Recreational Drugs

General

The acute use of alcohol and several other licit and illicit drugs can affect mental state and cognitive function. The chronic use of certain drugs may also increase the risk of cognitive impairment and perhaps dementia in later life. Currently available evidence indicates that mild to moderate alcohol consumption is not associated with increased risk of cognitive decline and may in fact have a protective effect against dementia, although heavy, long-term consumption is likely to have a negative impact on cognitive function. Longer-term smoking is associated with increased risk of cognitive impairment and possibly dementia. The chronic use of benzodiazepines has been associated with increased risk of cognitive impairment and the chronic use of cannabis may impair intellectual abilities(1) There is evidence that some drugs contribute to the causal pathway that leads to the development of cognitive impairment and perhaps dementia in later life but currently available data do not support the introduction of a separate diagnostic category of drug-induced dementia (such as alcohol-related dementia).

A new study suggests that for those who regularly use ecstasy or other recreational drugs, this kind of memory lapse is more common, uncovering potential links between memory deficits and cocaine for the first time (2). Memory tasks can be either time or event based, which means that the external trigger to remember could be in response to an event, or because it is time to do something. The distinction is important because these memory tasks use somewhat different brain processes. The researchers recruited 42 ecstasy/polydrug (muliple drugs used in combination) users (14 males, 28 females) and 31 non-users (5 males, 26 females) for the study -- all were students. The students were quizzed about their drug habits (including tobacco, cannabis, cocaine and alcohol), and given questionnaires to assess their everyday memory, cognitive failures and prospective and retrospective memory and then given a number of memory tests. The results showed that recreational drugs such as ecstasy, or the regular use of several drugs, affect users' memory functions, even when tests are controlled for cannabis, tobacco or alcohol use. Although ecstasy/polydrug users as a whole are aware of their memory problems they may be uncertain as to which illicit drug is behind the defects they perceive. The results suggest that these deficits are likely to be real rather than imagined and are evident in both time- and event-based prospective memory contexts. One interesting finding meriting further study was an association between recreational cocaine use and memory lapses.

Prospective memory performance is dependent on the brain's pre-frontal executive resources (executive functions involve planning, organization, and the ability to mentally juggle different intellectual tasks at the same time). A number of studies have shown in particular that event-based prospective memory tasks use the brain's frontopolar cortex, also known as the Brodmann area 10, or BA10, although scientists understanding of this region is still far from complete. (Present research suggests that it is involved in strategic processes in memory recall and various executive functions.) Event-based prospective memory tasks (prospective memory tasks are common in daily life and range from the relatively simple to extreme life-or-death situations) are also associated with the left superior frontal gyrus (RSG), which make up around a third of the brain's frontal area, and is linked with self-awareness. Time-based prospective memory tasks activate more diverse brain regions, including anterior medial frontal regions (linked to executive functions and personality), the right superior frontal gyrus (RSG) and the anterior cingulate (linked to many functions from heart rate regulation to cognitive functions and empathy). In addition, these time-based tasks also use the BA10 and the superior frontal gyrus as event based tasks do. The authors speculate that cocaine-related deficits observed on both the time- and event-based tasks might be due to cocaine's interaction with the BA10. Cocaine use could be associated with specific executive function deficits, which cause the prospective memory deficits. However, researchers have a difficulty when

investigating the drugs' effects on memory: which came first; the memory defects or the drug use? It is hard to rule out the possibility of pre-existing differences between users and non-users that originated before users tried drugs.

Another study has concluded that drug users show early Alzheimer's disease-related brain pathology that may be the basis for cognitive impairment and that neuroinflammation (*inflammation of the nervous tissuein the brain initiated in response to a variety of reasons eg.,infection, traumatic brain injury, toxins etc.*) is an early accompanying feature. This provides an opportunity to study the development of tau pathology in the human brain (3) (*Tauopathy belongs to class of neurodegenerative diseases associated with the pathological aggregation of* **tau** *protein in neurofibrillary or gliofibrillary tangles in the human brain*.) The most significant correlation for hyperphosphorylated tau deposition proved to be the level of microglial activation *(inflamatory response*). Whether changes in microglia are merely reactive to the rise in hyperphosphorylated tau, or to some other subtle pathology in drug-user brains, is unclear, but there is a parallel with Alzheimer's disease pathology (4) These data complement that of other investigators who have demonstrated cognitive decline in opiate abusers and who predicted that pathology in drug users is centred in the frontal and temporal cortices (5), and provide a pathological basis for these observations

Opiates Generally

Some medical experts have been suggesting that as the Baby Boomer generation ages we will begin to see alarmingly high rates of dementia. Experts suggest that the rampant drug abuse of the 1960s and early 1970s may be one cause of the increasing rates of dementia (12) Opiates are a class of drugs that includes morphine, heroin, codeine, Vicodin, oxycodone and Percodan. Medically, these drugs are used to control pain. When individuals use them for recreation or without regard for the doctor's instructions, addiction can occur. Opiates are extremely addictive. When abused they cause intense sensations of pleasure, which the addict is driven to repeat. They are also physically addictive and cause severe physical withdrawal symptoms if stopped suddenly. Many of the short-term health effects of opiate use are due to the fact that addicts will do anything to achieve the elusive "high." Because chronic use of opiates leads to physical tolerance, addicts need to use more and more of the drug each time, which can easily lead to a fatal overdose. The fact that the "high" becomes more difficult to achieve over time is what drives most opiate addicts to eventually administer their drugs by intravenous injections. Directly injecting the drug into the bloodstream will produce a more intense high than any other form of administration. Once an addict starts injecting drugs, the health risks of addiction skyrocket. Addicts will use dirty needles if clean ones aren't available, even though they know they can contract fatal infectious diseases like HIV, hepatitis and sepsis.

Regular use of opiates causes changes in the brain. Many regular opiate users become severely depressed due to the impact opiates have on the pain/pleasure centers of the brain. Some studies suggest that regular opiate users also suffer from significant cognitive impairment. Some opiate addicts have such severe cognitive impairment that they develop overt dementia. If these patients are hospitalized and taken off opiates, they usually regain some of their cognitive functioning. However, some of the brain damage may be permanent. In addition to direct damage to the brain from the drugs, addicts are at a very high risk of contracting HIV. HIV can cause dementia (AIDS-complex dementia). Severe nutritional deficiencies, such as vitamin B deficiencies, can also cause dementia. Addicts are often severely malnourished and are at risk of developing dementia from nutritional deficiencies. If caught early dementia due to nutritional deficiencies can be reversed, but it can become permanent.

Autopsy studies on deceased opiate addicts reveals that their brains are damaged, and the damage looks a lot like the damage associated with Alzheimer's (13). Individuals with early Alzheimer's have damage to the parts of the brain that are important for learning, memory and emotions, and these are the same areas that are found to be damaged by opiate abuse. The addicts in the autopsy study had an average age of 26. The researchers described the effect of the opiates on the brain as having prematurely aged the brain, making these young brains – and some of the addicts were as young as 17 – look like an 80-year-old's brain. Brains have a limited ability to heal. Once the damage is done, it is possible that even if these addicts enter rehab, they will always be at high risk of developing early-onset dementia or Alzheimer's disease.

One study that followed almost half a million men in Sweden from age 18 onwards measured the number that developed early-onset dementia. 487 of the men developed early-onset dementia at an average age of 54. The researchers studied possible risk factors that seemed to have contributed to developing early-onset dementia. Factors that were strongly linked to the development of early-onset dementia included alcohol and drug abuse.

Long-Term Health Effects Of Heroin And Opiate Addiction

Heroin and other opiates cause significant harmful changes in the body, and even for those who manage to outlive their drug use, the damage remains. Below are some of the long-term health effects of heroin and opiate addiction.

Memory Loss: Heroin in particular suppresses the brain's production of norepinephrine, an organic chemical that functions as both a hormone and a neurotransmitter. In the brain, norepinephrine is involved in the memory circuit. As the consumption of heroin suppresses the production of this vital chemical, it interrupts the memory circuit, decreasing the brain's capacity to receive, recall, and retain information. Long-term abstinence from heroin can restore these functions, though not always completely.

Impotence: Opiate abuse often results in diminished sex drive and impairs sexual behavior. Progressively more severe opiate addicts develop hypogonadism (diminished functional activity in the testes or ovaries), a condition that results in erectile dysfunction, menstrual irregularities, decreased libido, infertility, anxiety, and depression. Heroin impairs the brain systems that are activated during orgasm, making it impossible to achieve climax. Although stopping the use of heroin or opiates often restores sexual function, long-term abuse can impair it permanently.

Organ Failure: The use of heroin and opioids causes what's called "depressed respiration," or breathing that is slow, shallow, or irregular. This results in less oxygen to the body and reduced organ functioning. Additionally, some of the additives found in the drugs clog the small blood vessels leading to the organs, resulting in infection or tissue death in the vital organs.

Dementia: Chronic use of opiates often causes cognitive impairment to such a significant degree that it results in the development of dementia. In many cases, this impairment can be improved by stopping the drug use, but some of the brain damage may be permanent. Additionally, the nutritional deficiencies common in addicts put them at risk for developing dementia. Another significant finds in recent research is the link between depression and dementia. Although no conclusive evidence has been found as to why the two conditions are linked, these three theories continue to circulate:

- Depression may be an early sign of dementia
- Depression may damage the brain (by way of excess cortisol) and lead to dementia
- Depression may alter the brain and increase the risk of dementia

Alzheimer's Disease:Research has shown that brain damage in heroin and methadone abusers is strikingly similar to damage found in the brains of those with early Alzheimer's disease. The common areas of the brain affected by both Alzheimer's and heroin/methadone use are those involved with learning, memory, and emotional wellbeing.

Ecstacy (MDMA)

The recreational drug ecstasy, also known as “XTC” or “E,” Adam, X,Clarity, or Essence, is widely used by young people throughout the United States and Western Europe. It is a synthetic that can act as both a stimulant and hallucinogenic drug that enhances sensory processing, increases sexual sensations, and creates euphoric mood elevations in the user. The drug is an amphetamine derivative, with the pharmacologic name 3,4-methylenedioxymeth-amphetamine(MDMA). Its popularity has been enhanced by its close association with particular forms of music and dance venues and, despite well-publicized cases of MDMA-associated death, by the widely held belief that it is a“safe” drug. A review of the scientific literature, however, paints a very different picture of this drug, which is far from benign (6)

Evidence from human studies has accumulated more slowly, but it is becoming apparent that the toxic effect of MDMA on central serotonergic systems found previously in animal studies has a clear parallel in human users of the drug. There is now direct evidence of a lasting decrease in 5-HT uptake sites (a marker for the integrity of 5-HT nerve terminals) in human volunteers with a past history of MDMA abuse. Moreover, this decrease correlates positively with the extent of their self-reported previous exposure to the drug, and is in keeping with decreases in more general biochemical markers for central serotonergic activity reported elsewhere. (7). Positron emission tomographic (PET) imaging has revealed that the consequences of MDMA toxicity may be even more widespread than predicted from animal experiments. In addition to the hippocampal formation, both the amygdala and areas of neocortex may be affected byMDMA.

The manifestations of this neurotoxicity, in terms of altered cerebral function and behavioural change, range from deficits in verbal memory and reasoning, (8) to short-term memory (7) and visual memory (9). More general indices of intelligence are also adversely affected (10). The effects of MDMA on cognitive performance arising directly from drug-induced neurotoxicity may be compounded by indirect effects on the cerebral circulation. MDMA is known to cause major changes the level of neurotransmitters in the brain, such as serotonin and dopamine, which control our mood and behaviours. The role of serotonin in the body is to regulate mood, sleep, and stimulate or inhibit the release of particular hormones in the body. Amphetamines such as MDMA cause the release of serotonin in the body, creating a heightened sensory experience for the drug user.

It should not be surprising then that both acute and chronic treatments with MDMA produce cerebrovascular effects and it is evident that MDMA abuse is an important risk factor for cerebrovascular accidents (strokes) in young people. If these strokes are neurologically silent and subtle at first, they may only become apparent at a later date and develop into major deficits over the lifetime of an otherwise healthy individual. This effect may parallel the type of cognitive decline seen in patients with multi-infarct dementia. In particular, minor short-term deficits may be exacerbated by interaction with normal aging processes in the brain. The very scale of current usage - 3.4 million young Americans for example, have used the drug at least once - is such that the consequences of MDMA exposure may well develop into a **major health care problem** for the future.

In a Dutch study of 20 long-term Ecstasy users, all of whom were abstinent for at least two weeks before the study, and 20 non-users, researchers looked at three brain areas: the visual system lateral geniculate nucleus and Brodmann Areas (BA) 17 and 18 using fMRI (functional magnetic resonance imaging) scans during visual stimulation (11). The scans showed a direct, linear relationship between lifetime ecstasy use and activation in all three brain regions - the more ecstasy a subject had taken during their life, the greater was the activation seen in all three brain regions. This increased activation is interpreted as a sign of a less functional brain. This pattern of hyper-excitability is similar to that seen in studies of individuals at risk for or with early Alzheimer's disease. It does not

mean that ecstasy users are at risk of dementia, but that there's a loss of brain efficiency in both Ecstasy users and early Alzheimer's patients. This shift in cortical excitability may be chronic, long-lasting, and even permanent, which is a real worry. Another study found that in ten long-time Ecstasy users: their hippocampus measured, on average, more than 10% smaller than that of non-users The hippocampus is the area of the brain responsible for long-term memory. On average, the ecstasy users had not taken the drug for more than two months before undergoing the MRI scans, but had taken an average of 281 ecstasy tablets over the previous six and half years. The users also had an average 4.6 percent lower overall proportion of grey matter in the brain, which suggests that the effects of ecstasy may not be limited to the hippocampus.

Amphetamines; methylamphetamine (crystal meth)

Amphetamines are a group of drugs which vary in how powerful they are and how they are classified legally. The effects of crystal meth are similar to crack cocaine but they last longer. If you have experience of a mental health problem, you are more likely to experience negative effects. Long-term effects include agitation, confusion, aggression, psychosis and paranoia

Cannabis (marijuana, hemp, hashish, grass, skunk)

People take cannabis as a way of relaxing and getting high. The effects you experience will largely depend on whether you are used to taking the drug, how much you take, the type of cannabis you use and your genes If you have experience of anxiety and depression, you are more likely to experience negative side effects. This type of drug is a stimulant, depressant and hallucinogen. High doses may cause distorted perceptions, forgetfulness, distress, confusion and psychotic experiences (hallucinations or other unshared perceptions)

Cocaine, Crack Cocaine

Cocaine comes in two forms, cocaine powder, which is snorted and crack cocaine, which is smoked. Both forms may be injected. Cocaine is notoriously impure, and often contains other substances. Long-term effects include depression, anxiety, panic attacks, paranoia, irreversible brain damage, worsening of pre-existing mental health problems and repetitive movements. Cocaine is extremely addictive, and it is very difficult to stop taking it. If you have a mental health problem, cocaine can make this worse. intermittent use is addiction and is most prevalent in social situations.

Heroin (diamorphine)

Heroin is a painkiller, prescribed as diamorphine. The main effects are pain relief and euphoria but also depression. It is very addictive, and leads many people to crime to fund their use of it. The main problems with heroin arise because it is very addictive. Many drug treatment programmes are geared to helping people who are addicted to heroin and other opioid drugs.

Scientists at the University of Edinburgh (13) studied the brains of the deceased intravenous drug abusers of heroin and methadone and compared them to the brains of young people who were not drug users. The damaged nerve cells were in the areas of the brain involved in learning, memory, and emotional well-being, and were similar to damage found in the early stages of Alzheimer's disease. The study showed evidence of an increased risk of brain damage associated with heroin and methadone use, which may be highest in the young when individuals are most likely to acquire the habit. The brains of these young drug abusers showed significantly higher levels of two key proteins associated with brain damage. Tau protein, which in its soluble form is essential for communication

and transport within brain cells, had become insoluble in some cells, causing nerve cell damage and death in selected areas of the brain. Other nerve cells showed an accumulation of the amyloid precursor protein, which suggests that protein transport had been disrupted and the nerve cell functions affected. The study showed that drug abuse can lead to a build-up of proteins, which cause severe nerve cell damage and death in essential parts of the brain. The drug abusers in the study sadly died at a young age, but there are many others who don't realize the long-term effects that these drugs may be causing.

LSD (lysergic acid diethylamide, acid)

LSD is a synthetic drug that was first made in the 1940s. It causes random and sometimes frightening effects, known as a 'bad trip', which may be delayed. As LSD causes users to hallucinate and lose touch with their surroundings, it can cause users to do dangerous things (such as attempting to fly, for example). In some cases, people have died due to dangerous behaviour as a result of taking LSD.

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