Death rates from ecstasy (MDMA, MDA) and polydrug use in England and Wales 1996–2002


National Programme on Substance Abuse Deaths (np-SAD), Department Addictive Behaviour and Psychological Medicine, St George’s Hospital Medical School, London, UK

The present study reports on all deaths related to taking ecstasy (alone, or in a polydrug combination) occurring in England and Wales in the time frame August 1996–April 2002. Data presented here are based on all information recorded in the National Programme on Substance Abuse Deaths (np-SAD) database. The np-SAD regularly receives all information on drug related deaths in addicts and non addicts from coroners. A total of 202 ecstasy-related fatalities occurred in the chosen time-frame, showing a steady increase in the number of deaths each year. The ratio male:female was 4:1 and 3 of 4 victims were younger than 29. In 17% of cases ecstasy was the sole drug implicated in death and in the remaining cases a number of other drugs (mostly alcohol, cocaine, amphetamines and opiates) have been found. According to toxicology results, MDMA accounted for 86% of cases and MDA for 13% of cases; single deaths were associated with MDEA and PMA. This is the largest sample of ecstasy related deaths so far; possible explanations are given for the observed steady increase in ecstasy-related deaths and a tentative ‘rationale’ for this polypharmacy combination is then proposed. Copyright © 2003 John Wiley & Sons, Ltd.

KEY WORDS — ecstasy; MDMA; MDA; MDEA; PMA; polydrug abuse; drug-related deaths

INTRODUCTION

The UK accounts for most of the ecstasy (usually containing MDMA, or MDA or a few other entactogenic compounds) tablets seized in the EU (EMCDDA, 2002). According to Ramsay et al. (2001), lifetime use amongst 16–59 year olds in England and Wales was 5% in 2000, last year use was 2% and last month use was 1%. Similar levels applied in 2001/2 for last year and last month use. Rates were higher amongst males and amongst 16–24 year olds. For this age group, lifetime use rose from 8% in 1994 to 11% in 1996 and has since stayed at that level. Last month use rose across this period from 2% to 3.6%. Aust et al. (2002) provided a ‘best’ estimate of regular UK ecstasy users, which should be about 730 000 (680 000 in England and Wales). Very little is published about the settings of ecstasy use, although it would be fair to assume that most consumption takes place at clubs, ‘raves’ and other such venues. What is clear, though, is that for many, but certainly not all, young people attending clubs, drug use has become an integral part of their night out (Webster, 2002). Some information on use in such locations can be derived from reader surveys conducted by publications aimed at this population; Measham et al. (2001) found that 67% of respondents had used ecstasy in the previous 3 months. Rates of ecstasy use amongst those presenting for treatment for drug dependence varies from country to country within the UK. In England, the proportion of those reporting ecstasy use as one of their drugs of misuse was of about 5% in 2000 (Department of Health, 2002). The number of occasions on which ecstasy-type drugs were seized within the UK by law enforcement agencies rose 24-fold between 1990 and 2000. During the same period the quantity (in terms of doses/ tablets) seized rose 48-fold. In 2000, some 6630 individuals were cautioned by the police or dealt with by the...
courts for drug offences involving ecstasy-type drugs. This was a 23-fold increase on the 1990 figure of 286 (Corkery, 2002).

The risk of ‘overdose’ with XTC has been described in the UK since the early 1990s (Henry, 1992). The mechanisms of ecstasy death may be due to hyponatraemia (i.e. decrease of sodium plasma levels; this condition is usually the consequence of an excessive water intake and can lead to brain oedema; Parr et al., 1997), to hyperthermia (increase of body temperature, which can reach the levels of 42–43°C) and/or to the occurrence of a serotonin syndrome, characterized by mental confusion, myoclonus, rhabdomyolysis (disruption of striatal muscle cells), metabolic acidosis, tremor, behavioural hyperactivity (Parrott, 2002; Ghodse et al., 2001). Other adverse effects (tachycardia; increased blood pressure; over-arousal) are due to general noradrenaline/dopamine stimulation (Hedetoft and Christensen, 1999). It has been suggested that some individuals are possibly more at risk: very young women (Parr et al., 1997); users with CY2PD6 deficiency (Gilhooly and Daly, 2002).

Recently, a number of a few others entactogenic compounds (i.e. 4-MTA, PMA, 2-CT7) have entered both the European and the UK market (Bal and Griffin, 2002; Ramsey et al., 2001; Winstock et al., 2002). This may be the cause of a serious concern given that these so called ‘ecstasy-like’ molecules can show a higher toxicity than MDMA per single tablet, significantly increasing the chances of ‘ecstasy’ overdose (Ghodse et al., 2002).

Most of the data pertaining to ecstasy related fatalities are based on case-reports or small size case-series and, due to these uncertainties, the risk of using ecstasy varies (considerably) between one death in 2000 first time users to one death in 50 000 first time users (Gore, 1999).

A simultaneous administration (i.e. on the same occasion) of cannabis, alcohol, ecstasy, amphetamines and cocaine was relatively common among 3503 visitors of techno-parties in six different European metropolitan cities. The suggested polydrug occasional user model was characterized by at least three different psychoactive compounds taken on the same occasion; roughly 95% of the sample took ecstasy together with at least another psychoactive compound (Tossmann et al., 2001). Very similar results were reported among Canadian rave attendees (Gross et al., 2002). In general, it seems that ecstasy drug users tend to use not just cannabis, but a wide range of illicit drugs. Parrott et al. (2001), in comparing a group of heavy ecstasy users with five other (control) groups of individuals, found that consumers from the study group were heavy alcohol drinkers and tobacco smokers, but also the most extensive users of stimulants and hallucinogens (Milani et al., 2000). Brecht and von Maryhauser (2002) considered ecstasy use within its polydrug context (specifically with metamphetamine; MA) and found that ecstasy plus MA users differed from MA users who had never used ecstasy because they reported a lifetime history of more types of drugs and more drug related problems. An extensive poly-drug use in individuals who used ecstasy as a drug of preference was also confirmed by Fox et al. (2001) in the UK scene.

Whilst the risk of interaction of ecstasy with a few other compounds (i.e. alcohol, Pacifici et al., 2002; moclobemide, Vuori et al., 2003), has been recently pointed out, no systematic information regarding the toxicity and lethality of drugs in simultaneous administration, in the context of ecstasy abuse, is available in the literature.

The data presented here are based on all the information recorded in the National Programme on Substance Abuse Deaths (np-SAD) database regarding the deaths related to taking ecstasy (alone, or in combination with other compounds) occurring in England and Wales between August 1996 and April 2002.

MATERIALS AND METHODS

The np-SAD was established after the Home Office Addicts Index closed and, since then, it has regularly received coroners’ information on deaths related to drugs in addicts and non addicts in England and Wales (whilst recently being extended to Scotland and N Ireland).

To be recorded in the np-SAD database, these criteria must be met: the presence of one or more psychoactive substances directly implicated in death and/or a history of dependence of abuse of drugs and/or presence of controlled drugs at necropsy.

The response rate from coroners in England and Wales has been as high as 95% (Ghodse et al., 2002). Deaths related to ecstasy were defined as a coroner’s report including the text ‘ecstasy’, ‘XT’, and/or MDMA, MDA, MDEA, PMA or the other ecstasy-like compounds (Ghodse et al., 2002). It must be stressed here that the presence, per se, of an entactogenic compound in the toxicology results was not the only criteria used to define an ecstasy-related death, since coroners may have had access to other information (i.e. evidence from the scene; police investigations reports; witnesses’ statements etc) to arrive to their own final judgement.

Copyright © 2003 John Wiley & Sons, Ltd.

RESULTS

From August 1996 to April 2002, a total of 202 ecstasy-related fatalities (at an average rate of 3.4 deaths per month) have been identified (Figure 1). 1996–7: 12; 1998: 26; 1999: 40; 2000: 52; 2001–2: 72.

With respect to the sociodemographics of ecstasy-related victims (Table 1), 162 were males and 40 were females (M:F = 4.05:1). Twenty-nine (14.4%) subjects were in the 15–19 age band at the time of their death; 64 (31.7%) were in the 20–24 age band age band and 55 (27.2%) in the 25–29 one. Sixty-two (31%) victims were living on their own and 51 (25%) with their parents. Ninety-four subjects (47%) were employed and 20 (10%) were students. Ninety-nine (49%) victims died at home and 78 (39%) died in hospital. The most frequent verdicts given by coroners were: accident/misadventure 99 (49%) cases; dependence on drugs 27 (14%) cases; non dependent drug abuse 26 (13%) cases.

According to coroners reports, together with ecstasy, a number of different psychoactive compounds were implicated as well (Table 2): heroin: 27%; other opiates (co-proxamol; dihydrocodeine etc): 21%; alcohol: 19% of cases; cocaine: 13%; amphetamines: 12%; benzodiazepines: 9%. Finally, in 34 cases (17%) ecstasy was the sole drug found. In this particular subsample of ecstasy monodrug intoxication, verdicts given by coroners were: accident/misadventure 11 (65%) cases; abuse of drugs 5 (29%) cases and suicide 1 case.

Post-mortem toxicological confirmation was made available in 183 (90.6%) cases and the presence of one or more entactogenic compounds was confirmed in 167 (82.7%) cases. According to toxicology results, ecstasy and ecstasy-like compounds were represented as follows: MDMA was found in 143 (85.6%) cases, MDA in 22 (13.2%) cases; MDEA in 1 case (0.6%) and PMA in 1 (0.6%) case.

DISCUSSION

To the best of our knowledge, the present study comments on the largest sample size of ecstasy-related deaths in the scientific literature. It appears that, in England and Wales, a steady and constant increase of ecstasy-related fatalities has been observed in the time frame of this study (1996–2002) and also during the period 1993–2001 (Griffiths, 2003). There are a few possible explanations, not necessarily contradicting each other, for this increase: larger availability of ecstasy in the UK with respect to other European Union countries (EMCDDA, 2002); availability (as highlighted here) of more toxic (i.e. PMA) entactogenic compounds which can be indistinguishable from ecstasy/MDMA itself in terms of external appearance; higher reporting rate, on the coroners’ side, of ecstasy as an important issue in causing death in their own reports (possibly due to the high media interest on the issue in the UK). Moreover, a consistent decrease of the ecstasy tablets’ purchase costs has been taken into account as a significant factor. The price of ecstasy in the UK has fallen rapidly over the past decade or so. For example, in early 1994 the average price of a dose/tablet was £16.50, but by December 2001 this figure had more than halved to £7 (NCIS, 2002). The range of prices in London tend to be higher than in other areas, despite the fact it is closer to the source of supplies, i.e. Belgium and the Netherlands. If the effects of inflation are taken into account, the fall in real terms is even greater. Prices are believed to have fallen further in 2002.

Most ecstasy victims were employed men in their twenties who died at home, thus confirming preliminary suggestions from Schifano et al. (2003) who studied a smaller (n = 81) sample size of ecstasy-related fatalities reported from England and Wales. Of particular concern is the young age of the victims, since it appeared that roughly 3 out of 4 of them were younger than 29 and 1 out of 7 were younger than 19.

A non-negligible (17%) proportion of victims died after taking only ecstasy, a possibility which had been previously doubted (Giroud et al., 1997). More typically, however, the deceased took several different psychoactive compounds (most frequently: opiates, alcohol, cocaine, amphetamines) together with ecstasy.
People may have taken these different drugs to ‘boost’ the effects of the single compounds so that one could wonder about the ‘rationale’ of this polydrug abuse. Anecdotally, it appears that alcohol is taken with ecstasy at the beginning of the night to get a stronger/better ‘high’. In fact MDMA, whilst in the presence of alcohol, shows more significant physiopathological effects (Pacifici et al., 2002). Cocaine, amphetamines and/or additional ecstasy tablets are taken to maintain arousal and a state of alertness (the stimulant desired effects of ecstasy fade away in 2–4 h; Schifano et al., 1998). Finally, opiates and/or high (i.e. sedative) dosages of alcohol are taken in the last part of the night to ‘calm down’ before going home, since the untoward after-effects of ecstasy (namely: irritability and restlessness) persist well beyond the end of the empathogenic and entactogenic ‘pleasurable’ effects (Curran and Travill, 1997).

Apart from the symptomatic relief from ‘come down’ effects, there are other possible reasons for polydrug usage. First, ecstasy tablets are sometimes made up with different adulterants (ketamine; amphetamines etc.; Spruit, 2001). Second, the chronic high dosage ecstasy users experience a diminution of the desired effects over time, which leads to exploration of use of other stimulants and/or hallucinogens (Parrott, 2001).

One might wonder if, in this sample of ‘ecstasy polydrug abusers’ (Parrott et al., 2001) ecstasy was the sole agent which caused death or if the lethal event
was due to simultaneous drug use. On the other hand, also the reverse (i.e. the use of stimulants might confer some protective effects to those who overdosed with sedatives) might be true. At the moment, however, the Programme partially relies on the toxicological findings (which can inform on what has been taken in the 2–4 days before death) and can give only limited knowledge about which drugs had been taken on the last occasion. In all of these polydrug abuse cases, it is possible that ecstasy had at least a facilitating role in causing death.

A limitation of the present report is that no analytical attention was given to the role of the possible triggering environmental factors (i.e. overcrowding; hot settings etc). Moreover, it is possible that coroners were lacking uniformity in reporting. To address these issues, future studies will use not only coroners’ reports but also the original toxicology and pathology findings, normally available from the victims’ files which are stored in the different coroners’ offices throughout the country. Every file will then be studied and examined with the ‘psychological autopsy’ technique, an approach which is already in use by the np-SAD team to carry out ‘in-depth’ studies of specific local situations. With this approach, detailed information about some important areas will be possibly elicited: medical and psychiatric history of the victim; full history of his/her drug use; quantity of drugs taken on the last occasion and characteristics of the venues attended (if any) prior to death.

Further research should better describe, in larger scale samples, the clinical implications of ecstasy misuse in the context of a polydrug intoxication and should also address the issue of possible individual genetic vulnerability to ecstasy/stimulants deaths.

REFERENCES


