

# MDMA

## The Dark Side of Ecstasy

GREGORY N. HAYNER, PHARM.D.\* & HOWARD MCKINNEY, PHARM.D.\*\*

No one is quite sure how many doses of 3,4-methylenedioxymethamphetamine (MDMA) have been taken in the last 10 to 15 years, but by all estimates the number is in the hundreds of thousands (Seymour 1985). With the possibility of this many people taking a pharmacologic agent, one would expect that some portion of them would experience untoward side effects ranging from mild to severe. Compounding this is the fact that substances intended for popular recreational use are most often produced in clandestine laboratories with little or no quality control, so generally speaking users cannot be sure of the purity of what they are ingesting.

As expected, the Haight Ashbury Free Medical Clinic (hereafter referred to as the Clinic) and the various emergency rooms in the San Francisco Bay Area started hearing from MDMA users just as its use was coming into full swing in the summer of 1984. This article will discuss the range of side effects experienced after ingestion of MDMA in an attempt to put this drug into perspective as having both good *and* bad characteristics for the drug using public.

Being a derivative of methamphetamine, MDMA shares a dose-related sympathomimetic response with this compound. Bearing this in mind, the list of acute adverse reactions (see Table I) is not surprising in its content and most follow logically from the effects of sympathetic

stimulation and the fact that people vary quite a bit in their sensitivities to stimulants. Complicating this, however, is the fact that doses of the drug vary when they are purchased on the street. The samples sent out for analysis over the past year and a half by the Clinic have ranged from a low of 16 mg to a high of 150 mg. Thus, it is not always an easy matter for an individual to control his/her ideal dose, even if such a level were known. It is to be expected that occasional doses will be stronger than anticipated, leading more easily to unintentional overdose.

The most common of the side effects experienced with moderate doses (85-150 mg) appear in column A of Table I. These are seen fairly consistently at a variety of dose levels. Transient nausea—with an onset of about 30 minutes after ingestion and lasting for another 30 minutes—seems to be almost universal in MDMA users. The progression of this effect to vomiting seems to be more rare, and most people find it only slightly bothersome. Increased heart rate and some degree of increased blood pressure are also experienced by all subjects taking this drug. Other than insomnia, which is sometimes seen as a consequence of central nervous system stimulation, muscle hypertonicity seems to account for the balance of symptoms in this list.

The symptoms in column B of Table I are of acute reactions, which fortunately are much rarer than the others. These symptoms usually appear in people who are particularly sensitive to MDMA or who use doses that are in the upper range of "therapeutic" (100 mg and above). It has been found that these symptoms, as well as many other side effects associated with MDMA use, are more common after repeated doses. This seems to be especially

\*Chief Pharmacist, Haight Ashbury Drug Detoxification, Rehabilitation and Aftercare Project, 529 Clayton Street, San Francisco, California 94117.

\*\*Clinical Pharmacist in Toxicology, San Francisco Bay Area Regional Poison Control Center.

**TABLE I**  
**MDMA TOXICITY: THE RANGE OF SIDE EFFECTS**

|                                             | (A)                                                                                                                                                                                                                       | (B)                                                                                                                                                                                                                                                               |
|---------------------------------------------|---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <b>Acute Reactions at Therapeutic Doses</b> | Increased heart rate<br>Tremor<br>Tight jaw muscles<br>Bruxism<br>Nausea<br>Insomnia<br>Headache<br>Sweating                                                                                                              | Numbness and tingling in extremities<br>Luminescence of objects<br>Increased acuity to cold<br>Increased color acuity<br>Vomiting<br>Objects appear to shake<br>Floor appears to move<br>Visual hallucinations<br>Ataxia<br>Crying<br>Blurred vision<br>Nystagmus |
| <b>Overdose Reactions</b>                   | Tachycardia<br>Hypertension progressing to hypotension<br>Palpitations<br>Hyperthermia<br>Hypertonicity of the body<br>Disseminated intravascular coagulation<br>Renal failure<br>Visual hallucinations<br>Rhabdomyolysis |                                                                                                                                                                                                                                                                   |
| <b>Residual Effects</b>                     | Exhaustion<br>Fatigue<br>Depression<br>Nausea<br>Flashbacks<br>Numbness                                                                                                                                                   | Feeling of coldness in body<br>Anxiety attacks<br>Persistent insomnia<br>Rage reactions<br>Psychosis                                                                                                                                                              |

true if doses are taken within a few days of each other.

While the above reactions may be troublesome to the MDMA user, an overdose of this drug may be catastrophic or even fatal. Unfortunately, not enough human research has been done to determine what is the toxic dose for most people. What is emerging, however, is a picture of an unpredictable drug in this respect. It is a drug that can potentially kill at doses that were previously tolerated in susceptible individuals. Later in this article, two cases will be presented of people who have experienced very serious side effects as a consequence of MDMA use. Suffice it to say that from the list in Table I, death by overdose might ensue via several different mechanisms. Indeed, in one of the cases presented, several of these factors played a role concurrently in the severe reaction observed.

Of the calls and visits to the Clinic that were attributable to problems with MDMA, by far the largest percentage was due to residual effects experienced by many users. In many cases, these are people who have had experience with one or more hallucinogens in the past. What they expect from past experience is that most if not all of these symptoms will persist for no more than a day after their MDMA use. In contrast, what sometimes happens is that these residual symptoms persist for anywhere from several hours to two weeks after the drug is first consumed.

As a rule, only reassurance that these symptoms will subside on their own is necessary when counseling such individuals. Nevertheless, the tenacity with which many of these symptoms persist is very disturbing to most users. This is especially true when most neophytes are reportedly

told that MDMA is fairly innocuous in its residual effects. Some callers even report having seen therapists for extended periods of time in order to deal with some of these symptoms.

One interesting theory advanced to explain this phenomenon, at least for some of these cases of protracted symptomatology, is that the very effect that trained psychotherapists so desire as a therapeutic tool may be at fault. The release of suppressed emotional material when a person is on his/her own, and not in therapy, may be detrimental. Several people in contact with the Clinic have described "reliving" past traumatic experiences under the influence of MDMA. When confronted with these experiences in a nontherapeutic setting, these people found it imperative to seek help as a result. It is readily apparent that such a situation might be very detrimental to an individual's mental health (Landry 1986). Advice from the street cognoscenti appears to disfavor using this drug alone, but rather preferably with someone who is familiar with and/or has experienced its effects.

Psychosis, as a side effect of MDMA use, usually results as a consequence of using very high doses of the drug. Such symptoms have been seen when the reported dose taken was in excess of 200 mg. Its manifestation is a classic toxic psychosis, including paranoia, ideas of reference, and both auditory and visual hallucinations. As has been found in one such case, this psychosis can be both persistent and resistant to treatment with haloperidol. In addition, it is suspected that in susceptible individuals, a much lower dose might result in similar symptoms.

The question often arises as to the appropriateness of taking self-reports of side effects from MDMA at face value. After all, there are numerous substances that might be confused with this particular drug. As pointed out by Buchanan (1985) and Shulgin and Jacob (1982), some likely candidates for this confusion include MDA, MMDA, HMDA and HMDMA, which all have varying pharmacologies. This confusion might be inadvertent, as with HMDA and HMDMA, or a clever marketing ploy by a drug dealer not wishing to be behind the times.

In truth, there is probably no good way around this problem in the absolute sense. However, in examining the results of street drug analyses sent out by the Clinic, one may conclude that people can be fairly sure that they are purchasing the genuine article, at least in the San Francisco Bay Area. Of the samples analyzed for the Clinic over the past year and a half, only two out of 10 turned out to be something other than MDMA, and these were analyzed as MDA. Although the total number of samples tested in this way was small, they came to the Clinic over an extended period of time and from various locations. Thus, despite some conflicting accounts (Renfroe 1986), it seems rel-

atively safe to assume that most, if not all, of the people coming to the Clinic for a problem related to MDMA use had in fact taken that drug. Another piece of evidence would seem to be the consistency of these complaints and the known pharmacology of the drug.

## CASE REPORTS

One particular residual toxic effect that is especially dreaded from a behavioral point of view is toxic psychosis. The authors' clinical experience indicates that great bodily harm has been suffered at the hands of people experiencing stimulant-induced delusions. This fact, coupled with their generally erratic behavior, makes such individuals potentially dangerous to deal with. One such case presented recently.

### Case 1

A 34-year-old White male presented himself at the Clinic with a reported heroin habit (\$60-\$80 per day) coupled with Dilaudid® (4 mg, 3-4 times daily). He stated that his habit was only two weeks old and that he had come for treatment because if he did not, his private therapist would not see him. On further questioning, however, he also revealed a 10-month history of taking Ativan® (0.5 mg t.i.d.) on prescription, which had been changed to Valium® (10 mg t.i.d.) four weeks prior to admission. This latter medication had been cut off, by report, three days previously when his psychiatrist found out about his opiate habit.

On physical examination, he was found to be healthy and was started on a tapering schedule of phenobarbital (Hayner & Inaba 1983) and symptomatic medication for opiate withdrawal. Three days later, he was seen by the Clinic physician again for moderate agitation and acutely increased blood pressure. At this time, he admitted to self-administration of two "syringe-fuls" and ingestion of one half teaspoonful of MDMA orally over a 48-hour period two days prior to admission. The reported effects of this dose were visual hallucination and a "wired" feeling. A diagnosis of MDMA-induced toxic psychosis was made. His phenobarbital dose was increased by 90 mg/day and haloperidol (2 mg/day) was prescribed.

Five days later, after initial symptomatic improvement, a follow-up evaluation found him to be increasingly agitated and expressing fear that he might become violent. The dose of haloperidol was increased (2 mg b.i.d.) and the patient was asked to return the following day for reevaluation.

When he arrived at the Clinic the next day, he was found to exhibit a severe reaction with paranoia and auditory hallucinations. Attempts at calming him down verbally resulted in threats and violent gestures toward the

Clinic staff. As a result, a referral was made to the county mental health facility for inpatient care.

While the phenomenon of psychosis in patients undergoing sedative-hypnotic withdrawal is not unheard of, in this case the patient was adequately medicated to prevent such an occurrence. In addition, his psychiatrist related to the Clinic staff that his patient had no history of psychosis or of violent behavior. One is left to conclude that the most likely explanation for this reaction was toxicity associated with high doses of MDMA. As previously mentioned, clinical experience at the Clinic has shown numerous protracted reactions to this drug, some of them psychological in nature. There seems to be little reason to suppose that this type of event was not evident in this case as well.

## Case 2

A.B. is a 33-year-old White female who is employed full time as an editor for a large publishing house. Her previous medical history is notable for hepatitis contracted in Brazil in 1979, with complete recovery. In addition, she experiences asthma attacks once or twice a year (or less) that respond well to episodically administered medications. She has never had surgery or general anesthetics. Previous use of mind-altering substances includes three dozen ingestions of presumed LSD and four ingestions of psilocybin in 1970-71. No lasting adverse affects are reported or evident. She is an infrequent user of ethanol-containing beverages and smoked marijuana several times a week before her MDMA experience.

In the middle of July 1985, A.B. and C.D. (a long-time friend) and five other people took alleged MDMA together and had a pleasant experience. Each of the seven people took an estimated dose of 50-100 mg. Onset of pleasant feelings and a glow to people's faces occurred about 30 minutes after ingesting the dose. For the first hour they had some grating of teeth, surroundings appeared "bright and glowing," but that subsided after an hour. Then they were open and talkative. One person had transient nausea, but no other problems were reported. All of them felt hot, but no sweating or fever was noted. Time seemed compressed ("Three hours seemed like 15 minutes."). These effects wore off in four to five hours and for the next 24 hours A.B. felt very happy. She recalled that her pupils were dilated for about 24 hours. She thought it was the best time of her life and still remembers the pleasant event in detail.

The substance ingested was one of eight packets of white powder that were purchased together and distributed by C.D. to several people. On August 16, 1985, at 1500 hours, A.D. and C.D. took another packet, splitting the contents between them (estimated to be 60-65 mg),

mixed the powder in apple juice and drank the dose. Within 10 minutes, both A.B. and C.D. experienced the onset of altered perceptual effects and emotional changes. A.B. has stated that within the next 10 minutes she experienced visual hallucinations consisting of trees turning bright iridescent colors, then had a crescendo of her teeth chattering uncontrollably, sweating, feeling of dread, the sky turned black, the landscape became gray and burned "like it had been nuked," which all rapidly spiraled into unconsciousness. She stated that "it came on like a ton of bricks" and recalls nothing of the next 48 hours, except two brief memories of waking up, once in the emergency room (ER) and once in the critical care unit (CCU).

C.D., herself experiencing an altered mental status, realized A.B. was in trouble when she lost control of her perceived state of being after falling to the ground. At this time, C.D. "forgot her trip" and phoned for an ambulance that took A.B. to the ER. Later, C.D. reported no hallucinations or any recurrence of psychic effects after her initial suppression of the experience. For a week following this event, however, she reported problems sleeping, nausea, vomiting, emotional lability and nervousness. She then entered psychiatric therapy and is still attending weekly sessions.

It should be noted that records of the ambulance run could not be located. The course of this case will be presented in problem list fashion.

## Altered Mental Status

On arrival at the ER at 1536 hours, A.B. was described by the ER staff as hallucinating, very agitated, delirious, combative (requiring four-point restraints), having spontaneous vertical nystagmus, pupils seven millimeters reactive to light, skin pale, profuse diaphoresis, tonic (continuous contraction) arm movements and a rectal temperature of 41.6°C (106.88°F). She was given intravenous diazepam (6 mg) at 1605 hours.

A.B. became stuporous and was unconscious by 1630 hours, with no response to painful stimuli, but pupillary reflexes, corneals and doll's eyes preserved. These findings are consistent with a metabolic encephalopathy above the level of the midbrain. She was intubated nasotracheally and by 1700 hours was described as thrashing about and moving all extremities, quite agitated, alert to her name and looking in the direction of the speaker, with no focal neurologic deficit. At 1800 hours, when she was admitted to the CCU from the ER, she was very irritable and thrashing about, but would squeeze hand strongly on command.

At 1930 hours she was quieter, but still agitated and staring about with wide dilated pupils. Occasional anisocoria with L>R (left larger than right) was noted. By 2000

hours she was described as only "fairly agitated." At that time, the consulting neurologist described her as "an acutely agitated young woman who appears to be hallucinating. Pupils are widely dilated and somewhat asymmetric, with the right pupil being irregular in shape and the left slightly larger; both are poorly reactive to light; her eyes dart about, and she often appears quite frightened and seems to be looking at objects that she presumably is hallucinating. At times she moves her head as though she is experiencing auditory hallucinations as well. She startles readily when touched even lightly and appears to be even more frightened when approached. She looks at me, but her eyes quickly dart away and look in other directions at random in sudden jerking motions. She is warm to the touch, but only slightly diaphoretic. She was able to follow a few simple commands. For example, she would close her eyes, protrude her tongue, raise up her head, squeeze my fingers and move her feet on request. She also moved her extremities quite frequently on her own and was fighting at four-point restraints. No meningismus could be appreciated. She was unable to vocalize due to the endotracheal tube. She appears to see visual targets and responds to those presented to the periphery and also responds to auditory stimuli. Reflexes are hyperactive symmetrically, without pathologic reflexes. Coordinated and sensory testing could not be done at this time. Fundi were both well seen and are normal. No signs of trauma are evident about the head or neck."

A total of 10 mg of haloperidol (Haldol®) was given at the neurologist's suggestion. An hour later, at 2100, she was more sedated and less frightened. She slept off and on until 0400 hours of Day Two, when she easily opened her eyes and responded to commands, such as grips, nods, smiles and waves. By 1030 hours of Day Two she was somnolent, but easily available, quieted down, fully appropriate, no longer hallucinating, frightened or agitated. Pupils were three millimeters PERRL (pupils equally round and reactive to light). Grip strength was equal but weak. She was extubated at 1400 hours. She was still sleepy at 2100 hours when onset of intention tremor and overall muscular shakiness was noted.

On the morning of Day Three, the neurologist signed off the case, noting that she was fully recovered neurologically, except for a complete lack of recall for the previous two days. By 2100 hours she was walking in the hall with assistance, still sleepy but oriented and appropriate. As she became slowly more alert, her restlessness increased and some dizziness occurred when she walked. By 1500 hours of Day Four, she reported hallucinating whenever she closed her eyes, had difficulty talking and experienced a floating sensation. She was administered orally five milligrams of haloperidol at 2230 hours and slept off and

on until she was discharged from the hospital on Day Five at 1415 hours.

A.B. remained at home and off from work for a month, feeling unable to handle stress. She described feeling as if her "energy level was way down," and had bouts of depression with crying. She indicated that she felt like "I wasn't really myself." Six months later she began to "feel like her normal self," and friends noticed dramatic improvement. During the seventh month, she resumed smoking marijuana and on four occasions it induced disturbingly realistic memories of the MDMA dysphoria, so she stopped using it. Now, nine months later, she continues to feel slow improvement in her ability to handle stress, but it still not up to her pre-MDMA levels of stress tolerance and physical stamina.

### **Hyperthermia**

At 1605 hours of Day One in the ER, a rectal temperature of 41.6°C (106.89°F) was measured for A.B. She was bathed with tepid wet towels in the air-conditioned ER. Ice packs were applied to the armpits and groin. Cooling sponge baths were begun at 1630 hours and by 1700 hours her rectal temperature was 39.2°C (102.6°F). At 1715 hours, 10 gr acetaminophen was given rectally; and by 1910, temperature had dropped to 38.8°C (102°F). At 2000 hours, the bathing and sponging was replaced with a cooling blanket and another 10 gr acetaminophen was given at 2145 hours. By 2300 hours, the rectal temperature was 37.3°C (99.1°F) and the cooling blanket was turned off. By 0100 hours of Day Two, the rectal temperature was a normal 37°C (98.6°F) and remained normal except for a rise to about 38.2°C (100.8°F) from 1600 hours of Day Two to 0800 hours of Day Three. Thereafter it remained normal.

### **Tachycardia**

Throughout the first five hours in the hospital, A.B. had a sinus tachycardia with a rate of 130 to 150. Without specific therapy it dropped to a range of 95-120 until 0800 of Day Three when it rose to 130, which persisted until about 2000 hours of Day Three. Sinus tachycardia at a rate around 100 to 110 slowly resolved to normal over Days Four and Five.

### **Cardiac Pump Function, Hypotension, Respiratory Distress, Pulmonary Edema and Oliguria**

When A.B. was first examined in the ER, she had a blood pressure (BP) of 70/50, Heart Rate (HR) 150 and Respiratory Rate (RR) 36. An intravenous (i.v.) line was started in her right hand and one liter lactated Ringers (LR) was hung. Five hundred (500) milliliters ran in and then the i.v. unit plugged up. A second i.v. unit was

inserted into the left hand and a second liter of LR was hung. Blood was drawn for arterial blood gasses (ABG), laboratory and toxicology tests. Four ampules of naloxone and one ampule of D<sub>50</sub> (50% dextrose) were given i.v., but without response. She was given two milligrams of diazepam, three times by 1605 hours. She became stuporous for over 10 minutes and then became unconscious. BP dropped precipitously to 70 systolic. A nasotracheal tube was inserted at 1605 hours to provide oxygen. ABG was 7.37/37.5/43.7/18.6. A femoral vein catheter was inserted for rapid infusion of fluids. By 1630, one liter LR had been given via the femoral vein catheter, BP rose to 110/70 and A.B. began wheezing bilaterally despite increasing inspired oxygen concentration via T piece. Because of her history of asthma, she was given 120 mg methylprednisolone sodium succinate (Solu-Medrol®). Increasing respiratory distress and an ABG of 7.37/34/70/19.6 necessitated complete nebulizer therapy with isoetharine (Bronkosol®) at 1700 hours, producing an ABG of 7.28/44.6/305.6/20.9. Meanwhile, a No. 36 French Ewald tube was placed orogastrically and A.B. was lavaged with three liters normal saline, then 30 gm of activated charcoal and one bottle of magnesium citrate were given. A Foley catheter was inserted into the urinary bladder and about 60 cc concentrated urine was produced. By 1730 hours, about 2500 ml LR had been given, BP was 110/60, HR 130, RR 22, and temperature 39.2°C (102.6°F).

Some jugular venous distention (JVD) was noted while A.B. was lying flat, collapsed on inspiration or sitting. She was then admitted to CCU at 1800 hours. By 2000 hours she had JVD from clavicles to jaw line, lungs full, bronchial wheezes, rales in bases, urine dark, gritty and scant, BP 100/60-70, HR 128, RR 24. At 2230 furosemide given (40 mg i.v.), with instant production of profuse diuresis that continued until approximately 0400 of Day Two. By then there was no JVD or wheezing and respiratory distress slowly improved with aminophylline. She was extubated at 1400 hours of Day Two, when CXR (chest x-ray) showed some mild pulmonary vascular congestion. BP was 108/78, HR 98 and RR 16.

#### Analysis of Suspected Substance

The white powder that was brought in by the patient's friend was analyzed. Thin-layer chromatography showed that the unknown powder co-migrated with authentic MDMA (courtesy of A.T. Shulgin) and was 95 percent pure. Gas chromatography on a 10% apiezon-2% KOH column identified the powder as chromatographically pure MDMA. Ultraviolet spectra (200-400 nm) in 0.1 N HCl gave absorbance maxima of 208, 234 and 285 nm, corresponding to those of authentic MDMA. Infrared

TABLE II  
CONCENTRATION OF MDMA IN BODY FLUIDS

| Hours After Ingestion | Specimen | MDMA (mg/l) |
|-----------------------|----------|-------------|
| 2                     | Urine    | 410.0       |
| 5                     | Urine    | 816.0       |
| 120                   | Urine    | 1.0         |
| 5                     | Gastric  | 1070.0      |
| 2                     | Serum    | 6.5         |
| 5                     | Serum    | 7.0         |

spectroscopy for the unknown powder and MDMA were exact matches.

#### Analysis of Body Fluids

Serum, urine and gastric samples were preliminarily analyzed, then appropriately diluted and two milliliters of each was extracted with five milliliters of 1-chloral-butane at pH 10.5 (NaCO<sub>3</sub>). Each sample contained diphenhydramine (0.5 mg/l) as an internal standard. Evaporated residues were injected on a gas chromatographic equipped with a nitrogen-phosphorus detector and a glass column packed with Supelco 2250 DB. Operating temperatures were 250°C, 185°C and 300°C for the injector, oven and detector, respectively. Standard curves were linear for concentrations between 0.1 and 0.2 mg/l. Concentrations are given in Table II. Unextracted and extracted urine as well as gastric samples gave ultraviolet absorption maxima corresponding to those of MDMA. Only a small amount of MDMA was present in the urine (less than two percent of the total MDMA). The urine was also positive for amphetamines by immunoassay (EMIT-dau®) to those of MDMA—about 10 mg/l of MDMA is equivalent to the low calibrator cutoff of 0.3 mg/l of amphetamine.

#### CONCLUSION

Hopefully, further research on the pharmacokinetics of MDMA will allow interpretation of these values. However, this is the first recorded case of severe toxicity resulting from MDMA ingestion alone. There were no other drugs present on examination of the samples by immunoassay.

This case is disturbing in that a near fatal reaction followed the ingestion of a presumed average dose of MDMA, similar to the doses consumed by many other people who do not experience significant physical or psychic distress. This patient has no family history of malignant hyperthermia, nor is there any history of neuroleptic drugs predisposing to neuroleptic malignant syndrome.

In regard to this case, it simply is not known what triggered this devastating response to MDMA in this healthy young woman. Like any other drug, MDMA should be recognized as a substance with both beneficial effects and undesirable side effects. Before this drug is used clinically, it should be subjected to the same rigorous characterizations of pharmacology, pharmacokinetics and toxicology as all other drugs that are approved for human use. While many people will no doubt continue to

self-experiment with MDMA and other drugs, the occasionally intense enthusiasm for such experimentation should be tempered by the realization that there is risk involved that must be considered along with any proposed benefit.

#### ACKNOWLEDGMENTS

The authors are grateful to Drs. Shulgin, Jacob and Osterloh for their analysis of the samples.

#### REFERENCES

- Buchanan, J. 1985. Ecstasy in the emergency department. *Clinical Toxicology Update of San Francisco Regional Poison Control Center* Vol. 7(4).
- Hayner, G.N. & Inaba, D.S. 1983. A pharmacological approach to outpatient benzodiazepine detoxification. *Journal of Psychoactive Drugs* Vol. 15(1-2): 99-104.
- Landry, M. 1986. Personal communication.
- Renfro, C.L. 1986. MDMA on the street: Analysis Anonymous®. *Journal of Psychoactive Drugs* Vol. 18(4).
- Seymour, R.B. 1985. MDMA: Another view of Ecstasy. *PharmChem Newsletter* Vol. 14: 1-2, 8-9.
- Shulgin, A.T. & Jacob, P., III. 1982. Potential misrepresentation of 3,4-methylenedioxyamphetamine (MDA). *Journal of Analytical Toxicology* Vol. 6: 71-75.