Crack & Heroin

Day One

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1. Introduction

The pack will aid service providers to build competencies that will help to develop crack and heroin services or orientate generic services to becoming more successful in working with this client group.

2. Cocaine and Heroin Timeline

‘Coca’ comes from the Tiwakanu word ‘Khoka’ meaning ‘The Tree’, the Sumerians refer to opium as Hul Gil, the 'joy plant.'

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.
3400 BC	The opium poppy is cultivated in lower Mesopotamia. The Sumerians would soon pass along the plant and its euphoric effects to the Assyrians. The art of poppy-culling would continue from the Assyrians to the Babylonians who in turn would pass their knowledge onto the Egyptians.
3000 BC:	Coca chewing is practiced 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.
1300 BC	In the capital city of Thebes, Egyptians begin cultivation of opium thebaicum, grown in their famous poppy fields. The opium trade flourishes during the reign of Thutmose IV, Akhenaton and King Tutankhamen. The trade route included the Phoenicians and Minoans who move the profitable item across the Mediterranean Sea into Greece, Carthage, and Europe.
1100 BC	On the island of Cyprus, the "Peoples of the Sea" craft surgical-quality culling knives to harvest opium, which they would cultivate, trade and smoke before the fall of Troy.
460 BC	Hippocrates, "the father of medicine", dismisses the magical attributes of opium but acknowledges its usefulness as a narcotic and styptic in treating internal diseases, diseases of women and epidemics.
330 BC	Alexander the Great introduces opium to the people of Persia and India.
300 BC	Opium is used by Arabs, Greeks, and the Romans as a sedative and soporific.
400 AD	Opium from the Egyptian fields at Thebes, is first introduced to China by Arab traders.
1000	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).
1300’s	Opium disappears for two hundred years from European historical record. Opium had become a taboo subject for those in circles of learning during the Holy Inquisition. In the eyes of the Inquisition, anything from the East was linked to the Devil.
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 brings about the fall of the Incan civilisation. The estimated population of the Incan empire 14,000,000

1500's The Portuguese, whilst trading along the East China Sea, take on the smoking of opium. The effects were instantaneous, as they discovered, but it was a practice the Chinese considered barbaric and subversive.

1505. The first reports of coca use reach Europe.

Early 1500's First commercial production of coca by Europeans as holders of Spanish land grants are allowed to make their tax payments in coca leaves.

1527 During the height of the Reformation, opium is reintroduced into European medical literature by Paracelsus as laudanum. These black pills or "Stones of Immortality" were made of opium thebaicum, citrus juice and quintessence of gold and prescribed as painkillers.

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 1500's 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.

1600's Coca first brought to Western culture. Some Europeans chewing coca leaf for stimulation. Residents of Persia and India begin eating and drinking opium mixtures for recreational use. Portuguese merchants carrying cargoes of Indian opium through Macao direct its trade flow into China.

1606 Ships chartered by Elizabeth I are instructed to purchase the finest Indian opium and transport it back to England.

1662. Abraham Cowley writes a poem titled ‘A legend of Coca’. This is the first independent mention of coca in English literature.

1680 English apothecary, Thomas Sydenham, introduces Sydenham's Laudanum, a compound of opium, sherry wine and herbs. His pills along with others of the time become popular remedies for numerous ailments.

1689 Use of tobacco-opium mixtures (madak) begins in the East Indies (probably Java) spreads to Formosa, Fukien and the South China coast.

1700’s The Dutch export shipments of Indian opium to China and the islands of Southeast Asia; the Dutch introduce the practice of smoking opium in a tobacco pipe to the Chinese. Use of hashish, alcohol, and opium spreads among the population of occupied Constantinople

1708. German physician and botanist Herman Boerhaave first mentions coca with regards to a use as medicine.

1729 Chinese emperor, Yung Cheng, issues an edict prohibiting the smoking of opium and its domestic sale, except under license for use as medicine.

1750 The British East India Company assumes control of Bengal and Bihar, opium-growing districts of India. British shipping dominates the opium trade out of Calcutta to China.

1753 Linnaeus, the father of botany, first classifies the poppy, Papaver somniferum - 'sleep-inducing', in his book Genera Plantarum.

1767 Opium from Bengal continues to enter China despite the edict of 1729 prohibiting smoking. The British East India Company's import of opium to China increases in frequency from 200 chests annually in 1729 to a staggering two thousand chests of opium per year. Tariffs are collected on the opium.

1772 The East India Company establishes a limited monopoly over Bengal opium; the company has general control but the operation is in the hands of contractors, who advance company funds to the farmers, purchase the opium produced, and sell it to the company which then auctions it off to merchants in Calcutta. British companies are the principal shippers.

1779 First mention of actual opium trading in opium at Canton, China.

1780 British traders establish an opium depot at Macao. Another Imperial edict prohibits consumption of opium and reiterates prohibition of its sale.

1787 Trade in opium is still less important than trade in commodities; directors of the East India Company, recognizing China's objections to the importation of opium, make offers to prohibit the export of Indian opium to China. However, company representatives in Canton declare that the Chinese are never sincere in their declared intentions of suppressing illicit traffic, as long as the officials issue prohibitory edicts with one hand and extend the other to receive bribes from the illegal trade.

1793 The British East India Company establishes a total monopoly on the opium trade. All poppy growers in India were forbidden to sell opium to competitor trading companies.

1799 The 1799 edict increases traffic through Macao and other areas beyond government control enabling unprecedented growth. The British declare only their legitimate cargo, leave opium on board to be picked up by Chinese merchants who smuggle it ashore in small, fast boats. China's emperor, Kia King, bans opium completely, mak-

ing trade and poppy cultivation illegal. A strong edict by authorities at Canton, supporting the emperor's decree of 1796, forbids opium trade at that port. A concurrent drive against native poppy growing is initiated. Opium becomes an illicit commodity.

1800's Patent medicines and opium preparations such as Dover's Powder were readily available without restrictions. Indeed, laudanum (opium mixed with alcohol) was cheaper than beer or wine and readily within the means of the lowest-paid worker. As a result, throughout the first half of the 19th century, the incidence of opium dependence appears to have increased steadily in England, Europe and the United States. Working-class medicinal use of opium-bearing nostrums as sedatives for children was especially prominent in England. However, despite some well known cases among 19th century English literary and creative personalities (Thomas de Quincey, Byron, Shelley, Coleridge, and Dickens) recreational use was limited, and there is no evidence that use was so excessive as to be a medical or social concern.

1800 The British Levant Company purchases nearly half of all of the opium coming out of Smyrna, Turkey strictly for importation to Europe and the United States. Opium becomes identified with official corruption, criminals and antigovernment secret societies. An edict prohibits domestic cultivation and repeats the prohibition against importing opium. China develops an anti-opium policy, at least on paper. Edicts continue to be issued reiterating prohibitions against importation, sale, and consumption of opium.

1803 Friedrich Sertuerner of Paderborn, Germany discovers the active ingredient of opium by dissolving it in acid then neutralizing it with ammonia. The result: alkaloids - principium somniferum or morphine. This may have been the first plant alkaloid ever isolated and set off a firestorm of research into plant alkaloids. Within half a century, dozens of alkaloids, such as atropine, caffeine, cocaine, and quinine, had been isolated from other plants and were being used in precisely measured dosages for the first time.

1819 Writer John Keats and other English literary personalities (Thomas De Quincey, Confessions of an English Opium-eater) experiment with opium intended for strict recreational use - simply for the high and taken at extended, non-addictive intervals.

1827 E. Merck & Company of Darmstadt, Germany, begins commercial manufacturing of morphine.

1830 The British dependence on opium for medicinal and recreational use reaches an all time high as 22,000 pounds of opium is imported from Turkey and India.

1832 Codeine was extracted from opium.

1835. First accurate drawing of coca appears in popular English press. Drawing by Sir William Hooker who was then the director of Kew Gardens.

1839 Opium and its preparations are responsible for more premature deaths than any other chemical agent. Opiates account for 186 of 543 poisonings, including no fewer than 72 among children. Lin Tse-Hsu, imperial Chinese commissioner in charge of suppressing the opium traffic, orders all foreign traders to surrender their opium. In response, the British send expeditionary warships to the coast of China, beginning the First Opium War.

1841 The Chinese are defeated by the British in the First Opium War. Along with paying a large indemnity, Hong Kong is ceded to the British.

1843 Dr. Alexander Wood of Edinburgh discovers a new technique of administering morphine, injection with a syringe. He finds the effects of morphine on his patients instantaneous and three times more potent.

1852 The British arrive in lower Burma, importing large quantities of opium from India and selling it through a government-controlled opium monopoly.

1853 The hypodermic needle was invented.

1856 The British and French renew their hostilities against China in the Second Opium War. The importation of opium is now legalized.

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. Many copies of Vin Mariani were made including Marza Wine which was made in East London. The Dutch are growing coca in Indonesia and the British in Ceylon. Germany and the USA are getting crops from plantations in South America.

1874 English researcher, C.R. Wright first synthesizes heroin, or diacetylmorphine, by boiling morphine over a stove. In San Francisco, smoking opium in the city limits is banned and is confined to neighbouring Chinatowns and their opium dens.

1876. Race walkers in England chew coca leaves to enhance their performance.

1877. 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.

1878 Britain passes the Opium Act with hopes of reducing opium consumption. Under the new regulation, the selling of opium is restricted to registered Chinese opium smokers and Indian opium eaters while the Burmese are strictly prohibited from smoking opium.

1883. Dr. Theodor Aschenbrandt, a German army physician, secures a supply of pure cocaine from the pharmaceutical firm of Merck, issues it to Bavarian soldiers during their manoeuvres, and reports on the beneficial effects of the drug in increasing the soldiers' ability to endure fatigue.

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’s rashness and endorsement of cocaine led to a close friend, Fleischl-Marxow, dying in a very short period of time from using both cocaine and morphine in a ‘speedball’ combination. Freud also treats his depression with cocaine, and reports feeling "exhilaration and lasting euphoria, which is in no way differs 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.

1890 U.S. Congress, in its earliest law-enforcement legislation on narcotics, imposes a tax on opium and morphine. Tabloids owned by William Randolph Hearst publish stories of white women being seduced by Chinese men and their opium to invoke fear of the 'Yellow Peril', disguised as an "anti-drug" campaign.

1895. Heinrich Dreser working for The Bayer Company of Elberfeld, Germany, finds that diluting morphine with acetyls produces a drug without the common morphine side effects. Bayer begins production of diacetylmorphine and coins the name "heroin"

As head of Bayer's pharmacological laboratory, he was responsible for the launch of two drugs: aspirin, which the world now consumes 40 billion tablets a year, the world's most successful legal drug; and heroin, the most successful illegal one.

There were heroin pastilles, heroin cough lozenges, heroin tablets, water-soluble heroin salts and a heroin elixir in a glycerine solution.

Late 1800's The manufacture of refined cocaine starts. Coca plantations start to appear in areas other than South America, such as India and Indonesia.

1898 The Bayer Company introduce heroin as a substitute for morphine.

Early 1900's 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. The composition of Coca-Cola is changed, caffeine replacing the cocaine it contained until this time. Other tonics voluntarily withdrew from the market. Products containing cocaine had to be labelled as poisons. There was no distinction made between the cocaine content in elixirs, that tended to be weaker, and the stronger, more potent patent medicines, that had more adverse effects.

The philanthropic Saint James Society in the U.S. mounts a campaign to supply free samples of heroin through the mail to morphine addicts who are trying give up their habits.

1902 In various medical journals, physicians discuss the side effects of using heroin as a morphine step-down cure. Several physicians would argue that their patients suffered from heroin withdrawal symptoms equal to morphine addiction.

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 as snuff. Early cocaine snorting was known as 'cocaine snuffing. U.S. Congress bans opium.

1906 The first Pure Food and Drug Act becomes law; until its enactment, it was possible to buy, in stores or by mail order medicines containing morphine, cocaine, or heroin, and without their being so labelled. China and England finally enact a treaty restricting the Sino-Indian opium trade. Several physicians experiment with treatments for heroin addiction. Dr. Alexander Lambert and Charles B. Towns tout their popular cure as the most "advanced, effective and compassionate cure" for heroin addiction. The cure consisted of a 7 day regimen, which included a five day purge of heroin from the addict's system with doses of belladonna delirium.

1909 The first federal drug prohibition passes in the U.S. outlawing the importation of opium. It was passed in preparation for the Shanghai Conference, at which the US presses for legislation aimed at suppressing the sale of opium to China.

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 along with opium on the international drugs agenda.

1914. Cocaine and heroin sales are restricted under the Harrison Act in the U.S.A. the Act requires doctors, pharmacists and others who prescribed narcotics to register and pay a tax. Start of 1st world war. 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 U.K. 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.

1923	The U.S. Treasury Department's Narcotics Division (the first federal drug agency) bans all legal narcotics sales. With the prohibition of legal venues to purchase heroin, addicts are forced to buy from illegal street dealers.
1924	The Heroin Act made manufacture and possession of heroin illegal.
1925	In the wake of the first federal ban on opium, a thriving black market opens up in New York's Chinatown.
Early 1930's	Japan is worlds leading cocaine producer, followed by the U.S.A., Germany, Great Britain and then France. China recovering from the British opium trade now has 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".
1940's	During World War II, opium trade routes are blocked and the flow of opium from India and Persia is cut off. Fearful of losing their opium monopoly, the French encourage Hmong farmers to expand their opium production.
1944	Nazi researchers used concentration camp inmates to test a cocaine-based "wonder drug" they hoped would enhance the performance of German troops.
1945	Burma gains its independence from Britain at the end of World War II. Opium cultivation and trade flourishes in the Shan states.
1950's	Cocaine starting to regain popularity. Is used by the 'Famous set' where it begins to get its rich drug image, also very expensive at this time. Production has now shifted from Japan to South America. U.S. efforts to contain the spread of Communism in Asia involves forging alliances with tribes and warlords inhabiting the areas of the Golden Triangle, (an expanse covering Laos, Thailand and Burma), thus providing accessibility and protection along the southeast border of China. In order to maintain their relationship with the warlords while continuing to fund the struggle against communism, the U.S. and France supply the drug warlords and their armies with ammunition, arms and air transport for the production and sale of opium. The result: an explosion in the availability and illegal flow of heroin into the United States and into the hands of drug dealers and addicts.
1960's	Again regaining popularity with the drug revolution of the 60's era. Media yet again play down its dependent qualities as in the late 1800's. The importation of cocaine to the US is starting to be controlled by the Cuban's. Burma outlaws opium.
Early 1970's	Freebase cocaine starts to be used. At first it is washed with ether and then ammonia. Columbians are now starting to gain control of the American and world market. USA flooded with cocaine, especially Miami and New York. U.S. involvement in Vietnam is blamed for the surge in illegal heroin being smuggled into the States. To aid U.S. allies, the Central Intelligence Agency (CIA) sets up a charter airline, Air America, to transport raw opium from Burma and Laos. As well, some of the opium would be transported to Marseille by Corsican gangsters to be refined into heroin and shipped to the U.S via the French connection. The number of heroin addicts in the U.S. reaches an estimated 750,000.
1973	President Nixon creates the DEA (Drug Enforcement Administration) under the Justice Dept. to consolidate virtually all federal powers of drug enforcement in a single agency.

Mid 1970's Saigon falls. The heroin epidemic subsides. The search for a new source of raw opium yields Mexico's Sierra Madre. "Mexican Mud" would temporarily replace "China White" heroin until 1978.

Late 1970's Crack cocaine use starts in the United States. Crack becomes more popular because it is easier to mass-produce with the use of bicarbonate of soda and a microwave oven. It is initially named 'ready rock' or 'garbage freebase' in California and is then named crack once it has hit New York street market.

During the 1970's a small group of doctors in California prescribe 'Esterene' to chronic rheumatoid arthritis sufferers. Esterene was freebase cocaine prepared for nasal application.

1978 The U.S. and Mexican governments find a means to eliminate the source of raw opium - by spraying poppy fields with Agent Orange. The eradication plan is termed a success as the amount of "Mexican Mud" in the U.S. drug market declines. In response to the decrease in availability of "Mexican Mud", another source of heroin is found in the Golden Crescent area- Iran, Afghanistan and Pakistan, creating a dramatic upsurge in the production and trade of illegal heroin.

1982 Comedian John Belushi dies of a heroin-cocaine- "speedball" overdose.

1983. Crack cocaine starts to be sold in London. Crack utilises established dope dealing networks to begin to establish a foothold.

1984 U.S. State Department officials conclude, after more than a decade of crop substitution programs for Third World growers of marijuana, coca or opium poppies that the tactic cannot work without eradication of the plants and criminal enforcement.

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

1992 Colombia's drug lords are said to be introducing a high-grade form of heroin into the United States.

1993. Crack/cocaine overtakes heroin in estimated importation amounts. 10 years after its introduction the first outreach services for crack users start to appear in London and Nottingham. Twenty-three-year-old actor River Phoenix dies of a heroin-cocaine overdose, the same "speedball" combination that killed comedian John Belushi.

1994. 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. The Golden Triangle region of Southeast Asia is now the leader in opium production, yielding 2,500 tons annually. According to U.S. drug experts, there are new drug trafficking routes from Burma through Laos, to southern China, Cambodia and Vietnam.

1998 A survey by Columbia University found that 21 per cent of violent felons in state prisons committed their crimes under the influence of alcohol alone, only 3 per cent were under the influence of crack or cocaine.

2000. Crack and cocaine use increasing in the UK. It is being used as far south as Jersey and as far north as Aberdeen. It has spread to injecting users and is now also being distributed through heroin dealing networks. It is also gaining popularity within the dance drug culture / bars and clubs. 17 years after its introduction to the U.K. it is still marginalised as a drug with regards to treatment and national strategy.

2002 The first UK conference on the combined use of crack and heroin held in London by COCA and Release.

2003. Crack and cocaine use is now beginning to be taken seriously by various government departments such as 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. Opium and heroin production increased in Afghanistan.
Cocaine becoming third drug of choice in UK recreational market.

3. Who is using crack and heroin?

Though crack and heroin users may only rank as less than one percent of the total population and the money spent on illicit drugs is an estimated £6.6 billion annually (a large percentage of it being spent on crack and heroin) it still does not take a particular kind of trait or characteristic in a person to pick up these drugs. Put simply, some people will try them, some not. Research consistently points to social deprivation, lack of education and opportunity as major causes of drug use, but these are not the only factors as members of the middle and upper-class can develop drug problems. Most professionals within the drug field realise that a whole range of issues are at stake for users who cannot afford to pay for private treatment. Even if some of these users have a tenancy, prospects for training, education and employment they may still have continual drug problems and therefore need additional support.

Markets change and diversify and this in itself changes the attitudes, norms and values attached to those particular markets. The opposite is also true; a shift in people's drug taking behaviour can also have an impact on the market. The process involves a number of reciprocal factors which make current trends difficult to quantify. Also, politicians and the media are integral to the development or reinvention of certain drugs. Like the spread of methcathinone (synthetic khat) in the USA which was due to a politician making its use public, or the media portrayal of heroin ('heroin chic'), in New York in the early 1990's which transformed heroin from being perceived as a 'dirty' drug to a 'cool' one.

In Britain the usual stereotypes attributed to injecting heroin users are that of white, working class males with zero qualifications. Though heroin use affects all classes and all sectors of society, (the author Will Self, for instance, was found using heroin on John Majors election campaign jet) surprisingly, this stereotype is difficult to dispel as most injectors of heroin are white. This is in synch with the evidence presented from current research. Heroin users presenting to prescribing and needle exchange services, or many drug services in general, are predominately white UK. This may be due to a range of historical and cultural factors. Though injection of drugs cuts across all cultures, both medically and non-medically, injecting users from BME communities form a small percentage of overall injecting users. Smoking, 'piping' or 'chasing' drugs on the other hand is widespread through all communities and smoking heroin as been identified in the South Asian communities, to what extent is unknown. Smoking of any illicit drug would be difficult to quantify because smoking in general is a more socially acceptable form of administrating drugs than injecting.

On the other hand the stereotypes and media portrayal of crack users tend to be that of black males who are normally associated with guns, violence and robbery. Crack dealers tend to be 'Yardies' and cocaine users are generally viewed as white middleclass or upper, business or media types. Cocaine dealers have been stereotyped as being glamorously violent, but it is usually the Cubans or Colombians who are portrayed in this light and not white UK 'businessmen', shipping in large consignments of cocaine into the UK via containerised cargo. Unlike white heroin injectors, the racial stereotypes attributed to crack and cocaine users are not as clear cut and can be effectively broken down.

In general, crack, cocaine and heroin use transcends across all cultural, age and social groups. Though there is an element of truth in most stereotypes, they fail to grasp the whole picture. Psychologically stereotypes are a shortcut route to information. We do not need to get into all the complicated facts, an image will usually suffice. For when we try to analyse the current situation - from chaotic to recreational drug use - stereotypes are no longer useful, or for that matter, necessary.

As current research stands, there are no tidy racial stereotypes that distinguish current users from other users except possibly the route of administration. The differences in administration, like injecting or smoking, probably tell us nothing about the drugs themselves but only their route of distribution and possible harm it may cause. These differences are useful, on the other hand, in informing interventions if needed by users. For example, opiate based prescribing services and nee-

dle exchange services target chaotic opiate and opiate injecting users, who are predominately white UK. If services widened their target to smokers (whatever the method of inhalation), both in treatment and in harm reduction, and include primary stimulant users in their remit, there may be more representation from BME communities. At lot of the crack specific projects have been successful in this respect. What needs to change in this instance is the parameters of opiate based services and not our understanding of culturally specific drug taking.

It is therefore important that agencies consider the wider spectrum of users and their need when setting up services as they may be targeting a market that has significantly changed. Although government statistics will place the majority of crack and heroin use in deprived areas there needs to be awareness that most of this evidence is rooted within the criminal justice system and is not identifying those who are not supporting their habit through criminal activity.

Types of crack and heroin users:

Crack / Cocaine and Heroin: This combination may happen in different patterns:

Crack main drug →
Heroin for come down
Heroin Main drug →
Crack for an up
Crack and Heroin →
Speedball
Crack and methadone → Methadone (boring)

The crack market, it appears, is now utilising heroin distribution networks and visa versa. Dealers are diversifying their products and are selling both heroin and crack which may have contributed to increased levels of speedballing.

Speedballing

Speedballing traditionally involved the simultaneous injection of cocaine and heroin. The combined use of cocaine and heroin is not new and dates back to the 1930s. Previous to this cocaine was combined with morphine. Sigmund Freud's friend Fleischl-Marxow was speedballing in 1884. The cocaine and heroin combination was also used in medical circles in the 1950's and went by the name of the Brompton Cocktail. Speedballing first attracted widespread attention in the USA when the comedian John Belushi died from speedballing in 1982.

In the past most speedballers primary drug of choice was heroin, these users added cocaine in the injection as a "treat" when they had the extra cash to buy it. From anecdotal evidence from opiate based service providers across the UK speedballing with this type of client group is on the increase, whereby opiate I.V. injectors are now injecting crack, not cocaine, as a treat.

Speedballing is usually associated with injecting users. However there have been reports of heroin and crack use within dance culture and the recreational market. It should be noted that it is possible to combine both drugs through a pipe where users are layering the crack and heroin between different gauzes and 'blasting' it with a blow torch ('burner'). It is also possible to run the two drugs on foil or smoke them in a 'joint'. This method does not solely reside with chaotic users or primary heroin users and is seen as a more socially expectable way of administering the drug.

Though there is anecdotal and empirical information that states that you can be a light recreational user of heroin, for instance some of the Australian studies point to this, we as yet do not see this present in the UK recreational market. Piping/smoking crack, on the other hand has been identified in the mainstream recreational market but is still relatively small. However, this is not

to say that there are no light recreational users of heroin. Light recreational or recreational users of any illicit drug are notoriously difficult to quantify for the simple reason that these types of users rarely access treatment and are not identified within criminal justice statistics, unless they get caught.

There is anecdotal information to suggest that high grade heroin is being imported into the UK for the sole purpose of smoking. This could indicate that there could be a growing market for recreational heroin users.

4. Types of cocaine and heroin

Cocaine

The coca leaf has been chewed in South America for over 3000 years. It wasn't until the mid 1800's 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 preference.

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 they are 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 and forms a paste commonly known in South America as 'Basuco'.

To refine it further the coca paste is again put through a various chemical processes to produce its acid salt state or cocaine hydrochloride. Cocaine hydrochloride is usually cut during the process / 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: Process of manufacture as above.

- Form - Powder (Salt Form).
- 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 - £40 - £50 per gram.
- Purity - Average around 50%
- Cuts - Most common cuts are Lignocaine hydrochloride and phenacetin

Freebase Cocaine:

There are three basic methods to freebase cocaine; these are Ether, Ammonia and Baking Soda (baking powder) Methods:

Freebase Cocaine: Ether Method, Ammonia Method

This process was first developed by drug dealers in the 1970's 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 left 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. Come down can be very rapid and low.
- Cost - Mainly self-manufactured, but if sold same price as crack (£10 - £20 per 'rock').
- Purity - Average between 70% to 90% (but can be lower)

Crack Cocaine: Baking Soda Method.
This involves a similar process to that of 'freebase' but uses bicarbonate of soda instead of ether or ammonia. 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 (but can also be ingested).
- Effect - As with freebase (Starts to take effect within 5 – 10 seconds if smoked).
- Cost - £10 - £20 per rock, some people will sell it for £5 but these are smaller rocks.
- Purity - Average around 80% but may be decreasing

Black Cocaine: This type of cocaine gets its name from the use of black magnetic iron particles and potassium thiocyanate to mask the cocaine when it is being transported through customs. It enables it to pass colour tests and can also bypass sniffer dogs. The mixture will then be put through a process to extract the cocaine before it is sold on the market. However the resulting street cocaine / crack may be a little darker due to the chemicals used to mask it.

General: 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 get you drunk. There is no safe way to take cocaine, they all have their dangers and complications according to the route used.

Cocaine and the law: Crack and cocaine are controlled under the Misuse of Drugs Act and is categorised as a class 'A' drug. It is illegal to produce, supply and possess and can only be legally used for certain medical purposes such as a local anaesthetic for plastic surgery. It is also illegal to allow premises to be used for the dealing or production of crack or cocaine. Legally users could be sentenced to 7 years imprisonment for possession and life for supplying crack cocaine. Although it is rare to find such heavy sentences being given out it is not unusual for users to serve a couple of years imprisonment for possession of half a sixteenth (£40 - £50 or 2 – 3 'rocks') of crack cocaine especially if it is connected with another crime.

6.1 Cocaine Acid and Alkaloid Forms:

There are basically two different states of cocaine:

Form	Acid or Alkali	Type
Base form	Alkaloid	Freebase or Crack Cocaine
Salt form	Acid	Cocaine Hydrochloride or crack prepared for injection using an acid

When cocaine is first produced it is in its base form and is therefore an alkaloid. 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 (alkaloid). In its alkaloid state it is far easier to smoke as the melting point has been reduced hence the process before smoking.

Preparation for injection:

Crack cocaine or freebase cocaine in its alkaloid state does not dissolve in water. It also does not return back to a hydrochloride state when it has been prepared for injection using vinegar, citric acid or vit C. The state depends upon the acid used.

When acids are used to convert cocaine into an injectable form the cocaine is being converted into an acid form (salt). But the form of the cocaine is dependent on the type of acid used.

- Vit C - changes crack into -cocaine ascorbate
- Citric Acid - changes crack into -cocaine citrate
- Vinegar - changes crack into -cocaine acetate

Cocaine hydrochloride is in an acid salt form so does not need to have an acid added to it as with crack and will dissolve in water alone.

Information from:

- Yale School of Medicine, Department of Epidemiology and Public Health 2002
- National Institute of Drug Addiction, USA 2002

6.2 Other forms:

The development of the poly drug culture in the UK / change in dealing networks also means 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 but can be combined in injection. Coke also dissolve's in alcohol.	Produces cocaethelyne in the liver which in itself interacts with reward system to produce a 'high'.	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 others effect leading to a very intense high. Also prolongs the 'comedown'.	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 'comedown'.	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'. Can also be combined in a smokeable 'rock'.	This combination feelings of euphoria combined with 'out of body' experiences.	Recreational mainly but can also be used by chronic and binge users when combined in a 'rock'.
Cocaine & Ecstasy (dynamite)	Usually the ecstasy is taken orally and the cocaine snorted	Cocaine boosts the euphoric effect that can be felt on ecstasy.	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.	Recreational use.
Cocaine & Steroids	Both drugs taken separately	Both drugs can cause complications with moods	Recreational use.
Crack & amphetamine	Can be taken separately or may be combined in a 'rock'. Speed rocks tend to be pinkish in colour.	Both these drugs work in a similar way, but amphetamines releases dopamine rather than prevents re-uptake.	Recreational, chronic and binge patterns of use.

Opium

The source of opium and all derivatives such as morphine and heroin is the opium poppy, Papaver somniferum. The genus, Papaver, is the Greek word for "poppy." The species, somniferum, is Latin for "sleep-inducing."

The largest licit opium producing regions in the world today are in government-regulated opium farms in India, Turkey, and Tasmania. The major illicit or underground growing areas are in Southwest Asia (Afghanistan, Pakistan, and Iran) and in the highlands of Mainland Southeast Asia (Bur-

ma, Laos, Vietnam, and Thailand) popularly known as the “Golden Triangle.” The opium poppy is also grown in Colombia, Mexico, and Lebanon. Afghanistan and then Burma are the largest illicit opium producing countries in the world.

The opium poppy grows in temperate, warm climates with low humidity, and requires only a moderate amount of water. The poppies produce flowers. Only the pod portion of the plant can produce the opium alkaloids. About two weeks after the flower petals fall from the pods the scoring of the pods (also known as incising, lancing, or tapping) commences.

Approximately a depth of one millimetre is desired for scoring. Using a tool designed to cut to that depth, scoring ideally starts late in the afternoon so that raw opium can ooze out of the pod and slowly dries on the surface overnight. The opium gum is then collected.

Before opium is smoked, it is usually cooked. Uncooked opium contains impurities which detract from a smooth-smoking product. The raw opium is placed in a pot of boiling water where the sticky opium alkaloids quickly dissolve. The solution is strained through cloth to remove the impurities. A clear brown liquid, sometimes called “liquid opium,” is left and is actually opium in solution. This liquid then is reheated until the water turns to steam. When the water has evaporated, a thick paste remains. This paste is called “prepared opium,” “cooked opium,” or “smoking opium” and it is dried in the sun until it has a putty-like consistency. It is now ready for smoking or eating.

Opium, either raw or cooked, will not degrade, or otherwise spoil, for an indefinite period of time, as long as it remains relatively dry and cool. There are cases of opium being stored on a shelf for ten years without deterioration.

To make heroin, manufacturers must first extract the morphine from the opium, before converting the morphine to heroin. Morphine extraction is quite simple, requiring only a few chemicals and a supply of water. Morphine sometimes is extracted from opium in small clandestine laboratories, which are set up near opium poppy fields.

Since the morphine base is about one-tenth the weight and volume of raw opium, it makes sense to reduce the opium to morphine before transporting the product from the field to a heroin laboratory for later distribution.

Raw or cooked opium contains more than 35 different alkaloids, including morphine and codeine.

Morphine

In 1803, the German pharmacist F. W. Serturner isolated the principal alkaloid in opium by dissolving opium in acid and neutralising it in ammonia. He named the substance morphium (morphine) after Morpheus, the Greek god of dreams. The invention of the syringe and the discovery of other alkaloids of opium soon followed.

Crude morphine is sometimes referred to as heroin number one and prior to heroin conversion, heroin number two. Morphine is medically one of the most effective drugs known for the relief of severe pain and remains the standard against which new painkillers are measured.

It is stated that morphine is up to 1,000 per cent stronger than opium. It is pharmaceutically produced in tablet, suppository and injectable ampoule form. Usually the form found on the street is from medical stock.

Codeine

Codeine was extracted from opium in 1832. Codeine is the most widely used, naturally occurring narcotic in medical treatment in the world. This alkaloid is found in opium in concentrations ranging from 0.7 to 2.5 percent.

Codeine is medically prescribed for the relief of moderate pain and cough suppression. Codeine produces less sedation, and respiratory depression than morphine, and is usually taken orally. It is made into tablets either alone or in combination with aspirin or acetaminophen.

As a cough suppressant, codeine is found in a number of liquid preparations. Codeine is also used to a lesser extent as an injectable solution for the treatment of pain. Codeine products can find their way onto the illegal market.

Codeine is also the starting material for the production of two other painkillers, dihydrocodeine and hydrocodone. Dihydrocodeine is one of the most commonly prescribed painkillers in the UK

Heroin

In 1874 English researcher CR Adler Wright first isolated and developed an opium-based and supposedly non-addictive substitute for morphine. Morphine and acetic anhydride are heated together for approximately six hours to produce impure diacetylmorphine. The Bayer Pharmaceutical Company of Germany was the first to commercially produce this new drug under the brand name Heroin.

Form – Can come in several forms from brown or black tar coloured alkaloid to white hydrochloride salt. Its appearance varies considerably depending on the amount of refining and the manufacturing process which tends to be illegal.

Route - Heroin can be smoked, snorted injected or inhaled (which involves heating rock or powdered heroin on aluminium foil and inhaling the fumes, referred to as 'chasing the dragon' or ‘booting’ as in bootlace for chase.

Effect - 8 – 10 seconds if injecting). Slower if smoked.

Purity levels can reach 90 per cent by the time heroin leaves the refineries. Pure heroin is rarely sold on the streets, though in recent years the purity has risen to an average of approximately 50 per cent with a purity range of 2 to 90 per cent.

Heroin base
Also known as “crude heroin.” Heroin base is morphine base that has undergone acetylation. Sometimes called Southeast Asian heroin no. 2. Not easily soluble in water, and therefore not in-

jectable in this form. This form of heroin can be smoked. Heroin base may be further refined to either no. 3 or no. 4 heroin.

Heroin hydrochloride
Formed when heroin base is treated with hydrochloric acid. Usually in powder or crystal form, that easily dissolves in water and therefore suitable for injection. Sometimes called Southeast Asian heroin no. 4.

Heroin no. 3
A smokeable form of heroin. Not as highly refined as no. 4. Colour ranges from purple to tan to off-white. It may also be injected intravenously.

Heroin no. 4
An injectable form of heroin. Also known as heroin hydrochloride (as above) or China White. Highly refined heroin. Usually a fine white powder, flakes, or crystals. May be smoked or snorted.

Methadone

German scientists synthesized methadone during World War II because of a shortage of morphine due to trade routes being disrupted. Although chemically unlike morphine or heroin, methadone produces many of the same effects for longer hours without the ‘rush’.

It is prescribed in the UK and other counties as a substitute for heroin dependent users. It is available in oral solutions, tablets (physepton), and injectable formulations. Methadone's effects can last up to 20 - 24 hours, thereby permitting once-a-day oral administration in heroin reducing and maintenance programs. The most common form of administration is oral, in the form of supervised consumption. In the United Stated LAAM, another synthetic opioid, is prescribed for heroin dependency, with effects lasting longer than methadone.

Fentanyl

First synthesized in Belgium in the late 1950s.It is stated that fentanyl is fifty times stronger than heroin but the euphoric effects are less than those from morphine.

When used in surgery machines help patients breathe, unlike on the street where many users have been found dead with the syringe still hanging out of their arm. Illicit use of pharmaceutical fentanyls first appeared in the mid-1970s in the medical community and continues to be a problem in the United States. To date, over twelve different analogues of fentanyl have been produced clandestinely and identified in illegal drug markets in the U.S.

Buprenorphine

Buprenorphine is a partial agonist and is prescribed under the brand name of Subutex. It is found to be effective in heroin withdrawal and prevent the need to use heroin. It was also used illegally as a heroin substitute on the street in the 1980's.

Buprenorphine can be administered orally. A tablet is placed under the tongue. The tablet dissolves over 3-7 minutes and is absorbed straight into the bloodstream from the mouth. (The tablets do not work if swallowed into the stomach.) It is usually prescribed as a once daily dose taken under supervision.

Naltrexone

Naltrexone is a blocker. It blocks the effects of opiate drugs such as heroin or morphine. It is used in the treatment of opioid dependency but has recently been used with drinkers. In clinical trials which evaluated the effectiveness of naltrexone, patients who received naltrexone were twice as successful in remaining abstinent and avoiding relapse as patients who received a placebo. Naltrexone should not be used with pregnant women, individuals with severe liver or kidney damage or with clients who cannot remain abstinent for at least five days prior to taking naltrexone. Also, people who are dependent on opioid drugs must stop their drug use at least seven days prior to starting the medication.

Naloxone

Naloxone is an antagonist. It prevents or reverses the effects of opioids, including respiratory depression, sedation and hypotension. Naloxone works by competing against other opioids at the receptor sites.

Naloxone has not been shown to produce tolerance nor to cause physical or psychological dependence. No short-term toxicity has been observed but long-term safety has not been investigated. There is no clinical experience with naloxone overdoses in humans. Suboxone is buprenorphine and naloxone combined.

Lofexidine

Lofexidine reduces the symptoms of withdrawal from opiate dependency during supervised detoxification. It is presumed that opiate withdrawal symptoms such as diarrhoea, sweating, cramps, chills, etc are caused by a temporary excess of noradrenaline. Lofexidine reduces the effect of noradrenaline, thus reducing opiate withdrawal symptoms. Lofexidine does not suppress all opiate withdrawal symptoms, in particular tiredness and sleeplessness. The course is administered either over five days, which is the standard five day detox or as a seven-to-twelve-day programme.

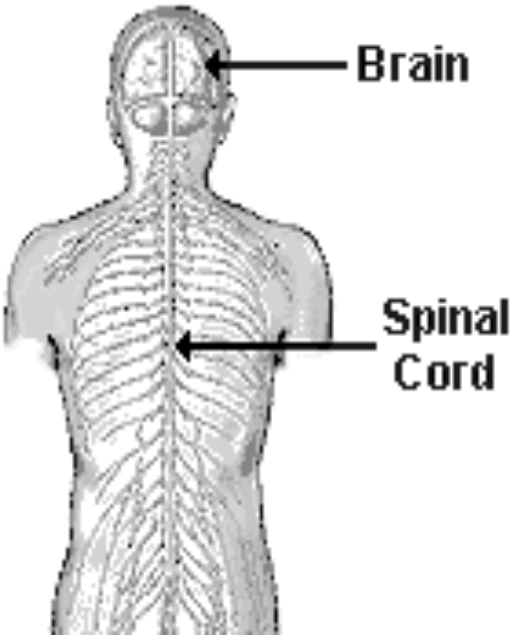
5. How crack and heroin work

The effects of any drug (including crack, cocaine or heroin) vary from one person to the next. It depends on many factors including a persons size, weight and health, how the drug is administered, how much of it is actually taken, whether the individual is used to taking it, the person's mood at that particular time and whether other drugs have been used in combination. The effects also depend on the environment in which the drug is used, for example, whether the person is by themselves or with others.

The Nervous System

To understand the effects of stimulants and narcotics on the human body we must first understand how the nervous system functions. The nervous system is a complex network of tissue that branches out from the brain and spinal cord. Its operation controls all our actions and reactions. It enables us to adjust to our changing environment.

The nervous system operates by receiving signals from all parts of the body, relaying them to the brain and spinal cord, and then sending the signals out again to muscles and body organs.

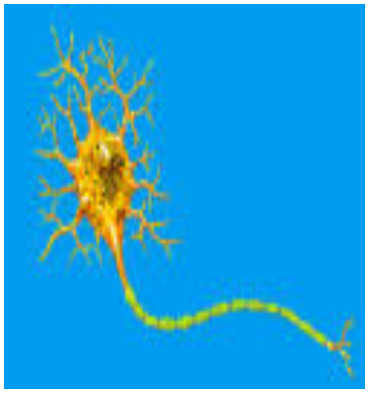


The nervous system has two divisions: the central nervous system and peripheral nervous system.

The central nervous system, consists of the brain and spinal cord. The peripheral nervous system comprises cranial nerves, controlling face and neck; spinal nerves, radiating to other parts of the body; and autonomic nerves, which form a subsystem regulating the muscles of heart, glands, lungs, stomach, etc.

Stimulant and sedative drugs will affect the nervous system in different ways but suffice to say that stimulant drugs will stimulate the central nervous system and narcotic drugs will depress or sedate the central nervous system.

Neurons



The basic unit of the nervous system is the nerve cell (neuron). Of the100 billion plus neurons in humans, half are in the brain.

The neuron is not a simple cell. It consists of a cell body (soma), containing the cell nucleus; dendrites that branch out from the cell, (extensions that receive incoming signals) and the axon, (a long cell extension that carries long distance signals).

A neuron works by receiving chemical signals-some excitatory, some inhibitory-through its dendrites and sending electrical impulses along its axon. Stimulants and narcotics work on neurons in specific ways.

How does crack/cocaine work?

Crack and cocaine are thought not to be physically dependent in the way that we understand heroin dependence, it can however, 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 and heightens the senses and enables the body to work at peak performance. It does this by:

Increasing heart rate: This is to increase the blood flow around the body, which also increases the speed of which oxygen gets to muscles.

Increasing breathing rate: Short and shallow breaths increase the amount of oxygen in the blood stream.

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 is getting hotter and sweating is the body's the 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 / cocaine or are just about to score. When they do use crack / cocaine they are again releasing adrenaline because of cocaine's effect on the neurotransmitter noradrenalin 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 & auditory hallucinations, impaired cognitive ability (due to lack of sleep), severe anxiety and paranoia. The environment that someone is using in can also affect these feelings. For instance if used in a hostile environment like a crack house or with someone they don't trust then the feelings of anxiety and paranoia can be worse.

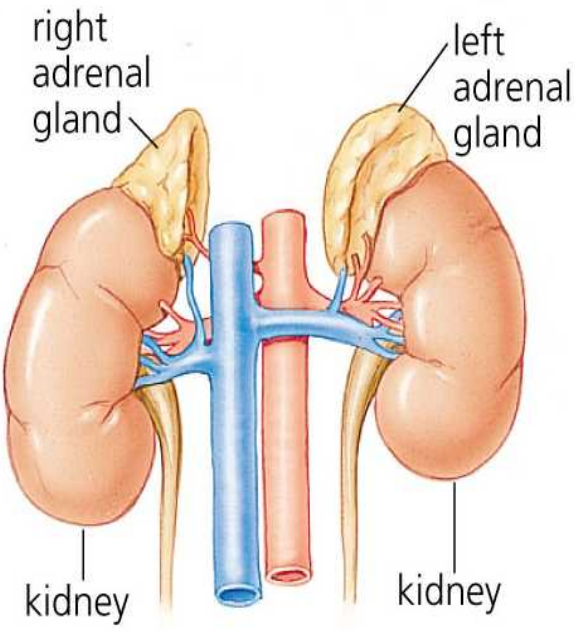
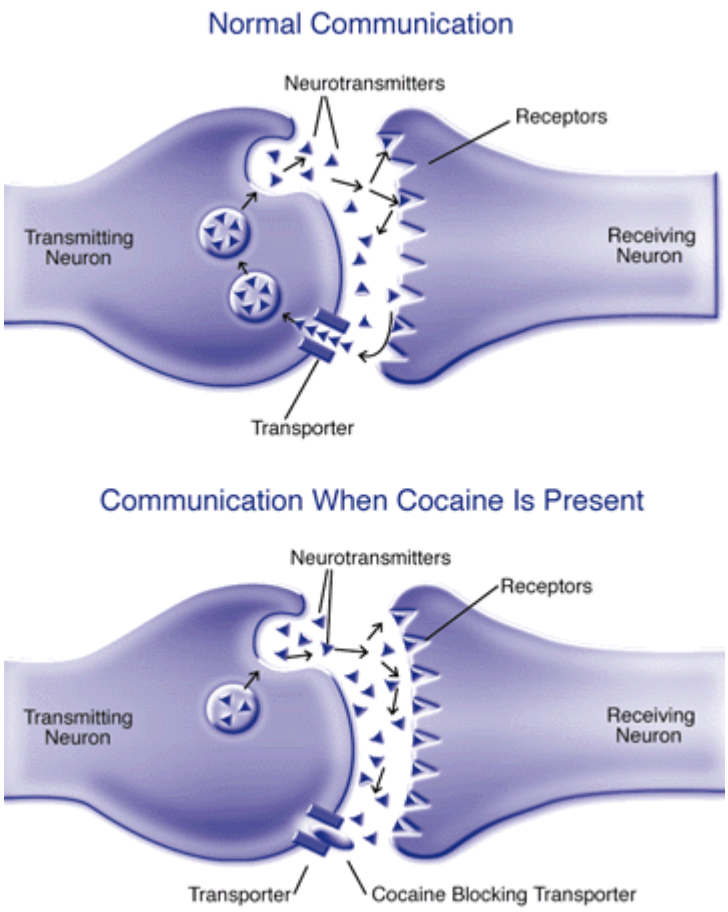


Image source: Dictionary of English Language

Dopamine and Serotonin:

The ‘high’ experienced when taking crack or cocaine is produced by 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 fire across a gap between each cell and attach onto receptor sites. Once the message has been received a transporter cell then collects up the neurotransmitters so that the levels in these chemicals remain 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 can't get back. 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 high they are aiming for. In this way all that is really happening is that they are kidding themselves into thinking that 'this will be the one' and the next, and the…

Image source: NIDA Website

The depletion of dopamine is partly responsible for the 'come down' or 'crash' making users feel bad and reinforcing the need for another hit, then another and another etc. Depletion in these neurotransmitters can also cause a chemical depression, which can sometimes combine with bad things happening in their lives (loss of job, 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 using 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.

Cravings and compulsion to use:

The urge to use crack or cocaine comes from a combination of the affects 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 (initial adrenaline release) 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 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.

7.1 Adrenaline, Dopamine and serotonin

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	Initial release: (first high / buzz) Reward and reinforcement
• Increased heart rate • Faster breathing • Sweating • Shaking / can't stay still • Butterflies / sickness in stomach	• Very strong first high • Feelings of confidence • Euphoric / orgasmic • Compulsion to use again
Prolonged release: (continued use can cause the following)	Prolonged release: (depletion of dopamine)
• Can't sleep • Don't want to eat • Increased anxiety ('wired' or 'prang') • Harder to think clearly • Hallucinations (also to do with brain chemicals) • Paranoia	• Repeated compulsion to use • Buzz getting shorter and lower • Comedown or 'crash' • Loss of interest in things not related to cocaine • Mood swings • Depression

How does heroin work?

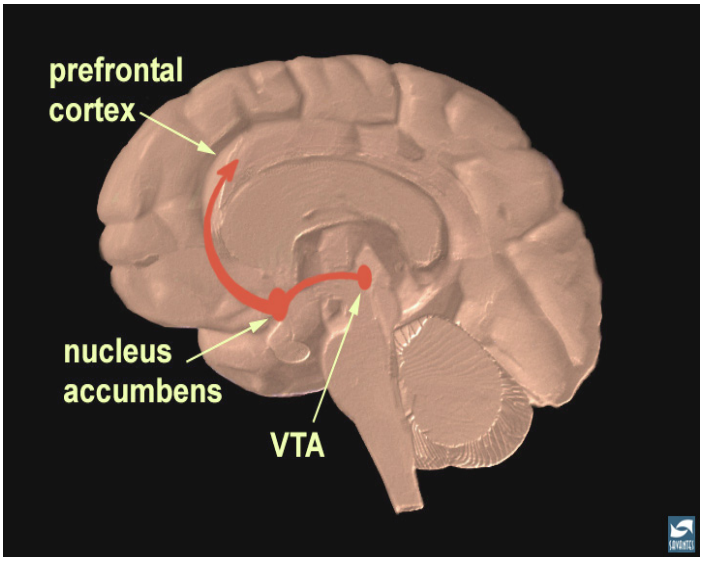
In 1973, scientists discovered that the brain had receptors for opiates and that the equivalent of opiates was produced naturally in the brain. These receptors were located in parts of the brain important for breathing, pain and emotions. The discovery of opiate receptors in the brain raised the question as to why neurons would have such receptors. A couple more years down the line scientists further discovered the brain manufactured its own opiates known as "endorphins." Endorphins are always present within the brain, but release is increased when people and animals are in pain or under stress.

Not all of the mechanisms by which heroin and other opiates affect the brain are fully understood. Neurology is not an exact science. However, there are large numbers of opioid receptors in the mesolimbic system, which is responsible for feelings of happiness, relaxation, fearlessness and tolerance to pain. When the receptors are flooded with morphine, the user experiences a sensation of pain-free euphoria and relaxation.

Heroin, (diacetylmorphine), crosses the blood-brain barrier quickly, where, after being changed into morphine, it acts by attaching itself to opioid receptors. Opiates also attach themselves to receptors in the spinal cord and other locations within the brain where pain is experienced. Binding to these opioid receptors creates an analgesic effect within the pain pathway, reducing the pain felt.

Opiates work similar to crack and cocaine in the sense that they both stimulate a "pleasure system" in the brain. Opiates indirectly affect “dopamine," whereby cocaine directly affects dopamine. These midbrain dopamine neurons are located in the ventral tegmental area which projects to an-

other structure called the nucleus accumbens which then projects to the cerebral cortex.



The body builds tolerance to the effects of heroin quickly. Increased amounts of the drugs needs to be consumed to have the desired effects as enzymes within the brain responsible for firing other neurons become accustomed to the presence of the opiate. More of the opiate is needed so that the enzymes can fire in the desired way.

If heroin is continuously consumed the brain again becomes accustomed to the presence of morphine. This is called dependency. When heroin use ceases then the user will experience withdrawals symptoms. The neurons become over activated because they have relied on the presence of morphine which is no longer in the system.

As previously mentioned. Heroin has sedative, painkilling and euphoric effects as the pain pathways in the body also cut across the reward circuits within the brain. It is easy to understand why heroin is used to take the edge off a crack binge, but what is happening when both are combined together in a ‘speedball’? There is limited amount of research into this area but it is stated by users that the high of both drugs combined produces an euphoria that surpass both crack/cocaine and heroin when used in isolation. Below is a table which shows the effects of both drugs when used separately.

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; this is the part of the brain that controls among other things, feelings of pleasure. When dopamine has been released it will attach 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 reuptake 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. Nervous System: Cocaine works with the sympathetic part of the nervous system, which is concerned with outside stimulus such danger and anticipation. The 'Fight and Flight' response is part of this and releases adrenalin into the body. Cardiovascular System: Cocaine increases the heart rate through the release of adrenalin and at the same time releases a chemical called endothelin which reduces the size of blood vessels (not a good combination). Respiratory System: Again cocaine stimulates the respiratory system through the release of adrenalin, especially when the user is craving or experiencing a bad 'come down'. Dependency: Physical dependency to cocaine is debatable. Cravings are triggered because of thoughts of using rather than a physical need for the drug.	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 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: 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: 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's 'Rest and Digest'. Dependency: Heroin causes a physical dependency because the brain adapts itself to accommodate the regular use of this chemical. Cravings are often associated with periods of physical withdrawal.

Speedballing

Speedballers report that the effect sought is the rush of crack or cocaine injection combined with the mellowing and soothing effect of heroin. The crack or cocaine hits first and then the heroin 'kicks' in so there is no sudden comedown. Some users call this the 'parachute' comedown and would agree that they are more satisfied with the high from speedballing.

Effects of Heroin and Cocaine on dopamine

Heroin-Enhanced Dopamine Activity

Heroin increases the neuronal firing rate of dopamine cells. The heroin user experiences the enhanced dopamine activity as mood elevation and euphoria.

Cocaine-Enhanced Dopamine Activity

Cocaine inhibits the reuptake of dopamine. This increases the availability of dopamine in the synapse and increases dopamine's action on the receptors. The enhanced dopamine activity produces mood elevation and euphoria.

Combined Heroin- and Cocaine-Enhanced Dopamine Activity

Because heroin and cocaine work on different parts of the mesolimbic dopamine neurons, they can be combined to produce even more intense dopamine activation. The heroin increases firing and dopamine release, while the cocaine keeps the released dopamine in the synaptic gap longer thereby intensifying and prolonging its effects.

Reinforcing effects of cocaine and heroin combination

Experiments on rhesus monkeys have shown that when cocaine was combined with heroin, the heroin increased the potency of cocaine as a reinforcer. Because both cocaine and heroin function as reinforcers, it is possible that the reinforcing effects of cocaine and heroin combinations are enhanced compared to either cocaine or heroin alone. However, this research is not conclusive.

Cloning

Recent research in both areas of investigation (opioids and cocaine) is now crossing over into the realm of genetics. Scientists are now beginning to clone re-uptake pumps to get a better idea of how cocaine - or possible future substitutes or blockers – bind to the transporter sites. In the case of heroin and other opiates, geneticists are now cloning opioid receptors, again to get a better understanding on how opioids bind to these sites.

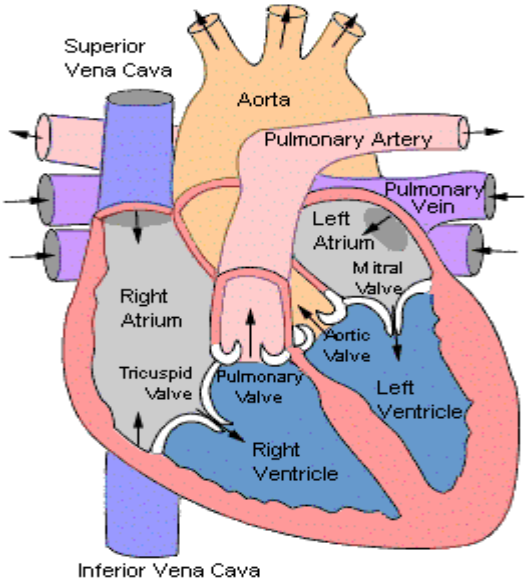
6. Health implications

Crack and Cocaine

Crack and cocaine can damage your 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.

Effects on the heart:

Heart failure can happen to anyone taking crack or cocaine, it does not matter how much they are taking or how long they have been using for. 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 down to frequent cocaine use. When taking crack or cocaine, you can increase the risk of having a possibly heart attack by 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 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)
- Arrhythmia's (erratic heart beat)
- Ashen grey skin (poorly oxygenated blood)
- Current heart problems (Made worse by cocaine)
- Other drugs that may be used in conjunction with cocaine such as Viagra and alcohol (can increase the stress upon the heart)

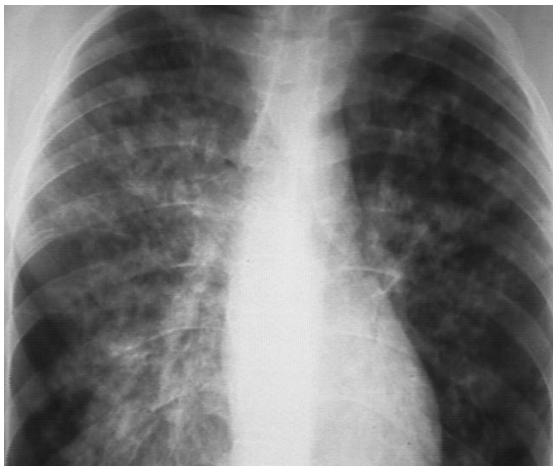
Sodium Bicarbonate (used to ‘wash’ cocaine to turn it into crack) may have some effect upon the heart putting it under further stress. And cocaethylene, a chemical that is produced in the liver when using crack / cocaine and alcohol together, also exerts more pressure on the cardiovascular system, than if cocaine were just taken on its own.

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 the blood supply to parts of the brain and also in some cases cause delicate blood vessels to break (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 that are 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 barotraumas - Air escaping lungs (by holding in crack smoke)
- Foreign bodies in lungs - Poor pipes, no gauzes used
- 'Crack Lung' - Cough, shortness 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 (this can be made worse by the impaired immune system).

Tuberculosis may be a new risk factor for crack and cocaine users as there is emerging evidence from the USA (University of Texas-Houston Health Science Centre) that is suggesting that there is increased chances of catching TB. This is 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 sure way of sure diagnosis is through a chest x-ray or skin test.

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

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 Hep 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 don't work as effectively as they should). This cell helps fight off infections throughout the body. Prolonged use can lead to 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 client has stopped using crack or cocaine.

Excited Delirium: (ED)
Excited delirium (agitated delirium) is thought to be caused by the build up of dopamine in certain areas of the brain after repeated binges of crack or cocaine. The symptoms of ED include (below) and may be followed by a heart attack:

- Bizarre or violent behaviour (incoherent shouting)
- Hyperactivity (lots of energy)
- Hypothermia (inability to regulate body temperature)
- Extreme paranoia

Pregnancy:
Crack or cocaine use is definitely <u>not</u> 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 that they bottle-feed.

It is vitally important that if someone has used when they are pregnant that 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.

Psychiatric Issues:
Some diagnosed psychiatric disorders can appear to get better 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 individuals are receiving the appropriate support from mental health professionals. Psychiatric illnesses that may be complicated by the use of crack or cocaine are:

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

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)
- Hypothermia (increased body temperature)
- Can exacerbate asthma and increase attacks
- Complications with epilepsy and sickle cell (increased attacks)

8.2 Crack and blood borne viruses (BBVs)

The issue of BBV's in connection with crack and cocaine use has to a large extent been ignored unless the route of use is injecting and even then important elements are not being addressed. There is a need to challenge this and disseminate information to users who are 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 and recent research into crack and the commercial sex industry (Mainliners, 2002) has highlighted that some working girls / boys are willingly having unprotected sex for an increased price to support their habit.

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.

Recent 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 affects HIV in two ways;

1. Cocaine can double the amount of HIV infected cells
2. Cocaine can deplete the number of CD4 T-Cells by up to nine times

The above combination can obviously have a dramatic affect upon the health of an individual who is HIV positive and taking cocaine, whether it is on a recreational basis or dependent use.

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

Injecting:
As mentioned above 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 lips becoming chapped. These can often be picked producing open wounds and the virus 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 nasal mucus membranes can occur causing the nose to bleed. The practice of sharing straws to ‘snort’ is quite common leading to the possibility of blood to blood transmission via the straw.

Heroin

The effect on users is more intense in the few minutes after injection (the 'rush') after which follow several hours of lethargy and sleepiness. During this time;

- The user may feel nauseous and may vomit, though the nausea doesn't distress them,
- Intense pleasure: heroin may cause a rush of intense pleasure and a strong feeling of well-being.
- Pain relief: heroin relieves physical pain. After using heroin, feelings of pain, hunger or sexual urges are diminished.
- Users may have trouble urinating,
- Drowsiness increases, as the quantity used increases, the user may feel warm, heavy and sleepy, eyelids may droop, close and open again, (they are 'gouching'),
- The user may sweat and itch, scratching themselves continually,
- Breathing becomes slower than normal,
- the pupils of their eyes contract (the eyes are 'pinned'),
- The user may become constipated.

When the effect wears off, the person may feel slightly drowsy for a day or so. After that they return to normal. However, if they start using heroin on a daily basis after a period – usually a few weeks – tolerance to the drug develops. The body's natural way of responding to this excess of opiates is to reduce the number of opioid receptors in the brain.

When heroin is no longer in the body there are a number of physical and biochemical changes which give rise to withdrawal symptoms, including: muscle cramps, irritability, anxiety, abdominal pains, chills, nausea, diarrhoea, sweating, sniffing, sneezing, weakness and insomnia.

These extremely uncomfortable sensations begin within 12 hours of not using, and peak after two to four days; subsiding after about a week. Death from withdrawal is rare.

Overdose

Using a large quantity of heroin can be fatal. Breathing slows down, body temperature drops, and heartbeat becomes irregular.

Overdose may occur if:

- too much heroin is injected, or it is a strong batch; or
- heroin is combined with other depressive drugs.

Most overdoses occur as a result of poly-drug use.

While unconscious, the person may also inhale and choke on their own vomit, which could cause a chest infection, long-term problems or death.

Short-Term Effects	Long-Term Effects
• "Rush" initial hit • Depressed respiration • Clouded mental functioning • Nausea and vomiting • Suppression of pain	• Dependence • HIV/AIDS and hepatitis B and C • Collapsed veins • Bacterial infections • Abscesses • Infection of heart lining and valves

Long-term use

Heroin is relatively non-toxic to the body in its pure form, causing little damage to body tissue and other organs. However, over long term use, users may become dependent on it. One of the most detrimental long-term effects of heroin is dependency.

Dependency is characterized by compulsive drug seeking behaviour and use, and by neurochemical changes in the brain. Heroin also produces tolerance and physical dependence, which are also powerful motivating factors for compulsive use. Users gradually spend more time and energy in searching, obtaining and using the drug. .

Physical dependence can develop with higher doses of the drug. With physical dependence, the body adapts to the presence of the drug and withdrawal symptoms occur if use is reduced abruptly. Symptoms of withdrawal include restlessness, muscle and bone pain, insomnia, diarrhoea, vomiting, cold flashes with goose bumps ("cold turkey"), and leg movements (kicking the habit). Withdrawal symptoms peak between 24 and 48 hours after the last dose of heroin and subside after about a week. However, some people have shown persistent withdrawal signs for many months.

Psychological problems can persist for longer periods. Craving and relapse can occur weeks and months after physical withdrawal symptoms are long gone. Patients with chronic pain who need opiates to function (sometimes over extended periods) have few if any problems leaving opiates after their pain is resolved by other means. This may be because the patient in pain is simply seeking relief of pain and not the rush sought by the illicit heroin user. In this case the ‘knowing’ is a factor in dependency. Knowing that if they have another hit of heroin then withdrawal symptoms will disappear. Recent neurological studies suggest that dependency and addiction affect two different parts of the reward system in the brain.

Consequences of chronic heroin use include scarred and/or collapsed veins, bacterial infections of the blood vessels and heart valves, abscesses and other soft-tissue infections, and liver or kidney

disease. Lung complications (including various types of pneumonia and tuberculosis) may result from the poor health condition of the user as well as from heroin's depressing effects on respiration.

Problems are more likely to occur if heroin is injected, for example, DVTs, skin, heart and lung infections and diseases such as hepatitis, HIV and other blood-borne viruses may arise. Sharing needles and the process of breaking down ‘brown’ with an acid are still ongoing practices. Many of the ‘cuts’ in street heroin may include substances that do not readily dissolve in acid and result in clogging the blood vessels that lead to the lungs, liver, kidneys, or brain.

Heroin users also report high rates of hopelessness, anti-social behaviour and self-harm. Depression can be both reactive and chemical, especially if heroin is combined with other drugs. Users can neglect such things as housing and food and, combined with reduced appetite, this can lead to malnutrition, low self esteem and depression.

Pregnancy:

As Dominic Streatfeild mentioned in his book ‘Cocaine’ most of the ‘crack babies’ photographed during the ‘crack baby’ scare of the 1980’s were in fact babies born from poly-drug or opiate using mothers.

Heroin can cross the placenta and an unborn baby can become dependent on the drug. Babies of heroin-dependent mothers can suffer withdrawal symptoms after they are born. They often need special care in hospital.

Using heroin while pregnant can harm an unborn child. These babies are often born underdeveloped. They suffer from breathing problems and infections in the first few weeks of life.

Heroin can cause premature labour. Babies may be born so early that they need intensive care. The baby's poor health can also be associated with the poor health and nutrition of their mothers.

Injecting heroin also increases the risk of HIV infection and other disease for both mother and baby.

Pregnant women who want to stop taking heroin need to be very careful. Sudden withdrawal from heroin may harm the baby and cause poor growth, miscarriage or premature labour.

Speedballing

Dangers occur with this combination because:

- Neurologists may have found that when combined heroin can increase the potency of cocaine and visa versa by acting on the cocaine mu agonists and increasing its capacity as a reinforcer.
- Heroin lowers respiration rates and in high doses so can cocaine, leading to dangers of respiratory failure.
- Opiates can lower the threshold for brain seizures and cocaine can cause brain seizures.
- The affects of cocaine can interfere with the user’s ability to judge how much heroin they have really had, increasing the potential for accidental overdose. Amounts of each drug should be lowered when used in combination. Because of cocaine’s initial effects on the cardiovascular system it is unlikely that overdoses will occur immediately after injection as with normal heroin overdoses.

The information in this pack is taken from a variety of different sources and written from a drug workers point of view. It is not meant to be a definitive document and the authors would advise that information be constantly checked as it can become out of date very quickly.

References:

Models of Care: For the Treatment of Drug Users, NTA, 2002

Quads: Professional Competencies for Alcohol and Drug Workers, Drugscope, 2000

Commissioning cocaine / crack treatment, NTA, 2002

Crack and Cocaine: Good Practice Guidelines for Drug Treatment and Testing Orders, A. Gray, D. Taegtmeyer, Commissioned by London Probation Service, 2002

Process and Intervention Chart, James, Robin & Gray, COCA 1999

A cognitive Behavioural Approach: Treating Cocaine addiction, Kathleen M. Carroll, NIDA, 2001

Report of an independent review of drug treatment services, Task Force to Review Services for Drug Misusers, Department of Health, 1996

Needs analysis of Carers and parents of African Caribbean and Asian Substance Users, Orchid Research Tas Gooden 1998

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

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

Expert Stimulant Group Findings, NTA, Unpublished 2002

National Crack Cocaine Treatment and Response Strategy, G. Haynes, T. Bottomley, A. Gray, Unpublished, 2000

National Cocaine Treatment Study: Report To The Task Force, M. Donmall, N. Seivewright, J. Douglas, T. Draycott and T. Millar, Manchester University 1995

Street Business: The Links Between Sex And Drug Markets T. May, M. Edmonds, M. Hough and C. Harvey, Home Office 1999

Drug Testing Arrestees, Home Office Research and Statistics Directorate, Home Office 1998

Drug and Alcohol National Occupational Standards (DANOS), Healthwork UK, 2002

A Brief History of Cocaine, Steven B. Karch

Cocaine, an unauthorised biography, Dominic Streatfeild, (2001), Virgin Publishing Ltd

Carmen Bernard The Inca's: Empire of Blood and Gold (1998) Thames and Hudson.

Misuse of Drugs Act (1976).

Michael Donmall, Nicholas Seivewright, Julie Douglas, Tamara Draycott and Tim Millar. National Cocaine Treatment Study: Report to the Task Force (1995) Manchester University.

Inciardi, J.A. and Harrison, L.D. Heroin in the Age of Crack Cocaine (1998) Sage.

American Drugs Enforcement Agency Black Cocaine (1998) DEA Briefing.

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

Paul A. Garris, Mark Wightman, William Rand Kenan Jr. Dopamine Not Brain Pleasure Source (1999) University of North Carolina – Chapel Hill.

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

Karen I. Bolla. How Cocaine Use Hurts Brain Function (1999) Journal of Neuropsychiatry and Clinical Neuroscience.

Meryem Tuncel, MD et al, "Mechanism of the Blood Pressure—Raising Effect of Cocaine in Humans" From the Department of Internal Medicine, Hypertension Division, and Donald W. Reynolds Cardiovascular Clinical Research Center, UT Southwestern Medical Center, Dallas, Texas 2002

Zickler P "Cues for Cocaine and Normal Pleasures Activate Common Brain Sites", NIDA NOTES 2002

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

Garavan, H.; et al "Cue-induced cocaine craving: neuro-anatomical specificity for drug users and drug stimuli". American Journal of Psychiatry, 2000.

Stocker S, "Cocaine's Pleasurable Effects May Involve Multiple Chemical Sites", NIDA NOTES, 2002

ND Volkow, JS Fowler, AP Wolf, R Hitzemann, S Dewey, B Bendriem, R Alpert and A Hoff. Changes in brain glucose metabolism in cocaine dependence and withdrawal (1991) American Journal of Psychiatry

Murray Mittleman. Cocaine Use Ups Heart Attack Risk (1999) Circulation, Journal of the American Heart Association.

David A. Severance. Cocaine and Heart Disease (1999) Medical College of Wisconsin – Healthlink.

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

Phillip Coffin. Research Brief: Cocaine and Pregnancy (1999) the Lyndesmith Centre – Drug Policy Foundation.

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

Murray A. Mittleman, M.D., et al., "Triggering of Myocardial Infarcation by Cocaine," Circulation, June 1, 1999,

Hannah Wunsch, "Cocaine Use Transiently Increases Heart-Attack Risk," The Lancet, June 5, 1999

Adnan I. Qureshi, MD; M. Fareed K. Suri, MD; Lee R. Guterman, PhD, MD; L. Nelson Hopkins, MD, "Cocaine Use and the Likelihood of Nonfatal Myocardial Infarction and Stroke", Circulation, January 2001

Brody SL; Slovis CM; Wrenn KD, "Cocaine-related medical problems", American Journal of Medicine, April 1990

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

Stocker S, "Cocaine Abuse May Lead To Strokes and Mental Deficits", NIDA NOTES, July 1988

Brookoff D, Rotondo MF, Shaw LM, et al, "Cocaethylene levels in patients who test positive for cocaine", Annual of Emergency Medicine, March 1996

Khalsa ME; Tashkin DP; Perrochet B. "Smoked cocaine: patterns of use and pulmonary consequences". Journal of Psychoactive Drugs, July - September 1992,

Burnett LB et al, "Toxicity, Cocaine" Emedicine Journal, July 2002

Baldwin G, "Cocaine Dramatically Accelerates HIV Infection", The Journal of Infectious Diseases, February 2002

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

Chavkin, W. "Cocaine and pregnancy: Time to look at the evidence". JAMA: Journal of the American Medical Association. 2001

Thurman, S.K., Brobeil, R.A., Duccette, J.P., & Hurt, H., "Prenatally Exposed to Cocaine: Does the Label Matter?" Journal of Early Intervention, 1994

Jaffe JH: Cocaine related disorders. Comprehensive Textbook of Psychiatry. Kaplan HI, Sadock BJ (eds). Baltimore, Williams & Wilkins, 6th Ed, 1995

Di Paolo N, Fineschi V, Di Paolo M, Wetly CV, Garosi G, Del Vecchio MT, Bianciardi G, "Kidney vascular damage and cocaine" Clin Nephrol May 1997
Marc L. Copersino, MA, Mark R. Serper, PhD. Co-morbidity of Schizophrenia and Cocaine Abuse: Phenomenology and Treatment, (1998) Hofstra University.

Cocaine Toxicity (1997) West Midlands Poisons Unit.

Dr Woody Hayes. Safe Handling of Ammonia (2000) Ohio State University.

Arnold M. Washton Cocaine Addiction: Treatment, Recovery and Relapse Prevention (1989) Norton.

The Blenheim Project. What's the Crack (1998) Blenheim Project Publications

LGC. LGC Drugs News. (2003) Issue 10

Special thanks goes to Canadian Harm Reduction Association; Lindsmith Policy Centre, USA; Gary Sutton, Release; Tony D'Agostino, COCA and Free Clinic, Antwerp.

Health References:

Murray A. Mittleman, M.D., et al., "Triggering of Myocardial Infarcation by Cocaine," Circulation, June 1, 1999,

Hannah Wunsch, "Cocaine Use Transiently Increases Heart-Attack Risk," The Lancet, June 5, 1999

Adnan I. Qureshi, MD; M. Fareed K. Suri, MD; Lee R. Guterman, PhD, MD; L. Nelson Hopkins, MD, "Cocaine Use and the Likelihood of Nonfatal Myocardial Infarction and Stroke", Circulation, January 2001

Brody SL; Slovis CM; Wrenn KD, "Cocaine-related medical problems", American Journal of Medicine, April 1990

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

Stocker S, "Cocaine Abuse May Lead To Strokes and Mental Deficits", NIDA NOTES, July 1988

Brookoff D, Rotondo MF, Shaw LM, et al, "Cocaethylene levels in patients who test positive for cocaine", Annual of Emergency Medicine, March 1996

Khalsa ME; Tashkin DP; Perrochet B. "Smoked cocaine: patterns of use and pulmonary consequences". Journal of Psychoactive Drugs, July - September 1992,

Burnett LB et al, "Toxicity, Cocaine" Emedicine Journal, July 2002

Gray A, "Freebasing Cocane", COCA Update, August 2001

Baldwin G, "Cocaine Dramatically Accelerates HIV Infection", The Journal of Infectious Diseases, February 2002

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

Chavkin, W. "Cocaine and pregnancy: Time to look at the evidence". JAMA: Journal of the American Medical Association. 2001

Thurman, S.K., Brobeil, R.A., Duccette, J.P., & Hurt, H., "Prenatally Exposed to Cocaine: Does the Label Matter?" Journal of Early Intervention, 1994

Jaffe JH: Cocaine related disorders. Comprehensive Textbook of Psychiatry. Kaplan HI, Sadock BJ (eds). Baltimore, Williams & Wilkins, 6th Ed, 1995

Di Paolo N, Fineschi V, Di Paolo M, Wetly CV, Garosi G, Del Vecchio MT, Bianciardi G, "Kidney vascular damage and cocaine" Clin Nephrol May 1997

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