

**NAC**  
National Addiction Centre



# *Dangerousness of Drugs*

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## **Introduction**

This publication has been prepared for the Department of Health by the National Addiction Centre. The authors are David Best, Louisa Vingoe & John Strang. (Special thanks to Joanne Bell of the DH Drug Prevention Projects Programme for her help in preparing the document for publication.)

## **Rationale & aims**

It is generally acknowledged that there are dangers associated with drug use, however that may be about as far as it is possible to go without encountering disagreement and controversy. The questions that generate this dissension are based on the reader's priorities (e.g. death rates versus costs to society), the availability of appropriate evidence, the interpretation of that evidence where it does exist and in the way that secondary outcomes (crime, employment, blood-borne disease and so on) are attributed to any drug.

Therefore, depending on the criterion selected, the evidence used to measure danger and the interpretation of that evidence, a 'league table' of dangers may look radically different from one in which other choices are made. What we will attempt to do is to provide a context for the question by examining some definitional and methodological issues before undertaking the task of assessing the dangers associated with particular drugs. We will then attempt to provide a structured, tabular analysis of the dangers directly associated with individual drugs, followed by a commentary on the strengths and limitations of this approach.

However, this tells only part of the story as drug effects relate not only to chemical properties, but also to the pattern and context of their use. Although some consideration of the ways in which drugs are used, such as route of administration, will be included in the tables, the next section will focus more specifically on the context of drug use. The context will be defined in terms of prevalence and using populations to facilitate the subsequent recommendations and conclusions.

## **Definitions**

The definition of danger to be used in this report is the actual or potential exposure to harm, or the risk that certain individuals or circumstances will increase the possibility of harm. While it is important to recognise the dynamic nature of danger, it is possible to establish predisposing danger factors that increase the risk of harm.

This framework for assessing dangerousness, in which the key concepts are risk and harm, is based on the work of Scott (1977) and Brooks (1984). Alaszewski et al (1994) have argued that risk is “the possibility that a given course of action will achieve an undesired outcome or some undesired outcome will develop” and so risk can be understood in terms of potential negative outcomes. This gives rise to an actuarial model that offers the possibility that drug dangers may be assessed in a probabilistic manner.

Scott (1977) has suggested that for such a systematic analysis of danger to occur, it is necessary to specify clearly the behaviour of concern, the potential damage likely from that behaviour and the probability that it will occur under given circumstances. For Brooks (1984) the key variables are the nature of the harm involved, its magnitude, its imminence, its likelihood and its frequency. However, he also suggested that it was important to account for situational conditions that affect the likelihood of the harm occurring and the impact of social interventions that influence the impact of the harm.

While this provides us with a general framework within which we can consider the concept of danger, there are some factors that are particularly salient when considering drug use. Hall (1999) has argued that an appraisal of the personal and public health impact of drug use must account for the prevalence of use, the relative risk of harm and the base rate of the adverse effect. In other words, the danger of the drug is related to both the prevalence of its use and the likelihood of any harms – this is an issue we will return to when examining the ‘capture rates’ for different drugs.

Jaffe (1985) has provided an initial taxonomy for assessing the dangers associated with drugs and the key factors related to this. He has argued that the variability in hazard is a consequence of the drug, the dose, the route of administration, the setting as well as the expectations and experiences of the user. The crucial point he makes for the current investigation is that while certain risks may be limited to more intensive use patterns, others can occur during experimental or recreational use.

The European Monitoring Centre for Drugs & Drug Addiction (EMCDDA) published the guidelines for the risk assessment of new synthetic drugs in 1999 which includes the following taxonomies:

Sources of hazard emanating from:

- Properties of the substance (pharmacology & toxicity)
- Measures of social control (regulatory policies & informal norms)
- Modalities of drug use (patterns & context of use)
- Individual characteristics of user (age gender genetic personality)

Hazardous effects of drugs:

- On the user:
  - Biological (toxicity, dependence)
  - Psychological (functional impairment, effects on personality)
  - Behavioural (neglect of social roles, violence etc)
- On the social environment:
  - Family – micro level (disruption, neglect, violence)
  - Neighbourhood & community – meso level (public disorder & insecurity)
  - Society at large – macro level (effects on the economy, public health & judicial systems)

English (1995) also emphasises the distinction between drug associated effects and drug caused effects. Here the distinction is between those things that are associated with use of the drug (social ostracism, criminal involvement) and those directly caused by the drug (intoxication or withdrawal). This distinction is useful although it may often be a question of attribution whether a particular outcome is explained in terms of preceding substance use. This is one of a number of critical methodological questions that has shaped the development of the investigation.

Single et al (2000) used a different approach to examining risk on the basis of the number of deaths and hospital admissions that could be attributed to alcohol, tobacco and illicit drugs in Canada in 1995. In this year they calculated that 6,507 deaths and 82,014 hospital admissions were attributed to alcohol, 34,728 deaths and 194,072 hospital admissions to tobacco and 805 deaths and 6,940 hospital admissions to illicit drugs. They concluded that substance abuse accounted for 20.0% of total deaths, 22.2% of total potential years of life lost and 9.4% of all hospital admissions, although the authors acknowledge that these estimates are low compared to those made in a number of previous studies. This provides the context against which we should place the importance of understanding the total costs of substance use and quantifying its breakdown across substance types.

## Methodological issues

To start with the question of attribution of causality, it is important to note that this applies to both 'drug-associated' effects and 'drug-caused' effects. One particularly good example of this, as a 'drug-associated' effect, is the relationship between drugs (heroin especially) and crime, in which the complexity of the association precludes a simplistic assertion that criminal involvement is a danger of using drugs. On the other hand, the recent court cases between tobacco companies and cancer victims have highlighted the problems of asserting causality for what would appear a straight-forward health outcome. The problems are twofold, the first relates to the number of potential mediating variables, while the second is about our confidence in asserting causal status to factors that may be separated by both time and circumstances.

One of the most illuminating examples of this comes from the 'death data' associated with different drugs. It relates not only to tobacco but also to alcohol, where a powerful lobby, the alcohol industry, has an obvious interest in minimising the number of deaths that are attributed to alcohol. This political pressure acts only to confound what is already a complex question of aetiology – a person who dies from heart disease may well have had their heart weakened by prolonged excessive drinking, but may also have had a poor diet, little exercise and a stressful lifestyle. In this way, alcohol may well be an enabling condition rather than the single causal determinant, complicating the question of accounting. The recording of this death and its inclusion in the statistics of alcohol dangers is therefore not simply a question of monitoring but of political decision-making, custom and practice and the dominant belief models about the relationships between events.

This is, however, complicated by the way in which data are recorded and information is gathered. Both Brooks (1984) and Jaffe (1985) emphasised the issue of frequency and prevalence of behaviour, yet this is something around which we have limited information for drug use, relying on epidemiological indicators of prevalence for the baseline against which to measure dangers. The problem this creates is that, if the prevalence of an outcome, like treatment-seeking or mortality is known for all drugs, yet prevalence of use is not known for certain substances, then it is not possible to calculate the incidence rate of the problem comparatively across drugs. This is, in part, a result of the legal situation in which many drugs are acquired illicitly and so neither the total population of users nor the total amount consumed can be readily estimated.

Even for a legal substance like alcohol, the quantity and prevalence question cannot be readily calculated. Although it is possible to record the total amount of alcohol sold legally, this excludes illegally imported alcohol, that consumed out of the country by UK citizens and tells us little about patterns and prevalence of alcohol consumption. For this reason, we frequently have to rely on research evidence – either from national surveys or from specific population investigations. The problem with these surveys is partly about representativeness (i.e. is the sample obtained an accurate reflection of the total population) and partly about the type of information obtained. People may not accurately report their drug use because of inadequate memory, the desire to create a good impression or concerns about how the information may be used, while questions about use may not pick up the kinds of problems that are associated with substance use.

Thus, a fundamental problem for this kind of research relates to the accuracy and comparability of the measures to be used. This, in part, reflects problems inherent in gathering certain types of data, but also is a consequence of the work not having been done. In other words, the difficulty of interpretation of risk or prevalence data are confounded by issues of confidence – if there is limited evidence available, or the evidence available is out of date or based on atypical populations, then the issue of comparability becomes especially problematic.

The issue of comparability is not only a question of quality of information (how reliable, representative, up-to-date, etc) but of the type of indicators that are measured across different drugs and by sources with different objectives. Thus, two of the major reference sources utilised in the literature analysis for the project are Home Office statistics on drug misuse (from which the mortality data have been drawn) and the British Crime Survey, a national household survey. The Home Office data are based on reported cases, so they reflect the system of recording and reporting used (changes in these methods will alter the results obtained), as well as the decision-making of those who present the original information (is this a drug death? Does this person qualify as an addict?).

In contrast, the BCS is a voluntary participation survey in which the data are compiled with the participants' consent and so is prey to the structural limitations inherent in self-report. Thus while one source is restricted by the methods of reporting and recording the other is prey to the limitations and biases of self-report. This means that questions of confidence are not the same across sources of information as they reflect the differing

objectives of those for whom the data are prepared. For this reason, we have attempted to analyse our data in the context of a qualitative study in which a literature trawl has been supplemented by expert interviews.

## Methods

A small number of initial interviews were conducted with addiction specialists (3 senior academics at the National Addiction Centre – Professor John Strang, Professor Michael Gossop and Dr Michael Farrell) to discuss the key components of the project, identify target substances and isolate key sources of information. These interviews informed both the subsequent literature search and the design of the interview schedule.

While a far greater range of substances could have been identified (including caffeine, khat and a number of prescribed drugs that have been abused including ketamine and dihydrocodeine), it was felt that it was important to restrict the project to the drugs or classes of drug that have the greatest impact on health and social behaviour. The list of drugs identified are listed in Box 1 below:

### Box 1: Target substances identified for consideration

- **Alcohol**
- **Amphetamines**  
(amphetamine sulphate, dexamphetamine, methamphetamine)
- **Amphetamine type stimulants & novel synthetic drugs**  
(MDMA & analogues, ketamine, GHB)
- **Anabolic-androgenic steroids**
- **Benzodiazepines**  
(temazepam, diazepam, nitrazepam, flunitrazepam)
- **Cannabis**
- **Cocaine hydrochloride**  
(cocaine powder)
- **Freebase cocaine**  
(crack/rock cocaine)
- **Hallucinogens**  
(LSD, psilocybe mushrooms)
- **Opiates**  
(heroin, methadone)
- **Tobacco**
- **Volatile substances**

## Literature search

The list of substances targeted became the focus for the first wave of the literature in which review articles considering key effects, risks and dangers were sought, along with basic information concerning the pharmacology of each of the targets. Key journal articles and books were abstracted from one of the following information sources:

- Psychlit, Medline, BIDS (social science citation index) computerised databases
- National Addiction Centre archive
- Institute for the Study of Drug Dependence (ISDD), now Drugscope, library.

The literature on dangers associated with drug use is large and it has therefore been necessary to be selective in the texts chosen to inform this project. Following the initial trawl of overview articles and texts, more detailed literature searches were carried out to complement the suggestions and recommendations of the interviewees.

The following key search terms were used, but the search strategy focussed primarily on substance specific review articles.

The selection criteria were:

- The material concerned dangers associated with use of the target drugs
- The material either took the form of books by acknowledged experts in the field or was published in peer-reviewed journals
- The material was recent (published within the last 10 years).

We have also tried to be flexible in our approach. In this regard, older material has been included on occasion. In such cases, our rationale was that the topic was important, but poorly served by the literature that fulfilled the inclusion criteria.

## Expert interviews

The initial set of interviews – to define the questions and to establish parameters for the project – were conducted with expert advisors with the National Addiction Centre/Institute of Psychiatry. These interviews were also used to target the interviewees to be contacted for the substance-specific interviews. The initial round of interviews were almost unstructured to allow the interviewees to describe and

prioritise what they felt were the key elements of the study and how we should go about conducting the project. This also enabled us to establish a more structured pro forma for each of the interviewees who were approached about the dangers associated with specific drugs.

For each of the target drugs, one or two experts were identified in this initial phase and recruited to participate (see Appendix 2 for interview schedule). Interviews were tape-recorded and subsequently transcribed. None of the individuals approached refused to participate. Similarly, none of the interviewees objected to being tape-recorded. Interviews were approximately 45 minutes in length.

## Results

Dangers associated with use of the target substances were initially categorised as chronic or acute, and further classified under the main domains of physical dangers (morbidity and mortality), psychological/psychiatric dangers and social/contextual negative effects.

When exploring chronic effects, additional questions about the 'addictiveness' of each substance were included. Participants were asked to describe this in terms of how addictive the substance is, the likelihood and circumstances of physical dependence, as well as evidence of tolerance and withdrawal in chronic users.

To reduce abstraction, participants were also asked to consider the factors that would mediate or moderate the main effects of the substance. This was an attempt to account for individual or group vulnerabilities and dangers associated with the ways in which drugs were used (such as the route of administration and popular combinations). However, it is important to note that there are also factors which may reduce the risk associated with particular drugs and these were included as moderating variables. The main framework for this analysis is outlined in Box 2 opposite:

## Box 2: Framework for typology of dangerousness of drugs

### **Acute adverse effects – dangers regardless of frequency of use**

Physical

- Mortality
- Morbidity

Psychological/psychiatric

Social

### **Chronic adverse effects – dangers that are cumulative with increased use**

Physical

- Mortality
- Morbidity

Psychological/psychiatric

Dependence, tolerance, withdrawal

Social

### **Factors that may mediate or moderate dangers**

- **Aspects of ingestion** (route of administration, dose and purity)
- **Combination use** (use with other drugs either concurrently or consecutively)
- **Availability** (how easily accessible is the substance and how this impacts upon use)
- **Legal situation** (both the law and its implementation around use of the substance)
- **Social context** (consequences of set, setting & social milieu on the dangerousness)
- **Age & developmental issues** (the likely impact of age of onset & use on danger)
- **Individual vulnerability** (particular individuals or groups susceptible to specific harms)
- **Incapacitation** (the effect of imprisonment or treatment on patterns of use – including the substitution of other drugs)

The consideration of target drugs was then completed by an estimation of the adequacy of the information and the severity or likelihood of each risk factor for each drug.

### **Substance specific dangers**

When evaluating and assessing the possible dangers of drugs, due to the vast amount of information obtained from expert interviews and literature searches, we decided to present the results in the form of tables. Three basic tables were designed (acute dangers, chronic dangers, mediating and moderating factors) and completed for each of the 11 target substances.

## Alcohol consumption tables

Table 1a: Acute adverse effects of alcohol consumption.

ALCOHOL	
ACUTE DIVERSE EFFECTS DANGERS OF ALCOHOL REGARDLESS OF FREQUENCY USE	
PHYSICAL	
MORTALITY	MORBIDITY
<ul style="list-style-type: none"> <li>• non drinkers - large dose coma death</li> <li>• death by asphyxiation, alcohol poisoning</li> <li>• death by depressing respiratory centre in medulla</li> <li>• acute pancreatitis</li> <li>• cardiovascular deaths</li> <li>• increased mortality associated with accidents (road traffic, swim, fire, falls)</li> </ul>	<ul style="list-style-type: none"> <li>• reduced audiovisual acuity</li> <li>• ataxia, decrease coordination &amp; loss of balance</li> <li>• drowsiness, loss of consciousness</li> <li>• gastritis, diarrhoea, nausea, vomiting, oesophageal reflux, dehydration</li> <li>• sleep disturbance</li> <li>• hypoglycaemia</li> <li>• arrhythmia (irregular heart beat)</li> <li>• Mallory-Weiss syndrome</li> <li>• flushing</li> <li>• hypothermia</li> <li>• diuresis (discharge of urine in excess)</li> <li>• increased morbidity associated with accidents</li> </ul>
	PSYCHIATRIC PSYCHOLOGICAL
	<ul style="list-style-type: none"> <li>• psychomotor &amp; cognitive impairment (memory, planning, judgement)</li> <li>• reduced inhibitions</li> <li>• argumentative, aggressive</li> <li>• anterograde amnesia, blackout</li> <li>• suicidal ideation intensified with alcohol</li> </ul>
	SOCIAL
	<ul style="list-style-type: none"> <li>• accident (road traffic, swim, fire, falls)</li> <li>• disinhibition, engaging in high risk behaviour (dangerous driving, unsafe sexual practices, use of other substances of abuse), victim of crime</li> <li>• acute intoxication possibly resulting in aggressive &amp; violent behaviour, disorderly conduct, criminal acts</li> <li>• job loss due to intoxication</li> <li>• relationship problems</li> </ul>

Table 1b: Chronic adverse effects of alcohol consumption.

ALCOHOL	
CHRONIC ADVERSE EFFECTS DANGERS OF ALCOHOL THAT ARE CUMULATIVE WITH INCREASED USE	
PHYSICAL	
MORTALITY	MORBIDITY
<ul style="list-style-type: none"> <li>• cardiovascular disease</li> <li>• cardiomyopathy</li> <li>• coronary heart disease</li> <li>• cardiac arrhythmia</li> </ul> <p><u>cerebrovascular disease</u></p> <ul style="list-style-type: none"> <li>• stroke</li> </ul> <p><u>cancers</u></p> <ul style="list-style-type: none"> <li>• oropharynx</li> <li>• larynx</li> <li>• oesophagus</li> <li>• liver</li> <li>• breast</li> </ul> <ul style="list-style-type: none"> <li>• liver cirrhosis</li> </ul> <ul style="list-style-type: none"> <li>• increased risk of premature mortality from accidents, suicide, violence</li> </ul>	<ul style="list-style-type: none"> <li>• toxic/nutritional disorders, vitamin deficiency</li> <li>• gastritis, gastric ulcer</li> <li>• peripheral neuropathy</li> <li>• poly neuropathy</li> <li>• hypertension</li> <li>• pancreatitis</li> <li>• liver cirrhosis</li> <li>• alcoholic hepatitis</li> <li>• oesophageal varices</li> <li>• alcoholic cardiomyopathy</li> <li>• myopathy, muscle weakness &amp; pain</li> <li>• complications in pregnancy and delivery - spontaneous abortion, still birth, foetal alcohol syndrome</li> </ul>
	<ul style="list-style-type: none"> <li>• brain injury</li> <li>• anterior lobe cerebellar degenerative disease</li> <li>• Wernicke encephalopathy (vitamin B1 thiamine deficiency – reversible)</li> <li>• Korsakoffs psychosis (memory defect, impaired ability to plan &amp; organise), with continued drinking in the absence of vitamin supplementation may produce irreversible cognitive impairment</li> <li>• psychotic symptoms during intoxication or withdrawal (depression, paranoia, anxiety)</li> <li>• memory loss, blackouts</li> <li>• loss of self esteem</li> </ul>
	<p><u>dependence syndrome</u></p> <ul style="list-style-type: none"> <li>• moderate dependence potential</li> </ul> <p><u>withdrawal syndrome</u></p> <ul style="list-style-type: none"> <li>• can be fatal</li> <li>• seizures/fits</li> <li>• tremors</li> <li>• anxiety</li> <li>• paranoia</li> <li>• hallucinations</li> </ul> <p>tolerance</p> <ul style="list-style-type: none"> <li>• tolerance to toxic effects may not develop in parallel with tolerance to central nervous system (CNS) depression – increase the likelihood of drug induced organ damage</li> <li>• increased capacity to metabolize alcohol (declines after several weeks abstinence)</li> </ul>
	<p style="text-align: center;"><b>DEPENDENCE TOLERANCE WITHDRAWAL</b></p>
	<p style="text-align: center;"><b>PSYCHIATRIC PSYCHOLOGICAL</b></p>
	<p style="text-align: center;"><b>SOCIAL</b></p> <ul style="list-style-type: none"> <li>alcohol and performance</li> <li>• impaired occupational performance in adults – (absenteeism, poor performance, workplace accidents, financial hardship)</li> <li>• impaired educational achievements in adolescents</li> <li>alcohol related crime</li> <li>• under the influence, while intoxicated or during withdrawal period</li> <li>• acquisitive crime including prostitution</li> <li>• domestic violence, abuse, marital separation, divorce</li> <li>children of parents with <u>alcohol problems</u></li> <li>• more often taken into care</li> <li>• more prone to anxiety &amp; low self esteem</li> <li>• girls higher incidence of depression in childhood &amp; adolescence</li> <li>• boys raised incidence of anti social behaviour</li> <li>• perform poorly at school</li> <li>• more likely to get into trouble with the law</li> <li>• at greater risk of developing a drinking problem in adulthood</li> <li>• child abuse (emotional, physical, sexual) associated with heavy drinking parents</li> </ul>

Table 1c: Factors that mediate & moderate dangers associated with alcohol consumption.

ALCOHOL							
FACTORS THAT MEDIATE (INCREASE) OR MODERATE (REDUCE) DANGERS ASSOCIATED WITH ALCOHOL USE							
ROUTE OF ADMINISTRATION PURITY DOSE	COMBINATION USE (concurrent use, consecutive use)	AVAILABILITY	CONTEXT SETTING	AGE DEVELOPMENTAL ISSUES	INDIVIDUAL VULNERABILITY	INCAPACITATION LEGAL SITUATION	
<ul style="list-style-type: none"> <li>• coma &amp; death in inexperienced drinker</li> <li>• rare examples of intravenous use</li> <li>• designer drinks – rapid intoxication</li> </ul>	<p><b>concurrent use</b></p> <ul style="list-style-type: none"> <li>• link between cigarette smoking &amp; alcohol use</li> <li>• use in combination with sedatives, opiates, cannabis, amphetamines</li> <li>• alcohol used in combination with opiates = risk factor for overdose by respiratory depression</li> </ul>	<ul style="list-style-type: none"> <li>• cross tolerance with benzodiazepines</li> <li>• alcohol may be used when other drug of choice is not available</li> </ul>	<ul style="list-style-type: none"> <li>• alcohol use perceived differently by different societies (wet vs dry cultures)</li> </ul>	<ul style="list-style-type: none"> <li>• alcohol use in adolescence is risk factor for drug use</li> <li>• Farrington (1990) – heavy drinking as a risk factor for persistence of delinquent career up to age 32</li> </ul>	<ul style="list-style-type: none"> <li>• Chinese race lack of enzyme (ALDH2) causes flushing</li> <li>• genetic factors predisposition to problem alcohol use</li> <li>• diabetic may become hypoglycemic as a result of alcohol use</li> </ul>	<ul style="list-style-type: none"> <li>• NOT controlled under Misuse of Drugs Act 1971</li> </ul>	

## Amphetamine consumption tables

Table 2a: Acute adverse effects of amphetamine consumption.

<b>AMPHETAMINES</b> amphetamine sulphate, dexamphetamine, methamphetamine	
ACUTE ADVERSE EFFECTS DANGERS OF AMPHETAMINES REGARDLESS OF FREQUENCY OF USE	
PHYSICAL	
MORTALITY	MORBIDITY
<p><u>excitation syndrome</u></p> <ul style="list-style-type: none"> <li>• hyperthermia</li> <li>• tachycardia followed by circulatory collapse with fatal outcome</li> </ul> <p><u>vascular accidents</u></p> <ul style="list-style-type: none"> <li>• increase in blood pressure</li> <li>• cerebral haemorrhage or myocardial infarction with increased risk of mortality</li> </ul> <p><u>cerebral convulsions &amp; coma</u></p> <ul style="list-style-type: none"> <li>• cardiovascular shock &amp; fatal outcome</li> <li>• mortality associated with methamphetamine use is greater than that with amphetamine</li> </ul>	<p><u>acute intoxication</u></p> <ul style="list-style-type: none"> <li>• pupil dilation</li> <li>• headache</li> <li>• dyskinesia</li> <li>• nausea, abdominal cramps</li> <li>• dry mouth</li> <li>• sweating</li> <li>• decreased appetite</li> <li>• dose related increase in body temperature</li> <li>• increased breathing rate, blood pressure, heart rate (possible arrhythmia)</li> <li>• naive user – dizziness, tremor, irritability, confusion, hallucinations</li> </ul>
PSYCHIATRIC PSYCHOLOGICAL	SOCIAL
<ul style="list-style-type: none"> <li>• drowsiness</li> <li>• reduced ability to concentrate</li> <li>• judgement &amp; learning impaired</li> <li>• dysphoria</li> <li>• anxiety, depression</li> <li>• irritability, aggression</li> <li>• toxic delirium with amnesia</li> </ul> <p><u>acute paranoid psychosis</u></p> <ul style="list-style-type: none"> <li>• psychotic reaction similar to acute paranoid schizophrenia (vivid visual, auditory, or tactile hallucinations, paranoid ideation possibly resulting in aggressive behaviour)</li> <li>• toxic syndrome may develop after ingestion of a single dose in sensitive individuals, risk not dose related, substantial number of cases reversible</li> </ul>	<ul style="list-style-type: none"> <li>• driving impaired – increased risk of road traffic accident</li> <li>• paranoid &amp; psychotic behaviour may be accompanied by violent behaviour, aggressiveness, hostility, physical assault, homicide</li> <li>• relationship problems</li> </ul>

Table 2b: Chronic adverse effects of amphetamine consumption.

AMPHETAMINES amphetamine sulphate, dexamphetamine, methamphetamine	
CHRONIC ADVERSE EFFECTS DANGERS OF AMPHETAMINES THAT ARE CUMULATIVE WITH INCREASED USE	
PHYSICAL	
MORTALITY	MORBIDITY
<p><u>excitation syndrome</u> <u>hyperthermia</u></p> <ul style="list-style-type: none"> <li>• tachycardia followed by circulatory collapse</li> </ul> <p><u>vascular accidents</u></p> <ul style="list-style-type: none"> <li>• increased blood pressure</li> <li>• cerebral haemorrhage or myocardial infarction</li> </ul> <p><u>cerebral convulsions &amp; coma</u></p> <ul style="list-style-type: none"> <li>• cardiovascular shock &amp; fatal outcome.</li> <li>• depression leading to suicide</li> </ul>	<ul style="list-style-type: none"> <li>• health effects of lack of food &amp; sleep – lower resistance to disease</li> <li>• possible neurotoxic damage</li> </ul> <p><u>route specific dangers</u></p> <ul style="list-style-type: none"> <li>• use by injection - see Table 12</li> <li>• smoking - see Table 14</li> </ul>
PSYCHIATRIC PSYCHOLOGICAL	DEPENDENCE TOLERANCE WITHDRAWAL
<p><u>organic brain syndrome</u></p> <ul style="list-style-type: none"> <li>• impairment of memory &amp; ability to concentrate</li> </ul> <p><u>chronic paranoid psychosis</u></p> <ul style="list-style-type: none"> <li>• psychotic reaction similar to paranoid schizophrenia – hallucinations, paranoid ideation possibly resulting in aggressive behaviour. potentially reversible.</li> </ul> <p><u>personality changes</u></p> <ul style="list-style-type: none"> <li>• irritability</li> <li>• suspiciousness</li> <li>• dysphoria</li> <li>• anxiety</li> <li>• paranoid psychosis</li> <li>• depression</li> <li>• restlessness</li> <li>• delirium</li> <li>• depersonalisation</li> <li>• feelings of persecution</li> <li>• lethargy</li> </ul> <p><u>neurological disorders</u></p> <ul style="list-style-type: none"> <li>• behaviour stereotypes - mechanical hyperactivities</li> <li>• perseveration, stereotype motor phenomena eg teeth grinding</li> </ul>	<ul style="list-style-type: none"> <li>• high abuse potential due to mood elevating properties</li> </ul> <p><u>dependence syndrome</u></p> <ul style="list-style-type: none"> <li>• moderate dependence potential</li> </ul> <p><u>withdrawal syndrome</u></p> <ul style="list-style-type: none"> <li>• rarely life threatening</li> <li>• symptoms – depression (risk of suicide), seclusiveness, craving, fatigue, lassitude, lack of energy, sleep disturbance</li> </ul> <p><u>tolerance</u></p> <ul style="list-style-type: none"> <li>• to euphorogenic, anorectic, hyperthermic, cardiovascular effects</li> </ul>
SOCIAL	SOCIAL
	<ul style="list-style-type: none"> <li>• driving impaired (increased risk of road traffic accident)</li> <li>• paranoid &amp; psychotic behaviour may be accompanied by violent behaviour, aggressiveness, hostility, physical assault, homicide</li> <li>• relationship problems</li> <li>• impairs occupational performance in adults &amp; educational achievements in adolescents</li> </ul> <p><u>involvement in crime</u></p> <ul style="list-style-type: none"> <li>• property crime – shoplifting, burglary, fraud</li> <li>• motivator/facilitator role in violent/organised crime eg football hooliganism</li> </ul>

Table 2c: Factors that mediate & moderate dangers associated with amphetamine consumption.

AMPHETAMINE amphetamine sulphate, dexamphetamine, methamphetamine							
FACTORS THAT MEDIATE (INCREASE) OR MODERATE (REDUCE) DANGERS ASSOCIATED WITH AMPHETAMINE USE							
ROUTE OF ADMINISTRATION PURITY DOSE	COMBINATION USE (concurrent use, consecutive use)	AVAILABILITY	SOCIAL CONTEXT SETTING	AGE DEVELOPMENTAL ISSUES	INDIVIDUAL VULNERABILITY	INCAPACITATION LEGAL SITUATION	
<p><u>route specific dangers</u></p> <ul style="list-style-type: none"> <li>• use by injection - see Table 12</li> <li>• smoking - see Table 14</li> <li>• amphetamine purity low in UK 4–9%</li> <li>• increased mortality associated with methamphetamine use</li> </ul>	<p><u>concurrent use</u></p> <ul style="list-style-type: none"> <li>• increased risk of withdrawal fits when amphetamines are used in combination with benzodiazepines &amp;/or alcohol</li> </ul>	<ul style="list-style-type: none"> <li>• no available evidence</li> </ul>	<ul style="list-style-type: none"> <li>• amphetamine aggregation toxicity in hot, crowded settings may result in overheating, exhaustion</li> </ul>	<ul style="list-style-type: none"> <li>• often characterised as a 'gateway' drug (Kandel, 1990) although small pockets of 'career' amphetamine use in the UK</li> </ul>	<p><u>chronic paranoid psychosis</u></p> <ul style="list-style-type: none"> <li>• genetic predisposition to psychotic reaction</li> </ul>	<ul style="list-style-type: none"> <li>• Misuse of Drugs Act 1971 Class A - amphetamines prepared for injection. Class B - oral amphetamines, methamphetamine</li> </ul>	

## Amphetamine type stimulants and novel synthetic drugs tables

Table 3a: Acute adverse effects associated with use of amphetamine type stimulants & novel synthetic drugs (MDMA & analogues, ketamine, GHB).

AMPHETAMINE TYPE STIMULANTS & NOVEL SYNTHETIC DRUGS (MDMA & analogues, ketamine, GHB)			
ACUTE ADVERSE EFFECTS DANGERS OF AMPHETAMINE TYPE STIMULANTS & NOVEL SYNTHETIC DRUGS (MDMA & analogues, eg ketamine, GHB) REGARDLESS OF FREQUENCY OF USE			
MORTALITY	PHYSICAL		SOCIAL
	MORBIDITY	PSYCHIATRIC PSYCHOLOGICAL	
<p><u>GHB</u></p> <ul style="list-style-type: none"> <li>lose consciousness as difficult to get dose right &amp; varies in strength</li> </ul> <p><u>MDMA &amp; analogues</u></p> <ul style="list-style-type: none"> <li>deaths caused by heatstroke, dehydration, surge in antidiuretic hormone so unable to pass excess water through kidneys consequence is brain may swell and cause illness, liver damage, stroke</li> </ul>	<ul style="list-style-type: none"> <li>nausea, vomiting</li> <li>fainting</li> <li>overheating, dehydration</li> <li>headache</li> <li>dry mouth &amp; throat</li> <li>increased blood pressure</li> <li>loss of appetite</li> <li>difficulty with bodily coordination</li> </ul> <p><u>GHB</u></p> <ul style="list-style-type: none"> <li>confusion</li> <li>muscle tremors</li> <li>coma</li> <li>breathing difficulties</li> </ul> <p><u>MDMA</u></p> <ul style="list-style-type: none"> <li>inhibit orgasm in men &amp; women</li> <li>inhibit male erection</li> </ul>	<ul style="list-style-type: none"> <li>anxiety, panic attacks</li> <li>confusion</li> <li>depression</li> <li>insomnia</li> <li>restlessness</li> <li>fatigue</li> <li>anorexia</li> <li>paranoia</li> <li>visual &amp; auditory hallucinations rare</li> </ul> <p><u>MDMA</u></p> <ul style="list-style-type: none"> <li>suggestions that MDMA mildly interferes with cognition after acute administration</li> <li>idiosyncratic psychotic episodes not dose related</li> </ul> <p>substance specific risks.</p> <ul style="list-style-type: none"> <li>4 MTA greater propensity to cause visual hallucinations than MDMA</li> </ul>	<ul style="list-style-type: none"> <li>no evidence of major harmful social consequences such as family or other social relations, problems concerning education, employment or marginalisation</li> <li>no evidence linked to disorderly conduct, acquisitive crime or violence</li> <li>arrest for possession or dealing (buying for friends) Class A drug</li> <li>disinhibition increase risk of unsafe sexual practices</li> <li>difficulty with bodily coordination (dangerous to drive, operate machinery)</li> <li>accidents may result from sleep deprivation</li> </ul>

Table 3b: Chronic adverse effects associated with the use of amphetamine type stimulants & novel synthetic drugs (MDMA & analogues, ketamine, GHB).

## AMPHETAMINES

### amphetamine sulphate, dexamphetamine, methamphetamine

#### CHRONIC ADVERSE EFFECTS

DANGERS OF AMPHETAMINE TYPE STIMULANTS & NOVEL SYNTHETIC DRUGS (MDMA & analogues, eg ketamine, GHB) THAT ARE CUMULATIVE WITH INCREASED USE				
PHYSICAL		PSYCHIATRIC PSYCHOLOGICAL	DEPENDENCE TOLERANCE WITHDRAWAL	SOCIAL
MORTALITY	MORBIDITY			
<ul style="list-style-type: none"> <li>not known - insufficient evidence base</li> </ul>	<ul style="list-style-type: none"> <li>some reports of increased susceptibility to minor ailments – colds, flu, sore throats</li> <li>possible liver damage</li> </ul>	<ul style="list-style-type: none"> <li>long term use MDMA may be associated with mild memory impairment</li> <li>repeated use MDMA may have long lasting effects on mood and personality characteristics such as aggressiveness and impulsivity</li> </ul> <p><u>animal studies</u></p> <ul style="list-style-type: none"> <li>MDMA is toxic to serotonin terminals in brain (in humans may increase risk of depression or other mental illness later in life)</li> <li>dose related neurotoxicity</li> </ul>	<p><u>no dependence syndrome</u></p> <ul style="list-style-type: none"> <li>group of binge users who may consume large quantities of tablets over 2–3 day period may fulfil criteria for dependence</li> <li>low dependence potential</li> <li>tolerance potential</li> </ul>	<ul style="list-style-type: none"> <li>not known – insufficient evidence base on long-term heavy users</li> </ul>

Table 3c: Factors that mediate & moderate dangers associated with the use of amphetamine type stimulants & novel synthetic drugs (MDMA & analogues, ketamine, GHB).

<b>AMPHETAMINE TYPE STIMULANTS &amp; NOVEL SYNTHETIC DRUGS (MDMA &amp; analogues, ketamine, GHB) USE</b>						
<b>FACTORS THAT MEDIATE (INCREASE) OR MODERATE (REDUCE) DANGERS ASSOCIATED WITH AMPHETAMINE TYPE STIMULANTS &amp; NOVEL SYNTHETIC DRUGS (MDMA &amp; analogues, ketamine, GHB) USE</b>						
ROUTE OF ADMINISTRATION PURITY DOSE	COMBINATION USE (concurrent use, consecutive use)	AVAILABILITY	SOCIAL CONTEXT SETTING	AGE DEVELOPMENTAL ISSUES	INDIVIDUAL VULNERABILITY	INCAPACITATION LEGAL SITUATION
<ul style="list-style-type: none"> <li><u>route specific</u></li> <li>use by injection - see table 12</li> <li>intranasal - see table 13</li> <li>rare for ecstasy</li> <li>influence of set &amp; setting has greater impact at a lower dose. As dose increases pharmacological properties of the drug override the effect of set &amp; setting</li> </ul>	<ul style="list-style-type: none"> <li>use of ecstasy may increase individual's social network to include drug scene, peer networking, other drugs more available (route to polydrug use)</li> <li><u>concurrent use</u></li> <li>ecstasy in combination with other stimulant drugs – increase the possible cardiotoxicity &amp; hyperthermic effects of ecstasy</li> <li>ecstasy in combination with cocaine – increased risk neurotoxicity</li> <li>ecstasy in combination with alcohol – reduce perceived level of intoxication – might think ok to drive (impair task performance)</li> <li>consecutive use</li> <li>anecdotal reports of ecstasy users using benzodiazepines or heroin to self medicate adverse effects (especially crash period)</li> </ul>	<ul style="list-style-type: none"> <li>market well informed &amp; shift patterns of use depending on:                             <ul style="list-style-type: none"> <li>rumours regarding pill content</li> <li>media campaigns adverse publicity regarding neurotoxicity</li> <li>subjective effects of lower dose MDMA</li> <li>changes in price of ecstasy &amp; other drugs – possibly substitute with drugs like amphetamine &amp; cocaine</li> </ul> </li> </ul>	<ul style="list-style-type: none"> <li>amphetamine aggregation toxicity - overcrowding &amp; overheating at unregulated dance events</li> <li>synthetic stimulants increase aggression therefore potential for violence in social situations (especially in combination with alcohol). However, anecdotally little evidence to suggest ecstasy involved in violent crime</li> </ul>	<ul style="list-style-type: none"> <li>no clear evidence on relationship between uptake of ecstasy and use of other drugs or drug careers</li> </ul>	<ul style="list-style-type: none"> <li>individual vulnerability for acute physical problems – previous history of epilepsy, cardiovascular or cerebrovascular disease</li> <li>poor metaboliser status – 2 phenotypes in population of the enzyme that metabolises ecstasy – however influence of metaboliser status unknown</li> <li>psychological vulnerability increased with previous or current history of psychiatric illness, or family history</li> </ul>	<ul style="list-style-type: none"> <li>Misuse of Drugs Act 1971 Class A - MDMA &amp; MDA family (MDMA, MDA, MDEA, MBDB, MMMA)</li> </ul>

## Anabolic-androgenic steroids tables

Table 4a: Acute adverse effects associated with the use of anabolic-androgenic steroids (AAS).

ANABOLIC-ANDROGENIC STEROIDS			
ACUTE ADVERSE EFFECTS DANGERS OF ANABOLIC-ANDROGENIC STEROIDS REGARDLESS OF FREQUENCY OF USE			
PHYSICAL		PSYCHIATRIC PSYCHOLOGICAL	SOCIAL
MORTALITY	MORBIDITY		
<ul style="list-style-type: none"> <li>• few recorded cases in UK of mortality directly linked to AAS use</li> <li>• several use cases of HIV fatalities related to AAS use (Brower, 1994)</li> </ul>	<ul style="list-style-type: none"> <li>• <u>route specific dangers</u></li> <li>• use by injection - (see table 12)</li> <li>• steroid users may have additional risks regarding injection:               <ul style="list-style-type: none"> <li>• intramuscular injection is administered in the buttocks out of sight of the user therefore their technique may be more clumsy &amp; increase chances of infection</li> <li>• chance of sharing injecting equipment may be increased as larger bore needles are needed for intramuscular injection &amp; these may not be as easily available as the narrower gauge intravenous varieties</li> </ul> </li> </ul>	<ul style="list-style-type: none"> <li>• aggression, violence</li> </ul>	<ul style="list-style-type: none"> <li>• not known - insufficient evidence base</li> </ul>

Note: Almost all of the side effects of anabolic steroids are dose dependent and are more likely when prolonged administration occurs.

Table 4b: Chronic adverse effects associated with the use of anabolic-androgenic steroids.

ANABOLIC-ANDROGENIC STEROIDS				
CHRONIC ADVERSE EFFECTS DANGERS OF ANABOLIC STEROIDS THAT ARE CUMULATIVE WITH INCREASED USE				
MORTALITY	PHYSICAL		SOCIAL	
	MORBIDITY	PSYCHIATRIC PSYCHOLOGICAL		DEPENDENCE TOLERANCE WITHDRAWAL
<ul style="list-style-type: none"> <li>premature accumulation of fats in arteries, death due to effect on heart &amp; blood vessels</li> <li>HIV deaths</li> </ul>	<ul style="list-style-type: none"> <li>hypertension</li> <li>liver &amp; heart damage</li> <li>growth stunting in adolescence</li> <li>cancer rare but liver, prostate &amp; kidney been reported</li> <li>acne, alopecia</li> <li>increased risk of tendon damage through exercise</li> <li>suggested link between steroid use and immunosuppression – no evidence</li> <li>sleep disorders</li> </ul> <p><u>males</u></p> <ul style="list-style-type: none"> <li>inhibiting effect on normal testicular function – reduced testosterone production &amp; sperm production, testicular atrophy</li> <li>enlargement of prostate gland or prostate cancer</li> <li>excessive testosterone and oestrogen levels leading to gynaecomastia (development of breast tissue may occur)</li> <li>effects reversible if use discontinued</li> </ul> <p><u>females</u></p> <ul style="list-style-type: none"> <li>masculinising/virilisation effects (increased growth of hair on body &amp; face, deepening of voice, enlargement of clitoris, increased libido, reduced breast size)</li> <li>menstrual irregularities or amenorrhoea</li> <li>reduced fertility</li> <li>effects in women irreversible</li> </ul> <p><u>route specific dangers</u></p> <ul style="list-style-type: none"> <li>use by injection - (see table 12)</li> </ul>	<p>personality changes</p> <ul style="list-style-type: none"> <li>argumentative</li> <li>impetuous</li> <li>moody</li> <li>suspicious</li> <li>antisocial behaviour</li> <li>paranoia</li> <li>aggression</li> <li>violence</li> <li>depression</li> </ul>	<ul style="list-style-type: none"> <li>evidence of dependence syndrome</li> <li>withdrawal symptoms unclear</li> </ul>	<ul style="list-style-type: none"> <li>limited evidence suggests increased access to stimulants</li> <li>sharing of injecting equipment with training partners</li> </ul>

Table 4c: Factors that mediate &amp; moderate dangers associated with the use of anabolic–androgenic steroids.

<b>ANABOLIC–ANDROGENIC STEROIDS</b>						
<b>FACTORS THAT MEDIATE (INCREASE) OR MODERATE (REDUCE) DANGERS ASSOCIATED WITH ANABOLIC–ANDROGENIC STEROID USE</b>						
<b>ROUTE OF ADMINISTRATION PURITY DOSE</b>	<b>COMBINATION USE (concurrent use, consecutive use)</b>	<b>AVAILABILITY</b>	<b>SOCIAL CONTEXT SETTING</b>	<b>AGE DEVELOPMENTAL ISSUES</b>	<b>INDIVIDUAL VULNERABILITY</b>	<b>INCAPACITATION LEGAL SITUATION</b>
<b>route specific</b> <ul style="list-style-type: none"> <li>• use by injection - see table 12</li> </ul>	<b>concurrent use</b> <ul style="list-style-type: none"> <li>• range of other drugs (prescribed, illicit) are used to counteract the side effects or augment the desired effects of steroids. Examples include:               <ul style="list-style-type: none"> <li>• diuretics – counteract fluid retention caused by steroids &amp; sharpen definition of skeletal muscle contours</li> <li>• tamoxifen – helps to reduce gynaecomastia</li> <li>• human chorionic gonadotrophin – increases secretion of testosterone</li> </ul> </li> <li>• some evidence of cross over to stimulant use</li> </ul>	<ul style="list-style-type: none"> <li>• variability in quality of imports</li> <li>• widely available in both legal and illegal gym facilities</li> </ul>	<ul style="list-style-type: none"> <li>• gyms</li> <li>• use by firemen, policemen etc but most common use by bodybuilders</li> </ul>	<ul style="list-style-type: none"> <li>• significant reproductive problems with adolescent use</li> <li>• growth stunting in adolescents</li> </ul>	<ul style="list-style-type: none"> <li>• dispositional tendency to aggression</li> </ul>	<ul style="list-style-type: none"> <li>• Misuse of Drugs Act 1971 Class C - anabolic - androgenic steroids</li> </ul>

## Benzodiazepines (temazepam, diazepam, nitrazepam) tables

Table 5a: Acute adverse effects associated with the use of benzodiazepines (temazepam, diazepam, nitrazepam).

<b>BENZODIAZEPINES</b> temazepam, diazepam nitrazepam			
ACUTE ADVERSE EFFECTS DANGERS OF BENZODIAZEPINES REGARDLESS OF FREQUENCY OF USE			
PHYSICAL		PSYCHIATRIC PSYCHOLOGICAL	SOCIAL
MORTALITY	MORBIDITY		
<p><u>overdose</u></p> <ul style="list-style-type: none"> <li>• prolonged sleep, coma, impairment of breathing, death</li> <li>• especially when used in combination with alcohol or heroin</li> </ul>	<p><u>route specific</u></p> <ul style="list-style-type: none"> <li>• use by injection - see Table 1.2</li> </ul>	<ul style="list-style-type: none"> <li>• depress mental activity &amp; alertness</li> <li>• drowsiness</li> <li>• lethargy</li> <li>• memory loss</li> <li>• disinhibition</li> <li>• chaotic paranoid behaviour</li> <li>• aggression, violent behaviour</li> </ul>	<ul style="list-style-type: none"> <li>• in high risk behaviour – unsafe sexual practices</li> <li>• aggressive &amp; violent behaviour</li> <li>• criminal activity</li> </ul>

Note: 2 different aspects to benzodiazepine abuse –

- over prescribing &/or inappropriate prescribing of benzodiazepines has resulted in large numbers of patients becoming dependent on them
- abuse of benzodiazepines occurs on the street often by intravenous injection of formulations designed for oral administration guidance on prescribing – purchased on black market or from legitimate receivers of prescriptions, theft from health centres or pharmacies, obtain prescriptions by deception false names etc.

Table 5b: Chronic adverse effects associated with the use of benzodiazepines (temazepam, diazepam, nitrazepam).

<b>BENZODIAZEPINES</b> temazepam, diazepam, nitrazepam				
<b>CHRONIC ADVERSE EFFECTS</b> DANGERS OF BENZODIAZEPINES THAT ARE CUMULATIVE WITH INCREASED USE				
<b>PHYSICAL</b>		<b>PSYCHIATRIC PSYCHOLOGICAL</b>	<b>DEPENDENCE TOLERANCE WITHDRAWAL</b>	<b>SOCIAL</b>
<b>MORTALITY</b>	<b>MORBIDITY</b>			
<ul style="list-style-type: none"> <li>low rates of direct benzodiazepines mortality but implicated in significant proportion of opiate overdose fatalities and evidence of mortality in combination with alcohol</li> </ul>	<b>route specific dangers</b> <ul style="list-style-type: none"> <li>use by injection - see Table 12</li> </ul>	<ul style="list-style-type: none"> <li>depression</li> <li>anxiety</li> <li>attention deficit</li> <li>loss of sleep</li> <li>loss of volitional control</li> </ul>	<b>Dependence syndrome</b> <ul style="list-style-type: none"> <li>moderate dependence potential</li> <li>2 groups – long term prescribed and illicit use</li> <li>tolerance potential</li> </ul> <b>withdrawal syndrome</b> <ul style="list-style-type: none"> <li>convulsions – possibly fatal</li> <li>insomnia</li> <li>dysphoria, anxiety, irritability, depression, malaise</li> <li>decreased concentration</li> <li>muscle twitching, tremors</li> <li>depersonalisation</li> <li>nausea &amp; vomiting</li> <li>perceptual hypersensitivity/distortions</li> <li>headaches</li> </ul>	<ul style="list-style-type: none"> <li>social use rare among long-term prescribed users</li> <li>use among polysubstance users, adolescent users and street population users increases their engagement with “grey markets” (markets of prescribed drugs)</li> </ul>

Table 5c: Factors that mediate & moderate dangers associated with the use of benzodiazepines (eg temazepam, diazepam, nitrazepam).

<b>BENZODIAZEPINES</b> temazepam, diazepam, nitrazepam							
FACTORS THAT MEDIATE (INCREASE) OR MODERATE (REDUCE) DANGERS ASSOCIATED WITH BENZODIAZEPINE USE							
ROUTE OF ADMINISTRATION PURITY DOSE	COMBINATION USE (concurrent use, consecutive use)	AVAILABILITY	SOCIAL CONTEXT SETTING	AGE DEVELOPMENTAL ISSUES	INDIVIDUAL VULNERABILITY	INCAPACITATION LEGAL SITUATION	
route specific • use by injection - see table 12  <u>temazepam gel capsules</u> • injecting complications • blocking peripheral veins in arms, legs, skin abscesses, deep vein thrombosis	concurrent use • benzodiazepines used in combination with opiates – increased risks of overdose • benzodiazepines used in combination with alcohol – increased risk of withdrawal fits – can be fatal • opiate users take benzodiazepines to augment the effects of weak illicit heroin	• prescribing practices • dose related factors • effectiveness of importation policies	• not known - insufficient evidence base	• early use of benzodiazepines has been noted in some adolescent groups but this has tended to be localised	• pre-existing depressant drug use	• Misuse of Drugs Act 1971 Class C - benzodiazepines	

Table 6a: Acute adverse effects of cannabis consumption.

<b>CANNABIS</b>	
<b>ACUTE ADVERSE EFFECTS DANGERS OF CANNABIS REGARDLESS OF FREQUENCY OF USE</b>	
<b>PHYSICAL</b>	
<b>MORTALITY</b>	<b>MORBIDITY</b>
<ul style="list-style-type: none"> <li>• no danger of fatal overdose</li> <li>• no confirmed cases of human deaths</li> </ul>	<ul style="list-style-type: none"> <li>• irritant effects of smoke on respiratory system (coughing, sore throat, bronchospasm in asthmatic people)</li> <li>• facial flushing</li> <li>• abdominal pain, nausea, vomiting</li> <li>• cannabis use can cause tachycardia &amp; in some cases increase blood pressure while in a reclining position but increase pressure when standing. Whilst this would not present problems for young healthy users, the older user, perhaps with cardiovascular or coronary artery disease should be aware of the potential risks</li> </ul>
<b>PSYCHIATRIC PSYCHOLOGICAL</b>	<b>SOCIAL</b>
<ul style="list-style-type: none"> <li>• effects of cannabis upon mental state vary considerably between individuals &amp; are determined by dose taken, route of administration, expectations of the user, concomitant use of other drugs, the users emotional state, whether individual is suffering from a psychiatric illness</li> <li>• temporary psychological distress (especially naive users) – dysphoria, anxiety, confusion, drowsiness, depression, panic attacks, perceptual distortion (hallucinations), amnesia/forgetfulness, confusion of thought processes, impaired judgement, agitation, hypomanic symptoms, short – lived &amp; reversible psychotic reaction</li> </ul>	<ul style="list-style-type: none"> <li>• intoxication impairs cognitive &amp; perceptual motor coordination &amp; performance, information processing, short term memory, signal detection &amp; tracking behaviour &amp; prolongs reaction time – therefore impair ability to drive (evidence that drivers aware of impairment and more cautious), operate machinery, make decisions</li> </ul>

Table 6b: Chronic adverse effects of cannabis consumption.

CANNABIS		CHRONIC ADVERSE EFFECTS DANGERS OF CANNABIS THAT ARE CUMULATIVE WITH INCREASED USE			
MORTALITY	PHYSICAL		PSYCHIATRIC PSYCHOLOGICAL	DEPENDENCE TOLERANCE WITHDRAWAL	SOCIAL
	MORBIDITY				
<p>contributory cause of <u>cancers of the aerodigestive tract</u></p> <ul style="list-style-type: none"> <li>• mouth</li> <li>• tongue</li> <li>• throat</li> <li>• oesophagus</li> <li>• lung</li> </ul> <p><u>chronic respiratory disease</u></p> <ul style="list-style-type: none"> <li>• chronic bronchitis</li> <li>• lung damage</li> </ul>	<ul style="list-style-type: none"> <li>• persistent sore throat</li> <li>• use can inhibit reproductive functions &amp; disrupt ovulation, sperm production &amp; sperm function</li> <li>• no evidence of structural change in brains of heavy long term cannabis users</li> <li>• effects of cannabis on human immune function not known (possible suppression of immune system)</li> <li>• no conclusive evidence that cannabis causes cancer in humans but may be an important risk factor for the development of respiratory cancer (cofounders – smoke tobacco as well or use tobacco as vehicle for smoking cannabis resin)</li> <li>• like tobacco, cannabis smoke is highly likely to be harmful to foetal development &amp; should be avoided by pregnant women. Although there is a raft of studies suggesting that babies born to cannabis smoking mothers weigh less than the offspring of a control group &amp; that children of cannabis smoking mothers may face developmental problems, the research thus far has been unable to untangle the effects of smoking &amp; other factors from that of cannabis use per se.</li> </ul>	<ul style="list-style-type: none"> <li>• no severe or grossly debilitating impairment in cognitive function (subtle cognitive impairment in higher cognitive functions of memory, learning processes, attention &amp; organization &amp; the integration of complex information – may or may not be reversible after abstinence)</li> <li>• clear evidence of an association between cannabis use &amp; schizophrenia, but the significance of this association is unclear: cannabis can exacerbate the symptoms of schizophrenia in affected individuals &amp; is linked with relapse in schizophrenia.</li> <li>• insomnia, depression, aggression anxiety</li> <li>• social withdrawal, apathy, amotivational syndrome – controversial</li> </ul>	<p>dependence syndrome</p> <ul style="list-style-type: none"> <li>• generally considered to be a drug of very low dependence potential</li> <li>• query dependence syndrome (Wayne Hall 1 in 10)</li> </ul> <p><u>withdrawal syndrome</u></p> <ul style="list-style-type: none"> <li>• mild. symptoms may include irritability, anxious mood, physical changes – tremor, perspiration, nausea, sleep disturbance</li> </ul> <p><u>tolerance</u></p> <ul style="list-style-type: none"> <li>• to psychoactive &amp; physical effects unlikely to occur unless there is sustained heavy exposure</li> </ul>	<ul style="list-style-type: none"> <li>• chronic heavy use may cause subtle impairments in occupational &amp; educational performance of adults (poor performance due to apathy, impaired motivation, lack of energy BUT confounding factors)</li> <li>• financial difficulties</li> </ul>	

Table 6c: Factors that mediate &amp; moderate dangers associated with cannabis consumption.

<b>CANNABIS</b>							
<b>FACTORS THAT MEDIATE (INCREASE) OR MODERATE (REDUCE) DANGERS ASSOCIATED WITH CANNABIS USE</b>							
<b>ROUTE OF ADMINISTRATION PURITY DOSE</b>	<b>COMBINATION USE (concurrent use, consecutive use)</b>	<b>AVAILABILITY</b>	<b>SOCIAL CONTEXT SETTING</b>	<b>AGE DEVELOPMENTAL ISSUES</b>	<b>INDIVIDUAL VULNERABILITY</b>	<b>INCAPACITATION LEGAL SITUATION</b>	
<ul style="list-style-type: none"> <li>• can be eaten or smoked</li> <li>• <u>eating of cannabis</u> makes dosages difficult to regulate</li> <li>• unpleasant reaction more difficult to avoid</li> <li>• <u>smoking of cannabis</u></li> <li>• see Table 10</li> </ul>	<ul style="list-style-type: none"> <li>• smoking with tobacco</li> </ul>	<ul style="list-style-type: none"> <li>• widely available across the UK and internationally – no clear evidence of both police or custom interventions on supply</li> <li>• also difficult to police as a result of home-grown cannabis</li> </ul>	<ul style="list-style-type: none"> <li>• used across wide range of social and age contexts</li> <li>• therapeutic use possible</li> <li>• self-medication among psychiatric patients</li> <li>• widely used by problem drug users</li> </ul>	<ul style="list-style-type: none"> <li>• regular use of cannabis may encourage users to progress to other forms of drug abuse, although the likelihood of this occurring is more related to the lifestyle &amp; personality of the individual than the effect of cannabis itself</li> </ul>	<ul style="list-style-type: none"> <li>• increased risk of experiencing psychotic reactions in vulnerable individuals – genetics</li> <li>• precipitate relapse schizophrenia</li> <li>• adversely affect course of schizophrenia</li> <li>• stimulating effects of THC on cardiovascular system can be detrimental to individuals with cardiovascular or</li> </ul>	<ul style="list-style-type: none"> <li>• Misuse of Drugs Act 1971 Class B - cannabis, cannabis resin</li> </ul>	

## Cocaine hydrochloride (cocaine powder) or freebase cocaine (crack/rock cocaine) tables

Table 7a: Acute adverse effects associated with the use of cocaine hydrochloride (cocaine powder) or freebase cocaine (crack/rock cocaine).

<b>COCAINE HYDROCHLORIDE (cocaine powder) FREEBASE COCAINE (crack/rock cocaine)</b>			
ACUTE ADVERSE EFFECTS			
DANGERS OF COCAINE HYDROCHLORIDE (cocaine powder) & FREEBASE COCAINE (crack/rock cocaine) REGARDLESS OF FREQUENCY OF USE			
PHYSICAL		PSYCHIATRIC PSYCHOLOGICAL	SOCIAL
MORTALITY	MORBIDITY		
<ul style="list-style-type: none"> <li>mortality rare from cocaine hydrochloride use alone – adverse effects are unlikely to lead to death</li> <li><u>cardiovascular crisis</u></li> <li>death from respiratory or heart failure – rare</li> <li><u>allergic reaction from intravenous use of cocaine</u></li> <li>anecdotal citations – possibly caused by additives in street cocaine</li> <li>toxic reactions (eg cardiovascular crisis) are dose related and are therefore more likely to occur from smoking crack cocaine than intranasal use of cocaine hydrochloride</li> </ul>	<p><u>cardiovascular</u></p> <ul style="list-style-type: none"> <li>dose dependent increase blood pressure &amp; body temperature, accelerated heart rate &amp; breathing, disturbed heart rhythm – ventricular fibrillation, chest pain, shortness of breath, respiratory arrest, heart attack</li> </ul> <p><u>neurological</u></p> <ul style="list-style-type: none"> <li>stroke</li> <li>seizure</li> <li>headaches</li> </ul> <p><u>musculoskeletal</u></p> <ul style="list-style-type: none"> <li>muscle spasms</li> <li>tremor</li> </ul> <p><u>gastrointestinal</u></p> <ul style="list-style-type: none"> <li>abdominal pain</li> <li>nausea</li> <li>vomiting</li> </ul> <ul style="list-style-type: none"> <li>increased sexual appetite &amp; desire</li> </ul> <p><u>route specific dangers</u></p> <ul style="list-style-type: none"> <li>use by injection - See Table 12</li> <li>intranasal use - See Table 13</li> <li>smoking - See Table 14</li> </ul>	<ul style="list-style-type: none"> <li>restlessness, agitation</li> <li>impaired mental functioning</li> <li>sleep disturbance</li> <li>anxiety</li> <li>paranoia</li> <li>grandiosity</li> <li>transient psychotic reactions</li> <li>hallucinations (visual, auditory, tactile) after large doses</li> <li>aggression &amp; possible violence (especially associated with crack cocaine use)</li> </ul>	<ul style="list-style-type: none"> <li>disinhibition &amp; increased sexual desire – possibility of high risk sexual behaviour (unprotected penetrative sex, vigorous intercourse, risk of sexually transmitted diseases, viral transmission)</li> <li>women exchange sex for crack, engage in high risk sex when using the drug</li> <li>aggression &amp; possible violence (especially associated with crack cocaine use)</li> </ul>

Table 7b: Chronic adverse effects associated with the use of cocaine hydrochloride (cocaine powder) or freebase cocaine (crack/rock cocaine).

COCAINE HYDROCHLORIDE (cocaine powder) FREEBASE COCAINE (crack/rock cocaine)				
CHRONIC ADVERSE EFFECTS DANGERS OF COCAINE HYDROCHLORIDE (cocaine powder) & FREEBASE COCAINE (crack/rock cocaine) THAT ARE CUMULATIVE WITH INCREASED USE				
PHYSICAL		PSYCHIATRIC PSYCHOLOGICAL	DEPENDENCE TOLERANCE WITHDRAWAL	SOCIAL
MORTALITY	MORBIDITY			
<p><u>crack cocaine</u></p> <ul style="list-style-type: none"> <li>coronary arteries fur with fatty deposits leading to premature heart attacks</li> </ul>	<ul style="list-style-type: none"> <li>malnutrition &amp; weight loss</li> <li>obstetric complications, increased risk of birth defects in child</li> <li>chronic use diminishes sexual appetite &amp; ability – reversible after abstinence</li> </ul> <p><u>route specific dangers</u></p> <ul style="list-style-type: none"> <li>use by injection - see Table 12</li> <li>intranasal use - see Table 13</li> <li>smoking - see Table 14</li> </ul>	<ul style="list-style-type: none"> <li>anxiety, depression</li> <li>obsessional rituals/preoccupation, repetitive behaviours</li> <li>sleep disturbance (decrease quantity &amp; quality of sleep)</li> <li>irritability, restlessness</li> <li>auditory hallucinations</li> <li>paranoid delusions &amp; psychosis</li> <li>hyperexcitability</li> <li>exhaustion</li> </ul> <p><u>toxic syndrome</u></p> <ul style="list-style-type: none"> <li>psychotic reaction similar to acute paranoid schizophrenia – vivid auditory tactile hallucinations, picking &amp; excoriation of skin, delusions of parasitosis, paranoid ideation</li> </ul> <ul style="list-style-type: none"> <li>blood flow deficit in brain leading to cognitive problems</li> </ul>	<p><u>dependence syndrome</u></p> <ul style="list-style-type: none"> <li>dependence potential may vary according to mode of administration:               <ul style="list-style-type: none"> <li>intranasal powder – high/moderate</li> <li>smoked crack – very high</li> </ul> </li> </ul> <p><u>withdrawal syndrome</u></p> <ul style="list-style-type: none"> <li>mild to moderate, intra &amp; inter individual variation in type &amp; severity of problems:               <ul style="list-style-type: none"> <li>symptoms – craving, lassitude, lack of energy, hyperphagia, depression, dysphoric mood, fatigue, unpleasant dreams, insomnia or hypersomnia, increased appetite, psychomotor retardation, agitation, anxiety, restlessness, irritability, aggression</li> </ul> </li> </ul> <ul style="list-style-type: none"> <li>craving – possibly different in magnitude for cocaine hydrochloride &amp; freebase/crack cocaine</li> </ul>	<ul style="list-style-type: none"> <li>financial difficulties</li> <li>context of purchasing drug on the street associated with danger around street culture, increased probability of violence</li> <li>women exchange sex for crack, engage in high risk sex when using the drug</li> </ul>

Table 7c: Factors that mediate & moderate dangers associated with the use of cocaine hydrochloride (cocaine powder) or freebase cocaine (crack/rock cocaine).

<b>COCAINE HYDROCHLORIDE (cocaine powder) FREEBASE COCAINE (crack/rock cocaine)</b>						
<b>FACTORS THAT MEDIATE (INCREASE) OR MODERATE (REDUCE) DANGERS ASSOCIATED WITH COCAINE HYDROCHLORIDE (cocaine powder) &amp; FREEBASE COCAINE (crack/rock cocaine) USE</b>						
ROUTE OF ADMINISTRATION PURITY DOSE	COMBINATION USE (concurrent use, consecutive use)	AVAILABILITY	SOCIAL CONTEXT SETTING	AGE DEVELOPMENTAL ISSUES	INDIVIDUAL VULNERABILITY	INCAPACITATION LEGAL SITUATION
<p><u>route specific dangers</u></p> <ul style="list-style-type: none"> <li>• use by injection - see Table 12</li> <li>• effects of cocaine by injection are relatively brief and therefore users may inject frequently increasing likelihood of sharing &amp; viral exposure</li> <li>• intranasal use - see Table 13</li> <li>• smoking - see Table 14</li> <li>• risk of acute death higher for crack cocaine than cocaine hydrochloride due to rapidity of onset - route specific smoking</li> </ul>	<ul style="list-style-type: none"> <li>• chronic use of cocaine may be a risk factor for use of heroin</li> <li>• concurrent use</li> <li>• alcohol &amp; cocaine used in combination potentiate one another by production of enzyme - this combination is most commonly seen in cocaine related deaths</li> <li>• mixture of cocaine &amp; heroin (speedball) frequently mentioned in fatal emergency room admissions</li> <li><u>consecutive use</u></li> <li>• reports of use of heroin after cocaine to manage negative effects after prolonged use</li> </ul>	<ul style="list-style-type: none"> <li>• cocaine now more available, cheaper &amp; of greater purity - possibly accounts for increases in prevalence of use</li> </ul>	<ul style="list-style-type: none"> <li>• cocaine and crack likely to be used in very different social settings</li> <li>• crack most commonly associated with existing problem drug users</li> <li>• cocaine powder more widespread and used functionally in both employment and social settings</li> </ul>	<ul style="list-style-type: none"> <li>• clear evidence of cocaine as culmination of developmental pathway</li> </ul>	<ul style="list-style-type: none"> <li>• individuals with pre existing ischemic heart disease, cocaine can have an apparently sympathomimetic effect on the heart increasing myocardial oxygen demands to the extent that angina pains occur and sometimes myocardial infarction</li> <li>• snorting or smoking can exacerbate asthma</li> <li>• significant exacerbating effect on individuals with pre-existing mental health problems</li> </ul>	<ul style="list-style-type: none"> <li>• Misuse of Drugs Act 1971 Class A - cocaine</li> </ul>

## Hallucinogens (LSD, psilocybe eg mushrooms) tables

Table 8a: Acute adverse effects associated with the use of hallucinogens (LSD, psilocybe eg mushrooms).

HALLUCINOGENS (LSD, psilocybe eg mushrooms)		
ACUTE ADVERSE EFFECTS DANGERS OF HALLUCINOGENS (LSD, psilocybe eg mushrooms) REGARDLESS OF FREQUENCY OF USE		
PHYSICAL		SOCIAL
MORTALITY	MORBIDITY	
<ul style="list-style-type: none"> <li>• risk of injury &amp; accidental death</li> </ul> <p><u>LSD</u></p> <ul style="list-style-type: none"> <li>• one case of fatal overdose has been reported</li> </ul> <p><u>mushrooms</u></p> <ul style="list-style-type: none"> <li>• fatal poisoning due to mistaken identity</li> </ul>	<ul style="list-style-type: none"> <li>• self harm, accidents or violence while intoxicated</li> </ul> <p><u>LSD</u></p> <ul style="list-style-type: none"> <li>• common effects: adrenergic 'fight or flight' effects – tachycardia, flushing, dry mouth, sweating, exhaustion, tiredness, weakness</li> <li>• rare effects: ataxia, convulsions, hyperpyrexia</li> </ul> <p><u>mushrooms</u></p> <ul style="list-style-type: none"> <li>• nausea, vomiting, stomach pains, dizziness</li> </ul>	<p style="text-align: center;"><b>PSYCHIATRIC PSYCHOLOGICAL</b></p> <ul style="list-style-type: none"> <li>• dysphoria</li> <li>• unpleasant distortions in shapes &amp; colours</li> <li>• frightening illusions, delusions or hallucinations</li> <li>• anxiety, panic, depression</li> <li>• dizziness, disorientation</li> <li>• impaired concentration</li> <li>• short lived psychotic episode (hallucinations, paranoia)</li> <li>• precipitate relapses in schizophrenia</li> </ul>

Table 8b: Chronic adverse effects associated with the use of hallucinogens (LSD, psilocybe eg mushrooms).

HALLUCINOGENS (LSD, psilocybe eg mushrooms)			
CHRONIC ADVERSE EFFECTS DANGERS OF HALLUCINOGENS (LSD, psilocybe eg mushrooms) THAT ARE CUMULATIVE WITH INCREASED USE			
MORTALITY	PHYSICAL		SOCIAL
	MORBIDITY	DEPENDENCE TOLERANCE WITHDRAWAL	
<ul style="list-style-type: none"> <li>limited evidence base</li> </ul>	<ul style="list-style-type: none"> <li>no known physical dangers associated with long term LSD use</li> </ul>	<p><u>post exposure effects</u></p> <ul style="list-style-type: none"> <li>post hallucinogen perceptual disorder (flashbacks – unwanted recurrence of previous hallucinatory experience days or months after use)</li> <li>depression</li> <li>feelings of isolation</li> <li>tiredness</li> <li>delirium</li> </ul> <p><u>psychosis</u></p> <ul style="list-style-type: none"> <li>result of chronic use – query drug induced condition or unmasking of a latent mental illness</li> </ul>	<ul style="list-style-type: none"> <li>social cognitive dysfunction</li> </ul>
		<p><u>tolerance</u></p> <ul style="list-style-type: none"> <li>develops rapidly to behavioural effects &amp; sensitivity returns after comparable drug free interval, tolerance to cardiovascular effects less pronounced</li> <li>very low dependence potential</li> <li>no withdrawal symptoms</li> </ul>	

Table 8c: Factors that mediate & moderate dangers associated with the use of hallucinogens (LSD, psilocybe eg mushrooms).

HALLUCINOGENS (LSD, psilocybe eg mushrooms)							
FACTORS THAT MEDIATE (INCREASE) OR MODERATE (REDUCE) DANGERS ASSOCIATED WITH HALLUCINOGENS (LSD, psilocybe eg mushrooms) USE							
ROUTE OF ADMINISTRATION PURITY DOSE	COMBINATION USE (concurrent use, consecutive use)	AVAILABILITY	SOCIAL CONTEXT SETTING	AGE DEVELOPMENTAL ISSUES	INDIVIDUAL VULNERABILITY	INCAPACITATION LEGAL SITUATION	
mushrooms • fatal poisoning due to mistaken identity	• not known insufficient evidence base	• seasonal and localised availability of mushrooms • LSD widely available	• risk of injury, accident if intoxicated in dangerous surroundings e.g. river, high building	• not known insufficient evidence base	• psychosis as a result of chronic use – query drug induced condition or unmasking of a latent mental illness	• Misuse of Drugs Act 1971 Class A - lysergide (LSD)	

## Opiates (heroin, methadone) tables

Table 9a: Acute adverse effects associated with the use of opiates (heroin, methadone).

OPIATES (heroin, methadone)	
ACUTE ADVERSE EFFECTS DANGERS OF OPIATES (heroin, methadone) REGARDLESS OF FREQUENCY OF USE	
PHYSICAL	
MORTALITY	MORBIDITY
<p><u>fatal overdose</u></p> <ul style="list-style-type: none"> <li>depression of breathing rate &amp; blood pressure resulting in respiratory arrest</li> <li>most common correlates of overdose are: <ul style="list-style-type: none"> <li>long history of opiate dependence</li> <li>high level of opiate dependence</li> <li>recent abstinence (eg prison, detoxification release)</li> <li>polydrug use (particularly with alcohol and benzodiazepines)</li> <li>being male</li> <li>being older (most fatalities occur in those in their 30's)</li> </ul> </li> <li>little evidence that opiate overdose fatality is strongly linked with drug purity</li> <li>while drug treatment generally provides a protective effect, there is a significantly enhanced risk in the first two weeks of methadone treatment</li> </ul>	<ul style="list-style-type: none"> <li>opiates cause little psychomotor or cognitive impairment in tolerant user</li> </ul> <p><u>common</u></p> <ul style="list-style-type: none"> <li>depressed nervous system activity</li> <li>constipation</li> <li>nausea</li> <li>vomiting</li> <li>drowsiness</li> <li>sedation</li> <li>decreased consciousness</li> <li>mental confusion</li> </ul> <p><u>infrequent</u></p> <ul style="list-style-type: none"> <li>sweating</li> <li>facial flushing</li> <li>pruritus, dry mouth</li> <li>hallucinations, dysphoria</li> <li>urinary retention</li> <li>headache</li> </ul> <p><u>rate</u></p> <ul style="list-style-type: none"> <li>complications associated with non fatal overdose eg hypoxia causing brain damage</li> </ul> <p><u>route specific dangers</u></p> <ul style="list-style-type: none"> <li>use by injection - see table 12</li> <li>smoking - see Table14</li> <li>spongiform encephalopathy – major neurological problems in heroin smokers/chasers not seem to occur in injectors</li> </ul>
PSYCHIATRIC PSYCHOLOGICAL	SOCIAL
<ul style="list-style-type: none"> <li>no acute psychological adverse effects in contrast to other drugs</li> </ul>	<ul style="list-style-type: none"> <li>few acute social adverse effects in contrast to other drugs</li> <li>intoxication may increase risk of causing or being exposed to accidents</li> <li>disinhibition &amp; subjectively enhanced sexual performance can result in increased sexual activity &amp; increased risk of viral infection, sexually transmitted diseases, unwanted pregnancies</li> </ul>

Table 9b: Chronic adverse effects associated with the use of opiates (heroin, methadone).

OPIATES (heroin, methadone)				
CHRONIC ADVERSE EFFECTS DANGERS OF OPIATES (heroin, methadone) THAT ARE CUMULATIVE WITH INCREASED USE				
PHYSICAL		PSYCHIATRIC PSYCHOLOGICAL	DEPENDENCE TOLERANCE WITHDRAWAL	SOCIAL
MORTALITY	MORBIDITY			
<ul style="list-style-type: none"> <li>increased mortality risk from overdose &amp; route specific hazards</li> <li>suicide rate higher than general population</li> </ul>	<ul style="list-style-type: none"> <li>non injected opiates carry little risk of chronic adverse health effects</li> <li>modest suppression of hormone levels</li> <li>suppression of immune system but confounded by social deprivation, malnutrition</li> <li>chronic constipation</li> <li>respiratory complaints</li> <li>menstrual irregularity</li> <li>malnutrition</li> <li>tooth decay</li> <li>decreased sexual desire &amp; performance</li> <li>query over increased risk of miscarriage, fetal death, low birth weight, withdrawal symptoms in newborn, developmental consequences</li> </ul> <p>route specific dangers</p> <ul style="list-style-type: none"> <li>see Tables 12, 13, 14</li> </ul>	<ul style="list-style-type: none"> <li>opiates are NOT causally linked to chronic psychiatric disorders but the following are associated with opiate use:                             <ul style="list-style-type: none"> <li>depressive disorder is common among opiate addicts but difficult to attribute causality</li> <li>instability of mood</li> <li>anorexia</li> <li>lethargy</li> </ul> </li> </ul>	<p>dependence syndrome</p> <ul style="list-style-type: none"> <li>very high dependence potential</li> </ul> <p><u>tolerance</u></p> <ul style="list-style-type: none"> <li>characterised by shortened duration &amp; decreased intensity of central nervous system (CNS) depressant effects, marked elevation in average lethal dose</li> </ul> <p><u>withdrawal syndrome</u></p> <ul style="list-style-type: none"> <li>rarely life threatening</li> <li>dependent on dose, interval between doses, duration of use, physical &amp; psychological health</li> <li>symptoms include – lacrimation, rhinorrhoea, yawning, sweating, sleep disturbance, dilated pupils, anorexia, gooseflesh, restlessness, irritability, tremor, sneezing, weakness, depression, nausea, vomiting, abdominal cramps, pains in bones, muscles, muscle spasms</li> </ul> <ul style="list-style-type: none"> <li>methadone withdrawal qualitatively similar to withdrawal from heroin but develops more slowly, is more prolonged &amp; less intense</li> </ul>	<ul style="list-style-type: none"> <li>poor living conditions</li> <li>poor health &amp; diet</li> <li>disrupted relationships</li> <li>involvement in crime</li> <li>high percentage of violent deaths at the hands of others</li> <li>institutionalisation of opiate dependent clients in methadone treatment                             <ul style="list-style-type: none"> <li>chronically slow, query around substitution treatment prolonging addiction, social poverty, narrowing of repertoire</li> </ul> </li> </ul>

Table 9c: Factors that mediate & moderate dangers associated with the use of opiates (heroin, methadone).

**OPIATES**  
(heroin, methadone)

**FACTORS THAT MEDIATE (INCREASE) OR MODERATE (REDUCE) DANGERS ASSOCIATED WITH OPIATE (heroin, methadone) USE**

ROUTE OF ADMINISTRATION PURITY DOSE	COMBINATION USE (concurrent use, consecutive use)	AVAILABILITY	SOCIAL CONTEXT SETTING	AGE DEVELOPMENTAL ISSUES	INDIVIDUAL VULNERABILITY	INCAPACITATION LEGAL SITUATION
<p>overdose</p> <ul style="list-style-type: none"> <li>increased risk of fatal &amp; nonfatal overdose due to fluctuations in purity of illicit heroin, adulterated illicit methadone</li> </ul> <p><u>fatal anaphylactoid reaction</u></p> <ul style="list-style-type: none"> <li>rare – results from intravenous injection of heroin containing impurities</li> </ul> <p><u>route specific dangers</u></p> <ul style="list-style-type: none"> <li>see tables 12, 13, 14</li> <li>injecting: local problems resulting from injection of methadone linctus or tablets</li> <li>smokin: respiratory complaints eg asthma in heroin chasers/smokers</li> <li>inadequate calculation of dose – either between formulation (eg blue vs green) or from heroin</li> </ul>	<p>concurrent use</p> <ul style="list-style-type: none"> <li>increased risk of overdose if used in combination with alcohol or other CNS depressants</li> <li>cyclozine (an antiemetic present in diconol) used in combination with methadone causes disorientation, gross intoxication</li> <li>interaction between opiate use &amp; use of prescribed drugs – antiepileptic/anticonvulsant &amp; antituberculosis medications decrease the methadone levels in the body – higher dose of methadone required. Conversely, protease inhibitors used in the treatment of HIV/AIDS increase methadone levels in the body – lower dose of methadone required</li> </ul>	<ul style="list-style-type: none"> <li>heroin has decreased in price in the UK</li> <li>methadone is now more readily available on prescription &amp; illicitly in Britain</li> <li>28,776 methadone patients in UK (EMCDDA 1998)</li> </ul>	<ul style="list-style-type: none"> <li>injection of opiates when alone increases the risk of fatal overdose as no one is present to resuscitate or get help</li> <li>methadone's slow onset may induce naive users to increase dose leading to overdose (particularly for polydrug use)</li> </ul>	<ul style="list-style-type: none"> <li>recent increase heroin use young people</li> <li>in settings with limited heroin use or high stigma, methadone can be route to opiate use &amp; injection</li> </ul>	<ul style="list-style-type: none"> <li>possible individual molecular genetic vulnerability to effects of opiates, the risk of dependence, the risk of overdose &amp; sensitivity to harms</li> </ul>	<ul style="list-style-type: none"> <li>tolerance decreases after abstinence &amp; individuals are at increased risk of overdose post treatment or incarceration</li> <li>increased risk of overdose on induction into methadone treatment due to pharmacokinetics of methadone</li> <li>Misuse of Drugs Act 1971 Class A - heroin, methadone</li> </ul>

Table 10a: Acute adverse effects of tobacco consumption.

TOBACCO		
ACUTE ADVERSE EFFECTS DANGERS OF TOBACCO REGARDLESS OF FREQUENCY OF USE		
PHYSICAL		SOCIAL
MORTALITY	MORBIDITY	
<p><u>fatal nicotine toxicity</u></p> <ul style="list-style-type: none"> <li>• rare, occurs in children nonsmokers</li> </ul> <p><u>accidental death</u></p> <ul style="list-style-type: none"> <li>• fires are an important cause of accidental death that may result from careless smoking</li> </ul>	<p><u>Sympathetic overactivation</u></p> <ul style="list-style-type: none"> <li>• Palpitations, sweating, tremor, nausea, dizziness – especially novice users</li> <li>• irritant effects of smoke on respiratory system</li> <li>• oral tobacco use – irritant effects on site of absorption</li> <li>• injury resulting from fires</li> </ul>	<ul style="list-style-type: none"> <li>• social stigma</li> <li>• financial difficulties</li> </ul>
PSYCHIATRIC PSYCHOLOGICAL		
<ul style="list-style-type: none"> <li>• increased anxiety</li> <li>• mood disturbance</li> <li>• increased irritability during periods of enforced abstinence</li> </ul>		

Table 10b: Chronic adverse effects of tobacco consumption.

TOBACCO		CHRONIC ADVERSE EFFECTS DANGERS OF TOBACCO THAT ARE CUMULATIVE WITH INCREASED USE			
MORTALITY	PHYSICAL		PSYCHIATRIC PSYCHOLOGICAL	DEPENDENCE TOLERANCE WITHDRAWAL	SOCIAL
	MORBIDITY				
<p>cardiovascular disease</p> <ul style="list-style-type: none"> <li>• coronary heart disease</li> </ul> <p>cerebrovascular disease</p> <ul style="list-style-type: none"> <li>• blood clots</li> <li>• stroke</li> </ul> <ul style="list-style-type: none"> <li>• peripheral vascular disease</li> </ul> <p>cancers</p> <ul style="list-style-type: none"> <li>• aerodigestive tract – mouth, tongue, throat, oesophagus, lungs</li> </ul> <p>chronic respiratory disease</p> <ul style="list-style-type: none"> <li>• chronic bronchitis</li> <li>• chronic obstructive lung disease</li> <li>• emphysema</li> </ul> <p>accident</p> <ul style="list-style-type: none"> <li>• fires are an important cause of accidental death that may result from careless smoking</li> </ul>	<p>cancers strongly linked to smoking</p> <ul style="list-style-type: none"> <li>• cancer of lung, mouth, pharynx, larynx</li> <li>• cancer of oesophagus, bladder, kidney, pancreas</li> <li>• cancer of stomach, liver, cervix, nose, lip</li> </ul> <p>other diseases linked to smoking</p> <ul style="list-style-type: none"> <li>• chronic obstructive airways disease</li> <li>• pneumonia</li> <li>• myocardial infarction</li> <li>• aortic aneurysm</li> <li>• ischaemic heart disease</li> <li>• peripheral vascular disease</li> <li>• cerebrovascular accidents</li> <li>• peptic ulcer</li> <li>• periodontal disease</li> <li>• osteoporosis</li> <li>• cataracts</li> </ul> <p>minor ailments</p> <ul style="list-style-type: none"> <li>• decreased exercise tolerance weight loss</li> <li>• halitosis</li> <li>• increased susceptibility to coughs &amp; colds</li> <li>• increased signs of aging</li> </ul> <p>reproductive disorders</p> <ul style="list-style-type: none"> <li>• decrease fertility in male &amp; female</li> <li>• smoking in pregnancy – increased risk miscarriage, perinatal mortality, low birth weight</li> </ul>	<ul style="list-style-type: none"> <li>• mood disorders</li> </ul>	<p>dependence syndrome</p> <ul style="list-style-type: none"> <li>• high/moderate dependence potential</li> </ul> <p>withdrawal syndrome</p> <ul style="list-style-type: none"> <li>• craving for nicotine</li> <li>• anxiety</li> <li>• irritability</li> <li>• emotional lability</li> <li>• inability to concentrate</li> <li>• insomnia</li> <li>• increased appetite</li> </ul> <p>tolerance</p> <ul style="list-style-type: none"> <li>• rapid development of tolerance to adverse effects eg nausea</li> <li>• acute tolerance to effects on heart rate</li> <li>• no tolerance to peripheral vasoconstriction</li> <li>• acute tolerance to subjective sensations</li> </ul>	<ul style="list-style-type: none"> <li>• limited evidence - some evidence of stigmatisation leading to loss of esteem and confidence</li> </ul>	

Table 10c: Factors that mediate &amp; moderate dangers associated with tobacco consumption.

TOBACCO							
FACTORS THAT MEDIATE (INCREASE) OR MODERATE (REDUCE) DANGERS ASSOCIATED WITH TOBACCO USE							
ROUTE OF ADMINISTRATION PURITY DOSE	COMBINATION USE (concurrent use, consecutive use)	AVAILABILITY	SOCIAL CONTEXT SETTING	AGE DEVELOPMENTAL ISSUES	INDIVIDUAL VULNERABILITY	INCAPACITATION LEGAL SITUATION	
<ul style="list-style-type: none"> <li>• content of cigarette</li> <li>• use of filters in some cigarettes</li> <li>• increased tobacco use in pipes and "roll-ups"</li> <li>• various as a function of puff intensity</li> </ul>	<ul style="list-style-type: none"> <li>• cigarette smoking is a relapse risk in drinkers</li> <li>• cigarette smoking may have facilitatory effects in opiate addicts</li> </ul>	<ul style="list-style-type: none"> <li>• age limit of 16 on purchase</li> <li>• increased availability and reduced price associated with illegal importation of duty-free tobacco</li> </ul>	<ul style="list-style-type: none"> <li>• widespread smoking among opiate users – 93% among methadone patients and frequent use among drinkers</li> </ul>	<ul style="list-style-type: none"> <li>• early onset of smoking and drinking clearly linked to earlier onset and more regular use of illicit drugs in adolescents</li> </ul>	<ul style="list-style-type: none"> <li>• stimulating effects of nicotine on cardiovascular system can be detrimental to persons with cardiovascular or respiratory disease</li> </ul>	<ul style="list-style-type: none"> <li>• NOT controlled under Misuse of Drugs Act 1971</li> </ul>	

## Volatile substances tables

Table 11a: Acute adverse effects associated with the use of volatile substances.

<b>VOLATILE SUBSTANCES</b>			
<b>ACUTE ADVERSE EFFECTS DANGERS OF VOLATILE SUBSTANCES REGARDLESS OF FREQUENCY OF USE</b>			
<b>PHYSICAL</b>		<b>PSYCHIATRIC PSYCHOLOGICAL</b>	<b>SOCIAL</b>
<b>MORTALITY</b>	<b>MORBIDITY</b>		
<ul style="list-style-type: none"> <li>• toxicity varies greatly with the specific substance &amp; causes of fatalities are unclear:</li> <li>• most fatalities involve cardiac arrhythmia or accident (falls, drowning, fire)</li> <li>• individuals may lose consciousness &amp; die through choking on vomit</li> <li>• danger from suffocation if place plastic bag over head to inhale</li> <li>• intense cooling in mouth caused by squirting lighter fuel down throat may result in laryngeal spasm blocking airways &amp; causing death by asphyxiation</li> </ul>	<ul style="list-style-type: none"> <li>• adverse effects vary greatly with the specific substance &amp; mode of administration:</li> <li>• flushed face &amp; neck</li> <li>• cold sweats</li> <li>• loss of balance, unsteadiness, lack of co-ordination</li> <li>• fainting</li> <li>• headache</li> <li>• nausea, vomiting</li> <li>• confusion, dizziness, disorientation</li> <li>• tachycardia, palpitations</li> <li>• drowsiness, sedation, unconsciousness</li> <li>• risk of accidental injury while intoxicated</li> </ul> <p style="text-align: center;">route specific dangers</p> <ul style="list-style-type: none"> <li>• see Table 14</li> </ul>	<ul style="list-style-type: none"> <li>• confusional states, disorientation</li> <li>• distorted perceptions, delusions, hallucinations, pseudohallucinations</li> <li>• aggression, agitation, fear</li> </ul>	<ul style="list-style-type: none"> <li>• accident (road traffic, swim, fire, falls)</li> <li>• disinhibition, engaging in high risk behaviour (dangerous driving, unsafe sexual practices), victim of crime</li> <li>• acute intoxication possibly resulting in aggressive &amp; violent behaviour, disorderly conduct</li> <li>• relationship problems</li> <li>• impairment of educational achievements in adolescents</li> </ul>

Notes: The most widely used volatile substances are glues, thinners, aerosols, paints & lighter fuel.

Volatile substances are unique amongst substances of abuse as the main users are children & adolescents (10 – 18 years of age). The exception to this is the nitrites, which tend to be used by a different population (gay men aged 18+ in club settings or as sex aid). Nitrites are therefore not included in the following table.

Table 11b: Chronic adverse effects associated with the use of volatile substances.

VOLATILE SUBSTANCES				
CHRONIC ADVERSE EFFECTS DANGERS OF VOLATILE SUBSTANCES DRUGS THAT ARE CUMULATIVE WITH INCREASED USE				
PHYSICAL		PSYCHIATRIC PSYCHOLOGICAL	DEPENDENCE TOLERANCE WITHDRAWAL	SOCIAL
MORTALITY	MORBIDITY			
<ul style="list-style-type: none"> <li>no evidence available</li> </ul>	<p><u>chronic medical problems</u></p> <ul style="list-style-type: none"> <li>no consistent pattern unclear why some suffer and others not</li> </ul> <p><u>adverse effects reported</u></p> <ul style="list-style-type: none"> <li>peripheral and central neurological damage</li> <li>renal failure</li> <li>hepatotoxicity</li> <li>severe gastrointestinal upset</li> <li>muscle damage</li> <li>very long term (e.g. 10 years) solvent misuse might result in lasting impairment of brain function affecting especially control of movement</li> </ul> <p><u>substance specific</u></p> <ul style="list-style-type: none"> <li>petrol - lead poisoning</li> </ul>	<ul style="list-style-type: none"> <li>decreased ability to concentrate</li> <li>insomnia</li> <li>nightmares</li> </ul>	<p><u>tolerance</u></p> <ul style="list-style-type: none"> <li>develops within 2-3 weeks of continual use but is lost after a few days of abstinence</li> </ul> <p><u>dependence</u></p> <ul style="list-style-type: none"> <li>low dependence potential</li> </ul> <p><u>withdrawal syndrome</u></p> <ul style="list-style-type: none"> <li>irritability</li> <li>headaches</li> </ul>	<ul style="list-style-type: none"> <li>crime - theft of volatile substances or money to buy volatile substances</li> <li>accident (road traffic, swim, fire, falls)</li> <li>disinhibition, engaging in high risk behaviour (dangerous driving, unsafe sexual practices), victim of crime</li> <li>acute intoxication possibly resulting in aggressive &amp; violent behaviour, disorderly conduct</li> <li>relationship problems</li> <li>impairment of educational achievements in adolescents</li> </ul>

Table 11c: Factors that mediate & moderate dangers associated with the use of volatile substances.

VOLATILE SUBSTANCES						
FACTORS THAT MEDIATE (INCREASE) OR MODERATE (REDUCE) DANGERS ASSOCIATED WITH VOLATILE SUBSTANCE USE						
ROUTE OF ADMINISTRATION PURITY DOSE	COMBINATION USE (concurrent use, consecutive use)	AVAILABILITY	SOCIAL CONTEXT SETTING	AGE DEVELOPMENTAL ISSUES	INDIVIDUAL VULNERABILITY	INCAPACITATION LEGAL SITUATION
<p>route specific dangers</p> <ul style="list-style-type: none"> <li>oral (inhalation, swallowing) - see Table 14</li> </ul>	<ul style="list-style-type: none"> <li>use with alcohol and other central nervous system (CNS) depressants will bring increased risk of breathing asphyxiation and death</li> </ul>	<ul style="list-style-type: none"> <li>volatile substances widely available in the household &amp; shops (newsagents, chemists, supermarkets)</li> </ul>	<ul style="list-style-type: none"> <li>risk of injury when consumed alone in potentially dangerous locations - eg near water (risk of drowning), on a high building (risk of falls)</li> <li>if an individual suffers an arrhythmia whilst alone may result in fatality as no one is present to perform resuscitation</li> </ul>	<ul style="list-style-type: none"> <li>volatile substances are unique amongst substances of abuse as the main abusers are children and adolescents (10 – 18 years of age)</li> </ul>	<ul style="list-style-type: none"> <li>co-morbidity</li> <li>neurocognitive deficits</li> <li>poor general physical health</li> <li>increased risk of involvement with problem alcohol and tobacco use</li> </ul>	<ul style="list-style-type: none"> <li>NOT controlled under Misuse of Drugs Act 1971</li> </ul>

## **Strengths & limitations of tabular approach**

The tables above provide a comprehensive assessment of the main categories of danger that apply to the target drugs selected. This permits a certain amount of comparability between drugs as the probability of the main classes of danger is indicated for each of the drugs listed – thus, while there is a high dependence risk and mortality danger among opiate users, there is little evidence to suggest chronic psychiatric problems in this population.

In contrast, no attempt has been made to rank order the target substances, even within each of the domains specified. This is because the dangers are not uni-dimensional nor do they generally occur in isolation. The purpose of following each list of drug dangers with a list of mediating and moderating effects is to avoid over-simplifying the dangers associated with substances and also to suggest some of the areas that may be appropriate for intervention to reduce dangers among those who may be unwilling to stop their use of a particular drug.

However, there are fundamental limitations to this approach – drugs are not, of themselves, dangerous, with the risk residing in the interaction between the substance, the individual, the method of consumption and the context of use. Among the main variables that will shape risk relating to substance are amount and purity, mediated by physiological and psychological factors in the user (such as tolerance, expectation and body mass), whether the drug is swallowed or injected or whether it is used in a safe and familiar environment. While it is not possible to list all of these possible combinations for each of the drugs (and more crucially for all of the possible drug interactions), the public health anxieties around blood-borne diseases would suggest that paying some attention to the issue of route of drug administration may be illustrative.

## **Route specific dangers**

One of the key variables mediating dangers is route of administration. Substances that are injected are associated with risks for blood-borne diseases, not only HIV, but also Hepatitis B and Hepatitis C. Use by injection is also associated with local site damage including skin abscesses, gangrene and lymphoedema. In addition, these problems can be exacerbated by both adulterants in the drug and by poor injecting technique, possibly resulting in deep vein thrombosis. Furthermore, chronic injecting can lead to vein loss resulting in the use of

particularly dangerous injecting sites such as the groin or neck. Thus, one possibility for intervention is to persuade entrenched users to switch to a different route of administration.

Although less dangerous than injecting, there are health problems associated with the other main routes of administration. Intranasal use (snorting) is associated with impaired breathing, nosebleeds and ulceration or inflammation of nasal mucosa, while swallowing of tablets may result in the longer term in liver damage. The key here may be education about the effects of use by different methods (see Tables 12-14 below):

Table 12: Adverse effects of the use of drugs by injection.

### **Route Specific Adverse Effects Use of Drugs by Injection**

#### **Local infections (skin and injection site)**

skin abscess, cellulitis, necrotising fasciitis, gangrene, septic thrombophlebitis, lymphoedema caused by adulterants/contaminants in street drugs, clumsy or unhygienic injecting technique.

#### **Bacterial infections leading to distant problems**

joint & bone infections osteomyelitis, septic arthritis septicaemia (possible consequence – endocarditis).

#### **Viral infection from sharing contaminated injecting equipment**

HIV, hepatitis B, hepatitis C.

#### **Blood vessel occlusion**

Inert adulterants used to dilute drug eg non soluble particles such as talc, starch and chalk may not all be removed by filtration & may become microemboli in the bloodstream. These particles may form granulomas in the lung which may impair gaseous diffusion giving rise to dyspnoea, hypoxia, pulmonary hypertension or emphysema. Embolisation of insoluble particles can also cause retinopathy (accumulation of obstructive particles in retinal blood vessels and impairment of sight can occur) & thrombus formation (deep vein thrombosis).

#### **Stigmata**

Repeated intravenous injection over a prolonged period of time may cause needle marks, scarring due to abscesses, bruising, discolouration of skin along lines of veins due to insoluble particles accumulating within the skin.

### **Rare adverse effects**

#### **Injection of air embolus**

Potential hazard leading to heart inefficiency & possible failure but difficult to achieve via hand held syringe (unlikely at street level).

#### **Intra arterial injection of irritant substances containing solid particles**

Swelling distal to injection site, pain, discolouration, sensory and motor deficit. Subsequent pattern of events will depend on the site of injection & the tissues affected eg thrombosis of digits leading to gangrene, deep vein thrombosis, haemorrhage.

#### **Direct irritant effects of drug**

Most drugs are not themselves irritant, the exception being temazepam which causes irritation of tissues or veins after injection leading to abscess, tissue necrosis, venous fibrosis, phlebitis. Irritant effects of injectable preparations are largely attributed to adulterants or additives eg citric acid to aid dissolution in heroin, ammonia in crack cocaine. Other potentially irritant adulterants in street drugs include quinine & sodium bicarbonate. Talcum powder may be used as cutting agent in heroin & damages organs where it collects eg talc lung.

### **Substance specific & route specific adverse effects**

#### **Temazepam gel capsules**

Temazepam gel (no longer prescribed in the form of gel-filled capsules in the UK) may solidify in blood vessels after injection causing ischaemia &/or act as a focus for thrombus formation. Temazepam is insoluble and solid particles may cause vascular blockade via microembolism. Severe rhabdomyolysis has been described necessitating fasciotomy or limb amputation and causing renal failure. Other effects include deep vein thrombosis, pulmonary embolus and critical ischaemia of digits leading to amputation.

#### **Cocaine**

Cocaine injectors may be at particular risk as the drug has local anaesthetic properties which can mask the pain of damage.

#### **Anabolic-androgenic steroids**

steroid users may have additional risks regarding injection as intramuscular injection is administered in the buttocks out of sight of the user therefore their technique may be more clumsy and increase chances of infection. In addition, the chance of sharing injecting equipment may be increased as larger bore needles are needed for intramuscular injection of these viscous solutions & these may not be as easily available as the narrower gauge intravenous varieties.

Table 13: Adverse effects associated with intranasal use of drugs.

### **Route Specific Adverse Effects Intranasal Use (Snorting)**

Damage to nasal passages leading to:

- impaired breathing
- minor nosebleeds
- irritation & possible perforation of nasal septum
- ulceration of nasal mucosa
- vasoconstriction of mucous membranes & subsequent vasodilation sometimes causing rhinitis (inflammation of mucous membrane)
- dental erosion if substance snorted through nose then into mouth
- local anaesthetic effect of cocaine resulting in difficulty swallowing.

Table 14: Adverse effects associated with oral use of drugs.

### **Route Specific Adverse Effects Use of Drugs by Oral Route (Swallowing, Smoking, Inhalation)**

#### **Cannabis**

irritant effects of smoke on respiratory system (cough, sore throat, bronchospasm in asthmatic people).

#### **cocaine**

general respiratory problems due to vasoconstrictive effects, coughing, wheezing, chest pain, black sputum, lung damage.

Risk of acute death higher for crack cocaine than cocaine hydrochloride due to rapidity of onset – route specific smoking.

#### **volatile substances**

intense cooling in mouth, throat may cause laryngeal spasm blocking airways & subsequent death by asphyxiation.

Inhalation of volatile material from a plastic bag may result in hypoxia and neurological impairment.

Irritant properties of solvents can produce erythema around mouth & nose & inflammation of perioral abrasions or spots, coughing, lacrimation & salivation.

#### **opiates (heroin, methadone)**

respiratory problems may result from chasing heroin eg asthma spongiform encephalopathy – extremely rare major neurological problems in heroin smokers/chasers not seem to occur in injectors

### Reading the substance specific tables

The tables are designed to provide both overviews about the types of difficulty that are associated with experimental or regular use of a range of drugs, classified according to a number of key domains of risk. They represent a reference guide for establishing the main types of health, psychological and social problems that are likely to follow from different patterns of use. Here it is possible to examine main effects according to drug type, frequency of use and the primary domain influenced by the use.

The following table (c, for each of the substances) attempts to indicate why other factors – associated with either the users or the context of use – are likely to shift the risks outlined in the two tables above. The authors sought to compromise in the complexity of the information provided so that general overview by substance could be interpreted by the reader in terms of the riskiness associated with the individual users and the factors that surround the using occasion.

As is evident from the tables, there are different profiles of danger across the classes of substances, which partly reflects 'real' differences between the drug classes but which is also indicative of the different priorities for investigation and the amounts of information available about certain drugs. For some drugs, like the new synthetic drugs this is because of their relative youth, but for others, like the hallucinogens and volatile substances, it is a result of the low priority accorded them by research scientists and policy-makers alike. For other substances, like anabolic steroids and benzodiazepines, the situation is complicated by the bifurcation in use patterns, with differing patterns of 'danger' for those who chronically use at therapeutic dose levels and those who use, for shorter periods of time but use massively increased doses. For example, it is not unknown for bodybuilders to use 200 times the recommended dose of an anabolic steroid.

In order to gain an accurate picture of the potential dangers associated with use of certain substances, we also require a probability risk estimation to assess the likelihood of an adverse effect occurring in any one individual. This is the area in which prevalence of use impacts upon the salience of certain types of danger. Hall (1999) highlighted the fact that the danger of a drug is related to both the prevalence of its use and the likelihood of any harms.

### **An alternative approach – ranking the dangers**

In their chapter in “The Health Effects of Cannabis” (Kalant et al, 1999), Hall, Room and Bondy undertake a comparison of the health and psychological risks of alcohol, cannabis, nicotine and opiates. They do however point out a number of limitations with this approach:

1. difficulties in making causal inferences about the use of a drug and adverse effects.
2. lack of information about the extent or seriousness of drug risks.
3. the difficulties of making comparative appraisals of the public health significance of identified risks.
4. the recognition that different drugs are used in different ways.
5. the difficulty of predicting the consequences of changes in either the prevalence of use of specific drugs or in their routes of administration.

Their first summary was of the “main adverse affects of regular heavy use of the most harmful form of each type of drug, as commonly used for non-medical purposes” (p487). They did this firstly on the basis of a literature review, differentiating between important effects (in terms of number of heavy users affected, marked as \*\*) or those effects that are less well established or less important numerically (marked as \*), see Table 15:

**Table 15: Hall et al (1999) assessment of comparative adverse effects for heavy users of the most harmful form of alcohol, nicotine, opiates and cannabis.**

	Cannabis	Alcohol	Tobacco	Heroin
Traffic and other accidents	*	**		*
Violence and suicide		**		
Overdose death		*		**
HIV and liver infections		*		**
Liver cirrhosis		**		
Heart disease		*	**	
Respiratory disease	*		**	
Cancer	*	*	**	
Mental illness	*	**		
Dependence/addiction	**	**	**	**
Lasting effects on the foetus	*	**	*	*

A second tier of assessment was carried out by asking two American experts, Neal Benowitz and Jack Henningfield, to rate the four substance types on five dimensions related to the capacity of each drug to produce addiction and casualties (Hilts, 1995). In Table 16 below, the lower the score, the greater the likelihood comparatively (ie 1 is the most likely to lead to this problem and 4 the least).

**Table 16: Comparative ratings of the dependence potential of cannabis, alcohol, tobacco and heroin (Hall et al, 1999).**

	Cannabis	Alcohol	Tobacco	Heroin
Presence and severity of withdrawal symptoms	4	1	3	2
Reinforcement: Capacity to get users to use again and again	4	2	3	1
Tolerance: How much more needed by a regular user for the same effect	4	3	2	1
Dependence: Difficulty quitting and avoiding relapse: perceived need to use	4	3	1	2
Intoxication: Impairment of motor abilities, distortion of thinking and mood	3	1	4	2

In the table above cannabis is rated as having the lowest ‘addictive’ potential on four of the five criteria identified, with heroin most strongly linked to reinforcement and tolerance, tobacco to dependence and alcohol to intoxication and withdrawal severity.

### Prevalence issues

When interpreting prevalence statistics we must remember that consumption of illicit drugs is not spread uniformly across age groups. The years between the ages of 16 and 35 are consistently found to be the peak periods for illegal drug consumption. In fact, above the age of 35 experience of illicit drugs is reasonably rare. If we look at prevalence statistics for the general population in the younger age groups we find elevated levels of use of all of the target illicit substances (see figures 1 - 4). However, this age effect is not apparent when looking at prevalence rates of alcohol and tobacco use (see figures 5 & 7). Use of these substances remains at a fairly constant level across age groups, although quantity of alcohol consumed decreases with age (see figure 6).

The British Crime Survey 1998 calculated population estimates of number of 16-29 and 16-24 year olds using selected drugs in last year and last month. Although the basis for the calculation of these estimates may be challenged (in particular, the grouping together of heroin, methadone, cocaine and crack), this kind of information provides us with a platform against which to assess a number of the critical parameters of danger.

**Table 17: 1998 estimates of number 16-29 year olds using drugs England & Wales.**

Substance	Best Estimates	
	Last Year	Last Month
cannabis	2,390,000	1,455,000
opiates +	310,000	105,000

Notes:

Total number of 16-29 year olds in England and Wales 1998 = 10,400,000  
The category opiates + includes heroin, methadone, cocaine and crack cocaine.

It would be extremely important to the completion of an assessment of dangerousness to have accurate prevalence and patterns data, against which to judge the prevalence of adverse outcomes and to establish both age- and quantity-related parameters for harm. However, our attempts at assessing prevalence rates are beset by both logical and methodological issues (what is the appropriate time period for assessment, the reliability of self-report, the adequacy of information sources and so on) that this information can be regarded as indicative at best. For this reason, our understanding of the rate of occurrence of each adverse outcome is limited by a failure of contextualisation – we cannot know the frequency of non-harmful preceding behaviours against which to measure, compare and assess. This has led to the use of triangulated methodologies in an effort to overcome these problems and a reliance on ‘objective’ measures such as fatality rates as a method of grounding these ephemeral and imprecise prevalence estimates.

### Data on drug-related deaths

One of the questions that concerns policy makers, parents and users alike relates to the most extreme form of use – namely, what are the chances of death resulting from use. Drug related

mortality measures one of the more extreme consequences of drug use, but one that seems relatively free from measurement problems. There are however, two problems in considering death data – one that relates to cause and one that relates to attribution. The causal question results from the distance of time between cause and the effect – if an individual’s heart is weakened by chronic heavy drinking and they die from a heart attack, it is not obvious whether or not this is a ‘alcohol-related’ death. This has been the subject of much politicised debate around smoking mortality. A second, related issue, concerns the proximal attribution of the death – thus, in the case of overdose, the death may be recorded as a heroin death in spite of the presence of excessive quantities of alcohol or benzodiazepines. Thus, even death data must be considered in terms of the recording practices employed.

All deaths in England and Wales which are sudden, unexpected or not natural and those for which the cause is unknown must be referred to a coroner for further investigation. The coroner’s function is to establish the circumstances and cause of death. The coroner orders a post mortem to be conducted, collects information on the deceased from a variety of sources (police, medical records, relatives, friends, witnesses) and gives a verdict on the cause of death. The coroner’s certificate is sent to registrar of births and deaths who register the death using the information provided on the coroner’s certificate. The Office for National Statistics (ONS) receives a copy of the information on this registration form and uses this to compile their database on drug related deaths.

### **Box 3: Problems interpreting drug related mortality data.**

- the deceased may be long term addict or occasional recreational user
- death may be accident, suicide or possibly homicide
- death may be due to direct, indirect or long term effects of drug use
- dependent drug use is not always recorded as cause of death in situations such as where drug addict dies in fire, road traffic accident, of viral infection (HIV, hepatitis)
- drugs involved may be controlled drugs, prescribed substances or a mixture
- the drug may not be detected at post mortem or recorded on death certificate
- whether a drug is detected may depend on which part of body sample is taken from
- whether a drug is detected may depend on how soon after death post mortem is carried out

- there is much variation between coroners in facilities, resources and workloads
- what is recorded as verdict/cause of death is at the discretion of coroner (drug use may be omitted for relatives stigma).

Coroners certificates do not include information on:

- how or where the drugs were obtained, quantities of drugs involved, where the drugs were taken, route of administration
- whether a toxicological exam was carried out within the post mortem.

Problem of attribution given polydrug using repertoires. When more than 1 substance has been used:

- only those drugs which are tested for will be detected
- no indication of relative quantities is provided
- no indication is given of which substance is likely to be responsible for the death
- if both substances are mentioned on the death certificate, the death is recorded more than once under each substance.

**Table 18: Number of deaths where target substance mentioned on death certificate. Source: Office for National Statistics database on drug related deaths.**

SUBSTANCE	ANNUAL NUMBER DEATHS 1997
ALCOHOL	28,000 over 3000 cases alcohol specified on death certificate Source: Alcohol Concern
AMPHETAMINES	40
ANABOLIC STEROIDS	no figures available
BENZODIAZEPINES (temazepam, diazepam)	temazepam 104 diazepam 122 nitrazepam 14
CANNABIS	13
COCAINE HYDRO- CHLORIDE (cocaine powder)	cocaine 38
FREE BASE COCAINE (crack/rock cocaine)	no figures available
AMPHETAMINE TYPE STIMULANTS & NOVEL SYNTHETIC DRUGS (ecstasy)	ecstasy 11
HALLUCINOGENS (LSD, magic mushrooms)	1
VOLATILE SUBSTANCES	78
TOBACCO	120,000 Source: Action on Smoking & Health ASH,1997
HEROIN	255
METHADONE	421
HEROIN AND/OR MORPHINE	445

Note: Figures quoted for heroin or morphine deaths (441) may contain heroin deaths due to rapid decay of heroin into morphine in body. Death data for drugs refers to drugs present in bloodstream at post-mortem examination and therefore cannot be assumed to be causes of death, at least in a direct sense.

### An alternative approach to risk: 'capture rates'

The issue of the relative impact of prevalence of use on danger is the basis for the capture rate approach. Although it is important to know prevalence, it is just as important to be able to work out how many of those who try a drug will go on to use it regularly or to become dependent on it – the 'capture rate' for a drug. Much of this information comes from the American National Comorbidity Survey (Anthony, Warner and Kessler, 1994). In a national household survey, they asked about lifetime use and lifetime dependence for a range of psychoactive substances. An estimated 24% of the total sample had developed tobacco dependence at some point in their lives, 14% alcohol dependence and 7% dependence on an illicit drug. However, significantly more people had used alcohol or tobacco than had ever used illicit drugs. Therefore, the authors also calculated the proportion of those who had ever used a drug who had gone on to develop dependence (see table 19 below):

**Table 19: Prevalence, dependence & 'capture' rates by target substance.**

Drug	Proportion who have used %	Proportion who have developed dependence %	Proportion of dependence among users %
Tobacco	75.6	24.1	31.9
Heroin	1.5	0.4	23.1
Cocaine	16.2	2.7	16.7
Alcohol	91.5	14.1	15.4
Cannabis	46.3	4.2	9.1

Although alcohol is most commonly used, transition from use to dependence for alcohol is relatively low. In contrast, almost one third of those who have ever smoked a cigarette and almost all of those who have ever tried heroin have gone on to become dependent. In contrast, while almost half of those surveyed have tried cannabis, less than 10% of these have gone on to become dependent. What this would suggest is that tobacco has the greatest potential for dependence followed by heroin, then cocaine and alcohol. Cannabis has the lowest 'addictability' of all the drugs listed above.

However, the capture rate approach may be slightly misleading in that it assumes that the people who have ever tried heroin

are the same as the people who have ever tried alcohol so that the capture score is a property of the drug and not of the user. Yet we know that being offered drugs in adolescence has been associated with poor neighbourhoods (Crum et al, 1996), with divorced parents (Grady et al, 1986) and with prior use of alcohol or tobacco (Stenbacka et al, 1993). For this reason, the frequency of the shift from experimentation to dependence reflects not only the addictiveness of the drug but the characteristics of those who are willing to experiment with it.

Yet the capture rate approach is a particularly promising method for those who wish to study the longitudinal dangers associated with a range of substances. Thus, there is no reason why this approach should be restricted to the relationship between experimenters and dependent users. An entire capture risk chain could be, in principle, calculated in which the start point is the first time the drug is offered to an individual, followed by first use, then regular use, then dependent use, and so on. Similarly, a capture equation could be made for first use to particular forms of morbidity and mortality. This method would permit an actuarial approach in which 'hit rates' could be calculated for substance effects according to the requirements of the policy makers.

### **General developmental issues & danger**

The second implication of the capture rate approach is that it suggests that drug dangers can be characterised as aspects of drug-using careers. This approach, borrowed from criminology, suggests that many young people will start using drugs and committing crimes in the early teenage years, their use will peak in late adolescence and generally decline through their early twenties. Studies have shown that those convicted at an earlier age (10-16 years) tended to be the most persistent offenders, commit the most offences and have longer criminal careers (Home Office Statistical Bulletin, 1987; Tracy and Kempf-Leonard, 1996; Farrington and Maughan, 1999).

The developmental approach assumes that, across populations, there are predictable patterns of deviant careers in which most people will flirt with delinquent behaviours (such as drug use) during adolescence, but will 'grow out' of these in early adulthood. This will be true for all but around 5% of young delinquents, who will develop long-term and serious problems associated with their adolescent delinquency. In contrast, most adolescents will go through a brief spell of independence-assertion, called 'adolescent-limited delinquency', during which they will reject the value system of their parents. This will lead

to a period in which deviance is valued, petty crime committed, where excessive drinking is commonplace and where recreational drug use occurs. In general, early adulthood signals the end of this period, with employment and marriage the most frequent catalysts.

Thus, there is a danger period for two aspects of substance use – initiation and escalation that are not independent of each other. The developmental approach has generally involved a consideration of ‘risk’ factors as the key determinants of harm or danger. These include background characteristics such as parental drug use and family income, anti-social personality, low intelligence and other factors that may increase the risk of all kinds of lifetime problems. On the other hand, there are also contemporary-contextual factors that influence the decisions made here and now about whether to use a drug. These may include availability, opportunity, peer influence and expectancies about what the drug will do. This distinction allows us to incorporate both general factors that will shape risk-taking behaviour across the life course with factors that will determine the outcome of a particular risk situation.

### **General discussion**

The first point to make here is that the dangerousness of an individual substance is difficult to abstract from the context of its use – a context that is likely to include the individual taking the drug, their expectations and beliefs about the drug, the society that defines these beliefs and the likelihood of sanctions and the state of the individual at the time of consuming the drug. This state will reflect not only predisposition (biological and psychological) but other forms of substance use that have been engaged in before or at the same time as the target drug.

The classification that has been provided in the central drug tables has adopted a *ceteris paribus* assumption that is only viable when making generalisations of this sort. These are only of background utility to the practitioner who is faced with a real user in a dangerous situation. However, this does not mean that there is no benefit to taxonomies of this sort – they form the basis on which actuarial calculations of the likelihood of particular negative outcomes can be calculated. The dangerousness of a drug cannot be generalised across all situations – the criterion specified and the method of calculating both the likelihood of and the extent of the negative outcome clarified so that it is consistent with the objectives of the policy-maker initiating the assessment of danger.

This is why the capture rate approach offers such a potentially useful method of calculation. As it requires the clear specification of the outcome (e.g. mortality rate) and the delineation of the calculation criteria (e.g. as a proportion of all problem users or lifetime users), the comparability between populations and between substances is less evidently skewed. Similarly, for the policy-maker attempting to assess the adequacy of the statistical information available, it permits a clear delineation of the gaps in the data necessary to make this form of calculation.

A further point this raises is about the temporal aspect of measurement. If it is accepted that the dangerousness of a drug is not exclusively a function of the pharmacological properties of the substance, then there are likely to be ephemeral factors (such as availability and purity) that will influence the likelihood of particular negative outcomes. The ability to measure shifts in these danger outcomes is also crucial to understanding shifting risk patterns, the efficacy of public health interventions and changing patterns of drug use. To this end it is critical not only that measures of dangerousness are maximised, it is also crucial that they are obtained consistently across time.

### **Implications**

Considerable work is required to provide an adequate answer to some of the problems of assessment set out in this project – some of which are epidemiological and some of which are under-pinned by limitations in our current knowledge of the ways in which drugs are used. However, there are some intrinsic logical issues that prevent clear delineation of risks by substance use:

- a. factors related to the substance – in particular, the quantity and purity of the drug consumed.
- b. how this relates to factors in the consumer – their physiological frame and state, their history of consumption and consequences for tolerance, and psychological factors including expectations and psycho-adaptation to the drug. Individual factors will also be mediated by ‘career’ variables including age and developmental state as well as other use forms.
- c. combination use – the concurrent or consecutive use of several drugs both within and across drug classes provides an enormous confounding effect on the prediction of effects.

d. route of administration – while it is generally acknowledged that use by injection carries the most immediate risk it should not be assumed that other routes – smoking and swallowing in particular – are without hazards.

What this implies is that the actuarial calculation of risk associated with any given substance is a multi-faceted assessment embedded within the typical use patterns and circumstances commonly undertaken in particular societies. This is partly a reflection on both societal and sub-cultural beliefs and preferences, but will also be impacted upon by the legal framework within which use occurs. Thus, there is a fundamental dysjunction between the risks associated with readily available legal drugs such as tobacco and alcohol, and the illicit drugs, for which a criminal justice component is inherent in the profiling of dangers. Thus, while swallowing cocaine may be generally less harmful than its injection, this is not the case for the cocaine dealer who swallows a package to avoid criminal detection.

What has been outlined above is an attempt to provide a basic matrix in which the more commonly occurring effects are presented for the main psychoactive drugs along two dimensions – acute versus chronic and physical versus psychosocial. This is no more than an illustrative guide based on the experiences and acquired knowledge of academics and clinicians and is therefore biased in the direction of medical and psychiatric effects. As with the Hall et al (1999) review, the current project does not attempt to enumerate the positive effects that may be associated with substance use, although it readily acknowledges that part of the risk relates to this reinforcing quality and the ‘functionality’ of much substance use.

The guide provided, particularly in the tables, reflects a summary of the published literature and the views of the key informants, but each of these sources of information is restricted by the limitations in our knowledge base. Perhaps the most important of these is epidemiological in that, without baseline levels of use reporting, we do not have adequate denominators against which to assess the risk probabilities of adverse event occurrence. There is considerable scope for building on this work by improving our understanding of the ‘why’ questions at a social, psychological and anthropological level, of developing our clinical and physiological awareness of impact and of using these factors as hazard predictors. However, this is a huge task that assumes societal stasis and, for this reason, is unrealistic. It is hoped that, within a relatively short period of time, our

research technologies and their assimilation within the broad multi-disciplinary frame of addiction science will significantly improve on the data presented here. This can never be a precise science and individual variation will inevitably confound our attempts at precision. This does not mean that the attempt is not worthwhile and it is the bases of our approximations that will decide the viability of such risk assessments.

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