Amphetamine

NH ₂	
Systematic (IUPAC) name	
(±)-1-phenylpropan-2-amine	
Identifiers	
CAS number	300-62-9 ^[1] 405-41-4 ^[2]
ATC code	N06 BA01 ^[3]
PubChem	CID 3007 ^[4]
DrugBank	DB00182 ^[5]
ChemSpider	13852819 ^[6]
Chemical data	
Formula	C ₉ H ₁₃ N
Mol. mass	135.2084
SMILES	eMolecules ^[7] & PubChem ^[8]
Synonyms	alpha-methyl benzene ethanamine, alpha-methyl phenethyl amine, beta-phenyl-isopropylamine
Physical data	
Melt. point	285–281 °C (545–538 °F)
Solubility in water	50–100 mg/mL (16C°) mg/mL (20 °C)
Pharmacokinetic data	
Bioavailability	Oral 20-25%; nasal 75%; rectal 95–99%; intravenous 100%
Protein binding	15–40%
Metabolism	Hepatic (CYP2D6) ^[9]
Half-life	12h average for d-isomer, 13h for l-isomer
Excretion	Renal; significant portion unaltered
Therapeutic considerations	
Pregnancy cat.	C(US)

Amphetamine

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Legal status	Controlled (S8) (AU) Schedule II (CA) ? (UK) Schedule II (US) Prescription only
Routes	Oral, Intravenous, Vaporization, Insufflation, Rectal, Sublingual
\checkmark (what is this?) (verify) ^[10]	

Amphetamine (USAN) or **amfetamine** (INN) is a psychostimulant drug that is known to produce increased wakefulness and focus in association with decreased fatigue and appetite. Amphetamine is chemically related to methamphetamine and lisdexamfetamine, a class of potent drugs that act by increasing levels of dopamine and norepinephrine in the brain, inducing euphoria.^[11] ^[12] The class includes prescription CNS drugs commonly used to treat attention-deficit hyperactivity disorder (ADHD). It is also used to treat symptoms of traumatic brain injury (TBI) and the daytime drowsiness symptoms of narcolepsy, postural orthostatic tachycardia syndrome (POTS) and chronic fatigue syndrome (CFS). Initially, amphetamine was more popularly used to diminish the appetite and to control weight. Brand names of the drugs that contain, or metabolize into, amphetamine include **Adderall**, **Vyvanse**, and **Dexedrine**, as well as **Benzedrine** in the past.

The drug is also used recreationally and as a performance enhancer. Recreational users of amphetamine have coined numerous street names for amphetamine, such as **speed** and **crank**. The European Monitoring Centre for Drugs and Drug Addiction reports the typical retail price of amphetamine in Europe varied between \in 3 and \in 15 (\$4 to \$21.55 USD) a gram in half of the reporting countries.^[13] The name *amphetamine* is derived from its chemical name: **a**lpha-**m**ethyl**phenet**hyl**amine**.

History

Amphetamine was first synthesized in 1887 by the Romanian chemist Lazăr Edeleanu in Berlin, Germany.^[14] He named the compound phenylisopropylamine. It was one of a series of compounds related to the plant derivative ephedrine, which had been isolated from Ma-Huang that same year by Nagayoshi Nagai.^[15] No pharmacological use was found for amphetamine until 1927, when pioneer psychopharmacologist Gordon Alles resynthesized and tested it on himself, in search of an artificial replacement for ephedrine. From 1933 or 1934 Smith, Kline and French began selling the volatile base form of the drug as an inhaler under the trade name Benzedrine, useful as a decongestant but readily usable for non-medical purposes.^[16] One of the first attempts at using amphetamine as a scientific study was done by M. H. Nathanson, a Los Angeles physician, in 1935. He studied the subjective effects of amphetamine in 55 hospital workers who were each given 20 mg of Benzedrine. The two most commonly reported drug effects were "a sense of well being and a feeling of exhilaration" and "lessened fatigue in reaction to work".^[17] During World War II amphetamine was extensively used to combat fatigue and increase alertness in soldiers. After decades of reported abuse, the FDA banned Benzedrine inhalers, and limited amphetamine to prescription use in 1965, but non-medical use remained common. Amphetamine became a schedule II drug under the Controlled Substances Act in 1971.

The related compound methamphetamine was first synthesized from ephedrine in Japan in 1918 by chemist Akira Ogata, via reduction of ephedrine using red phosphorus and iodine. The pharmaceutical Pervitin was a tablet of 3 mg methamphetamine which was available in Germany from 1938 and widely used in the *Wehrmacht*, but by mid-1941 it became a controlled substance, partly because of the amount of time needed for a soldier to rest and recover after use and partly because of abuse. For the rest of the war, military doctors continued to issue the drug, but less frequently and with increasing discrimination.^[18]

In 1997 and 1998,^[19] ^[20] researchers at Texas A&M University claimed to have found amphetamine and methamphetamine in the foliage of two Acacia species native to Texas, *A. berlandieri* and *A. rigidula*. Previously, both of these compounds had been thought to be human inventions. These findings have never been duplicated, and the analyses are believed by many biochemists to be the result of experimental error, and as such the validity of the report has come into question. Alexander Shulgin, one of the most experienced biochemical investigators and the discoverer of many new psychotropic substances, has tried to contact the Texas A&M researchers and verify their findings. The authors of the paper have not responded; natural amphetamine remains most likely an unconfirmed

discovery.^[21]

Contraindications

Amphetamine elevates cardiac output and blood pressure making it dangerous for use by patients with a history of heart disease or hypertension. Amphetamine can cause a life-threatening complication in patients taking MAOI antidepressants. The use of amphetamine and amphetamine-like drugs is contraindicated in patients with narrow-angle glaucoma or anatomically narrow angles. Like other sympathomimetic amines, amphetamine can induce transient mydriasis. In patients with narrow angles, pupillary dilation can provoke an acute attack of angle-closure glaucoma. These agents should also be avoided in patients with other forms of glaucoma, as mydriasis may occasionally increase intraocular pressure.^[22]

Amphetamine has been shown to pass through into breast milk. Because of this, mothers taking amphetamine are advised to avoid breastfeeding during their course of treatment.^[23]

Major neurobiological mechanisms

Primary sites of action

Amphetamine exerts its behavioral effects by modulating several key neurotransmitters in the brain, including dopamine, serotonin, and norepinephrine. However, the activity of amphetamine throughout the brain appears to be specific;^[24] certain receptors that respond to amphetamine in some regions of the brain tend not to do so in other regions. For instance, dopamine D2 receptors in the hippocampus, a region of the brain associated with forming new memories, appear to be unaffected by the presence of amphetamine.^[24]

The major neural systems affected by amphetamine are largely implicated in the brain's reward circuitry. Moreover, neurotransmitters involved in various reward pathways of the brain appear to be the primary targets of amphetamine.^[25] One such neurotransmitter is dopamine, a chemical messenger heavily active in the mesolimbic and mesocortical reward pathways. Not surprisingly, the anatomical components of these pathways—including the striatum, the nucleus accumbens, and the ventral striatum—have been found to be primary sites of amphetamine action.^[26] ^[27]

The fact that amphetamine influences neurotransmitter activity specifically in regions implicated in reward provides insight into the behavioral consequences of the drug, such as the stereotyped onset of euphoria.^[27] A better understanding of the specific mechanisms by which amphetamine operates may increase our ability to treat amphetamine addiction, as the brain's reward circuitry has been widely implicated in addictions of many types.^[28]

Endogenous amphetamines

Amphetamine has been found to have several endogenous analogues; that is, molecules of a similar structure found naturally in the brain.^[29] l-Phenylalanine and β -Phenethylamine are two examples, which are formed in the peripheral nervous system as well as in the brain itself. These molecules are thought to modulate levels of excitement and alertness, among other related affective states.

Dopamine

Perhaps the most widely studied neurotransmitter with regard to amphetamine action is dopamine, the "reward neurotransmitter" that is highly active in numerous reward pathways of the brain. Various studies have shown that in select regions, amphetamine increases the concentrations of dopamine in the synaptic cleft, thereby heightening the response of the post-synaptic neuron.^[30] This specific action hints at the hedonic response to the drug as well as to the drug's addictive quality.

The specific mechanisms by which amphetamine affects dopamine concentrations have been studied extensively. Currently, two major hypotheses have been proposed, which are not mutually exclusive. One theory emphasizes amphetamine's actions on the vesicular level, increasing concentrations of dopamine in the cytosol of the pre-synaptic neuron.^{[29] [31]} The other focuses on the role of the dopamine transporter DAT, and proposes that amphetamine may interact with DAT to induce reverse transport of dopamine from the presynaptic neuron into the synaptic cleft.^{[25] [32] [33] [34]}

The former hypothesis is backed by data demonstrating that injections of amphetamine result in rapid increases of cytosolic dopamine concentrations.^[34] Amphetamine is believed to interact with dopamine-containing synaptic vesicles in the axon terminal. Amphetamine is a substrate for a specific neuronal synaptic vesicle uptake transporter called VMAT2. When amphetamine is taken up by VMAT2, the vesicle releases dopamine molecules into the cytosol in exchange. The redistributed dopamine is then believed to interact with DAT to promote reverse transport.^[29] Calcium may be a key molecule involved in the interactions between amphetamine and VMATs.^[31]

The latter hypothesis postulates a direct interaction between amphetamine and the DAT. The activity of DAT is believed to depend on specific phosphorylating kinases, such as protein kinase c, specifically PKC- β .^[34] Upon phosphorylation, DAT undergoes a conformational change that results in the transportation of DAT-bound dopamine from the extracellular to the intracellular environment.^[33] In the presence of amphetamine, however, DAT has been observed to function in reverse, spitting dopamine out of the presynaptic neuron and into the synaptic cleft.^[32] Thus, beyond inhibiting reuptake of dopamine, amphetamine also stimulates the release of dopamine molecules into the synapse.^[25]

In support of the above hypothesis, it has been found that PKC- β inhibitors eliminate the effects of amphetamine on extracellular dopamine concentrations in the striatum of rats.^[34] This data suggests that the PKC- β kinase may represent a key point of interaction between amphetamine and the DAT transporter.

Serotonin

Amphetamine has been found to exert similar effects on serotonin as on dopamine.^[35] Like DAT, the serotonin transporter SERT can be induced to operate in reverse upon stimulation by amphetamine.^[36] This mechanism is thought to rely on the actions of calcium ions, as well as on the proximity of certain transporter proteins.^[36]

The interaction between amphetamine and serotonin is only apparent in particular regions of the brain, such as the mesocorticolimbic projection. Recent studies additionally postulate that amphetamine may indirectly alter the behavior of glutamatergic pathways extending from the ventral tegmental area to the prefrontal cortex.^[35] Glutamatergic pathways are strongly correlated with increased excitability at the level of the synapse. Increased extracellular concentrations of serotonin may thus modulate the excitatory activity of glutamatergic neurons.^[35]

The proposed ability of amphetamine to increase excitability of glutamatergic pathways may be of significance when considering serotonin-mediated addiction.^[35] An additional behavioral consequence may be the stereotyped locomotor stimulation that occurs in response to amphetamine exposure.^[30]

Other relevant neurotransmitters

Several other neurotransmitters have been linked to amphetamine activity. For instance, extracellular levels of glutamate, the primary excitatory neurotransmitter in the brain, have been shown to increase upon exposure to amphetamine. Consistent with other findings, this effect was found in the areas of the brain implicated in reward; namely, the nucleus accumbens, striatum, and prefrontal cortex.

Additionally, several studies demonstrate increased levels of norepinephrine, a neurotransmitter related to adrenaline, in response to amphetamine. This is believed to occur via reuptake blockage as well as via interactions with the norepinephrine neuronal transport carrier.^[37]

Pharmacology

Chemical properties

Amphetamine is a chiral compound. The racemic mixture can be divided into its optical isomers: levo- and dextro-amphetamine. Amphetamine is the parent compound of its own structural class, comprising a broad range of psychoactive derivatives, from empathogens, MDA (3,4-Methylenedioxyamphetamine) and MDMA (3,4-Methylenedioxy-N-methamphetamine) known as ecstasy, to the *N*-methylated form, methamphetamine known as 'meth', and to decongestants such as ephedrine (EPH). Amphetamine is a homologue of phenethylamine.

At first, the medical drug came as the salt racemic-amphetamine sulfate (racemic-amphetamine contains both isomers in equal amounts). Attention disorders are often treated using Adderall or a generic equivalent, a formulation of mixed amphetamine and dextroamphetamine salts that contain

- 1/4 dextro-amphetamine saccharate
- 1/4 dextro-amphetamine sulfate
- 1/4 (racemic amphetamine) aspartate monohydrate
- 1/4 (racemic amphetamine) sulfate



Amphetamine molecular structure. Methamphetamine has the same structure, with the addition of a methyl group attached to the nitrogen.



Pharmacodynamics

Amphetamine has been shown to both diffuse through the cell membrane and travel via the dopamine transporter (DAT) to increase concentrations of dopamine in the neuronal terminal.

Amphetamine, both as d-amphetamine (dextroamphetamine) and l-amphetamine (or a racemic mixture of the two isomers), is believed to exert its effects by binding to the monoamine transporters and increasing extracellular levels of the biogenic amines dopamine, norepinephrine (noradrenaline) and serotonin. It is hypothesized that d-amphetamine acts primarily on the dopaminergic systems, while l-amphetamine is comparatively norepinephrinergic (noradrenergic). The primary reinforcing and behavioral-stimulant effects of amphetamine, however, are linked to enhanced dopaminergic activity, primarily in the mesolimbic dopamine system.

Amphetamine and other amphetamine-type stimulants principally act to release dopamine into the synaptic cleft. Amphetamine, unlike similar dopamine acting stimulant cocaine, does not act as a ligand but does slow reuptake by a secondary acting mechanism through the phosphorylation of dopamine transporters.^[38] The primary action is through the increased amphetamine concentration which releases endogenous stores of dopamine from vesicular monoamine transporters (VMATs), thereby increasing intra-neuronal concentrations of transmitter. This increase in concentration effectively reverses transport of dopamine via the dopamine transporter (DAT) into the synapse.^[39] In addition, amphetamine binds reversibly to the DATs and blocks the transporter's ability to clear DA from the synaptic space. Amphetamine also acts in this way with norepinephrine (noradrenaline) and to a lesser extent serotonin.

In addition, amphetamine binds to a group of receptors called Trace Amine Associated Receptors (TAAR).^[40] TAAR are a newly discovered receptor system which seems to be affected by a range of amphetamine-like substances called trace amines.

Effects

Physical effects

Physical effects of amphetamine can include reduced appetite, increased/distorted sensations, hyperactivity, dilated pupils, flushing, restlessness, dry mouth, erectile dysfunction, headache, tachycardia, increased breathing rate, increased blood pressure, fever, sweating, diarrhea, constipation, blurred vision, impaired speech, dizziness, uncontrollable movements or shaking, insomnia, numbness, palpitations, and arrhythmia. In high doses or chronic use convulsions, dry or itchy skin, acne, pallor can occur.^[41] ^[42] ^[43]

Occasionally amphetamine use in males can cause an odd and sometimes startling effect in which the penis when flaccid appears to have shrunk due to vasoconstriction. Upon erection the penis returns to normal size.^[45] However, this may simply be an urban myth. "There are no published scientific reports which provide objective evidence that penile shrinkage occurs as an effect of amphetamine use."^[46]

Young adults who abuse amphetamine may be at greater risk of suffering a heart attack. In a study published in the journal Drug and Alcohol Dependence,^[47] researchers examined data from more than 3 million people between 18 and 44 years old hospitalized from 2000 through 2003 in Texas. After controlling for cocaine abuse, alcohol abuse, tobacco use, hypertension, diabetes mellitus, lipid disorders, obesity, congenital defects, and coagulation defects, they found a relationship between a diagnosis of amphetamine abuse and heart attack.^[48]

Psychological effects

Psychological effects can include euphoria, anxiety, increased libido, alertness, concentration, energy, self-esteem, self-confidence, sociability, irritability, aggression, psychosomatic disorders, psychomotor agitation, hubris, excessive feelings of power and invincibility, repetitive and obsessive behaviors, paranoia, and, with chronic and/or high doses, amphetamine psychosis can occur.^[41]

Based on a study in rats, amphetamine use during adolescence may impair adult working memory.^[49]

Withdrawal effects

Withdrawal from chronic use of amphetamine can include anxiety, depression, agitation, fatigue, excessive sleeping, increased appetite, short temper, psychosis and suicidal thoughts.^[50]

Overdose

An amphetamine overdose is rarely fatal but can lead to a number of different symptoms, including psychosis, chest pain, and hypertension.

Psychosis

Abuse of amphetamines can result in a stimulant psychosis that can present as a number of psychotic disorders (i.e. paranoia, hallucinations, delusions). The intensity and duration of symptoms may vary, but unlike true psychotic disorders (i.e. schizophrenia), stimulant psychoses are not considered to be permanent and will eventually resolve upon discontinuation of the drug's use.

Dependence and addiction

Tolerance is developed rapidly in amphetamine abuse, therefore periods of extended use require increasing amounts of the drug in order to achieve the same effect.^[51]

Performance-enhancing use

Amphetamine is used by some college and high-school students as a study and test-taking aid.^[52] Amphetamine works by increasing energy levels, concentration, and motivation, thus allowing students to study for an extended period of time.

Amphetamine has been, and is still, used by militaries around the world. British troops used 72 million amphetamine tablets in the second world war^[53] and the RAF used so many that "Methedrine won the Battle of Britain" according to one report.^[54] American bomber pilots use amphetamine ("go pills") to stay awake during long missions. The Tarnak Farm incident, in which an American F-16 pilot killed several friendly Canadian soldiers on the ground, was blamed by the pilot on his use of amphetamine.^[55] A nonjudicial hearing rejected the pilot's claim.

Amphetamine is also used by some professional,^[56] collegiate^[57] and high school^[57] athletes for its strong stimulant effect. Energy levels are perceived to be dramatically increased and sustained, which is believed to allow for more vigorous and longer play. However, at least one study has found that this effect is not measurable.^[58] The use of amphetamine during strenuous physical activity can be extremely dangerous, especially when combined with alcohol, and athletes have died as a result, for example, British cyclist Tom Simpson.

Amphetamine use has historically been especially common among Major League Baseball players and is usually known by the slang term "greenies".^[59] In 2006, the MLB banned the use of amphetamine. The ban is enforced through periodic drug-testing. However, the MLB has received some criticism because the consequences for amphetamine use are dramatically less severe than for anabolic steroid use, with the first offense bringing only a warning and further testing.^[60] [61] [62]

Amphetamine was formerly in widespread use by truck drivers^[63] to combat symptoms of somnolence and to increase their concentration during driving, especially in the decades prior to the signing by former president Ronald Reagan of Executive Order 12564, which initiated mandatory random drug testing of all truck drivers and employees of other DOT-regulated industries. Although implementation of the order on the trucking industry was kept to a gradual rate in consideration of its projected effects on the national economy, in the decades following the order, amphetamine and other drug abuse by truck drivers has since dropped drastically. (See also Truck driver—Implementation of drug detection).

Detection in body fluids

Amphetamine is frequently measured in urine as part of a drug abuse testing program, in plasma or serum to confirm a diagnosis of poisoning in hospitalized victims, or in whole blood to assist in the forensic investigation of a traffic or other criminal violation or a case of sudden death. Techniques such as immunoassay may cross-react with a number of sympathomimetics drugs, so chromatographic methods specific for amphetamine should be employed to prevent false positive results. Chiral techniques may be employed to help distinguish the source of the drug, whether obtained legally (via prescription) or illicitly, or possibly as a result of formation from a prodrug such as lisdexamfetamine or selegiline. Chiral separation is needed to assess the possible contribution of 1-methamphetamine (Vicks Inhaler) toward a positive test result.^[64] [65]

Cultural impact of amphetamine

From the 1960s onward, amphetamine has been popular with many youth subcultures in Britain (and other parts of the world) as a recreational drug. It has been commonly used by mods, skinheads, punks, goths, gangstas, and casuals in all night soul and ska dances, punk concerts, basement shows and fighting on the terraces by casuals.

The hippie counterculture was very critical of amphetamines due to the behaviors they cause; in an interview with the Los Angeles Free Press in 1965, beat writer Allen Ginsberg commented that "Speed is antisocial, paranoid making, it's a drag... all the nice gentle dope fiends are getting screwed up by the real horror monster Frankenstein speed freaks who are going round stealing and bad-mouthing everybody".^[67]

In literature

The writers of the Beat Generation used amphetamine extensively, mainly under the Benzedrine brand name. Jack Kerouac was a particularly avid user of amphetamine, which was said to provide him with the stamina needed to work on his novels for extended periods of time.^[68]

Scottish author Irvine Welsh often portrays drug use in his novels, though in one of his journalism works he comments on how drugs (including amphetamine) have become part of consumerism and how his novels Trainspotting and Porno reflect the changes in drug use and culture during the years that elapse between the two texts.^[69]

Amphetamine is frequently mentioned in the work of American journalist Hunter S. Thompson. Speed appears not only amongst the inventory of drugs Thompson consumed for what could broadly be defined as recreational purposes, but also receives frequent, explicit mention as an essential component of his writing toolkit,^[70] such as in his "Author's Note" in *Fear and Loathing on the Campaign Trail '72*.^[71]

"One afternoon about three days ago [the publishers] showed up at my door with no warning, and loaded about forty pounds of supplies into the room: two cases of Mexican beer, four quarts of gin, a dozen grapefruits, and enough speed to alter the outcome of six Super Bowls. ... Meanwhile, [...] with the final chapter still unwritten and the presses scheduled to start rolling in twenty-four hours . . . unless somebody shows up pretty soon with extremely powerful speed, there might not *be* a final chapter. About four fingers of king-hell Crank would do the trick, but I am not optimistic."

In mathematics

Famous mathematician Paul Erdős took amphetamine, and once won a bet from his friend Ron Graham, who bet him \$500 that he could not stop taking the drug for a month.^[72] Erdős won the bet, but complained during his abstinence that mathematics had been set back by a month: "Before, when I looked at a piece of blank paper my mind was filled with ideas. Now all I see is a blank piece of paper." After he won the bet, he promptly resumed his amphetamine habit.

In music

Many songs have been written about amphetamine, for example in the track entitled "St. Ides Heaven" from singer/songwriter, Elliott Smith's self-titled album. *Semi Charmed Life* by Third Eye Blind also references amphetamine. Another blatant example would be the song simply labelled "Amphetamine" by Alternative rock band Everclear. It has also influenced the aesthetics of many rock'n'roll bands (especially in the garage rock, mod R&B, death rock, punk/hardcore, gothic rock and extreme heavy metal genres). Hüsker Dü, Jesus and Mary Chain's and The Who were keen amphetamine users early in their existence. Land Speed Record is an allusion to Hüsker Dü's amphetamine use.

Many rock'n'roll bands have named themselves after amphetamine and drug slang surrounding it. For example Mod revivalists, The Purple Hearts named themselves after the amphetamine tablets popular with mods during the 1960s,

as did the Australian band of the same name during the mid 1960's. The Amphetameanies, a ska-punk band, are also named after amphetamine, but also imitate its effects. Dexy's Midnight Runners, of number one hit "Come On Eileen" are named after Dexedrine. Motörhead named themselves after the slang for an amphetamine addict.

In film

Producer David O. Selznick was an amphetamine user, and would often dictate long and rambling memos under the influence of amphetamine to his directors.^[73] The documentary *Shadowing The Third Man* relates that Selznick introduced Third Man director Carol Reed to the use of amphetamine, which allowed Reed to bring the picture in below budget and on schedule by filming nearly 22 hours at a time.^[74]

Garrett Scott's documentary *Cul-de-Sac: A Suburban War Story* has a brief history of the manufacture and spread of amphetamine, and of its effects.^[75]

In the film *Requiem for a Dream*, Ellen Burstyn portrays Sara Goldfarb, an elderly widow who becomes addicted to weight-loss amphetamine pills. After suffering from amphetamine psychosis, she is hospitalized against her will, undergoes electro-convulsive therapy, and later on was confined at a mental asylum.^[76]

The title of the 2009 movie *Amphetamine* plays on the double meaning of the word in Chinese - besides the name for the drug it also means 'isn't this his fate?' which figuratively ties to the movie's plot. The word is transliterated as 安非他命 - "ān fēi tā mìng" - and as commonly happens with transliteration of non-Chinese terms each character has independent meaning as an individual unrelated word.

In the 1972 film *Ciao! Manhattan*, Edie Sedgwick portrays Susan Superstar, a fictional version of herself, who discusses her addiction with drugs, mainly Amphetamines. In one particular scene, she talks about the exhilaration of her drug addiction in the infamous "Speed Monologue."^[77]

Legal status

- In the United Kingdom, amphetamines are regarded as Class B drugs. The maximum penalty for unauthorized possession is five years in prison and an unlimited fine. The maximum penalty for illegal supply is fourteen years in prison and an unlimited fine. Methamphetamine has recently been reclassified to Class A, penalties for possession of which are more severe (7 years in prison and an unlimited fine).^[78]
- In the Netherlands, amphetamine and methamphetamine are List I drugs of the Opium Law, but the dextro isomer of amphetamine is indicated for ADD/ADHD and narcolepsy and available for prescription as 5 and 10 mg generic tablets, and 5 and 10 mg gel capsules.
- In the United States, amphetamine and methamphetamine are Schedule II drugs, classified as CNS (central nervous system) stimulants.^[79] A Schedule II drug is classified as one that has a high potential for abuse, has a currently-accepted medical use and is used under severe restrictions, and has a high possibility of severe psychological and physiological dependence.

Internationally, amphetamine is a Schedule II drug under the Convention on Psychotropic Substances.^[80]

Prodrugs

A number of substances have been shown to produce amphetamine and/or methamphetamine as metabolites, including amphetaminil, benzphetamine, clobenzorex, dimethylamphetamine, ethylamphetamine, famprofazone, fencamine, fenethylline, fenproporex, furfenorex, lisdexamfetamine, mefenorex, mesocarb, prenylamine, propylamphetamine, and selegiline, among others.^[81] ^[82] These compounds may produce false positives for amphetamine on drug tests.^[81] ^[82]

See also

- Psychostimulant
- Methamphetamine
- Dextroamphetamine
- Adderall
- Lisdexamfetamine
- Amphetamine psychosis
- Attention-deficit hyperactivity disorder
- Methylphenidate
- Benzylpiperazine
- Clandestine chemistry
- Ethylamphetamine
- Phenethylamine
- Propylamphetamine
- Releasing agent

External links

- CID 5826^[83] from PubChem (D-form—dextroamphetamine)
- CID 3007^[4] from PubChem (L-form and D, L-forms)
- CID 32893 ^[84] from PubChem (L-form—Levamphetamine or L-amphetamine)
- List of 504 Compounds Similar to Amphetamine (PubChem)^[85]
- EMCDDA drugs profile: Amphetamine (2007)^[86]
- Drugs.com Amphetamine ^[87]
- Asia & Pacific Amphetamine-Type Stimulants Information Centre [88]
- U.S. National Library of Medicine: Drug Information Portal Amphetamine [89]

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