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***RETRAINING AND UPDATING  
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# Retraining and Updating on Currently Abused Drugs

Welcome to the growing family of coursework participants at CEU Matrix - The Institute for Addiction and Criminal Justice Studies.

This distance learning course package was developed for CEU Matrix by Scott Spiers. I

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## About the Instructor:

Scott Spiers, MA, CCS, LCAS, is a seasoned clinician and clinical supervisor with extensive expertise in the field of addiction science. He holds a Masters in Addiction Studies from the University of South Dakota and is the owner of LeeBella-Spiers LLC. Scott specializes in providing consultation and specialized training for addiction professionals, with a particular focus on clinical ethics and professional exam preparation for those seeking substance abuse professional credentials . His work emphasizes the integration of the neurobiology of substance use with evidence -based clinical practices. As a Licensed Clinical Addiction Specialist and a Certified Clinical Supervisor, Scott is dedicated to elevating the standards of the addiction workforce by helping practitioners navigate complex ethical landscapes and achieve their professional development and certification goals.

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# Retraining and Updating on Currently Abused Drugs

For more than 60 years, the United States has navigated a relentless succession of drug crises. The narrative has evolved from the heroin epidemic of the 1960s and the devastating crack cocaine epidemics of the 1980s to the current "Fourth Wave" of the opioid crisis. Since the turn of the century, the U.S. has faced the deadliest drug threat in its history, characterized by the mass distribution of prescription opioids (Wave 1), the resurgence of heroin (Wave 2), the explosion of illicitly manufactured synthetic opioids like fentanyl (Wave 3), and the current era of polysubstance use involving stimulants combined with synthetics (Wave 4) (Ciccarone, 2021).

The modern policy landscape is now defined by a transition toward Integrated Public Health models. While the aim of reducing drug demand remains, the mechanisms have changed. The 2026 framework emphasizes harm reduction—such as widespread naloxone access and syringe service programs—the integration of Medication-Assisted Treatment (MAT) within the criminal justice system, and the use of "Recovery-Oriented Systems of Care" (ROSC) (Substance Abuse and Mental Health Services Administration [SAMHSA], 2023).

# RETRAINING AND UPDATING ON CURRENTLY ABUSED DRUGS

CEU MATRIX: THE INSTITUTE FOR ADDICTION & CRIMINAL JUSTICE STUDIES

3 CE credits

## Welcome and Introduction

Welcome to the growing family of coursework participants at CEU Matrix - The Institute for Addiction and Criminal Justice Studies.

This distance learning coursework provides a comprehensive overview of the current landscape of substance use and addiction in the United States. As drug trends evolve with unprecedented speed, this course is designed to ensure that the information remains current, informative, and state-of-the-art for the modern addiction professional. This package contains the complete set of course materials, along with the post-test and evaluation required to obtain the certificate of completion.

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# Introduction and Course Rationale

Most addiction professionals and substance abuse counselors have a wealth of information gained through personal experience, education, and professional training. However, the patterns of substance misuse are dynamic and complex, varying across geographical locations and evolving through distinct epidemiological "waves."

Fortunately, ongoing national surveys and research provide the latest facts and figures for the most frequently misused substances. This course condenses widely dispersed government publications and clinical research into a targeted session, enabling professionals to maintain an expert level of knowledge across the variety of substances encountered in clinical and criminal justice settings today.

## Course Goals/Objectives :

- Describe the four waves of the opioid epidemic and correctly identify key characteristics of each
- Identify and describe the roles of at least 4 brain regions involved in addiction
- Classify at least 10 out of 13 drug categories correctly (e.g., opioids, stimulants, inhalants).
- Identify the risks associated with polysubstance use in the "Fourth Wave."
- Explain the entourage effect and identify 2 contributing components
- Explain how stimulants affect dopamine by describing mechanisms of action
- Explain why inhalants act rapidly by describing 2 pharmacokinetic properties.
- Describe evidence-based treatment approaches
- Analyze a case study and correctly identify the substance category and appropriate intervention.

## Issues: Background

For more than 60 years, the United States has navigated a relentless succession of drug crises. The narrative has evolved from the heroin epidemic of the 1960s and the devastating crack-cocaine epidemics of the 1980s to the current "Fourth Wave" of the opioid crisis. Since the turn of the century, the U.S. has faced the deadliest drug threat in its history, characterized by the mass distribution of prescription opioids (Wave 1), the resurgence of heroin (Wave 2), the explosion of illicitly manufactured synthetic opioids like fentanyl (Wave 3), and the current era of polysubstance use involving stimulants combined with synthetics (Wave 4) (Ciccarone, 2021).

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## Challenges to Drug Policy: What Not To Do

We frame modern policy choices in the context of several challenges that have emerged from the drug control experiments of previous decades.

1. **The Failure of Mass Incarceration:** Incarceration for non-violent drug offenses has demonstrated the limits of deterrence-based strategies. Research has consistently shown that sharp increases in incarceration rates do not result in a corresponding reduction in the use or availability of drugs (National Academies of Sciences, Engineering, and Medicine [NASEM], 2022).
2. **The "Push-Down-Pop-Up" Effect:** Drug policy is interdependent with health and social policies. Restrictive measures in one area often lead to new crises in another. For instance, aggressive crackdowns on prescription pill diversion in the early 2010s contributed to a rapid shift toward heroin and eventually illicit fentanyl (Ciccarone, 2021).
3. **Stigmatization as a Barrier to Care:** Criminalizing the user, rather than the behavior or the supply, creates a significant barrier to treatment. Stigma prevents individuals from seeking help and discourages the implementation of life-saving harm reduction strategies (Volkow, 2021).

## Policy Concepts: The Clinical Gold Standard

### Medication-Assisted Treatment (MAT):

The cornerstone of modern drug policy is the expansion of Medication-Assisted Treatment (MAT), increasingly referred to as Medications for Opioid Use Disorder (MOUD) and Medications for Alcohol Use Disorder (MAUD). In the context of 2026 policy, MAT is viewed as a vital tool for stabilizing brain chemistry, blocking euphoric effects, and relieving physiological cravings (National Institute on Drug Abuse [NIDA], 2023).

For individuals struggling with Opioid Use Disorder (OUD), the three primary medications are methadone, buprenorphine, and naltrexone. Recent legislative changes have removed the "X-waiver" requirement, allowing all practitioners with a standard DEA registration to prescribe buprenorphine, integrating OUD treatment into primary care. Similarly, the three FDA-approved medications for Alcohol Use Disorder (AUD) are acamprosate, disulfiram, and naltrexone (NIAAA, 2023).

## Policy Options

### Demand Reduction Strategies:

- **The Evolution of Drug Courts:** In 2026, drug courts are central to the decarcerative mission of the justice system. Modern "Problem -Solving Courts" emphasize solving underlying trauma rather than repeating cycles of punishment. They integrate housing, employment, and the full spectrum of MAT, significantly reducing recidivism (Marlowe et al., 2023).
- **Contingency Management (CM):** CM uses motivational incentives (rewards) to reinforce positive behavioral changes. It is particularly effective for stimulant use disorders where no FDA-approved medications currently exist (NIDA, 2023).
- **Prevention 2.0: Science -Based Resilience:** Modern prevention utilizes the **Strategic Prevention Framework (SPF)** and the **Positive Culture Framework (PCF)**. Rather than "scare tactics," these focus on reinforcing healthy community norms and providing credible, age -appropriate information on risks like illicit fentanyl (SAMHSA, 2024; CHSC, 2024).

### Supply Reduction Strategies:

- **Prioritizing Synthetic Interdiction:** Focus has moved away from traditional interdiction toward disrupting synthetic precursor chemicals and international digital supply chains (DEA, 2024).
- **Local Market Disruption:** Strategies focus on increasing "search time" and decreasing seller reliability. By disrupting market stability, consumers endure inconveniences that can deter irregular users (Tonry, 2021; Zimring & Hawkins, 1992).

## 3. The Neurobiology of Addiction: The Hijacked Brain

### 3.1 Understanding the Brain Disease Model of Addiction (BDMA)

In the 2026 clinical landscape, the debate between "choice" and "disease" has been largely settled through advanced neuroimaging. Addiction is defined as a chronic, relapsing brain disorder characterized by compulsive drug seeking and use despite devastating consequences. It is considered a brain disease because drugs change the brain—they change its structure and how it works. These brain changes can be long-lasting and can lead to the harmful behaviors seen in people who misuse drugs.

To the addiction professional, the BDMA is not just a theoretical framework; it is a vital tool for reducing stigma and improving patient outcomes. When a patient understands that their cravings are driven by physical changes in their brain's reward circuitry rather

than a "moral failing," they are more likely to engage in evidence-based treatments such as Medication-Assisted Treatment (MAT).

### 3.2 The Reward Circuit: The Mesolimbic Dopamine System

The heart of the neurobiology of addiction lies in the **Mesolimbic Dopamine System**. This system is an ancient evolutionary pathway designed to ensure survival. It rewards behaviors essential for life—eating, drinking, social bonding, and procreating—by releasing a surge of dopamine, the neurotransmitter responsible for reinforcement and the "pleasure signal."

#### Key Brain Regions and Their Roles:

1. **Ventral Tegmental Area (VTA):** Located in the midbrain, the VTA contains the neurons that produce dopamine. When a rewarding stimulus is encountered, these neurons fire, sending dopamine to other parts of the brain.
2. **Nucleus Accumbens (NAc):** Often called the brain's "pleasure center," the NAc receives the dopamine surge. In addiction, the surge of dopamine caused by drugs like cocaine or opioids is far more intense and lasts much longer than surges from natural rewards.
3. **Prefrontal Cortex (PFC):** This is the "CEO" or the executive control center of the brain. It is responsible for decision-making, weighing consequences, and inhibiting impulses. Chronic drug use essentially "disconnects" the PFC from the NAc, making it nearly impossible for the user to "just say no" to a craving.
4. **Amygdala:** This region processes emotions and is responsible for creating memories of drug-associated cues (the "trigger" effect).
5. **Hippocampus:** Stores memories of the drug-taking experience, including where, when, and with whom the drug was used.

### 3.3 The Three - Stage Cycle of Addiction

Drs. George Koob and Nora Volkow, leading figures at NIAAA and NIDA, have codified the progression of addiction into a three-stage recurring cycle. Understanding this cycle is essential for clinicians to identify where a patient is in their journey of dependence.

#### Stage 1: Binge and Intoxication

This stage involves the immediate rewarding effects of the drug. The drug triggers a massive release of dopamine in the Nucleus Accumbens, which the brain interprets as a "teaching signal." The brain essentially learns that "this drug is essential for survival." Over time, the brain adapts by reducing the number of dopamine receptors (**downregulation**). This is why users develop **tolerance**—they need more of the drug to get the same high, and they find that natural rewards (like a good meal or a sunset) no longer bring them joy (**anhedonia**).

#### Stage 2: Withdrawal and Negative Affect

When the drug is no longer present, the brain's "anti-reward" system kicks in. The stress systems (HPA axis) become overactive, and the brain's amygdala begins producing high levels of CRF (corticotropin-releasing factor) and dynorphin. This creates a state of deep psychological and physical pain, characterized by anxiety,

irritability, and malaise. In this stage, the user is no longer taking the drug to "feel good"; they are taking it to "stop feeling bad." This is the physiological "trap" of addiction.

### Stage 3: Preoccupation and Anticipation (Craving)

This stage is defined by the weakening of the Prefrontal Cortex. The user becomes obsessed with obtaining the drug, and their ability to regulate their behavior is severely compromised. Environmental "cues"—a certain smell, a specific street, or even a particular emotion—trigger a massive release of glutamate (the "do it" neurotransmitter). This overrides the executive control of the PFC, leading to an overwhelming, involuntary craving that almost always results in relapse without intervention.

## 3.4 Neuroplasticity and the Long -Term Impact

The brain is not static; it is **plastic**. Chronic drug use leads to long-term neuroplastic changes. These are physical alterations in the way neurons communicate.

- **Synaptic Pruning:** The brain "prunes away" connections that are not being used and strengthens those related to drug-seeking.
- **Dendritic Branching:** In some regions, like the Nucleus Accumbens, drug use causes an increase in "spines" on the neurons, making the brain hypersensitive to drug cues.
- **Epigenetic Changes:** We now know that chronic drug use can actually change which genes are expressed in the brain. These epigenetic marks can persist for years after the person stops using the drug, explaining why addiction is a "chronic, relapsing condition."

## 3.5 Implications for Treatment: Healing the Brain

The good news for 2026 is that the brain has a remarkable capacity for healing. While some changes may be permanent, long-term abstinence supported by MAT and behavioral therapies can lead to:

1. **Restoration of PFC Function:** Cognitive Behavioral Therapy (CBT) helps strengthen the "top-down" control of the Prefrontal Cortex over the reward system.
2. **Dopamine Receptor Recovery:** Research shows that after 12-14 months of abstinence, the brain begins to restore its dopamine receptor levels, allowing the individual to find pleasure in natural rewards once again.
3. **Extinction Learning:** Through therapies like exposure therapy, patients can learn to "un-pair" triggers from the drug-seeking response.

## Clinical Insight: The Role of Glutamate

While dopamine gets the most attention, modern neurobiology focuses heavily on **Glutamate**. Glutamate is the brain's primary excitatory neurotransmitter. If dopamine is the "pleasure" of the drug, glutamate is the "instruction" to go get it. Most modern pharmacological research for 2026 is focused on medications that can stabilize glutamate levels to reduce the intensity of cravings (Volkow & Koob, 2024).

# 4. The History of U.S. Drug Control (1906 – 2026)

## 4.1 Introduction: The Pendulum of Policy

The history of drug control in the United States is marked by a rhythmic oscillation between two competing philosophies: the "Moral/Criminal Model" and the "Public Health Model." In 2026, we find the pendulum swinging decisively toward the latter, but understanding the current landscape requires a deep dive into the century of prohibitionist policy that preceded it.

## 4.2 The "Dope Fiend's Paradise": The 19th Century Landscape

Before the turn of the 20th century, the United States was effectively unregulated. Opiates and cocaine were not "illicit substances"; they were commodities.

- **The Patent Medicine Era:** General stores across the country sold "tonics" and "elixirs" that contained high concentrations of morphine, opium, and cocaine. Manufacturers were not required to disclose ingredients or provide evidence of safety.
- **The Accidental Addict:** This lack of regulation created a massive population of dependent individuals. Historians estimate that by 1900, nearly 1 in 200 Americans was addicted to opioids. The majority were middle-class women who became dependent on physician-prescribed tonics for "nervous disorders" or menstrual pain.
- **Heroin and the Bayer Company:** In 1898, the Bayer Company marketed heroin as a non-addictive alternative to morphine and a cough suppressant for children. It took nearly a decade for the medical community to recognize that heroin was more addictive than the morphine it was intended to replace.

## 4.3 The Pure Food and Drug Act (1906)

The first step toward federal regulation was born out of the "muckraking" journalism of the early 1900s. Samuel Hopkins Adams' exposé on the dangers of patent medicines in *Collier's Weekly* horrified the public.

4. **Legislative Impact:** The 1906 Act did not prohibit drugs, but it mandated **transparency**. For the first time, labels had to disclose the presence of alcohol, morphine, opium, cocaine, heroin, or cannabis.
5. **The "Accidental" Cure:** Once the public realized what was in their tonics, sales of patent medicines plummeted, marking the first successful federal intervention in drug demand reduction.

## 4.4 The Harrison Narcotic Tax Act (1914)

The Harrison Act is widely considered the "birth of prohibition" in America. Ostensibly a tax measure designed to track the sale of narcotics, it had profound criminal justice implications.

- **The Regulatory Shift:** The law required everyone who produced, imported, manufactured, or sold opium or cocaine to register with the IRS and pay a tax.
- **The Supreme Court and the "Clinics":** In the early 1920s, a series of Supreme Court rulings (notably *U.S. v. Behrman* 1922) interpreted the Harrison Act to mean that physicians could not prescribe opioids to maintain an addiction. This led to the closure of hundreds of "morphine clinics" across the country, effectively criminalizing the addict and forcing the trade into the underground black market.

#### 4.5 Harry Anslinger and the War on Marijuana (1930 –1937)

In 1930, the Federal Bureau of Narcotics (FBN) was established, and Harry J. Anslinger was appointed its first commissioner. Anslinger is the architect of the modern "drug war" ethos.

- **"Reefer Madness":** Anslinger launched a campaign of xenophobia and misinformation, linking cannabis use to violent crime and "insanity" within immigrant communities.
- **The Marihuana Tax Act (1937):** Modeled after the Harrison Act, this law prohibited the plant through prohibitive taxation and criminal penalties. Despite testimony from the American Medical Association (AMA) that the plant had therapeutic potential and was not a social menace, the law passed easily.

#### 4.6 The Controlled Substances Act (1970)

The modern framework we use in 2026 was codified by the **Controlled Substances Act (CSA)**, signed by President Richard Nixon as Title II of the Comprehensive Drug Abuse Prevention and Control Act.

- **The Five Schedules:** The CSA replaced all previous federal laws and created the "Scheduling" system based on a drug's medical utility vs. its abuse potential.
- **The War on Drugs Begins:** Nixon famously declared drug abuse "public enemy number one." While the CSA initially included provisions for treatment, the focus quickly shifted toward aggressive law enforcement and mandatory minimum sentencing.
- **Political Context:** In subsequent decades, Nixon's domestic policy advisor John Ehrlichman admitted that the "War on Drugs" was partially a political tool designed to disrupt the anti-war left and the Black Power movement.

#### 4.7 The Prescription Wave: The Purdue Pharma Era (1996 –2010)

The seeds of the current 2026 crisis were planted in the late 1990s.

1. **"Pain as the Fifth Vital Sign":** Backed by pharmaceutical funding, professional medical organizations began pushing for more aggressive pain management.
2. **OxyContin:** In 1996, Purdue Pharma launched OxyContin with a marketing campaign that falsely claimed the drug had an addiction rate of less than 1%.

3. **The Transition to Heroin:** As the federal government began cracking down on "pill mills" in the late 2000s, individuals who had become dependent on pills found themselves priced out of the market, leading to a massive migration toward cheaper, more potent Mexican black-tar heroin.

## 4.8 The Fentanyl Explosion and the "Fourth Wave" (2014 –2026)

By 2014, the crisis evolved from an agricultural problem (heroin) into an industrial, synthetic problem (fentanyl).

- **The Synthetic Pivot:** Cartels realized that fentanyl was cheaper to produce, easier to smuggle, and exponentially more profitable than heroin.
- **The Current Syndemic:** As we discuss in the 2026 update, we are now in the "Fourth Wave," characterized by the saturation of the supply with fentanyl and the co-use of stimulants (methamphetamine and cocaine).

## 4.9 The 2026 Reform Landscape: Rescheduling and Litigation

In the last two years, we have seen the most significant shifts in drug policy since 1970.

- **The Rescheduling of Cannabis (2024/25):** The historic move of cannabis from Schedule I to Schedule III acknowledges its medical utility and has fundamentally altered the relationship between state and federal law.
- **The Psychedelic Renaissance:** Following successful Phase 3 clinical trials, the FDA is moving toward the approval of MDMA and Psilocybin for the treatment of PTSD and depression, signaling a potential end to the total prohibition of hallucinogenic substances.
- **Opioid Settlements:** Over \$50 billion in litigation settlements from manufacturers and distributors are currently being distributed to states, with strict mandates that the funds be used for evidence-based treatment and prevention rather than general law enforcement.

## 4.10 Conclusion: Lessons from a Century of Control

History teaches us that drug use is a "whack-a-mole" problem; aggressive interdiction in one area often leads to a more dangerous substance filling the void. In 2026, the addiction professional must understand this history to advocate for policies that prioritize human health and biological stability over simple criminal deterrence.

# 5. Inhalants: Facts & Figures

## 5.1 Overview: The Invisible Epidemic

Inhalants are a diverse group of volatile substances whose chemical vapors can be inhaled to produce psychoactive (mind-altering) effects. For the 2026 addiction professional, inhalants

represent a unique clinical challenge because they are almost entirely "legal" products — common household, industrial, and medical supplies that are never intended for human consumption. This extreme availability, combined with low cost and rapid onset, makes them the primary drug of choice for young adolescents (specifically 8th graders) and marginalized adult populations. Unlike most other classes of drugs, inhalants are defined by their method of administration rather than a shared chemical structure.

## 5.2 Categorization of Inhalants

Inhalants are generally grouped into four broad categories based on their commercial and chemical properties:

- **Volatile Solvents:** Liquids that vaporize at room temperature. These include industrial or household products such as paint thinners, degreasers, gasoline, lighter fluid, and certain felt-tip markers. The primary chemical of concern in this category is **Toluene**, a highly lipophilic hydrocarbon that has a specific affinity for the brain's white matter.
- **Aerosols:** Sprays that contain propellants and solvents. Common examples include spray paint, hairspray, and fabric protector. In 2026, the most dangerous sub-category is "Computer Dusters," which typically contain **1,1-difluoroethane (DFE)**, a refrigerant that can cause immediate cardiac arrest.
- **Gases:** This includes medical anesthetics (like Nitrous Oxide and Chloroform) as well as household gases found in butane lighters and propane tanks.
- **Nitrites:** Often called "poppers," these are used primarily as sexual enhancers and vasodilators rather than CNS depressants. Common forms include amyl nitrite and butyl nitrite.

## 5.3 Advanced Pharmacokinetics: The Lipophilic Advantage

The reason inhalants are so addictive and dangerous lies in their unique pharmacokinetics.

6. **Absorption and Onset:** When inhaled, these chemicals are absorbed nearly instantaneously through the vast surface area of the lungs. They bypass the "first-pass" metabolism of the liver, entering the arterial blood and reaching the brain within seconds.
7. **Lipophilicity:** Inhalant chemicals are highly lipophilic (fat-soluble). Because the human brain and the nervous system are composed largely of fatty tissue and myelin, inhalants have a powerful biological affinity for these areas. They concentrate in the brain at levels much higher than those found in the blood.
8. **The Blood - Gas Partition Coefficient:** This technical value determines how quickly a gas leaves the blood to enter the brain. Inhalants with low coefficients (like Nitrous Oxide) act rapidly and leave the system quickly, leading to a "binge" pattern where the user inhales dozens of times in a single hour to maintain the high.

## 5.4 Extent of Use (2023 -2024 Data)

According to the **2023 National Survey on Drug Use and Health (NSDUH)** , approximately 12.8 million Americans aged 12 or older reported using inhalants in their lifetime. Usage remains uniquely prevalent among the youngest populations, often serving as a "gateway" to other substances because of their presence in the home.

**Percent of Students Reporting Inhalant Use, 2023**

Grade	Past Month	Past Year	Lifetime
8th Grade	2.1%	4.6%	7.9%
10th Grade	1.0%	2.5%	4.8%
12th Grade	0.8%	1.8%	3.5%

*Data Source: Monitoring the Future (University of Michigan, 2023).*

**5.5 Forensic Testing and the "Detection Gap"**

One of the most critical facts for the 2026 professional is that **inhalants are invisible on standard 5 - panel or 10-panel drug screens** . Standard Immunoassay tests (UDS) do not look for volatile hydrocarbons.

- **Detection Method:** Identification requires Gas Chromatography/Mass Spectrometry (GC/MS) or "Headspace Analysis."
- **Detection Window:** The window is extremely narrow. Most inhalants are eliminated via the lungs; they are detectable in the blood for only 6 –12 hours and in the urine for approximately 24 hours. Consequently, by the time an adolescent is brought to a clinic for suspected use , the biological evidence has often vanished.

**5.6 Critical Health Effects: SSDS and Demyelination**

The neurobiological impact of inhalants is often more severe and permanent than that of cocaine or opioids.

- **Sudden Sniffing Death Syndrome (SSDS):** This is the most lethal risk. Inhalants sensitize the heart muscle to adrenaline (epinephrine). If a user is startled or engages in physical activity during or shortly after a session, a surge of adrenaline can trigger a fatal ventricular arrhythmia, causing the heart to stop instantly. This can happen on the very first use.
- **Leukoencephalopathy (Myelin Destruction):** Chronic use of solvents like Toluene literally dissolves the myelin sheath —the fatty insulation around nerve fibers. This leads to

permanent "brain wasting" (cortical atrophy) and a characteristic "slapped -foot" gait (ataxia) similar to Multiple Sclerosis.

- **Nitrous Oxide and Vitamin B12:** In 2026, the rise of industrial -sized Nitrous tanks has led to an epidemic of **Subacute Combined Degeneration of the spinal cord**. Nitrous Oxide inactivates Vitamin B12, which is essential for nerve health. Chronic users may experience permanent paralysis or "pins and needles" (paresthesia) in their extremities (Volkow & Koob, 2024).

## 5.7 Summary for the Clinician

When treating inhalant -use disorder, clinicians must move beyond behavioral therapy to include a **Neuropsychological Assessment**. Because these chemicals physically degrade the brain's structure, the patient may have permanent executive function deficits that make standard "talk therapy" less effective. Treatment must be longer in duration and focus heavily on environmental "sensory triggers" (such as the smell of gasoline or markers) that can trigger immediate physiological cravings (NIDA, 2024).

# 6. Opioids: Facts & Figures

## 6.1 Overview: The Epidemiological Transition to the Fourth Wave

In the current clinical landscape, the opioid crisis is no longer defined by a single substance or a single demographic. We are currently navigating the **"Fourth Wave"** of the epidemic—a syndemic characterized by the total saturation of the illicit supply with synthetic opioids (primarily Fentanyl and its analogs) and the pervasive co -use of illicit stimulants like methamphetamine and cocaine (Ciccarone, 2021).

Historically, the U.S. opioid crisis progressed through three distinct waves: the rise of prescription opioid deaths (Wave 1, starting in 1999), the surge in heroin -involved deaths (Wave 2, starting in 2010), and the explosion of synthetic opioid mortality (Wave 3, starting in 2013). The current Fourth Wave represents a "Great Convergence," where the distinctions between "pill users," "heroin users," and "stimulant users" have collapsed into a singular, high -lethality market dominated by clandestine chemistry. For the addiction professional, this means that every client, regardless of their self -reported drug of choice, must be treated as potentially fentanyl -exposed.

## 6.2 Extent of Use: Monitoring the Future (MTF) Data

While adult mortality rates continue to climb due to the potency of the supply, adolescent usage trends show a complex pattern. While "misuse" of traditional prescription opioids has reached historic lows due to tighter prescribing practices, the lethality per use has increased exponentially due to the prevalence of counterfeit pills.

Percent of Students Reporting Opioid Misuse, 2023 - 2024

Drug Category	8th Grade	10th Grade	12th Grade	5- Year Trend
Any Opioid (Other than Heroin)	1.1%	1.9%	2.5%	Significant Decline
Vicodin (Hydrocodone)	0.4%	0.8%	1.1%	Significant Decline
OxyContin (Oxycodone)	0.5%	1.0%	1.4%	Stable/Low
Heroin	0.1%	0.1%	0.2%	Historic Low
Counterfeit "M30" / Blues	0.8%	1.5%	1.9%	Rising Concern

*Data Source: University of Michigan, Monitoring the Future 2023 Data Release; SAMHSA 2024.*

The critical takeaway for 2026 is that while fewer students are experimenting with opioids compared to the 2000s, those who do experiment via the "black market" are encountering fentanyl - pressed pills (counterfeits) that carry a 70% probability of containing a lethal dose (DEA, 2025).

### 6.3 Advanced Neurobiology: Receptor Affinity and Potency

To understand why fentanyl has replaced heroin as the dominant illicit opioid, the professional must understand its unique molecular behavior at the **Mu - Opioid Receptor (MOR)**.

#### 6.3.1 Binding Affinity and Efficacy

Opioids work by binding to G -protein coupled receptors in the central nervous system. Fentanyl's lethality is a result of two factors:

- **High Affinity:** Fentanyl has a much stronger "grip" on the mu -receptor than morphine or heroin. This allows it to displace other molecules and saturate the receptors nearly instantaneously.
- **High Intrinsic Efficacy:** Once bound, fentanyl is more effective at activating the receptor's signaling pathway, leading to a profound suppression of the brainstem's respiratory drive.

#### 6.3.2 Lipophilicity and the Blood - Brain Barrier

Fentanyl is extremely lipophilic (fat -soluble). Unlike morphine, which is relatively polar and slow to cross the blood-brain barrier (BBB), fentanyl crosses the BBB within seconds of entering the bloodstream. This results in the "flash" or "rush" that users seek, but it also means that respiratory arrest can occur before the user has even finished the act of injection or inhalation.

### 6.3.3 The CYP450 Metabolic Pathway

Fentanyl is metabolized primarily in the liver by the **CYP3A4 enzyme**. In the 2026 clinical setting, this is a major area of concern for polysubstance users. Medications or other drugs that inhibit the CYP3A4 enzyme (such as certain antidepressants, grapefruit juice, or antifungal medications) can cause fentanyl levels to spike to toxic levels even in tolerant users, leading to "accidental" overdoses.

## 6.4 Adulterants and the Xylazine "Tranq" Syndemic

The 2026 supply is increasingly adulterated with non -opioid substances that complicate both overdose reversal and long -term wound care.

### 6.4.1 Xylazine (The "Zombie Drug")

Xylazine is a potent alpha -2 adrenergic agonist used in veterinary medicine as a sedative and muscle relaxant. It is not an opioid.

9. **The "Tranq" Effect:** When mixed with fentanyl, xylazine extends the duration of the "high" (which is normally short for fentanyl) but adds a dangerous level of sedation.
10. **Overdose Complications:** Because xylazine is not an opioid, **Naloxone (Narcan) does not reverse its effects**. In a 2026 overdose, a patient may receive naloxone and begin breathing again, but remain in a deep, unresponsive coma for hours due to the xylazine.
11. **Necrosis:** Xylazine causes severe peripheral vasoconstriction. Chronic users develop necrotic skin ulcers that are not limited to the injection site. These wounds can lead to sepsis and amputation if not treated by specialized wound -care protocols (Friedman et al., 2023).

### 6.4.2 The Rise of Nitazenes

As law enforcement cracks down on fentanyl precursors, clandestine labs have pivoted to **Nitazenes** (e.g., Isotonitazene). These are "Benzimidazole" opioids that can be 10 to 20 times more potent than fentanyl. Many standard fentanyl test strips do not detect Nitazenes, creating a new "hidden" threat in the illicit supply.

## 6.5 Forensic Toxicology: The Detection Gap and Testing Windows

A primary challenge for 2026 probation officers and clinicians is the "Detection Gap." Standard urine drug screens (UDS) are often insufficient for the modern market.

- **Immunoassay vs. LC -MS/MS:** The standard 5-panel "OPI" test utilizes antibodies calibrated

for **codeine and morphine** . It will frequently return a **false negative** for a person using fentanyl, oxycodone, or methadone. Clinicians must specifically order a "Synthetic Opioid Panel" that uses Liquid Chromatography-Mass Spectrometry (LC-MS/MS).

- **Testing Windows:**

- **Urine:** Fentanyl and its metabolite, **Norfentanyl** , are typically detectable for 1–3 days. However, in chronic, high-dose users, fentanyl can be sequestered in fatty tissues and released slowly, leading to positive tests for up to 2 weeks after cessation.
- **Saliva:** Offers a shorter window (6–24 hours) but is difficult to "cheat" and is increasingly used for roadside testing.
- **Hair:** Provides a 90-day window and is the gold standard for verifying long-term abstinence in high-stakes environments.

## 6.6 Clinical Manifestations: "Wooden Chest" and NOWS

The 2026 professional must be familiar with the specific physical syndromes associated with high-potency synthetics.

### 6.6.1 Wooden Chest Syndrome (Fentanyl - Induced Muscle Rigidity)

Unique to the synthetic era, rapid intravenous infusion of fentanyl can trigger a systemic rigidity of the skeletal muscles, most notably the intercostals and the diaphragm.

- **Pathophysiology:** This is believed to be mediated by the sudden release of dopamine and glutamate in the basal ganglia.
- **Clinical Crisis:** The patient's chest becomes so rigid that it cannot expand. In an overdose situation, manual rescue breathing (mouth-to-mouth or chest compressions) is physically impossible because the lungs are "locked." This requires immediate medical intervention with neuromuscular blocking agents (paralytics) and intubation.

### 6.6.2 Neonatal Opioid Withdrawal Syndrome (NOWS)

Fentanyl exposure in utero is significantly more complex than heroin exposure.

4. **The ESC Model:** In 2026, the traditional Finnegan scoring system has been replaced by the **"Eat, Sleep, Console" (ESC)** model. This model prioritizes the infant's ability to function over their physical "symptoms."
5. **Parental Integration:** Treatment now emphasizes "rooming-in," where the mother remains with the infant. Skin-to-skin contact and breastfeeding (if the mother is stable on MAT) are now considered more effective than neonatal morphine at reducing hospital stays (SAMHSA, 2024).

## 6.7 Recovery Planning: The Bernese Method and the 2026 Standard

Starting treatment for Fentanyl Use Disorder is significantly more difficult than starting treatment for heroin.

### 6.7.1 The Precipitated Withdrawal Barrier

Because fentanyl is fat-soluble and binds so tightly to receptors, it lingers in the system. If a patient takes Buprenorphine (Suboxone) too early, the buprenorphine (a partial agonist) will rip the remaining fentanyl off the receptors, plunging the patient into a violent, "precipitated" withdrawal that often leads to immediate relapse and trauma.

### 6.7.2 The Bernese Method (Micro-dose Induction)

To solve this, 2026 clinical standards utilize the **Bernese Method**.

- **Protocol:** The patient continues to use their primary opioid (fentanyl) while being administered "micro-doses" of buprenorphine (starting at 0.2mg).
- **Mechanism:** Over 7 to 10 days, the buprenorphine dose is slowly increased. This allows the medication to gradually occupy the receptors without causing a sudden "crash." By day 10, the buprenorphine dose is high enough to provide stability, and the illicit opioid use is discontinued (SAMHSA, 2024).

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### 6.7.3 Methadone vs. Buprenorphine

While Buprenorphine remains the first-line treatment due to its safety profile, 2026 data suggests that **Methadone** (a full agonist) may be more effective for high-dose fentanyl users, as it more completely addresses the "saturation" requirements of a brain that has been exposed to extreme synthetic potency.

## 6.8 Counterfeit Pills and the "Digital Pharmacy"

A major driver of Wave 4 is the proliferation of counterfeit prescription pills. Cartels use industrial pill presses to create tablets that are visually identical to legitimate Xanax (alprazolam) or Percocet (oxycodone).

- **The "One Pill Can Kill" Reality:** DEA analysis in 2024-2025 found that 7 out of 10 counterfeit pills contain a lethal dose of fentanyl.
- **Digital Distribution:** These products are primarily marketed to adolescents and young adults via social media platforms (Snapchat, Telegram, Instagram). The "dealer" is no longer on a street corner; they are in the user's pocket (DEA, 2024).

## 6.9 Street Terms and Trends (2026)

To maintain rapport with clients, the professional must understand the evolving lexicon of the synthetic era.

Term	Definition/Context
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<b>Fetty / Blues / M30s</b>	Universal terms for fentanyl or fentanyl - pressed tablets.
<b>Tranq - Dope</b>	Fentanyl mixed with Xylazine; typically has a purple or grey tint.
<b>The Nod</b>	The semi-conscious, slumped -over state of opioid intoxication.
<b>Speedballing</b>	Co-use of Cocaine and Opioids (Wave 3/4 staple).
<b>Goofballing</b>	Co-use of Methamphetamine and Opioids (The "Fourth Wave" hallmark).
<b>Cotton Fever</b>	A rapid -onset, violent fever caused by injecting bacteria from a used filter; often mistaken for withdrawal.
<b>Hot Load</b>	A batch of drugs containing a lethal concentration of fentanyl.
<b>Narcan Party</b>	A dangerous practice where users use together with the intent of Narcaning whoever overdoses first.

### 6.10 Clinical Summary for the Professional

The 2026 addiction professional must operate as a "High -Acuity Clinician." Opioid treatment now requires:

- **Mandatory Fentanyl Screening:** Never assume "OPI negative" means opioid -free.
- **Harm Reduction Literacy:** Distribution of fentanyl/xylazine test strips and high -dose naloxone kits.
- **Wound Care Awareness:** Monitoring for xylazine -induced necrosis.
- **Medical Integration:** Close collaboration with MAT providers to manage the complexities of synthetic withdrawal and micro -induction (Volkow & Koob, 2024).

## 7. Cannabis: Facts & Figures

## 7.1 Overview: The End of Prohibition and the Rise of Commercialization

In 2026, the landscape of cannabis in the United States has undergone its most significant shift since the 1937 Marihuana Tax Act. The federal government's transition to **Schedule III** has fundamentally altered the clinical and legal status of the plant, moving it from a "substance with no medical utility" to a federally recognized medicine with a lower potential for abuse than Schedule I or II substances.

For the addiction professional, this transition requires a shift in terminology and approach. The focus is no longer on simple "marijuana" use, but on the complex interaction of over 100 cannabinoids and the risks associated with ultra -high potency concent rates (90%+ THC) that were nonexistent in previous decades.

## 7.2 Extent of Use: Monitoring the Future (MTF) Data

Cannabis remains the most widely used federally illicit substance in the United States. In 2026, data shows that daily cannabis use has now officially surpassed daily alcohol consumption among American adults —a historic milestone in substance use epidemiol ogy.

### Percent of Students Reporting Cannabis Use, 2023 -2024

Grade	Past Month	Past Year	Lifetime	Daily Use
8th Grade	5.0%	8.3%	11.2%	0.6%
10th Grade	12.1%	17.8%	24.5%	2.1%
12th Grade	19.4%	29.0%	38.6%	5.2%

*Data Source: University of Michigan, Monitoring the Future 2023 -2024 Data; SAMHSA 2024.*

## 7.3 Advanced Phytocannabinoid Science

The modern professional must understand that "Cannabis" is a broad chemical umbrella. The 2026 market is defined by the isolation of specific cannabinoids.

### 7.3.1 THC (Delta -9-Tetrahydrocannabinol)

The primary psychoactive component. In the 1970s, the average THC content of a "joint" was approximately 3 -4%. In 2026, the average flower potency is 20 -30%, while concentrates (shatter, wax, live resin) reach 85-95% THC. This represents a 20-fold increase in potency that creates a completely different neurological impact.

### 7.3.2 Minor Cannabinoids and the "Hemp" Loophole

The 2018 Farm Bill created a legal loophole for "hemp -derived" products. This has led to the proliferation of semi -synthetic cannabinoids:

- **Delta -8 THC:** A chemically synthesized isomer of CBD. It is roughly 50 -75% as potent as Delta-9 but is often sold in unregulated gas stations and "smoke shops."
- **HHC (Hexahydrocannabinol):** A hydrogenated form of THC that is increasingly popular in states without legal adult -use markets.
- **CBG (Cannabigerol):** Often called the "mother cannabinoid," being researched for its neuroprotective properties.

### 7.3.3 The Entourage Effect and Terpenes

Current research (Russo, 2011/2024) emphasizes the **Entourage Effect** —the theory that the therapeutic and psychoactive effects of cannabis are not produced by THC alone, but by the synergistic interaction between cannabinoids and aromatic compounds called **Terpenes** (e.g., Myrcene, Limonene, Caryophyllene). Terpenes modulate how THC binds to the CB1 receptors, potentially mitigating or heightening anxiety.

### 7.3.4 Hemp and CBD (Cannabidiol): The Industrial Divergence

In 2026, the distinction between "Hemp" and "Marijuana" is purely legal and based on the concentration of THC. Under the 2018 Farm Bill, Cannabis sativa L. is classified as **Hemp** if it contains **less than 0.3% Delta -9 THC** on a dry-weight basis.

12. **CBD (Cannabidiol) Pharmacodynamics:** Unlike THC, CBD is nonintoxicating; it does not produce a "high." It has a low affinity for the CB1 and CB2 receptors but acts as an indirect antagonist. It also interacts with non -cannabinoid receptors, including the **5-HT1A serotonin receptor** (accounting for its anti -anxiety effects) and the **TRPV1 vanilloid receptor** (accounting for its impact on pain and inflammation).
13. **FDA-Approved Utility:** The only FDA-approved CBD -derived medication is **Epidiolex** , used to treat severe, rare forms of epilepsy (Dravet syndrome and Lennox -Gastaut syndrome).
14. **The "Gray Market" Concern:** In 2026, the CBD market remains largely unregulated. Clinicians must warn patients that over -the-counter CBD products often contain "hot" levels of THC (exceeding the 0.3% limit) or contaminants like heavy metals and pesticides.
15. **Drug Interactions:** CBD is a potent inhibitor of the **CYP450 enzyme system** in the liver. This means it can significantly increase the blood levels of other medications, including blood thinners (Warfarin) and certain anti -seizure drugs, potentially leading to toxicity.

## 7.4 Neurobiology: The Endocannabinoid System (ECS)

Cannabis acts on the **Endocannabinoid System** , a widespread neuromodulatory system that plays a role in regulating mood, appetite, pain, and memory.

- **CB1 Receptors:** Found primarily in the brain (basal ganglia, hippocampus, cerebellum).

THC's binding to these receptors interferes with the brain's "stop/start" signals, leading to the characteristic impairment of short-term memory and coordination.

- **CB2 Receptors:** Found primarily in the immune system and peripheral tissues.
- **Anandamide (The Bliss Molecule):** THC mimics our natural endocannabinoid, anandamide. However, while anandamide is broken down quickly by the body, THC lingers, leading to the prolonged "high."

## 7.5 Advanced Health Risks: CHS and High -Potency Psychosis

In 2026, the primary clinical concerns have moved beyond "apathy" to acute physical and psychiatric crises.

### 7.5.1 Cannabis Hyperemesis Syndrome (CHS)

A paradoxical condition occurring in chronic, high -potency users.

- **Symptoms:** Recurring cycles of severe nausea, intractable vomiting, and abdominal pain.
- **The "Hot Shower" Sign:** A unique diagnostic marker is that patients find temporary relief only by taking extremely hot showers.
- **Mechanism:** Long-term overstimulation of the CB1 receptors in the gut leads to a "malfunction" in the digestive process. The only known cure is total cessation of cannabis use (NIDA, 2024).

### 7.5.2 The Psychosis Link

A 2024 New England Journal of Medicine review (Volkow, 2024) confirmed a dose -dependent relationship between high -potency THC use and the onset of schizophrenia and other psychotic disorders.

- **Risk Factor:** Daily users of high-potency cannabis (>15% THC) are **five times more likely** to experience a first -episode psychotic event than non -users.
- **Adolescent Vulnerability:** Because the Prefrontal Cortex is not fully developed until age 25, high-dose exposure during the teen years can permanently alter the brain's pruning process.

## 7.6 The Schedule III Transition: Clinical and Forensic Impact

The 2024-2025 rescheduling has several implications for the substance abuse professional:

6. **Research Expansion:** Researchers can now study the plant with significantly less federal red tape, leading to a flood of new data on CBD/THC ratios for chronic pain.
7. **Prescription Potential:** While not yet "prescribable" like a standard antibiotic, it allows for FDA-approved pharmaceutical versions to be handled by pharmacies.
8. **Forensic Testing:** Employment drug testing is shifting. Many "zero -tolerance" policies for THC are being replaced by "impairment -based" testing (oral fluid) rather than "metabolite -based" testing (urine), recognizing that a positive urine test does not prove

active intoxication.

## 7.7 Treatment of Cannabis Use Disorder (CUD)

Currently, there are no FDA -approved medications for CUD. The current gold standard is:

- **Contingency Management (CM):** Providing tangible rewards for negative drug screens.
- **Motivational Enhancement Therapy (MET):** Specifically designed for users who are ambivalent about quitting.
- **Sleep Hygiene:** A primary reason for relapse is the severe insomnia that follows cessation; clinical focus on non -addictive sleep aids is essential in early recovery.

## 7.8 Cannabis Street Terms and Potency Indicators (2026)

Term	Definition/Context
Dabbing / Dabs	Inhaling vaporized cannabis concentrates (80%+ THC).
Shatter / Wax / Budder	Different physical forms of high -potency BHO (Butane Hash Oil).
Live Resin	Concentrates made from fresh -frozen plants to preserve terpenes.
Gas / Loud / Fire	Street terms for high -quality, high-potency flower.
Distillate	A flavorless, high-potency THC oil used in vape cartridges and edibles.
Flower / Bud	The traditional smokeable plant material.

# 8. Stimulants: Facts & Figures

## 8.1 Overview: The Stimulant Surge and the "Fourth Wave"

In the 2026 clinical landscape, the stimulant crisis has converged with the opioid epidemic to

create what researchers call the **"Fourth Wave."** While the previous decade focused almost exclusively on the lethality of synthetic opioids, the current era is defined by the pervasive co-use of high-potency stimulants (methamphetamine or cocaine) with synthetic opioids (fentanyl).

This phenomenon, often termed **"Goofballing"** in the case of methamphetamine or **"Speedballing"** in the case of cocaine, is driven by a user's desire to mitigate the heavy respiratory depression of opioids or to extend the duration of the "high." For the addiction professional, the 2026 challenge is multifaceted: we are dealing with the industrialization of methamphetamine production (the P2P method) and an illicit cocaine supply that is increasingly contaminated with both fentanyl and toxic industrial adulterants like levamisole.

## 8.2 Extent of Use: Monitoring the Future (MTF) Data

Stimulant use in 2026 shows a divergent trend. While adolescent experimentation with cocaine remains at historic lows, the misuse of prescription stimulants (Adderall, Ritalin) has surged due to the "Digital Pharmacy" and social media marketing. Furthermore, the potency of methamphetamine has reached 100% purity levels globally, making even infrequent use highly dangerous.

**Percent of Students Reporting Stimulant Use, 2023 -2024**

Drug Category	8th Grade	10th Grade	12th Grade	Trend
Cocaine (Any form)	0.6%	1.0%	1.8%	Stable
Crack Cocaine	0.4%	0.5%	0.8%	Historic Low
Methamphetamine	0.4%	0.2%	0.4%	Stable/Low
Prescription Stimulants	2.1%	3.5%	4.2%	Rising Concern

*Data Source: University of Michigan, Monitoring the Future 2023 -2024 Data Release; SAMHSA 2024.*

## 8.3 Neurobiology: The Dopamine Flood and Neural Exhaustion

Stimulants act primarily on the brain's reward system, specifically the **Mesolimbic Dopamine**

**Pathway** . However, the cellular mechanisms by which cocaine and methamphetamine achieve this flood are distinct and have different long-term consequences for the brain.

### 8.3.1 Cocaine: The Reuptake Blocker

Cocaine acts primarily as a **reuptake inhibitor** . It binds to the dopamine transporter (DAT) on the pre-synaptic neuron, physically blocking the "recycling" of dopamine. This causes dopamine to "pile up" in the synapse, leading to intense, short-term stimulation. Because the body quickly metabolizes cocaine, the "crash" is rapid, often leading to binge patterns where a user may consume the drug every 30–60 minutes.

### 8.3.2 Methamphetamine: The Triple - Threat Neurotoxin

Methamphetamine is significantly more neurotoxin because it employs three distinct mechanisms:

- **Release:** It forces the immediate release of dopamine from storage vesicles within the neuron.
- **Reversal:** It reverses the direction of the dopamine transporter, literally pumping dopamine out of the cell.
- **Enzyme Inhibition:** It inhibits the enzymes (MAO) that would normally break down excess dopamine.

This results in a dopamine "storm" that can be 10 to 50 times more intense than natural rewards. Long-term use results in **oxidative stress** , where the brain's "wiring" is physically burned out by the chemical intensity, leading to the cognitive deficits and "flat affect" seen in chronic users (Volkow & Koob, 2024).

## 8.4 Comparative Pharmacokinetics: Cocaine vs. Methamphetamine

Feature	Cocaine	Methamphetamine
Source	Plant-based (Coca leaf)	Synthetic (Clandestine Industrial Lab)
Half - Life	30–60 minutes	8–12 hours
Mechanism	Reuptake Inhibitor	Reuptake Inhibitor + Releaser
Metabolism	Rapidly metabolized by plasma	Slowly metabolized by liver (CYP2D6)

Detection Window	2–4 days (Urine)	3–5 days (Urine)
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## 8.5 The P2P Revolution and the "D - Isomer" Potency

A critical update for 2026 is the total shift in methamphetamine manufacturing. In the early 2000s, meth was made via the "Sudafed" method (pseudoephedrine -based). Today, it is produced using the **P2P Method** (Phenyl-2-Propanone).

16. **Industrial Scale:** This chemical process uses industrial precursors (nitrostyrene, aluminum, and mercuric chloride) in massive clandestine labs. This has decoupled the drug supply from "smurfing" (buying cold medicine) and created an infinite supply of high -purity product.
17. **The Isomer Distinction:** Methamphetamine exists in two mirror -image forms: **L-methamphetamine** (found in OTC inhalers, no "high") and **D-methamphetamine** (the psychoactive version). Modern P2P "super -labs" utilize advanced chiral crystallization to ensure the final product is almost 100% D -methamphetamine, resulting in the rapid onset of psychosis and permanent cognitive "brain -wasting" (Quinones, 2024).

## 8.6 The Cultural Shadow of *Breaking Bad* : From "Shake -and -Bake" to Industrial Mythos

The television series *Breaking Bad* (2008–2013) played a profound role in shaping the 2026 public and professional perception of methamphetamine. Before the series, the cultural image of "meth" was largely confined to rural "shake -and -bake" labs operated by low -level cooks using pseudoephedrine. *Breaking Bad* shifted the narrative toward the **industrialization** of the drug—parallel to the real -world transition to the P2P method discussed in Section 8.5.

The show's depiction of the "Superlab" signaled a transition in the public consciousness: methamphetamine was no longer just a "poor man's cocaine" made in a trailer, but a high -purity product of sophisticated clandestine chemistry controlled by international cartels. This mirrored the actual epidemiological shift where clandestine labs moved from domestic kitchens to massive industrial facilities in Mexico.

Furthermore, the show popularized the "Blue Sky" myth —the idea that high -purity methamphetamine is blue. In reality, pure methamphetamine is colorless or white; a blue tint usually indicates the presence of impurities or the intentional addition of food coloring by dealers seeking to "brand" their product. This "branding" effect has been observed in 2026 forensic seizures, where dealers add dyes to exploit the cultural cachet of the series. While the show humanized the "cook" character, it also educated a generation on the devastating physical toll of the drug (e.g., the "meth mouth" and skin sores depicted in the character Wendy), yet it simultaneously created a dangerous "prestige" around high -potency D -

methamphetamine that clinicians still contend with today (Quinones, 2024).

## 8.7 Adulterants: Levamisole, Fentanyl, and Counterfeit Pills

The "purity" of the 2026 stimulant market is an illusion. Adulteration is now systemic.

- **Levamisole (The "Skin -Rot" Adulterant):** Approximately 80% of cocaine seized in 2025 - 2026 contains levamisole, a veterinary dewormer. In humans, levamisole causes **Agranulocytosis** (a severe drop in white blood cells) and necrotic skin ulcerations, particularly on the ears and nose.
- **The "Accidental" Fentanyl Exposure:** Because of shared processing equipment in clandestine labs, cocaine is frequently cross -contaminated with fentanyl. For a stimulant user with no opioid tolerance, this result is almost always a fatal overdose.
- **Counterfeit Prescription Stimulants:** Due to widespread legal shortages of Adderall and Ritalin in 2023-2026, many students and professionals have turned to the "Digital Pharmacy" (social media). These black-market "Addys" are almost universally composed of methamphetamine and fentanyl -pressed filler, leading to a new demographic of "accidental" addicts (DEA, 2024).

## 8.8 Advanced Health Effects: Cardiovascular Crisis and Hyperthermia

- **Cardiovascular Hammering:** Cocaine causes intense "vasoconstriction" (narrowing of blood vessels) while simultaneously spiking heart rate and blood pressure. This creates a "hammering" effect on the heart wall, leading to myocardial infarction (heart attack) and aortic dissection even in physically fit, young adults.
- **Neurotoxic Hyperthermia:** Methamphetamine use can cause the body's internal temperature to skyrocket to 105 –108 degrees Fahrenheit. This "cooking" of the brain is a primary driver of permanent neurological damage and acute organ failure.
- **"Meth Mouth" and Formication:** Methamphetamine causes severe xerostomia (dry mouth) and "bruxism" (teeth grinding). Combined with the drug's acidity, this leads to rapid dental liquefaction. Furthermore, the sensation of "Meth Mites" (formication) causes users to pick at their skin, leading to chronic MRSA infections.

## 8.9 Forensic Detection and Clinical Markers: Excited Delirium

- **Metabolic Targets:** For cocaine, the target for urine screening is **Benzoyllecgonine** , a metabolite that remains detectable for days after the parent drug has left the system.
- **Hyperactive Delirium (2026 Clinical Standard):** Clinicians are now trained to recognize "Hyperactive Delirium with Severe Agitation." Symptoms include superhuman strength, imperviousness to pain, and extreme hyperthermia. This is a life -threatening medical emergency. In 2026, the standard of care is immediate sedation (typically with Ketamine or Midazolam) and aggressive cooling to prevent cardiac arrest (Frontiers in Psychiatry, 2025).

## 8.10 Treatment: Contingency Management and the 2026 Vaccine Research

Unlike opioids, there are currently **no FDA - approved medications** (like methadone) to treat stimulant use disorder.

9. **Contingency Management (CM):** This is the only evidence-based treatment with high efficacy rates. It utilizes "motivational incentives" (tangible rewards or vouchers) to reinforce negative drug screens. In 2026, federal guidelines have increased the allowable value of these rewards due to their overwhelming success in stimulant recovery (NIDA, 2024).
10. **The Matrix Model:** A 16-week intensive behavioral program that combines group therapy, individual counseling, and family education.
11. **The Stimulant Vaccine:** In 2026, Phase III clinical trials are underway for a "Cocaine Vaccine" that produces antibodies to trap the drug in the bloodstream, preventing it from reaching the brain. While not yet standard, it represents the future of stimulant treatment.

## 8.11 Stimulant Street Terms and Slang (2026)

Term	Substance/Context
Work / Girl / White	Common terms for cocaine powder.
Clear / Glass / Tina	Common terms for high -purity crystal methamphetamine.
Goofball	The "Fourth Wave" hallmark: Methamphetamine + Fentanyl.
Speedball	The classic combination: Cocaine + Fentanyl (or Heroin).
Clouding / Twirling	The act of vaping or smoking methamphetamine in a glass pipe.
Bumping	Consuming small "bumps" of powder via the nose throughout the day.
Over - amping	A non-fatal stimulant overdose characterized by extreme paranoia and physical distress.

## 8.12 Clinical Summary for the Professional

Addiction professionals must approach stimulants with high clinical acuity. Because of the P2P - driven potency, the window between "recreational use" and "permanent psychosis" has narrowed. Recovery planning should include a **Neuropsychological Assessment** to determine if the patient has the executive function required for "talk therapy," or if they require a "stabilization phase" focusing on biological restoration and basic cognitive functioning first.

# 9. Prescription Drugs: Facts & Figures

## 9.1 Overview: The Counterfeit Paradigm Shift

In the 2026 clinical landscape, the term "prescription drug abuse" has undergone a fundamental transformation. For decades, the primary concern for addiction professionals was the **diversion** of legitimate pharmaceuticals —the "cabinet raiding" era where individuals accessed diverted OxyContin, Vicodin, or Xanax from family members or through "doctor shopping." However, in 2026, the illicit market is defined by the **Counterfeit Era**.

The majority of substances sold on the street, through social media, or via encrypted messaging apps as "prescription pills" (e.g., Xanax, Adderall, Percocet) are, in reality, clandestinely manufactured tablets. These products contain high -potency synthetic opioids (primarily fentanyl) or unregulated "designer" benzodiazepines (such as bromazolam or etizolam). This shift represents a high -acuity risk for the professional: a patient reporting a "Xanax problem" may clinically present with a fentanyl overdose, and a student seeking "Adderall" for academic performance may inadvertently consume a lethal dose of methamphetamine.

## 9.2 Extent of Use: Monitoring the Future (MTF) Data

Adolescent trends in 2026 show a significant decline in the misuse of legitimate diverted pharmaceuticals due to tighter prescribing regulations (PMP systems), but a sharp increase in the availability and lethality of counterfeits.

Percent of Students Reporting Prescription Drug Misuse, 2023 - 2024

Drug Category	8th Grade	10th Grade	12th Grade	2026 Trend
Any	4.2%	7.8%	9.4%	Stable

<b>Prescription Drug</b>				
<b>Sedatives/Tranquilizers</b>	1.1%	2.0%	3.1%	Declining (Diverted)
<b>Prescription Stimulants</b>	2.1%	3.5%	4.2%	Rising (Counterfeit)
<b>Counterfeit "M30" Pills</b>	0.8%	1.5%	1.9%	High - Acuity Risk

*Data Source: University of Michigan, Monitoring the Future 2023 -2024 Data Release; SAMHSA 2024.*

### 9.3 The Telehealth Stimulant Surge and the "Digital Pharmacy"

One of the most significant contributors to the 2026 stimulant crisis was the "Telehealth Surge" that began in 2020 and peaked in 2023 –2024. This era was defined by the intersection of loosened federal regulations and the rise of venture -capital -backed tel ehealth startups.

#### 9.3.1 The Loosening of the Ryan Haight Act

During the COVID-19 public health emergency, the DEA suspended enforcement of the Ryan Haight Act, which previously required an in -person medical evaluation before a controlled substance could be prescribed. This allowed for the rapid expansion of remote A DHD diagnoses and the widespread prescribing of Adderall (amphetamine salts) and Ritalin (methylphenidate) via smartphone apps.

#### 9.3.2 The Shortage and the Illicit Pivot

By 2023, the sudden explosion in demand, combined with manufacturing quotas and supply chain failures, led to a multi -year national shortage of ADHD medications. Patients who had been stabilized on telehealth -prescribed stimulants suddenly found themselves unable to fill their prescriptions at local pharmacies.

- **The Transition to the Dark Web:** This "forced abstinence" drove a significant portion of the population toward the illicit market.
- **The Methamphetamine -Pressed "Addy":** Cartels exploited this vacuum by flooding the market with orange tablets pressed to look identical to 30mg Adderall. DEA forensic analysis in 2025 confirmed that **over 95% of black -market "Adderall"** contains methamphetamine and/or fentanyl rather than legitimate amphetamine salts. This has introduced methamphetamine to a demographic (students and white -collar

professionals) that would traditionally have avoided "hard" illicit stimulants.

## 9.4 Sedative -Hypnotics: The Benzodiazepine Crisis

Benzodiazepines (e.g., Xanax, Valium, Ativan) remain a pillar of the prescription market, primarily for the treatment of generalized anxiety and insomnia. In 2026, the clinical focus has moved toward the synergy of CNS depression and the emergence of **Designer Benzos**.

### 9.4.1 The Rise of Bromazolam and the "Non - Opioid Overdose"

As federal oversight on traditional benzodiazepines increased, clandestine labs pivoted to unregulated analogs.

18. **Bromazolam and Etizolam:** These substances are often found in counterfeit "Xanax" bars. They are significantly more potent and have much longer half-lives than alprazolam.
19. **The Naloxone Barrier:** In 2026, many "overdoses" involve a combination of fentanyl and designer benzodiazepines. Because naloxone only works on opioid receptors, it will restore the patient's breathing but will not "wake them up" from the profound sedative effect of the benzodiazepine. This leads to a dangerous state of prolonged unconsciousness and aspiration risk.

### 9.4.2 The "GABAA Trap" and Seizure Risks

Benzodiazepines act as **positive allosteric modulators** of the GABAA receptor. Chronic use leads to the downregulation (turning off) of these receptors.

- **The Withdrawal Crisis:** Unlike opioids, which are miserable but rarely fatal in withdrawal, benzodiazepine withdrawal is a medical emergency. The sudden removal of the "braking" effect of GABA leads to a "Glutamate Storm," which can trigger grand mal seizures, status epilepticus, and death.
- **Clinical Standard 2026:** Abrupt "cold turkey" detoxification from benzodiazepines is strictly contraindicated. The **Ashton Manual** protocols, which involve transitioning the patient to a long-acting benzodiazepine (like Diazepam) for a slow, months-long taper, remain the gold standard for stabilizing the central nervous system.

## 9.5 The Counterfeit Pill Crisis: "One Pill Can Kill"

The 2026 professional must be an expert in identifying the "M30" or "Blues" phenomenon, which has replaced heroin as the primary entry point for opioid use.

- **Industrial Manufacturing:** Cartels utilize industrial-grade pill presses to mimic the appearance, color, and imprints of 30mg Oxycodone tablets (the "M30" Mallinckrodt brand).
- **The "Chocolate Chip Cookie" Effect:** Clandestine labs lack the precision of pharmaceutical manufacturing. Fentanyl is often unevenly mixed into the "dough" of the tablet filler. This creates "hot spots"—one pill from a batch may contain no fentanyl, while the next pill

contains 5mg, a dose capable of killing an adult with no tolerance.

- **Psychological Deception:** By using the pill form, cartels capitalize on the "Safety Bias." Users perceive a tablet as being cleaner, safer, and less "addictive" than a powder or a needle, allowing for easier initiation into opioid use among adolescents.

## 9.6 Advanced Toxicology and the Detection Gap

A major challenge for 2026 clinicians and criminal justice professionals is that standard drug testing technology has not kept pace with clandestine chemistry.

- **Immunoassay Failures:** Standard "BZO" (Benzodiazepine) urine screens utilize antibodies calibrated for older drugs like Valium or Xanax. They frequently return a **false negative** for designer analogs like Etizolam or Flualprazolam.
- **Confirmatory Testing:** A "negative" drug screen in a patient showing clear signs of sedative intoxication (slurred speech, staggering gait, pinpoint pupils) must be followed by **LC-MS/MS** (Liquid Chromatography - Mass Spectrometry) confirmatory testing to identify the specific synthetic compounds involved.
- **The Testing Windows:**
  - **Short -acting (Xanax):** 2–3 days in urine.
  - **Long -acting (Valium/Designer Analogs):** Up to 30 days due to storage in fatty tissues.
  - **Fentanyl -Pressed Pills:** Because these contain fentanyl, the narrow 24 -hour window for fentanyl detection is the primary forensic priority.

## 9.7 Clinical Management: The Integrated Standard

Management of prescription drug misuse requires a "Double -Stabilization" approach.

- **Addressing Co -Occurring Disorders:** Data indicates that approximately 70% of individuals misusing prescription sedatives or stimulants have an underlying, often untreated, psychiatric condition (anxiety, ADHD, or PTSD). Treatment must integrate behavioral therapy with psychiatric management .
- **MOUD for Prescription Opioids:** For those dependent on counterfeit or diverted opioids, the 2026 standard has moved entirely away from "short -term detox" toward long -term **Medication for Opioid Use Disorder (MOUD)** using buprenorphine. This stabilizes the reward circuitry and reduces the risk of death by 50% (Volkow & Koob, 2024).
- **Cognitive Remediation:** Chronic prescription drug misuse results in significant executive function impairment —often described as "brain fog." Recovery plans must include cognitive exercises to help patients regain memory, focus, and decision -making abilities.

## 9.8 Prescription Drug Street Terms (2026 Update)

Term	Substance/Context
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<b>Blues / M30s</b>	Counterfeit fentanyl -pressed oxycodone tablets.
<b>Bars / Hulks / Ladders</b>	Street names for alprazolam (Xanax) based on pill shape/color.
<b>Addys / Beans</b>	Prescription stimulants (increasingly counterfeit methamphetamine).
<b>Z- Bars</b>	Counterfeit Xanax bars containing the designer benzo Bromazolam.
<b>Scripting</b>	The act of forging prescriptions or manipulating doctors for pills.
<b>Pharm - Party</b>	A dangerous ritual where various pills are mixed in a bowl and consumed at random.
<b>The "Digital Pharmacy"</b>	Telegram, Snapchat, or Instagram accounts used to sell counterfeit pills.

### 9.9 Clinical Summary for the Professional

The contemporary addiction professional must operate under the assumption that **the pill form is no longer a guarantee of pharmaceutical safety**. The "one pill can kill" reality is not a slogan; it is a clinical fact driven by the saturation of the market with synthetic analogs. Every prescription drug case must be screened for fentanyl, every sedative case must be monitored for seizure risk, and every stimulant case must be evaluated for the cardiovascular impact of methamphetamine.

## 10. Steroids and Performance - Enhancing Drugs (PEDs): Facts & Figures

### 10.1 Overview: The Shift to Performance and Image Enhancing Drugs (PIEDs)

In the contemporary clinical environment, the category of "steroids" has evolved into a broader classification known as **Performance and Image Enhancing Drugs (PIEDs)**. While traditional Anabolic-Androgenic Steroids (AAS) remain a staple of the illicit market, the landscape is now

dominated by a new generation of synthetic compounds, including **SARMs**, peptides, and metabolic modulators.

Unlike the opioid or stimulant crises, which are often driven by a desire for euphoria or escape, PIED use is primarily driven by **body image pathology** and the pressure of the "attainability myth" perpetuated by social media fitness culture (Pope et al., 2024). For the addiction professional, this requires a specialized understanding of both the physiological impact of hormonal manipulation and the underlying psychological complex known as Muscle Dysmorphia.

## 10.2 Extent of Use: Monitoring the Future (MTF) Data

While often overshadowed by more "visible" drugs, PIED use remains a significant public health concern, particularly among adolescent males and young adults entering the military or competitive athletics.

### Percent of Students Reporting Steroid Use, 2023 - 2024

Grade	Past Month	Past Year	Lifetime	Trend
8th Grade	0.3%	0.7%	1.1%	Stable
10th Grade	0.4%	0.8%	1.4%	Stable
12th Grade	0.5%	1.1%	2.0%	Stable

*Data Source: University of Michigan, Monitoring the Future 2024 Data Release; SAMHSA.*

**Clinical Note:** These figures likely underrepresent the true extent of use, as they often do not account for the consumption of **SARMs**, which are frequently marketed as "legal supplements" rather than steroids, leading to significant self-reporting bias (NIDA, 2024).

## 10.3 Classification of Modern PIEDs

### 10.3.1 Anabolic - Androgenic Steroids (AAS)

AAS are synthetic derivatives of the male hormone testosterone. They are designed to maximize the **Anabolic** (muscle-building) effects while minimizing the **Androgenic** (masculinizing) effects, though it is biologically impossible to decouple the two entirely.

- **Injectables:** Testosterone Cypionate, Deca -Durabolin (Nandrolone), and the extremely potent **Trenbolone** (often referred to as "Tren").

- **Orals:** Anadrol, Winstrol (Stanozolol), and Dianabol (D -Bol). Orals are particularly dangerous due to their "C -17 alpha-alkylation," which allows them to survive the first pass through the liver but causes extreme hepatotoxicity (Achar et al., 2010).

### 10.3.2 SARMs: The "Selective" Frontier

Selective Androgen Receptor Modulators (SARMs) represent the most significant market shift in the last decade. Compounds like **Ostarine (MK -2866)** and **Ligandrol (LGD -4033)** are designed to bind selectively to androgen receptors in muscle and bone tissue without affecting the prostate or liver.

20. **The Gray Market:** SARMs are sold openly online as "Research Chemicals" or "Not for Human Consumption."
21. **The Clinical Reality:** Despite the "selective" marketing, SARMs cause significant suppression of natural testosterone and carry many of the same cardiovascular risks as traditional steroids (FDA, 2023).

### 10.3.3 Peptides and Growth Factors

This category includes **Human Growth Hormone (hGH)** and newer peptides like **BPC-157** or **Ipamorelin**. These are used to accelerate recovery and increase fat metabolism. While they do not provide the same raw mass as AAS, they are often "stacked" to create a leaner, more "aesthetic" physique.

## 10.4 The Neurobiology of "Roid Rage" and Dependence

The impact of PIEDs on the brain is distinct from that of classic drugs of abuse. AAS do not produce an immediate dopamine "rush" in the Nucleus Accumbens, which is why many users deny they have an addiction.

### 10.4.1 The GABAA and Glutamate Interaction

High doses of AAS modulate the **GABAA** and **Glutamate** receptors in the amygdala —the brain's emotional processing center (Volkow & Koob, 2024). Chronic exposure leads to:

- **Hyper - reactivity:** An increased sensitivity to perceived threats or social slights, manifesting as "Roid Rage."
- **Neurotoxicity:** AAS induce apoptosis (cell death) in hippocampal neurons, which can lead to long - term deficits in memory and emotional regulation (Basaria, 2010).

### 10.4.2 The HPTA Axis Shutdown

The brain's Hypothalamic - Pituitary - Testicular Axis (HPTA) operates on a negative feedback loop. When a user introduces synthetic testosterone, the brain detects the excess and shuts down the body's natural production. This leads to the characteristic "crash" during the withdrawal phase, characterized by deep depression, suicidal ideation, and sexual dysfunction (SAMHSA, 2024).

## 10.5 Pathophysiology: The Systemic Cost of Potency

- **Cardiovascular Destruction:** Steroids increase LDL (bad cholesterol) and decrease HDL (good cholesterol) to near -zero levels. More critically, they cause **Left Ventricular Hypertrophy** —an enlargement of the heart wall that can lead to sudden cardiac arrest in users as young as their 20s (Achar et al., 2010).
- **Hepatic Impact:** Oral AAS cause "Peliosis Hepatis"—the formation of blood -filled cysts in the liver which can rupture and cause fatal internal bleeding.
- **Endocrine Disruption:** In males, excess testosterone is converted to estrogen via the **Aromatase** enzyme, leading to **Gynecomastia** (the development of female breast tissue). In females, use leads to irreversible **Virilization** , including deepening of the voice, clitoral enlargement, and facial hair growth.

## 10.6 The Psychology of Use: Muscle Dysmorphia

A core component of PED addiction is **Muscle Dysmorphia** , often called "Reverse Anorexia" or "Bigorexia" (Pope, Gruber, & Choi, 1997).

- **The Pathology:** Individuals perceive themselves as small or weak even when they are exceptionally muscular.
- **The Cycle:** This distorted body image drives the "Stacking" and "Cycling" behaviors. The user becomes dependent on the drug not for euphoria, but to maintain a body image that they feel is never "good enough."

## 10.7 Forensic Detection and the "Clear" Market

Standard employment drug screens do **not** detect PIEDs. Testing for steroids requires highly specialized Liquid Chromatography -Mass Spectrometry (LC -MS).

12. **Testing Challenges:** Because many SARMs and designer steroids are chemically similar to natural hormones, labs must look for specific synthetic metabolites.
13. **The Rise of "TRT" Cloaking:** Many users in professional settings now claim to be on "Testosterone Replacement Therapy" (TRT) to justify a positive test for testosterone, even when their levels are in the "supraphysiological" range.

## 10.8 Clinical Management: Post -Cycle Therapy (PCT) and Recovery

Treatment for PIED use disorder must be medically supervised because of the risk of HPTA shutdown.

- **Medical Stabilization:** Clinicians may use medications like **Clomiphene** or **HCG** to "restart" the body's natural testosterone production during a taper (SAMHSA, 2024).
- **Psychiatric Integration:** Depression during the "off -cycle" is high-acuity. SSRIs and intensive behavioral therapy for Muscle Dysmorphia are essential.
- **Hormone Monitoring:** Ongoing blood work is required to monitor liver enzymes, lipid panels, and hormonal stabilization.

## 10.9 PIED Street Terms and Slang

Term	Definition/Context
Juice / Gear / Sauce	General terms for anabolic steroids.
Tren / The King	Refers to Trenbolone, known for extreme strength and side effects.
D-Bol	Dianabol; the most common oral steroid used by beginners.
Stacking	Taking two or more different PIEDs simultaneously.
Cycling	Taking drugs for a period (e.g., 8–12 weeks), followed by a break.
Blast and Cruise	A pattern of high-dose use followed by "maintenance" doses rather than stopping.
Gyno / Bitch Tits	Street terms for gynecomastia.
SARMs / Liquid Gold	Often refers to the liquid-suspended SARMs sold online.

## 10.10 Clinical Summary for the Professional

The addiction professional must view PIED use through a **biopsychosocial lens**. It is a disorder characterized by endocrine disruption, neurobiological aggression, and deep-seated body image pathology. Screening for steroids should be mandatory for any client presenting with sudden, inexplicable changes in muscle mass, severe cystic acne, or "unprovoked" aggressive outbursts. Recovery is not just about stopping the drug; it is about restoring the body's hormonal homeostasis and the patient's psychological self-image (Pope et al., 2024).

# 11. Club Drugs: Facts & Figures

## 11.1 Overview: The Shifting Paradigm of "Party" Drugs

In the contemporary clinical landscape, the category of "Club Drugs" has evolved far beyond the rave subculture of the 1990s. This group represents a pharmacologically diverse set of

substances—including empathogens (MDMA), dissociatives (Ketamine), and sedative-hypnotics (GHB/Rohypnol)—primarily used to enhance social connection, alter sensory perception, or induce states of detachment.

A major shift in the current era is the **"Medicalization of the Macabre."** Substances that were once strictly viewed through the lens of forensic toxicology are now being integrated into clinical settings as breakthrough therapies for treatment-resistant depression and Post-Traumatic Stress Disorder (PTSD). For the addiction professional, this creates a "messaging paradox": a substance may be a life-saving medicine in a controlled clinical environment while remaining a high-risk neurotoxin in the unregulated illicit market (Volkow & Koob, 2024).

### 11.2 Extent of Use: Monitoring the Future (MTF) Data

While the use of traditional club drugs like MDMA has stabilized, the misuse of Ketamine and the emergence of high-potency synthetic cocktails have seen a notable uptick in older adolescent and college-aged populations.

Percent of Students Reporting Club Drug Use, 2023 - 2024

Drug Category	8th Grade	10th Grade	12th Grade	Trend
MDMA (Ecstasy/Molly)	0.6%	1.4%	2.1%	Stable
Ketamine (Special K)	0.4%	0.7%	1.2%	Rising
GHB	0.1%	0.2%	0.3%	Low/Stable
Rohypnol	0.1%	0.2%	0.3%	Low/Stable
Pink Cocaine (Tusi)	<0.1%	0.3%	0.6%	Emerging

Data Source: University of Michigan, Monitoring the Future 2024 Data Release; SAMHSA.

### 11.3 MDMA (3,4-Methylenedioxymethamphetamine)

MDMA acts as both a stimulant and a mild hallucinogen. It is chemically classified as an **empathogen** or **entactogen** because of its unique ability to increase feelings of emotional openness and communal bonding.

### 11.3.1 Neurobiology: The Serotonin and Oxytocin Surge

MDMA works by reversing the direction of the serotonin transporter (SERT), triggering a massive flood of **Serotonin** into the synapse. It also moderately increases Norepinephrine and Dopamine.

- **The Bonding Hormone:** Unique to MDMA is its ability to stimulate the release of **Oxytocin** from the hypothalamus. This accounts for the profound pro-social effects and the "hug drug" reputation.
- **The "Tuesday Blues":** Because the brain has a finite supply of serotonin, a single high dose of MDMA can deplete these stores for up to a week. This results in severe post-use depression and irritability, often referred to by users as "Suicide Tuesday."

### 11.3.2 The Clinical vs. Illicit Divide

In the current medical landscape, pharmaceutical-grade MDMA is utilized in highly structured psychotherapy sessions to help PTSD patients process trauma without being overwhelmed by fear. However, street-level "Molly" is rarely pure. Forensic analysis frequently reveals that Molly contains no MDMA at all, instead consisting of synthetic cathinones (Bath Salts), methamphetamine, or even fentanyl (NIDA, 2024).

## 11.4 Ketamine: The NMDA Dissociative

Ketamine is a dissociative anesthetic that has seen a massive clinical and illicit resurgence in the present era.

### 11.4.1 Mechanism of Action: NMDA Antagonism

Unlike most drugs of abuse that target the dopamine system, Ketamine is a non-competitive **NMDA receptor antagonist**. By blocking glutamate signals, it disconnects the thalamus from the cerebral cortex, creating a state of "dissociative anesthesia."

22. **The "K-Hole":** At high doses, users experience a total loss of sensory awareness and motor control, often described as a near-death or "out-of-body" experience.
23. **Esketamine (Spravato):** The FDA approval of S-ketamine for depression has destigmatized the drug, but illicit use remains dangerous due to the risk of "accidental" injuries during dissociative states.

### 11.4.2 Pathophysiology: Ketamine Cystitis

A specific clinical marker of chronic Ketamine use is **Ketamine-Induced Cystitis**. The drug's metabolites are highly caustic to the bladder wall. Chronic users experience "K-cramps," frequent urination, and bloody urine. In severe cases, the damage is permanent, necessitating surgical bladder removal or the lifelong use of a catheter (Achar et al., 2010).

## 11.5 GHB and Rohypnol: The Sedative-Hypnotics

These substances are potent CNS depressants often associated with "Drug -Facilitated Sexual Assault" (DFSA) due to their ability to induce rapid unconsciousness and anterograde amnesia.

### 11.5.1 GHB (Gamma -hydroxybutyrate)

GHB acts primarily on the **GABAB receptor** .

- **The Synergy Risk:** GHB has an extremely narrow "therapeutic window." The difference between a social high and a fatal coma can be as little as one extra milliliter. When combined with alcohol, the respiratory depression is synergistic and often fatal.
- **Forensic Challenge:** GHB is naturally occurring in the human body and has a half -life of less than 30 minutes. It is usually cleared from blood and urine within 12 hours, making forensic detection in assault cases nearly impossible unless samples are taken immediately.

### 11.5.2 Rohypnol (Flunitrazepam)

A powerful benzodiazepine (the "Roofie") that is 10 times more potent than Valium. While not legally prescribed in the U.S., it is smuggled across borders. Modern tablets often contain a blue dye that becomes visible when dissolved in clear liquids, though this is not a universal safety feature across all generic illicit versions.

## 11.6 The Rise of "Tusi" (Pink Cocaine)

A major emerging threat in the modern illicit market is **"Tusi." \* The Chemical Cocktail:** Despite the name, Tusi rarely contains cocaine. It is a synthetic mixture, typically consisting of Ketamine, MDMA, and caffeine, dyed bright pink with food coloring and flavored with strawberry scents.

- **Predictability Risk:** Because Tusi is a "trash-can drug" (a mix of whatever is available in the lab), users have no way of knowing the ratio of dissociative to stimulant. This leads to high rates of "bad trips," cardiac distress, and non -responsive sedation.

## 11.7 Advanced Health Risks: Hyperthermia and Hyponatremia

- **Hyperthermia:** MDMA and other club drugs interfere with the body's thermoregulation. In crowded, poorly ventilated club environments, body temperatures can spike to dangerous levels, leading to Rhabdomyolysis (muscle breakdown), kidney failure, and death.
- **Hyponatremia:** Because MDMA causes the release of the antidiuretic hormone (vasopressin), users may stop urinating while simultaneously drinking excessive amounts of water to stay "safe." This leads to "water intoxication," where the brain swells (cerebral edema), which can be fatal.

## 11.8 Forensic Testing and the Detection Gap

The contemporary professional must be aware that standard 5-panel and 10-panel drug screens **do not detect** most club drugs.

- 14. **Specialized Panels:** Clinicians must specifically request an "Ecstasy/MDMA" panel to detect relevant metabolites.
- 15. **Ketamine:** Requires specialized testing for **Norketamine**.
- 16. **GHB:** Requires specialized gas chromatography which is not available in standard clinical labs.

### 11.9 Club Drug Street Terms and Slang

Term	Substance/Context
Molly / Adam / X	MDMA in powder or tablet form.
Special K / Vitamin K	Ketamine.
Liquid X / G	GHB.
Tusi / Pink Cocaine	Synthetic ketamine/MDMA cocktail.
Candy - Flipping	Co-use of MDMA and LSD.
Hippy - Flipping	Co-use of MDMA and Psilocybin.
The G - Sleep	The deep, coma-like state induced by GHB.

### 11.10 Clinical Summary for the Professional

The modern addiction professional must navigate the "Psychedelic Renaissance" with clinical balance. While we acknowledge the medical potential of MDMA and Ketamine in psychotherapy, we must remain vigilant regarding the neurotoxic, cardiovascular, and forensic risks of their illicit counterparts. Treatment must focus on acute medical stabilization, long-term serotonin restoration, and addressing the profound post-use mood disruptions that characterize this class of drugs (SAMHSA, 2024).

## 12. Cocaine & Crack: Facts & Figures

### 12.1 Overview: The Resurgence and the "Fourth Wave"

In the current clinical landscape, cocaine remains one of the most significant public health challenges in the United States. While much of the recent focus has been on synthetic opioids,

cocaine is currently in a "Global Resurgence" phase. Production in South America has reached record highs, resulting in a supply that is both cheaper and purer than at any point in the last three decades (United Nations Office on Drugs and Crime [UNODC], 2024).

For the contemporary professional, cocaine must be understood through the lens of the **"Fourth Wave"** of the overdose crisis. This era is defined by the pervasive cross-contamination of the cocaine supply with synthetic opioids (fentanyl). For many users—particularly those who consider themselves "recreational" powder cocaine users—the lack of opioid tolerance means that even a trace amount of fentanyl contamination can result in a rapid, fatal overdose (Volkow & Koob, 2024).

## 12.2 Extent of Use: Monitoring the Future (MTF) Data

While the "crack epidemic" of the 1980s has faded from the headlines, usage rates for cocaine remain stable, with a notable shift in how the drug is perceived by younger populations.

### Percent of Students Reporting Cocaine Use, 2023 - 2024

Grade	Past Month	Past Year	Lifetime	Trend
8th Grade	0.2%	0.6%	1.0%	Stable
10th Grade	0.4%	1.0%	1.6%	Stable
12th Grade	0.7%	1.8%	2.5%	Stable

*Data Source: University of Michigan, Monitoring the Future 2024 Data Release; SAMHSA.*

**Clinical Insight:** While prevalence rates are lower than during the 1980s peak, the **mortality rate** associated with cocaine use has spiked. This is not due to an increase in users, but due to the increased lethality of the adulterated supply (Volkow & Koob, 2024).

## 12.3 Neurobiology: The Dopamine Reuptake Trap

Cocaine is a powerful central nervous system (CNS) stimulant that derives its addictive potential from its direct impact on the brain's reward architecture, specifically the **Mesolimbic Dopamine System** (Volkow et al., 2023).

### 12.3.1 Mechanism of Action

Unlike methamphetamine, which triggers the *release* of dopamine, cocaine acts primarily as a **reuptake inhibitor**.

- **Blocking the Transporter:** Cocaine binds to the dopamine transporter (DAT) on the pre-synaptic neuron.
- **Synaptic Accumulation:** By blocking the "vacuum cleaner" that normally removes dopamine from the synapse, cocaine causes dopamine to accumulate to supraphysiological levels.
- **Continuous Signaling:** This flood of dopamine continuously stimulates the post-synaptic receptors, producing the characteristic euphoria, increased energy, and hyper-vigilance.

### 12.3.2 The "Crash" and Anhedonia

Because cocaine prevents the recycling of dopamine, the brain's supply of the neurotransmitter is eventually exhausted. When the drug wears off, the user enters a state of "dopamine bankruptcy." This leads to **Anhedonia** —the inability to feel pleasure from natural rewards (eating, social interaction, sex) —which drives the compulsive "binge" behavior to restore the high (Koob & Volkow, 2024).

## 12.4 Pharmacokinetics: Powder vs. Crack

The primary difference between cocaine hydrochloride (powder) and cocaine base (crack) is the route of administration and the speed of onset.

24. **Cocaine Hydrochloride (Powder):** Usually insufflated (snorted) or injected. Snorting results in a slower onset (3–5 minutes) as the drug must cross the mucous membranes. The high lasts 15–30 minutes.
25. **Cocaine Base (Crack):** Created by processing powder cocaine with baking soda and water to remove the hydrochloride salt. This allows the drug to be smoked.
26. **The "Flash":** Inhaled crack reaches the brain in less than 8 seconds, producing an immediate, intense "flash" or "rush." However, the high is exceptionally short (5–10 minutes), leading to rapid-fire binge cycles and a much higher potential for immediate dependence.

## 12.5 Adulterants: The Levamisole and Fentanyl Syndemic

In the modern market, "pure" cocaine is virtually non-existent. Two specific adulterants define the current toxicological risk.

### 12.5.1 Levamisole (The "Skin-Rot" Adulterant)

Approximately 80% of the cocaine supply is currently adulterated with **Levamisole**, a veterinary dewormer (Larocque & Hoffman, 2023).

- **The Clinical Risk:** In humans, levamisole causes **Agranulocytosis** —a severe depletion of white blood cells that leaves the user vulnerable to life-threatening infections.
- **Necrosis:** It also causes "Vasculitis," leading to purple, necrotic skin patches, typically on the earlobes, nose, and cheeks. This is a primary diagnostic marker for the contemporary

cocaine user (Achar et al., 2010).

### 12.5.2 Fentanyl Contamination

The most lethal trend in the current market is the presence of fentanyl in cocaine. This is often "accidental" cross-contamination occurring in clandestine labs that process multiple substances. Because cocaine users often do not use opioids, they have no physiological tolerance, making even a microscopic amount of fentanyl fatal (SAMHSA, 2024).

### 12.6 Systemic Health Effects

- **Cardiovascular "Hammering":** Cocaine is unique because it is both a stimulant (increasing heart rate) and a vasoconstrictor (narrowing blood vessels). This "squeezes" the heart while forcing it to work harder, leading to myocardial infarction (heart attack) and aortic dissection even in young adults (Achar et al., 2010).
- **"Crack Lung":** Smoking crack causes acute thermal injury to the airways and alveolar hemorrhage, manifesting as chest pain, fever, and coughing up blood.
- **Neurological Impact:** Chronic use is linked to "Micro-strokes" and a significant increase in the risk of hemorrhagic stroke.

### 12.7 Clinical Crisis: Excited Delirium

Contemporary clinicians must be trained to recognize **Hyperactive Delirium with Severe Agitation** (formerly termed Excited Delirium).

- **Symptoms:** Extreme agitation, superhuman strength, hyperthermia (body temperatures over 105°F), and a total lack of response to verbal de-escalation.
- **Management:** This is a medical emergency. The risk of sudden cardiac arrest is high. Current standards of care involve rapid chemical sedation (e.g., Ketamine or Midazolam) and aggressive physical cooling to prevent multi-organ failure (Frontiers in Psychiatry, 2025).

### 12.8 Forensic Toxicology and Detection

17. **Metabolite Testing:** Cocaine has a very short half-life (~1 hour). Therefore, drug screens do not look for cocaine itself, but for its primary metabolite, **Benzoyllecgonine**.

18. **Detection Windows:**

- **Urine:** Detectable for 2 –4 days.
- **Chronic Use:** In heavy users, metabolites can be sequestered in fatty tissues and remain detectable for up to 10 days.
- **Hair:** Provides a 90-day window and is the gold standard for verifying long-term abstinence in legal or high-stakes employment settings.

### 12.9 Treatment: Behavioral Mastery and the Medical Gap

A primary challenge for the contemporary addiction professional is the existence of the

"Medical Gap." Unlike Opioid Use Disorder (OUD) or Alcohol Use Disorder (AUD), there are currently **no FDA-approved medications** (such as methadone or naltrexone) that are indicated for the treatment of Cocaine Use Disorder. This puts the entire burden of recovery on high-acuity behavioral interventions and neuro-rehabilitation.

### 12.9.1 Contingency Management (CM): The Behavioral Gold Standard

The single most effective intervention for stimulant use is **Contingency Management (CM)**. CM is based on the principles of operant conditioning, specifically positive reinforcement (Petry, 2024). Because cocaine "hijacks" the brain's reward system, standard talk therapy is often insufficient in early recovery because the brain's internal reward signals (dopamine) are "bankrupt." CM provides an external, tangible reward to compete with the immediate reinforcement of the drug.

- **Mechanics of Reinforcement:** Patients receive "incentives" (vouchers, gift cards, or small prizes) for objective proof of drug-free behavior, typically confirmed by urine drug screens.
- **The "Escalation" Principle:** To encourage long-term abstinence, the value of the rewards typically increases with each consecutive negative drug screen. If a patient tests positive, the reward value resets to the baseline. This creates a high psychological cost for relapse.
- **Fishbowl vs. Voucher-Based:** Two common models exist. In the **Voucher-Based Reinforcement (VBR)** model, the patient receives a specific monetary value. In the **Fishbowl (Prize-Based)** model, the patient earns "draws" from a bowl for negative tests, with the chance to win prizes of varying values (from \$1 to \$100). Research indicates the Fishbowl model is highly effective and often more sustainable for community-based clinics (NIDA, 2024).
- **Policy Shifts:** In the present era, federal and state health authorities (including the OIG and HHS) have raised the "allowable caps" for these incentives, acknowledging that the clinical benefits of CM in preventing stimulant-related deaths far outweigh the cost of the rewards.

### 12.9.2 The Matrix Model: Intensive Outpatient Structure

For high-acuity cocaine and crack users, the **Matrix Model** provides the necessary structure to stabilize a compromised Prefrontal Cortex (Rawson et al., 2024). Developed specifically for stimulant users, this 16-week intensive outpatient program (IOP) integrates multiple therapeutic modalities:

- **Individual Counseling:** Focuses on immediate behavioral change and preventing "binge" triggers.
- **Early Recovery Skills Groups:** These groups meet twice weekly to teach "hunger management" for cravings and environmental avoidance.
- **Family Education Groups:** Crucial for repair, as chronic cocaine use often involves severe financial and trust-based trauma within the family unit.

- **Social Support Groups:** Transitioning the patient into "Cocaine Anonymous" (CA) or other 12-step/peer -support frameworks.
- **Relapse Prevention:** Focusing on the "Dopamine Trap" and educating the patient that they may feel emotionally "flat" (anhedonia) for 12 –18 months while their brain heals.

### 12.9.3 Cognitive -Behavioral Therapy (CBT) and CRA

While CM provides the motivation, **Cognitive -Behavioral Therapy (CBT)** provides the tools. CBT helps patients identify the "Antecedents" (triggers) and "Consequences" of their use (SAMHSA, 2024).

- **Community Reinforcement Approach (CRA):** This 24-week intervention is often paired with CM. CRA works by systematically restructuring the patient’s environment. It helps the user build a "pro-social" life where natural rewards (employment, healthy relationships, hobbies) eventually replace the chemical reward of the drug.

### 12.9.4 The Pharmacological Frontier: Research and Vaccines

Despite the current lack of FDA approval, pharmacological research is focused on three areas:

- **Glutamate Stabilization:** Researchers are investigating medications like **Topiramate** and **Modafinil** to stabilize the glutamate "storm" that causes involuntary cravings.
- **Dopamine Transporter Blockers:** Investigating substances that can "shield" the dopamine transporter from cocaine's binding without producing a high themselves.
- **The Cocaine Vaccine:** Perhaps the most promising development is the **Anti -Cocaine Vaccine** . Currently in Phase III trials, this vaccine produces antibodies that "trap" the cocaine molecule in the bloodstream, preventing it from crossing the blood -brain barrier (NIDA, 2024). If a vaccinated individual uses cocaine, they feel no effect, effectively breaking the reinforcement cycle.

### 12.9.5 Neuro -Rehabilitation: Healing the "CEO"

Because cocaine use results in significant executive function impairment, modern treatment must include **Cognitive Remediation** . This involves exercises designed to strengthen the Prefrontal Cortex, improving memory, impulse control, and decision -making. Clinicians must realize that in the first 90 days of recovery, the "CEO" of the patient's brain is offline; treatment must be directive, structured, and highly supportive until biological restoration begins (SAMHSA, 2024).

## 12.10 Cocaine Street Terms and Slang

Term	Definition/Context
Work / Girl / White	Common terms for powder cocaine.

<b>Rock / Candy / Hard</b>	Common terms for crack cocaine.
<b>Speedball</b>	The lethal combination of Cocaine and Fentanyl (or Heroin).
<b>Bumping</b>	Using small amounts of powder intranasally throughout a social event.
<b>Freebasing</b>	An older term for smoking the pure cocaine base.
<b>Cooker</b>	A person who converts powder cocaine into crack.

### 12.11 Clinical Summary for the Professional

The modern addiction professional must treat every cocaine case as a high -acuity medical risk. Due to the saturation of the supply with fentanyl and levamisole, the "recreational" user is a relic of the past. Clinical focus must remain on cardiovascular monitoring, rapid identification of hyperactive delirium, and the implementation of robust behavioral reinforcement through Contingency Management (SAMHSA, 2024).

## 13. Alcohol: Facts & Figures

### 13.1 Overview: The Legal Paradox and Societal Normalization

Alcohol remains the most widely used and misused psychoactive substance in the United States, presenting a unique challenge for the addiction professional. Unlike the illicit "street" drugs discussed in previous modules, alcohol is a legal, socially celebrated, and economically significant commodity. This normalization creates a significant barrier to treatment; many individuals do not seek help until they reach a state of advanced physiological crisis because their consumption patterns —though high -risk—are mirrored and even encouraged by their social and professional peers.

In the contemporary landscape, alcohol contributes to over 178,000 deaths annually in the U.S. (CDC, 2024). It is a leading cause of preventable death, ranking just behind tobacco and the fentanyl -driven overdose crisis. The present era is defined by a "Bi modal Trend": while overall consumption is declining among younger generations, "High -Acuity Bingeing" and alcohol -related liver disease are reaching record highs among middle -aged adults, particularly women. The economic cost of excessive alcohol consumption is estimated at over \$250 billion per year, driven largely by lost workplace productivity and healthcare expenses (Sacks et al., 2024).

## 13.2 Epidemiology: The Gen Z "Sober Curious" Shift and the Binge Paradox

A profound demographic shift is occurring in the 2020s. Data from the **Monitoring the Future (MTF)** survey and other longitudinal studies indicate that Gen Z and younger Millennials are consuming significantly less alcohol than previous generations did at the same age.

### 13.2.1 The "Sober Curious" Movement and Wellness Culture

There is a growing cultural emphasis on "wellness," "bio -hacking," and "mindful drinking." Younger populations are increasingly aware of the long -term health risks associated with alcohol, including its classification as a Group 1 carcinogen. The rise of "hangxiety" (anxiety induced by alcohol withdrawal) has become a common topic in social media discourse, leading many to adopt "Dry January" or "Sober October" as permanent lifestyle changes. Furthermore, the legalization of cannabis in many states has provided a "social substitute" that many young adults perceive as safer and more compatible with a productive lifestyle.

### 13.2.2 The Binge Paradox: High -Intensity Consumption

Despite fewer young people drinking, those who *do* drink are engaging in more dangerous patterns of consumption. The "Binge Paradox" refers to the fact that while the frequency of drinking occasions is down, the intensity of those occasions is up.

- **"Borging":** This contemporary trend involves mixing a "Blackout Rage Gallon" (BORG) —a gallon-sized container filled with half water and half vodka, flavored with liquid caffeine and electrolytes. While marketed as a "harm reduction" tool because of the added water and electrolytes, the sheer volume of ethanol (typically 750ml of vodka) consumed in a single session leads to extreme, life -threatening BAC levels (White et al., 2024).
- **Blackout Culture:** The goal for a subset of the contemporary youth population is not just intoxication, but total memory erasure (anterograde amnesia), which indicates a dangerous level of CNS suppression.

Percent of Students Reporting Alcohol Use, 2023 - 2024

Grade	Past Month Use	Binge Drinking (Past 2 Weeks)	Trend
8th Grade	5.8%	2.1%	Historical Low
10th Grade	13.5%	5.5%	Declining
12th Grade	22.1%	11.2%	Declining

*Data Source: University of Michigan, Monitoring the Future 2024 Data Release.*

### 13.3 Product Innovation: The RTD, Seltzer, and Palatability Revolution

The alcohol industry has responded to declining beer sales by innovating new product categories that "mask" the taste of ethanol, making them more accessible to non-traditional drinkers and adolescents.

#### 13.3.1 The "Health Halo" of Hard Seltzers

Products like **White Claw** and **Truly** have revolutionized the market by framing alcohol through the lens of fitness. By using "Low Calorie," "Low Carb," and "Gluten-Free" labels, these products create a "Health Halo." This psychological trick dissociates the beverage from its identity as a metabolic toxin. The clear, carbonated nature of these drinks makes them easy to consume rapidly, leading to a faster "spike" in BAC compared to beer or wine, which contain more complex carbohydrates that slow absorption.

#### 13.3.2 "Alcopops" and Palatability Engineering

Modern "Ready-to-Drink" (RTD) cocktails and "Alcopops" (e.g., **Mike's Hard Lemonade**) utilize sugar and artificial flavorings to bypass the body's natural "aversion response" to the acrid taste of ethanol.

27. **Lowering the Barrier to Entry:** For an adolescent's developing palate, the sweetness of these drinks allows for high-volume consumption before the brain can signal intoxication.
28. **The Sugar-Ethanol Synergy:** The combination of high-fructose corn syrup and ethanol creates a "double-hit" on the liver. The liver must prioritize the metabolism of ethanol (converting it into acetaldehyde), which causes the sugar to be stored as fat, leading to the rapid development of non-alcoholic fatty liver disease (NAFLD) and alcohol-associated liver disease (ALD) even in young users (Volkow & Koob, 2024).

### 13.4 Neurobiology: The GABA/Glutamate Seesaw and the Kindling Effect

Alcohol is a central nervous system (CNS) depressant that exerts its primary effects by disrupting the balance between inhibitory and excitatory neurotransmitters.

#### 13.4.1 GABA (The Brakes)

Alcohol is a **positive allosteric modulator of GABA<sub>A</sub> receptors**. It enhances the inhibitory effect of GABA, which slows down brain activity, leading to sedation, decreased motor coordination, and anxiolysis. Chronic use leads to **downregulation**—the brain reduces its natural GABA production and receptor density to compensate for the constant presence of the drug.

#### 13.4.2 Glutamate (The Gas)

Alcohol is an **NMDA receptor antagonist**. It suppresses glutamate, the brain's primary

excitatory neurotransmitter. To maintain homeostasis, the brain "upregulates" its glutamate receptors—effectively installing more "gas pedals." This is why a person with severe AUD feels "shaky" or "wired" when they stop drinking; their brain is physically over-excited.

### 13.4.3 The Kindling Effect and Neurotoxicity

The **Kindling Effect** is a critical concept for modern clinicians. It refers to the phenomenon where each subsequent cycle of alcohol withdrawal becomes more severe and more dangerous than the last. Each "detox" session without long-term stabilization sensitizes the brain's glutamate systems.

- **Clinical Result:** A patient who had mild tremors during their first detox may experience grand mal seizures or **Delirium Tremens (DTs)** during their fifth detox, even if they drank the same amount. This neuro-excitability also causes permanent damage to the hippocampus and cerebellum, leading to the "wet brain" symptoms of Wernicke - Korsakoff Syndrome (Koob & Volkow, 2024).

## 13.5 Pathophysiology: The Crisis in Women's Health and Forensic Markers

The current era has seen an unprecedented rise in alcohol-associated liver disease (ALD) among women in their 20s and 30s. This surge is driven by both sociocultural shifts in consumption and unique biological vulnerabilities.

### 13.5.1 The Biological Vulnerability and "Telescoping" Effect

Biological females face higher risks from alcohol for several distinct physiological reasons:

- **Lower Water Content:** Alcohol is water-soluble. Women generally have a lower percentage of total body water than men of equal weight, resulting in a higher concentration of alcohol in the blood even when adjusting for dosage (NIAAA, 2024).
- **Enzymatic Differences:** Women have lower levels of **alcohol dehydrogenase (ADH)**—the enzyme in the stomach lining that begins the breakdown of ethanol before it reaches the bloodstream. This means a woman absorbs approximately 30% more alcohol into her bloodstream than a man drinking the same amount.
- **The Telescoping Phenomenon:** Research indicates that women experience a "telescoping" effect, where the time between the initiation of alcohol use and the development of alcohol-related physical complications is significantly shorter than in men (White et al., 2024). This results in more rapid progression to cirrhosis, alcoholic cardiomyopathy, and cognitive decline.
- **Hormonal Interaction and Cancer Risk:** Alcohol spikes estrogen levels, which is directly linked to an increased risk of breast cancer. Even moderate consumption (one drink per day) increases the risk of breast cancer by 5% to 15% (CDC, 2024).

### 13.5.2 Advanced Forensic Markers: Direct vs. Indirect Biomarkers

Standard Breathalyzer and Blood Alcohol tests only detect alcohol while it is currently in the

system. To monitor long-term recovery, contemporary clinicians utilize a two-tiered approach to biomarkers.

- **Ethyl Glucuronide (EtG):** This metabolite can be detected in urine for up to 80 hours after consumption. It is highly sensitive, though clinicians must be aware of "false positives" from incidental exposure to hand sanitizers (NIAAA, 2024).
- **Phosphatidylethanol (PEth):** A phospholipid formed only in the presence of ethanol. PEth provides a 2-3 week "window" of consumption and is nearly impossible to "cheat" via incidental exposure, making it the current gold standard for high-stakes professional monitoring (e.g., for physicians or pilots in recovery).

## 13.6 Contemporary Treatment for Alcohol Use Disorders (AUD)

In the present era, the treatment of AUD has transitioned from a strictly behavioral "12-step" model to an **Integrated Medical - Psychosocial Model**. We no longer wait for a patient to "hit rock bottom"; instead, we intervene early using pharmacological and digital tools.

### 13.6.1 Pharmacotherapy: The Three FDA - Approved Pillars

19. **Naltrexone (Vivitrol/ReVia):** An opioid antagonist that blocks the endorphin receptors that alcohol stimulates. It "de-couples" the pleasure from the act of drinking (Petry, 2024).
  - **The Sinclair Method (TSM):** A contemporary approach where patients take Naltrexone one hour *before* they intend to drink. Over months, this leads to "pharmacological extinction," where the brain "un-learns" its craving for alcohol because the reward is blocked.
20. **Acamprosate (Campral):** Designed to "level out" the GABA/Glutamate seesaw. It reduces the "protracted withdrawal" symptoms —anxiety, insomnia, and restlessness—that typically lead to early relapse. It is most effective for patients who have already achieved abstinence.
21. **Disulfiram (Antabuse):** A "deterrent" medication that blocks the enzyme acetaldehyde dehydrogenase. If a patient drinks, toxic acetaldehyde builds up in the system, causing violent vomiting and headache.

### 13.6.2 Off -Label Pharmacological Innovations

Clinicians are increasingly utilizing medications that show high efficacy in reducing heavy drinking days:

- **Gabapentin:** Often used to manage the anxiety and sleep disturbances of early recovery.
- **Topiramate:** Acts on glutamate and GABA systems to reduce the "obsessive" quality of alcohol cravings.

## 13.7 Dual Diagnosis: The Anxiety - Depression Loop and Differential Diagnosis

Alcohol is frequently used as a form of "self-medication" for untreated anxiety or depression.

However, because alcohol is a depressant, it ultimately worsens these conditions. Clinicians must determine whether psychiatric symptoms are "primary" or "substance-induced" (Volkow et al., 2024).

### 13.7.1 Distinguishing Primary vs. Substance - Induced Symptoms

- **The Clinical Waiting Period:** Contemporary standards of care suggest that a definitive primary psychiatric diagnosis should not be made until the patient has achieved 4 to 6 weeks of medically supervised abstinence (DSM -5-TR; SAMHSA, 2024). During this window, substance -induced symptoms often begin to dissipate as the GABA and Glutamate systems return to homeostasis.
- **Longitudinal Assessment:** A primary disorder is typically indicated if symptoms preceded the onset of heavy drinking or persisted during previous periods of long -term sobriety.
- **The "Biological Trap":** Alcohol provides temporary relief from anxiety by spiking GABA, but as it wears off, the brain's compensatory glutamate surge creates "rebound anxiety" or "hangxiety." This cycle eventually renders the brain incapable of regulating mood without the substance, a state known as **allostasis** (Koob & Volkow, 2024).

### 13.8 Summary for the Addiction Professional

The contemporary professional must recognize that "the face of alcoholism" has changed. It is no longer defined strictly by the "skid row" image, but by the high -functioning professional using high-sugar RTDs, the student "borging" at a university party, and the wellness-focused seltzer drinker. Screening must be universal, and clinicians must be aware that the palatability and "Health Halo" of modern alcoholic beverages have shortened the timeline from "first use" to severe pathology (SAMHSA, 2024).

## 14. Kratom and Herbal Substances: Facts & Figures

### 14.1 Overview: The "Natural" Marketing Myth

In the contemporary clinical landscape, a new frontier of substance use has emerged involving "herbal," "botanical," or "gas station" drugs. These substances—primarily **Kratom** and **Tianeptine**—are often marketed as safe, natural alternatives to traditional pharmaceuticals or as "legal highs." However, their pharmacology is far from benign.

The present era is defined by the "Regulatory Gap." Because these substances are often sold as dietary supplements or "not for human consumption," they bypass the rigorous safety testing required for FDA-approved medications. For the addiction professional, this creates a high-acuity challenge: patients may believe they are using a "natural supplement" while they are, in biological reality, maintaining a severe opioid or GABAergic dependence.

## 14.2 Kratom (*Mitragyna speciosa*)

Kratom is a tropical tree native to Southeast Asia. In the current market, its leaves are ground into powder, encapsulated, or sold as highly concentrated liquid extracts.

### 14.2.1 Biphasic Pharmacology

Kratom is unique because its effects are dose-dependent, acting on different neurotransmitter systems based on the amount consumed:

- **Low Dose (Stimulant Effect):** At doses of 1–5 grams, the primary alkaloid **Mitragynine** acts as a stimulant, increasing energy, alertness, and sociability. It appears to interact with adenosine receptors, similar to caffeine.
- **High Dose (Opioid Effect):** At doses above 5 grams, the alkaloid **7-hydroxymitragynine** becomes dominant. This compound is a partial **mu-opioid receptor agonist**. It produces sedation, analgesia (pain relief), and euphoria nearly identical to traditional opioids (Volkow & Koob, 2024).

### 14.2.2 The Scheduling Controversy

The DEA attempted to place Kratom into Schedule I in 2016 but withdrew the proposal following intense public and legislative pushback. Currently, Kratom remains federally legal but is banned in several states and municipalities. This "gray market" status leads to significant issues with **Adulteration**; forensic testing of street-level Kratom often reveals high concentrations of heavy metals (lead/nickel) or the addition of synthetic opioids like O-desmethyltramadol to boost potency (FDA, 2024).

## 14.3 Tianeptine: "Gas Station Heroin"

Tianeptine is a tricyclic compound used as an antidepressant in some European and Latin American countries, but it is not approved for any medical use in the United States. In the modern illicit market, it is sold in convenience stores and gas stations under brand names like **Tianaa**, **Zaza Red**, or **TD Red**.

29. **Mechanism of Action:** While structurally a tricyclic, Tianeptine is a potent **full mu-opioid receptor agonist**.
30. **The Withdrawal Crisis:** Tianeptine withdrawal is reported by clinicians to be significantly more severe than traditional heroin or oxycodone withdrawal. Because it also affects glutamate and serotonin pathways, the withdrawal syndrome includes not only the standard "opioid flu" but also severe cardiovascular instability, profound depression, and suicidal ideation (NIDA, 2024).
31. **High-Acuity Risk:** Users often rapidly escalate their dosage to 100 times the therapeutic limit to achieve an opioid high, leading to rapid-onset liver toxicity and respiratory failure.

## 14.4 Phenibut: The Russian Anxiolytic

Phenibut is a synthetic GABA analog originally developed in the Soviet Union. It is marketed online as a "nootropic" (smart drug) or anxiolytic.

- **Neurobiology:** Phenibut is a **GABAB agonist**, similar to Baclofen.

- **The Dependency Trap:** Tolerance to Phenibut develops with extreme rapidity. Users who take it for anxiety often find themselves physically dependent within 7 to 10 days. Withdrawal is high-acuity and mimics benzodiazepine or alcohol withdrawal, carrying a high risk of grand mal seizures and acute psychosis (SAMHSA, 2024).

## 14.5 Forensic Detection and the Detection Gap

A primary challenge for the contemporary professional is that **none of these substances appear on standard drug screens.**

- **Kratom:** Requires a specific "Mitragynine" panel.
- **Tianeptine:** Requires specialized liquid chromatography-mass spectrometry (LC-MS) testing that is not standard in most clinical settings.
- **Clinical Marker:** A patient who presents with clear signs of opioid intoxication (pinpoint pupils, respiratory depression) but returns a "clean" 12-panel drug screen is a high-probability candidate for Kratom or Tianeptine misuse.

## 14.6 Clinical Management and Treatment: Navigating the Botanical Threshold

The clinical management of herbal and "gas station" substance use disorders requires an integrated approach that acknowledges their multi-receptor activity. Because these substances are often unregulated, clinicians must prepare for unpredictable withdrawal trajectories and high levels of psychological "denial" based on the substances' legal and "natural" status.

### 14.6.1 MAT Integration for Kratom and Tianeptine

While no specific FDA-approved medication exists exclusively for Kratom or Tianeptine cessation, the use of **Medication-Assisted Treatment (MAT)**, specifically **Buprenorphine/Naloxone (Suboxone)**, has emerged as the contemporary gold standard for stabilization (Volkow et al., 2024).

- **The Buprenorphine Anchor:** For Kratom users, Buprenorphine acts as a higher-affinity partial agonist that stabilizes the mu-opioid receptors previously occupied by mitragynines. Because Kratom has a relatively short half-life, induction can typically begin when the patient reaches a Moderate Clinical Opiate Withdrawal Scale (COWS) score of 12 or higher.
- **Tianeptine Induction Challenges:** Tianeptine presents a more complex challenge. Because it is a *full* mu-opioid agonist and also has tricyclic-like effects on serotonin and glutamate, users often consume massive quantities (up to 10 grams per day). Standard buprenorphine induction may cause **precipitated withdrawal** if not managed carefully. Recent case studies suggest that "Micro-Induction" or the **Bernese Method** may be safer for high-dose Tianeptine users, allowing for a gradual displacement of the full agonist over 7 to 10 days (SAMHSA, 2024).
- **Maintenance vs. Tapering:** For long-term recovery, many clinicians recommend a maintenance phase of at least 6 to 12 months to allow for the restoration of opioid receptor density before attempting a slow taper (NIDA, 2024).

### 14.6.2 The Phenibut GABAB Taper

The management of Phenibut dependence is a life-safety priority due to the risk of status epilepticus and acute delirium.

22. **Cross-Tapering with Baclofen:** Since Phenibut is a GABAB agonist, the most effective clinical strategy often involves cross-tapering the patient onto **Baclofen**, which has a similar receptor profile but a more predictable half-life and medical oversight.
23. **Seizure Prophylaxis:** For patients with extremely high-dose Phenibut habits, the integration of long-acting benzodiazepines (such as Diazepam) or anticonvulsants (such as Gabapentin) is necessary to manage the voltage-gated calcium channel (VGCC) excitability that characterizes Phenibut withdrawal. Unlike opioid withdrawal, Phenibut detox often requires an inpatient setting to monitor for cardiovascular instability and hallucinations (Koob & Volkow, 2024).

### 14.6.3 Managing the "Antidepressant Crash"

Tianeptine and Kratom both possess secondary mechanisms that impact mood regulation. Tianeptine, in particular, affects the reuptake of serotonin and modulates glutamate. When a user stops Tianeptine, they experience a "Crash" that is qualitatively different from standard opioid withdrawal.

- **Psychiatric Stabilization:** Patients often report profound, sudden-onset suicidal ideation and "un-real" anxiety. Clinicians must be prepared to use non-addictive psychiatric medications (such as SSRIs or Buspirone) alongside MAT to stabilize the patient's mood during the first 30 to 60 days of abstinence.

### 14.6.4 Overcoming the "Natural Supplement" Identity

A major behavioral hurdle in this population is the patient's self-identity. Many users of "herbal" substances do not view themselves as "drug users" or "addicts" in the traditional sense.

- **Cognitive Reframing:** Therapy must focus on **Safety Literacy**. Patients need to understand that the "natural" label on a bag of Kratom or a bottle of Zaza is a marketing term, not a biological guarantee. Psychoeducation regarding the lack of quality control—including the risk of heavy metal poisoning and synthetic adulteration—is vital to breaking the psychological attachment to the "botanical" narrative.
- **Behavioral Support:** Cognitive Behavioral Therapy (CBT) and Motivational Interviewing (MI) should be tailored to address the "Gateway Myth"—the idea that these substances are a safe bridge to recovery from harder drugs, when in reality, they often prolong the cycle of neurological instability (Petry, 2024).

## 14.7 Herbal Street Terms and Slang

Term	Substance/Context
The Leaf / Ketum	Common terms for Kratom.

<b>Gas Station Heroin</b>	Refers specifically to Tianeptine (Tianaa/Zaza).
<b>Whites/Greens/Reds</b>	Different strains of Kratom (categorized by vein color and perceived effect).
<b>The "Natural High"</b>	A deceptive marketing term used to normalize botanical abuse.
<b>Zaza / Red Bullets</b>	Popular branding for high-dose Tianeptine extracts.

### 14.8 Summary for the Professional

The contemporary addiction professional must stay vigilant regarding "convenience store" pharmacology. We are moving into an era where high-potency opioids and sedatives are sold alongside candy and energy drinks. Screening for these substances should be standard practice for any client presenting with "opioid-negative" withdrawal or unexplained seizures, and treatment must involve aggressive pharmacological stabilization to restore the brain's neurochemical balance (SAMHSA, 2024).

## 15. Nicotine: Facts & Figures

### 15.1 Overview: The Nicotine Renaissance and the "Clean" Marketing Paradigm

In the contemporary clinical landscape, nicotine has undergone a profound "re-branding" driven by rapid technological innovation in delivery systems and a sophisticated shift in marketing psychology. While traditional combustible cigarette use has reached historic lows — a triumph for 20th-century public health — the total consumption of nicotine remains high, and in some demographics, is actually increasing. This resurgence is driven by the proliferation of **Electronic Nicotine Delivery Systems (ENDS)** and **Oral Nicotine Pouches**.

The 2026 era is defined by the "Clean Nicotine" narrative. Manufacturers have successfully decoupled nicotine from the "dirty," carcinogenic image of tobacco smoke, framing it instead as a "nootropic" (cognitive enhancer), a stress-management tool, or a high-tech lifestyle accessory. For the addiction professional, this presents a unique challenge: we are treating a generation that is often more heavily dependent on ultra-potent nicotine salts than previous generations were on cigarettes, yet these users do not identify with the traditional "smoker" label and often perceive their use as relatively harmless (Volkow & Koob, 2024).

## 15.2 Epidemiology: Monitoring the Future (MTF) Data and the Generational Shift

The transition from combustible tobacco to electronic and oral delivery is most visible in the adolescent and young adult populations. The 2026 data indicates that while the "War on Smoking" was won, the "War on Nicotine" has entered a more complex, invisible phase.

### Percent of Students Reporting Nicotine Use, 2023 - 2024

Product Category	8th Grade	10th Grade	12th Grade	2026 Trend Analysis
Combustible Cigarettes	1.1%	1.5%	2.5%	Historic Low; "Social Stigma" is the primary deterrent.
Nicotine Vaping (ENDS)	7.0%	11.4%	17.6%	Stabilizing at high levels; primary mode of initiation.
Oral Nicotine Pouches	1.8%	3.2%	5.1%	<b>Rapidly Rising</b> ; the "Zyn" phenomenon in schools.
Vaping (Daily Use)	2.5%	4.8%	9.2%	Indicates high physical dependency/nicotine salt saturation.

*Data Source: University of Michigan, Monitoring the Future 2024 Data Release; SAMHSA 2024.*

**Clinical Insight:** The surge in "Daily Use" among 12th graders (nearly 1 in 10) is particularly concerning to the 2026 professional. This frequency suggests that the "pulse" delivery of nicotine salts has fundamentally altered the addiction trajectory, moving users from experimentation to physiological dependence much faster than traditional cigarettes (NIDA, 2024).

## 15.3 Neurobiology: nAChRs and the Hijacked Survival Circuit

Nicotine is a potent psychoactive alkaloid that mimics the neurotransmitter acetylcholine. It acts as a powerful agonist at the **nicotinic acetylcholine receptors (nAChRs)**, which are distributed widely throughout the central and peripheral nervous systems.

### 15.3.1 The Molecular Mechanism of Reward

- **Receptor Binding:** When nicotine enters the bloodstream, it reaches the brain in approximately 7 to 10 seconds. It binds specifically to the **alpha-4-beta-2 ( $\alpha 4\beta 2$ )** subtype of nAChRs located on the dopaminergic neurons in the **Ventral Tegmental Area (VTA)**.
- **The Dopamine Surge:** This binding triggers an immediate, supraphysiological release of dopamine in the **Nucleus Accumbens (NAc)**. Unlike the slow rise of dopamine from natural rewards, nicotine provides a "spike" that reinforces the behavior of use almost instantly.
- **Glutamate and GABA Modulation:** Nicotine also increases glutamate release (which facilitates the "learning" of the addiction) while simultaneously desensitizing GABAergic inhibitory neurons. This creates a "double-hit" where the reward signal is amplified and the "brakes" on the system are removed (Volkow et al., 2023).

### 15.3.2 The Adrenaline Response and Cardiovascular Stress

Nicotine also stimulates the adrenal glands to release **epinephrine (adrenaline)**. This accounts for the "kick" users feel—a sudden increase in heart rate, blood pressure, and blood glucose. In the present era of high-concentration salts, this cardiovascular "hammering" is occurring continuously throughout the day, leading to early-onset vascular stiffness in users as young as 18 (CDC, 2024).

### 15.3.3 The Adolescent Brain: "Pruning Interference"

The most critical 2026 clinical focus is the impact of nicotine on the **Developing Prefrontal Cortex (PFC)**.

32. **Synaptic Pruning:** During adolescence, the brain undergoes a process of "pruning" where inefficient connections are removed to streamline cognitive function.
33. **Hard-Wiring for Addiction:** Nicotine exposure during this window interferes with pruning and "hard-wires" the reward system to be hyper-reactive. This increases the lifetime risk of developing other substance use disorders (the "Gateway Effect" is biologically rooted in this receptor up-regulation).
34. **Cognitive Deficits:** Chronic nicotine use in adolescence is linked to permanent deficits in attention, impulse control, and emotional regulation, as the PFC never fully develops its "top-down" inhibitory control over the reward centers (Koob & Volkow, 2024).

## 15.4 The Evolution of Delivery: From Freebase to Nicotine Salts

Understanding the 2026 landscape requires an analysis of the chemical engineering shift from

the "3rd Generation" (refillable tanks) to the **"4th Generation" (Nicotine Salts/Disposables)** .

### 15.4.1 The Chemistry of Nicotine Salts

Traditional cigarettes and early e-cigarettes used "freebase" nicotine. Freebase nicotine is alkaline (high pH), which makes the vapor "harsh" on the throat, naturally limiting how much a user can inhale.

- **The Benzoic Acid Innovation:** Modern devices (like Juul and subsequent disposables) add an organic acid (typically **benzoic acid** ) to the nicotine. This creates a "salt" version that is pH-neutral.
- **Clinical Result:** The vapor is smooth and non-irritating, allowing manufacturers to increase nicotine concentrations to levels previously impossible (50 mg/mL or 5%). This allows for a "bolus" of nicotine to reach the brain with the same speed and intensity as a combustible cigarette, but without the immediate cough reflex (FDA, 2024).

### 15.4.2 Pharmacokinetics of the "Spike"

Nicotine salts produce a blood-nicotine curve that mimics the "spike and crash" of a cigarette. This rapid decline in blood levels is what drives the compulsive need for the next "hit." In 2026, the addiction professional must recognize that "salt users" are often consuming the equivalent of 1 to 2 packs of cigarettes per day via a single disposable device.

## 15.5 Oral Nicotine Pouches: The "Zyn" Phenomenon

A major emerging threat in the present era is the use of tobacco-free oral nicotine pouches (e.g., **Zyn, On!, Rogue** ). These products consist of nicotine powder and flavorings in a small, permeable pouch placed between the lip and gum.

### 15.5.1 The "Hidden" Addiction

Unlike vaping, which produces a visible cloud, oral pouches are entirely discrete. This allows for "24/7 use" in environments where nicotine was previously restricted, such as classrooms, workplaces, and public transportation.

- **Continuous Saturation:** Users often "park" a pouch in their mouth for hours at a time, leading to a continuous state of nicotine saturation in the brain.
- **Oral Absorption:** Nicotine is absorbed through the oral mucosa, bypassing the lungs entirely. While this reduces pulmonary risk, it significantly increases the risk of periodontal disease, gum recession, and oral lesions (NIAAA, 2024).

### 15.5.2 The "Upper Decky" Culture

Social media (specifically TikTok and Instagram) has "gamified" pouch use. The culture involves "packing a lip" or an "upper decky" (placing the pouch in the upper lip). This lifestyle branding has successfully marketed nicotine to a demographic of "fitness enthusiasts" and "productivity

hackers" who would never have considered smoking a cigarette.

## 15.6 Advanced Health Effects: The Systemic Impact

The 2026 professional must move beyond the "lung cancer" narrative to address the systemic impact of high -potency nicotine.

- **Vascular Aging:** Nicotine is a potent vasoconstrictor. Chronic use leads to **Endothelial Dysfunction** —the blood vessels lose their ability to dilate. This accelerates vascular aging, leading to erectile dysfunction in young men and early -onset hypertension in both genders (CDC, 2024).
- **EVALI and Flavor Toxicity:** While the 2019 EVALI outbreak was largely linked to THC additives, the "flavor chemicals" in nicotine vapes (such as cinnamaldehyde and diacetyl) are known to cause inflammatory responses in the lung tissue. "Popcorn Lung" (Bronchiolitis Obliterans) remains a long-term risk for high -volume flavor users.
- **Metabolic Disruption:** Nicotine interferes with insulin sensitivity. In the current era, we are observing a link between heavy vaping and an increased risk of Type 2 Diabetes among young adults (Volkow & Koob, 2024).

## 15.7 Forensic Toxicology and Monitoring

Monitoring nicotine use in 2026 requires an understanding of specific metabolites and the "Detection Gap."

24. **Cotinine (The Primary Marker):** Nicotine has a very short half -life (~2 hours). Therefore, testing focuses on **Cotinine** , which has a half-life of 15 to 20 hours.
  - **Urine:** Detectable for 2 –4 days.
  - **Blood:** Detectable for 1 –2 days.
  - **Hair:** Detectable for up to 90 days.
25. **Anabasine Testing: The NRT Distinction:** A major challenge in 2026 is distinguishing between a patient who is "cheating" with tobacco and a patient who is using Nicotine Replacement Therapy (NRT).
  - **The Marker: Anabasine** and **Anatabine** are alkaloids found in the tobacco plant but are **not** present in pharmaceutical -grade nicotine (patches/gum).
  - **Clinical Value:** If a patient tests positive for Cotinine but negative for Anabasine, they are successfully using NRT. If they test positive for both, they are still using tobacco products (NIAAA, 2024).

## 15.8 Contemporary Treatment: The 2026 Integrated Standard

Treatment for Nicotine Use Disorder has moved from "Cold Turkey" to a high -acuity, integrated model involving pharmacological stabilization and digital intervention.

### 15.8.1 Pharmacotherapy: The Three Pillars

- **Varenicline (Chantix):** A partial agonist at the  $\alpha 4\beta 2$  receptor. It "shields" the receptor from nicotine (reducing the reward of a slip) while providing a low-level dopamine release (reducing withdrawal). In 2026, Varenicline remains the most effective single-medication therapy.
- **Combination NRT:** The current gold standard is the **"Patch Plus"** method.
  - **The Patch:** Provides 24-hour baseline nicotine levels to prevent systemic withdrawal.
  - **The Gum/Lozenge:** Provides "as-needed" relief for acute, high-intensity cravings.
- **Cytisine:** An emerging botanical alkaloid (widely used in Europe and gaining traction in the U.S. in 2025-2026) that acts similarly to Varenicline but with fewer side effects (SAMHSA, 2024).

### 15.8.2 Digital Behavioral Therapy and AI Support

Because nicotine use is deeply ritualistic, 2026 treatments utilize **Just - In- Time Adaptive Interventions (JITAs)** .

- **AI Quit -Bots:** Smartphone apps use GPS data and heart-rate monitoring (via wearables) to predict cravings. If a user is near a vape shop or their heart rate spikes (indicating stress), the app provides an immediate behavioral intervention or "urge-surfing" exercise.
- **Gamification of Cessation:** Apps that track "Money Saved," "Life Minutes Gained," and "Lung Capacity Restored" are highly effective for the Gen Z demographic (SAMHSA, 2024).

### 15.8.3 The Challenge of "Salt Withdrawal"

Clinicians must be aware that withdrawal from 5% nicotine salts is significantly more intense than withdrawal from 12mg cigarettes. Patients often report "brain fog," extreme irritability, and severe insomnia. In 2026, the professional must be prepared to use higher doses of NRT or longer tapers to accommodate this high-potency dependency.

## 15.9 Nicotine Street Terms and Slang (2026 Update)

Term	Definition/Context
Zynning / Packing a Lip	Using oral nicotine pouches.
Ghosting	Inhaling a vape and holding the breath until no visible cloud is exhaled (to hide use).
The Buzz	The acute nicotine rush, often only achievable after a period of abstinence.
Modding	Using high-wattage, refillable devices (now

	largely replaced by disposables).
<b>Juice / Salt</b>	The nicotine liquid used in devices.
<b>Upper Decky</b>	A nicotine pouch placed in the upper lip.
<b>The Pouch Gap</b>	The period of time between removing one pouch and inserting the next.
<b>Vape - Tongue</b>	A loss of taste/smell caused by chronic over-use of flavor - heavy vapes.

## 15.10 Clinical Summary for the Professional

The 2026 addiction professional must view nicotine through a high -acuity, "continuous use" lens. The transition to salts and pouches has created a dependency profile that is more physiologically entrenched than the "pulse" use of cigarettes. Screening must be universal and must include oral nicotine products. Recovery planning must address the "lifestyle branding" of "Clean Nicotine" and focus on restoring the brain's prefrontal inhibitory control through a combination of pharmacological stabilization and A I-supported behavioral mastery (Volkow & Koob, 2024).

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## QUIZ

1. What long-term changes occur in the brain due to chronic drug use according to the document?
  - A. Neuroplastic changes
  - B. Increased blood flow
  - C. Enhanced memory retention
  - D. Decreased dopamine production
2. What is the significance of the 'Fourth Wave' in the context of drug use as mentioned in the document?
  - A. It marks the beginning of drug legalization
  - B. It is characterized by the saturation of the supply with fentanyl
  - C. It indicates a decrease in drug-related deaths
  - D. It represents a shift towards natural remedies
3. What is the primary effect of chronic inhalant use on the nervous system as described in the document?
  - A. Increased synaptic connections
  - B. Improved emotional regulation
  - C. Enhanced cognitive abilities
  - D. Destruction of the myelin sheath
4. What legislative change has allowed more practitioners to prescribe buprenorphine for Opioid Use Disorder?
  - A. Removal of the 'X-waiver' requirement
  - B. Introduction of new drug courts
  - C. Increased funding for addiction treatment
  - D. Legalization of cannabis
5. What is the role of the hippocampus in the context of addiction as per the document?
  - A. It processes emotions
  - B. It regulates dopamine levels
  - C. It stores memories of drug-taking experiences
  - D. It controls physical movements
6. What is the primary concern regarding the 2026 supply of opioids and their adulterants?

- A. Increased purity of heroin
- B. Adulteration with non-opioid substances
- C. Decreased availability of prescription opioids
- D. Reduction in overdose deaths

7. What is the unique diagnostic marker for Cannabis Hyperemesis Syndrome (CHS)?

- A. Severe abdominal pain
- B. Recurring cycles of nausea
- C. Intractable vomiting
- D. Temporary relief from hot showers

8. What is the mechanism of the Bernese Method used in 2026 for treating opioid dependence?

- A. Gradual increase of methadone
- B. Immediate cessation of opioids
- C. Micro-doses of buprenorphine
- D. High doses of naloxone

9. What significant change occurred in the classification of cannabis in 2026?

- A. It was classified as a Schedule I substance
- B. It was moved to Schedule III
- C. It was banned entirely
- D. It was recognized as a non-medical substance

10. What is the primary reason for the rise in fentanyl-related deaths according to the document?

- A. High binding affinity and efficacy of fentanyl
- B. Increased prescription of opioids
- C. Decreased availability of heroin
- D. Increased use of methadone

11. What is the primary risk associated with the P2P-driven potency of stimulants according to the document?

- A. Increased recreational use
- B. Narrowed window between recreational use and permanent psychosis
- C. Higher rates of prescription drug misuse
- D. Decreased cognitive function

12. What is the recommended approach for addiction professionals when dealing with stimulant use?

- A. Focus solely on detoxification
- B. Utilize short-term therapy
- C. Conduct a Neuropsychological Assessment
- D. Avoid any form of therapy

13. What significant change occurred in the treatment of prescription opioid dependence by 2026?

- A. Shift to long-term MOUD with buprenorphine
- B. Increased use of methadone
- C. Return to short-term detox
- D. Focus on cognitive remediation only

14. What is the primary concern regarding counterfeit prescription drugs as highlighted in the document?

- A. They are less potent than real drugs
- B. They are more affordable
- C. They are always safe to use
- D. They can lead to immediate overdose

15. What is the effect of chronic prescription drug misuse on cognitive function?

- A. Improves decision-making abilities
- B. Leads to significant executive function impairment
- C. Has no effect on cognitive function
- D. Enhances memory retention

16. What is the primary psychological condition associated with PIED use that necessitates specialized understanding in treatment professionals?

- A. Anxiety Disorders
- B. Bipolar Disorder
- C. Muscle Dysmorphia
- D. Post-Traumatic Stress Disorder

17. What is the consequence of excessive testosterone conversion to estrogen in males using anabolic steroids?

- A. Increased muscle mass
- B. Enhanced libido

- C. Improved mood
- D. Gynecomastia

18. What is the primary mechanism by which cocaine exerts its addictive potential?

- A. Dopamine release
- B. Dopamine reuptake inhibition
- C. Serotonin release
- D. Norepinephrine inhibition

19. What is a significant health risk associated with MDMA use in poorly ventilated environments?

- A. Hypothermia
- B. Hyperthermia
- C. Dehydration
- D. Hyponatremia

20. What is the term used to describe the phenomenon where users of MDMA experience severe post-use depression?

- A. Suicide Tuesday
- B. Withdrawal Syndrome
- C. Post-Use Dysphoria
- D. Cognitive Dissonance

21. What is the role of the Anti-Cocaine Vaccine in cocaine treatment?

- A. It enhances the effects of cocaine
- B. It produces antibodies that trap cocaine in the bloodstream
- C. It increases dopamine levels
- D. It stabilizes glutamate levels

22. What is a significant risk associated with cocaine use mentioned in the document?

- A. Increased social interactions
- B. Development of non-alcoholic fatty liver disease
- C. Severe depletion of white blood cells
- D. Improved executive function

23. Which of the following substances is investigated as a dopamine transporter blocker?

- A. Topiramate
- B. Fentanyl

- C. Gabapentin
- D. Modafinil

24. What is the primary diagnostic marker for contemporary cocaine users?

- A. Increased heart rate
- B. Purple necrotic skin patches
- C. High blood pressure
- D. Elevated liver enzymes

25. What is the estimated economic cost of excessive alcohol consumption in the U.S. per year?

- A. \$100 billion
- B. \$500 billion
- C. \$250 billion
- D. \$1 trillion

26. What is the primary reason for the psychological attachment to the 'botanical' narrative surrounding Kratom and Tianeptine?

- A. Lack of quality control and risk of poisoning
- B. High effectiveness in treating addiction
- C. Natural origin of the substances
- D. Marketing strategies targeting young adults

27. Which therapy is recommended to address the 'Gateway Myth' associated with Kratom and Tianeptine?

- A. Family Therapy
- B. Psychoanalysis
- C. Cognitive Behavioral Therapy (CBT)
- D. Exposure Therapy

28. What is the primary pharmacological action of nicotine as described in the document?

- A. It acts as a stimulant
- B. It is a potent vasoconstrictor
- C. It enhances GABA activity
- D. It reduces dopamine release

29. What is the significance of the 'Clean Nicotine' narrative in the context of nicotine use?

- A. It promotes the use of traditional cigarettes

- B. It emphasizes the dangers of nicotine addiction
- C. It discourages the use of nicotine altogether
- D. It frames nicotine as a cognitive enhancer

30. What is the recommended approach for managing Phenibut dependence according to the document?

- A. Cross-tapering with Baclofen
- B. Immediate cessation without tapering
- C. Using herbal supplements
- D. Increasing the dosage gradually

Name: \_\_\_\_\_

- |                         |                         |
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| 1. [A] [B] [C] [D] [E]  | 16. [A] [B] [C] [D] [E] |
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| 3. [A] [B] [C] [D] [E]  | 18. [A] [B] [C] [D] [E] |
| 4. [A] [B] [C] [D] [E]  | 19. [A] [B] [C] [D] [E] |
| 5. [A] [B] [C] [D] [E]  | 20. [A] [B] [C] [D] [E] |
| 6. [A] [B] [C] [D] [E]  | 21. [A] [B] [C] [D] [E] |
| 7. [A] [B] [C] [D] [E]  | 22. [A] [B] [C] [D] [E] |
| 8. [A] [B] [C] [D] [E]  | 23. [A] [B] [C] [D] [E] |
| 9. [A] [B] [C] [D] [E]  | 24. [A] [B] [C] [D] [E] |
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| 14. [A] [B] [C] [D] [E] | 29. [A] [B] [C] [D] [E] |
| 15. [A] [B] [C] [D] [E] | 30. [A] [B] [C] [D] [E] |