CRACK & COCAINE

LEVEL 1 HANDBOOK

TD consultancy

CRACK AND COCAINE

Level 1: Foundation

TD Consultancy

2026 Edition

www.tonydagostino.co.uk

1. Introduction and How to Use This Pack

The pack will aid service providers in building competencies and knowledge bases to develop specific crack and cocaine services or to orient generic services to be more successful in working with this client group.

Working with crack and cocaine users does not mean learning and developing totally new skills. What it does mean is that there is a shift in how treatment and support are thought about.

How to Use This Training Pack

This pack is intended for use as a learning tool and for reference after the training course has been completed. It is not intended for use in isolation from the training course.

Not all the material provided in this pack will be relevant to all services. Much will depend on the services they offer and the context in which they are provided.

Agencies and individuals will also need to take into account the regional differences in patterns of use within their catchment area, the type of service being offered and the target group.

UPDATE: Capability Framework for the Drug and Alcohol Treatment and Recovery Workforce

This training resource aligns with the Capability Framework for the Drug and Alcohol Treatment and Recovery Workforce (NHS England / Office for Health Improvement and Disparities, 2024), which underpins the 10-Year Strategic Plan for the Drug and Alcohol Treatment and Recovery Workforce 2024–2034.

The framework provides guidance on the knowledge, skills and behaviours required for core roles within LA-commissioned drug and alcohol treatment and recovery services. This manual supports workforce development towards those capabilities and is intended to be used as part of a broader training, supervision and professional development plan.

Audience	Guidance
Managers of services	Managers of treatment services can use this pack to help identify the skills and staff mix required to develop or improve services for crack and cocaine users.
Staff of services	Staff of treatment services can use the sample tools and forms to work with crack and cocaine users.

2. Crack and Cocaine History Timeline

Main Coca Growing Regions

'Coca' comes from the Tiwakanu word 'Khoka' meaning 'The Tree'.

5000 BC	Fragment of pottery found by archaeologist Ronald K. Siegel depicts the use of coca leaf in an event that took place around this period.
3000 BC	Coca chewing is practised throughout South America to help with working at high altitude and to stave off hunger. It is thought to be a gift from the Gods. Also used in embalming and trepanning (as a local anaesthetic). Also some evidence to suggest that it was used in Egypt at the same time.
1000 AD	Incan civilisation begins. The civilisation eventually rises to incorporate 75% of the Pacific coast of South America. Coca is very important to the Inca's and is incorporated into almost every aspect of daily life including their measurement of distance and time. Cocada: The time it takes to use a wad of coca leaves and the distance covered in that time (approximately 45 minutes).

1400	Coca plantations are operated by the Incas in Peru. Chewed coca used on wounds as an anaesthetic.
1492	Columbus strikes land which he mistakes for China and begins a process which will swiftly bring about the fall of the Incan civilisation. The estimated population of the Incan empire 14,000,000.
1505	The first reports of coca use reach Europe.
Early 1500s	First commercial production of coca by Europeans as holders of Spanish land grants are allowed to make their tax payments in coca leaves.
1548	Silver miners getting through over half a million kilograms of coca per year.
1552	Because of inaccurate reports on coca the Catholic Church decide that they want to ban the use of the leaf (probably one of the first anti-drug campaigns).
Mid 1500s	Forced labourers working in Spanish silver mines are kept well supplied with coca leaves. Roughly 8% of Europeans living in Peru are involved in the coca trade.
1577	The Church withdrew their petition to ban coca some years earlier when a 10% tax was levied on its purchase. This tax was given to the Church and helped support its growth in South America.
1600s	Coca first brought to Western culture. Some Europeans chewing for stimulation.
1662	Abraham Cowley writes a poem titled 'A Legend of Coca'. This is the first independent mention of coca in English literature.
1708	German physician and botanist Herman Boerhaave first mentions coca with regards to a use as medicine.
1835	First accurate drawing of coca appears in popular English press. Drawing by Sir William Hooker who was then the director of Kew Gardens.
1860	Cocaine is first isolated from the coca leaf by Albert Neiman. He receives his PhD for his efforts.
1870	Vin Mariani (cocaine wine) is on sale throughout France and also exported. Bottles contain 7.2 mg per ounce of wine. The Dutch are growing coca in Indonesia and the British in Ceylon. Germany and the USA are getting crops from plantations in South America.
1876	Race walkers in England chew coca leaves to enhance their performance.

1878	Cocaine is held up as a wonder drug that can cure the morphine addiction faced by many American Civil War veterans. Dr W.H. Bentley publishes these findings in the Therapeutic Gazette.
1884	Cocaine is used as a local anaesthetic in eye surgery and receives much attention from the medical profession. Freud publishes 'On Coca' in which he recommends that cocaine be used in a variety of psychiatric conditions. More importantly, Freud saw cocaine as a cure for morphine dependency. Freud also treated his depression with cocaine, and reported feeling "exhilaration and lasting euphoria, which is in no way different from the normal euphoria of the healthy person. You perceive an increase in self-control and possess more vitality and capacity for work. In other words, you are simply more normal, and it is soon hard to believe that you are under the influence of a drug." [Quoted in Ernest Jones, The Life and Work of Sigmund Freud, Vol. 1, p. 82]
1884	Coca-Cola is first introduced by John Pemberton. The drink contained two main stimulants, coca leaves and kola nuts, hence its name.
Late 1800s	The manufacture of refined cocaine starts. Coca plantations start to appear in areas other than South America, such as India and Indonesia.
Early 1900s	The Pure Food and Drug Act was passed, forming the Food and Drug Administration and giving it power to regulate foods and drugs, and requiring labelling of contents on foods and drugs. Cocaine is withdrawn from tonics due to its adverse effects and addictive properties. Products containing cocaine had to be labelled as poisons.
1905	Snorting cocaine starts to become popular, particularly in the United States. It is carried around in small ornate boxes and snorted in a similar fashion to snuff. Early cocaine snorting was known as 'cocaine snuffing'.
1910	The first case of nasal damage due to cocaine snorting reported in American medical literature. Harrods selling cocaine over the counter.
1911	5,000 cocaine and heroin related deaths are reported by the U.S. government. To prolong the opium trade Britain forces the U.S.A. to put cocaine on the international drugs agenda.
1914	Cocaine and opiates are banned as illegal substances in the U.S.A under the Harrison Act. Start of First World War in Europe. Cocaine given to British troops in the form of 'Forced March Tablets'.
1916	The Defence of the Realm Act introduced which makes it illegal for cocaine and morphine to be sold to British Armed Forces.
1920	Cocaine is banned as an illegal substance in the UK under the Dangerous Drugs Act. This started an illegal importation trade. Some enterprising

	traffickers used homing pigeons sent from France to bring in a gram at a time.
1920s	Evidence of a 'modern' underground drug scene emerging in Soho, London.
Early 1930s	Japan is world's leading cocaine producer, followed by the U.S.A., Germany, Great Britain and then France. China recovering from the British opium trade now had to endure Japan flooding the market with cocaine. Hitler condemns the use of cocaine, a popular society drug in the 1920s that the Nazis called "devil's stuff".
1944	Nazi researchers used concentration camp inmates to test a cocaine-based "wonder drug" they hoped would enhance the performance of German troops.
1950s	Cocaine starting to regain popularity. Used by the "famous set" where it begins to get its rich drug image; also very expensive at this time. Production has now shifted to South America, though Peru was legally producing cocaine from the 1880s to 1950.
1960s	Again regaining popularity with the drug revolution of the 60s era. Media yet again play down its dependent qualities as in the late 1800s. Importation starting to be controlled by the Cubans.
Early 1970s	Freebase cocaine starts to be used. At first it is washed with ether and then ammonia. Colombians are now starting to gain control of the American and world market. USA flooded with cocaine, especially Miami and New York.
Late 1970s	Crack cocaine use starts in the United States. It is initially named 'ready rock' or 'garbage freebase' and later is called crack once it hits the New York street market. Crack becomes more popular because it is easier to mass-produce with the use of bicarbonate of soda and a microwave oven. During the 1970s a small group of doctors in California prescribe 'Esterene' to chronic rheumatoid arthritis sufferers. Esterene was freebase cocaine prepared for nasal application.
1983	Crack cocaine starts to be sold in London. Crack utilises established dealing networks to begin to establish a foothold.
1989	Robert Stutman addresses ACPO conference and helps introduce the strong stereotypes associated with crack. In one presentation he introduced the ideas that crack was: almost instantly addictive; particularly attractive to young people; an incredibly violent drug; mainly controlled by Jamaicans.

1993	Crack/cocaine overtakes heroin in estimated importation amounts. Ten years after its introduction the first outreach services for crack users start to appear in London and Nottingham.
1995	Task force report on crack and cocaine published and outlines that crack users do not see drug services as being for them. First structured programmes for crack and cocaine users open.
1998	A survey by Columbia University found that 21% of violent felons in state prisons committed their crimes under the influence of alcohol alone, and only 3% were under the influence of crack or cocaine.
2000	Crack and cocaine use are increasing in the UK. Used as far south as Jersey and as far north as Aberdeen. Spread to injecting users and now also being distributed through heroin dealing networks. Also gaining popularity within the dance drug culture and bars and clubs. Seventeen years after its introduction to the UK it is still marginalised as a drug with regards to treatment and national strategy.
2003	Crack and cocaine use is now beginning to be taken seriously by various government departments such as the NTA, Home Office and Cabinet Office. This is helping to ensure that the issue is discussed and thought about. However the stereotypes and myths surrounding the use of this drug still continue to interfere with the development of rational policies and practice.
2004	Crack pilot projects launched.
Late 2000s–2010s	Throughout the 2000s, crack remains concentrated among the most marginalised opiate-using populations but begins to appear more widely in some urban night-time economies. By the mid-2010s, official estimates show an 8–10% rise in the number of people using crack in England compared with the early 2010s.
2011–2017	Public Health England and the Home Office report a statistically significant increase in estimated crack users in England from about 166,600 in 2011–12 to around 180,700 in 2016–17 (up 8.5%). Among people who inject drugs, crack use rises from 39% in 2013 to 53% in 2017.
Late 2010s – County Lines Era	Crack becomes closely linked with 'county lines' supply models, with organised crime groups expanding crack distribution from major cities into smaller towns and coastal areas. Treatment data show a sharp rise in adults entering treatment for crack between 2015–16 and 2017–18 (around a 19% increase). The practice of 'cuckooing' – where dealers take over vulnerable people's homes to use as drug dealing bases – becomes widespread.

2020–2023	Opiate and crack cocaine use together affects an estimated 300,000+ people in England. Drug-poisoning data show 1,118 cocaine-related deaths in England and Wales in 2023, nearly ten times the level in 2011 (112 deaths). By 2024, this rises further to 1,279 deaths.
2022	The UK Government launches 'From Harm to Hope: a 10-year drugs plan to cut crime and save lives', committing over £3 billion to expand treatment, disrupt county lines, and reduce drug-related deaths.
2023–2026	Updated OHID prevalence estimates for 2022–23 report 310,718 opiate and/or crack users in England (rate of 8.5 per 1,000 population). Highest rates in North East (12.8), Yorkshire and the Humber (11.6), and North West (11.3). NDTMS data for 2024–25 shows 329,646 adults in contact with treatment services, with 19% entering treatment for crack cocaine problems.

3. Who Is Using Crack and Cocaine?

Both crack and cocaine are very widely used drugs and transcend across all cultural, age and social groups. It is therefore important that agencies consider the wider spectrum of users and their needs when setting up services.

Although government statistics will place the majority of crack use in deprived areas, there needs to be awareness that most of this evidence is rooted within the criminal justice system and does not identify those who are not supporting their habit through criminal activity.

Type of User	Characteristics
The Light Recreational User	• Does not buy crack or cocaine • Uses small amounts at social occasions • Does not have a pattern of use • Value systems are unaffected • Has minimal contact with crack or cocaine
The Recreational User	• Buys crack or cocaine infrequently, usually to share with friends • Uses crack or cocaine socially when available as a recreational diversion • Begins to establish a recognisable pattern of use • Value systems are unaffected • Relationships and work are not affected • Does not show psychological or physiological signs of use • Begins to make contacts to obtain crack or cocaine
The Binge User	• Actively seeks crack or cocaine and will buy increased quantities • Plans social activities involving crack or cocaine • A recognisable pattern of use is firmly established, with the user isolating themselves from others • Value systems change • Relationships and work become affected • May begin to sell drugs to maintain supply • Binge behaviour may include perceived loss of control over drug use, using large quantities at one time and life-threatening patterns of use • May show psychological and physiological signs of use • May combine crack or cocaine with other drugs • Finances may be negatively affected

The Chronic User	• Will consume as much as possible • Demonstrates a life-threatening pattern of use • Values are entirely centred on crack/cocaine use • Relationships and work significantly affected or non-existent • Extreme psychological and physiological signs of use present (depression, weight loss, seizure, depersonalisation)
The Dependent User with Complex Needs (NEW)	• Multiple and overlapping needs including homelessness, mental health conditions, involvement with the criminal justice system, and physical health problems • Often long-term crack and/or opiate use • May experience trauma, domestic abuse, or exploitation • Treatment presentations characterised by high attrition, chaotic engagement, and complex safeguarding concerns • Requires coordinated multi-agency support (housing, mental health, social care)

**Information from Wheeler Street Crack Outreach Team*

INTERSECTIONALITY AND INEQUALITIES

Evidence from the OHID prevalence estimates (2022–23) and NDTMS data highlights significant inequalities in crack use and treatment access. People experiencing homelessness, those with care experience, and those involved in the criminal justice system are disproportionately represented in crack treatment populations. Research by the Race Equality Foundation has identified that Black and minoritised communities face additional barriers to accessing treatment, including stigma, cultural inappropriateness of services, and systemic racism within health systems. In 2024–25, 19% of all adults entering treatment reported crack cocaine as a problem substance, with 32,399 people starting treatment for crack.

4. Types of Cocaine

The coca leaf has been chewed in South America for over 3,000 years. It was not until the mid-1800s that the active ingredient was isolated and the first cocaine was manufactured. Since then cocaine has been re-invented in many ways according to markets and users' preferences.

There are around 200 species of erythroxylon plants. At least 17 produce cocaine. Only two of them, erythroxylon coca and erythroxylon novogranatense, typically yield enough cocaine to justify commercial cultivation and can be harvested four times a year.

When the coca leaf is harvested, it is put into large vats, crushed, pressed (similar process to making wine) and then put through a manufacturing process that includes the use of kerosene and ammonia. This removes the active ingredient, forming a paste commonly known in South America as 'Basuco'.

To refine it further, the coca paste is again subjected to various chemical processes to produce its salt form, cocaine hydrochloride. Cocaine hydrochloride is usually cut during the process and route of importation into the UK.

Cuts are often made with substances that can mimic the anaesthetic effect of cocaine or look similar to cocaine.

Cocaine Hydrochloride: The Salt Form

Chemical Nature: A Salt

- (somewhat similar structure to table salt).

Solubility: Highly water-soluble.

- Dissolves in mucous membranes (snorting) or water (injecting).

Melting Point: Very High.

Why it isn't smoked often:

- High heat destroys most of the psychoactive properties before vaporisation. It burns rather than turning into smokable vapour.
- Known as powder, sniff, toot, coke, snow etc.

Cocaine Hydrochloride

- Route: Mainly snorted (but can also be injected and ingested)
- Effect: Starts to take effect within a few minutes and gradually rises to full high in 15–30 minutes. Come-down is also more gradual.
- Cost: £30–120 per gram (updated 2024–25)
- Purity: Average now 70–90%;
- Cuts: Most common cutting agents now include levamisole, benzocaine, lidocaine, caffeine, and lactose. Note that as purity has increased, cutting has decreased.

Freebase Cocaine

There are three basic methods to freebase cocaine: the Ether Method, the Ammonia Method, and the Baking Soda Method.

The Ether and Ammonia methods were first developed by drug dealers in the 1970s to test the purity of cocaine hydrochloride by removing the hydrochloride (salt). Ether or Ammonia is combined with water and cocaine and then heated. The crystallised form of cocaine is now returned to a base form, making it easier to smoke.

- Form: Crystallised cocaine (Base Form)
- Route: Mainly smoked (but can also be injected)
- Effect: Starts to take effect within 5–10 seconds, giving a short and very intense high (4-5 mins). Come-down can be very rapid and low.
- Cost: Mainly self-manufactured, but if sold, same price as crack (£10–20 per 0.1 to 0.2 of a gram 'rock')

- Purity: Average between 70% to 90% (but can be lower)

Crack Cocaine: Baking Soda Method

This involves a similar process to 'freebase', but uses bicarbonate of soda instead of ether or ammonia. Crack is technically freebase. The name 'crack' comes from the fact that the bicarbonate of soda is not as efficient as ether or ammonia at freeing the 'base' and residues of salt and bicarb are left, causing it to crackle when smoked. This form of cocaine can be easily manufactured at home, leading to its popularity and abundance.

- Form: Crystallised cocaine (Base Form)
- Route: Mainly smoked or injected
- Effect: As with freebase (starts to take effect within 5–10 seconds if smoked)
- Cost: As with freebase £10–20 per rock; some people will sell it for £5 but these are smaller rocks
- Purity: Typically 60–80%; quality directly reflects the purity of the source powder cocaine

General note: The differences between these types of cocaine are similar to the differences between types of alcohol. They all have different tastes and strengths, but at the end of the day, they all produce intoxication. There is no safe way to take cocaine; they all have their dangers and complications according to the route used.

CUTTING AGENTS AND ADULTERATION RISKS

The landscape of cocaine adulteration has changed significantly. Levamisole, originally a veterinary anti-parasitic, is found in a high proportion of cocaine samples. It is associated with agranulocytosis (severe reduction in white blood cells), skin necrosis, and vasculitis. Of emerging concern, the ACMD has issued alerts regarding the potential of synthetic opioids (including nitazenes) to contaminate stimulant drug supplies, though this remains rare in the UK compared to North America. Drug checking services such as WEDINOS play an increasingly important role in monitoring these risks.

The Chemical Evolution of a Substance

To treat and understand cocaine use, one must understand the drug's journey from a plant leaf to a processed chemical.

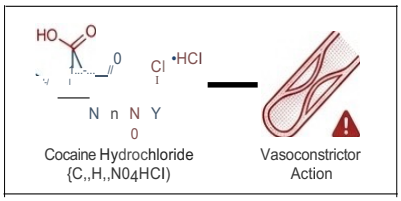
1. The Origin (The Leaf)

A natural vasodilator used for altitude adaptation in the Andes.

Vasodilation Mechanism

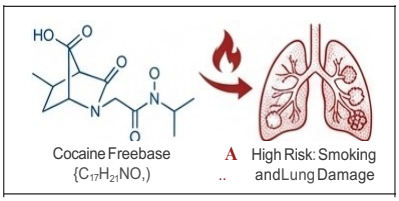
2. The Salt (Powder)

A chemical extraction (Cocaine Hydrochloride) that is water-soluble and acts as a vasoconstrictor.



3. The Base (Crack/Freebase)

A chemical conversion to the alkaloid state lowers the melting point for smoking.



Cocaine Base and Salt Forms

There are basically two different states of cocaine:

When cocaine is first produced, it is in its base form. Hydrochloric acid is then used in a process to turn it into a salt form, which is now cocaine hydrochloride.

When ammonia, ether or bicarbonate of soda are used in the preparation of freebase or crack, the cocaine is being returned to its base form. In its base form, it is far easier to smoke as the melting point has been reduced, hence the process before smoking.

Form	State	Type
Base form	Base	Freebase or Crack Cocaine
Salt form	Neutral	Cocaine Hydrochloride or crack prepared for injection using an acid

Preparation for Injection

Crack cocaine or freebase cocaine do not dissolve in water. They must be prepared for injection with citric acid or vitamin C.

When acids are used to convert crack cocaine into an injectable form, the cocaine is converted into salt form. But the form of the cocaine is dependent on the type of acid used:

- Vitamin C – changes crack into cocaine ascorbate
- Citric Acid – changes crack into cocaine citrate

Cocaine hydrochloride is in a salt form, so it does not require an acid to be added and dissolves in water alone.

Polydrug Use

The development of the polydrug culture in the UK and changes in dealing networks mean that both crack and cocaine have become more widely available and have increased drug combinations and routes of use:

Drug / Combination	Routes	Effect	Types of User
Cocaine & Alcohol	Usually cocaine snorted and alcohol oral.	Produces cocaethylene in the liver, which in itself interacts with the reward system to produce a 'high'. Systematic reviews show an 18–25 times greater risk of sudden death compared to cocaine alone.	One of the most common combinations in the UK. Recreational, binge and chronic users.

Crack & Heroin (snowball, speedball)	Can be taken one after the other by smoking or injecting routes. Can also be combined together in injectable form.	When taken together cocaine and heroin seem to boost each other's effect leading to a very intense high. Also prolongs the come-down.	This form of use is usually associated with chronic users. However there have been reports of heroin use within dance culture.
Crack & Cannabis	Crack can be added to a joint along with cannabis and smoked.	A less intense 'high' with cannabis alleviating the come-down.	Recreational and sometimes used in clubs or by dealers because of the decreased intensity.
Cocaine & Ketamine (CK1)	Usually snorted alternately or in a combined 'line'.	This combination produces feelings of euphoria combined with 'out of body' experiences.	Recreational mainly, but can also be used by chronic and binge users.
Cocaine & MDMA/Ecstasy (updated)	Usually the ecstasy is taken orally and the cocaine snorted.	Cocaine boosts the euphoric effect. Risk of serotonin syndrome – a potentially life-threatening condition – particularly if combined with SSRIs. Both drugs increase heart rate and body temperature.	Mainly recreational but can also fit into binge patterns of use.
Cocaine & Viagra	Cocaine snorted and Viagra taken orally.	Cocaine can heighten sexual experiences as can Viagra. Combined use significantly increases cardiovascular risk.	Recreational use.
Cocaine & Steroids	Both drugs taken separately.	Both drugs can cause complications with moods. Increased cardiovascular risk.	Recreational use.

Crack & Amphetamine	Can be taken separately or may be combined in a 'rock'.	Both these drugs work in a similar way, but amphetamines release dopamine rather than prevent re-uptake.	Recreational, chronic and binge patterns of use.
Cocaine + GHB/GBL	Both taken orally or cocaine snorted with GHB/GBL ingested.	In chemsex contexts, cocaine may be used to counteract the sedating effects of GHB/GBL, creating a dangerous cycle of stimulant and depressant use. Risk of GHB/GBL overdose is high. Associated with sexual health risks including STI transmission.	Recreational, chronic and binge patterns particularly in chemsex settings.
Crack + Benzodiazepines (including street benzos)	Crack smoked and benzodiazepines taken orally or injected.	Street benzodiazepines may contain unpredictable doses or be adulterated with nitazenes (synthetic opioids). Combined depressant and stimulant use increases risk of fatal overdose. ACMD alerts (2024) have highlighted growing nitazene contamination.	Chronic, complex needs users. High fatality risk.
Crack + Synthetic Opioids	Risk of unintentional exposure through contaminated supply rather than intentional combination.	Nitazenes have been detected in drug samples across England and Wales, though contamination of stimulant supplies remains uncommon in the UK.	All user types; risk is primarily unintentional.

COCAINE AND THE LAW

Crack and cocaine are controlled under the Misuse of Drugs Act 1971 and are categorised as Class A drugs. It is illegal to produce, supply, and possess them. Cocaine can only be legally used for certain medical purposes such as a local anaesthetic for plastic surgery. Maximum penalties: possession – 7 years imprisonment and/or unlimited fine; supply/production – life imprisonment and/or unlimited fine. The Sentencing Council updated its Drug Offences Definitive Guideline in 2021.

5. How Cocaine Works

The first thing to say about crack and cocaine is that they are not physically dependent in the way that we understand heroin dependence; however, they create a very strong psychological dependence. Crack and cocaine work by triggering the release of chemicals that are already present in the body. It is important to note that these chemicals are part of the body's response to danger and pleasure.

Adrenaline

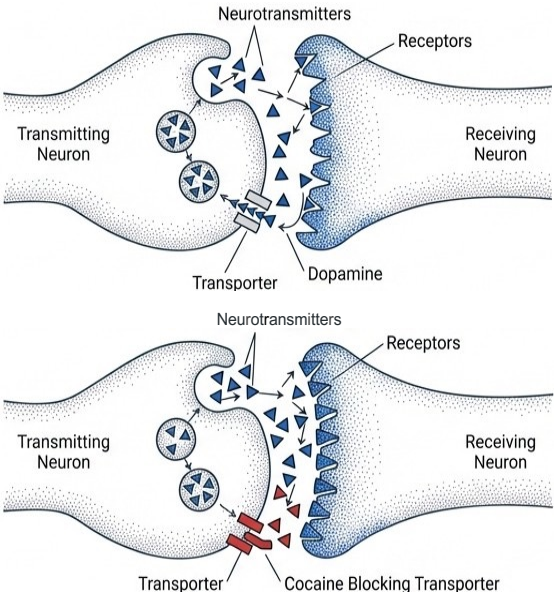
Adrenaline is normally released as part of a response to danger or excitement, heightening the senses and enabling the body to perform at peak. It does this by:

- Increasing heart rate: This increases blood flow throughout the body, which also speeds the delivery of oxygen to muscles.
- Increasing breathing rate: Short and shallow breaths increase the amount of oxygen in the bloodstream.
- Butterflies in the stomach: This is due to blood leaving the stomach and being diverted to the arms and legs, where it is most needed.
- Sweating: The body gets hotter, and sweating is its cooling system.
- Shaking: This is due to the increased energy ready for release. Muscles are primed and ready to go into action.

Users may interpret the above symptoms as the feelings they get when craving for crack or cocaine, or when they are just about to use. When they do use crack or cocaine, they are again releasing adrenaline because of cocaine's effect on the neurotransmitter noradrenaline that controls the adrenal system. The persistent release of adrenaline caused by cocaine use can lead to decreased need for sleep, loss of appetite, visual and auditory

hallucinations, impaired cognitive ability (due to lack of sleep), severe anxiety and paranoia. The environment that someone is in can also affect these feelings. For instance, if used in a hostile environment like a crack house or with someone they do not trust, then the feelings of anxiety and paranoia can be worse.

Neurobiology: The Mechanics of the 'High'



- **Step 1. Normal Function:** Neurotransmitters (Dopamine) released, signal sent, then reabsorbed (reuptake).
- **Step 2. Cocaine's Action:** Acts as a Reuptake Inhibitor. It blocks the transporter channels.
- **Step 3. Result:** Dopamine floods the synapse, firing continuously.
- **Step 4. The Crash:** Brain depletes supply of neurotransmitters, causing depression/exhaustion.

Dopamine and Serotonin

The 'high' experienced when taking crack or cocaine is produced by the chemicals dopamine and serotonin. Cocaine changes the way the brain works by changing the way the nerve cells (neurones) communicate with each other. Nerve cells in the brain normally send messages to each other using chemicals called neurotransmitters. These neurotransmitters cross a synaptic gap and bind to receptor sites. Once the message has been received, a transporter cell collects the neurotransmitters to keep their levels balanced.

Dopamine and serotonin are neurotransmitters that help control the feelings of pleasure and are released by the use of cocaine. But by taking cocaine, the transporter cell is blocked and does not return these neurotransmitters. This leads to the extended feelings of pleasure that are experienced when taking cocaine, and also ultimately leads to the 'downs' experienced by causing a depletion in these chemicals because they cannot be replenished. Imagine getting a brand new credit card: you have extended spending power for a period of time, you have fun, and then the bill arrives through your letterbox.

'Chasing that high' is a lost cause because the more that people use, the more blocks they are putting in place and the less dopamine they have. After their first hit, they will be on a downward spiral, and it is impossible to reach the heights they are aiming for. In this way, all that is really happening is that they are convincing themselves that 'this will be the one' and the next, and the next...

The depletion of dopamine is partly responsible for the 'come down' or 'crash', making users feel bad and reinforcing the need for another hit. Depletion in these neurotransmitters can also cause a chemical depression, which can sometimes combine with bad things happening in their lives (loss of a job, a partner, etc.) and lead to suicidal thoughts. It may also lead to users experiencing severe mood changes.

Combination

The combination of increased adrenaline levels and low dopamine levels after a period of use can produce the feelings of being 'wired' or 'prang'. Users may at this stage use a 'downer' drug like alcohol, cannabis or heroin to help them cope with this feeling.

Chasing the Original High

Many users report chasing their early highs even though this may have been years ago, and their experience tells them that they will not achieve those feelings again.

Adrenaline, Dopamine and Serotonin: Summary Chart

Below is a chart that will explain further how crack and cocaine affect both the mind and body:

Adrenaline	Dopamine and Serotonin
Initial release: (craving, anticipation) Danger and excitement • Increased heart rate • Faster breathing • Sweating • Shaking / cannot stay still • Butterflies / sickness in stomach	**Initial release: (first high / hit)** Reward and reinforcement • Very strong first high • Feelings of confidence • Euphoric / orgasmic • Compulsion to use again
Prolonged release: (continued use can cause the following) • Cannot sleep • Do not want to eat • Increased anxiety ('wired' or 'prang') • Harder to think clearly • Hallucinations (also related to brain chemicals) • Paranoia	**Prolonged release: (depletion of dopamine)** • Repeated compulsion to use • Buzz getting shorter and lower • Come-down or 'crash' • Loss of interest in things not related to cocaine • Mood swings • Depression

Cravings and Compulsion to Use

The urge to use crack or cocaine comes from a combination of the effects of adrenaline and dopamine. To begin with, adrenaline is usually released by a 'trigger' (something that is associated with crack or cocaine use), such as meeting someone they use with, emotional feelings or getting the money to use. This causes the symptoms described above, and suddenly they can be on the 'mission' to use and feeling agitated or full of anticipation at the thought of using. However, when they have used it once, the compulsion to use is created by dopamine. Dopamine works within the primitive areas of the brain and is partly responsible for the drive that we experience to seek food and have sex, etc. Taking crack or cocaine exaggerates this drive and reinforces drug-seeking behaviour, leading to continued use of the drug even when users know that the 'high' cannot be reached again.

Crack and Cocaine vs Heroin: Comparison

Crack and Cocaine	Heroin
Effects on the Brain: Cocaine works by stimulating pleasure-giving neurotransmitters. One of the main neurotransmitters affected by cocaine is dopamine. It stimulates the neurons to release dopamine in the limbic system, the part of the brain that controls feelings of pleasure. When dopamine has been released it attaches itself to the corresponding nerve cells receptor stimulating a pleasurable response. It is then normally taken back to the neuron that released it. Cocaine blocks this re-uptake causing dopamine to continue stimulating the receptor, which in turn leads to a higher, more pronounced feeling of pleasure. In the long term this depletes dopamine, causing changes in brain function such as depression and mood swings.	**Effects on the Brain:** The limbic system, brainstem and spinal cord have nerve cells that respond to endorphins. The brain naturally releases endorphins when the body is undergoing pain or stress. Large amounts of endorphins flood the space between nerve cells, inhibiting the neurons from firing and thus creating an analgesic effect. They can also stimulate the neurons leading to a feeling of euphoria. Heroin contains a metabolised version of morphine that mimics endorphins and binds onto endorphin-receptor sites. Because morphine is more powerful than natural endorphins the brain has no control over release, so it builds dependence. When heroin is taken away a chemical imbalance is created causing the feelings of withdrawal.
Nervous System: Cocaine works with the sympathetic part of the nervous system, which is concerned with outside stimulus such as danger and anticipation. The 'Fight or Flight' response is part of this and releases adrenaline into the body.	**Nervous System:** Heroin works with the parasympathetic part of the nervous system. This is responsible for the opposite effect of the sympathetic nervous system and produces a 'Rest and Digest' response in the mind and body.

Cardiovascular System: Cocaine increases the heart rate through the release of adrenaline and at the same time releases a chemical called endothelin which reduces the size of blood vessels. **Respiratory System:** Cocaine stimulates the respiratory system through the release of adrenaline, especially when the user is craving or experiencing a bad come-down. **Addiction:** Physical addiction to cocaine is debatable. Cravings are triggered because of thoughts of using rather than a physical need for the drug.	**Cardiovascular System:** As well as depressing the activity of the nervous system, heroin also depresses the cardiovascular system. Heart rate lowers and the blood vessels are widened giving the feeling of warmth. **Respiratory System:** When heroin is used the respiratory system is depressed, slowing down breathing. Rather than 'Fight or Flight' it is 'Rest and Digest'. **Addiction:** Heroin causes a physical addiction because the brain adapts itself to accommodate the regular use of this chemical. Cravings are often associated with periods of physical withdrawal.

Updated Neuroscience (2016–2025)

The Koob and Volkow model of addiction (Lancet Psychiatry, 2016) describes three stages of the addiction cycle – binge/intoxication, withdrawal/negative affect, and preoccupation/anticipation – each mediated by distinct brain circuits. Cocaine addiction involves dysregulation of the reward system (ventral striatum), the extended amygdala (stress and negative emotions), and the prefrontal cortex (executive function and decision-making).

The role of glutamate has become increasingly recognised. Chronic cocaine use disrupts glutamate homeostasis in the nucleus accumbens, particularly through downregulation of the cystine-glutamate exchanger on glial cells. This possibility contributes to compulsive drug-seeking behaviour and vulnerability to relapse.

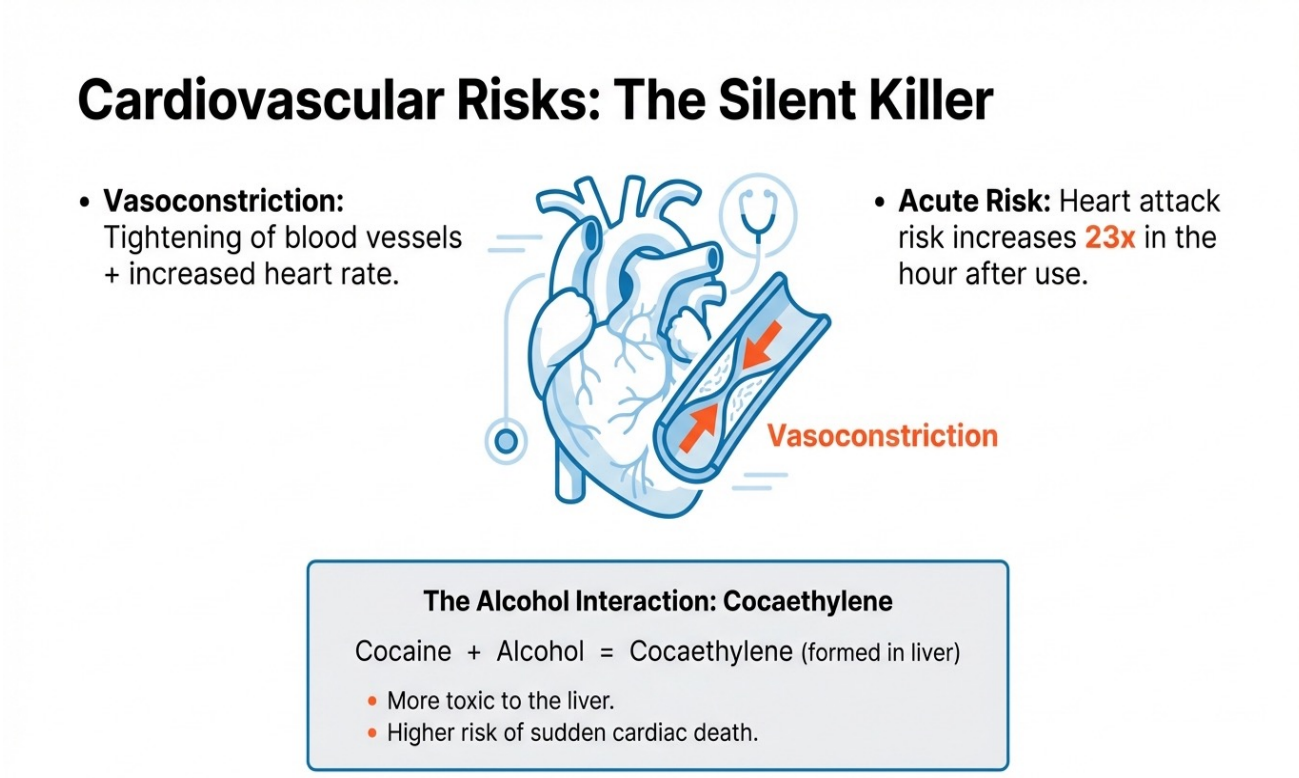
The orbitofrontal cortex (OFC) shows significant dysfunction in cocaine users, contributing to impaired decision-making and impulsivity. fMRI and PET scan studies have demonstrated that cue-induced craving activates the amygdala, anterior cingulate cortex, and dorsolateral prefrontal cortex.

Diagnostic Framework

Under the DSM-5-TR (2022), cocaine addiction is classified as Stimulant Use Disorder, graded as mild, moderate, or severe based on the number of criteria met. The ICD-11 (WHO, 2022) uses the term 'Disorders due to use of cocaine' with distinct categories for harmful use and dependence. Importantly, the ICD-11 now formally recognises a cocaine withdrawal syndrome, with symptoms including dysphoria, fatigue, increased appetite, psychomotor retardation or agitation, vivid unpleasant dreams, and sleep disturbance.

6. Health Implications

Crack and cocaine can damage health in many ways, and in some instances, these can be fatal. Some of these risks can be increased by the way that the drug is used and also by the route of use. The bottom line is that there is no safe way to use it.



Heart failure can happen to anyone taking crack or cocaine, regardless of how much they are taking or how long they have been using. People who already have heart disease or heart defects are at an even greater risk if they use the drug. Some American studies have shown that around 25% of all heart attacks in people between the ages of 18–45 are linked to frequent cocaine use. When taking crack or cocaine, the risk of a potentially fatal heart attack increases by around 23 times in the hour after use, especially if alcohol has been used in conjunction.

The increased risk of heart attack can come from a number of factors, including:

- Increased adrenaline (released because of cocaine use)
- High blood pressure (increased heart rate caused by adrenaline)
- Constricted blood vessels (cocaine releases endothelin, which constricts blood vessels)
- Hardening of the arteries (caused by cocaine use)
- Weakened heart (congestive heart failure)
- Arrhythmias (erratic heartbeat)
- Ashen grey skin (poorly oxygenated blood)
- Current heart problems (made worse by cocaine)

- Other drugs used in conjunction with cocaine, such as Viagra and alcohol, can increase stress upon the heart)

Sodium bicarbonate (used to 'wash' cocaine to turn it into crack) may also exert further stress upon the heart. And cocaethylene, a chemical produced in the liver when using crack or cocaine and alcohol together, also exerts more pressure on the cardiovascular system than if cocaine were taken alone.

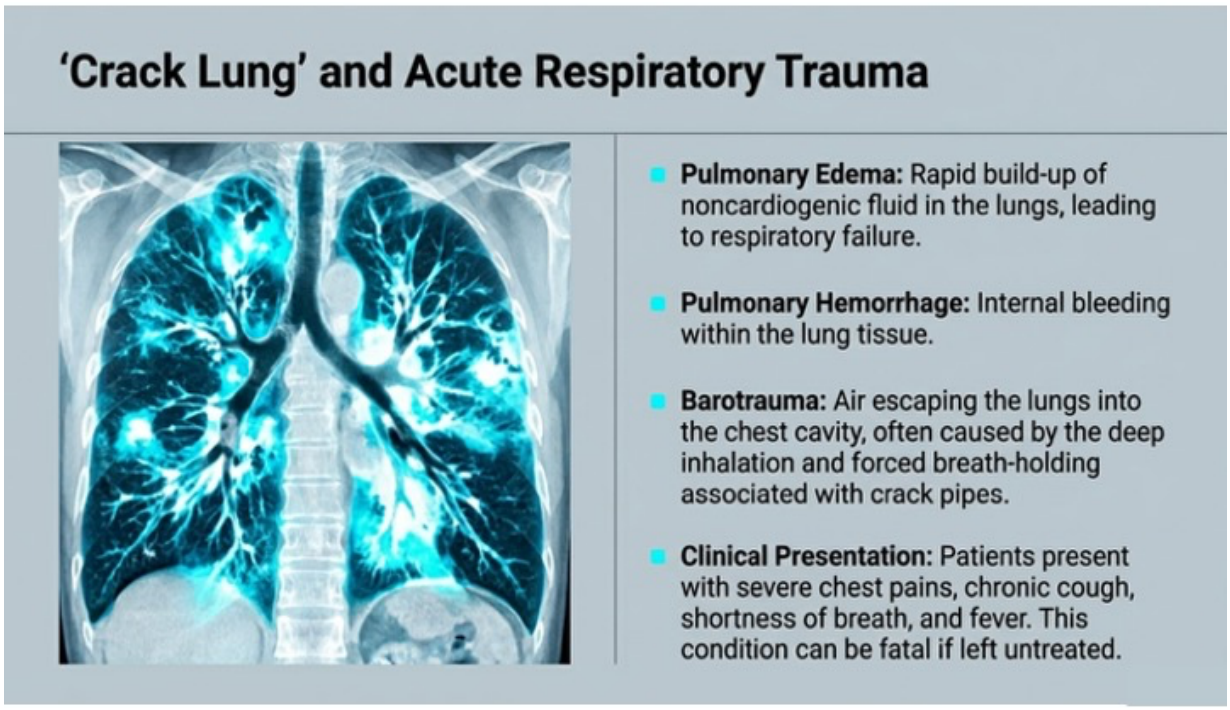
UPDATED CARDIOVASCULAR EVIDENCE

A 2020 systematic review (Nature Scientific Reports) found that chronic cocaine use is associated with concentric ventricular hypertrophy, decreased left ventricular end-diastolic dimension, and increased wall thickness – changes consistent with diastolic dysfunction. A 2025 Yale forensic pathology study found that nearly 90% of cocaine-related deaths had pre-existing atherosclerotic or hypertensive heart disease, compared to only 40% in fentanyl deaths.

Strokes and Seizures

Strokes are thought to be caused by the constriction of blood vessels and the repeated increase in blood pressure. These combined factors can sometimes cut off blood supply to parts of the brain and, in some cases, cause delicate blood vessels to rupture (causing bleeding in the brain). Blackouts and seizures may also be caused by the above, coupled with high body temperatures.

Respiratory System



Taking crack or cocaine can cause many lung problems. These problems are not just isolated to smoking crack, as injecting crack or cocaine can also cause lung problems. Some of the problems associated with the use of crack or cocaine include:

- Pulmonary oedema – build-up of fluid in the lungs
- Pulmonary haemorrhage – bleeding in the lungs
- Pulmonary barotrauma – air escaping lungs (by holding in crack smoke)
- Foreign bodies in lungs – poor pipes, no gauzes used
- 'Crack Lung' – cough, shortness of breath, fever, inflamed lungs

Crack use can affect the cilia (small hairs) that line the main tubes of the lungs. These help to clean the lungs and prevent infections, which in turn leads to crack and cocaine users being more susceptible to bronchitis, pneumonia, pleurisy etc.

Tuberculosis may be a risk factor for crack and cocaine users. Evidence suggests increased chances of contracting TB, probably due to impaired immune systems, long spells within enclosed environments (crack houses etc), poor diet and reluctance to present for medical interventions. The symptoms of TB are similar to those of someone heavily using crack or cocaine so may not be identified. The only certain way of diagnosis is through a chest x-ray or skin test.

Damage to the lungs may also be caused by deep inhaling ammonia (freebase rocks).

Long-term crack smoking is associated with accelerated decline in lung function and increased risk of chronic obstructive pulmonary disease (COPD). OHID drug-related harms data confirms high rates of respiratory illness among people who smoke crack.

Liver Damage

If alcohol is used in conjunction with cocaine then the stress upon the liver will become increased as a liver-toxic substance called cocaethylene is produced. If users are Hepatitis C positive then the stress exerted upon the liver could have more serious consequences.

Immune System

Crack and cocaine impair the immune system by damaging CD4 T Cells (they do not work as effectively as they should). This cell helps fight off infections throughout the body. Prolonged use can lead to a depletion in vitamins (particularly C and E), minerals and amino acids (the building blocks for neurotransmitters). Poor diet and unhealthy lifestyle can also contribute to a poor immune system. This should recover once the person has stopped using crack or cocaine.

Mental Health

Some diagnosed psychiatric disorders can appear to improve with the use of crack or cocaine; this does not mean that the issue has gone away, as when the use of crack or cocaine stops these conditions may reappear. It is therefore vitally important that if there

has been a psychiatric diagnosis made in the past that the individual is receiving the appropriate support from mental health professionals. Psychiatric illnesses that may be complicated by the use of crack or cocaine:

- Attention Deficit Hyperactivity Disorder (cocaine may act as self-medication)
- Paranoia and anxiety disorders (cocaine can make these worse)
- Bipolar disorder (manic depression)
- Schizophrenia (dopamine theory may indicate possible medication action)
- Depression and suicidal thoughts
- Visual and auditory hallucinations
- Compulsive and eating disorders
- Crack/cocaine-induced psychosis

UPDATED MENTAL HEALTH EVIDENCE

Cocaine-induced psychosis affects an estimated 5–10% of regular cocaine users. Updated ONS/OHID data shows cocaine is increasingly implicated in suicide deaths. NICE Clinical Guideline CG178 (Psychosis and schizophrenia) should be referenced when managing cocaine-induced psychotic episodes. ADHD self-medication with cocaine is well-documented; evidence supports consideration of stimulant prescribing (such as lisdexamfetamine/Vyvanse) as a harm reduction strategy for people with confirmed ADHD and co-occurring cocaine use disorder.

Pregnancy

Crack or cocaine use is definitely not advisable during pregnancy as taking any substance during this time could have an adverse effect. Many of the studies regarding issues such as 'crack baby syndrome' have now been shown to be overblown and more to do with public and professional reactions to crack being used during pregnancy than factual evidence.

However, crack and cocaine use during pregnancy may cause:

- Miscarriage (high blood pressure)
- Low birth weight (under-nourishment)
- Premature birth
- Disturbed behaviour in new-born babies (possibly high adrenaline levels)

Cocaine can be passed on to the child through breast milk, so it is advisable that if clients continue to use after the birth of their child they bottle-feed.

It is vitally important that if someone has used when they are pregnant they receive proper medical attention and look after themselves during the course of the pregnancy. Avoiding proper medical care, not eating properly, smoking cigarettes and drinking alcohol can all have a major effect upon the health of the baby during pregnancy.

UPDATED PREGNANCY EVIDENCE

Post-2010 longitudinal research has further debunked 'crack baby syndrome'. Studies including the Maternal Lifestyle Study (NIDA) show that prenatal cocaine exposure effects are largely attributable to confounding factors such as poverty, poor nutrition, tobacco and alcohol use, and inadequate antenatal care. Current NICE antenatal care guidelines emphasise non-judgemental support and safeguarding frameworks.

Blood-Borne Viruses (BBVs)

The issue of BBVs in connection with crack and cocaine use has, to a large extent, been ignored unless the route of use is injecting. There is a need to challenge this and to disseminate information to users at risk.

HIV: HIV can be spread by the sharing of injecting equipment (as with heroin use) and also by the practice of unsafe sex. Some crack and cocaine users may have multiple partners. The main transmission route for HIV amongst crack and cocaine users is either through sharing contaminated needles or risky sexual behaviour. There is a tendency generally for risk-taking behaviour to increase when taking cocaine, which in itself could increase the likelihood of the above transmission routes. Research from the University of California has discovered that cocaine not only influences risk-taking behaviour and consequent possible transmission, but it also affects the AIDS viral load in the blood. Cocaine can double the amount of HIV infected cells and can deplete the number of CD4 T-Cells by up to nine times.

HCV: The dangers of contracting Hepatitis C are not confined to intravenous drug use. If Hepatitis C positive, cocaine use itself can exert strain upon the liver, and if alcohol is also used, the immune system can be impaired.

Injecting: Cocaine use can increase risk-taking behaviour, and anecdotal information suggests that injecting users of cocaine who are fully aware of safer injecting behaviour can ignore this when caught up in the chaos and compulsion of using.

Smoking: The use of crack can seriously dehydrate the body, leading to the lips becoming chapped. These can often be picked, producing open wounds, and a virus may be transmitted by pipe sharing. Some pipes can also cut the mouth when smoking, again increasing the risk.

Snorting: When cocaine is snorted on a regular basis, damage to the nasal mucus membranes can occur, causing the nose to bleed. The practice of sharing straws to 'snort' is quite common, potentially leading to blood-to-blood transmission via the straw.

UPDATED BBV EVIDENCE

UKHSA surveillance data shows ongoing HIV and Hepatitis C transmission risk among people who use crack, particularly through shared injecting equipment and sexual transmission. The NIHR-funded SIPP (Safe Inhalation Pipe Provision) study, launched in 2022, is the first UK evaluation of crack pipe distribution as a harm reduction intervention, operating with police approval in Bristol, Nottingham, and Mansfield. Current UK law prohibits crack pipe supply under Section 9A of the Misuse of Drugs Act 1971, but reform is being advocated. Directly acting antiviral (DAA) treatment for Hepatitis C is now available and highly effective (cure rates >95%), and drug treatment services should actively facilitate HCV testing and treatment referral.

Other Health Issues

- Stomach pains and digestive disorders
- Weight loss (usually happens with people using on a daily basis) can become more complicated if combined with an eating disorder.
- Kidney damage
- Skin problems (poor diet, depletion in vitamins, burns from smoking etc)
- Hyperthermia (increased body temperature)
- Can exacerbate asthma and increase attacks
- Complications with epilepsy and sickle cell disease (increased attacks)

Dangers of Use

There are many dangers associated with the use of crack and cocaine, and to understand them better, we need to look at how the drug is administered.

Severe Complications:

1. Strokes, including subarachnoid and intracerebral haemorrhage and cerebral infarcts.
2. Numerous cardiovascular complications, for example, myocardial infarction, ventricular arrhythmias and cardiac arrest.
3. Intestinal ischaemia.

Chronic Effects:

General – Anorexia, weight loss, malnutrition, dehydration, tremor, psychosis, severe depression and isolated convulsions. Anaesthetic effects on nerve endings can increase the risk of physical harm and numbness of the mucous membranes.

Smoking – Haemoptysis, black sputum, chest pains, lung damage, respiratory failure (freebase/ammonia).

Snorting – Loss of smell, increased susceptibility to upper respiratory tract infections, sinusitis, erosions and nasal perforations.

Injecting – Local abscess and the usual infectious conditions associated with intravenous drug use. Danger also occurs from the injecting site becoming anaesthetised, veins collapsing and increased risk-taking behaviour. There is also an increased risk of septicaemia due to an impaired immune system.

Ingestion – In some cases, clients may ingest large amounts of cocaine. This can be intentional due to chasing the high or due to paranoia. Large amounts of cocaine in the system can lead to overdose. If you have a client that you know or fear has ingested a large amount of cocaine, they need to be taken to accident and emergency services as quickly as possible (within 1 hour of ingesting). Phenothiazines and Haloperidol should be avoided as they can lower the threshold for convulsions.

ACUTE BEHAVIOURAL DISTURBANCE (FORMERLY 'EXCITED DELIRIUM')

The term 'excited delirium' has been retired as a medical diagnosis. The American Medical Association adopted a policy opposing its use in 2021, the American College of Emergency Physicians formally rejected it in 2023, and the American Psychiatric Association has stated it is "too non-specific to meaningfully describe a person". These organisations have highlighted concerns about the term's disproportionate application in cases involving Black individuals in police custody. The Royal College of Emergency Medicine (RCEM) now recommends the term 'acute behavioural disturbance' or 'hyperactive delirium with severe agitation'. Practitioners should be aware that stimulant toxicity can present with agitation, hyperthermia, tachycardia, and bizarre behaviour, and requires immediate emergency medical intervention rather than restraint alone.

7. Medications and Therapeutic Interventions

Conventional prescribed medication has not had much success in working with this client group. Other failures have been due to inappropriate prescribing, such as methadone (there is no need to prescribe a physically addictive drug for problems with a non-physically addictive one), unless they are also using opiates alongside.

Promazine

Promazine remains occasionally used in some services but is **rarely recommended as first-line treatment**. In paediatric doses, promazine appears to have some beneficial effects when people are first coming off crack or cocaine: although it is an anti-psychotic medication, the small doses mean that clients are not heavily sedated, but sufficient to take the edge off craving and work with increased adrenaline levels. Any prescribing should follow local clinical governance protocols, and prescribers should be aware of the limited evidence base.

CONTINGENCY MANAGEMENT – NICE-RECOMMENDED INTERVENTION

Contingency Management (CM) is recommended by NICE for crack and cocaine use disorder (NICE NG58, Drug misuse in over 16s: psychosocial interventions, 2007, reviewed 2016). CM uses positive reinforcement (typically vouchers or small financial incentives) to reward drug-free urine samples or treatment attendance. The Department of Health and Social Care rolled out an Incentivised Substance Free Living scheme in 2023–24 as part of the From Harm to Hope strategy. A 2021 JAMA Network Open meta-analysis confirmed CM as the most effective treatment for active cocaine use. Despite NICE recommendations, UK implementation remains limited due to resource constraints, ethical concerns, and cultural barriers.

Pharmacological Evidence (2015–2025)

Medication	Evidence Summary	Status
Naltrexone	Emerging evidence for cocaine use disorder, particularly in combination with disulfiram for people with co-occurring alcohol dependence.	*Experimental*
Modafinil	Meta-analysis of 11 RCTs shows no overall superiority over placebo, but may benefit subgroups without alcohol use disorder.	*Experimental*
Bupropion	Some evidence when combined with contingency management; may enhance CM effects.	*Experimental*
Disulfiram (Antabuse)	Mixed results; may reduce cocaine use in people not dependent on alcohol. Originally identified in Yale studies combining disulfiram with buprenorphine.	*Under investigation*
Topiramate	Meta-analysis of 5 RCTs shows increased abstinence (low strength evidence); combination with mixed amphetamine salts shows promise.	*Experimental*
N-Acetylcysteine (NAC)	2024 meta-analysis shows reduction in craving ratings. Works by modulating glutamate homeostasis. Most effective in already-abstinent patients.	*Promising adjunct*
Lisdexamfetamine (Vyvanse)	Licensed for ADHD; may indirectly reduce cocaine use in people with confirmed ADHD-cocaine comorbidity.	*Licensed for ADHD only*

Tirzepatide & Cocaine Use

A study published in April 2026 looked at whether tirzepatide could affect cocaine use. Researchers found that it reduced the brain's dopamine spike triggered by cocaine, and also made rats less likely to take cocaine, less driven to seek it out, and less motivated to use it. The scientists concluded that because tirzepatide works on two brain receptors at once (GLP-1 and GIP), it's worth exploring as a possible treatment for cocaine addiction in humans.

Psychosocial Interventions

Cognitive Behavioural Therapy (CBT): NICE-endorsed for cocaine use disorder. Focuses on identifying triggers, developing coping strategies, and relapse prevention.

Motivational Interviewing (MI): Evidence-based approach for enhancing motivation to change. Particularly useful for people ambivalent about treatment engagement.

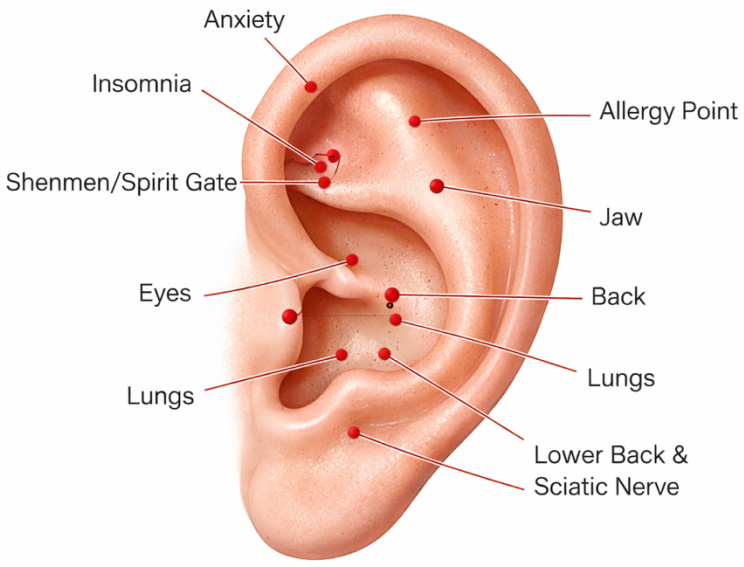
Community Reinforcement Approach (CRA): A comprehensive behavioural programme that rearranges vocational, social, and recreational reinforcers to support recovery.

Mindfulness-Based Relapse Prevention (MBRP): Growing evidence base. A 2022 RCT combining ketamine infusion with MBRP showed reduced cocaine cravings and relapse risk.

Exercise Interventions: Emerging evidence supports structured exercise programmes as adjunct treatment, with aerobic exercise shown to reduce craving and improve mood regulation.

Alternative Therapies

At present, alternative therapies offer a useful complementary approach for agencies working with this client group. They are relatively low-cost, often accessible, and can work by reducing adrenaline levels (calming the client down), thereby reducing craving levels. This can help build up drug-free time, which, in turn, will help brain chemistry return to normal.



Acupuncture Points

Acupuncture: Five-point detox acupuncture can calm a client suffering from cravings very quickly and has an accumulative effect. Problems arise because it can only be given within the agency and usually during specified times. A proportion of clients may have needle phobias. Full-body acupuncture is also effective.

Essential Oils: These can be used in the agency and also by the client. Lavender and Geranium are good all-around oils and are fairly inexpensive. Camomile, Frankincense and Ylang Ylang also work well. They can also be placed in the bath and used with a massage.

Massage: All types of massage are effective in helping clients become calmer and also serve as an incentive to bring clients into the project.

Homoeopathy: This seems to work well with symptoms of stress, anxiety and emotional problems. Always inform the homoeopath of any other treatments the client may be receiving, as these can affect the treatment.

Echinacea Drops: Helps boost the body's natural immune system. Can be used in the early stages of recovery to support an impaired immune system.

The authors would like to underline that before any medication is prescribed, expert advice should be sought on the exact doses and a full medical history obtained. As complementary therapies are very often used with crack and cocaine users, this needs to be included in the medical history and cross-referenced to see if there are any negative effects with the proposed course of medication.

8. Appendices: Herbal Tea Recipes

Sleep, Detox and Lung teas are probably the most useful complementary supports. They can be given to clients to use at home and in the evenings, when cravings are usually strongest. Make sure that the client knows how to make them properly, as some people have been known to make the teas very strong in the belief that this will work better. Lung Tea should not be given to clients who have liver problems, so if in doubt, do not give it.

CLINICAL CAVEAT

These herbal tea preparations are supplementary, non-pharmacological supports and should never replace clinical treatment. They may help manage some symptoms of withdrawal and craving but are not evidence-based treatments for cocaine use disorder. Practitioners should advise clients to inform medical staff about any herbal remedies being used.

DRUG INTERACTION WARNING: ST JOHN'S WORT

St John's Wort (Hypericum perforatum) has significant drug interactions, particularly with antiretroviral medications used in HIV treatment, hormonal contraceptives, and some psychiatric medications. Clients who are HIV-positive or taking prescribed medication should be advised to check the NHS Medicines A–Z or BNF (British National Formulary) before using herbal preparations containing St John's Wort.

Sleep Tea

Herb (1 part each, i.e. 1 oz)	Latin Name	Use
CHAMOMILE	*Matricaria recutita*	Anti-inflammatory, antispasmodic, mild bitter, sedative
SKULLCAP	*Scutellaria laterifolia*	Nerve tonic, anxiolytic, sedative, mild bitter, diaphoretic
MOTHERWORT	*Leonurus cardiaca*	Cardiac tonic, sedative
DAMIANA	*Turnera diffusa*	Nerve tonic, stimulant, anxiolytic, antidepressant
PASSIFLORA	*Passiflora incarnata*	Sedative, antispasmodic, hypotensive

RED CLOVER BLOSSOM	*Trifolium pratense*	Oestrogenic, expectorant, skin depurative
VERVAIN	*Verbena officinalis*	Nerve tonic, sedative, antispasmodic, cholagogue

Detox Tea

Herb	Latin Name	Use
CHAMOMILE (2 parts)	*Matricaria recutita*	Anti-inflammatory, antispasmodic, mild bitter, sedative
SKULLCAP (1 part)	*Scutellaria laterifolia*	Nerve tonic, anxiolytic, sedative, mild bitter, diaphoretic
CATMINT (1 part)	*Nepeta cataria*	Diaphoretic, antispasmodic, sedative
PEPPERMINT (1 part)	*Mentha piperata*	Expectorant, antiviral, carminative, antispasmodic
YARROW (1 part)	*Achillea millefolium*	Circulatory, diaphoretic, anti-inflammatory, antispasmodic
ELDER (1 part)	*Sambucus nigra*	Anti-inflammatory, diuretic, vascular, diaphoretic, astringent, anti-catarrh
HOPS ¼ part (omit if feeling depressed)	*Humulus lupulus*	Sedative, bitter digestive, diuretic

Lung Tea

Herb (1 part each)	Latin Name	Use
HYSSOP	*Hyssopus officinalis*	Antispasmodic, sedative, anticatarrh

COMFREY	*Symphytum officinalis*	Demulcent, tissue healer, expectorant
MARSHMALLOW	*Althaea officinalis*	Demulcent, expectorant
PEPPERMINT	*Mentha piperata*	Expectorant, antiviral
WILD CHERRY BARK	*Prunus serotina*	Anti-tussive, astringent, sedative
COLTSFOOT	*Tussilago farfara*	Expectorant, demulcent, antispasmodic
YARROW	*Achillea millefolium*	Circulatory, diaphoretic, antispasmodic
ST JOHN'S WORT (only if infection)	*Hypericum perforatum*	Nervous system tonic, vulnerary, antidepressant, antiviral, antibacterial

Glossary of Herbal Terms

Term	Meaning
Vulnerary	Healing of wounds
Anxiolytic	Relieving anxiety
Depurative	Purifying, cleansing
Diaphoretic	Promotes sweating (cleansing)
Carminative	Eases gripe pain – expels flatulence
Cholagogue	Stimulates bile flow
Bitter	Digestive stimulant
Demulcent	Soothes mucus membranes
Antitussive	Anti-cough reflex
Expectorant	Expels catarrh and mucous

Nephritic	Soothes kidneys
Antilithic	Reduces formation of calcii stones

Always advise clients to seek appropriate medical attention and always inform medical staff of any complementary therapies used.

9. References

Policy and Epidemiology

HM Government. (2022). From harm to hope: A 10-year drugs plan to cut crime and save lives. https://www.gov.uk/government/publications/from-harm-to-hope-a-10-year-drugs-plan-to-cut-crime-and-save-lives

Office for Health Improvement and Disparities. (2025). Opiate and crack cocaine use: Prevalence estimates 2022 to 2023. OHID.

Office for National Statistics. (2025). Deaths related to drug poisoning in England and Wales: 2024 registrations. ONS. https://www.ons.gov.uk/peoplepopulationandcommunity/birthsdeathsandmarriages/deaths/bulletins/deathsrelatedtodrugpoisoninginenglandandwales/2024registrations

Office for Health Improvement and Disparities. (2025). Adult substance misuse treatment statistics 2024 to 2025. OHID.

National Crime Agency. (2024). National strategic assessment of serious and organised crime. NCA.

Race Equality Foundation. (2021). Improving drug treatment services for Black and minoritised communities. London: Race Equality Foundation.

Pharmacology and Neuroscience

Koob, G. F., & Volkow, N. D. (2016). Neurobiology of addiction: A neurocircuitry analysis. Lancet Psychiatry, 3(8), 760–773.

Koob, G. F. (2018). Addiction and stress: An allostatic view. Neuroscience & Biobehavioural Reviews.

American Psychiatric Association. (2022). Diagnostic and statistical manual of mental disorders (5th ed., text rev.). APA.

World Health Organisation. (2022). International classification of diseases (11th rev.). WHO.

Garris, P. A., Wightman, M., & Kenan, W. R. (1999). Dopamine not brain pleasure source. University of North Carolina – Chapel Hill.

Volkow, N.D., Mullani, N., Gould, K.L., Adler, S., & Krajewski, K. (1988). Cerebral blood flow in chronic cocaine users: A study with positron emission tomography. British Journal of Psychiatry, 152, 641–648.

Volkow, N.D., Fowler, J.S., Wolf, A.P., Hitzemann, R., Dewey, S., Bendriem, B., Alpert, R., & Hoff, A. (1991). Changes in brain glucose metabolism in cocaine dependence and withdrawal. American Journal of Psychiatry.

Childress, A. R., et al. (1999). Limbic activation during cue-induced cocaine craving. American Journal of Psychiatry.

Garavan, H., et al. (2000). Cue-induced cocaine craving: Neuroanatomical specificity for drug users and drug stimuli. American Journal of Psychiatry.

Crossman, A. R., & Neary, D. (2000). Neuroanatomy. Churchill Livingstone.

Bolla, K. I. (1999). How cocaine use hurts brain function. Journal of Neuropsychiatry and Clinical Neuroscience.

Treatment

National Institute for Health and Care Excellence. (2007, reviewed 2016). Drug misuse in over 16s: Psychosocial interventions (NG58). NICE.

Ronsley, C., et al. (2020). Pharmacotherapeutic strategies for treating cocaine use disorder. Addiction, 115(10).

Nocito Echevarria, M. A., et al. (2017). N-acetylcysteine for treating cocaine addiction: A systematic review. Psychiatry Research.

Gaval-Cruz, M., & Bhatt, D. (2024). Effect of N-acetylcysteine on craving in substance use disorders. Frontiers in Pharmacology.

Carroll, K. M. (2001). A cognitive behavioural approach: Treating cocaine addiction. NIDA.

Otto, C., et al. (1998). Auricular acupuncture as an adjunctive treatment for cocaine addiction. American Journal on Addictions, 7(2), 164–170.

Donmall, M., Seivewright, N., Douglas, J., Draycott, T., & Millar, T. (1995). National cocaine treatment study: Report to the task force. Manchester University.

Health

Mega, J. L., et al. (2020). Cocaine, cardiomyopathy, and heart failure: A systematic review and meta-analysis. Scientific Reports, 10.

Diagnostics. (2025). Cocaine-induced cardiac alterations: Histological and immunohistochemical analysis.

Mittleman, M. A., et al. (1999). Triggering of myocardial infarction by cocaine. Circulation, June 1999.

Wunsch, H. (1999). Cocaine use transiently increases heart-attack risk. The Lancet, June 1999.

Qureshi, A. I., et al. (2001). Cocaine use and the likelihood of nonfatal myocardial infarction and stroke. Circulation.

Brody, S. L., Slovis, C. M., & Wrenn, K. D. (1990). Cocaine-related medical problems. American Journal of Medicine.

Brookoff, D., et al. (1996). Cocaethylene levels in patients who test positive for cocaine. Annals of Emergency Medicine.

Khalsa, M. E., Tashkin, D. P., & Perrochet, B. (1992). Smoked cocaine: Patterns of use and pulmonary consequences. Journal of Psychoactive Drugs.

Baldwin, G. (2002). Cocaine dramatically accelerates HIV infection. The Journal of Infectious Diseases.

Harris, M., et al. (2024). Safe inhalation pipe provision (SIPP): Protocol for a mixed-method evaluation. Harm Reduction Journal.

American Medical Association. (2021). New AMA policy opposes 'excited delirium' diagnosis. AMA.

Coffin, P. (1996). Cocaine and pregnancy. New York: Lindesmith Center.

Chavkin, W. (2001). Cocaine and pregnancy: Time to look at the evidence. JAMA.

Thurman, S. K., Brobeil, R. A., Duccette, J. P., & Hurt, H. (1994). Prenatally exposed to cocaine: Does the label matter? Journal of Early Intervention.

Landry, M. J. (1992). An overview of cocaethylene, an alcohol-derived, psychoactive, cocaine metabolite. Journal of Psychoactive Drugs.

Historical and Policy Context

Karch, S. B. A brief history of cocaine. CRC Press.

Streatfeild, D. (2001). Cocaine: An unauthorised biography. Virgin Publishing.

Bernard, C. (1998). The Incas: Empire of blood and gold. Thames and Hudson.

Misuse of Drugs Act 1971. Her Majesty's Stationery Office.

Inciardi, J. A., & Harrison, L. D. (1998). Heroin in the age of crack cocaine. Sage.

Gray, A., & Taegtmeyer, D. (2002). Crack and cocaine: Good practice guidelines for drug treatment and testing orders. Commissioned by London Probation Service.

Gray, A. (2001). Freebasing cocaine. COCA Update, August 2001.

National Treatment Agency. (2002). Commissioning cocaine/crack treatment. NTA.

National Treatment Agency. (2002). Models of care: For the treatment of drug users. NTA.

Haynes, G., Bottomley, T., & Gray, A. (2000). National crack cocaine treatment and response strategy. Unpublished.

May, T., Edmonds, M., Hough, M., & Harvey, C. (1999). Street business: The links between sex and drug markets. Home Office.

Sangster, D., Shiner, M., Patel, K., & Sheikh, N. (2002). Delivery of drug services to black and minority ethnic groups. London: DPAS.

Wise, R. A., Bauco, P., Carlezon, W. A., Jr., & Trojniar, W. (1992). Self-administration and drug reward mechanisms. Annals of the New York Academy of Sciences.

Jaskiw, G. E., & Weinberger, D. R. (1992). Dopamine and schizophrenia. Seminars in the Neurosciences.