Theories of addiction: Causes and maintenance of addiction

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Overview: Theories of addiction

In attempting to explain why people become dependent on drugs, a variety of different approaches have been taken. What follows is a summary of three different areas of explanation. The first concentrates on the neurobiological effects of drugs, and explains drug dependence in biological terms. The second approach is psychological, with explanations concentrating on behavioural models and individual differences. The final approach is sociocultural, with explanations concentrating on the cultural and environmental factors that make drug dependence more likely. As will become clear, there are a variety of approaches to the question of why people become dependent on drugs. These are not mutually exclusive.

Neuroscientific theories

Neuroscientific theories require an understanding of the effects of drugs on the brain, and Box 4.1 outlines the actions of each of the major drug classes. Different drugs clearly have different primary actions on the brain, but two major pathways – the dopamine reward system and the endogenous opioid system – have been implicated as common to most drugs (Koob & LeMoal, 1997; Nutt, 1997).

Box 4.1 Molecular and cellular effects of drug action

Alcohol

Alcohol has several primary targets of action, and identifying the mechanisms of action has proved to be a difficult task. Acute administration of alcohol leads to increases in inhibitory transmission at gamma-amino-butyric acid (GABA-A) channels, increased serotonin (5HT-3) function, dopamine release and transmission at opiate receptors, and a reduction of excitatory transmission at the NMDA subtype of the glutamate receptor (Altman et al., 1996; Markou, Kosten, & Koch, 1998).

Nicotine

Nicotine is an agonist at the nicotinic receptor – that is, it activates the nicotinic receptor. Nicotinic receptor activation results in increased transmission of a number of neurotransmitters including acetylcholine, norepinephrine, dopamine, serotonin, glutamate, and endorphin (Benowitz, 1998).

Cannabis

The main active ingredient in cannabis is Δ^9 –tetrahydrocannabinol (Δ^9 -THC), which acts as an agonist at the cannabinoid receptor in the brain. This action results in the prevention of the uptake of dopamine, serotonin, GABA, and norepinephrine (Comings et al., 1997). The cannabinoid (CB1) receptor is most common in the hippocampus, ganglia, and cerebellum (Comings et al., 1997).

Opiates

The brain's endogenous opioid system constitutes peptide including endorphins and enkephalins, which are stored in opiate neurons and released to mediate endogenous opiate actions (Altman et al., 1996; Nutt, 1997).

Opiate drugs act as agonists at three major opiate receptor subtypes; *μ* (mu), δ(delta), and *κ* (kappa). The mu receptor appears to be the subtype important for the reinforcing effects of opiate drugs (Altman et al., 1996; Di Chiara & North, 1992). Mu receptors are largely located on cell bodies of dopamine neurons in the ventral tegmental area (VTA), the origin of the mesolimbic dopamine system; and on neurons in the basal forebrain, particularly the nucleus accumbens (Altman et al., 1996; Di Chiara & North, 1992). Delta opiate receptors may be important for the potentiation of the control of reinforcers over behaviour (Altman et al., 1996). There is some evidence that kappa opiate receptors are involved in the aversive effects associated with withdrawal symptoms of opiates (Altman et al., 1996).

Psychomotor Stimulants

Cocaine

Cocaine binds to dopamine, noradrenaline, and serotonin transporters (Altman et al., 1996), but it is thought that cocaine's blockage of dopamine re-uptake is the most important element mediating its reinforcing and psychomotor stimulant effects. This has been supported by recent evidence showing that dopamine D1-like receptors may play an important role in the euphoric and stimulating effects of cocaine. A D1 antagonist significantly attenuated the euphoric and stimulating effects of cocaine, and reduced the desire to take cocaine, among cocaine-dependent persons (Romach et al., 1999).

Box 4.1 Continued

Amphetamine

Amphetamine acts to increase monoamine release, as well as to increase release of dopamine, with secondary effects occurring in the inhibition of dopamine re-uptake and metabolism (Altman et al., 1996; Stahl, 1996). Similarly to cocaine, the enhanced release and inhibited re-uptake of dopamine is thought to be most important for amphetamine's reinforcing effects (Altman et al., 1996).

Benzodiazepines

Benzodiazepines act by binding with sites on the GABA-A/benzodiazepine receptor (Altman et al., 1996). This results in an increase in chloride conductance through chloride channels, thus enhancing inhibitory transmission. Increased dopamine transmission has been found in the VTA following acute benzodiazepine administration (Altman et al., 1996), but decreased dopamine levels occur in the nucleus accumbens.

Dopamine reward system

The mesolimbic–fronto cortical dopamine system (containing the mesolimbic and mesocortical dopamine systems) is regarded as a critical pathway in brain reward (Nutt, 1997; Wise, 1996). Dopamine has been implicated in the reinforcing effects of alcohol, with alcohol use resulting in the direct stimulation of dopamine and also an indirect increase in dopamine levels (Altman et al., 1996). It is also thought that the behavioural rewards of nicotine, and perhaps the basis of nicotine dependence, are also linked to the release of dopamine in the mesolimbic pathway (Benowitz, 1998; Markou et al., 1998). Following administration of nicotine, increased dopamine is released in rats, and lesions in the mesolimbic dopamine pathway lead to reduced self-administration of nicotine (Altman et al., 1996).

Cannabis was long considered an "atypical" drug, in that it did not interact with the brain's reward system. However, research has revealed that the active component of cannabis, Δ^9-tetrahydrocannabinol (A^9-THC), produces enhancement of brain-stimulation reward in rats, at doses within the range of human use (Gardner, 1992). Studies have also revealed cannabinoid receptors in areas associated with brain reward, and that Δ^9-THC increases dopamine levels (Adams & Martin, 1996; Gardner, 1992). This suggests that cannabis does in fact interact with the dopaminergic system. Cocaine's effects have also been related to an increase in dopamine function (Bergman, Kamien, & Spealman, 1990; Caine & Koob, 1994; Spealman, 1990; Spealman, Bargman, Madras, & Melia, 1991).

Endogenous **opioid** system

There is evidence that the brain's endogenous opioid system may play an important role in drug use and misuse. Exogenous opiates such as heroin, morphine, and codeine act as opiate receptor agonists, and readily cause tolerance and dependence. Adaptation of opiate receptors occurs quite readily after chronic opiate use, as is seen in the need to use larger amounts to achieve pain relief or euphoria. Further, the opiate antagonist naloxone will quickly induce withdrawal symptoms if administered.

Research is increasingly suggesting that the opioid system may be involved in the rewarding effects of other psychoactive substances. One form of therapy for alcohol dependence is the use of the opiate antagonist naltrexone, which has been shown to block the reinforcing properties of alcohol, suggesting that the endogenous opioid system may play an important role in the rewarding effects of alcohol. Recent research suggests that long-term tobacco-smoking may cause changes in the responsivity of the endogenous opioid system, which leads to an increased likelihood of developing nicotine dependence (Krishnan-Sarin, Rosen, & O'Malley, 1999). Research has also found that doses of naloxone reverse the enhancement of brain reward caused by the active component of cannabis, Δ^9-THC (Gardner, 1992).

The dopaminergic and opioid systems have been characterized by some theorists as playing **two** different functions (Di Chiara & North, 1992). The dopaminergic pathway is associated with the incentive, preparatory aspects of reward, which are experienced as thrill, urgency, or craving. In contrast, the opioid system is associated with the satiation and consummatory aspects of reward, such as rest, blissfulness, and sedation (Di Chiara & North, 1992).

Biological factors

One area of research has concentrated on exploring biological characteristics that underlie drug dependence. These can be grouped into **two** kinds of explanations; one which examines individual differences in liability to drug dependence because of genetic characteristics, and one which accounts for drug dependence in terms of changes that occur in the brain due to chronic drug administration.

Genetic factors

One hypothesis concerning drug dependence is that people may inherit an increased likelihood *(vulnerability)* of developing dependence on substances. The question of whether or not such vulnerability exists has been examined in the form of numerous family studies, adoption studies, and twin studies.

Family studies of alcohol use disorders suggest that such disorders do cluster in families (Kendler, Davis, & Kessler, 1997; Merikangas, 1990; Merikangas et al., 1998). In a recent study, over one-third (36%) of the relatives of persons with an alcohol use disorder were also diagnosed with an alcohol use disorder (abuse or dependence), compared to 15% of the relatives of controls (Merikangas et al., 1998). This relationship was stronger in a study that examined the rate of alcohol *dependence* among *siblings:* among subjects identified with alcohol dependence, 50% of male siblings met criteria for alcohol dependence, compared to 20% of controls' male siblings; the respective rates for female siblings were 24% and 6% (Bierut et al., 1998). Clearly, alcohol use disorder is likely to occur in more than one family member.

Similar aggregation has been found for other drug use disorders (Bierut et al., 1998; Merikangas et al., 1998). Among persons whose predominant problematic drug was cannabis, 13% of relatives also had a cannabis use disorder, compared to 2.4% of controls' relatives. The comparative rates for opiates were 10% vs. 0.4% and, for cocaine, 7.5% vs. 0.8% (Merikangas et al., 1998).

While these studies suggest that substance use disorders cluster within families, family studies do not allow us to separate the effects of genetic and environmental influences. The clustering may occur simply because the siblings share the same environment rather than any underlying genetic cause. The separate contribution of genes and environment can be teased apart in studies of adopted children and of monozygotic and dizygotic twins.

Adoption studies examine rates of disorder among adoptees, given their biological and adoptive parents' disorder status. This allows evaluation of the effects of genetic (biological parents' status) and environmental (adoptive parents' status) effects on vulnerability to substance use disorders. Research suggests that there is a significant genetic factor that influences adoptees' vulnerability to alcohol use disorders (Bohman, Sigvardsson, & Cloninger, 1981; Cloninger, Bohman, & Sigvardsson, 1981; Goodwin, Schulsinger, Hermansen, Guze, & Winokur, 1973; Heath, 1995).

Researchers have attempted to develop models of vulnerability to substance use disorders, in which vulnerability is the product of genetic and/or environmental factors. Research with twins suggests that there is a significant genetic component (heritability) that increases the likelihood of dependence on a range of substances. For example, twin studies have produced estimates of the heritability of alcohol dependence ranging from 39 to 60% of the total variance (Heath, 1995; Kendler et al., 1997; Kendler, Heath, Neale, Kessler, & Eaves, 1992; Kendler, Neale, Heath, Kessler, & Eaves, 1994; Prescott & Kendler, 1999; Prescott, Neale, Corey, & Kendler, 1997; True et al., 1999). Similarly, the heritability of smoking persistence has been estimated at 53% (Heath & Martin, 1993), and that for nicotine dependence between 60 and 70% (Kendler et al., 1999a; True et al., 1999). Research examining dependence on other drugs has revealed significant heritability estimates for cannabis dependence (Kendler & Prescott, 1998b; Tsuang et al., 1998) and dependence on heroin, sedatives, and stimulants (Kendler & Prescott, 1998b; Tsuang et al., 1996, 1998).

These findings suggest a further issue: Do persons have a vulnerability towards one specific drug, or is there a more general vulnerability to a class of drugs or, indeed, to any psychoactive substance? In one discussion of this question, researchers concluded: "There is no definitive evidence indicating that individuals who habitually and preferentially use one substance are fundamentally different from those who use another" (Tarter & Mezzich, 1992). A recent study found that among the relatives of persons with substance use disorders, rates of *all* substance use disorders were higher than those among the relatives of controls (Merikangas et al., 1998).

Other recent research involving male twins has also examined the issue of a common genetic vulnerability to substance misuse (True et al., 1999; Tsuang et al., 1998). One of these studies examined the genetic and environmental contributions to illicit substance abuse and dependence (cannabis, stimulants, sedatives, opiates, and psychedelics) (Tsuang et al., 1998). It found that there was a significant common genetic component (16% of the variance for heroin, 22% for cannabis, stimulants, sedatives, and 26% for psychedelics). Around one-third of the variance in this common vulnerability was caused by genetic effects. A similar analysis of alcohol and nicotine dependence (True et al., 1999) suggested that there was a significant common genetic vulnerability ($r = 0.68$) to both nicotine and alcohol dependence among male twins, with 26% of the total variance in the

risk for alcohol dependence shared with genetic risk of nicotine dependence.

Genetic characteristics

The exact nature of these genetic vulnerabilities has been the subject of increasing research. Thus far, no single candidate genes have been discovered that are directly related to drug abuse (Altman et al., 1996); it is likely that these influences may involve multiple genes or incomplete expression of several major genes (Kendler, 1999; Schuckit, 1999). For example, there is recent evidence to suggest a relationship between tobacco-smoking and genes involved in dopamine regulation (Lerman et al., 1999; Pomerleau & Kardia, 1999; Sabol et al., 1999). Research examining the gene for the brain's cannabinoid system (CNR1) found that variants of the CNRl gene were associated with cannabis, cocaine, and heroin dependence (Comings et al., 1997).

Neuroadaptation

One theory of drug dependence is based on the concept of neuroadaptation (Koob & LeMoal, 1997). Neuroadaptation refers to changes in the brain that occur to oppose a drug's acute actions after repeated drug administration. This may be of **two** types: *within-system adaptations,* where the changes occur at the site of the drug's action, and *between-system adaptations,* which are changes in different mechanisms that are triggered by the drug's action. When drugs are repeatedly administered, changes occur in the chemistry of the brain to oppose the drug's effects. When this drug use is discontinued, the adaptations are no longer opposed; the brain's homeostasis is disrupted (Koob & LeMoal, 1997).

Essentially, this hypothesis argues that tolerance to the effects of a drug and withdrawal when drug use stops are both the result of neuroadaptation (Koob, Caine, Parsons, Markou, & Weiss, 1997). Animal models have shown that stressful stimuli activate the dopamine reward system, so vulnerability to relapse from abstinence is hypothesized to occur. As a result, drug use continues in an attempt to avoid the symptoms that follow if drug use stops (Koob & LeMoal, 1997).

While, traditionally, conceptualizations of drug dependence focused on physical withdrawal symptoms, more recent formulations have begun to concentrate on the presence of more motivational symptoms, such as dysphoria, depression, irritability, and anxiety. It has been hypothesized that these negative motivational symptoms

are manifestations of neurobiological changes, and that these changes signal "not only . . . the beginning of the development of dependence, but may also contribute to vulnerability to relapse and may also have motivational significance" (Koob et al., 1997, p. 53). This approach hypothesizes that, after chronic drug use, changes occur in the dopamine reward system and the endogenous opioid system.

Psychological theories

Psychological approaches to the explanation of drug dependence have often been based on concepts that are common to those of other syndromes of behaviour involving compulsive or impulsive behaviours, such as obsessive–compulsive disorder or gambling (Miller, 1980). In particular, emphasis is given to the fact that there is impaired control over use and continued use despite usage problems. There are a variety of psychological approaches to the explanation of drug dependence, including emphasis on learning and conditioning (behavioural models), cognitive theories, pre-existing behavioural tendencies (personality theories), and models of rational choice.

Behavioural theories

Behaviourist models of addiction focus on directly observable behaviour. One group concentrates on the fact that behaviour is maintained (or made more likely) by the consequences (reinforcers) of such behaviour (West, 1989). Drug self-administration is then an example of ***instrumental behaviour*** because the activities of persons (or animals in an experiment) are instrumental in obtaining the consequences (the drug's effects).

Research with animal subjects has shown that when drugs are available, drug-naive animals will self-administer them, often to excess (Institute of Medicine, 1996). This observation has led to the development of the ***drug self-administration model.*** Drugs might be reinforcing in **two** general ways: through the direct effects of drugs on some sort of reinforcement system in the brain; or through its effects on other reinforcers (such as social or sexual reinforcers) or behavioural effects (such as increased attention) (Altman et al., 1996).

Research with animals has shown that they will self-administer most drugs (except LSD and Δ^9-THC) in instrumental paradigms

(Altman et al., 1996), and this finding has been replicated with many species of animal and a variety of routes of administration (Institute of Medicine, 1996).

In using these animal research models, it has been possible to control the history of use (learning) and current environmental conditions of use (cues). As a result, it has been shown that both of these factors are important in the development of persistent use or abuse of drugs (Barrett & Witkin, 1986).

These findings point to another group of behaviourist theories, which focus on *classical conditioning* (Greeley & Westbrook, 1991; Heather & Greeley, 1990) and this sort of learning has been found to play an important part in the development and maintenance of addictive behaviours. *Cue exposure theory* is based on classical conditioning theory, and argues that cues are important in the development and maintenance of addictive behaviour (Drummond, Tiffany, Glautier, & Remington, 1995; Heather & Greeley, 1990). A cue that has previously been present when drugs were administered will be more likely to elicit a conditioned response (cue reactivity). This is thought to underlie craving, and may explain why someone who was dependent on a substance but has been abstinent for some time experiences strong cravings (Heather & Greeley, 1990).

The number of cues that may be associated with addictive behaviours is potentially infinite. Exteroceptive cues occur before the use of a drug, and may include the smell of an alcoholic drink, the sight of a needle, or may even constitute the time of day when drugs are typically taken. Interoceptive cues include such things as the effects of a drug on the brain's receptors, mood cues such as depressed affect (Greeley, Swift, & Heather, 1992), or cognitions such as beliefs about drug effects (Drummond et al., 1995).

The response to these cues may be autonomic, behavioural, or symbolic-expressive (Drummond et al., 1995). Autonomic responses that have been observed in cue exposure experiments include changes in heart rate, temperature, and salivation; symbolic-expressive responses that have been observed include self-reported drug-craving and urges to use drugs; and behavioural responses include an increased likelihood to use drugs.

Cognitive theories

There are a number of theories that explain drug dependence in terms of cognitive constructs. One theory proposes that *self-regulation*

is an important factor in the development of drug use problems. Self-regulation has been described as taking "planful action designed to change the course of one's behaviour" (Miller & Brown, 1991), the "executive (i.e. non-automatic) capacity to plan, guide and monitor one's behaviour flexibly, according to changing circumstances" (Diaz & Fruhauf, 1991). Self-regulation involves planning, taking into account social and physical factors as well as one's own goals, and acting appropriately. Addictive behaviours are seen as the result of having an excessive reliance on external structures – in the case of drug dependence, excessive reliance on substance use – to maintain a physical and psychological balance.

Personality theories

Some theorists argue that certain people are more prone to addiction through a so-called "addictive personality". Hans Eysenck has discussed this in terms of a ***psychological resource model,*** whereby the habit of drug-taking is developed because the drug used fulfils a certain purpose that is related to the individual's personality profile (Eysenck, 1997). For such people, drug-taking behaviour – or, more specifically, "addiction" – holds benefits even though there are negative consequences that occur after some time.

According to Eysenck, there are three major and independent personality dimensions: P (psychoticism), N (neuroticism), and E (extraversion) (Eysenck & Eysenck, 1985). The psychoticism dimension refers to an underlying propensity to functional psychosis, which lies along a continuum from "altruistic" to "schizophrenic" (Eysenck, 1997). Some of the characteristic traits of this dimension are aggression, coldness, egocentricity, impersonality, and impulsivity. The neuroticism dimension refers to a propensity towards emotional lability: some of the traits of neuroticism are moodiness, irritability, and anxiety. Genetic factors are theorized to play an important role in determining these personality dimensions; indeed, studies have shown that the major personality dimensions have high heritability (Eley & Plomin, 1997; Eysenck, 1997).

There has been extensive examination of the relationship between drug dependence and these personality dimensions. Research examining the link between E and drug dependence has revealed inconsistent findings; in a review of research on this topic, 10 studies found a negative relationship, 2 found a positive correlation, and **12** found no significant relationship (Francis, 1996). In contrast, there has been considerable research suggesting that persons with dependence

on a range of substances – alcohol, heroin, benzodiazepines, nicotine – have higher than normal N and P scores (Francis, 1996).

This research suggests that persons who are more moody, irritable, and anxious (high N scores), and those who are more impulsive and aggressive (high P scores), are also more likely to have substance use problems. However, on the basis of correlational studies such as those above, it is difficult to make any statements about the nature of the relationship between these and substance use problems. It may be the case, for example, that persons who develop problematic drug use become more irritable, moody, or aggressive as a result of changes that occur due to their drug use. Furthermore, research examining the genetic contributions to personality suggests that dimensions such as N may indicate vulnerability to psychopathology *in general,* not simply a tendency to have an "addictive personality" per se.

Other research, conducted with longitudinal studies of children, has examined personality attributes that predict 'substance use at a later stage and has found that, in general, adolescents who are more rebellious and have less conventional attitudes are more likely to drink, smoke, and use illicit drugs (Institute of Medicine, 1996).

Rational choice theories

One group of theories examines the problem of why people voluntarily engage in self-destructive behaviour (Elster & Skog, 1999). One of the central elements of drug dependence is the fact that the individuals have impaired control over their use of the substance. This may manifest itself in continued use despite a wish to reduce or stop use of the drug, to use greater amounts of the drug than intended, or to use the drug for longer periods than intended (American Psychiatric Association, 1994). This difficulty may be greater in certain contexts – for example, if an alcohol-dependent person has not had a drink for some time and walks past a bar.

Some would argue that this represents a form of "weakness of will": that addiction is an example of behaving "against one's own better judgement" (Davidson, 1985; Pears, 1984). For such theorists, drug-dependent persons have a choice of two options, both of which may be evaluated in terms of their future consequences. They realize that one option is superior, yet choose the other; for instance, an individual may have made the decision to stop drinking and yet accept a drink from someone even though the individual knows, at that particular time, that he or she should not.

As Elster and Skog (1999) point out, the problem with this approach is that it is difficult to know whether such a person knew *at the* ***time*** *of acceptance* that choosing the drink was the less preferred option. The person may have made this considered decision before the party, and regretted accepting the drink after the party, but it is difficult to ascertain whether he or she thought so at the time of accepting the drink.

In contrast, some theorists argue that drug-dependent persons ***do*** make rational choices in their continued use of drugs. The aim of these theories is to explain how rational people can become "knowingly trapped in a consumption pattern . . . and . . . once they realise that their current lifestyle is actually suboptimal (i.e. not the best thing, all things considered), still continue to act the same way" (Skog, 1999).

A number of attempts to explain this paradox have centred on people's ability to weigh present and future benefits – in other words, their ability to consider the immediate rewards associated with drug use, weighed against the longer-term benefits of abstention (Ainslie, 1992; Becker & Murphy, 1988; Herrnstein & Prelec, 1992). One approach has been to argue that people have a limited ability to consider future benefits – "cognitive myopia" (Hermstein & Prelec, 1992); hence, the choice to use drugs at one particular time is rational, given the considered options. Two other approaches have argued that while people are able to consider a range of future benefits, present and future benefits are differentially weighted, with greater weight given to the present (Ainslie, 1992; Becker & Murphy, 1988).

Contextual factors

There are a number of social and environmental factors that have been strongly related to substance use and substance use disorders. These are in keeping with the findings of twin studies, which show that while there is a strong genetic component to vulnerability to drug dependence, there is also a substantial environmental component (Kendler & Gardner, 1998; Kendler, Karkowski, & Prescott, 1999b; Kendler & Prescott, 1998a). A range of these factors are outlined below.

There is much evidence to suggest that people with antisocial behaviour are more likely to have or develop substance use problems. Adolescents with conduct disorders are significantly more likely to

develop substance use disorders than those without such conduct problems (Cicchetti & Rogosch, 1999; Gittelman, Mannuzza, Shenker, & Bonagura, 1985). In general, it appears that the earlier, more varied and more serious a child's antisocial behaviour, the more likely will it be continued into adulthood, with substance misuse considered as one of these antisocial behaviours (Costello, Erkanli, Federman, & Angold, 1999; Robins, 1978). Furthermore, children or young people with anxiety or depressive symptoms are more likely to begin substance use at an earlier age, and to develop substance use problems (Cicchetti & Rogosch, 1999; Costello et al., 1999; Henry et al., 1993; Loeber, Southamer-Lober, & White, 1999).

The peer environment also has a large influence on the drug-taking behaviour of individuals. Drug use usually begins with peers, and peer attitudes to drug use have been shown to be highly predictive of adolescent drug use (Fergusson & Horwood, 1997; Hoefler et al., 1999; Newcomb, Maddahian, & Bentler, 1986) perhaps because those who use drugs are more likely to choose to spend time with peers who also use drugs. There is, however, no evidence concerning the influence of peers on the development or maintenance of drug ***dependence*** (Institute of Medicine, 1996).

Families also have a strong effect on the likelihood that people will develop substance use problems (Hawkins, Catalano, & Miller, 1992; Lynskey & Hall, 1998a) and this may occur in a number of ways. First, modelling of substance use by parents and other family members has been shown to affect the chances of the substance use behaviour of adolescents. For example, parents' drug use has been associated with the initiation and frequency of alcohol and cannabis use (Hawkins et al., 1992), while older brothers' drug use and attitudes towards drug use have been associated with younger brothers' drug use (Brook, Whiteman, Gordon, & Brook, 1988). Second, there is evidence that if parents hold permissive attitudes towards the use of drugs by their children, their children will be more likely to use drugs (Hawkins et al., 1992). Third, the nature of family relationships has an effect on the likelihood that adolescents will develop problematic drug use. The risk of substance misuse is higher if there is family discord, poor or inconsistent behavioural management techniques by parents, or low levels of bonding within the family (Hawkins et al., 1992).

The sociocultural background of a person will also affect the likelihood that he or she will develop substance use problems; for example, people who come from lower socioeconomic backgrounds are more likely to have problematic use of a range of drugs (Anthony,

Warner, & Kessler, 1994; Hall, Johnston, & Donnelly, 1999a). Those who have completed fewer years of education, or who have performed poorly in school, are also much more likely to have problematic substance use. Also, people who have grown up in an area in which there are high rates of crime, where drugs are readily available, and who have associated with delinquent peers, are much more likely to have drug misuse problems (Institute of Medicine, 1996).

Summary

The neurochemistry of chronic drug use is being more clearly described and understood. It appears that key pathways in the brain are involved in substance use and dependence, and research has revealed that changes in the brain's balance and neurotransmitter function occur after chronic drug use. So why do some people become dependent on drugs? Genetic factors do appear to play a part. Significant genetic components have been found to play a part in dependence on many of the most commonly used licit and illicit substances, suggesting that some persons are more vulnerable than others to developing drug use problems. There is also consistent evidence that certain environmental factors will increase the likelihood of problematic substance use, such as economic disadvantage, family conflict, modelling of drug use, or parents' permissive attitudes towards drug use, as well as conduct and emotional problems at an early age.

Psychological approaches to the issue of substance dependence have attempted to explain some of the behavioural and cognitive phenomena thought to underlie problematic substance use. Some theories – such as those proposing a personality type more disposed to addiction, or those characterizing the "rational" addict – have less importance for clinicians. However, learning theories hold considerable importance: they can be empirically tested, and supporting evidence suggests that learning plays an important role in the development and maintenance of substance use problems. In the same way, learning may be used to overcome these problems.

Clearly, there have been a number of approaches to explain why some people become dependent on psychoactive substances. Each level of explanation – genetic, psychological, or sociocultural – has been supported by empirical research, but these different levels remain to be integrated into a more comprehensive model of addiction.

Currently, the biopsychosocial model of the causes of addictive behaviours forms the basis of most treatment responses to addictions (Marlatt & VandenBos, 1997). In contrast to the disease model, the biopsychosocial model sees "addiction" as a complex behaviour pattern having biological, psychological, sociological, and behavioural components. These include the subjective experience of craving, short-term gratification at the risk of longer-term harm, and rapid change in physical and psychological states. Addictive behaviour is distinguished from other problem behaviours by the individual's overwhelming, pathological involvement in drug use, intense desire to continue using the drugs, and lack of control over his or her drug use. This theory provides the principles for the treatment responses to addiction outlined in the next sections of this book.