

Phencyclidine (PCP)

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1. Core Definition

Phencyclidine (PCP) is a synthetic dissociative anesthetic and hallucinogen, originally developed for medical use but now predominantly associated with illicit recreational consumption. Known colloquially as "angel dust," among other street names, PCP belongs to the arylcyclohexylamine class of chemicals. Its pharmacological profile is complex, primarily acting as an N-methyl-D-aspartate (NMDA) receptor antagonist, which interferes with the normal functioning of neurotransmitters in the brain, particularly those involved in memory, learning, and perception. This disruption leads to a profound sense of detachment from one's surroundings and self, a characteristic effect known as dissociation.

The unique psychoactive properties of PCP manifest across a spectrum of effects, depending on the dosage, route of administration, and individual user factors. At lower doses, it can induce euphoria, feelings of numbness, and altered body image, accompanied by a noticeable sluggishness and dulled senses. However, as the dose increases, the drug's effects escalate significantly, leading to more severe psychological and physical manifestations. These can include visual and auditory hallucinations, profound thought disorganization, severe agitation, and intense paranoia, often culminating in behaviors that are erratic, unpredictable, and potentially dangerous to both the user and others.

Beyond its immediate psychoactive effects, PCP poses significant health risks. It is a highly addictive substance, and its chronic use can lead to severe psychological dependence. Acute intoxication can precipitate a range of serious and potentially deadly consequences, including seizures, coma, hyperthermia, and rhabdomyolysis. The drug's capacity to induce states resembling schizophrenia, characterized by delusions and paranoia, underscores its potent neurotoxic and psychotomimetic potential, making it a substance of considerable concern within public health and medical communities.

2. Etymology and Historical Development

The chemical compound phencyclidine was first synthesized in 1956 by chemists at Parke, Davis and Company, with its name deriving from its chemical structure: **phenyl cyclohexyl isopropylamine**. Initially, it was explored for its potential as an anesthetic agent, offering promise due to its ability to induce analgesia and a trance-like state without significant respiratory depression. Following successful preclinical trials, it was introduced into clinical practice under the

brand name **Sernyl** in the late 1950s and early 1960s, primarily for human surgical procedures and later for veterinary medicine.

Despite its initial therapeutic promise, the medical utility of PCP for humans proved to be short-lived. A significant drawback emerged from its post-operative effects, which frequently included severe psychological disturbances such as agitation, delirium, hallucinations, and a prolonged emergence reaction that could last for several hours or even days. These adverse psychotomimetic effects were deemed unacceptable for human surgical applications, leading to its withdrawal from human clinical use in 1965. It continued to be used for a period in veterinary medicine as an anesthetic and tranquilizer under the name **Sernylan** but was also eventually discontinued for animal use in 1978 due to widespread diversion for illicit purposes.

Following its discontinuation from legitimate medical practice, PCP rapidly found its way into the illicit drug market, particularly gaining prominence in the 1960s and 1970s. Its availability and potent psychoactive effects contributed to its rise as a significant drug of abuse, becoming widely known by its infamous street name, "angel dust." The drug's ability to induce profound dissociation and altered states of consciousness, combined with its relatively easy synthesis, cemented its place as a problematic recreational substance. In response to the escalating public health and safety concerns, PCP was classified as a Schedule I controlled substance in the United States in 1978, indicating a high potential for abuse and no accepted medical use, thereby imposing stringent legal restrictions on its manufacture, possession, and distribution.

3. Key Characteristics

Mechanism of Action as an NMDA Receptor Antagonist: The primary pharmacological action of PCP is its non-competitive antagonism of the N-methyl-D-aspartate (NMDA) receptor, a type of ionotropic glutamate receptor crucial for synaptic plasticity, learning, and memory. By blocking the ion channel associated with the NMDA receptor, PCP prevents the influx of calcium ions into neurons, thereby disrupting normal glutamatergic neurotransmission. This blockade is thought to be central to its dissociative and anesthetic effects, interfering with the brain's ability to process sensory input and integrate information, leading to the characteristic sense of detachment from reality. Furthermore, PCP also exhibits other neurochemical effects, including inhibition of dopamine, norepinephrine, and serotonin reuptake, and agonism at sigma opioid receptors, contributing to its complex and varied psychoactive profile.

Dose-Dependent Psychoactive Effects: PCP's effects are highly dependent on the dose consumed. At **low doses** (typically 1-5 mg), users may experience mild euphoria, numbness in extremities, generalized analgesia, motor incoordination, a blank stare, and a distorted perception of body image and reality. Verbal communication may become slurred, and users might exhibit nystagmus (involuntary eye movements). As the dose increases to **moderate levels** (5-10 mg),

the dissociative effects become more pronounced, often accompanied by visual and auditory hallucinations, thought disorganization, agitation, paranoia, and a propensity for violent or erratic behavior. Memory impairment and amnesia for the drug experience are also common. At **high doses** (10 mg or more), the effects can mimic acute schizophrenia, involving severe psychosis, profound confusion, catatonia, and intense delusions. Physical manifestations can escalate to seizures, coma, severe hyperthermia, and potentially life-threatening cardiovascular and respiratory depression.

Physical and Neurological Manifestations: Beyond its psychological effects, PCP elicits a range of distinct physical and neurological signs. Common findings in intoxicated individuals include horizontal and vertical nystagmus, hypertension (high blood pressure), tachycardia (rapid heart rate), and hyperthermia (elevated body temperature). Muscular rigidity, often presenting as a "waxy flexibility," can also be observed. The drug's effects on the central nervous system can lead to ataxia (lack of voluntary coordination of muscle movements), dysarthria (difficulty speaking), and generalized motor dysfunction. In severe cases, particularly with high doses, PCP intoxication can induce status epilepticus, cardiac arrhythmias, and profound respiratory depression, necessitating immediate medical intervention. The duration of effects can be surprisingly prolonged, sometimes lasting for several days, due to PCP's high lipophilicity and enterohepatic recirculation, which contribute to its slow elimination from the body.

4. Significance and Impact

Phencyclidine has had a profound and multifaceted impact across various sectors, most notably in public health, psychiatry, and criminal justice. During its peak period of abuse in the 1970s and early 1980s, PCP became a significant public health crisis, contributing to a surge in emergency room visits related to drug intoxication, psychiatric crises, and traumatic injuries resulting from erratic behavior. The unpredictable and often violent nature of PCP intoxication posed unique challenges for law enforcement and emergency medical personnel, requiring specialized protocols for managing agitated and potentially dangerous individuals under its influence. Although its prevalence has decreased since its peak, PCP remains a persistent concern, with ongoing reports of its illicit manufacture and use.

In the realm of medical research, particularly psychiatry, PCP has been an invaluable pharmacological tool for understanding the neurobiological underpinnings of psychotic disorders, especially schizophrenia. Its ability to induce a state resembling acute schizophrenia in both humans and animal models, largely through its NMDA receptor antagonism, has provided crucial insights into the glutamate hypothesis of schizophrenia. This hypothesis posits that hypofunction of NMDA receptors plays a significant role in the pathophysiology of the disorder, offering targets for the development of novel antipsychotic medications. Researchers utilize PCP to model cognitive deficits, negative symptoms, and positive symptoms observed in schizophrenia, advancing our

understanding of brain function and potential therapeutic strategies.

The social impact of PCP extends to the criminal justice system, where it has been linked to various forms of criminal activity. The drug's propensity to induce extreme paranoia, agitation, and a distorted perception of pain can lead users to engage in violent, self-injurious, or otherwise dangerous acts without inhibition. This has resulted in numerous arrests, emergency detentions, and legal challenges associated with its possession, distribution, and the behaviors exhibited by individuals under its influence. The long-term consequences of PCP abuse, including persistent cognitive deficits, mood disturbances, and the potential for flashbacks, further burden healthcare systems and social support services, highlighting the enduring challenges posed by this potent substance.

5. Debates and Criticisms

One of the enduring aspects of PCP's impact has been the proliferation of various myths and misconceptions surrounding its effects, which have often been sensationalized in popular culture. Perhaps the most prominent of these is the notion that PCP grants users "superhuman strength" or makes them impervious to pain, leading to extraordinary feats of violence. While PCP does induce analgesia and can diminish inhibitions, making individuals less responsive to pain and more prone to aggressive behavior, there is no scientific evidence to support claims of superhuman strength. Such narratives, while contributing to the drug's notorious reputation, can also complicate efforts to effectively manage and treat intoxicated individuals, as emergency responders may approach with unwarranted fear or misunderstanding of the actual pharmacological effects.

From a clinical perspective, managing acute PCP intoxication presents significant challenges. The combination of severe agitation, unpredictable aggression, and potentially life-threatening physiological effects requires rapid and skillful intervention. Debates often arise regarding the most effective sedative agents and supportive care measures for managing acute overdose, particularly in settings where patient cooperation is minimal. The goal of treatment is primarily supportive, focusing on stabilization of vital signs, control of agitation, and protection against self-harm or harm to others. Pharmacological interventions often involve benzodiazepines to manage agitation and seizures, and careful monitoring for complications such as hyperthermia, rhabdomyolysis, and kidney failure.

Furthermore, the addictive potential of PCP and the complexities of treating chronic abuse are subjects of ongoing concern. While physical dependence is not as clearly defined as with some other substances, psychological dependence can be profound, leading to compulsive drug-seeking behavior. Long-term treatment often involves comprehensive behavioral therapies, counseling, and support groups, addressing not only the addiction itself but also co-occurring mental health conditions that may have contributed to or resulted from PCP use. The residual cognitive and

psychiatric effects that can persist long after cessation, such as memory problems, depression, and anxiety, underscore the need for integrated and sustained care models to support recovery and improve the long-term prognosis for individuals affected by PCP addiction.

Further Reading

[Phencyclidine - Wikipedia](#)

[Phencyclidine \(PCP\) - National Institute on Drug Abuse \(NIDA\)](#)

[PCP - DEA Diversion Control Division](#)

[Phencyclidine Toxicity - StatPearls - NCBI Bookshelf](#)

[PCP overdose - MedlinePlus \(National Library of Medicine\)](#)

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