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Indian Psychiatric Society Publication



Recent Advances in Psychiatry

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CHAPTER 4

Designer Drugs

Omkar Nayak, Avinash De Sousa

INTRODUCTION

Designer drugs, also known as "Legal Highs" are substances having psychotropic effects that are marketed and distributed for recreational use. Designer drug use has increased significantly in the past decade, especially among the young adults leading to significant problems for some users. The incidence of designer drug problems in emergency departments, hospitals, and other medical settings is largely unknown as per literature. Only a small percentage of those who use designer drugs, usually come into contact with the healthcare system, even in developed countries like USA. Having knowledge of designer drugs can help the clinicians to recognize common adverse reactions and life-threatening consequences. This chapter provides an overview of the main mechanisms of action and adverse effects of currently available designer drugs.[1]

STIMULANTS

Stimulants such as amphetamine, 3,4-methylenedioxymethamphetamine (MDMA), and cocaine, are among the most popular drugs of abuse of misuse. Methylphenidate and dextroamphetamine are some other examples of stimulants. Stimulants cause modulation of monoaminergic neurotransmission by interacting with norepinephrine, serotonin, and dopamine transporters. They also interact with monoaminergic receptors and other targets. Different selectivity and potency at different transporters result in different pharmacological effects, different clinical potencies, and different abuse liabilities. The number of available designer stimulants is constantly increasing and their use can cause various physiological complications and disturbances in mood.

Amphetamines

In addition to traditional amphetamines that have been used both medically and recreationally, several amphetamine drugs without approved medical uses have become available. MDMA is the most popular amphetamines

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designer drug. It was a first synthesized by Merck in 1912 as precursor in new chemical pathway. In 1980s, it became popular as recreational drug under the street name "Ecstasy" which led to ban of MDMA in most countries soon afterward. Currently, it is a promising agent for treatment of post-traumatic stress disorder.[2]

Mechanism of Action

Most amphetamines are substrate-type monoamine releasers, having a potent effect on the norepinephrine transporters. In addition to it, many amphetamines predominantly act at dopamine transporter and serotonin transporter (DAT vs. SERT) resulting in greater reinforcing effects and higher abuse liability. Those amphetamines like MDMA which has a more action on SERT as compared to DAT have a comparatively lesser abuse liability. Amphetamine also acts as substrates at vesicular monoamine transporters and inhibits the monoamine oxidase (MAO). These drugs have also been reported to interact with various monoaminergic receptors including serotonergic and adrenergic receptors and trace-amine-associated-receptor-1 which negatively modulates monoaminergic neurotransmission.

Adverse Effects

For traditional amphetamines, mainly sympathomimetic adverse effects can be expected. These include dry mouth, anxiety, insomnia, headache, mydriasis, bruxism, hypertension, tachycardia, hyperthermia, chest pain, palpitations, anorexia, nausea, vomiting, and abdominal pain. Hyperthermia may lead to potentially severe adverse effects like disseminated intravascular coagulation, renal failure, and rhabdomyolysis. Hepatotoxicity is a potentially fatal adverse effect that has been associated with the use of amphetamines and MDMA is a designer drug that has been most frequently linked to liver injury. Cardiotoxicity is another potential complication of amphetamine use, largely attributable to sympathomimetic activation, additionally also to hyperthermia. Monoamine depletion and reactive species contribute to neurotoxicity of amphetamines. However, despite extensive research, the extent to which different amphetamines are neurotoxic remains largely unknown.

Cathinone and Pyrovalerone Derivatives

Cathinone designer drugs are derivatives of β-keto amphetamine cathinone, an alkaloid that is found in the leaves of the *Catha edulis* plant. The large scale recreational use of cathinone's is a relatively new phenomenon, although several compounds have been known for long time. Several synthetic cathinones have been investigated for their medicinal potential mostly

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as antidepressants or anorectic agents but only a few were ever marketed because of concerns about abuse. Pyrovalerone derivatives represent a subgroup of synthetic cathinones based on structure of pyrovalerone, which was developed in the 1960s as a treatment option for lethargy, fatigue and obesity. As a result of their initial misleading marketing as "bath salts", synthetic cathinone's are still often referred to by that term.[3]

Mechanism of Action

The psychoactive effects of these drugs are primarily medicated by interactions with monoamine transporters. They are partially of fully effective substrate-type releasers at one or several monoamine transporters, but some compounds such as pyrovalerone derivatives are transporter inhibitors. Mephedrone has additionally been shown to mediate monoamine release via organic cation transporter 3 (OCT 3) indicating that cathinones target both high affinity and low affinity high capacity transporters. Similarly, to amphetamines, cathinone drugs also interact with several adrenergic and serotonergic receptors. Compared to amphetamines, however, cathinone designer drugs have been shown to interact less potently with trace-amine-associated-receptor-1 (TAAR-1) and vesicular monoamine transporter 2 (VMAT-2). These less potent interactions at TAAR-1 may result in higher risk of cathinone dependence compared with amphetamines.

Adverse Effects

The adverse effects include mainly sympathomimetic toxicity which manifests as agitation, tachycardia, hypertension, lower levels of consciousness, hallucinations, hyponatremia, chest pain, palpitations, and nausea. Rarely, severe adverse effects such as seizures, significant peripheral organ damage, and rhabdomyolysis have been reported. Numerous cathinone-related fatalities have been reported in literature.

Benzofuran and Indole Derivatives

Benzofuran and indole derivatives are analogs of MDMA and its metabolite 3,4-methylenedioxyamphetamine (MDA).[4]

Mechanism of Action

They cause norepinephrine uptake inhibition. They also have moderate to high selectivity in inhibiting 5-hydroxytryptamine (5-HT) vs. dopamine uptake, often with substrate activity at the transporters. Furthermore, affinity at adrenergic, serotonergic, and histaminergic receptors, and partial agonist at 5-HT2B receptors have been reported for these designer drugs.

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Adverse Effects

They may cause side effects like agitation, insomnia headache, drowsiness, dry mouth, dry eyes, bruxism, hyperthermia, tachycardia, palpitations, nausea diarrhea, hot flashes, and clonus of hands and feet. They can also cause psychological symptoms including visual and auditory hallucinations, depression, anxiety, panic attacks, paranoia, and psychosis.

Aminoindanes

Aminoindanes are conformationally restricted analogs of amphetamines that were originally investigated as bronchodilators, analgesic, and anti-Parkinsonian agents and subsequently as dugs with psychotherapeutic value. The desired psychoactive effects of aminoindane designer drugs include euphoria, mild distortion of vision, time and space, a greater intensity of perceptions and colors, empathy, and arousal.[5]

Mechanism of Action

They are monoamine transporter substrates with relevant affinity for adrenergic, dopaminergic, and serotonergic receptors.

Adverse Effects

Self-reported adverse effects of aminoindane designer drugs include agitation, anxiety, panic attacks, headache, insomnia, hallucinations, and tachycardia. Three fatal cases were reported due to MDAI intake [5,6-methylenedixy-2-aminoindone], As per the literature, serotonin syndrome could have been a factor that contributed to death. The likelihood of serotonergic toxicity of aminoindanes in humans has not been investigated, but signs of serotonin syndrome were reported for a high dose of MDAI in rats.

Piperazines

Piperazine designer drugs are widely sold as legal party pills or powder and appeared as pure substances or adulterants in pills that are sold as “ecstasy” because of their somewhat MDMA-like pharmacological profile. Various therapeutic drugs have a piperazine moiety and some piperazine designer drugs have a history of medical use; for example 1-benzylpiperazine (BZP) has been investigated as an anthelminthic agent and antidepressant and meta-chlorophenylpiperazine [mCPP] is an active metabolite of different antidepressants.[5]

Mechanism of Action

Piperazine designer drugs exert mixed effects at monoamine transporters. All these substances bind to several serotonergic, adrenergic, dopaminergic and histaminergic receptors with sub micro molar or low micro molar affinity.

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Adverse Effects

Adverse effects of piperazine designer drugs are mostly sympathomimetic including agitation, insomnia, headaches, dizziness, dilated pupils, hyperthermia, tachycardia, nausea, urine retention, and inducible clonus.

In addition to sympathomimetic toxicity, dissociative symptoms, visual and auditory hallucinations, and psychological symptoms (e.g., short temper, confusion, anxiety, depression, and paranoia) have been associated with the use of piperazine designer drugs. Furthermore, toxic seizures have been frequently observed in patients who were admitted to emergency department after the use of BZP-containing party pills. Seizures may also occur at lower doses as per literature. Other severe adverse effects of BZP include hyponatremia, severe combined metabolic and respiratory acidosis, hepatic injury, renal failure, disseminated intravascular coagulation, and rhabdomyolysis. A case of severe hyperthermia with resultant multiorgan failure and a case of hyponatremia that led to fatal brain edema were reported for the concomitant use of piperazine designer drugs and MDMA.

Phenidate Derivatives

Derivatives of the piperidine prescription drug methylphenidate have appeared as designer drugs with substitutions at the "phenyl ring" and different lengths of the carbon side chain phenidate derivatives may be used to induce euphoria or as cognitive enhancers. When insufflated, the pharmacological and subjective-effect profile of methylphenidate is similar to cocaine and phenidate derivatives may therefore be used as substitutes to cocaine.[6]

Mechanism of Action

Similar to methylphenidate, phenidate derivatives act as potent NET and DAT inhibitors that are avoid of any substrate activity. Some less potent interactions with SERT and adrenergic and serotonergic receptors have been reported but are not likely to play a relevant role in the psychoactive actions of most phenidate derivatives.

Adverse Effects

Adverse effects of phenidate derivatives are similar to amphetamines and include agitation, anxiety, hypertension, tachycardia, and palpitations. Because their onset of action is relatively slow when taken orally, the nasal insufflation or injection of phenidate derivatives is common, especially in heavy users. Nasal pain and septum perforations after insufflation and infections after intravenous injections may occur.

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Aminorex Analogs

Various analogs of anorectic agent aminorex have become available as designer drugs. Aminorex was first marketed as an over-the-counter appetite suppressant in parts of Europe in the 1960s but was withdrawn a few years later due to an epidemic of chronic pulmonary hypertension that was associated with many fatalities. Aminorex analogs, like 4-methylaminorex (4-MAR), cause euphoria, mental and physical stimulation, sociability, empathy, arousal, and changes in visual perception.[7]

Mechanism of Action

Aminorex analogs are potent inhibitors of norepinephrine, dopamine, and serotonin reuptake.

Adverse Effects

Adverse effects of aminorex agents include agitation, dysphonia, insomnia, amnesia, panic attacks, psychosis, hallucinations, facial spasms, dilated pupils, foaming at the mouth, dry mouth, jaw clenching, elevations of body temperature, sweating, tachycardia, nausea, and restless legs. Pulmonary hypertension has been associated with recreational use of 4-MAR.

Phenmetrazine Derivatives

Phenmetrazine is a reinforcing stimulant which was previously used as an appetite suppressant before it was eventually withdrawn from the market. They represent relatively understudied class of drugs among which 3-fluorophenmetra (2-3-FPM) use appears to be the most widespread.[8]

Mechanism of Action

Phenmetrazine is a reinforcing stimulant. It is a substrate at the norepinephrine transporter (NET) and DAT with minor substrate activity at the SERT. Ring methylated phenmetrazine derivatives were reported to have greater potency at the SERT, in addition to activity at NET and DAT.

Adverse Effects

They elicit stimulatory toxicity similar to amphetamines. Clinical features include both miotic and dilated pupils, hypertension, and hypotension. Intravenous use of 3-FPM may result in severe vasoconstriction.

Thiophene Designer Drugs

Various analogs of amphetamines and cathinones with a theophany group that replaces the phenyl ring have appeared as designer drugs. To date, most

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pharmacological studies and toxicological reports involve methiopropamine (MPA) the thiophene analog of methamphetamine.[9]

Mechanism of Action

Methiopropamine is a quasi-equipotent inhibitor of norepinephrine and dopamine uptake and was reported to interact with various serotonergic, adrenergic, dopaminergic, N-methyl-D-aspartate, and sigma 1 receptors.

Adverse Effects

Methiopropamine use has been associated with significant acute toxicity and psychotic, cardiovascular, and gastrointestinal symptoms, including agitation, anxiety, confusion, insomnia, visual hallucinations, tachycardia, palpitations, nausea, vomiting, and chest tightness. There may be elevations in levels of creatine kinase.

SEDATIVES

Synthetic Opioids

While being essential for pain treatment, the nonmedical use of opioids has been a public health threat for centuries and includes the recreational use of illegal substances, the abuse of prescription medications, and drug adulteration with nonpharmacological opioids. Opioids induce euphoria, anxiolysis, feelings of relaxation, and drowsiness. Repeated use leads to development of dependence.[10]

Mechanism of Action

Synthetic opioids interact with G-protein-coupled opioid receptors in the brain as partial to full agonists at all, Delta and Kappa opioid receptors subtypes, with selectivity for the ϒ opioid receptors. The agonist at ϒ opioid receptors drives the main pharmacological effects of opioids, including euphoria, analgesia, respiratory depression, and development of dependence.

Adverse Effects

Adverse effects include dizziness, a lower level of consciousness, miosis, central nervous system depression, respiratory depression, pulmonary edema hypoxia, bradycardia, pruritus, nausea, vomiting, constipation, agitation, hypertension, and tachycardia. Serotonergic toxicity is one adverse effect that needs to be considered for opioid designer drugs when compared with other serotonergic agents. The competitive opioid receptor antagonist naloxone rapidly reverses central and peripheral effects of opioids and is thus an effective antidote for opioid toxicity. The initial care of patients who

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are intoxicated with designer drugs should include maintenance of airway, breathing, and circulation. Naloxone should be administered as soon as possible.

Designer Benzodiazepines

Benzodiazepine abuse is frequent. The main reasons for such abuse is to facilitate sleep, cope with stress, ease effects of stimulants, self-treat withdrawal symptoms, and get a high. When taken in combination with opioids, they enhance the euphoric effects of opioid use. Since 2007, several benzodiazepine designer drugs have become available, some of which are precursors or metabolites of prescription benzodiazepine. Their effects resemble those of prescription benzodiazepine.[11]

Mechanism of Action

They mediate their effects through interactions with (GABA) receptors, like prescription benzodiazepines. Benzodiazepines enhance the effect of GABA as positive allosteric modulators by binding to a receptor site that is different from binding site of GABA.

Adverse Effects

The concurrent use of benzodiazepines and other depressants such as alcohol and opioids may produce prolonged and potentially fatal respiratory depression. Reported adverse effects of designer benzodiazepines include agitation, hyperthermia, tachycardia, fatigue, impairment of thinking, confusion, dizziness, drowsiness, lethargy, amnesia, blurred vision, blurred speech, and palpitations. It can cause auditory and visual hallucinations, delirium, seizures, and coma at high doses. Chronic use may lead to development of tolerance and dependence. Withdrawal symptoms such as anxiety, panic attacks, restlessness, insomnia, seizures, and life-threatening conclusions may follow the abrupt cessation of chronic designer benzodiazepine use.

Gamma-aminobutyric Acid Analogs

Gamma hydroxybutyrate (GHB) is a short-chain fatty acid analog of the inhibitory neurotransmitter GABA. It has become popular among drug users because of its ability to induce feelings of euphoria and relaxation, reduce social anxiety, and increase sexual drive. Other structural analogs of GABA that have become available as designer drugs include gamma-butyrolactone (GBL), 1,4-butanediol (1,4-BD), gamma-hydroxyvaleric acid, 4-amino-3-phenyl-butyne acid (phenibut).

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Mechanism of Action

The subjective benefits of GHB and its analogs outweigh adverse effects only over a narrow range of doses. Adverse events include a lower level of consciousness, hypothermia, respiratory depression, aspiration, bradycardia, and gastrointestinal upset. Some other nonsedative adverse events include agitation, seizures, and myoclonus. These adverse events typically have a relative short duration and are usually managed with supportive care. Abrupt cessation after regular use may trigger a potentially life-threatening withdrawal syndrome that can manifest as agitation, anxiety, confusion, disorientation, paranoia, aggression, insomnia, auditory and visual hallucinations, tremors, sweating, hypertension, and tachycardia. Benzodiazepines appear to be the treatment of choice for withdrawal from GHB and its analogs.

Synthetic Cannabinoids

The endocannabinoid system is involved in various physiological functions including cognition, behavior, memory, motor control, pain sensation, appetite, cardiovascular parameters, gastrointestinal motility, and immunoregulation.[12] The term "cannabinoid" refers to the class of compounds produced by *Cannabis sativa* and *Cannabis indica* and endogenous and exogenous ligands that interact with G protein-coupled cannabinoid type 1 (CB1) and CB2 receptors. CB1 receptors are mainly expressed in the brain and modulate neurotransmitter signaling, whereas CB2 receptors are abundant in immune tissues. The synthetic cannabinoids were developed to study human endocannabinoid receptor systems in the second half of 20th century. Today they represent the largest and most structurally diverse class of designer drugs and some of these compounds are similar to phyto- and endocannabinoids. Synthetic cannabinoids are often referred to as "spice" based on the first branded synthetic cannabinoid product. They are commonly applied to dried leaves that mimic cannabis. Desired effects of synthetic cannabinoids include relaxation, euphoria, and disinhibition, thus not significantly differing from desired effects of D^9-tetrahydrocannabinol (D^9THC), the main psychoactive component of cannabis.

Mechanism of Action

Various synthetic cannabinoids have been reported to bind to CB1 and CB2 receptors with higher efficiency at both receptors compared with D^9THC. Based signaling at cannabinoid receptors or the disruption of mitochondrial homeostasis may play a role in the difference between the clinical effects of D^9THC and synthetic cannabinoids, but research in this area is still in its infancy. The predominance of CB1 receptors in the central nervous system compared to CB2 receptors indicates that they (CB1 receptors) mainly mediate the psychoactive effects of synthetic cannabinoids. Possible effects of

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synthetic cannabinoids on noncannabinoid receptors and different signaling pathways have not yet been discovered, but cannot be ruled out.

Adverse Effects

The common adverse effects of synthetic cannabinoids include agitation, drowsiness, dizziness, confusion, hallucinations, hypertension, tachycardia, chest pain, nausea, and vomiting. They typically have short duration and require only symptomatic or supportive treatment. Severe clinical complications that have been reported to be associated with synthetic cannabinoid use include convulsions, delirium, ischemic stroke, intracranial hemorrhage, pulmonary embolism, pneumonia and pulmonary infiltrates, respiratory depression, ventricular and supraventricular arrhythmias, myocardial ischemia and infarction, takotsubo cardiomyopathy, liver injury, acute kidney injury, rhabdomyolysis, and hyperemesis syndrome. Furthermore, various psychiatric adverse effects have been reported including paranoia, psychosis, ideations of self-harm, and suicide.

PSYCHEDELICS

Serotonergic psychedelics induce alterations of perception and cognitive states in users. Traditional psychedelics such as phenylethylamine, tryptamines, and psilocybin and the ergot alkaloid LSD (lysergic acid diethylamide) have a history of being used as illicit black market drug. The psychedelic effects of these drugs are medicated mainly by 5-HT2A agonist. Affinity for 5-HT2A and 5-HT2C receptors is correlated with amount of drug that induces psychedelic effects in the humans.[13]

Phenylethylamines

Derivatives of mescaline comprise a large amount of psychedelic designer drugs. The widest spread phenylethylamines are 2,5-dimethoxyphenylethylamines.

Mechanism of Action

They interact with serotonergic receptors with highest affinity for 5-HT2A receptors. Some derivatives have higher affinity for 5-HT2A and 5-HT2C receptors and lower affinity for 5-HT1A receptors compared to their 2C analogs.

Adverse Effects

Most of the frequently reported adverse effects include agitation, hallucinations, confusion, drowsiness, mydriasis, aggression, hyperthermia, hypertension, and tachycardia. Severe adverse effects include seizure, coma, acute psychosis, cerebral edema, long lasting severe neurological impairment, serotonin syndrome, prolonged respiratory failure, renal failure, multiorgan failure, metabolic acidosis, and rhabdomyolysis.

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Tryptamines

The core structure of tryptamine designer drugs contains an indole ring that is connected to an amino group by an ethyl side chain. They have recently regained interest for their therapeutic use.

Mechanism of Action

5-HT2A receptor agonist plays a key role in mediating the psychedelic effects of naturally occurring and synthetic tryptamine psychedelic. Most traditional and novel tryptamine psychedelics bind to 5-HT1A and 5-HT2A receptors with similar affinity. Some tryptamines are slightly more selective at one or the other receptor subtype. They have been also shown to bind at various other targets in vitro like the adrenergic, dopaminergic, histaminergic receptors. They have interactions at NET, DAT as well as SERT. Substrate activity at VMAT has also been described for tryptamine psychedelics. Tryptamines are prone to metabolism by MAO and MAO inhibitors counteract extensive degradation of tryptamine after oral use.

Adverse Effects

They alter the perception and can induce psychological disturbances in users, including acute psychosis. Adverse events include restlessness, disorientation, clouding of consciousness, confusion, hallucinations, amnesia, catalepsy, mydriasis, tachypnea, tachycardia, and hypertension. Some tryptamines can produce prolonged delusions. In severe cases the use of tryptomer designer drugs has resulted in acute renal failure and rhabdomyolysis.

Lysergamides

Several derivatives of LSD have been described in the scientific literature and such derivatives are increasingly emerging as designer drugs. The LSD-derived designer drugs such as 1-acetyl LSD, 1-propionyl-LSD, and 1-butyryl-LSD have been shown to be metabolized to LSD, in vitro and are thus considered precursors of LSD with very similar effects.

Mechanism of Action

5-HT2A receptor activation mediates the behavioral effects of LSD analogs. Additionally, 5-HT1A receptor activation likely contributes to the qualitative effects of lysergamide designer drugs, similar to LSD and tryptamine psychedelic.

Adverse Effects

Acute physiological effects of LSD include imbalance, difficulty concentrating, feelings of exhaustion, dizziness, headache, dry mouth, lack of appetite, and nausea.

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CONCLUSION

Various types of designer drugs are available in the market. It is important to know and spread information about their effects and side effects so that their misuse and abuse can be prevented or treated accordingly.

TAKE-HOME POINTS

- Designer drugs are a new emerging class of substances that are being used and abused more and more by patients in India and all over the world.
- The number of these drugs keeps increasing as new drugs are being produced as derivatives and synthetic preparations of older existing ones.
- Clinicians must be aware of designer drugs and their types to help in the management of patients that abuse these substances.

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Recent Advances in Psychiatry

The field of psychiatry has been ever-evolving and the evolution in the recent past has been faster than ever. Concerns around mental health are finally taking the driver's seat in society, making it all the more necessary for mental health professionals to be abreast with the recent advances in the field. Recent advances theory papers are there in postgraduate examinations and this book should provide a comprehensive reading for most of the topics covered under this paper. This book has chapters from renowned people working in the area; having firsthand knowledge and experience.

It has 21 chapters covering a wide range of topics. The chapters are on connectomics of the brain, stem cell research, probiotics in relation to psychiatry, telepsychiatry, cyberpsychiatry, newer drugs, mental health laws and insurance in India, ketamine in psychiatry, and ecopsychiatry to name a few. The editors include psychiatry postgraduate teachers and practitioners with varied expertise who are sure this book will give a rich reading experience on recent advances in psychiatry.



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