not monitor all drugs, and the range of drugs monitored varies between systems.

Coordination of prescribing is important in a collaborative-care model to avoid excess or missed supply.

Overview of interventions for disorders of substance use and addictive behaviours

Overview of interventions for disorders of substance use and addictive behaviours

Establishing a therapeutic relationship that engages the person (and ideally those close to them) is central to the management of substance use and addictive behaviours. Use an approach based on [trauma-informed care](#) and awareness of [stigma](#).

The most important element of managing disorders of substance use or addictive behaviours is establishing a therapeutic relationship.

The range of interventions for people with a disorder of substance use or addictive behaviour includes:

- [ensuring the safety](#) of the person and those around them
- [brief interventions](#)
- [psychosocial interventions](#)
- drug therapy to help reduce or stop substance use and prevent relapse—available for managing [tobacco use and nicotine dependence](#), [opioid dependence](#) and (once abstinence is achieved) [alcohol use](#)
- [gradual weaning of medications](#)
- short-term [withdrawal management in disorders of substance use](#)
- [harm reduction](#)
- [long-term care](#) to address broad aspects of health care and offer regular opportunities for a person to review their options.

Ensuring the safety of a person with a disorder of substance use or addictive behaviour

Ensuring the safety of a person with a disorder of substance use or addictive behaviour

Substance use and addictive behaviours can pose risk of acute harms to the person (such as self-harm, overdose [accidental or deliberate], suicide, falls and other accidents) and those around them (through impacts on driving, childcare, fitness to work and acute behavioural disturbances).

Safe management of acute behavioural disturbance in disorders of substance use or addictive behaviours

Safe management of acute behavioural disturbance in disorders of substance use or addictive behaviours

If a person with a disorder of substance use or addictive behaviour is highly agitated, see [Approach to managing acute behavioural disturbance](#) for advice on assessing safety, reducing risk of harm to the person and staff, verbal de-escalation and pharmacological management. [Precipitating factors for development of acute behavioural disturbance](#) includes acute medical conditions to consider as causes of acute behavioural disturbance.

For advice on managing acute medical aspects of intoxication, including support of the airway and circulation, see [Overview of treatment for poisonings](#). Substance-specific advice on intoxication is also available in the Toxicology Guidelines (eg [Opioid poisoning](#), [Benzodiazepine poisoning](#)). Mixed intoxication from multiple substance ingestion increases risk.

For advice on assessing capacity and considering issues of consent, see [Informed consent and capacity in substance use and addictive behaviour](#).

Even mildly intoxicated patients may pose risk to themselves and others (eg if planning to drive or be responsible for children while affected by alcohol). Healthcare workers have a duty of care to prevent a patient from taking such risks. Options to ensure the safety of an intoxicated person include:

- contacting a friend or family member to take them home where they will have support from a carer (or to an emergency department if further monitoring or intervention is needed)
- calling an ambulance if urgent transfer to an emergency department or mental health facility is needed
- calling the police if the person's behaviour is too aggressive for other measures, if the person is a risk to themselves or others, or if the person absconds.

If unsure how to manage the situation, seek specialist advice from a [drug and alcohol clinical advisory phone service](#).

Child and family safety in disorders of substance use and addictive behaviours

Child and family safety in disorders of substance use and addictive behaviours

When seeing a patient with a disorder of substance use or addictive behaviour who has children in their care, consider whether their ability to provide safety and care is impaired, and whether the level of risk requires a mandatory notification. For advice on mandatory notification of child abuse and neglect, see the [Australian Institute of Family Studies website](#). For help locating a child protection case worker in each state and territory, see the [Department of Social Services website](#).

Disorders of substance use and addictive behaviours can exacerbate family and intimate partner violence. For further information, see the Royal Australian College of General Practitioners (RACGP) [guideline on abuse and violence](#) (The White Book).

Fitness to drive and disorders of substance use

Fitness to drive and disorders of substance use

A person cannot hold an unconditional driving licence if they have certain medical conditions, including:

- dependence on (or heavy frequent use of) alcohol or other substance(s) that are likely to impair safe driving [\[Note 4\]](#)
- a history of a seizure.

The [AustRoads website](#) has advice on assessing fitness to drive, advising patients about driving, and when to consider notifying the licensing authority directly about concerns regarding a person's fitness to drive.

Discussions about driving depend on a therapeutic alliance and agreement of a plan for regular review of fitness to drive. The review plan should be agreed before treatment starts. Advise patients who are planning a short-term withdrawal intervention that they should not drive until the withdrawal process has been completed; this applies whether or not they take medication to manage withdrawal. Other contraindications to driving include feeling sedated or unstable drug use.

If a person has had a seizure suspected to result from substance withdrawal, advise the person not to drive until cleared by a specialist. Licensing for resuming driving may be conditional on continued abstinence and monitoring by an addiction specialist. Explain the importance of abstaining to reduce recurrence risk.

Note 4: Driving may be impaired by deficits in short-term memory and learning, perceptual-motor skills (ability to combine perceptual information with motor skills), visual search and scanning strategies, executive functions (mental flexibility, problem-solving, planning, prioritising, focusing attention, sustaining or shifting focus, or controlling impulsivity).

Occupational implications of disorders of substance use

Occupational implications of disorders of substance use

Substance use poses risk for operating potentially dangerous machinery, working in hazardous environments and occupations such as truck driving, public transport operators, pilots, oil rig and mining employees, and healthcare workers. Some workplaces (eg in the mining and airline industries) operate with a 'zero tolerance' policy that does not permit any substance use while an employee is working; random workplace alcohol or other drug testing is undertaken to monitor use.

It is a professional requirement that a healthcare practitioner functions without the influence of alcohol or other drugs, but workplace monitoring usually does not occur unless intervention from the relevant registering board has been required. The [Australian Health Practitioner Regulation Agency \(AHPRA\) website](#) has advice on raising concerns about a healthcare practitioner and on sourcing support for a practitioner who may be seeking help for substance use.

Brief interventions for disorders of substance use and addictive behaviours

Brief interventions for disorders of substance use and addictive behaviours

Brief interventions use techniques such as motivational interviewing, in which the clinician helps a person reflect on the pros and cons of their substance use, gaming or gambling and to uncover their motivation to change. [Table 22.4](#) provides examples of questions. The clinician can offer brief advice on options for next steps and work with the person to agree a plan.

Table 22.4 Motivational interviewing questions for managing disorders of substance use and addictive behaviours

Brief version: The following questions can be used within a 5-minute discussion.

Question	Rationale
What do you like about your [substance use or behaviour]?	This question is unexpected as most patients would be expecting a lecture. It gives an opportunity to listen and build rapport and may give valuable information that helps to understand the context of the behaviour. It may also be important to consider how to replace this function if the behaviour were to stop.
What don't you like about your [substance use or behaviour]?	This question is critical as it draws out the internal motivation for change.
[Add your own concerns if appropriate]	Add your own concerns about the behaviour if you believe this might help to tip the balance towards change and not increase resistance.

Briefer version: The following questions can be used within a 1- to 2-minute discussion.

Question	Rationale
On a scale of 1 to 10 where 10 is a lot, how much do you want to [change the substance use or behaviour]?	Patient usually pick a number higher than 2. Regardless of the answer, you can usually ask the next question.
Why so high; why is it not [a lower number]?	The patient tells you why they want to change (argues for change).
So what do you want to do about it?	The patient states how to move towards change. Aim to get a commitment to a plan, which might range from action to change, an agreement to return to discuss further, or an agreement for the issue to be raised again later.

Reproduced with permission from The Royal Australian College of General Practitioners from: Sim MG, Wain T, Khong E. Influencing behaviour change in general practice: Part 2 – Motivational interviewing approaches. Aust Fam Physician 2009;38(12):986–89. Available at www.racgp.org.au/getattachment/201f2425-e6e8-4965-83c5-9017a92d2ec8/Influencing-behaviour-change.aspx [Accessed 22 September 2022].

For more information about motivational interviewing, see:

- Sim MG, Wain T, Khong E. Influencing behaviour change in general practice - Part 1 - brief intervention and motivational interviewing. Aust Fam Physician 2009;38(11):885-8. [URL](#)
- Sim MG, Wain T, Khong E. Influencing behaviour change in general practice - Part 2 - motivational interviewing approaches. Aust Fam Physician 2009;38(12):986-9. [URL](#)
- a self-directed [online course at Flinders University](#).
- the [Insight website](#)—this includes substance-specific brief motivational interviewing tools (available as tear-off forms, which can be ordered) with a takeaway summary.

Psychosocial supports in disorders of substance use and addictive behaviours

Psychosocial supports in disorders of substance use and addictive behaviours

Psychosocial support addresses the complex needs of people with substance use and addictive behaviours; it includes:

- practical assistance with housing, financial difficulties, legal and employment issues, social difficulties
- psychological interventions
- peer support.

It is important to address a person's immediate needs before considering structured behavioural interventions. Offer support regardless of whether a person adopts other therapies such as drug treatments. A flexible approach is needed to work with a patient to regularly review achievable goals. Printable patient information on how to access a range of supports is available in [Patient resources and support organisations for substance use and addictive behaviour](#).

[Psychological interventions](#) have a major role in the treatment of substance use and addictive behaviours. They can assist in reducing or stopping use of substances and addictive behaviours, and increase retention in treatment. Psychological interventions may include:

- cognitive behavioural therapy—this explores negative thoughts, provides training in social skills and managing cravings, sometimes by graded exposure to situational cues
- acceptance and commitment therapy—this focuses on accepting and managing distressing emotions and thoughts
- contingency management—this is a behavioural therapy that rewards individuals for positive behavioural change
- family interventions and family therapy.
- motivational enhancement therapy—this uses [motivational interviewing](#) with structured feedback, planning and building motivation to change.

While most support is provided on an outpatient basis, for some patients, residential rehabilitation may be beneficial; see [Long-term care in disorders of substance use and addictive behaviours](#).

Weaning for management of disorders of substance use

Weaning for management of disorders of substance use

Weaning (deprescribing) describes the gradual reduction of medication doses, until an appropriate safer dose is reached, or the drug is stopped. Staged supply (dispensing small amounts of medication) can support weaning; see [Safe prescribing and supply during planned substance withdrawal in a home setting](#) for more detail. Advice is available in these guidelines for weaning [benzodiazepines](#), [zopiclone](#), [pregabalin](#) and [gabapentin](#), [quetiapine](#) and [other antipsychotics](#) and [sedating antihistamines](#).

For some patients with [chronic pain](#), weaning opioids is appropriate; the Pain and Analgesia Guidelines provide advice on [deprescribing opioids and adjuvants](#). [Medication-assisted treatment of opioid dependence](#) can also be considered for some patients with chronic pain.

Withdrawal management in disorders of substance use

Withdrawal management in disorders of substance use

In these guidelines, the term ‘withdrawal management’ describes short-term interventions (eg over 7 days) that provide support (and sometimes medications to manage symptoms) to reduce substance use safely, usually with the aim of stopping use. Gradual reduction in use is considered separately in substance-specific topics and includes [weaning of medications](#).

Unplanned withdrawal can occur when a person is suddenly unable to use a substance, for example when admitted to hospital for an acute medical or surgical illness. The risk of complications is increased in unplanned withdrawal, particularly with delayed diagnosis of alcohol or benzodiazepine withdrawal; seek [specialist advice](#) as outlined in substance-specific topics.

The following sections outline how best to plan for and implement safer short-term withdrawal interventions.

Choice of setting for planned withdrawal management in substance use

Choice of setting for planned withdrawal management in substance use

Planned withdrawal management can take place at the patient’s home, in a community residential withdrawal unit or in a hospital with medical monitoring of withdrawal. Choosing the appropriate setting for planned withdrawal management depends on the level of associated risk. This varies with substance, method of ingestion, extent of use, number of substances used, duration of use, and individual factors (eg age, physical and mental health). A history of symptoms in previous withdrawals is generally a good indicator of risks for subsequent attempts. Guidance for selecting a suitable withdrawal setting is outlined below; see substance-specific topics for additional considerations. Work with the patient in assessing their needs and preferences for a suitable setting. Seek local specialist advice on the best location for management of withdrawal that is predicted to be moderate to severe.

Suggested **requirements for withdrawal management in a home setting** include the following:

- Withdrawal is predicted to be of mild to moderate severity with no medical or psychiatric contraindications.
- The patient wants home management and can comply with instructions, including avoiding driving until the withdrawal process is complete.
- The patient has a safe and supportive environment.
- The GP, pharmacist or other involved healthcare professionals are able and willing to provide frequent reviews, which may include [supervised dosing and staged supply of medications](#).
- A carer is available to support the patient, supervise medication and call for help if deterioration occurs, using an agreed deterioration plan (eg to phone an agreed health professional to escalate care, which may involve a home visit or transfer to a community residential withdrawal unit).
- The patient can arrange their responsibilities and commitments to give themselves a period of minimal stress and sufficient time for withdrawal.

Withdrawal management in a **community-based residential withdrawal unit** (with staff trained in withdrawal monitoring but without 24-hour a day medical support) may be suitable if the person:

- is predicted to experience mild to moderate withdrawal
- is in an unsupportive home environment
- is living alone
- is homeless
- has had more than one unsuccessful attempt at withdrawal at home
- has dependent use of more than one substance (polysubstance use).

Withdrawal management in a **hospital setting with close medical monitoring** may be suitable if the person has:

- an acute medical, surgical or psychiatric condition
- uncertain drug use or a history of a condition that indicates a need for close medical monitoring
- a history of severe or complicated withdrawal.

Safe prescribing and supply during planned substance withdrawal in a home setting

Safe prescribing and supply during planned substance withdrawal in a home setting

Comply with [legal requirements](#) when prescribing medications to manage substance withdrawal, including use of real-time prescription monitoring.

If a patient is being managed collaboratively, communication between prescribers is important to co-ordinate prescribing and avoid excess or undersupply.

If short-term treatment with a benzodiazepine, zolpidem, zopiclone or an antipsychotic is required to manage withdrawal symptoms, only prescribe the number of tablets required for the course (not a full pack) to limit risk of dependence or overdose. Seek specialist advice if considering prescribing combinations of any of these drugs. Warn patients about potential sedation and advise them to avoid concurrent use of alcohol or other drugs with sedative effects. Warn patients not to drive until the course of medication and the withdrawal process are completed; for more information on driving, see [Fitness to drive and substance use](#).

Consider supervised dosing or staged supply at the patient’s local pharmacy. **Supervised dosing** is an arrangement in which a patient attends a pharmacy and is given their medication to take in the pharmacy. This is useful when a prescriber wants to ensure the medicine is taken at a certain time by a specific person as directed by their clinician. **Staged supply** refers to dispensing small amounts of medicine (eg daily, second daily, weekly) [\[Note 5\]](#) to support the patient to better manage and maintain the prescribed dose. Pharmacists can be reimbursed for staged supply by becoming an approved provider; see the [Pharmacy Programs Administrator website](#). Supervised dosing and staged supply need to be negotiated with the patient and the pharmacist. They can be used as a condition of prescribing to support the patient to achieve their goals and maintain safety. It is important to convey that these options are additional support to assist a person to take medication safely and are not coercive or punitive.

Note 5: The dispensing interval needs to be negotiated with the patient and pharmacist and depends on the patient’s stability (ie risk of overdose), whether the person has a responsible carer to supervise dosing, and practical considerations (eg opening hours and travel time to the pharmacy).

Harm reduction in substance use and addictive behaviours

Harm reduction in substance use and addictive behaviours

Harm reduction strategies aim to reduce the harmful consequences of substance use or addictive behaviours for the individual, those close to them and the community. Offer advice on harm reduction to people who use substances, gamble or game. Some people will not choose to stop substance use or addictive behaviours but will consider measures to limit risk. They may be open to revisiting their options for behavioural change later, particularly if information about harm reduction is provided in a nonjudgemental way. The general [principles of care](#) in chronic disease management are important, particularly maintaining a therapeutic relationship and a sense of hope.

Interventions for harm reduction that clinicians can offer are outlined in [Figure 22.2](#). A [printable summary of harm reduction actions](#) that patients can undertake is available.

Additional specific harm reduction advice for patients is available for [gamma-hydroxybutyrate \(GHB\)](#).

Figure 22.2 Interventions by clinicians for harm reduction in substance use and addictive behaviours

Educate patients about ways to reduce harms from use of alcohol or other drugs, gambling or gaming; see the printable patient information sheet [Table 22.5](#).

Offer [thiamine supplementation](#) in managing alcohol use to limit neurological damage.

Review vaccination status—consider the need for vaccination against hepatitis B and human papilloma virus to reduce risks from unprotected sex (and in the case of hepatitis B, other routes of transmission, such as by sharing needles).

Offer access to opioid overdose prevention ([naloxone](#)).

Offer advice on sexual health, including [contraception](#), pregnancy testing, and prevention and management of [sexually transmitted infections](#), including [HIV pre-exposure prophylaxis \(PrEP\)](#) and [HIV postexposure treatment \(PEP\)](#).

Offer prenatal, pregnancy and breastfeeding care to limit risk of substance use to a fetus or infant; refer early to services experienced in management of substance use in pregnancy and breastfeeding individuals.

Treat organ damage from substance use (eg smoking-related lung disease, alcohol-related liver disease).

Offer referral for:

- dietary management of malnutrition
- dental care (see [Examination and investigations in assessing substance use or addictive behaviour](#) for a list of dental harms)
- physiotherapy and occupational therapy aspects of falls prevention
- [psychosocial support](#) to manage impact on finances, family and child welfare.

Table 22.5 How to get help and reduce harms from alcohol and other drugs or gambling—patient information

[Printable table](#)

This table lists some ways you can use alcohol or other drugs or gamble more safely.

Whether it's for yourself or someone you're worried about, you can get free confidential information, support and practical help 24 hours a day 7 days a week anywhere in Australia. This table suggests some of the things you can ask about on the phone lines or websites.

For any **alcohol or other drug concerns**, call the national Alcohol and Drug Information Service on **1800 250 015** or go to <https://www.counsellingonline.org.au>.

For any **gambling concerns**, call the National Gambling Helpline on **1800 858 858** or go to <https://www.gamblinghelponline.org.au>.

Finding drugs

Examples of how you could be harmed	Possessing or buying street drugs can cause you legal trouble.
	People who sell drugs or others buying drugs might assault you.
Some steps to consider or ask about	Find out your legal rights and how to get legal advice (these are not the same in all states or territories).
	Avoid unfamiliar drug dealers and locations.
Paying for drugs, alcohol or gambling	
Examples of how you could be harmed	Costs of drugs, alcohol or gambling could put you in debt or lead you into crime.
	Ask for help to manage your money and any debts.
	Avoid paying for drugs, alcohol or gambling with credit cards.
Some steps to consider or ask about	Find out how: <ul style="list-style-type: none"> • gambling venues can help you limit your spending • to put a spending limit on your credit or debit card • to get yourself barred from going into specific gambling venues.

Getting drugs into your body

Examples of how you could be harmed	If you inject drugs, you are at risk of infections from: <ul style="list-style-type: none"> • bloodborne viruses (eg hepatitis B, hepatitis C, HIV) • bacteria causing skin inflammation or infection and other damage (eg to heart valves).
	Smoking drugs can damage your lungs.
	Using a pipe can cause mouth burns.
	Find out about your nearest needle and syringe programs .
Some steps to consider or ask about to reduce risk from injecting	Learn about safer injecting; see the Touchbase toolkit .
	Inhale or swallow 'ice' (crystal meth) instead of injecting it.
	Consider switching from smoking cannabis to using a dry-herb cannabis vaporiser (a medical device from a pharmacy, not any other type of vaping device); do not use cannabis oil in a vaping device.
Some steps to consider or ask about to reduce harm from smoking drugs	If using a pipe, use a Pyrex one to avoid mouth burns, and clean it regularly.
	Change the water in a bong (water pipe) after each use to avoid breathing in bacteria.
	Do not share pipes because this can spread infections.
Not looking after yourself when using drugs, alcohol or gambling	
Examples of how you could be harmed	Not eating well could make you lose weight, get infections and damage your general health.
	Tooth grinding, dry mouth (from some drugs) and not brushing can damage teeth and gums.

Sleeping less or irregularly can damage your physical and mental health.

Ask about practical help you might need (eg with housing or food). You can anonymously search the [AskIzzy website](#) for local help in a crisis.

Aim to set up regular patterns for:

Some steps to consider or ask about

- eating nonsugary foods
- drinking enough water
- mouth care (tooth brushing, flossing, use of lip balm and sugar-free gum)
- resting and sleep.

Intoxication (being drunk or high)

Ask for medical help with any physical or mental health concerns.

Overdose can cause severe harms or death.

Examples of how you could be harmed

Your work, home duties or schooling might be affected.

You or others around you could become aggressive or violent.

You could risk unsafe sex, needle sharing, drunk-driving or drug-driving and crime.

Have someone with you who isn't drinking or using.

Plan in advance how you'll get home safely.

Know how to call an ambulance and give first aid; see the [Australian Red Cross First Aid app](#).

Write what drugs you've taken on the back of your hand in case you pass out; this could help you get the right emergency care.

Keep track of how much alcohol you drink or how much you use of any drug (apps can help).

Cut down the number of times you use more than one drug (including alcohol) at a time.

Find out which ways of getting drugs into your body will give you a smoother high.

Space alcoholic drinks out with water or other nonalcoholic drinks.

Space out bongs or cut down the amount of cannabis in each one.

Some steps to consider or ask about

If using oral cannabis (edibles) try a small amount and wait 1 to 2 hours before having more.

Use cool packs and rest to help with anxiety from stimulant (eg 'ice') use.

Ask about take-home naloxone (an antidote to opioids like heroin or prescription painkillers) if you or someone you know uses these drugs.

Plan your drug or alcohol use to allow time for other life activities and duties.

Reduce risk of violence by staying out of overcrowded clubs, raves or other events.

Carry condoms and lube.

Find out about other contraceptives and ways to reduce risk of infections (eg HIV) and have regular sexual health checks.

Leave the car at home if you are going to use drugs or drink while out. Don't 'drink-drive', 'drug-drive' or operate machinery while drunk or high.

Having a hangover (crash) after using alcohol or drugs

Examples of how you could be harmed

Your work, home duties or schooling might be affected.

Some steps to consider or ask about

Plan your drug use or drinking to allow time for other life activities and duties.

Try to get enough food and sleep.

Going into withdrawal from alcohol or drugs

Examples of how you could be harmed

You could become unwell and have seizures (fits), hallucinations and distress (depending on what you drink or use and how much you have).

If planning withdrawal, organise to do it with medical help to make it safer and more comfortable.

Some steps to consider or ask about

Plan (with medical help) for what to do if you have unexpected withdrawal.

Long-term care in disorders of substance use and addictive behaviours

Long-term care in disorders of substance use and addictive behaviours

Long-term management of disorders of substance use or addictive behaviours focuses on maintaining a therapeutic relationship and offering a range of strategies, including:

- case management—a co-ordinated approach that considers physical and mental health, social networks, sexual health, nutritional advice and dental care
- access to a range of outpatient [psychosocial supports](#)
- revisiting options for [interventions](#) to reduce use or addictive behaviours as the situation and goals change
- residential rehabilitation.

Residential rehabilitation communities are drug and alcohol-free therapeutic communities that offer withdrawal and relapse prevention during stays of generally 1 to 24 months. Interventions include individual or group counselling, peer support and help re-integrating into the community. Residential rehabilitation may benefit people who specifically seek a long-term commitment or those who have not had success with other therapies, but evidence is limited.

Considerations for specific populations in substance use and addictive behaviours

Considerations for specific populations in substance use and addictive behaviours

Specific populations may have particular risks from substance use or addictive behaviours; some may be more vulnerable to harms at lower levels of use, or harms that are more likely to be overlooked. Interventions and resources specific to their situation may be relevant, such as referral to a specialist or collaboration with a specialist service. Considerations include:

- [age](#)
- [gender and sexuality](#)
- [Aboriginal or Torres Strait Islander origin](#)
- [other cultural and language considerations](#)
- [mental illness](#)
- [chronic pain](#)
- [other populations](#) such as patients with other comorbidities, developmental disabilities, people recently released from prison, and specific occupational groups.

Pregnancy and breastfeeding considerations in substance use are discussed in the substance-specific topics in these guidelines.

Age considerations in substance use and addictive behaviours

Age considerations in substance use and addictive behaviours

The developing brain of an **adolescent** is more sensitive than the adult brain to long-term damage from substance use and addictive behaviours; decision-making and academic performance can be harmed and the risk of substance dependence in adulthood is increased by early substance use. Some young people present because of others' concerns about their substance use. [Family therapy](#) may be needed to agree goals and the pace at which changes are made. Activity therapy (programs that engage young people in fun activities while also providing psychoeducation) has been very effective in some situations, for example in treating [volatile inhalant use](#). For resources to support management of substance use and addictive behaviours in young people, see [Substance use and addictive behaviours in young people: additional resources for clinicians](#).

In **older people**, physical and mental illnesses (including chronic pain, grief and isolation), multiple medications and adverse effects increase risk of harms (eg cognitive impairment and falls) from substance use or addictive behaviours. Long-term substance use increases the risk of age-related disease (eg cognitive decline, which can limit the use of treatments that require learning new skills). Tobacco causes most harm, followed by alcohol. The prevalence of hazardous alcohol use is rising in people older than 50 years, and those older than 70 years are most likely to report daily drinking. Ask also about illicit drug use; older people may not be considered likely to use drugs, but one-third of those on [treatment for opioid dependence](#) are 50 years or older. Consider the effect of age when assessing and managing substance use, because older people are at greater risk of toxicity (eg from [benzodiazepines](#), [zolpidem](#) and [zopiclone](#)). The [Older Wiser Lifestyle \(OWL\) Program](#) is the first drug and alcohol service specific to the needs of adults aged 60 years and older.

Gender and sexuality considerations in substance use and addictive behaviours

Gender and sexuality considerations in substance use and addictive behaviours

Gender and sexuality affect a person's level of risk of harm, types of harms and their needs for a particular approach to their health care.

In populations of [diverse gender](#), stigma, discrimination and trauma related to sexuality or gender are common contributors to a high prevalence of substance use. Populations of diverse sexuality or gender may engage in 'chemsex' (using substances such as [gamma-hydroxybutyrate \(GHB\)](#), [stimulants](#) [eg ecstasy or metamfetamine] or amyl nitrite [a [volatile inhalant](#)] to enhance sexual experience). Consider seeking advice from or referral to a sexual health expert for help with [harm reduction measures](#) for sexual health. For links to information on trans health for individuals and healthcare providers, see [Trans and gender diverse health](#). For links to alcohol and other drug services with a specific focus on diverse gender or sexuality, see [Substance use in populations of diverse sexuality or gender: additional resources for clinicians](#).

Females develop disorders of substance use or addictive behaviours earlier than males and experience harms at lower levels of substance use. Females are more likely to be introduced to substance use by their intimate partner or peer groups, and to engage in sex work to support their use. Although the prevalence of [gambling disorder](#) is lower in females than males generally, females who play poker machines are at higher risk of harm than males. Females benefit from services that consider gender-specific needs such as the opportunity for single-gender peer support as well as support of pregnancy, breastfeeding and family needs (eg supporting childcare and development of parenting skills).

Males have higher prevalence of substance use and drug-related death (including overdose and chronic illness) than females. Disorders of gambling and gaming are also more prevalent in males than females.

Considerations for Aboriginal and Torres Strait Islander peoples with disorders of substance use and addictive behaviours

Considerations for Aboriginal and Torres Strait Islander peoples with disorders of substance use and addictive behaviours

In Aboriginal and Torres Strait Islander peoples, severe intergenerational trauma through dispossession is a potent driver of substance use and harms. Gambling is more common than in the general population, and treatment-seeking is low. Tobacco is the most used substance. Alcohol use is less prevalent than in the general population, but people who use alcohol (or other substances [[Note 6](#)]) have a higher risk of harms. For resources to support culturally sensitive management of smoking, alcohol and other drug use, see [Substance use in Aboriginal and Torres Strait Islander peoples: additional resources for clinicians](#).

Note 6: The prevalence of cannabis use is second to tobacco.

Considerations for other culturally and linguistically diverse populations in substance use and addictive behaviours

Considerations for other culturally and linguistically diverse populations in substance use and addictive behaviours

People from culturally and linguistically diverse backgrounds are less likely to use substances or gamble than the general population, but individuals who do are often at higher risk of harms. A history of torture, trauma, grief and loss increases risk. The sensitivity of treatment services to language and cultural needs is important for improving access.

Mental illness considerations in substance use and addictive behaviours

Mental illness considerations in substance use and addictive behaviours

Comorbid psychiatric conditions are common in people with disorders of substance use and addictive behaviours. Resources to aid management of mental illness in substance use are available at [Dual Diagnosis Australia and New Zealand](#). For resources to support management of people with mental illness and gambling harms, see [Contact details for specialist gambling therapy services for referral](#).

Mental illness and tobacco use

Mental illness and tobacco use

Despite high rates of smoking, people with severe mental illness are less frequently offered help in managing their tobacco smoking, often due to the misconception that change is not possible in this population. Conditions such as schizophrenia are not contraindications to reducing or stopping smoking.

Patients may need closer monitoring for exacerbation of psychiatric symptoms as they reduce their smoking, particularly if nicotine withdrawal symptoms are not well-controlled. High doses of nicotine replacement therapy may be needed as nicotine dependence is more likely to be severe than in the general population. Varenicline is appropriate for the management of tobacco smoking in people whose psychiatric condition is stable; use bupropion with caution in people with bipolar disorder as there is a risk of mania.

Smoking promotes CYP1A2 metabolism of some psychotropic drugs, in particular clozapine; lower doses may be required for patients taking these drugs as they reduce their smoking. See the [Quit website](#) for a table of interactions and management advice.

Mental illness and cannabis use

Mental illness and cannabis use

Cannabis use may increase the risk of developing schizophrenia, particularly in people who start at a young age or have heavy use. Cannabis can contribute to relapse of mental illness in people with schizophrenia, bipolar disorder or depression. Many patients with mental illness or social difficulties use considerable quantities of cannabis. Charting episodes of cannabis use and relapse may be a motivational tool, showing how use leads to hospitalisation and other adverse outcomes.

Cannabis withdrawal can unmask undiagnosed psychiatric illness, which can present as a [first episode of psychosis](#), trigger a re-emergence or precipitate deterioration of symptoms. Consider the need to admit a patient to manage withdrawal if they are at risk of these events.

Chronic pain in substance use and addictive behaviours

Chronic pain in substance use and addictive behaviours

Chronic pain is a common comorbidity in people with a disorder of [opioid use](#), [pregabalin or gabapentin use](#) or [cannabis use](#).

The Pain and Analgesia guidelines provide advice on the [role of opioids in chronic noncancer pain](#) and the [role of adjuvants for chronic noncancer pain](#). Consider ways to reduce opioid use (eg weaning [deprescribing] or medication-assisted treatment of opioid dependence) for all patients with chronic pain because of the risk of harms (even when the person is adhering to the prescriber's advice) and seek specialist advice for complex situations.

It can be challenging to assess whether a patient with chronic pain has a [disorder of substance use](#). A possible or known disorder of substance use in a patient with chronic pain is an indication for specialist advice or referral to evaluate the options for [weaning medications](#) or [medication-assisted treatment of opioid dependence](#). [Key resources](#) has links to clinical advisory services on substance use referral information for state- and territory-based services.

Some patients use cannabinoids (cannabis products derived from plants or synthetic forms of cannabis) for management of chronic pain, but cannabinoid use is associated with a risk of developing a disorder of substance use. For advice on the use of cannabinoids in chronic pain, see [Cannabinoids](#).

Consider alternative pain management strategies with specialist advice if planning any substance withdrawal for patients with chronic pain.

Other populations to consider in substance use and addictive behaviours

Other populations to consider in substance use and addictive behaviours

Some populations with specific diagnoses may be overlooked in screening for substance use or addictive behaviours if clinicians are not aware that prevalence is increased. For example, substance use is more prevalent in people with **attention deficit hyperactivity disorder**, **autism spectrum disorder** and mild to moderate **developmental disability** compared to the general population.

People recently released from prison are very vulnerable to overdose in the first few weeks after release, because of homelessness, poor access to health care and reduced drug tolerance following imprisonment. Help to address this could include prescription of [medication-assisted treatment of opioid dependence](#), or referral to a specialist opioid treatment service. Other social supports that may be relevant include services that address homelessness, domestic violence or gambling. [Services Australia](#) has a list of websites to assist with housing, food, legal help and drug and alcohol services, including the free and anonymous [AskIzzy website](#), which people can use in a crisis to find local help.

Consider the **occupation** of all patients and whether substance use poses a risk, either for themselves or the community. People in whom this may be a concern include commercial vehicle drivers, airline personnel, defence force personnel, healthcare workers, hospitality workers (eg chefs, publicans), mining employees and construction workers. Resources that may be helpful in supporting people in these occupations are listed in [Substance use in occupational groups: additional resources for clinicians](#). See also [Fitness to drive and disorders of substance use](#) and [Occupational implications of disorders of substance use](#).

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[\[X\] Close](#)

Harms of tobacco smoking and nicotine dependence and benefits of intervention

Harms of tobacco smoking and nicotine dependence and benefits of intervention

Tobacco smoking, driven by [nicotine dependence](#), is the leading preventable cause of death in Australia, associated with almost 20 500 deaths per year. It is a major risk factor for stroke, coronary heart disease, peripheral vascular disease, chronic obstructive pulmonary disease, cancer, and many other illnesses in people who smoke and those exposed to others' smoking. The risks of most harms from tobacco smoking correlate with cumulative exposure. Other forms of tobacco use (chewing, use of tobacco plugs and inhaling 'heat-not-burn' tobacco) are also harmful and are managed in the same way as tobacco smoking. Although nicotine vaping products cause [harms](#), they are often used as [second-line therapy](#) when other measures are not successful or suitable in the management of nicotine dependence.

The prevalence of daily smoking in Australia has declined from 24% in 1991 to 11% in 2019, largely because of public health measures, such as plain packaging and price increases. However, the prevalence of daily tobacco smoking remains much greater in some groups, including people:

- with harmful or dependent use of other substances (66%)
- with serious mental illness, such as schizophrenia and related conditions (62%)
- of Aboriginal and Torres Strait Islander origin (38%),
- who are unemployed (24%)
- who live in regional areas (19%) and remote areas (23%).

Considerations for some specific populations in the management of tobacco smoking are discussed in [Overview of substance use and addictive behaviours](#). Advice specific to [management of tobacco smoking during pregnancy](#) and [management of tobacco smoking during breastfeeding](#) is included in this topic.

Like other forms of [substance dependence](#), nicotine dependence is a chronic relapsing illness; more than half of all people who smoke try to change their smoking behaviour every year. Without treatment, most individuals will not be able to make long-term changes; however, with treatment, even highly nicotine-dependent people can change their smoking behaviour. Making changes has short- and long-term benefits to mental, physical and financial health, as outlined in [Table 22.6](#). Managing tobacco smoking and nicotine dependence is important in people with other disorders of substance use, especially the use of [cannabis](#), [alcohol](#) and [stimulants](#).

Table 22.6 Health benefits associated with stopping smoking

[Printable table](#)

Time since stopping smoking	Health benefits
within 1 day	<ul style="list-style-type: none"> • level of carbon monoxide in the blood will drop and more oxygen will reach the heart and muscles
within 1 week	<ul style="list-style-type: none"> • the lung's natural cleaning system will start to recover and become better at removing mucus, tar and dust from the lungs • there will be higher blood levels of protective antioxidants (eg vitamin C)
within 1 month	<ul style="list-style-type: none"> • the body will be better at healing cuts and wounds

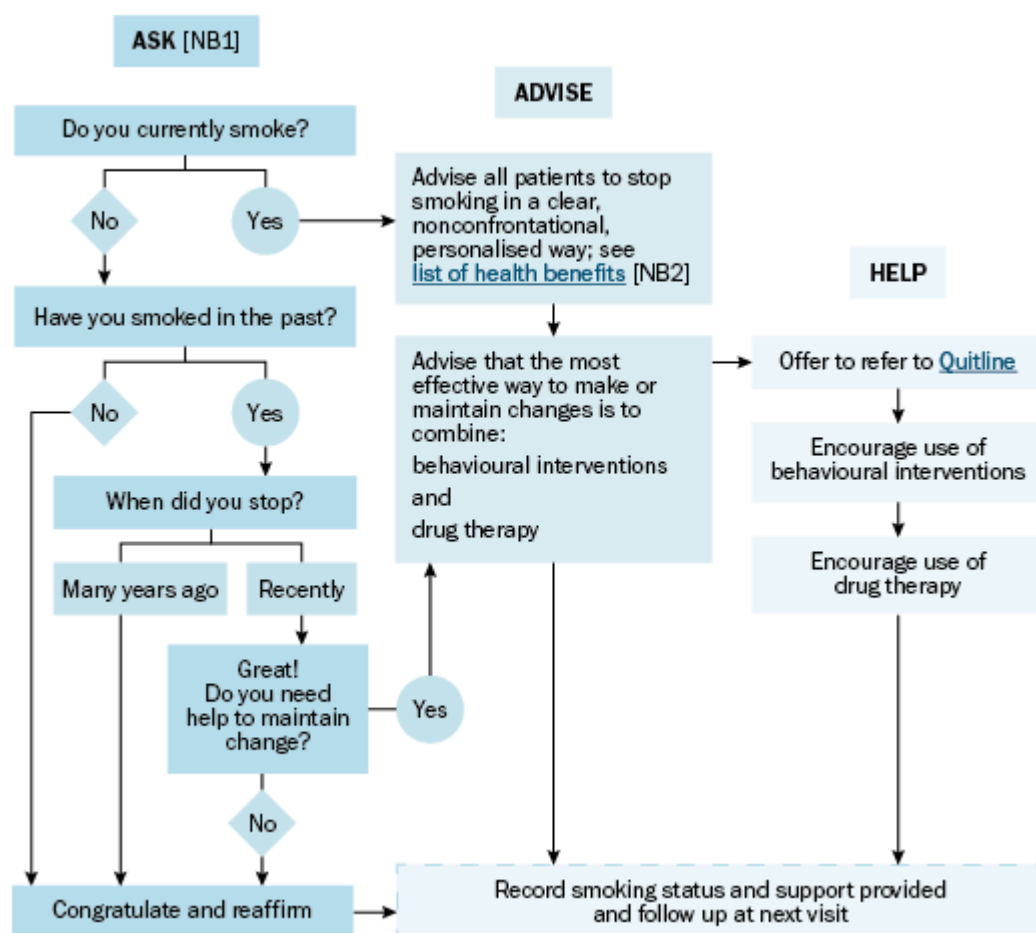
Time since stopping smoking	Health benefits
within 2 months	<ul style="list-style-type: none"> • there will be less coughing and wheezing • the immune system will have started to recover • blood will be less thick and sticky and blood flow to the hands and feet will improve
within 6 months	<ul style="list-style-type: none"> • lungs will no longer produce the extra phlegm caused by smoking
after 1 year	<ul style="list-style-type: none"> • lungs will be healthier and breathing will be easier, making it easier to exercise
within 2 to 5 years	<ul style="list-style-type: none"> • there will be a large drop in risk of heart attack and stroke; this risk will continue to gradually decrease
within 5 years	<ul style="list-style-type: none"> • risk of cervical cancer will return to that of a person who has never smoked
after 10 years	<ul style="list-style-type: none"> • risk of lung cancer will be markedly lower than that of a person who continues to smoke; this risk will continue to decline (provided the disease is not already present)
after 15 years	<ul style="list-style-type: none"> • risk of heart attack and stroke will be close to that of a person who has never smoked

Overview of management of tobacco smoking and nicotine dependence

Overview of management of tobacco smoking and nicotine dependence

The guiding principles of managing tobacco smoking are **ask, advise, help**, as outlined by the Royal Australian College of General Practitioners (RACGP) publication [Supporting smoking cessation: A guide for health professionals](#). This approach is summarised in [Figure 22.3](#).

Figure 22.3 Approach to screening, assessment and interventions for tobacco smoking



NB1: For more detail, see [Screening and assessment of tobacco smoking and nicotine dependence](#).

NB2: Although the goal of intervention is stopping smoking, for some people, an interim target of cutting down is more achievable, can offer benefit (eg saving money) and can act as a step towards stopping.

Adapted with permission from: Quit. Helping patients to stop smoking: a guide for general practitioners [factsheet]. Version 3.0 (November 2022). <https://www.quitcentre.org.au/clinical-tools>. Source © 2022 Cancer Council Victoria.

[Overview of substance use and addictive behaviours](#) explains key [principles of care](#) for a person with a disorder of substance use. Establishing a therapeutic relationship that engages the person (and ideally those close to them) is central to the management of substance use.

The most important element of treatment for substance use is a therapeutic relationship.

Specialist advice on any aspects of care for patients with any disorder of substance use is available and contact is encouraged; see [Contact details for substance use clinical advisory services for clinicians](#).

Specialist advice is available by phone on any aspect of the management of substance use; contact is encouraged.

No amount of tobacco use is considered safe, but making changes to smoking is an achievable step towards stopping. Many people feel demoralised by previous unsuccessful efforts to quit but may be engaged by offers of help to change or control their smoking. Offer patients active assistance and encouragement, whether they are aiming to reduce or stop their smoking or use of nicotine or tobacco. Acknowledging the small wins along the way (the cigarettes not smoked and the money saved) maintains motivation.

Although most literature on managing tobacco use is derived from studies of people who smoke, it is reasonable to apply this to those who use tobacco in other ways because the shared factor perpetuating use is

nicotine dependence.

The most effective approach for managing smoking and nicotine dependence is the combination of [behavioural interventions](#) and [drug therapy](#). Anyone who uses tobacco or nicotine can benefit from interventions, but highly dependent people are most likely to need drug interventions to make lasting change. The most effective management of tobacco smoking and nicotine dependence is drug therapy combined with behavioural strategies.

Advise the patient that it is not necessary to set a quit date, although some people prefer to do so. Each patient can decide their rate of reduction, depending on how well their urges to smoke are controlled by the interventions, and their preferences and circumstances.

For patients who have completed the initial course of drug therapy, offer a further course if they wish to continue the drug, as this increases the likelihood of reducing or stopping use and may prevent relapse. Alternatively, offer a different strategy to promote further attempts at reducing or stopping.

Tobacco smoking affects the metabolism of caffeine [\[Note 1\]](#) and many drugs, including antipsychotics (which are most affected), antidepressants, and some antithrombotic drugs (eg warfarin). These pharmacokinetic changes are caused by chemicals in tobacco other than nicotine. Pharmacodynamic drug interactions (interactions that affect the mechanism of a drug's action) (eg with benzodiazepines) are mainly caused by nicotine. When a person is reducing or stopping smoking, some medications may require dose adjustment (generally a reduction); for management advice, see Quit Centre's [Clinical tools and guidelines](#).

Specific considerations are relevant for the management of smoking in people who are [pregnant](#) or [breastfeeding](#). For other specific groups, see [Considerations for specific populations in substance use and addictive behaviour](#).

Note 1: When a person stops smoking, caffeine metabolism decreases causing its plasma concentration to remain elevated for longer; consider the possibility of [caffeine toxicity](#) if a person has high caffeine intake.

Screening and assessment of tobacco smoking and nicotine dependence

Screening and assessment of tobacco smoking and nicotine dependence

Ask all new patients routinely and others (adolescents and adults) opportunistically and periodically about tobacco smoking or vaping as part of a general screen for disorders of substance use and gambling; these disorders are common (and often co-exist) and people are reluctant to disclose them, often due to fear of stigma. [Screening and assessment of substance use and addictive behaviours](#) outlines history-taking (including use of the [ASSIST-Lite tool](#)), examination, and investigations that should be considered in a broad review of substance use and addictive behaviour.

Ask if a patient smokes cigarettes (including roll-your-own cigarettes with [cannabis](#)), uses 'heat-not burn' tobacco products, chews tobacco or uses [nicotine vaping products](#). Document the number of cigarettes smoked daily as a baseline; this is useful to increase motivation by tracking the savings a patient makes as they reduce or stop smoking.

The most useful questions to identify **high nicotine dependence** include:

- How soon after waking is your first cigarette (or other nicotine intake)? (Smoking within 30 minutes of waking is the best indicator of high dependence)
- Have you experienced [cravings or other withdrawal symptoms](#) for cigarettes while using drug therapy to manage previous efforts to cut down or stop smoking?
- Do you smoke (or use other forms of nicotine) as much as usual when you are unwell?

Figure 22.4 Symptoms of nicotine withdrawal

Withdrawal can be recognised by 2 or more of the following symptoms starting within 24 hours of nicotine reduction or stopping; some features can last 3 to 4 weeks [NB1]:

- cravings for nicotine [NB2]
- anxiety
- irritability or restlessness
- reduced concentration
- malaise
- increased cough
- dysphoria
- mouth ulceration
- insomnia
- increased appetite.

NB1: Withdrawal symptoms occurring during previous drug therapy for tobacco smoking are markers of high nicotine dependence.

NB2: Cravings for nicotine can persist for months after the withdrawal period.

Smoking within 30 minutes of waking is the strongest predictor of high nicotine dependence, regardless of total cigarettes smoked in the day.

Smoking within 30 minutes of waking is the most important marker of high nicotine dependence.

Awareness of the 'time to first cigarette' after waking is important to avoid undertreating a patient with high nicotine dependence who smokes comparatively few cigarettes [Note 2]. This is a shift from previous advice that placed more emphasis on the number of cigarettes smoked per day; the number of cigarettes smoked per day does not correlate well with abstinence at 6 months or biomarkers of nicotine ingestion (eg the serum concentration of cotinine, a metabolite of nicotine). A person who smokes 10 cigarettes per day and starts within 30 minutes after waking may have a higher serum cotinine concentration than a person who smokes 20 cigarettes per day but starts later than 30 minutes after waking. Duration and cumulative amount of smoking remain important to assess the risk of harms (eg cardiovascular disease and lung cancer) from cumulative tobacco exposure.

Note 2: A person can inhale a large amount of nicotine from a few cigarettes by adjusting their inhaling technique.

Behavioural interventions for tobacco smoking and nicotine dependence

Behavioural interventions for tobacco smoking and nicotine dependence

Behavioural interventions are important in every management plan for tobacco smoking and nicotine dependence. They can be accessed by referral to a service such as [Quitline](#), which offers individually tailored counselling; approaches include cognitive behavioural therapy, acceptance and commitment therapy and motivational interviewing. Financial cost is the most common motivation for reducing or stopping smoking, but effects on physical health, social stigma, medication efficacy or mental health may also be important. Referral to a service is more likely to engage a patient with that service than simply providing the person with their contact details.

See [Psychosocial supports in disorders of substance use and additive behaviours](#) for a brief summary of therapies used in behavioural interventions. [Figure 22.5](#) lists additional steps that clinicians can take to support behavioural change in tobacco smoking and nicotine dependence.

Figure 22.5 Ways to support behavioural change in tobacco smoking and nicotine dependence

Encourage the patient by reinforcing that change is possible and support is available; resources include:

- Quitline counselling — available nationally on 137 848
- [MyQuitBuddy](#) app — provides help in setting goals, tracking progress and accessing support.

Identify cues that trigger a craving, for example having an alcoholic drink.

Discuss the nature of cravings as time-limited intrusive thoughts and feelings, and explain how to ‘surf the waves’ of cravings; a guided activity is available on the [Insight website](#).

Suggest the use of strong peppermints or other sweets for managing cravings and keeping the mouth busy.

Reassure the patient that any weight gain that may accompany smoking reduction is still associated with reduced risk of death; the weight gain (compared to people who have never smoked) is usually less than 3 kg.

Recommend brief periods of exercise of any form.

Suggest progressive muscle relaxation, deep controlled (box) breathing or any other relaxation technique.

Suggest that patients break down the financial cost of smoking and see where money saved could be used instead. Visual aids can be useful, such as keeping a separate account or jar of money or tracking savings on an app.

Recommend that patients revisit motivations for change and reflect on the impact of changes made (eg money saved, control over decision-making gained).

Acknowledge that nicotine dependence is a [chronic relapsing illness](#).

Overview of drug therapy for tobacco smoking and nicotine dependence

Overview of drug therapy for tobacco smoking and nicotine dependence

Most people require drug therapy to make lasting changes to tobacco use, but those who have ‘low nicotine dependence’ may prefer to try behavioural intervention alone before considering adding drug therapy. People are considered to have low dependence if they use tobacco more than 60 minutes after waking, particularly if they do so only in cue-driven situations (eg when out with friends in a bar).

Options for drug therapy in managing tobacco smoking and nicotine dependence are outlined in [Figure 22.6](#). First-line options are:

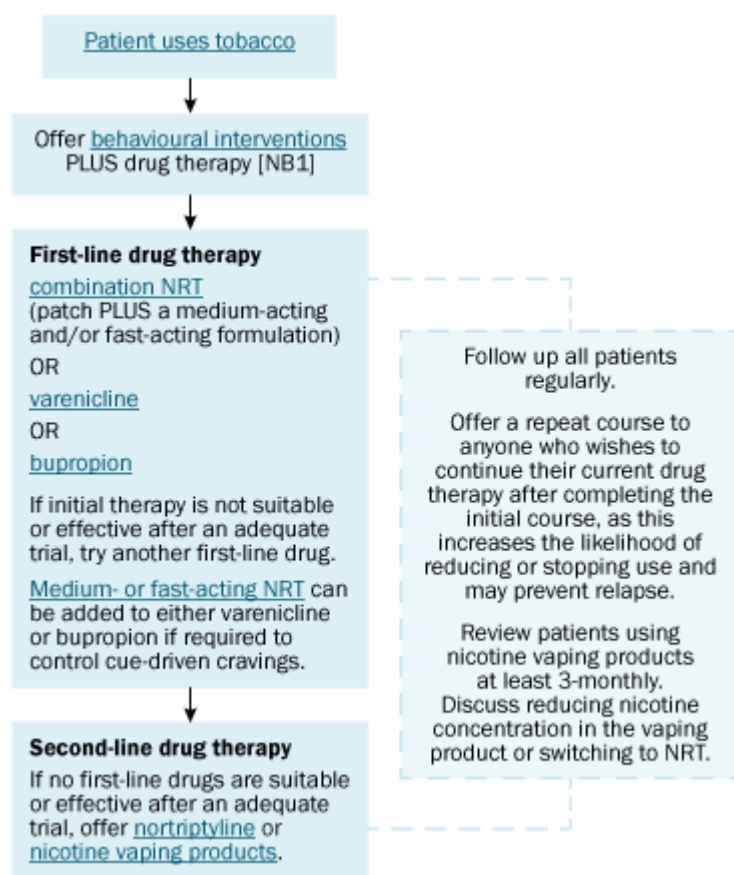
- [combination nicotine replacement therapy \(NRT\)](#) (use of a slow-acting NRT [nicotine patch] with a medium-acting form [eg gum, lozenge or inhalator] and/or a fast-acting form [nicotine mist spray])
- [varenicline](#)
- [bupropion](#).

[Medium- or fast-acting NRT](#) can be safely added to [varenicline](#) or [bupropion](#) if required to control cue-driven cravings. Data is lacking on combining bupropion with varenicline.

For a discussion of how to choose a therapy, see [Factors affecting choice of drug therapy in tobacco smoking and nicotine dependence](#).

If one first-line drug therapy is not suitable or effective after an adequate trial, consider another first-line drug before considering [nortriptyline](#) or [nicotine vaping products](#).

Figure 22.6 Overview of drug therapy to manage tobacco use and nicotine dependence



NRT = nicotine replacement therapy

NB1: People who smoke later than 60 minutes after waking are regarded as having low nicotine dependence; they may choose to try behavioural interventions alone first, especially if they smoke mainly in specific cue-driven situations (eg when out with friends in a bar). However, drug therapy can be offered if they want to start it together with behavioural interventions, or if behavioural interventions alone are not effective.

Factors affecting choice of drug therapy for tobacco smoking and nicotine dependence

Factors affecting choice of drug therapy for tobacco smoking and nicotine dependence

The **most effective** drug therapy is the nicotine receptor agonist varenicline, closely followed by combination nicotine replacement therapy (combination NRT). Each of these is more effective than NRT monotherapy or bupropion.

The **most widely available** and **best tolerated** form of drug therapy is NRT. It is less likely to cause adverse effects and drug interactions than varenicline and bupropion. Medium- or fast-acting NRT can be added to treatment with varenicline or bupropion if required to control cue-driven cravings.

Bupropion is an option when combination NRT and varenicline are not suitable, though it is less effective. Bupropion is contraindicated in patients with a history of seizures, eating disorders and those taking irreversible monoamine oxidase inhibitors.

Nortriptyline is an option when first-line drug therapies (NRT, varenicline and bupropion) are not suitable or were not effective after an adequate trial. Nortriptyline is a second-line choice, mainly due to adverse effects and drug interactions. Nortriptyline has similar efficacy to bupropion but is not approved by the Therapeutic Goods Administration (TGA) for the management of tobacco smoking.

Nicotine vaping products are an alternative for use when first-line drug therapies (NRT, varenicline and bupropion) are not suitable or were not effective after an adequate trial. However, evidence for efficacy is