

Recreational drugs in India

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ABSTRACT

Substance use has been present in India since many millennia, and the type and pattern of substances being abused have seen changes over time. In the review, we look at the traditional recreational substance and then describe the newer and emerging recreational drugs in India.

KEYWORDS: Addiction, alcohol, India, opioids, recreational drugs

Introduction

India is the second most populous nation in the world. The sheer number of individuals residing in the country makes it home to one of the largest substances using population. Substance use is often influenced by local factors which affect the demand, supply, and actual use of a set of substances.^[1,2] The locally relevant factors vary across geographical regions, periods of time and population strata. India lies in South Asia bounded by Pakistan in the North and Myanmar in the East. The geographical location has forced India to become the transit point of many illegal recreational agents to the world. The traditional substances such as cannabis, alcohol, and opium have given way to newer drugs such as heroin, cocaine, methylenedioxymethylamphetamine (MDMA), and synthetic psychoactive substances. Their use has been increasing at an alarming rate among the youth in India.

Table 1 summarises the prevalence of recreational drugs in different ages and Table 2 highlights the states consuming the common intoxicants.

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Traditional Agents

Alcohol

Use of alcohol has been in vogue in India since ancient times.^[3] Alcohol has been described as the drink of the gods (“soma”) in the Vedas. The *puranas* as well as epics (Mahabharata and Ramayana) have references to use of alcohol. Historical texts also denounce the heavy use of alcohol and drunkenness. The ancient Indian society was aware of the hazards of alcoholism. Indian Physician Charaka (about 300 B.C.) remarks moderate use of alcohol being healthy and recommends treatment for heavy users of alcohol.^[3]

This changed with the arrival of Mughals. The sale of alcohol was decreed, however, use of alcohol started to gain momentum again with the rise of British Empire in India. After independence, directive principles of state policy mandated government initiated efforts to restrict alcohol consumption. Recent trends indicate increasing use of alcohol in India,^[4] attributed to conglomeration and expansion of the alcohol industry, covert marketing strategies, and “westernization” of the Indian society.

Ethanol (CH₃CH₂OH) is a water-soluble compound that rapidly crosses cell membranes, resulting in ready equilibration between intra- and extra-cellular concentrations.^[5] Its absorption occurs mainly in the proximal intestinal tract, namely, in the stomach (70%) and the duodenum (25%) while only a small percentage occurs in the remaining intestinal tracts.^[5] 90% of

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ingested ethanol is metabolized in acetaldehyde along three liver enzymatic pathways:

1. Liver alcohol dehydrogenase (90%),
2. Microsomal ethanol oxidizing system (8-10%), and,
3. Catalase (0-2%).^[6]

Diagnosis of acute alcohol intoxication (Diagnostic and Statistical Manual of Mental Disorders IV edition) is based on:

- Recent alcohol ingestion;
- Clinically significant maladaptive behavioral or psychological changes developing during or shortly after alcohol ingestion and including inappropriate sexual or aggressive behavior, unstable mood, impaired judgment, and impaired social or occupational functioning; and
- One or more of the signs that develop during or shortly after alcohol use:
 1. Slurred speech;
 2. Lack of coordination;
 3. Unsteady gait;
 4. Nystagmus;
 5. Impairment of attention or memory;
 6. Stupor or coma.

Several factors can influence the extent of acute alcohol intoxication; besides the amount of alcohol ingested, individual body weight and tolerance to alcohol, the percentage of alcohol in the beverage, and the period of alcohol ingestion seem to be particularly important.^[7]

Symptoms are usually related to the blood alcohol concentration [Table 3]. In alcohol-dependent patients who develop tolerance to alcohol as a result of repeated exposure to ethanol, these effects may become reduced.^[8]

Acute alcohol intoxication is able to cause several metabolic alterations, including hypoglycemia, lactic acidosis, hypokalemia, hypomagnesemia, hypoalbuminemia, hypocalcemia, and hypophosphatemia.^[5] Cardiovascular effects include tachycardia, peripheral vasodilation, and volume depletion. Severe intoxication can lead to atrial or ventricular tachyarrhythmias and new-onset atrial fibrillation after acute alcohol ingestion (holiday heart syndrome).^[9,10] Life-threatening respiratory consequence is respiratory depression. Gastrointestinal effects include nausea, vomiting, diarrhea, abdominal pain secondary to gastritis, peptic ulcer, or pancreatitis.^[11,12] Vomiting can lead to hyponatremia.^[13] Symptoms usually include nausea, vomiting, and abdominal pain.

Table 1: Prevalence of substance use among Indian males aged 12-60

Substances	Ever use (life time) (in %)	Current use (last 1-month) (in %)	Dependent user as per ICD-10 criteria (in %)
Tobacco	57.9	55.8	—
Alcohol	25.9	21.4	3.60
Cannabis	4.1	3	0.77
Opiates	1.0	0.7	0.15
Sedative and hypnotics	0.1	0.1	—

Table 2: States with high use of substances in India

Alcohol	NHS: Himachal Pradesh, Haryana, Arunachal Pradesh, Manipur, Nagaland DAMS: Uttar Pradesh, Maharashtra, Kerala
Opioids	NHS: Punjab, Rajasthan, Haryana, Himachal Pradesh, Nagaland, Mizoram DAMS: Punjab, Rajasthan, Uttar Pradesh, West Bengal
Cannabis	NHS: Manipur, Bihar DAMS: Uttar Pradesh, Bihar

NHS = According to national household survey, DAMS = Drug abuse monitoring system

Table 3: Main clinical symptoms in acute alcohol intoxication according to BAC

Symptoms	BAC
Impairment in some tasks requiring skill	BAC <50 mg/dL (10.9 mmol/L)
Increase in talkativeness	
Relaxation	
Altered perception of the environment	BAC >100 mg/dL (21.7 mmol/L)
Ataxia	
Hyper-reflexia	
Impaired judgment	
Lack of coordination	
Mood, personality, and behavioral changes, nystagmus	
Prolonged reaction time	
Slurred speech	
Amnesia	BAC >200 mg/dL (43.4 mmol/L)
Diplopia	
Dysarthria	
Hypothermia	
Nausea	
Vomiting	
Respiratory depression	BAC >400 mg/dL (86.8 mmol/L)
Coma	
Death	

BAC = Blood alcohol concentration

Diagnosis is based on history, including the quantity and the type consumed the time course of the symptoms, the circumstances, and injuries. Physical examination must include vital signs as, hydration, and signs of chronic alcoholism-related liver cell dysfunction (capillary prominence, spider naevi, telangiectasias, palmar erythema, and muscular atrophy).^[14] Alcohol levels can also be determined by breath analysis^[15] or with a saliva dipstick, although these methods are less reliable.^[16] Levels of free ethanol in blood and ethanol conjugates in urine can be measured.^[17]

Many times, acute ingestion young adults are accompanied by concomitant recreational drug use. This possibility should be considered and ruled out in an intoxicated adult presenting in the ER.

The management is aimed at stabilizing the patient. Airway assessment and patency is paramount. Respiratory function should be carefully assessed, and one should be vigilant for respiratory depression and airway obstruction. Placement of the patient in a lateral position prevents aspiration. Intravenous (IV) fluid should be administered to hydrate the patient. Hypoglycemia and correction of electrolyte imbalances are the priority. IV solution containing dextrose, folate, thiamine, and multivitamins is used (1 l of 5% dextrose and 0.45% sodium chloride, 2 g of magnesium sulfate, 1 mg of folate, and 100 mg of thiamine).^[5] In agitated and violent patients, sedatives can be used. Droperidol or haloperidol is preferred. Use of excessive sedation could lead to respiratory depression and hypotension. Physical restraints should be avoided. Specific drug for the treatment of acute alcohol intoxication is metadoxine (pyridoxol 1-2-pyrrolidone-5-carboxylate). Metadoxine appears to accelerate ethanol metabolism^[18] due to by increasing acetaldehyde dehydrogenase activity,^[19] ethanol and acetaldehyde plasma clearance and urinary elimination of ketones.^[20] In a recent study, single IV injection of metadoxine (900 mg IV) significantly decreased the half-life of ethanol in the blood and showed a faster rate of ethanol elimination. It is not yet available in India.

Cannabis

Cannabis use has been prevalent in India since a long time, and ancient Indian texts describe medicinal use of cannabis.^[21] The indigenous species *Cannabis indica* grows freely in the cool hilly areas of lower Himalayas. Traditionally, use of cannabis has been socially sanctified on certain Hindu festivals (*Holi* and *Shivratri*). Consumption of cannabis has been prevalent in the form of oral intake of leaves (*bhanga*) and smoked forms (*charas* and *ganja*). Controlled sale of cannabis through legal outlets was allowed in parts of northern India. However, after India became a signatory to the Single Convention on Narcotic Drugs, move toward declaring cannabis illegal was made. Cannabis was later classified as illicit substance and effectuated penalties for sale, purchase and possession of cannabis. Despite the ban, occasional nonrecreational cannabis still remains to be prevalent, especially among the younger population.^[22]

Marijuana (*charas* and *ganja*) comes from leaves, stems, and dried flower buds of the cannabis plant. Bhang comes from the crushed fresh leaves. Hashish is a resin obtained from flowering buds of the hemp plant.^[23]

Majority of cannabis users in India start abusing cannabis in their adolescence, most quit but some develop dependence.^[24,25] Cannabis abuse among younger subjects is associated with poor academic performance, increased school dropout, psychosis, violence, aggression, sexual encounters, accidents, and crime.^[26-28] Long-term cannabis abuse has been associated with conduct disorders, attention-deficit hyperactivity disorder, and learning disorders.^[29]

The primary psychoactive constituent of cannabis, delta 9-tetrahydrocannabinol (Δ^9 -THC). Δ^9 -THC acts on several intracellular targets including opioid and benzodiazepine receptors, prostaglandin synthetic pathway, protein and nucleic acid metabolism.^[30,31] Cannabinoids (CBs) exert various physiological effects by interacting with specific CB receptors present in the brain and periphery.^[32] THC is rapidly absorbed through lungs after inhalation. It quickly reaches high concentration in blood^[33] maximum Δ^9 -THC plasma concentration was observed approximately 8 min after onset of smoking while 11-OH-THC peaked at 15 min and THC-COOH at 81 min. This Δ^9 -THC concentration rapidly decreases to 1-4 ng/mL within 3-4 h.^[34]

In comparison to smoking and inhalation, after oral ingestion, systemic absorption is relatively slow resulting in maximum Δ^9 -THC plasma concentration within 1-2 h which could be delayed by few hours in certain cases.^[35,36]

Behavioral effects of cannabis include feeling of euphoria, relaxation, altered time perception, lack of concentration, and impaired learning.^[37-39] Memory and mood changes are dominant. Panic and paranoid reactions have been reported. Physiological effects include rapid changes in heart rate and diastolic blood pressure, conjunctival suffusion, dry mouth and throat, increased appetite, vasodilatation and decreased respiratory rate.^[40,41] Cannabis also affects the immune and endocrine system; and its abuse is associated with lung damage and electroencephalography alterations.^[38,42-44] Cannabis appears to affect the same reward systems as alcohol, cocaine, and opioids,^[43] therefore potential for dependence.

No fatal overdoses with cannabis use have been reported.^[45,46]

Marijuana overdose treatment is usually symptomatic. Drugs can be used to reduce heart rate and curb any panic attacks.^[45] Since overdose can be mixed drug overdose, there could be serious and even fatal consequences. Efforts should be made to identify all substances and manage accordingly.

Opium

Opium was apparently introduced in India by the Arabs around the 9th century A.D. Thereafter, use of opioids gradually spread over northern India, especially among the rich and the powerful. Use of opioids in the form of raw opium (*afeem*) and poppy husk (*doda*) became acceptable as it relieved pain, caused relaxation and euphoria.^[47] Opium use was also popular amongst women and children but in limited amounts. Farmers used opium during the harvest season to alleviate fatigue and enhance productivity. A large proportion of opiate addicts still continue to use opium in limited quantities as a way of life. Although still prevalent in Northern India raw opium use has been largely replaced by newer illicit opioids.

Opium is the dried latex obtained from the opium poppy (*Papaver somniferum*). It contains approximately 12% of the analgesic alkaloid morphine, which is processed chemically to produce heroin and other synthetic opioids.^[48] The latex also contains the closely related opiates codeine and thebaine.

Morphine is the most prevalent and important alkaloid in opium. It binds to and activates mu opioid receptor in the brain, spinal cord, stomach, and intestine.^[49] Morphine has effects like opium but is responsible for most of its harmful effects such as lung edema, respiratory difficulties, coma, or cardiac or respiratory collapse. Regular use can lead to drug tolerance or physical dependence.^[48]

Heroin (diacetylmorphine) is derived from the morphine and is 2-3 times more potent. Heroin causes euphoria, reduces anxiety and has central nervous analgesic properties. It is a highly addictive drug. Pure heroin is a white powder with a bitter taste. Illicit heroin is available as brownish powder and is usually “mixed/cut” with other drugs or substances. Since it is difficult to know the actual strength of the drug, abusers are at risk of overdose or death. Heroin is more often injected, however, it may also be smoked, sniffed, or orally ingested.^[48]

Effects of morphine/heroin

There is a feeling of euphoria (the “rush” with injection of heroin) accompanied by a warm flushing of the skin, a dry mouth, and heavy extremities. Following this, there could be a wakeful and drowsy state.^[50] Mental functioning becomes clouded. There could be nausea, pinpoint pupils, and slow shallow respiration. Overdose may result in severe respiratory depression, hypotension, muscle spasms, convulsions, coma, and death. IV heroin use is complicated by other issues such as the sharing of contaminated needles, the spread of HIV/AIDS, hepatitis, and toxic reactions to heroin impurities.

Heroin overdose is a medical emergency that requires treatment with naloxone. IV naloxone reverses the respiratory depression within 2 min.^[49] Usual dose is 0.4 mg IV or IM and can be repeated till a maximum of 2 mg. Repeated doses may be required as naloxone is shorter acting agent (30-120 min) as compared to the opioid. Supportive therapy with respiratory support (oxygen/ventilation), IV fluids, and other adjunctive medications may be required.^[50,51]

For chronic use/addiction, oral methadone, buprenorphine, and naltrexone (intramuscular depot injection) are treatment options.

Table 4 summarizes the common toxidrome observed with recreational drugs and Table 5

Table 4: Common toxidrome observed with recreational drugs

Sympathomimetic toxidrome
Drugs causing this syndrome: Amphetamines; Cocaine; MDMA
CNS excitation: Euphoria, hallucinations, agitation, psychosis, delirium, seizures
Neuromuscular excitation: Hyper-reflexes, tremor
Autonomic effects: Hyperthermia, diaphoresis, flushing, mydriasis
Cardiovascular effects: Tachycardia, hypertension, myocardial depression, arrhythmias
Gastrointestinal effects: Nausea, vomiting, diarrhea
Opiate toxidrome
Drugs causing: Opium, opiates and derivatives
CNS depression: Drowsiness to coma
Respiratory depression, hypercapnia
Psychological disturbance
Depersonalization, disorientation, decreased inhibition, altered mood, memory impairment, lack of attention
Sedative-hypnotic toxidrome
Drugs causing this: Benzodiazepines, barbiturates, ethanol
CNS: Depressed, reflexes are normal or depressed
Respiratory rate: Decreased or even respiratory arrest
Heart rate and blood pressure: Usually normal
Toxic autonomic syndromes
Drugs causing: GHB, methadone
CNS: Agitated, dilated pupils
Vitals: Hyperthermia, tachycardia, dry flushed skin

highlights the pharmacology and symptomatology associated with commonly used recreational drugs. Table 6 gives the management of various recreational drugs.

Newer recreational agents

The last half of the century has seen the emergence of newer substances of abuse in India. The use of traditional forms of opioids has been supplanted by use of newer opioids. On the other hand, club drugs,

volatile solvents, and stimulants have made significant inroads among select population groups. The newer and emerging drugs of abuse in India are discussed below.

Newer opioids

The use of raw opium was supplanted by use of other substances in last few decades. Use of heroin expanded after 1970s due to a better “kick” than opium, could be injected for a “rush” and was less bulky than opium, and opium husk so was easier to

Table 5: Clinical profile and pharmacology of recreational drugs

	MDMA	GHB	Ketamine	Rohypnol
Manner of use	Tablet/Capsule; ingested orally, can be crushed/ snorted/ dissolved/injected	Liquid; often mixed with alcohol – effects amplified	Liquid – injected, added to items to be smoked. Powder – dissolved in drinks, smoked, snorted or dissolved and injected	Tablet; typically ingested orally; crushed and snorted
Onset of action; T _{1/2}	30 to 60 min 5.8 ± 2.2 h	15-30 min after an oral dose; 22-28 min	Variable; elimination t _{1/2} 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.	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.	Low dose: relaxation (K-land); Higher dose: dream-like state, hallucinations, visual distortions, sensation of near-death experiences (K-land); 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.
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.	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.	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.	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

Adapted from Chakraborty K, Neogi R, Basu D. Club drugs: Review of the 'rave' with a note of concern for the Indian scenario. *Indian J Med Res* 2011;133:594-604.

Table 6: Management of suspected recreational drug ingestion**Sympathomimetic toxidrome**

Record vital signs and temperature.
 Keep airway patent, oxygenate and check oxygen saturation/arterial blood gases.
 Establish vascular access.
 Collect blood samples for serum chemistry.
 Get electrocardiogram, check QTc.
 Collect blood and urine samples for drug toxicology screen.
 Check urine sodium, osmolality, myoglobinuria.
 Collect blood for creatine kinase.
 Neuro-imaging if needed (GCS <8).
 Consider “coma cocktail” 50 mL of dextrose D50W, thiamine 100 mg IV, and naloxone IV.
 Avoid flumazenil if mixed dose suspected.
 Use activated charcoal carefully only after airway is secure.
 Pharmacological restraint with short-acting benzodiazepines.
 Drug abuse-related hypertension, tachycardia to be treated with benzodiazepines.
 Beta-blockers should be avoided in these patients.

transport. Steady supply of heroin from regions like Golden Crescent (Afghanistan and Central Asia), and Golden Triangle (South East Asia) has led to gradually greater use of heroin in India. This has resulted in increased injectable drug use, which was an uncommon problem previously.

There has been the emergence of prescription opioid misuse.^[52,53] Pentazocine, buprenorphine, morphine, fentanyl, and tramadol are the injectable forms; while dextropropoxyphene, tramadol, diphenoxylate, buprenorphine, oxycodone, hydrocodone bitartrate, and codeine are the oral forms of prescription opioids being abused. Adolescents represent an important vulnerable group for development of prescription opioid misuse.^[54]

Opioids and morphine derivatives are very popular as they can relieve pain and help a person feel better after hard days job, especially in the fields. They also cause feelings of euphoria, and relaxation but can lead to drowsiness, confusion, nausea, and respiratory complications.

Stimulants

These stimulants speed up the body’s nervous system and create a feeling of energy. They are also called “uppers” because of their ability to make you feel very awake.^[55] Stimulants have the opposite effect of depressants.

- Oral/tablet forms.
- Cocaine.
- Methamphetamines.
- Amphetamines.
- Inhalants.
- Glues.

- Paint thinner.
- Gasoline.
- Aerosol sprays.

Cocaine

Cocaine is the prototype stimulant. It increases alertness, feelings of well-being and euphoria, energy and motor activity, feelings of competence and sexuality.^[56] Common side effects are anxiety, fever, paranoia, restlessness, and tooth grinding.^[56] With prolonged use, it can cause itching, tachycardia, hallucinations, and paranoid delusions. If taken as an overdose, it can result in tachycardia, tremors, convulsions, hyperthermia, myocardial infarction, stroke, and heart failure.^[56,57]

Chronic use results in depression with suicidal ideation. Irreversible neurocognitive dysfunction can result with chronic abuse. Chronic intranasal usage (snorting) can degrade the nasal septal cartilage.^[57] Cocaine withdrawal includes vivid and unpleasant dreams, insomnia or hypersomnia, anger, increased appetite, and psychomotor retardation or agitation.^[57]

Overdose can result in tachyarrhythmias and life-threatening hypertension. Death could result from respiratory failure, stroke, cerebral hemorrhage, or heart-failure. Cocaine-induced hyperthermia may cause muscle cell destruction and myoglobinuria resulting in renal failure.^[56] Emergency treatment consists of reducing the heart rate and blood pressure by administering a benzodiazepine sedation agent, such as diazepam. Physical cooling (ice, cold packs) and paracetamol may be used to treat hyperthermia. Supportive therapy in the form of hydration and oxygen/ventilation may be required.^[56]

Amphetamine and similar agents

In recent times, amphetamine-type stimulants (ATS) are also making gradual inroads into India. The numbers of young injecting drug users who are using ATS has been observed to be rising.^[58] Use of ATS is associated with the use of chased heroin, and other high-risk behaviors. Stimulant drugs increase the release of excitatory neurotransmitters and thus produce a higher level of wakefulness and a more radically altered mood. That is why these stimulant drugs are sometimes known as “speed”.^[59] When the effects of a stimulant wear off, the user is typically left with feelings of sickness and a loss of energy. These drugs have a strong addictive potential, and constant

use can result in addictions and severe neurological/cognitive dysfunction on the user.

Methadone is a synthetic stimulant drug (ATS class). It has similar stimulant effects but also produces side effects like teeth grinding (commonest). It is abused widely in UK^[60] similar to amphetamine, MDMA and cocaine it causes euphoria, stimulation, elevated mood, decreased hostility, improved mental function, and mild sexual stimulation. Side effects include visual problems due to dilated pupils,^[61] poor concentration, teeth grinding, amnesia, hallucinations, delusions, and erratic behavior.^[59] Other side effects include raised body temperature, tachycardia, respiratory distress, loss of appetite, increased sweating, anxiety, paranoia and depression.^[59]

Inhalants

The last half century has also seen the gradual expansion in the usage of volatile solvents in India. The use of volatile solvents seems to be primarily restricted to younger age groups. Most users discontinue the use of volatile solvents with age. Street children are a vulnerable group of volatile solvent users,^[62] it is used to reduce hunger, decrease pain, and feel euphoric. Inhalants are sniffed or huffed and give the user immediate results. Unfortunately, these immediate results can also result in sudden mental damage. When inhalants are taken, the body becomes deprived of oxygen, causing a rapid heartbeat.^[62] Other effects include liver, lung, and kidney problems, affected sense of smell, difficulty walking and confusion. Prolonged use of inhalants has been associated with significant neurological dysfunction.^[62]

Depressants

Depressants usually slow down activity in the central nervous system. These drugs are also called “downers” because they slow the body down and seem to give feelings of relaxation. Depressant drugs, like alcohol and heroin, work in much the same way on mood and personality but activate inhibitory chemical messengers.^[52] Depressants are available as prescription drugs to relieve stress and anger, although drowsiness is often a side effect. The “relaxation” felt from these drugs is not a healthy feeling. Repeated use of such drugs over an extended period of time can cause the body to adjust the amount of naturally occurring inhibitory chemicals it produces.^[53] This leads to the phenomena of tolerance. More and more of the drug has to be taken in order to get the desired effect. The user ultimately ends up

developing physical drug dependence. These patients may become agitated and can develop respiratory arrest.

Alcohol is the prototype agent which has been consumed since ages as a relaxant but in high doses can cause significant depressant action. The other examples are.

Prescription drugs

- Barbiturates.
- Benzodiazepines.
- Flunitrazepam.
- Tranquillizers.

Illicit/street drugs

- Gamma-hydroxybutyrate (GHB).
- Methaqualone.
- Flunitrazepam.

Flunitrazepam (Rohypnol)

Is an intermediate-acting benzodiazepine very similar to diazepam.^[55] It is used for short-term treatment of insomnia, as a premedication and for inducing anesthesia. Its effects include sedation, muscle relaxation, antianxiety, and antiseizure properties. It can also cause partial amnesia. It is to be noted that the sedative effects are 10 times stronger than diazepam. Effects start in 15-20 min after administration and last for up to 4-6 h.^[55] Due to its abuse potential, it is not legally allowed to be used or possessed in many countries, including India. In India, it is popular amongst robbers, for robbing passengers in buses and trains after mixing with drinks/food. It can also be abused as “date rape drug.” To prevent its misuse, the standard drug now has a blue core instead of being white. This can help detect tampering with drinks.^[60] Its abuse is common amongst teenagers and students as it can cause profound intoxication, and modulate the effects of cocaine or heroin if co-ingested.^[60]

Abuse in the higher dose, repeatedly can result in drowsiness, dizziness, lack of motor control, in-coordination, slurred speech, confusion, and gastrointestinal disturbances. Due to longer half-life effect can last for up to 12 h or more. Higher doses can also cause respiratory depression.^[55,60] Chronic use can result in dependence and withdrawal syndrome on discontinuation.

It can be detected as benzodiazepine in routine tox screens but can be detected for up to 72 h, not beyond that. Since there could be partial or complete amnesia,

patient may not remember to have taken/given the drug and timing correctly.^[55] Treatment is supportive. Airway and respiration need to be supported while maintaining hydration. The patient may need to be kept in the hospital for up to 72 h.^[60] Chronic abuser should be offered detoxification.

Gamma-hydroxybutyrate/gamma-butyrolactone

It is commonly available as a clear liquid, white powder (dissolved in water), tablet, or capsule. Common recreational drug in the west and is usually co-ingested with alcohol or other recreational agents. Since it is tasteless and odorless it can be mixed in food or drinks and is also notorious as “date rape drug.” GHB is structurally related to gamma-aminobutyric acid. It inhibits dopamine release and activates tyrosine hydroxylase, to increase dopamine levels in the brain. It causes Euphoria, increased sex drive, and tranquility in the user.^[63,64]

Adverse effects related to GHB ingestion is sweating, loss of consciousness, nausea, hallucinations and amnesia.^[65] Overdose patients commonly present in the emergency with sedation, seizures, coma, severe respiratory depression and death.

Routine use can lead to addiction and stoppage may result in severe withdrawal manifesting with insomnia, anxiety, tremors, and sweating.

It can be easily detected by routine urine toxicology screens but due to a short half-life may become undetectable in 12 h after ingestion.

Treatment of overdose is largely supportive. Since co-ingestion of opiate is common, “coma cocktail” may precipitate withdrawal in chronic users.^[60]

For chronic users with withdrawal symptoms, benzodiazepines, antihypertensive medications, and/or anticonvulsants should be used under strict medical supervision.^[65,66] Baclofen is also used for treatment for GHB withdrawal but is not Food and Drug Administration approved.^[66]

Hallucinogens

Hallucinogenic drugs, like *lysergic acid diethylamide* (LSD) and certain “magic” mushrooms, affect those areas of the brain which control sensory perception and thought patterns. They do this by altering the way in which the messages are received and interpreted. The change in mood or personality brought about by

hallucinogenic drugs is more likely to be influenced by the set and setting of the drug use than the purely pharmacological action of the drugs themselves within the central nervous system. After taking hallucinogens, switching emotions of is a frequent effect. Hallucinogens affect the body’s self-control, such as speech and movement, and often bring about hostility. Other negative side effects of these drugs include increased heart rate, higher blood pressure, cardiac dysfunction and hormonal changes.

Types of drugs include

- LSD.
- Mescaline.
- Psilocybin.
- Cannabis.
- Magic mushrooms.

Lysergic acid diethylamide

It is one of the most powerful hallucinogens available. It is made from a fungus called ergot. LSD is available as a liquid or tablet and is taken orally.^[67] It produces hallucinations that may be pleasant or unpleasant. Overdose symptoms usually include changes in mood, thought and perception.^[67] Symptoms after ingestion can include true hallucinations or pseudo hallucinations (illusions resulting from the misinterpretation of actual experiences), synesthesia (which create a sensory crossover experience), unpredictable emotions, panic reactions, psychoses, extreme depression, despair, intense fear, and terrifying thoughts. The effect usually lasts for hours.^[67]

In a case with severe overdose, vomiting, and collapse can occur with signs of sympathetic overactivity like hyperthermia, eventually leading to coma and respiratory arrest. Patients may develop rhabdomyolysis and renal failure if physically restrained.^[67] Treatment of an acute overdose is symptomatic. It is aimed at maintaining vitals and hydration. Chronic users may develop tolerance, requiring higher doses to achieve a high.^[67]

Psilocybin/magic mushrooms

These are known as psychedelic mushrooms as they contain psychedelic drugs. Biological genera containing psilocybin include *Copelandia*, *Galerina*, *Gymnopilus*, *Inocybe*, *Mycena*, *Panaeolus*, *Pholiotina*, *Pluteus*, and *Psilocybe*.^[68] These are commonly used or abused for their psychedelic effects since ages and are still popular. However, it is pertinent to note that a normal mushroom injected with psychedelic drugs/

agents can be sold to an unsuspecting naïve customer as magic mushroom.

Psilocybin is broken down after ingestion to psilocin, which is responsible for the psychedelic effects.^[68] Concentration of Psilocybin varies in various species, making it unpredictable to predict the effect. It difficult to abuse magic mushrooms as they are often taken within a short period.^[69] Magic mushrooms are not known to cause physical or psychological dependence.^[70] since the concentration can vary effects last for variable time, from 2 to 3 h till 6-8 h, or they may be perceived to last longer due to mind alteration.^[71]

Changes in audio, visual, and tactile senses are apparent within 30 min to an hour after ingestion. There is a change in visual perception like enhancement and contrasting of colors, auras or “halos” around light sources, increased visual acuity, changing shapes, colors, and trails behind moving objects. There could be synesthesia, clarity, and loudness of voice/sound and deep meaning of lyrics.^[72] like LSD, the trip is dependent on the time and situation of the user.

Most feared toxicity is from poisonous wild mushrooms, which can be mistaken as magic mushrooms and can lead to severe hepatotoxicity and even death.

Since there is no effective antidote, the toxicity is treated symptomatically.

Club drugs

Are the new range of drugs which seem to have a dual action. These are the stimulant psychedelics, of which ecstasy is the most well-known. They are the new threats and are being taken up by the new generation due to its effects and availability. Classic club drugs are amphetamines and cocaine, but the newer club drugs typically include substances like ecstasy, gamma-hydroxy-butyrate, ketamine, which are now being primarily used in rave parties.^[65] These drugs are used to prevent fatigue during long spells of dancing in raves (using amphetamines or cocaine) or for intensifying the partying experience (using ecstasy and ketamine). These drugs are generally not consumed on a regular basis but are typically taken only during social gathering. The use of these drugs is currently restricted to metropolitan cities, parts of Goa and some regions of Himachal Pradesh.^[73] The use seems to be gradually expanding in the affluent adolescent and young-adult population.

Ecstasy or methylenedioxyethylamphetamine

It belongs to a family of synthetic compounds related to the amphetamines. It has stimulant properties like amphetamine, but it also has certain effects in common with LSD. Produces a mixture of the stimulant and mild psychedelic effects.^[55,60] It is an indirect serotonin agonist, induces the serotonin release and blocks its uptake, leading to “serotonergic syndrome,” much the same way as LSD. Users feel happier and it increasing their feelings of empathy for others. Commonly ingested but can be injected/snorted too. It is difficult to detect on routine toxicological screens. Toxicity or overdose can manifest with autonomic dysfunction and agitation. Since there is no antidote, treatment is symptomatic.^[55,60]

Ketamine

It is a derivative of phencyclidine. Used as a dissociative anesthetic in emergency and surgery. Illicit *ketamine* is widely used in Western and some South Asian countries.^[74] It can be taken orally or injected but typically used as a nasal inhaler. A typical abuser can be recognized by observing traces of white powder residue in nares.

It is often used along with methamphetamine, cocaine and/or heroin. Its use as a recreational agent has gone up in dance parties and club raves.

It binds to the N-methyl-D-aspartate receptor site and blocks it. Its use results in altered perception, memory, and cognition. Commonly occurring side effects are tachycardia, palpitations, hypertension, and respiratory depression.^[74] Patients may also sustain grievous injuries due to pain insensitivity. Its use also has been linked to sexual assaults in females.

Frequent use can lead to dependence similar to cocaine or amphetamine dependence. Tolerance builds rapidly, and users become psychologically dependent on it.^[60] Withdrawal may occur after chronic use and usually presents as chills, sweating, excitation, hallucinations, watering of eyes, and cravings.^[74]

It is not detected in routine toxicology screens and may give a false positive result for Phencyclidine.^[60]

Overdose treatment is usually symptomatic. Airway patency and breathing need to be controlled. Benzodiazepines may be used to control seizures or agitated behavior.^[60]

Conclusion

Addiction in India contributes significantly to the global burden of substance use disorders. Though alcohol, cannabis, opioids, and tobacco are the most prominent substances of use in India, other substances are gradually emerging. The research on substance use disorder in India is constrained by the lack of trained manpower and funding. Though control measures have been instituted and implemented, further work needs to be done to effectively challenge the problem of addiction in India.

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