MacLean, K.A., Johnson, M.W., & Griffiths, R.R. (2015). Hallucinogens and Club Drugs. In M. Galanter, H.D. Kleber, & K.T. Brady (Eds.), Textbook of Substance Abuse Treatment, Fifth Edition (pp. 209-222). Arlington, VA: American Psychiatric Publishing.

Hallucinogens and Club Drugs

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Hallucinogens are a pharmacologically diverse group of compounds, including plant- and animal-sourced compounds and synthetic drugs that produce unique alterations of consciousness (Table 15–1). These psychoactive substances produce a profile of changes in thoughts, perceptions, and emotions, often including profound alterations in the perception of reality, that are rarely experienced except in dreams, naturally occurring mystical experiences, and acute psychosis. Naturally occurring hallucinogenic agents have been used for millennia by indigenous cultures in rites of passage, divination, and healing ceremonies (Ott 1996; Schultes et al. 2001) and continue to be used as sacramental and recreational drugs. Examples include psilocybin (the main psychoactive constituent in the *Psilocybe* genus and several other genera of mushrooms), dimethyltryptamine (DMT, one of the main psychoactive constituents in the plant admixture ayahuasca), mescaline (the main psychoactive constituent in peyote cactus), and salvinorin A (the main psychoactive constituent of *Salvia divinorum*, a plant in the mint family). Anticholinergic hallucinogens such as scopolamine (the main psychoactive constituent in the *Datura* genus and several other genera of plants) have an ancient history of sacramental use in Asia and South America, but have limited modern recreational use because of their unpleasant hallucinogenic effects (Ott 1996). Synthetic hallucinogens include lysergic acid diethylamide (LSD), dissociative anesthetics (phencyclidine [PCP], ketamine, dextromethorphan [DXM]), methylenedioxymethamphetamine (MDMA, often contained in the illicit drug ecstasy), and designer hallucinogens (chemical analogues of known hallucinogens; see Table 15–2 later in this chapter).

TABLE 15–1. **Hallucinogens**

Category	Example	Common names of drug or drug medium[a]
Classic hallucinogen		
Tryptamine	Lysergic acid diethylamide (LSD)	Acid, L, blotter, tabs
	Lysergic acid amide (LSA)	Morning glory seeds, Hawaiian baby woodrose seeds
	Psilocybin	Magic mushrooms, 'shrooms
	Dimethyltryptamine (DMT)	Dimitri, businessman's trip, elf spice
	5-Methoxy-DMT	5-MeO, toad venom, 5pice
	Alpha-methyltryptamine (AMT)	Love pills, trip, IT-290
Phenethylamine	Mescaline	Mescalito, peyote, San Pedro, Peruvian Torch
	Dimethoxymethylamphetamine (DOM)	STP (Serenity, Tranquility, Peace)
Entactogen	Methylenedioxymethamphetamine (MDMA)	Ecstasy, E, X, Molly
Dissociative anesthetic	Phencyclidine (PCP)	Angel dust, Sherms, embalming fluid
	Ketamine	K, Special K, cat tranquilizer
	Dextromethorphan (DXM)	Robo-tripping, DM
Atypical hallucinogen	Salvinorin A	Salvia divinorum, Ska Maria Pastora, Sally-D
	Scopolamine	Scospace, zombie drug, jimson weed, Datura, henbane

[a]Common names and slang terms for hallucinogenic compounds may differ depending on geographic location and demographic background of the patient. Several of the common names have been obtained from the Web site www.erowid.org.

Hallucinogen use is relatively low compared with other drug classes; however, a nontrivial number of individuals report regular use of hallucinogens. In the 2011 U.S. National Survey on Drug Use and Health, approximately 1 million people 12 years and older reported using a hallucinogen (broadly defined) in the past month (Substance Abuse and Mental Health Services Administration [SAMHSA] 2012).

Many hallucinogens are currently classified in the United States as Schedule I compounds (i.e., compounds having high abuse potential and no accepted safe medical use). However, animal studies and epidemiological data indicate low to moderate risk of addiction for the classic serotonergically mediated hallucinogens (Anthony et al. 1994; Fantegrossi et al. 2004). Some clinical findings suggest potential medical uses for some of the Schedule I hallucinogens, such as in the treatment of alcoholism (LSD; Krebs and Johansen 2012), posttraumatic stress disorder (MDMA; Mithoefer et al. 2013), and anxiety related to terminal illness (psilocybin; Grob et al. 2011). Some hallucinogenic drugs are not designated Schedule I and have accepted medical uses, such as ketamine (for anesthesia), dextromethorphan (for cough suppression), and scopolamine (for nausea and motion sickness).

Club drug is a term used to describe a range of compounds, including various hallucinogens, stimulants, sedatives, and other drugs that have been associated with dance clubs, bars, raves, and outdoor music festivals. The term *club drug* is a misnomer, however, in that drugs that are used primarily in bars and clubs (e.g., alcohol, tobacco) are not included in this category, whereas hallucinogens are often used in private or in small-group settings. Nevertheless, the National Institute on Drug Abuse has identified the following compounds as club drugs: LSD, ketamine, MDMA, methamphetamine, γ-hydroxybutyrate (GHB), and flunitrazepam (Rohypnol). This chapter addresses the first three compounds, which fall within the broad classification of hallucinogens. Methamphetamine is covered in Chapter 12, "Neurobiology of Stimulants," and Chapter 14, "Clinical Management: Methamphetamine."

Classic Hallucinogens

Classic hallucinogens are divided into two chemical classes: 1) *tryptamines*, such as LSD, psilocybin, and DMT, and 2) *phenethylamines*, such as mescaline. Classic hallucinogens are agonists at postsynaptic serotonin type 2 receptors (primarily 5-HT_{2A}), are recognized by animals trained to discriminate the chemical dimethoxymethylamphetamine, and produce a generally similar profile of subjective effects (Glennon et al. 1984; Nichols 2004).

Subjective Effects

Hallucinogens may be taken orally, snorted, smoked, or injected. The time course and intensity of effects can vary greatly depending on route of administration and type of compound. For example, smoked DMT produces intense psychological effects that begin about 15 seconds following inhalation and subside within 15 minutes. A common oral dose of LSD (e.g., 100–200 μg) produces moderate to strong effects within 1–2 hours, and the effects largely subside after about 7–8 hours, with residual effects lasting 12 hours or more. Clinical management of hallucinogenic effects is greatly aided if one can ascertain the

time of ingestion and identity of the ingested substance(s).

Hallucinogens produce robust changes in perception, cognition, affect, and somesthesia. Perceptual effects can include visual illusions, intensification of colors, visions of geometric patterns and detailed scenes with eyes closed, and synesthesia (e.g., listening to music produces the perception of seeing colors). Despite the term *hallucinogen*, frank visual and auditory hallucinations (e.g., vivid sensory experiences that appear without apparent stimuli) are uncommon, although perceptual alterations (i.e., illusions) are common. Perception of time and space may also be strongly altered, with individuals reporting extreme slowing of time and changes in their body size (e.g., very large or very small) relative to the environment. Other effects may include intense emotions (e.g., awe, joy, fear, grief), changes in the importance or meaning of people and objects in the environment, thoughts concerning one's personal history or life circumstances, changes in sense of reality and self, and spiritual experiences (Griffiths et al. 2006, 2008, 2011). Physiological effects are modest and may include dizziness, nausea, drowsiness, paresthesia, blurred vision, dilated pupils, and increases in heart rate and blood pressure.

Treatment of Acute Effects

Acute hallucinogenic effects rarely require medical treatment. However, the effects of hallucinogens vary greatly across individuals and occasions of use, and even experienced users may have unpredictable adverse psychological reactions. The most likely adverse effect for which someone would seek medical attention is anxiety or panic during the period of acute drug action. "Bad trips" are characterized by intense negative emotions such as nervousness, fear, dysphoria, and paranoia. Verbal reassurance (e.g., reminding the patient that he or she is safe), physical contact (holding the patient's hand), and a calm environment are usually effective in managing adverse reactions. Music, deep breathing, meditation, and guided imagery techniques may be helpful. Findings from laboratory studies indicate that when strong interpersonal support is provided during acute drug effects (Johnson et al. 2008), patients retrospectively view periods of fear and distress as personally meaningful life experiences (Griffiths et al. 2011).

There are no approved medications to reverse acute hallucinogenic effects. Severe agitation can be treated with benzodiazepines. Antipsychotic medications can also be used, although these may entail added risks such as dysphoria (e.g., with risperidone) and paradoxical increases in hallucinogenic effects (e.g., with haloperidol) (Vollenweider et al. 1998). Hypertensive crisis can be treated with intravenous labetalol. Serotonin syndrome, a toxic and potentially fatal condition involving excessive levels of serotonin in the brain, may occur in individuals who have ingested a classic hallucinogen along with a monoamine oxidase inhibitor (as in ayahuasca brew; Callaway and Grob 1998). The major symptoms of serotonin syndrome include increased autonomic activity (mydriasis, tachycardia, increased blood pressure), delirium, nausea, and vomiting. A treatment approved by the U.S. Food and Drug Administration for serotonin syndrome is cyproheptadine, a serotonergic antagonist.

Long-Term Consequences

Classic hallucinogens are abused (i.e., used in such a way that jeopardizes the

safety or well-being of the user or others); however, they are not typically considered drugs of dependence or addiction because they do not engender compulsive drug seeking and are not associated with a known withdrawal syndrome. Classic hallucinogens possess relatively low physiological toxicity and have not been shown to result in organ damage or neuropsychological deficits. In a review of hundreds of research reports on the nonmedical use of psychoactive drugs (including alcohol and nicotine), Gable (1993) concluded that psilocybin had the lowest levels of acute toxicity and dependence potential of the 20 drugs that were studied.

A rare yet possible risk of hallucinogens is prolonged adverse psychological reactions, such as psychosis and depression. Individuals with personal or family histories of psychiatric conditions, particularly schizophrenia or mania, are believed to be at an increased risk for hallucinogen-induced psychosis (Strassman 1984). A meta-analysis of human laboratory studies of psilocybin (Studerus et al. 2011) found that prolonged adverse reactions, such as persisting psychosis and depression, were rare. Among 110 healthy participants, none had an incident of psychotic reactions or precipitation of schizophrenia spectrum disorders and only one experienced depression and anxiety that lasted for several weeks after receiving a moderate dose of psilocybin. The risk that hallucinogens can precipitate enduring psychiatric illness such as schizophrenia in vulnerable individuals cannot be excluded completely.

The most commonly associated long-term risk of classic hallucinogen use is hallucinogen persisting perception disorder (HPPD), frequently referred to as "flashbacks." A flashback involves unexpectedly reexperiencing the perceptual, emotional, or somatic effects of a previous hallucinogen experience. For the psychiatric diagnostic criteria for HPPD to be met, perceptual disturbances must last beyond the normal duration of drug effects and must be clinically distressing or impairing. In a Web-based survey of more than 15,000 hallucinogen users (Baggott et al. 2011), 4% of respondents reported reoccurring perceptual effects that were distressing or impairing. HPPD or other perceptual abnormalities are infrequent in clinical research and are most common in illicit recreational use, which may include polydrug use. The occurrence of perceptual abnormalities in illicit drug users may also be due to underlying psychiatric disorders.

Medical and Therapeutic Applications

In the 1950s and 1960s, thousands of individuals were administered classic hallucinogens, including LSD and psilocybin, as part of research studies and clinical treatment. Findings suggested that classic hallucinogens could be administered safely in a controlled environment and had potential therapeutic applications in the treatment of addiction (Krebs and Johansen 2012) and the treatment of pain, anxiety, and distress associated with terminal illness (e.g., Grob et al. 2013). However, recreational hallucinogen use escalated in the late 1960s, leading to the placement of hallucinogens into Schedule I of the 1970 Controlled Substances Act, which created barriers for human research. More recently, double-blind clinical trials have examined hallucinogens in the treatment of mood disorders, such as depression and anxiety related to advanced-stage cancer (Grob et al. 2011). Double-blind, placebo-controlled studies of healthy individuals have indicated that hallucinogens can re-

liably occasion spiritual experiences that are associated with positive behavior change, increases in the personality trait of openness, and improvements in personal well-being, life satisfaction, and interpersonal relationships (Griffiths et al. 2006, 2008, 2011; MacLean et al. 2011), suggesting possible avenues for future clinical application.

Salvia Divinorum (Salvinorin A)

Salvia divinorum is a psychoactive mint plant that has been used for centuries in ethnomedical practices in Mexico and has gained increased popularity as a recreational drug (Ott 1996). The main psychoactive ingredient is salvinorin A, a selective κ-opioid agonist with no activity at 5-HT_{2A} receptors, making it pharmacologically distinct from classic hallucinogens (Roth et al. 2002).

Subjective Effects

The dry leaves of *S. divinorum* are commonly smoked, although some users report psychoactive effects from chewing fresh leaves and holding them in the mouth for extended periods of time (Ott 1996). Findings from double-blind, placebo-controlled human laboratory studies of inhaled salvinorin A indicate intense subjective effects that only partially overlap with those of classic hallucinogens (Johnson et al. 2011; MacLean et al. 2013). At high doses, salvinorin A produces marked changes in visual, auditory, and tactile perception; disruptions in vestibular and proprioceptive processing; memory impairments and lack of awareness of the surrounding environment; reliving of childhood memories; and interactions with imaginary beings. Physiological effects are minimal and include sweating and chills. Some individuals, including first-time users and experienced hallucinogen users, are disinclined to continue using *S. divinorum* because of the psychological intensity and the bizarre, sometimes unpleasant nature of the subjective effects. Surveys suggest that many users use the substance only a few times ever (Baggott et al. 2010).

Treatment of Acute Effects

Because the effects of smoked *S. divinorum* are relatively brief (resolving within about 10–15 minutes), it is unlikely that a medical professional would encounter a patient who is experiencing acute drug effects. Depending on the intensity of effects, individuals may be completely unresponsive and unaware of the environment. Some individuals may be confused and exhibit erratic, potentially dangerous behavior such as falling. Thus, the caregiver should focus on keeping the person physically safe until the drug effects have worn off. Interpersonal support and hand holding may be helpful once the patient is lucid and can communicate about his or her experience.

Long-Term Consequences

A small percentage of users report persistent anxiety after *S. divinorum* use; however, a large percentage of users report persisting positive psychological effects (Baggott et al. 2010). In one laboratory study of medically and psychologically healthy hallucinogen users (Johnson et al. 2011; MacLean et al. 2013), there was no evidence of depression, psychiatric symptoms, or perceptual disturbances 1 month after participants completed a regimen of 17 doses of salvinorin A (1–3 doses per week on average). There are two case reports of persisting psychotic-type episodes associated with *S.*

divinorum use, although both cases were complex and difficult to attribute to use of this drug (cf. Johnson et al. 2011). The theoretical possibility remains that exposure to *S. divinorum* in those predisposed to psychotic process may trigger a psychotic episode, as with classic hallucinogens (Johnson et al. 2008). Although experienced hallucinogen users show increased ratings of "liking" after salvinorin A administration in controlled studies (e.g., MacLean et al. 2013), addiction to *S. divinorum* is not commonly reported in surveys of users (Baggott et al. 2010).

Medical and Therapeutic Applications

Nonhuman laboratory studies have characterized the behavioral and discriminative effects of *S. divinorum* and salvinorin A, and there is interest in using salvinorin A or related κ-opioid agonists in the treatment of chronic pain and cocaine drug dependence (Sheffler and Roth 2003). The intense subjective effects observed in humans make it unlikely that inhaled salvinorin A could be used as a medical treatment. However, the development of other formulations of salvinorin A and orally administered κ-opioid agonists with fewer side effects may prove fruitful.

MDMA (Ecstasy)

MDMA is a synthetic drug that is structurally similar to both amphetamine and mescaline and is known for its unique mix of mood-enhancing, stimulant-like, and hallucinogenic effects (Shulgin and Nichols 1978). MDMA exerts most of its psychoactive effects by promoting the release of serotonin and other monoamines (norepinephrine and dopamine), as well as by preventing the reuptake of serotonin. In the early 1980s, before MDMA became a popular recreational drug and was added to the list of Schedule I controlled compounds, there was some interest in using it as a therapeutic tool (see Johansen and Krebs 2009).

Subjective Effects

MDMA is typically taken orally in tablet or capsule form, with onset of effects about 30–60 minutes after administration and effects lasting around 4–6 hours. MDMA has been labeled an entactogen for its ability to promote increases in positive mood, as well as feelings of interpersonal openness, trust, and empathy (Johansen and Krebs 2009; Nichols et al. 1986). Physiological effects of MDMA include dilated pupils and increases in heart rate and blood pressure. Due to its mood-enhancing and stimulant-like effects (e.g., increases in energy), MDMA has been a drug of abuse at clubs and all-night dance parties called raves.

Treatment of Acute Effects

With MDMA, as with classic hallucinogens, acute psychiatric complaints such as anxiety and panic can be managed well with interpersonal support and administration of benzodiazepines in the case of extreme agitation. The most significant risks posed by MDMA are physiological. Sympathomimetic effects can lead to cardiac complications including tachycardia, arrhythmia, stroke, cerebral hemorrhage, and myocardial infarction. Other serious adverse effects associated with MDMA use are hyperthermia and hyponatremia (P. Halpern et al. 2011).

Long-Term Consequences

Heavy and extended use of MDMA may produce changes in attention, memory, and mood, as well as depressive symp-

toms, anxiety, and difficulty sleeping (McCann and Ricaurte 2007). Numerous studies in rats and nonhuman primates indicate that high or repeated doses of MDMA can damage serotonergic axons as a result of oxidative stress (Ricaurte et al. 2000). However, there is controversy within the scientific community as to how well the toxic dose regimens used in nonhuman animal research relate to doses typically used by humans.

In evaluating human research with illicit users, it is important to consider that the street drug ecstasy often contains other drugs besides MDMA and sometimes does not contain any MDMA (Baggott et al. 2000). Findings from cross-sectional studies in humans suggest differences in cognitive performance, brain activity, and markers of serotonin function between ecstasy users and control subjects. However, mixed results have been reported in studies that have equated ecstasy users and non–ecstasy users in terms of polydrug use and demographic factors (e.g., Erritzoe et al. 2011; J. H. Halpern et al. 2011). There is little conclusive evidence in humans that MDMA causes neurotoxicity or clinically relevant impairments in brain function at doses (e.g., 100–200 mg) and total exposures (e.g., one to three sessions) used in clinical trials.

Medical and Therapeutic Applications

MDMA was originally used as an aid in psychotherapy during the late 1970s and early 1980s. Although MDMA has remained a Schedule I compound since 1985, it has been administered to nearly 500 individuals in the context of research studies and federally approved clinical trials. Initial clinical trials suggest that MDMA may be a safe and effective treatment for posttraumatic stress disorder, with patients exhibiting clinically significant relief from symptoms for years after initial treatment (Mithoefer et al. 2013).

Dissociative Anesthetics

PCP, ketamine, and DXM are dissociative anesthetics that have hallucinogenic effects. In contrast to classic hallucinogens, dissociative anesthetics such as PCP and ketamine have considerable addiction (e.g., compulsive drug-seeking) liability (Lerner and Burns 1978). These compounds exert their psychoactive effects by blocking *N*-methyl-D-aspartate (NMDA) glutamate receptors. PCP was previously used for both human and veterinary anesthesia, although medical use was discontinued because of reports of psychosis, agitation, and dysphoria after surgery (Bey and Patel 2007). PCP was popular as a recreational drug during the 1970s; its popularity has declined since the 1980s, although it is still used nonmedically (Bey and Patel 2007). Ketamine is widely used for human and veterinary anesthesia and has also been used as a recreational drug (Morgan et al. 2010; Muetzelfeldt et al. 2008). DXM is a widely available over-the-counter cough suppressant that has been used for more than 40 years (Bem and Peck 1992). Survey and epidemiological data show that DXM has been used as a recreational drug since the mid-1960s (Bem and Peck 1992).

Subjective Effects

Dissociative anesthetics are commonly taken orally, intravenously, or intranasally. PCP produces brief, dissociative psychotic-like reactions, including catatonic-like states and extreme agitation. The subjective effects of ketamine and

DXM overlap with the effects of classic hallucinogens, including perceptual distortions, changes in sense of time and space, alterations in body awareness, and spiritual experiences (Reissig et al. 2012). Anecdotally, some users report more dissociative effects with dissociative anesthetics (e.g., classic out-of-body experiences) than with classic hallucinogens. Unlike classic hallucinogens, dissociative anesthetics are associated with strong motor impairment (Reissig et al. 2012).

Treatment of Acute Effects

Clinical support of distress associated with ketamine and DXM is similar to the methods used with classic hallucinogens. For DXM, which is usually consumed orally, activated charcoal is effective for gastrointestinal decontamination. Naloxone (an opioid antagonist) has been recommended for treatment of DXM overdose (Chyka et al. 2007).

In emergency department settings, the management of PCP intoxication can be complex. It should involve initial assessment and stabilization of breathing, circulation, temperature, and neurological condition. A dissociative phenomenon can occur during which PCP users exhibit dangerous, violent, and/or psychotic-like behaviors (Marrs-Simon et al. 1988). Individuals with a history of psychosis are more likely to exhibit assaultive behavior after PCP use (McCardle and Fishbein 1989). Physical restraint may increase the risk of rhabdomyolysis. Although antipsychotic medications can have dangerous interactions with PCP, haloperidol and lorazepam can be used if the patient poses a serious threat to self or others. Cardiac monitoring should be continuous, because intracerebral hemorrhage can result from hypertension. Prolonged seizure can be treated with intravenous benzodiazepines or phenobarbital (Bey and Patel 2007). The effects of PCP can last for several days.

Long-Term Consequences

Survey studies of frequent ketamine users indicate memory loss, decreased sociability, physical health problems (e.g., gastric pain, cystitis), and addictive/dependent behaviors such as using ketamine without stopping and taking steadily increasing doses (Muetzelfeldt et al. 2008). Neuropsychology studies of ketamine users have confirmed memory-related cognitive deficits, as well as dissociative symptoms and depression in frequent and abstinent users (Morgan et al. 2010). However, findings from human laboratory studies show no evidence that controlled administration of a small number of doses of ketamine produces persistent psychotic states or changes in aspects of psychopathology in healthy individuals (Gouzoulis-Mayfrank et al. 2005). Likewise, administration of DXM to regular hallucinogen users did not produce persistent psychosis, distress, or visual disturbances (Reissig et al. 2012).

Some data from rat studies suggest that NMDA antagonists can induce pathological changes in cerebrocortical neurons (Olney et al. 1989). There is no evidence of neurotoxic effects in humans. Chronic PCP use may lead to a form of mental impairment called *phencyclidine organic mental disorder*, which is characterized by memory deficits, confusion, assaultiveness, visual disturbances, and speech difficulty (Weaver and Scholl 2007).

Medical and Therapeutic Applications

In addition to its accepted medical use in human and veterinary anesthesia, ketamine may be an effective treatment for substance abuse and depression. In clinical trials, relatively low or moderate

TABLE 15–2. **Designer hallucinogens**

Category[a]	Example	Common name(s) of drug[b]
Tryptamine	5-MeO-DiPT	Foxy, foxy methoxy
	4-AcO-DMT	Psilacetin
Phenethylamine	2C-B	Nexus, bees, bromo
	2C-I	Smiles
	2C-E	Europa
	2C-T-7	Blue mystic, 7th heaven
	25I-NBOMe	25-I, N bomb
Dissociative anesthetic	Methoxetamine	MXE, mexxy, roflcopter
	4-MeO-PCP	Methoxydine

[a]Categorization is based on chemical structure. The subjective effects of the listed designer hallucinogen may only partially resemble the effects of classic hallucinogens (tryptamines and phenethylamines) or dissociative anesthetics. Acute risks, including toxicity and potential for overdose, may be greater for some designer hallucinogens than for traditional hallucinogens.
[b]Several of the common names have been obtained from the Web site www.erowid.org.

(i.e., nonanesthetic) doses of intravenous ketamine have been shown to promote long-term abstinence in both alcohol- and heroin-dependent populations (Krupitsky and Grinenko 1997). Double-blind, placebo-controlled studies have demonstrated the efficacy of ketamine in producing rapid (within hours) antidepressant effects that are clinically robust in patients with treatment-resistant major depression (Zarate et al. 2006).

Designer Hallucinogens

Designer drugs (sometimes marketed as "research chemicals") (Table 15–2) are synthesized in clandestine laboratories in an attempt to circumvent national and international drug laws. Designer drugs with hallucinogenic properties are analogues of known tryptamines (Shulgin and Shulgin 1997), phenethylamines (Shulgin and Shulgin 1991), and dissociative anesthetics.

Designer drugs are commonly purchased through anonymous, online distribution sites, and many newly synthesized compounds remain legal.

The profile of subjective effects of a given designer drug will usually be most similar to drugs from the same chemical class. For example, the designer drug 2-CB is a phenethylamine analogue that has hallucinogenic properties similar to the serotonergically mediated phenethylamine mescaline. The methods used for clinical management of acute effects are similar to those used with classic hallucinogens, MDMA, or dissociative anesthetics. Complicating matters, however, the physical and psychological risks of many designer hallucinogens are unknown. Compounds that are chemically similar to relatively benign classic hallucinogens may be toxic or even fatal. Nevertheless, confirmed fatalities are relatively rare and usually result from lack of knowledge about the identity or appropriate dose of a given drug. Because of the lack of basic safety data in nonhumans, rigorous human research trials with designer hallucinogens are nonexistent.

Key Points

- Hallucinogens are a pharmacologically diverse group of compounds that produce a unique profile of changes in thoughts, perceptions, and emotions, often including profound alterations in the perception of reality that are rarely experienced except in dreams, naturally occurring mystical experiences, and acute psychosis.
- Acute hallucinogenic effects do not often require pharmacological treatment. The most likely adverse effect of hallucinogens is anxiety or panic, which can be managed well with interpersonal support. Benzodiazepines and antipsychotic medication can be used in cases of extreme agitation.
- Although hallucinogens are drugs of abuse, there are accepted medical uses for some hallucinogens (e.g., ketamine), and clinical research trials indicate potential therapeutic effects on anxiety in cancer patients (e.g., psilocybin) and patients with posttraumatic stress disorder (e.g., MDMA).
- Designer hallucinogens—analogues of classic hallucinogens (tryptamines and phenethylamines)—may have greater physiological toxicity and long-term risks than classic hallucinogens.

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Suggested Web Resources

Erowid: A Web-based repository for up-to-date information on psychoactive compounds, including hallucinogens and designer drugs. Available at: http://www.erowid.org.

National Institute on Drug Abuse: Important information and resources on club drugs. Available at: http://www.drugabuse.gov/drugs-abuse/club-drugs.

Suggested Readings

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