

The Encyclopedia of Meth

A Scientific, Medical, and Harm Reduction Reference

EDITION I

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Notice, Scope & Disclaimers

What this book deliberately does not contain

This reference is written for clinical, scientific, public-health, legal, and educational use. It **does not** provide methods, procedures, reagent lists, quantities, reaction conditions, equipment specifications, or precursor-sourcing information for the manufacture of methamphetamine. It **does not** provide dosing schedules, routes, or techniques intended to facilitate non-medical use. These omissions are intentional and absolute. Where the history of manufacture or precursor regulation is discussed, it is treated at the level of policy and historical record only, without operational detail.

Purpose

The aim of this encyclopedia is to consolidate, in one rigorous and readable volume, what the scientific and medical literature establishes about methamphetamine: its chemistry and pharmacology, its history, its medical uses and harms, its toxicology and neuroscience, the epidemiology of its use, the evidence base for treatment and harm reduction, and the legal and social frameworks that surround it. It is intended for healthcare professionals, researchers, students, public-health workers, harm-reduction practitioners, policymakers, journalists, and informed members of the public.

Not medical or legal advice

Nothing in this volume constitutes medical, clinical, or legal advice, nor does it establish a clinician–patient or attorney–client relationship. Clinical decisions must be made by qualified professionals with reference to current guidelines, local regulations, and the circumstances of the individual patient. Drug laws vary by jurisdiction and change over time; legal questions should be directed to a qualified professional in the relevant jurisdiction.

On sources and verification

This work follows a live-verification standard: factual claims are grounded in primary and authoritative secondary sources, and figures that change over time (prevalence, mortality, market data) are presented as time-stamped and are flagged where they require periodic re-verification. Citations are intended to be checkable; no source has been invented.

Where the literature is genuinely uncertain or contested, the text says so rather than manufacturing false confidence.

On language

This book uses person-first, non-stigmatizing language wherever possible ("a person who uses methamphetamine" rather than pejorative labels). This is not a cosmetic preference: the evidence indicates that stigmatizing language measurably affects clinical judgment and care-seeking. Terms in wide clinical or street use are defined for clarity, not endorsed as appropriate descriptors of people.

A note on harm reduction

Substantial portions of this book describe harm-reduction science. Documenting an intervention that reduces death, injury, or disease among people who already use drugs is not the same as encouraging drug use, and the two should not be confused. Harm reduction is a mainstream public-health framework endorsed by major national and international health bodies; it is presented here as evidence, with its evidence base and its limits both made explicit.

How to Use This Encyclopedia

A reader's map to fourteen parts, forty chapters, and eight appendices.

The encyclopedia is organized to move from the molecule outward to the person, the population, and the policy. Part I introduces the subject and the conventions used throughout. Part II is historical. Parts III through V treat the physical science — chemistry, pharmacology, and neuroscience. Parts VI through IX turn to the human body and mind: medicine, public health, addiction science, and toxicology. Parts X through XII address forensic science, the social context, and law and policy. Part XIII surveys the research frontier, and Part XIV provides reference apparatus — glossary, FAQ, timeline, a statistics atlas, and a comprehensive index.

Each chapter is intended to stand alone for a reader who needs a single topic, while cross-references and a shared glossary knit the volume together. Readers seeking a clinical orientation may begin with Parts IV, VI, and IX; those seeking a public-health or policy orientation may begin with Parts VII, XI, and XII; chemists and analysts will find Parts III and X most directly relevant. The non-specialist is encouraged to read Part I in full before sampling elsewhere, because it establishes the terminology and the interpretive stance that the rest of the book assumes.

Throughout, validated findings are distinguished from projections, hypotheses, and contested claims. Where a number is current as of a particular date, that date is given. Where the science is provisional, the text marks it as provisional. The reader should never have to guess whether a statement reflects settled knowledge or an open question.

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PART I

Introduction

Why This Encyclopedia Exists

A subject this consequential deserves to be understood on the basis of evidence rather than fear.

Few substances occupy as contradictory a place in modern life as methamphetamine. It is, simultaneously, a molecule with a legitimate and FDA-approved medical indication; one of the most damaging drugs of the contemporary overdose era; a fixture of wartime history; a subject of intense neuroscientific research; and a focus of some of the most punitive drug policy ever enacted. A reader who wishes to understand it must therefore hold several truths at once, and resist the pull toward any single, simplifying story. The purpose of this book is to make that possible.

Scope and limitations

This encyclopedia covers the chemistry, history, pharmacology, neuroscience, medicine, public health, addiction science, toxicology, forensic science, sociology, and law of methamphetamine. It is deliberately broad, because the drug cannot be understood within any single discipline: the chemistry is unintelligible without the pharmacology, the pharmacology is incomplete without the clinical picture, and the clinical picture is meaningless outside the social and legal context in which people actually use the drug.

The limitations are equally deliberate. As stated in the front matter, this volume contains no synthesis instructions, no precursor-sourcing guidance, and no information designed to facilitate non-medical use. The reader interested in how methamphetamine is made will find here only what the historical and policy record requires — that certain precursor chemistries existed, that they were regulated, and that regulation reshaped illicit supply — without any of the operational detail that would convert history into a manual. This is not an oversight to be corrected in a later edition. It is a fixed boundary that defines the kind of book this is.

Harm reduction philosophy

Harm reduction is a set of practical strategies and principles aimed at reducing the negative consequences associated with drug use. Its central premise is that, while abstinence may be the safest choice and a worthy goal, a substantial number of people will use drugs regardless of legal prohibition or moral exhortation, and that public health is best served by reducing the death, disease, and injury that befall those people in the meantime. Harm reduction does not require a person to stop using in order to deserve care, dignity, or access to services.

This stance is sometimes misread as endorsement. It is not. A seatbelt does not endorse dangerous driving; naloxone does not endorse opioid use; clean syringes do not endorse injection. Each is an intervention that reduces a specific, measurable harm in a population that exists whether or not we approve of it. The harm-reduction chapters of this book describe what works, what the evidence shows, and where the limits of these interventions lie — including honest acknowledgment that harm reduction is not a treatment for addiction and does not claim to be.

Scientific neutrality

The molecule is value-neutral; the social phenomenon is not. A great deal of confusion follows from conflating the two. The pharmacological action of methamphetamine at the dopamine transporter is a matter of biochemistry, indifferent to politics. The question of how a society should respond to methamphetamine use is a matter of values, evidence, and trade-offs about which reasonable people disagree. This book aims to be rigorously neutral about the former and scrupulously fair about the latter: presenting the science as the science stands, and presenting policy debates by giving the strongest version of competing positions rather than a caricature of the one the author finds least congenial.

Neutrality here does not mean false balance. Where the evidence is one-sided, the text says so. Where a popular belief is simply wrong, the text corrects it. Neutrality means refusing to let sensationalism, moral panic, or ideological commitment distort the reporting of what is actually known.

Terminology

A small vocabulary recurs throughout the book and is worth fixing at the outset.

Methamphetamine — the substance *N*-methylanphetamine, a synthetic central-nervous-system stimulant of the substituted-amphetamine class. Often shortened to "meth."

Amphetamine-type stimulants (ATS) — an umbrella category used in epidemiology and drug policy that groups amphetamine, methamphetamine, and related stimulants; useful for population data but too coarse for pharmacology.

Crystal meth / "ice" — high-purity methamphetamine hydrochloride in a coarse, translucent crystalline form, typically associated with smoking; chemically the same molecule as other forms but differing in purity and presentation.

Enantiomers (d- and l-) — the two mirror-image forms of the molecule. The dextrorotary (d-) enantiomer is the potent central stimulant; the levorotary (l-) enantiomer is a far weaker central stimulant historically used as a nasal decongestant. Chapter 2 treats this in detail.

Person who uses drugs — the preferred descriptor for an individual, in place of stigmatizing labels. The distinction matters clinically and is defended in Chapter 30.

Common myths

Public understanding of methamphetamine is shaped heavily by alarmist media, and several widespread beliefs do not survive contact with the evidence. A few are flagged here and examined more fully in later chapters.

"One use causes instant, permanent addiction." Addiction is a process, not an event. Methamphetamine has high addictive liability, and some people progress rapidly, but the claim that a single exposure inevitably produces lifelong dependence is not supported; vulnerability is shaped by genetics, environment, mental health, dose, route, and pattern of use (Parts VIII and XI).

"Brain damage from methamphetamine is total and irreversible." Chronic heavy use is associated with measurable changes in brain structure and function, including in dopaminergic markers, but imaging studies also document partial re-

covery of some markers with sustained abstinence. The picture is one of serious harm with meaningful, if incomplete, capacity for recovery (Part V).

"All methamphetamine is the same." Pharmaceutical methamphetamine is an enantiopure, dose-defined, quality-controlled product. Illicit methamphetamine is of variable purity and composition and may contain adulterants. The molecule may be identical; the risk profile of an unknown street sample is not (Chapter 2).

"'Meth mouth' is purely chemical corrosion of the teeth." The severe dental disease associated with methamphetamine use is multifactorial — reduced salivary flow, bruxism, prolonged intoxication, poor nutrition and oral hygiene, and high-sugar intake all contribute. The drug itself is not a simple acid dissolving enamel (Chapter 14).

What Is Methamphetamine?

A single molecule, two mirror images, and a wide gulf between the pharmacy and the street.

Chemical identity

Methamphetamine is the common name for *N*-methylamphetamine, also written as *N*-methyl-1-phenylpropan-2-amine and historically as desoxyephedrine. Its molecular formula is $C_{10}H_{15}N$, giving a molar mass of approximately 149.23 grams per mole for the free base. Structurally it belongs to the substituted amphetamines, a subclass of the phenethylamines — the same broad chemical family that includes amphetamine, the catecholamine neurotransmitters, and a number of other psychoactive compounds. Methamphetamine is, quite literally, amphetamine bearing an additional methyl group on the nitrogen atom; that single methyl group increases the molecule's lipid solubility and its ability to cross the blood–brain barrier, which contributes to its potency relative to amphetamine.

Molecular structure

The molecule can be read as three parts. A benzene (phenyl) ring forms the aromatic core. Attached to it is a two-carbon ethyl chain bearing a methyl branch at the second carbon — the phenethylamine backbone with an alpha-methyl substituent that distinguishes amphetamines from simpler phenethylamines and slows their metabolism by monoamine oxidase. At the end of that chain sits an amine nitrogen, and in methamphetamine that nitrogen carries a methyl group (the "*N*-methyl" of the name). The alpha-methyl group and the *N*-methyl group together account for much of how methamphetamine differs pharmacologically from the body's own stimulant neurochemicals, which it structurally resembles.

Chirality

The carbon bearing the methyl branch is a stereocenter, which means methamphetamine exists as two non-superimposable mirror-image forms, or enantiomers. These are not a chemical curiosity; the distinction is central to the drug's pharmacology, its legal status, and its appearance in over-the-counter products.

The **dextrorotary** form — d-methamphetamine, dextromethamphetamine, the (S)-(+)
enantiomer — is the potent central-nervous-system stimulant. It is the active ingredient in the pharmaceutical product Desoxyn and the form most associated with both the therapeutic and the harmful central effects of the drug. The **levorotary** form — l-methamphetamine, levomethamphetamine, the (R)-(–)
enantiomer — is a much weaker central stimulant whose effects are predominantly peripheral (vasoconstriction); it was for decades the active component of certain over-the-counter nasal decongestant inhalers, where it was labeled by chemical name rather than as "methamphetamine."^{5,6} A **racemic** mixture contains both enantiomers in equal proportion. This single fact — that "methamphetamine" can mean a potent stimulant, a weak decongestant, or a fifty-fifty blend depending on chirality — is a recurring source of confusion in toxicology, drug testing, and public discussion, and it returns in Parts IV and X.

Physical properties

Methamphetamine free base is an oily, volatile liquid at room temperature. The form encountered as a pharmaceutical and as an illicit solid is the hydrochloride salt, a white-to-translucent crystalline material that is freely soluble in water. The high-purity coarse crystalline presentation of the hydrochloride salt is what is colloquially called "ice" or "crystal." The salt is considerably more stable and water-soluble than the free base, which is why it is the form used both medically and illicitly. Methamphetamine hydrochloride melts in the region of roughly 170–175 °C. Beyond melting point and solubility, the salient physical properties for later chapters are its solubility behavior (relevant to formulation and to analysis, Chapters 5–6) and its volatility (relevant to routes of administration and to environmental contamination, Chapter 27).

Property	Value / description

Chemical name	<i>N</i> -methamphetamine (<i>N</i> -methyl-1-phenylpropan-2-amine)
Molecular formula	C ₁₀ H ₁₅ N
Molar mass (free base)	≈ 149.23 g/mol
Chemical class	Substituted amphetamine / phenethylamine
Stereochemistry	One stereocenter; d- (S,+) and l- (R,−) enantiomers; racemate possible
Common solid form	Hydrochloride salt — white/translucent crystalline solid, water-soluble
Pharmacological class	CNS stimulant; sympathomimetic monoamine releaser / reuptake inhibitor

Pharmacological classification

Methamphetamine is classified as a central-nervous-system stimulant and a sympathomimetic amine. Mechanistically — developed fully in Part IV — it acts principally by increasing synaptic concentrations of the monoamine neurotransmitters dopamine and norepinephrine (and, to a lesser degree, serotonin). It does this chiefly by entering the presynaptic neuron, interacting with the vesicular monoamine transporter (VMAT2) to displace neurotransmitter into the cytoplasm, and reversing the direction of the plasma-membrane transporters (the dopamine, norepinephrine, and serotonin transporters: DAT, NET, SERT) so that they release rather than reuptake their substrate. It is also an agonist at the trace amine-associated receptor 1 (TAAR1). The net effect is a large, sustained rise in extracellular dopamine and norepinephrine, which underlies both the drug's effects and much of its toxicity.

Pharmaceutical versus illicit methamphetamine

It is the same molecule; it is not the same substance as encountered. Pharmaceutical methamphetamine — marketed in the United States as Desoxyn — is enantiopure dextromethamphetamine hydrochloride, manufactured to defined purity,

dispensed in known unit doses, FDA-approved for narrowly defined indications, and subject to stringent controls.^{1,5} Illicit methamphetamine is of unknown and variable purity, may be racemic or predominantly d-, and may contain manufacturing residues or adulterants; it is consumed in unknown quantity by routes that change its risk profile. Many of the harms attributed to "methamphetamine" in the public imagination are inseparable from this context of uncertainty, unregulated dose, high-risk routes, and the conditions of illicit use — a point this book returns to repeatedly, because confusing the molecule with the street reality leads to bad science and bad policy alike.

PART II

History

Discovery and Early Chemistry

From an ancient medicinal shrub to a wartime industrial stimulant, in a little over half a century.

Natural precursor compounds

The story begins not with methamphetamine but with ephedra. Plants of the genus *Ephedra* — known in Chinese medicine as *má huáng* — have been used for respiratory complaints for a very long time. In 1885 the Japanese chemist Nagai Nagayoshi, then working in Germany, isolated and identified the plant's active stimulant alkaloid, ephedrine.^{4,7} Ephedrine and its stereoisomer pseudoephedrine are sympathomimetic amines in their own right, with bronchodilator and decongestant activity, and they are close chemical relatives of amphetamine and methamphetamine. This relationship — that methamphetamine could be derived from naturally occurring ephedrine — is the thread that connects the herbal pharmacopoeia of antiquity to the synthetic stimulant of the twentieth century, and, much later, the thread that drove precursor-control policy (Chapter 4).

Early synthesis

In 1893, Nagai Nagayoshi first synthesized methamphetamine from ephedrine.^{1,2,7} The compound he obtained was, however, not in the stable, readily handled form by which the drug is known today. That step came in 1919, when Akira Ogata, another Japanese chemist, first produced methamphetamine in crystalline (hydrochloride) form.^{1,2,5} Ogata's achievement — a more stable, water-soluble crystalline product — is conventionally treated as the origin point of methamphetamine as a usable drug substance. In keeping with this book's stated boundaries, the historical record of which precursor chemistry was involved is acknowledged without any procedural detail; the relevant historical fact is simply that an ephedrine-derived route yielded a crystalline product, and that this product proved far more practical than the earlier liquid form.

Industrial development

Crystalline methamphetamine moved from laboratory novelty to commercial pharmaceutical over the following two decades. In Japan, the compound was brought to market by the pharmaceutical industry under brand names including Philopon (Hiropon).² In Germany, researchers at the Berlin-based Temmler-Werke developed their own production method in the 1930s, which was patented in the latter part of that decade, and in 1938 Temmler introduced methamphetamine to the market under the brand name Pervitin.^{2,5} The near-simultaneous commercialization of a powerful, cheap, orally active stimulant in two industrializing societies on the eve of a world war set the stage for the drug's first mass deployment.

Wartime use

The Second World War was methamphetamine's introduction to mass use. Pervitin was distributed widely to German military personnel, valued for its capacity to suppress fatigue and sustain alertness during sustained operations; it became sufficiently associated with armored and air crews to acquire the nickname "Panzerschokolade" — tank chocolate.^{4,9} Enormous quantities circulated: contemporary accounts describe many millions of tablets issued during the rapid campaigns of 1939–1941.⁴ The Allied powers relied principally on amphetamine rather than methamphetamine — the British and Americans issued Benzedrine — while methamphetamine saw heavy use in Germany and Japan.^{4,9,10} Reports also record that senior figures, including Adolf Hitler, received injections of stimulants and other agents from personal physicians during the war.⁸ The wartime experience seeded large civilian supplies and patterns of use that would shape the post-war decades, most acutely in Japan, which experienced a significant methamphetamine epidemic after 1945 as military stockpiles entered civilian hands.

Medical commercialization

Alongside its military use, methamphetamine and its relatives entered ordinary medical practice. The amphetamine-containing Benzedrine inhaler was marketed in the United States from the early 1930s for nasal congestion and asthma, initially available without prescription, and its euphoric and energizing side effects were

soon discovered by the public.¹⁰ Methamphetamine itself was marketed under names such as Methedrine. In December 1944, the U.S. Food and Drug Administration approved Desoxyn (methamphetamine hydrochloride, as the dextro isomer), initially for a broad set of indications that were later narrowed substantially.^{1,5,12} Other branded methamphetamine products, such as Obetrol, were marketed for weight control. By mid-century, methamphetamine had thus completed a remarkable arc — from an ephedrine derivative of academic interest, to a wartime industrial stimulant, to a widely prescribed pharmaceutical — well before the harms of large-scale use and diversion forced a reckoning.

Meth Through History

How a prescribed stimulant became a controlled substance, an illicit market, and a fixture of the modern overdose era.

Military applications

The military thread, begun in Chapter 3, did not end in 1945. Methamphetamine and related stimulants were administered to soldiers in subsequent conflicts — for example, methamphetamine was given to United States troops during the Korean War of the early 1950s.⁴ The broader military interest in pharmacological fatigue management persisted into the modern era, though contemporary armed forces have generally turned to other agents, such as dextroamphetamine and later modafinil, for "go-pill" use under medical supervision. The military history matters here for two reasons: it normalized stimulant use on a mass scale, and it left behind both supply and demand that fed the postwar civilian markets.

Medical prescribing

The 1950s and 1960s were the high-water mark of legal stimulant prescribing in much of the industrialized world. Methamphetamine and amphetamine products were prescribed widely for weight loss, depression, fatigue, and a range of vaguely defined complaints. The very properties that made them commercially successful — mood elevation, appetite suppression, wakefulness — also made them prone to diversion, escalation, and dependence. Liberal prescribing produced a large population of people exposed to the drugs and a steady leakage of pharmaceutical supply into non-medical channels, and by the late 1960s the medical and regulatory consensus had begun to turn against this pattern of use.

Counterculture

As pharmaceutical stimulants became culturally available, a distinct pattern of high-dose, often injected use emerged in the 1960s, associated in the American

imagination with the "speed freak" and with scenes such as San Francisco's Haight-Ashbury. The slogan "speed kills" dates from this period and reflected a growing awareness, even within drug-using subcultures, that heavy methamphetamine use carried severe physical and psychological costs. This era marked the beginning of the drug's migration from the pharmacy and the doctor's office toward the illicit market — a migration that tightening regulation would accelerate.

The War on Drugs

The regulatory turn was decisive. In the United States, the Controlled Substances Act of 1970 placed methamphetamine under federal control as a Schedule II substance — a category reserved for drugs with accepted medical use but high potential for abuse and dependence — a status it retains.^{1,14} Internationally, amphetamine-type stimulants were brought under the United Nations Convention on Psychotropic Substances of 1971.¹⁴ As legal pharmaceutical supply contracted, illicit production expanded to meet continuing demand. A particularly consequential dynamic was precursor control: because illicit manufacture had relied heavily on ephedrine and pseudoephedrine — the very decongestant compounds descended from Nagai's 1885 discovery — regulators moved to restrict access to these chemicals. In the United States, measures culminating in the Combat Methamphetamine Epidemic Act of 2005 placed pseudoephedrine-containing products behind the pharmacy counter and limited quantities of sale.^{11,14} Restriction of these precursors did not end illicit supply; it reshaped it, contributing to a shift toward alternative precursor chemistries and toward large-scale production concentrated in clandestine operations outside domestic borders. This is the level of detail appropriate to a policy history; the operational specifics remain, by design, outside the scope of this book.

Modern trends

The contemporary picture is shaped by three intertwined developments, examined quantitatively in the Statistics Atlas (Chapter 39) and the epidemiology chapter (Chapter 16). First, the dominant illicit product in many markets shifted toward

high-purity methamphetamine produced by alternative precursor routes, accompanied in several regions by falling prices and rising purity — a combination that increases harm. Second, methamphetamine became deeply embedded in patterns of polysubstance use, including the co-use of methamphetamine and synthetic opioids such as fentanyl, with serious implications for overdose risk. Third, stimulant-involved deaths rose markedly in the overdose statistics of the late 2010s and the 2020s, marking the emergence of what some public-health analysts describe as a stimulant phase of the broader overdose crisis. Specific figures for prevalence, purity, price, and mortality change from year to year and are therefore presented, with their dates and sources, in the dedicated data chapters rather than asserted here, in keeping with this book's commitment to time-stamping the numbers that move.

References — Parts I–II

References are listed in order of first citation. Sources current as of 2026; figures subject to periodic re-verification are so noted in the text.

1. Yasaei R, Saadabadi A. *Methamphetamine*. StatPearls. Treasure Island (FL): StatPearls Publishing; updated 2025. NCBI Bookshelf ID NBK535356.
2. ChemistryViews. "Who Was the First to Synthesize Methamphetamine?" ChemistryViews / Wiley-VCH, December 2024.
3. Reserved for analytical-chemistry sources (Part III).
4. Release (UK). "Methamphetamine — History." Release: Drugs, the Law & Human Rights.
5. *Levomethamphetamine*. Reference summary of enantiomer pharmacology, decongestant history, and FDA labeling changes (l-desoxyephedrine → levmetamfetamine, 1998).
6. DrugBank. "Levmetamfetamine (DB09571)" — L-enantiomer pharmacology and OTC decongestant use.
7. *Nagai Nagayoshi* — biographical and scientific record: isolation of ephedrine (1885); synthesis of methamphetamine from ephedrine (1893).
8. Ohler N., and contemporary historical accounts of stimulant use by military and political figures in the Second World War.
9. Historical accounts of Pervitin distribution to German forces ("Panzerschokolade") and production peaks, 1939–1941.
10. HISTORY (A&E). "History of Meth: Crystal, Bust & Timeline." Includes Benzedrine inhaler marketing (Smith, Kline & French, early 1930s).
11. U.S. Combat Methamphetamine Epidemic Act of 2005 (precursor control; pseudoephedrine point-of-sale restrictions); shift toward phenyl-2-propanone-based illicit production, as documented in policy and investigative literature.
12. Desoxyn (methamphetamine hydrochloride) FDA prescribing information; original NDA approval December 1944 (Abbott), with subsequent narrowing of approved indications to ADHD and short-term obesity.
13. U.S. Drug Enforcement Administration — scheduling of methamphetamine (Schedule II) under the Controlled Substances Act of 1970.
14. United Nations Convention on Psychotropic Substances (1971); international control framework for amphetamine-type stimulants. See also UNODC *World Drug Report* (current edition) for global ATS market data.

End of Installment I — Front Matter & Parts I–II

*Parts III–XIV (Chapters 5–40) and Appendices A–H continue the volume,
following the structure set out in the Contents.*