

2.4 EFFECTS

Effects generally appear within twenty to sixty minutes when the user experiences a 'rush' usually described as mild but euphoric. This 'rush' may last from a few minutes to half an hour or not occur at all, depending on the user's mental set and the environment, the dose ingested, and the quality of the MDMA. After the rush, the high levels off to a plateau usually lasting from two to three hours and followed by a gradual 'coming down' sensation, ending with a feeling of fatigue. Insomnia, however, may persist long after the fatigue stage, depending on the dosage and the user (Beck & Morgan 1986:293). The effects of one tablet or dose last anywhere between one and twelve hours (median five hours) with residual effects lasting up to thirty-two hours (Solowij, Hall & Lee 1992:1165).

The effects of ingesting the average dose of MDMA (75–150 mg) can be divided into positive and negative psychological and physical categories.

Table 2.2 Reported effects of average doses of MDMA

Negative psychological/ behavioural traits	Positive psychological/ behavioural traits
Poor concentration Anxiety/restlessness Visual hallucinations Fear of loss of control Paranoia Panic attacks	Euphoria Elevated self-esteem Closeness to others/empathy Talkativeness Overall sense of well-being Acceptance Greater self-insight Heightened sensuality
Negative physiological traits	Positive physiological traits
Elevated systolic/diastolic blood pressure Muscle hypertonicity Elevated heart rate Jaw clenching Transient nausea Insomnia Dehydration Hot/cold flushes Nystagmus (flickering of the eyes)	Increased energy level Heightened sensory perception Desire to be in constant motion Appetite suppression High level of stimulation

(Source: Elk 1996:351)

2.4.1 *Positive psychological effects*

The most universal and consistent psychological effect reported by MDMA users is a 'positive mood state'. The second most commonly reported effect is 'activation' or energy (Solowij, Hall & Lee 1992:1166). Users describe a dramatic drop in defence mechanisms and increased empathy for others. Integrated with the stimulant effect, this generally produces an increase in intimate communication (Beck & Morgan 1986:293). Other perceived positive psychological effects included a sense of euphoria, elevated self-esteem, feelings of spirituality, closeness to others and open-mindedness. These qualities are reflected in the slang terms used for MDMA such as *Ecstasy* and the *hug drug* (Elk1996:352).

2.4.2 *Negative psychological effects*

The 1990s saw the growing popularity and widespread use of Ecstasy as a recreational drug, resulting in escalating reports of an apparent association between Ecstasy use and a diverse range of psychological symptoms and psychiatric disorders (McGuire & Fahy 1991:391). Ecstasy has also been associated with lasting adverse neuropsychiatric sequelae in humans who have taken repeated doses (Schifano & Magni 1994:763). According to McCann and Ricaurte (1991:302), while under the influence of Ecstasy, users may sometimes experience confusion, disorientation, anxiety, panic attacks, depression, insomnia, depersonalisation (the feeling that one is not 'real' and that one is an outside observer of one's mental processes, one's body, or parts of one's body, (DSM-IV 1994:488)), derealisation (where the environment appears to be unreal and devoid of the usual emotional component), perceptual disorders and hallucinations, paranoia, and psychotic characteristics. It is possible that some of these effects may continue for a period after the drug has worn off.

Several researchers have found that MDMA causes alterations and sometimes permanent damage to serotonin-regulated systems in the brains of experimental animals (Cohen 1995:1143). Battaglia, Yeh and de Sousa (1988:270) reported that large doses of MDMA repeatedly injected into laboratory animals decreased the levels of the neurotransmitter (chemical messenger) in the brain called *serotonin*, and to a lesser degree dopamine, and damaged the nerve terminals from which serotonin was released (see Section 2.6 'Neurotoxicity' for more detail). These effects were dose-related and recovery was incomplete. Cohen (1995:1143) believes that Ecstasy may have altering effects on serotonergic mechanisms in the human brain as well. Jansen

(1997:113) notes that there is some limited evidence of serotonin deficits in human Ecstasy users.

According to Jansen (1997), the relevance of animal studies to humans taking one or two Ecstasy tablets occasionally has been questioned. However, the animal studies do suggest that persons taking large amounts of Ecstasy for several days may be at some risk of persistently low serotonin (Jansen 1997:113). Many of the adverse effects reported have been well documented in the literature as having originated from abnormal neurotransmission of serotonin in the brain. According to Cohen (1995:1143), alterations in neurotransmitter systems, including brain serotonin, have been commonly associated with depression, anxiety, headaches, sleep disorders and sexual functioning. It has been suggested that heavy users of Ecstasy may be at increased risk of developing psychological problems of this nature (Jansen 1997:113).

Several subjects disclosed being in psychotherapy and to be taking prescribed medications such as sertraline (Zoloft) and fluoxetine (Prozac) to help alleviate symptoms induced by Ecstasy. These medications were reported to be effective in managing side-effects following MDMA use. The efficacy of these particular medications further suggests that MDMA has an altering effect on the mechanism responsible for both the release and repackaging of serotonin, especially since these medications, amongst others, are known to enhance the neurotransmission of serotonin (Cohen 1995:1143).

In attempting to explain adverse reactions to Ecstasy, the focus has, to a great extent, been upon possible brain chemical changes. Jansen (1997:114) points out that there has been an inclination to ignore the fact that Ecstasy releases emotions and can therefore have a definite influence upon the psychodynamic balance of the mind. One of the main concepts in psychodynamics is that anxiety-provoking material 'unacceptable' to waking consciousness is repressed into the unconscious, from where it may make itself known through dreams and other ways. Defences are put up against this material. Some psychotherapies may entail bringing such material to the fore so that it can be worked through and released (Jansen 1997:114). Jansen (1997:114) maintains that if these defences against disturbing material in the psyche are removed in a non-psychotherapeutic context, there may be little possibility of working through the material or containing it.

Beck and Morgan (1986:298) observed a delayed anxiety disorder in a few initiate users of MDMA. In most cases, MDMA was taken in a non-professional setting for quasi-therapeutic reasons. The indications ranged from 'a mild anxiety or concentration difficulties, to a full-blown disorder such as a panic attack with hyperventilation and tachycardia, phobic disorders, parathesias or other anxiety states' (Seymour in Beck & Morgan 1986:289). Seymour (in Beck & Morgan 1986:289) explains

that through taking MDMA, much of their repressed anxiety, hostility, guilt or other so-called negative feelings were released into their conscious minds. Prior to the time that this suppressed material was released into conscious consideration, they were probably protected by their normal defence mechanisms. After the release of this material, they are undefended and conscious of what emotional and psychological work needs to be done ...

These initial findings stress a growing danger of unsuccessful attempts at 'self-therapy' by people who take the chance of intensifying their emotional problems with unsupervised sessions. Possible consequences thereof may be the range of symptoms linked with the neuroses – such as anxiety, depression and insomnia – the very symptoms that are also associated with Ecstasy use (Jansen 1997:114).

Since much of the information available about adverse reactions to MDMA is in the form of single case studies and short, uncontrolled studies, Jansen (1997:115–120) underlines several key issues to bear in mind when considering such publications:

2.4.2.1 Pill composition – Was the drug taken actually MDMA?

Jansen (1997:115) maintains that authors who say that a person took MDMA should try to produce toxicological proof (tests of the pills taken or at least a urine test) to back up this allegation, as many pills sold as 'Ecstasy' have been shown to contain other drugs instead, sometimes in dangerous combinations. Other substances normally found in place of MDMA are methylenedioxyethylamphetamine (MDEA), MDA (predecessor to MDMA), MBDB, MDE, amphetamine, ketamine, LSD, caffeine and other chemical agents. Some pills contain no psychoactive substances at all. (Refer to Figure 2.1 for the pill contents.) Regarding effects, MDEA (MDE) has a shorter duration of action (two hours) and is more amphetamine-like, having less emotional effects than MDMA. MDEA may show a profile more similar to amphetamine in terms of adverse effects such as paranoia, agitation and anxiety. MBDB is quite like MDMA but is depicted by some as less intense with a greater 'cognitive' element. MDA is far more psychedelic (LSD-like) and is regarded to be more toxic. Concerning amphetamine additives, the connection between amphetamine use and paranoid psychosis is well established (Jansen 1997:115). Ketamine is known to produce an out-of-body experience and can be very hallucinogenic.

It is thus apparent that some of the adverse effects which have been ascribed to Ecstasy may occur as a result of adulterated or 'dodgy E' rather than pure MDMA.



Figure 2.1 Pill composition

2.4.2.2 *The role of polydrug use*

Jansen (1997:115) points out that the pure Ecstasy user is a very rare being. Most people who take Ecstasy also use other drugs, some of which are clearly associated with the risk of mental health consequences. This detail is hardly ever stressed in the case reports ascribing a psychiatric disorder to Ecstasy use, and other drug use is often dispelled in a few lines. The significance of polydrug use has been confirmed by a study of drugs taken at Raves where polydrug use was the norm amongst people who favoured Ecstasy. The preferred other drugs were cannabis, hallucinogens (LSD, magic mushrooms, ketamine), amphetamines (speed) and hypnotics (sleep-inducing drugs) (Brown, Jarvie & Simpson 1995:170). (See Figures 3.5–3.9).

2.4.2.3 *The issue of causality*

Jansen (1997:119) asserts that many of the published reports draw cause-and-effect conclusions which are not backed by the facts presented, that is, they conclude that Ecstasy consumption *caused* the symptoms rather than being *associated* with the symptoms. He affirms that it is helpful to consider whether the criteria suggested by Strassman (1984) and by Brabbins and Poole (1996) are met for research of this nature before concluding that Ecstasy *did* in fact *cause* the mental disorders described. The criteria proposed by Strassman (in Jansen 1997:119) are as follows:

There is a tendency for people with poorer pre morbid adjustment, a history of psychiatric illness and/or treatment, a greater number of exposures to psychedelic drugs, drug-taking in an unsupervised setting, a history of polydrug use, and self-therapeutic and/or peer pressure submission motive for drug use, to suffer these complications.

According to Jansen (1997:119), one need also weigh up the probability of a chance association. Brabbins and Poole (in Jansen 1997:120) point out the importance of recognising 'that among the large group of drug users within the general population, a proportion will become mentally ill regardless of any supposed psychotomimetic properties of drugs'. Since depression and anxiety are common states in the general population, it is a statistical certainty that a percentage of persons who take Ecstasy will develop depression irrespective of whether or not they took the substance. Anxiety, panic attacks and all of the other symptoms associated with Ecstasy use also occur in the non-Ecstasy-using population (Jansen 1997:119).

2.4.3 Adverse psychological effects of Ecstasy use

2.4.3.1 Psychotic phenomena

Not very often Ecstasy may produce a state of intoxication which mirrors a psychosis, such as paranoia. However, this does not usually last for more than a few days and seems to be quite rare (Williams, Meager & Galligan 1993:44). Granted that Ecstasy is not a hallucinogen in most people, it can sometimes cause hallucinations, particularly in higher doses (Beck & Morgan 1986:291). As regards serious mental illness, such as prolonged psychosis, Jansen (1997:120) maintains that there is currently a lack of accurate statistics. According to Nichols (in Jansen 1997:121), Ecstasy releases dopamine in a similar way to amphetamine and cocaine, and thus might be expected to increase the risk of psychotic illness in a comparable way to other psychostimulants, but perhaps not to the same extent.

McGuire and Fahy (1991:697) report that they have noted apparent connections between the onset of psychotic symptoms and Ecstasy use. However, Jansen (1997:121) points out that this study is based only on two cases that also involved other substances and there was no toxicological confirmation of pill content. Nevertheless, there are several other reports (Schifano & Magni 1994:763–765) and, combined, the evidence is representative of a risk. At present the size of that risk is unknown. However, Jansen (1997:121) believes it likely to be relatively small.

2.4.3.2 Anxiety disorders and panic attacks

Rare episodes of hyperventilation have been noted. These almost always occur during the onset of the experience as part of a generalised panic reaction. Reassurance that the phase is transitory generally eases this problem (Beck & Morgan 1986:297). Many reports from people who have experienced negative effects in connection with taking Ecstasy suggest that the recurring theme may be anxiety disorders rather than depression. This idea is supported by the published clinical reports in which types of anxiety disorder seem to be more common than depression (Jansen 1997:121).

2.4.3.3 Depression

A short spell of low mood associated with the 'comedown' is to be expected, although experienced users are inclined to bypass this by taking other drugs (Jansen 1997:122), such as smoking marijuana. Severe Ecstasy use is also at times followed by a longer-lasting depression (Benazzi & Mazzoli 1991:1 520). However, it is uncertain whether the chronic use of Ecstasy might not have

been a type of self-therapy or self-treatment of a pre-existing depression rather than actually causing the depression (Jansen 1997:122). Because of the association between mood and serotonin, depression may be anticipated on theoretical grounds.

2.4.3.4 Cognitive deficits

Research into drug-induced cognitive deficits (impaired memory, attention and concentration) is not easily done well, since the number of probable confounding variables is high. Jansen (1997:122) stresses the necessity to control for the use of other drugs, especially regular marijuana smoking and for the effects of any mood disorder upon cognition. If subjects have been told to abstain from all drugs for several weeks, a withdrawal syndrome may result which could confound tests carried out during this time. Jansen (1997:122) asserts that all statements of cognitive deficits should be followed by proof that the urine tests of the subjects were free of drugs and their metabolites (in particular marijuana metabolites which can take at least four weeks to disappear from urine).

A report of memory deficits in connection with Ecstasy use has been made by Bolla, McCann and Ricaurte (1998). (See Section 2.6.2.5 on memory impairment for more detail.)

2.4.3.5 Sleep disturbance

Insomnia for a few days after taking Ecstasy is quite common, but in some instances this has continued for months (Elk 1996:353) with excessive dreaming and sometimes nightmares (Jansen 1997:124). A persistent reduction in stage 2 sleep has been confirmed by Allen, McCann and Ricaurte (1994:562), although the subjects in this study were not deemed to be suffering from sleep disorders. (See Section 2.6.2.3.a on sleep electroencephalogram data for more detail.)

2.4.3.6 The 'busy head' syndrome

People who have ingested large amounts of drugs such as LSD, Ecstasy and ketamine for a prolonged period may develop a mental state which involves a high level of internal, 'mental' imagery but no perceptual disorder (Jansen 1997:123). Attention and concentration are, however, impaired, which may result in a poor memory due to failure to attend to new information. The person may be described as 'lacking focus'. Mostly, anxiety-generating situations appear to intensify the imagery (Jansen 1997:123).

2.4.3.7 Flashbacks

Flashbacks have been reported by some Ecstasy users (Creighton, Black & Hyde 1991:713). Flashbacks may be distinguished from psychotic disorders by their episodic nature, frequently of very short duration (seconds or minutes) and by their duplication (sometimes exact) of previous drug-related experiences (ICD-10 1992:83). Flashbacks have the likelihood of occurring following very traumatic drug experiences. This adds importance to the suggestion that some flashbacks are anxiety-related. One of the cases cited by Creighton, Black and Hyde (1991:713) includes a woman who had been raped while under the influence of MDMA. According to Jansen (1997:124), the possibility that flashbacks are, in fact, due to persisting changes in the brain as a result of Ecstasy use is significantly decreased by noting that a wide range of drugs, with very different action in the brain (such as LSD and ketamine), have also been linked to flashbacks. This, once again, shows the importance of polydrug use in these reports.

All the same, negative psychological effects are described by users to be less severe than those of hallucinogens and are reported less frequently. These effects are seen more commonly with higher dosages of MDMA and are thought to be more frequent in subjects with predisposed sensitivity to the drug (Elk 1996:352). Symptoms for both acute high-dose and chronic low-dose problems seem to ease with stopping use and resuming healthy living patterns (Beck & Morgan 1986:298).

2.4.4 Tolerance versus dependency versus abuse patterns

An important issue to examine is the potential for dependency and/or abusive use patterns. The most frequent use of MDMA normally happens during the first months following the initial experience. After first exposure, some people will continually try to re-experience the positive aspects of the drug. However, this abusive cycle is inclined to be short lived as the frequent use of MDMA almost always produces a strong dysphoric reaction, which is only made worse with continued use (Beck & Morgan 1986:297). The positive or pleasurable effects of Ecstasy diminish with frequent use. While the pleasurable effects decrease, side-effects tend to increase, both with frequent use and with high doses of the drug (Solowij, Hall & Lee 1992:1170). High doses of MDMA occasionally produce a variety of symptoms ranging from a 'caffeine-like' state of nervous restlessness accompanied by a 'jumbling' of thought, mood and behaviour (Seymour in Beck & Morgan 1986:298). With smaller amounts of

MDMA, psychopathology is hardly ever displayed, although some restlessness, anxiety and insomnia may occur (Beck & Morgan 1986:298).

The increasing number of unpleasant side-effects combined with an almost total loss of desired effects occurs with greater rapidity and intensity than they do with other more commonly abused substances (Beck & Morgan 1986:298). Hayner and McKinney (1986:345) too report that the unpleasant side-effects are experienced more readily following repeated doses, especially within a few days of one another. There appears to be a point at which the unpleasant side-effects increase to the extent where they outweigh the pleasurable effects originally sought by users of the drug. Because of this, recreational users report that they usually use MDMA once every several weeks (Elk 1996:353). Most likely, this unusual and sporadic pattern of use is one of the reasons that MDMA is believed not to be physically addictive. There have been no cases of physical addiction reported to date (Elk 1996:353).

Ecstasy appears to be subject to the development of tolerance and tachyphylaxis (the rapidly decreasing response to a drug after administration of a few doses) and this clearly has some relevance to its openness for dependence (Solowij, Hall & Lee 1992:1170). As expressed from a pharmacist's perspective, Riedlinger (1985:169) stated that 'there is no evidence at any rate, that MDMA is physically addictive ... the drug's possible side-effects ... are more likely to discourage frequent use or high dosage abuse'. However, whilst intensity of use increases the more severe side-effects, more intense users tend to keep using, as do dysfunctional users of any drug (Solowij, Hall & Lee 1992:1170).

2.4.4.1 Problematic Ecstasy use

There are certainly those who have taken Ecstasy on a daily basis irrespective of tolerance effects, for prolonged periods (McGuire & Fahy 1991:697). It is far more common, however, for 'problematic' Ecstasy use to involve consumption of the drug in 48-hour weekend binges with four to five days in between (Jansen 1997:125). The day after taking Ecstasy – if individuals have had a reasonable amount of sleep – a large number of users feel quite 'uplifted' in mood. However, this cheery mood generally starts dissolving by the second day and by the third day low mood, which can be quite severe, and irritability are common. This persists into the fourth day, with relative recovery of mood occurring on the fifth day. The cycle often repeats itself with Ecstasy use on the sixth and seventh days (Jansen 1997:125). Thus some people may seem to be constantly affected by the drug, even if they take it only on weekends.

With frequent use, the patterns of use may begin to have the appearance of a dependency problem, especially in persons who are taking 25 pills Thursday to Monday, month after month (Jansen 1997:125). *The Tenth*

Classification of Mental and Behavioural Disorders (ICD-10) (1992:75) states that it is not necessary to take a drug every day before a dependency syndrome can be identified, nor is physical withdrawal essential to the diagnosis. The reality that Ecstasy may be linked to tolerance, dependence and withdrawal syndromes will surprise those users who only take the drug occasionally in a relaxed setting (Jansen 1997:125).

2.4.5 Physical effects

The physical effects of MDMA are more closely related to those of amphetamines than those of hallucinogens. The amphetamine-like effects include dilated pupils, dry mouth and throat, tension in the lower jaw (trismus), involuntary grinding of the teeth (bruxism) and overall stimulation (Beck & Morgan 1986:293). The side-effects are less 'annoying' when a small or moderate dose of MDMA is taken by a healthy individual. According to Beck and Morgan (1986:293), MDMA exerts a strong paradoxical effect of relaxation, drawing less attention to the side-effects.

The universal physical effect of MDMA that is reported as positive by users is that of a high level of stimulation, described as feeling energetic or the desire to be in constant motion. Following these reported stimulant effects were heightened sensory perception and appetite suppression (Elk 1996:352). Combined with its stimulant properties, MDMA is seen as perfect for the now well-established Rave scene, which involves people dancing for hours on end in clubs and warehouses.

2.4.5.1 Negative physical effects

Consistent negative physical effects reported by MDMA users include nausea and occasional dizziness, often during the initial onset of the high (Beck & Morgan 1986:296). This feeling of nausea results in actual vomiting in some users. Many also complain of having an intense lower back pain at the onset of ingestion (Cohen 1995:1140). Other negative effects commonly reported are flickering of the eyes, muscle hypertonicity (stiffness), and an elevated pulse rate and blood pressure. Less frequently reported are tremors, dry mouth, insomnia, hot and cold flushes (Elk 1996:352), headaches, and blurred vision (van Aerts 1997:94). One of the most common annoying effects is a tension of the jaw muscles (trismus), often progressing to involuntary grinding of the teeth (bruxism). A way of relieving jaw tension is to chew gum (Eisner 1989:120) or to suck on lollipops. This has become a common sight in the Rave setting where young and old alike sport lollipops. There is also a connection between taking Ecstasy and a desire to smoke excessively, which may be associated with the effect of the drug upon dopamine pleasure systems in the brain. Respiratory

complaints are common when the smoker is moving from a hot dance environment to the cold night outside (Jansen 1997:124).

Most of these side-effects subside within twenty-four hours. However, complaints of muscle tension in the jaw may continue for two days to six weeks, blurred vision up to three days and psychological effects such as insomnia, depression and anxiety up to eight days (van Aerts 1997:94).

Individuals on Ecstasy become dehydrated and should be drinking water or juice throughout the experience. Unfortunately, some choose to drink alcohol, which increases the dehydration. As with other stimulants, persons under the influence of MDMA are often capable of consuming large amounts of alcohol with few noticeable effects until a little while later. Thus, alcohol excess probably plays a role in the next day's 'hangover' (Beck & Morgan 1986:296). The potentially toxic interaction between MDMA and alcohol warrants further investigation.

MDMA may exert an adverse action on the immunological response of some individuals. This effect is most often associated with repeated high doses, especially in people who have used the drug over a long period of time (Beck & Morgan 1986:296). Long-term users often recount increasingly uncomfortable and prolonged 'burn-out' periods, sometimes lasting two or more days. Many individuals have also reported a greater susceptibility to various ailments, particularly sore throats, colds, influenza and herpes outbreaks. Latent infections in the female genito-urinary tract can become activated. These reactions appear to be rare in initiate users and persons in good physical and mental health (Beck & Morgan 1986:296).

Researchers, using the limited information available, have identified the following medical conditions as possible contra-indications to MDMA use: diabetes, diminished liver function, epilepsy, glaucoma, heart disease, hypertension (high blood pressure), hypoglycaemia (low blood sugar), hyperthyroidism (overactive thyroid) and pregnancy (Beck & Morgan 1986:297). Since MDMA increases the blood pressure and raises the pulse rate, it may be harmful for people with cardiac problems and hypertension. In such cases the likelihood of cardiac arrhythmias, cardiac arrest and having a stroke is increased (Saunders 1997:85). MDMA also taxes the liver and may increase the probability of hepatitis and jaundice in those who have diminished liver function (Saunders 1997:83). Although there is no known effect on blood sugar, MDMA does increase energy levels and this may be harmful in diabetics since diabetics need to adjust their sugar intake or insulin dose to allow for physical activity (Saunders 1997:86). The neurochemical or electrochemical changes in the brain induced by taking MDMA can trigger epileptic fits (Saunders 1997:86). The use of any stimulant when pregnant is not advisable. It appears that the use of Ecstasy when pregnant may increase the risk of congenital abnormalities, especially heart defects, in the babies born. Further

research into a larger number of pregnancies is nevertheless essential in order to establish firmly whether MDMA itself causes these defects (*The Natal Witness* 1999:09).

2.4.6 Acute physical reactions

Acute or toxic physical and psychological effects seem to be more frequent or exacerbated with higher doses of MDMA, and also with combinations of MDMA and other drugs (Elk 1996:352). According to Henry, Jeffreys and Dawling (1992:386), the predominant toxicity patterns that emerge from the medical literature are hyperthermia (overheating or heat-stroke), convulsions, disseminated intravascular coagulation (DIC) (blood clotting in the blood vessels, rhabdomyolysis (dissolution of skeletal muscle) and acute renal (kidney) failure (ARF). DIC and rhabdomyolysis may be brought about by the hyperthermic condition while rhabdomyolysis can also be caused by ARF. Acute liver failure is another serious complication reported in association with the use of MDMA and can also precipitate from a hyperthermic condition (van Aerts 1997:92). Other acute effects reported by Hayner and McKinney (in Elk 1996:353) include vomiting, visual hallucinations, tachycardia (increased heart rate), hypertonicity of the body, hypotension or hypertension (low/high blood pressure) and palpitations. Fatal reactions to MDMA are usually cardiac in nature as acute intoxication usually results in adrenalin-like overactivity and overstimulation of the heart (Elk 1996:353). (See Table 2.3.)

Again, these reactions have been seen with high doses, combinations with other drugs or in users with predisposing conditions. Although associated with relatively few overdoses or deaths, MDMA's neurotoxic potential is cause for concern. The most controversial issue surrounding the safety of MDMA is its effects on the brain chemicals serotonin and dopamine and its possible neurotoxicity (Elk 1996:352–353). (See Section 2.9 for more detail.)

2.4.6.1 MDMA deaths

It is important to recognise that the number of deaths related to MDMA is relatively small compared with the likely frequency of its use. In the UK, the Ecstasy-related death rate per ten thousand (10 000) 15- to 24-year-old users is between 0,2 per cent and 5,3 per cent (Gore 1999:01). Nevertheless, MDMA deaths are especially puzzling as they are unpredictable. In some cases, other people appear to have taken similar quantities of Ecstasy from the same source as the overdose victim, with only minor toxic effects. One theory is that variations in metabolism of the drug caused by genetic differences or concurrent use of other drugs may result in differential susceptibility to MDMA overdose (White, Bochner & Irvine 1997:117).

The causes of death after MDMA ingestion are not well documented. Certainly, hyperthermia and its consequences seem to be of major importance, and results of animal studies suggest that environmental temperature may be a critical determinant of susceptibility (Gordon in White, Bochner and Irvine 1997:117). This is the basis for recommendations about access to cool environments or 'chill rooms' in nightclubs and Raves. Henry (in Saunders 1993:02) believes that the cause of death is due to overheating, dehydration and exhaustion from dancing in hot clubs without drinking enough fluids. He maintains that ravers dance on, feeling fine in conditions that would otherwise send them gasping for air and water, meanwhile increased body temperature can lead to strokes and internal bleeding. According to McFadyean (1997:75), the risk is reduced for people who look after themselves by drinking plenty of water and cooling off before they overheat. However, the risk is greater for those who use high and frequent doses.

Be that as it may, excessive consumption of fluids has also been cited as the cause of death. The reason for such excessive fluid consumption is not understood, although MDMA is known to induce thirst. In addition, high doses of amphetamine and amphetamine derivatives induce repetitive behaviours in animals and humans. It is possible that the combination of thirst and repetitive behaviour patterns leads to excessive fluid intake. If urine output is also low, because of dehydration, impending renal failure and (possibly) other unidentified causes, then there is considerable potential for fluid overload and its consequences (White, Bochner & Irvine 1997:117).

It is now well recognised that hyperthermia plays a central role in these events and body temperature control is therefore an important means in preventing the serious conditions already mentioned. Providing the body with enough fluid is one way of achieving this. However, it should be stressed that excessive drinking of water may lower the ionic strength (salt concentration) of the body fluids and cause tissues to swell (cerebral oedema), and can eventually lead to death (van Aerts 1997:93). Matthai *et al.* (in van Aerts 1997:93) described two cases that were shown to have developed mild cerebral oedema (abnormal accumulation of fluid in brain tissue) due to unrestricted water intake after ingesting Ecstasy. When very thirsty while on Ecstasy, it is therefore wiser to drink isotonic fluids instead of solely water.

Although raving for hours in a hot environment may aggravate the onset of a hyperthermic condition, it should be noted that MDMA by its pharmacologic action may lead to a rise in body temperature by itself. Severe reactions such as hyperthermia and DIC were rare at the time it was used in more relaxed settings in the USA in the 1980s (van Aerts 1997:93). Nevertheless, observations of this type amongst ravers have recently become all too familiar in British medical journals (Randall 1992:1505). Cardiac arrhythmia (irregularities in the heart rhythm) are often also noted in emergencies cases that are brought

in and are probably another way by which death may result, particularly in those that are predisposed by having cardiac abnormalities. The increase in the blood pressure and rise in heart rate caused by MDMA may be harmful in people with heart problems (van Aerts 1997:93). (See Table 2.3.)

Table 2.3 MDMA toxicity in humans

Case	Identity	Dose and environment	Presentation	Blood MDMA	Outcome	References in Podraza (1999:10–12)
1	–	15 tablets of E in 36 hours	urinary tract retention	–	returned to normal	Bryden <i>et al.</i> 1995
2	18 yrs M	3 tablets of E at a concert	T 40 °C, DIC, R	1,26 mg/l	deceased	Campkin and Davies 1992
3	19 yrs F	MDMA capsule at a Rave	hyperthermia	detected	returned to normal	Nimmo <i>et al.</i> 1993
4	17 yrs M	2 tablets of Ecstasy at a party	T 41 °C, DIC	detected	deceased	Henry <i>et al.</i> 1992
5	18 yrs M	3 tablets of E at a club	T 41,8 °C, dec. BP	0,36 mg/l	deceased	Henry <i>et al.</i> 1992
6	20 yrs M	3 tablets of E at a club	T 40 °C, DIC, R, ARF	0,24 mg/l MDA, MDEA and amphetamine	returned to normal	Henry <i>et al.</i> 1992
7	21 yrs F	several tablets of E at a party	T 41 °C, DIC, R, ARF	0,11 mg/l	died after liver transplant	Henry <i>et al.</i> 1992
8	30 yrs M	10 days after taking E at a party	BP 190/100, ARF, fluid overload	–	haemodialysis, died of cardiac arrest	Bingham <i>et al.</i> 1998
9	20 yrs M	MDMA capsule	T 40 °C, dec. BP, DIC, R, ARF	1,16 mg/l, MDA and amphetamine	deceased	Henry <i>et al.</i> 1992
10	18 yrs F	Ecstasy, amphetamine and alcohol at a Rave	T 42 °C, R, DIC	MDA and amphetamine	liver damage	Jones <i>et al.</i> 1994
11	25 yrs F	3 tablets of E	T 41,9 °C, R, DIC, hypoglycaemic	–	returned to normal	Montgomery and Myerson, 1997
12	18 yrs M	5 tablets of E at a party	T 42,1 °C	detected	deceased	Henry <i>et al.</i> 1992

Table 2.3 Continued

Case	Identity	Dose and environment	Presentation	Blood MDMA	Outcome	References in Podraza (1999:10–12)
13	20 yrs M	18 tablets of E at a Rave	Hyperthermia	4,05 mg/l	returned to normal	Roberts <i>et al.</i> 1993
14	24 yrs M	200 mg of E	T 40,2°C, R	–	returned to normal	Sinarajah and Lavies 1992
15	25 yrs F	1 tablet of E and alcohol at a party	T 41,9 °C, DIC, hypoglycaemic	–	returned to normal	Williams and Unwin 1997
16	19 yrs M	MDMA at a club	T 43,3 °C, DIC, R	MDMA and amphetamine	deceased	Screaton <i>et al.</i> 1992
17	20 yrs F	2 tablets of E at a Rave	T 42 °C, dec. BP	2,3 mg/l	deceased	Mueller and Korey 1998
18	16 yrs F	1 tablet of E	T 42 °C, dec. BP, DIC, acidosis	0,424 mg/l, stomach 28,0 mg/l	deceased	Chadwick <i>et al.</i> 1991
19	32 yrs F	100–150 mg of E	T 41,6 °C, DIC, dec. BP, R	0,65 mg/l	returned to normal	Brown and Osterloh 1987
20	17 yrs M	10 tablets of E and alcohol at a club	T 42 °C, dec. BP, DIC	0,23 mg/l	deceased	Dar and McBrien 1996

Key: ARF = acute renal failure; BP = blood pressure; C = celsius; dec. = decrease; DIC = disseminated intravascular coagulation; E = Ecstasy; F = female; M = male; MDA = methylenedioxyamphetamine; MDEA = methylenedioxyethylamphetamine; R = rhabdomyolysis; T = temperature.

*Fahal *et al.* (1992:29) suggest that a blood MDMA level > 0,2 mg/l is definitive of serious toxicity. Bost (in Podraza 1999:12) supports this conclusion stating a fatal range of 0,95 mg to 2,0 mg/l.

(Source: Adapted from Podraza (1999:10–12) (<http://www.maps.org/research/mdma/podraza.html>))

2.4.6.2 The serotonin syndrome

A number of articles reporting on the adverse reactions associated with the use of Ecstasy in a recreational setting have implicated the serotonin syndrome. Sternbach (1991) and Bodner, Lynch and Lewis (in Podraza 1999:09) state that the syndrome is diagnosed when a known central serotonergic agent is administered resulting in at least three of the following complications:

- 1 Mental status or behavioural change which may include confusion, agitation, hypomania or coma.
- 2 Alteration in muscle tone or neuromuscular activity which may include uncoordination, shivering, tremor, hyperreflexia (exaggeration of reflexes),

myoclonus (twitching or spasm of a muscle or group of muscles) and rigidity.

- 3 Autonomic instability which may include diaphoresis (profuse perspiration), tachycardia, hypertension or hypotension.
- 4 Hyperpyrexia (exceptionally high fever as in heat-stroke).
- 5 Diarrhoea.

Sternbach (1991) and Bodner, Lynch and Lewis (in Podraza 1999:09) further state that when the serotonin syndrome can be diagnosed in the presence of elevated temperature, possible complications include DIC, rhabdomyolysis, cardiac arrhythmias, renal failure, seizures, coma and death. The syndrome has been specifically diagnosed in several cases of Ecstasy-related toxicity and deaths, and appears to be an accurate deduction considering that MDMA is a known central serotonergic agent.

2.4.7 Adverse psychological and physical effects

Contrary to the media's many reports of extreme adverse reactions to Ecstasy, Solowij, Hall and Lee (1992:1170) maintain that in reality they are quite rare. According to these authors (1992:1170), the cases being reported in the clinical literature present extreme exaggeration of the physiological side-effects of Ecstasy, such as hyperthermia and ataxia (impairment of motor control), or with symptoms of toxic psychosis. Often, these symptoms are triggered by some precipitating factor such as a pre-existing medical condition (Dowling, McDonough and Bost 1987:1616) or arise due to extremely high doses being consumed, sometimes with other concurrent drug use (McGuire & Fahy 1991:697).

An animal study conducted by Battaglia, Yoh and de Sousa (1988:270) suggested that the dosage and the number of exposures to MDMA greatly contribute to the neurotoxicity and degeneration of neural serotonin uptake sites. However, Cohen (1995:1143) did not find any relation between an individual's number of exposures to MDMA and recurring symptomatology. This would suggest that side-effects attributed to MDMA may be independent of the number of times one has ingested the drug and that even minimum exposure may elicit adverse symptomatology.

According to Jansen (1997:114), the observation that the duration of Ecstasy use and dosage are not currently related to the probability of developing such symptoms tends to support an examination of psychological causes, and suggests that the current focus on neurotransmitter changes may be misguided – particularly when considering the noticeable lack of change in the behaviour of animals following chronic high-dose injections of Ecstasy. Many reports from persons who have had adverse psychiatric reactions in

association with Ecstasy describe only having taken a few doses. All the same, it is still possible that rigorous scientific studies will eventually establish a link between at least some adverse effects and dose or duration of Ecstasy use (Jansen 1997:114).

It is possible that some users will experience idiosyncratic or allergic reactions to MDMA (Solowij, Hall & Lee 1992:1170). It has been implied that a combination of individual sensitivity or susceptibility and dose may explain the cases of adverse reactions that are serious enough to be documented (Hayner & McKinney 1986:345). Since Ecstasy is a sympathomimetic substance, it is to be expected that some adverse physical side-effects occur. (A sympathomimetic substance is a central nervous stimulant (CNS) that mimics adrenaline responses, <http://2000.01>.) Nevertheless, the majority of people describe these negative effects as mild, if they were experienced at all. Ecstasy produced no more severe side-effects than other widely used drugs such as amphetamine and hallucinogens (Solowij, Hall & Lee 1992:1170).

Even if most users do not experience distressing side-effects from using Ecstasy, there may still be grounds for caution in that the long-term consequences of even problem-free use are as yet unknown.

2.5

MDMA AND THE BRAIN – HOW ECSTASY AFFECTS THE BRAIN

Ecstasy largely affects serotonin-producing nerve cells in the brain. These nerve cells communicate with their neighbours by releasing the chemical messenger serotonin, which transmits electrical signals from one nerve cell to the next. Serotonin neurons originate in the raphe nucleus near the base of the brain or brain stem. By means of long, strand-like extensions known as *axons* these neurons project to practically every area of the central nervous system, including the fore brain and spinal chord (Ricaurte 1997:01–02.) When Ecstasy is ingested, the release of serotonin by these nerve cells may be responsible for the overall sense of well-being and feelings of empathy, happiness and perceived insight that are experienced.

The axon is a long shaft of the cell across which the electrical signal is transmitted. (Axons can extend to as much as 30 cm long.) The dendritic tree composed of dendrites is that part of the neuron which receives input from other nerve cells. The cell body is the part of the cell containing the nucleus and is responsible for the production of the chemical substances (neurotransmitters) that neurons use to communicate with one another (Banich 1997:04). Usually, serotonin is released when an electrical signal travels from

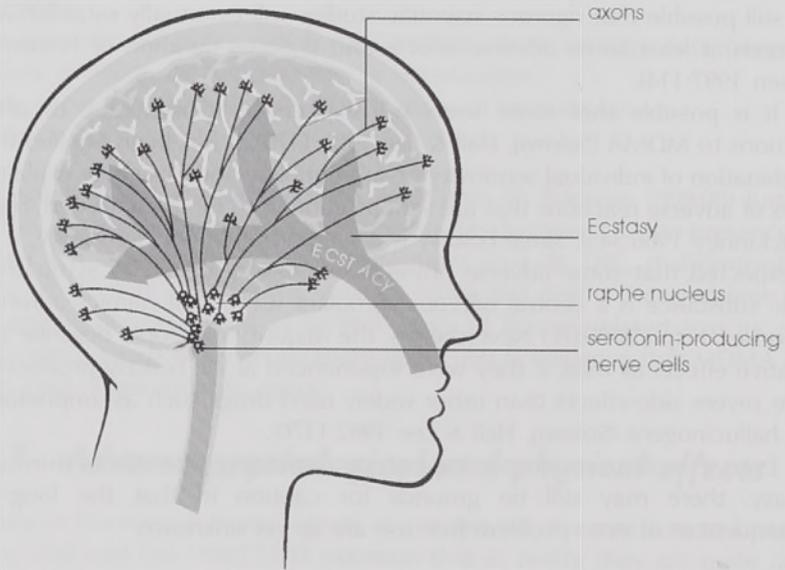


Figure 2.2 Diagrammatic representation of how Ecstasy affects the brain

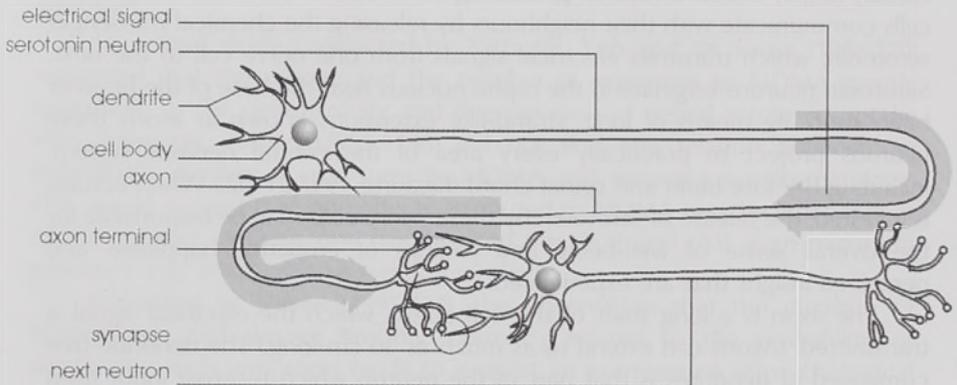


Figure 2.3 The basic parts of a neuron

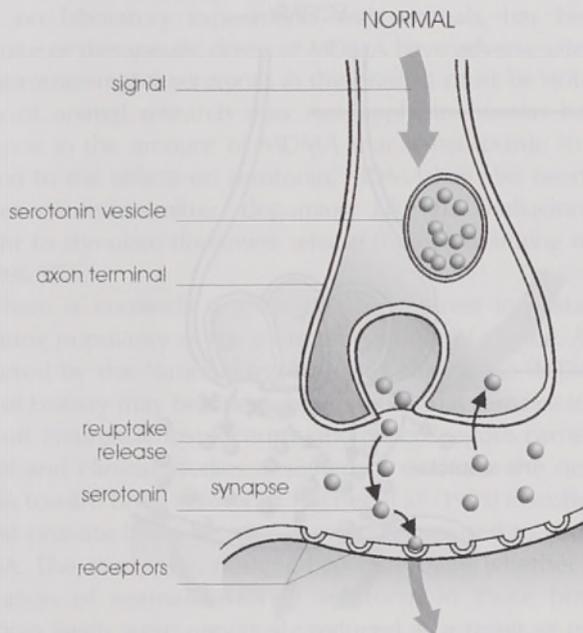


Figure 2.4 A normal serotonin vesicle

the cell body down the axon. Serotonin is stored in tiny vesicles clustered at the ends of these axons and is deposited into a small gap called the *synapse* (Cloud & Ratnesar 2000:65).

The electrical signal causes the filled synaptic vesicles to burst open releasing the chemical serotonin into the area between the neurons called the *synaptic cleft* (Banich 1997:04). Some of the serotonin is absorbed by receptors on the neighbouring neuron. At this point the chemical signal is transformed back into an electrical signal to be passed down the stimulated neuron to other dendrites. These, in turn, trigger other neurons. This process continues to form long networks of activated brain cells (Eisner 1989:158). The rest of the serotonin is metabolised (broken down) by enzymes or reabsorbed by the releasing neuron (Cloud & Ratnesar 2001:65).

When individuals take Ecstasy, MDMA causes the nerve cells to release all the stored serotonin at once, even without an electrical signal. The chemical floods the synapse, overwhelming the serotonin receptors. MDMA blocks the reuptake of serotonin, thereby preventing the reabsorption of serotonin back

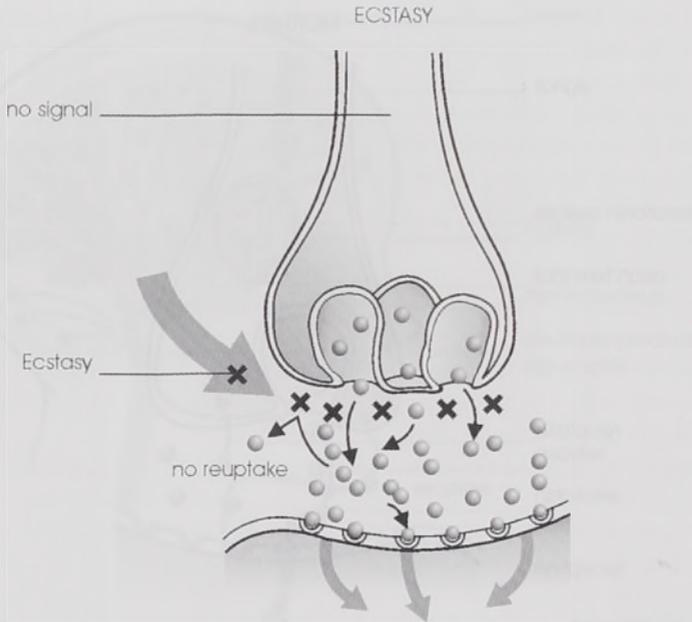


Figure 2.5 An Ecstasy-affected vesicle

into the neuron and further increasing the concentration in the synapse (Granquist 1992:01). The acute 'rush' of serotonin may cause damage to the axonal endings. Most studies suggest that nerve endings die off, but some indicate that they may grow back abnormally. These axons may return in denser formation and may no longer reach the areas of the brain in which they are needed (Cloud & Ratnesar 2000:65) thereby making it impossible for the serotonin nerve cells to communicate with neighbouring cells the way they normally would (Ricaurte 1997:02). (Refer to Figure 2.6 for representation of axonal damage.)

2.6 NEUROTOXICITY

A growing body of literature suggests a neurotoxic effect of MDMA on serotonergic nerve terminals. According to Elk (1996:352), the most controversial

effect of MDMA at this time is its possible irreversible neurotoxicity. A suggestion, based on laboratory experiments with animals, has been made that even moderate or therapeutic doses of MDMA have adverse effects on the amount of the neurotransmitter serotonin in the brain. It must be noted, however, that the results of animal research may not apply to human beings because of the difference in the amount of MDMA that is neurotoxic to rats and humans. In addition to the effects on serotonin, MDMA has also been suspected to act on another neurotransmitter, dopamine. Like the hallucinogen LSD, MDMA is thought to stimulate dopamine release thus contributing to behavioural toxicity (Elk 1996:352).

There is currently a great deal of interest in Ecstasy as a result of its increasing popularity as the recreational drug of choice. According to research supported by the National Institute of Drug Abuse (NIDA) in the USA, heavy users of Ecstasy may be risking brain injury that remains long after the high has worn off. Neuroscientist Ricaurte and his colleagues carried out a series of pre-clinical and clinical studies designed to evaluate the neurotoxic potential of MDMA toward brain serotonin. Fischer *et al.* (1995) investigated the regrowth of rat and primate brain neurons previously exposed to extremely large doses of MDMA. The study was designed to determine whether there was long-term restoration of normal levels of serotonin in those brain regions in which serotonin levels were previously reduced as a result of exposure to very large amounts of MDMA. Also examined was whether the regrowth of serotonin nerve terminals (reinnervation) restores the original brain structures.

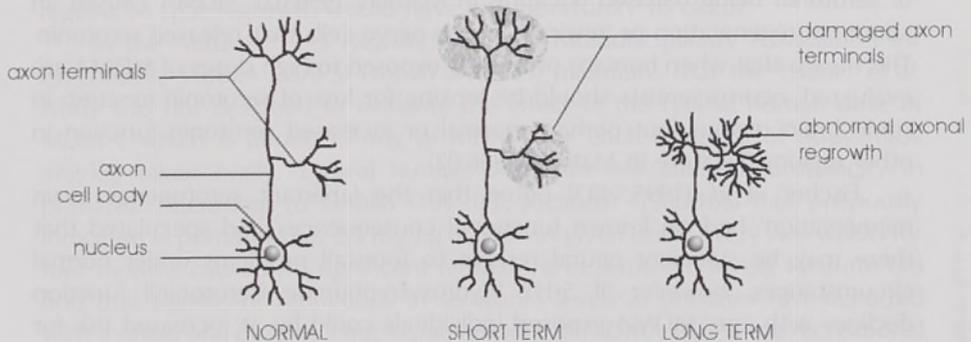


Figure 2.6 *Abnormal regrowth of damaged axon terminals*

Note: The illustration on the left shows a normal neuron. The shaded area in the middle illustration shows the axon terminals of the neuron that are damaged by MDMA. The illustration on the right shows how 12 to 18 months after being damaged by MDMA, serotonin-producing nerve fibres have regrown excessively in some areas and not at all in others (Mathias 1996:01).

Fischer *et al.* (1995:5476) determined that a single dose of MDMA only slightly higher than the size of doses normally taken by humans significantly damaged brain cells that produce serotonin. Ricaurte (in Mathias 1996:01) reported that 12 to 18 months after the brains of squirrel monkeys had been damaged by MDMA, serotonin-producing nerve fibres had regrown abnormally in some brain regions and failed to regrow at all in others.

According to Ricaurte (in Mathias 1996:02), the doses of MDMA that some people take recreationally closely approach the doses known to produce neurotoxic effects in animals. The major question is whether the neuronal changes seen in animals from MDMA exposure occur in human beings who use the drug. To help answer that question, Ricaurte conducted separate clinical studies using brain imaging techniques to evaluate the possibility of long-term brain damage in humans who have previously used MDMA. (See Section 2.6.2.4 for positron emission tomography scans.) These studies also assessed the potential functional consequences of such neuronal damage on aspects of mood, movement, memory, impulse control, aggression and sleep cycles (Mathias 1996:02).

Ricaurte (in Mathias 1996:02) maintains that determining the functional consequences of MDMA exposure 'may be more complex than previously thought'. The long-term study with squirrel monkeys showed that in some brain areas, such as those containing structures involved in memory and learning, damaged neurons failed to recover. However, in other brain areas, particularly those involved in controlling functions such as sleep and appetite, damaged neurons regrew excessive nerve fibre, resulting in an overabundance of serotonin being released (Ricaurte in Mathias 1996:02). MDMA caused an abnormal regeneration or 'rewiring' of the nerve cells that released serotonin. This means that when humans previously exposed to high doses of MDMA are evaluated, neuroscientists should be looking for loss of serotonin function in some brain regions, but perhaps normal or increased serotonin function in other regions (Ricaurte in Mathias 1996:02).

Fischer *et al.* (1995:5483) noted that the 'aberrant serotonergic brain reinnervation' had no known functional consequences and speculated that there may be sufficient neural reserve to forestall problems under normal circumstances, however 'if 5-HT [hydroxytryptamine (serotonin)] function declines with age, MDMA-exposed individuals could be at increased risk for developing age-related cognitive impairment'. The results are further evidence that people using high doses of MDMA may be putting themselves at significant risk of brain injury.

Researchers are now trying to determine why the nerve cells grow back normally, abnormally or not at all and whether the damaged nerve tissue disrupts mood, memory and other functions associated with serotonin (Cramer 1995:01).

2.6.1 *Implications of animal studies for human use*

Doblin (1995:03) maintains that in order to evaluate what implications the Fischer *et al.* (1995) study has for humans using MDMA for recreational or therapeutic purposes, the following questions must be addressed:

- 1 How does the amount of MDMA administered to the animals relate to human use patterns?
- 2 What are the consequences of MDMA-caused serotonin reductions in animals?
- 3 What evidence is there that MDMA causes serotonin reductions in humans?
- 4 If there are MDMA-caused serotonin reductions in humans, what are the consequences?

2.6.1.1 *Animal versus human doses*

Doblin (1995:03) explains that the Fischer *et al.* (1995) study was designed to determine whether there was long-term restoration of normal levels of serotonin in those brain regions in which serotonin levels were previously reduced as a result of exposure to very large amounts of MDMA, and to investigate whether the regrowth of serotonin nerve terminals (reinnervation) restored the original brain structures in the rats and primates. It was therefore necessary to cause large initial reductions in serotonin levels in multiple brain regions so that regrowth would have an opportunity to occur.

In the Autumn 1995 Newsletter of the Multidisciplinary Association for Psychedelic Studies (MAPS) Doblin (1995:03) maintains that the Fischer *et al.* study was not designed to evaluate the effect of the typical human dose of MDMA, which is about 1,7 mg of MDMA for each kilogram of body mass (mg/kg) taken orally. Typical human doses do not cause neurotoxicity in primates. According to Doblin (1995:03), Ricaurte (1988:166) had previously indicated in primates that 2,5 mg/kg of MDMA given orally every two weeks for four months caused no significant reductions in serotonin levels. Ricaurte did find that significant reductions in serotonin levels in primates first occurred with a single oral dose of 5 mg/kg, an amount of MDMA that some recreational users do take. This dose produced no reductions in most primate brain regions tested two weeks after administration, however, there was a 21 per cent reduction in serotonin in the thalamus and a 16 per cent reduction in the hypothalamus. Thus, the 'no effect' level in primates for serotonin reductions is somewhere between an oral dose of 2,5 mg/kg and 5,0 mg/kg. Whether there is a direct connection between these initial reductions in serotonin levels and

structural damage (neurotoxicity) has been questioned. In addition, no associated functional or behavioural consequences have been noted either from these minor and localised reductions or from the larger reductions caused by the higher doses administered to the primates in this experiment (Doblin 1995:03).

In order to cause substantial serotonin reductions in multiple primate brain regions, it was necessary to administer a subcutaneous injection of 5 mg/kg twice daily, four days in a row, for a total of eight injections. According to Doblin (1995:04), the relevance of the data from this study to the human therapeutic or recreational use of MDMA is not clear, since most people ingest MDMA orally and not by injection. Furthermore, it is practically unheard of for someone to use MDMA for four days in a row because tolerance to the desired positive effects develops that cannot be overcome by increasing the amount ingested, thereby distinguishing MDMA from drugs such as cocaine or heroin.

The 5 mg/kg dose of MDMA injected in the primates is almost three times larger than the typical human dose of 1,7 mg/kg. In an earlier study, Ricaurte (1988:166) indicated that subcutaneous injection of MDMA is roughly twice as toxic as oral administration. Doblin (1995:04) therefore argues that each injection given to the primate is equivalent to slightly less than six times the typical oral human dose. Since there were eight injections, each primate received the rough equivalent of forty-five times the amount of MDMA that a person would ingest in a typical MDMA session. This estimate is very tentative, since it multiplies dose, frequency and route of administration effects, even though there may not be a linear relationship between these factors and serotonin reductions. Furthermore, the typical human dose varies from person to person depending on the person's height and weight. O'Callaghan (1993 in Doblin 1995:04) uses the smaller figure of twenty-five times the typical dose to estimate the relationship between the doses given to the primates in this study and the typical human dose.

Data from the Fischer *et al.* (1995) study can be used to develop hypotheses about the effects of MDMA in humans but no clear conclusions can be drawn because there are marked species-dependent differences in response to the administration of drugs. For example, rats respond differently to MDMA from mice in some studies. In this study, the rats responded differently from the primates in that most rats – but only some primates – re-established normal serotonin levels. Primate data is most useful in estimating the effect of a drug in humans, but even primate data needs to be confirmed by human studies (Doblin 1995:04). Neither the relative safety nor risk of MDMA can be determined conclusively without human studies.

2.6.1.2 Consequences of serotonin reductions in animals

The long-term functional or behavioural consequences in animals who have been administered large amounts of MDMA are still unknown. No obvious impairments have been noted (Doblin 1995:05).

2.6.2 Human studies

In human beings it is very difficult to assess the state of brain serotonin nerve cells. At present, this can only be done by studies of 5-hydroxyindoleacetic acid (5-HIAA) in the cerebro-spinal fluid (CSF) and positron emission tomography (PET) studies of serotonin transporters (Ricaurte 1997:03).

2.6.2.1 Studies of 5-hydroxyindoleacetic acid in cerebro-spinal fluid

CSF studies involve measuring 5-HIAA, the breakdown product of serotonin, in spinal fluid. CSF is simply the fluid that bathes the brain and the spinal cord. Once serotonin produced by nerve cells has been released, it is metabolised (or broken down) into 5-HIAA, which accumulates in the spinal fluid. Spinal fluid is easily obtained in living humans by doing a lumbar puncture. In order to determine if 5-HIAA in the CSF could serve as a marker of MDMA neurotoxicity, Ricaurte and McCann (1997:03) first carried out a series of studies in monkeys. When 5-HIAA in the spinal fluid of the monkeys given MDMA was measured, it was found that if that monkey had a 70 to 90 per cent reduction of serotonin and 5-HIAA in the brain, that same animal had about a 50 to 60 per cent loss of 5-HIAA in the spinal fluid. According to Ricaurte, two important points arise from these studies. One is that 5-HIAA can be used as an indirect marker for MDMA-induced serotonin neurotoxicity in primates and the other is that the degree of loss or depletion of 5-HIAA in the spinal fluid tends to underestimate the degree of loss of 5-HIAA in the brain (Ricaurte *et al.* 1997:03).

Ricaurte and his colleagues then conducted a similar study in a group of people who had used MDMA extensively in the past. On average, these individuals reported using MDMA about ninety-five times over a period of five years. The group of people that were investigated had used MDMA four times a month roughly every week. Normally, they reported taking a dose of 170 mg (an estimated dose), which translates to one or two tablets every time they used Ecstasy. Individuals were also asked when the last time they had taken the drug was. On average, the group investigated had stopped using MDMA about four months before. The MDMA group was compared with a control group

that was reasonably well matched for size (there were 28 subjects in the control group and 30 in the MDMA group), age, height, weight and level of education. The number of men and women in the two groups was also comparable (Ricaurte 1997:04).

What was found in the spinal fluid of these people was reminiscent of what was found in the spinal fluid of the MDMA-tested monkeys. There was a reduction in the amount of 5-HIAA in the CSF of the MDMA group. In the control group, the CSF 5-HIAA concentration was about 15 ng/ml, whereas in the MDMA group it was reduced to almost 10 ng/ml (Ricaurte 1997:04), that is, the MDMA users had roughly 32 per cent less serotonin metabolite in their spinal fluid on average than the group of controls (Doblin 1995:03). This was a statistically significant change. Markers for dopamine and norepinephrine or noradrenaline were not affected.

It should be stressed that this data does not establish definite evidence of serotonin neurotoxicity in MDMA-exposed individuals. CSF 5-HIAA is only an indirect chemical measure of the serotonin nerve cells in the brain. Additional studies are needed to assess the neurotoxic potential of MDMA in humans further (Ricaurte 1997:04). Nevertheless, CSF data does suggest that MDMA may produce neurotoxic effects in humans.

2.6.2.2 Evidence for serotonin reductions in humans

There is no conclusive evidence demonstrating that MDMA causes serotonin reductions in humans. Studies using spinal taps and brain scans to evaluate people before and after administration of MDMA will be needed to determine definitively whether MDMA causes serotonin reductions in humans. Doblin (1995:04) maintains that in order to put McCann and Ricaurte's CSF findings into context, it is important to note that the normal range of serotonin metabolites in spinal fluid is quite large. Some people naturally have twice as much or more than others, thus a difference of 32 per cent between groups, although statistically significant, is a relatively small shift within the normal range of serotonin metabolite levels.

According to Doblin (1995:04), whether the 32 per cent difference can be ascribed to MDMA use is questionable, largely because the serotonin metabolite levels of the MDMA users were not measured before they began to use the drug. Doblin (1995:04) points out that this study used a matched control group design instead of pre- and post-measures on the same subjects, therefore the difference in serotonin levels could be due to uncontrolled factors resulting from an imprecise matching process. For example, some personality factors such as risk-taking behaviour (eg illegal drug use) have been linked to lower serotonin metabolite levels. He also notes that the volunteers in this study had exposure to other drugs as well as Ecstasy (MDMA), while the

control group was relatively drug naïve. Furthermore, MDMA sold illegally is often impure. Serotonin reductions, if they occurred as a result of drug use, could be due to impurities and not to MDMA itself (Doblin 1995:04).

2.6.2.3 Consequences of serotonin reductions in humans

While McCann *et al.* (1994) found lower serotonin metabolite levels in MDMA users compared with controls, no harmful functional or behavioural differences between the subjects in the MDMA and control groups were found. In fact, the MDMA users exhibited less hostile and impulsive personality traits and increased harm avoidance, constraint and control than the members of the control group (Doblin 1995:05). This finding is particularly surprising, since it runs contrary to previous research which has associated low levels of serotonin with increased violent and impulsive behaviour. Possibly, this finding is due to MDMA's psychological effect of empathy rather than any long-term change in serotonin (Doblin 1995:05).

a Sleep electroencephalogram (EEG) data

A study by Allen, McCann and Ricaurte (1994:560) examined the EEGs of human users because of the function of serotonin in sleep. MDMA was found not to cause gross abnormalities in the quality of sleep in human users, suggesting that the systems responsible for sleep were intact. MDMA also did not change rapid eye movement (REM) or stages 3 and 4 slow wave sleep (SWS) periods. According to Granquist (1995:03), this is not what would be expected from experience with chemical or anatomical lesioning of the serotonergic systems in animals.

Sleep EEG data from this study indicated that the MDMA group averaged 19 minutes less total sleep a night than members of the control group. MDMA users had about 37 minutes less of stage 2 non-REM sleep, generally considered to be of lesser importance than other stages of sleep in terms of restorative function. MDMA users actually spent about 18 minutes more than controls in the stages of sleep considered essential for physical and biological restoration, stages 3 and 4 non-REM sleep and REM sleep (Doblin 1995:05).

The fact that MDMA does not reduce REM and SWS, while reducing the lighter stage 2 sleep, may indicate that MDMA users experience better-quality sleep. REM and SWS are considered important states in sleep (being linked to memory and psychiatric health), while stages 1 and 2 sleep are not generally regarded as being important (Granquist 1995:03). The sleep patterns of the MDMA users could perhaps be considered more efficient and more restorative than those of the control group, because they went more quickly into deep sleep (Doblin 1995:05).

At present there is no evidence of harmful neurotoxic effects in the current population of MDMA-exposed people.

2.6.2.4 Positron emission tomography (PET) scans

Advancement in neuro-imaging techniques, such as PET scans, and the development of carbon-11 [¹¹C] called McN-5652 – a radioligand that selectively labels the serotonin transporter (a structural element of brain serotonin neurons) – has made it possible to see and assess the status of brain serotonin neurons in living human beings (McCann *et al.* 1998:02). The brain scans of drug users have yielded the first direct evidence that recreational Ecstasy use can trigger long-lasting changes in the human brain. PET was used to scan the brains of 15 MDMA users. For comparison, the brains of 15 people who had used other drugs such as cocaine, heroin and marijuana but had never taken MDMA, were also scanned (Concar 1997:01).

A key difference came to light when the researchers injected participants with the radioligand [¹¹C]McN-5652, a radioactive substance designed to ‘light up’ in the presence of healthy serotonin (5-HT) synapses. Radioligand [¹¹C]McN-5652 binds to a protein that transports serotonin across cell membranes (Concar 1997:01). The control subjects had normal levels of the transported protein but the MDMA users had deficiencies in all areas of the brain thereby indicating that recreational MDMA use can lead to large, dose-related decreases in the brain serotonin transporter (McCann *et al.* 1998:03). Taken in combination with results of previous studies showing selective decreases in concentrations of CSF 5-HIAA in MDMA users, and similar findings in MDMA-treated animals with documented neurotoxic lesions, these data suggest that human MDMA users are susceptible to MDMA-induced brain serotonin neural injury (McCann *et al.* 1998:07). (See Figure 2.7.)

All participants in the MDMA group reported that they had refrained from using MDMA or other psychoactive drugs for at least three weeks before the study, which suggests that the decreases seen in brain [¹¹C]McN-5652-labelled serotonin transporter sites were not due to pharmacological effects of MDMA or other drugs (McCann *et al.* 1998:07). The results do not, however, rule out the possibility that decreased serotonin transporter binding sites are secondary to pre-existing differences in serotonin function in MDMA users compared with controls, but since none of the MDMA users had a neuropsychiatric disorder in which serotonin has been implicated, McCann *et al.* (1998:07) maintain that this possibility is unlikely. Finally, although most of the MDMA users had experimented with other recreational drugs, none was a known serotonin neurotoxin in human beings and was not likely to account for changes in serotonin binding (McCann *et al.* 1998:07).

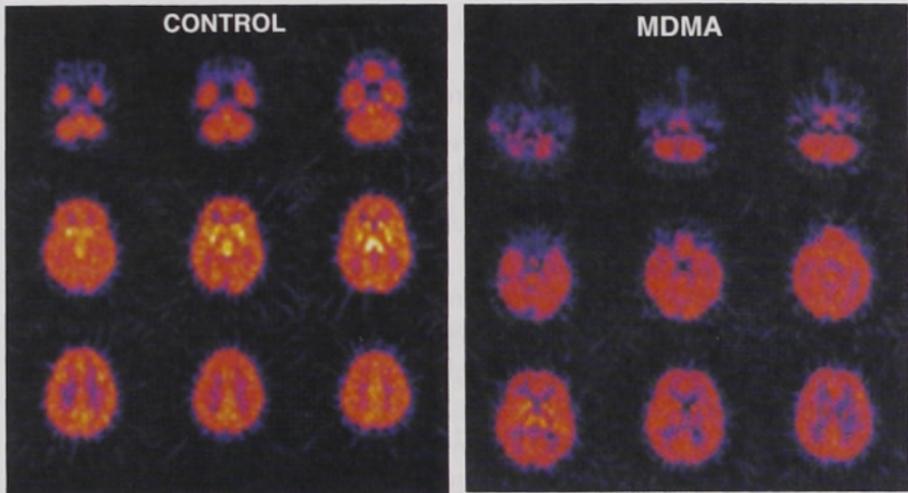


Figure 2.7 Axial PET images of participants in the control group and MDMA group showing distribution of specific $[^{11}\text{C}]\text{McN-5652}$ binding

Note: PET images were acquired 55 to 95 minutes after tracer injection (McCann *et al.* 1998:06).

McCann *et al.*'s (1998:07) findings do not draw conclusions about reversibility or permanence of MDMA-induced changes in brain serotonin transporters. Although no correlation between the length of abstinence and the extent of decrease in $[^{11}\text{C}]\text{McN-5652}$ binding was found, McCann *et al.* believe MDMA-induced changes may be reversible. Sample sizes and various other factors could have contributed to the apparent absence of recovery. More MDMA users with varied durations of abstinence and drug exposure histories must be studied to show whether serotonin terminal structure and function return to normal over time. Studies in non-human primates show that MDMA-induced changes in serotonin terminal markers persist for longer than one year after doses of MDMA similar to those used by some human recreational MDMA users (McCann *et al.* 1998:07).

In short, the data suggests that people who use MDMA as a recreational drug are 'unwittingly putting themselves at risk of developing brain serotonin neural injury' (McCann *et al.* 1998:08). In addition, systematic studies of MDMA-exposed individuals with highly selective brain serotonin transporter deficits may give important insights into the functional role of brain serotonin in human behaviour. According to McCann *et al.* (1998:08), the potential functional consequences of MDMA-induced brain serotonin neurotoxic lesions are not yet clear, but may include depression, anxiety, memory disturbance

and other neuropsychiatric disorders in which brain serotonin has been implicated.

The following questions regarding recreational Ecstasy use therefore arise:

- 1 What does all of this mean to ravers who love their Ecstasy?
- 2 How do these findings apply to the average recreational users, most of whom are quite moderate in their usage, that is, they do not use it every weekend?

There are a number of different opinions as to the relevance of the serotonin neural damage. Doblin (in RaveSafe 1998:01), mentions that

an important point to note is that the subjects were tested for psychiatric disorders, such as anxiety and depression, and all were found to be normal. In other words, these reductions in transporter binding relative to the control group existed without any anxiety and depression, as established by the experimenters themselves. This is in line with animal experiments which show that considerable persistent changes do not result in persistent behavioural changes in these animals. They cannot be distinguished from controls. While Ecstasy may cause some brain changes, the evidence for depression and anxiety as a long-term time bomb is entirely lacking especially if the control group are other drug users. So far, these changes in the serotonin transporter are without proven effect.

Parry (in RaveSafe 1998:02) of the Medical Research Council in South Africa commented that the same could be said for alcohol. Alcohol causes major neurological damage as a result of causing a thiamine (vitamin B12) deficiency, but only in very large doses over a long period of time. While no precise information on the quantity and frequency of Ecstasy use among the participants is available, Parry (in RaveSafe 1998:02) attests it can be assumed that participants used at least one pill a week. According to McCann *et al.* (1998:05), participants in the PET study had generally used MDMA on more than 200 occasions and over a four- to five-year period.

Parry (in RaveSafe 1998:02) further points out that the seriousness of the effect of long-term (eg one year) or permanent loss of functioning of serotonin neurons is not very clear. What McCann *et al.* (1998) say is that it *might* have broad implications for many neuropsychiatric illnesses in which brain serotonin neurons have been implicated, for example, depression, anxiety and cognitive dysfunction. McCann (in Concar 1997:02), nevertheless, maintains that the brain scans provide clear evidence that MDMA can damage serotonin synapses in humans and her message to ravers is 'if you're going to use MDMA, use it in moderation'. According to Parry (in RaveSafe 1998:02), the bottom line is that Ecstasy use 'does affect your brain (structurally) in ways which could be

permanent (or at least long lasting) and this may affect you psychologically and in other ways'.

2.6.2.5 *Memory impairment*

Memory function in MDMA-exposed individuals merits special examination for various reasons (Bolla, McCann & Ricaurte 1998:02):

- 1 Brain serotonin (5-HT) appears to play a role in mnemonic or memory function.
- 2 In animals, MDMA severely damages 5-HT axons in the hippocampus and other brain regions implicated in learning and memory (eg the thalamus).
- 3 Case reports of memory impairment in some MDMA users and in several studies suggest that MDMA users have impaired verbal memory function.

Since previous studies have included subjects who may recently have used MDMA or other centrally acting nervous system drugs, it is not completely clear whether deficits in MDMA users depict neurotoxic effects of MDMA, pharmacologic effects of drugs or drug withdrawal (Bolla, McCann & Ricaurte 1998:02).

The purpose of the study conducted by Bolla, McCann and Ricaurte was to determine whether memory deficits existed in MDMA users who were drug-free for at least two weeks and if they do, whether memory deficits are dose-related. Furthermore, this study examined whether memory deficits in MDMA users correlate with decrements in CSF 5-HIAA which serves as a reliable indicator of MDMA-induced brain 5-HT neurotoxicity in non-human primates (Bolla, McCann & Ricaurte 1998:02). Twenty-four abstinent MDMA users and twenty-four control subjects were compared on several standardised tests of memory, after matching subjects for age, gender, educational level and vocabulary score (verbal intelligence).

Bolla, McCann and Ricaurte (1998:07) found that abstinent MDMA users have a deficit in visual and verbal memory, and that higher average monthly doses of MDMA are associated with greater decrements in memory function. Furthermore, the results indicate that lower levels of CSF 5-HIAA (an indirect measure of central 5-HT function), are associated with poorer memory performance, suggesting that MDMA-induced brain serotonin neurotoxicity may account for memory impairment in MDMA users. Lastly, the results indicate that both baseline intelligence and gender influence the effects of MDMA on memory function. Women were less susceptible than men to MDMA dose-related decreases in memory (Bolla, McCann & Ricaurte 1998:08).

These findings are generally consistent with reports of memory problems in previous studies, although some important differences are evident. Counter to findings in a previous report issued by Parrott *et al.* (1998) in which persons

with low MDMA exposure (ten or fewer doses) showed memory deficits, only subjects with high total monthly MDMA dosages were found to have memory deficits in this study. Bolla, McCann and Ricaurte (1998:07) feel the differences between the two studies may be attributed in part to the fact that subjects in their study abstained from psychoactive drugs (including MDMA) for at least two weeks. Thus acute or partial residual drug effects, or drug withdrawal, may have caused the memory disturbances noted in previous studies. Another possibility is the subjects in the study by Parrott et al. (1998) may have used extremely high doses of MDMA, causing brain 5-HT neurotoxicity despite the small number of separate drug exposures. Since some people attending Raves report using doses of MDMA that are clearly neurotoxic in non-human primates, the latter possibility cannot be excluded (Bolla, McCann & Ricaurte 1998:07).

The observation that higher exposures to MDMA are associated with memory impairment is consistent with findings in animals, indicating that higher dosages of MDMA produce greater neurotoxic lesions. Significantly, only individuals with more profound decrements in CSF 5-HIAA (presumably reflecting a greater extent of 5-HT injury) displayed obvious difficulties with memory function. These results correspond with a growing body of literature that denotes that large lesions (>80 per cent) of neural systems are often necessary for functional deficits to be apparent (Bolla, McCann & Ricaurte 1998:08).

Results from this study also indicate that individuals with lower intellectual abilities (ie vocabulary scores) display greater decrements in memory performance with higher doses of MDMA. Similar interactions are seen in individuals exposed to other neurotoxins such as solvents and aluminium. Bolla, McCann and Ricaurte (1998:08) explain this effect by the concept of *cognitive reserve*, which assumes that individuals with higher intellect have a higher threshold for developing neurocognitive effects after brain insult. As regards the gender discrepancy pertaining to the MDMA dose-related decrease in memory function (where women were less susceptible than men), studies in adults 19 to 50 years old have found that women tend to have better memory abilities whereas men tend to have better reasoning abilities (Bolla, McCann and Ricaurte 1998:08). The Bolla, McCann & Ricaurte results are consistent with these reports because gender differences were also seen in control subjects, with women performing better than men.

A few possible limitations of this study should be pointed out. As with all retrospective studies, the possibility exists that pre-existing differences between MDMA users and non-users underlie differences in memory function and 5-HIAA. Thus, people with low CSF 5-HIAA may be predisposed to use MDMA and to have memory problems. However, Bolla, McCann & Ricaurte (1998:08) assert that the dose-related decreases in both CSF 5-HIAA (similar to those that

have been found in non-human primates) and memory function make this unlikely. Since several subjects in the control group also used recreational drugs (although not MDMA), a tendency to use drugs cannot completely explain the biological and behavioural differences found in MDMA users in this study (Bolla, McCann & Ricaurte 1998:08).

The observation that higher exposures to MDMA are associated with cognitive deficits is clearly worrying given the widespread use of Ecstasy amongst the youth, as these effects could cause problems for students in both secondary and tertiary education who are studying and preparing for examination. Nevertheless, no single line of evidence can be taken as conclusive proof that MDMA is neurotoxic in humans. More studies are needed to depict better the neurotoxic potential of MDMA in humans and its functional consequences.

2.7 ETHICAL DILEMMA

An important issue raised at the 1998 Novartis Foundation Press Conference is the difficulty of reaching conclusions from data produced from retrospective research on humans, rather than prospective research. This is the difference between studying the brains of humans who say they have taken Ecstasy in the past (whether recently or not), and studying the brains of humans before and after actually giving them MDMA. In the former case there are obvious methodological difficulties – differences in the brains of MDMA users compared to a non-using control group could be pre-existing. Another problem is pill composition – a lack of certainty about what the users have actually taken (Novartis Foundation Press Conference 1998:02).

Nevertheless, despite the clear shortcomings of retrospective research, prospective research is not being carried out because of perceived ethical difficulties. Ricaurte (Novartis Foundation Press Conference 1998:02) mentioned two problems he saw with this kind of research on humans. Firstly, he believed it would be unethical to ask subjects to take part in a study the purpose of which would be to 'see whether or not we can destroy serotonin nerve terminals in your brain'. Be that as it may, Ravers are taking Ecstasy on dance floors all over the world every weekend and there is unlikely to be a shortage of people willing to volunteer for MDMA research. In all honesty, not to do this kind of research might even be seen as being unethical.

Ricaurte's second problem concentrated on what he saw as the lack of medical necessity to test MDMA in this way, as there is still no documented evidence that MDMA has any medical use. Grob, an American psychiatrist,

who is considering the possible uses of MDMA within therapy, specifically for treating post-traumatic stress disorder, pointed out the catch-22 nature of this argument. He maintains that 'there is no documented evidence because putative medical application has not been put to rigorous testing. There have been no authorised, sound, methodological investigations, in large part because of the concerns Ricaurte is raising about neurotoxic potential' (Novartis Foundation Press Conference 1998:03). Hence, there is no evidence because the studies have not been done, but the studies cannot be done until there is some evidence to support them.

Nonetheless, it is crucial that research continues on the exact mechanism of MDMA-induced toxicity and also research into the possible beneficial therapeutic uses of MDMA, so that the risks and benefits of MDMA can be accurately balanced.

2.8 CONCLUSION

While the popularity of MDMA is escalating, especially amongst students and young people in the Rave party and club scene, research studies reinforce concerns that Ecstasy use can affect the brain some time after the immediate effects of the drug have worn off. The real fear is that Ecstasy may cause long-term permanent effects on the human brain in much the same way as observed in animal experiments. This remains unproven but many experts see such long-term effects as a bigger potential threat to public health than the much-more-publicised short-term risks (British Parliamentary Office of Science and Technology (POST) report in McFadyean 1997:72).

MDMA is no exception to the rule that every drug has potentially serious side-effects. As with any substance, some people tend to be very sensitive to small quantities of MDMA. Others may take unusually large amounts, especially in recreational contexts. Concern is expressed that the doses taken by some young people at Raves considerably exceed the normal 'advisable' human dose of 1,7 mg/kg. It would therefore not be surprising if some people took enough MDMA to cause long-term reductions in their levels of serotonin in some brain regions (Doblin 1995:05), to place them at a higher risk for developing the wide array of neuropsychiatric disturbances in which serotonin neurons have been implicated, such as depression, mood swings, impulse control problems, aggressive tendencies, sleep disturbances, cognitive dysfunction and anxiety disorders.

Although research studies do not prove beyond doubt that these are likely outcomes, they are a warning to Ecstasy users. (See Figure 2.8.) The challenge

If you're gonna take **E** know the deal

If you take E now, know that you are part of a massive experiment because the long term effects cannot be determined with certainty. Unlike other pills, E has not been medically tested.

What is known is that some people have suffered allergic reactions to E. Some ravers have died suddenly from insufficient fluid intake and heat exhaustion even on low doses.

There is some evidence that taking E might cause neural damage in the brain resulting in possible susceptibility to neuropsychiatric disturbances at a later stage. Nobody can be absolutely sure this will happen but it's some risk for an all night "jol".

There's some confusion about how much water to drink on E. When dancing, slowly sip about a pint of water or Energade an hour to replace lost fluids and sodium. Do not drink alcohol as this dehydrates you even more.

Remember to take breaks from dancing and to chill out regularly.

Figure 2.8 A warning to Ecstasy users

for educators, drug counsellors and others dealing with young people using Ecstasy is to convey the message that in later years they may be susceptible to neuropsychiatric disturbances as a result of their Ecstasy use. How effective this message will be depends on how one gets through to an individual. Caution is nonetheless imperative with recreational Ecstasy use until further studies can ascertain the extent to which the drug is indeed dangerous in humans.

CHAPTER 3

RAVES AND THEIR CULTURE

3.1 INTRODUCTION

In South Africa's youth culture a phenomenon known as *Rave* – a combination of energetic 'techno' music, lasers, visual effects, clothes and an assortment of drugs – exists. Driven by the idealism of PLUR, ravers portray themselves as friendly individuals, looking out for one another to ensure a good time and create a positive ambience. Sometimes recreational drugs are used to enhance the experience. In tune with the music, ravers 'groove' to the beats, thriving on the energy of the tracks played by the disc jockey (DJ). Dancing hard into the night, ravers tire into the early morning, exhausted from their energetic rituals (<http://1998:01>). This chapter will consider what Raves are, as well as the underlying philosophy of a raver. What is causing thousands of young people to gather in empty warehouses or nightclubs and listen to music from 22:00 until 8:00? Who are the ravers? Is the Rave movement new or is it some form of 'archaic revival'?

3.2 WHAT IS A RAVE?

3.2.1 Contemporary definition of Rave

A Rave is a social event, primarily an all-night dance party open to the general public, where loud techno music is played and many participants indulge in a number of different chemicals. According to Brown and Behlendorf (1995:03), the participants experience a sense of community and elevated consciousness through the hearing of music and responding to it through dance, a positive

change of mood and both spoken and unspoken interaction with other participants.

Brown and Behlendorf (1995:03) maintain that Raves tend to comprise the following key elements:

- venue which may be a warehouse, open field, dance club or other exotic location
- at least one large, amplified stereo sound system
- skilled DJs who provide a continuous mix of dance-orientated electronic music, usually techno, house, or jungle music
- colourful moving lights, lasers and strobes
- night-time hours, usually from 22:00 or 23:00 until sunrise
- attendance of at least 50 people (varies widely from region to region around the world; some Raves routinely attract over 10 000)
- use of recreational drugs among a percentage of the participants (varies widely from Rave to Rave; some Raves are substance-free)
- non-use of alcohol (varies from Rave to Rave)
- selling of non-alcoholic 'smart drinks', T-shirts and DJ mix tapes
- retro and 'little kid' fashions
- 'chill out' areas or rooms featuring ambient music.

Raves are advertised via flyers, posters, word of mouth, Internet mailing lists and Web pages. Places where flyers can be found include independent compact disc (CD) or record stores, alternative clothing shops, Internet cafés, nightclubs and other student 'hangouts' near universities or technikons. The name of the Rave appears on the flyers, however, the word *Rave* is not usually displayed due to negative connotations applied by people unfamiliar with the scene (Brown & Behlendorf 1995:08). Many Rave flyers use pagan and religious symbolism. Enlightenment is a common theme as well as love and kindness (Stiens 1997:12). (See Figure 3.1.) Raves do not only take place in urban areas. They often take place in small towns, rural areas, out in the desert, on rooftops, in parking garages, on the beach, or anywhere where people want to dance all night long (Brown & Behlendorf 1995:08).

While raving may be collective in nature, it is nevertheless a highly subjective experience (Stiens 1997:01). One person's best Rave may be another's worst. It is important to bear this in mind when considering the following series of quotes:

Raves are a place where people can go to be themselves, where everyone is accepted for who they are, where people will give you a smile and a hug even if you don't know them well, where you can escape reality and live together in harmony with others for a while, where you can dance your ass off all night until you see the sun rise, where you leave having more friends

than when you came in, where the music hits you hard and won't stop, where everyone is equal, where you can sit if you want to, dance if you want to, talk if you want to, hug if you want, clap or whistle or just space out or anything ... (LaGassa 1994:01).

A Rave ... superficial people all around ... just a pathetic excuse to take loads of drugs and act like you belong in pre-school ... pretentious regression (A respondent).

People that don't know what Raves are think that they are about drugs, but that is not what it is about. Undeniably there are people who do drugs here, but people do drugs everywhere ... This is about being able to do your own thing without judgement – total freedom. It is an emotional release, totally refreshing ... (A raver).

Cyberlights ... frolicking all night long ... becoming one with the music, one with the cosmos ... all-knowing unity (A raver).

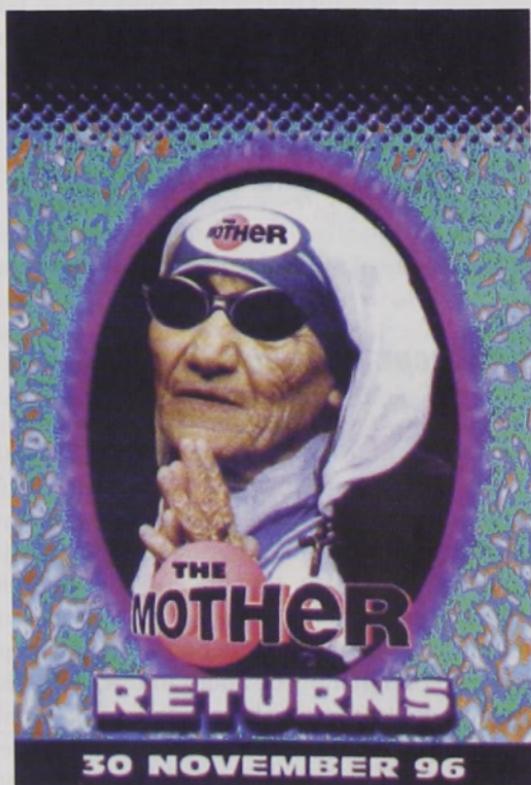


Figure 3.1 Rave flyer

At the Rave, I spent an entire twelve hours being cold, lonely and misunderstood by the drug-addled, happy-faced space kids who were content to boogie down all night long to the monotonous thumping that shook my skull but not my ass ... The only way to have fun at a Rave is to do massive amounts of drugs which make you happy and able to enjoy the constant mental assault that is techno music and spend the whole night dancing (Hoffman 1997:01).

... collective soul destruction ... drugged up ... more drugs ... the more the better ... loneliness ... reputation ruin ... (A respondent).

There are no bad vibes ... just one common goal – to have as much fun as possible. Fun is to be taken very seriously, life is too short to worry unnecessarily, so we just dance. Take a night off from life and just have fun ... (A raver).

A myriad of amazing, high individuals buzzing on the same drug ... pure bliss ... intimacy ... ecstasy ... beautiful! (A raver).

A modern-day rainbow gathering, straight, gay, transsexual, tolerance, smiles, understanding, PLUR – the new faith, selfless, universal oneness, evolution (A raver).

Rave is more than music plus drugs ... To the participant it feels like a religion; from the standpoint of a mainstream observer, it looks more like a sinister cult (Reynolds 1998:xviii).

At the end of the day it is just one big, stupid party!! (A raver).

3.2.2 *The concept of Rave*

The actual concept of Rave is nothing new. Some people claim Raves are a more sophisticated form of a primal culture (Hoy 1998:02). According to Behlendorf (Brown & Behlendorf 1995:11), 'at the base level, raves are very comparable to American Indian religious ceremonies (pow-wows) and also to the concept of Shamanism (navigating consciousness) in Eskimo and Siberian society, where music is the key towards pulling oneself into a unique, emotional and psychological state ... in which one experiences washes of sensations and visions ...'. Similarly, a large part of the concept of modern Rave parties is employing a combination of audio (music) and visual stimuli (lights and lasers) to 'elevate people into an altered state of physical or psychological existence' (Behlendorf 1995:09). (See Section 3.9.1.) Others allege that Raves are simply the re-emergence of the hippies, an 'Age of Aquarius-style utopian togetherness with a technological feel' (Beckerling in Hoy 1998:03).

For many people, a Rave is not simply an all-night party but rather a form of lifestyle, ritualised behaviour and beliefs (Reynolds 1998:xviii). Dance culture has long been home to two highly contrasting versions of what Rave is all about. On the one hand, there is the transcendentalist or New Age hippy with psychedelic talk of higher planes of consciousness and a 'oneness' with humanity and the cosmos. On the other hand, Ecstasy and Rave music fit into a happening 'rush culture' of adolescent excitement and cheap thrills: playstation video games, skateboarding, bungee jumping, other extreme sports and blockbuster movies whose story lines are simply weak frameworks for the display of spectacular special effects (Reynolds 1998:xix-xx).

3.3 *WHY RAVE?*

Although the flowing of emotions and group hugs at a Rave are a large part of the Ecstasy experience, this 'closeness' is spread into an overall atmosphere of friendliness: you connect with the group you came with but also with people you have never met (Reynolds 1998:xxv). Behlendorf (1995:10) affirms that 'what distinguishes raves is the concept of the shared experience; a feeling of unity often arises and people are open and friendly to one another. There is a loss of that "pretentious" attitude that is omnipresent in other kinds of clubs and even in life in general. People are celebrated for who they are, not what they aren't.'

In a world of post-modernism where young people are constantly vying for acceptance, belonging and love, Raves are offering what they are looking for. The need for purpose and meaning is important to most adolescents. Raves provide young people with a step towards individualism but without losing the security of society and their peer groups (Hoy 1998:03). Furthermore, Raves are perceived as a loose arena of free ideas where people come out and are allowed to express themselves: 'you are allowed to be a freak'.

There are three essential aspects common to every Rave. The following section deals with the physical aspect, namely the music, visual effects, drugs and people. The spiritual and the psychological aspects will be discussed later.

3.4 *RAVE MUSIC*

Rave music is generally characterised as all music that is dominated by a bass beat of approximately 115 to 160 beats per minute (BPM), with the most

common being about 120 to 140 BPM. Trance, goa trance, house, acid house, techno, ambient, progressive, deep house, hardcore techno, garage, speed garage and jungle are all styles of music associated with the Rave culture. 'Chill out' areas at Raves (where ravers rest from dancing) often feature a combination of the above, along with ambient or even classical music. According to Reynolds (1998:xxvii), all music sounds better on Ecstasy. It is clearer and more distinct, but also instantly overwhelming. 'You feel like you are dancing inside the music; sound becomes a fluid medium in which you are immersed' (Reynolds 1998:xxvii).

Regardless of Ecstasy's musical applications, it is the driving electronic dance music that sets the tone and builds the anticipation at Raves (Morgan 1998:04). Skilled DJs are an integral part of Raves, occupying centre stage and drawing the attention of the ravers as they spin one track into another. Generally, the purpose of the music played at Raves is to make people dance. However, Brown and Behlendorf (1995:09) believe it is more than that. The music has to 'take people to another place'. It has to 'calm the conscious mind while at the same time stimulating the subconscious, as well as the body'.

DJs must be sensitive to the 'spirit of the Rave' to know how to 'build up' and when to 'break down' the emotional tension of the crowd (Fourie 1999:31). The DJ strives to create an environment in which people in the crowd lose their inhibitions, let go and forget about the people around them. 'They hoot, holler and put their arms in the air ... kind of like in churches ... when the vibe



Figure 3.2 The 'deck'

reaches that point and people are just so into it' (DJ Sense in Morgan 1998:04). (See Figure 3.2.)

Dancing is, to an extent, yet another reiteration of the music. Stiens (1997:08–09) compares the music that the DJ spontaneously creates to a text which the dancer interprets through body movements. The beat is the driving force. Whether people are doing highly choreographed dance moves, or whether they are simply thrusting their bodies back and forth, ceases to matter. It is losing oneself to the beat and becoming one with the music by letting the music control one's movements.

3.5 VISUAL EFFECTS

A Rave is just not complete without its visual effects – the skilful use of lights, lasers and video projections. (See Figure 3.4.) Lasers focused onto moving mirrors controlled by a computer, enable an assortment of shapes, designs and logos to be drawn. The video imagery projected on the walls, linking everything from animated cartoons and 'trippy' computer graphics to film clips, is speeded up to match the beat of the music closely (Hoy 1998:05). Light fixtures, called *cyberlights*, move the lights in patterns and project them through different



Figure 3.3 'Arms in the air'



Figure 3.4 The laser effect

coloured metal filters. With the correct filters, cyberlights can create the illusion of textures (Berko 1997:03). The atmosphere at a Rave is charged not only by the music but also by the 'back to the sixties' and 'dawning of the Age of Aquarius' imagery (Hoy 1998:04). Extraterrestrial (alien), psychedelic, as well as Eastern religious iconography are a familiar sight.

The music and lights at a Rave tilt the MDMA experience towards the drug's sensory effects. With its mildly pre-hallucinogenic feel, Ecstasy makes colours, sounds, smells, tastes and tactile sensations more vivid. The experience combines clarity and brightness (Reynolds 1998:xxv). The combination of Rave music and visual effects creates a synergy that can take a raver into an altered state of awareness. Behlendorf (1995:10) asserts that the hypnotic effect of techno music, combined with the progressions of Rave DJs, can become quite intoxicating, resulting in what could closely be compared to a religious or mystical experience. (See Section 3.9.1.) The altered state may simply help one forget about who one is, but ultimately it helps one escape the world and all its problems (Hoy 1998:05).

3.6 DRUGS

To decontextualise Ecstasy use in South Africa from its predominant setting within the Rave culture is both unproductive and misleading. It is clear that Ecstasy and Rave culture go hand in hand. The subject of drugs at Raves is very controversial. Some wonder whether the Rave scene would have been more easily accepted by the public had the presence of drugs not been so high, while others wonder how Raves could ever come about without them (Behlendorf 1995:11). Anyone who has been to a Rave knows the 'thrill of catching a stranger's eye, making contact through the shared glee of knowing that you're both buzzing off the same drug-music synergy' (Reynolds 1998:xxv).

There is an indisputable connection between recreational drugs and Raves. There are many reasons for this situation. Brown and Behlendorf (1995:11) maintain that some of these reasons may include, but are not limited to, the

- presence of drugs throughout youth culture
- sensory and empathetic enhancements drugs add to the experiences of raving
- expectations of some ravers about what they are 'supposed' to be doing at Raves
- energy provided by drugs to help people stay up all night dancing
- desires of some ravers to escape or to return for a night to a carefree, childlike existence
- relatively safe, comfortable and stimulating environment provided by Raves
- inexperience and immaturity of young adults, out on their own for the first time, who want to indulge in the 'forbidden fruit', so to speak.

These factors together sometimes create an overwhelming pressure on ravers to indulge in recreational drugs. There are, however, significant risks that are often ignored by the general raving community. As discussed in Chapter 2, a small percentage of the population is prone to allergic reactions to Ecstasy (MDMA) and some ravers have died suddenly after taking low dosages of MDMA.

Although drugs have been part of the Rave scene since the beginning, it is possible to go to a Rave and not do drugs. For every raver who chooses to enhance his or her experience with drugs, there is a raver who chooses not to (Brown & Behlendorf 1995:12). Regardless, one cannot separate the Rave scene from drug use. Ecstasy became the raver drug of choice. Ecstasy broke down barriers of communication, enhanced pleasure and sensation, and made music

physically pleasurable. Strangers became people to be loved. Ecstasy broke down egos. It was a perfect fit with the happy family that the Rave scene was trying to create (Stiens 1997:10). For many young ravers, the pleasures of Ecstasy considerably surpassed any potential dangers that the drug may possess; thus amongst many Rave-goers Ecstasy became known as the 'friendly drug'.

Dance music and club life play an increasingly prominent part in the lifestyles and choices of many of today's youth. MDMA, far from being an expensive and short-lived fad, quickly established itself as a major part of certain drug-using circles in South Africa. No one is quite sure just how many people are using Ecstasy regularly in this country, though some put the number who have tried Ecstasy at 500 000 (Jonker 1996, South African Police Drug Conference). This should come as no surprise for there is little worthwhile information available regarding Ecstasy, despite a plethora of recent studies of MDMA in the USA. Estimates on usage vary widely. However, researchers now believe that a 'significant' number of young people are familiar with what has been generically termed 'dance drugs' (Redhead 1993:11).

Newcombe (in Redhead 1993:11) argues that 'Every weekend ... an estimated 20 to 30 thousand people go to house music clubs and parties, known as "Raves". Several thousand take drugs such as cannabis, Ecstasy, amphetamine and, or LSD.' Research carried out in Brighton (UK), indicated

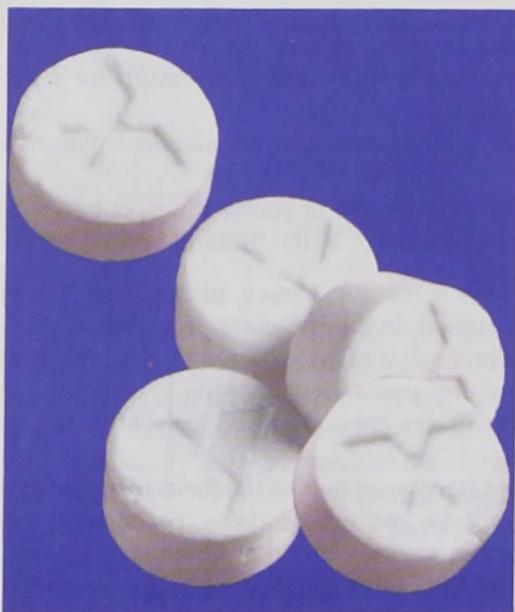


Figure 3.5 Ecstasy ('E', ecky)



Figure 3.6 Cocaine (charlie, schnarf, coke)



Figure 3.7 LSD (acid, candy)

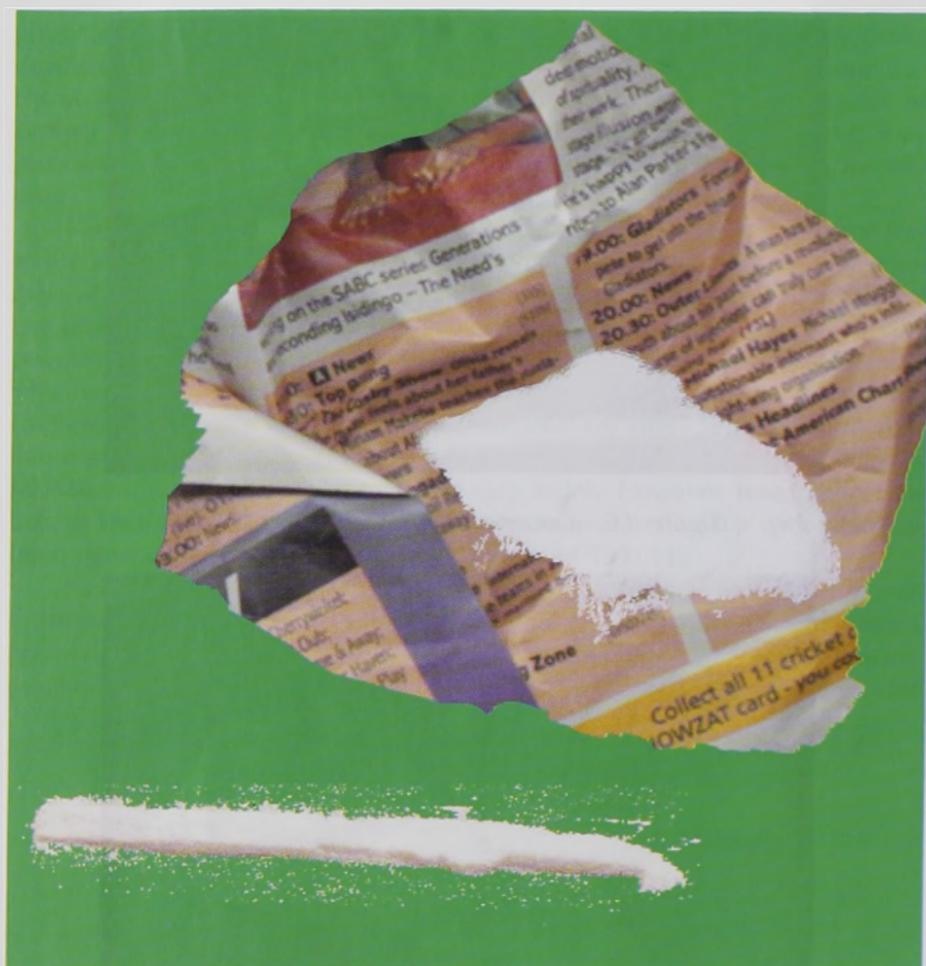


Figure 3.8 *Speed (amphetamine, whizz)*

that 62 per cent of those who regularly go to nightclubs had stated that they had used drugs recently. The Brighton study concludes that 'use of drugs is considered by many young pleasuredomers as a valid component of their leisure, along with their dress, style, choice of friends, music and clubs.' Other drug researchers argue that Ecstasy (MDMA) or to the ravers, just 'E', is the raver's cultural choice (Redhead 1993:11).

Ecstasy has also dominated contemporary pop music. References to 'E' permeate much of today's music: for example ED 209's *Acid to Ecstasy* (Redhead 1993:11–12) and Happy Monday's album *Pills'N'Thrills and Bellyaches*



Figure 3.9 Cannabis (marijuana, spliff, ganja, doob)

(Reynolds 1998:93). Ecstasy is also proving important for some young people. It is increasingly replacing more traditional drugs, such as marijuana and LSD, in becoming their introduction to illegal drug use (Redhead 1993:12). Not surprisingly therefore, many fears have been expressed about Ecstasy – fears concerning its chemical make-up and its after-effects both in the short and the long term.

Drugs are a frequent topic of debate amongst ravers. Some think that drugs should be done away with entirely, others think that only drugs that increase the vibe, such as Ecstasy, marijuana and acid (LSD), should be allowed. Still others think that the Rave scene is about personal choice and if society decides which drugs are good and which drugs are bad, it is imposing personal morals on others (Stiens 1997:10). Drugs on the Rave scene include MDMA (Ecstasy), LSD, marijuana, cocaine, speed, ketamine, alcohol, amylnitrate (poppers) and even 'natural' alternatives such as herbal Ecstasy (Cloud 9) and Midnite Flite. (See Figures 3.5 to 3.9.)

Raves created a mass recreational drug culture and fed a craving for all-night dancing. The energy released by Ecstasy felt 'radical' but it was not targeted against the social status quo (Reynolds 1998:48). According to Reynolds (1998:48), Rave was more like a withdrawal from normality, a subculture based on what Melechi (in Reynolds 1998:48) characterises as a kind of 'collective disappearance'. There existed this whole society of people who lived at night and slept during the day. Gray (in Reynolds 1998:48) refers to this idea as 'turning the ordinary world completely on its head ... almost like slipping into a parallel universe'.

3.7 THE PEOPLE WHO ATTEND RAVES

Since Rave culture adopts individualism – a 'come as you are' – mentality, practically anybody can be a raver. A typical raver can be male or female who may or may not be heterosexual. Many are university or technician students or scholars. Others have jobs. Some are professional people. Some ravers may be as young as 13 or as old as 50 (Brown & Behlendorf 1995:04) but most are between 17 and 25 years of age (Stiens 1997:09). They probably come from middle-class families and are reasonably well educated (Stiens 1997:09). Yet, none of those participants fall under one stereotype. Certainly, there are many confused teenagers and apathetic 'twenty-somethings', but there are also professional people. Ravers are some of the most diverse people one will ever meet. Unlike other drug scenes, Rave scenes do not result in participants dropping out of life (Brown 1998:10).

Raves also tend to reflect the racial diversity of the general population. Asian, black, coloured and white – it all seems not to matter. Ravers insist that 'inclusion' is central to the scene. Raves became a cross-roads where unlikely subcultures would meet (Melechi in Redhead 1993:36). Ecstasy was undoubtedly the catalyst of this coming together. A new atmosphere of sociability began to emerge, a sense of community that expressed itself in new

revivals of contact (hugging, kissing and massaging) and exchange (drinks, cigarettes, joints and poppers). As Melechi (in Redhead 1993:36) so aptly commented: 'If club culture had before celebrated an ecstasy of selflessness and oblivion, the new ecstasy was one of belonging and togetherness, of brothers and sisters in a "Promised Land".'

Many people use Raves as escapes, as 'weekend excursions' from their otherwise stressful or mundane school and home life (Brown & Behlendorf 1995:04). Infantilism is generally very predominant among ravers. Lollipops, baby's dummies, glow sticks, cuddly toys, backpacks and shirts adorned with cartoon characters are common. In a sense, this embodies the culture. It is the regaining of innocence and forgetting about problems for a while. It is a recreation of that time in their life when play was the most important thing and problems did not seem to matter (Stiens 1997:09). Raves and Ecstasy represent 'a fantasy of liberation, an escape from identity. A place where nobody is but everybody belongs' (Melechi in Redhead 1993:37).

The Rave has always been portrayed as a place where the rampant use of 'E' takes place. No one can deny drug use at Raves. The use of Ecstasy is becoming more frequent and is one of the real issues that educators need to be concerned about. Another issue would be the underlying philosophy of Raves. Perhaps the question to ask is why more and more young people are being drawn to the Rave culture and its practices.

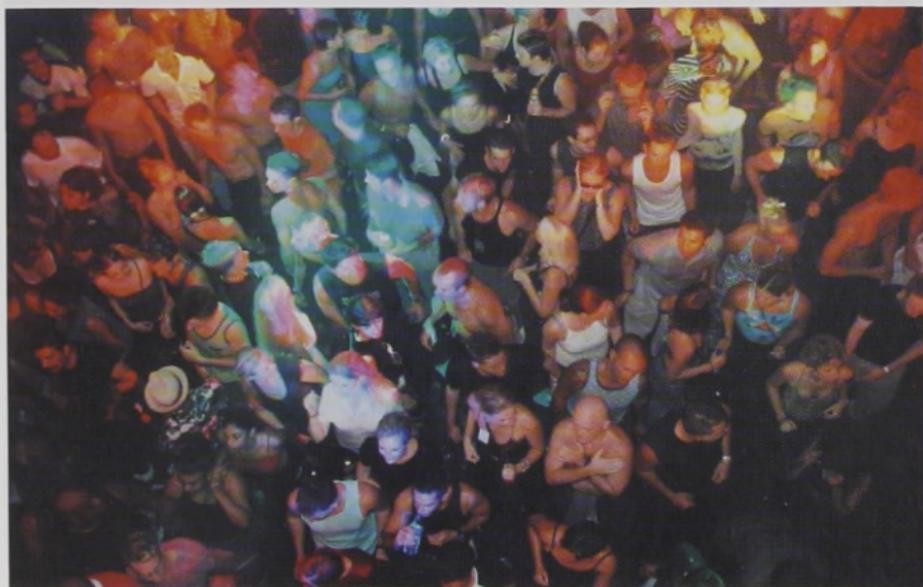


Figure 3.10 Ravers