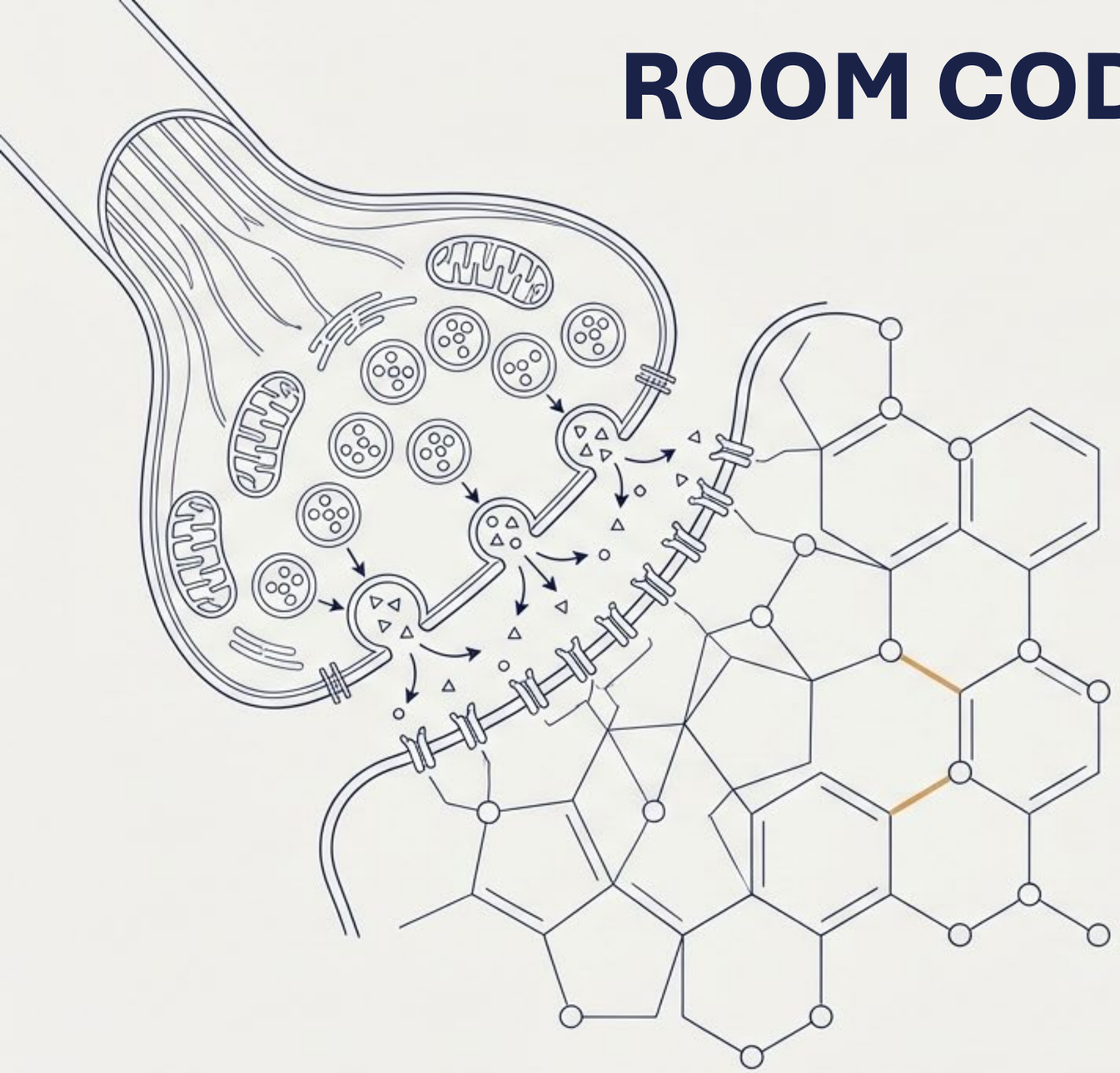
ROOM CODE = 2007



The Crystal Clear Story: Science, and Impact of Methamphetamine

Bridging the historical shift in illicit production with the clinical realities of neurotoxicity, psychosis, and evidence-based treatment.

Hildebrando “Brando” Mireles III, LPC-S, PhD Candidate
Chief Clinical Officer – Camino Real

Disclosure to Learners

2026 Texas Council Conference

June 10-12, 2026

Successful Completion

To successfully complete this activity, you must:

- Miss no more than 15 minutes of the activity or each session.
- Complete and submit the post-activity evaluation.

Continuing Education

Continuing education is approved for these professions:

- Licensed Psychologists (LP)
- Physicians (CME)
  This activity has been planned and implemented in accordance with the accreditation requirements and policies of the Texas Medical Association (TMA) through the joint providership of The Texas Department of State Health Services Continuing Education Program and Texas Council of Community Centers. The Texas Department of State Health Services Continuing Education Program is accredited by TMA to provide continuing medical education for physicians.

  The Texas Department of State Health Services Continuing Education Program designates this live activity for a maximum of 6.00 *AMA PRA Category 1 Credits*™. Physicians should claim only the credit commensurate with the extent of their participation in the activity.

TEXAS Health and Human Services

Texas Department of State Health Services

Commercial Support & Disclosure of Conflict of Interest

This event received no commercial support.

TEXAS Health and Human Services

Texas Department of State Health Services

Non-Endorsement Statement & Off Label Use

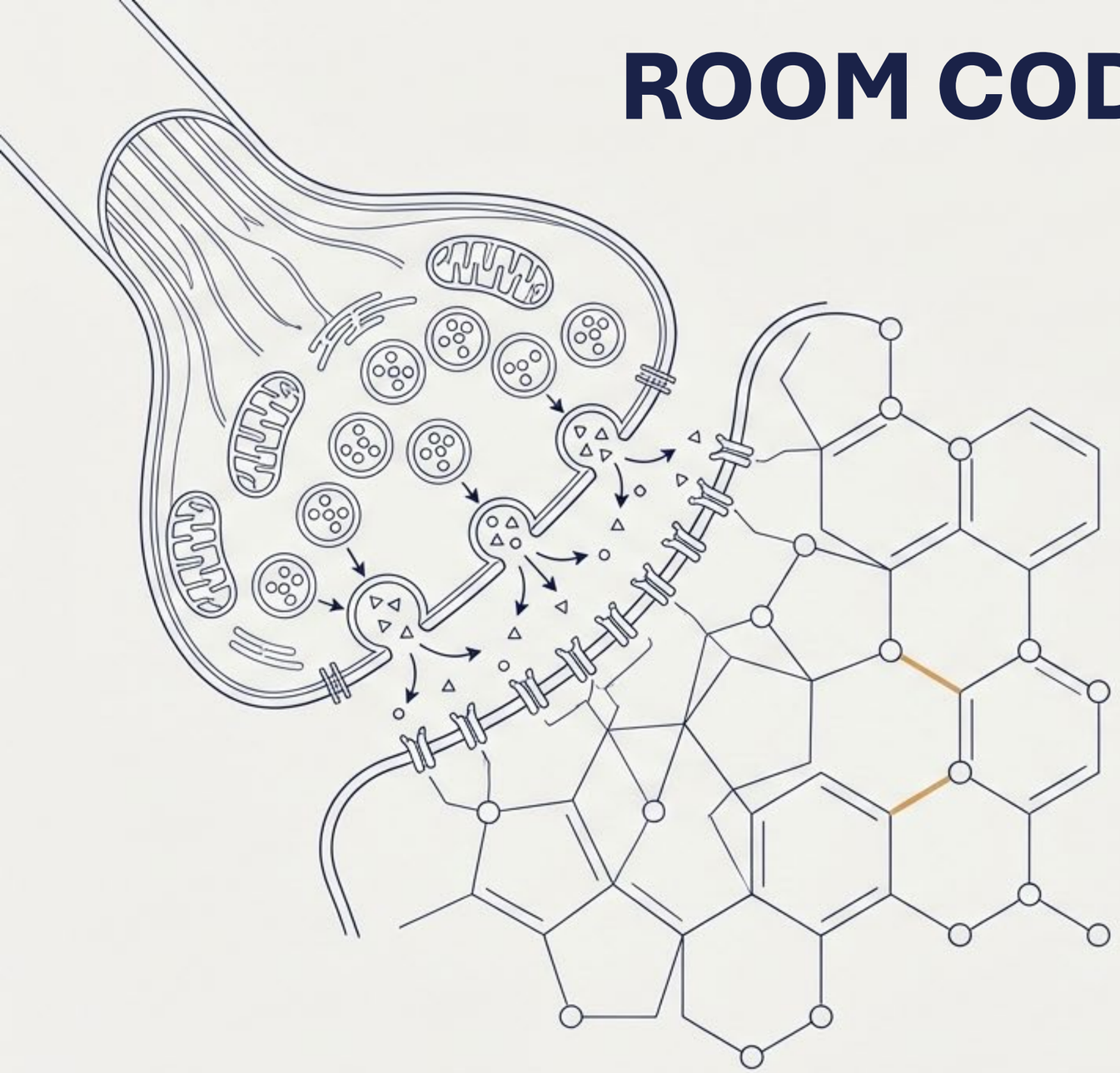
Accredited status does not imply endorsement of any commercial products or services by the Texas Department of State Health Services, Texas Medical Association, or American Nurses Credentialing Center.

The speakers did not disclose the use of products for a purpose other than what it had been approved for by the Food and Drug Administration.

TEXAS Health and Human Services

Texas Department of State Health Services

ROOM CODE = 2007



The Crystal Clear Story: Science, and Impact of Methamphetamine

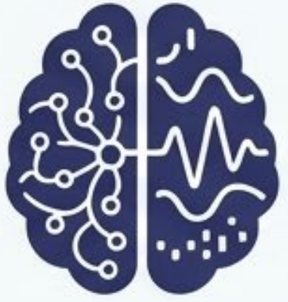
Bridging the historical shift in illicit production with the clinical realities of neurotoxicity, psychosis, and evidence-based treatment.

Hildebrando “Brando” Mireles III, LPC-S, PhD Candidate
Chief Clinical Officer – Camino Real

Learning Objectives

Summarize the origins of methamphetamine, methamphetamine, its early uses, and how its role evolved over time in medicine and illicit markets.

Describe the key chemical components of methamphetamine and how they interact with the central nervous system to produce stimulant effects.



Assess the short-term and long-term physiological and psychological effects of methamphetamine use, including addiction, organ damage, and mental health consequences

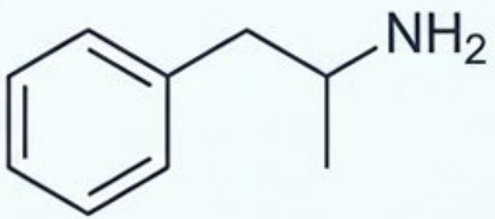
Evaluate evidence-based intervention and treatment approaches for methamphetamine and other stimulant use disorders, including considerations for co-occurring substance use.

What is Methamphetamine “Meth”?

What is Methamphetamine?

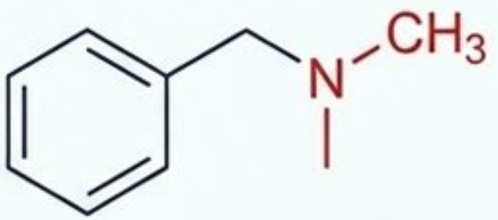
Crystal Methamphetamine

Amphetamine (e.g., Adderall)



Base stimulant structure.

Methamphetamine (Meth)



Added Methyl Group = More Potent & Faster Action.

Methamphetamine is chemically similar to amphetamine but the added methyl group allows it to enter the brain more rapidly, resulting in a more intense and longer-lasting effect on the central nervous system.

Meth is synthetic meaning it is composed chemically by “Cooks.”

Meth Origins / History

The Botanical and Chemical Origins

The original Breaking Bad

The Ephedra Shrub

- Contains natural alkaloids. Utilized for over 5,000 years in traditional Chinese medicine for respiratory ailments and asthma.

1885 / 1893

Japanese chemist Nagayoshi Nagayoshi isolates ephedrine and synthesizes methamphetamine, creating an N-methylated derivative with potent CNS effects.

1919

Akira Ogata refines the synthesis process using red phosphorus and iodine to reduce ephedrine, creating the world's first crystallized methamphetamine.

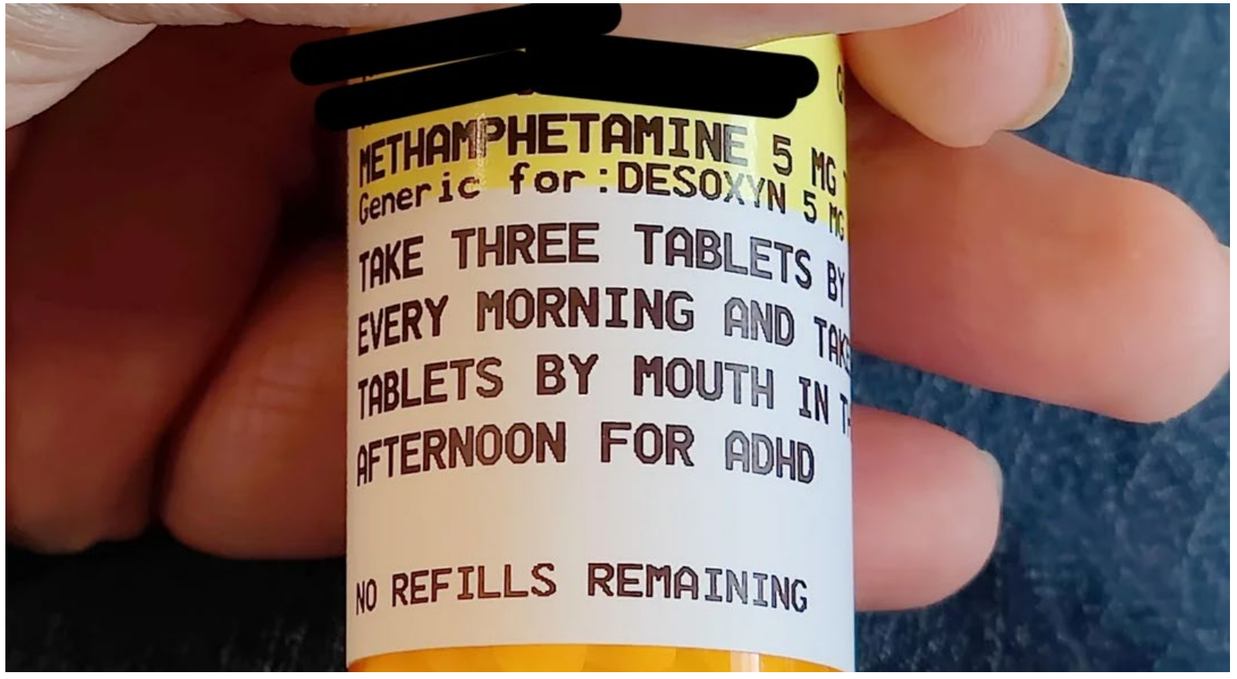
Pharmaceutical Products: The Amphetamine Family

From legitimate medicine to illicit crisis — tracing methamphetamine and amphetamine through the pharmaceutical record

Brand Name	Active Ingredient	Classification	Primary Indication	Era / Current Status
Pervitin	Methamphetamine HCl	**METHAMPHETAMINE**	Military stimulant / fatigue	1938 (Temmler, Germany) — Discontinued
Desoxyn	Methamphetamine HCl	**METHAMPHETAMINE**	ADHD / Obesity (last-line)	FDA approved 1943 — Still available (Sched. II)
Obetrol	Methamphetamine + Amphetamine salts	**METH + AMPHETAMINE**	Obesity	FDA 1960 — Withdrawn 1973; became Adderall
Benzedrine	Amphetamine sulfate	**AMPHETAMINE**	Nasal congestion / Narcolepsy	1933 (Smith, Kline & French) — Discontinued
Dexedrine	Dextroamphetamine sulfate	**AMPHETAMINE**	ADHD / Narcolepsy	Pre-1982 — Generic still available (Sched. II)
Adderall / Adderall XR	Mixed amphetamine salts (75:25 d:l)	**AMPHETAMINE**	ADHD / Narcolepsy	FDA 1996 — Widely prescribed (Sched. II)
Vyvanse	Lisdexamfetamine dimesylate (prodrug)	**AMPHETAMINE (prodrug)**	ADHD / Binge Eating Disorder	FDA 2007 — Widely prescribed (Sched. II)
Evekeo	Amphetamine sulfate (50:50 d:l)	**AMPHETAMINE**	ADHD / Narcolepsy / Obesity	FDA 2012 — Currently available (Sched. II)

■ **METHAMPHETAMINE** | ■ **AMPHETAMINE** | All products listed are Schedule II controlled substances under the CSA


BENZEDRINE
(Reg. U.S. Pat. Off.)
INHALER
Accepted by the Council on Pharmacy and


METHAMPHETAMINE 5 MG
Generic for: DESOXYN 5 MG
TAKE THREE TABLETS BY
EVERY MORNING AND TA
TABLETS BY MOUTH IN T
AFTERNOON FOR ADHD
NO REFILLS REMAINING


Psyche und Kreislauf
Depressionen
Hypotonie
Müdigkeit
Narkolepsie
postoperative
Rekonvaleszenz
Pervitin
Pervitin
1-Phenyl-2-methylamino-propan-hydrochlorid


XR
20 mg

The Wartime Catalyst & Civilian Epidemics

1930s | World War II | 1950s–1960s

1930s: Medicalization

Introduction of the Benzedrine inhaler by Smith, Kline, and French for nasal congestion and asthma. The euphoric side effects are rapidly discovered.

World War II: The Wartime Catalyst

Widespread institutional distribution. Axis and Allied forces deploy methamphetamine tablets (e.g., Pervitin) to enhance troop endurance, ward off fatigue, and stimulate kamikaze pilots and factory workers.

1950s–1960s: Civilian Epidemics

Post-war surplus floods civilian markets. Prescribed for obesity (Obetrol) and normalized in Beatnik culture as ‘bennies’ before severe psychiatric consequences forced the FDA to mandate prescriptions in 1959.

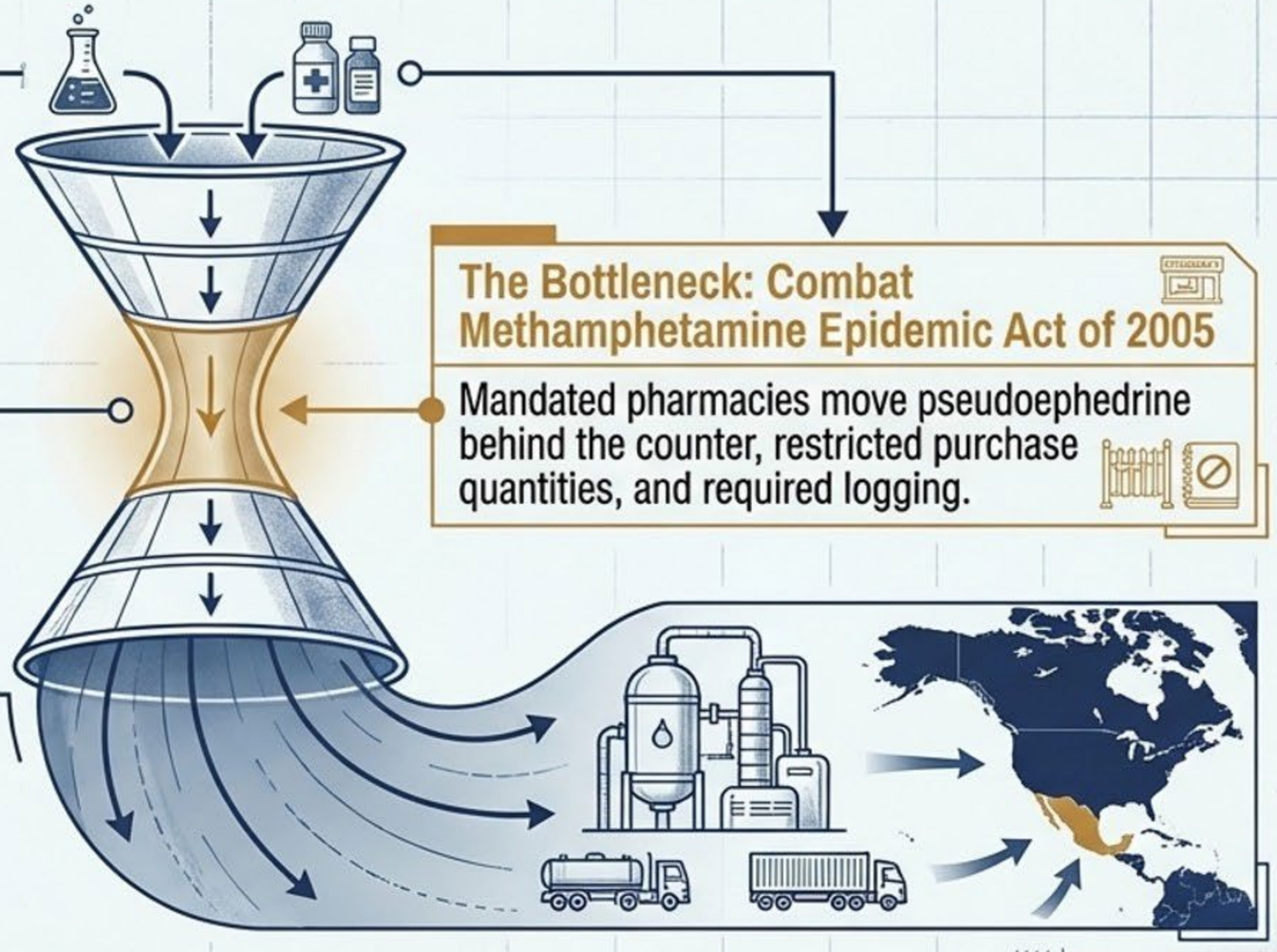
The Legislative Squeeze & The Market Vacuum

Pre-2005: The "Shake-and-Bake" Era

Widespread domestic clandestine labs utilizing easily accessible pseudoephedrine from over-the-counter cold medications.

The Bottleneck: Combat Methamphetamine Epidemic Act of 2005

Mandated pharmacies move pseudoephedrine behind the counter, restricted purchase quantities, and required logging.



The Unintended Consequence: Industrial Expansion

The collapse of small-scale domestic production created a market vacuum. Transnational Criminal Organizations (TCOs) in Mexico rapidly filled this void, permanently shifting the manufacturing paradigm to industrial scales.

The Evolution of Methamphetamine: From Ephedra to the P2P Paradigm

1885–1960s: Origins and Global Institutionalization

Tracing over a century of development, this timeline tracks methamphetamine's journey from its 19th-century synthesis to its institutional use in World War II, followed by its cultural normalization and the modern emergence of high-potency "Super Meth." It highlights how chemical precursors and regulations have fundamentally shaped the drug's availability and clinical impact.

1885–1919: The Foundation of Synthetic Stimulants

Nagai Nagayoshi isolates ephedrine (1885) and synthesizes meth (1893) before Akira Ogata creates the first crystallized form (1919).

1930s–1940s: Pharmaceuticals and the Frontlines of War

Benzedrine and Pervitin are mass-produced and issued to military forces to enhance endurance and alertness during WWII.

1950s–1960s: Medical Use and Cultural Normalization

Methamphetamine becomes a common prescription for obesity (Obetrol) and a staple of the "Beat Generation" culture.

1970–Present: Regulation and the Industrial Shift

1970–2005: Regulatory Crackdowns and Precursor Shifts

The 1970 Controlled Substances Act and 2005 Combat Meth Act stricify regulate usage and cold medicine precursors.

1980s–1990s: The Rise of Clandestine Laboratories

Domestic illegal labs pivot to using pseudoephedrine found in over-the-counter medications to circumvent chemical restrictions.

Modern Era: The Industrial "Super Meth" Crisis

Mexican Transnational Criminal Organizations shift to P2P synthesis, producing a high-potency, highly neurotosic product.

Precursor Evolution and Synthesis Methods

Era	Primary Precursor	Dominant Synthesis Method
1885 – 1970s	Ephedrine	Medicinal/Pharmaceutical
1980s – 2005	Pseudoephedrine	Clandestine "One-Pot" / Shake-and-Bake
2010s – Present	Phenyl-2-Propanone (P2P)	Industrial-Scale TCO Production

How to recognize a meth lab:

Recognizing a Clandestine Methamphetamine Laboratory

Environmental Red Flags

Chemical Odors

- Ammonia (cat urine), ether, acetone, paint thinner, rotten eggs, or vinegar

Property Modifications

- Blacked-out or foil-covered windows, unusual ventilation (fans in winter), elaborate security measures

Dead Vegetation

- Burn pits, dead spots in grass from chemical dumping

Unusual Activity

- Frequent visitors at odd hours, excessive trash with chemical containers

Equipment & Chemical Indicators

Precursor Materials

- Large quantities of cold medicine (pseudoephedrine), lithium batteries torn apart, drain cleaner, starter fluid

Lab Equipment

- Glass cookware with residue, plastic bottles with tubes, rubber hoses, funnels, respiratory masks

Waste Residue

- Red-stained coffee filters, multi-layered liquids, white powdery residue on surfaces

Anhydrous Ammonia Tanks

- Gas cylinders with blue-discolored brass valves (chemical reaction marker)

Clinical Exposure Symptoms

Acute Exposure Signs

- Respiratory distress, coughing, chest tightness, eye and skin irritation, chemical burns

Neurological Symptoms

- Headache, dizziness, confusion, disorientation, nausea

Chronic / Residential Exposure

- Persistent respiratory illness, skin rashes, neurological deficits in occupants and especially children

Pediatric Vulnerability

- Children at highest risk due to floor-level exposure, hand-to-mouth behavior, and developing organ systems

⚠ SAFETY IMPERATIVE

Do NOT enter, touch, or investigate a suspected lab. Treat as a HazMat scene. Evacuate, call 911, and decontaminate any exposed persons. One pound of meth produces six pounds of toxic waste. Residual contamination persists in walls, carpets, and HVAC systems long after a lab is dismantled.

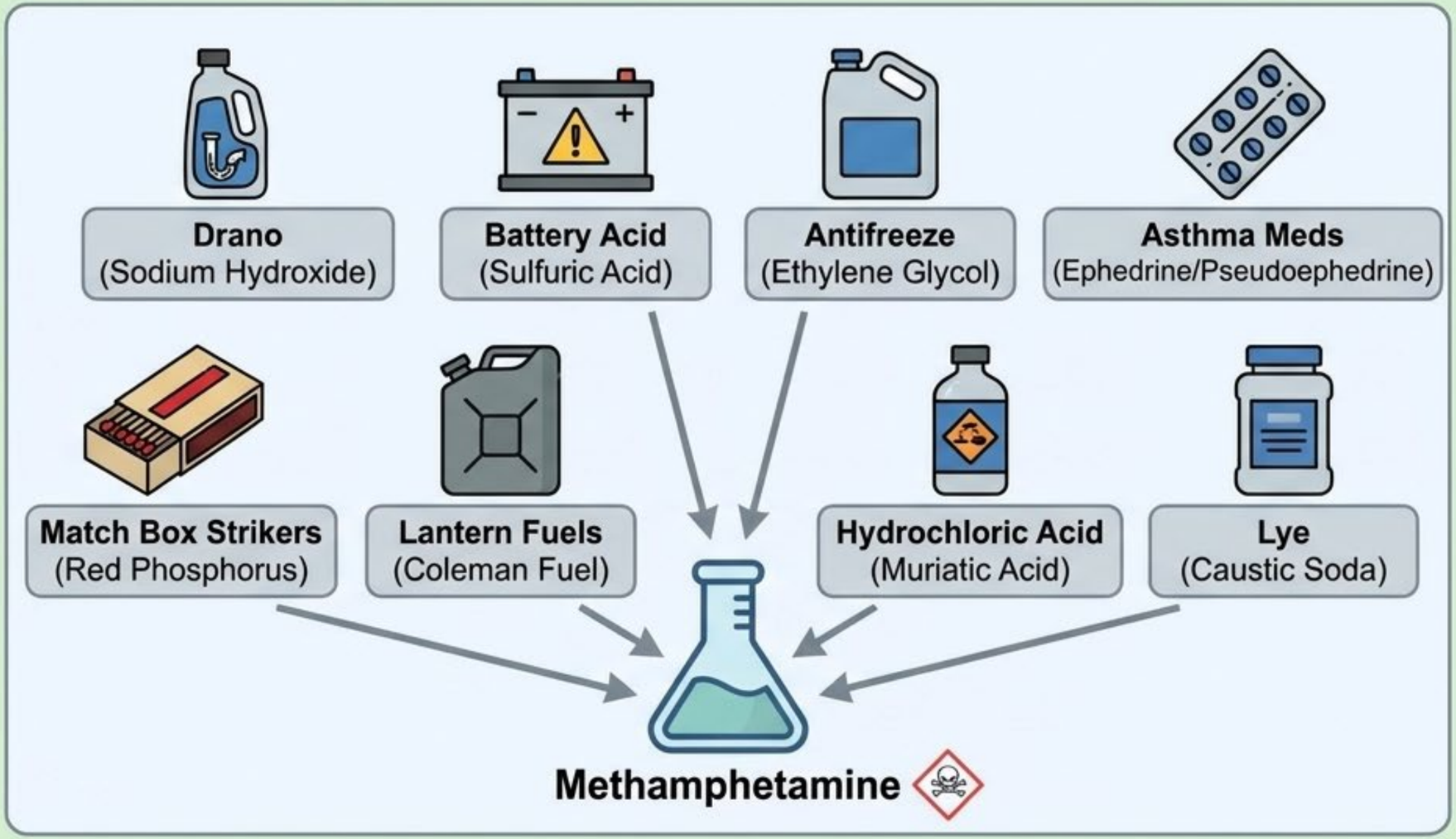
Sources: DOJ National Drug Intelligence Center; NC State Bureau of Investigation; CDC/NIOSH; EPA Voluntary Guidelines for Meth Lab Cleanup

The Chemistry Behind Meth

The Production Shift: Ephedrine vs. P2P "Super Meth"

Dimension	The Ephedrine Era	The P2P Era
Precursor Chemical	Pseudoephedrine (from cold medicine)	Phenyl-2-Propanone (an industrial chemical)
Manufacturing Scale	Small, domestic "shake-and-bake" labs	Mexican TCOs operating massive clandestine industrial facilities
Chemical Impurities	Lithium, battery acid	Cyanide, nitrostyrene, racing fuel, lead
Clinical Presentation	Social/talkative euphoria, gradual physical decline	Rapid onset of severe paranoia, immediate social isolation, pronounced neurotoxicity

The Toxic Ingredients of Methamphetamine



Takeaway: Methamphetamine is produced using highly volatile and poisonous common household chemicals, posing severe health and environmental risks.

Family Impacts and Risks

Children's Exposure & Fumes

Danger of children's exposure to toxic chemical fumes; severe implications for respiratory and neurological development.

Prenatal Risks & Explosion

Risks of pregnant women exposure affecting fetal health. High risk of explosions, fires, and structural damage.

Chemical Burns on People & Pets

Direct contact causes severe chemical burns on adults, children, and pets (pets are not immune).

Weapons & Unsanitary Conditions

Exposure to weapons, unsanitary conditions, and hazardous waste in production environments.

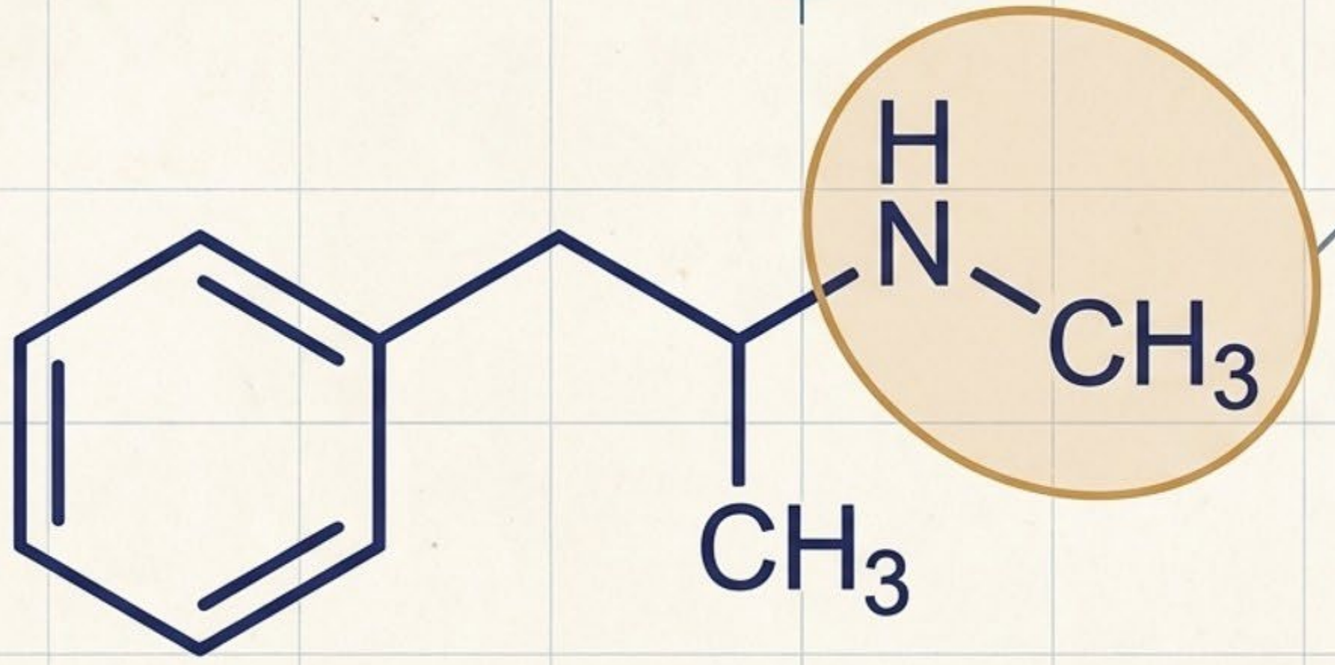
Drug Schedules I-V: Potential for Abuse & Examples

HIGH
Potential for Abuse

LOW
Potential for Abuse

SCHEDULE I	No Accepted Medical Use	Heroin, LSD, Ecstasy
SCHEDULE II	High Abuse Potential, Severe Restrictions	Cocaine, Fentanyl, Cocain, Fentanyl, METHAMPHETAMINE
SCHEDULE III	Moderate to Low Abuse Potential	Ketamine, Anabolic Steroids
SCHEDULE IV	Low Abuse Potential	Xanax, Valium
SCHEDULE V	Lowest Abuse Potential	Cough preparations with codeine

Chemical Architecture & The Lipophilicity Factor



The Crucial Methyl Addition

Methamphetamine ($C_{10}H_{15}N$) shares a phenylethylamine core with amphetamine. This specific methyl addition significantly enhances lipid solubility.

Clinical Impact: Ultra-Rapid Penetration

High lipophilicity enables the molecule to aggressively bypass the Blood-Brain Barrier (BBB), resulting in an immediate, intense, and highly neurotoxic effect on the Central Nervous System.

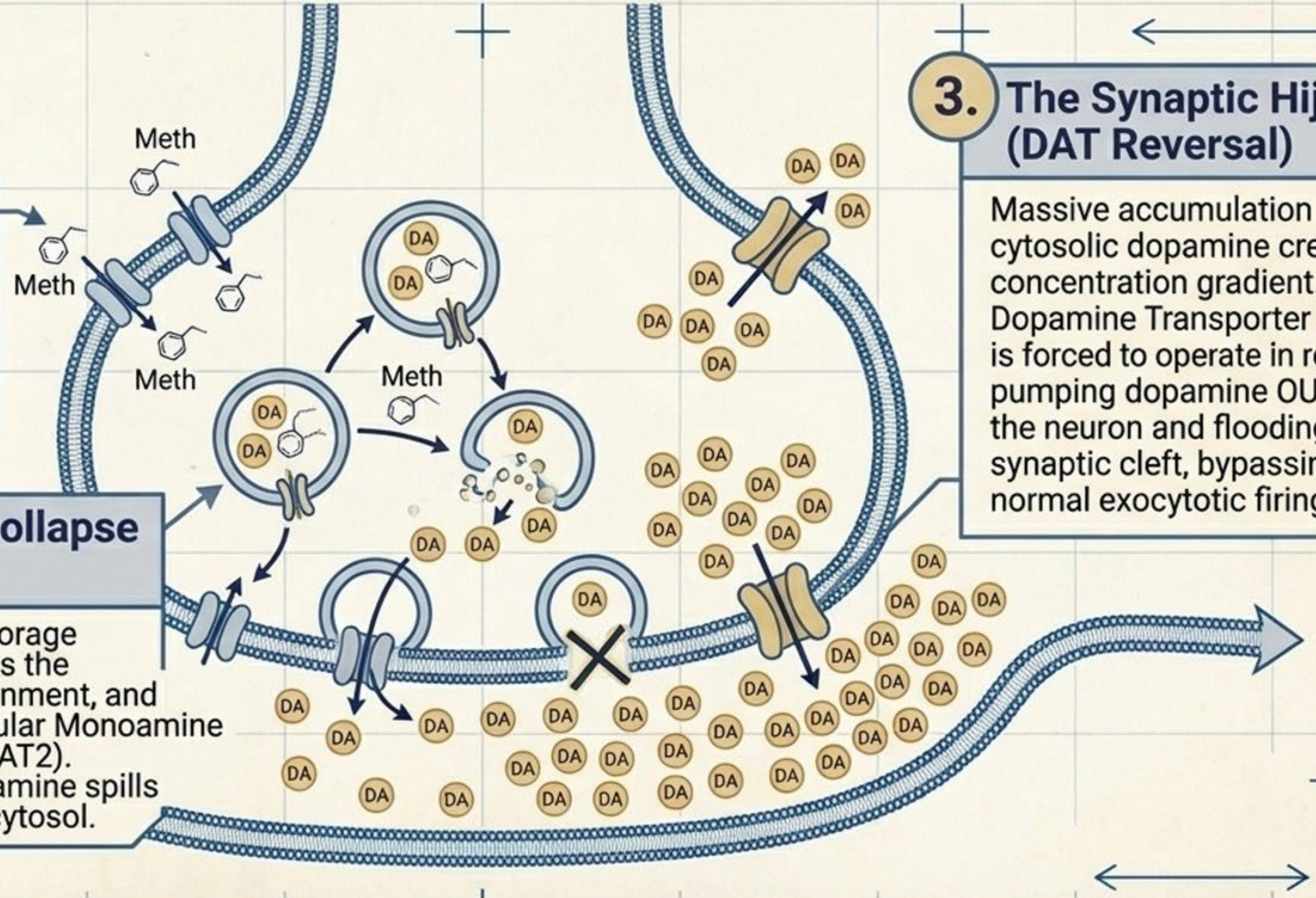
The Synaptic Hijack: VMAT2 & DAT Reversal

1. Infiltration

Methamphetamine readily diffuses across the neuronal membrane, entering the presynaptic terminal.

2. Vesicular Collapse (VMAT2)

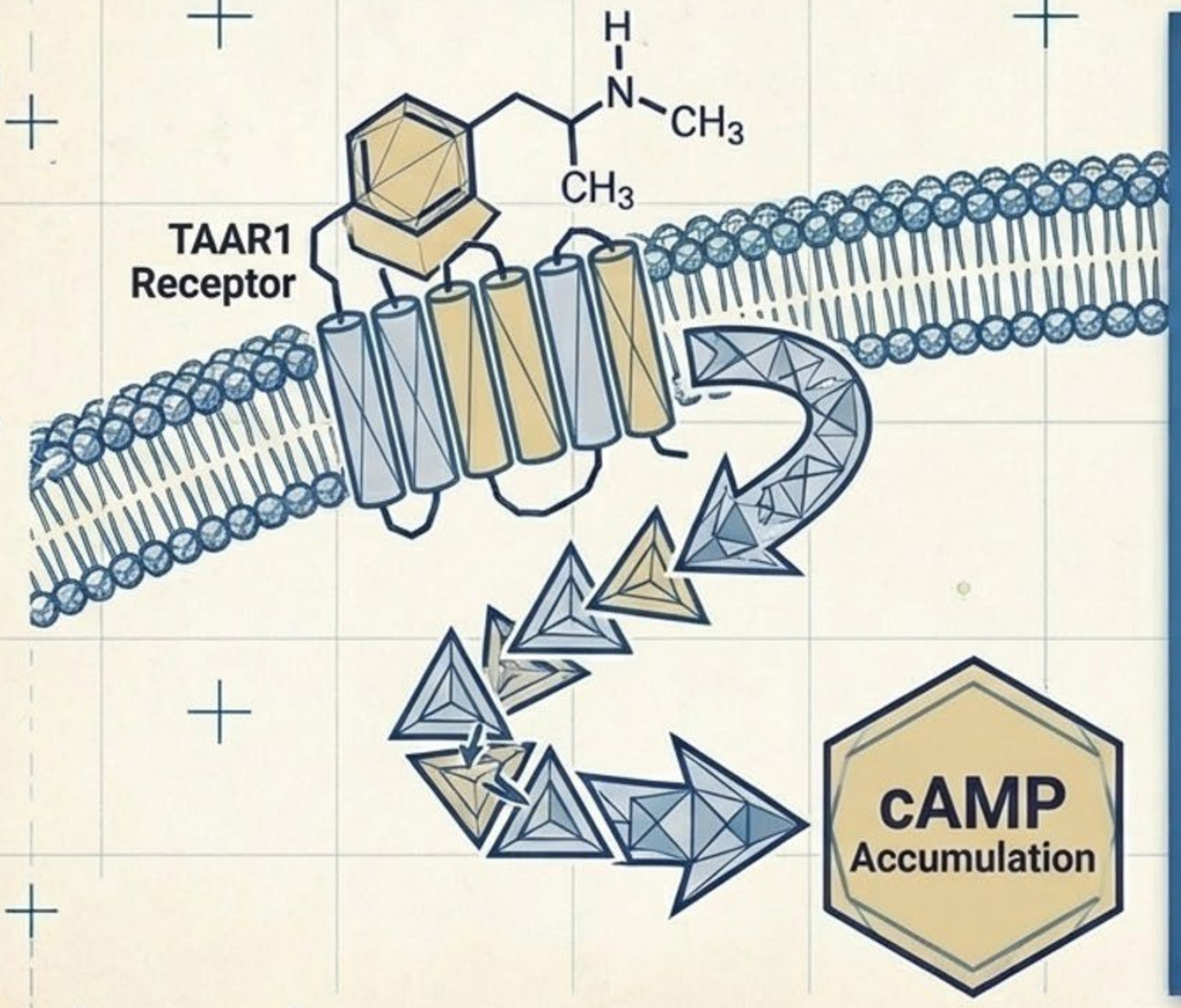
The drug enters storage vesicles, alkalinizes the intracellular environment, and disrupts the Vesicular Monoamine Transporter 2 (VMAT2). Sequestrated dopamine spills into the neuronal cytosol.



3. The Synaptic Hijack (DAT Reversal)

Massive accumulation of cytosolic dopamine creates a concentration gradient. The Dopamine Transporter (DAT) is forced to operate in reverse, pumping dopamine OUT of the neuron and flooding the synaptic cleft, bypassing normal exocytotic firing.

TAAR1 Agonism: Beyond Transporter Blockade



1. The Target: TAAR1

Methamphetamine acts as a full agonist at the presynaptic Trace Amine-Associated Receptor 1 (TAAR1).

2. The Signaling Cascade

Activation stimulates G-alpha-s proteins, driving the rapid, massive accumulation of intracellular cyclic AMP (cAMP).

3. The Duality of TAAR1

Modulation: Inhibits dopamine uptake and modulates firing rates of dopaminergic neurons in the Ventral Tegmental Area (VTA).

Vulnerability: Alterations in these pathways are directly implicated in profound neurotoxicity and impaired thermoregulation in chronic abuse.

Short/Long Term Physiological and Psychological Effects of Meth Use

Acute Effects & The Hyperthermic Cascade

Stage 1: Acute Intoxication

Supraphysiologic monoamine release causes intense euphoria, tachycardia, and a massive adrenergic surge.

Stage 2: Severe Hyperthermia

Core body temperatures dangerously elevate, often reaching 39–42°C (102–107°F), severely disrupting cellular homeostasis.

Stage 3: Metabolic Impairment

Extreme heat damages hepatocyte mitochondria. The liver's ability to metabolize ammonia via the urea cycle is critically impaired.

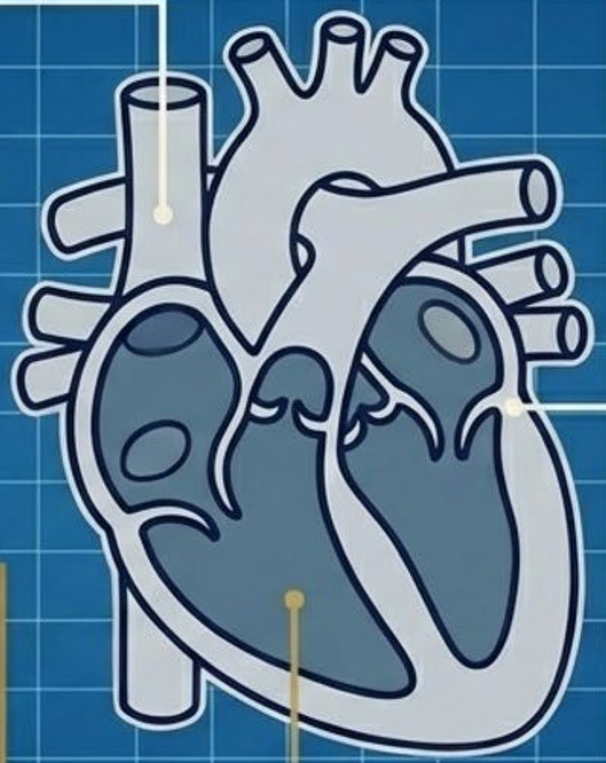
Stage 4: Systemic Oxidative Stress

Plasma ammonia concentrations rise, exacerbating the generation of Reactive Oxygen Species (ROS) and triggering widespread, systemic cellular injury.

Cardiotoxicity & Systemic Organ Strain

Hemodynamic Instability

Acute use triggers severe hypertension, tachycardia, and vasospasm. This extreme strain directly leads to myocardial ischemia or acute myocardial infarction.



Methamphetamine-Associated Cardiomyopathy

Chronic abuse drives profound, irreversible structural changes: dilation of the ventricles, extensive cardiac fibrosis, and progressive, end-stage heart failure.

Lethal Arrhythmias

Altered catecholamine signaling and massive adrenergic surges disrupt the heart's electrical pathways, frequently leading to lethal ventricular arrhythmias.

Pathophysiology of Renal Failure & Rhabdomyolysis

1. Muscle Necrosis (Rhabdomyolysis)

Extreme hyperthermia, severe agitation, and vasoconstriction lead to rapid skeletal muscle breakdown.

2. Myoglobin Release

Damaged muscle fibers release massive amounts of the protein myoglobin directly into the bloodstream.

3. Renal Obstruction

Myoglobin filters into the kidneys, precipitating and forming obstructive “myoglobin casts” within the renal tubules.

4. Acute Kidney Injury (AKI)

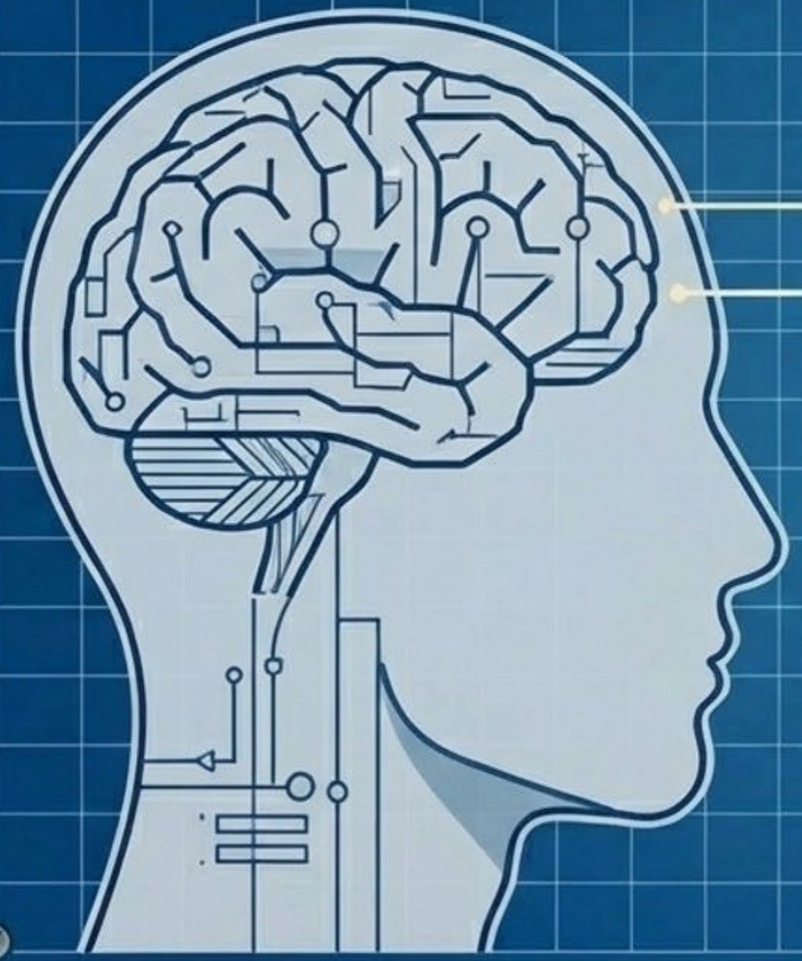
Toxic tubular necrosis and reduced renal blood flow trigger acute renal failure.

Clinical Diagnostic Markers:

Detection is confirmed via critically elevated serum levels of creatine kinase, urea, cystatin C, and NGAL.

The Psychiatric Paradigm: MIPD

Methamphetamine-Induced Psychosis (MIPD) driven by P2P formulations.



Rapid Onset Paranoia

Extreme, sudden-onset delusions of persecution. Patients exhibit high levels of agitation and suspicion, completely lacking insight into their condition.

Pronounced Hallucinations

Unlike other primary psychoses, MIPD is heavily characterized by visual and tactile hallucinations, such as formication (the severe, distressing sensation of insects crawling under the skin).

Social Disintegration & The Kindling Effect

Results in incoherent speech and intense social isolation. Previous episodes of stimulant-induced psychosis lower the neurological threshold, making future psychotic breaks highly likely even with microscopic doses.

Differential Diagnosis: MIPD vs. Primary Schizophrenia

Symptom Dimension	MIPD (Meth-Induced Psychosis)	Primary Schizophrenia
Positive Symptoms: Hallucinations	Predominantly visual and tactile (e.g., formication).	Predominantly auditory (e.g., voices conversing).
Positive Symptoms: Delusions & Thought	High rates of incoherent speech, intense, rapid paranoia, and acute agitation.	Pronounced, structured thought disorder, thought broadcasting, and thought projection.
Negative Symptoms	Milder negative symptoms; less severe affective flattening.	Pronounced negative symptoms; severe blunted affect, emotional withdrawal, and alogia (poverty of speech).

Clinical Warning: Up to 30% of persistent MIPD cases may transition to a formal schizophrenia diagnosis at long-term follow-up.

Intervention

The Standard of Care: Behavioral Interventions

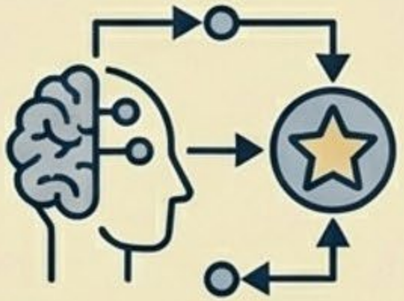
The Pharmacological Gap

Establish clearly that there are currently zero FDA-approved medications medications specifically for the treatment of Methamphetamine Use Disorder (MUD). Medicine alone cannot cure this.

The Standard of Care

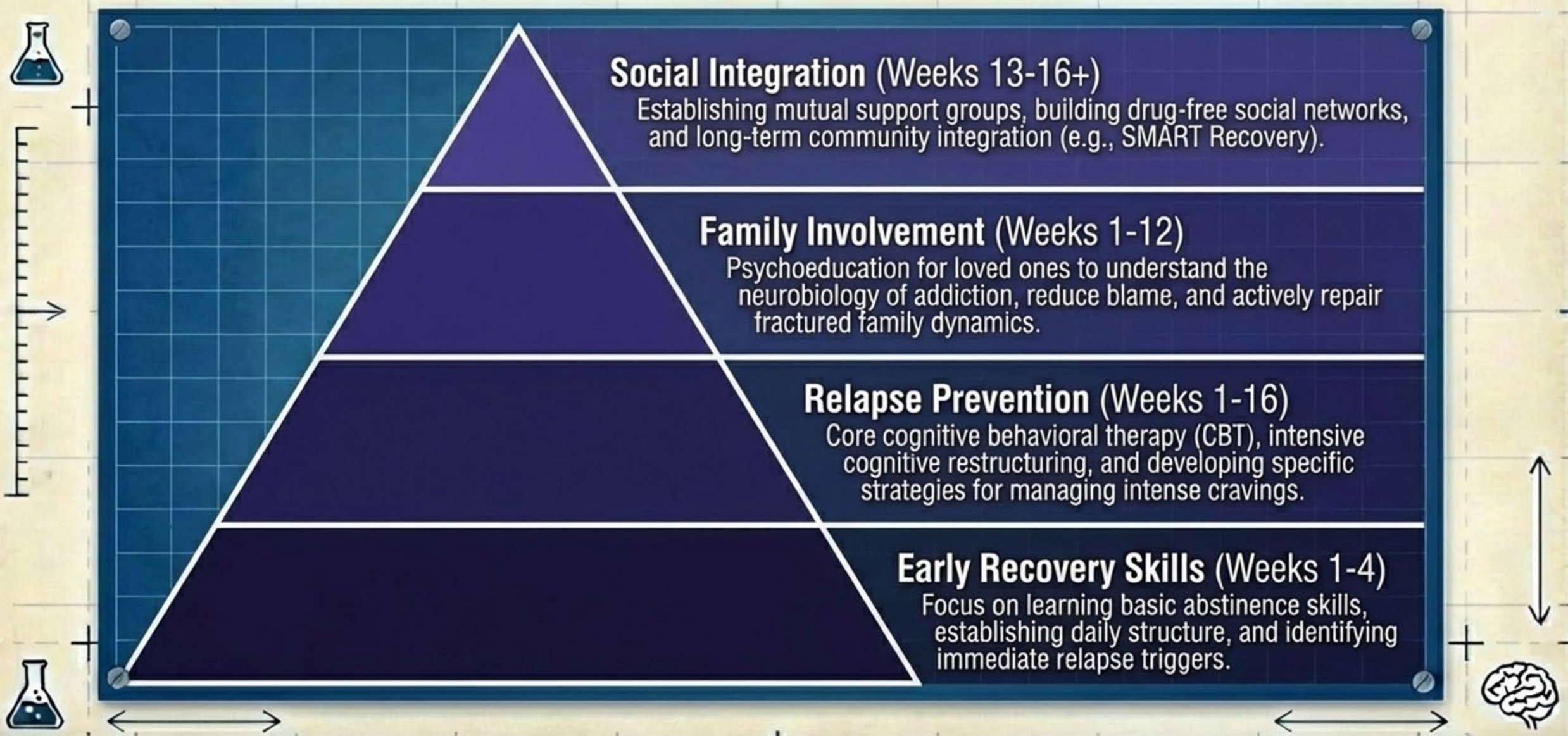
In the absence of pharmacotherapy, highly structured behavioral and psychosocial therapies remain the primary, evidence-based frontline treatment for achieving abstinence.

Contingency Management (CM)



The most effective evidence-based intervention. CM utilizes motivational incentives (tangible rewards) to reinforce objective evidence of abstinence (negative drug tests), directly counteracting the brain's hijacked reward system.

The Matrix Model: A 16-Week Multicomponent Protocol



Clinical Blueprint

Emerging Pharmacotherapy: The NIH ADAPT-2 Trial

Addressing the urgent clinical gap in MUD pharmacotherapy

The Intervention (Combination Therapy)

Injectable Naltrexone

An extended-release opioid antagonist designed to severely modulate the craving and reward pathways in the brain.

Oral Bupropion

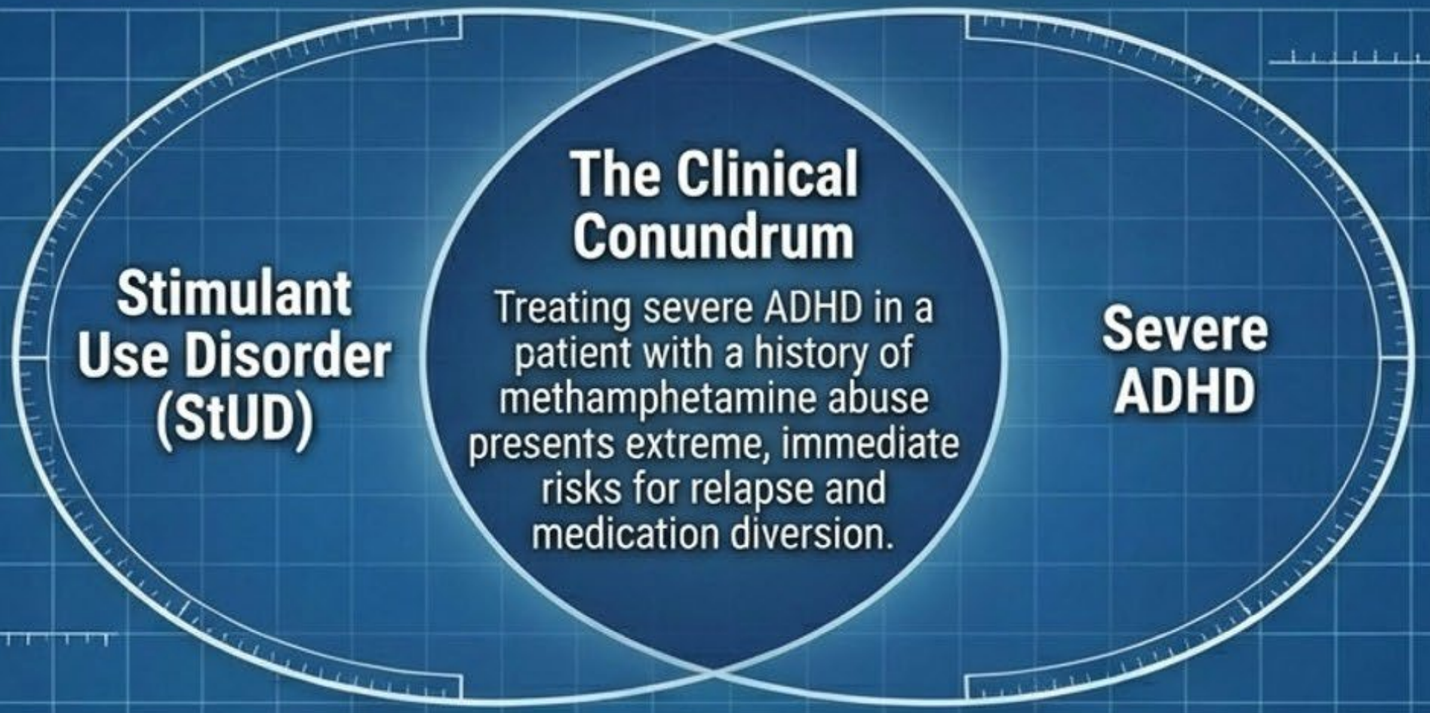
An antidepressant that increases extracellular dopamine and norepinephrine levels, effectively mitigating acute withdrawal dysphoria.

The Clinical Outcome

The ADAPT-2 combination demonstrated a statistically significant reduction in methamphetamine use among trial participants, offering the most promising bridge to a pharmacological standard of care to date.

Clinical Blueprint

Clinical Complexity: Managing Co-Occurring ADHD



Evidence-Based Management Strategy

First-Line Risk Mitigation	Pharmacological Options	Non-Stimulant Alternatives
Strictly avoid the prescription of short-acting, immediate-release stimulants due to their high abuse liability and rapid euphoric onset.	Utilize long-acting formulations (e.g., Extended-Release Mixed Amphetamine Salts [MAS-ER]) to provide steady therapeutic levels without the reinforcing euphoric 'rush'.	Leverage medications like Atomoxetine to effectively treat ADHD symptoms while entirely circumventing the dopaminergic abuse liability.

Clinical Blueprint

The ‘Fourth Wave’ Synthesis: A Compounded Crisis

Vector 1: The Neurotoxic Baseline

The historical shift to industrial P2P “Super Meth” has created a massive population suffering from profound neurotoxic vulnerability, cardiovascular damage, and volatile psychiatric instability.

The Clinical Imperative

Comprehensive treatment must aggressively integrate evidence-based behavioral therapies (Matrix/CM) with immediate harm reduction. Widespread distribution of Fentanyl Test Strips and Naloxone is mandatory. We must ensure ensure survival to enable recovery.

Vector 2: The Opioid Collision

This vulnerable population is now colliding directly with the “Fourth Wave” of the overdose epidemic—lethal synthetic opioid (fentanyl) contamination directly within the illicit stimulant supply.

References

- Arkansas Department of Human Services. (n.d.). *Origins of meth*. Me Over Meth. https://humanservices.arkansas.gov/divisions-shared-services/shared-services/office-of-substance-abuse-and-mental-health/me-over-meth/origins-of-meth/
- CADCA. (2023). *Methamphetamine: Everything, everywhere, and its prevention*. Practical Theorist 14. https://www.cadca.org/wp-content/uploads/2023/06/CADCAPT14.pdf
- Healthline. (2022, July 1). *Can meth use cause renal failure?* https://www.healthline.com/health/can-meth-cause-renal-failure
- HISTORY.com Editors. (2017, June 7). *History of meth*. History. https://www.history.com/articles/history-of-meth (Updated 2025, May 28).
- Lindsay, D., et al. (2024). The ASAM/AAAP clinical practice guideline on the management of stimulant use disorder. *Journal of Addiction Medicine*, *18*(1), 1–56. https://doi.org/10.1097/ADM.0000000000001299
- Mirza, S. A., Mohammed, N. S., Ali, Z. Q., & Mohamed, A. S. (2025). The effects of methamphetamine intoxication on acute kidney injury in Iraqi male addicts. *Toxicology Reports*, *14*, 102065. https://doi.org/10.1016/j.toxrep.2025.102065
- National Institute on Drug Abuse. (n.d.). *Tobacco, Alcohol, Prescription medication, and other Substance use (TAPS) tool*. National Institutes of Health. https://nida.nih.gov/taps2
- National Institutes of Health. (2021, January 13). *Combination treatment for methamphetamine use disorder shows promise in NIH study*. https://www.nih.gov/news-events/news-releases/combination-treatment-methamphetamine-use-disorder-shows-promise-nih-study
- Patel, P., & Saadabadi, A. (2025, December 13). *Methamphetamine*. StatPearls. https://www.ncbi.nlm.nih.gov/books/NBK535356/
- Pyramid Healthcare. (2021, September 16). *What can meth do to my body?* https://www.pyramid-healthcare.com/blog/what-can-meth-do-to-my-body/
- Ramli, F. F., Rejeki, P. S., Ibrahim, N. I., Abdullayeva, G., & Halim, S. (2025). A mechanistic review on toxicity effects of methamphetamine. *International Journal of Medical Sciences*, *22*(3), 482-507. https://doi.org/10.7150/ijms.99159
- Tanz, L. J., Miller, K. D., Dinwiddie, A. T., Gladden, R. M., Asher, A., Baldwin, G., Nesbit, B., & O'Donnell, J. (2025). Drug overdose deaths involving stimulants — United States, January 2018–June 2024. *MMWR. Morbidity and Mortality Weekly Report*, *74*(32), 491–499. http://dx.doi.org/10.15585/mmwr.mm7432a1
- Trivedi, M. H., Walker, R., Ling, W., Dela Cruz, A., Sharma, G., Carmody, T., Ghitza, U. E., Wahle, A., Kim, M., Shores-Wilson, K., Sparenborg, S., Coffin, P., Schmitz, J., Wiest, K., Bart, G., Sonne, S. C., Wakhlu, S., Rush, A. J., Nunes, E. V., & Shoptaw, S. (2021). Bupropion and naltrexone in methamphetamine use disorder. *The New England Journal of Medicine*, *384*(2), 140–153. https://doi.org/10.1056/NEJMoa2020214
- Wearne, T. A., & Cornish, J. L. (2018). A comparison of methamphetamine-induced psychosis and schizophrenia: A review of positive, negative, and cognitive symptomatology. *Frontiers in Psychiatry*, *9*, 491. https://doi.org/10.3389/fpsyt.2018.00491
- Yang, X., Wang, Y., Li, Q., Zhong, Y., Chen, L., Du, Y., He, J., Liao, L., Xiong, K., Yi, C., & Yan, J. (2018). The main molecular mechanisms underlying methamphetamine-induced neurotoxicity and implications for pharmacological treatment. *Frontiers in Molecular Neuroscience*, *11*, 186. https://doi.org/10.3389/fnmol.2018.00186