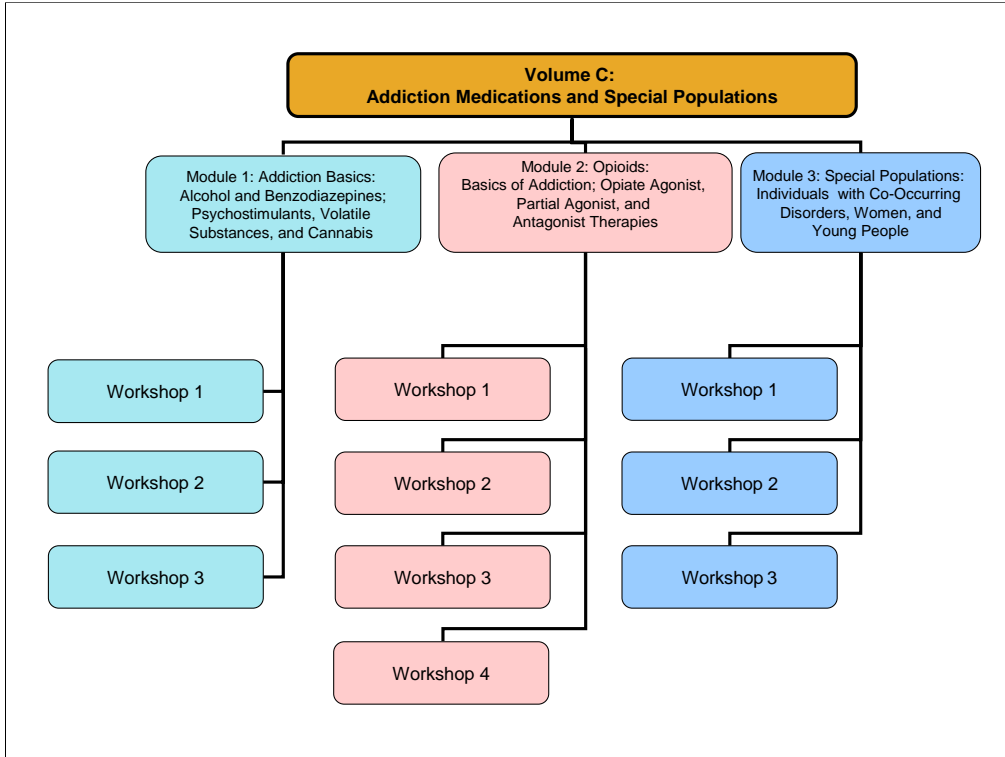


# Volume C: Addiction Medications and Special Populations



Treatnet Training Volume C: Module 1 – Updated 19 February 2008



Module 1: Addiction Basics:  
Alcohol and Benzodiazepines; Psychostimulants;  
Volatile Substances and Cannabis



## Module 1: Training goals



1. Increase knowledge of the medical and addiction-related problems associated with alcohol, benzodiazepines, psychostimulants, volatile substances and cannabis.
2. Learn the appropriate medical detoxification and post detoxification pharmacotherapies appropriate to treat these substance use disorders.
3. Promote the use of these techniques by practionners and organizations

## Module 1: Workshops

**Workshop 1:** Addiction Basics

**Workshop 2:** Alcohol & Benzodiazepines;  
Medical Issues, Detoxification  
Approaches

**Workshop 3:** Psychostimulants, Volatile  
Substances and Cannabis

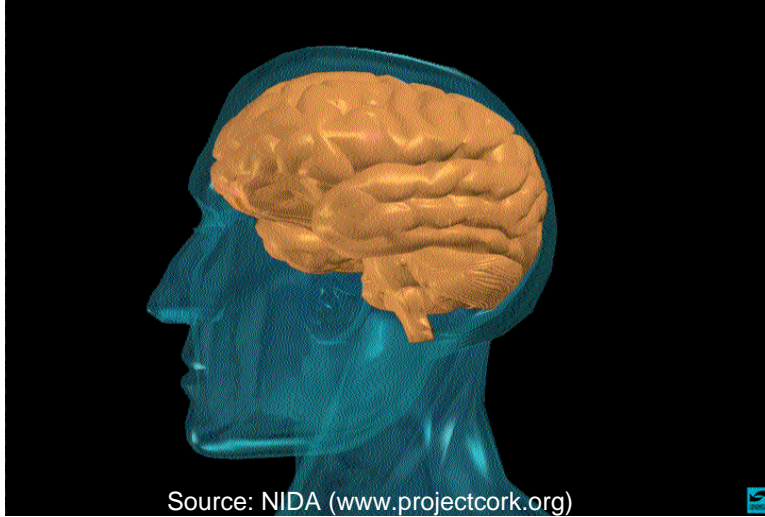
## Icebreaker: Drugs in my country

- What is the main consumed drug in your country?
- What are the main problems that this drug is creating among people in your country?



15 minutes

# Workshop 1: Drug Abuse and Addiction



Source: NIDA ([www.projectcork.org](http://www.projectcork.org))



## Pre-assessment

Please respond to the pre-assessment questions in your workbook.

(Your responses are strictly confidential.)



10 minutes





# Training Objectives



At the end of this training you will be able to:

1. Understand basic principles and concepts of drug abuse and dependence.
2. Understand the basic pharmacology of alcohol, benzodiazepines, psychostimulants, volatile substances and cannabis
3. Understand the specific role of pharmacotherapy for overdose, withdrawal treatments, maintenance treatments and relapse prevention treatments.
4. Understand clinical populations and treatment settings where pharmacotherapies can be used.

# Why do people initiate drug use?

## Key Motivators

- Fun (pleasure)
- Forget (pain amelioration)
- Functional (purposeful)

(NCETA, 2004)

Also initiation starts through:

- Experimental use
- Peer pressure



## Notes

While there are many reasons for the initiation into use of both licit and illicit drugs and continuation of their use, key motivators pivot around three main factors (for fun, to forget, for functional reasons). These motivators are not mutually exclusive. A person may take drugs for any or all of the reasons shown.

It's important to note that a person may not be aware that these are the underpinning drivers of their drug use. For example, a young woman who finally gets into treatment, after being referred by her general practitioner (medical doctor), finds that she has experienced traumatic childhood events (childhood sexual abuse is very common among women in AOD treatment) that have left her affected and that such events are integrally linked to her problematic drug use.

(Australian National Centre for Education on Training and Addiction [NCETA], 2004)

## Understanding young people's motivation to use drugs



1. Risk-takers / pleasure seekers
2. Socially disconnected
3. Self-medicators

### Notes

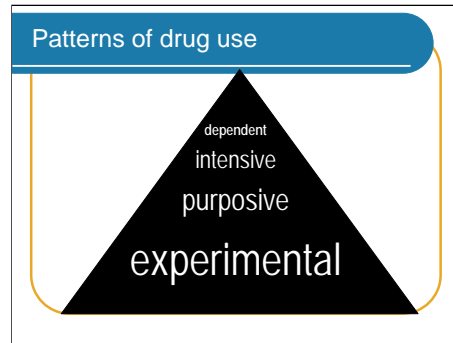
- Different people have different motivations for drug use. A good clinical assessment and good rapport with the patient are required to establish the motivations for use by individual patients.
- Risk-taking is part of the human experience. Developmental psychology tells us that healthy development involves, indeed necessitates, a degree of risk-taking. For some young people, the lure of illicit (and also licit) drug use is seen as “risky” and therefore appealing.

## Types of drug users

Enormous variability and range include:

- Experimenters
- Social users
- Regular heavy users
- Dependent users





## Notes

This figure displays the various drug use patterns. People can move through a spectrum of drug use patterns but do not necessarily progress from one stage to the next. Reasons for changing patterns of use are complex and usually reflect a balance between the benefits and the costs of using.

### Experimental Use/Single or Short Term Use

This category generally describes the “curious” or naïve and those people seeking new experiences. Experimental use is mostly conducted in the presence of friends or family and may occasionally be seen as a “rite of passage” (e.g., the first drink or cigarette). For many, the first drug use experience is not worth repeating.

### Social/Recreational/Occasional Use

Drug use for social or recreational pleasure usually occurs:

- in an environment of relaxation
- during specific social occasions, such as celebrations, special events e.g., drinking with friends following a regular or social activity (e.g., wedding, sporting event), smoking cannabis with friends while listening to music.

It usually occurs with people who:

- are experienced with the drugs they are using
- know what suits them
- are aware of circumstances surrounding use.

Occasional use harms tend to result from user inexperience or lack of tolerance (overdose or intoxication-related harms).

### Purposive Use

Drug use for specific purpose, or under specific circumstances. Usually persons are aware of what they are using and are knowledgeable about drugs, their effects and impact.

### Intensive/Regular Use

Intensive or regular use patterns tend to be associated with a need to achieve relief, or a need to achieve good performance. This pattern involves more frequent and routine use, and the person may use the drug when alone or in social situations where others are not partaking. Harms related to intensive use include physical effects resulting from long-term use or effects on finances, relationships, employment etc.

### Dependent Use

Use is characterised by physical and/or psychological dependence and an inability to cease use without experiencing significant physical or emotional distress. Harms can include those associated with withdrawal, social isolation and physical, social and emotional dysfunction.

Source: adapted from Peard, J., Lintzeris, N., & Churchill, A. 1996, *From Go To Whoa: Amphetamines and Analogues, The Trainer's Package for Health Professionals*, Commonwealth Department of Health and Human Services, AGPS, Canberra, p. 37.

## Factors that influence drug use

There are at least three different categories of factors to consider:

- predisposing factors
- precipitating (enabling) factors
- perpetuating (reinforcing) or maintaining factors

### Notes

For every individual, in each sociocultural context, there is a range of differing predisposing, precipitating, and perpetuating factors that shape and drive drug use.

Predisposing factors might include economic deprivation (and drugs can provide a source of income), or personal trauma (death of a loved one, early childhood sexual abuse). Research has identified many predisposing factors that make some individuals more vulnerable to the uptake of drugs.

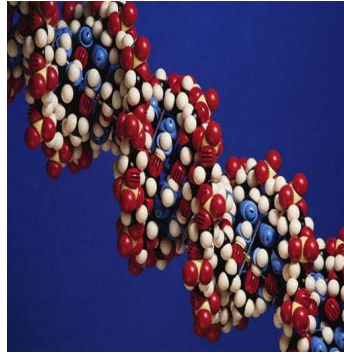
Precipitating factors might include availability and the desire to be accepted by a group of friends who use drugs or drink heavily.

Perpetuating factors or maintaining factors include the reinforcing power of certain drugs (note the very reinforcing qualities of tobacco and alcohol, before even considering heroin). Image and other secondary social reinforcers for behaviour (beyond biological phenomena) are also important to consider. Finally, dependence is a crucial factor in the perpetuation of some drug-using behaviours (again, think about tobacco).

While general medical practitioners may not be in a position to influence any of these factors, having an understanding of their impact on a particular patient and their drug-using career may be helpful in shaping the type of support offered.

## Drugs and genes

- While psychological theories account for a large proportion of the behaviours related to drug use, other factors are also important
- It is increasingly recognised that genes play an important role in an individual's response to drugs and the propensity for the development of dependence



## Environmental factors

- A range of environmental factors impact on drug use, including price and availability of both licit and illicit drugs
- Other environmental factors include prenatal problems, early childhood experiences, family relationship and bonding, and early educational opportunities.
- Cultural norms around drug use also act as powerful determinants of the use of both licit and illicit substances

**Notes:**

Cultural norms can be described as cultural acceptability or cultural sanction of drug use.



## Psychoactive drugs (1)

Psychoactive drugs are generally defined as substances that alter:

- mood
- cognition (thoughts)
- behaviour



### Notes

This addresses drugs that have psychoactive properties, that is, licit and illicit drugs that affect mood, thought, and behaviour, specifically:

- drugs that are used in a recreational or social manner
- drugs that have the potential for creating physical or psychological dependence
- drugs that may result in drug-related harms
- volatile substances, some prescription medications, and alcohol and tobacco.

## Psychoactive drugs (2)

- Affect mental processes and behaviour
- Affect thought processes and actions
- Alter perceptions of reality
- Change level of alertness, response time, and perception of the world
- Achieve effects by interacting with the central nervous system (CNS)

(Source: Carmichael, C. 2001, *The DISE Manual – A Resource for Directions in Illicit Substance Education*, Queensland Alcohol and Drug Research and Education Centre [QADREC], University of Queensland, Brisbane.)

## Psychoactive drug use

- Is a common activity
- Is part of a range of human behaviours
- Can be classified in many ways, including legal status, drug effects
- Alters mood or consciousness, although there are other ways to achieve this:
  - e.g., skydiving, meditation, extreme (and non-extreme) sports, sex. Children, for example, love to alter their consciousness by spinning around.

### Notes

Drugs have been used by almost every society at different points in time. History shows that different cultures have experimented with drugs and also demonstrates the important role of drug use in society, politics, and religion.

All societies allow some form/s of mood alteration. However the most common “mind-altering” activity by far is drug use.

(Source: Weil, A., & Rosen, W. 1993. *From Chocolate to Morphine: Everything You Need to Know About Mind Altering Drugs*. Boston, New York: Houghton Mifflin Company.)

## Views about AOD-related issues

Our thinking about alcohol and other drug (AOD) related issues is informed by factors such as:

- experience
- culture
- education
- religion
- family / environment
- legislation

### Activity Link

Ask participants to select scenarios that are the most or least objectionable to them. Ask them to consider which of the above factors influenced their reactions, attitudes, or perceptions in each case.

## Drug classifications



Psychoactive drugs may be classified according to their:

1. *Status*

- legal
- chemical
- medical
- social

2. *Action and properties*

- depressant
- stimulant
- hallucinogenic
- etc.

### Notes

Psychoactive drugs are categorized according to a number of classification schemes.

## Classifying psychoactive drugs

Depressants	Stimulants	Hallucinogens
Alcohol	Amphetamines	LSD, DMT
Benzodiazepines	Methamphetamine	Mescaline
Opioids	Cocaine	PCP
Solvents	Nicotine	Ketamine
Barbiturates	Khat	Cannabis (high doses)
Cannabis (low doses)	Caffeine	Magic mushrooms
	MDMA	MDMA

### Instructions:

1. Explain the table of classifying psychoactive drugs to your audience.
2. Provide some “street names” of the drugs in your geographic area.
3. Also ask your audience to provide “street names” for the different drugs.

### Notes

There are various drug classifications. These are largely arbitrary groupings, but they provide a useful ready reference tool for approximating relative drug effects, possible harms, and potential withdrawal features. The classification shown in this slide is an example only.

### Limitations of classifications

Classifications are intended as a general guide only, as variations in effects and intensity may occur for drugs within the same class. For example, although ecstasy produces similar effects to amphetamines, it is not as intense, and it may have additional hallucinogenic effects for some people.

For the purposes of this training resource, cannabis has been placed in the central nervous system (CNS) depressant category because of its primary effects as a CNS depressant, and in the hallucinogens category because at high doses cannabis may produce hallucinogenic effects. Opioids have been classified as CNS depressants as a result of their primary effect on the CNS.

### Abbreviations in slide

- DMT: N,N-Dimethyltryptamine
- LSD: Lysergic acid diethylamide
- PCP: phencyclidine

(Source: Victoria Police. 2002. *Custodial Drug Guide: Medical Management of People in Custody with Alcohol and Drug Problems*. Mornington, Victoria, Australia: Custodial Medicine Unit, Victoria Police.

## Drug use and health



### Patients with drug problems:

- often have multiple health and social problems
- expect doctors to ask and provide information about alcohol and drug issues – failure to inquire may lead to medical malpractice in some situations

(Source: Hamilton, M., & Cape, G. 2002, History of Drug Use and Drug Policy Responses. In Hulse, G., White, J., & Cape, G. (eds.), *Management of Alcohol and Drug Problems*. South Melbourne, Australia: Oxford University Press, pp. 3–16.)

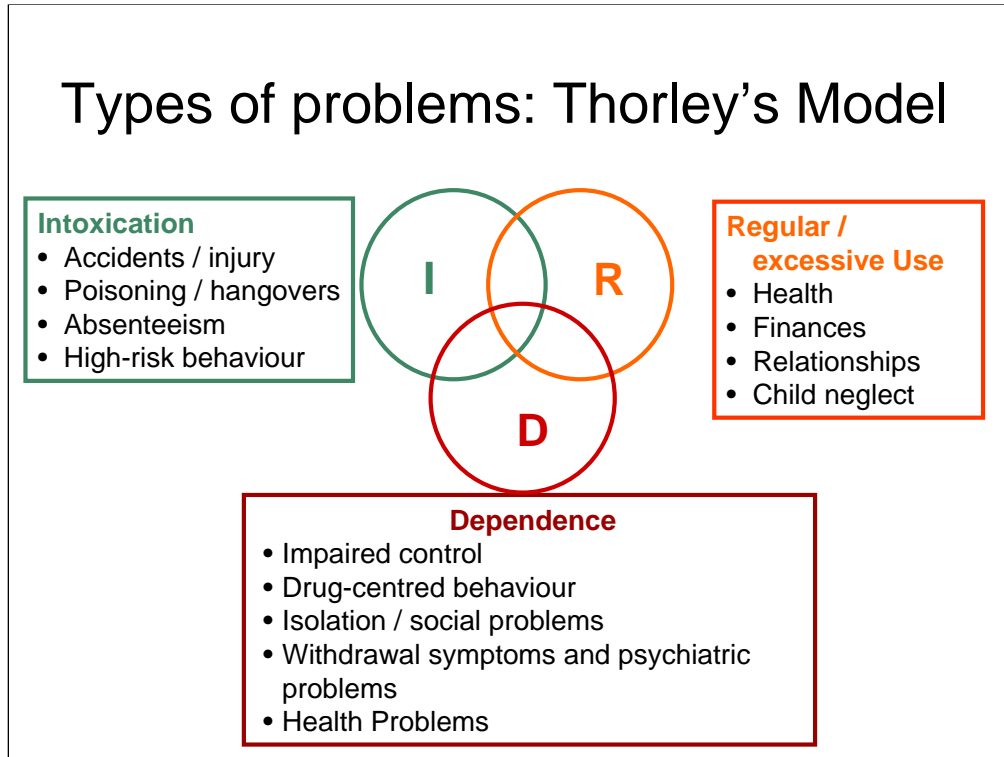
## Types of problems (1)



- Different patterns of drug use result in different types of problems
- Because individuals have different genetic make ups and early experiences, they may respond differently to drugs and have a different risk for drug abuse and dependence
- Drug use may affect all areas of a patient's life, and problems are not restricted to dependent drug use



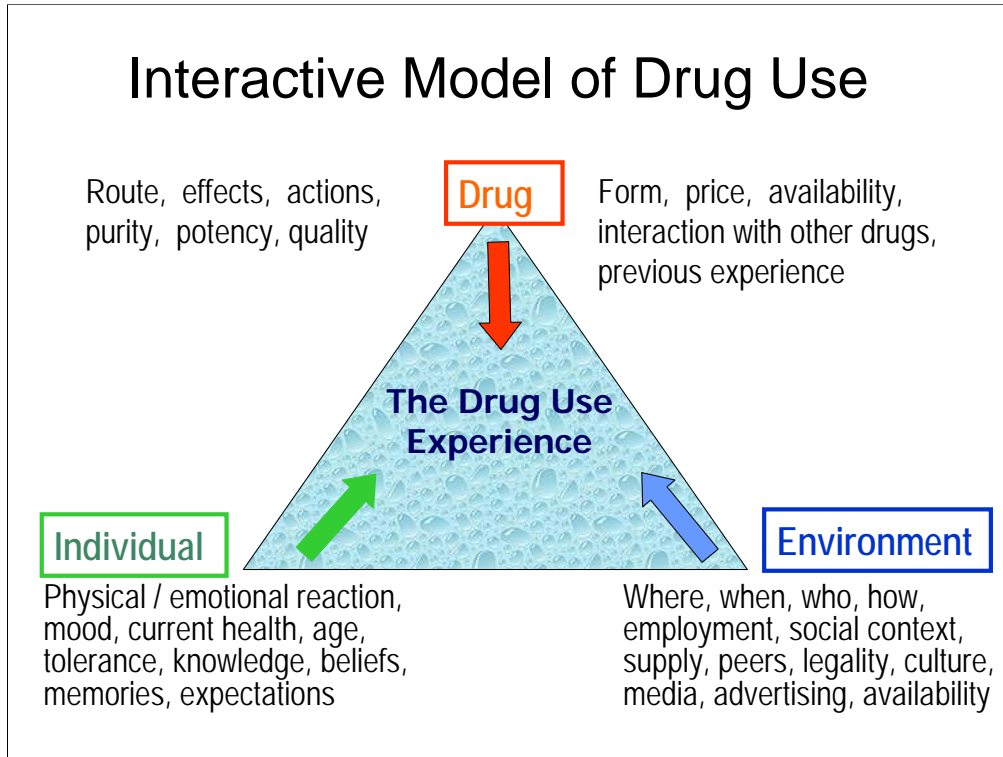
## Types of problems: Thorley's Model



### Notes

It is important for the trainer to be thoroughly acquainted with Thorley's model (shown in this slide). Each of the three domains of use provides a valuable emphasis for problem identification related to most drugs across sub-groups of the population. (Note: nicotine tends not to produce problems of intoxication)

Drug-related harms are commonly perceived to be mostly associated with dependence. However, a significant proportion of harms arise for both individuals and the community as a consequence of the actions of people who are not dependent, or who are only mildly dependent, on drugs. An inappropriate emphasis on **dependence** may result in the proportionally greater number of non-dependent users with problems related to **intoxication** (particularly among young people) and **regular use** going undetected. This model can be readily adapted to all psychoactive drugs. It is a useful aid when working with patients to help both the practitioner and patient understand specific harms related to drug use.



### Notes

#### ***The Interactive Model***

This model presents a *systems* perspective of drug use and provides the overall context for the drug-taking *experience*. It combines Zinberg's interactive model of drug-related harm with principles of Social Learning Theory. The Interactive model suggests that the interaction between the *Drug*, *Environment*, or *Individual* is crucial to the overall drug use experience. Each factor cannot be considered alone.

Asking about each domain during assessment and treatment planning can provide:

- a more accurate picture of the role of AOD use and related problems
- an insight into the personal experiences of patients
- information or cues about possible or relative harms
- a guide to treatment interventions.

The importance or weight given to any one factor within a domain will vary from person to person and situation to situation. Although some factors may overlap into other categories (e.g., drug availability may be an environmental or drug factor), the most important issue is that each domain is considered.

This model can be used for working with individuals, families, or whole communities, and forms the basis of public health approaches to health promotion, prevention, and access to health care.

## Important terminology



1. Harmful use
2. Physical dependence vs. addiction
3. Psychological craving
4. Tolerance
5. Withdrawal symptoms
6. Neurotransmitters and receptors

## What is harmful use? (ICD-10)

A pattern of psychoactive substance use that is damaging to physical and / or mental health.

### Notes

“ICD-10” stands for “International Statistical Classification of Diseases and Related Health Problems, 10<sup>th</sup> Edition.

## What is drug addiction?



Drug addiction is a complex illness characterised by compulsive, and at times, uncontrollable drug craving, seeking, and use that persist even in the face of extremely negative consequences.

(NIDA, 1999)

### Notes

#### ***Addiction***

Addiction is a state in which an organism engages in a compulsive behavior, even when faced with negative consequences. This behavior is reinforcing, or rewarding. A major feature of addiction is the loss of control in limiting intake of the addictive substance. The most recent research indicates that the brain's reward pathway may be even more important in the craving associated with addiction, compared to the reward itself. Scientists have learned a great deal about the biochemical, cellular, and molecular bases of addiction; it is clear that addiction is a disease of the brain.

(NIDA, 1999)

## Characteristics of addiction

- Compulsive behaviour
- Behaviour is reinforcing (rewarding or pleasurable)
- Loss of control in limiting intake



(NIDA; [www.projectcork.org](http://www.projectcork.org))

## Psychological craving

Psychological craving is a strong desire or urge to use drugs. Cravings are most apparent during drug withdrawal.



## Tolerance

A state in which a person no longer responds to a drug as they did before, and a higher dose is required to achieve the same effect.





## Withdrawal (1)

A period during which somebody addicted to a drug or other addictive substance reduces their use or stops taking it, causing the person to experience painful or uncomfortable symptoms

OR

A person takes a similar substance in order to avoid experiencing the effects described above.

## Withdrawal (2)

When a drug is removed, physical and / or mental disturbances may occur, including:

- Physical symptoms
- Emotional problems
- Cognitive and attention deficits
- Aggressive behavior
- Hallucinations
- Convulsions
- Death

## DSM IV criteria for substance dependence

Three or more of the following occurring at any time during the same 12 month period:

- Tolerance
- Withdrawal
- Substance taken in larger amounts over time
- Persistent desire and unsuccessful efforts to cut down or stop
- A lot of time and activities spent trying to get the drug
- Disturbance in social, occupational, or recreational functioning
- Continued use in spite of knowledge of the damage it is doing to the user or others

(DSM-IV-TR, American Psychiatric Association, 2000.)

### Notes

“DSM-IV-TR” stands for “Diagnostic and Statistical Manual of Mental Disorders, 4<sup>th</sup> Edition, Text Revision”

## ICD-10 criteria for dependence

**Dependence: 3 or more of the following:**

- (a) strong desire or sense of compulsion to take the substance;
- (b) difficulties in controlling substance-taking behaviour in terms of its onset, termination, or levels of use;
- (c) a physiological withdrawal state;
- (d) evidence of tolerance;
- (e) progressive neglect of alternative pleasures or interests
- (f) persisting with substance use despite clear evidence of overtly harmful consequences

### Notes

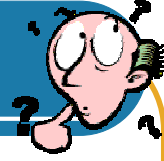
“ICD-10” stands for “International Statistical Classification of Diseases and Related Health Problems, 10<sup>th</sup> Edition.”

### Additional information:

A definite diagnosis of dependence should usually be made only if three or more of the following have been present together at some time during the previous year:

- (a) a strong desire or sense of compulsion to take the substance;
- (b) difficulties in controlling substance-taking behaviour in terms of its onset, termination, or levels of use;
- (c) a physiological withdrawal state when substance use has ceased or been reduced, as evidenced by: the characteristic withdrawal syndrome for the substance; or use of the same (or a closely related) substance with the intention of relieving or avoiding withdrawal symptoms;
- (d) evidence of tolerance, such that increased doses of the psychoactive substances are required in order to achieve effects originally produced by lower doses (clear examples of this are found in alcohol- and opiate-dependent individuals who may take daily doses sufficient to incapacitate or kill nontolerant users);
- (e) progressive neglect of alternative pleasures or interests because of psychoactive substance use, increased amount of time necessary to obtain or take the substance or to recover from its effects;
- (f) persisting with substance use despite clear evidence of overtly harmful

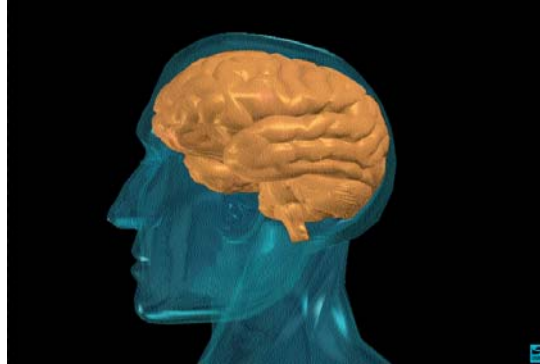
## To avoid confusion



- In this training, “**addiction**” will be the term used to refer to the pattern of continued use of drugs despite pathological behaviours and other negative outcomes
- “**Dependence**” will only be used to refer to physical dependence on the substance as indicated by tolerance and withdrawal as described above

## Addiction = Brain Disease

Addiction is a brain disease that is chronic and relapsing in nature.



(NIDA; [www.projectcork.org](http://www.projectcork.org))

### Instructions

Indicate that you will explain how the brain basically works and how and where drugs such as heroin and cocaine work in the brain.

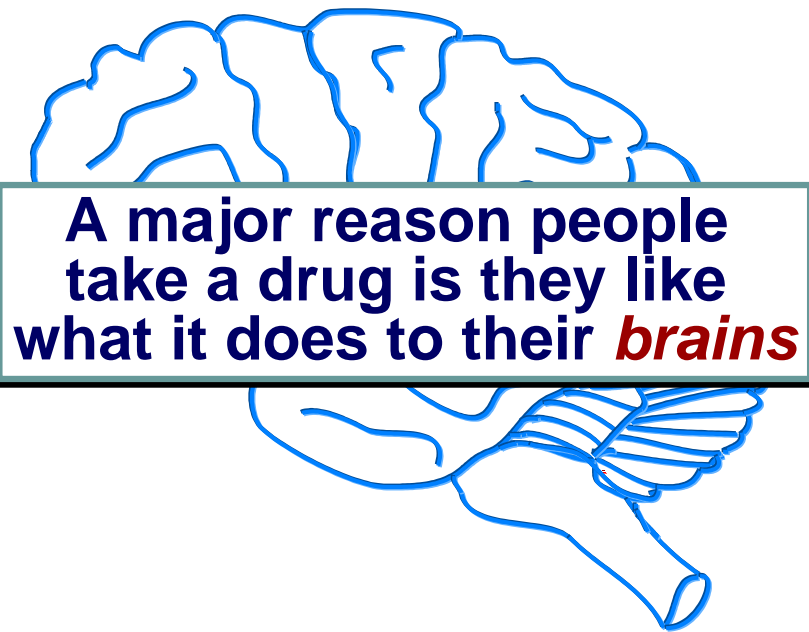
### The brain is the most complex organ in the body.

The brain is made up of a complex network of billions of nerve cells called *neurons*, as well as other kinds of cells, all protected by the bones of the skull. The typical brain weighs only about 3 pounds, but it is the source of most qualities that make you who you are. Neurons in the brain and spinal cord are part of the nervous system and act as a body's "Command Central."

The brain is constantly active, even when we are asleep. As a matter of fact, asleep or awake, the brain requires 20 percent of the heart's output of fresh blood and 20 percent of the blood's oxygen and glucose to keep functioning properly. Glucose is a type of sugar that is our brain's primary fuel.

The brain produces enough electrical energy to power a 40-watt light bulb for 24 hours. That's a lot of energy for a human organ a little bigger than a softball.

Photo courtesy of the NIDA Web site. From *A Slide Teaching Packet: The Brain and the Actions of Cocaine, Opiate, and Marijuana*.



A major reason people take a drug is they like what it does to their *brains*

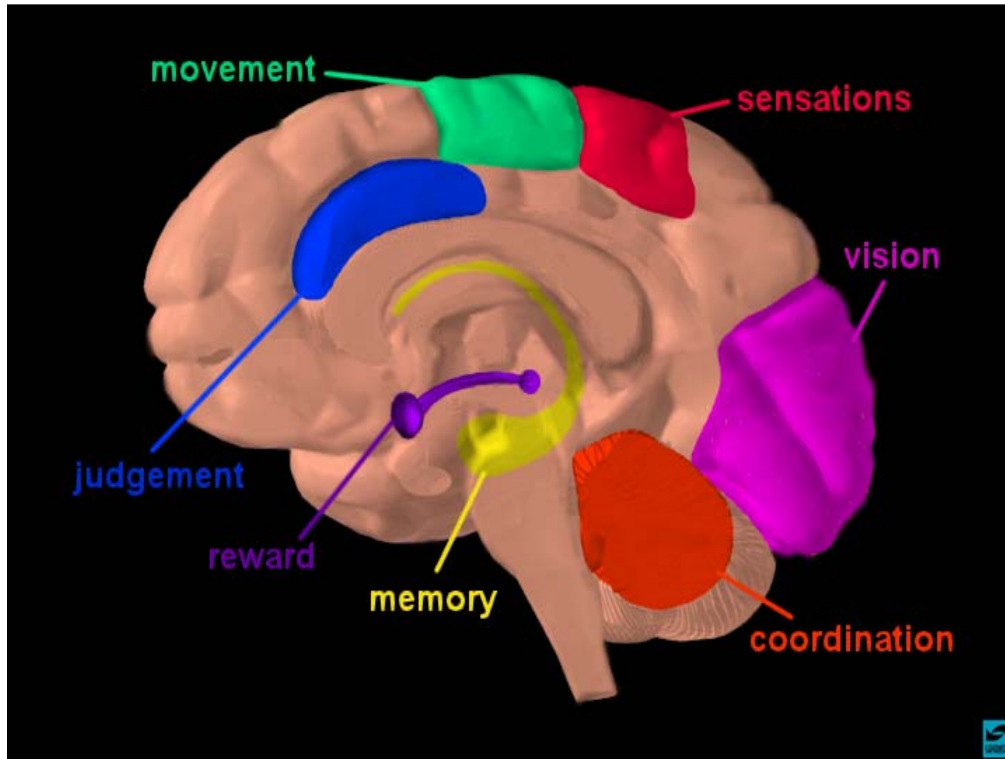
## How the reward system works



### Notes

Natural rewards such as food, water, sex, and nurturing allow people to feel pleasure when eating, drinking, procreating, and being nurtured. Such pleasurable feelings reinforce the behavior so that it will be repeated. Each of these behaviors is required for the survival of the species. There is a pathway in the brain that is responsible for rewarding behaviours. This can be viewed in more detail in the next slide.



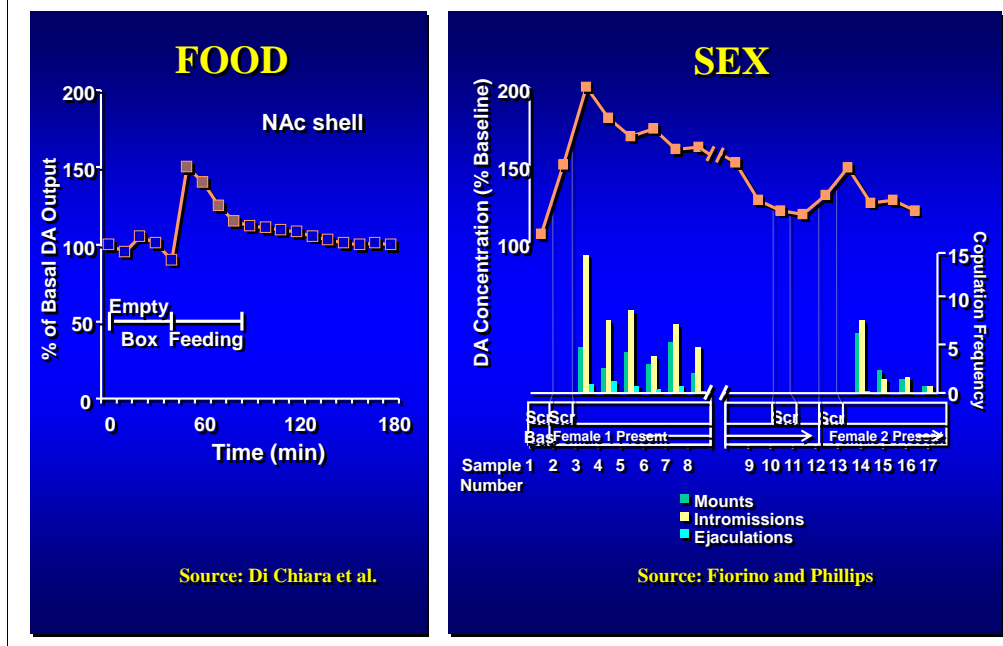


### Instructions

1. Explain that certain parts of the brain govern specific functions.
2. Point to areas such as the sensory (blue), motor (orange), and visual cortex (yellow) areas of the brain to highlight their specific functions.
3. Point to the cerebellum (pink), for coordination, and to the hippocampus (green), for memory.
4. Indicate that nerve cells or neurons connect one area to another via pathways to send and integrate information.
5. Explain that the distances that neurons extend can be short or long.
6. Point to the reward pathway (orange). Explain that this pathway is activated when a person receives positive reinforcement for certain behaviours ("rewards").
7. Indicate that you will explain how this happens when a person takes an addictive drug.
8. As another example, point to the thalamus (magenta). This structure receives information about pain coming from the body (magenta line within the spinal cord), and passes the information up to the cortex.

(Source: NIDA)

## Natural rewards elevate dopamine levels



### Notes

Natural rewards, such as food and sex, cause our brains to release dopamine.

## Activating the system with drugs



(NIDA; [www.projectcork.org](http://www.projectcork.org))

### Instructions:

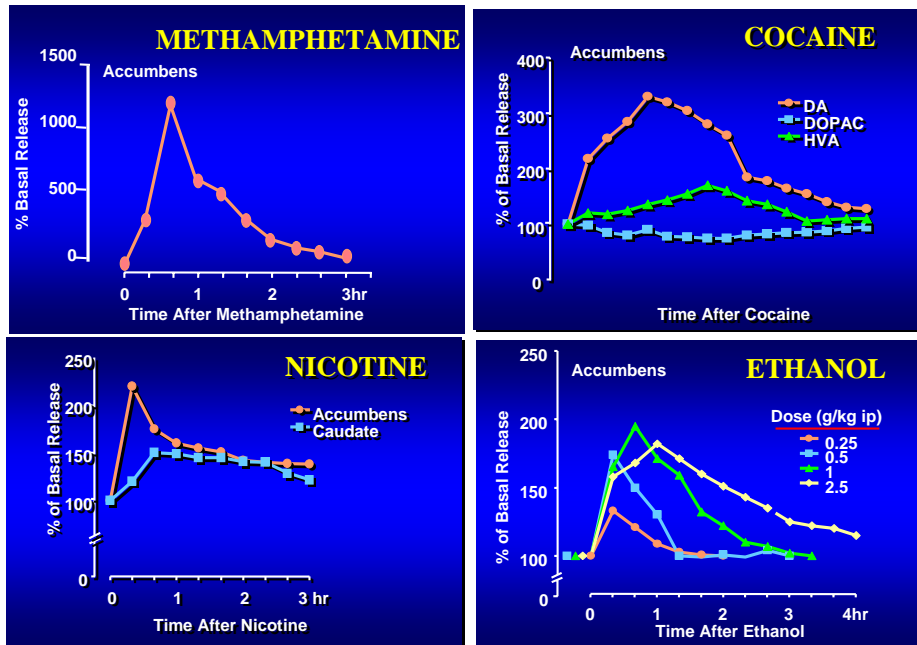
1. Explain that the discovery of the reward pathway was achieved with the help of animals such as rats. Rats were trained to press a lever for a tiny electrical jolt to certain parts of the brain.
2. Point out that when an electrode is placed in the nucleus accumbens, the rat keeps pressing the lever to receive the small electrical stimulus because it feels pleasurable. This rewarding feeling is also called positive reinforcement.
3. Explain that drugs have the same positive reinforcement effect by activating the reward system artificially (not natural rewards).

(Source: NIDA.)

### Additional information:

*Activation of the reward pathway by an electrical stimulus:* The discovery of the reward pathway was achieved with the help of animals such as rats. Rats were trained to press a lever for a tiny electrical jolt to certain parts of the brain. Show that when an electrode is placed in the nucleus accumbens, the rat keeps pressing the lever to receive the small electrical stimulus because it feels pleasurable. This rewarding feeling is also called positive reinforcement. Point to an area of the brain close to the nucleus accumbens. Tell the audience that when the electrode is placed there, the rat will not press the lever for the electrical stimulus because stimulating neurons in a nearby area that does not connect with the nucleus accumbens does not activate the reward pathway. The importance of the neurotransmitter dopamine has been determined in these experiments because scientists can measure an increased release of dopamine in the reward pathway after the rat receives the reward. And, if the dopamine release is

## Effects of Drugs on Dopamine Release



Source: Shoblock and Sullivan; Di Chiara and Imperato

### Instructions:

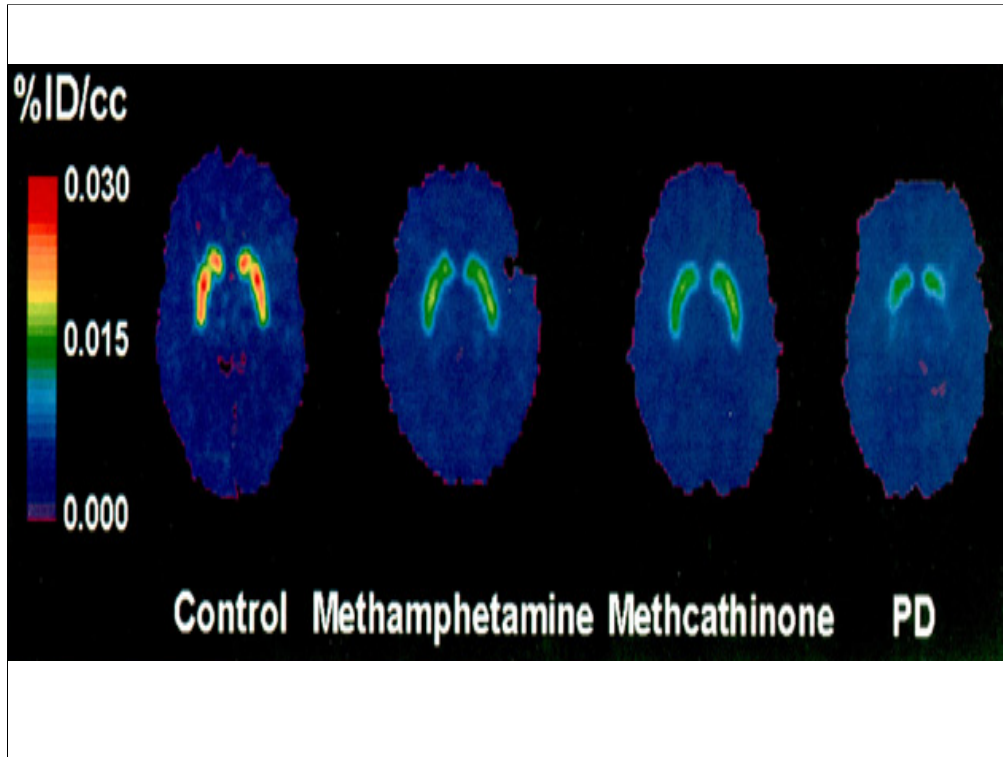
1. Explain that drugs also cause a release of dopamine.
2. Review the curve of dopamine release for each drug (methamphetamine, cocaine, nicotine and ethanol)
3. Note that methamphetamine causes a release of dopamine that is far greater than the release caused by natural rewards (e.g., food and sex) or alcohol, nicotine, or cocaine.

## Why can't people just stop drug use?

When people first try drugs, it is usually a voluntary decision, but after using the drug for a while, it is no longer voluntary.

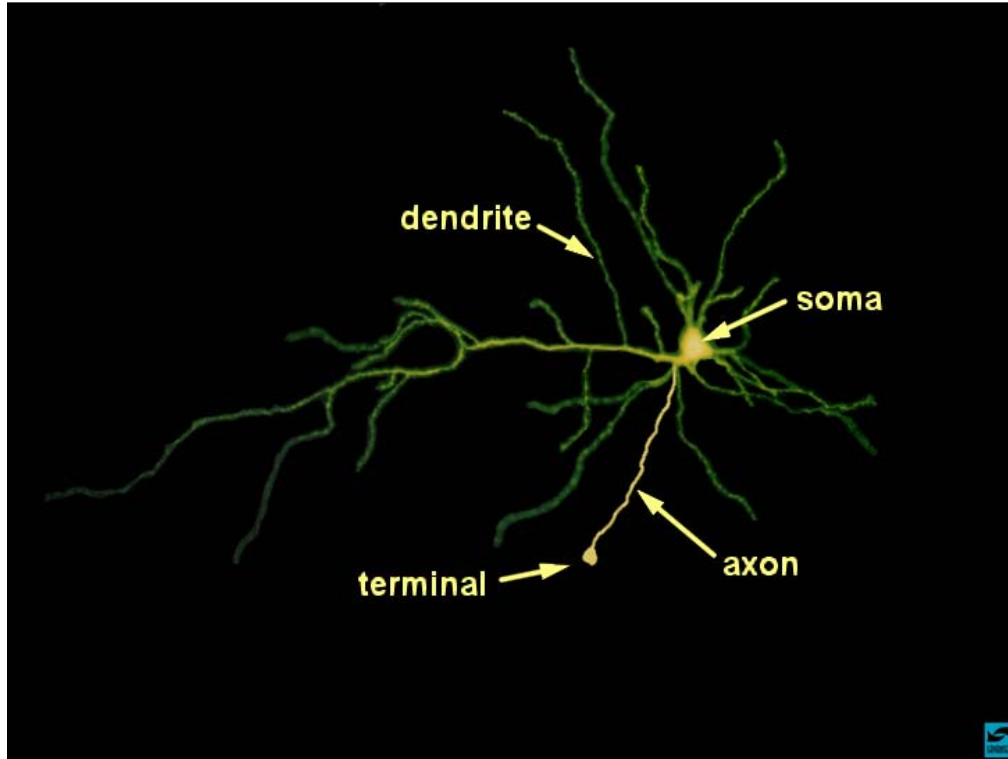
Why can't people stop?





### Notes

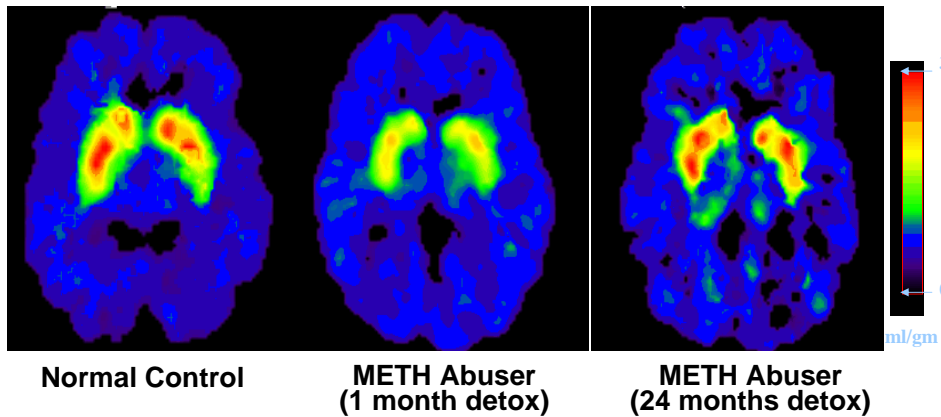
PET scans compare dopamine transporter density in a control subject, a methamphetamine user (1 month abstinent), a methcathinone user, and a patient with Parkinson's Disease. Chronic methamphetamine use has been shown to alter dopamine transporter density.



### Notes

The anatomy of a neuron. Neurons communicate with each other by releasing neurotransmitters, such as dopamine, from the terminals.

## Partial Recovery of Brain Dopamine Transporters in Methamphetamine (METH) Abuser After Protracted Abstinence



(Volkow, N.D., et al. 2001. Journal of Neuroscience 21, 9414-9418.)

### Notes

There is some recovery in dopamine transporter levels in methamphetamine abusers after a 2-year period of abstinence.



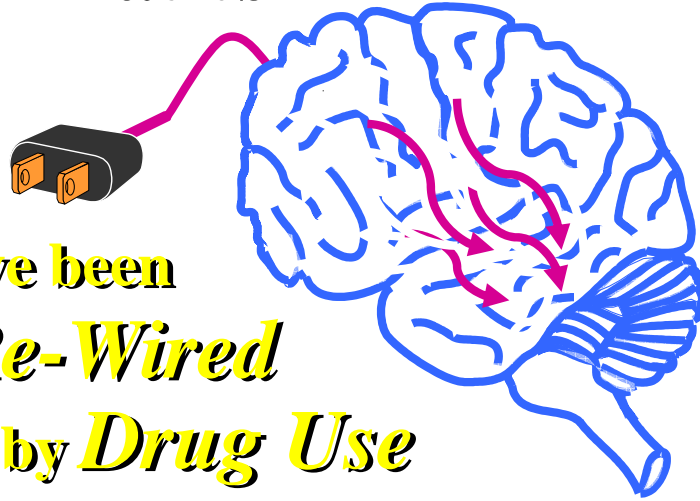
*Because...*

*Their Brains*

*have been*

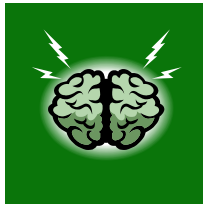
*Re-Wired*

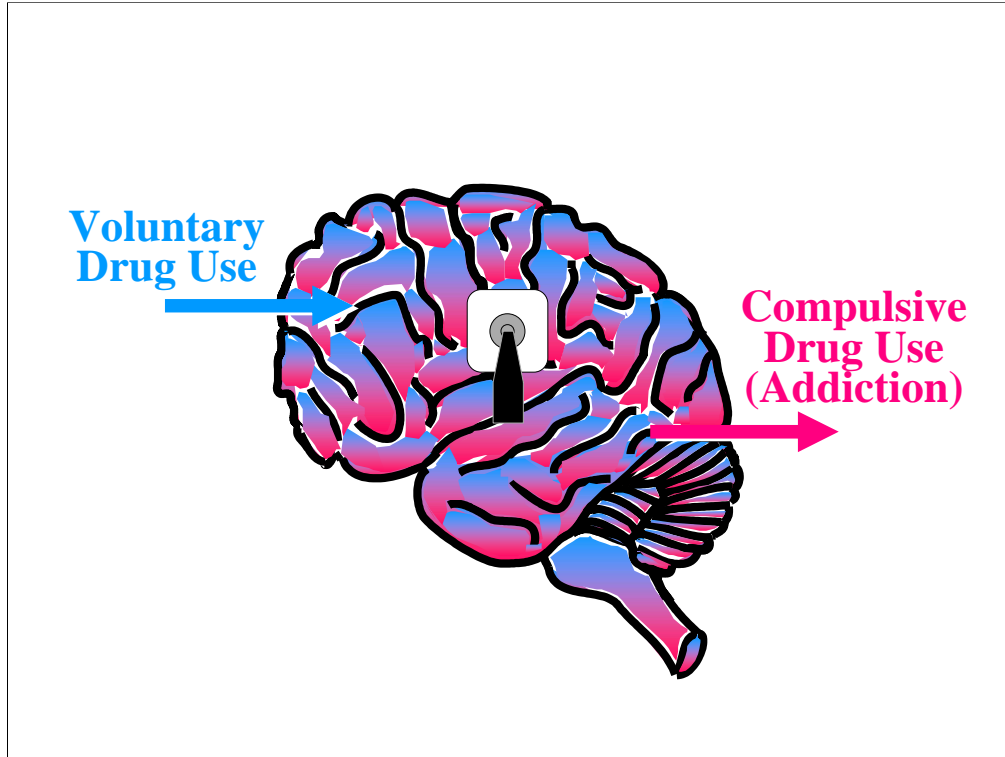
*by Drug Use*



## Why can't people just stop drug use?

Prolonged drug use changes the brain in fundamental and long-lasting ways!





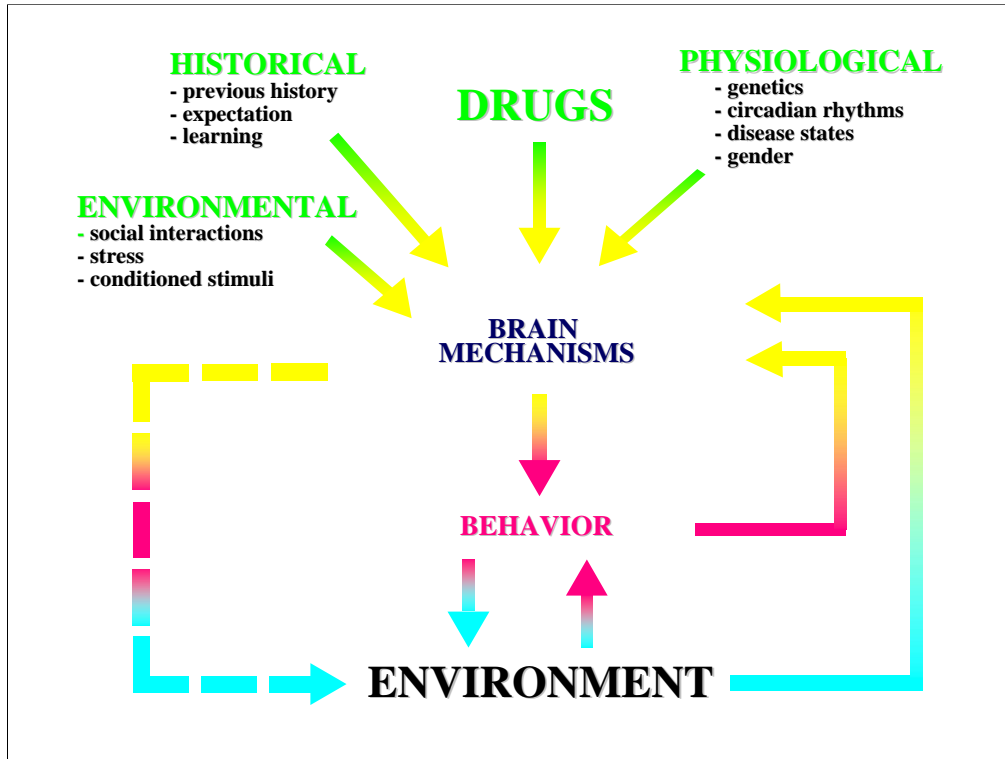
**Notes**

Repeated drug use alters the brain in such a way that the user is transformed from a voluntary user to an addicted individual.

**Addiction is, Fundamentally,  
A Brain Disease**

**...*BUT***

**It's Not Just A Brain Disease**



### Notes

Addiction is not just a brain disease. The historical, environmental, physiological and behavioral components must also be considered and addressed.

Questions?



Comments?

## Post-assessment

Please respond to the post-assessment questions in your workbook.

(Your responses are strictly confidential.)

10 minutes



Thank you for your time!

End of Workshop 1



Volume C, Module 1, Workshop 2:  
Alcohol & Benzodiazepines; Medical Issues,  
Detoxification Approaches, Pharmacotherapies



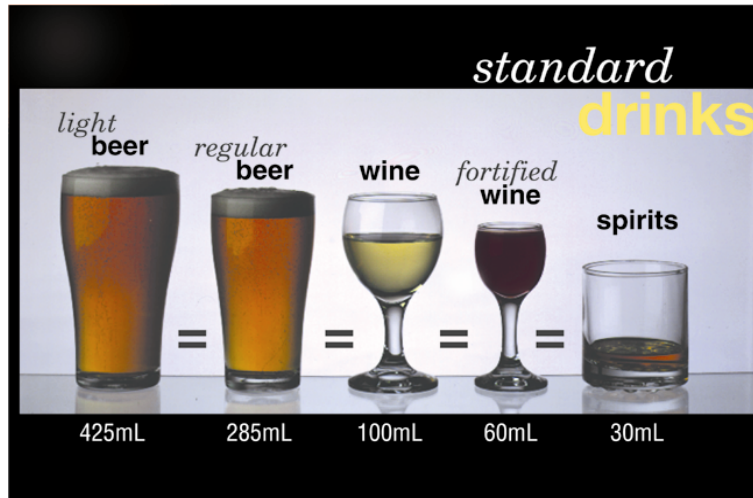
# Training objectives



## At the end of this training you know:

1. Acute and chronic effects of **alcohol and benzodiazepines**, the medical and psychiatric dangers associated with intoxication, overdose, withdrawal, and interactions with other substances
2. Treatment protocols to treat intoxication and overdose
3. Withdrawal approaches and protocols
4. Necessary treatments following detoxification
5. Proper setting and support services needed to properly conduct withdrawal treatments

# Alcohol



## Notes

This slide set contains information for general practitioners (GPs) on:

- assessment of patterns of drinking
- population effects and high-risk groups
- pharmacokinetics
- effects and alcohol-related harm
- types of problems and drug-alcohol interactions
- history and assessment
- screening and assessment tools
- brief intervention and treatment matching.

Please adapt this slide set where needed to meet the learning/information needs of your GP group(s). For instance, you may wish to include cases and question prompts that focus attention on what GPs encounter in their day-to-day practice.

## Acute alcohol-related harms

Physical injury and psychological harms and death arise from:

- |   |   |  |
|---|---|--|
| <ul style="list-style-type: none"><li>• Falls</li><li>• Physical assaults</li><li>• Sexual assaults</li><li>• Domestic violence</li><li>• Traffic accidents</li><li>• Occupational &amp; machinery injuries</li></ul> | <ul style="list-style-type: none"><li>• Fires</li><li>• Drowning</li><li>• Child abuse</li><li>• Unprotected sex leading to STDs and HIV</li><li>• Overdose</li></ul> | <ul style="list-style-type: none"><li>• Comorbidity</li><li>• Dehydration</li><li>• Sleep disturbances</li><li>• Raised blood pressure</li><li>• Shortness of breath</li></ul> |
|---|---|--|

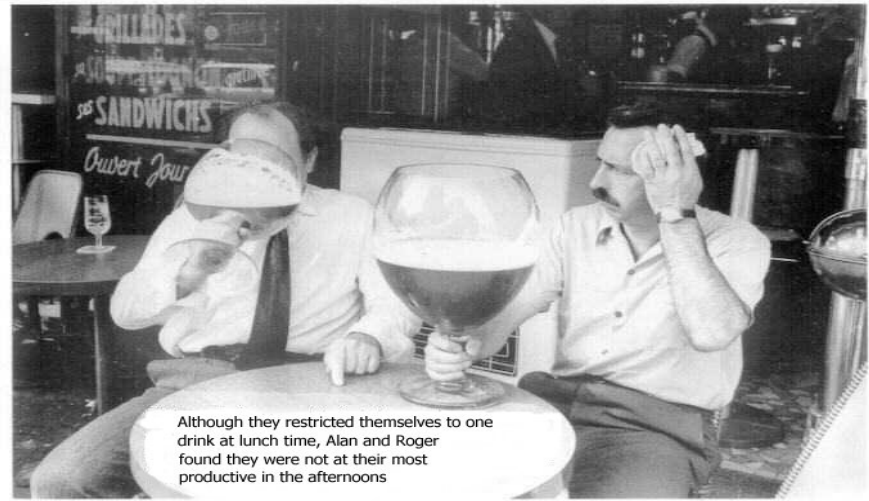
# Alcohol



- Still the most popular “drug”
  - In some societies over 80% of population drinks
- 8% drink daily, peak in males +60 yrs (23%). 40% drink weekly.
- At-risk drinking now defined as:
  - risks of harm in the long term (chronic harm)
  - risks of harm in the short term (acute harm)

Source: AIHW (Australian Institute of Health and Welfare) 2002, *2001 National Drug Strategy Household Survey: First Results*, Drug Statistics Series No. 9, AIHW cat. no. PHE 35. AIHW, Canberra, <http://www.aihw.gov.au> [accessed 5 September 2003].

## A standard drink

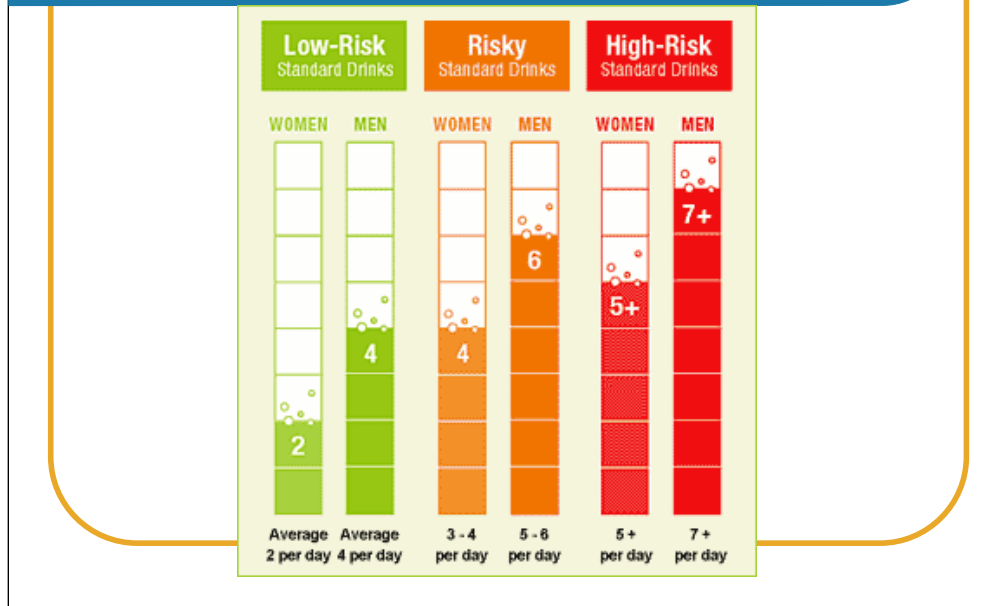


### Notes

Different surveys sometimes use different measures of a “standard drink.” Many patients will not know what a standard drink is, or will find it difficult to calculate their own consumption in these terms. GPs have an important role to play here in helping patients learn about risk levels and the associated impact on health.

Most people significantly under-estimate how much a standard drink is (usually by about 50%-100%). Most survey data about alcohol consumption levels are therefore highly conservative.

## Risky drinking levels (for chronic harm)



### Notes

This slide shows the risk associated with drinking levels for chronic harm. The levels considered to be high risk for acute harm are >7 standard drinks for women and >11 standard drinks for men.

Source: <http://www.therightmix.gov.au/sdg.htm>

## Alcohol-induced memory loss



- Teenagers (28.4%) were most likely to have a memory loss incident following drinking:
  - 4.4% reported “blackouts” occurring on weekly basis
  - 10.9% reported “blackouts” on a monthly basis
- Memory loss occurred after drinking for:
  - 12% male drinkers aged > 40 years
  - 7% female drinkers aged > 40 years
  - 20% - 30% of all other age groups

### Notes

Memory loss is an important assessment indicator for short-term risk of harm among young people.

### Assessment/Screening Link

The AUDIT (Alcohol Use Disorders Identification Test) can assist with alcohol assessment and screening to identify problems such as memory loss after a session of alcohol consumption and patterns of drinking that place people at high risk of short-term harm (“binge-patterns”).

Source: AIHW (Australian Institute of Health and Welfare) 2002, 2001 *National Drug Strategy Household Survey: Detailed Findings*, Drug Statistics Series No. 11, AIHW cat. no. PHE 41, AIHW, Canberra.



## Predisposing factors for high-risk drinking

- Family history of alcohol problems
- Childhood problem behaviours related to impulse control
- Poor coping responses in the face of stressful life events
- Depression, divorce, or separation
- Drinking partner
- Working in a male-dominated environment

### Notes

There is little evidence to support the notion of an addictive personality. However, certain factors predispose people to increased risk of high regular alcohol intake. Some of these predisposing factors are similar to those for developing a mental illness. They include a variety of environmental factors and genetic contributions in a proportion of individuals (although estimates of the importance of genetic contributions to AOD-related problems vary considerably).

## Concurrent mental health problems

Alcohol may:

- exacerbate existing mental health problems
- interact with prescribed medications
- reduce or exacerbate the effect of certain medications
- reduce patient compliance with treatment regimens

### Notes

Alcohol use in comorbid patients is high. It is important to be aware of the effects alcohol use may have on patients with concurrent mental health problems. Any drug use complicates both the pathology of the condition and its treatment.

Sources: Holmwood C. 2002, *Comorbidity of Mental Disorders and Substance Use: A Brief Guide for the Primary Care Clinician*, Primary Mental Health Care Australian Resource Centre (PARC), Department of General Practice, Flinders University, Adelaide.

McDermott, F., & Pyett, P. 1993, *Not Welcome Anywhere, People who have Both a Serious Psychiatric Disorder and Problematic Drug or Alcohol Use*, vol. 2, Victorian Community Managed Mental Health Services Inc., Melbourne.

## Women and alcohol



Women are more susceptible to the effects of alcohol due to:

- smaller physical size
- decreased blood volume
- lower body water to fat ratio
- reduced ADH activity in gastric mucosa (hence reduced stomach metabolism of alcohol).

Resulting in:

- earlier development of organ damage
- increased risk of intoxication related harms; e.g., assault, injury.

### Notes

- Physiological differences in women (relative to males) result in higher blood alcohol concentrations and increased likelihood of organ damage, e.g., cirrhosis. Gastric ADH levels in females are 50% of those in males.
- Drinking increases the risk for breast cancer.

## Fetal Alcohol Syndrome (FAS)

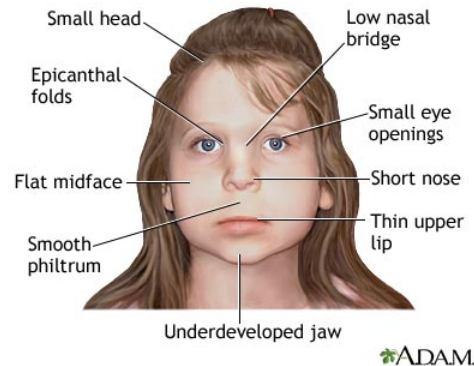
Increasing prevalence of risky drinking by young women has raised concerns about fetal alcohol syndrome / effects.



Notes:

# FAS Diagnosis

1. Prenatal or postnatal growth retardation
2. Brain dysfunction (intellectual retardation, poor muscle tone, irritability)
3. Facial dysmorphism
  - Microcephaly
  - Microphthalmia (smallness of the eye)
  - Thin upper lip



## Instructions:

1. Explain that the worldwide incidence of Fetal Alcohol Syndrome (FAS) is 1-3 births per 1,000 live births.
2. Explain that for a diagnosis of FAS, one abnormality from the 3 listed in the slide must be present
  1. Prenatal or postnatal growth retardation; failure to thrive (weight, length, and/or head circumference less than the 10<sup>th</sup> percentile)
  2. Central nervous system dysfunction, including intellectual, neurologic, and behavioural deficits manifested as mild to moderate mental retardation, hypotonia (poor muscle tone), irritability in infancy, and later hyperactivity in childhood. Mental abnormality occurs in 85% of FAS children, and although IQ scores vary, affected children rarely show normal mental ability.
  3. Facial dysmorphism (structural abnormalities) including at least two of three characteristics:
    - a) Microcephaly (head circumference less than 10<sup>th</sup> percentile)
    - b) Microphthalmia (abnormal smallness of the eye) or short palpebral fissures, ptosis (drooping eyelid), strabismus (imbalance of the eye muscles), or epicanthal folds (folds of the skin of the upper eyelid over the eye)
    - c) Poorly developed philtrum, thin upper lip (vermillion border), short upturned nose, or flattening or absences of the maxilla (upper jaw).

(Source: U.S. Department of Health and Human Services. Public Health Services. Substance Abuse and Mental Health Services Administration (1993). *Improving Treatment for Drug-Exposed Infants*. Tip 5. DHHS Publication No., (SMA) 93-2011.)

## Notes:

Even small amounts of alcohol such as one drink a day while pregnant could harm the baby.

# Pharmacokinetics

**5 minutes**  
to affect  
brain

2% excreted  
unchanged in  
sweat, breath,  
& urine

- *Rapidly absorbed* into blood by stomach (20%) and small intestine (80%)
  
- *Metabolised* by liver (95% – 99%)  
     alcohol → acetaldehyde → acetic acid & H<sub>2</sub>O → CO<sub>2</sub>
  
- *Distributed* in body fluids (not fat)
  
- 1 standard drink per hour raises BAC by about 0.01–0.03 g%.

## Notes

The ethanol molecule is small and highly water soluble.

Rate of absorption depends on gastric emptying time - the rate of alcohol absorption is slow in the stomach and faster in the small intestine. Food in the stomach delays emptying and hence slows alcohol absorption with lower peak BAC being reached.

- beverages <30% alcohol are rapidly absorbed but
- beverages > 40% alcohol cause gastric irritation and pyloric spasm resulting in delayed stomach emptying
- carbonated drinks neutralise stomach hydrochloric acid and speed up gastric emptying

The major metabolic pathway is via ADH, which becomes saturated at low alcohol concentrations. Induction of microsomal CYP2E1 occurs with chronic alcohol consumption to become a more significant metabolic pathway. It may also increase metabolism of other drugs.

Takes about 1 HOUR to clear 1 STANDARD DRINK  
 (average rate 7.5 grams/hour, range 4-12 grams)

## Alcohol: Effects on the brain



- No single receptor. Alcohol interacts with and alters function of many different cellular components.
- Primary targets are GABA, NMDA glutamate, serotonin, and ATP receptors
- Stimulates dopamine and opioid systems
- Effects of chronic consumption are opposite to acute because of homeostatic compensation

### Notes

#### Alcohol effects on neurotransmitters

↑GABA (inhibitory): acute - ↓anxiety, ataxia, sedation, amnesia  
chronic - tolerance, withdrawal, craving

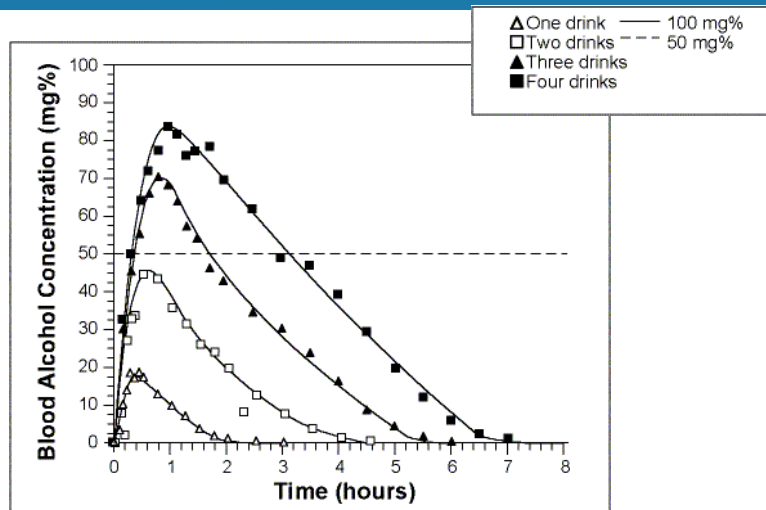
↓glutamate (excitatory): acute - intoxication  
chronic - up-regulation → seizures, withdrawal, craving

↑5HT<sub>3</sub>: acute - intoxication, nausea & vomiting  
chronic - reinforcement

Opioid receptors (direct/indirect → dopamine release)

acute - euphoria  
chronic - reinforcement

# Alcohol Metabolism



National Institute on Alcohol Abuse and Alcoholism (NIAAA)

## Additional information:

Metabolism is the body's process of converting ingested substances to other compounds. Metabolism results in some substances becoming more, and some less, toxic than those originally ingested. Metabolism involves a number of processes, one of which is referred to as oxidation. Through oxidation, alcohol is detoxified and removed from the blood, preventing the alcohol from accumulating and destroying cells and organs. A minute amount of alcohol escapes metabolism and is excreted unchanged in the breath and in urine. Until all the alcohol consumed has been metabolized, it is distributed throughout the body, affecting the brain and other tissues. Studying alcohol metabolism can also help us to understand how this process influences the metabolism of food, hormones, and medications.

Women and men absorb and metabolize alcohol differently. Women have a higher blood alcohol concentration (BAC) after consuming the same amount of alcohol as men. Women are more susceptible to alcoholic liver disease, heart muscle damage, and brain damage. The difference in BACs between men and women has been attributed to women's smaller amount of body water. An additional factor contributing to the difference in BACs may be that women have lower activity of the alcohol metabolizing enzyme ADH in the stomach, causing a larger proportion of the ingested alcohol to reach the blood. The combination of these factors may render women more vulnerable than men to alcohol-related diseases.

(Source: National Institute on Alcohol Abuse and Alcoholism No. 35; PH 371 January 1997. Available at: <http://pubs.niaaa.nih.gov/publications/aa35.htm>)



## Effects of alcohol intoxication

<b>.01-.02</b>	Clearing of head
<b>.02-.05</b>	Mild throbbing rear of head, slightly dizzy, talkative, euphoria, confidence, clumsy, flippant remarks
<b>.06-1.0</b>	↓ inhibitions, ↑ talkativeness, ↓ motor co-ord, ↑ pulse, stagger, loud singing!
<b>0.2-0.3</b>	Poor judgement, nausea, vomiting
<b>0.3-0.4</b>	Blackout, memory loss, emotionally labile
<b>0.4+</b>	Stupor, breathing reflex threatened, deep anaesthesia, death

### Notes

The euphoria and stimulation of behaviour at low concentrations is possibly related to noradrenaline and dopamine release. Pleasurable effects are a result of dopamine and endogenous opioids (mechanism unclear).

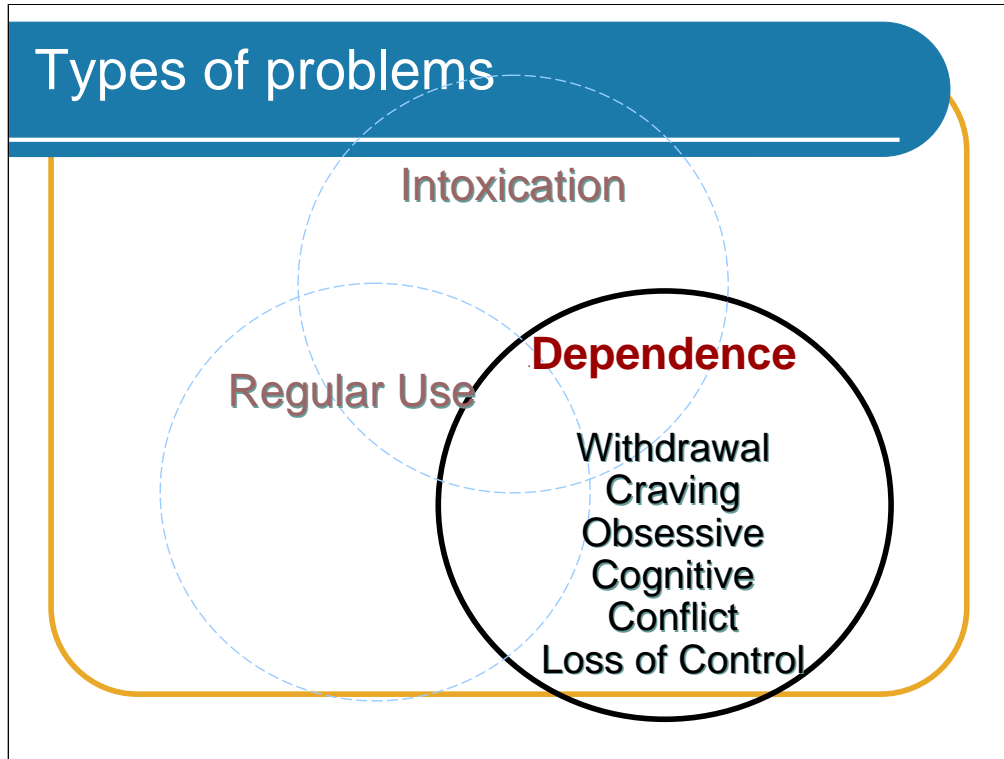
Alcohol enhances the effects of GABA (inhibitory neurotransmitter) at the GABA-A receptor resulting in anxiolysis, muscle relaxation, and sedation. It also inhibits the effects of glutamate (excitatory transmitter) through effects on NMDA receptor - thought to contribute to amnesic and cerebral depressant effects.

Serotonin receptor stimulation leads to both pleasurable and nauseating effects of alcohol.

Adverse health consequences - acute inhibition of brain function:

- reduced arousal
- reduced coordination & movement
- reduced ADH from pituitary
- vomiting (+ inhalation)
- unconsciousness
- death from respiratory depression.

Death may result from depression of the medullary respiratory centre and is usually preceded by stupor and coma.



### Notes

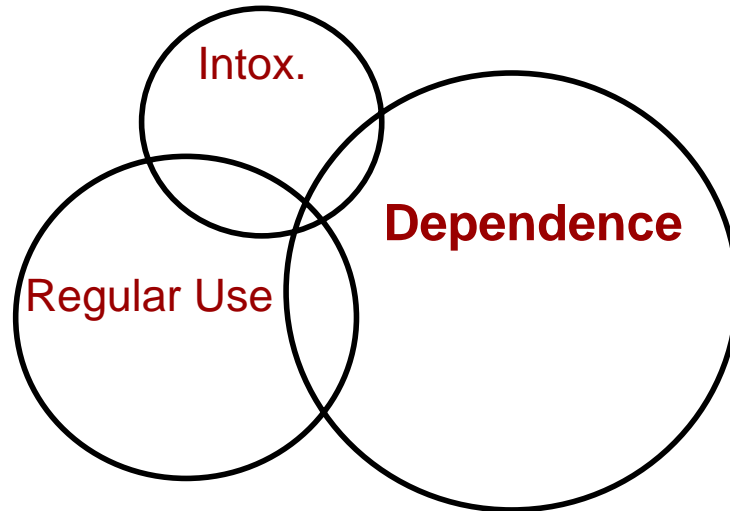
#### Dependence

- While people who are dependent on drugs often have problems related to intoxication and regular use, a specific set of problems that relate to being dependent or addicted can also be identified.
- “Cognitive Conflict” relates to the ambivalence experienced when there is a discrepancy between what a person is doing and what they would like to be doing. This is seen as a hallmark feature of addiction by Jim Orford in his book *Excessive Appetites*.

Speaker notes © 1998, Jon Rose.

‘Problems of Drug Use’ [PowerPoint presentation], Western Australian Alcohol and Drug Authority (WAADA), [www.drugnet.org](http://www.drugnet.org).

## Types of problems: Clinical samples



### Notes

#### Clinical Samples

While clinical samples often have a large proportion of people who have problems of dependence, this is not representative of the general population. It is important not to generalise from the types of problems seen in the clinical settings (e.g., dependence) to the general population (where intoxication and regular use are more common).

Speaker notes © 1998, Jon Rose.

'Problems of Drug Use' [PowerPoint presentation], Western Australian Alcohol and Drug Authority (WAADA), [www.drugnet.org](http://www.drugnet.org).

# Binge Drinking

## Binge drinking can lead to:

- increased risk taking
- poor judgment / decision making
- Misadventure / accidents
- increased risky sexual behaviour
- increased violence
- suicide.

### Notes

The International Center for Alcohol Policies says that diverse definitions of binge drinking exist. "Within the field of epidemiology, for example, there is disparity regarding the amount of alcohol that needs to be consumed in order to be drunk ([Binge Drinking: Key Facts and Issues](#), International Center for Alcohol Policies). A generally accepted definition of binge drinking is 5 or more drinks for men and 4 or more for women per occasion. Heavy binge drinking includes three or more such episodes in 2 weeks.

(Source: *Binge Drinking: Key Facts and Issues*. International Center for Alcohol Policies available at: <http://www.icap.org/PolicyIssues/BingeDrinking/KeyFactsandIssues/tabid/196/Default.aspx>).

- 40% of 16–17 year olds are binge drinkers. Alcohol is responsible for the majority of deaths in 15–34 age group.
- Some binge drinkers are less tolerant than regular drinkers creating a risk for greater harm, including alcoholic poisoning.
- Acute GI effects include oesophagitis, gastritis, GI bleeding, or pancreatitis (with repeat binges).
- Acute CVS effects include atrial or ventricular arrhythmias and myocardial infarction (with pre-existing ischaemic heart disease).

### CNS

Death results from profound depression of the medullary respiratory centre. Profound intoxication with anaesthesia or coma is likely at BAC  $\geq$  0.25%. Lethal levels range from 0.3%–0.5% in humans although some highly tolerant individuals survive these high concentrations.

### CVS

Alcohol (30–60 grams) decreases myocardial contractility and causes peripheral dilation (of central origin) resulting in a mild drop in blood pressure and a compensatory increase in sympathetic nervous system activity. This leads to increased heart rate and cardiac output with an increase in blood pressure. Cardiac oxygen consumption increases with alcohol use (especially when combined with exercise) leading to increased risk of myocardial infarction.

Paroxysmal tachycardia is especially related to binge drinking, a syndrome known as "holiday heart."

(Source: Lopatko, O., McLean, S., Saunders, J., Young, R., Robinson, G. & Conigrave, K. 2002 'Alcohol' in Hulse, G., White, J. & Cape, G. (eds.), *Management of Alcohol and Drug Problems*, Chap. 10, Oxford University Press, South Melbourne.)

...continued from previous page.

## **CVA**

This is a complex relationship. It is clear that heavier consumption (>40 grams per day for men, >20grams per day for women) is a risk factor for CVA, particularly for haemorrhagic stroke. There is some suggestion that low level intake may protect against ischaemic stroke by increasing HDL cholesterol.

**Cardiomyopathy** develops with consistent high intake (>80 grams per day over a period of years).

**Peripheral neuropathy** develops as a consequence of direct toxic effect of ethanol and thiamine deficiency.

**Impotence** results from direct toxic effects with suppression of pituitary and hypothalamic function, increased oestrogen and decreased testosterone.

## **Breast cancer**

There is a moderately strong and consistent dose-response relationship between intake and risk – 35% higher in 30–40 grams per day and 67% higher in >40 grams per day compared with those who drink little or none. Suggestion that drinking in later life is relatively more risky.

## **Fetal alcohol syndrome**

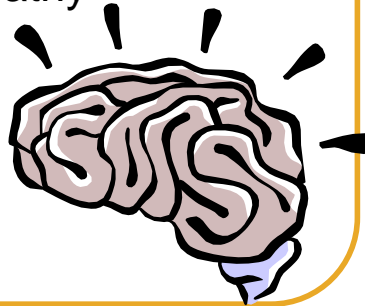
A high level consumption can contribute to adverse outcomes for unborn child. At higher levels (+ intoxication), there is a general dose-response relationship. Harms include fetal death, congenital malformation, IUGR and behavioural deficits. An average of one standard drink per day is considered the level below which there is no discernible evidence for fetal harm (see NHMRC, 2001).

Source: NHMRC (National Health and Medical Research Council) 2001, *Australian Alcohol Guidelines: Health Risks and Benefits*, Commonwealth of Australia, Canberra.

Hulse, G., White, J. & Cape, G. (eds.), 2002, *Management of Alcohol and Drug Problems*, Oxford University Press, South Melbourne.

## Alcohol-related brain injury

- Cognitive impairment may result from consumption levels of >70 grams per day
- Thiamine deficiency leads to:
  - Wernicke's encephalopathy
  - Korsakoff's psychosis
- Frontal lobe syndrome
- Cerebellar degeneration
- Trauma



### Notes

- Cognitive impairment may result from consumption levels of >70 grams per day.
- Thiamine deficiency leads to:
  - Wernicke's encephalopathy
    - 6<sup>th</sup> nerve palsy, ataxia, confusion
  - Korsakoff's psychosis
    - memory deficit, apathy, confusion, confabulation, poor judgment.
- Frontal lobe syndrome.
- Cerebellar degeneration.
- Trauma, e.g., subdural haematoma, haemorrhage (subarachnoid/intracerebral).

Source: Alcohol Related Brain Injury: A guide for General Practitioners and other health workers,  
<http://www.dasc.sa.gov.au/resources/documents/Alcohol%20Related%20Brain%20Injury%20GP.pdf> [accessed 29 September 2003]

## Interventions and treatment for alcohol-related problems

- Screening and assessment → individualised interventions
- Brief intervention and harm reduction strategies
- Withdrawal management
- Relapse prevention / goal-setting strategies
- Controlled drinking programs
- Residential programs
- Self-help groups

### Notes

Many of these treatments and interventions can be successfully implemented in a GP setting; e.g., brief intervention and provision of self-help materials (available from local and national specialist services). Other approaches may include referral to specialist agencies; e.g., GPs may prescribe a pharmacotherapy and involve the local drug and alcohol counselling service.

Controlled drinking may not be appropriate if a patient is clearly alcohol-dependent but can be useful for those with risky levels and patterns of use who are not yet dependent.

Trainers may wish to discuss the services available in their local area.

## Brief Intervention

### Consider the patient's:

- perspective on drinking
- attitudes towards drinking goals
- significant others
- short-term objectives

### Provide:

- information on standard drinks, risks, and risk levels
- encouragement to identify positive alternatives to drinking
- self-help manuals
- follow-up session

Source: adapted from Clarke, C., Bowen, J. & Carnegie, J. 2002, *Alcohol Training Resource*, Department of Human Services, Melbourne.



## Two steps towards alcohol brief intervention (BI)

### 1. Screening

- For example, the alcohol AUDIT, a 10-item questionnaire

### 2. Intervention

- Information
- Brief counselling
- Advice
- Referral (if required)

## Notes

Brief Interventions:

- are for people drinking at risky levels, prior to the establishment of dependence or significant health and social problems
- provide education, support, motivation
- involve assessment (e.g., use of the Alcohol Use Disorders Identification Test ('AUDIT') questionnaire to detect pattern of consumption).

### Provide intervention/feedback

Provide brief standardised information:

- the AUDIT score (see next slide)
- an interpretation of drinking risk
- advice to reduce risky drinking
- a definition of low-risk drinking - follow NHMRC guidelines
- an alcohol information pamphlet
- if within the "high-risk" category offer:
  - brief counselling
  - a self-help manual
  - referral/shared care for further management of alcohol problems where this is warranted and where patient agrees.

## AUDIT – The FLAGS approach

After administering the AUDIT, use  
“**FLAGS**”:

- **F**eedback results
- **L**isten to patient concerns
- Provide **A**lcohol education and information
- **G**oals of treatment – identify and plan
- **S**trategies discussed and implemented



### Feedback

- Offer advice and information, not therapy
- Be prepared for some discussion
- Be informative, not judgemental.

**Offer self-help booklet:** (e.g., WAADA 1995, *Drinker's Guide to Cutting Down or Cutting Out*, Drug and Alcohol Services Council (DASC), Adelaide)

if: the score is 10 or more, or you believe benefit will be gained from offering the booklet

- there is a willingness to reduce drinking
- you have time to go through the booklet briefly.

### Consider referral:

- if score is  $>13$  (men) or  $\geq 13$  (women)
- where there is evidence of physical or social problems
- the person may be dependent. They should not stop drinking abruptly if withdrawal is likely.

### Handout

*Using the AUDIT score with the FLAGS approach for treatment interventions.*

Trainers may wish to provide participants with copies of locally used and available self-help materials plus referral information.

## Harm-minimising strategies

### Benefits of cutting down or cutting out:

- save money
- be less depressed
- lose weight
- less hassles for family
- have more energy
- sleep better
- better physical shape

### Reduce the risk of:

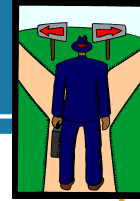
- liver disease
- cancer
- brain damage
- high blood pressure
- accidents
- injury
- legal problems

### Notes

See Handout 8: *Tip Sheet for Cutting Down Alcohol Consumption*.

Ideally, patients should identify for themselves the advantages of cutting down/ceasing drinking. The patient's reasons for change (rather than the GP's reasons) are the most important influences in motivating, promoting, and maintaining changes in behaviour.

# Choosing a treatment option



## Severity

No major lifestyle disruptions, not severely dependent

Major lifestyle disruptions, significant dependence

## Goal

Reducing consumption /controlled (or even Abstinence)

Abstinence

## Treatment options

For example:

- outpatient counselling
- group or individual work (skills training, relapse prevention)
- marital and family therapy
- loss and grief counselling
- self-help / support groups

Above options plus:

- withdrawal management
- pharmacotherapy
- residential rehabilitation

## Notes

Ideally, abstinence should be the ultimate goal for those who have problems controlling their drinking and for those who are dependent as well. Nonetheless, abstinence is a more suitable goal for those who are severely dependent, or for those whose alcohol use has caused major problems.

Many strategies can be valuable for both groups described above (although the goals may be different).

Consider the benefits of undertaking a shared care relationship with:

- alcohol and drug workers
- psychologists
- social workers etc.

It may be necessary to consider the use of a pharmacotherapy such as acamprostate or naltrexone for those who are dependent, as covered later in this topic. Also it may be necessary to consider co-morbid conditions such as anxiety and depression, which may be a cause or consequence of drinking.

# Withdrawal

Usually occurs 6–24 hours after last drink:

- tremor
- anxiety and agitation
- sweating
- nausea and vomiting
- headache
- sensory disturbances – hallucinations

Severity depends on:

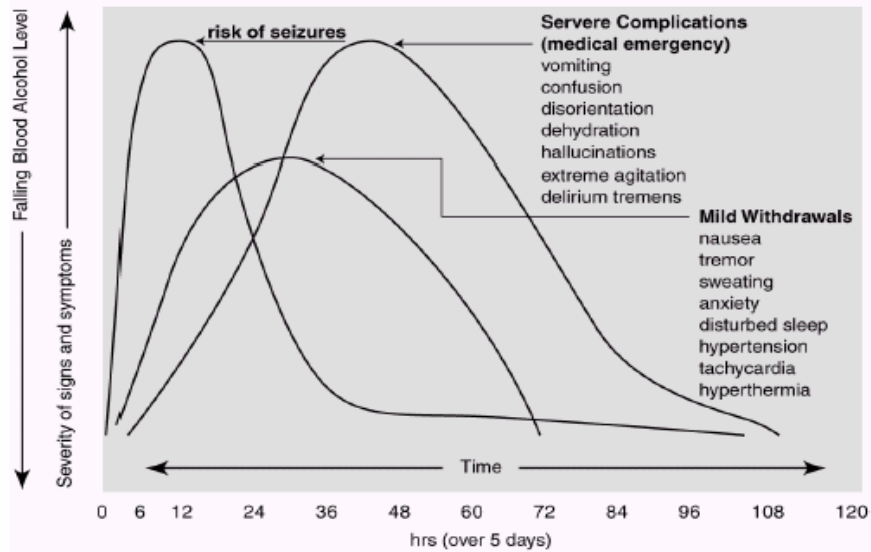
- pattern, quantity and duration of use
- previous withdrawal history
- patient expectations
- physical and psychological wellbeing of the patient (illness or injury)
- other drug use/dependence
- the setting in which withdrawal takes place

## Notes

Handout: CIWA-AR Withdrawal Scale.

See: NCETA 2004, *Alcohol and Other Drugs: A Handbook for Health Professionals*, Australian Government Department of Health & Ageing, Canberra.

## Progress of Alcohol Withdrawal from Time of Last Drink



(Source: deCrespigny & Cusack (2003)  
Adapted from NSW Health Detoxification Clinical Practice Guidelines (2000–2003))

Source: DeCrespigny, C. & Cusack, L. 2003, *Alcohol, Tobacco & Other Drugs Guidelines for Nurses and Midwives: A Framework for Policy and Standards* Flinders University and Drug and Alcohol Services Council (DASC), Adelaide.

## Treatment of alcohol withdrawal symptoms

### Medications for Symptomatic Treatment

- Diazepam
- Thiamine & multivitamins
- Antiemetic
- Analgesia (e.g., paracetamol)
- Antidiarrhoeal



### Notes

Diazepam regime recommendations vary but are usually 40–50 mg/day in divided doses for the first couple of days, reducing to 0 by the end of 4–5 days. Administration of diazepam to individuals under the influence of alcohol should be done with caution, since paradoxical reactions can result.

Because oral thiamine is poorly absorbed in patients with a pattern of chronic alcohol consumption, high doses (ideally parenterally) (100 mg im) should be considered in the first instance.

Thiamine  $\geq 100$  mg daily.

Main principles for carers:

- maintain orientation, provide reassurance
- use simple commands, brief explanations, repetition
- treat and manage symptoms (headache, diarrhoea, generalised aches and pains, nausea and vomiting etc.)
- encourage fluids and light meals
- ensure calm, uncluttered, comfortable environment with dim lighting, comfortable clothing and clean bedclothes
- avoid sudden movements, noise, unnecessary actions.

## Post-withdrawal management

### Treatment options:

- retain in treatment, ongoing management
- seek referral

### Considerations:

- patient's wants (abstinence or reduced consumption, remaining your patient)
- severity of problems

### Pharmacotherapies:

- acamprosate
- naltrexone
- disulfiram

Source: Clarke, C., Bowen, J., & Carnegie, J. 2002, *Alcohol Training Resource*, Department of Human Services, Melbourne.



## Naltrexone and Acamprosate

- Effective.
- Work well with variety of supportive treatments, e.g., brief intervention, CBT, supportive group therapy.
- Start following alcohol withdrawal. Proven efficacy where goal is abstinence, uncertain with goal of moderation.
- No contraindication while person is still drinking, although efficacy uncertain.
- Generally safe and well tolerated.

## Clinical guidelines



### Naltrexone 50 mg daily:

- indicated especially where strong craving for alcohol after a priming dose
- ↓ likelihood of lapse progressing to relapse
- LFTs < x3 above normal
- side effects: nausea in the first few days

### Acamprosate 600 mg (2 tabs) tds:

- indicated especially when susceptible to drinking cues or drinking triggered by withdrawal symptoms
- low potential for drug interactions
- need normal renal function
- side effects: diarrhoea, headache, nausea, itch

### Notes

- Because naltrexone is potentially hepatotoxic, it is considered better when LFTs (transaminases) are not more than three times above normal limits. Monitoring liver function recommended.
- Since acamprosate is excreted by the kidneys, normal renal function is necessary. It is not metabolised by the liver, so there is low likelihood of drug interactions. The dose is reduced if individuals weigh less than 60 kg (to 2 mane then 1 at lunch and dinner).
- Neither drug causes severe reaction if alcohol is ingested while drug being taken.

## Disulfiram

- Acetaldehyde dehydrogenase inhibitor – 200 mg daily
- → unpleasant reaction with alcohol ingestion (depending on dose)
- Indications: alcohol dependence + goal of abstinence + need for external aid to abstinence
- Controlled trials: ↑ abstinence rate in first 3–6 months
- Best results with supervised ingestion & contingency management strategies
- Caution when using with patients who have significant symptoms of depression

### Notes

Disulfiram (Antabuse®) is not a first line agent. It is best reserved for those for whom other strategies have not worked and for those who would be motivated by the knowledge that they cannot drink until at least 5 days after ceasing the medication. If alcohol is consumed, the reaction is extremely unpleasant and potentially fatal.

Low dose (100 mg) induces milder unpleasant reaction. Another approach is to titrate to dose, which induces a reaction of sufficient severity. Disulfiram can be considered first line as it is more effective than naltrexone and acamprosate but the unpleasant effects may be difficult to tolerate. There are medical precautions such as for ischaemic heart disease, psychosis, and others.

# Benzodiazepines

*“Benzodiazepines:  
the opium of the masses”*

(Source: Malcolm Lader, *Neuroscience*, 1978)

## Notes

This slide set contains information for GPs on:

- background, medical use & problems
- epidemiology
- doctor shopping
- properties
- effects & harms including overdose
- assessment
- withdrawal & treatment matching.

Please adapt this slide set where needed to meet the learning/information needs of your GP group(s). For instance, you may wish to include cases and question prompts that focus attention on what GPs encounter in their day to day practice.

Source: Cape, G., Hulse, G., Robinson, G., McLean, S., Saunders, J., Young, R. & Martin, J. 2002, 'Sedative-Hypnotics', in Hulse, G., White, J. & Cape, G. (eds.) 2002, *Management of Alcohol and Drug Problems*, ch. 11, Oxford University Press, South Melbourne.

## Benzodiazepines: History

1950s	Invented by Swiss chemists who identified its sedative effects
1950s–60s	Chlordiazepoxide (Librium) marketed as a safer alternative to barbiturates; along with newer benzodiazepines (BZDs), promoted as having no dependence-inducing properties!
1970s–80s	BZDs most commonly prescribed drug class in the world
1990s on	Some decline in the number of prescriptions due to problems related to dependence and reduced therapeutic value. Generally safer than barbiturates; problems are with longer term and polydrug use
1998	8.89 million prescriptions dispensed

Sources: Cape, G., Hulse, G., Robinson, G., McLean, S., Saunders, J., Young, R. & Martin, J. 2002, 'Sedative-Hypnotics', in Hulse, G., White, J. & Cape, G. (eds.) 2002, *Management of Alcohol and Drug Problems*, ch. 11, Oxford University Press, South Melbourne.

Victoria Police 2002, *Custodial Drug Guide: Medical Management of People in Custody with Alcohol and Drug Problems*, Custodial Medicine Unit, Victoria Police, Mornington, Victoria.

## General medical / psychiatric indications for benzodiazepine use

- Anxiolytic – chronic / phobic anxiety & panic attacks
- Sedative and hypnotic – sleep disturbance & anaesthesia / premedication
- Anticonvulsant – status epilepticus, myoclonic & photic epilepsy
- Muscle relaxant – muscle spasm / spasticity
- Alcohol withdrawal

Sources: Cape, G., Hulse, G., Robinson, G., McLean, S., Saunders, J., Young, R. & Martin, J. 2002, 'Sedative-Hypnotics', in Hulse, G., White, J. & Cape, G. (eds.) 2002, *Management of Alcohol and Drug Problems*, ch. 11, Oxford University Press, South Melbourne.

Victoria Police 2002, *Custodial Drug Guide: Medical Management of People in Custody with Alcohol and Drug Problems*, Custodial Medicine Unit, Victoria Police, Mornington, Victoria.

## Exercise: Case study

After the recent and unexpected death of her husband from an MI, Shirley, aged 62, presented for you to check her cardiac state as she fears a similar fate to her husband's.

She is afraid to go out alone, and she fears going to sleep as she is scared she will not wake up. She experiences occasional non-radiating chest pain. She has been taking sleeping tablets for several years and finds that they are now no longer working.

## Patterns of use

- BZDs are one of the most prescribed drugs
- 4% of all prescriptions from general practitioners are for benzodiazepines (BZDs)
- Predictors for BZD prescription include:
  - being female
  - being elderly
  - being an established patient
  - attending a busy doctor, or a doctor in inner urban area
- Over 40% of prescriptions given to people  $\geq 70$  years old
- Night time use tends to increase with age
- 58% of current users report daily use for  $\geq 6$  months

### Notes

In Australia, the prevalence of BZD usage and dependence is not clear, although BZDs are prescribed at the rate of six million PBS prescriptions (not including hospital and repatriation benefits) per year. Up to 2% of the Australian adult population may be daily and long-term users of BZDs.

Pharmaceutical records suggest that enough BZD is prescribed to enable 3% of the population to use every day (Cape et al., 2002, p.224).

Sources: Carr, N. 1998, 'I Just Want Some Valium, Doc' *Australian Family Physician*, vol. 27, no. 9, pp. 817–821.

Cape, G., Hulse, G., Robinson, G., McLean, S., Saunders, J., Young, R. & Martin, J. 2002, 'Sedative-Hypnotics', in Hulse, G., White, J. & Cape, G. (eds.) 2002, *Management of Alcohol and Drug Problems*, ch. 11, Oxford University Press, South Melbourne.



## BZDs and long-term use

- Long-term use is common and associated with:
  - altered use patterns (from nighttime to daytime use)
  - excessive sedation
  - cognitive impairment
  - increased risk of accidents
  - adverse sleep effects
  - dependence and withdrawal (even at therapeutic doses)
- BZDs have an additive effect with alcohol / other CNS depressants, increasing the risk of harm
- BZDs have limited long-term efficacy

### Notes

Benzodiazepines are usually not a solution to presenting problems, as they have limited long-term efficacy and high dependence-producing potential. For some people, even short-acting night sedation can lead to daytime use (i.e., when taken to avoid withdrawal). Similarly, people quickly learn that continuance of use means that withdrawal can be avoided.

Long-term use is common and associated with:

- excessive sedation
- cognitive impairment
- increased risk of accidents
- adverse sleep effects
- dependence and withdrawal (even at therapeutic doses).

When used with alcohol and other CNS depressants, BZDs have an additive effect, increasing the risk of harm.

## BZD and illicit drug use

- Illicit BZD use is usually oral, although around 5% are likely to inject (usually males)
- Often 2<sup>nd</sup> drug of choice for illicit drug users, as BZDs assist withdrawal from opioids, stimulants, and alcohol
- Estimated around 70% of people using  $\geq 50$  mg per day are polydrug users, who tend to:
  - be younger
  - have higher daily doses and higher lifetime exposure
  - use in combination with other CNS depressants to increase intoxication
  - prefer fast-acting BZDs (diazepam, flunitrazepam)
  - may convert form to enable injection

### Notes

Flunitrazepam (Rohypnol) is generally no longer available. However, requests for the drug may indicate suspicion of illicit use.

People involved in illicit use are more likely to have several legal sources from whom they obtain prescriptions, and be familiar with a variety of different BZD types.

Sources: Cape, G., Hulse, G., Robinson, G., McLean, S., Saunders, J., Young, R. & Martin, J. 2002, 'Sedative-Hypnotics', in Hulse, G., White, J. & Cape, G. (eds.) 2002, *Management of Alcohol and Drug Problems*, ch. 11, Oxford University Press, South Melbourne.

Victoria Police 2002, *Custodial Drug Guide: Medical Management of People in Custody with Alcohol and Drug Problems*, Custodial Medicine Unit, Victoria Police, Mornington, Victoria, pp. 1-15 – 1-20.

## Benzodiazepines: Half-life

<b>Benzodiazepines</b>	<b>Half-life (hrs) 2 [active metabolite]</b>	<b>Appr. Equivalent Oral dosages (mg)<sup>3</sup></b>
Alprazolam (Xanax, Xanor, Tafil)	6-12	0.5
Diazepam (Valium)	20-100 [36-200]	10
Clonazepam (Klonopin, Rivotril)	18-50	0.5

### Instructions:

1. Read the slide to your audience.
2. Explain what “half-life” is. Half-life is a numerical expression of how long it takes for a drug to clear out of the body. It can also be described as the time taken for blood concentration to fall to half its peak value after a single dose. The half-life of active metabolites is shown in square brackets in the slide. This time may vary considerably between individuals.
3. Explain that although diazepam is fast-acting, it has a long half-life, as seen in the table. Other preferred drugs more like flunitrazepam with a short half life include alprazolam (Xanax).

Note: 10 mg of diazepam (valium) are given.

(Source: Ashton, H. (2002). *Benzodiazepines: How they work and how to withdraw*. Available at: <http://www.benzo.org.uk/manual/index.htm>)

## Pharmacodynamics

- Rapidly absorbed orally (slower rate of absorption IM)
- Lipid soluble - differences determine rate of passage through blood brain barrier, i.e.,
  - ↑ lipophilic → ↑ speed of onset
- Duration of action variable –
  - ↑ lipophilic → ↓ duration of action due to distribution in adipose tissue

### Notes

Benzodiazepines are:

- rapidly absorbed orally (slower rate of absorption IM)
- lipid soluble. Any differences determine the rate of passage through the blood brain barrier; i.e., ↑lipophilic →↑ speed of onset.

Duration of action is variable – increased lipophilic → decreased duration of action due to distribution in the adipose tissue. BZDs are metabolised in the liver, mostly undergoing oxidative transformation prior to conjugation with glucuronic acid for urinary excretion.

Elimination half life (drug & active metabolites) ranges from 8 to 60 or more hours. If the drug has short half-life with no active metabolites, a steady state is rapidly attained with minimal accumulation.

BZDs enhance GABA (main inhibitory neurotransmitter), which bind to postsynaptic GABA-A receptors, opening chloride channels (which leads to hyperpolarisation) and inhibiting actions of the neurone (making the neurons more difficult to excite).

Specific neuronal membrane receptors for BZD are closely associated with synaptic GABA receptors.

Receptors are distributed through the CNS, concentrated in reticular formation and limbic systems, and peripheral binding sites.

Further understanding of the effects of BZDs on receptor subgroups may lead to the development of non-sedating anxiolytic BZDs.

## Metabolism

- Metabolised in the liver – mostly oxidative transformation prior to conjugation with glucuronic acid for urinary excretion
- Elimination half life (drug & active metabolites) ranges from 8 – >60 hours, if short half life & no active metabolites, it rapidly attains steady state with minimal accumulation

# Neurotransmission

- Potentiate neurotransmission mediated by GABA (main inhibitory neurotransmitter), therefore neurons are more difficult to excite
- Specific neuronal membrane receptors for BZD closely associated with synaptic GABA receptors
- Receptors distributed through CNS, concentrated in reticular formation & limbic systems, also peripheral binding sites
- Further understanding of the effects of BZDs on receptor subgroups may lead to the development of non-sedating anxiolytic BZDs

## Effects: Low dose

### Short term:

- Sedation
- Anxiety relief
- Anticonvulsant properties
- Can usually attend daily business (though should not drive in first 2 weeks of treatment)

### Other effects:

- Drowsiness, lethargy, fatigue
- Impaired concentration, coordination, memory
- Reduced ability to think and learn
- Clumsiness, ataxia
- Depression
- Mood swings
- Blurred vision and / or vertigo
- Light-headedness
- Nausea, constipation, dry mouth, loss of appetite

### Notes

Different BZDs do not vary significantly in their effects. Variation is mainly in their course/duration of action.

Sources: Cape, G., Hulse, G., Robinson, G., McLean, S., Saunders, J., Young, R. & Martin, J. 2002, 'Sedative-Hypnotics', in Hulse, G., White, J. & Cape, G. (eds.) 2002, *Management of Alcohol and Drug Problems*, ch. 11, Oxford University Press, South Melbourne.

Victoria Police 2002, *Custodial Drug Guide: Medical Management of People in Custody with Alcohol and Drug Problems*, Custodial Medicine Unit, Victoria Police, Mornington, Victoria.

## Effects: High dose

### Short term

- Sedation
- Intoxication
- Drowsiness

### Other effects

- Paradoxical excitement
- Mood swings
- Hostile and erratic behaviour

### Toxicity

- Performance deficits
- Emotional blunting
- Muscle weakness
- Sensitivity
- Potentiates other drugs
- Euphoria, hypomania

Higher doses generally result in:

- sedation
- sleepiness
- slower or delayed actions and responses
- appearance of little muscle strength (some people).

Progressively larger doses produce effects similar to alcohol and barbiturates – disinhibition, excitation, feeling “good,” reduction of anxiety. Some report loss of memory.

Paradoxical effects:

- while uncommon, they may lead to extremely uninhibited behaviour whereby the user undertakes actions uncharacteristic of them (e.g., shoplifting, assault, child battering). An increased number of people using BZDs at high doses report feeling invincible. Some people report deliberately seeking BZDs in order to conduct crime.
- Performance deficits:
- memory (especially anterograde amnesia)
- motor incoordination, decreased reaction time, ataxia – impaired performance on complex tasks (e.g., driving performance can be severely affected)
- elderly especially prone to cognitive and neuromuscular effects of BZD.

Emotional blunting:

- inhibition of arousal centres of the brain. Hence long-term users of BZD may report anhedonia
- BZDs are reported to interfere with the grieving process.

Other effects include:

- hypotonia, headaches, sensitivity reactions, potentiation other CNS depressants, menstrual irregularities, breast engorgement, euphoria, restlessness, and hypomanic behaviour.

Sources: Cape, G., Hulse, G., Robinson, G., McLean, S., Saunders, J., Young, R. & Martin, J. 2002, 'Sedative-Hypnotics', in Hulse, G., White, J. & Cape, G. (eds.) 2002, *Management of Alcohol and Drug Problems*, ch. 11, Oxford University Press, South Melbourne.

Victoria Police 2002, *Custodial Drug Guide: Medical Management of People in Custody with Alcohol and Drug Problems*, Custodial Medicine Unit, Victoria Police, Mornington, Victoria.



## Drug + alcohol interactions



- CNS depressants, e.g., benzodiazepines → Confusion, depressed respiration
- Antipsychotics, antidepressants → Decreased metabolism, toxicity & CNS depression
- Opioid analgesics, antihistamines (some) → CNS depression
- Hypoglycaemics (chlorpropamide), metronidazole, cephalosporins (some) → Facial flushing, headache

### Notes

There are a large number of potential drug-alcohol interactions. This slide depicts a few of the more common ones.

## Overdose

- Benzodiazepines are the most commonly implicated drug in overdose cases
- On their own, unlikely to cause death despite causing respiratory depression
- Serious / potentially fatal implications when used in combination with other CNS depressants

Sources: Cape, G., Hulse, G., Robinson, G., McLean, S., Saunders, J., Young, R. & Martin, J. 2002, 'Sedative-Hypnotics', in Hulse, G., White, J. & Cape, G. (eds.) 2002, *Management of Alcohol and Drug Problems*, ch. 11, Oxford University Press, South Melbourne.

Victoria Police 2002, *Custodial Drug Guide: Medical Management of People in Custody with Alcohol and Drug Problems*, Custodial Medicine Unit, Victoria Police, Mornington, Victoria.

## Overdose response

Overdose depresses the conscious state and respiratory system. Airway management and assisted ventilation is necessary.

### *Flumazenil®*

- a BZD antagonist which reverses BZD overdose, though contraindicated outside the emergency department
- precipitates seizures in:
  - chronic BZD users
  - pre-existing epilepsy
  - tricyclic antidepressant users
  - concurrent amphetamine or cocaine users

Source: Victoria Police 2002, *Custodial Drug Guide: Medical Management of People in Custody with Alcohol and Drug Problems*, Custodial Medicine Unit, Victoria Police, Mornington, Victoria.

## Assessment

- Review BZD medication
  - initial reasons for use
  - type of BZD, response to, and patterns of use
  - side-effects reported or observed
  - current / past withdrawal history and symptoms
- Obtain physical history (concurrent medical problems)
- Mental health history (e.g., depression)
- Other drug (and alcohol / prescription drug) use
- Discuss options
  - risks of continued and prolonged use
  - withdrawal potential / risks, management options

### Notes

Assess for withdrawal experience after cessation of short-term use accompanied by short periods of abstinence. Common symptoms include sleep disturbance, early morning waking, anxiety, tremor, sweating, sometimes panic attacks or hyperventilation. Relief from discomfort upon reinstatement of use is a reliable marker of dependence.

Distinguish low- from high-risk use. High-risk use is generally associated with polydrug use.

Ask about intravenous use.

Short-term, high-level binges do not usually indicate significant long-term risk.

Identify risk for acute or dangerous symptoms, e.g., panic attacks, seizures, psychosis.

Be aware of agitation or depression in patients.

Source: Alliance of NSW Divisions GP Liaison Project, 'Detoxification from Benzodiazepines in General Practice', Tip Sheet Series, [www.answd.com.au](http://www.answd.com.au).

Victoria Police 2002, *Custodial Drug Guide: Medical Management of People in Custody with Alcohol and Drug Problems*, Custodial Medicine Unit, Victoria Police, Mornington, Victoria.

## Dependence

Two groups of patients are especially likely to develop dependence.

1. Low dose dependence occurs among women and elderly prescribed low doses over long time periods (up to 40% experience withdrawal symptoms)
2. High dose dependence occurs among polydrug users

## Withdrawal

- 40% of people on long-term therapeutic BZD doses will experience withdrawal if abruptly ceased
- Symptoms occur within 2 “short-acting” to 7 day “long-acting” forms
- BZD withdrawal:
  - is not life-threatening & usually protracted
  - initial symptoms / problems re-emerge on cessation
  - issues usually more complicated on cessation
- Seizures uncommon (unless high dose use or abrupt withdrawal, ± alcohol use)
- Two main groups of users:
  - prescribed (older women)
  - high level, erratic polydrug use

### Notes

#### Seizures

Seizures are most likely after abrupt withdrawal from high-dose, high efficacy, short half-life BZD, but can occur with others.

Seizures are rare in low dose forms except for:

- concomitant use of medications that lower seizure threshold (e.g., Tricyclics)
- use of other CNS depressants (e.g., alcohol)
- old age
- pre-existing seizure disorder not adequately treated.

Pre-existing seizure disorders which are properly treated do not increase seizure risk in BZD withdrawal, though latent conditions may be unmasked when abruptly stopped.

Seizure may occur 24 hours (e.g., oxazepam) up to 7 days (e.g., diazepam) after cessation of drug use, depending on half life. (Victoria Police, 2002)

“Rebound” or reinstatement of symptoms for which BZD was originally prescribed may occur for 2–3 days following abrupt withdrawal of short-term BZD use. Onset is generally related to the half-life of the drug.

Some will find that following withdrawal, the symptoms of the original condition will return. This is often difficult to distinguish from “rebound” phenomena. Treatment may not necessarily entail a return to BZD therapy.

Following long-term BZD treatment of more than one year, 50–60% of patients treated for anxiety, and up to 95% of patients treated for panic disorder, will report recurrent symptoms (Victoria Police, 2002).

Long-term BZD use will alter sleep patterns.

Source: Victoria Police 2002, *Custodial Drug Guide: Medical Management of People in Custody with Alcohol and Drug Problems*, Custodial Medicine Unit, Victoria Police, Mornington, Victoria.

## Withdrawal severity

Severity of withdrawal is dependent on:

- pattern and extent of use  
(duration, quantity, type (half-life))
- withdrawal experience  
(prior symptoms, success, complications)
- coexisting physical / mental health problems

## 3 Areas of BZD withdrawal

### **Anxiety and anxiety-related symptoms**

- anxiety, panic attacks, hyperventilation, tremor
- sleep disturbance, muscle spasms, anorexia, weight loss
- visual disturbance, sweating
- dysphoria

### **Perceptual distortions**

- hypersensitivity to stimuli
- abnormal body sensations
- depersonalisation/derealisation

### **Major events**

- seizures (grand mal type)
- precipitation of psychosis



## Withdrawal management

- Obtain an accurate consumption history
- Calculate diazepam equivalent and substitute. Reduce gradually over 6–8 weeks (or longer, e.g., 3–4 months)
- Reduce dose by a fixed rate at weekly intervals (usually 10%–20% initially, then 5%–10% / week, or slower when dose at 15 mg or less).
- Supervision of long-term BZD reductions (3-4 months)
- Dose at regular times
- Regularly review and titrate dose to match symptoms
- If symptoms re-emerge, dose may be plateaued for 1–2 weeks, or increased before reduction resumed
- Provide support, *not* pharmacological alternatives for conditions such as insomnia and anxiety.

### Notes

To date, BZD withdrawal scales have not been validated. Therefore, use clinical judgement and observation (Victoria Police, 2002).

In the GP setting, 50 mg/day would probably be the maximum dose you would want to use. If the dose equivalent is higher or there are other complicating factors, consider specialist referral or assistance.

Sources: Alliance of NSW Divisions GP Liaison Project, 'Detoxification from Benzodiazepines in General Practice', Tip Sheet Series, [www.answd.com.au](http://www.answd.com.au).

Victoria Police 2002, *Custodial Drug Guide: Medical Management of People in Custody with Alcohol and Drug Problems*, Custodial Medicine Unit, Victoria Police, Mornington, Victoria.

## Outpatient withdrawal protocol

- **Consider outpatient withdrawal management:**
  - if willing, committed, compliant, and has adequate social supports
  - if taking  $\leq 50$  mg diazepam equivalent or therapeutic doses
  - if no previous history of complicated withdrawal
  - if able to attend weekly reviews
- **Develop an individualised withdrawal plan considering:**
  - psychosocial factors
  - coping skills
  - previous attempts
  - counselling / referral needs

Source: Alliance of NSW Divisions GP Liaison Project, 'Detoxification from Benzodiazepines in General Practice', Tip Sheet Series, [www.answd.com.au](http://www.answd.com.au).

## Inpatient withdrawal protocol



Inpatient withdrawal management is necessary if the patient:

- is using  $\geq 50$  mg diazepam equivalent for  $>14$  days
- has a history of alcohol or other drug use or dependence
- has concurrent medical or psychiatric problem
- has a history of withdrawal seizures
- if significant symptoms are predicted
- is in an unstable social situation
- has previous poor compliance / doubtful motivation
- is in concurrent methadone stabilisation

## Drug interactions

BZDs either potentiate / increase effects or interfere with metabolism or absorption of:

- alcohol
- antidepressants and antihistamines
- disulfiram, cimetidine, erythromycin
- anticonvulsants
- anticoagulants, oral diabetic agents
- cisapride

## Exercise: Case study

Meg, a 47-year-old woman, always has alcohol on her breath and frequently falls. She moved into the suburb a few months ago and is well known at the local liquor shop and hotel. She denied alcohol use until a recent fracture and hospital admission. Since her discharge, she has started drinking again, mostly spirits.

She presents to you late one afternoon seeking benzodiazepines.

*As her doctor, how will you respond?*

*If her alcohol use continues, how can harm be reduced?*

Questions?



Comments?

Thank you for your time!

End of Workshop 2

Volume C, Module 1, Workshop 3:  
Psychostimulants, Volatile Substances, and  
Cannabis : Medical Issues and Treatment  
Approaches





## Training objectives



### **At the end of this training you will:**

- Understand acute and chronic effects of psychostimulants, volatile substances, and cannabis and the medical and psychiatric dangers associated with intoxication, overdose, withdrawal, and interactions with other substances.
- Know treatment protocols to treat intoxication and overdose
- Know withdrawal approaches and protocols
- Know about necessary treatments following detoxification
- Know proper setting and support services needed to properly conduct treatments

# Stimulants



## Stimulants

**Description:** A group of synthetic and plant-derived drugs that increase alertness and arousal by stimulating the central nervous system. Although MDMA (ecstasy) has some hallucinogenic properties, it is often classified as a stimulant

**Medical Uses:** Short-term treatment of obesity, narcolepsy, and hyperactivity in children

**Method of Use:** Intravenous, intranasal, oral, smoking

## Types of stimulant drugs

### **Amphetamine Type Stimulants (ATS)**

- Amphetamine
- Dexamphetamine
- Methylphenidate
- Methamphetamine (“speed,” “crystal,” “ice,” “yaba,” “shabu”)

## Types of stimulant drugs

### Cocaine Products

- Cocaine powder (generally sniffed, injected, smoked on foil)



- "Crack" (smoked)



## Types of stimulant drugs

### Methyldioxymethamphetamine (MDMA)

(A synthetic drug with psychostimulant and hallucinogenic properties)

- Commonly referred to as **ecstasy**. Sold in tablet form
- Estimated to be 10 million users worldwide

## Scope of the ATS problem worldwide

- According to surveys and estimates by WHO and UNODC, ATS is the most widely used category of illicit drugs in the world except for cannabis.
- Worldwide, there are an estimated 26 million or more regular users of amphetamine, methamphetamine, or ecstasy, as compared to approximately 16 million heroin users and 14 million cocaine users.
- Methamphetamine accounts for over 90% of the ATS used worldwide



## Methamphetamine vs. cocaine

- Cocaine half-life: 2 hours
- Methamphetamine half-life: 10 hours
- Cocaine paranoia: 4 - 8 hours following drug cessation
- Methamphetamine paranoia: 7-14 days
- Methamphetamine psychosis - May require medication / hospitalisation and may not be reversible
- Neurotoxicity: Appears to be more profound with amphetamine-like substances



## Acute stimulant effects

### *Psychological*

- Increased energy
- Increased clarity
- Increased competence
- Heightened feelings of sexuality
- Increased sociability
- Improved mood
- Powerful rush of euphoria - freebase and intravenous only

## Acute stimulant effects

### *Physical*

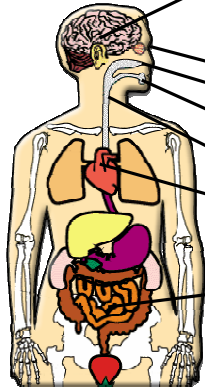
- Increased heart rate
- Increased pupil size
- Increased body temperature
- Increased respiration
- Cardiac arrhythmias
- Constriction of small blood vessels
- Decreased appetite
- Decreased need for sleep

## Chronic stimulant effects

### *Physical*

- Weight loss / anorexia
- Sleep deprivation
- Respiratory system disease
- Cardiovascular disease
- Headaches
- Severe dental disease
- Needle marks and abscesses - intravenous only
- Seizures

## Long-term effects of stimulants



- > Strokes, seizures, and headaches
- > Irritability, restlessness
- > Depression, anxiety, irritability, anger
- > Memory loss, confusion, attention problems
- > Insomnia
- > Paranoia, auditory hallucinations, panic reactions
- > Suicidal ideation
- > Sinus infection
- > Loss of sense of smell, nosebleeds, chronic runny nose, hoarseness
- > Dry mouth, burned lips
- > Worn teeth (due to grinding during intoxication)
- > Problems swallowing
- > Chest pain, cough, respiratory failure
- > Disturbances in heart rhythm and heart attack
- > Gastrointestinal complications (abdominal pain and nausea)
- > Loss of libido
- > Malnourishment, weight loss, anorexia
- > Weakness, fatigue
- > Tremors
- > Sweating
- > Oily skin, complexion





## Meth use leads to severe tooth decay



**“Meth Mouth”**

Source: *The New York Times*, June 11, 2005

## Prenatal meth exposure



Preliminary findings on infants exposed prenatally to methamphetamine (MA) and nonexposed infants suggest...

- Prenatal exposure to MA is associated with an increase in SGA (small for gestational size).
- Neurobehavioural deficits at birth were identified in NNNS (Neonatal Intensive Care Unit Network Neurobehavioral Scale) neurobehaviour, including dose response relationships and acoustical analysis of the infant's cry.

(Source: Lester et al., 2005)



## Chronic stimulant effects

### *Psychological*

- Severe anxiety
- Paranoia
- Psychosis
- Irritability
- Confusion
- Desire to isolate
- Memory impairment
- Inability to concentrate
- Loss of control
- Aggressiveness

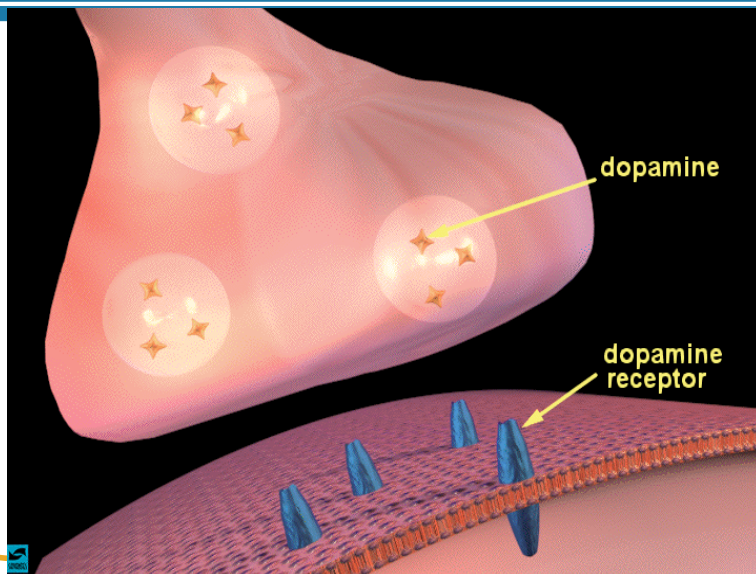
## Methamphetamine: Psychiatric consequences

- Paranoid reactions
- Protracted memory impairment
- Depressive/dysthymic reactions
- Hallucinations
- Psychotic reactions
- Panic disorders
- Rapid addiction

## Stimulant withdrawal symptoms

- Depression
- Difficulty concentrating
- Increased need for sleep / insomnia
- Memory dysfunction
- Anxiety
- Decreased sex drive
- Low energy
- Irritability
- Headache
- Craving

## Synaptic activity



### Notes

Amphetamines (including methamphetamine) are synthetic substances structurally related to naturally occurring adrenaline and ephedrine. Amphetamines activate the central nervous system (CNS) and sympathetic nervous system (SNS), increasing synaptic concentrations of excitatory neurotransmitters and/or inhibiting their reuptake. The monoamines commonly affected by amphetamines are:

- dopamine
- noradrenaline
- serotonin

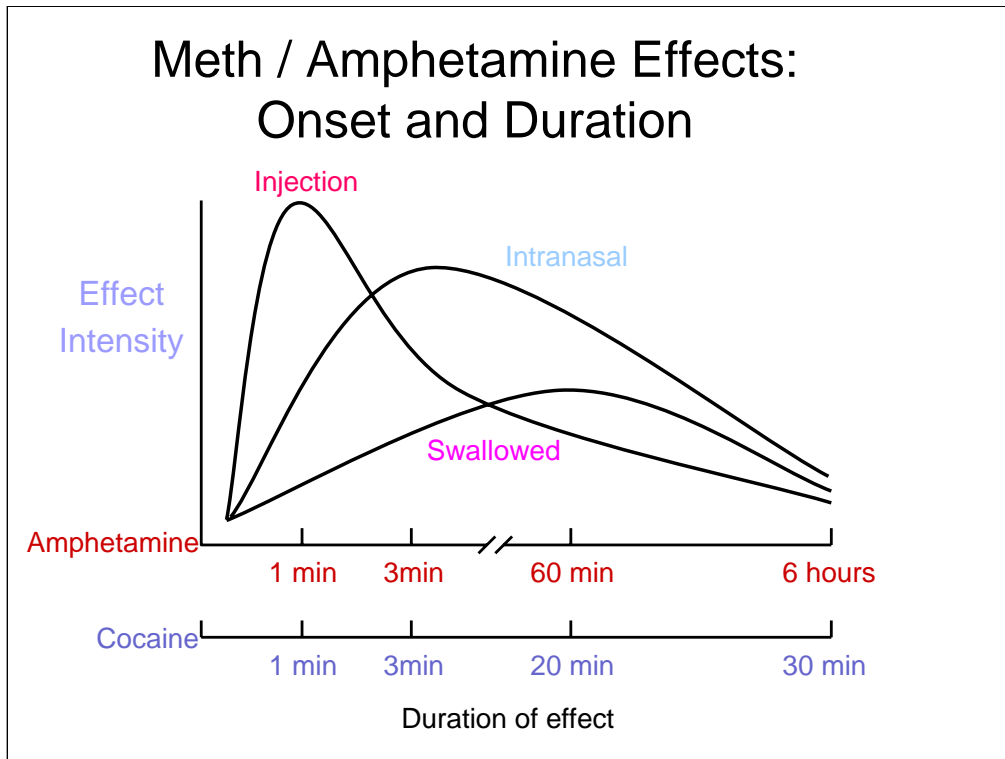
Through stimulating neurotransmitter release and preventing reuptake, amphetamine use results in:

- *CNS effects*: euphoria; increased sense of wellbeing, confidence and physical activity; improved cognitive and physical performance; suppression of appetite and a decreased need for sleep.
- *SNS effects*: increased blood pressure, tachycardia or reflex bradycardia, increased core temperature.

Sources: Latt, N., White, J., McLean, S., Lenton, S., Young, R. & Saunders, J. 2002, 'Central Nervous System Stimulants', in Hulse, G., White, J. & Cape, G. (eds.) 2002, *Management of Alcohol and Drug Problems*, ch. 8, Oxford University Press, South Melbourne, pp. 124–140.

Victoria Police 2002, *Custodial Drug Guide: Medical Management of People in Custody with Alcohol and Drug Problems*, Custodial Medicine Unit, Victoria Police, Mornington, Victoria.

Diagram Source: [www.nida.nih.gov](http://www.nida.nih.gov)



#### Notes

The graph shows psychostimulant effects according to drug type (amphetamine and cocaine) and route of administration. Although the effect is similar across drug types, the duration of effect varies.

*Injecting* has the strongest, most immediate effect as it is delivered to the brain within seconds of administration. It is also considered the most cost-effective method (in terms of intensity and speed of onset), although its duration of effect tends to be shorter than other methods.

Injecting has the highest risk of immediate harm, with risks related to:

- extreme intoxication (overdose, psychosis)
- physical effects (neurological, cardiac)
- emotional wellbeing (risk-taking behaviour, injecting, overconfidence, mood swings)

*Smoking* has a similar onset and duration of effect as injecting. Both cocaine and methamphetamine can be smoked.

*Intranasal use/snorting* has a weaker, but longer lasting effect.

*Swallowing* requires the drug to be metabolised by the liver and GI system before delivery to other major organs. Hence has the longest and weakest effect.

Swallowing has the lowest risk of harm because of slower onset of action but it does last the longest. It is important when swallowing to wait for the full effect of the first dose prior to having additional doses, otherwise the risk of overdose is increased.

Source: adapted from Peard, J., Lintzeris, N., & Churchill, A. 1996, *From Go To Whoa: Amphetamines and Analogues, The Trainer's Package for Health Professionals*, Commonwealth Department of Health and Human Services, AGPS, Canberra, p. 37.

# Amphetamine Effects

Mild	Moderate	Toxic
<p>Feel good Alert Energy Confidence</p>	<p>Feel great Increased libido Increased stamina No need for sleep</p>	<p>Extreme agitation Incoherence Increased temperature Dehydration Thought disorder Violent aggression Stroke Heart attack</p>
<p>Sleeplessness Reduced appetite Dry mouth</p>	<p>Crash Suspicion Headache Teeth grinding Anxiety</p>	

## Notes

The figure shows the expanded array of negative effects from amphetamines as frequency of use increases.

Effects often perceived as negative by health professionals may not necessarily be perceived that way by someone used to experiencing and managing those effects. For example, teeth grinding, dry mouth, or headache may be perceived by the person using “speed” as a “manageable irritation.” Other effects (such as effects on sleep or appetite) may be desired. Increased alertness and mood are generally sought-after effects readily gained from mild doses.

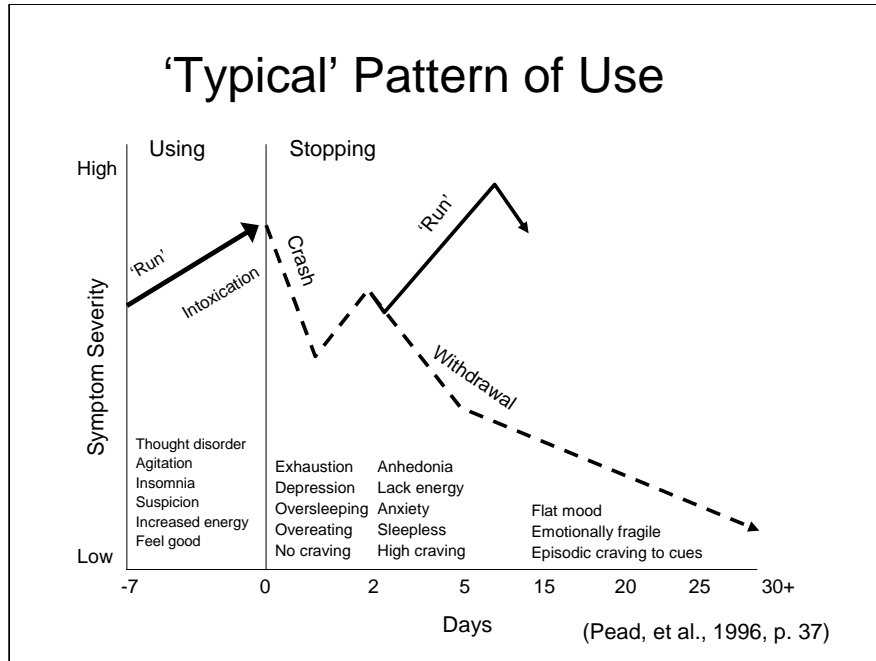
Moderate effects include increased confidence, libido or stamina. However, the trade-off may be an inability to perform or a “hangover-like crash” the following day.

Although higher doses may be associated with elevated mood, increasingly negative effects occur at high doses, e.g., agitation, delusions or markedly increased body temperature.

Recent references to “speed” are most likely to refer to methamphetamine rather than the formerly more common amphetamine sulphate.

Sources: adapted from Peard, J., Lintzeris, N., & Churchill, A. 1996, *From Go To Whoa: Amphetamines and Analogues, The Trainer's Package for Health Professionals*, Commonwealth Department of Health and Human Services, AGPS, Canberra, p. 26.

Latt, N., White, J., McLean, S., Lenton, S., Young, R. & Saunders, J. 2002, ‘Central Nervous System Stimulants’, in Hulse, G., White, J. & Cape, G. (eds.) 2002, *Management of Alcohol and Drug Problems*, ch. 8, Oxford University Press, South Melbourne, pp. 124–140.



## Notes

Effective GP interventions require the identification of drug type and patterns of use (level and frequency).

The pattern shown above is relatively common.

**A “run” or binge** may last a few days to a week before the negative effects (e.g., sleeplessness, agitation, anxiety, delusions, hallucinations, tolerance, exhaustion from lack of sleep and food) outweigh the positive effects (e.g., alertness, raised mood). Some patients may seek or use other depressant drugs (e.g., alcohol or benzodiazepines) to reduce the effects of intoxication or to manage the after-effects of use. Others may experience an acute paranoid state following repeated high dose use. Methamphetamine may be used regularly (up to 3 times a day) for months at a time.

**The “crash”/amphetamine hangover** may occur even after a single-occasion of use, or on cessation of use, and may last one to two days. Main features include restless oversleeping, depressed mood, exhaustion, overeating, lethargy and absence of desire for amphetamines. Concurrent hazardous or harmful patterns of alcohol use may result in an alcohol hangover.

*Withdrawal* follows cessation of regular, sustained use. This is generally a phase of considerable discomfort, commencing two to three days after cessation of use and lasting weeks to months. General effects include flat mood, sleeplessness, craving, agitation, aggression and possible recurrence of delusional thoughts and hallucinations.

*Prolonged withdrawal* is frequently avoided by many users, who choose to return to speed use rather than experience unwanted effects. A prolonged episode of withdrawal is commonly described, whereby emotional unresponsiveness to pleasant events (anhedonia), or episodic craving may continue.

...continued from previous page.

### **Mental health**

For example, hyperactive, emotional lability, psychosis/paranoia, evidence of depression and suicidal ideation.

### **Laboratory investigations**

Urine drug screen may be valuable. Detects recent use (metabolites of speed in urine for up to 48–72 hours, cocaine metabolites for 24–36 hours ). In addition to routine Ix, CPK, cardiac enzymes, serum troponin concentrations if appropriate.

### **Other**

Disinhibition and poor decision-making may lead to risk-taking behaviour, e.g., assess for consequences of unsafe sex, injecting etc.

Sources: adapted from Pead. J., Lintzeris, N., & Churchill, A. 1996, *From Go To Whoa: Amphetamines and Analogues, The Trainer's Package for Health Professionals*, Commonwealth Department of Health and Human Services, AGPS, Canberra.

Latt, N., White, J., McLean, S., Lenton, S., Young, R. & Saunders, J. 2002, 'Central Nervous System Stimulants', in Hulse, G., White, J. & Cape, G. (eds.) 2002, *Management of Alcohol and Drug Problems*, ch. 8, Oxford University Press, South Melbourne, pp. 124–140.



## Management of toxic reactions

### Priorities are:

- maintain airway, circulation, breathing
- control elevated body temperature (hydration, cold water, ice)
- control seizures (IV diazepam)
- manage psychotic symptoms (antipsychotics)
- reassurance, support, comfort, minimal stimulation

Treatment depends on patient's condition on presentation.

### Notes

Refer to NCETA 2004 'Cocaine'. *Alcohol and Other Drugs, A Handbook for Health Professionals*, Chap. 8, Commonwealth Department of Health & Ageing, Canberra, for further information.

## Activity: Case study

Rory, a 24-year-old student, presents with persistent headache, lethargy, and unexplained weight loss. He is “burning the candle at both ends,” working in a bar and studying, and states that “life is pretty hectic” at present. Speed helps him get things done.

*Describe a brief intervention for Rory.*

# Psychostimulant Withdrawal

Crash (Days 1–3)	Peak symptoms (Days 2–10)	Residual symptoms (from 1–8 weeks)
<ul style="list-style-type: none"> <li>• exhaustion</li> <li>• depression</li> <li>• oversleeping</li> <li>• no cravings</li> </ul>	<ul style="list-style-type: none"> <li>• dysphoria</li> <li>• lack energy</li> <li>• increased appetite</li> <li>• generalised aches and pains</li> <li>• re-emergence of mild psychotic features, including: misperceptions paranoid ideation hallucinations anxiety.</li> <li>• sleeplessness</li> <li>• high craving</li> </ul>	<ul style="list-style-type: none"> <li>• episodic craving</li> <li>• insomnia</li> </ul> <p>Fluctuating:</p> <ul style="list-style-type: none"> <li>• irritability</li> <li>• agitation</li> <li>• restlessness</li> <li>• dysphoria</li> <li>• lethargy</li> <li>• amotivation</li> </ul>

From Pead et al. (1996, p. 84)

Source: adapted from Pead, J., Lintzeris, N., & Churchill, A. 1996, *From Go To Whoa: Amphetamines and Analogues, The Trainer's Package for Health Professionals*, Commonwealth Department of Health and Human Services, AGPS, Canberra, p. 84.

# Withdrawal treatment

## Immediate withdrawal treatment

- setting (outpatient or inpatient)
- supportive environment, information, and reassurance
- provide ongoing monitoring
- plan long-term management strategies

## Planning for prolonged withdrawal

- anticipate it will be prolonged (i.e., affecting sleep, mood, cravings)
- plan for lapse and relapse

### Notes

#### Immediate withdrawal treatment

Assess suitability of home versus inpatient withdrawal according to support available in the home, medical and psychological functioning and psychological and emotional stability. Ideally, home withdrawal should take place in a supportive, quiet place where emotional and physical support can be provided by a trusted person who is not currently using drugs

Psychosocial supportive care, information, and reassurance should be provided by:

- qualified health professional/s (e.g., outpatient support, GP, home withdrawal support nurse, social worker, case management/shared care strategy) to assist the patient to develop psychosocial strategies for coping with relapse and lapses, for managing mood and sleep disturbances and to assist with managing anger or irritation over subsequent months
- psychosocial support includes managing cravings, sleeping patterns, anhedonia, emotional lability
- 24-hour help line (ADIS)

Inpatient treatment is the most appropriate option when a person is experiencing medical or psychiatric complications as a result of use of amphetamines. Residential or inpatient care may be indicated when supportive environments are unavailable. Long-term residential programs may demand that patients attend services “drug free” including being free from prescribed medications. Hence medications for ongoing management of depression or relapse, for example, may not be acceptable. Check with the program in the first instance.

Source: adapted from Peard, J., Lintzeris, N., & Churchill, A. 1996, *From Go To Whoa: Amphetamines and Analogues, The Trainer's Package for Health Professionals*, Commonwealth Department of Health and Human Services, AGPS, Canberra.

## Pharmacotherapies for psychostimulant withdrawal

- Aim to decrease discomfort
- Benzodiazepines
  - assist sleep or reduce anxiety and agitation
  - avoid long-term prescribing
- Antipsychotics and Antidepressants
  - available research shows limited efficacy



### Notes

Pharmacotherapies (general aim is to reduce discomfort):

#### Benzodiazepines

- sleeplessness and anxiety may be prolonged during withdrawal; prepare patient for this. Avoid long term prescribing of benzodiazepines to prevent benzodiazepine dependence.

#### Dopamine agonists

- have only been studied in relation to cocaine, with mixed reviews of their effect on cravings and withdrawal.

#### Antidepressants

- tricyclic antidepressants are generally not helpful and are not recommended unless there is a prior diagnosed affective condition, or on advice from the patient's psychiatrist. SSRIs may be helpful. Symptoms such as tearfulness and suicidal thoughts often resolve within a week of withdrawal.

#### Antipsychotics

- have only been studied in relation to cocaine as an anticraving agent, with limited effect.
- Haloperidol may be indicated for anxiety and distress resulting from features of psychosis. Amphetamine-induced symptoms of psychosis tend to resolve after about 7 days. Should mental health problems persist, refer for specialist psychiatric assessment.

Pharmacotherapies indicated if intoxicated, or during withdrawal:

- if severely agitated (benzodiazepine, e.g., Diazepam).
- if experiencing symptoms of psychosis (antipsychotic, e.g., Haloperidol).

Source: adapted from Peard, J., Lintzeris, N., & Churchill, A. 1996, *From Go To Whoa: Amphetamines and Analogues, The Trainer's Package for Health Professionals*, Commonwealth Department of Health and Human Services, AGPS, Canberra.

## Promising pharmacotherapies?

- Newton, T. et al (Biological Psychiatry, Dec, 2005)  
**Bupropion** reduces craving and reinforcing effects of methamphetamine in a laboratory self-administration study.
- Elkashef, A. et al (Neuropsychopharmacology, 2007)  
**Bupropion** reduces meth use in an outpatient trial, with particularly strong effect with less severe users.
- Tiihonen, J. et al (recently completed; reported at the ACNP methamphetamine satellite meeting in Kona, Hawaii) **Methylphenidate SR** (sustained release) has shown promise in a recent Finnish study with very heavy amphetamine injectors.

## Low threshold treatment services for MSM methamphetamine users

- Street outreach and field workers in clubs and bath houses
- Needle exchange
- Drop-in centres for food, medical services
- Housing for homeless methamphetamine users
- HIV risk reduction groups employing peer and professional counselling
- No empirical evidence at this point

### Notes

“MSM” is the acronym for “men who have sex with men.”

## Activity: Case study

Kylie, a 33-year-old lawyer, recently discovered she was pregnant. She has an active work and social life, and consequently, tends to eat poorly. The pregnancy was unplanned. She is concerned about the health of her baby and her lifestyle that precludes regular eating habits.

*How would you incorporate an AOD history into your consultation?*

*What triggers may lead you to suspect psychostimulant use?*



# Cocaine

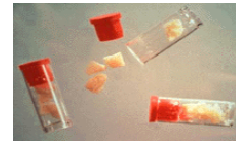
- Alkaloid from plant leaf of *Erythroxylon coca*
- Known as *coke*, *charlie*, *snow*, *okey doke*
- Sold in '*lines*'
- Central nervous system stimulant with local anaesthetic actions
- Also stimulates the sympathetic nervous system
- Blocks reuptake of dopamine, noradrenaline, and serotonin



Cocaine



Crack



Crack in vials

## Notes

The plant *Erythroxylon coca*, chewed by South American Indians for its stimulant effects, is primarily available in Peru and Bolivia.

Cocaine is extracted from the coca leaf and exported in the form of a salt, *cocaine hydrochloride*. The salt is a white, odourless, crystalline powder with a bitter taste.

Cocaine base is extracted from the powder to form rocks or crystals known as "crack" or "freebase." When smoked, the subjective effects are almost immediate.

Blockade of neurotransmitter reuptake results in increased concentration at post-synaptic receptor sites.

Dopamine is thought to be responsible for the reinforcing effects.

Sources: Latt, N., White, J., McLean, S., Lenton, S., Young, R. & Saunders, J. 2002, 'Central Nervous System Stimulants', in Hulse, G., White, J. & Cape, G. (eds.) 2002, *Management of Alcohol and Drug Problems*, ch. 8, Oxford University Press, South Melbourne, pp. 124–140.

Victoria Police 2002, *Custodial Drug Guide: Medical Management of People in Custody with Alcohol and Drug Problems*, Custodial Medicine Unit, Victoria Police, Mornington, Victoria.

Picture source: The DEA. <http://www.usdoj.gov/dea/index.htm>

## Cocaine: Metabolism

- Rapid onset of action (2–8 minutes respectively)
- Peak blood levels occur in 5–30 minutes
- Action is brief:
  - half-life of 15–30 minutes if injected
  - half-life of up to 30 minutes if snorted
- Metabolised by liver, 1%–2% excreted unchanged in urine
- Inactive metabolites can be detected in:
  - blood or urine for 24–36 hours after use
  - hair for weeks to months after use

## Cocaine: Acute and chronic effects

Very similar to those associated with methamphetamine. Since the half-life of cocaine is much shorter, in comparison to methamphetamine there is:

- Somewhat less severe neurotoxicity
- Somewhat lower frequency of drug-induced psychosis
- Somewhat shorter protracted withdrawal symptoms

## Cocaine: Symptoms of withdrawal

- Dysphoria (rather than depression), which may persist (up to 10 weeks). Plus at least two of:
  - fatigue
  - insomnia / hypersomnia
  - psychomotor agitation
  - craving
  - increased appetite
  - vivid unpleasant dreams
- Withdrawal tends to peak 2–4 days following cessation of use.

## Cocaine pharmacotherapy



- Disulfiram has been shown to reduce cocaine use significantly in non-alcohol using cocaine-dependent individuals. However, further research is needed.
- There is substantial use of other medications for “treating” short- and long-term effects of cocaine use. However, controlled research shows no evidence to support use of these medications.

## Cocaine: Withdrawal management

- Non-stimulating / non-threatening environment
- Possible suicide precautions
- To date, no effective pharmacotherapies for withdrawal management
- Prescribed medications:
  - Short-term use of benzodiazepines (anxiety, agitation, promote sleep)

## Psychostimulant interventions

- Be non-judgemental, do not insist on abstinence
- Engage and retain patient in treatment
- Understand patient's treatment goals
- Tailor intervention to suit patient, including level and intensity of referrals
- Offer flexible service delivery, consistent with a patient's changing goals and needs
- Provide psychosocial support
- Address concurrent mental health needs, e.g., anxiety, bipolar, or attention deficit disorders are common with cocaine use.

## Treatments for stimulant-use disorders with empirical support

- **Cognitive-Behavioral Therapy (CBT)**
- **Community Reinforcement Approach**
- **Contingency Management**
- **12-Step Facilitation**
- **Brief Cognitive Behavioral Therapy**
- **Matrix Model**

All have demonstrated efficacy for the treatment of cocaine and / or methamphetamine dependence.



## Volatile Substances



## Volatile substances



- Commonly referred to as 'inhalants', 'solvents', 'solvent based products'
- Common terms include 'chroming,' 'huffing,' 'sniffing,' 'bagging'
- Comprise a group of chemical compounds that change from a liquid or semi-solid to gaseous state when exposed to air
- Inhalation of the vapour through the mouth or nose produces a psychoactive effect (intoxication and euphoria).

### Notes

The term "volatile substance" has been used in this topic as it is a term that is inclusive and applies to a wide range of substances that are inhaled. Other terms used include glues, solvents, inhalants or "chroming," which do not describe the breadth of substances used in this fashion.

See also Volatile Substances handouts.

## What substances are used?

- Inhalants are found in hundreds of products at supermarkets, newsagencies, hardware stores, and industrial sites
- 4 categories of inhalants:
  - Solvents
  - Aerosols
  - Gases
  - Nitrites



### Notes

Four categories of inhalants:

1. Volatile solvents – toluene and xylene are the common compounds found in a multitude of inexpensive, accessible products used for common household and industrial purposes. These include paint thinners and removers, dry-cleaning fluids, degreasers, petrol, glues, contact adhesives, plastic cement, correction fluids and felt-tip markers.
2. Aerosols – pressurised aerosols can contain halons and freons (fluorocarbon propellants). Increasingly butane may be used to protect the ozone layer. Products include spray paints, deodorant and hairsprays, insect sprays, vegetable oil sprays for cooking and fabric protector spray.
3. Gases – medical anaesthetic gases include ether, chloroform, halothane and nitrous oxide (“laughing gas”). Household gases can include commercial products containing gas fuels such as butane cigarette lighters, bottled domestic gas and cylinder propane gas.
4. Nitrites – act primarily to dilate blood vessels and relax the muscles rather than acting directly on the central nervous system. They include amyl nitrite and butyl nitrite. Primarily used as sexual enhancers.

## Pharmacology

- High lipid solubility promotes rapid absorption from the lungs
- Acute intoxication occurs after 3–5 minutes (10–15 breaths are sufficient)
- Peak plasma concentration reached in 15–30 minutes
- Half-life varies from hours to days
- Metabolised in kidneys and liver
- Accumulate in lipid rich organs (i.e., liver, brain)
- Crosses placental barrier

### Notes

Fat solubility results in ready absorption from the blood into high fat tissues, including nerve cells. This action results in generalised reduction of nerve membrane functioning, which causes CNS depression.

CNS damage identified in long-term, chronic users includes:

- damage to white matter
- cortical atrophy
- cerebellar damage
- peripheral neuropathy
- optic atrophy
- hearing loss

Toluene is the most harmful volatile substance.

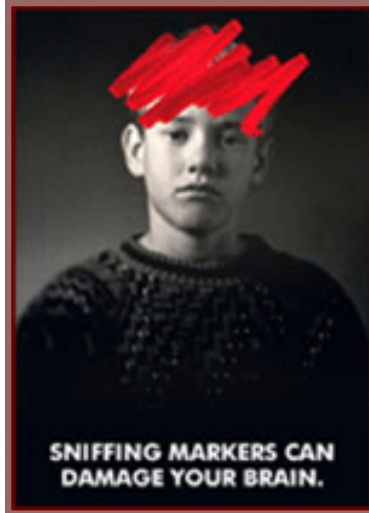
### Maternal and neonatal concerns/issues

Fat solubility results in crossing of placental barrier. Fetal toluene exposure is associated with:

- oral cleft and microcephaly
- spontaneous abortion
- fetal growth retardation
- low birth weight
- prematurity
- developmental delays.

See: NCETA 2004, *Alcohol and Other Drugs, A Handbook for Health Professionals*, 3rd edn., Commonwealth Department of Health & Ageing, Canberra.

Highest prevalence among  
14- to 17-year-olds



## Appeal of volatile substances

- Inexpensive
- Readily available despite legislation precluding sale to minors
- Can be packaged in small, discrete containers
- Create both rapid intoxication and rapid resolution of intoxication (can use and still return home sober)

## Who uses inhalants?

### **Lack of good epidemiological data, however data from Australia indicates:**

- highest prevalence among 14- to 17-year-olds (c.f., older adults)
- a small percentage try, but most cease use after a few attempts
- primarily a short-term, experimental activity by young males (however, female use is increasing)
- recreational users tend to combine solvents and cannabis with ecstasy, speed, or LSD
- not restricted to Indigenous communities, but Indigenous youth (compared with non-Indigenous) tend to:
  - show greater habitual use
  - use more frequently
  - use over a longer period
- use of solvents is of international concern

### **Notes**

Volatile substance users are mostly young teenagers. Occasionally there are reports of use by young children (6–12 yrs) and older individuals (i.e., over 30 years). Greatest trend is for teens over 12 years.

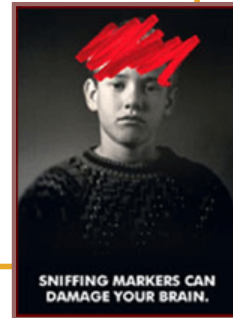
While predominantly an activity undertaken by young males, there is a trend towards increased use by young females.

Sources: Goldfields South East Health Region 2002, *Management of Inhalants in the Goldfields – Industry Information Package*, Kalgoorlie, WA Country Health Service, Government of Western Australia.

Victoria Police 2002, *Custodial Drug Guide: Medical Management of People in Custody with Alcohol and Drug Problems*, Custodial Medicine Unit, Victoria Police, Mornington, Victoria.

## Why do youth use volatile substances?

- *“Because it’s fun and exciting”*
- *“I like the way it makes me feel – I feel drunk”*
- *“It takes away my bad feelings”*
- *“I wanted to be part of the gang”*
- *“My brothers were doing it so I wanted to try it”*
- *“Because I want to do something my parents don’t like”*
- *“Because it’s easy to get and I’m not allowed to get alcohol”*



### Notes

These quotes are from Australian Indigenous youth about their reasons for starting to use volatile solvents.

Source: ADAC 2000, *Petrol Sniffing and Other Solvents*, Booklets 4, ADAC, South Australia, [www.adac.org.au](http://www.adac.org.au)



## Patterns and methods of use

### 3 major patterns of use:

- experimental / occasional
- social
- long-term dependent / chronic

### Methods of use:

- sniffing
- huffing
- bagging

### Notes

Sniffing or inhaling from a container gives the lowest vapour concentration - much vapour dissipates into the air.

Huffing refers to saturating material (e.g., sleeve cuff, handkerchief, collar, lapel, rag) which is held against nose or mouth or sometimes in the mouth. The practice is designed to be unobtrusive.

Bagging involves inhaling vapours from a plastic or paper bag, which is held over the mouth or nose. Bags are alternately collapsed and inflated to obtain greatest vapour concentration. Some people place a larger plastic bag over the head to prevent further vapour loss. A particularly dangerous practice – to be strongly discouraged.

Sources: NCETA 2004, 'Volatile Substances', *Alcohol and Other Drugs, A Handbook for Health Professionals*, 3rd edn., ch. 10, Commonwealth Department of Health & Ageing, Canberra.

Victoria Police 2002, *Custodial Drug Guide: Medical Management of People in Custody with Alcohol and Drug Problems*, Custodial Medicine Unit, Victoria Police, Mornington, Victoria.

## Cues for detecting recent use



- Red, watery eyes
- Sneezing & coughing (URTI-like symptoms)
- Chemical smell or odour on breath
- Glue, solvent, or paint stains on clothing, fingers, nose, or mouth
- Apparent intoxication / altered behaviour / risk taking
- Incoherence, confusion
- Poor coordination
- Excessive sweating
- Unusual spots, marks, rashes and sores around nose and mouth
- Excessive nasal secretions, constantly sniffing

### Notes

As with other psychoactive substances, a list of signs that may suggest drug use should be viewed with extreme care, to prevent otherwise ordinary reactions to situations (distress, lack of sleep, challenging behaviour, etc.) being misinterpreted by health workers or parents.

While some signs may appear obvious (smell of petrol or paint on clothes, nasal sores, chemical smell on breath etc.), other signs (such as tiredness, anxiety, irritability, poor school performance) may be the result of a range of other problems. Creating distrust between either parents or health workers will disrupt relationships and prevent discussion of a range of issues or concerns that may or may not be associated with the use of psychoactive drugs.

GPs are advised to establish good communication and rapport with young persons and parents where volatile use is suspected.

Source: ADAC 2000, *Petrol Sniffing and Other Solvents*, Booklet 4, ADAC, South Australia, [www.adac.org.au](http://www.adac.org.au).

## Volatile effects – short term

### Desired effects

- Euphoria
- Excitation
- Exhilaration
- Sense of invulnerability
- Disinhibition

### Negative acute/ short-term effects

- Drowsiness
- 'Flu-like' symptoms
- Nausea and vomiting
- Headaches
- Diarrhoea, abdominal pain
- Unpleasant breath
- Nosebleeds and sores
- Reckless behaviour

### Notes

For most users, most effects are achieved after a few breaths and occur within an hour after inhaling.

Hangovers and headaches may occur after the immediate effects have passed, lasting several days. Hangovers from inhaling are generally less severe and less common than those caused by alcohol.

Larger quantities may result in disorientation and lack of coordination, visual distortions, and possibly blacking out.

Tolerance rapidly develops, within several days, and the person requires increased amounts to achieve the desired effect.

Source: Goldfields South East Health Region 2002, *Management of Inhalants in the Goldfields – Industry Information Package*, Kalgoorlie, WA Country Health Service, Government of Western Australia.

See: NCETA 2004, *Alcohol and Other Drug: A Handbook for Health Professionals*, Australian Government Department of Health & Ageing, Canberra.

## Volatile effects – high doses

### Effects at high doses

- Slurred speech
- Poor coordination
- Disorientation, confusion
- Tremor
- Headaches
- Delusions
- Visual distortions or hallucinations
- Unpredictable behaviour, *then*:
  - ataxia
  - stupor
  - final stages (seizures, coma cardiopulmonary arrest, death)

## Volatiles - overdoses

### **High Doses** put user at risk for:

- Convulsions, seizures, coma
- Respiratory depression
- Cardiac arrhythmias

### **Injury or death** can occur from:

- Risk-taking behavior (drowning, falls, etc.)
- Suffocation
- Aspiration of vomit
- Burns, explosions
- Poisoning, organ failure (chronic use)
- Laryngeal spasm (Butane), respiratory arrest
- Lead poisoning (gasoline / petrol)

## Tolerance and dependence

- Tolerance develops rapidly with regular use
- Psychological and physical dependence, while rare, may also occur

### Notes

Tolerance develops rapidly with regular use.

Dependence is rare compared with the number of people who have tried inhaling volatile substances, although psychological dependence may occur when using inhalants takes precedence over other activities and responsibilities.

There is some evidence that a withdrawal syndrome may be experienced among long-term regular users on abrupt cessation. Symptoms include anxiety, depression, agitation, loss of appetite, irritation, aggressive behaviour, dizziness, tremor, nausea and vomiting, headache and abdominal cramps.

Source: Victoria Police 2002, *Custodial Drug Guide: Medical Management of People in Custody with Alcohol and Drug Problems*, Custodial Medicine Unit, Victoria Police, Mornington, Victoria.

# Withdrawal

- **Onset and duration**
  - not classified in DSM IV but features of possible “withdrawal syndrome” may commence 24-48 hours after cessation of use
- **Withdrawal Symptoms**
  - sleep disturbances
  - tremor
  - irritability and depression
  - nausea
  - diaphoresis
  - fleeting illusions
- **Treatment**
  - symptomatic

## Notes

Some users report withdrawal symptoms (as shown on the slide). Medical intervention is rarely required although some individuals may benefit from symptomatic treatment and treatment for sores or irritant effect of vapour around eyes, nose and mouth.

Withdrawal may occur 24–48 hours after cessation of use, and last for 2–5 days.

Source: Victoria Police 2002, *Custodial Drug Guide: Medical Management of People in Custody with Alcohol and Drug Problems*, Custodial Medicine Unit, Victoria Police, Mornington, Victoria, p. 2-28.

## Problems with long-term use

Patients may present with a variety of symptoms as a consequence of long-term use, including:

- chronic headache
- sinusitis, nosebleeds, increased nasal secretions
- diminished cognitive function
- ataxia
- chronic coughing
- chest pain or angina
- tinnitus
- extreme tiredness, weakness, dizziness
- depression / anxiety
- shortness of breath
- indigestion
- stomach ulcers



## Complications from long-term use

### CNS complications

- acute encephalopathy
- chronic neurological deficits
- memory, thinking
- hearing loss, and loss of sense of smell
- nystagmus
- motor impairment, especially secondary to lead poisoning
- peripheral nerve damage

### Other systems

- *Renal* – nephrolithiasis, glomerulopathies
- *Hepatic* – reversible hepatotoxicity
- *Pulmonary* – e.g., pulmonary hypertension, acute respiratory distress
- *Cardiovascular* – e.g., VF, arrhythmias, acute cardiomyopathy
- *Haematological* – e.g., blood dyscrasias

### Notes

Lead poisoning is less commonly encountered since the introduction of unleaded fuel.

Source: Victoria Police 2002, *Custodial Drug Guide: Medical Management of People in Custody with Alcohol and Drug Problems*, Custodial Medicine Unit, Victoria Police, Mornington, Victoria, p. 1-35.

## Impact

Use of volatile substances (as with use of other psychoactive drugs) impacts not only personal health but also:

- families
- workplace safety
- community (e.g., anti-social behaviour)

## Responding to intoxication

- Ensure fresh air
- Be calm, and calming
- Don't chase, argue, use force
- Persuade to cease sniffing (if able to understand)
- Take person to a safe environment
- Don't attempt to counsel while intoxicated
- Follow-up with parents
- If drowsy or heavily intoxicated
  - consider the best environment for the individual and monitor physical and mental health

### Notes

Response will depend on the person's situation, your relationship with that person, and whether the person is alone or in a group.

If uncertain what to do, seek advice from:

- major hospital Emergency Department
- Poison Information Service.

Source: ADAC 2000, *Petrol Sniffing and Other Solvents*, Booklet 4, ADAC, South Australia, [www.adac.org.au](http://www.adac.org.au).

## Interventions

- Brief intervention
- Harm reduction
- Counselling
- Group counselling
  - Family support and counselling
  - Be involved in developing community responses (e.g., Drug Action Teams)

*Avoid lectures to school / youth groups – evidence suggests it may increase curiosity and level of use.*

### Notes

Brief Intervention includes providing education to individuals who are currently using and encouragement of involvement in other activities.

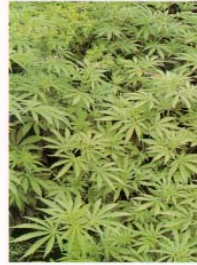
A cautionary note: provision of information/education to non-users can lead to uptake.

- Harm Reduction responses include information regarding:
  - safer use (changing from harmful to less harmful products, safer methods of use)
  - monitoring use
  - safety while using (compare risks of flammable versus non-flammable products, environment)
  - development of tolerance
  - reduction of acute and chronic tissue toxicity and damage
  - non-use of plastic bags (bags can cause asphyxia)
- Counselling may be valuable, but insure context of use and family situation is assessed
- Group counselling may be useful if part of group activity
- Family support and counselling. Family involvement is especially important for Indigenous families
- Community involvement by GPs.

Sources: ADAC 2000, *Petrol Sniffing and Other Solvents*, Booklet 4, ADAC, South Australia, [www.adac.org.au](http://www.adac.org.au).

NCETA 2004, *Alcohol and Other Drugs: A Handbook for Health Professionals*, Australian Government Department of Health & Ageing, Canberra.

# Cannabanoids



Marijuana

Hashish

# Cannabis



- The most widely used illicit drug
- The drug most likely to be seen in general medical practise
- Generally an experimental or recreational drug, but the most common illicit drug of dependence
- Use is common among polydrug users
- 70% of all drug-related offences relate to cannabis

THC or delta-9-tetrahydrocannabinol is the active ingredient of cannabis

## Notes

This slide set contains introductory information about the forms and routes of cannabis administration, prevalence of use, its properties, detection, peak effect and effects of use (acute and long-term).

Sources: Hulse, G., White, J. & Cape, G. 2002, *Management of Alcohol and Drug Problems*, Oxford University Press, South Melbourne, pp. 141–157.

Victoria Police 2002, *Custodial Drug Guide: Medical Management of People in Custody with Alcohol and Drug Problems*, Custodial Medicine Unit, Victoria Police, Mornington, Victoria.

## Case study

Mark is a 23-year-old unemployed labourer who presents ostensibly with fatigue. On examination, some psychotic symptoms are apparent.

Upon questioning, he says he has been smoking 30 cones of cannabis a day.

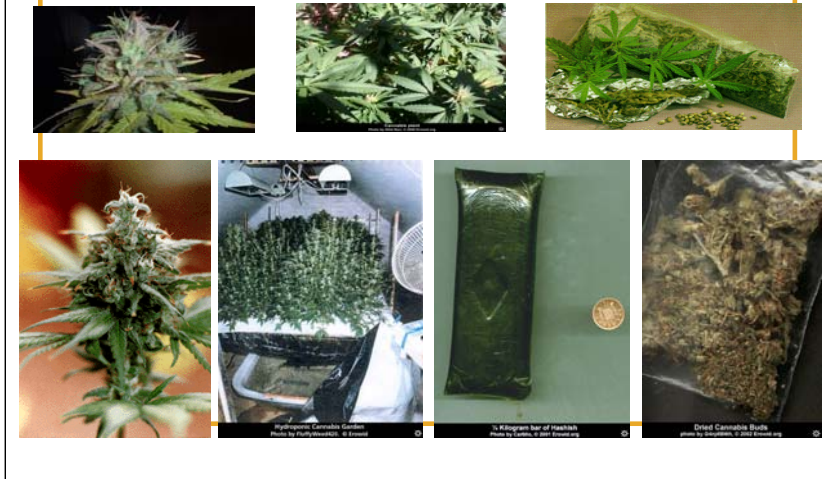
He is restless, with significant mood swings, racing thoughts and paranoia but no real features of lasting psychosis.

*Is his presentation consistent with his drug use?*

*How long are his symptoms likely to last?*

*What advice might you give him regarding future use?*

## Cannabis: Forms



### Notes

- A) Cannabis Head/Flower/Bud
- B) Cannabis Plant
- C) Cannabis Leaves & Dried Cannabis
- D) Cannabis Head/Flower/Bud

- E) Hydroponically Grown Cannabis
- F) Hashish Bar
- G) Dried Cannabis Buds (Head or Flower).

- Cannabis plants are annuals, well suited to warm conditions. Male and female forms contain THC, although the flowers of the smaller female plant contain higher concentrations. Hydroponic varieties tend to produce higher concentrations of THC.  
Source: <http://www.erowid.org/index.html>
- Dried flowers/leaves/buds (*marijuana/ganja*): 1–15% THC (depending on genetic and environmental factors)
- Extracted dried resin, sometimes mixed with dried flowers and pressed into a cube (*hashish*): around 10%–20% THC
- Extracted oil using an organic solvent (*hashish oil*): 15–30% THC
- Route of administration can affect dose:
  - smoked (joint, pipe, bong, bucket bong): 50% absorbed, peak concentration 10–30 mins, lasts 2–4 hours
  - ingested (cake, biscuits): 3–6% absorbed, peak concentration 2–3 hours, lasts up to 8 hours

Sources: Todd, F., McLean, S., Krum, H., Martin, J., & Copeland, J. 2002, 'Cannabis', in Hulse, G., White, J. & Cape, G. (eds.) 2002, *Management of Alcohol and Drug Problems*, ch. 9, Oxford University Press, South Melbourne, pp. 141–157.

Victoria Police 2002, *Custodial Drug Guide: Medical Management of People in Custody with Alcohol and Drug Problems*, Custodial Medicine Unit, Victoria Police, Mornington, Victoria.



## Cannabis: Properties

- Frequently, but erroneously, classified as a narcotic, sedative, or hallucinogen. Sits alone within a unique class.
- Degree of effects determined by the THC concentration of specific cannabis material used.
- Major active constituent is THC (delta-9-tetrahydrocannabinol).
  - rapidly absorbed and metabolised when smoked, less so when ingested (1–3 hours for psychoactive effects).
- Attaches to specific cannabinoid receptors (endogenous brain molecule – anandamide).

### Notes

THC is only one of 60 cannabinoids present in the plant *Cannabis sativa*, but it is the one responsible for its psychoactive effects.

The metabolite 11-carboxy-THC remains at high plasma levels for some hours after absorption. This metabolite is inactive, but is what is measured in urine drug screens. Presence of this metabolite merely confirms recent use. Its presence does not indicate patterns of use, dependence, or intoxication.

Blood levels of THC vary and do not necessarily match the effects experienced. The presence of THC in urine does not necessarily indicate recent use.

(Todd et al., 2002, p. 143)

When used orally, the onset of effects is delayed and may vary considerably from those experienced when smoked. To reduce the severity and duration of intoxication, advise those who use oral forms to wait for the effects to commence (up to half an hour or more) before taking another dose.

THC is not water soluble and therefore not suited for use by injection. THC is lipophilic - taken up and stored by body lipids. Slow elimination of metabolites results in detection of THC metabolites in urine for weeks or months after use.

Approximate detection times for:

- one-off use = 2 days
- 3 times per week use = 2 weeks
- daily use = 2–4 weeks
- heavy daily use = 4–6 weeks, for some up to 12 weeks

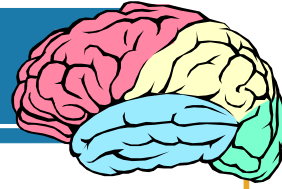
Usual cut-off detection point is 100 nanograms – but lower for defence personnel and prisoners.

Source: Todd, F., McLean, S., Krum, H., Martin, J., & Copeland, J. 2002, 'Cannabis', in Hulse, G., White, J. & Cape, G. (eds.) 2002, *Management of Alcohol and Drug Problems*, ch. 9, Oxford University Press, South Melbourne, pp. 141–157.

Additional information is available at:

<http://www.cdc.gov/mmwr/preview/mmwrhtml/00000138.htm>

## Cannabis: Brain receptors



- Two types of cannabinoid receptors  
CB<sub>1</sub> & CB<sub>2</sub>
  - CB<sub>1</sub> receptors in brain (cortex, hippocampus, basal ganglia, amygdala) and peripheral tissues (testes, endothelial cells)
  - CB<sub>2</sub> receptors associated with the immune system
- Most cannabis effects are via THC acting on CB<sub>1</sub> receptors, which facilitate activity in mesolimbic dopamine neurones

# Cannabis: Forms & routes



## Forms include:

- dried flowers/leaves / buds (marijuana/ganja)
  - 1% – 24% THC (depending on genetic and environmental factors)
- extracted dried resin, sometimes mixed with dried flowers and pressed into a cube (hashish)
  - around 10% – 20% THC
- extracted oil using an organic solvent (hashish oil)
  - 15% – 30% THC

## Route of administration can affect dose:

- smoked (joint, pipe, bong, bucket bong,  $\delta$  dose )
  - 50% absorbed, peak concentration 10 – 30 mins, lasts 2 – 4 hours
- ingested (cake, biscuits)
  - 3% – 6% absorbed, peak concentration 2 – 3 hours, lasts up to 8 hours

## Notes

Joint – a cigarette containing cannabis or cannabis + tobacco.

Pipe – a tube with a “cone”/reservoir at the end. There are many different styles.

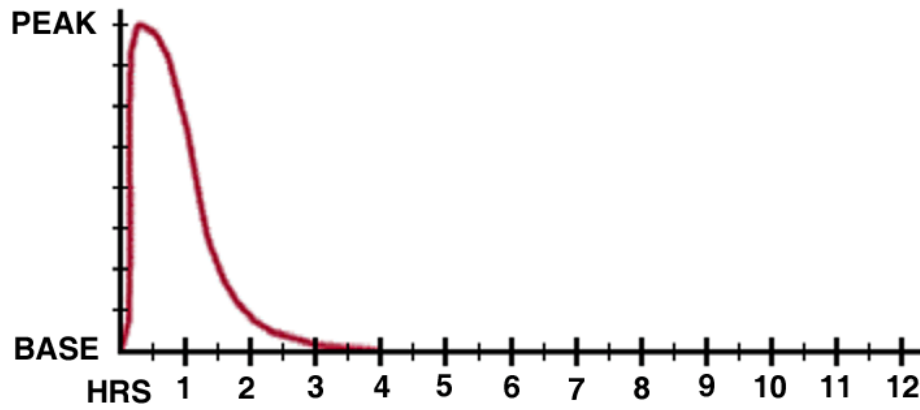
Bong – a “cone” attached to a vessel containing water with a mouthpiece.

Bucket bong – similar to a bong but uses water pressure to force smoke into the lungs.

Source: Todd, F., McLean, S., Krum, H., Martin, J., & Copeland, J. 2002, ‘Cannabis’, in Hulse, G., White, J. & Cape, G. (eds.) 2002, *Management of Alcohol and Drug Problems*, ch. 9, Oxford University Press, South Melbourne, pp. 141–157.

Victoria Police 2002, *Custodial Drug Guide: Medical Management of People in Custody with Alcohol and Drug Problems*, Custodial Medicine Unit, Victoria Police, Mornington, Victoria.

## Cannabis: Time to Peak Effect



Source: [www.erowid.org](http://www.erowid.org)

## Cannabis: Acute effects

- Analgesia
- Euphoria, altered concentration, relaxation, sense of calm or wellbeing, disinhibition, confusion
- Increased appetite, thirst
- Heightened visual, auditory and olfactory perceptions, inability to appropriately interpret surroundings
- Reduced intra-ocular pressure (used for glaucoma treatment)
- Nausea, headaches
- With consistent use, URTIs
- Problems associated with intoxication

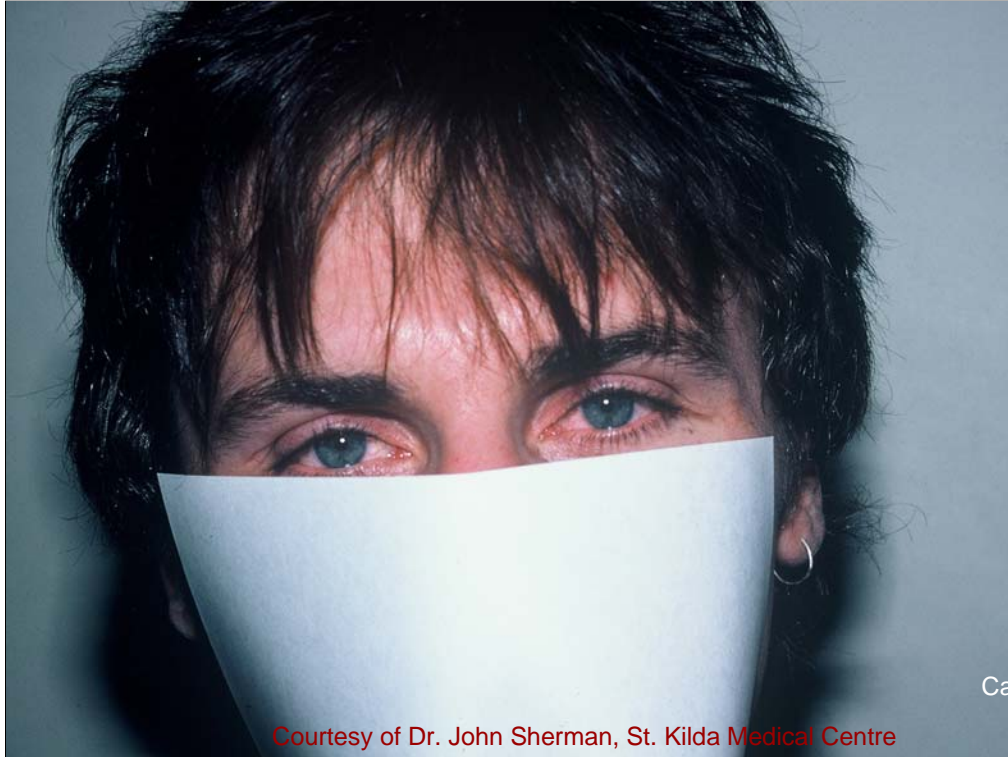
*Cannabis overdose does not result in death.*

### Notes

This list describes some of the possible physiological or psychological effects that may occur as a result of using cannabis. However, the relative severity of these effects or their consequences may vary enormously according to personal makeup, previous use, experiences and expectations, and a range of environmental or other factors.

For GPs, gaining an understanding of the impact, benefits, and harms for the person using psychoactive drugs, rather than focusing on the drug itself, is a key step in gaining credibility and developing a relationship with patients who use psychoactive drugs.

Remember that adverse effects (considered of clinical importance from the perspective of a GP), may have little relevance for the person using the drug.



**Notes**

Reddened Conjunctiva – Cannabis Intoxication

Cannabis use can cause infused vasculature/red eyes.

## Short term, high-dose effects

### Cannabis also affects:

- Short-term memory
- ability to learn and retain new information
- task performance
- balance, stability, mental dexterity
- the cardiovascular and respiratory systems

### Short-term, high-dose use may result in:

- synaesthesia
- pseudo- or true hallucinations
- delusions, feelings of depersonalisation
- paranoia, agitation, panicky feelings, “psychosis”

### Notes

Synaesthesia – melding of one sensory modality with another.

Pseudo-hallucinations are those whereby the person is able to tell that the hallucinations are not real. A true hallucination is where the person is unable to recognise that their hallucinations are not real.

Cardiovascular and respiratory system effects include tachycardia, vasodilation, hypotension, arrhythmias. and bronchodilation.

A short-lived psychotic state associated with a high dose. It usually resolves within a week of abstinence. It may be difficult to distinguish from the precipitation of psychosis in those with a predisposition to mental illness.

Sources: Todd, F., McLean, S., Krum, H., Martin, J., & Copeland, J. 2002, 'Cannabis', in Hulse, G., White, J. & Cape, G. (eds.) 2002, *Management of Alcohol and Drug Problems*, ch. 9, Oxford University Press, South Melbourne, pp. 141–157.

Victoria Police 2002, *Custodial Drug Guide: Medical Management of People in Custody with Alcohol and Drug Problems*, Custodial Medicine Unit, Victoria Police, Mornington, Victoria.

## Long-term effects



- CNS
  - Respiratory system
  - Cardiovascular system
- Immune system
- Endocrine and reproductive systems
- Adverse social outcomes
  - Mental health problems
  - Cognitive impairment
  - Dependence

### Notes

**CNS** problems associated with intoxication.

#### **Respiratory system**

- bronchitis, asthma, sore throat, chronic irritation (e.g., COAD, exacerbates asthma).
- cannabis contains more tar than cigarettes. Cannabis smoke may be more highly carcinogenic than tobacco smoke. Many cannabis users are also dependent tobacco users.
- harms are mostly associated with the route of administration (smoking); e.g., chronic bronchitis. Smoking can result in mutagenic and carcinogenic histopathological changes of the parenchyma and epithelial cells.

#### **Cardiovascular system**

- Increases heart rate but decreases strength of contraction.
- people with cardiovascular disease may experience a decreased exercise tolerance.

#### **Immune system**

- animal studies suggest that chronic cannabis use results in immunosuppression, although findings are inconclusive for humans.

#### **Cognitive impairment**

- depression, anxiety, rapid mood changes reported.
- precipitation of schizophrenia.
- effects may be subtle, but include effects on memory, attention, organisation, and integration of complex information. Although the current evidence suggests that these effects are not grossly debilitating, their reversibility is unknown.



## Cannabis and psychosis

- THC may exacerbate symptoms of schizophrenia through increase in dopamine release
- THC likely precipitates schizophrenia in those vulnerable, i.e., those with a personal or family history of schizophrenia
- Some reports of onset of cannabis-associated schizophrenia in individuals without family risk factors

## Cannabis dependence

- The “cannabis dependence syndrome,” while now clearly described, is perceived as less pronounced than for other drugs (i.e., opioids, alcohol)
- Not yet listed in DSM IV
- Variation in frequency, duration of use and dose result in difficulty predicting rapidity, development, and duration of withdrawal

### Notes

#### **Cannabis dependence syndrome**

Characterised by a variety of cognitive, physical, and behavioural symptoms, e.g., poor impulse control/inability to control use, continued use despite evidence of problems, withdrawal syndrome (anxiety, depression, mood swings, sleep disturbance, memory problems, non-specific physical discomfort) and tolerance (Palmer 2001).

Concerns are emerging that dependence may develop rapidly in younger people and be more severe than previously thought.

It is estimated that 2 joints per day for 3 weeks is sufficient to induce withdrawal symptoms after cessation in some people, although in others daily use for several years has not resulted in withdrawal symptoms on cessation.

Sources: Palmer, B. 2001, *Alcohol and Drug Withdrawal: A Practical Approach. A Manual for Doctors to Assist in the Treatment of Patients Withdrawing from Alcohol and Other Drugs*, Next Step Specialist Drug and Alcohol Services, Mt Lawley, Perth, Western Australia, [www.nextstep.health.wa.gov.au](http://www.nextstep.health.wa.gov.au).

Todd, F., McLean, S., Krum, H., Martin, J., & Copeland, J. 2002, ‘Cannabis’, in Hulse, G., White, J. & Cape, G. (eds.) 2002, *Management of Alcohol and Drug Problems*, ch. 9, Oxford University Press, South Melbourne, pp. 141–157.

## Withdrawal symptoms

- Anxiety, restlessness, irritability, agitation
- Racing thoughts
- Mood swings and increased aggression
- Feelings of unreality
- Fear, sometimes paranoia
- Anorexia, stomach pain
- Weight loss
- Increased body temperature
- Nausea and salivation
- Drowsiness, through disturbed sleep, and an increase in vivid dreams

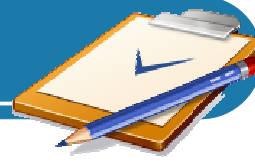
### Notes

While not recorded as a diagnosis in the DSM-IV, the cannabis dependence syndrome is well described. Cannabis withdrawal tends to be less pronounced than that of other drugs (e.g., alcohol and opioids) and may take longer to become established. There is significant variation in symptomatology according to frequency and duration of use, and the rapidity and severity of withdrawal. Some (e.g., Todd et al., 2003) argue that younger people are increasingly reporting dependence of a greater severity than previously.

In assessing and managing cannabis withdrawal, keep in mind that many polydrug users also tend to use cannabis. Assess for polydrug use, as withdrawal from a range of drugs will increase the severity and nature of withdrawal.

Source: Todd, F., McLean, S., Krum, H., Martin, J., & Copeland, J. 2002, 'Cannabis', in Hulse, G., White, J. & Cape, G. (eds.) 2002, *Management of Alcohol and Drug Problems*, ch. 9, Oxford University Press, South Melbourne, pp. 141–157.

# Assessment



## Assessment should focus on:

- drug type, history, route, pattern of use, expenditure
- tolerance, dependence, potential for withdrawal
- history or evidence of psychiatric sequelae
- health complications of cannabis use
- psychosocial context of use (time spent using, obtaining drug, social impact, etc.)
- previous attempts to cut down or quit

## Assessment tools:

- SDS
- ASSIST

## Notes

### Drug history:

- pattern and frequency of use
- number of hours spent intoxicated per day
- cost
- activities undertaken while intoxicated

Part of plant (e.g., bud/head or leaf) and type (e.g., ordinary, hydroponically grown, skunk).

Route of administration (e.g., joint, pipe, bong, bucket bong, oral (cakes, biscuits, butter)).

Brief psychosis precipitated by cannabis is controversial, though the precipitation of a comorbid psychiatric disorder (e.g., schizophrenia) is well documented. Onset of the disorders often occurs in early adulthood coinciding with cannabis use. Thorough investigation of psychotic symptoms and family history are important.

Examination of respiratory function may be useful. Significant respiratory problems such as emphysema, chronic bronchitis or exacerbation of asthma may be evident.

Spirometry may be considered to provide feedback to a user regarding the acute consequences of smoking cannabis (alone or mixed with tobacco).

Acute cardiovascular signs may also be present, either related to panic (e.g. hypertension, tachycardia) or an exacerbation of angina pectoris.

ASSIST source: WHO (World Health Organization) unpublished, Assist Version 2.1 [2003].

Cannabis source: NRDGP (Northern Rivers Division of General Practice ) no date, *11 Cannabis Questions*, <http://www.nrdgp.org.au/directory/documents/23/cannabis-1.pdf>.

SDS source: Gossop, M., Darke, S., Griffiths, P., Hando, J., Powis, B., Hall, W. & Strang, J. 1995, 'The Severity of Dependence Scale (SDS): Psychometric Properties of the SDS in English and Australian Samples of Heroin, Cocaine and Amphetamine Users', *Addiction*, vol. 90, issue 5, pp. 607–614.

## Treatment approaches (1)

### **Brief Advice**

- Provide information on the harms associated with:
  - intoxication
  - long-term, regular use of cannabis
- Provide advice on reducing or ceasing use:
  - 'Delay', 'Distract', 'Avoid', 'Escape', and dealing with 'Lapses'
- Adopt brief motivational and cognitive-behavioural techniques to manage withdrawal and craving
- Other strategies may include:
  - exercise, stress management, relaxation, hobbies, diet, friends.

***Early intervention may be more effective than education.***

### **Notes**

Some people at the severe end of the dependence spectrum or with co-morbid disorders may be helped by referral, consultation, or shared care arrangements with specialist AOD and/or psychiatric services.

## Treatment approaches (2)

No specific pharmacotherapies are available yet for managing cannabis withdrawal or relapse.

### Notes

There has been a substantial increase in the number of cannabis smokers seeking professional assistance to quit, or to manage cannabis-related problems.

There are no specific pharmacotherapies available for the management of cannabis withdrawal or relapse prevention. Short-term sedative-hypnotics may be helpful if withdrawal symptoms are severe and antipsychotic medications may assist in the treatment of psychosis (if antipsychotics are used, ensure prophylaxis to prevent extrapyramidal side-effects).

### Psychosocial interventions

Psychosocial interventions for cannabis use disorder are still in their infancy. Most interventions used for cannabis dependence have been adapted from alcohol interventions. Psychosocial interventions are of greater benefit than no therapy.

Even one session of cognitive behavioural therapy can produce clinically significant reductions in the frequency and amount of cannabis use and related problems among severely dependent users (Copeland et al., 2001). Studies show that 6–9 sessions of cognitive behavioural therapy produce more favourable outcomes than brief motivational interventions, especially with more severely dependent users.

Source: Copeland, J., Swift, W., Howard, J., Roffman, R., Stephens, R. & Berghuis, J. 2001, 'A Randomised Controlled Trial of Brief Interventions for Cannabis Problems Among Young Offenders', *Drug and Alcohol Dependence*, vol. 63(s1), s32.

## Treatment approaches (3)

- **Relapse prevention can be achieved through:**
  - supportive treatment
  - regular follow-up
  - encouraging patient to follow-up treatment with counselling or support groups
  - use of self-help tools and techniques
- **Harm reduction can be promoted by:**
  - assisting patients to identify harms and possible solutions
  - discussing risks associated with driving or work
  - discussing possible psychosis with those predisposed to such

### Notes

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There are no specific pharmacotherapies available for the management of cannabis withdrawal or relapse prevention. Short-term sedative-hypnotics may be helpful if withdrawal symptoms are severe and antipsychotic medications may assist in the treatment of psychosis (if antipsychotics are used, insure prophylaxis to prevent extrapyramidal side-effects).

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## Withdrawal management

- No specific pharmacotherapies for managing cannabis withdrawal or relapse
- Effectively managed as an outpatient, however severe dependence may require specialised assistance
- engage in brief interventions, including relapse prevention and problem solving skills
- consider shared care with psychologists and / or experienced AOD workers



## Pharmacology for withdrawal



Medications may be useful for a limited time:

- sedative / hypnotics

e.g., diazepam 5 – 10 mg qid prn,  
temazepam, 10 – 20 mg nocte for a few  
days

- antipsychotics (for severe agitation or  
psychosis)

e.g., haloperidol or novel agents

### Notes

It may sometimes be useful to prescribe relatively small amounts of hypno-sedatives, e.g., diazepam, to assist with severe agitation and anxiety in the first few days.

Long-term use of anxiolytics is contraindicated unless specifically recommended by a treating psychiatrist.

Antipsychotic agents may be employed in response to psychotic symptoms in the short term. If antipsychotics are used, insure prophylaxis to prevent extrapyramidal side-effects. If symptoms do not settle within a week, psychiatric review is recommended.

Questions?



Comments?

## Post-assessment

Please respond to the post-assessment questions in your workbook.

(Your responses are strictly confidential.)

10 minutes



Thank you for your time!

End of Workshop 3

Thank you for your time!

End of Module 1