Raves and Club Drugs

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Cover Illustration by Tish Berchtold Klus

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Raves and Club Drugs

Shannon C. Miller, MD, CMRO

INTRODUCTION

Youth attendance at rave events and clubs continues to be highly popular. The prevalence of illegal drugs at these events is well known. Up to 70% of attendees may be using illegal drugs. According to one survey, more than 80% of attendees at a “circuit” party had taken illegal drugs that day, with an average of 3 drugs per person. The term club drugs results from the strong association of specific illegal drugs and many dance clubs. Most of these drugs are Schedule I substances under the Controlled Substances Act. Most are fraught with highly dangerous medical complications—including death—in addition to addiction. Dehydration due to the combination of psychomotor stimulants, relentless dancing, and poor fluid intake has been implicated in a variety of club- and rave-related deaths. Moreover, the variety and number of over-the-counter and legal drugs present at raves and clubs are significant. Attempts by intoxicated, exhausted, sleep-deprived attendees to operate motor vehicles home during the early morning rush hour can prove deadly. Awareness of these drugs and related issues is of particular importance to the practice of specific medical specialties, including pediatrics, psychiatry, family practice, emergency medicine, addiction medicine, and addiction psychiatry, as well as general practice.

THE CLUB SCENE AND THE ANATOMY OF A RAVE

A “club” may be defined as any dance establishment, whereas a “rave” refers to the actual activity therein. Raves, however, may occur anywhere. Raves are organized social events that usually include music and dancing and are often organized around a specific theme (eg, environmental concerns, spiritual concerns) for a specific audience—usually teens and young adults. The culture behind this movement has its roots in rebellious youth subculture. From the Beatnicks and the Greasers of the 1950s, the Hippies of the 1960s, and the Punkers of the 1980s, youth has long had an interest in establishing a set of behaviors different from those of society. Most often this is reflected in the adoption of unique, intense behaviors (eg, “free love,” body piercing) and is expressed in unique styles of clothing and music. At times, this has also included illegal drug use.

Club music has its roots in the Punk Rock sounds of the 1980s, characterized by thrashing electronic sounds and stage antics. This evolved into New Wave music (a cleaner, more technologically sophisticated sound). As home music synthesizing equipment became less expensive, emphasis shifted from a band consisting of musicians to a new music creator, the DJ. DJs typically produced music without lyrics. But without lyrics, a lead singer, nor any musicians or stage antics, the music was not marketable and thus was found primarily “underground”—thus, the appeal to the youth counterculture.

This “techno” sound became more focused within circuit parties—dance parties for gay men. The popular use of cocaine at these events in the 1980s was replaced by MDMA, or ecstasy, which provided a more desired blend of energy (important for extended dancing and staying out late) together with an empathic or “loving” feeling. The development of the Roland TB-303 synthesizer allowed DJs to create music inexpensively. In addition, this synthesizer had a unique ability to offer long music sequences featuring drum machines with a fast tempo (140–200 beats per minute)—highly desired by those using stimulants such as MDMA.

In 1988, Great Britain experienced the “Second Summer of Love” (as termed by British musical journalists). MDMA had just arrived from the United States. Because MDMA was sold as tablets with logos imprinted by the drug’s makers, they resembled pharmaceutical grade prescription medications, thus appearing “safe” (Figure 1). MDMA was devoid of the stigmata of illegal drugs of abuse, such as being used intravenously, carrying the risk of HIV infection, or lacking standardized packaging or a standardized form. MDMA’s pleasant psychotropic effects caused it to be viewed as a “nice” drug, and the “happy face” symbol became the icon of

Note: The views expressed in this article are those of the authors and do not reflect the official policy or position of the United States Air Force, Department of Defense, or the US Government.
Raves and MDMA. This 1990s version of the 1960s youth subculture blended various idealized themes—peace, love, unity, respect, and harmony—giving rise to the term, “feeling PLURHY.”

Although the MDMA “high” may last up to 8 hours, parties held in clubs (organized business establishments) in England would be broken up when the clubs were forced to close within locally allotted business hours. Party promoters thus migrated their activities from clubs to locations with little oversight or restrictions, such as deserted warehouses, empty fields, and unused subway stations. These events were now popularly termed raves. Raves jumped back across the Atlantic Ocean to the United States and occur today in many parts of the world.

Elements common to most raves include a promoter (who funds, advertises, and profits from the event), specific clothing, and rave paraphernalia (including certain drugs). Raves vary in size, and the venue is usually secret. They generally take place between the hours of 4:00 AM and daybreak. Organized security may be present. (At some raves, stigmatized drugs such as cocaine and heroin are specifically confiscated.) Many raves are advertised in a clandestine fashion by word of mouth only, some via more public means (particularly the Internet). Many see the logistical challenge of locating a rave as a fundamental aspect of the rave itself. Rave advertisements are shown in Figure 1.

Clothing at raves is highly functional. Headbands and hats manage perspiration. Sneakers and baggy pants facilitate dancing and also help to manage perspiration. Backpacks contain rave paraphernalia. An androgynous theme is common in dress, as is one of infancy, including wearing pony tails and hair barrettes and carrying infant toys (Figure 2). Glow sticks and glitter provide visual stimulation to enhance the dissociative effects of club drugs. Face masks may be worn to reduce dust exposure at raves held in condemned places; menthol ointment is often smeared inside to enhance the psychological effects of drugs. Pacifiers reinforce the infancy theme and are utilized to mitigate bruxism caused by MDMA intoxication. Lollipops may also be used for this purpose and may be adulterated with lysergic acid diethylamide (LSD).

Beverages available at raves include “smart drinks,” which contain ginseng or tryptophan to purportedly counterbalance cognitive slowing from illegal drug effects. γ-Hydroxybutyrate (GHB) is commonly smuggled into raves as a solute in bottled water. “Go-go” drinks containing various substances thought to have aphrodisiac properties (eg, ginseng, yohimbine, sildenafil, guarana) are not uncommon. “Power drinks” may be sold to facilitate marathon dancing. These drinks provide carbohydrates as well as caffeine, amino acids, and ephedrine (either synthetic or derived from natural sources)—potentially resulting in dangerous sympathomimetic synergism. “Herbal ecstasy” (various preparations of Ephedra sp.) is erroneously seen by users as a legal, safe alternative to MDMA. The use of such stimulants may be fraught with deadly side effects when combined with excessive exercise, dehydration, and MDMA or other sympathomimetics. In an effort to minimize such dangerous effects, “chill-out” rooms are offered at better-organized raves; often air-conditioned, these rooms contain showers and bottled water and may offer relaxation techniques.

Most ravers are between the ages of 15 and 25 years, white, and middle class. In a study that took place in New York City, 70% of rave attendees were white, 20% were Hispanic, and 10% were black. These demographics
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General Characteristics

Those who attend raves or club events and use psychotropics generally desire certain drug qualities. Stimulants are desired to enhance the ability to dance for prolonged periods (several hours at a time) and to very fast-paced music, as well as for mood enhancement. Dissociatives and illusionogens are desired to enhance the effects of colored laser-lights and hypnotic music. Sedatives are used to “smooth-out” overstimulation. Sedatives also can loosen inhibitions and contribute to the “PLURHY” scene. Rave culture frowns upon stigmatized drugs such as cocaine and heroin, as well as alcohol, because these drugs are felt to contribute to the perception of drug use for narcissistic needs, rather than for group cohesion and enlightenment. The prototypical rave drug is MDMA, as it has qualities of psychomotor stimulation, euphoria, perceptual distortion, and empathic connectedness. The most common illegal drug found at raves in one study, however, was cannabis.

The Drug Abuse Warning Network (DAWN), a national voluntary drug abuse surveillance system that monitors emergency room visits and medical examiner data showing deaths attributable to drug use within 21 metro areas, suggests that the use of illegal drugs associated with clubs and raves has seen a statistically significant increase since 1994 (Figure 3). Each emergency room visit by a patient in which specific drugs are related to the cause of the visit is counted as an emergency room “mention.” Per DAWN data, most emergency room mentions involving club drugs also involve a second or third club drug. The most common commented drug is alcohol. Limitations of the DAWN data include (1) under-reporting of those drugs less likely to cause death or emergency room visits, (2) possible favoring of the reporting of “less-experienced” users who end up sick or dead, (3) sampling locations that are primarily outside of rave events, and (4) the data do not necessarily provide a record of drug use prevalence, but rather bad outcomes.

MDMA

MDMA (3,4-methylenedioxymethamphetamine) was developed and patented by Merck Pharmaceuticals in the early 1900s as an experimental drug with possible use as an appetite suppressant. It was never tested in humans. It was used uncommonly in the 1970s by both psychotherapists and clergy as a means to better empathize with those whom they treated or counseled. The United States Food and Drug Administration (FDA) recently approved a randomized, double blind, placebo-controlled protocol to study the safety and efficacy of MDMA-assisted psychotherapy in stimulating therapeutic processing of traumatic experiences in posttraumatic stress disorder.

MDMA is a Schedule I drug. Most of the world’s MDMA is manufactured illicitly in Amsterdam; however, production in the United States has increased. MDMA production and trafficking is a highly lucrative business. Street names include ecstasy, E, adam, clarity, empathy, lover’s speed, X, and XTC. A single tablet costs 20 to 25 cents to manufacture and sells for $20 to $40. Typically, 1 or 2 tablets are taken per dose, each tablet containing 60 to 120 mg of MDMA. MDMA is popular in part because it is readily available, cheap, and is not stigmatized. In a UK survey of 16- to 29-year-olds, the prevalence of MDMA use was 9% overall, whereas in those who attended raves, it was 91%. More recently, there has been a trend towards injecting or snorting MDMA.

In general, the effects of MDMA are similar to those of both stimulants and hallucinogens. However, MDMA is preferred by those who attend raves owing to its facilitation of empathic connectedness as well as for the

psychomotor stimulation it provides, allowing users to dance for prolonged periods at rave events. MDMA intoxication initially involves a “rush” (30–45 seconds), a sense of sudden clarity of mind, intensification of perceptions (objects appear brighter and crisper), and an inner sensation of happiness. People seem lovable exactly as they are. As the effect wears off, users commonly administer a booster dose. A “plateau” of effect will be maintained for a half an hour up to 3 hours, making the repetitive, trance-like music played in clubs or at raves seem very pleasurable. The effect gradually wears off over the next 3 to 6 hours, leaving the user in a state of depressed emotions and sluggishness.12

Adverse effects of MDMA include anxiety, bruxism, malignant hypertension, tachycardia, dehydration, exhaustion, hyperthermia (107°–109°F), rhabdomyolysis, stroke, seizure, paranoia, heart/kidney failure, dependence, and death. Although MDMA alone is well known to disable the body’s temperature homeostasis, two related substances, PMA (paramethoxyamphetamine) and PMMA (paramethoxynmethamphetamine), are believed to be responsible for the extreme body temperature dysregulation that has been seen in some recent cases of MDMA-associated hyperthermic death.13

A key recent concern is the striking finding of serotonergic neuronal injury associated with MDMA use. This is dose-dependent, but may occur with as few as 4 doses per month (1 per weekend).14 Neuropsychological testing of abstinent MDMA users has shown deficits in attention, memory, learning, and IQ.14 Some of these deficits have continued in primate models despite 7 years of abstinence.14 The mechanism involves a number of serotonergic effects of MDMA on specific brain circuits: serotonergic vesicular release, serotonin and norepinephrine reuptake blockade, 5-HT2A receptor stimulation, dopaminergic release, and serotonin reuptake pump depletion. The closer the animal is to humans phylogenetically, the more permanent the damage is.15,16 Chronic use may cause neuronal serotonin depletion and may account for the “Tuesday blues”—the hangover or crash typical the day after intoxication. This involves feeling drained and depressed, and may last for up to 5 days, often resulting in attempts to re-dose. Selective serotonin reuptake inhibitors have been shown to attenuate the pleasurable effects of MDMA, thus perhaps reducing relapse risk in users.16,17 MDMA’s effects on the serotonergic system may affect cerebral blood flow, which has been shown to be reduced in human MDMA users (Figure 4, page 8); this effect, however, may be transient.18 Studies on neonatal rats suggest that MDMA use during the third trimester of pregnancy may cause memory and learning problems in newborns, persisting into adulthood.19

Very recent research suggests dopaminergic injury from MDMA use may be even more severe than serotonergic injury (Figures 5 and 6).20 Individuals who use MDMA may be at risk for substantial neurotoxicity if they use 2 or 3 sequential doses, hours apart, on a single occasion, as is often the case in recreational settings. This injury, together with the decline in dopaminergic function known to occur with age, may put these individuals at increased risk for developing Parkinsonism and other neuropsychiatric diseases involving brain dopamine/serotonin deficiency, either as young adults or later in life.20

Drug users have been known to rely on a pill’s logo (used by a drug supplier or dealer to identify their product) as reassurance that a pill known to be “safe” in the past (ie, with minimal side effects and adulterants) will be safe to purchase in the future. A 2-year European study of pills sold as MDMA with 69 different logos found that 30 contained MDMA only (doses ranging widely, from 2 mg to 149 mg), 8 contained adulterants (aspirin, caffeine,ephedra, amphetamines), and 7 contained no MDMA.11 MDMA concentration may vary as much as 70 times.21 Thus, many harm reduction organizations offer home MDMA testing kits, as well as mail-in pill testing services. In addition, in Holland, free, anonymous on-site MDMA testing has been offered as a harm reduction strategy.

A recent addition to the club drug scene is BDMPEA (2-[4-bromo-2,5-dimethoxy-phenyl]-ethylamine). Street names include nexus, venus, bromo, 2 CB, toonies, and MFT. Similar to MDMA pharmacodynamically, BDMPEA is 10 times more potent, thus carrying a high risk of overdose, particularly in users purchasing it as MDMA.

Routine urine drug testing panels typically include methamphetamine and amphetamine; MDMA may or may not be included and thus may need to be specifically requested. Testing for BDMPEA requires a specific request.

GHB

γ-Hydroxybutyrate (GHB) was introduced into the dietary supplement market in the spring of 1990. Initially, it was considered a safe dietary supplement for use as a body building aid, antidepressant, sleep aid, and sexual enhancer. It was quickly found responsible for a significant number of adverse events (including deaths), and was banned from over-the-counter sale and listed as a Schedule I substance by the FDA later that year. Medical emergencies related to GHB increased sharply during the latter part of the 1990s, with a notable demographic shift toward users younger than 20 years.22
Figure 5. MDMA and serotonergic neurotoxicity. Neurotoxic effects on squirrel monkeys (Saimiri sciureus) of an MDMA regimen modeled closely after one often used by MDMA users at all-night dance parties. (A–C) Graphs illustrating lasting reductions (2 weeks after exposure) in regional brain serotonin (5-HT), 5-hydroxyindoleacetic acid (5-HIAA, serotonin’s major metabolite), and the serotonin transporter (SERT), respectively. Results shown represent the mean ± SEM (n = 3 animals per group). Fc = frontal cortex, Pc = parietal cortex, Tc = temporal cortex, Oc = occipital cortex, Hc = hippocampus, Cd = caudate nucleus, Put = putamen, DPM = disintegrations/min. Asterisk designates P < 0.05, determined by individual comparison to control after 1-way analysis of variance showed an F value with P < 0.05. Anatomic studies support these observations, showing reductions in the density of serotonin- and SERT-immunoreactive (SERT-IR) axons in some cortical regions. Items D and E illustrate 5-HT (D) and SERT-IR (E) axons in the parietal cortex of a control monkey (left) and a monkey treated with MDMA 2 weeks previously (right). Dark-field photomicrographs of the coronal plane are shown; scale bar = 100 µm. (F) (see page 8). (Reprinted with permission from Ricaurte GA, Yuan J, Hatziidimitriou G, et al. Severe dopaminergic neurotoxicity in primates after a common recreational dose regimen of MDMA [‘‘ecstasy’’]. Science 2002;297:2260–3. © 2002 American Association for the Advancement of Science.) CLICK HERE for terms of use and citation of this material.
GHB is used to produce sensations of euphoria, relaxation, and sexual enhancement. Street names include Georgia home boy, grievous bodily harm, growth hormone booster, liquid ecstasy, nature’s Quaalude, salty water, somatomax, and easy lay. GHB is most often sold dissolved in water as an “energy drink” at clubs and raves. Street purity of GHB can vary by as much as a factor of 100. GHB is inexpensive and simple to manufacture.

γ-Butyrolactone (GBL), a club drug in its own right, is often sold and ingested because it is metabolized to GHB. Chemically different from GHB, it is available as a dietary supplement.23

Because of unpredictable “drop attacks” (ie, sudden, unexplained loss of consciousness) and an LD₅₀ of only 5 times the intoxicating dose, adverse events with the use of GHB are common. They are more common in daily users and in multiple drug users. In a published survey of 42 GHB users, 66% reported experiencing loss of consciousness, 28% reported accidental overdose, and 45% reported amnesia after GHB use.24 Tolerance and withdrawal symptoms, including delirium, are often reported.24,25 GHB is usually included in drug tests by request only.

GHB is both a precursor and a metabolite of GABA (γ-aminobutyric acid), but has no strong GABA-receptor affinity. It is an endogenous short chain fatty acid with inhibitory properties. GHB has a dual effect, both as a central nervous system (CNS) depressant and as an anabolic agent. GHB stimulates pituitary growth hormone release at levels 7 times above normal physiologic levels.24 Onset of action is within 15 minutes; half-life is short (27 minutes). A 2-g dose induces deep sleep; 4 g induces coma. GHB neuromodulates an increase in opioidergic activity. In addition to activity in cholinergic, serotonergic, and GABA neurons,24 GHB has a biphasic effect in dopaminergic neurons, initially reducing dopamine levels, then increasing them.26

GHB has been investigated for its therapeutic potential. It has been studied for use in narcolepsy27 and has been approved as Xyrem (sodium oxybate) for the treatment of cataplexy associated with narcolepsy in the FDA’s orphan drug program. Although illicitly made GHB is a Schedule I drug, Xyrem is a Schedule III drug, subject to distribution protocols for restricted access. GHB has also been studied for use in treating other substance disorders.28–30 GHB has been shown to reduce alcohol consumption and suppress withdrawal.31,32

Endogenous GHB may have a role in the disease of addiction. Both GHB and the spasmylic drug baclofen are GABA(β) receptor agonists but have no direct effect on GABA(α) receptors (despite GHB’s known sedative-hypnotic effects). Baclofen lacks significant addiction
Figure 4. Reduced regional cerebral blood flow (rCBF) in human MDMA users. (Reprinted with permission from Chang L, Grob CS, Ernst T, et al. Effect of ecstasy [3,4-methylenedioxymethamphetamine (MDMA)] on cerebral blood flow: a co-registered SPECT and MRI study. Psychiatry Res 2000;98(1):15–28.)

Figure 5. (F) Radioisotopically labeled SERT in the coronal section of a control monkey and a monkey treated with MDMA 2 weeks previously. The scale on the right shows the density of binding sites designated by color expressed in nanocuries (nCi) per mg of tissue. (Reprinted with permission from Ricaurte GA, Yuan J, Hatzidimitriou G, et al. Severe dopaminergic neurotoxicity in primates after a common recreational dose regimen of MDMA ["ecstasy"]. Science 2002;297:2260–3. © 2002 American Association for the Advancement of Science.) CLICK HERE for terms of use and citation of this material.

Figure 6. (E) Radioisotopically labeled dopamine transporter (DAT) in the coronal section of a control monkey and a monkey treated with MDMA 2 weeks previously. The scale on the right shows the density of binding sites designated by color expressed in nCi/mg of tissue. Morphologic studies (not shown) revealed corresponding reductions in the density of striatal DAT- and tyrosine hydroxylase (TH)-immunoreactive (IR) axons throughout the striatal complex. (Reprinted with permission from Ricaurte GA, Yuan J, Hatzidimitriou G, et al. Severe dopaminergic neurotoxicity in primates after a common recreational dose regimen of MDMA ["ecstasy"]. Science 2002;297:2260–3. © 2002 American Association for the Advancement of Science.) CLICK HERE for terms of use and citation of this material.
potential (unlike GHB). Thus, GHB-specific receptors may have properties unique to addiction. GHB-specific receptors have been identified in the hippocampus, cerebral cortex, various dopaminergic structures, and hypothalamus.24

**Ketamine, PCP**

Ketamine and phencyclidine (PCP), both arylcyclohexylamines, are dissociative anesthetics. PCP (a Schedule II drug) is a non-competitive NMDA (N-methyl-D-aspartate) receptor antagonist. Thus, glutamate-mediated CNS functioning is blocked, resulting in dissociation and hallucinations. Other signs of PCP intoxication include increased vital signs, agitation, nystagmus (vertical or horizontal), and delirium. PCP is inexpensive to manufacture; street names include angel dust, super grass, PeaCe pill, and rocket fuel. PCP was developed in the 1950s and marketed in the 1960s as the anesthetic Sernyl. Because of difficult to manage intra- and postoperative hallucinations and agitation, it was restricted to veterinary use only; it was removed from the market entirely in the 1970s because of diversion as a drug of abuse.

Similar to PCP, ketamine (a Schedule III drug) also is an NMDA receptor antagonist that has been used for veterinary and pediatric surgical anesthesia since the 1970s. However, ketamine’s minimal cardiac, respiratory, and agitation complications make it safer for surgical use. Ketamine’s popularity as a drug of abuse stems from its dissociative, dreamy, catatonic states that it produces. Street names include cat valium, K, special K, vitamin K, and ket nip. Ketamine is usually taken orally, but can also be injected.

Symptoms of ketamine intoxication can mimic non-substance induced psychotic disorders, such as schizoaffective disorder, a specific drug testing and a careful history-taking to exclude substance-induced psychotic disorders usually results in accurate diagnosis. Low doses of ketamine have been associated with cognitive dysfunction; higher doses have been associated with hypertension, delirium, poor motor control, depression, and respiratory decompensation. The LD₅₀ of ketamine is 30 times its anesthetic dose.35

Both ketamine and PCP are often soaked or sprayed onto the leaves of smoked substances (e.g., marijuana, tobacco, oregano, parsley, mint). They are highly lipophilic and easily absorbed. PCP is routinely included in most routine drug screens. PCP can be detected for 1 to 2 weeks after use, and sometimes longer in more heavy users. Ketamine is usually included in drug tests by request only.

**Dextromethorphan**

Dextromethorphan is a semi-synthetic morphine derivative antitussive agent sold in more than 140 non-prescription products.34 Some of the most common products abused are Coricidin HBP Cough and Cold (“C-C-C”) tablets, which contain the largest amount of dextromethorphan per dosage unit on the market, and Nyquil. Street names include dex, DXM, C-C-C, and Robo.

Dextromethorphan binds to sigma opioid receptors (related to physiologic tolerance and dysphoria in opioid users), and its metabolite, dextrorphan, is an NMDA receptor antagonist. If enough dextromethorphan is consumed (usually doses of 4 ounces or more of a dextromethorphan-containing cough syrup) or if the user is a fast metabolizer of dextromethorphan, accumulated dextrorphan can produce PCP-like effects caused by the NMDA antagonism. It affects serotonergic systems, perhaps causing 5-HT₁A agonist effects. A number of addiction effects have been demonstrated.

Cases of dextromethorphan abuse and dependence are frequently seen clinically, but are not often reported in the literature.35 The over-the-counter nature of these products facilitates adolescent use. The “high” is described as an “out of body” experience. Dissociative and perceptual distortions can occur. At higher doses vital sign elevation and delirium may result, including choreothetoid movements and seizures. Vomiting may result in aspiration. Drug testing requires a specific request to test for dextromethorphan. If dextromethorphan abuse is suspected, acetaminophen levels should also be ordered, because many of these preparations contain this added ingredient.

**LSD**

LSD is an indolealkylamine that was first synthesized in 1938. Although it is usually categorized as an hallucinogen, in strict terms, it is better described as an illusionogen. Its illusion-producing effects are possibly due to its 5-HT₂₅₆ agonism. A Schedule I drug, it is one of the most potent substances of abuse, measured in micrograms. However, unusually rapid tolerance develops to its effects, thus perhaps protecting against rapid readministration (as no “high” would be achieved) and resultant classic addiction behaviors. In addition to illusions, LSD can cause a sense of detachment from reality as well as a sense of enmeshment with surroundings. Synesthesias (in which one sensory perception takes on the qualities of another, such as sounds experienced as colors) are fairly unique to this drug class. These sensory perturbations may result in disturbing, distorted, or
false environmental interpretations, resulting in panic episodes or other psychiatric sequelae.

LSD intoxication begins approximately 1 hour after ingestion, peaks at 2 to 4 hours, and resolves after approximately 12 hours. In the 1940s and 1950s, it was accepted as a pharmacological vehicle for clinicians (who self-administered it) to better understand schizophrenia. Prior to the Controlled Substances Act of 1970, it was prescribed for a wide variety of psychiatric disorders. A Schedule I drug, LSD has no medical use, has a high abuse potential, and has a lack of safety when used under medical supervision.

LSD is water soluble, clear, and odorless, and can be packaged as crystals, pills (similar to colored candy dots sold on a paper backing), or gelatin (sold in sets of molds called “window panes”); dissolved into sugar cubes, and impregnated into colored designs printed on paper that can be dissolved (“blotter paper”—the most commonly sold form). Common street names include acid, microdots, mind detergent, sugar, windowpanes, and names based on the paper design being sold (eg, unicorns, Bart Simpsons). Drug testing requires specific request.

Flunitrazepam

Flunitrazepam is a highly potent, short-acting benzodiazepine legally available only outside of the United States, where it is marketed as Rohypnol. It is often smuggled across the border from Mexico (where it can be prescribed). Flunitrazepam is used as a primary intoxicant at raves or to counterbalance the psychomotor agitation of stimulants. Street names include Mexican Valium, roches, forget me pill, date rape pill, but may also be crushed and snorted (more common in England).37 dissolved under the tongue, dissolved in liquid and injected, or mixed into marijuana and smoked. Swallowing the pill with coffee, soda, or fruit juice reportedly enhances its effect. Side effects may include hypotension, dizziness, visual problems, confusion, gastrointestinal disturbances, urinary retention, paradoxical excitement/aggression, and addiction.

Flunitrazepam has qualities that make it a sought-after “date rape” drug. However, studies suggest that the use of flunitrazepam for this purpose is relatively uncommon. The most commonly used drug to facilitate sexual assault is alcohol. The prevalence of drugs cited in date rape cases is as follows, in decreasing order: alcohol, marijuana, cocaine, GHB, benzodiazepines, opiates, barbiturates, methamphetamines/amphetamine, and flunitrazepam.

Psilocybin, Psilocin

Found primarily in mushrooms in the genus Psilocybe, these compounds are tryptamines similar in structure to serotonin. Likely owing to their 5-HT2A agonist properties, they can exert psychiatric effects akin to LSD; however, the “high” is described by users as being more “natural” in feel than LSD. Tolerance develops rapidly, and cross-tolerance exists with LSD. Psilocin is significantly more active than psilocybin in effect, but psilocin is also far less stable and often oxidizes when the mushroom is dried for use, and therefore is usually present in trace amounts only (compared to a psilocybin concentration of 0.2% to 0.4%).40 Drug testing for psilocybin and psilocin requires specific request. Use of psilocybin mushrooms (eaten or brewed) dates back to 1000 to 500 BCE, particularly by Aztecs. In 1957, Sandoz Pharmaceuticals assisted researchers in extracting the active substances.

Poisonous mushrooms are quite similar in appearance to psilocybin mushrooms, and thus toxicity from the ingestion of misidentified mushrooms is an important risk in psilocybin users. Psilocybes mushrooms tend to stain blue when touched or cut.

HARM REDUCTION PROGRAMS

Harm reduction is an evolving set of techniques aimed at reducing the complications associated with harmful substance use without focusing on making the user drug-free. Efforts focused on club drugs and raves have included various means of promoting the points illustrated in Table 1, such as handing out overdose alert cards at raves (Figure 7).4 Harm reduction organizations in the United States include the Harm Reduction Coalition (http://www.harmreduction.org/home.html) and DanceSafe (http://www.dancesafe.org/).
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Table 1. Harm Reduction Messages Aimed at Rave Attendees

- Replenish fluids and sodium (500 mL/hr if dancing, 250 mL/hr if not) to minimize dehydration
- Take regular dancing breaks to cool down
- Know the risks of adulterated drugs
- Don’t trust pill logos
- Know signs of toxicity from club drugs
- Avoid alcohol
- Ensure a medical team is on site at the event
- Don’t attend the event alone (buddy system)
- Beware of date rape

Adapted from Weir E. Raves: a review of the culture, the drugs and the prevention of harm. CMAJ 2000;162:1843–8.

SUMMARY

The use of club drugs such as MDMA, GHB, and ketamine has been rising in alarming rates over the past decade in a new social context (raves) with an old theme (youth and counterculture). These drugs are usually not tested for in routine medical urine drug screens. Knowledge regarding their epidemiology, pharmacology, usage patterns, medical complications, and the historical context within which they are used is important to all clinicians working in general medical practice, pediatrics, psychiatry, and in the addiction subspecialties.

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