



Home Office

**MDMA ('ecstasy'):
A REVIEW OF ITS HARMS
AND CLASSIFICATION UNDER
THE MISUSE OF DRUGS ACT
1971**

Advisory Council on the
Misuse of Drugs

ACMD

Advisory Council on the Misuse of Drugs

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February 2008

Rt Hon Jacqui Smith MP
Home Office
2 Marsham Street
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Dear Home Secretary,

The Advisory Council on the Misuse of Drugs (ACMD) recently considered that a review of MDMA ('ecstasy') would be timely as there is a much greater body of evidence regarding the harms and misuse of MDMA since the Council last provided its advice to Ministers in 1996. I have pleasure in enclosing the Council's report.

The use of MDMA is undoubtedly harmful. I would therefore like to emphasise that the Council continues to be concerned about the widespread use of MDMA; particularly among young people.

Due to its prevalence of use, MDMA is a significant public health issue and we believe that criminal justice measures will only have limited effect. You will wish to note that the Council strongly advises the promulgation of public health messages. It is of vital importance that issues of classification do not detract from messages concerning public health.

Forensic evidence shows that MDMA is by far the most commonly seized of the 'ecstasy-like' drugs. MDMA is presently generically classified in Class A under the Misuse of Drugs Act with other 'ecstasy-like' drugs. The ACMD has not extended this review to other compounds within the generic classification since their use is considerably less than that of MDMA.

In reviewing the evidence of the harmfulness of MDMA to individuals and society, the Council's collective view is that the balance of harms most closely equates to that of other substances in Class B.

Despite the current generic definition of 'ecstasy-like' drugs, it is not envisaged that the Council's recommendation concerning the classification of MDMA and subsequent changes to the legislation would be difficult to enact. For example, in New Zealand, MDMA was previously generically classified, but MDMA has recently been re-scheduled in a lower classification. This model retains other 'ecstasy-like' compounds in the higher classification.

The report includes a number of research recommendations. The Council believes that the outcomes of research commissioned in these areas will positively contribute to the evidence base for the development of specific policies for tackling the misuse of MDMA. We applaud the on-going development of the cross-government drugs research strategy and hope that these recommendations will contribute to its development.

The production of this report has been greatly aided by valuable contributions from a wide range of organisations and experts. The Council is particularly grateful to those experts who provided written and oral evidence.

Yours sincerely,

A handwritten signature in black ink, consisting of a large, stylized initial 'D' followed by a series of smaller, connected loops and a final horizontal stroke.

Professor David Nutt FMedSci
ACMD Chairman

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Preamble

3,4-Methylenedioxyamphetamine (MDMA), colloquially known as 'ecstasy', is a Class A drug, the use of which increases the feeling of euphoria and induces a heightened sense of intimacy with others.

The Advisory Council on the Misuse of Drugs (ACMD) considers that a review of MDMA would be timely given: that there is a greater body of scientific evidence now available since the ACMD's last advice to Ministers; the further information available on current use; and the length of time since the ACMD last provided advice to Ministers.¹ Indeed, a recent inquiry by the House of Commons Science and Technology Select Committee into the Government's handling of scientific advice, risk and evidence in policy making (House of Commons Science and Technology Select Committee, 2006) recommended that the ACMD should use the now expanded knowledge base to review the classification of 'ecstasy' under the Misuse of Drugs Act 1971. This report represents the output of a review of MDMA which was conducted by the ACMD during 2008.

1 1996 ACMD advice to Ministers regarding 'ecstasy'.

1. Background

- 1.1** The Advisory Council on the Misuse of Drugs (the Council) is established under the Misuse of Drugs Act 1971. The Council's current membership is shown in Annex A. Additional experts also attended the Council's meetings to assist in the preparation of this report (Annex B).
- 1.2** The Council is required under the Misuse of Drugs Act 1971 *"to keep under review the situation in the United Kingdom with respect to drugs which appear to them likely to be misused and of which the misuse is having or appears to them of having effects sufficient to constitute a social problem"*.
- 1.3** Substances that are controlled under the Misuse of Drugs Act 1971 are grouped into one of three classes in a system of relative-based harms:
- Class A** (the most harmful) includes cocaine, diamorphine (heroin), 3,4-methylenedioxymethylamphetamine ('ecstasy'), lysergic acid diethylamide (LSD), and methylamphetamine.
- Class B** (intermediate category) includes amphetamine, barbiturates, codeine and methylphenidate.
- Class C** (less harmful) includes benzodiazepines, buprenorphine, anabolic steroids, gamma-hydroxybutyrate (GHB) and ketamine.
- 1.4** As devised, this system of classification, which is based on the harmfulness to individuals and society, serves to determine the penalties for the possession and supply of controlled substances. The current maximum penalties are as follows:
- Class A drugs:** For possession – 7 years' imprisonment and/or an unlimited fine; for supply – life imprisonment and/or fine.
- Class B drugs:** For possession – 5 years' imprisonment and/or an unlimited fine; for supply – 14 years' imprisonment and/or fine.
- Class C drugs:** For possession – 2 years' imprisonment and/or an unlimited fine; for supply – 14 years' imprisonment and/or fine.
- 1.5** In 1977, MDMA and other ring-substituted phenylethylamines were generically classified under the Misuse of Drugs Act as Class A drugs. Some other countries, at about a similar time, took the same generic approach, including the Republic of Ireland and New Zealand, whereas others enacted drug-specific rather than generic controls. Subsequent amendments to the New Zealand legislature have re-classified MDMA within Class B Part 2 of their legislation, yet 3,4-methylenedioxyamphetamine (MDA) and 3-methoxy-4,5-methylenedioxyamphetamine (MMDA) (other drugs considered 'ecstasy-like') have been retained in Class A.

- 1.6** MDMA was given Class A status as at the time it was considered to be a hallucinogen like LSD, which had also been placed in Class A when the Misuse of Drugs Act was introduced in 1971. Subsequent experience, however, showed that MDMA does not usually cause hallucinations (Green *et al.*, 2003), although these may sometimes occur as an adverse effect (Davison and Parrott, 1998). MDMA has no recognised medicinal use and is therefore placed in Schedule 1 of the Misuse of Drugs Regulation (2001).
- 1.7** This report is based on a review of the literature, including journal articles, books and other literature. The ACMD also considered oral and written evidence (Annexes C and D respectively) submitted by organisations and individuals with particular expertise and special interest in MDMA.

2. Introduction

- 2.1** MDMA is a ring-substituted phenylethylamine, a chemical derivative of amphetamine. Other related compounds include MDEA (3,4-methylenedioxyethylamphetamine) and MDA (3,4-methylenedioxyamphetamine), and together these are colloquially known as 'ecstasy' (often abbreviated to E, X or XTC) although there is a range of synonyms that are used. For the purposes of this review, we focus on MDMA as currently this is by far the most commonly seized of these drugs (Forensic Science Service, 2008a and b, see paragraph 2.5).
- 2.2** Safrole is the primary precursor for the illicit manufacture of MDMA and is a natural product found in sassafras oil (Forensic Science Service, 2008a; Dal Cason, 1990). Safrole and a number of other MDMA precursors are subject to controls under European Union (EU) regulations. There are several methods by which MDMA may be illicitly manufactured, mainly via the precursor 3,4-methylenedioxyphenyl-2-propanone (3,4-MDP2P), which is prepared from safrole. There are very limited licit requirements for 3,4-MDP2P and therefore the vast majority of this precursor is obtained by illicit manufacture rather than diversion from legitimate sources.
- 2.3** In the late 1980s, 'ecstasy' was associated with the rave culture. However, it then became more widely available in many dance clubs and other venues during the 1990s. Data from the British Crime Survey (BCS) indicate that use since 1996 has remained relatively stable (British Crime Survey, 2008).
- 2.4** Use of ecstasy is broadly considered 'recreational' by the majority of users, rather than a drug of daily or dependent use (Measham *et al.*, 2001; Measham, 2004). A large proportion of individuals who use MDMA have previously, or are concurrently (often in the same night), using other drugs, particularly alcohol, nicotine/tobacco, cannabis or amphetamine (Gross *et al.*, 2002; Riley *et al.*, 2001). There are, therefore, substantial problems in attributing field observations of any purported 'effect' solely to 'ecstasy'.
- 2.5** Data from the Forensic Science Service show that 99% of all police seizures of 'ecstasy' analysed contain MDMA (Forensic Science Service, 2008a). Similarly, Dutch data (Drugs Information and Monitoring System project) found that since 2000 over 95% of all samples of 'ecstasy' contained MDMA (R Niesink, *pers. comm.*).
- 2.6** MDMA has a distinct pharmacology that differs from that of amphetamines and other stimulants (e.g. cocaine) in that it produces a sense of warmth and empathy with others, which is why it was considered, in the 1950s, to assist in psychotherapy (reviewed in Greer and Tolbert, 1990; Holland, 2001; Pentney, 2001).
- 2.7** Like other stimulants, MDMA produces an increase in drive and energy which encourages and allows users to dance for long periods. The empathy-producing action has led some to call MDMA an 'empathogen' or 'entactogen'. MDMA's empathogenic properties are thought to be

due to the release of serotonin² (5-hydroxytryptamine (5HT)) in the brain, of which MDMA causes the release to a much greater extent than other psychostimulants (e.g. cocaine or amphetamine). The ‘energy-increasing’ effects are thought to reflect brain dopamine³ release as this is the main action of other psychostimulants; these dopamine-releasing effects are more prominent at higher doses. However, in general, other psychostimulants give rise to a considerably greater release of dopamine, which probably explains why they are more likely than ‘ecstasy’ to cause dependence and paranoia (see paragraph 5.3).

- 2.8** Tolerance to some of the psychological actions of MDMA are reported in some human users of high doses of the drug (Parrott, 2005), which can lead to users taking more tablets to get the desired effect. Rats exposed to high doses of MDMA exhibit a temporary tolerance to the serotonin-releasing and behavioural stimulant effects of subsequent doses of MDMA (Baumann *et al.*, 2008; Brennan and Schenk, 2006); this may represent an animal model for the human tolerance.
- 2.9** Most MDMA is currently sold as tablets, the rest as a white or off-white powder or very occasionally as crystals (Forensic Science Service, 2008a). Tablets are often prepared to a high standard using the same technology as for prescription medications. Toxic constituents as by-products of production or through contamination are rare, but tablets may have other substances added, possibly to alter their effects (see paragraph 2.11).
- 2.10** Based on current evidence, the ‘ecstasy’ consumed in the UK is produced almost exclusively in northern Europe (Association of Chief Police Officers, 2008; Serious Organised Crime Agency, 2008). Home-based laboratories are rare but have been uncovered by police operations as have tableting facilities. The chemicals used in MDMA synthesis, though less dangerous than those used to synthesise methylamphetamine, can still present hazards (such as fires) to the manufacturer and local inhabitants.
- 2.11** The constituents of tablets sold as ‘ecstasy’ have changed over time. In past years, ‘ecstasy’ tablets may have had singularly, or in combination, MDMA, MDEA or MDA as the major psychoactive constituents. In more recent years, the majority of seized tablets sold as ‘ecstasy’ contain MDMA as the primary psychoactive component. However, recent seizures of MDMA tablets have also been found to contain other psychoactive ingredients including ketamine and benzylpiperazine. For example, around 9% of tablets seized between July 2007 and June 2008 contained a piperazine-type drug (Forensic Science Service, 2008b). In addition, there are more recent reports of other stimulants being added to alter the psychoactive properties of the MDMA. An example is some Canadian seizures where methylamphetamine was present (Forensic Science Service, 2008a). This will give users a greater stimulant effect and may be included to induce MDMA users onto the more addictive drug.

2 Serotonin is a neurotransmitter that plays an important role in the modulation of mood, anger, aggression, body temperature, sleep, sexuality and appetite (see Glossary).

3 Dopamine is a neurotransmitter that is involved in the regulation of motor function, energy and drive, appetite, drug-liking and drug dependence and psychosis (see Glossary).

- 2.12** The average content of MDMA in tablets has reduced over the past decade from around 100mg per tablet to about 40mg; this may go some way to explain the increase in the average number of tablets used per user over this period. However, seizures of tablets have shown that the MDMA content of a given tablet can vary considerably (Forensic Science Service, 2008a and b).
- 2.13** The price of 'ecstasy' tablets has fallen over recent years and currently tablets can cost as little as £2.30, most commonly sold in batches of 3–5 for £10 (DrugScope, 2006; 2007; 2008). MDMA powder or crystal costs about £35–40 per gram and is swallowed, sometimes by dabbing a moistened finger into the packet containing the powder, or less often wrapped in a piece of cigarette paper and swallowed (F Measham *pers. comm.*). Despite the considerably higher price of MDMA powder over tablets, the higher price is seen by some as an indicator of higher quality as well as having associations with higher kudos due to its greater cost (Measham and Moore, 2009). It is not yet clear from research the extent to which users are switching from tablets to powder or adding powder to their drug repertoires. MDMA powder is not usually insufflated (snorted) as it causes sneezing, pain and nosebleeds. MDMA cannot be smoked and is very rarely injected intravenously.
- 2.14** MDMA was originally used in the USA as an agent to assist in psychotherapy (Holland, 2001; Pentney, 2001); this use ceased once it became illegal in the USA. In the UK, MDMA was placed in Schedule 1 of the Misuse of Drugs Regulations 1975 (and as subsequently amended) on the grounds it had no recognised medicinal use.⁴ MDMA has also been used by some people with severe Parkinson's disease to reduce disabling tremor (Concar, 2002). Subsequent research in a mouse model of this disease has shown MDMA to be particularly effective in reducing symptoms (Sotnikova *et al.*, 2005).

4 The ACMD makes recommendations for both classification (Misuse of Drugs Act 1971) and scheduling (Misuse of Drugs Regulations 2001). Medicinal use is covered as appropriate in the scheduling. However, the Medicines Act 1968 (under the auspices of the Medicines and Healthcare products Regulatory Agency (MHRA)) will cover the medical use of any given drug.

3. Epidemiology of MDMA use, seizures and enforcement

- 3.1** MDMA is an illegal drug and so data have been obtained from formal surveys (such as the BCS) and less formal surveys such as internet-based questionnaires and analysis of samples handed in at night clubs and dance venues.
- 3.2** It has been estimated by the Association of Chief Police Officers (ACPO) that between 2.5 and 5 million MDMA tablets are taken every month in the UK (Association of Chief Police Officers, 2008). In 2003/04, it was also estimated that nearly 60 million 'ecstasy' tablets (95% confidence interval: 32.7 to 86.3 million) were consumed annually (Home Office, 2006a), which is consistent with the ACPO estimates. It is uncertain, however, whether these estimates have changed over time. Information on average consumption by individual users is unavailable to test fully how these estimates relate to the number of 'ecstasy' users (see paragraph 3.3). It is likely also that use increases during the summer music festival season and during holidays at resorts where clubbing is popular. It is of concern that many young people from the UK are introduced to MDMA use while abroad on holiday.
- 3.3** The BCS (England and Wales) shows that the level of 'ecstasy' use has remained fairly stable over time with few statistically significant changes from one year to the next. The BCS estimates that, in 1996, 1.7% of 16 to 59-year-olds had used 'ecstasy' in the last year. Estimated use peaked in 2002/03 with a reported 2.2% of 16 to 59-year-olds having used 'ecstasy', this figure falling to 1.8% in 2006/07 (Home Office, 2008).
- 3.4** The 2006/07 BCS estimates that 1.8% of 16 to 59-year-olds had used 'ecstasy' in the last year (Home Office, 2007). In the same survey, among adults aged 16–24, 4.8% reported having taken 'ecstasy' in the last year. An earlier survey – the Offending, Crime and Justice Survey (OCJS) conducted in 2003 reported a larger proportion of the population using 'ecstasy' than the BCS. Thus, in 2003 the weighted survey estimates were approximately 3% for the OCJS and 2% for the BCS in the last year (Home Office, 2006a).
- 3.5** Data from the 2006 Scottish Crime and Victimization Survey and the 2006/07 Northern Ireland Crime Survey show that 3.2% and 0.9% of 16 to 59-year-olds surveyed, respectively, had taken 'ecstasy' in the last year (Scottish Government Social Research, 2007; Northern Ireland Office Statistics and Research Branch, 2007).
- 3.6** Use data for 16 to 24-year-olds from the BCS for England and Wales are shown in Table 1 (Home Office, 2007). These are likely to be minimum estimates as they do not take account of the under-reporting of population surveys such as the BCS. Use in older age groups is lower, though not unknown (Anonymous, 2001). Cocaine may be taking over from 'ecstasy' as the preferred drug of young clubbers, as judged by the higher use of this Class A stimulant (Table 1) and increasing medical problems (see Section 4).

Table 1. Reported drug use by 16 to 24-year-olds, data from the British Crime Survey 2006/07 (Home Office, 2007)

Drug	Used at all last year (estimated)	Frequent use i.e. more than once a month (estimated)
Ecstasy	272,000 (4.8%)	42,000 (0.7%)
Amphetamines	207,000 (3.5%)	25,000 (0.4%)
Cocaine powder	373,000 (6.0%)	95,000 (1.7%)

- 3.7** Among 11 to 15-year-olds, data for England from 2007 show that 1.8% report ever having used 'ecstasy' (the percentage figure for use in the last year increases with age) (National Centre for Social Research, 2008). 'Ecstasy' use within the 11 to 15-year-old age group in England has remained at similar levels since 2001 (National Centre for Social Research, 2008). Data from Scotland (Scottish Schools Adolescent Lifestyle and Substance Use Survey (SALSUS), 2006) show that the percentage of 15-year-olds who reported ever having used 'ecstasy' was 5%. The data from the Scottish survey show the prevalence of reported use in the last month was 3% for 15-year-olds and 1% for 13-year-olds (SALSUS, 2006).
- 3.8** Ongoing surveys of the drug use of club customers from 1997 (Measham, Aldridge and Parker, 2001) to 2008 (Measham and Moore, 2009) suggest that 'ecstasy' use by clubbers has not declined in this 10-year period. However, in recent years there appear to be growing trends in (a) increased dosage of 'ecstasy' by club customers, and (b) the use of a growing repertoire of other illicit drugs alongside MDMA in the night-time economy, dance club and festival scene (Gross *et al.*, 2002; Measham and Moore, 2009). As well as alcohol, cocaine and to a lesser extent amphetamines may be co-consumed for their stimulant properties, and cannabis, ketamine or benzodiazepines may be used later in the evening – usually in private households – reportedly (a) to prolong the socialising by attending post-club 'chill-out' parties and 'breakfast clubs', and (b) to reduce the negative effects of the stimulant drug 'comedown' phase (Moore and Measham, 2008). In some cases, such drug combinations are likely to add to health risks, as each are themselves potentially harmful, although little research has been done on the consequences of such poly-drug use (Measham and Moore, 2009).
- 3.9** As with all drugs, differing policing priorities and targets do not allow relative comparison of seizures of drugs in the same class. However, we were informed that 'ecstasy' possession has a lower priority than some other Class A drugs, specifically heroin and cocaine. Data from 2004 show that 'ecstasy-type' possession offences attracted a greater number of cautions (37%) than those for possession of other Class A drugs (crack cocaine (15%) or heroin (17%)) though not cocaine powder (37%) (Home Office, 2005a).⁵ Estimates comparing consumption and seizure rates from 2003 suggest that less than 10% of 'ecstasy' tablets are seized, compared with nearly 25% of cannabis and 12% of heroin (Home Office, 2006a). However, more recent data from the Home Office indicate that seizures of 'ecstasy' have increased by 22% since 2005 (Home Office, 2008).

⁵ The greater number of cautions may suggest that 'ecstasy' is dealt with more leniently, i.e. cautions are given rather than charges brought.

3.10 Sentencing data show that the percentage of persons receiving an immediate prison sentence for either MDMA supply, intent to supply or possession offences is less than those for other Class A drugs (Table 2) (Sentencing Guidelines Council, 2007). The data suggest that the courts treat offences concerning MDMA more leniently than those of other Class A drugs.

Table 2. Percentage of persons sentenced to immediate custody for offences related to selected drugs (2007)

	Supply	Intent to supply	Possession
MDMA	52% (70) ⁶	56% (273)	5% (68)
Cocaine	67% (344)	73% (814)	4% (206)
Heroin	72% (809)	80% (835)	9% (413)
Crack cocaine	81% (197)	79% (248)	9% (104)
Other Class A drugs	93% (442)	69% (193)	10% (97)

⁶ (n) number of persons.

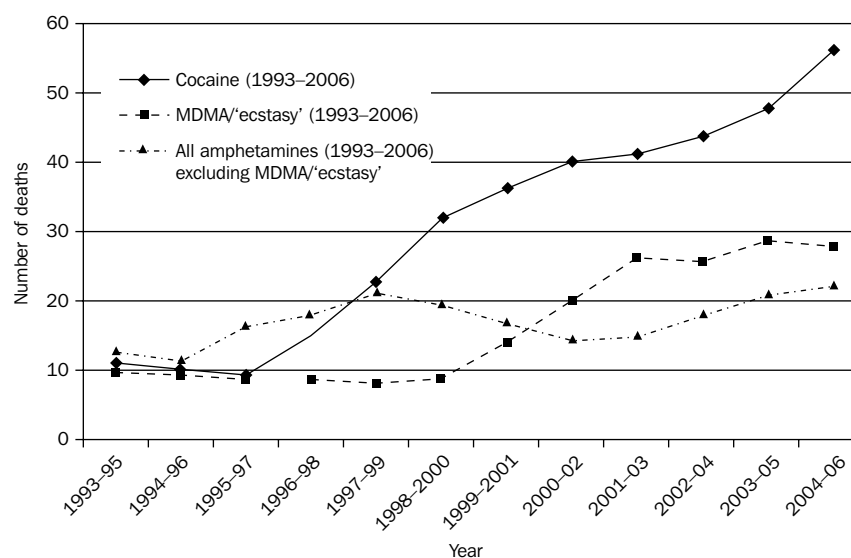
4. Physical harms

- 4.1** MDMA has undoubted harms, causing direct toxicity especially when taken in high doses. However, many of the other physical harms of MDMA are associated with behaviours in which the users subsequently engage, such as energetic dancing for long periods.
- 4.2** Published literature provides a heterogeneous picture, with case reports detailing acute complications including death occurring after limited exposure (including consumption of a single tablet) (Rogers *et al.*, 2009). Presentations to accident and emergency departments after taking MDMA are usually associated with poly-substance use (80% with alcohol, 24% cocaine and 21% ketamine) (Dargan, 2008).
- 4.3** Admission data from Newcastle (Dargan, 2008) show that the number of admissions due to MDMA between 2000 and 2007 varies between 22 and 35 per year. This is compared with around 15 per year for amphetamines and, following a recent increase, over 30 per year for cocaine. Data from presentations to St Thomas' Hospital, London (2005 to 2008) show that, for those agents classed as recreational drugs, MDMA was the third most common drug behind cocaine and GHB, being involved in a total of 382 presentations (Dargan, 2008). However, of these MDMA presentations, only 52 were as sole drug; 85% involved co-ingestants, of which alcohol, GHB and ketamine were the most common.
- 4.4** The total number of admissions to hospital due to MDMA (alone or in combination) is not known. But, if the data provided by St Thomas' and Newcastle hospitals are considered indicative, it is likely to be of the order of several thousand per year. By way of comparison, there were over 57,000 recorded hospital admissions in 2006/07 with a primary diagnosis of alcohol poisoning and 846 with a primary diagnosis of cannabis poisoning (Department of Health/National Treatment Agency for Substance Misuse, 2008). Estimates for all hospital admissions to which alcohol contributes are over 800,000 per year with over 200,000 admissions with alcohol-specific conditions.
- 4.5** Data obtained from the National Poisons Information Service (NPIS) show that among Class A drugs MDMA, historically, has been the most common drug of misuse where information has been accessed (National Poisons Information Service, 2008). However, the proportion of telephone enquiries related to MDMA acute toxicity fell sharply between 2004/05 and 2006/07. In contrast, the proportion of those enquiries relating to cocaine has increased over the same period and is currently a more common drug for enquiry than MDMA (National Poisons Information Service, 2008). The NPIS data, however, are limited in providing any indication of the true incidence of toxicity cases.
- 4.6** MDMA overdose has a profile of toxicity similar to, but with somewhat less severe outcomes than that seen with amphetamines and cocaine (Dargan, 2008). Cardiovascular effects (elevated blood pressure and heart rate) are prominent and consistent with the amphetamine-like nature of MDMA; epileptic seizures are sometimes seen. Cocaine has a similar toxicity profile, but has a higher rate of cardiac problems associated, especially myocardial infarction, particularly when taken with alcohol (Devlin and Henry, 2008). On rare occasions, use of amphetamines, cocaine and MDMA can lead to intracerebral and subarachnoid haemorrhage (Gledhill

et al., 1993; McEvoy *et al.*, 2000) and it would appear that, in the majority of reported cases, the haemorrhage appeared to be related to an underlying vascular malformation.

- 4.7** MDMA is often taken in night/dance clubs and settings where the temperature may already be high and the individual is engaged in prolonged dancing. These factors, coupled with MDMA use, can be dangerous, especially if associated with dehydration – sometimes leading to exertional hyperpyrexia/hyperthermia (raised body temperature). This was the explanation for some of the first MDMA fatalities which occurred in dance clubs when users had danced for prolonged periods in high temperatures while drinking very little water. In 1996, the ACMD acted on these incidents and issued advice to Ministers and suggested guidance to users to ensure adequate hydration when dancing for long periods (Advisory Council on the Misuse of Drugs, 1996). This was coupled with guidance to local authorities and club owners to provide free water and ‘chill-out’ rooms, to reduce such incidents. New safe clubbing guidelines – *Safer Nightlife* – have recently been issued by the London Drug Policy Forum (2008).
- 4.8** Water intoxication (with secondary low blood sodium levels – hyponatraemia) is a condition also associated with the use of MDMA. This can be as a result of excessive water intake, in an attempt to prevent dehydration after taking MDMA. In some people, MDMA may cause excessive secretion of antidiuretic hormone, which makes the kidneys retain water, so aggravating the consequences of excessive water intake (Devlin and Henry, 2008).
- 4.9** Data presented to the ACMD identified nine published case reports of fatalities due to hyponatraemia between 1997 and 2002 and one in 2006 (Rogers *et al.*, 2009). Twenty-four case series or case reports involving non-fatal hyponatraemia were also identified. All fatal cases were in women aged between 16 and 21. The propensity for women to be disproportionately affected is probably due to the lower ratio of body water to body mass in women.
- 4.10** Cases of acute liver injury (hepatitis) are occasionally reported. These can be secondary to hyperthermia or caused by direct hepatotoxicity from the drug; in the latter case, it may re-occur if MDMA is taken again (Devlin and Henry, 2008).
- 4.11** The National Programme on Substance Abuse Deaths (np-SAD) maintains the Special Mortality Register (SMR). The dataset is unlikely to be fully complete as it records the voluntary submissions of coroners’ reports for England and Wales and there are differences in the way coroners, or their pathologists, incorporate findings. The General Mortality Register (GMR) is a database maintained by the Office for National Statistics (ONS) based on information from death certificates and coroners’ reports. Accuracy of the dataset relies on the information recorded by the coroner. Full toxicological data on all of the drugs detected at post-mortem are not always cited on the death certificate, and in some situations it can be difficult to ascribe the drug(s) responsible for the death (Hickman *et al.*, 2007).
- 4.12** Between 1999 and 2001, the data from the GMR show a rise in drug-related deaths, where ‘ecstasy’ was the sole drug mentioned. Thereafter, the number of deaths attributed to ‘ecstasy’ reached a plateau while both cocaine- and, to a lesser extent, amphetamine-related deaths continued to rise (Figure 1).

Figure 1. General Mortality Register drug-related deaths, 1993 to 2006 (sole drug mentioned): three-year rolling averages for cocaine, MDMA/'ecstasy' and amphetamines (excluding MDMA/'ecstasy') (Rogers *et al.*, 2009)



4.13 Data from the np-SAD for the period 1997 to 2006 recorded that MDMA was implicated in a mean of 50 deaths per year and around 10 where it was considered the sole drug (Rogers *et al.*, 2009). Data from ONS using the GMR in the period 1993 to 2006 record a mean 33 deaths per year where MDMA is implicated and 17 where it was considered the sole drug (Table 3) (Rogers *et al.*, 2009). The difference between the GMR and np-SAD figures will be due to the differences in data reporting and data sources used.

Table 3. Annual number of deaths recording illicit drugs, General Mortality Register, 1993 to 2006)

	Mean annual deaths (%) – sole drug	Mean annual deaths – co-use drug mentions
Heroin and morphine	447 (65.6)	622
Methadone	150 (22.0)	276
Cocaine	31 (4.6)	86
All amphetamines	34 (4.9)	70
MDMA/'ecstasy'	17 (2.5)	33 ⁷
Cannabis	1 (0.2)	14
GHB	1 (0.2)	2

Source: ONS

4.14 Table 3 shows the number of drug-related deaths for selected causes either as the sole drug or as one of the drugs involved. There are fewer deaths implicating MDMA than several other Class A drugs (such as heroin, methadone and cocaine) and a similar number of deaths due to amphetamines.

7 Alcohol was also recorded in an annual average of six co-drug use deaths involving ecstasy.

- 4.15** Data from the General Register Office for Scotland (GRO) show that, between 1995 and 2007, there was an average of 2.5 deaths a year which involved only ‘ecstasy’, or only ‘ecstasy-type’ drugs, or only these and alcohol (General Register for Scotland, 2007).
- 4.16** Np-SAD data suggest that, for those deaths where MDMA has been implicated, the individuals tend to be younger with a greater likelihood of being employed. This is in contrast to those deaths where amphetamine is implicated. Fatalities where ‘ecstasy’ is implicated also tend to be more associated with concurrent alcohol and cocaine use and less with heroin and methadone use than those from amphetamines.
- 4.17** It is particularly difficult to estimate the risk of taking any given MDMA dose due to the lack of information on the average level of consumption and dose-response relationship between tablet intake and increased risk of overdose, as well as uncertainty surrounding the number of ‘ecstasy’ users. For example, in 1995/96 a 25-fold range was estimated for ‘ecstasy’-related death among 15 to 24-year-olds of between one in 2,000 and one in 50,000 users (Gore, 1999). Equally, if we assume that there are 1.2 million adult ‘ecstasy’ users and that approximately 60 million tablets are consumed annually (Home Office, 2006a) then the risk of death per person and per tablet is: one in 39,000 and one in 1.8 million respectively, if all deaths mentioning ‘ecstasy’ are included; and one in 76,000 and one in 3.5 million respectively, if only those deaths solely mentioning ‘ecstasy’ are included.
- 4.18** In attempting to quantify the intrinsic fatal toxicity risk of MDMA, as measured by the ratio of deaths to availability, we looked at mortality data from the ONS for the period 2003 to 2007. Three separate measures of an index of fatal toxicity (T1, T2 and T3⁸) were calculated as the total number of cases in which the drug was mentioned on death certificates⁹ divided by, respectively: (i) the number of users of that drug (T1). The number of users (16 to 59-year-olds) was derived from the BCS (Home Office, 2004; 2005b; 2006b; 2007) based on the estimated number of users in the last year over the same period; (ii) seizures by law enforcement agencies (T2). Drug seizure data were taken from Home Office (2008); and (iii) estimates of the market size of each drug in England and Wales (T3). Market size was derived from Home Office data (Home Office, 2006b). The data were then normalised such that, for each scale, heroin = 1,000. Values of T1, T2 and T3 are listed in Table 4. For each scale, amphetamine, MDMA and cocaine have a broadly similar fatal toxicity, which is considerably lower than that of heroin.

Table 4. Indices of fatal toxicity (T) for the period 2003 to 2007

Drug	T1	T2	T3
Heroin/opiates	1,000.0	1,000.0	1000.0
Cocaine	10.9	163.0	92.0
MDMA	4.6	118.0	99.0
Amphetamine	5.0	95.0	106.0
Cannabis	< 1.0	2.0	< 1.0

⁸ We note that there are caveats that must be considered when using all three of these availability metrics.

⁹ We note that a drug mention on a death certificate does not necessarily indicate causality.

4.19 A study of all drug-related deaths in Scotland during the 1990s found that every death where MDMA was involved was reported in the newspapers (Forsyth, 2001). Deaths due to other drugs were much less likely to be reported; for example, only one in 50 were reported for diazepam and for amphetamine it was one in three. The skewed reporting of 'ecstasy' against the landscape of other drug-related harms and deaths is a real phenomenon and may heavily impact on public perception.

5. Acute psychological effects

- 5.1** MDMA produces a sense of well-being and empathy (indeed, when it was originally used in psychotherapy it was called 'Empathy'). Unlike other stimulants (especially crack cocaine and methylamphetamine), MDMA does not predispose users to violence and users do not usually present problems for policing, even when in large gatherings. MDMA is not a hallucinogen like LSD or psilocybin, as users maintain a sense of contact with reality, and it is not normally a cause of 'bad trips' (Green *et al.*, 2003), although visual hallucinations may sometimes occur as an adverse effect (Davison and Parrott, 1998).
- 5.2** In common with amphetamine and many of its derivatives, MDMA improves arousal, energy, attention and concentration effects – opposite to the impairing effects of alcohol (Ramaekers and Kuypers, 2006). Although MDMA usually reduces anxiety, in some cases it can lead to panic attacks (Whitaker-Azmitia and Aronson, 1989). Irritability in the comedown period after MDMA use is widely reported as is a period of relatively low mood – the 'mid-week crash' (Parrott and Lasky, 1998) (see Section 8).
- 5.3** MDMA differs from other stimulants in that it rarely causes paranoid feelings or aggression, both of which are significant problems in amphetamine and cocaine users. The reasons for this are not fully understood but probably relate to MDMA having a predominant action on serotonin pathways in the brain, whereas the other stimulants act predominantly through dopamine (Iversen, 2008).
- 5.4** The effects of MDMA on psychomotor function have been studied during driving performance. Studies on MDMA alone have shown that it can improve some aspects of driving and impair others (Ramaekers *et al.*, 2006; Kuypers and Ramaekers, 2008). This contrasts with alcohol which impairs on all measures and leads to impulsively impaired judgement. There are cases of driving offences associated with MDMA use, but these are few in relation to the number of users, and very many fewer than those attributable to alcohol (Association of Chief Police Officers, 2008).
- 5.5** Laboratory studies of acute MDMA administration, using an 80mg dose in human volunteers, have revealed that, under the influence of MDMA, there is an impairment on word list learning of one to two words out of a total of 20 words, an effect similar to that found at the maximum legal blood alcohol concentration for driving (80mg/100ml) (Curran, 2008).
- 5.6** Like amphetamines, MDMA has been found to improve impulse control and sustained attention – an effect opposite to that of alcohol (Iversen, 2008).
- 5.7** However, evidence from self-reporting studies demonstrates that memory problems have been attributed to 'ecstasy' use in mainly 'moderate' and 'heavy' users (Parrott, 2002) The degree of self-reported psychobiological problems following MDMA use is to an extent determined by the more extreme the physical exertion of the user, with more exertion leading to more reported problems (Parrott, 2006 *et al.*). 'Novice' or short-term users (in terms of lifetime usage) generally remain unimpaired regarding memory or other psychobiological problems which are attributed to 'ecstasy' (Parrott, 2006).

6. Sub-chronic and chronic psychological effects – does MDMA produce long-term harms to the brain?

- 6.1** Many approaches to considering whether MDMA use produces long-term harms to the brain have been conducted. These include measures of serotonin neurochemistry and receptor function, imaging studies on brain volume, brain metabolism and neurotransmitter receptors and measures of the vulnerability of the brain serotonin system to depletion (Reneman, 2008).
- 6.2** Early rat studies on the pharmacology of MDMA found that as well as elevating serotonin it also damaged serotonin neurons (those that release serotonin) in the brain (reviewed by Green *et al.*, 2003). Subsequent studies in non-human primates produced similar findings (Hatzidimitriou *et al.*, 1999), although in mice dopamine neurons were also affected. Although the doses used in these studies were considerably higher than those typically taken recreationally, these preliminary findings raise concerns that MDMA might produce similar nerve cell damage in humans.¹⁰ However, a recent non-human primate study using dosing similar to that seen in humans showed no effect (Fantegrossi *et al.*, 2004).
- 6.3** A systematic review of the observational evidence (Rogers *et al.*, 2009) emphasises that reported results should be considered in the context of methodological flaws in studies. These authors note that consistency may also be reduced by publication bias, selective reporting of outcomes and interdependence of some outcome measures. Importantly, within-study imbalances in the use of other drugs and alcohol could explain some of the effects seen, confounding being most consistently seen with alcohol (Rogers *et al.*, 2009).
- 6.4** Statistically significant alterations in some brain-imaging measures have been reported. Their magnitude is generally less than comparable findings in alcohol, cocaine and methylamphetamine misusers and the clinical relevance of these findings is unclear.
- 6.5** As some animal studies have found that high doses of MDMA can induce long-term changes in serotonin nerves in the brain (Green *et al.*, 2003), the largest body of imaging research has focused on trying to determine whether similar changes occur in humans. Although humans use relatively much lower dose levels than those used in the animal experiments, it is possible that the human brain might be more susceptible to damage.
- 6.6** It is theoretically possible to measure damage to serotonin nerves in living human brain using neuro-imaging with radioactive tracers that bind to the serotonin reuptake sites; a reduction in tracer binding is therefore evidence for a reduced number of serotonin nerve terminals i.e. consistent with damage to the serotonin nerves as seen in non-human primates (Hatzidimitriou *et al.*, 1999). Such human tracer binding studies are difficult as they are subject to a number of potential confounds, especially the use of other drugs and the effects of residual MDMA in

¹⁰ Note that the most high-profile study of non-human primates was retracted because it subsequently emerged that methylamphetamine not MDMA was used (Ricaurte *et al.*, 2002; 2003).

the brain which would tend to reduce tracer binding and so mimic nerve terminal damage. Moreover, different tracers with different characteristics have been used which complicates comparisons between studies. Overall, there is evidence for a reduction of tracer binding in various brain regions in users of MDMA which is correlated with dose (McCann *et al.*, 1998; Reneman *et al.*, 2001; McCann *et al.*, 2005, Buchert *et al.*, 2003). The studies suggest that women might be more affected than men (Buchert *et al.*, 2003). Earlier studies found this reduction in tracer binding tended to be less or not present in ex-users (McCann *et al.*, 2005; Thomasius *et al.*, 2006) and a very recent UK study using the current state-of-the-art tracer has found no difference between ex-users and controls (Selvaraj *et al.*, in press). Taken together, the serotonin imaging data suggest that MDMA use may alter tracer binding to serotonin nerve terminals in the short term but that this is not permanent.

- 6.7** No evidence for any effect on the dopamine system has been found, which distinguishes MDMA from stimulants such as methylamphetamine and cocaine (Volkow *et al.*, 2001a and b).
- 6.8** One study has looked at measures of brain metabolism and found changes in the frontal cortex in ‘very heavy’ users who had consumed more than 700 tablets (Reneman, 2008). A new prospective magnetic resonance imaging (MRI) study in the Netherlands performed several different brain MRI measurements over a period of several years, during which some of the study group had taken MDMA (Reneman, 2008). The authors found that MDMA use caused a significant change in fibre tract density in the thalamus, but this was not dose related and the relevance of these changes are unknown.
- 6.9** An extensive systematic assessment of observational data on the recreational use of MDMA by Rogers *et al.* (2009) examined studies that compared MDMA users versus poly-drug users and MDMA users versus drug-naïve controls with separate analyses for current MDMA users and ex-users. The review found that there was a small but consistent negative effect of ‘ecstasy’ on cognitive and psychomotor function across a large number of controlled observational studies (over 100). The authors considered these effects tended to be ‘small’ in magnitude, noting that the mean scores of ‘ecstasy’-exposed cohorts were commonly still in the ‘normal range’. Former ‘ecstasy’ users frequently showed deficits that matched or exceeded those seen among current users. The statistically significant differences reported were most apparent on memory domains and on focused but not sustained attention. Self-rated measures of performance gave bigger effects than objective measures, which suggests a degree of self-concern in those volunteering for research studies. The authors of the report suggest that such measures could bias research findings (Rogers *et al.*, 2009).
- 6.10** There has been one prospective study where a group of young people were tested on a range of measures before any had taken MDMA and then were re-tested two to three years later (Schilt *et al.*, 2007). A significant reduction in the improvement of performance on a verbal memory task when repeated two to three years later was found in a group that had used MDMA (fewer of the ‘ecstasy’ users improved than the controls). However, the absolute level of scores was very high in all tests and did not differ between the users and non-users. The Council was presented with conflicting interpretations as to the potential clinical relevance of this data. No significant changes were seen in the other tests

conducted and in some tests the MDMA users 'improved' more than the non-users (Schilt *et al.*, 2007).

- 6.11** The literature reviewed by Rogers *et al.* (2009) suggests that, on average, 'ecstasy'-exposed cohorts tend not to exhibit exposure effects that take them outside of normal ranges. Because such literature describes cohorts, rather than individuals, it is not possible to say whether there are individual cases in which clinically relevant deficits are apparent, and none of the studies identified in the review (Rogers *et al.*, 2009) concerned themselves with defining and reporting the incidence of clinically relevant differences.
- 6.12** The MDMA findings are rather different from those of studies of methylamphetamine and cocaine users where impulse control, planning and attentional (rather than memory) processes are affected, often to a pronounced and clinically relevant degree (Volkow *et al.*, 2001a and b).

7. Dependence

- 7.1** Generally speaking, a person is said to be psychologically dependent on a substance if they experience problems controlling the amount and/or frequency of their use and continue to use in spite of adverse consequences. Physical dependence may also occur when a person requires a higher dose to get a desired effect (tolerance) or experiences withdrawal symptoms (World Health Organization, 1996). Unlike amphetamines and cocaine, there appears to be little evidence for long-term physical dependence on MDMA (Nestler, 2005; Iversen, 2008), but some withdrawal effects in the form of low mood appear to be commonly experienced (see Section 8).
- 7.2** Although it is unusual, some users develop a compulsive pattern of MDMA use, where there may be some degree of tolerance and dose escalation. This compulsive use pattern does not appear to be of the same nature as in amphetamines and cocaine. The relative lack of dependence liability probably reflects the significantly different pharmacology of MDMA to other stimulants; MDMA has more effect on brain serotonin and less effect on brain dopamine function.
- 7.3** Nevertheless, some regular MDMA users do seek specialist treatment for MDMA-related problems and assistance to reduce or stop their use. Currently these represent 1% of all treatment seekers to services, compared with 3% for amphetamines and 11% for cocaine/crack cocaine (see Table 5 taken from the published statistics from the National Drug Treatment Monitoring System (England) for 2007/2008).

Table 5. Primary drug of misuse by age at triage: 2007/08 (England)

Drug	Aged less than 18 years at triage		Aged 18 years or over at triage		N	All persons	
	N	%	N	%		N	%
Heroin	773	5	122,749	66	123,522	61	31
Methadone	15	<0.5	10,097	5	10,112	5	34
Other opiates	43	<0.5	5,404	3	5,447	3	34
Benzodiazepines	59	<0.5	2,029	1	2,088	1	36
Amphetamines	383	2	5,320	3	5,703	3	32
Cocaine	861	5	11,752	6	12,613	6	27
Crack cocaine	168	1	10,826	6	10,994	5	32
Hallucinogens	58	<0.5	344	<0.5	402	<0.5	24
Ecstasy	490	3	569	<0.5	1,059	1	18
Cannabis	12,865	78	13,422	7	26,287	13	18
Solvents	321	2	178	<0.5	499	<0.5	15
Barbiturates	<5	<0.5	39	<0.5	39	<0.5	35
Major tranquilisers	<5	<0.5	39	<0.5	39	<0.5	36
Antidepressants	<5	<0.5	171	<0.5	171	<0.5	37
Other drugs	65	<0.5	1,631	1	1,696	<0.5	41
Poly use; no details	65	<0.5	153	<0.5	218	<0.5	29
Drug-free at triage	273	2	745	<0.5	1,018	1	28
Total (clients)	16,450	100	185,460	100	201,907	100	

7.4 The Scottish Drug Misuse Database (SDMD) offers a profile of drug misusers based on reports submitted on individuals when they first attend a service for assessment of their drug misuse problems (Drug Misuse Information Scotland, 2007). The data from the SDMD show that less than 1% of the total number seeking treatment for the use of illicit drugs reported 'ecstasy' use as their main drug of misuse. The equivalent percentage of those seeking treatment for the use of heroin was 64%.

8. MDMA and mental health effects

- 8.1** MDMA-associated depressive symptoms appear to typically follow weekend use and have been termed the ‘mid-week crash’ (Parrott and Lasky, 1998). These feelings are generally mild and quickly resolve, although some users have been reported to take selective serotonin reuptake inhibitor (SSRI) antidepressants to mitigate the effects (Farre *et al.*, 2007).
- 8.2** A concern has been raised that extensive, chronic MDMA use can lead to clinical depression, perhaps through changes in brain serotonin function discussed in Section 6. The evidence is currently equivocal – most studies do not find significantly increased levels of clinical depression in current or ex-MDMA users; however, when combined, the available evidence suggests that there is a small but significant exposure effect (Rogers *et al.*, 2009). One study has found that scores on depression rating scales in MDMA users were somewhat elevated compared with non-users and this was most marked in those with a specific genotype of the serotonin reuptake site (Roiser *et al.*, 2005). Although, even in the most affected group, these ratings did not fall within the range considered symptomatic of clinical depression.
- 8.3** Some people with clinical depression find that MDMA can acutely lift their mood, albeit only transiently (B. Sessa, *pers. comm.*). Although it is unlikely that much MDMA use is for such self-medication, the scheduling status of MDMA has discouraged systematic clinical research work in this area. Recently there have been two clinical trials showing that MDMA can accentuate the benefits of psychotherapy in the treatment of chronic post-traumatic stress disorder (PTSD) (Bouso *et al.*, 2008, Mithoefer *et al.*, 2008).

9. Societal harms

- 9.1** While MDMA clearly can have a major impact on some users and their families, there are few data suggesting negative impacts on society when directly compared with the other widely used Class A drugs, namely heroin and cocaine. Policing priorities in relation to possession (as discussed in Section 3) appear to reflect this.
- 9.2** MDMA users are more likely to be in employment than heroin, cocaine and amphetamine users (Rogers *et al.*, 2009) and usually fund their drug purchases from their own income rather than from acquisitive crime (Association of Chief Police Officers, 2008).
- 9.3** In contrast to alcohol and stimulants, there are few public order offences deriving solely from the use of MDMA (Association of Chief Police Officers, 2008).
- 9.4** 'Ecstasy' use has been implicated in only a very small proportion of serious sexual assault cases (0.65%) (ACPO, *pers comm.*, 2008). Compared to 'ecstasy', there are over four times as many recorded victims of serious sexual assault under the influence of heroin and nearly three times as many under the influence of cocaine. In cases where the perpetrators are recorded as being, or are believed to be, under the influence of 'ecstasy', the figures for 'ecstasy' and heroin are similar.
- 9.5** There is evidence of the involvement of organised crime in the trafficking of MDMA both into and within the UK. There is less certainty with regard to the relative extent to which organised criminal groups specialise in such commodity dealing or whether the trafficking of MDMA is part of the multi-commodity nature of organised crime where profit and risk are assessed against both the commodity and the market. At a local level, supply of MDMA is prominently, though not exclusively, based within the night club environment.
- 9.6** It is not known what impact, if any, the classification of MDMA as Class A has on criminal activity. Downgrading would reduce the maximum sentence for production or supply from life to 14 years. However, data suggest that downgrading would not require concomitant provision of greater leniency by the judiciary, as in 2006 there was not one case of possession with intent to supply where the sentence given exceeded 10 years. Whether separating MDMA from other Class A drugs could have health and societal benefits through separating drug markets and reducing 'one-stop-shop' drug dealers that encourage heroin and crack cocaine/cocaine use has been suggested, but is not certain.

10. Discussion

10.1 The original classification of MDMA in 1977 under the Misuse of Drugs Act 1971 as a Class A drug was carried out before it had become widely used and with limited knowledge of its pharmacology and toxicology. Since then use has increased enormously, despite it being a Class A drug. As a consequence, there is now much more evidence on which to base future policy decisions.

Physical harms

10.2 Use of MDMA is undoubtedly harmful. High doses may lead to death: by direct toxicity, in situations of hyperthermia/dehydration, excessive water intake, or for other reasons. However, fatalities are relatively low given its widespread use, and are substantially lower than those due to some other Class A drugs, particularly heroin and cocaine. Although it is no substitute for abstinence, the risks can be minimised by following advice such as drinking appropriate amounts of water (see Annex E).

10.3 Some people experience acute medical consequences as a result of MDMA use which can lead to hospital admission, sometimes with the requirement for intensive care. MDMA poisonings are not currently increasing in number and are less frequent than episodes due to cocaine.

10.4 MDMA appears not to have a high propensity for dependence or withdrawal reactions although a number of users seek help through treatment services.

10.5 MDMA appears to have little acute or enduring effect on the mental health of the average user, and unlike amphetamines and cocaine, it is seldom implicated in significant episodes of paranoia.

10.6 There is presently little evidence of longer-term harms to the brain in terms of either its structure or function. However, there is evidence for some small decline in a variety of domains, including verbal memory, even at low cumulative dose. The magnitude of such deficits appears to be small and their clinical relevance is unclear. The evidence shows that MDMA has been misused in the UK for 20 years but it should be noted that long-term effects of use cannot be ruled out.

10.7 Overall, the ACMD judges that the physical harms of MDMA more closely equate with those of amphetamine than of heroin or cocaine.

Societal harms

10.8 MDMA use seems to have few societal effects in terms of intoxication-related harms or social disorder. However, the ACMD notes the very small proportion of cases where 'ecstasy' use has been implicated in sexual assault.

10.9 Disinhibition and impulsive, violent or risky behaviours are not commonly seen under the influence of MDMA, unlike with cocaine, amphetamines, heroin and alcohol.

- 10.10** The major issue for law enforcement is 'ecstasy's' position, alongside other Class A drugs, as a commodity favoured by organised criminal groups. It is therefore generally associated with a range of secondary harms connected with the trafficking of illegal drugs.

11. Recommendations

11.1 The ACMD emphasises that a harm minimisation approach to the widespread use of MDMA should be continued. In 1996, the ACMD provided advice to Ministers that outlined important public health messages to be promulgated. The key messages remain the same but have been revised in light of current data – these are attached at Annex E.

11.2 MDMA is used widely as part of the club scene alongside other recreational drugs. The ACMD acknowledges the FRANK website and associated campaigns; however, there is a need for the ‘media-savvy’ user population, in particular, to receive accurate information from a credible and reliable source on risk and harm reduction advice. Such advice on the dangers of use of MDMA with other drugs (poly-drug abuse) should be developed with a special emphasis on guidance for young people in reducing harm. Access to the *Safer Nightlife* guidance for those who work within the night-life environment should also be encouraged (London Drug Policy Forum, 2008).

Recommendation 1: A harm minimisation approach to the widespread use of MDMA should be continued.

Recommendation 2: Access to the *Safer Nightlife* guidance should be encouraged.

11.3 In particular, children and young people should receive adequate education on the risks and support to encourage abstinence, but noting that support, information and advice should be available if they are users.

Recommendation 3: Young people should receive adequate education on the risks of using MDMA to support and encourage abstinence.

11.4 Parents/carers, teachers and those working in the criminal justice system should be informed about the risks of MDMA how these compare with those of other drugs.

Recommendation 4: Parents/carers, teachers and those working in the criminal justice system should be informed about the risks of MDMA and how these compare with those of other drugs.

11.5 Poisons databases should be refined to capture whether MDMA has been used as powder or tablets. Data on psychoactive adulterants in MDMA tablets and powders, especially other Class A drugs, should be determined on a more systematic basis.¹¹

Recommendation 5: Better data should be captured regarding the form and constituents of seized MDMA.

¹¹ The ACMD acknowledges that the Forensic Science Service already collects some data.

Classification

- 11.6** In advising the Government on the classification of a substance, the Council is required, under the terms of the Misuse of Drugs Act, to consider its harmfulness to individuals and society. There is no legal basis for the Council, in advising on classification, to take into account matters such as the message that is conveyed to the public, or the consequences for policing priorities.
- 11.7** The Council's recommendation on classification is ultimately a carefully considered collective judgement. This judgement is based upon the relative harmfulness of substances within the classification system. However, the Council must strongly emphasise that the recommendation on classification should not detract from the very real harms of MDMA nor the public health messages that should be promulgated.
- 11.8** It is the Council's collective view that the balance of harms of MDMA most closely equates to substances in Class B. After consideration of the totality of evidence, the majority of Council members did not recognise the harmful effects of MDMA as commensurate with those of Class A drugs.
- 11.9** The Council's view is that MDMA should be maintained in Schedule 1 – having no recognised medicinal use – of the Misuse of Drugs Regulations 2001 (see also Recommendation 13).

Recommendation 6: MDMA should be re-classified as a Class B drug.

Research recommendations

- 11.10** More and improved knowledge of the effects of MDMA upon brain mechanisms, including research utilising brain imaging studies, is required. This should include concurrent psychomotor and cognitive measures of the effects of MDMA on the brain to explore the possibility of MDMA-induced neurotoxicity. Comparative studies with other stimulant drugs and replication studies in longitudinal cohorts scanned pre- and post-drug use, especially controlling for other drugs, are needed. This research would consolidate knowledge on the relative harms of MDMA in relation to other drugs and so provide a better evidence base for policy decisions in future.
- Recommendation 7:** Research is required into the effects of MDMA upon brain mechanisms.
- 11.11** Improved data are required on the extent and nature of MDMA use in under-16s, and on the evolution of the patterns and profile of MDMA use, especially in relation to that of other drugs, particularly in combination. This research would help inform education messages and the provision of health interventions to this group.
- Recommendation 8:** Improved data are required regarding the nature and extent of MDMA use in under-16s.
- 11.12** Further research regarding MDMA should quantify its relative risks and also gather information on public attitudes to MDMA in comparison with other drugs.

Recommendation 9: Research should quantify the relative risks of, and public attitudes towards, MDMA in comparison with other drugs.

- 11.13** Information on potential alterations in risk and harms from the use of MDMA with other drugs – especially alcohol, cocaine and ketamine – should be obtained.

Recommendation 10: More information should be gathered on the risk and harms from concurrent use of MDMA and other substances.

- 11.14** More research should be focused on the role of vulnerability factors that may make individuals more prone to the harms of MDMA. These should include studies of results of polymorphisms of genes coding for enzymes such as production of COMT (catechole-O-methyl transferase) and MAO (monoamineoxidase) which affect serotonin and dopamine metabolism and have been suggested to modulate the effects of MDMA.

Recommendation 11: More research should be focused on the role of vulnerability factors that may make individuals more prone to the harms of MDMA.

- 11.15** The potential value of a national scheme to allow drug testing of MDMA tablets/powder for individuals' personal use, such as the Dutch Information and Monitoring System (DIMS), should be explored. The scheme would have the potential for reducing harm (by promulgating harm reduction advice) and would be an important means of developing better monitoring data on drug misuse.

Recommendation 12: Consideration should be given to developing a national scheme for the purpose of testing MDMA with a view to providing harm reduction advice and developing monitoring data.

- 11.16** It is important that research into the medicinal uses of MDMA is not disadvantaged by the legislation (Schedule 1 of the Misuse of Drugs Regulations 2001). Government should keep under review any further clinical efficacy data that emerge.

Recommendation 13: Research into the medicinal use of MDMA should not be disadvantaged by the legislation and the position of MDMA in Schedule 1 of the Misuse of Drugs Regulations 2001.

Acknowledgements

The ACMD is particularly grateful to those who gave evidence at its specially convened meetings.

The ACMD was greatly assisted in its assessment of the evidence base of the harmful health effects of 'ecstasy' by a systematic review of observational evidence, conducted by the Health Technology Assessment (HTA) group – Peninsula Technology Assessment Group (PenTAG).

The ACMD is thankful to the ACMD Secretariat for their administrative support.

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Annex A: Members of the Advisory Council on the Misuse of Drugs

Member	Position
Professor David Nutt FRCP, FRCPsych, FMedSci Chair	Edmund J Safra Professor of Neuropsychopharmacology and Head of the Department of Neuropsychopharmacology and Molecular Imaging at Imperial College London
Dr Dima Abdulrahim	Senior Researcher, Research Briefings Manager, National Treatment Agency
Lord Victor Adebowale CBE	Chief Executive, Turning Point
Mr Martin Barnes	Chief Executive, Drugscope
Dr Margaret Birtwistle	Specialist General Practitioner, Senior Tutor – Education and Training Unit, St George’s Hospital and Forensic Medical Examiner
Commander Simon Bray	Commander, Metropolitan Police
Dr Simon Campbell CBE, FRS, FMedSci	Scientific consultant. Formerly Senior Vice President for Worldwide Discovery and Medicinal R&D Europe, Pfizer
Mr Eric Carlin	Chief Executive, Mentor UK
Ms Carmel Clancy	Principal Lecturer in Mental Health and Addictions, Middlesex University
Professor Ilana Crome MB, ChB, MD, FRCPsych	Academic Director of Psychiatry, Professor of Addiction Psychiatry, Keele University
Ms Robyn Doran	Mental Health Nurse and Director of Operations, North-West London Mental Health Trust
Dr Clare Gerada MBE, FRCP, FRCGP, MRCPsych	General Practitioner and Primary Care Lead for Drug Misuse, Royal College of General Practitioners
Mr Patrick Hargreaves	School Inspector, Drugs and Alcohol Adviser, County Durham Children and Young People’s Services
Ms Caroline Healy	National Adviser for the commissioning of mental health services for children in secure settings, Department of Health
Dr Matthew Hickman	Reader in Public Health and Epidemiology, Department of Social Medicine, University of Bristol
Professor Leslie Iversen FRS	Professor of Pharmacology, Oxford University
Dr Leslie King	Adviser to the Department of Health and the European Monitoring Centre for Drugs and Drug Addiction

Member	Position
Professor Michael Lewis	Professor of Oral Medicine, Cardiff University
Mr David Liddell	Director, Scottish Drugs Forum
Dr John Marsden	Reader in Addiction Psychology
Mr Peter Martin CBE	Independent consultant in substance misuse
Mr Trevor Pearce QPM	Director of Enforcement, Serious Organised Crime Agency
District Judge Justin Phillips	District Judge, Drugs Court London
Mr Richard Phillips	Independent consultant in substance misuse
Dr Ian Ragan	Executive Director of European Brain Council; formerly Executive Director Neuroscience Research, Eli Lilly UK
DCC Howard Roberts	Deputy Chief Constable, Nottinghamshire Police
Dr Mary Rowlands	Consultant psychiatrist in substance misuse, Exeter
Dr Polly Taylor	Veterinary surgeon, Cambridgeshire
Ms Monique Tomlinson	Freelance consultant in substance misuse
Mrs Marion Walker	Pharmacist and Clinical Director, Substance Misuse Service, Berkshire Healthcare NHS Foundation Trust
Mr Arthur Wing	Assistant Chief Officer, Sussex Probation Area

**Annex B: Co-opted experts attending meetings of the
Advisory Council on the Misuse of Drugs**

Mr Ric Treble LGC Forensics

Dr Mike White Forensic Science Service

Annex C: Experts submitting oral evidence to the Advisory Council on the Misuse of Drugs, September and November 2008

Professor Valerie Curran	Professor of Psychology, University College London
Dr Paul Dargan (and Professor Simon Thomas)	Consultant physician, Guy's and St Thomas' Poisons Unit, London (Professor of Clinical Pharmacology and Therapeutics, Wolfson Unit of Clinical Pharmacology, Newcastle University)
Dr Leslie King	Forensic scientist, Co-ordinator, 'new psychoactive substances', Reitox/EMCDDA Focal Point, Department of Health
Professor Andrew Parrott	Professor of Psychology, Swansea University
Dr Liesbeth Reneman	Radiologist, Academic Psychiatric Centre (AMC), Amsterdam
Mr Gabriel Rogers	Research Fellow, Health Technology Assessment, Peninsula Technology Assessment Group
Professor Fabrizio Schifano	Professor of Clinical Pharmacology and Therapeutics, University of Hertfordshire
Mr Ric Treble	Scientist, LGC Forensics

Annex D: Organisations and affiliated individuals submitting written evidence to the Advisory Council on the Misuse of Drugs

Association of Chief Police Officers

Beckley Foundation

Europe Against Drugs

Federal Highway Research Institute, Germany (H Schulze)

Forensic Science Service

Government departments (Home Office, Department of Health)

Guy's and St Thomas' Poisons Unit

Her Majesty's Revenue and Customs

Maastricht University, The Netherlands (KPC Kuypers and JG Ramaekers)

National Centre for Social Research – Survey of smoking, drinking and drug use among young people in England

National Poisons Information Service

NHS, National Services Scotland – Scottish Schools Adolescent Lifestyle and Substance Use Survey (SALSUS)

Peninsula Technology Assessment Group

Sentencing Guidelines Council

Transform Drugs Policy Foundation

UK Drugs Policy Commission

University of East London (JD Turner, M Milani and K Soar)

University of Liverpool (D Cole)

University of Swansea (A Parrott)

The Council also considered correspondence from members of the public

Annex E: Public health guidance

The advice below is updated from the ACMD's previous consideration of ecstasy and is directed at three groups of people – MDMA users, parents/carers and friends of users – although much of the advice is common to all groups.

The ACMD strongly suggests that government departments promulgate this advice as appropriate.

USERS

1. Ecstasy is a potentially dangerous drug so the only risk-free option is not to take it. Also, some ecstasy tablets contain other substances which may also be harmful.
2. It is more dangerous to take ecstasy if you have previously had epileptic fits or mental illness or you have had a previous bad experience with the drug.
3. Ecstasy itself can and does occasionally lead to immediate death through one of a number of causes. For example, through sudden cessation of the heartbeat, blood clots in blood vessels all over the body followed by uncontrolled bleeding, and sudden kidney failure. There have been about 200 ecstasy-related deaths in the last 10 years.
4. Ecstasy is often taken in clubs and at dance events. The most serious dangers are dehydration (loss of water) and heatstroke, which can be fatal. Hot environments combined with constant dancing for long periods of time will cause the body temperature to rise even more after taking ecstasy. **Water consumption is not an antidote to ecstasy.** However, **excessive** water consumption after taking ecstasy can also lead to medical complications and some deaths have occurred from this cause. Hence the advice about water consumption we have given below is very important (see paragraph 10). Combined with mineral intake, water consumption is an antidote to dehydration caused by over-exertion.
5. Ecstasy can sometimes cause epileptic fits, panic attacks and confusion, conditions which need to be medically treated. There is some evidence to suggest it can, in some people, cause damage to the heart and liver as well.
6. The more ecstasy you take in a session, the more severe the effects are likely to be. The use of other substances at the same time (including alcohol) increases the likelihood and severity of immediate problems.
7. It has been suggested that regular use of ecstasy over months or years may cause brain damage and make you more prone to mental or depressive illness in later life.
8. You may be offered other drugs which it is claimed will make taking ecstasy safer. There is no evidence that taking other drugs at the same time reduces the dangers.

9. If you take ecstasy you can reduce (but not eliminate) the immediate risks as follows:
 - (i) If you are dancing (or are in unusually hot conditions), drink about a pint of water over each period of an hour – sipped regularly rather than in one go. Salt levels in the body should also be kept up by eating salty snacks, or drinking fruit juice, fizzy drinks or sports drinks; **water is an antidote to dehydration, not to ecstasy.**
 - (ii) If you are not dancing, drink no more than a small glass of water per hour.
10. If you feel unwell after taking ecstasy or are particularly affected by any of the following, you should seek medical help:
 - (i) you are not passing urine normally and regularly despite drinking sensibly (see above);
 - (ii) you feel sick;
 - (iii) you feel light-headed or wobbly;
 - (iv) your vision becomes blurred;
 - (v) you feel panicky; or
 - (vi) you feel depressed.
11. Ecstasy affects judgement and you should not operate machinery, drive a vehicle or partake in any safety-critical activity while under its influence. It may also affect other decisions you make, for example about sexual activity.
12. For more information you can call FRANK on 0800 77 66 00 or visit the website at www.talktofrank.com/.

PARENTS/CARERS

1. Remember, your children may be taking ecstasy without you knowing it.
2. Young people take the drug because it makes them feel energetic, happy, sociable and comfortable in the company of others. It is not only taken at festivals, clubs and dance events; it is also taken at parties and at home.
3. Ecstasy can sometimes cause epileptic fits, feelings of persecution, panic attacks, blurred vision and kidney malfunction. These conditions strike individuals unpredictably but treatment is usually effective provided it is obtained quickly enough. People who have previously suffered epileptic fits or mental illness are at particular risk. In some rare cases the medical effects of taking ecstasy can result in death.
4. Ecstasy is often taken in clubs and at dance events. The most serious dangers are dehydration (loss of water) and heatstroke, which can be fatal. Hot environments combined with constant dancing for long periods of time will cause the body temperature to rise even more after taking ecstasy. Water consumption is not an antidote to ecstasy. Combined with mineral intake, water consumption is an antidote to dehydration caused by over-exertion. However, excessive water consumption after taking ecstasy can also lead to medical complications and some deaths have occurred from this cause. Hence the advice about water consumption we have given below is very important.

5. If your child takes ecstasy he or she can reduce (but not eliminate) the immediate risks as follows:
 - (i) If dancing (or in unusually hot conditions), by drinking about a pint of water over each period of an hour – sipped regularly rather than in one go. Salt levels in the body should also be kept up by eating salty snacks, or drinking fruit juice, fizzy drinks or sports drinks; water is an antidote to dehydration, not to ecstasy.
 - (ii) If not dancing, by drinking no more than a small glass of water per hour.
6. If your child unexpectedly feels unwell through feeling sick, light-headed or wobbly, panicky or depressed, or his/her vision becomes blurred, you should seek medical help and consider whether the taking of ecstasy has possibly brought the problem on.
7. It has been suggested that regular use of ecstasy over months or years may cause brain damage and make users more prone to mental or depressive illness in later life.
8. You should talk to your children about ecstasy and other drugs, including alcohol and tobacco. The Department of Health produces material that provides advice on how to do this. The material is available from doctors' surgeries, in libraries, from some chemists and through FRANK (on 0800 77 66 00 or www.talktofrank.com/) and also from the Department of Health, PO Box 410, Wetherby LS23 7LN.

FRIENDS OF USERS

1. If you know your friend has taken ecstasy that evening/recently, try and discourage them from taking any more.
2. Users can lose their common sense and indulge in irrational, repetitive behaviour. If you see evidence of this behaviour, calmly try to persuade them to stop. Don't be afraid to seek help from others, including medical help.
3. Ecstasy can sometimes have serious consequences. For example, it can bring on epileptic fits, feelings of persecution, panic attacks, blurred vision and kidney malfunction. These conditions happen unpredictably but treatment is usually effective, provided it is obtained quickly enough. People who have previously suffered epileptic fits or mental illness are at particular risk. In rare cases it can lead to death.
4. You may be able to help friends reduce (but not eliminate) the immediate risks as follows:
 - (i) If they are dancing (or are in unusually hot conditions), encourage them to drink about a pint of water over each period of an hour – sipped regularly rather than in one go. Salt levels in the blood should be kept up by encouraging them to eat salty snacks, or to drink fruit juice, fizzy drinks or sports drinks; water is an antidote to dehydration, not to ecstasy.
 - (ii) If they are not dancing, encourage them to drink no more than a small glass of water per hour.

5. When dancing in hot conditions the most serious dangers are dehydration (loss of water) and heatstroke, which can be fatal. This is because if someone dances constantly for hours and hours, their body temperature will rise even more after taking ecstasy. However, water is not an antidote to ecstasy, but combined with mineral intake (e.g. salty snacks, fruit juice or sports drink) it is an antidote to dehydration caused by over-exertion. However, excessive water consumption after taking ecstasy can also lead to medical complications and some deaths have occurred. Therefore, the advice about water consumption is very important.
6. If your friend notices he or she is not passing urine normally, feels sick or otherwise feels unwell, you should seek medical help. You should tell the assisting services that, if you believe or know they have taken ecstasy, that this has possibly brought the problem on.
7. If you are at a dance event and you see evidence of worrying behaviour by a friend:
 - (i) try and ensure that they do not get too hot;
 - (ii) take regular breaks;
 - (iii) give them drink soft drinks to sip regularly (see 5 above); and
 - (iv) consider getting them away from loud music and strobe lighting because these can affect them as well.

Glossary

5HT: 5Hydroxytryptamine – cf. Serotonin (see definition below).

Adulterants: substances added to illicit powders and tablets; they may be pharmacologically active or relatively inert and are usually less expensive than the drug itself.

Amphetamines: when used in this report, means amphetamine and methylamphetamine unless specified.

COMT: catechole-O-methyl transferase: an enzyme that is involved in the breakdown of dopamine.

Cutting agents: the term is synonymous with adulterants.

DIMS: Dutch Information and Monitoring System: a scheme developed in the Netherlands for appraising and tracing new substances. The network also provides information to users.

Dopamine: a neurotransmitter involved in the regulation of motor function, energy and drive, appetite, drug-liking and drug dependence and psychosis.

MAO: monoamine oxidase: an enzyme that is involved in the breakdown of dopamine and serotonin.

MDA: 3,4-methylenedioxyamphetamine.

MDEA: 3,4-methylenedioxyethylamphetamine.

Serotonin: a neurotransmitter involved in many brain functions including regulation of mood and anxiety, eating, sleeping, sexual function and cognition. Also known as 5HT.



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Produced by COI on behalf of the Home Office.
January 2009. Ref: 292015

ISBN 978-1-84726-868-6



5051688012835