ADDICTION DEFINED 1

CHAPTER OVERVIEW

This chapter will initially provide a brief overview of what addiction is currently understood to mean, and how this meaning has evolved over time. It will then describe the various constituent components of addictive behaviour, and how this behaviour is influenced by interacting biopsychosocial factors. Finally, the psychiatric classification systems that are clinically utilised to diagnose addiction will be described and considered.

This chapter will:

- Outline the use of terminology in addiction
- Outline the components of addiction
- Discuss factors contributing to addiction
- Describe diagnostic nomenclature

LEARNING AIMS OF THE CHAPTER

- To understand factors in addiction
- To appreciate the elements of diagnosis and the use of different diagnostic systems
- To understand the main components that the addiction concept is comprised of

WHAT IS ADDICTION?

First consider these three illustrative people:

CASE 1

Cynthia is a 55 year old woman. She grew up in a household where both parents were regular heavy drinkers. Cynthia was bullied at school and lacks confidence in herself. She is divorced and lives alone, and supports herself by working in a low paid administration job. She has three grown up children who rarely visit, and a strained distant relationship with her elderly mother who lives in the area. Cynthia is a daily drinker, and wine is her drink of choice. She feels nervous if she thinks her supplies are running low. She drinks roughly three quarters to one bottle of wine a day. She has a few friends that she goes drinking with a couple of times a month, but the rest of the time she drinks alone. Cynthia is able to turn up for work on time and carry out the duties required in her job, although she sometimes makes minor errors. She is often moody, and snaps at her work colleagues if she is challenged about her work. On occasional nights out with friends or colleagues she can easily consume three bottles of wine over the course of the evening. She feels tired and overwhelmed by life sometimes. Her children worry about her drinking and have tried to persuade her to cut down. Cynthia considers her alcohol use her own business and completely acceptable. She would not dream of ever touching cannabis, cocaine, ecstasy, heroin or any other 'drug'. She considers these other drugs as dangerous and unacceptable and has little sympathy for their users. She would never consider seeking help for reducing her alcohol intake, and does not consider that drinking may have contributed to other problems in her life.

CASE 2

Michael is a young man aged 23. He started to use alcohol and other drugs around age 14. Michael is shy and socially awkward. He has always felt different from other people, and finds it hard to communicate his feelings. Alcohol and other drugs alter his mood and help him to feel accepted by his peers. However, Michael is unable to control his drink and drug use. He has ended up in hospital Accident and Emergency departments several times as a consequence of excessive use. He is well aware that his substance use causes significant problems in his life, and his own pattern of use differs from the way his friends use substances. Although he has sought professional help for his problems, and has a loving and supportive family, he has so far been unable to prevent the repeating cycle of abstinence and relapse that has dominated recent years. Michael fights what he describes as a constant battle with the side of himself that wants to use and the side that wants to stop. This struggle drains his energy and makes him feel tired and depressed. Sometimes he contemplates suicide as a way to end this struggle. He finds it difficult to tell new people that he meets about his problems with alcohol and other drugs for fear of being distrusted and judged. As a young person living in a society in which the majority of socialising involves alcohol use, or takes place in venues where alcohol is readily available, he finds it very difficult to put this problem out of his mind. Even if he switches on the television or opens a magazine he is exposed to frequent images and references to alcohol.

CASE 3

Peter is a 35 year old marketing executive living in London. He grew up with a father who was a cocaine and heroin addict, and a mother who was twenty years younger and had severe mental health problems. Although his parents were not deliberately abusive, he was neglected regularly, and was exposed to a world of crime and drugs from a young age. As a child he developed an extrovert and entertaining persona as a way to gain attention and avoid the feelings of disconnection he experienced. Peter has used cocaine, amphetamines and alcohol to varying degrees, since around age 13. Despite his outwardly happy and outgoing persona, he feels deeply out of place in the world, and suffers from strong feelings of self-loathing. He uses substances to manage and numb these uncomfortable feelings. He also frequently gambles, seeks out casual sex and uses prostitutes, for the same reasons.

Peter has many acquaintances but no close friends. He has never had a stable relationship, though he has had many one-night stands. He tells those around him this is a lifestyle choice, but deep down he feels lonely and ashamed at his inability to connect with the world around him. Although his substance use has led him to be erratic and unpredictable at times, natural ability and charm have allowed Peter to do reasonably well at work. He has drunk alcohol

and used drugs at work for many years, however in his line of work people doing this is not entirely uncommon. Recently his substance use has escalated, and he is regularly not turning up to work at all, and risks losing his job. Peter knows he has a problem. He wants to stop his addictive behaviours, but he does not know how to live in any other way.

We have included these three cases (loosely based on actual people with personal details altered) at the start of this chapter because they illustrate some of the complexity around defining addiction. Subjective judgements are involved both on the part of the addicted individual and others, and these judgements cannot fail to be influenced by peer group and societal norms.

Come back to consider the following questions in relation to some or all of these three cases when you have finished reading this chapter and the other chapters in this book.

- Do you think Cynthia is addicted to alcohol?
- Are other people affected by these individuals' substance use?
- Do you consider Cynthia's attitude to alcohol use compared to the use of other substances to be reasonable? If so why?
- Where have Cynthia's opinions and assumptions about substance use originated? Where have your own opinions come from?
- Do you think that genetic vulnerability and/or an underlying psychiatric disorder may be influencing substance use in these particular cases? Why do you think so?
- What are the similarities and differences between Cynthia's, Michael's and Peter's substance use?
- Who do you think stands a better chance of recovery?
- How much responsibility should society take for the 'choices' made by Cynthia, Michael and Peter? Who is to blame? Where does responsibility lie?

The term 'addiction' is defined according to the historical, social and cultural context in which it is used, and it can be defined in slightly different ways for different purposes. The meaning and assumed implication behind the use of this word have also changed throughout history (Alexander, 2008). At various points in time, addiction has been thought of as a moral weakness, a medical condition, a socially determined behaviour, and the product of a dysfunctional brain (Rehm, 2014). There is still no clear consensus on the definition of the word because it involves both objective and subjective elements. Addiction is generally understood to mean a loss of self-control and compulsion with regard to an activity or substance.

In the current context, addiction can be defined as:

> A repeated powerful motivation to engage in a purposeful behaviour that has been acquired as a result of previously engaging in that behaviour, with significant potential for unintended harm. (adapted from West and the EMCDDA, 2013)

This definition manages to define addiction, but does not try to explain or describe the underlying mechanisms and associated symptoms, which can vary in their presence and severity between individuals and between different addictive agents (e.g. behavioural addictions and substance addictions).

Addiction can refer to the pathological consumption of alcohol, tobacco and other drugs, but can also encompass other behaviours, for example, compulsive eating, gambling, shopping and internet use. There is evidence for some underlying commonalities in the neurotransmitters and brain circuits involved in some substance and non-substance related behavioural addictions such as gambling (which will be explored in Chapter 8), however, throughout this book we will be primarily referring to drug addiction (*inclusive of alcohol addiction*) unless otherwise stated.

Consider the American Society of Addiction Medicine's (2018) definition of addiction, which is described in their Public Policy Statement (released April, 2011) as follows:

> Addiction is a primary, chronic disease of brain reward, motivation, memory and related circuitry. Dysfunction in these circuits leads to characteristic biological, psychological, social and spiritual manifestations. This is reflected in an individual pathologically pursuing reward and/or relief by substance use and other behaviours.
>
> Addiction is characterized by inability to consistently abstain, impairment in behavioural control, craving, diminished recognition of significant problems with one's behaviours and interpersonal relationships, and a dysfunctional emotional response. Like other chronic diseases, addiction often involves cycles of relapse and remission. Without treatment in recovery activities, addiction is progressive and can result in disability or premature death.

This definition of addiction acknowledges what has been learned about the underlying neuroanatomical, neurocognitive and neurochemical basis of addiction through scientific research in recent decades. This definition could be interpreted as reductionist if it is applied without thinking about the influence of the wider socio-cultural context within which it was

originally formulated and is currently placed. This definition is widely influential in the addiction field.

It is worth thinking critically about how this definition is worded. Dysfunctional brain circuitry is described as leading to the various manifestations of addiction. However, although dysfunctional brain circuitry is clearly involved in addiction, the interrelationships between contributory factors to addiction are not clear-cut. There is also an inherent danger in defining addiction as a disease in that it overemphasises the biological explanation, at the expense of psychosocial explanations, and consequently may adversely influence the formulation of effective treatment strategies. It has been argued (Booth Davis, 1997; Peele, 1985; Pickard et al., 2015; Satel & Lilienfeld, 2013) that the 'brain disease' model of addiction obscures the dimension of personal choice, the capacity to respond to incentives, and the fact that people use drugs for often valid reasons, such as self-medication of emotional states, or to tolerate the otherwise intolerable aspects of themselves and/or their lives. It also does not explain or encompass factors that influence treatment-free recovery. There is no doubt that defining addiction will remain a controversial area because it has at its core the concept of personal responsibility (and its perceived erosion).

Different explanations of addiction can be simultaneously accurate and compatible with each other. Seemingly divergent aspects of addiction are worthy of exploration and potentially useful in aiding overall understanding of this condition and its variation both between individuals and within individuals. Addiction is best understood from an interdisciplinary perspective and examined at multiple levels of explanation, from the biological through to the societal.

COMPONENTS OF ADDICTION

There are certain features which are traditionally associated with addiction and it is thought that an individual will repeatedly cycle through these stages which are outlined below:

- Initial use, which is usually associated with pleasure and reward
- Escalating use/binge/intoxication
- Increasing dependence, tolerance, and loss of control
- Adverse effects if substance use or activity ceases or is prevented (including physical withdrawal effects)
- Craving for the substance or activity
- Relapse to substance use and repetition of behaviour

The addictive cycle is initially driven by impulsive use, and over a period of time the behaviour becomes increasingly compulsive. Initial use is often associated with short-term pleasure and reward, whereas as addiction takes hold over time; behaviour becomes increasingly motivated by alleviation of the discomfort associated with abstinence.

Much of what was originally understood about addiction came from early research on heroin and alcohol addiction, and much has been learned subsequently, with regard to addiction to these, and other drugs. This early research led to a definition of addiction which focused on the ability of a substance to produce physical withdrawal symptoms. When a person takes a substance for a period of time their body adapts to the substance and they become physically dependent on the substance to feel 'normal'. Alcohol and heroin both result in distinctive and identifiable physical symptoms when their regular heavy use is discontinued. For example, the effects of heroin include euphoria, constipation and relaxation. Habitual users of heroin become physically dependent on the drug and develop what is known as *tolerance* to some of the drug's effects. Drug tolerance is when larger and larger amounts of the drug are needed in order to experience the same effects.

This is caused by compensatory mechanisms that occur in the body as a result of drug intake and are the body's attempt to achieve homeostasis (maintain a biologically optimum steady state). These compensatory mechanisms are opposite to the effects of the drug itself, so when drug use is abruptly discontinued these effects are experienced acutely and are felt as *withdrawal* symptoms.

In the case of heroin use, withdrawal is experienced as agitation, diarrhoea, vomiting and anxiety, amongst other symptoms, which are opposite to some of the drug's effects. The severity and length of the withdrawal syndrome will vary according to drug type, individual difference factors, drug dose and duration of use.

It was originally believed that the drug addict was driven to take the drug to alleviate these unpleasant withdrawal symptoms, resulting in a feeling of physical *craving* (a powerful feeling of longing for, or 'wanting') which would drive further drug-seeking and drug-taking behaviour. Although alleviating unpleasant withdrawal symptoms undoubtedly contributes to drug addiction, it does not explain why relapse to drug use often occurs a long time after the physical withdrawal symptoms cease. It is now thought that physical withdrawal, although unpleasant, is not what drives addictive behaviour. Many addictive drugs such as cocaine, marijuana and amphetamines are highly addictive in the absence of prolonged or severe withdrawal symptoms, and these drugs have therefore been described as psychologically addictive. Physical and psychological withdrawal can be important components of addictive behaviour but other factors are also involved.

The exact mechanisms underlying the addictive process are not well understood, and the study of addiction is complicated by the fact that it encompasses the study of the harmful effects of many different drugs, with both overlapping and distinct modes of

action. Although the contribution of differing factors varies between individuals and with different drugs, research evidence has identified that different drugs of abuse also share many common features at the neurobiological and behavioural levels (Kelley & Berridge, 2002).

THE ROLE OF COGNITION IN ADDICTION

Cognitive processes such as attention, perception and memory, both explicit and implicit, are considered to be central to drug addiction (Baker et al., 2004; Tiffany, 1990), and cognitive control can be recruited to either support or inhibit drug use (Curtin et al., 2006). One of the commonalities of addictive drugs is their ability to invoke reactivity to environmental drug cues. Cue reactivity is the array of responses that are observed when drug users or former drug users are exposed to stimuli previously associated with drug effects. These cues could include particular people, places, or drug-associated behavioural rituals, for example, always lighting a cigarette whilst talking on the phone, or always having a drink with a smoke. Over time using the phone would automatically cause a craving for a cigarette through repeated association, and having a drink would cause a craving to smoke. Cues could also include particular emotions associated with drug taking such as low mood, depression and anxiety. Associative learning (involving perception, attention and memory processes) is thought to play a crucial role in drug craving and relapse to drug use in the absence of acute physical withdrawal. This is proposed to occur through the mechanisms of classical and instrumental conditioning to drug cues. These drug associated cues can themselves elicit powerful neurophysiological effects in the absence of the drug itself.

Several theories of addiction propose that reactivity to drug-related cues is an important feature of drug dependence. The incentive-sensitisation theory (Robinson & Berridge, 1993) incorporates associative learning but places particular emphasis on the *sensitisation* of brain motivational systems to drugs and drug associated cues. Sensitisation is a process whereby, over repeated exposure, a greater effect is observed. Addictive drugs produce both positive and negative reinforcement effects on behaviour depending upon at which stage of the addictive cycle they are taken. Reinforcement refers to the effect that a particular stimulus has on a behaviour that preceded it, and it means that the behaviour is more likely to be repeated. That behaviour is effectively strengthened. This is particularly true for behaviour resulting in immediately rewarding effects, and drugs that produce an immediate 'high' (or other pleasurable effect) are more positively reinforcing and addicting (Volkow et al., 2000). Negative reinforcement occurs when an unpleasant stimulus is removed as a result of the

preceding behaviour. In the case of drug consumption, this can be escape from an unpleasant emotional state, such as low mood, or from unpleasant psychological and/or physical withdrawal symptoms. Negative reinforcement is considered to be an important factor in driving drug use, craving and relapse (Baker et al., 2004).

WHICH PARTS OF THE BRAIN ARE INVOLVED IN ADDICTION?

Research on addiction has shown that addictive drugs can interact with, subvert and alter neural circuits in the brain which are associated with naturally rewarding motivated behaviours such as eating, drinking and sexual activity (Kelley & Berridge, 2002). These behaviours have evolved to be motivationally potent to ensure survival. In particular, the mesotelencephalic dopamine system is implicated in mediating the pleasurable effects of drugs.

The release of the neurotransmitter dopamine is thought to occur in response to nearly all addictive drugs, and also in response to drug-associated cues. Large and rapid increases in dopamine in the limbic system are involved in the initial reinforcing effects of most drugs of abuse. Research has focused on dopamine, but other neurotransmitter systems including serotonin, glutamate and GABA are also known to be important in the transition to addiction. Neuro-adaptive changes resulting from chronic drug use may result in changes in dopamine receptor levels which drive further drug consumption. Recent research using a neuroimaging technique has shown that individuals at high familial risk of addiction show a blunted dopamine response following a dose of amphetamine, suggesting a possibility that this diminished response either develops early in drug use history or is a pre-existing familial vulnerability trait (Casey et al., 2014).

Evidence from neuroimaging studies has greatly enriched understanding of brain regions and processes involved in addictive behaviour (Goldstein & Volkow, 2002; Volkow et al., 2003). The findings from both human and pre-clinical research have shown that much of the neural circuitry involved in addiction is involved in motivation, the processing of rewards, and in reward-related decision making and impulse control (Bechara, 2005; Kalivas & Volkow, 2005). Circuits necessary for insight and social behaviours are also affected, resulting in addicted individuals making poor behavioural choices despite awareness of their negative consequences (Forbes & Grafman, 2010; Volkow et al., 2011). Disruption of this circuitry is thought to result in the development and maintenance of addiction, with different addictive substances influencing different parts of this circuitry (reviewed by Reid & Lingford-Hughes, 2006).

WHAT FACTORS INFLUENCE THE TRANSITION TO ADDICTION?

Of the large numbers of people who are exposed to, experiment with and regularly use addictive drugs, it is only a small minority in whom drug use escalates to addiction. There are many factors that interact to increase or decrease the likelihood that an individual will become an addict, but there is no clear causal pathway.

Risk factors for addiction vulnerability include some of the factors considered below that *interact with each other* to a greater or lesser degree and with the pharmacological properties of the drug itself. Ultimately addiction will result if the factors that confer addiction vulnerability outweigh the factors that may protect against it. This list is not exhaustive and these and other factors influencing addiction vulnerability will be considered in more detail in subsequent chapters in which theoretical models of addiction will be examined.

GENETIC PREDISPOSITION

There is no single gene that predisposes a person to addiction. As addiction can involve numerous different substances and behaviours, any genetic influence is a result of differences in multiple genes. For example, genes may influence pharmacological factors such as individual differences in the regulation of enzymes involved in drug metabolism. This may indirectly affect how sensitive an individual is to a particular drug effect and how it is subjectively experienced, and hence its addictive potential for that individual. Genes affecting individual differences in the expression of dopamine receptors and other neurotransmitters and their receptors may also influence addiction vulnerability in this manner.

The genetics of addiction have been studied using family, adoption and twin studies, and the role of heritable influences has been estimated for different substances and also across substances. For example, the variation in liability to nicotine dependence has been estimated at between 33–71% (Edwards et al., 2011; Kendler et al., 1999; Lessov et al., 2004; Lyons et al., 2008) and alcohol dependence at between 48–66% (Heath & Martin, 1994; Heath et al., 2001; Kendler et al., 1992). Another study (Merikangas et al., 1998) has shown that the percentage of relatives with a substance use disorder is approximately eight times higher in those related to an addicted person compared to those related to a non-addicted person.

DEVELOPMENTAL FACTORS

Developmental factors are important components of addiction vulnerability and there is increasing evidence that the adolescent brain is a critical period of heightened vulnerability to any

environmental insult, including exposure to potentially neurotoxic drugs. This is because adolescence is a developmental period during which significant physiological, psychological and behavioural changes occur, including puberty, and its associated hormonal and emotional challenges.

The adolescent brain is still extensively developing and maturing which may make it particularly sensitive to drug effects and also to social and environmental pressures. In adolescence synaptic connections undergo a 'pruning' process, increased myelination of nerve cells occurs, and the communication between key regions of the brain is enhanced and becomes more efficient. Importantly, the frontal lobes (and their connections) – which are critical to complex thought, decision making and inhibition of more impulsive behaviour – are still undergoing development. This developmental period coincides with an increased desire to engage in and ability to access what are perceived as more adult social behaviours, including drug use. Increased peer-directed social interaction and influence typify the teenage years, and increased rates of impulsivity and risk taking occur in adolescence (Spear, 2000). Increased sensitivity to drug reward and a decreased sensitivity to drug withdrawal also occur at this time (O'Dell, 2009). Disruption of the brain circuits involved in behaviour control by addictive drugs during this critical developmental period may cause lasting damage, which increases the risk for the onset and maintenance of mental health problems, including addiction.

Most research on the developmental effects of drug use has been carried out on alcohol and nicotine, because these addictive drugs are legal in most societies and so are more easily accessible to teenagers and are usually the first drugs they try.

Recent longitudinal studies on adolescent alcohol use suggest that there are both pre-existing differences in the brains of those who later engage in heavy alcohol use and also damaging effects of this use. This has been demonstrated for working memory (Squeglia et al., 2012), response inhibition (Wetherill et al., 2013) and brain regions impacting on behaviour control, language and spatial tasks (Squeglia, Rinker, et al., 2014). A recent study (Squeglia, Jacobus, et al., 2014) showed that compromised inhibitory functioning in 12 to 14 year olds was related to more frequent and intense alcohol and marijuana use by late adolescence (17 to 18 years). These authors suggest that tests of inhibitory performance could help to identify those at risk for initiating heavy substance use during adolescence. Prospective and longitudinal studies on adolescent exposure to nicotine also suggest that adolescent exposure to nicotine increases the risk of mental health disorders, including anxiety and depression, and increases the risk for developing addiction (reviewed by Counotte et al., 2011).

SOCIAL CONTEXT

Social learning and social and cultural context play an extremely important role in vulnerability to drug use and addiction. How a drug is experienced is related to an interaction between the pharmacological properties of the drug itself, the set (personal factors) and the

setting, including the views and beliefs of family and the wider social and cultural community towards a particular substance, as well as the views and beliefs of minority drug-using subcultures (Zinberg, 1986).

An example of the importance of social context and context specific drug use is provided by a unique study of heroin use by American servicemen in the Vietnam War (Robins et al., 1975). Heroin was considered at the time to be one of the most addictive and dangerous drugs. American servicemen in Vietnam were widely exposed to heroin and many became addicted after regular use. This led to a belief that on returning to America there would be problems relating to heroin use in this population. In reality these fears did not materialise, and only a small percentage of war veterans had heroin use problems on their return. These studies showed the widespread use of heroin by soldiers in Vietnam was related to specific contextual factors, such as easy access to high quality heroin, drug-using peers, the lack of alternative recreational substances, the absence of social censure, and the fact that serving in Vietnam (and its related stresses) were seen as unrelated to their lives in America (Robins, 1993).

It has been claimed that modern industrial capitalism, with its associated social upheavals such as rapid urbanisation, mass migration and modern warfare, has led to a dismantling of the cultural structures and social ties that would have more easily helped to control drug use in the past (Samson, 2004). It has also been argued (Alexander, 2008), that from a historical perspective, addiction is better understood as a societal rather than an individual problem. Alexander's 'dislocation theory of addiction' attributes the current increase in both substance and behavioural addictions to a lack of psychosocial integration (referred to as social dislocation) in the modern world. Psychosocial integration, in this context, means an interdependence between an individual and their society, and is proposed to fulfil the human need for both individual autonomy and social belonging. The competition and individualism inherent in the current globalising free market economy is proposed to have resulted in increasing feelings of alienation in both rich and poor, and an attendant poverty of the spirit. This is more readily visible when the powerful presence and influence of a majority culture adversely affects a minority indigenous culture, for example, as has happened to ethnic Canadian Indians in Vancouver, where it is argued that social dislocation has led to increases in drug and alcohol problems.

A number of studies have examined drug self-administration in laboratory rats housed in spacious and sociable conditions compared to isolated rats housed in standard laboratory cages (e.g. Alexander et al., 1978; Bozarth et al., 1989; Hadaway et al., 1979; Raz & Berger, 2010). These experiments aim to model the effect of social and environmental deprivation, and its associated stress, on drug use in humans. The results of these studies show a marked decrease in drug self-administration in animals housed in socially-enriched environments compared to isolated animals in standard cages, and provide indirect evidence to support the effect of social and environmental stress in the pathogenesis of addiction (see below).

STRESS AND THE CO-OCCURRENCE OF OTHER PSYCHIATRIC DISORDERS WITH ADDICTION

The role of both acute and chronic stress in triggering drug use and relapse is confirmed by the personal accounts of many people. Stress can manifest itself through traumatic life events and through unhappy or disrupted social relationships, for example with family, work colleagues or peer group. Evidence from animal and human research studies has repeatedly demonstrated that the hormonal system involved in regulating the stress response – the hypo-thalamic-pituitary adrenal axis (HPA axis) – is critically important in drug use, relapse and addiction (Koob, 2010; Koob & Le Moal, 2001; Lovallo, 2006; Stephens & Wand, 2012). The HPA-axis controls secretion of the hormone cortisol, which is a 'master hormone' that not only responds to stress, but also regulates multiple body organs for optimal functioning, as well as being involved in regulating mood and emotions.

Large-scale epidemiological studies (Grant, Hasin et al., 2004; Grant, Stinson et al., 2004) have revealed comorbidities of 21–29% for mood disorders, 22–25% for anxiety disorders, and 32–70% for personality disorders. All these disorders are associated with aberrations of HPA axis function. Thus, pre-existing factors which impact on biological stress systems may precipitate drug use. For addicts with co-morbid psychiatric disorders it is likely that substance use provides a habitual and temporarily effective means for coping with psychological stress in the short term, and is consistent with a self-medication view of addiction (Khantzian & Albanese, 2008).

HOW IS ADDICTION DIAGNOSED?

There are two main systems that are currently used to diagnose mental disorders, including addiction, which comes under the definition of 'substance use disorder' in these systems. These systems are the ICD and the DSM. The ICD is an abbreviation of *International Statistical Classification of Diseases and Related Health Problems* (World Health Organisation, 1992), and is the official classification system used throughout the world. The current version is the ICD-10, and a revised version (ICD-11) is in process at the time of writing and expected to be completed in 2018. The ICD is the system that is used to classify most disorders in medicine, including substance dependence and other mental disorders which come under the section called 'Mental and Behavioural Disorders'. The DSM is an abbreviation of the *Diagnostic and Statistical Manual of Mental Disorders*, and is the standard classification of mental disorders used by the American Psychiatric Association for clinical diagnosis in America, and is also used worldwide. The current version of the DSM is DSM-5, which was

released in 2013. Some of the differences between the DSM and the ICD are given below (adapted from Tyrer, 2014):

1. The ICD is the official world classification, whereas the DSM is the US classification (although it is also used in many other countries).
2. The ICD is intended for use by all health practitioners, whereas the DSM is used primarily by psychiatrists and researchers.
3. Special attention is given in the ICD to primary care and low- and middle-income countries, whereas the DSM is focused mainly on secondary psychiatric care in high-income countries.
4. The ICD-11 is planned to focus on clinical utility and intends to reduce the number of diagnoses, whereas the number of diagnoses has increased with each successive revision of the DSM (although there was a reduction of three diagnoses between DSM-4 and DSM-5).
5. The ICD provides diagnostic descriptions and guidance but does not employ operational criteria, the DSM depends on more clearly defined operational criteria: as a result of this the DSM is considered by some to be a more reliable (but not necessarily more valid) system, while the ICD allows for more clinical discretion in making diagnoses.

THE ICD-10

In the ICD, harmful use is distinguished from the dependence syndrome which can be considered analogous to addiction. The block (labelled F10 – F19) covers mental and behavioural disorders due to psychoactive substance use covering: alcohol, opioids, cannabinoids, sedative hypnotics, cocaine, other stimulants including caffeine, hallucinogens, tobacco, volatile solvents, multiple drug use, and use of other psychoactive substances.

The following extract shows the ICD-10 definitions for harmful use and dependence syndrome, and letters in brackets refer to ICD codes:

> Harmful use
>
> A pattern of psychoactive substance use that is causing damage to health. The damage may be physical (as in cases of hepatitis from the self-administration of injected drugs) or mental (e.g. episodes of depressive disorder secondary to heavy consumption of alcohol).

> The diagnosis requires that actual damage should have been caused to the mental or physical health of the user. Harmful patterns of use are often criticized by others and frequently associated with adverse social consequences of various kinds. The fact that a pattern of use or a particular substance is disapproved of by another person or by the culture, or may have led to socially negative consequences such as arrest or marital arguments is not in itself evidence of harmful use. Acute intoxication (see F1x.0), or "hangover" is not itself sufficient evidence of the damage to health required for coding harmful use. Harmful use should not be diagnosed if dependence syndrome (F1x.2), a psychotic disorder (F1x.5), or another specific form of drug- or alcohol-related disorder is present.

This next extract comes from the ICD-10 diagnostic guide for *dependence syndrome:*

> A cluster of physiological behavioural, and cognitive phenomena in which the use of a substance or a class of substances takes on a much higher priority for a given individual than other behaviours that once had greater value. A central descriptive characteristic of the dependence syndrome is the desire (often strong, sometimes overpowering) to take psychoactive drugs (which may or may not have been medically prescribed), alcohol, or tobacco. There may be evidence that return to substance use after a period of abstinence leads to a more rapid reappearance of other features of the syndrome than occurs with nondependent individuals.
>
> Diagnostic guidelines: A definite diagnosis of dependence should usually be made only if **three or more** of the following have been present together at some time during the previous year:
>
> a. a strong desire or sense of compulsion to take the substance;
>
> b. difficulties in controlling substance-taking behaviour in terms of its onset, termination, or levels of use;
>
> c. a physiological withdrawal state when substance use has ceased or been reduced, as evidenced by: the characteristic withdrawal syndrome for the substance; or use of the same (or a closely related) substance with the intention of relieving or avoiding withdrawal symptoms;
>
> d. evidence of tolerance, such that increased doses of the psychoactive substances are required in order to achieve effects originally produced by

lower doses (clear examples of this are found in alcohol- and opiate-dependent individuals who may take daily doses sufficient to incapacitate or kill nontolerant users);

e. progressive neglect of alternative pleasures or interests because of psychoactive substance use, increased amount of time necessary to obtain or take the substance or to recover from its effects;

f. persisting with substance use despite clear evidence of overtly harmful consequences, such as harm to the liver through excessive drinking, depressive mood states consequent to periods of heavy substance use, or drug-related impairment of cognitive functioning; efforts should be made to determine that the user was actually, or could be expected to be, aware of the nature and extent of the harm.

(Extract from *The ICD-10 Classification of Mental and Behavioural Disorders: Diagnostic Criteria for Research*, 2018, © WHO.)

THE DSM

The DSM was first published in 1952 and has been revised several times. Since its inception it has had widespread influence in the USA and elsewhere on how disorders are diagnosed, treated and investigated. The current version, DSM-5, was published in 2013 after a fourteen-year revision process which aimed to overcome some of the problematic issues identified in DSM-4-TR (Hasin et al., 2013). This involved literature reviews and extensive new data analyses by a work group consisting of 12 scientists and clinicians with additional input from consultants and advisers, and through comments obtained through the DSM-5 website.

The revised chapter in the DSM-5 is now called 'Substance Related and Addictive Disorders'. This change in terminology has been implemented to avoid the potential confusion that had arisen from earlier versions of the DSM, which had favoured use of the term 'dependence' over 'addiction'. Some scientists had considered 'addiction' to be derogatory and 'dependence' was chosen by a narrow margin of only one vote for the DSM-3-R in 1987, and subsequently incorporated unchanged into the the DSM-4-TR in 1994 (O'Brien, 2011). However, dependence does not necessarily mean addiction, and can be used to refer purely to the physiological adaptation that occurs when a substance is taken over time, rather than the compulsive drug-seeking behaviour that characterises addiction.

The major change implemented in the DSM-5 is the combining of DSM-4 abuse and dependence criteria into a single disorder, measured on a continuum from mild to severe. Other changes included: dropping legal problems as a diagnostic criterion because of its poor diagnostic utility; adding craving as a diagnostic criterion to increase consistency with ICD-10 (where it is included); adding cannabis and caffeine withdrawal syndromes; aligning tobacco use disorder criteria with other substance use disorders; and including gambling disorders in the same chapter as substance-related disorders. Gambling disorder is the sole condition in a new category on behavioural addictions, and is included in the same chapter as substance use disorders in recognition of research findings that have provided evidence for similarities between these disorders in their clinical expression, brain origin, co-morbidity, physiology, and treatment (APA, 2013).

Section III of the manual includes disorders for which further research is required before they can be considered as diagnosable disorders, and internet gaming disorder and caffeine use disorder are included in this section in DSM-5. Each specific substance is addressed as a separate use disorder (apart from caffeine, which is not considered as a diagnosable substance use disorder at present), but the same overarching criteria are used for diagnostic purposes. There are ten separate classes of substances included: 1) alcohol; 2) caffeine; 3) cannabis; 4) hallucinogens – phencyclidine or similar acting aryl cyclohexylamines; 5) inhalants; 6) opioids; 7) sedatives, hypnotics and anxiolytics; 8) stimulants; 9) tobacco; 10) other.

The overarching criteria used for diagnosis of substance use disorder in the DSM-5 are listed below using alcohol as an example of a substance use disorder:

> A problematic pattern of alcohol use leading to clinically significant impairment or distress, as manifested by at least two of the following, occurring within a 12-month period:
>
> 1. Alcohol is often taken in larger amounts or over a longer period than was intended.
> 2. There is a persistent desire or unsuccessful efforts to cut down or control alcohol use.
> 3. A great deal of time is spent in activities necessary to obtain alcohol, use alcohol, or recover from its effects.
> 4. Craving, or a strong desire or urge to use alcohol.
> 5. Recurrent alcohol use resulting in a failure to fulfil major role obligations at work, school, or home.

6. Continued alcohol use despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of alcohol.
7. Important social, occupational, or recreational activities are given up or reduced because of alcohol use.
8. Recurrent alcohol use in situations in which it is physically hazardous.
9. Alcohol use is continued despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by alcohol.
10. Tolerance, as defined by either of the following:
    a. A need for markedly increased amounts of alcohol to achieve intoxication or desired effect.
    b. A markedly diminished effect with continued use of the same amount of alcohol.
11. Withdrawal, as manifested by either of the following:
    a. The characteristic withdrawal syndrome for alcohol
    b. Alcohol (or a closely related substance, such as a benzodiazepine) is taken to relieve or avoid withdrawal symptoms.

CONSIDERATION OF CURRENT CLASSIFICATION SYSTEMS

Substance use disorders in both systems are defined by a set of criteria, where no single criterion is necessary or sufficient to diagnose addiction. Considerable overlap is observed between the DSM-5 and ICD-10 dependence criteria as would be expected, and this is desirable for diagnosis of the same condition. However, this also means that similar criticisms are applicable to both systems.

A major change in the DSM-5 has been the move away from splitting abuse criteria and dependence criteria as separately diagnosable syndromes. This has resulted in a more continuous classification system, with severity being measured by the number of diagnostic criteria. This move towards a more continuous system, and removal of the abuse diagnosis as a separate syndrome (which was previously considered to precede the diagnosis of dependence), is supported by statistical analysis, indicating that they represent the same underlying condition (Hasin et al., 2013). However, diagnostic criteria are not weighted in either the

DSM or the ICD systems, and a diagnosis is based on an individual having a minimum number of criteria. For DSM-5 the threshold is set at two or more for mild, four or five for moderate, and six or more criteria for a diagnosis of severe substance use disorder. It is therefore possible for people with very different defining criteria to be given an identical diagnosis. Both systems involve a degree of subjective interpretation, both by the individual being diagnosed and by the clinical or research practitioner. They are essentially a set of descriptive symptoms that provide a common language for diagnosis, and their major drawback is their lack of specificity (see Lilienfeld et al. 2013 for a detailed review of further problems associated with the DSM system).

Some of this diagnostic subjectivity could be addressed if objective measures, applicable to substance use disorders, were available. However, although there are biological correlates of addiction, these are currently of no diagnostic value in either system. Unlike mental disorders, many medical disorders have an identifiable biological basis that can confirm disease. Although the threshold of diagnosis can be arbitrarily defined even in medical diseases, defining a threshold for diagnosis is more subjective for mental disorders. The evidence for the use of biomarkers for substance use disorder diagnoses was reviewed for possible inclusion in DSM-5, but was rejected. This decision was based on the inability of current biomarkers to be diagnostically useful. For example, although useful for detection and confirmation of drug use, the limited time window applicable for current measures of drugs and associated metabolites in blood, urine, sweat, saliva and hair, prevents their use for diagnosing substance use disorder. Despite the repeated association of certain genetic variants with substance use disorders, these associations cannot be used for diagnosis due to their small effects and inconsistency across different populations. Also, evidence from neuroimaging studies such as positron emission tomography (PET) and functional magnetic resonance imaging (fMRI), showing group differences between substance users and controls, is not considered specific enough to diagnose substance use disorders in individual cases, and there is also considerable overlap with other psychiatric disorders. This absence of any reliable or valid biomarker for the diagnosis of addiction is ironic, considering the widespread influence of the prevailing biomedical paradigm.

OTHER DIAGNOSTIC SYSTEMS

RESEARCH DOMAIN CRITERIA

The Research Domain Criteria project (RDoC) was initiated by the US's National Institute of Mental Health (NIMH, 2008). It is an expansive framework for organising research from different scientific disciplines and across different levels of analysis. It differs considerably in

its approach compared to the ICD and DSM systems, and is compatible with a biologically-based conceptualisation of addiction as a disorder of dysfunctional brain circuitry.

This extensive research framework is in its infancy at present, but it is hoped that eventually it will lead to a more precise classification system for psychiatric diagnosis (Insel, 2014). The RDoC framework comprises a matrix of broad psychobiological domains that correspond to brain-based circuits that are relevant to psychopathology. These domains include negative valence systems (e.g. fear, anxiety, loss) and positive valence systems (e.g. approach motivation, reward responsiveness, reward learning, habit), with different subcategories in each domain (see Cuthbert, 2014). These domains can be experimentally studied using different types of research tools and strategies. The research paradigm under which the investigation is being conducted is also taken into consideration.

An example of this approach is a recent review of abnormal reward functioning across substance use disorders and major depressive disorder (Baskin-Sommers & Foti, 2015). This review synthesised available data from preclinical, electrophysiological and neuroimaging literature on reward processing, and organised information under the key reward constructs within the Positive Valence Systems domain of the RDoC matrix. These authors suggest that examining reward functioning across clinically diverse samples, rather than within limited diagnostic categories, may ultimately be more clinically useful.

Although this approach will undoubtedly increase understanding of the biological correlates of addiction, it also faces several methodological and conceptual challenges, and has been criticised for underplaying the importance of the social, cultural and psychological dimensions of mental health (Bracken et al., 2012; Lilienfeld, 2014).

HEAVY USE OVER TIME

In Europe there has been a drive by some scientists to implement the concept of *heavy use over time* as the key criterion for defining substance use disorders (Rehm et al., 2013). Proponents of this view argue that this concept underlies all current definitions of substance use disorder criteria. It is argued that the physiological, psychological, social, behavioural, and other health consequences of substance dependence, as defined by the current DSM and ICD criteria, are nearly all associated with 'heavy use over time'. For example, tolerance and withdrawal are argued to be physiological consequences of heavy use over time, whereas craving is argued to be a psychological consequence. Other consequences of heavy use over time include relinquishing social, occupational, and recreational activities in favour of drug use. It is also argued that physical and health consequences arise from heavy use over time, in a dose-dependent manner, for example, diseases such as cirrhosis of the liver result from

increasing heavy alcohol use over time. Data obtained from epidemiological surveys demonstrate a high correlation between level of consumption and criteria used for diagnosis of substance use disorders. It is also argued that high levels of consumption are more associated with adverse mortality and morbidity outcomes than current diagnostic criteria, and are therefore far more relevant to public health. Proponents of 'heavy use over time' suggest that a definition based on a continuum such as consumption level (with defined thresholds for diagnosis, as currently occurs for example with blood pressure) will reduce the stigma attached to the current diagnoses of substance use disorders.

Although there is undoubtedly some merit in this approach, it is not without criticism. Heather (2013) argues that the addiction concept is necessary to direct theory and research towards understanding the essential paradox of addictive behaviour – continued use despite knowledge of harmful consequences – and this is not addressed by invoking 'heavy use over time'. Saunders (2013) also questions the ability of 'heavy use over time' to replace the concept of substance use disorders, because it fails to capture the clinical experience as described through cognitions, behaviours, actions and physiological responses, and argues that a quantification of heavy use is problematic in itself. For example, although commercial alcohol and tobacco products can be accurately quantified, underreporting of use is a common problem in clinical practice, whereas other features of the dependence syndrome can be more readily communicated by substance users and observed by others (e.g. impaired control, continued use despite harmful consequences). Quantification with regard to illicit substances presents an even greater challenge, where the absence of quality control means that it is difficult to ascertain the amount of drug taken. Bradley and Rubinsky (2013) advocate that consumption measures should be included in the diagnostic criteria for substance use disorders rather than replace them, and suggest that this approach would lead to the identification of a greater number of people who could be helped to reduce their high-risk drinking.

SUMMARY

- Addiction is thought to result from a combination of biological factors that interact with environmental contexts. The definition and popular understanding of what addiction means have varied throughout history.
- Addiction generally consists of several stages through which an affected individual repeatedly cycles. These stages broadly consist of initial use, escalating use, tolerance, abstinence, craving and relapse. Initial substance use tends to be motivated by reward seeking (positive reinforcement), but in its later stages substance use can often end up being motivated by relief from discomfort (negative reinforcement).

- The exact mechanisms underlying the addictive process are not well understood. It is likely that the contribution of differing factors varies between individuals, and also within an individual, depending on which stage of the addictive cycle they are engaged in. Factors which influence addiction include: genetic predisposition; developmental factors; social context; exposure to stress; and co-occurring psychiatric problems. These factors are not mutually exclusive and interact to varying degrees.
- The current classification systems used to diagnose addiction are the DSM-5 and the ICD-10. These classification systems are not ideal, due to the fact that they are descriptive and entail subjective judgement. There is general agreement that some kind of classification system is necessary, but the nature and content of these systems will continue to evolve, and there is potential for future revision as more information becomes available.

QUESTIONS

- What is addiction?
- How is addictive behaviour diagnosed?
- Who becomes addicted?