

## Review Article

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# Club drugs: review of the 'rave' with a note of concern for the Indian scenario

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**'Club drugs' which include Ecstasy, gamma-hydroxybutyrate (GHB), ketamine, and Rohypnol (flunitrazepam) have become popular with participants in 'raves', because they are perceived to enhance energy, endurance, sociability and sexual arousal. These drugs vary in their pharmacologic properties, physiological and psychological effects, and potential consequences. The use of club drugs by young people has increased in the last decade, and continue to get modified and evolve, making them very difficult to monitor. Further, these drugs are not picked up by routine drugs screening procedures, thereby making these popular with the criminals. India, which is in a phase of social transition, also faces this rising menace. Despite the nature and extent of this problem, this area has been under-researched. Data from India are sparse barring a few newspaper and police reports. Keeping abreast of current trends in club drug use prepares the clinician to recognize the clinical effects of club drug use, to manage club drug related emergencies, and to generate social awareness.**

**Key words** Club drugs - Ecstasy - gamma-hydroxybutyrate - ketamine - rave drugs - rohypnol

### Introduction

'Raves' are parties with loud, electronic "techno-rock" music, laser light shows, and all-night dancing held in clandestine locations, including warehouses, nightclubs, and farm fields<sup>1</sup>. These first became popular in Great Britain in the late 1980s. The underground or noncommercial music featured at raves which is produced by computers and include little or no vocals is distinct from the music played at conventional nightclubs. Following bans in some countries the rave parties moved in to legitimate nightclubs. A raver is a person who has an exciting and uninhibited social life and regularly goes to raves. Not all ravers use drugs;

however, many illicit drugs are available at raves and are used liberally to enhance the "vibe"<sup>2</sup>. The words like 'rave drugs', 'club drugs' and 'party drugs' have been used interchangeably in the literature. The U.S. National Institute on Drug Abuse (NIDA) in its "Community Alert on Club Drugs," defined "club drugs" as Ecstasy (3,4-methylenedioxyamphetamine, or MDMA), gamma-hydroxybutyrate (GHB), ketamine, Rohypnol (flunitrazepam), methamphetamine, and lysergic acid diethylamide (LSD)<sup>3</sup>. On the other hand, the U.S. Office of National Drug Control Policy identifies four specific club drugs: MDMA, GHB, ketamine, and Rohypnol<sup>4</sup>. Methamphetamine and LSD have been inconsistently included in the category of the club drugs. In addition,

these two drugs have a longer history of misuse in comparison to the other four which came to the scene much later (MDMA being first reported in 1985).

In this review the term 'club drugs' will be used in reference to Ecstasy, GHB, ketamine, and Rohypnol to avoid further confusion. These drugs are being used in an expanding variety of venues by groups who differ in terms of age, gender, sexual orientation, and race/ethnicity<sup>5</sup>. Each of these drugs has very different pharmacologic properties, physiological and psychological effects, and potential consequences (Table I). Despite evidence that suggests an increase in the population level use of these drugs, research on the population prevalence of their use is limited. For Ecstasy the prevalence has been estimated between 0.9 per cent in France to 19.7 per cent in Australia<sup>24,25</sup>. The others are much less prevalent - 0.9-4.7 and 0.7-2.6 per cent for GHB and ketamine respectively, depending upon the population and age group studied<sup>26-28</sup>. One study reported 20 per cent of American youths aged 16-23 having ever used one or more of these club drugs<sup>29</sup>. Further, polysubstance abuse is common among users of club drugs, often used in combination, particularly with marijuana or alcohol<sup>29-31</sup>. It has been found that, club drug use is common among youths in treatment for substance abuse and has spread beyond the rave culture<sup>32</sup>. A study assessing the pattern of club drug initiation among gay and bisexual men suggests that the sequencing of club drug use may be better explained by socialization processes in the gay community than by Gateway Theory, which has been traditionally used to explain patterns of drug use in the population<sup>33</sup>.

Studies on short- and long-term effects of these club drugs on animals have shown that these drugs can be damaging in multiple ways, and some drugs (particularly MDMA) can damage specific neurons in the brain<sup>5</sup>. Moreover, routine drug screens do not pick up various club drugs, and gas chromatography-mass spectrometry (GC-MS) testing must be requested to confirm the presence of the specific drug, otherwise many cases of sexual assault and driving under the influence may go undetected. This review describes the brief history, neurobiology, pharmacology, toxicity and side effect profile, treatment strategies of club drugs with specific reference to the Indian scenario.

*Data search methodology:* The data search strategies included electronic databases as well as hand-search of relevant publications or cross-references. The electronic search included PubMed and other search engines (*e.g.* Google Scholar, PsychINFO, *etc.*). Cross-searches

of online websites and hand-search of key references yielded other relevant material. The search terms used, in various combinations, were: club drugs, rave drugs, rave parties, Ecstasy, Ketamine, Rohypnol, GHB, street names, pharmacology, toxicology, psychiatric disorders and management.

### **Ecstasy (3,4-methylenedioxymethamphetamine, MDMA)**

Ecstasy (also called X, M, E, XTC, rolls, beans, Clarity, Adam, lover's speed, hug drug) is a synthetic, psychoactive drug with both stimulant and hallucinogenic properties similar to methamphetamine and mescaline. MDMA was developed in 1914 as an appetite suppressant, but animal tests were unimpressive, and it was never tested in humans<sup>16</sup>. In the 1970s and 1980s, MDMA was thought to be a useful adjunct to psychotherapy due to the altered state of consciousness it produced<sup>34</sup>. Despite the lack of scientific evidence, the Multidisciplinary Association for Psychedelic Studies (MAPS) is currently supporting the progression of research to investigate the effectiveness of MDMA as a therapeutic adjunct to psychotherapy<sup>35</sup>. It has been a Schedule I drug in USA since 1985 having a high potential for abuse. There is no currently accepted medical use of this drug in treatment in the USA, and there is a lack of accepted safety for use of this drug under medical supervision<sup>36</sup>.

MDMA can be taken through various routes<sup>6</sup> (Table I). Most tablets containing MDMA are produced in clandestine laboratories in Belgium, Luxemburg, or in Asia. The simplest method of manufacturing MDMA is through 3,4-methylenedioxyphenyl-2-propanone (PMK), a commercially available ketone. Other common precursors include saffrol, isosaffrol, piperonal, and safrole (often from sassafras oil)<sup>37</sup>. A review of surveys on the contents of Ecstasy tablets<sup>38</sup> found in the 1980s and early 1990s that there were few problems with purity, and tablets nearly always contained MDMA. During the mid-1990s, the majority of Ecstasy tablets continued to contain MDMA, but others contained 3,4-methylenedioxyamphetamine (MDA), 3,4-methylenedioxyethamphetamine (MDEA), or amphetamine drug mixtures. However, between 4 and 20 per cent of tablets contained other drugs. During the late 1990s, the proportion of tablets containing MDMA increased to between 80 and 90 per cent purity, and in the early 2000s, purity levels have reached 90 to 100 per cent<sup>38</sup>. MDMA is being increasingly used in combination with ketamine and selective serotonin reuptake inhibitors (SSRIs)

**Table I.** Pharmacological and clinical profile of club drugs

	MDMA	GHB	Ketamine	Rohypnol
Manner of use	Tablet/Capsule; ingested orally, can be crushed/ snorted/ dissolved/injected <sup>20</sup>	Liquid; often mixed with alcohol – effects amplified <sup>20</sup>	Liquid – injected, added to items to be smoked. Powder – dissolved in drinks, smoked, snorted or dissolved and injected <sup>7</sup>	Tablet; typically ingested orally; crushed and snorted
Onset of action; T <sub>1/2</sub>	30 to 60 min 5.8 ± 2.2 h	15-30 min after an oral dose; 22-28 min	Variable; elimination t <sub>1/2</sub> is ~2 h	15-20 min 18 - 26 h
Therapeutic use	Not strongly supported	To treat cataplexy associated with narcolepsy	Veterinary anaesthetic, also used as anaesthetic in India for humans	To treat insomnia
Clinical effects	Feelings of empathy, energy, psychomotor drive, self-confidence, depression, derealization, depersonalization, well being, positive mood, heightened perception & sensory awareness, increase in the sensuality of sexual experiences, inhibition of orgasm and erectile dysfunction, mydriasis <sup>8-12</sup> .	10 mg/kg: euphoria, amnesia, and hypotonia; 20-30 mg/kg: somnolence; > 50 mg/kg: unconsciousness and coma; anxiety reduction, increases relaxation, enhances libido, agitation, nystagmus, ataxia, vomiting, muscle spasms, effects similar to alcohol <sup>13</sup> .	Low dose: relaxation (K-land); Higher dose: dream-like state, hallucinations, visual distortions, sensation of near-death experiences (K-land) <sup>7,14,15</sup> ; nystagmus, increased tone, purposeful movements, amnesia, hallucinations, sympathomimetic symptoms, delirium.	1- or 2-mg dose: reduces anxiety, inhibition, and muscular tension; Higher doses: anterograde amnesia, lack of muscular control, and loss of consciousness <sup>16</sup> .
Toxic effects	Irritability, fatigue, nausea, loss of appetite, weight loss, tachycardia, hypertension, tremors, tics, jaw clenching, serotonin-syndrome, anxiety, bruxism, thought disorder/psychosis, difficulty concentrating, hyperthermia, hyponatremia, hypertension, dysrhythmias, liver toxicity, ataxia, neurotoxicity, rhabdomyolysis, disseminated intravascular coagulation (DIC), seizures, death <sup>17-20</sup> .	Sleep induction, tremors, agitation, seizures, GI symptoms, CNS & respiratory depression, dizziness, confusion, hallucinations, apnoea, bradycardia, unconsciousness, sudden reversible coma with abrupt awakening and violence, death <sup>7,21</sup> .	Increased heart rate, hypertension, cognitive and psychomotor impairment, nausea, respiratory depression, immobility, anxiety, dissociation, depression, recurrent flashbacks, delirium, amnesia, schizophrenic symptoms, loss of consciousness, respiratory depression, catatonia, death <sup>1,7,14,22,23</sup> .	Decreased body temperature and blood pressure, sedation, cognitive & psychomotor impairment, visual disturbances, dizziness, confusion, GI disturbances, urinary retention.
Neural mechanisms	Serotonin, noradrenergic, dopaminergic, cholinergic	Dopaminergic, cortical, hippocampal	Serotonin, dopamine, norepinephrine, calcium channels	Chloride channels
Dependence liability	No dependence	Can produce physical dependence	No dependence	Can produce physical dependence
Long term sequelae	Possible: cognitive deficiencies, brain neurotoxicity	Unknown	cognitive difficulties (attention, learning, memory)	Depression, memory loss, mental confusion, paradoxical agitation, stomach disorders

like fluoxetine and sertraline, which produces a rush initially, prolongs the pleasurable effect, and results in easier comedown following a high<sup>39,40</sup>.

MDMA has multiple effects on central nervous system (CNS). However, its principle effects are on the serotonin system where it is an indirect serotonin agonist. MDMA inhibits tryptophan hydroxylase,

which decreases serotonin production<sup>13</sup>. It induces the release of serotonin and also blocks serotonin reuptake. These effects are thought to be related to the observed depression, anorexia, agitation, and marked feelings of empathy reported in association with use of the drug. Because MDMA depletes serotonin stores in neurons, subsequent doses produce

diminished euphoria and increase adverse effects such as depression and agitation<sup>41</sup>. In addition to its effects on serotonin neurotransmission, MDMA has also been shown to affect the noradrenergic, dopaminergic, and cholinergic neurotransmitter systems<sup>6</sup>. It is generally agreed that MDMA is a human neurotoxin, although the mechanism of action remains an active area of research<sup>42</sup>.

A multisite study exploring the psycho-economic factors of Ecstasy consumption has found that monetary and opportunity cost, but not income, significantly predicted Ecstasy use<sup>43</sup>. A recent study has found that low risk perception, high perceived behavioural control of obtaining Ecstasy (an estimated Ecstasy procurement time less than 24 h), and current Ecstasy dependence predicted future Ecstasy use<sup>44</sup>.

Clinical effects of MDMA have been varied ranging from increased energy to depression<sup>8-12</sup> (Table I). Ecstasy use can also bring about a range of negative physical side effects while under the influence of the drug and during the comedown period<sup>17-20</sup> (Table I). By 1997 there were 53 reports in the UK of Ecstasy producing severe acute toxicity resulting in death<sup>45</sup>. MDMA ingestion directly causes a rise in antidiuretic hormone. Heat from the exertion of dancing in a crowded room coupled with the MDMA-induced hyperthermia can lead easily to excessive water intake and severe hyponatremia, to which young women appear to be particularly susceptible<sup>46,47</sup>. There is no antidote for MDMA, only supportive care similar to treatment of amphetamine or methamphetamine overdose has some benefit<sup>19</sup>. No withdrawal syndrome from MDMA has been reported<sup>13</sup>. Till the beginning of this decade, the use of MDMA has been implicated in 27 deaths in the U.S. and more than 50 in Europe, usually related to severe dehydration, strokes, hyperthermia, and hyponatremia<sup>48</sup>.

The relationship of MDMA and psychiatric disorders has also been the focus of attention of the researchers. MDMA-associated reductions of serotonergic neurotransmission in those at risk for depression, bulimia nervosa, alcoholism, *etc.*, may push the individual over the threshold for psychopathology<sup>49</sup>. The National Drug and Alcohol Research Centre in Australia issued an warning for people who are at high risk of problems from Ecstasy: those with or with a family history of any heart disorder or cardiovascular disease; psychiatric condition, including depression, manic-depression, schizophrenia, and anxiety disorders; kidney disease; neurological or

nervous system impairment (*e.g.*, Parkinson's disease, Tourette's syndrome); taking any antidepressant; taking weight-loss medication; or taking medication for blood pressure<sup>50</sup>. Studies have found that MDMA use may be associated with deficits in executive functioning (planning, initiation, self-regulation of goal-directed behavior), verbal memory and tasks known to be sensitive to temporal functioning<sup>51,52</sup>.

Laboratory identification of MDMA is difficult. As many as one-third of immunoassay urine screens have failed to detect it in standardized specimens<sup>53</sup>, although some cross-reactivity with amphetamines may occur if the concentration is high. Toxicologists have now developed procedures for detection or quantification of MDMA and its metabolites<sup>54</sup>.

### **GHB (Gamma-hydroxybutyrate)**

GHB is available as a clear liquid, white powder (dissolved in water), tablet, or capsule and can be made in private residences with ingredients and recipes obtained on the Internet<sup>14</sup>. Street names include "G", "Liquid Ecstasy," "Scoop," "Easy Lay," "Georgia Home Boy," "Grievous Bodily Harm," "Liquid X," "Goop," "Gib," "Soap," and "Nitro"<sup>16</sup>. Overdose is common because the often unknown strength of the solution. Toxicity of GHB is increased if taken with alcohol or other CNS depressants<sup>55</sup>.

GHB was first synthesized in France in 1960 as an anesthetic but later achieved popularity as a recreational drug and a nutritional supplement marketed to bodybuilders<sup>56</sup>. Nonprescription sales in the United States were banned in 1990 because of adverse effects, including uncontrolled movements and depression of the respiratory and CNS<sup>57</sup>. It is now a Schedule I drug in the U.S. and Schedule IV of the 1971 UN Convention. In 2002, sodium oxybate, a formulation of GHB, was approved for the treatment of narcolepsy and classified as schedule III<sup>16</sup>. Illegal GHB and its precursors, GBL (gamma butyrolactone) and 1,4-BD (1,4-butanediol), can be obtained over the Internet and sometimes are marketed as solvents such as ink jet printer fluid or as GHB alternatives in health food stores, gyms, raves, and nightclubs. Chemistry kits, reagents, and recipes are available on the web to convert the precursors into GHB<sup>58</sup>.

GHB is a naturally occurring neurotransmitter in the brain. Structurally, it is related to GABA (gamma-amino butyric acid) and is a metabolite of GABA. Although it does not bind to GABA-A receptors, it does bind noncompetitively to GABAs but only when

present in amounts larger than that occurs naturally. In addition, there are also high- and low-affinity GHB receptor sites that are highly specific for GHB and their distribution differs from GABA receptors. The highest density of these receptors is in the hippocampus, cortex, and dopaminergic areas (striatum, olfactory tracts, and substantia nigra). GHB inhibits dopamine release and activates tyrosine hydroxylase, that together act to increase central dopamine levels, which could be associated with the reinforcing effects of GHB<sup>59</sup>.

GHB was suggested for medical use in anaesthesia, obstetrics, and psychiatry (including possible use for alcohol and narcotic withdrawal symptoms) in the 1960s<sup>60</sup>. More recently, its role has been explored in the treatment of alcohol and opiate withdrawal, fibromyalgia, and narcolepsy<sup>16,61-63</sup>. GHB is misused at doses ranging from 2.5-30 g<sup>64</sup>. By the 1990s, GHB was being marketed for illicit use in weight control management<sup>19</sup> and its purported anabolic properties and associated muscle growth<sup>14</sup> that made it a popular drug with body builders<sup>6</sup>. In addition, GHB has been implicated for its use in association with sexual assault because victims have difficulty resisting the assault due to the level of intoxication produced. The associated memory problems and the fact that it clears from the body quickly (within 12 h) make detection difficult and increase the complexity of attempts to prosecute for which GHB derived its infamous label of 'date rape' drug<sup>7</sup>. Much interest has been generated regarding driving difficulties caused by GHB. It has been found that, two symptoms that most commonly caused driving difficulties were rapid loss of consciousness or onset of stupor, known among users as "G-napping" and periods of anterograde amnesia<sup>65</sup>.

Illicit use of GHB has been associated with little consistency and precision in the doses consumed<sup>13</sup> (Table I). Adverse effects related to GHB involve CNS, respiratory, cardiovascular and gastrointestinal systems (Table I)<sup>7,13</sup>. The Drug Enforcement Administration (DEA) has documented at least 71 GHB-related deaths<sup>20</sup>. GHB should be used with caution by HIV-seropositive patients with predisposition to seizure disorders or with opportunistic infections that may lower seizure thresholds. GHB may also cause severe nausea, vomiting, and gastrointestinal tract irritation that may complicate antiretroviral therapy and affect adherence to the regimen for HIV drugs<sup>66</sup>.

There is a fair amount of overlap in what appears to be adverse symptoms due to acute effects, and withdrawal symptoms. Dependence on GHB has

been described as developing after regular use, *i.e.*, every 2 h round-the-clock for 2 months to 4 yr<sup>67</sup>. Withdrawal symptoms include insomnia, muscular cramping, tremor, perspiration, anxiety, and feelings of doom<sup>68</sup>. Withdrawal from higher doses includes bowel bladder incontinence and blackouts<sup>69</sup>. Treatment includes high doses of short-acting benzodiazepines (*e.g.*, lorazepam) and trazodone for mild withdrawal symptoms and barbiturates, high-dose of short-acting benzodiazepines, along with a mood stabilizer (such as gabapentin) and low-dose antipsychotic for higher severity of withdrawal symptoms. In addition, restraints can be used for protection of the patient and medical staff<sup>67,70</sup>.

GHB is detected by routine urine screens, but other immunoassays for GHB and GBL are not available. These are reliably detected by specific requests for GC-MS, but timing is important, as GHB is rapidly excreted as carbon dioxide through exhalation<sup>13</sup>. GHB has a half-life of 27 min<sup>71</sup> and it is virtually undetected in the urine 12 h after ingestion<sup>19</sup>. Since GHB continues to be produced after death, it should not be stored in tubes containing citrate, but should be collected in tubes containing sodium fluoride<sup>72</sup>. In cases of chemical submission, both urine and blood should be analyzed, since GHB is present longer in urine than in blood, and a detailed case history should be obtained. A sensitive and specific GC-MS method using selective ion monitoring has been developed for the quantification of GHB in blood<sup>73</sup>.

### **Ketamine**

Ketamine, a derivative of phencyclidine, is an anaesthetic that has been approved for human and animal use, both in trauma and emergency surgery as well as veterinary medicine. Illicit ketamine is also known as Special K, Vitamin K, K, kit-kat, keets, super acid, super K, cat valiums and jet. It can be taken through various routes<sup>7</sup> (Table I). A typical method uses a nasal inhaler, called a "bullet" or "bumper"; an inhalation is called a "bump". Ketamine often is taken in "trail mixes" of methamphetamine, cocaine, sildenafil citrate (Viagra), or heroin<sup>74</sup>. Both popular and research accounts indicate that the recreational use of ketamine has widened in the context of nightclubs, dance parties, and raves<sup>75</sup>.

Ketamine is an N-methyl-D-aspartate (NMDA) receptor antagonist. It binds to the same NMDA receptor site as phencyclidine, located in the calcium channel, leading to a blockade in calcium flow through

these channels. Decreased excitatory amino acid neurotransmission mediated by NMDA receptors through calcium channel blockade has been associated with altered perception, memory, and cognition<sup>22</sup>. NMDA blockade is associated with increased dopamine release in prefrontal cortex and midbrain. NMDA blockade resulting from ketamine binding has also been linked to activation of serotonin systems, particularly serotonin 1A receptors. It has also been suggested that ketamine, through its binding to the NMDA receptor, can inhibit the reuptake of serotonin, dopamine, and norepinephrine, although the mechanism underlying this action is not entirely clear<sup>19</sup>.

Ketamine is a Schedule III controlled substance in USA whereas in India it is placed under Schedule H. Ketamine users often take several sequential doses of the drug in order to maintain psychotropic effects over time. Anaesthesia doses are 2-10 mg/kg while recreational doses can range between 50-100 mg<sup>48</sup>.

When misused, subjective experience depends on the dose of the drug consumed<sup>7,14,15</sup>. Along with “desired” effects, users also commonly experience untoward effects (Table I). They also may experience tachycardia, palpitations, hypertension, and respiratory depression with apnoea<sup>16</sup>. Its use has also been associated with unintentional injuries that can occur because the user is insensitive to pain<sup>1</sup>. In addition, it has been associated with sexual assault because of its dissociative effect in humans<sup>14</sup>.

Acute adverse effects are many<sup>1,7,14,15</sup> (Table I). Effects due to chronic misuse include cognitive difficulties in areas such as attention, learning, and memory<sup>14</sup>. Taffe *et al*<sup>76</sup> concluded that recreational exposure to subanaesthetic doses of ketamine was likely to induce wide-ranging compromise of cognitive function ranging from memory to attentional to motor domains. There are very few deaths by “pure” ketamine overdose recorded. Of the 87 ketamine-linked deaths in New York City, none was purely due to the use of ketamine<sup>77</sup>. There are cases of accidental injections with 10 times the amount required for surgery, with no obvious, lasting effects<sup>78</sup>.

Frequent users often take ketamine in a pattern of cyclical binges similar to cocaine or amphetamine dependence. Tolerance builds rapidly and can be very high<sup>79</sup>. Users can become psychologically dependent, with craving and a high tolerance, but there is little documented evidence of a physiological withdrawal syndrome<sup>79,80</sup>.

Ketamine is not detected in routine drug screens, and clinicians should be aware that immunoassays for PCP may cross-react with ketamine assays<sup>19</sup>. High-performance liquid chromatography (HPLC) is required to reliably detect ketamine<sup>48</sup>.

### **Rohypnol (Flunitrazepam)**

Rohypnol comes in pill form and is typically taken orally, although reports of it being ground and snorted are also available<sup>7</sup>. Street names include Roofies, Rophies, Roche, Forget-me Pill, Circles, Mexican Valium, Rib, Roach-2, Roopies, Rope, Ropies, Ruffies, and Roaches<sup>14</sup>. Rohypnol is a benzodiazepine manufactured by Roche Laboratories, it is available in more than 60 countries in Europe and Latin America for preoperative anaesthesia, sedation, and treatment of insomnia. In the United States where it is not legally available, imported Rohypnol came to prominence in the 1990s as an inexpensive recreational sedative and a “date rape” drug<sup>81</sup>. It is odourless and tasteless and is easily dissolved in beverages<sup>7</sup>, allowing a perpetrator to add it to the beverage of a potential victim. Flunitrazepam has had a long history of abuse by heroin and cocaine addicts. The manufacturers, Hoffman-La Roche, are now adding a blue dye to the pill that will be visible if added to a beverage<sup>14</sup>.

Similar to other benzodiazepines, flunitrazepam is a GABA agonist. As such, it mediates inhibitory neurotransmission in the brain and spinal cord<sup>82</sup>. Benzodiazepines bind to the GABA receptor, opening the chloride channels of neurons and resulting in an influx of chloride and hyperpolarization of the cell. This decreases the excitability of the cell producing sedation, anticonvulsant activity, and anxiety reduction.

Clinical effects of flunitrazepam are similar to benzodiazepines<sup>16</sup> (Table I). The effects are much greater with the concurrent ingestion of alcohol or other sedating drugs<sup>16</sup>. Rohypnol has been suggested to have a high abuse liability, higher, some say, than other benzodiazepines<sup>83</sup>. Like other benzodiazepines, chronic use of Rohypnol can produce dependence. The withdrawal syndrome includes headache, tension, anxiety, restlessness, muscle pain, photosensitivity, numbness and tingling of the extremities, and increased seizure potential<sup>84</sup>.

Overdoses can lead to loss of consciousness and respiratory depression that can be life threatening<sup>85</sup>. Supportive measures include use of activated charcoal to absorb drug in the gastrointestinal tract as well as respiratory support<sup>86</sup>. Flumazenil is a specific

benzodiazepine antagonist that may be administered to reverse the effects of flunitrazepam toxicity<sup>85</sup>.

While a standard component of most urine drug screens is testing for benzodiazepines, flunitrazepam is administered in such small amounts and distributed so rapidly that detection methods commonly fail. Typical toxicological tests can only detect flunitrazepam in blood and urine for up to 72 h after ingestion due to quick metabolism and elimination<sup>5</sup>.

### Club drugs: Indian scenario

Indian data on club drugs are very limited with little efforts being made to gather systematic data regarding the same. The first nationwide survey to obtain information on extent, pattern and magnitude of substance abuse in the country indicated new emerging trend of substance use in India with amphetamine like substances (ATS) are being more used in regions like Goa and Ahmedabad<sup>87</sup>. Most reports regarding club drugs are from newspaper articles; hence there is an urgent need for verified, authentic research data.

A recent assessment by United Nations Office on Drugs and Crime (UNODC)<sup>88</sup> has found that after substantial increases in the late 1990s, the use of synthetic drugs (*e.g.*, amphetamines and Ecstasy) in North America, Europe and Oceania has stabilized, albeit at high levels. But the problem has shifted to new markets, particularly in East and South-East Asia and the Middle East over the past few years. With technological advancement and particularly the information technology sector coming up in a big way in India (often as outsourcing for overseas-based multinational companies), suddenly there is a neo-rich young generation. This is often coupled with the need to escape temporarily from the severe work pressure and social isolation created by this lifestyle. With drug licensing and controlling authorities focusing more on licit and traditional illicit drugs (*e.g.*, opioid, cannabis), club drugs have caught the fancy of this neo-rich young generation. Table II lists the various factors/reasons behind the significance of this new and emerging phenomenon in the Indian drugs scenario and why we should be concerned.

The rave parties of Goa are said to be started by the Hippies<sup>89</sup>. Earlier Rave parties meant loud music, alcohol and cannabis abuse. Since the late eighties, psychedelic culture in the northern village of Anjuna became increasingly concentrated on free outdoor parties with a particular subgenre of electronic dance music, which by 1994 was known as Goa trance

**Table II.** Significance of club drugs in the emerging Indian drug abuse scenario

1. These are often associated with status symbol amongst the neo-rich youth
2. These are often perceived to be safe or benign compared to the "hard drugs" such as heroin, cocaine, *etc.*
3. Some of these drugs (*e.g.*, GHB, Rohypnol) are notorious as "rape drugs" and their use is associated with crimes like date-rape, party rape, robbery, *etc.*
4. Unlike cannabis, opium or heroin, these are usually imported or smuggled from neighbouring countries
5. Some of these (*e.g.*, MDMA) are directly neurotoxic in short and long term use
6. Contrary to their popular perception, all these drugs can have substantial toxic effects on several systems of the body and can be potentially lethal
7. These are difficult to detect through routine toxicological screening (GHB is an exception)
8. These are notorious for spreading sexually transmitted diseases (STDs) and HIV infection because of their intravenous route of administration
9. These are devoid of any antidote (flunitrazepam is an exception)
10. Management needs to be started early and can be difficult

and later became much darker, more minimal and aggressive, called psy-trance<sup>89</sup> Rave parties in Goa happen in every tourist season (November to May) which are attended mainly by foreigners from the UK, Israel, Germany, France and Japan<sup>90</sup>. The bars organizing such parties sell Ecstasy or LSD<sup>91</sup>. In last few years upper-class Indians have massively taken to Ecstasy and clubbing and there are more women amongst them<sup>89</sup>. Later on, with government interventions and regulatory norms, drug abuse came down as these were declared illegal by law. Goa police recently admitted about the unorganised and the organised channels for ketamine in Goa<sup>92</sup>. The CK1 pill is one of the trendy party drugs manufactured locally in Goa. The pill is a combination of cocaine and the anaesthetic ketamine. CK1, also known by its street names Blizzard and Calvin Klein, is easily available in the north Goa beach belt.

In the north, Himachal Pradesh's Kullu valley is now known for its full-moon-night jungle rave parties. A large number are Israelis, most of them fresh from the frazzle of military service dance to psychedelic music on full moon nights and smoking hashish<sup>91</sup>.

Bangalore is baptized the Silicon Valley of India and has turned into a rave hotspot<sup>93</sup>. The spot lights

in the clubs create an atmosphere between cosy and disco, everybody drinks, most smoke and a few take psychedelic drugs<sup>93</sup>. Most raves in the city do get busted by the police<sup>94</sup>.

In Pune, 280 people were arrested during a pre-dawn raid on a rave party in March 2007. The ravers were allegedly using California drops<sup>95</sup>. A California drop is acid that is put on a stamp, which is then chewed; the cost of each drop is put between INR 350 and 500. Like the Irish woman suspected of supplying drugs at the Pune party, foreigners have been arrested for peddling illegal LSD, Ecstasy and cocaine at various rave parties in Bangalore and Mumbai<sup>90</sup>.

Sometimes police directly raid rave parties in plain clothes and catch ravers red-handed. People who are found in the Rave Party are often booked under Section 27 of the Narcotic Drugs and Psychotropics Substances Act of 1985<sup>96</sup> and Section 294 of the Indian Penal Code<sup>97</sup>.

### Management of club drugs abuse

Because club drugs are illicitly obtained and often are adulterated or substituted, these are usually known as unknown substances. In the ever-changing world of illegal drug distribution, Internet Web sites can be helpful in identifying the rapidly changing appearances of these substances<sup>16</sup>. Urine and serum toxicology screens may not be able to detect club drugs. For example, urine screening does not detect MDMA, though it does detect its metabolite, MDA. Ravers often present with concurrent ingestion of drugs with different pharmacological profile, which may include stimulant and depressant drugs. Therapist should always make an attempt to gather information from as many sources as possible regarding what was ingested and in what form.

No standard treatment protocol has been identified for club drug overdose. Basic management should include cardiac monitoring, pulse oximetry, urinalysis, and performance of a comprehensive chemistry panel to check for electrolyte imbalance, renal toxicity, and possible underlying disorders<sup>16</sup>. Every effort should be made to control seizures. Gastrointestinal decontamination with activated charcoal and a cathartic may be useful in acute exposures if the drug was taken orally within the previous 60 min. Severe hypertension can be treated with labetalol, phentolamine, nitroprusside, or similar agents. Hyperthermia should be treated immediately with tepid water bathing and fanning. The serotonin antagonists chlorpromazine and cyproheptadine appear to be effective in mild to

**Table III.** Harm reduction strategies for raves (adapted from Weir, 2000)<sup>2</sup>

- 
- Replenish fluids and sodium (500 ml/h if dancing, 250 ml/h if inactive)
  - Take breaks from dancing
  - Know the risks of adulterated drugs and the inaccuracies of logos
  - Know the signs and symptoms of toxicity
  - Avoid alcohol
  - Ensure medical centre and team is on-site
  - Do not attend a rave alone; contract with a friend to look out for each other
- 

moderate cases of serotonin syndrome<sup>98</sup>. There are no specific antidotes for ingestion of club drugs, except for Rohypnol, which has already been mentioned. In view of the above, an alternative is to encourage harm reduction strategies (Table III) by ensuring that buildings meet safety and health standards, adequate security is provided to accommodate the large number of attendees and ravers are educated about health effects by trained volunteers. NIDA website ([www.clubdrugs.org](http://www.clubdrugs.org))<sup>99</sup> also provides useful information regarding club drugs. UNODC's Global Synthetics Monitoring: Analyses, Reporting and Trends (SMART) Programme aims to provide quality information on ATS, such as patterns of trafficking and use, and will provide the international community with the evidence needed to take more targeted action in areas of weakness<sup>88</sup>.

### Conclusions

Club drugs are a menace to the society. Their use, other than for strictly medical or approved research purposes, should be prohibited through legislation and awareness generation. Even though the "club drug" phenomenon was identified early, scientific information about these drugs, their identification, and short- and long-term effects are still evolving. The lack of research-based information on the adverse effects of these drugs has led to the emergence of a range of websites that may or may not provide accurate information. India has a huge teenage population which is being targeted by foreign drug peddlers to flourish their business. Club drugs continue to be modified and evolve, making them very difficult to monitor. It is useful to know what drugs are being used in the community. Information can be gleaned from teens themselves at both routine and emergency visits, from local substance abuse programmes, and from the police. Only collective effort can stop this menace from engulfing the society. All health professionals should remain well informed regarding club drugs and their management protocol.



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