What is addiction?

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Addiction is a modern disease, which is not always well managed by health professionals. This is particularly so when they have high expectations that most people with an addiction are cured as a result of formal interventions. When these cures are not forthcoming the professional can become frustrated, and a feeling of helplessness may descend upon patients and clinicians alike, fuelled by mutual distrust and judgement. This paper takes a long look at what addiction is, with the hope that a more caring, as well as a more realistic, approach to assisting people with addiction can be facilitated.

Diagnosis

Over the past 20 years diagnostic systems have been dominated by the American Psychiatric Association's *'Diagnostic and Statistical Manual'* (DSM). The DSM has undergone three revisions during this time; DSMIII,[1] to DSMIII-R[2] to DSMIV[3] to DSMIV-TR.[4] The most important shift for addiction was from the DSMIII to the DSMIII-R.[5] The diagnostic criteria for substance dependence in DSMIII-R (1987) represented a trans-Atlantic consensus on the concept, based on a landmark paper 10 years earlier.[6] Rather than view dependence as primarily based on whether there are physiological features, tolerance or withdrawal, the previous USA concept, the DSMIII-R (1987) description was a major concession by the Americans to European thinking that dependence was a syndrome that involved dyscontrol, salience and compulsion to use drugs, just as much as it involved tolerance and withdrawal; which essentially are features reflecting brain neuroadaptation to regular drug use.

Addiction has not been used as a diagnostic term in the past 20 years, being replaced in the major diagnostic systems – ICD9[7] and DSMIII (1980) – by the term 'dependence'. Adopting the term dependence collapsed the false dichotomy between physical and psychological addiction that had previously prevailed, but the term addiction never really went away. In fact, over the past 10 years or so the term has undergone a revival. This has been in significant part due to the neurobiological revolution that has brought us to the point of now being able to glimpse the brain pathology. Addiction has fitted the emerging understanding of brain pathology better than the term 'dependence', which has continued to be associated with the physiological features of tolerance and withdrawal; but moreover, patients have generally preferred the term 'addiction' over the rather limp sounding 'dependence'. The sound of the word 'addiction' (like 'cancer') alludes to something one should take immediate note of; something one should fear getting.

Addiction as an erosion of free will

The term addiction is derived from the Latin addictus referring to the relationship a slave had with his/her master – an enslavement. The question, *'How much "free will" does a person with addiction have?'* invites discussion about the degree of enslavement and non-autonomous, compulsive behaviour that patients with the disorder suffer from. This is important information that will break down the long-held, erroneous view that addiction is simply a self-inflicted disorder of weak-willed people and therefore any suffering on their part can be discounted and conveniently ignored. As information accumulates, it is becoming clearer that addiction is more like other serious psychiatric conditions, such as

schizophrenia, than we ever previously thought, in which a person's behaviour has become driven by disordered brain processes diminishing their normal ability to consciously plan and guide rational, adaptive behaviour.

Using the standard continuum of drug use, from 'no use' through 'safe use', 'hazardous use', 'problem use' and 'dependence' it is clear that dependence in itself might include mild degrees of enslavement in which a person's 'free will' is still largely intact. Measurement of the intactness of executive functions in this regard is still in its infancy and certainly not available for clinical use at the present time. However, a person may meet the necessary three of the seven criteria of dependence (DSMIV-TR 2000) (see Table 1), be therefore diagnosed with dependence but will not have 'addiction' in terms of serious erosion of 'free will' that is becoming part of the new conceptualisation of addiction. A useful addition to the standard continuum of drug use is to divide 'dependence' into 'mild dependence' and 'moderate-severe dependence' to make this distinction. Addiction is then equated with 'moderate-severe dependence' to indicate a disorder in which a person's 'free will' has been eroded to the extent that a threshold has been reached beyond which the likelihood of a person responding with compulsive drug seeking behaviour to associated cues is high.

Table 1. Diagnostic criteria for substance dependence (based on DSMIV-TR) mapped onto four key elements of addiction – dyscontrol, salience, compulsion to use, physiological features

Dyscontrol

1. Substance is often used more than intended.
2. Unsuccessful attempts to cut down or control use.

Salience

3. Much time is spent in substance use.
4. Important activities are given up or reduced.

Compulsion to use

5. Continued substance use despite knowledge of associated medical or psychological problems.

Physiological features

6. Acquired tolerance.
7. Withdrawal symptoms and/or relief use.

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The psychology of addiction

Twenty years ago addiction was conceptualised to be the result of two fundamental psychological processes. The brain was largely uncharted territory in the minds of addiction clinicians. The first process was positive reinforcement in which a person is likely to repeat a behaviour that is rewarding, in this case drug use. A person repeatedly uses drugs because of an anticipated enjoyable 'high'. However, once the person continues to regularly engage in drug use, adaptation occurs and then sooner or later, in the absence of the drug, they begin experiencing withdrawal symptoms. Subsequent drug use relieves the discomfort of withdrawal symptomatology. This is negative reinforcement; using the drug to avoid the negative consequences of abstinence and feel normal. The primacy of withdrawal symptomatology in the concept of dependence 20 years ago was a reflection of this psychological process being viewed as central to addiction. Interestingly, this concept of addiction still has wide currency in wider health, and indeed educated public circles, that essentially people with addiction continue to use drugs primarily to avoid withdrawal symptoms. If this was true then the treatment of addiction would be relatively easy; it would primarily consist of a managed withdrawal programme, detoxification. However, the rate of relapse following withdrawal in people with moderate-severe dependence is high, about 95% within two years of detoxification (alcohol and opioid dependence).[8] The theory doesn't accord with reality.

Although positive and negative reinforcement remain as key elements in the progression of voluntary drug taking to a state of addiction, they are limited in themselves in explaining the increasing autonomous nature of drug seeking behaviour by people with addiction. About 10 years ago, appearing through the academic mist was increasing reference to changes in the brain consolidating cue-based associated learning[9] and this has been the key to understanding what addiction is from a psychological perspective; and most importantly, what has helped explain why addiction is, relatively, so hard to treat.

There is still a significant gap between knowledge of addiction from a psychological perspective and what is known from a neurobiological perspective. However, in terms of the latter, there have been major strides made over the past 20 years coming out of basic neuroscience. Three main strands that together begin to form a coherent picture are:

- the evolution of the human brain;
- neural pathways focused around the reward pathways; and
- the nature of consciousness.

Evolution

The vastness of time is the key to understanding the extraordinary process of evolution through the process of natural selection in which *'...each slight variation, if useful, is preserved'*.[10] There are only a few like Steven Hawking who appear able to grasp 13.7 billion years of the space/time continuum.[11] Complex life (post-Cambrian) has provided about 550 million years for the 'blind watchmaker' to do the business[12] and produce the glorified virus known as homo sapiens. We may not be able to run faster, see further, or live longer than other animals, but we can think and anticipate the future better than other animals. This thinking ability has given us an overwhelming biological advantage here on planet Earth, at least in terms of other animals; viruses and bacteria remain by far the most enduring life form.[13]

The human brain consists of the cognitive processes of a fish embedded in a reptile, wrapped in a mammal, overlaid by a primate. We are top of the tree for primate thinking ability in terms of intuition, creativity and presumed consciousness. The tension between the instinctual reptilian core of our brains and the much newer overlay of neocortical functioning is at the basis of a neurobiological understanding of addiction.

Neural pathways

An early paper[14] titled *'Seeking drugs/alcohol and avoiding withdrawal: The neuroanatomy of drive states and withdrawal'* provided an exciting vision of future directions in knowledge of what addiction is from a neurobiological perspective. The authors wrote: *'Drug users feel as if they have acted to preserve the species, when in reality they have simply bypassed the normal behaviour reinforcement system'*. In other words, they were suggesting that people addicted to drugs, use drugs as if their lives somehow depended on it; that the process of addiction had hijacked the brain's mechanisms for mediating survival instincts, into the alternative service of drug taking.

Just how this process of addiction occurs, whereby voluntary drug use for pleasure is replaced by an involuntary compulsion to use drugs out of a distorted yet deep instinctual sense of need, remains a ripe topic of neurobiological research. However, the brain pathways involved in the process have certainly become increasingly clear, for example as described by Ronald Hammer.[15] The nucleus accumbens is placed at the centre as an integrating centre within the limbic system receiving dopaminergic inputs from neurons ascending from the ventral tegmental area of the midbrain. Dopaminergic neurons also ascend from the midbrain through to the prefrontal cortex. These dopaminergic neurons are considered the fundamental aspect of the brain's reward pathway. The nucleus accumbens also receives inputs from the hippocampus (memory) and amygdala (emotions) and once these and no doubt other inputs are processed in the nucleus accumbens, the activated output is initiation of drug seeking behaviour. The important point is that this initiation of drug seeking behaviour is from the limbic system, outside of consciousness.

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Consciousness

Consciousness is the link with the earlier question: *'How much "free will" does a person with addiction have?'* Benjamin Libet and colleagues undertook a series of experiments in the early 1980s in which subjects' conscious awareness of making a decision was detected against the appearance of readiness potentials measured by EEG, and found that there was a delay of about half a second between an unconscious 'decision' to act in response to an external stimulus and the development of consciousness of that decision.[16] In other words they demonstrated that we are all living (consciously) about half a second behind what we have already decided to do. Free will is essentially an illusion. However, we are used to living as if we have free will. Our conscious prefrontal cortex with its sense of being in control fills in the gaps and justifies 'decisions' we make unconsciously. Continuous spin doctoring by our observing prefrontal cortex provides psychological equilibrium. We retain the capacity to change direction once a certain course of action has been embarked upon unconsciously, but retrospective decision-making involving a split second conscious admission that a mistake has been made, is required. Applying Libet's discoveries to addiction may be critical to understanding addiction. The 'decision' to take drugs in an addicted state is made unconsciously, utilising the most powerful machinery of the brain for ensuring survival, taken over for the purpose. Consciousness kicks in half a second later allowing the person only a split second (about one tenth of a second) to admit the mistake and 'put the brakes on'. When the force behind the action is being experienced as if it were a life and death one, as it seems to be experienced by an addicted person, the likelihood of continuing with drug seeking behaviour is great. Addiction drives an autonomous state that is an exaggeration of the normal autonomous state we all are in. The belief in having free will, being in control, and initiating behaviour consciously, appears to be one of the great delusions of the human mind. We may have a limited degree of free won't,[17] but the human capacity to exercise free will is a concept in need of revision.

What causes addiction?

Evidence for a genetic influence in addiction has been well established now for over 20 years, the starting point being that alcoholism, as the prototypical addiction, was clearly found to be a familial disorder; it ran strongly in families. Even 20 years ago there was a substantial literature of various adoption and twin studies of alcoholics, attesting to the strength of a genetic component in aetiology.

One of the most fascinating elements of the story about what causes addiction has been the inbreeding experiments of rats. T K Li was an early leader in this research[18] in which animals were developed that not only preferred to drink an alcohol solution over water (alcohol preference), but liked to drink a lot of it (high volume vs low volume consumption). Selecting for interest in alcohol and then preference for alcohol over a series of successive generations can produce a rat that, despite not having been abused in early life or otherwise exposed to dysfunctional formative experiences, nevertheless 'likes' to drink a lot of alcohol and will spontaneously drink alcohol through to achieving cirrhosis of the liver. These rats develop tolerance to alcohol, will go into a withdrawal syndrome when deprived of alcohol following regular use and will exhibit 'relief drinking' (negative reinforcement) when provided with alcohol once again. It has been subsequent experimentation with these rats and other animals that has assisted human advancement of knowledge about alcohol and other drug use and addiction, most importantly providing data for the development of effective pharmacotherapies for treating addiction, beginning with naltrexone for alcohol dependence.

Search for the genes

The heritability of addiction has been known to be in the region of 50% for several decades, at least as much as the heritability of IQ and Type 2 diabetes. About half of this vulnerability appears to be a generalised genetic factor and half specific for the particular addiction under consideration, at least for alcohol, cannabis, cocaine and nicotine.[19] Over this time, there has been a vigorous search for the actual genes that confer the various aspects of the genetic vulnerability to addiction. The genes that provide the recipe for creating a human being derive from very early times. Habit formation is a specific cognitive ability in fish, a life form which speciated in the Ordovician period, about 500 million years ago,[20] and so it can be said relatively confidently that at least some of the genes associated with addiction (compulsive habit formation) are 500 million years old.

A key development in the understanding of genes has been the interaction of genes with the environment.[21] Genes are being constantly turned on and turned off, like light switches, in response to environmental events. Sir Francis Galton's famous phrase of more than 100 years ago, *'nature versus nurture'* is now collapsed into *'nature and nurture'*, a gene/environment interactional entity. No longer is the exploration focused on finding the genes, but finding which environmental influence or influences interacting with which gene or genes, contributes to the aetiology of addiction.

Why use the term 'addiction'?

First it is a favoured term by people with the disorder because it sounds like what they are suffering from. Second, it is the favoured word by those working in neurobiology to denote there is more to this disorder than tolerance and withdrawal, the traditional features of dependence. The third reason is more pragmatic. It is a simple word that can be used service-wise to denote a wide range of disorders and therefore be inclusive, not only of the various substance use disorders, including nicotine dependence, but can also be used to include the range of compulsive consumptive behavioural disorders, that are increasingly referred to as behavioural addictions. Pathological gambling is the most scientifically validated of these to date but also under active investigation are compulsive buying, addictive overeating, sexual addiction and others. If, in time, phenomenology, neurobiology, aetiology, clinical course and treatment are demonstrated to be similar then it is likely that the diagnostic systems will substitute addiction for dependence and incorporate both compulsive substance use and compulsive consumptive behaviours under an addiction diagnostic umbrella.

Conclusion

Addiction is unique to the human species and results from ancient genes interacting with modern human environments. These ancient genes originate from at least the time of the Ordovician Period (~500 million years ago) when fish evolved with the capacity of habit formation. Modern human environments feature technological advances such as the distillation of alcohol, tailor-made cigarettes, hypodermic needles, electronic gambling machines and intensely hedonic food. These can each provide a compelling stimulus to an integrating structure in the 'reptilian' part of the human brain, called the nucleus accumbens, which initiates automatic responses to seek and consume. If continued regularly, these compelling stimuli spark the development of compulsive behaviour in the individual, initiated outside of consciousness. Addiction runs a chronic relapsing course in the majority of sufferers. Commercialisation of addictive products in consumption based economies, fuels the problem of addiction in modern societies, our contemporary human environment.

A deeper understanding of what addiction is (hopefully) facilitates greater empathy for people with the affliction. It also provides a solid starting point for appreciating that addiction generally presents as a chronic relapsing disease and why it is difficult to cure.[22]

Competing interests

None declared.

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Missed opportunity

'The most troubling finding of the survey is that 94 percent of primary care physicians (excluding pediatricians) failed to include substance abuse among the five diagnoses they offered when presented with early symptoms of alcohol abuse in an adult patient.

Many of the other findings are also disturbing:

- *Most patients (53.7 percent) said their primary care physician did nothing about their substance abuse: 43 percent said their physician never diagnosed it, 10.7 percent believe their physician knew about their addiction and did nothing about it.*
- *Less than one-third of primary care physicians (32.1 percent) carefully screen for substance abuse.*
- *Only one in five (19.9 percent) primary care physicians consider themselves "very prepared" to identify alcoholism, only16.9 percent consider themselves "very prepared" to spot illegal drug use, and 30.2 percent consider themselves "very prepared" to spot prescription drug abuse.*
- *Most patients (54.8 percent) agreed that physicians do not know how to detect addictions.*
- *Most patients (54.5 percent) say that doctors prescribe drugs that could be dangerous to addicted individuals. Thirty (29.5) percent of patients said their physician knew about their addiction and still prescribed psychoactive drugs such as sedatives or Valium.*
- *The average patient was abusing alcohol, pills and/or illegal drugs for 10 years before entering treatment.*
- *Three out of four patients (74.1 percent) said their primary care physician was not involved in their decision to seek treatment and 16.7 percent said the physician was involved only "a little."'*

National Survey of Primary Care Physicians and Patients on Substance Abuse Conducted by the Survey Research Laboratory, University of Illinois at Chicago; April 2002. Available at: http://www.casacolumbia.org/Absolutenm/articlefiles/29109.pdf