Theory of Addiction

Robert West

with

Ainsley Hardy

University College London

Blackwell Publishing
Addiction Press
CONTENTS

Foreword vii
Acknowledgements ix

1 Introduction: journey to the centre of addiction 1
   In the beginning 1
   In the end 3
   What this book does 4
   The synthetic theory of addiction in brief 6

2 Definition, theory and observation 9
   Defining addiction (addiction is not an elephant) 9
      Four views on what is addiction 13
      Addiction and dependence 14
      Diagnosing and measuring addiction 14
      Theory and supposition 16
         Meehl’s insights into the failure of soft psychology to make progress 21
   ‘Big questions’ in the field of addiction 22
   Recapitulation 28

3 Beginning the journey: addiction as choice 29
   Addiction as a rational, informed choice based on stable preferences 29
      The RISC theory: one addict’s eye view 30
      Box 3.1 The myth of addiction 31
      Box 3.2 The Theory of Rational Addiction 32
      Box 3.3 The Self-medication Model of addiction 36
      Box 3.4 Opponent Process Theory 40
   Irrational, ill-informed choice and unstable preferences 46
      Rational ill-informed choice with unstable preferences: an addict’s eye view 47
      Box 3.5 Expectancy Theories 48
      Box 3.6 Skog’s Choice Theory 52
      Box 3.7 Slovic’s Affect Heuristic 54
      Box 3.8 Cognitive Bias Theories 56
      Box 3.9 Behavioural Economic Theory 59
      Box 3.10 Gateway Theory 64
Box 3.11 The Transtheoretical Model (TTM) of behaviour change 66
Box 3.12 Identity shifts and behaviour change 72
Addiction as the exercise of choice based on desires 73
Watering my Yucca plant and trying to overcome addiction 74

4 Choice is not enough: the concepts of impulse and self-control 75
Reports of feelings of compulsion 75
Powerful motives versus impaired control 76
Box 4.1 The Disease Model of addiction 76
Personality and addiction typologies 78
Box 4.2 Tri-dimensional Personality Theory 78
Self-efficacy 80
Box 4.3 Self-efficacy Theory 80
The transition from lapse to relapse 82
Box 4.4 The Abstinence Violation Effect 82
Impulse control 85
Box 4.5 Inhibition Dysregulation Theory 85
Self-regulation as a broadly based concept 87
Box 4.6 Self-regulation Theory 88
Urges and craving 88
Box 4.7 A Cognitive Model of Drug Urges 89
Addiction as a failure of self-control over desires and urges 90

5 Addiction, habit and instrumental learning 91
Instrumental learning 91
Box 5.1 Instrumental learning (operant conditioning) and addiction 92
Mechanisms underpinning instrumental learning 95
Box 5.2 The Dopamine Theory of drug reward 95
Box 5.3 Addiction arising from functional neurotoxicity of drugs 97
Classical conditioning 99
Box 5.4 Classical conditioning and addiction 99
More complex learning models 101
Box 5.5 Addiction as a learning/memory process 101
Box 5.6 Incentive Sensitisation Theory 102
Box 5.7 Balfour’s theory of differential drug effects within the nucleus accumbens 105
Social learning 106
Box 5.8 Social Learning Theory 106
Associative learning 108

6 Addiction in populations, and comprehensive theories 109
Addiction in populations 109
Box 6.1 Diffusion Theory 110

Comprehensive theories of addiction
Box 6.2 Excessive Appetites
Box 6.3 The Pathways Model of pathological gambling
What is addiction and how can we explain it?

7 Development of a comprehensive theory
A reminder of what we are talking about
Addiction as rational choice
Addiction as irrational choice
Addiction, compulsion and self-control
Addiction, instrumental learning and habit
Addiction, choice, compulsion and habit

8 A synthetic theory of motivation
The human motivational system
The five levels of motivation
The language of emotional states
Momentum and inertia
Three ways in which past experience affects motivation
The ‘representational system’ and consciousness
Self and self-control
Mental effort and motivational resources
What motivates us
The unstable mind
Flat-bottomed valleys and rating scales

9 A theory of addiction
Addiction is . . .
The pathologies underlying addiction
A return to some ‘big observations’ about addiction
The abnormalities underlying addiction
Effects of interventions
Recommendations and predictions regarding addiction interventions
Testing the theory
Conclusions

References
Index
FOREWORD
I want to thank Bill Bryson for reminding me what science is all about, and why I gave up work as a civil servant to pursue a career in research. His book, *The History of Nearly Everything*, was an inspiration to me. It reminded me that some of the most important scientific discoveries can be expressed in a few words and no mathematical symbols, that I might as well say what I really think, and, reassuringly, that I'll probably never know if my ideas had any real impact so I can at least pretend that they will once I'm dead and gone. I want to thank Lion Shahab for many ideas and for being a critical sounding board. I want to thank Mike Gossop for reminding me of the importance of the concept of habit. I want to thank Nick Heather, Keith Humphries, Jim Orford, Griffith Edwards, Andy McEwen and Christos Kouimtsidis for their insightful comments. I want to thank my sons: Jamie for being interested in my pontificating, Matthew for helping me come up with the graphic for the p.r.i.m.e. model, and Daniel for his dedication to drumming. I want to thank my wife Anne for putting up with my mental absence in the latter stages of writing this book.
Preface

I confess that I rarely read prefaces. My natural impatience impels me to turn straight to the first chapter and in any case I’m never quite sure what the point of them is. Now that I have a chance to write one, I may take a different view. Since I am writing this having just read the proofs of this volume and having given several presentations on key aspects of its content, it gives me a chance to explain some things about this book.

The end of Chapter 1 gives some kind of taste of what the theory of addiction and the book are about. The theory of addiction is in fact a theory of motivation and how the motivational system is distorted in the case of addiction. Having had a chance to try out different ways of communicating the theory I have devised another way that readers might find useful. In this scheme the theory has five key themes:

**Theme 1: The structure of the motivational system.** The theory argues that the motivational system consists of five levels: responses, impulses/inhibitory forces, motives (desires), evaluations (beliefs) and overarching plans, that all five levels are continuously subject to direct influence from our internal environment (emotional states, drives, arousal levels and thoughts) and external environment (stimuli and information), and that impulses/inhibitory forces are the final common pathway through which all higher level motivation operates.

**Theme 2: The moment-to-moment control of behaviour.** The theory notes that the motivational system is in a constant state of flux resulting from the influences within it and from the ever-changing internal and external environment. Entities such as beliefs and wants have a fleeting existence and, like memories, only occur when they are generated by whatever conditions operate at the time. What gives stability to human motivation are the brain structures and neural connections that lead to generation of similar (but not necessarily identical) states given similar conditions. Most psychological theories mistakenly ‘objectify’ concepts such as attitudes and ‘stages’ of readiness to change, ‘self-efficacy’ etc. and place too great an emphasis on their stability and permanence.

**Theme 3: Plasticity of the motivational system.** The theory draws attention to the various ways in which past experiences influence our motivational system. These are: habituation/sensitisation (lowering of heightening of responses as a result of repetition), associative learning (most notably Pavlovian and operant conditioning), and explicit memory/inference. All of these have profound effects on all elements of the motivational system. The theory seeks to frame these well-known phenomena in terms that show how habit mechanisms interact with conscious analytical processes to influence our responses.

**Theme 4: Identity.** The theory notes that our mental representations of ourselves are fundamental to our motivation. Identity influences our motivation in a number of ways. It is essential for us to have a sense of ourselves and what we want to be in order for self-control to operate. Categorical self-labels (e.g. smoker or non-smoker) to which we attach
value help prevent ‘behaviour creep’ (small deviations from desired behaviour patterns leading to larger ones). The emotions we experience when thinking about ourselves have profound motivational effects including the extent of desire for self-harm or self-protection. Finally, our ideas about what we are or are not capable of achieving (self-efficacy) influence actions that we attempt and the energy and commitment we devote to those actions.

**Theme 5: The unstable mind.** The theory proposes that the human mind has evolved to behave like a ‘fly-by-wire’ aircraft. That is to say, it is inherently unstable and exquisitely sensitive to small events and triggers. This hypersensitivity is kept in check by constant balancing input. A lack of balancing input is responsible for many phenomena observed from effects of sensory deprivation to ‘groupthink’, and in many cases, addiction. The propensity of a system to go down particular pathways, sometimes as a result of very small environmental events, together with apparently unpredictable switches of state are the province of ‘chaos theory’. It is proposed that chaos theory provides a much better system for modeling human behaviour than the more linear, box and arrow models that prevail in psychology. Put succinctly, the motivational system is more like a weather system than a plumbing system. The model provides an account of the process of lifestyle changes in terms of the development of motivational tension on which even quite small triggers than then act to create substantial changes in behaviour patterns.

I now want to make a point about the individual versus social focus of the new theory. As put forward in this book the theory focuses on the mind of the addict as the point where all the various forces promoting behaviour come together. However, I take the view that social forces of all kinds play a critical role in behaviour, and explanations at a social or cultural level are also critical to the development of our understanding. Even when they are not explicitly acknowledged in this book, social factors are catered for. For example, social reinforcers (rewards and punishments) are among the most powerful to operate on human beings. In addition our identity, which features prominently in the theory, derives in large part from our social interactions. Our social environment dictates the very way we construe our world and even what we do and do not think about. It a most basic level, if we never learn of a particular way of behaving it may never occur to us to do it. Related to this, so many of the triggers that make us think about behaviours at a given moment in time involve seeing other people doing things or being reminded of things that we may want to do. This means that explanations and predictions made using social constructs such as norms are very powerful, as are population level interventions which involve those constructs.

Turning now to the style of the book, the first thing to note is that I have tried to use repetition in the first six chapters, which cover the existing theories, as a means of helping readers who are not familiar with the concepts to grasp the ideas. This means that ideas are sometimes repeated inside and outside of the boxes that describe the theories. I have made Chapters 7 through 9, which deal with the synthetic theory of addiction, able to stand alone as far as possible so that readers who are interested primarily in the theory can read them without the need to reference the rest of the book. Again, this leads to a
certain amount repetition but for students and researchers who are not familiar with all of the disciplines covered in the book this should help with understanding.

Even as I write this I am working on better ways of communicating the theory. I am aware that many readers will not have the patience or interest to explore many of the implications of the proposals within the theory. A single sentence may be pregnant with implications left unstated. I am also aware that many readers will only want to take away from the theory some simple ideas that can be applied in their own research or practice. Therefore any readers who wish to see alternative accounts of the theory, explanations of elements of it, simplified descriptions of it or other chapters and papers relating to it can go to www.primetheory.com.

Theoretical development in the field of addiction and behavioural science may be on a cusp. If this book helps to tip us into a new way of thinking about the issues that is more integrative and less accepting of simplistic, narrow mechanistic accounts that do not fit with even common observation, so much the better.

Robert West
September 2005
Chapter 4
CHOICE IS NOT ENOUGH: THE CONCEPTS OF IMPULSE AND SELF-CONTROL

This chapter introduces the concept of impulses, urges, inhibitory forces, self-control and voluntary restraint. These concepts are required to explain the experiences of addicts who make an effort to exercise restraint and whose failure is often not a simple 'change of mind'. It introduces evidence that, at least in some cases, addiction is accompanied by impaired performance of brain pathways involved in response inhibition. The concepts on pathological changes in motives and states that impel and restrain behaviour provide a way of explaining patterns of behaviour that do not fit a simple choice model, however irrational that choice may be.

The last chapter took us from a Rational Choice Theory of addiction to an Irrational Choice Theory; but addiction was still conceived of as a choice process: a weighing up of the costs and benefits of an action and selection based on what is perceived to be the best outcome at the time. Under this view the idea that the behaviour is out of control is an illusion based on a failure to appreciate that the expressed desire to stop doing something at one time does not reflect the preferences operating at a later time after the attempt at restraint has begun.

Reports of feelings of compulsion

The problem with this view is that it does not accord with the experience of many addicts. At the point where they find themselves about to relapse back to their old ways, they frequently report a feeling of compulsion that is distinct from simple desire. It is not even that it is a 'strong desire': it is an urge that they are trying to resist. It also leads to neglect of the panoply of observational and research evidence for the importance of a failure of impulse control in the development and maintenance of addiction.

The concepts of compulsion, craving and self-control dominate what has been termed the Disease Model of addiction (see Jellinek 1960) (Box 4.1). This model proposes that the pathology underlying addiction involves changes in the brain that lead people to do things against their will.
Powerful motives versus impaired control

Now would be a good point to bring in a distinction that has not figured highly in the theories considered thus far but is made in theories that we will consider later that are dominant in the discipline of behavioural pharmacology. The distinction is between compulsion that arises because the impetus to engage in a behaviour is so strong versus compulsion that arises because the ability to exercise restraint or control is so weak.

To put it crudely, the question is how far an addict is someone who has such a strong desire for a drug, or indeed urge to use a drug, that it overwhelms powers of restraint that are unimpaired versus someone who has an admittedly strong desire or urge for a drug but an impaired ability to resist that desire. The definition of addiction as ‘impaired control’ implies the latter but most of the theorising on the subject assumes the former. However, in the seminal formulation of Edwards and Gross (1976) of the Disease Model of alcohol dependence, the emphasis on failure to exercise control is evident, as, incidentally, is the philosophical difficulty in proposing a lack of choice. Edwards and Gross (1976, p. 1060) state: ‘It is unclear, however, whether the experience is truly one of losing control rather than one of deciding not to exercise control.’

Box 4.1 The Disease Model of addiction

The Disease Model of addiction states that addiction involves pathological changes in the brain that result in overpowering urges.

The Disease Model of addiction seeks to explain the development of addiction and individual differences in susceptibility to and recovery from it. It proposes that addiction fits the definition of a medical disorder. It involves an abnormality of structure or function in the CNS that results in impairment (Jellinek 1960; Gelkopf et al. 2002). It can be diagnosed using standard criteria and in principle it can be treated.

Loss of control

Under this view an addicted individual will express a sincere desire to stop engaging in their addictive behaviour and will show every evidence of making strenuous efforts to stop doing it at the same time as s/he carries on nevertheless. In fact the loss of control is manifest over short and long time spans. Over a period of a few hours an alcoholic may begin a drinking session with the intention of having one or two drinks but finds that, according to his way of thinking, it is impossible to stop at that and more and more drinks are consumed: the power to resist has gone.

Over a longer time span, the alcoholic, or indeed the smoker, gambler or other drug addict, formulates a plan not to engage in the activity but after
a time (usually a short time) does in fact engage in the activity. The addict chooses to do one thing but does something else.

The importance of craving
At the heart of this theory is the concept of ‘craving’. In the disease model this has been defined as an ‘urgent and overpowering desire’ (Jellinek 1960). One way of thinking about this is as a feeling that impels the addict to take whatever steps are necessary and feasible to achieve the object of the addiction. However, it might even be proposed that it is a motivational state that goes beyond feelings: it overwhelms the individual in totality, dominating the thoughts, feelings and actions of the individual to the exclusion of all else.

This theory captures what seems to be the central phenomenology of addiction: a desire that is so strong and all-encompassing that it sweeps all other considerations before it in a myopic and single-minded search for the object of that desire. Even if in some sense there is a choice, it does not seem like it to the addict or to observers, and in the common understanding of the term there is no real choice, there is compulsion.

Self-cure
An observation that on the face of it poses difficulties for the disease model is that many ‘addicts’ stop engaging in the activity concerned without apparent difficulty. For years their behaviour has shown all the signs of compulsion but then one day they decide that enough is enough and cease.

It can only be presumed either that whatever abnormality there was in the individual’s brain that led to the compulsion suddenly normalised or that the individual was never addicted in the first place. Both of these are just about possible at least for some individuals. It is also possible that this phenomenon is not quite what it seems: that heavily addicted individuals do not actually recover spontaneously in this way and that a distinction needs to be made between individuals who are heavy and regular users of a drug or who engage in an activity frequently but who are not addicted.

Issues and evaluation
Just as Rational Choice Theory can be regarded as misleading in implying that addicts are merely exercising preferences, the Disease Model has been criticised as misleading in implying that addicts are impotent onlookers and the only way of stopping them doing it is physical restraint (Skog 2000).

As with the theories reviewed in the previous chapter, the main problem with this theory is observations that it does not account for. By focusing on compulsion, there are many aspects of addiction that it does not encompass. Specifically it does not begin to address the issues of choice and identity that were the insights presented in the previous chapter.
The question of why some people become addicted to particular activities while others do not is a thread running through all the preceding discussion. We have postulated that it may relate to a host of different factors that make the addictive behaviour more rewarding or abstinence more distressing.

**Personality and addiction typologies**

One model that focuses specifically on personality as a predisposing factor in addiction is Cloninger’s Tri-dimensional Theory (Cloninger 1987). It proposes three dimensions of personality: novelty seeking, harm avoidance, and reward dependence (see Box 4.2). The potential link between these dimensions and susceptibility to addiction are striking so it is surprising that research has not provided greater support for the theory (Howard et al. 1997).

**Box 4.2 Tri-dimensional Personality Theory**

*Cloninger’s Tri-dimensional Personality Theory argues that three fundamental dimensions of personality have direct implications for susceptibility to addiction and can also be used to divide addicts into subtypes (see Cloninger 1987).*

**Dimensional theories of personality**

Cloninger’s Tri-dimensional Personality Theory is an example of an approach to personality and its assessment that postulates that a small number of hypothetical dimensions of mental functioning explain a large amount of the variation that exists in the way in which people respond to their environment. It is from the same mould as Eysenck’s Three-factor Personality Theory (see, for example, Roos 1977a,b) and McRae and Costa’s Five-factor Theory (see, for example, Conway et al. 2003; Miller and Lynam 2003), both of which have also been used to try to explain susceptibility to addiction.

**Novelty seeking, harm avoidance and reward dependence**

The Tri-dimensional Personality Theory proposes three fundamental dimensions: novelty seeking, harm avoidance and reward dependence (see Cloninger 1987). It is argued that the interaction between these dimensions leads to different patterns of responses to novelty, punishment and reward, and that this has implications for dependence on alcohol and drugs. It is further proposed that there are two fundamental subtypes of alcoholic (Type I and Type II). These two subtypes differ according to the age at which alcoholism develops; the relative contributions of predisposing genetic and environmental factors; the gender and personality traits of the alcoholic; and whether co-occurring psychiatric disorders, such as antisocial behaviour, are present. It is proposed that Type I alcoholics have later onset, less genetic involvement, are more likely to be female and have fewer problem behaviours.
Evidence

In a review of studies that have examined Cloninger’s theory and its associated questionnaire assessment, novelty seeking was found to distinguish alcoholics from non-alcoholics and Type II from their Type I counterparts, and smokers from non-smokers. Tri-dimensional traits independently predicted early-onset alcohol abuse and serious delinquency and were significantly associated with concurrent substance abuse among adolescents. The review also found that most studies that compared non-alcoholic youth with positive and negative family histories of alcoholism reported non-significant differences between scores on a questionnaire designed to assess the three personality dimensions. Few alcoholics, cigarette smokers or sons of alcoholics displayed Type I (low novelty seeking, high harm avoidance, high reward dependence) or Type II (high novelty seeking, low harm avoidance, low reward dependence) personality profiles.

Thus novelty seeking does predict early onset alcohol abuse and criminality and discriminates alcoholics exhibiting antisocial behavior and persons with antisocial personality disorder (ASPD) from their non-antisocial counterparts. Findings for the harm avoidance and reward-seeking dimensions are less consistent though there is some support for the role of higher levels of harm avoidance in intensity of substance use (Howard et al. 1997).

More recently Sannibale and Hall (1998) evaluated Cloninger’s typology of alcoholism in a sample of 300 Australian men and women with a lifetime diagnosis of alcohol abuse/dependence. They found that the questionnaire measure used classified only 18% of the sample into either Type I or Type II. More women than men were classified as Type I (19% vs. 6%) but, contrary to expectations, similar numbers were classified as Type II problem drinkers (7% vs. 4%). As predicted, Type II problem drinkers had more symptoms of ASPD, more social consequences of drinking and higher sensation-seeking scores than Type I problem drinkers.

Issues and evaluation

Cloninger’s typology of alcohol dependence is of importance primarily because it seeks to ground a theory of addiction in a more general theory of personality. It also recognises and attempts to systematise the heterogeneity that exists even within dependence on a particular substance. It clearly captures some important distinctions but evidence has generally failed to support its broader contentions.

Other typologies have also been proposed that are quite close to Cloninger’s. Most notable is Babor’s classification into Type A and B alcoholics (see Babor et al. 1992; Litt et al. 1992; Brown et al. 1994). This typology was based on a cluster analysis of alcoholics in treatment rather than derived from a personality theory. The two types of alcoholic differed across 17 characteristics. Type A alcoholics were characterised by later onset, fewer childhood risk factors, less severe
dependence, fewer alcohol-related problems, and less psychopathological dysfunction. Type B alcoholics were characterised by childhood risk factors, familial alcoholism, early onset of alcohol-related problems, greater severity of dependence, polydrug use, a more chronic treatment history (despite their younger age), greater psychopathological dysfunction, and more life stress. The Type B alcoholics were less likely to be successful after treatment.

**Self-efficacy**

In the previous chapter the concept of ‘self-efficacy’ was alluded to but not discussed in detail. Self-efficacy, like addiction, is a social construct, and has been defined in different ways. In general it reflects our confidence in being able to achieve certain outcomes and has been proposed as playing a major role in changing behaviour. Put simply, if we do not think we can do something, we do not try or if we do try we give up easily. This concept can be fitted into a simple choice framework as a factor entering into the cost–benefit analysis. However, it probably fits a little better in the present discussion about compulsion and control (Box 4.3).

---

**Box 4.3 Self-efficacy Theory**

Overcoming addiction is related to the extent of an individual’s beliefs that they can organise and carry out activities in order to exercise restraint or achieve abstinence.

Self-efficacy is the level of an individual’s confidence in his or her abilities to organise and complete actions that lead to particular goals (Bandura 1977; Bandura et al. 1977). Four main hypotheses form the core of the theory:

- Levels of self-efficacy affect the goals that people pursue.
- Self-efficacy affects the level of effort used to achieve those goals.
- Self-efficacy affects how long people will persevere in pursuit of their goals when encountering barriers.
- Self-efficacy affects the likelihood of the goal being achieved.

Self-efficacy may be influenced by the success or failure that an individual has previously experienced on the task in question though it is not the only source of influence. Thus it should, according to the theory, predict future behaviour over and above what is known about past experience.

*This concept forms a part of many theoretical accounts including social learning theory and the abstinence violation effect described later. As with other theories it is drawn out here for the purpose of exposition.*
Generalised versus specific self-efficacy
Self-efficacy can be related to specific tasks (such as stopping heroin use) or more general. Self-efficacy can extend beyond behaviour to a person’s level of perceived control with regard to the individual’s thoughts, feelings and environment. The original Bandura Theory of Self-efficacy has been applied to addictive behaviours with some adaptation (see Marlatt 1996). It has been suggested that reduced self-efficacy may underpin loss of control in addiction (Brandon et al. 2004).

Marlatt (1996) has classified efficacy judgements in relation to the stages of drug and alcohol misuse. Resistance self-efficacy beliefs are the person’s own judgements of their ability to avoid substance abuse prior to its first use. Harm reduction self-efficacy beliefs involve judging one’s ability to reduce the risks of the drugs once addicted.

Craving and self-efficacy
Craving and self-efficacy are postulated by Marlatt to be reciprocally related, with high levels of craving being the most disruptive to the addict’s coping skills. Indeed, self-efficacy has been found to be inversely proportional to cigarette craving (Niaura 2000) and has also been found in some studies to be a predictor of success or failure of smoking cessation attempts even after controlling for concurrent smoking (Gwaltney et al. 2001).

Changes in self-efficacy
It is thought that the act of giving up a drug has a positive impact on the individual’s levels of self-efficacy. Carey and Kalra (1993) examined data from a sample of smokers over a 12-month period. Those who had successfully quit for 12 months had increased levels of self-efficacy, whereas those who continued smoking, or even attempted abstinence but relapsed, had decreased self-efficacy.

This has obvious parallels with the Identity Shift Theory described in the previous chapter. Self-efficacy ratings have also been reported to increase after an initial dose of methadone, to be unchanged when the individual is maintained on a stable dose of methadone, and to decline during the weaning stage (Reilly et al. 1995).

Issues and evaluation
The ideas behind Self-efficacy Theory fit well with natural observations and with a simple Rational Choice Theory of behaviour. It also seems appropriate to focus special attention on self-efficacy because of the possibility that it may exert a large influence on addictive behaviour.

Unfortunately, the evidence to date has not established how important self-efficacy per se is in maintaining addiction. For example, consider someone who has all the signs of being heavily addicted to alcohol but is utterly confident that he could control his drinking whenever he so chooses. Would that
confidence in itself be enough to enable him to overcome his addiction? This does not appear to have been tested but it seems unlikely.

Similarly, if we take a sample of smokers and assess their level of addiction carefully without reference to any beliefs about their ability to stop smoking, including observations of their past experiences of attempts to stop, would adding self-efficacy beliefs into the model substantially improve on the prediction of their ability to stop? Perhaps most importantly, we need evidence that an intervention that raises self-efficacy has a substantial impact on ability to exercise restraint.

The transition from lapse to relapse

We noted that the concept of self-efficacy permeated many of the choice-based models described in the previous chapter. It is also an important element of a theory that focuses on a particular phenomenon in addictive behaviour: the process by which a single lapse back to an old pattern of behaviour leads to full resumption of that behaviour. This process has been called the Abstinence Violation Effect (Box 4.4)

Box 4.4 The Abstinence Violation Effect

Precipitants of relapse are internal, stable and global attributions as to the causes of initial lapses – relapse is a learning experience that occurs due to inadequate coping resources.

The Abstinence Violation Effect (AVE) proposes that internal, stable and global attributions for the cause of a lapse of abstinence and concomitant feelings of guilt and loss of control increase the probability of a return to regular substance use. Cognitive factors play a crucial role in the likelihood of relapse. Marlatt argues that the Abstinence Violation Effect is a particularly destructive cognitive process (Marlatt 1979).

Genesis of the AVE

The AVE occurs when an individual views his or her drug use as a deviation from his or her commitment to absolute abstinence that elicits a state of ‘cognitive dissonance’. The individual attempts to resolve this dissonance by assuming that some intrinsic personal quality makes abstinence impossible (‘personal attribution’). Interpretations and attributions can then undermine future attempts at maintaining abstinence.

Effects of the AVE

The theory also has implications for how failure of an attempt at abstinence influences subsequent attempts. It argues that one of the most constructive
ways in perceiving relapse, for the individual at least, is to identify circum-
stantial factors that made it difficult to resist, permitting contingency plans
for the future to be developed. The AVE conceptualises relapse as a learn-
ing experience and that the individual’s reaction and appraisal of the lapse
will determine future commitments to abstinence.

Empirical evidence
It has been documented that alcoholics, smokers, opiate addicts, gamblers
and overeaters are more likely to lapse when they experience negative
emotional states (e.g. Cummings et al. 1985). Such findings support the propo-
sition that addictive behaviour is often engaged in because of its effective-
ness in controlling stress. Evidence for the importance of coping skills is
provided by Miller et al. (1996) who found that these were the strongest
predictors of relapse in a sample of alcoholics receiving outpatient care.

Walton et al. (1994) examined the role of attributions in the lapse and
relapse process following substance abuse treatment. In a study of inpatients
who had undergone 6 months of treatment they looked at the attributions
made by recovering drug users who were tempted to lapse but reminded
abstinent in comparison with those who lapsed and those who relapsed.
Predictions made by the Abstinence Violation Model were not supported in
so far as lapsers and relapsers were similar in terms of internal/external attrib-
utions following a return to drug use. However, relapsers made more stable
and global attributions compared with lapsers, and abstainers made more
internal and stable attributions regarding their abstinence, compared with
lapsers following their slip. Abstainers’ attributions for success in remain-
ing abstinent tended to be similar to the attributions made by relapsers for
their failure to remain abstinent (that is, for their relapse). These findings
highlight the complexity in their attribution process in early recovery.

Shiffman and others (1996) found in a study of smokers that the indica-
tors of the AVE, namely self-efficacy, attributions and affective reactions to
the lapse, generally failed to predict progression to relapse, but participants
who attempted restorative coping following a lapse were less likely to lapse
for a second time. Those whose lapses were triggered by stress progressed
more quickly on to a further lapse whereas those triggered by eating or drink-
ing or accompanied by alcohol consumption progressed more slowly. Those
who were heavily nicotine dependent were more likely to have a second lapse,
but neither the amount smoked in the first lapse nor its subjective reinforcement
predicted progression.

Birke et al. (1990) used data from a sample of alcoholics and cigarette
smokers to study the occurrence of the AVE predicting the likelihood of
‘full blown relapse’ following an initial lapse. No significant difference was
found in the attribution style of abstainers and relapsers. Negative affect (such
as anxiety or depression) and interpersonal conflict were found to be important
precipitants of relapse while social pressure was not found to be linked
to relapse.
Ross et al. (1988) examined the assumption that self-efficacy plays an important role in the resistance to relapse in the AVE. It was thought that combinations of unrealistically high standards and low self-efficacy for following those standards may predict the risk for relapse. Newly admitted alcoholics and individuals who had remained abstinent for 1 year showed no difference in their self-imposed high standards, but successfully abstinent alcoholics were found to have higher self-efficacy expectations than alcoholics who were early in the recovery process.

Positive findings have been reported from a study testing the AVE hypotheses in a sample of marijuana users who reported a lapse back to marijuana use following completion of either relapse prevention treatment or a social support group promoting abstinence (Stephens et al. 1994). It was found that more internal, stable and global attributions for the cause of the lapse and perceived loss of control were related significantly to concurrently reported lapse. Furthermore, internal and global attributions for lapses predicted marijuana use during the subsequent 6 months.

**Implications and interventions**

The Relapse Prevention Model has been developed out of the AVE concept. In this model no single treatment or package is endorsed owing to the diversity of attributions that may be attached to maintaining abstinence on an individual level. Instead, it is proposed that the therapist gain an overview of the wide range of different factors that can influence attitudes and expectations about a variety of drugs, and be willing to approach each individual’s dependence with an open-minded view about the underlying factors.

The relapse prevention framework identifies four elements for developing an appropriate package based on the components of AVE:

1. An in-depth assessment of the individual’s risk factors and discriminative stimuli (cues), as well as their existing coping skills and resources. The level of assessment required will be extensive, although further details will emerge and need to be added to the review over the course of the treatment.
2. Assisting the addicted individual in finding appropriate alternative strategies for avoidance or coping with risk situations. Strategies may consist of cognitive techniques (e.g. talking through difficult issues in order to identify the need and type of strategies required) and behavioural techniques (e.g. relaxation, distraction).
3. Enhancing the addict’s self-efficacy and skill in using alternative strategies, for example by assisting him or her to practise them and thereby identify and tackle potential difficulties.
4. Preparation for how to deal with a lapse may require discussion of the AVE, together with the development of a structured plan of what to do in such an eventuality (e.g. contact a particular person, go somewhere private for a period of reflection).
These four elements can take many different forms, depending on the characteristics of the therapist, the patient and the resources available.

Motivational interviewing
The relapse prevention approach has spawned a counselling technique known as ‘motivational interviewing’ (Miller and Johnson 2001). The idea of motivational interviewing is to engage the individual with the idea of sustaining and committing to their behaviour change in the longer term. This level of intervention relies on a dialogue between the therapist and the individual and a commitment to working together to identify risky situations and other problem areas and evaluate potential coping strategies in a supportive environment.

There is evidence that the motivational interviewing technique that is based on the relapse prevention model is of value (Rubak et al. 2005). For example, Allsop et al. (1997) randomised a sample of alcoholics to either a standard care package of basic drugs education, a relapse prevention package or extra discussion sessions. Those receiving the relapse prevention package showed significantly higher abstinence rates and spent longer drug free before relapse.

Issues and evaluation
Although not all of the postulates of the theory have been supported, the AVE theory captures some important features of the relapse process and accords well with observations in the real world. Its main tenets would need to be encompassed within any general theory of addiction. It is important to take note of Miller’s admonition, however (Miller 1996), that the concept of relapse itself is problematic and an oversimplification of a complex and highly varied process of transition from abstinence to resumption of an addictive behaviour.

Impulse control
The concepts of self-efficacy and the Abstinence Violation Effect draw attention to beliefs and feelings that undermine attempts at change. It has been noted that impulse control problems as a personality type are associated with extent of addictive substance use (Conway et al. 2003). The failure of self-control has been explored in a more general way in a number of theories. One of these (Box 4.5) describes links between neuroanatomical structures and inhibitory mechanisms.

Box 4.5 Inhibition Dysregulation Theory
Addiction involves a progressive dysregulation of ability to inhibit behaviour that is rewarded (Lubman et al. 2004).
Lubman et al. (2004) present an argument that suggests that the inhibitory system involving brain regions involved in aspects of response inhibition and selection underlies compulsive behaviours associated with drug addiction. They are not suggesting that addicted individuals are automatons under the direct control of substances or related stimuli acting on the brain, but rather that aspects of decision-making are compromised in perhaps either a direct way (i.e. a dysfunctional inhibitory system) or indirectly via a dysfunctional reward system.

**Commonalities between addiction and other disorders of control**

It is noted that substance abuse is common in patients suffering from schizophrenia, depression and obsessive compulsive disorder (OCD) (Conway et al. 2002). It is also noted that inhibitory processes involving frontal cortical structures are deficient in patients with schizophrenia, bipolar mood disorder, attention deficit hyperactivity disorder and OCD (Murphy 1990; Bannon et al. 2002). Some aspects of addiction appear quite similar to OCD (Modell et al. 1992) as clinical descriptions of both disorders describe an inability to inhibit intrusive repetitive thoughts (i.e. obsessions or cravings) and ritualistic, regimented behaviour (compulsions or drug-taking behaviour) and both result in significantly impaired functioning.

**Brain regions involved in inhibition**

Significant under-activity has been found in the orbitofrontal cortex and anterior cingulate cortex of cocaine addicts and alcoholics who continue to use and in those abstinent for long periods (Volkow et al. 1997). These regions become highly active during cue exposure (Grant et al. 1996) and during acute withdrawal (see Volkow et al. 1997).

The theory proposes that compulsive behaviour requires dysfunction within two highly interconnected cortical systems, the anterior cingulate cortex (ACC) and orbitofrontal cortex (OFC) which are critically involved in self-regulation and together form the core of the inhibitory system. It is suggested that increased incentive states alone are not sufficient for compulsive behaviour to occur, but require dysfunctional inhibitory processes within the OFC and ACC.

**Failure of inhibition and relapse**

The Inhibition Dysregulation Theory appears to be supported by the findings of Miller and Gold (1994) that craving was only cited in 7% of cases as a primary factor for relapse, with 41% citing impulsive action (i.e. reduced inhibitory control). It has also been suggested that a failure consciously to consider future consequences of behaviours (Altman et al. 1996) may be associated with the underactivity of the inhibitory system.

It is hypothesised that the inhibitory system is overwhelmed by motives, resulting in the release, or disinhibition, of behaviour that is unduly dominated by ‘pre-potent’ and ‘stimulus-driven’ tendencies in the presence of
addiction-related provocation. Lubman et al. (2004) explain further that this provocation leads to impulsivity (experienced as loss of control) with little consideration of adverse future consequences, resulting in recurrent compulsive drug taking. It is argued that clinical outcomes may be predicted more accurately by systematically probing the integrity of the inhibitory system under real-life motivational states, rather than measuring craving levels or inhibitory functions alone.

**Interventions to enhance inhibitory control**

Failure of inhibitory control is the basis of many self-help clinical interventions such as Alcoholics Anonymous, and overcoming this is a feature of the technique known as ‘motivational interviewing’ mentioned earlier. Pharmacotherapeutic approaches that target aspects of dependence such as substitution treatments or withdrawal-oriented therapy are also argued to be consistent with a model of inhibitory dysregulation.

**Inhibition dysfunction versus tolerance and withdrawal symptoms**

This theory is a move away from the tolerance and dependence explanations of addiction towards a behavioural, compulsive drug-seeking explanation, reinforced by the effects of the drug and the struggle for abstinence. Lubman et al. (2004) attempt to bring together disparate findings involving neuroadaptation and sensitisation of the dopamine reward system (see next chapter), drug cue reactivity (see next chapter) and malfunction of the inhibitory system to create an integrated model.

**Issues and evaluation**

We have located this theory in a chapter that introduces the concept of compulsion and self-control, but, like many of the theories reviewed thus far, it incorporates elements that include conscious choice, already discussed, and habit elements that will be considered further in the next chapter. What is clear is that there is evidence of a failure of inhibitory mechanisms in the development of at least some cases of addiction, and we have the beginnings of an understanding of the neurological substrate of this.

**Self-regulation as a broadly based concept**

An account of self-regulation in all its aspects is set out by Baumeister and colleagues (1994) (Box 4.6). This wide ranging review presents a persuasive case that self-regulation at many levels, from conscious restraint to response inhibition, plays a critical role in the balance of forces that operate on behaviour in all its manifestations. It notes that self-control is one aspect of wider self-regulation in which we self-consciously exercise our will in order to achieve particular goals.
Box 4.6 Self-regulation Theory

Actions arise from a hierarchy of multiple processes that may be in competition with each other. Higher processes involve more complex networks of meaningful associations and interpretations, and more distal or abstract goals. Self-regulation involves higher processes overriding lower processes. Self-regulation failure occurs when lower order processes win through.

Among the various theories that focus on impaired control or compulsion, Baumeister et al. (1994) have produced a highly developed account that emphasises the commonalities between addiction, poor self-management, obsessions, eating too much, aggression and many other behavioural problems. This theory allows for the possibility that there are stable individual differences in propensity to exercise self-control that may predate and contribute to development of addiction. Like the dysregulation of impulse control theory described earlier, it also allows for the possible effects of drugs of dependence on self-regulation and it also examines the effects of short-term influences such as tiredness, emotional state and environmental stimuli on self-regulation.

Issues and evaluation

The theory has enormous explanatory potential, for example with regard to the link between antisocial personality and addiction, mental illness and addiction, associations between different forms of addiction, and indeed the process of recovery from addiction.

It seems that we must include abnormalities of self-control in the theory of addiction. As noted earlier, the phenomenology of addiction also appears to involve a sense of urge or compulsion that may go beyond a weakening of impulse control mechanisms. It is now time to explore the concept of ‘urge’ in more detail.

Urges and craving

The term ‘urge’ has often been used synonymously with ‘craving’ but unfortunately this latter term has acquired many different meanings and this has tended to cloud the debate (Kozlowski and Wilkinson 1987). In common parlance ‘urge’ is a feeling of being impelled to do something. It is not necessarily the same as wanting to do it but often the two go together. A simple example of the difference between the two would be something like: ‘Sammy had a strong urge to go to the bathroom but wanted to carry on playing on his computer’. Tiffany (1990) has developed a theory about how urges develop and what role they play in maintaining drug use (Box 4.7).
Box 4.7  A Cognitive Model of Drug Urges

The Theory of Drug Urges proposes that compulsive drug use involves more than subjective feelings of craving. It involves enactment of highly automated action sequences that are driven by cue–response associations. Craving should be viewed as two related dimensions. One arises from a conscious attempt to block automated action sequences. The other is anticipation of pleasure from the behaviour (Tiffany 1990, 1999; Tiffany and Conklin 2000).

The theory proposes that craving represents the addict’s effortful cognitive processing devoted to interrupting drug use. It arises from attempts to block highly automated action sequences that are learned through repetition. Under this view the addict’s intention regarding whether or not to use drugs should influence features of reported craving. The model predicts that desire and intent to use drugs will be strongly ‘coupled’ in active smokers. However, desire and intention to smoke may become ‘uncoupled’ in individuals who are trying to quit smoking.

Two dimensions of urge

According to the theory, craving should be divided into two dimensions. One dimension is a feeling of urgent need linked with withdrawal symptoms and the other arises from expectation of pleasure from the activity concerned (Tiffany and Drobes 1991). Studies with smokers during ad lib smoking and abstinence have been claimed to support this view (e.g. Cox et al. 2001). However, the type of evidence used is only weakly related to the hypotheses.

One line of evidence comes from ‘factor analysis’: a statistical method for assessing the extent to which scores on a number of measures (e.g. ratings given in response to questionnaire items) can be explained in terms of one or more underlying dimensions.

Factor analysis has been applied to responses to a questionnaire devised to test the theory, the Questionnaire of Smoking Urges (QSU). Unfortunately, when used with questionnaires this statistical method is highly sensitive to the specific question wording to the extent of semantic overlap between questions, and to the sample and the circumstances in which the measurement is made. In addition it does not give a clear decision as to how many underlying dimensions there are: more or less arbitrary rules have to be devised to make that determination.

In the case of the QSU, proponents of the theory claim that there are two underlying dimensions that are correlated with each other. However, one could argue that there is just one underlying dimension on which is superimposed some additional systematic variation attributable to similarities in wording and style of the items.

The second line of evidence is that the two scores derived from the QSU respond differently to interventions that affect craving in different ways. Unfortunately the tests of this have not used the necessary statistical procedures.
that would tell us whether the pattern of responses are truly different or whether the difference could have occurred by chance. Thus it is not enough to show that an intervention significantly affects one scale and not another. It is necessary directly to show a statistically significant difference in the effect of the intervention on the two scales (i.e. an interaction).

**Automaticity and addiction**
As regards the part of the theory related to automated action sequences, the theory is designed to explain the relationship between environmental cues and reports of craving and the fact that relapse to an addictive behaviour often occurs in the absence of cravings. Research has shown that although absent-minded lapses to smoking do occur they are rare (Catley et al. 2000).

**Issues and evaluation**
It is clear that this theory, like others, stems from a need to integrate cognitive processing models with motivational systems that do not involve conscious awareness. The theory recognises that urges to engage in addictive behaviours do not simply derive from the anticipated pleasure that these will provide. The theory makes an important point about the urges potentially arising from an attempt to frustrate an automated action sequence. In this respect it is the flipside of the ‘restraint’ coin.

**Addiction as a failure of self-control over desires and urges**
We have now arrived at a theory of addiction in which we have added the concepts of self-control and compulsion to an irrational choice theory with unstable preferences. This is still a theory in which conscious choice is the final common pathway. Many of the theories reviewed thus far (including the last one reviewed) have included non-conscious, automatic or semi-automatic processes.

The reason for this is that there are still anomalies in a choice-type model. One is the observation, albeit not common, that addictive behaviours sometimes occur without conscious awareness being directed at them (e.g. lighting up a cigarette without thinking about it). More importantly, there seems to be a mismatch between the degree of urge to engage in some addictive behaviours and the apparent rewards that stem from them. The next chapter brings in the field of behavioural pharmacology as a possible means of addressing this weakness.
Chapter 3
BEGINNING THE JOURNEY:
ADDICTION AS CHOICE

This chapter starts the journey to the development of a comprehensive theory of addiction. It begins with the simplest common-sense model of rational, informed choice with stable preferences, moves on to a model of choice with unstable preferences, and then considers ways in which the choice might be irrational. Regarding addiction as a choice process goes a surprisingly long way towards explaining the major phenomena and most theories of addiction, and attempts at combating addiction derive from this approach. However, we have to conclude at the end of the chapter that addiction cannot be exclusively construed in terms of choices.

Addiction as a rational, informed choice based on stable preferences

Having established in the previous chapter broadly what it is we are talking about, let us now start our journey of trying to explain it. The most obvious starting point is a Rational Informed Stable Choice (RISC) model of behaviour.

Addiction as cost–benefit analysis

Under this model, we do things because we expect them to confer benefits and we know about and are willing to accept the adverse consequences. For most of us, this is the default model for explaining our own behaviour and that of other people. It provides the starting point for our thinking. Under the most extreme version of this theory there is no abnormality in the mental functioning of the addict, no disorder: there only appears to be. When we see a heroin addict living in squalor and suffering ill-health, we wrongly assume that this person would prefer not to live like this and only does so because s/he has lost control over the activity.

The importance of the addict’s eye view

Under this view, it may be that the addict would prefer to live differently, but among the options that s/he sees as actually open to him or her this is judged to be the best on offer at the time. When we see the alcoholic sleeping on the street, living a life of degradation in a drink-fuelled haze with the likelihood of an early death, it is wrong to assume that s/he does so because s/he cannot stop drinking. This life
may be preferable to his or her own life without alcohol. When we see smokers puffing on a cigarette in the cold outside the back door of their office building, risking lung cancer, heart disease and chronic bronchitis, it is wrong to believe that they do so because they cannot stop. Under a RISC model they prefer the life of a smoker to the alternative that is open to them – their own life as a non-smoker.

When we see the gambler who has lost his or her family and is living in debt to loan sharks but keeps returning to the betting shop in the hope of a big win that when it comes just provides more stake money, it is wrong to assume that there is something wrong with him or her. The behaviour seems to be out of control but according to a RISC analysis s/he prefers to live this way than what for him or her becomes more likely. On the other hand, I know that my activity [is causing problems for myself/is causing problems for other people/is viewed as morally wrong] so it serves me to present the activity as being outside my control.

‘Do you want to stop?’

According to this view it is misleading to ask the smoker or the alcoholic if they want to stop smoking and to use a contradiction between their responses and their behaviour as evidence of addiction. The smoker may well say that s/he wants to stop smoking, just as s/he may say that s/he would like to be a multi-millionaire. There are lots of things we would like. The real question is, ‘Does the prospect of becoming a non-smoker and all that this entails appeal to you more than continuing, for the time being, your life as a smoker?’ If the smoker says ‘Yes’ and continues to smoke, there would be at least a prima facie case against the RISC model.

Loss of control is a matter of perspective

According to this model the concept of loss of control is an illusion that stems from the failure of perspective on the part of onlookers. We see the life the addict leads and we imagine a life that would be so much better if the person were not engaging so frequently in the addictive behaviour.

However, we forget that the alternative life we imagine for the addict is not the alternative life the addict imagines for himself or herself. The pleasure or escape
that the addict obtains from a drug is worth whatever short- and long-term costs there might be for that individual. If a drug is particularly pleasurable for an individual, it could be worth the risk of an early grave. Even if a drug is not particularly pleasurable, if the addict’s emotional state or life circumstances as an ex-drug user are bleak and unhappy, it would be quite rational to choose to remain a user.

**Rational addiction theories**

There are a number of theories that fall within the Rational Choice model of addiction (see Vuchinich and Heather 2003). Box 3.1 gives an interesting example which highlights the observation, undoubtedly valid, that addiction is a social construct that serves a particular purpose for particular individuals at particular times. People may label themselves as addicted on one occasion and not on another depending on the benefits or costs of attaching these labels. However, it only highlights one feature of the application of the label ‘addiction’ and if it were proposed as a theory of addiction would fall foul of strong contradictory evidence as discussed later.

### Box 3.1 The myth of addiction

According to John Booth Davies (Davies 1992), addiction is a myth or legend similar to ‘possession’ that serves particular functions for society. Davies notes that the language that addicts use to describe their behaviour varies depending on the person to whom they are talking. If they are talking to health workers or the police, they use the language of addiction; their behaviour is out of control and they cannot help themselves.

In this way they minimise blame and punishment and maximise their chances of forgiveness, understanding and help. When so-called addicts talk to their peers they use a different kind of language that would be hard to distinguish from the language they use to talk about other behaviours such as shopping. They are exercising preferences that are understandable and rational given the kinds of people they are and the circumstances in which they live.

Attributing drug taking or other damaging excesses to addiction also serves the needs of people other than the addict. It serves the needs of those in the professions whose living depends on treating addicts as though they were ill. It serves the emotional needs of families of addicts who find it difficult to accept that someone they care for is behaving in a way that is harming themselves and others and otherwise would be morally wrong. It serves the needs of politicians and pundits who prefer not to make moralistic judgements about people.

### Issues and evaluation

This theory highlights an important feature of addiction that is easily overlooked: that the terminology used serves a function for the user. In this case, attributing certain activities to addiction enables the addict or other members...
of society to meet their physical or emotional needs regarding help and understanding rather than blame and personal responsibility. However, denial of the reality of the phenomena of addiction, including the feelings of craving and compulsion are contradicted by observation. These phenomena are not myths.

The RISC model can perhaps explain why drugs such as heroin and cocaine have a strong addictive potential: it is just that they are very pleasurable. It can explain why heroin use is so much less common than, say, marijuana use: it may be partly because of the greater severity of legal sanctions but is also explained by a greater fear of the adverse consequences – most people see heroin as a dangerous drug that they do not even want to try. It can explain why some people are more susceptible to heroin addiction: perhaps they place greater value on immediate pleasure or their lives without heroin are more bleak and meaningless.

It can perhaps explain why activities such as smoking that are only mildly pleasurable appear to be addictive: while young the prospect of possible death and illness in the distant future is not a sufficient deterrent to overcome the enjoyment experienced in the present; when old and infirm there is no reason to stop smoking because the damage is already done. Thus the costs of smoking are not judged to be particularly great early on, and later on it is too late to do anything about it.

Economists have developed a number of models that attempt to explain addiction as a societal phenomenon. These models are typically choice models and stem from a fundamental precept of economic theory, that at a population level consumer choices can be understood in terms of maximising ‘utilities’. That is, consumers will expend resources according to their judgements about benefits to them of the goods they are acquiring.

One of the most widely cited economic models of addiction is Becker and Murphy’s Theory of Rational Addiction (Box 3.2). This develops a number of equations relating to the consumption of addictive ‘goods’ on the assumption that addicts are making rational choices with stable preferences. This model appears to be contradicted by observations at both the individual and population level.

**Box 3.2 The Theory of Rational Addiction**

*Becker and Murphy (1988) have proposed an economic model of addiction that is based on the idea of stable rational preferences. Rationality is defined as a ‘consistent plan to maximise utility over time’ (p. 675).*

**Utilities**

Utility is a term that economists use to quantify benefits or losses as the person concerned sees them. It is different for different individuals and not the same thing as objective value. For example, $100 will have a different utility for a beggar than for a millionaire and even for the millionaire will have a
different utility when taken off the price of a house as when deducted from
the price of a hi-fi. Utility provides a single dimension on which to compare
things that are very different – which is of course necessary when making
choices. For example, when deciding to spend a windfall on a yacht or a house,
we have to convert the various desirable attributes of each to a single scale
so that they can be weighed against each other: that scale is utility.

**Addictive ‘goods’**
In the Theory of Rational Addiction (TRA), addiction is manifest as an increase
in consumption of a ‘good’ (as in ‘goods’, for example drugs, gambling or
anything that costs resources) as a result of past consumption. Thus an increase
in the present consumption leads to an increase in future consumption.

The model is supposedly able to explain patterns of consumption that include
bingeing and temporary and permanent abstention. According to this theory,
addiction is a term that can be applied to a wide range of ‘goods’ such as
gambling, watching TV, sex, and other people. In addictions to things that
cost money, it predicts that addicts will respond more strongly to permanent
than temporary price changes and that increased tension precipitates addiction.

**Harmful and beneficial addictions**
The theory makes a distinction between ‘harmful’ and ‘beneficial’ addictions.
Addiction to heroin and alcohol are harmful whereas addiction to religion
and jogging are beneficial. The difference between the two appears to lie in
the effect of consumption on an individual’s ‘stock’ of capital – resources.

**Addicts look ahead and maximise utilities**
According to this theory addicts are mostly rational consumers who look
ahead and behave in a way that maximises preferences that they hold and
that remain stable over time. This seems at odds with the observation that
preferences change over time with mood and time since last partaking of the
goods, but the proponents of the theory argue that it is not. They also claim
that what lies at the heart of addiction is an ‘unstable steady state’.

This sounds like a contradiction in terms, an oxymoron, but it is not. An
unstable steady state is one in which the behaviour changes over time as a
function of underlying conditions (expressed as parameters in their equations
– see below) that remain stable.

**What makes a ‘good’ addictive?**
So to the mechanism: the first thing to note is that theory is expressed as a
set of equations relating variables that are drawn from market economics
but which are used to represent psychological variables: stock, consumption,
depreciation, etc. This makes it difficult to interpret because the concepts
are similar to but not the same as those that we generally use when thinking
about behaviour; on the other hand they are not strictly economic variables
either. This allows the theory to get away with some questionable assumptions
on the one hand and to state some truisms as though they were genuine insights on the other.

So what follows is an attempt to translate the theory into language a non-economist can understand. According to the TRA, addiction arises from an interaction between individuals and goods. Individuals are generally more susceptible to harmful addictions to the extent that they disregard future consequences of their actions. They are more susceptible to beneficial addictions to the extent that they are more future-oriented. Goods are addictive for individuals to the extent that, for those individuals, they are reinforcing and they induce tolerance.

Goods are reinforcing for an individual if each time they are used they increase the likelihood that they will be used again. This is akin to the psychological notion of positive reinforcement: giving a hungry rat food if it presses a lever increases the likelihood of the rat pressing the lever again, and the rate of pressing will increase with each repetition of the sequence. The food is a positive reinforcer. However, it is much simpler and falls short of an adequate depiction of how rewards affect behaviour.

Tolerance represents a reduction in the utility of a good as a result of consumption. It is akin to, but again a much simplified version of, the concept of pharmacological tolerance in which there is physiological adaptation to ingestion of a drug so that it has less of an effect with repeated use. In reality tolerance occurs to both unpleasant and sought-after effects of some drugs and for other drugs it seems to occur primarily to the unpleasant effects. In fact, in the case of stimulants such as cocaine, the rewarding effect of the drug appears to increase with repeated use.

**Individual differences**

Individuals differ, not only in their propensity to reinforcement and tolerance, but also in the resources (income, for example) at their disposal to participate in the activity and how the activity affects these. One individual will be able to get drunk on a regular basis without damaging his or her livelihood while another will not. The non-economic costs of the activity will also differ; an individual who suffers severe hangovers after relatively light drinking will be less inclined to drink heavily.

**The dynamics of addiction**

Depending on the values of the various parameters that are plugged into the equations, an individual may exhibit an increase in consumption that then stabilises, a continuing increase followed by abrupt cessation, cycling through consumption and abstinence, bingeing, or merely brief flirtation with the activity followed by no further use. Drugs that are highly addictive are characterised by parameters that mostly lead to either high levels of consumption or complete abstinence.

Nicotine would seem to be such a drug. Becker and Murphy (1988) state that cocaine follows this pattern as well but actually that is not the case;
most cocaine use is at a relatively low level. Drugs that have relatively low addictive potential have parameters resulting in a unimodal distribution (with a single peak) that is below the level for harmful use but with a tail that includes very high levels of consumption that are harmful. According to the theory, looking at the consumption patterns of individuals those that are addicted typically show the bimodal (all or nothing) pattern of consumption.

The theory argues that older people should be ‘rationally myopic’ in that they have less time to live and therefore should be less concerned about the future. This illustrates rather nicely the difficulty in arriving at unequivocal, testable predictions in this area. It is obviously extremely rare for people to develop addictions in old age but this can then be explained by life circumstances and opportunity.

Theory predictions
The theory specifically predicts that a price increase will have a small initial effect on consumption but this will grow with time until a new steady state is reached. This appears to be contradicted by the evidence but then it all depends on the time scale over which one is looking and of course there will be some specific factors that will be important. The effect of price on cigarette consumption appears to be immediate and similar observations have been made for alcohol and heroin. However, the theory can always claim that the effect is progressive but over a relatively short time scale or that other factors also come into play.

The theory predicts that for individuals an addictive pattern of consumption will be triggered by stressful life-events. This is because the overall utility of the individual will be reduced and the marginal utility from consuming the addictive good relative to it will be greater.

One controversial postulate of the theory is that addicts are happier than they would be without their drug or addictive activity. It is argued that the reason that addicts generally appear to be more distressed than non-addicts is that distress has led them to addiction and keeps them in it. Again, this appears to be contradicted by studies with smokers, for example, which show that well-being does not diminish when they stop. But again, the conflicting observations can be explained away in terms of measures that are used or other confounding variables.

This is an economic theory and so deals with a conceptualisation of addiction that is different from that of the clinician, behavioural scientist or even member of the lay public. It deals with ‘consumption’ of a ‘good’. Its explanatory concepts are also somewhat different from those that most of us would understand, though in some cases they are presented as variants of psychological concepts such as ‘reinforcement’ and ‘tension’.

Issues and evaluation
The theory makes a lot of assumptions and allows itself a great deal of latitude in explaining patterns of consumption that the proponents believe to
occur, but, except in the most general of terms, the patterns of consumption observed are not those that the model predicts. In attempting to derive a mathematical description of consumption, the theory fails to do justice to the complexity of the phenomenon. Probably its most useful insight is that, when addicts say they want to stop using a drug or whatever, they do not necessarily mean that they would prefer their own life without drugs and they do not necessarily mean that they would like it to happen in the immediate future.

Self-medication and choice

We noted earlier that there are strong associations between drug use in particular and psychological problems, and that so-called addicts may be taking drugs as a means of coping with or ameliorating adverse life experiences. This approach has found its clearest expression in the Self-medication Model of addiction (Box 3.3).

This model has been proposed by many people in many forms. In one form it proposes that drug taking involves a cost–benefit analysis in which the benefits of the drug outweigh the costs. Apart from problems of mood disorder, we can expand this approach to any psychological deficits. For example, it has been proposed that smokers may smoke, at least partly, in order to combat problems they have in maintaining attention. It is argued that nicotine in particular helps with sustained attention (Warburton 1985, 1992). Related to this it has also been argued that smokers may have an underlying deficit in ‘sensory gating’ – tuning out irrelevant stimuli, and that nicotine helps to correct this disorder. In fact there is disagreement about the supposed beneficial effects of nicotine (West 1993) but that does not alter the fact that at least some researchers have claimed that it plays a role in why some people choose to smoke.

Box 3.3 The Self-medication Model of addiction

The Self-medication Model of addiction seeks to explain the development of addiction and individual differences in susceptibility to it. It proposes that individuals intentionally use drugs to treat psychological symptoms from which they suffer (e.g. Gelkopf et al. 2002).

Drugs as rational coping mechanisms

The Self-medication Model of addictive disorders derives from clinical observations and surveys of addicted individuals showing that they are predisposed to addiction if they suffer from unpleasant affective states and psychiatric disorders (Khantzian 1997; Farrell et al. 2001). The theory proposes that the particular drug an addict uses is not decided upon at random but is one that helps with the particular problem or problems that the person is struggling with. Therefore, initiation of drug use and the choice of drug are based on the drug effect sought by the individual. Drugs may
be chosen because they alleviate feelings of anxiety, help to control aggressive impulses, help to cope with psychotic symptoms and so on (Buckley 1998).

The model need not be limited to diagnosable psychological disorders. In principle it can be extended to any level and form of psychological need, however caused. For example, it could apply to anxiety and depression resulting from life circumstances. In a further variant of the model the drug or activity may not even make things better – they need only be judged to do so by the person concerned.

**Drug use and stress relief**

For example, most smokers will cite ‘stress relief’ as a major motivating factor and yet current smokers report higher levels of stress than never-smokers or even ex-smokers. Moreover, when smokers stop, their stress levels actually decrease and when they relapse they go up again (Cohen and Lichtenstein 1990). It is possible that each cigarette has an acute effect on stress, possibly because it relieves withdrawal symptoms that arise when the smoker cannot smoke, but there is a chronic effect in increasing stress (Parrott 1998).

Something similar may happen with alcohol. Acute intoxication without doubt can help people to ‘forget their troubles’, calm fears and ease pain. However, once the effect has worn off, there may be a rebound increase in anxiety. Moreover, repeated intoxication has an adverse effect on the life of the drinker which leads to increased stress and anxiety.

**Combating the side-effects of other drugs**

In a further development of the model, it has been proposed that some drugs may serve a purpose in alleviating the side-effects of drugs that are used to treat psychiatric disorder. This is particularly true for smoking, in which, for example, the effect of nicotine in increasing dopamine release at certain nerve terminals has been argued to help with the side-effect of neuroleptic drugs (Poirier et al. 2002).

**Possible mechanisms**

The Self-medication Model in its most general form covers short-term situational as well as chronic, environmental, short-term state and long-term trait problems that might be ameliorated by addictive drugs. Possible mechanisms and brain pathways underpinning self-medication effects have been proposed (Kassel et al. 2003).

**Issues and evaluation**

In its basic form, the Self-medication Theory requires that psychological disorders predate drug use and this is often though not always the case. The model cannot explain the many cases of addiction where there is no underlying pathology. Neither can it explain drug use in situations where there
are no psychological problems to be overcome. The model also fails to account for the extensive evidence of biological and psychological changes that occur with chronic drug use that appear to underpin compulsion.

Overall, the Self-medication Model fits well with a choice theory and can explain some of the phenomena of addiction but by no means all. Therefore, a synthetic theory of addiction should probably include the concept.

The issue of informed choice and future orientation

The view that many people who are classified as addicts are highly motivated by problems that they have in their lives seems inescapable. However, as a comprehensive model of addiction it is contradicted by the fact that many addicts show no evidence of underlying problems apart from their addiction. Thus the model should contribute to a comprehensive theory but cannot be one in itself.

Let us return to the basic concept of informed choice and addiction. According to this type of theory, when first introduced to a potentially addictive behaviour, addicts-to-be look at their current situation and at their future and what they think the activity or drug has to offer; they know about the risks of sampling an activity or drug and they are willing to take that risk. Once they have sampled the wares and they have a better idea of what it entails and what it delivers, their preferences are adjusted accordingly.

At all times these people are engaging in a process of weighing up costs and benefits. In some cases, they decide that the costs outweigh the benefits and they change their pattern of behaviour or stop it altogether. This theory involves ‘choice’ because addicts are aware of and deliberately select from a number of options. It is informed because they are aware of all the relevant information about risks and benefits. It involves rationality because the process by which it is used to arrive at a decision is one that maximises the chances of arriving at what for that person is the best outcome.

Under this model the addict is aware at the outset that s/he might become addicted and is willing to take the risk. In the beginning, the advantages of sampling the activity might be pleasant sensations, social approval, or escape from mental or physical pain. The risks are those of becoming addicted, and the physical and social harm that the activity might cause. This may include shame or embarrassment at being someone who gambles, smokes, takes heroin or whatever. Since we are still assuming that the individual is well informed, we presume that he or she is has a realistic appraisal of the likelihood of each of the various outcomes. However, as time goes on, the best appraisal of those likelihoods can change.

For example, someone who begins to use heroin may start with a view that the chances of becoming a compulsive injecting user are less than 50%. However, after having smoked heroin for a few months the person may notice that the consumption is escalating and reappraise the risk of becoming an addict. That in itself would not constitute a change in preference but for many people the more imminent prospect
of something bad happening will lead to a re-evaluation of exactly how bad it is. For some users, that prospect would be very unwelcome, so much so that they may choose to exercise restraint or even stop using altogether to avoid it. In other cases, the prospect of becoming an addict may not be a sufficient deterrent because the benefits of the drug are so great or the prospect of being an addict, given their life circumstances, is not of great concern.

In another example, a smoker may start smoking aware of and willing to accept the risks of illness and death in middle or old age. As the years pass, s/he may continue to feel in good health and come to believe that s/he will escape the major ill-effects of smoking so that the motivation to stop actually reduces. Then s/he may be diagnosed with lung cancer and still not stop because s/he believes that there is insufficient prospect of recovery to make it worth it.

What is rational choice?

It is worth reflecting some more on what rational choice actually is. It does not have to be sensible or adaptive. It only has to result from a weighing up of the costs and benefits as the decision-maker sees them. So it is rational, if often unwise, for a person to choose short-term gain over a possible longer term pain. A young person with a short time-horizon may simply not care about life after 30. In the throes of an alcohol binge an individual may not even care about the prospect of a hangover the following day. These are unwise choices but they are not irrational.

Habituation and withdrawal symptoms

Some, or possibly all, addictive activities appear to involve some kind of short-and/or long-term habituation and this might contribute to escalation of the activity and continued engagement in the activity. Postulating such a process is compatible with a Rational Choice Theory of addiction. The addict chooses to escalate the ‘dose’ because of a stable preference for a particular effect and continues to engage in the activity because of a choice to avoid or escape from withdrawal symptoms. Drug withdrawal, defined as a combination of signs and symptoms paired with abstinence, differs in manifestation across drug types in terms of magnitude, type and persistence.

For example, with opioid withdrawal, symptoms experienced upon cessation include nausea, lacrimation, perspiration, tremor, restlessness and yawning. Nicotine withdrawal symptoms include depressed mood, poor concentration, restlessness, increased appetite and irritability. The link between habituation and withdrawal symptoms has been elaborated in an important theory of addiction: the Opponent Process Theory (see Box 3.4). The theory could have been placed almost anywhere in this book but it is placed here because in theory it is compatible with a choice in which addicts consciously decide to continue to use a drug to avoid withdrawal symptoms.
Box 3.4 Opponent Process Theory

The drug reward process from repetitive drug use is upset by opponent processes that have a homeostatic function following drug euphoria to restore baseline levels. This leads to a reduction in the effect of the drug and withdrawal symptoms during abstinence.

The Opponent Process Model (Solomon and Corbit 1973, 1974; Solomon 1980) describes the positive and negative affective processes that underlie addiction. It proposes that internal reward processes are distorted by drug use and that drug-induced increases in reward threshold result in compulsive drug self-administration.

Maintenance of hedonic balance

The core tenet of the Opponent Process Model is that our central nervous system works to maintain hedonic balance. According to the theory, the primary ‘a-process’ for the hedonic drug effect is aroused by a stimulus. An ‘opponent loop’ generates a secondary ‘b-process’, which has an opposite effect to the hedonia aroused by the input. The loop generating the b-process is activated once a hedonic experience follows input. The b-process has a relatively slow build-up and decline (Figure 3.1).

The a- and b-processes combine to create the experienced hedonic state. The initial experience dominated by the a-process is labelled the A-state and

![Figure 3.1 A simplified view of the Opponent Process Model of addiction. The affective (hedonic or emotional) response to a stimulus (such as a drug) is the underlying a-process, which elicits the opponent b-process. These processes add together to create the pleasant A-state followed by an unpleasant B-state. On first use the pleasant A-state is large, followed by a small B-state. With repeated drug use the b-process increases in magnitude and duration, leading to an experience dominated by the unpleasant symptoms associated with withdrawal.](image)
the subsequent experience in which the b-process dominates is the B-state. Figure 3.1 shows the sequence of the peak primary reaction A, adaptation, steady level, after-reaction B and the decay of B.

The opponent b-process is activated indirectly via the activation of the a-process. It has an evocation threshold, a latency or recruitment time. Under certain conditions, the opponent process can be activated by events in memory and as a consequence of Pavlovian conditioning.

The temporal patterning of hedonic states
Thus according to the Opponent Process Model the temporal pattern of affective states caused by a stimulus has five distinctive features:

- The peak of primary hedonic process state, precipitated by stimulus onset.
- A period of hedonic or affective adaptation during which the intensity of hedonic state declines, even though stimulus intensity is maintained.
- A steady level of the hedonic process which continues as long as stimulus intensity is maintained.
- A peak of affective after-reaction, which quickly follows stimulus termination and whose quality is hedonically different from that of the primary hedonic state.
- Decay of the after-state.

Effect of drug use on the opponent process
The opponent process is strengthened by drug use and weakened by disuse, although the primary affective process is not affected by use. A b-process will acquire more power if frequently elicited. It will show a shorter latency of response to the a-process, a quicker rise, a higher asymptote and a longer decay time. In contrast, an a-process is a relatively stable, unconditioned reaction. Disuse weakens the b-process and it should return to its original magnitude whenever the a-process has not occurred for long time. Thus over time:

- The peak of A' (the prime denotes the situation after extended drug use) will be less intense because the latency of the b-process is decreased and its intensity increased.
- The steady level of A' (a–b) during maintained stimulation will be close to baseline and even below it in some cases.
- The peak of B should be more intense and long lasting.

Aversive states, manifesting themselves after the sudden termination of pleasurable inputs, become more intense with repeated experiences. Mild craving later becomes abstinence agony and intense craving. In addition, after many repetitions the steady level of pleasure produced by the continued presence of pleasurable stimulus input has decreased.

It is proposed that prolonged activity of an opponent process system, whether it be pleasurable or aversive, may cause psychological ‘stress’ in the
sense that many physiological resources may be required in order to keep the opponent process strong.

**Opponent processes and behavioural disorders**

It is expected that a constant demand might lead to the exhaustion of a particular overworked opponent process system or to the debilitation of other defence systems. Theories of mental disease, psychosomatic disease and behavioural disorders usually emphasise that aversive stimuli lead to stress, stress is aversive and both cause emotional disorders, psychosomatic illness and behavioural malfunctions. The Opponent Process Theory takes a different view. In this view, stress caused by aversive stimulation is only part of the story. There are behavioural disorders caused by lasting, repeated and intense b-processes in general.

**Pleasurable and aversive A-states**

The theory argues that A-states may be pleasurable or aversive and the corresponding B-state is the reaction to that and has the opposite hedonic value. Motivational systems involving pleasurable A-states and aversive A-states are similar. In both cases, the onset, maintenance and termination of the stimulus results in a certain amount of pleasure and a certain amount of displeasure. They mainly differ in terms of whether pleasure or displeasure comes first.

In the case of pleasurable A-states, it is proposed that the subsequent aversive B-state functions as a drive that energises behaviour and that the pleasurable A-states reward these behaviours. Electrical self-stimulation of rewarding brain sites, chemical self-stimulation with opiates and love relationships are argued to work in this way.

**Variability in activation of opponent processes**

Not all stimuli result in an opponent process. A non-opposed system would manifest no peak of the A-state, no adaptation and no appearance of a B-state after stimulus termination. It is argued that the hedonic state engendered by marijuana could be an example. As a chemical stimulus it precipitates a pleasurable A-state. However, there is no reported peak or adaptation nor are there supposedly aversive withdrawal symptoms or craving (though this now appears not to be true). Nausea is possibly an aversive A-state without a B component state.

There are argued to be wide variations in the strengths and variations of b-processes after repeated elicitations. Taste cravings as b-processes, for example, may last a few minutes; exhilaration following exercise on the other hand can last somewhat longer. Craving following withdrawal from opiates, alcohol or barbiturates can last for months.

**The example of opiate use**

Opiate use is given as a classic case of the operation of opponent processes. In the initial stages of opiate use, the user experiences an intense pleasurable
feeling, described as a ‘rush’, immediately after the opiate injection. The rush is then accompanied by a period of euphoria, but of reduced intensity. Later on, the individual experiences aversive withdrawal symptoms, together with a feeling of craving. Over several weeks, with repeated doses, the A-state begins to weaken, and at the same time the B-state intensifies and takes longer to return to baseline. The A-state becomes a state of just normal functioning as opposed to the experience of euphoric sensations. The B-state becomes more extreme and longer in duration.

The development of addiction

It is proposed that repeated use of some drugs is responsible for people finding themselves craving a substance which previously held little interest, and the model is argued to explain opiate, alcohol, barbiturate, amphetamine and nicotine addiction. In these cases:

- The B-state lasts a long time.
- The acquired B-state is highly aversive.
- The elicitation of the A-state is effective in causing rapid alleviation of the B-state.
- The user learns to use the drug which elicits the A-state in order to remove the B-state.

Addiction will not occur (even if A and B are experienced repetitively) if the properties of affective response to a drug are such that B regresses to baseline quickly – another dose is never needed to remove the effects of the aversive B-state as it quickly disappears anyway.

If the b-process is strengthened through repetition, withdrawal can be reinstated by a small dose of the drug. In the case of longer-term abstinence from the drug, the b-process decays but it is argued that the system may not return to normal and that there is always a vulnerability to relapse (Weiss et al. 2001).

Possible mechanisms

Suggestions for neural circuitry in relation to the opponent process model have included the suggestion that the positive a-process is caused by activation of mesolimbic dopamine projections to the nucleus accumbens and amygdala that mediate positive reinforcement and the b-process involves down-regulation in the mesolimbic dopamine system (Weiss et al. 2001).

Extensions of the model

The model has been extended and developed to account for complex temporal variations in the rewarding properties of stimuli. It has been proposed, for example, that a single drug administration may create a temporary decrease in reward threshold (Koob and Le Moal 1997). A second administration of the drug may produce an increased euphoric effect. This can lead
to repeated drug use prior to onset of addiction. After further repetitions internal processes oppose the drug-induced reduction in reward threshold, restoring its original pre-drug state.

Koob and Le Moal (1997) propose a ‘spiralling distress’ concept in which initial drug use reduces reward threshold making future use more likely. Then subsequent increases in reward threshold mean that periods when the drug is not available are accompanied by increasing levels of negative affect and greater emotional distress.

Koob and colleagues (Koob and Le Moal 1997; Koob et al. 1997) have also suggested that repeated drug use activates an additional b-process via the hypothalamic–pituitary axis stress system, causing release of corticotrophin releasing factor in the amygdala region as well as other stress responses. Under this view, it is hypothesised that an individual progresses into an addictive state of drug use if the addict originally began drug taking for the positive hedonic effects, which eventually progress into a predominantly negative hedonic state.

A further development of the model has been proposed in which it is argued that the b-process arises from Pavlovian conditioning. Siegel (1988) proposed a conditioning model based on the Opponent Process Theory. In that model the stimuli that accompany drug-taking trigger the opponent process by virtue of being associated with the drug effect. This derives from the observation that the experience of drug withdrawal in humans and other animals is more severe in the presence of stimuli that accompanied the drug experience.

**Opponent processes, choice and instrumental learning**

Note that although we have positioned the model in the part of the book that deals with rational choice, many proponents of the model would argue that it fits better within a later section dealing with instrumental learning. However, the model is in principle agnostic as to whether the distress that is presumed to motivate drug-seeking behaviour involves conscious choice or an automatic cue–response process.

**Issues and evaluation**

The model has considerable explanatory power that goes well beyond explanation of drug dependence. For example, it can explain acquired tastes (where the initial experience of something is unpleasant but apparently by virtue of that unpleasantness it then becomes much sought after). It can explain the dynamics of the hedonic experience in both the short and long term in response to drugs as diverse as cocaine and alcohol.

On the other hand it remains highly speculative and its flexibility, in terms of allowing very different parameters with different drugs and other stimuli, makes it difficult to test as a general concept.

Perhaps the major limitation is not with this particular theory but with its use as the dominant theory underpinning drug dependence. Although the
suggestion that addiction is motivated primarily by avoidance of distress makes intuitive sense, it seems unlikely that it is the only or even a major cause of loss of control of drug-seeking behaviour. There are just too many counter-examples.

In the realms of animal research there are many observations that cause difficulties for the theory. For example, Stewart and Wise (1992) conducted experiments in which rats were trained to respond for heroin infusions. The animals were then subjected to a period in which the drug was not administered. This was followed by a number of different experimental manipulations. In one of these, the a-process was activated by giving the rats a small injection of their drug prior to the test (priming injection). In the other condition, the b-process was activated by administration of naltrexone, which is an opioid antagonist drug that blocks opioid receptors in the brain and has the potential to induce withdrawal symptoms in individuals who are heroin dependent. Precipitated withdrawal, it is argued, represents a b-process and is expected to be the most powerful cause for reactivating drug-seeking behaviour. A priming drug injection turned out to be far more effective at reinstating drug-seeking than naltrexone administration.

Besides this, the timing of withdrawal states and relapse in humans and reinstatement of drug use in animals do not match up well. To account for this, a p-process has been proposed that decays slowly after long periods of abstinence. It has also been suggested that associative conditioning causes predictive drug cues to elicit conditioned tolerance and continued withdrawal essentially as conditioned b-processes. However, many human addicts report cues that often fail to elicit conditioned withdrawal. In addition, drug cues often elicit quite different effects such as conditioned feelings of a drug high (a-process), or feelings of drug craving by themselves.

The Opponent Process Model offers some intriguing possibilities but is only one of a number of possible ways in which individuals may become habituated to the effects of drugs or other rewarding experiences. Some of these mechanisms may lead to onset of adverse withdrawal symptoms when the drug or experience is not available.

For example, Peper (2004a,b) has provided a very full conceptual and mathematical account of the process of development of tolerance to drugs which is purported to explain many of the features observed in animals and humans. A detailed examination and critique of this new model is beyond the scope of this book. The point we wish to make here is only that a synthetic model of addiction needs to be able to accommodate such detailed models but at this stage must be agnostic on the precise details until more is known.

Opponent Process Theory is one example of a theory about how an individual comes to suffer negative consequences of drug use that then go on to drive further use. We have already alluded to the possibility, even likelihood, that in at least some cases an individual’s personal characteristics or circumstances create unpleasant
experiences that an addictive drug can be perceived as helping with. These include problems of depression, anxiety and possibly boredom.

One problem that is not so obvious is that of problems with intimacy (Keane 2004). It is argued that individuals with particular problems with intimacy in personal relationships may have their needs meet by an intense intimate relationship with a drug. This certainly fits with some observations about addiction such as the fact that experience of abuse as a child predisposes to addiction to drugs such as alcohol and heroin (e.g. Marcenko et al. 2000).

Policy implications of a Rational Addiction Theory

The policy implications of a Rational Informed Choice Theory of addiction tend toward a libertarian view that individuals should be left to make (and suffer the consequences of) their decisions without state interference. This view is typified by the law professor Viscusi who argues that the present adversarial approach to tobacco control is misplaced – we should present the information and let smokers decide (Viscusi 1998).

In the case of addictions in which harm is caused to others, this should be dealt with by the criminal justice system. Although proponents of this approach may recognise that use of an addictive drug leads to withdrawal symptoms, the addict was aware of this at the outset and should be held accountable for his or her earlier decision to experiment with the drug in spite of this. It might even be argued that offering treatment to reduce the severity of the effects of addictions is counter-productive because it shifts the balance of preference in favour of uptake and continuation.

Irrational, ill-informed choice and unstable preferences

The Rational Informed Choice Model cannot account for the fact that many addicts choose to exercise restraint and in many cases go to great lengths and expend time, effort and money to achieve this, and yet still fail. Many addicts also regret having started down the road that has led them to this state. However, a Rational Choice Model can still be entertained if one assumes that the addicts’ preferences change over time, and particularly between the time when a decision is made to restrain use and when that restraint has been exercised for a while. We also have to postulate that the addict is not very good at predicting what the consequences of trying to exercise restraint would be. If they were good at predicting it and were acting rationally, they would only try it when they were going to succeed.

The idea that addicts are fully able to predict their future reactions and that their preferences do not change was always going to be unrealistic. We are ill-informed about many other aspects of our lives and reactions and we also change our minds about things. Introducing unstable preferences clearly goes a long way to explaining why addiction develops and why attempts to restrain the activity fail: the preferences change.
Rational ill-informed choice with unstable preferences: an addict’s eye view

Time 1 (while still partaking)
I want to stop being a [drinker/smoker/heroin user/gambler/tranquilliser user/crack user]. This is because although it [gives me pleasure/makes me feel better/is part of who I am/helps me to cope with life/helps me do things I need to do], I realise that it [is doing me harm/might do me harm in the future/is doing harm to people I care about/may make people I care about unhappy].

Time 2 (during an attempt to exercise restraint or abstain completely)
I want to go back to my old ways because [I miss the benefits/I do not want to put up with the unpleasant consequences of abstinence].

How well informed are addicts?
There is in fact evidence that addicts are ill-informed. For example, smokers greatly overestimate the likelihood that they would be able to stop if they tried. They are also often unaware of the full range of adverse health effects of smoking. Many have the view that they will escape the unpleasantness of old age if they die young from smoking. In fact, smokers spend more of their shortened lives in disability and pain as a result of chronic smoking-related diseases. In effect, smoking brings old age on early – not just death. It is also the case that many alcoholics underestimate the adverse health consequences of their alcohol use and even deny that they have a problem.

Allowing for those who engage in addictive behaviours to misjudge the consequences of their actions greatly eases the task of explaining what is going on. It is possible that addiction arises because, while the immediate effects of the activity are known, the longer-term risks are not accurately judged. This may occur because the addict is not aware of the information or even that the addict’s awareness is biased by emotional needs.

Ignorance and education
It would be tempting to assert that lack of information about addictive behaviours helps to explain a higher prevalence among those in society who are less well educated. However, this does not work. In the case of smoking, for example, in countries such as the UK where there is a strong association with educational level, there is no difference between more and less well educated smokers in their desire to stop or indeed in the frequency of their attempts to stop. Also, there are countries where smoking prevalence is as high in doctors who are fully aware of the health risks as in other members of the population.

A great deal of the effort that goes into preventing and even treating addiction seems to assume that it is partly caused by lack of knowledge. It is presumed that providing more information on the harmful effects of addictive behaviours can
influence people to exercise restraint. This is not as easy an assumption to test as might be imagined. We pointed out earlier that on the face of it there is no evidence that people who are better informed are more motivated to exercise restraint. However, things are more complicated than that.

First of all, the studies are typically carried out in a society that is saturated with information about the harmful effects of addictive behaviours. One would have to have lived in a cave all one’s life not to have heard that excessive alcohol consumption can lead to an early grave. If just about everyone is aware of the harmful effects of an addictive activity, there is obviously minimal scope for showing that lack of knowledge is contributing to addiction.

Secondly, it is entirely possible that our measurement of how well informed addicts are is not up to the job. Information is not a simple quantity of which we have more or less. It consists of discrete items, some of which may affect our choice while others do not. Moreover, what affects our choices may be different for different people. For some people, showing them what they really look like when they are drunk or smoke may be more important than others for whom the prospect of disability in later life may be more motivating.

**Expectancies and addiction**

The idea that beliefs about the consequences of an activity, which may or may not be accurate, may contribute to addiction has been extensively explored in what are known as Expectancy Theories (Box 3.5).

---

**Box 3.5 Expectancy Theories**

‘Expectancies’ about the costs and benefits of an addictive activity are assumed to contribute to excessive use. These may be inaccurate. There is interest specifically in how far addicted individuals differ from non-addicted individuals in expectancies about the positive versus negative effects of the activity. Expectancies may involve more than beliefs. They may be ‘memory templates’ of the rewarding value of the addictive behaviour.

Expectancy Theories view drug use that escalates into addiction as being a result of the expectations an individual holds regarding the costs and benefits of the activity. Using alcohol consumption as an example, Expectancy Theories propose that the level of alcohol consumption is related to how much the person expects it will deliver a desired effect.

**Positive and negative expectancies**

It has been found that extent of drinking is positively correlated with positive expectancies and inversely associated with negative expectancies (Christiansen and Goldman 1983; Brown et al. 1987; Reich et al. 2004). Heavier drinkers report more positive expectancies than lighter drinkers (Southwick et al. 1981) and heavier drinking has been found to be associated with expectations
of social and physical pleasure, social assertion, tension reduction, greater sociability and enhanced cognitive and motor functioning (Brown 1985).

Alcoholics and non-problem drinkers have also been found to differ on measurements of global positive changes, sexual arousal, physical and social pressure, assertiveness, tension reduction and arousal aggression (Connors et al. 1986). Expectancies may be more consistently associated with quantity than frequency of drinking (Chen et al. 1994). Even when demographic variables that are known to contribute to drinking are accounted for (e.g. age and gender), expectancies have been found to be associated with drinking versus not drinking and with quantity of drinking (Mooney et al. 1987). However, the proportion of variance explained by expectancies is typically low: in the Mooney study it was 6% for drinking versus not drinking and 15% for quantity of drinking.

Alcohol expectancies have also been found to predict self-reported alcohol consumption in adolescents (Christiansen et al. 1989). The strongest predictors of behaviour are argued to be beliefs and expectancies that relate to specific actions. For example, ‘I will not drink alcohol at all (single behaviour) this week (time specific)’. Expectancy Theories have also been applied to the outcome of attempts to control addictive behaviours. For example, Brown (1985) found that higher positive outcome expectancies were associated with decreased likelihood of achieving a year’s abstinence and with treatment compliance.

It has been reported that in some populations men have stronger positive and weaker negative expectancies than women. In one study, men had higher levels of expectancy for tension reduction, social facilitation, activity enhancement and performance enhancement (Sher et al. 1996).

Self-efficacy expectancies
‘Self-efficacy’ expectations are particular kinds of beliefs that are postulated to reflect individuals’ beliefs in their ability to perform certain behaviours, i.e. smokers would be more motivated to quit smoking if they thought that they would be able to do it successfully. Generalised self-efficacy beliefs refer to the belief that an individual can cope with the demands of everyday life across a broad range of behaviours.

Expectancies may be more than beliefs
Some Expectancy Theories consider the core construct as much more than simple beliefs. Goldman and Darkes (2004) have argued that expectancies are part of the memory structures that organise input to the central nervous system and guide behaviour. Expectancies are also regarded as having a moderating function, for example in moderating the role of stress in alcohol consumption. Goldman and Darkes suggest that expectancies are the pathway through which genetic predisposition, social and cultural information, affective state, personality and so on influence drug use and abuse.
Brandon et al. (2004) have argued that in considering the quantity of information that must be processed and the required speed of processing for large amounts of information, the expectancy-based control systems are ‘automatic’, functioning through parallel processes that operate outside of conscious awareness.

Rather et al. (1992) have used the application of a mathematical information processing model to propose that affect (or expectancy) can be mapped within a two-dimensional model (e.g. arousal and valence) that represents patterns of expectancy activation.

Brandon et al. (2004) claim that, if expectancy-based control systems are unconscious and automatised, then this could account for loss of control and craving, behavioural tolerance and difficulty in achieving abstinence. It is proposed that, as addiction develops, the activity is influenced by conscious expectancies involving controlled processes (and assessable through self-report) and more by the unconscious expectancies involving automatic processes. On the other hand, the conscious accessibility of expectancies may relate to the effect that these have on behaviour.

It has been proposed that expectancies and actions may influence each other in a reciprocal fashion. For example, Smith et al. (1995) found that teenagers’ expectancies for social facilitation from alcohol and their drinking showed a bidirectional relationship with each other.

**Expectancies as part of a wider motivational system**

Expectancy Theories have been combined with more general motivational accounts. For example, it has been proposed that alcohol abuse can be predicted from a causal chain that includes alcohol consumption and ‘drinking to cope’ as proximal determinants and coping skills and positive alcohol expectancies as more distal determinants (Cooper et al. 1988).

Cooper et al. (1988) reported a study in which drinking to cope emerged as the most powerful predictor of high alcohol consumption, apparently exerting an influence via direct and indirect pathways. Coping styles that involved showing avoidance of emotion emerged as stronger predictors of alcohol abuse than problem-focused coping. The predictive value of coping was moderated by alcohol expectancies: avoidant style of coping with emotion predicted alcohol abuse only in drinkers expressing high levels of positive expectancies from alcohol.

Along the same lines, Fischer et al. (2003) found that positive expectancies of social facilitation from drinking moderated the effects of extraversion on drinking behaviour among a sample of undergraduate men and women.

**Manipulating expectancies**

Expectancies ought to be able to be manipulated, making them a suitable target for interventions. Tate et al. (1994) informed abstaining smokers that they should expect no complaints during abstinence, which led to fewer reported somatic complaints and less mood disturbance than controls not
primed with any expectancies. Participants told to expect somatic but not psychological complaints reported more numerous and severe somatic withdrawal symptoms than those not told to expect this. Therefore, manipulation of expectancies on quit day may affect the withdrawal experience.

Issues and evaluation
Where Expectancy Theories deal with conscious beliefs about the costs and benefits of addictive behaviours, the fact that they show some level of association between expectancies and behaviours may not seem to represent a major advance on the common-sense model with which we started the chapter.

Where expectancies are construed as something more amorphous or difficult to pin down as such ‘memory templates’ or automatic processes, it is not clear what the concept refers to – what correspondence it has with the real world. It offers a level of abstraction that takes it out of the phenomenological or the physiological. On the other hand, it is interesting to see that this kind of theory is incorporating in some way the kinds of learning mechanisms that we will be considering in Chapter 5, without which we do not have a comprehensive theory of addiction.

A major limitation of the research involving these theories is that it rarely uses addiction per se as a target variable; most often it uses level of consumption and sometimes it uses problematic use. Thus it is not clear how far expectancies play a specific role in the development of the loss of control.

The other problem faced by such theories is the one alluded to in the previous chapter about what constitutes support for a theory. The prediction of actual behaviour using even quite elaborate Expectancy Models is typically weak and this prediction is not compared with other formulations that make fewer assumptions. If we employ the method of looking for counterexamples to establish whether Expectancy Theories can provide a complete explanation of addiction, it seems that the theories are not sufficient.

Unstable preferences
It was pointed out earlier that it seems gratuitously limiting to postulate that underlying preferences do not change in addiction; after all they obviously change in many other areas of activity. People change their minds about what they like and dislike and what they want. One minute they choose to try to stop using a drug; at that time their thoughts are dominated by the negative aspects of the activity. Having then abstained for a while and facing the reality of the loss of reward and the discomfort associated with this, they change their mind and resume the behaviour. As Skog (2000) puts it: ‘what we observe is not an inability to choose, but choices governed by strong appetites and conflicting motives’ (p. 1309).

The concept of ‘approach-avoidance conflict’ is one that extends throughout motivational theory. At a distance something may look attractive but as one gets closer to it the unattractive features become more evident and dominate our thinking so
we retreat from it at which point the unattractive features become less salient and we approach again.

The related concept of choice with unstable preferences has been elaborated by Skog (2000) (Box 3.6). His main tenet is that what looks like compulsive use is no more than unstable preferences resulting from conflicting motives.

**Box 3.6 Skog’s Choice Theory**

This theory states that addiction can be regarded as a manifestation of conflicted choices that change as a function of the addict’s current preferences.

Skog’s Choice Theory of addiction seeks to explain addiction in terms of choices that people make rather than compulsion (Skog 2000, 2003). He points to the fact that in some sense addicts always have a choice. They are not physically forced to engage in the behaviour and do not require physical restraint. The appearance of lack of control arises because addicts change their minds. Their choices are conflicted and on some occasions the option to continue the behaviour dominates and on other occasions the option to exercise restraint dominates.

**Stability and consistency of choices**

Thus the theory postulates that individuals differ in the stability and consistency of their preferences. This is certainly in accordance with common observation and with systematic research. It is also postulated that addicts may have a propensity to more unstable preferences. This may arise because of the addiction or be a factor that predisposes to development of addiction. Skog suggests that this might be a fruitful area for future research and indeed it may be. However, it is not clear whether a propensity towards unstable preferences is needed to understand addiction or why it should be greater in addicts.

**The definition of choice**

To be fair, Skog goes beyond metaphysics in his definition of choice. He states that if an action is made with regard to its possible consequences, if it is future-oriented, it must involve choice (Skog 2000). However, it is doubtful whether anyone working in the field of addiction would want to argue that it is only physiological or physical compulsion that is the appropriate model. Indeed, there are probably few addiction experts who believe that an addict would on a given occasion partake of their addiction in the certain knowledge that it would kill them immediately. So addiction in some sense must involve choice, even if that choice is highly constrained.

**Issues and evaluation**

In proposing his Choice Theory Skog has argued that ‘from the point of view of empirical research, convincing evidence for inability to choose is still
missing’ (Skog 2000, p. 1131). This is an example of a statement that appears to establish a theory (one involving ‘compulsions’) as conflicting with observation but only does so under a particular interpretation of the terms with which proponents of the theory could reasonably disagree. In focusing on the instability of preferences Skog’s theory captures a fundamental feature of human motivation, but in framing behaviour solely within the context of choice it arguably misses key aspects of the flow of behaviour in which alternative courses of action are not considered.

As long as we are willing to accept that people can change their minds about things rather quickly and then change them back again, and that a choice can be rational even if the preferences that determine it are ultimately self-destructive, there is no reason to abandon a Rational Choice Theory of addiction.

The denotative and connotative meaning of choice

However, this involves placing an interpretation on the term ‘choice’ that is very narrow and arguably misleading. If someone puts a gun to your head and threatens to pull the trigger if you do not drink a large glass of whiskey, it is true that you have a choice but the imperative to do as you are told is frankly so strong that most people would say that they were compelled to do the deed. On the other hand, if someone bursts a balloon in front of your face unexpectedly you would not be able to stop yourself blinking and would have no choice in the matter.

So acceptance of the Rational Choice Theory approach depends on what we mean by ‘choice’ and what we mean by ‘rational’. This is something of a problem because the terms ‘rational’ and ‘choice’ have connotative as well as denotative meaning. ‘Rational’ carries connotations of calculating and purposeful, even adaptive; ‘choice’ carries connotations of free will and responsibility.

This is no doubt why this approach is so attractive to libertarian politicians who think that if people are too stupid, ill-informed or lacking in moral fibre to resist the ravages of drug addiction, it is their own fault; and it is attractive to businessmen and shopkeepers who make a good living out of manufacturing and selling legal drugs because they are merely satisfying the desires of their customers.

Judgement and decision-making

There is a very large literature on decision-making, how it is done and in what ways it is rational or irrational, and many excellent books on the subject (e.g. Baron 2000). Much of this research has direct relevance for Choice Theories of addiction. Rather than attempt to summarise the field here, we will focus on the question of how risks are perceived and how people compare positive and negative features of alternatives when they are qualitatively different.

Looking first of all at the perception of risk, a number of useful observations have been made. Perhaps the most notable contribution to the field has been made...
by Paul Slovic (Box 3.7). Slovic’s research has shown over the years how sensitive our judgements of risk are to the context in which those judgements are made and the manner used to elicit them. Of particular relevance here is the proposition, also made by others, that on most occasions when we have to judge risks we use feelings rather than analytical thought as the basis for our judgements.

**Box 3.7 Slovic’s Affect Heuristic**

Slovic distinguishes between intuitive and analytical methods of making judgements and argues that the influence of the intuitive system which is based on feelings is an important factor in the development and maintenance of addictive behaviours.

*The influence of ‘feelings’ on risk perception*

Slovic and his colleagues have shown over a number of years that the choices we make between options involving numerically specified probabilities depend on how we are asked to express that choice and the context in which the choice is set. Of particular relevance here is a specific view on the role of feeling in the perception of risk (Slovic et al. 2002). Some general observations are as follows.

We have two very different methods of making judgements: an intuitive system and an analytic. The intuitive system is rapid and influences our feelings about what it is that we are dealing with that in turn is related to our imagination of it. The analytic system uses procedures, including formal logic and calculation, to arrive at judgements. Most judgements are made using the intuitive method.

*How intuitive judgement can contribute to behaviours such as smoking*

The intuitive method has many features that help to explain why people engage in addictive behaviours such as smoking.

1. When risks involve an accumulation over many instances of a behaviour, these tend to be discounted. Small individual risks do not engage our feelings of fear.
2. Risks that occur at some indefinable point in the future tend not to be prominent in our thoughts and so do not engage with motivational feelings.
3. Our ability to imagine a possible outcome as something fearful critically affects our feeling of motivation to avoid it. General abstract concepts such as ‘harming health’, and numerical probabilities, do not generally provide the kind of imagery that is engaging. In fact, in numerical terms people greatly overestimate the risk of lung cancer as a result of smoking. The lifetime risk is in the region of 15% for a smoker whereas studies have found that people put the figure at more than 40% (Viscusi 2000). However, their emotive reaction to the prospect of getting lung cancer is much weaker than is warranted.
Related to this, currently experienced ‘visceral factors’ (drives such as hunger) have a much greater impact on our actions than mental representations of those factors in the future.

We tend to feel a greater sense of control over our fate than is warranted. In games of chance, for example, individuals often feel that there are ways in which they can influence events even when at some level they know this is impossible. The large majority of smokers believe, when they start, that they will smoke for no more than 5 years and yet this is obviously not what happens. Even adult smokers overestimate their likelihood of success if they were to try to stop.

Possibly related to this, most of us experience an optimism bias. We may accept that there are risks attaching to particular activities but perceive our own personal risks as lower than these.

When judging risks and making choices, the size of potential losses or gains affects the way that we use judgements of probability. When losses or gains are very large, we are much less sensitive to differences in probability. For example, even the tiniest possibility of a cataclysmic event can lead people to expend huge resources to try to avoid it. And on the other side, the extremely low probability of, say, winning the national lottery is discounted when the potential prize is extremely large.

The Affect Heuristic
When considering the value that we place on particular option, Slovic notes that much of our thinking is dominated, not by an objective evaluation of the costs and benefits but by ‘affect’ – positive or negative feelings that are attached to those options. He takes this idea from Damasio (1994) although the observation has been made many times and indeed is a central plank of theories of conditioning.

Affective reactions derive from simple associations and non-analytical considerations such as mere repetition. The Affect Heuristic proposed by Slovic is an experiential system for ascribing risks and benefits to particular options. Slovic proposes that our positive or negative feelings about an option determine our judgements about its risks and benefits. Put simply, if we feel positively about something, we tend to judge the risks arising from it as low and the benefits as high.

Issues and evaluation
Some people might consider that the view that feelings affect our judgements of risks and benefits is fairly obvious. Slovic’s contribution has arguably been in articulating this and the other sources of bias in a language that brings it within the domain of economic theory and cognitive psychology. His theory is extremely important in that it represents the beginnings of a synthesis between theories of motivation based on learning mechanisms and drives and classical decision theory.
Cognitive biases

The influence that feelings have on beliefs and evaluations can be extended to non-conscious mental processes. Bias can occur not just because we believe things we want to believe but also in the very way in which our attention and memory operate. That theme is the focus of what can be referred to as Cognitive Bias Theories of addiction (Box 3.8). These propose that addiction arises out of or is maintained by a tendency of the addict to pay greater attention to and selectively remember addiction-related information.

Box 3.8 Cognitive Bias Theories

Addiction is maintained by biases in the cognitive system, including beliefs, expectancies, self-efficacy, attributions and attention.

Cognitive bias and expectancies

The cognitive bias approach can be regarded as an extension of expectancy theory. The latter characterises addiction in terms of expectations, which may be accurate or inaccurate, for the individual concerned, about the future benefits and costs of engaging in the addictive activity. Cognitive Bias Theories propose that it is biases in beliefs and in attention and memory processes linked to these beliefs that are at the root of the problem of addiction.

Unconscious biases

Cognitive Bias Theories propose that expectancies that motivate behaviour reside in memory and that tasks that examine storage and recall of memory information can be used to detect biases that may be operating. A major advantage of this approach is that it does not rely on self-report and all the problems associated with this. It has been found, for example, that although smokers may endorse equal amounts of negative and positive outcome associations with smoking, they have a higher incidental recall of the positive outcomes.

Cognitive Bias Theories propose that the loss of control demonstrated by addicts can be explained by automatic and pre-conscious cue processing of stimuli related to the addiction. It is proposed that potential drug cues are evaluated ‘pre-attentively’. They are then prioritized and subsequently trigger ‘somatovisceral, behavioural and cognitive responses’. This information processing is subjected to automatic attentional and interpretative biases; these enhance the addict’s awareness of his or her physiological arousal as well as action tendencies and cognitions (Ryan 2002).

*This book has separated out Cognitive Bias Theories from Expectancy Theories for the purpose of exposition. In practice, as with so many of the theories in this book, there is considerable overlap in concepts and ideas.
Positive belief biases purportedly arise from the highly cue- and situation-dependent nature of memory structures. Experimental cognitive psychology techniques may be able to yield useful information about biases. Individuals may be aware of automatically triggered beliefs, but the ability to report these may be interfered with by a range of factors including cognitive dissonance and the demands of the situation.

Cognitive biases pertaining to addictive behaviours have been reported across a range of addictive behaviours, such as gambling (McCusker and Gettings 1997), cannabis use (Field et al. 2004), smokers (Waters et al. 2003) and heroin use (Franken et al. 2003).

**Attentional biases**

The Stroop colour naming task requires attention to the perceptual characteristics of words while suppressing processing of their meaning. In the original version of this task, people are presented with a list of colour words (e.g. ‘red’) written in colours different from the ones they are naming. They are asked to name as quickly as possible the colours in which the words are written. The task has been extended to non-colour words. Problem drinkers and smokers have shown a selective interference effect (increased colour naming times) for words semantically related to their addictive behaviour (e.g. ‘cigarette’ or ‘beer’) (Waters and Feyerabend 2000; Bruce and Jones 2004).

Visual probe tasks have also been found to add useful information about attentional responses to drug cues. Waters et al. (2003) found that a sample of heavy smokers were faster and more accurate in responding to a visual probe that replaced a smoking picture than to a neutral picture, indicating that they demonstrate an attentional bias to smoking cues. Attentional bias was also found to correlate with severity of cravings reported prior to undertaking the task, suggesting that the bias may be due to motivational processes.

In another visual probe study by Bradley et al. (2004) it was found that smokers showed an attentional bias for smoking pictures presented at exposures of 200 to 2000 milliseconds (ms) compared with non-smokers but not at exposures at very short latencies (17 ms). Smokers also showed greater preferences for smoking-related than control cues. They interpreted these findings as showing that the biases were not due to pre-conscious processing.

Similar attentional biases have been found with problem gamblers. Boyer and Dickerson (2003) found that participants who reported difficulty in controlling their gambling behaviour took significantly longer to name the colour of the words relating to poker machine gambling compared with those reporting that they could control their gambling behaviour.

**Different results with different types of bias**

It has been proposed that the three commonly used measures of cognitive bias (masked and unmasked Stroop test and visual probe tasks) may tap
different underlying mechanisms. Mogg and Bradley (2002) found that scores on these tasks by a group of smokers were not correlated. The participants were asked to abstain from smoking 12 hours prior to a first session and to smoke as usual before a second session. An attentional bias for smoking-related pictures was found on the visual probe task and for smoking-related words in the unmasked condition of the modified Stroop task. Self-reported urges to smoke were most strongly predicted by the latter, rather than the deprivation manipulation. No evidence for a pre-conscious bias for smoking cues was found.

Frequent alcohol use and frequent drinking for enhancement but not for coping or social reasons have been found to be associated with a bias to attend to alcohol reward cues and to disinhibited behaviour (Colder and O’Connor 2002).

**Issues and evaluation**

It is clear from experimental studies that individuals who engage more frequently in addictive behaviours have attentional and memory processes that prioritise stimuli relating to these behaviours. However, it has not been demonstrated that this represents an abnormality that causes the behavioural problem of impaired control or is a consequence of it.

In fact, as with Expectancy Theories, most of the research has focused on frequency of the behaviour as a target variable rather than loss of control. As with other theories mentioned in this volume, this one draws attention to an interesting phenomenon that may contribute to addiction in some cases but cannot be considered a comprehensive theory. The experimental method adopted shows associations but these only account for a small amount of the variance and there are many individuals who are addicted who do not show evidence of the biases proposed.

**Behavioural economics**

An interesting approach to the understanding of addiction seeks to borrow concepts from the field of economics and apply them to the psychology of choice behaviour. Vuchinich and Heather (2003) have produced an excellent edited volume that explores this in detail, and in fact we have already considered two theories that fall within the ambit of this approach (the Rational Addiction Model and Skog’s Choice Theory).

We should now consider behavioural economics more broadly. In its broader formulation, it is not entirely clear how far the mechanisms described in Behavioural Economic Theory are believed to operate within the realms of conscious decision-making but that is one interpretation so we will consider this theoretical approach here (Box 3.9).
Box 3.9 Behavioural Economic Theory

Economic conditions or principles influence the consumption of addictive substances, either determined by the availability of the drug or in terms of the decisions made by the individual regarding their drug use.

Addiction as a consumption behaviour

The Behavioural Economic Theory of addiction seeks to explain addiction in terms of the conditions that influence the consumption of addictive substances (Bickel et al. 1995). It views addiction as a process by which an increase in past consumption will determine an increase in current consumption and proposes that proportional changes in consumption can be explained by proportional changes in cost, defined broadly (Bickel et al. 1997). Behavioural Economic Theory describes addictive behaviour at both an individual and population level.

Trade-offs and interactions

Behavioural Economic Theory focuses particularly on the trade-off and interaction between different rewarding stimuli (reinforcers) and the resources that individuals will expend to experience those stimuli as a function of how far into the future those rewards will accrue.

Price elasticity

A central concept in economic theory is price elasticity. This is the ratio of the proportionate change in demand to the change in price. Inelastic consumption refers to a decrease in consumption that is proportionally less than price increases. Conversely, elastic consumption is a decrease in consumption that is proportionally greater than the increase in price. For example, a price elasticity of tobacco of −0.5 implies a 1% increase in price that, with all other factors remaining constant, would lead to a 0.5% reduction in demand. For the UK and US, the majority of studies have indicated that tobacco consumption is price inelastic, with a figure around 0.4–0.5 (Lewit 1989).

One major tenet of Behavioural Economic Theory is that behaviours that seek out a reinforcer become more frequent as ‘unit price’ falls where unit price is defined as the total resources, including price, time and effort, needed to acquire a particular ‘amount’ of a reinforcing stimulus. This is the case whether the change in unit price arises from changes in the response requirement per episode of reinforcement or from a change in the amount of reinforcement delivered per response.

It has been suggested that pharmacological and conditioned effects may contribute to this finding. Macenski and Meisch (1998) found that unit price predicted consumption even at low doses of orally administered cocaine: the taste of the solution had acquired conditioned reinforcing properties that allowed it to function as a reinforcer despite a very low dose.
Addiction as a shift in the demand curve

The behavioural economic view sees addiction as a change in the relationship between unit price and drug consumption. Simplistically this can be thought of as a shift to the right of a ‘demand curve’ as a dependent drug user consumes more at a given unit price. However, this requires a more detailed explanation as the slope of the function is subject to change. Flat functions or those with a minimal slope are hypothesised to show ‘inelastic demand’. It is predicted that as dependence progresses, the demand curve may shift to the right with drug demand becoming more inelastic with price changes having little effect on consumption. Higher intercepts and smaller slopes of demand curves associated with higher levels of dependence are expected to lead to stable patterns of intake and a greater difficulty in terms of controlling drug use.

Cross-price elasticity

The availability of one reinforcer may in principle have an effect on the demand for another reinforcer: ‘cross-price elasticity’ (Bickel et al. 1995). ‘Substitution’ refers to the situation in which the availability of one reinforcer reduces the consumption of another. A ‘complement’ is a reinforcer that increases the value of another.

There is little evidence to suggest that increasing the price of one substance results in consumers switching from one substance to another, as few significant cross-price effects have been found for alcohol or tobacco (Godfrey 1986). With regard to interactions between different substances, Jones (1989) found a subsystem of demand equations for tobacco, beer, wines and spirits using UK data for the period 1964–1983. Tobacco was found to be a compliment to four types of alcoholic drink, with the results suggesting that, for example, a 1% increase in tobacco prices would lead to a decrease in beer and wine consumption of 0.2% and a decrease in spirits and cider consumption of 0.5%, with all other factors remaining constant.

Delay discounting

The concept of delay discounting is an important feature of behavioural economic theory. It is the phenomenon in which an individual selects a smaller but more immediately available reward over a later but larger one. Under one view, delayed rewards are discounted exponentially with a constant decay parameter. Under another view, discounting follows a hyperbolic function in which the decay is greater later on (Madden et al. 1999).

There are numerous examples of delay discounting and some evidence that individual differences in the function underpinning it may account for propensity to develop addictive patterns of behaviour. In one study, discounting of delayed rewards by pathological gamblers was compared with the delayed discounting by non-gambling participants (Dixon et al. 2003) in a hypothetical choice task in which participants made repeated choices between $1000 available after a delay (varied from 1 week to 10 years across conditions) and
an equal or lesser amount of money available immediately. ‘Indifference points’
between immediate and delayed monetary rewards were calculated at each
delay condition by varying the amount of immediate money across choice
trials. The pathological gamblers discounted the delayed rewards more
steeply than did the control participants, favouring the more immediately
available rewards.

Discounting and impulsivity
Discounting has been proposed to account for the impulsivity evident in
drug-dependent individuals and the loss of control associated with drug use.
Impulsivity is related to behaviours other than addiction, which include delin-
quency, suicide, aggression, gambling and excessive spending.

Drug-dependent individuals have been shown to select brief but immediate
drug intoxication or relief of transient withdrawal symptoms over a variety
of prosocial but often deferred rewards (Madden et al. 1997). Also it seems
that intravenous drug users often choose to share hypodermic needles instead
of delaying their drug use until they have the opportunity to disinfect or obtain
clean needles. Besides claims that drug users exhibit impulsive behaviour and
that they score higher than controls on standardised measures of impulsiv-
ity, research has begun to compare the prevalence or degree of impulsivity
across different types of drug dependence disorders to assess whether this is
a general trait or whether impulsivity is specific to the type of reinforcer.

Loss of control
Loss of control is construed as somewhat different from impulsivity. In the
case of loss of control, drug-dependent individuals report that they would
prefer larger, more delayed rewards, but actually choose the smaller and
immediate reward (Bickel and Marsch 2001). The key difference between
loss of control and impulsivity is the inconsistency between behaviour and
expressed preference in the case of loss of control compared with the con-
sistent preference for an immediate smaller reward in the case of impulsivity.
Bickel and Marsch (2001) provide the example of drug-dependent individuals
expressing a strong preference for employment or family/friend relationships
over drug use, but after a short period of time they may use drugs instead
of going to work or spending time with friends or family.

Factors affecting discounting
It is suggested that educational level may play a role in the operation of delay
counting. Jaroni et al. (2004) found that less educated smokers were more
likely to discount future rewards than non-smokers.

State of tiredness may also play a role. There is evidence that the discounting
effect is exacerbated in participants who have been sleep deprived (Reynolds
and Schiffbauer 2004). This suggests that the operation of self-control requires
mental effort which in turn requires mental resources that are depleted under
sleep deprivation.
Polysubstance use, substitution and complementarity

Sumnall et al. (2004) examined the influence of price upon hypothetical purchases of alcohol, amphetamine, cocaine and ecstasy in a sample of poly-substance users. Self-reported demand for alcohol was inelastic. Amphetamine acted as a substitute for alcohol; cocaine was a compliment drug and ecstasy was found to be independent of alcohol price increases. Demand for amphetamine, on the other hand, was found to be elastic as its price increased, and alcohol was identified as a substitute for amphetamine. Other substances were found to be independent. Demand for cocaine was elastic. Alcohol and ecstasy were substitutes for cocaine, yet amphetamine was independent. Finally, demand for ecstasy was elastic and cocaine was substituted as ecstasy price increased. These results suggest that consumption involves socio-economic and psychopharmacological factors, although of course hypothetical situations and self-reports may not accurately represent actual behaviour.

In terms of ‘substitution’, it has been found that adolescents’ choice to smoke appears to depend on substitute reinforcers or complimentary activities to smoking and also individual differences in appraisal of the reinforcer value. For example, Audrain-McGovern et al. (2004) found that in a sample of adolescents the main ‘substitutes’ for smoking included school involvement, academic performance, physical activity and sports team participation. Complementary activities that influenced their decision to smoke included peer smoking or substance use.

Issues and evaluation

Behavioural economic theory offers a perspective on addiction that can be helpful. There is probably little in it that could not in principle be contained within other theoretical orientations but it does point to concepts that might otherwise be neglected. Of particular benefit are the concepts of the interaction between reinforcers with some acting as substitutes and some being complements.

One has to be very careful, however, in the application of economic ideas as general concepts. We cannot, for example, predict what will happen in terms of response substitution without knowing the precise purpose subserved by a particular reinforcer. We must also recognise that the worth of particular reinforcers is highly context dependent.

Finally, a detailed understanding is required of the behaviour one is dealing with; otherwise one can make some serious mistakes in interpretation of the evidence. A case in point is the price elasticity of cigarette consumption. We noted earlier that studies had found this to be in the region of $-0.4$ to $-0.5$. This means that a price rise in terms of purchasing power of 10% on average results in a reduction in consumption of about 4–5%. If one could address problems of black market sales, this puts tax increases at the top of the list of methods of reducing smoking in a population.

However, we have to recognise that a drop in consumption can arise either because smokers smoke fewer cigarettes, or because fewer people start...
smoking or because more smokers stop smoking altogether. There is less
evidence on the effect of price rises on smoking cessation but what there is
suggests that the price elasticity is more like $-0.2$.

One might still be satisfied that a reduction in consumption among those
continuing to smoke is worthwhile but when smokers reduce their consumption
in response to price rises, they do not end up inhaling less smoke – they smoke
the remaining cigarettes more intensively. Therefore, unless a price rise is able
to reduce the number of smokers, it appears not to have a useful effect on
the true amount of smoking. This is perhaps not surprising if smoking does
in fact represent nicotine-seeking behaviour with a preferred nicotine dose.

Some issues with mathematical and rule-based theories of choice

We noted earlier that there are many theories about behaviour that focus on beliefs
and evaluations and how these combine to influence behaviour. The most general
of these is probably one called Subjective Expected Utility Theory (see Baron 2000).
This is a mathematical model of the decision-making process in which for each
option being considered the decision maker examines how good or bad each of
the possible consequences is, then gives this a weighting according to how likely
he thinks it is to occur. The decision maker then adds up these ‘weighted utilities’
for each of the options being considered and chooses the one with the highest value.

Models as descriptions or as metaphors

Of course we know that people do not actually do this except on rare occasions,
but theorists have argued that they behave as though they do, subject to some notable
deviations. In the event, this model conflicts with how people actually think so it
cannot be applied in most real-life situations.

For example, our decision-making is generally much more haphazard. We do
not think numerically about the value of outcomes, and often we do not even think
about outcomes but about ‘characteristics’ of the options (for example, the aesthetic
qualities of a car we are thinking of buying) and so on.

These limitations have, surprisingly, not been of great concern to psychologists
and economists who are presumably willing to accept the patent inapplicability of
the model to real behaviour for the sake of something that can be expressed in
rather simple mathematical terms. A notable exception to this is found in the work
of Janis and Mann (1977) who developed a more realistic model in which the mode
of decision-making varies according to person and situation.

The importance of the moment

But another limitation has been noted and it is that actions are influenced by what
is going on at the time. This has been taken into account to a limited degree by
the Theory of Planned Behaviour, TPB (e.g. Hu and Lanese 1998) and to a greater
The Health Belief Model, HBM (see, for example, Garcia and Mann 2003). This addresses the issue by explicitly including the concept of ‘intention’. Unfortunately, intention in the TPB has been construed in terms of a more or less enduring state that may span anything from minutes to months. The HBM introduces the idea of ‘cues to action’ or triggers. These are immediate situational determinants of behaviours such as exercising, attending for screening or attempting to stop smoking. The limitation is that the model treats triggers as independent of the motives that lead individuals to consider the action in question.

Transitions from one addictive behaviour to another

There are a number of theories that focus on ways in which preferences change over time and as a result of experience. One such account relates to the observation that many users of dangerous drugs, such as heroin, began their drug taking with so-called ‘soft’ drugs such as cannabis. Changes in preference as a result of continuing drug use have a manifestation in a particular theory of its development that has gained wide currency among the public and politicians: the Gateway Theory (Box 3.10).

**Box 3.10 Gateway Theory**

This theory argues that becoming a user of one drug, usually a drug with less powerful effects and a lower propensity to addiction, causes an individual to be more susceptible to using another, stronger and potentially more addictive or harmful drug. One example is the view that using cannabis makes it more likely that one will go on to use heroin (e.g. Kandel et al. 1992).

This theory proposes a number of mechanisms by which this might occur. First, the ‘gateway’ drug could simply provide a taste of the reward that a more powerful drug has to offer. Secondly, an individual may develop tolerance to the gateway drug so that its effects become weaker, and in order to achieve the original reward the user is motivated to seek a stronger drug. This would be the same principle that in some cases underpins the transition from a weaker form of the same drug to a stronger form or a mode of delivery that yields a more powerful effect. Thirdly, by becoming involved with a group of people who use or deal in the gateway drug, the user may encounter a greater opportunity or social pressure to take the stronger drug.

**Soft to hard drugs**

The major observation that underpins the theory is that many of those who use ‘hard’ drugs such as heroin started with use of ‘soft’ drugs such as cannabis. However, transitions have also been examined between nicotine, alcohol and illicit drug use (Kandel et al. 1992; Lindsay and Rainey 1997; Kenkel et al. 2001; Beenstock and Rahav 2002; Chen et al. 2002; Tullis et al. 2003). The sequences appear to differ in different populations.
The problem of establishing causality
The problem is that mere observation of a transition from one drug to another does not necessarily imply a causal connection. It is equally plausible that the same characteristics that put people at risk of using ‘hard’ drugs also lead them to use ‘soft’ drugs, and use of soft drugs predates that of hard drugs as a result of availability or other environmental conditions.

One way to test a genuine causal relationship between use of one drug type and another is to assess how far factors that directly influence the first type (the gateway drug) at a population level later come to influence the second type. This has been done in one study in Israel in which it was found that price increases on tobacco reduced cannabis use, suggesting that tobacco may be a gateway to cannabis, but increase in the price of cannabis had no effect on later heroin use suggesting that in that country at that time cannabis was not a gateway drug for heroin (Beenstock and Rahav 2002). However, it is not difficult to come up with alternative explanations, and measurement error and choice of time scale may play a role in the findings.

Gateway Theory and behavioural economics
Gateway Theory has been formulated in terms of economic theory (Pacula 1997). It proposes that

‘in the case of multi-commodity habit formation . . . the marginal utility of initiating a new drug is higher when there is prior consumption of the other drug. Further, it is found that the individual will initiate drug consumption with that drug that has the lowest marginal cost. The particular sequencing of drug use that is observed in empirical data is explained by differences in the marginal cost of consuming legal and illegal drugs’ (from abstract).

Issues and evaluation
In terms of a general theory of addiction, the transition from use of one form of a drug to another needs to be able to be explained, but is likely to be highly dependent on the context. We are not yet in a position to say that using a ‘soft’ drug in itself makes it more likely that someone will start to use a ‘hard’ drug. In policy terms we do not know whether relaxing restrictions on soft drugs makes the prevalence of hard drug use more likely. In terms of addiction, we cannot yet say anything useful about the influence of use of one drug on development of addiction to another.

Recovery from addiction
A model that focuses more on attempts by individuals to change chronic behaviour patterns for the better is the Transtheoretical Model (TTM), sometimes called the Stages of Change or Cycle of Change Model. The model attempts to explain when, how and why individuals manage to do such things as stop smoking, restrain from
or stop drinking alcohol, engage in more exercise and so on (Box 3.11). It seeks to do this by combining a number of ideas from other theories into a single framework which regards such changes as a process of transition between ‘stages’.

Box 3.11 The Transtheoretical Model (TTM) of behaviour change

The TTM proposes that the process of recovery from an addictive behaviour involves transition through stages from ‘precontemplation’ in which no change is contemplated, through ‘contemplation’ in which it is contemplated in the foreseeable future, to ‘preparation’ in which plans are made for a definite attempt, to ‘action’ in which the attempt is made and then ‘maintenance’ in which the new pattern is established. Different processes are involved in the transition between different stages, and individuals can move backwards as well as forwards.

This model seeks to describe the processes that individuals go through in overcoming addictions and changing other chronic behaviour patterns. The Transtheoretical Model of behaviour change, also known as the Stages of Change (SOC) model, states that, with regard to chronic behaviour patterns such as smoking, individuals can be characterised as belonging to one of six ‘stages’ (Prochaska et al. 1985; Prochaska and Goldstein 1991; Prochaska and Velicer 1997): ‘precontemplation’ in which the individual is not thinking about changing in a defined period; ‘contemplation’ in which the individual is thinking about changing but not making any specific plans to change in a defined period; ‘preparation’ in which the individual intends to change; ‘action’ in which the individual is actively attempting to change; ‘maintenance’ in which the individual has engaged in the new behaviour pattern for a defined period; and ‘termination’ in which the individual has permanently adopted the new behaviour pattern. The ‘termination’ stage is a relatively recent addition to the model. The five main stages are described in more detail below.

**Precontemplation stage**

This stage represents individuals who are not interested in changing their behaviour and have no desire to do so in the immediately foreseeable future. Prochaska and DiClemente (1985) define the foreseeable future as a six-month time period, as this is the most far into the future that most people plan a specific health behaviour change. It is thought that the individuals who fall within this stage have a lack of awareness or appreciation of the specific behaviour. However, these individuals are aware of the effects of this behaviour upon their health, but avoid involvement in health behaviour change programmes as this may involve rationalisation of their behaviour. Individuals may be at this stage as a result of failure at the desired behaviour change and resent their past efforts. Potential for progression from the precontemplation to the contemplation stage may be mediated through
increasing awareness, which may be achieved through the mass media. Goals that are easily obtainable can increase self-efficacy or confidence that may also ensure progression to the contemplation stage.

**Contemplation stage**
Individuals at this stage are described as having the desire to change their behaviour within the next 6 months. Prochaska and DiClemente (1985) claim that this intention arises despite the individual’s having knowledge of potential barriers or constraints. A plan for behaviour change is then drawn up with these barriers in mind. The benefits of the desired behaviour change may be obvious to the individual, although the types of barrier that they encounter may be specific to each individual. Having inadequate finances to fund the behaviour change may be relevant for one individual whereas others may be concerned about the impact of the behaviour change in question upon their social activities. Individuals in this stage require extra attention, intervention or strategies. The individual’s progression towards the desired behaviour at a pace at which they feel most comfortable is emphasised and encouragement is required for motivation. Tailor-made messages are more preferable than general messages from the mass media at this stage.

**Preparation stage**
Individuals at this stage intend to make the behaviour change in the near future – that is to say, within the next month – and have usually made one previous attempt at the behaviour. The model has recently been changed so that a prior attempt to make the change is not necessary.

**Action stage**
The action stage may last from less than a month to as long as 6 months and is identifiable by a sufficient change of behaviour. This stage of change is usually the most identifiable as it involves a visible change in behaviour and usually receives the greatest external recognition. An individual in this stage will perceive the cons associated with the behaviour as greater than the plus points if they are to abstain from certain behaviours such as drug abuse or smoking. Or in the case of behaviour such as adherence to an exercise regime, the pros of the behaviour should exceed the cons. If the individual continues their pattern of behaviour, they will proceed to the maintenance stage.

**Maintenance stage**
This stage starts after the action stage and can last for several years. The individual’s level of self-efficacy is at its highest at this stage. In application of this model it is important to bear in mind the need for relapse prevention, in the form of a self-control programme designed to teach individuals who are trying to change their behaviour how to cope with the situation of relapse. Since this model is cyclical, it is possible for the individual to relapse back several stages rather than just the one stage.
Processes of change
The model further proposes that individuals progress through stages sequentially but usually revert to prior stages before achieving maintenance and then termination (Prochaska and Velicer 1997). The model also states that different processes are involved in moving between different stages (Prochaska and Velicer 1997). It argues that interventions to promote change should be designed so that they are appropriate to an individual’s current stage (Prochaska and Goldstein 1991). Moving an individual from one stage to another is purported to be a worthwhile goal because it will increase the likelihood that this person will subsequently achieve the termination stage (Prochaska and Goldstein 1991). Proponents of the model have argued that the model has revolutionised health promotion, claiming that interventions that are tailored to the particular stage of the individual improve their effectiveness (Prochaska and Velicer 1997).

Difficulties with the model
The following analysis draws primarily from research in smoking. It is in this area that the model was first developed and where much of the research relating to it has been carried out. To give some idea of the extent of the dominance of smoking, out of 540 articles found in PubMed using the search phrase ‘stages of change’, 174 also had ‘smoking’ in the abstract or title, 60 had ‘alcohol’, seven had cocaine, two had ‘heroin’ or ‘opiate’ and one had ‘gambling’.

Reservations have emerged about the model, many of which have been well articulated (Etter and Perneger 1999; Bunton et al. 2000; Whitelaw et al. 2000; Sutton 2001; Etter and Sutton 2002; Littell and Girvin 2002). One of these is a concern about the concept of the ‘stage’. The model draws arbitrary dividing lines in order to differentiate between the stages. This has to mean that these are not genuine stages.

For example, an individual who is planning to stop smoking is in the preparation stage if this is within the next 30 days (provided that the smoker has made a quit attempt that lasted 24 hours in the past 12 months) but only the contemplation stage if it is in 31 days’ time (Sutton 2001). Boundaries between so-called ‘stages’ are therefore just arbitrary lines in the sand and statements of the kind ‘x% of smokers are in the ‘contemplation stage’ have little useful meaning. They should not be taken to mean, as they so often are, that ‘x% of smokers are thinking about stopping smoking’.

Secondly, this approach to classifying individuals assumes that individuals typically make coherent and stable plans. People responding to multiple-choice questionnaires are compliant and generally will try to choose an answer, but this does not mean that they think about things in the terms set by the response options. Apart from those individuals who set a specific occasion or date for change (e.g. in a New Year’s resolution), intentions about change appear to be much less clearly formulated.
In what appears to be the first study of its kind, Larrabie (in press) found that more than half of reported quit attempts in a general practice sample involved no planning or preparation at all – not even going so far as to finish the current packet of cigarettes. Another recent study found considerable instability in intentions to stop smoking over short periods (Hughes et al. in press). A high level of instability in stages has also been found in other domains (De Nooijer et al. 2005).

Thirdly, it has been pointed out by others that the stage definitions represent a mixture of different types of construct that do not fit together coherently (e.g. time since quit, past quit attempts and intention) (Etter and Sutton 2002). It is not, as some of those using the model would like it to be, a statement of ‘readiness’ to change. Readiness or even preparedness is not actually assessed.

Fourthly, the model focuses on conscious decision-making and planning processes and draws attention away from what are known to be important underpinnings of human motivation. It neglects the roles of reward and punishment and of associative learning in developing habits that are hard to break (Baumeister et al. 1994; Mook 1996; Salamone et al. 2003).

Much of the problem of behaviour change arises from the fact that unhealthy habit patterns become entrenched and semi-automated through repeated reward and punishment (Robinson and Berridge 2003). These processes operate outside conscious awareness and do not follow decision-making rules such as weighing up costs and benefits. There is little or no consideration of the concept of addiction, which is clearly a crucial consideration when it comes to behaviours such as smoking.

Predictions made by the model have been found to be incorrect or less accurate than those of competing theories (Farkas et al. 1996; Herzog et al. 1999; Abrams et al. 2000). Strong claims have been made for the model (Prochaska and Velicer 1997) but the main body of evidence given in support of the theory is that individuals who are closer to maintenance at any one time are more likely to have changed their behaviour when followed up (e.g. Reed et al. 2005).

The relationship is often not strong, and by no means all studies find it (Hernandez-Avila et al. 1998; Littell and Girvin 2002) but the fact that it is present is given as evidence for the model. But this says no more than that individuals who are thinking of changing their behaviour are more likely to try to do so than those who are not, or that individuals who are in the process of trying to change are more likely to change than those who are just thinking about it. Put that way, it is just a statement of the obvious: people who want or plan to do something are obviously more likely to try to do it; and people who try to do something are more likely to succeed than those who do not.

Surprisingly, the proponents of the model appear not to report findings showing that the model is better at predicting behaviour than a simple question such as ‘Do you have any plans to try to . . . ?’ or even ‘Do you want
to . . . ?'. However, where others have made the comparison (e.g. SOC versus a simple contemplation letter that preceded it), little difference has been found (Abrams et al. 2000), or a simple rating of desire has been found to be better (Pisinger et al. 2005b). There have also been problems in the reliability of the assignment to categorical stages, as one might expect given that these are arbitrarily designated (Hodgins 2001). One might imagine that a scientific model would need to show an improvement at least on this kind of simple assessment.

Applications of the model
Proponents of the model may point to its having drawn attention to the fact that many people are not ready for interventions and progress can be made by moving them in the direction of changing their behaviour. However, in the years that the model has been in use, there appears to be no convincing evidence that moving an individual closer to action actually results in a sustained change in behaviour at a later date. In fact the history of behaviour change research is littered with studies that have succeeded in changing attitudes without accompanying changes in behaviour.

Where interventions have been developed that are based on the model, these have not proved more effective than interventions based on traditional concepts. A recent review comparing stop-smoking interventions designed using the SOC approach with non-tailored treatments found no benefit for those based on the model (Riemsma et al. 2003). Another review of the effects of applying the model to primary care behaviour change interventions has similarly found no evidence for a benefit (van Sluijs et al. 2004) and neither has there been found to be a benefit of applying the model in promotion of physical activity (Adams and White 2005). By contrast, there is good evidence that tailoring interventions in other ways, including triggers and motives, is more effective than untailored approaches (Lancaster and Stead 2002).

The popularity of the model
The popularity of the model can be put down to a number of factors. First, the seemingly scientific style of the assessment tool gives the impression that some form of diagnosis is being made from which a treatment plan can be devised. Secondly, the model also gives permission to go for ‘non-specific’ outcomes such as moving an individual from ‘precontemplation’ to ‘contemplation’ which is of no proven value. Thirdly, it provides labels to categorise people rather than using everyday language: an individual is a ‘precontemplator’ not ‘someone who is not planning on changing’. This appears to give the model scientific validation, which may or may not be founded on evidence.

Issues and evaluation
The above analysis suggests that the model tends to promote the wrong intervention strategy. For example, precontemplators tend to be provided
with interventions aimed at ‘moving them along’ the stages, for example by attempting to persuade them about the benefits of changing. However, if their apparent lack of interest in changing arises from their addiction, these individuals may respond favourably to the offer of a new and promising treatment as appears to have happened when the drug, Zyban, was launched as a smoking cessation aid (e.g. Zwar and Richmond 2002).

The model is also likely to lead to effective interventions not being offered to people who would have responded. There is now evidence in the case of smoking cessation that help should be offered to as wide a group as possible (Pisinger et al. 2005a,b) but the SOC model can be taken as giving permission to those attempting to promote behaviour change to give weak interventions or no intervention to ‘precontemplators’. This approach fails to take account of the strong situational determinants of behaviour. Behaviour change can arise from a response to a trigger even in apparently unmotivated individuals.

In the case of psychological theories for which there is accumulating evidence that they are not proving helpful, it is common to argue that better measurement is needed or that the theory has not been applied properly. This particular model is no exception (e.g. DiClemente et al. 2004). However, in the end, with some theories, one is often forced to accept that fundamental precepts of the theory are misplaced.

This analysis of the Transtheoretical Model is drawn mostly from West (2005), including large sections of text that are reprinted verbatim with permission.

So far in the development of a theory of addiction we have arrived at what can be summarised as follows: addiction results from choices that people make in which they weigh up the benefits as they see it of engaging in the activity with the perceived costs; their evaluations of the costs and benefits may be subject to biases of many kinds, including feelings associated with the behaviour and its outcomes and a priority attaching to the present over the future. In some cases at least, preferences for the activities are altered by habituation, sensitisation and/or physiological changes which increase the costs of abstinence. Although it can be incorporated within this account, it is worth now drawing attention to something in our lives that quite obviously is very special: ourselves.

Identity

There is a large body of literature on the topic of ‘identity’ or ‘self-labelling’. How we see ourselves is clearly of huge importance to our feeling of well-being and a major influence on our behaviour. This has been recognised in an interesting and insightful theory of behaviour change that encompasses addiction (Box 3.12).
Box 3.12 Identity shifts and behaviour change

Identity Shift Theory proposes that a value conflict develops as a result of increasing distress caused by behaviours. This prompts a small step towards behaviour change which if successful begins to lead to an identity shift. Increased self-awareness and self-confidence then fuel continued change (Kearney and O’Sullivan 2003).

The first step
Identity Shift Theory is used by its proponents to explain changes in a range of chronic behaviour patterns including overeating, smoking, excessive alcohol use and drug abuse. The version presented here developed out of a synthesis of the findings of a number of qualitative studies. The analyses revealed that key moments in achieving lasting behaviour change were a 'critical reappraisal of self and situation' which led to a small step towards change.

A new identity
If that was successful it produced ‘positive indicators’ of a new identity. More behaviour change followed leading to a positive feedback cycle in which the

![Diagram](image)
new identity strengthened the behaviour change that reinforced the identity. Like many of the other theories considered, this theory notes the importance of social and environmental influences that act as prompts or constraints. It notes further that the constraints are powerful and numerous, which is why attempts at behaviour change so often fail. Figure 3.2 shows the elements and influences operating within the model.

**Dissatisfaction plus trigger equals change**
One of the major insights of this theory is the concept of a build-up of dissatisfaction with the current situation and a trigger, which might be quite trivial or might be major, that often results in an immediate and unplanned step on the journey to behaviour change. This fits well with the evidence from Larrabie (in press) that many quit attempts involve no overt planning but result from triggers ranging from news of illness to weather conditions that prevent going out to buy a packet of cigarettes.

**Issues and evaluation**
This theory highlights what seems to be a pivotal concept in addiction. However, it is not a comprehensive account but focuses on particular features of behaviour change. If one were to propose it as a comprehensive theory, it would fall foul of apparent counter-examples in which behaviour change takes place without, for example, reference to longstanding goals.

The importance of identity in the development and recovery from addiction has been included in other theories. For example the Addicted Self Model (Fiorentine and Hillhouse 2000, 2004) proposes that the cessation of alcohol- and drug-dependent behaviour is more likely when the individual attributes failure to control the behaviour to a stable condition, disease, or some other permanent property of the self. The person has to accept that they must achieve complete abstinence.

**Addiction as the exercise of choice based on desires**
We have arrived at a theory of addiction in which the individual chooses in some sense to engage or not engage in the behaviour. The choice involves a weighing up of the costs and benefits of the behaviour which change over time and the appreciation of which changes over time. The costs and benefits may involve mental representations to which we do not have full conscious access. The choice is influenced by pharmacological and non-pharmacological factors, including one’s sense of self and what one wants to be, and possibly by biases in attention to and memory for stimuli related to the addictive behaviour.
Watering my Yucca plant and trying to overcome addiction

People who are not addicts often find it hard to understand the mind of the addict. In a TV interview I did recently I was asked in all seriousness: how can people still keep on smoking when they know it is so dangerous? The answer I gave was that of course they do want to stop smoking but even trying to make the change is not easy. But that answer does not help the non-addict really to know what it is like to be an addict, really to understand the addict’s perspective. So here is something that helps me put myself in the place of the addict.

I have a lovely and expensive Yucca plant. It was given to me as a present and I like it. I decorate my office. In my previous job my secretary used to water it. In my present job I do not have a secretary so I have to water it. Yucca plants are very hardy and do not need a lot of water but they do need some and this one is, even as I write this, dying through lack of water. How hard would it be for me to give it some water every now and then and keep it healthy? Not very. Have I been doing it? No. I have probably watered it five times in more than a year. Do I care that the plant will die? Yes I do.

So why on earth am I not watering it? While it was healthy-looking, on every occasion when the opportunity arose for me to water it – when I would think about it because I was going to the kitchen, I did not need to water it because it looked all right and I am so ‘rushed off my feet’ that I just do not have the time. It can wait. The trouble is it can always wait. Now it is in really bad shape, most of its leaves are brown and it must be close to death. Now perhaps it is too late. So I did not water it before because there was no single occasion when it really needed it at that moment and now I am not watering it because it’s probably too late to do anything about it. I’m in too deep.

There is nothing really pathological about this behaviour but for the sake of a very small effort I am killing something I like. You almost certainly have your ‘Yucca plant’, most people do. What are we talking about? Procrastination of course. We will do it, we mean to do it, we intend to do it . . . then there’s no point in doing it because it’s probably too late, and then it is actually too late. What will it take to make me to water my plant on a regular basis? Writing this piece is a start – maybe it is not too late to save it. But if we can turn a blind eye to our Yucca plants, imagine how much harder it is for the addict to make that life-changing decision to give up their addiction. The smoker, the drinker, the cocaine sniffer, are all OK . . . until they are probably not and then definitely not.

For the addict, of course, once the life-changing decision has been made to give up the addiction, the trouble really starts. For them, continuing to water their Yucca plant requires them to walk on hot coals to the watering hole and trudge back again laden down with a heavy ewer, all the while missing out of a party that is taking place across the street, not knowing if they are going to make it or even whether it is really worth it because the plant may die anyway. It is not really surprising that many of them do not make it.

Now I’m off to water my plant.
Chapter 2
DEFINITION, THEORY AND OBSERVATION

This chapter discusses the issue of definitions in the social and behavioural sciences. It adopts the prevailing definition of addiction as a starting point for discussion. It then examines what makes a good theory and finishes with a summary of some of the ‘big observations’ that a theory of addiction needs to accommodate.

Defining addiction (addiction is not an elephant)

This section explores the issue of definition of addiction and related concepts. It accepts the intention behind the current definition but suggests a particular wording to ensure that this intention is made clear.

What are definitions?
Definitions are rules for deciding whether or not a label should be applied to something. They are surprisingly difficult to formulate even for concrete objects such as furniture or large mammals. Addiction is an abstract concept. It has no objective existence and boundaries as would for example a chair or an elephant. Furthermore, it is socially defined which means that opinions can legitimately differ about what is the most suitable definition; it cannot be said that one definition is unequivocally correct and another one incorrect, only that one is more useful or is generally agreed upon by ‘experts’. Despite this, ‘addiction’ is one of the most important concepts in behavioural and clinical science.

The changing definition of addiction
The definition of addiction in authoritative texts on the subject has changed over the years. At one time it was defined as a state of physiological adaptation to presence of a drug in the body so that absence of the drug leads to physiological dysfunction which is manifest to the sufferer as unpleasant or even life-threatening ‘withdrawal symptoms’. An addict was someone who needed to take a drug in order to maintain normal physiological functioning.

For many members of the public and some researchers, this concept of addiction still holds sway. They have an image of the heroin addict suffering stomach cramps, runny nose and shivering or the alcoholic with hands shaking uncontrollably. In
some ways, this definition is attractive because it is clearly a physiological problem with a known or at least discoverable aetiology and mechanism of action. There is a physical abnormality that can be measured objectively.

Nowadays the term ‘addiction’ is applied to a syndrome at the centre of which is impaired control over a behaviour, and this loss of control is leading to significant harm. The fact that there is harm is important because otherwise addiction would be of limited interest. It certainly would not merit spending large sums of public money researching, preventing and treating it. There is impaired control in that an addicted individual feels a compulsion to engage in the activity concerned or else it takes on a priority in his or her life that seems excessive. In many cases the addicted individual expresses an apparently sincere desire not to engage in the activity but fails to sustain abstinence.

In this formulation, addiction does not just involve control: there is a syndrome that includes a heterogeneous collection of symptoms. This syndrome and associated diagnostic criteria are based on the alcohol dependence syndrome set out by Edwards and Gross (1976). Thus they still include withdrawal symptoms, cravings and tolerance (reduced drug effect with repeated use), of which more later.

The reason for the change in the way addiction is conceptualised is that withdrawal symptoms in themselves pose little social threat and are clearly not the main problem. Even where they are unpleasant or dangerous, they are of limited duration and can be treated. By contrast, the compulsion to use drugs or engage in particular behaviours poses a very serious long-term threat to the well-being of sufferers and others and is very difficult to tackle with interventions that are practicable and ethical. It is a much more deserving focus of attention.

Current variation in definitions of addiction

For some researchers, addiction only involves drug-taking behaviour. However, it is clear that other behaviours such as gambling can be addictive. Yet including other behaviours within the definition causes a problem. How do we differentiate addiction from involuntary tics on the one hand and obsessive compulsive disorder on the other? The solution seems to be inclusion of the term ‘reward-seeking’ in the definition. Thus the definition becomes: ‘a syndrome at the centre of which is impaired control over a reward-seeking behaviour’.

Arriving at a working definition

There is one further minor tweak of wording that is needed to implement what is presumably the intention behind the definition. The conventional formulation talks of ‘impaired control’. There is a risk that this might imply specifically that the disorder involves a weakening of self-control rather than an increase in drive to engage in the addictive behaviour. This is an empirical matter and is probably best avoided in the definition of the phenomenon. Therefore the preferred form of words is: ‘a syndrome in which a reward-seeking behaviour has become out of control’.
True addiction

One difficulty with the fact that addiction is socially defined is that people get caught up in the debate about the ‘true’ definition. Of course there are no true definitions; there are a variety of forms of words that can be used, each with slightly different nuances and all of which can be subscribed to by all but the most fastidious of researchers even though they may prefer another. On the other hand, there are definitions which do not fit the purpose at all because they exclude activities that need to be included or include activities that need to be excluded.

Thus, in the formulation proposed earlier, the term ‘reward seeking’ was used to enable exclusion of involuntary actions and obsessive compulsive disorder. Now it may be that the latter has commonalities with addiction but on current usage we believe that they are sufficiently different that they must be considered as separate phenomena.

And that brings us to another problem of the definition of socially defined constructs such as addiction. It is easy to get the object itself mixed up with things that are closely linked to it and may be taken as an ‘index’ of it but not actually part of it. An example may help to explain this. Addiction to smoking cigarettes can be described as ‘a trait of an individual in which the drive to smoke in opportunities when smoking is possible is so great that it overwhelms a strongly felt desire not to’.

One might argue that the only way of knowing whether someone is addicted is if they try to stop smoking and fail. However, what if they do not try? Are they not addicted? No, they are not ‘not addicted’ (sic), it is just that we cannot be sure if they are addicted or not. However, we can get a good idea by the fact that they are taking in a great deal of nicotine from their cigarettes or the fact that they light up a cigarette within minutes of waking every single morning. These are markers of addiction but they should not be part of the definition of it. Yet many smoking researchers treat these measures as though they are addiction.

Concepts of craving

An even more difficult example is that of ‘craving’. In common parlance this has a relatively straightforward meaning. It is a subjective feeling of a strong urge to do something. A person can say something like, ‘I have a craving for chocolate’ and someone else knows that this means that the person feels a powerful urge to eat some chocolate. Thus a ‘weak craving’ is an oxymoron. Someone who craves alcohol will often show all kinds of behaviours, including increased swallowing, increased heart rate, and sweating perhaps. They will become obsessed with thinking about alcohol and perhaps anticipate the pleasure and relief that will come from a large swig of whisky.

So researchers naturally come to think that some or all of these features should become part of the definition of craving. But why should they? What was wrong with the original definition accompanied by these other events as interesting correlates
of the phenomenon? If we go down the route of adding these other features, we find ourselves in a difficult position that not all those whom we wish to regard as craving, because, for example, they tell us that they badly need a drink, will show all or even some of these other signs. Worse still, craving for different things will manifest itself in different ways and so we can easily end up with different concepts of craving for different things that people crave.

Addiction and intoxication

Coming back to the definition of addiction, some have tried to argue that there must be intoxication for addiction to occur (Robinson and Pritchard 1992). Such arguments need not be founded on a search for truth; definitions and labels can have far reaching social, political and commercial consequences. In this case, the context was a discussion about whether cigarettes are addictive, and the proponents of the view that intoxication is required had received funding from the tobacco industry who at that time stood potentially to lose major court actions if juries accepted that cigarettes were addictive.

The proponents of the intoxication view were arguing that because nicotine is not intoxicating it is not addictive and so smokers were freely risking lung cancer in choosing to smoke – ‘because they like it’. This would mitigate the blame attaching to the tobacco industry for promoting and selling the only product in the world, apart from the suicide pill, that is lethal to the user when used exactly as intended by the manufacturer.

Individuals and professional groups are of course at liberty to define addiction in a way that meets their own needs. Some psychologists may feel comfortable with a psychological definition that emphasises the role of decision-making and choice, whereas behavioural pharmacologists may feel more comfortable with one that emphasises learning mechanisms and brain pathways.

Some smokers, who need to feel that they are in control, may prefer to think of addiction in terms that exclude smoking whereas others who need to feel comfortable about the fact that they have tried and failed to stop many times may be attracted to one that includes it. However, at some point we need to agree that a particular definition serves the purposes of research, clinical practice and policy better than others and that is what has happened with the adoption of a version of the Edwards and Gross conceptualisation.

When consensus fails

Another problem with addiction as a socially defined construct is that many people are not up to date with, or simply do not accept, the changing definitions. Thus many members of the general public, politicians, clinicians and even researchers may still subscribe to a definition that is outmoded. It was noted earlier that many people still subscribe to what was the consensual definition that focused on physiological dependence on a drug.
Four views on what is addiction

It is interesting to see what non-specialists regard as addiction. I asked a number of people with no formal grounding in the field of addiction or behavioural research to say what they thought addiction was. Here are four of the responses.

NB (Composer of a musical about alcohol dependence): ‘Addiction is, amongst many other things, probably as many things as there are addicts . . . 1. The constant attempt to try and fill a perceived lack of something, real or imagined, 2. A way of never having to say what you really feel . . . the consequence, or maybe the origination, being a physical, emotional and chemical dependence.’

CW (Theatre producer and manager): ‘An addiction is something we can’t stop doing without some kind of intervention; but then so are breathing and compulsive behaviours . . . perhaps there’s a lack of rational justification for the addiction and some sense that the addiction itself continues to “make us do it” . . . (and the judgement that the addiction is “bad”). Do we mix up addictions and compulsions? There seems to be a difference. Are addictions purely chemical and everything else compulsive behaviour? We usually recognise non-physical/chemical components to addictions but at the same time are familiar with an addict’s attempts to justify their addiction as part of the addiction itself. Common usage of terms like “physically addictive”, “psychologically addictive” and “addictive personality” suggests not only different kinds of addiction but also different attitudes to addiction. It is hard to keep a sense of “good” and “bad for you” out of it. So what am I trying to say? Addiction is something you don’t need to do (not something connected to survival) but you can’t stop yourself doing (not without help/suffering/effort) and it’s somehow self-perpetuating (it becomes a goal in itself). I’m reminded of philosophy classes (what would a philosopher say?), e.g. knowledge = justified, true belief – plus a connection between justification and what makes the belief true (to avoid counter-examples) and the ancient Greek concept of akrasia (weakness of the will, or choosing the lesser “good”) which is relevant to addiction (as soon as you analyse an example of weakness of the will it disappears because understanding it means it’s no longer irrational behaviour and so no longer weakness of the will – so is there such a thing?). Perhaps we do addicts a disservice understanding them and so justifying their addictions? We should rather tell them to . . .’

CC (Retired nurse): ‘Addiction is compulsive behaviour in a certain direction. It gives you pleasure. If a person is prone to addiction, if they are not addicted to one thing they will be addicted to something else. It is caused by a genetic susceptibility. The way to tackle addiction is to transfer it to something less harmful and reward them for doing it or put a more powerful motivation in place.’

CD (Author and actor): ‘For me, addiction is: security, only noticeable in its absence. A nag in the head. Can say nothing, do nothing, think nothing but “Want a fag”. Nothing else will hit the spot, but it doesn’t. It’s disappointing but the best thing at the same time And still thinking “The next one will do the trick”. A cigarette every half an hour is like having a hug for a dull and miserable life. The alternative is to whine constantly, and who wants to listen to that? So, smoke, smoke, smoke – cheaper than therapy.’

It is remarkable how these off-the-cuff, non-expert views capture much of the discussion that has gone into arriving at the current technical formulation.
Even though addiction is socially defined, there are some constraints that place absolute limits on what can be said. Thus, there is a safeguard against definitions that are motivated by biases or are ill-conceived: logic. Two statements that flatly contradict each other cannot both be true. Thus, someone who wants to propose that intoxication in which performance is impaired is necessary for addiction cannot also hold the view that methadone or cocaine are addictive because in doses commonly used they do not impair performance.

Addiction and dependence

It is useful to be able to distinguish between addiction as impaired control and ‘physical dependence’ as a state of physiological adaptation to a drug which then needs to be taken to prevent withdrawal symptoms. Some researchers like to talk about ‘psychological dependence’. This term can be useful if it refers to a state in which an individual, for whatever reason, feels that he ‘needs’ something. As such it is different in a subtle way from addiction which is a syndrome involving a behaviour and feelings.

The problem is that addiction and dependence are often used interchangeably; it is unlikely that we will ever be in a position where there is a strong enough consensus on the definition of and distinction between these terms to make a play for a formal definition.

Interestingly, in common parlance we tend to use different terminology for different addictions: we say ‘heroin addiction’ not ‘heroin dependence’ but ‘alcohol dependence’ not ‘alcohol addiction’. With benzodiazepines we tend towards ‘dependence’ rather than ‘addiction’. With nicotine and stimulants we feel about equally comfortable with either term. At the risk of using a language that reads rather strangely, this book will consistently use the term ‘addiction’ to refer to the syndrome at the heart of which is impaired behavioural control and try to avoid the term dependence. However, use of language sometimes provides clues to how well underlying concepts fit together and it is worth considering whether the different application of the terms ‘dependence’ and ‘addiction’ to different drugs reflects differences in the phenomenology of the problem.

Diagnosing and measuring addiction

A feature of the definition of addiction that currently prevails is that it is a clinical disorder and in fact it is a disorder of motivation. We also know that it is chronic and in many cases life-threatening. Like most psychiatric disorders, and many physical disorders, it is diagnosed by reference to a set of symptoms rather than an underlying pathology. There is no laboratory test or scan that can be used to say that an individual is suffering from addiction; the symptoms are the sole means of determining whether the disease is present in any one case. But the symptoms are only markers and for various reasons an individual may have some symptoms but not others.

There are two main sets of criteria in common use, the World Health Organisation’s ICD-10 and the American Psychiatric Association’s DSM-IV. Table 2.1 shows how these compare with each other.
Table 2.1 Diagnostic criteria for addiction.

**Diagnostic and Statistical Manual-IV (APA 1995)**

A maladaptive pattern of substance use leading to clinically significant impairment or distress as manifested by three (or more) of the following, occurring at any time in the same 12-month period:

1. Substance is often taken in larger amounts or for longer period than intended
2. Persistent desire or unsuccessful efforts to cut down or control substance use
3. A great deal of time is spent in activities necessary to obtain the substance (e.g. visiting multiple doctors or driving long distances), use the substance (e.g. chain smoking), or recover from its effects
4. Important social, occupational or recreational activities given up or reduced because of substance abuse
5. Continued substance use despite knowledge of having a persistent or recurrent psychological, or physical problem that is caused or exacerbated by use of the substance
6. Tolerance, as defined by either: need for larger amounts of the substance in order to achieve intoxication or desired effect; or markedly diminished effect with continued use of the same amount
7. Withdrawal, as manifested by either: characteristic withdrawal syndrome for the substance; or the same (or closely related) substance is taken to relieve or avoid withdrawal symptoms

**International Classification of Diseases-10 (WHO 1992)**

Three or more of the following must have been experienced or exhibited at some time during the previous year:

1. Difficulties in controlling substance-taking behaviour in terms of its onset, termination, or levels of use
2. A strong desire or sense of compulsion to take the substance
3. 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
4. Persisting with substance use despite clear evidence of overtely harmful consequences, depressive mood states consequent to heavy use, or drug-related impairment of cognitive functioning
5. Evidence of tolerance, such that increased doses of the psychoactive substance are required in order to achieve effects originally produced by lower doses
6. 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
Some problems with the DSM and ICD criteria

These diagnostic criteria leave considerable scope for interpretation. For example, how strong does a feeling of desire or urge need to be to count as craving? How severe do effects of withdrawal have to be to count as withdrawal ‘symptoms’? How harmful do the consequences have to be for them to count? The result is that determining whether or not an individual who engages in a behaviour is addicted cannot be specified objectively, but only by reference to judgements made in a clinical interview, or responses that the individual makes on a diagnostic questionnaire. The arbitrariness is compounded by the specification that only a subset of symptoms is required for the diagnosis to be made. In principle, two ‘addicts’ could have non-overlapping sets of symptoms. However, this use of ‘disjunctive’ symptom sets in diagnosis is essential because some of the criteria have little relevance to some types of addictive behaviour. For example, chronic tolerance is marked in the case of alcohol but less so for cocaine. Giving up activities because of the addiction is more relevant to intoxicating drugs such as alcohol than drugs such as nicotine that do not interfere with normal functioning.

This suggests that the current diagnostic criteria need further revision. That is something to which this book returns in the last chapter. It is worth noting at this point, however, that revision of a set of diagnostic criteria and conceptualisations is not something to be undertaken lightly because it creates a disjunction with the past. This affects prevalence estimates as well as estimates of such parameters as heritability. Rounsaville and colleagues provide a useful discussion of problems associated with current diagnostic criteria but stress the need for caution when revising them (Rounsaville 2002).

Theory and supposition

This section describes what a theory should be and what makes a good or bad theory. It points out that theories in the field of addiction are rarely tested adequately in this field. This is because the dominant research methodology does not allow it. However, there is an approach that does allow testing of theories and by this method at least some of the theories that have been proposed fail the test. The test is to find somewhere in the real world or the world of experiment a counter-example that conflicts with the theory. This may seem an obvious test but it has been used surprisingly rarely.

What makes a good theory?

Theories are central to science, but they form only a part of it. They are discrete, coherent accounts of a process that are arrived at by a process of inference, provide an explanation for observed phenomena and generate predictions. Much of science does not fall into this category because it consists of disparate observations or is descriptive rather than explanatory. The same is true for the field of addiction.
Most of what is known or believed could not be called theories. A good theory should:

- explain a related set of observations;
- generate predictions that can be tested;
- involve no more concepts or elements than are necessary;
- be comprehensible;
- be coherent;
- be internally consistent;
- not be contradicted by any observations.

All this sounds reasonable but actually it turns out to be a not very useful set of principles for theory development. The problem is that there are any number of different theories that could be developed and compete for our attention, each good in some ways and not so good in others. We end up with a situation where there are lots of theories each with their proponents and with new ones being developed that serve some new purpose but do not supplant ones that already have their proponents. If we want theory to progress, we have to have a set of rules for deciding that what we have at the moment is not up to the job and in what way.

**Theory development**

Theory development should ideally start with the simplest possible account and set a high threshold for seeking to supersede it. It should ask four questions of the existing body of theory:

- Is it contradicted by observations?
- Does it fail to encompass important relevant observations?
- Does it have more elements than are needed?
- Is it misleading?

The answers to those questions should provide the starting point for modification.

**Theory development in the social and behavioural sciences**

Unfortunately, the prevailing approach in the field of addiction, like behavioural and social science generally, has been to develop theories with a less than complete analysis of what is already in the literature. In practice, theories of addiction have typically been developed because a researcher has, very understandably, wished to emphasise a particular approach to understanding a set of phenomena, or out of a set of specific observations from which the researcher has wished to generalise.

It is very rare to see direct comparisons between theories, and when these are undertaken they do not usually result in a resolution. The result is that theories in this field tend to take a particular approach to a topic rather than attempting a
comprehensive coherent account of the topic, and they frequently (though not always) involve a number of propositions that are not connected in a coherent framework so that any one of them could be false without others being false.

In principle there is a major difference between a 'theory' and a 'model'. A model is a description or representation of a system, an object or characteristic set of events. It need not explain anything. A theory seeks to explain and predict by proposing the existence or operation of entities that have not been observed. In practice, the two often become confused and indeed it is not clear whether the proponents wish their 'account' of addiction to be regarded as one or the other or both. Therefore this volume considers both theories and models, and hybrids.

Test and testability

It was stated earlier that theories in behavioural sciences are rarely tested using a stringent system that would require them to be abandoned. This needs further explanation. There are two reasons for the problem: theories are rarely specified with enough precision to be sure that they could definitely not account for a particular set of observations, and secondly our formal measurement is rarely precise or reliable enough to be sure that we have disproved a prediction from a theory. As Meehl (1978) has pointed out (see below) in behavioural and social science the result is that theories go out of favour rather than being abandoned, and most researchers seem able to accept that multiple theoretical approaches can be applied. This seems democratic and reasonable but it is not conducive to progress.

A new approach is needed

Our approach to testing theories needs to be much more stringent. At present a researcher will claim that a theory is supported because it predicts an association between two variables and indeed a correlation is found of, let us say, 0.30 with a significance level of $p = 0.01$. But is this a good enough test?

Arguably not. Logic dictates that theories can only be truly tested by looking for and finding counter-examples. Examining whether a correlation is significant or not does not provide this opportunity. The correlation may not be significant because of weak measurement or interference from other influences or just sampling error, and it may be significant because of a common confounding variable, common measurement error or sampling error.

One of the clearest examples of this is in 'tests' of a model called the 'Trans-theoretical Model' (TTM) of behaviour change (Prochaska and Velicer 1997). This will be described in more detail later, but in essence the model states that individuals can be classified into 'stages' according to whether they are planning to make the change in some arbitrarily defined timescale or are indeed in the process of making the change or have made the change. The major tests of the model show that
individuals who are planning to change or are in the process of change are more likely to have changed when followed up later.

Yet this would be true even if this particular theory were false in all important respects, for example about the idea that individuals fitted into ‘stages’. In fact the association is weak and most individuals fail to show the hypothesised progression, but these are not considered as counter-examples that would lead to the theory being abandoned because the theory is not expressed with that level of precision. To date the predictions of the theory have not been compared with a much simpler model in which individuals are presumed just to have a particular level of desire for change that is more or less stable over time and when interacting with opportunities for change trigger the change attempt. In these respects the methods used to test the theory do not seem conducive to an incremental science.

Counter-examples and observations of natural behaviour

Many of the counter-examples that can be used to test theories can be found in observation of natural behaviour. It could be argued that we need a radical shift in our approach to the study of behaviour. Rather than assuming that the body of knowledge is what we find from experiments and formal studies with sporadic and unsystematic reference to ‘anecdotal evidence’, we take as a basic starting point ‘observation of nature’ and consider formal experiment as a means to help explain this.

Our theories must at the very least account for what we know to be true about behaviour in the real world. Developing elaborate theories about the fine detail of results of experiments that deal with essentially trivial behaviours will not provide us with the advances we seek. Observations of nature can in fact be very powerful for testing theories because of one principle: if just one example of behaviour observed in the real world conflicts with our theory, our theory must be wrong.

By these criteria, several dominant theories of behaviour must be wrong because counter-examples abound in the real world. The TTM, already mentioned, is a case in point. It states that people can be categorised into stages based on when they plan to make a change. However, one does not need to undertake extensive surveys to know that large numbers of people do not make plans in that way.

If just one smoker can be found who is thinking that he might try to stop smoking but has not decided definitely (and without being asked) whether it will be in 3 weeks, 3 months or 9 months, s/he contradicts one of the fundamental tenets of the theory. If one smoker can be found who was not thinking about stopping smoking one minute, and then the next minute, because of something he hears or sees, decides never to smoke again without making any plans at all, the theory is surely wrong.

Proponents may argue that this is not a theory about individuals, but then the status of the constructs becomes so ambiguous that the theory loses meaning. How many counter-examples would a theory that is not about individuals need before the theory could be considered wrong?
Another example is the Theory of Planned Behaviour (see Levin 1999). This asserts that behaviours arise from intentions, and intentions arise from a combination of attitudes to the behaviour (a weighted sum of evaluations of the consequences of the behaviour), subjective norms (beliefs about what important other people think about the behaviour weighted by motivation to comply) and perceived control (belief in ability to carry out the behaviour).

In practice, even when the measurements are taken in precisely the manner dictated by the theory, the variables concerned rarely explain more than about 10% of the variance. They explain a lot more of the variance in self-reports of behaviours that have already taken place but this could easily be because respondents do not want to make themselves look inconsistent. Does this mean that the theory is correct or incorrect? Proponents of the theory argue that any association that is significant supports the theory but this neglects the fact that most people in the study did not follow the predicted pattern. In fact the theory is contradicted by finding examples of instances in which individuals do not think in the terms defined by the theory; when deciding what to do they do not consider the consequences of their actions but use broader moral or ethical principles.

The weakest form of evidence for a theory

Probably the weakest form of test that researchers apply to theories is captured by the statement: ‘X observation is consistent with the theory that Y’. This is such a weak statement it should have no place in anything with aspirations to being a science. To use an extreme example for illustrative purposes: the observation that children put out stockings to be filled with presents at Christmas is consistent with the theory that Father Christmas is real and lives in the North Pole. Why do we use this kind of evidence? It appears that we do so because we want our audience to read more into the term ‘consistent’ than a literal interpretation warrants. For example, here is an abstract verbatim from a study on nicotine dependence (Mogg et al. 2005):

**Rationale:** Different theories of addiction make conflicting predictions about whether attentional and approach biases for smoking-related cues are enhanced, or reduced, as a function of the level of nicotine dependence.

**Objective:** These theoretical views were evaluated by examining cognitive biases in smokers.

**Methods:** We monitored the eye movements of 41 smokers (predominantly young adults, who smoked from one to 40 cigarettes per day) as they completed a visual probe task in which smoking-related and matched control pictures were presented. Participants also completed a stimulus–response compatibility task, which measured the tendency to approach smoking-related cues, and a rating task.

**Results:** Smokers with lower levels of nicotine dependence showed greater maintained attention and faster approach responses to smoking-related cues. Longer gaze times for smoking cues were associated not only with lower levels of nicotine dependence, but also with higher levels of craving.
Conclusions: Overall, the results seem consistent with an integrated ‘incentive-habit’ model of addiction.

Choosing this example is not intended to belittle this work from one of the world’s leading research groups. It is chosen just to illustrate a very common approach to linking theory to empirical evidence. There is no doubt that the study provided interesting data but arguably the use of the phrase ‘consistent with’ in the conclusion reveals that in fact the link between theory and data is extremely tenuous.

Meehl’s insights into the failure of soft psychology to make progress

The failure of psychology to make progress because of an inappropriate approach to the updating of theories in the light of observation was beautifully described by Meehl some 30 years ago (Meehl 1978). He observed that theories in soft psychology are neither refuted nor corroborated but fade away as people lose interest. There are many intrinsic difficulties with the subject matter that contribute to this. A reliance on ‘significance testing’ is partly responsible. Pretending that there is a relationship if it has a $p$ value of less than 0.05 (or any other value) and not if it does not, and interpreting patterns of $p$ values in terms of relationships that ‘exist’ or ‘do not exist’ is just nonsense. Consistency tests that aim to estimate numerical parameters are a much better approach.

The sensitising role of theory

The preceding analysis has presumed a Popperian view of theories as statements that are tested against evidence and potentially falsified. Science progresses, it is argued, by a closer and closer approximation to reality in both coverage and content. There is another view. Blumer (1969) argues for the ‘sensitising’ role of theory: to generate ideas and concepts that help understanding. It could be argued that it is this role that is being fulfilled in theories in psychology.

Thus the role of the Theory of Planned Behaviour, for example, is to alert us to the importance of specific attitudes to a behaviour – our beliefs about what people who matter to us think, and our confidence in being able to successfully complete the action – in determining our intentions to do something. It also emphasises the fact that intentions play an important role in attempts to stop smoking (Norman et al. 1999).

The fact that it fails to consider other important motives, or that in its specific formulation it makes claims about how people combine their motives that are clearly untrue, is less important than the way in which it sensitises us to these ideas.

This is a useful role for theory but if one is willing to propose a theory that even as it is being proposed conflicts with ‘big observations’ and therefore must be wrong, there is no hope for progress in theory development. In psychology, economics and other behavioural and social sciences, there is a remarkable tolerance for a multitude of theories, many of which overlap considerably, many of which deal with a small part of a system (e.g. the motivational system) that is in fact integrated and many of which are contradicted by big observations.
When on occasions a researcher seeks to compete one theory against another (see Farkas et al. 1996), it is considered almost impolite and is rarely if ever successful because the proponents of the losing theory can always find ways to claim that the competition was unfair or as a last resort that their theory is basically correct and can be modified to take account of apparently conflicting evidence rather than abandoned.

We can summarise the process of theory generation currently in operation as follows: a researcher has an insight which s/he believes is novel and has the intellectual capability and motivation to turn it into a theory; s/he does this with insufficient regard to other theories or big observations that would mean that the theory as stated must be incorrect even though the basic idea is useful; the ideas in the theory capture the imagination and appeal to a following who then start to use it even though it has not been rigorously tested against competing theories. Such theories play a useful sensitising role for the ideas being presented but at the expense of neglect of other ideas that are as or more important when considering the behaviour in question.

Perhaps one should not underestimate the importance of establishing a personal reputation or even financial reward in this process. It is easy to forget that researchers, just like anyone else, have personal motives for career advancement and the plaudits of our colleagues.

For theory development to proceed, what is needed is a much fuller analysis of what problems the proposed theory addresses and a recognition that, while the proposed theory may make statements that cannot currently be tested, it absolutely must not be contradicted by observations that are already available.

‘Big questions’ in the field of addiction

This section sets out some major observations that any comprehensive theory of addiction needs to be able to accommodate.

A comprehensive theory of addiction has at the very least to account for what we know about the phenomenon: and we do know a great deal. It has to account for the diversity of the experience of addiction, the social and economic data relating to addiction and major findings from the field of neuroscience. Throughout this volume we will try to draw attention to observations and systematic research that bear on the development of theory, but we will begin with a brief summary of some of the big questions.

Some big questions

(1) Why are some drugs and activities more addictive than others?

It has been estimated that at the start of the 21st century there are 200 million people using illicit drugs (primarily opium, heroin, cocaine, stimulants and cannabis)
of whom some 15 million are suffering from a ‘drug use disorder’. A further 76 million are suffering from alcohol use disorders and 1000 million people smoke tobacco of whom a majority could probably be classified as dependent (see www.who.int/substance_abuse/facts/en/ and United Nations Office on Drugs and Crime 2005).

All addictive drugs act directly on the central nervous system. Most are rewarding in the sense that a substantial proportion of users report enjoying their effects and some are only rewarding in the sense that they relieve unpleasant feelings. Even in those that provide enjoyment, the degree to which they are addictive is not directly correlated with the amount of enjoyment that users obtain from them, nor the strength of their other subjective effects (e.g. the extent of intoxication).

Nicotine from cigarettes produces modest effects and mild enjoyment but is apparently more addictive than cannabis, alcohol and cocaine. Some addictive drugs lead to physiological adaptation so that the body does not function properly when they are removed. But there are many drugs to which the body adapts so that sudden withdrawal leads to unwanted withdrawal symptoms that are not addictive. Addictive activities that do not involve drug-taking are also rewarding in the broad sense, at least to some people, but not every rewarding activity is addictive.

(2) **Why does addiction take repeated exposures to develop but then remain roughly at the same level with further exposure?**

It can take months or years from the initial sampling of an activity for addiction to develop (e.g. Robinson et al. 2004; Vitaro et al. 2004). In some cases the activity is sampled and then not tried again for some time. Then there appears to be a period that can be just a few months or several years while the severity of dependence increases. After that time there is no evidence of an increase. It should also be noted that the positive experience that addicts obtain from their drug is never abolished through habituation; even long-term heavy users of a drug, for example, obtain pleasurable sensations from it.

(3) **Why are some individuals, strata in society, ethnic groups and cultures more susceptible to addictions than others?**

Prevalence of almost all recognised addictions is higher in men than women but this is subject to cultural and temporal variation (e.g. Pomerleau et al. 1993; Burt et al. 2000; Zilberman et al. 2003). In the case of most addictive behaviours, it is associated with economic deprivation. There are also substantial cultural and regional variations. At an individual level onset of most addictions is more likely in people who show greater propensity to antisocial behaviour as well as anxiety and depression. Being subject to physical or sexual abuse as a child is strongly linked to development of addiction to illicit drugs and alcohol (see for example Wilsnack et al. 1997; Marcenko et al. 2000; Langeland et al. 2003).
(4) Why are many people able to stop engaging in an activity to which they have been addicted but very few are able to maintain a pattern of non-addicted use?

A large proportion of individuals addicted to alcohol, heroin, cocaine, nicotine and gambling appear eventually to stop engaging in the activity (Granfield and Cloud 2001; Klingemann and Sobell 2001; Russell et al. 2001). In many cases this seems to occur without outside help. On the other hand, for individuals who have developed an addicted pattern of use, the prospect of achieving a stable pattern of non-addicted use is slim. This is true for smoking (Hajek et al. 1995) and alcohol (Mann et al. 2005).

(5) Why are withdrawal symptoms not strongly related to failure to maintain abstinence?

Some addictive drugs such as heroin and alcohol have a very clear withdrawal syndrome involving unpleasant physical symptoms (Hughes et al. 1994). Withdrawal from other drugs such as nicotine clearly leads to a well defined set of psychological symptoms and some minor physical symptoms. With some drugs such as amphetamine it is less clear that there is a distinct syndrome but mood disturbance is common. But how important are these to the pattern of compulsive use observed? The evidence suggests that the relationship is modest at best.

(6) Why do some situations increase the likelihood of relapse when people are trying to stop the addictive activity?

Unpleasant events, periods when there is a lack of interesting things to do, situations and stimuli that have previously been associated with the activity and the sight of someone else engaging in the activity all increase the likelihood of relapse (e.g. Annis 1990).

(7) Why are some interventions and treatments for addiction more successful than others?

What we know about effective treatment for addiction can be summed up relatively easily: we know surprisingly little (Lingford-Hughes et al. 2004).

With heroin addiction we know that long-term substitution of methadone or buprenorphine can reduce or in some cases eliminate heroin use. We do not know whether or not psychological support such as counselling helps to reduce or eliminate heroin use. Neither do we know whether or not the use of a gradually diminishing dose of methadone or other drugs helps a heroin user to become completely drug free.

With alcohol addiction we know that a programme of psychological support of some kind (we do not know what) can increase the ability of alcoholics to remain abstinent for months or even years. We also know that the drug acamprosate can help.
With nicotine addiction, we know that a relatively brief programme of psychological support lasting for about 6 weeks increases the ability of smokers to remain abstinent for at least 12 months. The effect is not large: between 1 in 20 and 1 in 10 smokers stop smoking who would not otherwise have done. We also know that the various forms of nicotine replacement therapy (NRT) such as gum, skin patch and lozenge, taken for up to 12 weeks, have a broadly similar effect. Other drugs are similarly effective: the antidepressants bupropion (Zyban) and nortriptyline, the nicotinic receptor agonist varenicline, and the cannabinoid (CB1) receptor antagonist, rimonabant. Putting psychological support and NRT or Zyban together it seems that as many as 10–15% of addicted smokers can be helped to stop for at least 12 months over and above those who could have stopped anyway.

(8) Why do different addictive drugs follow particular consumption distributions in the population?

Alcohol has a pattern with a small proportion of total abstainers (in drinking cultures), together with a unimodal (with one peak) distribution with most of the population drinking at a level that is not harmful but a ‘tail’ that extends into very high and damaging levels of consumption. Heroin is used by only a very small minority of the population (probably less than 1% of the population in the US). These patterns, and patterns of use of other drugs, are discussed later in this chapter.

(9) Why do different addictive drugs show different temporal patterns of use?

Alcohol is mostly used in drinking sessions that occur during leisure time; these sessions are terminated at or below moderate levels of intoxication but often they continue until a high level of intoxication is reached. For some individuals, these ‘binges’ are frequent but still limited to leisure time. Some individuals drink most of the day at a level that causes intoxication. Smoking patterns tend to follow a pattern that reflects restrictions. Where it is permitted at all times and can be afforded easily, it tends to occur at regular intervals throughout the day, but where there are restrictions naturally it clusters around certain times and places. These patterns and temporal patterns of usage of other drugs are considered in more detail later.

The natural history of addiction in individuals

The development of addiction follows many different trajectories but there are some commonalities. An activity is sampled first of all, often with no particular intention to make it a regular thing. The individual is curious to see what it is like and unafraid that just sampling the activity will lead to serious lasting harm. This lack of fear may be because the individual does not believe that sampling the activity will lead to harm, or it may be because the person does not mind particularly about the harm that it could lead to, or it may be because the individual does not give the future any particular thought.
Initially the activity occurs at a relatively low rate and is linked to particular situations or particular times. The individual will report that the behaviour is enjoyable or serves some particular purpose. Then it becomes more frequent and the individual will seek out more opportunities to engage in it. This may mean giving up other activities. It may also mean engaging in additional activities to support the behaviour in question.

The enjoyment of the activity may or may not change during this period, but a sense of wanting to engage in the activity or even ‘needing’ to engage in it will develop. This does not necessarily occur at all times, but may be tied to situations or times when there is seen to be an opportunity to engage in the behaviour. If the activity cannot be engaged in, the individual will often experience unpleasant effects. In the case of some drug addictions, these are generally a combination of adverse mood and physical symptoms.

At some point the individual may notice (or have it drawn to his or her attention) that the behaviour is causing problems, and may seek to reduce its frequency or stop it altogether. This results in an increase in feelings of need to engage in the activity that has to be countered by restraint. In most cases, the restraint is insufficient to prevent a ‘lapse’ in which the behaviour re-occurs. This lapse then usually turns within a short period into a ‘relapse’ where the individual resumes his or her activity at approximately the same rate as before. The individual, feeling that he or she is unable to exercise restraint, will sometimes seek help with doing so. With the benefit of this help, the individual stands a better chance of maintaining abstinence, though relapse still occurs in the majority of cases. The longer an individual, with or without help, is able to maintain abstinence, the greater his or her likelihood of continuing to maintain it indefinitely.

More on the addictive potential of drugs and activities

Addiction occurs mostly to the activity of taking particular drugs, but it can also occur to other activities, most notably gambling. Drugs that cause addiction in a significant number of users are typically those that give an experience of pleasure or reduce feelings of anxiety. Table 2.2 gives a list of activities or consumables that at least some researchers consider are addictive to some people (see for example Irons and Schneider 1997; Bruinsma and Taren 1999; Holden 2001; Potenza 2001). It offers a possible view on the level of ‘addictive potential’ of these drugs or activities. In some cases, as with nicotine and heroin, this view is not controversial. In other cases, such as shopping and eating tasty food, views legitimately differ from the proposition that they have moderate addictive potential to none at all.

For the US, the National Comorbidity Survey provided estimates of dependence potential of some drugs using a national sample aged 15–54 years (Warner et al. 1995). The survey tallied lifetime prevalence of drug addiction using DSM-IIIR criteria and it also examined ‘ever use’. Tobacco dependence was by far the most prevalent of drug dependencies: among ever users (used at least once in a lifetime) 32% of tobacco users were dependent compared with 23% of heroin users, 17% of cocaine
Table 2.2  Drugs and activities that at least some people have seriously claimed to have addictive potential. Addictive potential in this case refers to the proportion of ever users who develop an addictive pattern of use at some point in their lives. At best only rough estimates are possible. High ~ >20%, moderate ~ 10% to 20%, low ~ 5% to 10%, very low ~ <5%.

<table>
<thead>
<tr>
<th>Drug/activity</th>
<th>Plausible estimate of ‘addictive potential’ (proportion of ever users who become addicted)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Those with at least some usable data</strong></td>
<td></td>
</tr>
<tr>
<td>Heroin</td>
<td>High</td>
</tr>
<tr>
<td>Methadone</td>
<td>High</td>
</tr>
<tr>
<td>Nicotine</td>
<td>High</td>
</tr>
<tr>
<td>Amphetamines</td>
<td>Moderate</td>
</tr>
<tr>
<td>Ecstasy</td>
<td>Moderate</td>
</tr>
<tr>
<td>Cocaine</td>
<td>Moderate</td>
</tr>
<tr>
<td>Alcohol</td>
<td>Moderate</td>
</tr>
<tr>
<td>Marijuana</td>
<td>Moderate</td>
</tr>
<tr>
<td>Benzodiazepines</td>
<td>Moderate</td>
</tr>
<tr>
<td>Gambling</td>
<td>Low</td>
</tr>
<tr>
<td><strong>Those with little usable data</strong></td>
<td></td>
</tr>
<tr>
<td>Inactivity</td>
<td>Moderate</td>
</tr>
<tr>
<td>Tasty food</td>
<td>Moderate</td>
</tr>
<tr>
<td>Barbiturates</td>
<td>Low</td>
</tr>
<tr>
<td>Inhalants</td>
<td>Low</td>
</tr>
<tr>
<td>Gamma hydroxy butyrate (GHB)</td>
<td>Low</td>
</tr>
<tr>
<td>Steroids</td>
<td>Low</td>
</tr>
<tr>
<td>Stealing</td>
<td>Low</td>
</tr>
<tr>
<td>Violence</td>
<td>Low</td>
</tr>
<tr>
<td>Diving</td>
<td>Low</td>
</tr>
<tr>
<td>Surfing</td>
<td>Low</td>
</tr>
<tr>
<td>Fast driving</td>
<td>Low</td>
</tr>
<tr>
<td>Exercise</td>
<td>Very low</td>
</tr>
<tr>
<td>Sexual behaviours</td>
<td>Very low</td>
</tr>
<tr>
<td>Playing computer games</td>
<td>Very low</td>
</tr>
<tr>
<td>Chocolate</td>
<td>Very low</td>
</tr>
<tr>
<td>Self-harm</td>
<td>Very low</td>
</tr>
<tr>
<td>Caffeine</td>
<td>Very low</td>
</tr>
<tr>
<td>Watching TV</td>
<td>Very low</td>
</tr>
<tr>
<td>Work</td>
<td>Very low</td>
</tr>
<tr>
<td>Shopping</td>
<td>Very low</td>
</tr>
</tbody>
</table>

NB: This is presented in a simplistic version that does not take account of mode of administration of drugs or cultural context. The purpose is simply to make concrete the idea that activities and drugs may differ in addictive potential.
users, and 15% of alcohol users. These percentages are based on those who ever used each drug for a non-medical purpose.

Table 2.3 shows rough estimates of ever users (by self-report) and use in the past month of illicit drugs in the UK in 2000. It also shows the approximate costs of regular monthly use. The percentage of users in the past month gives a possible upper estimate of the propensity of a drug to elicit regular use and possibly addictive use. What is noteworthy is that most of the figures are broadly in line with figures cited above from the US.

The major anomalies are ecstasy and cannabis in which other evidence shows that regular use is only a fraction of the figure for use in the past month, probably reflecting the lifestyle of the users. What is clear is that, even with the most addictive drugs, only a minority of people who try them develop anything that could be called chronic addiction.

We should not make too much of these kinds of quantitative estimates. It is clear that all the important parameters can be influenced by social and cultural factors as well as the purity and mode of administration of the drugs themselves. However, they do tell us what is possible in particular social climates in which it is known that addiction does occur.

Recapitulation

This chapter has attempted to set the scene for the development of a theory of addiction by exploring the changing definition of addiction, issues in the criteria used to diagnose addiction, what makes a good theory, how evidence has been and should be used in the development of theories, and some important observations about addiction that need to be accommodated by any comprehensive theory. Now it is time to begin the journey to a theory that is sufficiently broad to account for the phenomenon.
Chapter 1
INTRODUCTION: JOURNEY TO THE CENTRE OF ADDICTION

This book aims to develop our understanding of addiction by taking a journey to the development of a synthetic theory of addiction, beginning with the simplest possible common-sense approach and exploring how the theory needs to be extended or changed to take account of the available evidence. The goal is to end up with a theory that is parsimonious, coherent, original, stimulating and above all useful.

In the beginning

Many theories but little progress
In the beginning this book was going to be a compendium of addiction theories. Each one would be summarised and matched against various criteria. However, after a while it became apparent that this was going to be an unfulfilling task. It is not that there are no good theories out there; there are. There are many of them and almost all of them are insightful and capture important elements of what we understand as addiction. The problem is that each theory seemed to stem from an innovative idea that accounted for selected aspects of the problem but did not account for other features that existing theories already catered for quite well. There was little sense of progress in our understanding; little sense that we are engaging in ‘incremental science’.

Theory and observation
It was also clear that the relationship between theory and observation was following an approach that was not always appropriate. In behavioural science we have developed our methods from the paradigm of the natural sciences; but unfortunately they have not always served us well. In the prevailing paradigm, the primary source of evidence is the formal study: the survey or the experiment. Observation of behaviour in the natural habitat is usually relegated to the ‘anecdotal’. The problem with this is that the ‘big’ observations about what people do or never do become less important than percentages in surveys, ‘significant differences’ between groups, and ‘correlations’ between variables. In many cases these involve rather trivial behaviours in unrealistic laboratory situations or taking at face value people’s reports of their
attitudes, beliefs and behaviours on questionnaires or in interviews. Very often this gives us an inaccurate portrayal of real behaviour, thoughts and feelings about things that really matter. The responses are too often a pale shadow of, and bear little relationship to, what happens in the world at large.

Need for a synthetic theory

I decided therefore to try to develop a theory of addiction that could provide a conceptual framework within which the genuine insights provided by the existing theories could be placed. It would be a synthetic theory in the sense that it would attempt to pull together the accumulated wisdom. It would not attempt to explain everything there is to explain, but it would explain the ‘big observations’ and provide a conceptual system in which the existing theories could be located. It would be as parsimonious as possible: that is to say, it would only bring in additional elements if they are actually needed. It would be as coherent as possible: the ideas would relate naturally to each other and not be just a list of unconnected assertions.

A guiding principle

In attempting this task I was mindful of the words of Nick Heather (Heather 1998), which I can do no better than to quote verbatim:

‘addiction...is best defined by repeated failures to refrain from drug use despite prior resolutions to do so. This definition is consistent with views of addiction that see decision-making, ambivalence and conflict as central features of the addict’s behaviour and experience. On this basis, a three-level framework of required explanation is (needed) consisting of (1) the level of neuroadaptation, (2) the level of desire for drugs and (3) the level of ‘akrasia’ or failures of resolve...explanatory concepts used at the ‘lower’ levels in this framework can never be held to be sufficient as explanations at higher levels, i.e. the postulation of additional determinants is always required at Levels 2 and 3. In particular, it is a failure to address problems at the highest level in the framework that marks the inadequacy of most existing theories of addiction.’ (p. 3).

I was also very aware of the fact that there already exists in the literature a scholarly and eclectic account in the form of Jim Orford’s model ‘Excessive Appetites’ (Orford 2001). The new theory would seek to build on the work of Orford while paying close attention to the admonitions of Heather.

A psychological orientation

I am a psychologist by training and my field of research is tobacco. Both of these things will inevitably affect my approach and the examples I use. There is no use pretending otherwise. It was apparent to me in researching this book that it is difficult
for theorists to write convincingly in areas that are not their discipline (neuropharmacologists or economists writing about psychology for example), and equally difficult for researchers to demonstrate proficiency in areas of addiction that are not their specialty (the alcohol researcher writing about tobacco or the tobacco researcher writing about cocaine), and many do not even try. But try we must because if we do not we will fail to grasp what addiction is all about.

In the end

I decided that the book would be a journey from the most common-sense and simple explanation of addiction to one that is only as complicated as it needs to be. The narrative would be punctuated with references to many of the theories that have been proposed, together with some comments on these.

These would not be summaries; it would not be possible to do justice to the theories in the space available; some of them take up whole volumes. I would attempt to draw out the theories’ unique insights or important lessons that may be drawn from them. Where possible, the developing theory would use concepts that already exist in the minds of well informed non-specialists and use words that serve non-psychologists well in explaining and predicting each others’ behaviour. I would try to avoid the pitfall of needlessly constructing new terms or making up new meanings for existing ones.

This book uses the device of putting existing theories that it discusses in boxes. In some cases the theories are described very briefly and in others they are considered in much more detail. The level of detail is not related to the complexity or importance of the theory but only what is required to draw out the lessons for the purposes of taking forward the journey to a comprehensive theory of addiction.

The starting definition of addiction

The book starts with the conventional view of addiction as ‘impaired control over a reward-seeking (usually drug-taking) behaviour from which harm ensues’. It is not all-or-none, but a matter of degree. Its severity can be assessed, amongst other things, by the severity of subjective urges or cravings, a frequency or intensity of behaviour that is causing harm and failure of serious attempts to limit or cease the activity.

Addiction and motivation

With this in mind, it seems obvious that a theory of addiction needs to be based on a theory of motivation: it should be apparent that addiction is a disorder of motivation. Although many aspects of motivation are well understood, surprisingly there appears to be no truly synthetic theory that brings it all together. Therefore this book attempts to provide one. Hopefully the theory will have value outside the study of addiction.
Establishing base camp

To achieve the goals set out above is no small matter. To bring on board an expert readership made up of researchers who have their own ideas about how addiction should be construed is even more challenging. Nevertheless, the goal seems worthwhile and if the ideas presented are logical and contain enough new insights, perhaps this theory will provide a ‘base camp’ from which we can start an incremental science of addiction research. We can start replacing the parts that are contradicted by evidence with new, better parts, finding more coherent or simpler accounts that explain all the things that this theory explains, or adding new theories within the structure of this one.

What this book does

Many of the ideas in this book are quite novel and will take some getting used to. To try to help with the process of understanding it, what follows provides some pointers:

- This book develops a draft of a synthetic theory of addiction that draws into a single system the mechanisms underlying: learning through reward and punishment and by associations; feelings of compulsion and desire; the exercise of self-control, beliefs, decisions and plans.

  The theory is based on a synthetic theory of motivation that focuses on the moment-to-moment control of actions through causal pathways of varying lengths and levels of complexity from simple reflexes, through impulses and inhibitory forces, then desires, drives, and emotional states, to evaluations and plans. It emphasises the fact that for any element to influence behaviour, it must do so through impulses and inhibitory forces operating at the time.

- The book argues that the functioning of the brain has evolved to be inherently unstable; the motivational system is built like a ‘fly-by-wire’ aircraft with built-in instability that requires constant balancing input to keep it ‘on the straight and narrow’. This has the advantage of making us highly adaptive and creative but the disadvantage that, without balancing inputs, including devices and techniques to stabilise our mental processes, we readily develop maladaptive thought processes and behaviour patterns.

- The book argues that this pattern of activity can be understood in terms of the concepts of the ‘epigenetic landscape’ proposed by Waddington to explain embryological developments, and chaos theory, a mathematical approach to modelling systems such as weather patterns. In chaos theory, systems can descend into particular states (‘Lorenz attractors’ are examples of these) but still switch apparently unpredictably to other states or even move in a pseudo-random fashion between them.

- The book argues that addiction develops in susceptible individuals from a failure of balancing inputs leading the motivational system concerned into a condition such that particular forces have an unhealthy dominance.
The book tentatively suggests that addiction could be usefully viewed as a symptom rather than a unitary disorder. The idea is that addiction is associated with widely varying underlying pathologies and a number of different syndromes (such as the alcohol dependence syndrome). These pathologies involve disorders with varying combinations of abnormally strong impulses, abnormal drives, abnormal emotional states or abnormal mechanisms for restraint.

Sometimes the pathology is present in the individual quite independently of the addictive behaviour. Sometimes the pathology arises from a susceptibility of the individual to the effects of the addictive behaviour or drug. And sometimes it is the individual’s environment that is pathological and most ‘normal’ individuals would succumb in such situations.

Often the pathology shows itself as a syndrome that goes beyond addiction per se but involves other classical symptoms (as in the alcohol dependence syndrome already mentioned). But across the different types of addictive behaviour, the pathologies often interact with environmental conditions to result in widely varying manifestations of the symptoms from frequent, low intensity adoption of the addictive behaviour through to relatively infrequent bingeing. The same drug can lead to different patterns of addictive behaviour in different social and environmental conditions.

The book proposes a change to the assessment of addiction. It argues that the assessment should focus on gathering evidence for the degree of dominance of particular motivational forces. For clinicians, the next step after assessment of addiction would be try to determine where the pathology or pathologies lie and what are the prospects in the short and medium term for treating these. This would inform the decision about how much emphasis to place on treating the underlying pathology or simply suppressing the symptom (the addictive behaviour).

Further, if one is to treat the underlying pathology, what are the prospects for an acute treatment episode that will result in a lasting effect, will chronic treatment be required, or is the best model one of treatment episodes that are repeated as required? Symptomatic treatment involves harnessing additional motives to bolster restraint and minimise the manifestation of the impulses to engage in the behaviour. Treatment of the underlying pathologies involves pharmacological and psychological interventions to treat, or permanently normalise, the disorders of the motivational system.

The book proposes an approach to the development of population level interventions to prevent or control addictive behaviour that takes account of the whole of the motivational system (impulses, desires, evaluations and plans). It states that, equally importantly, interventions should be based on a calculus of the forces operating on individuals at times when the activity is currently occurring or being planned.

This book argues that our existing approach to theory development and testing is not conducive to ‘incremental science’ but rather a plethora of theories that have much in common but use slightly different formulations or that focus on just one aspect of the matter in hand and fail to address other important
aspects. Moreover the methods we use to test theories, such as correlation coefficients and regression, are most often not up to the job. We should be looking for counter-examples: a single genuine counter-example means that the theory must be wrong and prompts the search for improvements.

• This volume can do no more than establish a starting point for what it is hoped in future will be an incremental science of addiction, with new theoretical ideas being proposed that do a better job at explaining and predicting behaviour within a common integrative framework rather than just drawing attention to new insights that explain some things better but fail to address other observations that were adequately explained by previous theories.

The synthetic theory of addiction in brief

While reading the preceding paragraphs you will have been placing a particular interpretation on the words used and attempting to fit the ideas into your understanding of the area. You will also have been trying to think of ways in which the ideas are wrong or do not say anything particularly new. I do not expect to convince the discerning reader of the truth or worth of what the book is arguing without a great deal of explanation and discussion.

To start the ball rolling the following paragraphs will outline some of the claims made by the theory. This will involve some repetition. This is deliberate: ideas often take several exposures in different contexts to be understood – this is just the beginning.

Addiction is . . .

Addiction can be usefully viewed as a symptom rather than as a unitary disorder. (The distinction is semantic but has some value). It can arise from many different pathologies, and varies in severity and in its manifestations. Addiction involves a chronic condition of the motivational system (see below) in which there is an abnormally and damagingly high priority given to a particular activity.

The pathologies underlying addiction come in three basic types:

• Abnormalities in the motivational system that were not directly caused by the addictive activity (e.g. related to chronic anxiety, depression, low self-esteem, poor impulse control, etc.).
• Abnormalities in the motivational system caused by the addictive activity acting on susceptibilities in that system (e.g. sensitisation to the effects of stimulant drugs, tolerance and withdrawal symptoms, and mood disturbance arising from social effects of the behaviour).
• Pathological environments acting on essentially normal motivational systems that are not equipped to cope with them (e.g. sometimes the lifestyle of public icons, particular social relationships, people in chronically distressing circumstances).
The ‘motivational system’

The motivational system is the system of forces that energise and direct our actions. It operates at five levels of complexity: (1) simple responses, (2) impulses and inhibitory forces (experienced as urges and restraint when they come to conscious awareness), (3) motives (experienced as desires/wants when we are conscious of them), (4) evaluations (conscious beliefs about what is good or bad, right or wrong, useful or detrimental), and (5) plans (conscious mental representations of actions or collections of actions together with starting conditions and a more or less strong feeling of commitment).

Motives can influence behaviour only through impulses and inhibitions, evaluations can do so only through motives, and plans operate on motives and evaluations.

Impulses, motives and plans

Leaving aside simple reflex responses which are not of great interest here, actions result only from impulses and inhibitory forces operating at that moment in time, and these result from stimuli/information and from motives operating at that time. Thus motives operate through impulses and inhibitions, and evaluations operate through motives. Plans influence actions through impulses/inhibitions, motives or evaluations.

The way that the motivational system is structured imparts an inherent (though not paramount) primacy to the immediate environment in terms of influences on our actions and a primacy of desires and urges over evaluations and plans.

Motives and impulses derive from drives (e.g. hunger), generalised emotional states (e.g. happiness, sadness, excitement) and targeted emotional states (e.g. liking, disliking). The strength of a given motive derives from the strength of associated emotions and drives. The direction of the motive derives from the nature of the drives and whether the emotions are positive or negative.

Learning by association

Occurrence and repetition of particular associations within the motivational system lead to facilitative links being formed so that when one element occurs the other elements are triggered more readily. This is associative learning.

Stimulus–stimulus associations underlie ‘Pavlovian (classical) conditioning’ while stimulus–response–outcome (reward or punishment) associations underpin what is called ‘instrumental (operant) learning’. We must appreciate, however, that associative learning is a very general property of the brain which underpins creative thoughts and propositional learning and habits and skills. Thus, what we have come to think of as classical and operant learning are two examples of a wide range of possible types of association between mental activities.

The term habit is more generic and includes any activity that involves a significant element of automaticity through associative learning.
The unstable mind

The pathologies underlying addiction develop because the human mind and the physiological systems that give rise to it have evolved to be inherently unstable and require constant balancing input to prevent them heading off in unwanted directions. This instability is what makes humans highly responsive to environmental events, creative and adaptable but at the cost of a tendency to descend into maladaptive patterns of thought and behaviour in the absence of balancing input.

A useful way of visualising this is Waddington’s ‘epigenetic landscape’. This characterises the state of an organism as a ball rolling down a contoured landscape with valleys and plains. At bifurcations in the valleys small environmental forces can lead the ball down one path or another. Addiction represents a particularly deep valley which would require very powerful sustained input to escape from.

In principle this approach can be modelled using mathematical concepts of ‘chaos theory’. ‘Chaotic systems’ (such as weather) exhibit characteristic patterns: they involve periods of short- to medium-term stability punctuated by apparently unpredictable switches in state or periods of violent instability resulting from apparently small events; the paths that two systems follow can diverge markedly as a result of very small differences in their starting points; on occasions the system can become fixed in a particular state without the possibility of escape under any realistic conditions, while on other occasions it can apparently show this pattern only to suddenly switch state.

The motivational system seems to fit this model, and addiction represents a particular kind of activity of the system. For most people under most conditions the motivational system has checks and balances that prevent any one set of motivational forces dominating for a protracted period. However, some circumstances (a pre-existing pathology in the system, changes to a susceptible system stemming from a given set of actions, or a particular set of environmental inputs to the system) lead it to enter a state in which the inputs are not sufficiently balanced for that particular system and it is ‘attracted’ to an addicted state where a given set of motivational forces are inadequately balanced by competing influences.

This book is an essay. I have tried to communicate the ideas as best I can, but each one could be a book in itself and this text is not attempting to include that level of detail. Instead I aim to present a wider view of addiction theory. I hope I have done justice to the subject and, even if you do not agree with everything in the book, I still hope you find it stimulating.
Chapter 5

ADDICTION, HABIT AND INSTRUMENTAL LEARNING

This chapter takes the development of the theory one step further by incorporating the idea of a mechanism linking stimuli to responses that does not involve conscious choice. Research with other animals has shown that they will learn to perform simple actions repeatedly if these are followed by rewards such as the availability of food. Animals will also make repeated responses for drugs that are addictive for humans. This raises the possibility that part of the motivation to take addictive drugs involves a learning mechanism that predates in evolutionary terms the development of conscious decision-making. Thus addiction involves the development of a habitual behaviour pattern that is independent of any conscious evaluation that might be taking place about the costs and benefits of the behaviour. The impulses to engage in addictive behaviour that are generated by this mechanism can be so strong that they overwhelm the desire of the addicts to restrain themselves.

It was noted at the end of the previous chapter that some of the theories of addiction that work within a framework of choice theory have also found it necessary to postulate automatic, non-conscious processing to account for some of the phenomena of addiction. This chapter develops this idea using concepts of habit, instrumental (operant) learning and classical (Pavlovian) conditioning. These are ideas that are everyday currency in the world of behavioural pharmacology. There is in fact something of a gulf between this world and that of proponents of choice models of behaviour. Some kinds of conceptual link are made but only at a very superficial level.

Instrumental learning

In the world of the behavioural pharmacologist, addiction to drugs arises from the operation of reward and punishment. There are many different variants of this approach and this chapter will examine some of these in quite general terms. We begin with a simple account of instrumental learning in which no particular pathology is involved; impaired conscious control arises simply because this reinforcement mechanism operates at a level that is outside conscious control and so sets up motivational forces that come into conflict with consciously held preferences (Box 5.1).
Box 5.1 Instrumental learning (operant conditioning) and addiction

Dependence on drugs and other activities arises from a normal instrumental learning mechanism that operates outside conscious awareness. Drugs such as heroin and nicotine reward behaviour that leads to them. Neuroadaptation in some cases also means that abstinence is aversive and so drug taking is strengthened by escape and avoidance learning mechanisms.

Addictive behaviours become entrenched and difficult to stop through a process that can occur without the individual being aware of what is happening, and it does not involve an active decision-making process. It does not even require the individual to feel positive pleasure from the behaviour. The process, reinforcement, involves a part of the brain that evolved many millions of years ago because it 'trained' animals to engage in behaviours that help with survival and reproduction. There are two facets to this process, positive reinforcement and negative reinforcement.

Positive reinforcement
This is the process by which a rat learns to press a lever to obtain food or a dog is trained to sit up and beg for treats. Drugs of dependence tap into the motivational system underlying this and in effect train the user to sit up and beg for the drug (O’Brien et al. 1992). The drug acts as a reward or positive reinforcer. With repetition, the cue–response–reward association becomes stronger and stronger.

Negative reinforcement
This is the second element of the instrumental learning process. Whereas positive reinforcement involves seeking out rewarding stimuli, negative reinforcement involves escaping from or avoiding unpleasant stimuli – punishment (e.g. Lewis 1990; Schulteis and Koob 1996). This kind of learning can be very powerful. Just as with positive reinforcement, many millions of years ago animals evolved a motivational system that enabled them to learn to escape from or avoid noxious or painful stimuli. It requires no conscious decision-making: it is automatic. A rat can readily learn to avoid treading on a part of the cage floor which delivers a small electric shock. A child quickly learns not to touch a hot surface. Equally, one can train a rat to press a lever to prevent an electric shock from occurring or to turn off an electric shock.

Addictive drugs tap into this system because after a relatively short period of use the body adapts physiologically to the presence of the drug. From that point onwards, periods of abstinence lead to the body compensating for the drug when it is not actually present: the physiological systems become unbalanced. Even a relatively brief interval without the drug can lead to a characteristic and unpleasant withdrawal syndrome (see the discussion in Chapter 3). Taking the drug turns these symptoms off. Like the rat pressing
a lever to escape the shock, the drug user learns to take the drug to escape the withdrawal symptoms. Again, it is important to emphasise that according to the instrumental learning model this is not a conscious process and no decision-making is involved.

Occasional reinforcement
The instrumental learning account of habit training extends further than noting that animals will respond to obtain rewards or avoid punishment. Many of the symptoms of withdrawal mirror feelings all of us feel from time to time anyway, such as depressed mood. The drug user is not very well placed to distinguish between those feelings caused by withdrawal and those that arise from other causes. This means that sometimes taking the drug will relieve them and sometimes it will not.

One might imagine that this would weaken the association between taking the drug and those symptoms but, paradoxically, it strengthens it. Odd as this may seem, a behaviour that is only reinforced on some occasions becomes more deeply entrenched, and more resistant to change, than one that is reinforced on every occasion. Lever-pressing is more firmly established in a rat if it produces food only once every 5 or 10 presses on average than if it produces food every time.

It has been noticed that when an animal has to press a lever, say, 20 times to get a morsel of food, as the end of the sequence of responses approaches, the response rate increases. That is, the responding speeds up and becomes more energised as the reward approaches. This has parallels in a tendency for people to place increasing priority on action sequences as they near their completion. It raises the possibility that the force of addiction arises in part from the same mechanism.

The evolutionary advantage of the strength of learning from occasional reinforcement is quite clear. The world is an unpredictable place, and animals need to be able to learn adaptive behaviours that on average work to their advantage, rather than just those that work every time. The animal cannot know each time whether the behaviour will have the required effect and so has to be driven to persist with it as long as it works often enough to make it worthwhile. With drug use, the end result is that taking the drug need only reduce depressed mood, say, every now and then for the pattern of behaviour to become established and maintained. The same is true for positive reinforcement: even if only a small proportion of cigarettes are rewarding, the reinforcing power of the cigarette may be very strong.

Avoidance
The instrumental learning model contains another potentially important feature. Animals, including humans, will learn to avoid as well as escape from discomfort. This means that the response can be maintained at a high rate even when no discomfort is experienced (because the avoidance is successful). Drug users do not need to experience withdrawal symptoms to keep them
using the drug – the mere threat of these symptoms is enough to tap into the negative reinforcement mechanism. Again, according to the model, this does not involve a deliberate, conscious process, but one that typically operates outside of awareness. The behaviours driven by this process are performed urgently and compulsively.

**Cues**

The association between behaviour and reward becomes attached to particular cues (called ‘discriminative stimuli’). This means that the behaviour tends to occur primarily in the presence of those cues or ones somewhat like them. Thus a habit that is very strong in one situation may not be strong in another. Cues can be anything from the environmental context (being at home or at a party versus being at work) to a time of day (evening versus morning). According to this view, craving is the subjective manifestation of the learned habit at a particular time and in a particular context. Thus, when a habit has been learned in the context of a particular set of cues, those cues may come to increase craving and unless that craving is opposed by a conflicting motivational pressure, it causes the behaviour to occur.

**Secondary reinforcement**

The mechanisms described above are supplemented by a very important phenomenon which strengthens the power of reinforcers to control behaviour. It was demonstrated early in the 20th century that a neutral stimulus (such as a coloured light) could come to seem rewarding if it preceded a genuine reward (such as food). This process is known as ‘classical conditioning’ or ‘Pavlovian conditioning’ (after Pavlov, who demonstrated that salivation could be triggered in dogs as soon as a bell was sounded, even before food was presented, if the bell had previously been associated with food). It is the reason why certain smells, sounds and sights evoke pleasant feelings in some people but not in others.

These stimuli are called ‘secondary reinforcers’ because they derive their influence on behaviour only by association. However, there is evidence that they can increase the rate of responding for addictive drugs and may play an important role in dependence (Glautier and Drummond 1994).

Smoking provides a good example of this (Miyata and Yanagita 2001). For a smoker, one sensation that immediately precedes the nicotine hit is the ‘scratch’ of the smoke in the back of the throat. There is obviously nothing intrinsically pleasant about this, but smokers come to like it. Smokers do not know why this is the case, but it arises because this sensation has been so closely tied to the rewarding effect of nicotine. Besides this, the sight of the cigarette packet, the feel of the foil, the smell of the tobacco and so on all come to arouse a pleasant expectation of reward and form part of the addiction process. Similar effects are observed with drugs such as heroin and alcohol where stimuli associated with the pharmacological effects themselves become rewarding.
The power of the learning process
The strength of the learning process is influenced by a number of factors: the nature of the reinforcer itself, the schedule of reinforcement and also how long the schedule has been in operation. When one considers that a packet-a-day smoker repeats the processes of reinforcement 240 times a day (12 puffs for each of 20 cigarettes), 87,600 times a year, and 2,190,000 times over a 25-year packet-a-day smoking career, it is not difficult to see how this might entrench the behaviour very deeply. It may be that it is this repetition that is driving the strong addiction in this case.

Overall
According to a simple instrumental learning model, drug use or another addictive activity becomes a deeply entrenched behavioural pattern ultimately under the control of the rewarding or punishing stimulus but also intricately tied into behavioural and social forces, and under impaired voluntary control.

Issues and evaluation
Instrumental learning offers a very powerful and attractive explanation for many aspects of drug addiction. Of particular importance is the fact that the process does not require conscious choice or awareness. This can help to explain how a conflict might occur between conscious desire to exercise restraint and motivational forces impelling the behaviour.

Mechanisms underpinning instrumental learning
We know something of the neural circuitry underpinning positive reinforcement (see, for example, Nestler 2004). It is believed that whatever the drug or activity, ultimately the final common pathway through which reinforcement operates is the medial forebrain bundle and an important part of that is the mesolimbic pathway (Box 5.2).

Box 5.2 The Dopamine Theory of drug reward
There are many variations on the Dopamine Theory of Positive Reinforcement but they all propose that the action of dopamine on receptors in the nucleus accumbens plays a critical role.

The mesolimbic dopamine pathway
This is a theory about the mechanism by which addictive drugs exert their rewarding effects. There are numerous versions of the theory and our understanding of the circuitry is developing rapidly, but at its simplest it states that drugs with addictive potential increase the concentration of the neurotransmitter...
dopamine in a part of the brain known as the nucleus accumbens (NAcc). They further state that this increase in dopamine is necessary for addiction to occur. The nucleus accumbens lies towards the front of the brain and receives major input from a part of the midbrain called the ventral tegmental area (VTA) (Figure 5.1).

Drugs of dependence influence dopamine concentrations in the NAcc in a number of different ways (Figure 5.2). Opioids, nicotine and alcohol block the inhibitory control of GABA on the VTA leading to an increase in firing of neurones leading to the NAcc. Cocaine and amphetamines act directly

**Figure 5.1** Location of the mesolimbic dopamine pathway (see Tomkins and Sellers 2001).

Dopamine binding to receptors in the nucleus accumbens is central to the rewarding effect of stimuli

Cocaine and amphetamines block re-uptake of dopamine released by nerve terminals in the nucleus accumbens leading to more dopamine in the extracellular space; amphetamines also enhance release of dopamine

Opioids, nicotine and alcohol increase firing of nerves whose cell bodies are located in the ventral tegmental area; either through action on those cell bodies or by blocking inhibition by GABA interneurons

**Figure 5.2** Drug actions of the mesolimbic dopamine pathway (see Tomkins and Sellers 2001).
on the NAcc, blocking the natural process of re-uptake of dopamine that is released because of firing of the cells so that there is more dopamine in the extracellular space.

Neuroleptic drugs which block dopamine receptors disrupt self-administration of psychomotor stimulants, whereas drugs blocking the noradrenergic receptors are ineffective. Also lesions in the dopaminergic terminal field in the NA increase psychostimulant self-administration. The actions of opiates in the cell body region (enhancing dopamine cell firing) and the actions of psychomotor stimulants in the terminal region (enhancing dopaminergic synaptic activity) increase dopaminergic neurotransmission. It is also thought that this pathway is involved in secondary reinforcement and the effects of cues on drug-seeking behaviour.

**Issues and evaluation**

The above description paints a picture that is relatively clear cut but in fact there is still a great deal of uncertainty about these mechanisms, not least because it is not clear how far humans share the same kinds of response as rats and mice, bearing in mind that rats and mice are different from each other in some important respects.

The neural basis for negative reinforcement is less well understood though some have argued that it may involve the same neural substrate as positive reinforcement (Koob and Nestler 1997). There is some evidence that drugs of dependence such as nicotine and cocaine cause changes in the functioning of the dopamine reward system that may underpin anhedonia during abstinence, but how this then goes on to motivate behaviour is not clear.

Weiss and Koob (2001) talk of ‘functional neurotoxicity’ as the basis for drug dependence (Box 5.3) but this does not assume pathological changes to the motivational process, only that drugs act abnormally on it and create additional motivation through neuroadaptation.

---

**Box 5.3 Addiction arising from functional neurotoxicity of drugs**

*Chronic use of addictive drugs alters the functioning of brain reward circuitry and changes to other brain systems that lead to withdrawal symptoms and acquired drives.*

**Mechanisms underpinning withdrawal symptoms**

This approach broadens simple dopamine theory referred to above, in proposing that the acute reinforcing effects of addictive drugs involve the part of the basal forebrain called the extended amygdala which includes the nucleus accumbens and amygdala and neurotransmitters such as dopamine, opioid peptides, serotonin, GABA and glutamate (Weiss and Koob 2001).
Withdrawal from addictive drugs is proposed to cause unpleasant mood changes and dysregulation of brain reward systems involving some of the same neurochemical systems implicated in the acute reinforcing effects of drugs of abuse.

The ‘functional toxicity’ of the acute withdrawal state is accompanied by recruitment of the stress-related neurotransmitter system involving corticotrophin-releasing factor. During more prolonged abstinence, ‘post-acute withdrawal’, there may be continued dysregulation of the neural systems associated with drug reinforcement and stress. This may produce less pronounced but persistent functional neurotoxic effects and could be responsible for persistent vulnerability to relapse.

**Addictive drugs differ from natural reinforcers**

In a variant of this approach it is proposed that drug rewards have larger and more prolonged rewarding or psychostimulant effects than naturally occurring stimuli (Wise and Bozarth 1987). A recent study found that presentation of an aversive conditioned stimulus suppressed drug seeking in rats with limited cocaine self-administration, but no longer did so after an extended cocaine-taking history. In contrast, after equivalent extended sucrose experience, sucrose seeking was still suppressed by an aversive conditioned stimulus. The effect of cocaine was not due to impaired fear conditioning, nor to an increase in the incentive value of cocaine (Vanderschuren and Everitt 2004).

**Acquired drives**

It has been suggested that the development of a compulsive pattern of behaviour may stem from changes to drive mechanisms (Kostowski 2002). It may arise from a disturbed balance of the mechanism underlying drive-related behaviours, which controls appetitive reactions aimed at seeking out an addictive substance. It is proposed that drug addiction may involve a change in the mechanism of satisfaction of drives and states of satiation. It argues further that, to understand how the motivational processes are changed with the development of addiction, it is necessary to consider the mechanism of drive satisfaction and satiation states that occur in relation to what it calls the ‘consummatory reflex’.

When a given drive is satisfied, a state of ‘fulfilment’ results. This may stem from a so-called ‘antidrive’ mechanism. While a drive activity is characterised by general activation and tension, the drive satisfaction state is characterised by relaxation and relief. When a particular drive is satisfied, the other drives can then come into play. Hence the theory postulates that dysfunction of drive satisfaction leads to sustained activation related to the current drug-related drive, which blocks the operation of other drives. In effect, uncontrolled compulsive appetitive behaviour is released, and the operation of other drives is restrained, thus forcing the addict to focus on drug-related drive.
**Issues and evaluation**

This emphasis on drive states offers a very attractive and plausible account of at least part of the addictive process. Addictive activities, and particularly drugs, create acquired drives which are unlike other drives in that they are resistant to satisfaction and therefore maintain a priority over other drive states. In this regard it is noteworthy that individuals addicted to drugs as diverse as alcohol, heroin and nicotine show evidence of a reduction in drives relating to eating.

**Classical conditioning**

Several theorists have focused more closely on Pavlovian or classical conditioning and addiction (Box 5.4). These examine in more detail the role of cues in generation of impulses to engage in the behaviour. The overlap between the accounts makes it difficult to pick out any one of them as a theory.

**Box 5.4 Classical conditioning and addiction**

*Stimulus–stimulus associations play an important role in the development of withdrawal symptoms and urges to take addictive drugs.*

**Classical conditioning and craving**

Under a Classical Conditioning Model, drug craving arises because of a repeated pairing of environmental stimuli with drug effects (Drummond et al. 1990). For example, falling blood alcohol level (an unconditioned stimulus) induces a withdrawal syndrome including craving (unconditioned responses). After a period of abstinence it is possible for the stimuli associated with falling blood alcohol levels (conditioned stimuli) to elicit a conditioned withdrawal response which resembles alcohol withdrawal.

This account has been developed. Drummond (2001) has proposed that ‘cue-elicited craving’ (as a response to the environmental stimuli) is different from ‘withdrawal-related craving’ (craving as part of the unconditioned withdrawal syndrome). He also proposes that cue-elicited craving may be more predictive of a relapse than withdrawal-related craving as relapse can only occur in situations of drug availability which is when cue-elicited craving occurs. Classical conditioning has also been proposed as the basis for urges and relapse long after the acute withdrawal phase has passed (Childress et al. 1988; Azorlosa 1994).

**Classical conditioning and primary withdrawal symptoms**

It has been argued that classical conditioning could underpin occurrence of the primary withdrawal syndrome itself and particularly craving (Melchior
and Tabakoff 1984). The unconditioned stimulus is the drug effect, and in classical conditioning terms is thought of as a pharmacological challenge to internal regulation, which in some cases is compensated for by a response (unconditioned response, UCR) which reflects the animal’s defence against the drug-induced internal dysregulation.

A stimulus that precedes drug dose may then precipitate a defence reaction, termed a ‘compensatory CR’, when the drug effect is expected (see Siegel and Ramos 2002), which serves the purpose of maintaining this system of internal regulation as a response to the pharmacological challenge. The escalation of casual use to a level of use characteristic of addiction is explained by a strengthening of the learned associative link between the CS and UCS which increases the intensity of the CR. CRs that occur in the absence of a drug UCS will put the individual into a state of disequilibrium and induce withdrawal symptoms.

**Classical conditioning and tachyphylaxis**

The initial sensations associated with onset of drug actions could be interoceptive cues that predict the later full effect and so become conditioned stimuli themselves (McDonald and Siegel 2004). It is possible that these drug onset cues (DOCs) are important in development of addiction as they come to elicit the response at least as reliably as any external drug-associated cues.

**Cue exposure as a treatment**

A psychological treatment approach has been proposed based on the idea that cues associated with drug taking precipitate cravings and relapse. The treatment, cue exposure, presents the cues without the opportunity to engage in the drug-taking behaviour. It is thought that this may lead to extinction of the classically conditioned association of the cues and drug effect thereby reducing the craving (Drummond et al. 1990; Childress et al. 1993; Sayette and Hufford 1994).

At present, evidence that this treatment approach is effective is weak. However, it needs to be recognised that even if the underlying theory were correct, there is no guarantee that there is a practicable procedure that would be powerful enough to have a major impact on cue-elicited craving and relapse. One needs to bear in mind that the conditioning process will have involved many years of pairing, and the opportunity to extinguish the association will involve a relatively few sessions at best.

**Issues and evaluation**

There can be no doubt that classical conditioning plays a critical role in the experience of drug addiction and the dependence syndromes surrounding it. It seems likely that it plays a significant role in the motivation to continue the addictive activity but it is not yet clear whether the same mechanism can be used to help addicts to recover.
More complex learning models

Variants on a straightforward instrumental learning model have been proposed that may better explain the details of drug-seeking behaviour, at least in animals. One of these (Box 5.5) emphasises memory processes.

Box 5.5  Addiction as a learning/memory process

The development of more frequent drug-seeking behaviour involves multiple parallel learning and memory systems, not just a simple instrumental learning process (White 1996).

Three independent learning systems
According to this theory, all changes in behaviour, including the development of drug addiction, involve storage of new ‘information’ in the nervous system. This involves at least three more or less independent learning systems. Reinforcers operate on these systems in three ways: they activate the neural mechanisms involved in approach or avoidance responses; they produce states that are rewarding or aversive; and they alter or strengthen the representation of the information stored in these systems. Each addictive drug maintains self-administration by tapping into these mechanisms in different ways. Each involves different brain structures.

Three actions of reinforcers
Figure 5.3 shows the theory in schematic form. The three actions of reinforcers are listed in the horizontal band at the top labelled ‘Reinforcer actions’ (in the column on the left). Each addictive drug is capable of mimicking some or all of these functions of natural reinforcers. These reinforcing actions influence behaviour by acting on learning and memory systems.

The systems are shown in the band labelled ‘Memory systems’. Each system is named for a brain structure that is central to it. Each acquires a different type of behaviour or information: a one-word description of the kind of learning mediated in each system is below the name of its central structure. The kinds of behaviours these produce are described in the band labelled ‘Acquired behaviour or information’. The role of each of these behaviours in the addictive process is described in the band labelled ‘Implications for addiction’. White argues that most addictive drugs initiate more than one of these processes.

Issues and evaluation
This theory is an ambitious attempt to integrate classical and instrumental learning concepts and it also brings in conscious and non-conscious representational systems as would be necessary to understand human behaviour.
Research with other species is increasingly showing that instrumental and other forms of associative learning operate in complex ways and natural and drug reinforcers have effects that are only beginning to be understood. One aspect of this that has attracted a great deal of attention is the observation that some effects of addictive drugs have been shown to increase with exposure to the drug rather than decrease. This has formed the basis for the Incentive Sensitisation Theory of drug dependence (Box 5.6).

**Box 5.6 Incentive Sensitisation Theory**

Compulsive drug use results from the effects of drugs on the mechanism that establishes particular stimuli (cues) as triggers for appetitive behaviours. While habituation occurs to the hedonic value of drugs, sensitisation occurs to the effect of the drugs in establishing the salience of these cues. This creates a dissociation between how much pleasure a drug provides and the degree to which it is sought after.

The Incentive Sensitisation Theory (IST) of addiction focuses on how drug cues trigger excessive incentive motivation for drugs, leading to compulsive drug seeking, drug taking and relapse (Robinson and Berridge 1993, 2003).
It is based on the observation that ingestion of at least some addictive drugs actually increases the effect of those drugs on certain behaviours in animals (such as locomotor activity and drug self-administration).

**Sensitisation of incentive salience**

This theory proposes that while drug pleasure becomes less important, during the transition to addiction the incentive motivation to use the drug increases. This arises from the long-lasting consequences of drug-induced alterations in NAcc-related circuitry that mediate ‘incentive salience’ (Figure 5.4).

Incentive salience is a characteristic of the mental representation of a stimulus that can be characterised in terms of ‘wanting’ (a mental state that triggers behaviours that seek out a rewarding stimulus). The ‘wanting’ system can be activated implicitly and so can influence behaviour without a person necessarily having conscious emotion, desire or a goal.

For example, it has been reported that the brief subliminal presentation of faces expressing positive emotions (backward masked and so brief they do not cause any conscious feeling of emotion at the time they are presented) can activate implicit ‘wanting’ and so lead to an increase in subsequent consumption of a beverage (Winkielman et al. 2005). In addicts, doses of drugs that are too low to produce any conscious experience of pleasure can activate implicit ‘wanting’ as indicated by an increase in drug-seeking behavior (e.g. Lamb et al. 1991).
Incentive salience and ‘wanting’
Ingestion of addictive drugs causes the circuitry to become sensitised so that further ingestion produces a greater effect. Thus drug-associated cues lead to pathological ‘wanting’ because excessive incentive salience is attributed chiefly to them. The sensitised neural systems responsible for excessive incentive salience can be dissociated from neural systems that mediate the hedonic effects of drugs, that is, how much they are ‘liked’. In other words, ‘wanting’ is not ‘liking’. Hedonic ‘liking’ is a different psychological process that has its own neural substrates (e.g. NAcc opioid neurotransmission).

The role of Pavlovian conditioning
It is proposed that individuals are guided to incentive stimuli by the influence of Pavlovian stimulus–stimulus (S–S) associations, and this is psychologically separable from the symbolic cognitive systems that mediate conscious desire, declarative expectancies of reward, and behaviour-outcome representations (see Robinson and Berridge 2003).

Brain regions involved
In fact, it is argued that different brain systems mediate cognitive versus incentive salience forms of motivation. Prefrontal and other cortical areas primarily mediate cognitive forms of desire and act-outcome representations, whereas NAcc-related circuitry (especially dopamine-related systems) play a more important role in Pavlovian-guided attributions of incentive salience (see Robinson and Berridge 2003).

Issues and evaluation
This theory allows for the possibility that excessive wanting may be compounded in at least some addicts by drug-induced dysfunction in prefrontal cortical systems normally involved in decision-making, judgement, emotional regulation and inhibitory control over behaviour. Cognitive deficits in the ability to inhibit or properly assess the future consequences of one’s actions due to prefrontal dysfunction, combined with excessive incentive salience due to sensitisation of NAcc-related circuitry, lead to the compulsive pursuit of drugs out of proportion to the pleasure drugs provide and in the face of negative consequences.

The dissociation between wanting and liking is a powerful non-obvious statement that has a great deal of appeal. Moreover the explanation in terms of sensitisation of one part of the motivational system versus habituation of another part is an important theoretical statement.

From the point of view of addiction in humans, the dissociation between wanting and liking in the Incentive Sensitisation Theory represents an important step towards the goal of understanding why the link between addictiveness of a particular drug and the degree to which it gives enjoyment is weak. This idea has been expanded on in more detail by Balfour (Box 5.7).
Box 5.7 Balfour’s theory of differential drug effects within the nucleus accumbens

There are two major subdivisions of the nucleus accumbens which are presumed to be central to the rewarding effects of addictive drugs: the shell and the core. These mediate different parts of the instrumental learning process. Different effects of drugs on these different subdivisions may explain why different drugs show different patterns of addictive behaviour and in the case of nicotine why compulsive use can develop in the absence of powerful euphoriant effects.

A variant of the dopamine theory of reward has been proposed by Balfour (2004). It focuses on nicotine addiction but makes reference to addiction to other drugs. The theory attempts to relate the effects of nicotine on concentrations of dopamine in the extracellular space in the nucleus accumbens to its effects on the drug-seeking behaviour of animals. In doing so it offers an explanation for the fact that nicotine replacement therapies such as nicotine patches have only a small effect in improving the success rates of smokers wanting to quit.

Nucleus accumbens core and shell as separate systems

The theory notes that the nucleus accumbens consists of two major subdivisions: the shell and the core. It hypothesises that stimulation of the dopamine projections to the medial shell and the core of the nucleus accumbens play complementary roles in the development of addiction. Increased extra-synaptic dopamine in the medial shell confers hedonic properties on behaviours such as smoking which deliver nicotine, and this increases the probability that the response will be repeated as in the conventional model of operant learning.

However, nicotine also causes an increase in dopamine concentrations in the core and this confers ‘incentive salience’ to cues associated with delivery of the drug and leads to the development of stimulus–response (‘Pavlovian’) type behaviour in the presence of those cues. This effect of nicotine is enhanced by pre-treatment with nicotine. As Robinson and Berridge (2003) have proposed for psychostimulant drugs, there is sensitisation of this effect on dopamine levels in the accumbens core and this underpins the progression from normal to addictive use.

What is special about nicotine

Balfour argues that nicotine is different from other addictive drugs in a number of respects that account for the different pattern of behaviour observed. One consequence of nicotine’s actions is that the behaviour itself and the stimuli and cues associated with it are incentives in their own right. This would explain the observation that sensory characteristics of smoking such as the smell and feeling in the throat of the smoke being inhaled are sufficient to control behaviour in the short term (e.g. Pritchard et al. 1996).
Issues and evaluation

This is an elegant and insightful version of the Dopamine Theory of Drug Reward that, when applied to human behaviour, offers explanations of puzzling phenomena that go well beyond what simple common sense can provide. It suggests lines of preclinical and clinical research that could prove fruitful.

We have presented examples of the many formulations of the theories that attempt to explain addiction in terms of instrumental learning, supplemented by classical conditioning. These models have been developed to explain the behaviour of rats and other species in laboratory experiments but they have provided a wealth of ideas that may help in understanding human addiction.

Of particular value are the ideas that help explain why degree of addiction and degree of pleasure may be dissociated, why urges to engage in addictive activities are often affected by environmental triggers and the non-conscious processes that can lead to the behaviours in question.

The theories contain many speculative elements, especially when attempting to translate results from animal research to humans. From the point of view of developing a theory of human addiction, it is not clear how much further one needs to go than to acknowledge the existence of instrumental learning and classical conditioning mechanisms that operate outside of conscious awareness but influence behaviours and feeling states that can drive conscious behaviour.

Social learning

There is another aspect of learning that has been identified that needs to be included before any learning theory account can be considered complete. This is the propensity of humans to learn by both direct and vicarious experience. The details of this have been proposed in what has been called Social Learning Theory (Box 5.8).

Box 5.8 Social Learning Theory

Social Learning Theory extends the concept of instrumental learning as a basis for addiction to learning through observation and communication.

Social Learning Theory (SLT) (Bandura et al. 1977) describes the effect of cognitive processes on goal-directed behaviour in humans. It considers the human capacity for learning within a social environment through observation or listening to others.

Starting with simple instrumental learning

It proposes that excessive human drug use is determined by consequences of the actual drug taking. The learning element of SLT is simple operant
learning whereby an individual will repeat any behaviour that leads to a reward. Applied to drug use, a ‘positive event’ could be defined as the subjective euphoric effect that immediately follows a dose of a drug. However, some drug-taking experiences lead to adverse effects (e.g. nausea or disturbing flashbacks). The experience of a negative event following drug use is postulated to promote avoidance of drug taking in the future.

According to SLT, the more frequent or intense the drug-taking experience, the more habitual it becomes. Similarly, the more frequent or intense the negative drug-associated experience becomes, the greater the likelihood it will be avoided. It is suggested that the individual becomes motivated to take the drug more often in order to achieve the drug effect. Such an affect may even increase to the point at which it interferes with other activities needed to sustain normal life.

**Punishment and motivation to exercise restraint**
Attempts to overcome the habit are conceptualised as reflecting the strength of the punishing outcomes. Most habits produce mixed effects, some pleasant, others aversive, and so the addicts may find themselves in an approach-avoidance conflict, where motivation fluctuates between wanting to use and wanting to stop.

**Individual motives**
Where social learning begins to take on a special character is in recognising that different classes of drug exert different types of effect and the effects that are most rewarding will differ between individuals and their desires, which also depend upon their past history, personality traits and current life circumstances.

For example an individual who takes a drug as a reaction to a social problem will face different issues when attempting to overcome addiction compared with an individual who uses drugs in a social environment where all his or her acquaintances also use the drug.

**Application to relapse prevention**
Marlatt and George (1984) have applied SLT to understanding and treatment of addictions in the Relapse Prevention Model (see also the earlier discussion on the Abstinence Violation Effect). The Relapse Prevention Model focuses on the factors that will influence the success or failure of an attempt to maintain abstinence, but there is a great deal of overlap with processes that were involved in the initial development of an addiction.

Under the relapse prevention view, all addicts have a range of discriminative stimuli (cues) for drug use. After a period of abstinence, if the individual comes into contact with a cue s/he is at higher risk of relapse. If there are multiple cues present, then the risk escalates further. Marlatt and George (1984) propose a cognitive process whereby the cues arouse positive ‘outcome expectancies’ and thus trigger a motivation to use drugs.
The importance of personal resources

The ability of the addict to overcome such cognitive pressures depends upon factors such as their strength of will not to use the drug, their knowledge of alternative strategies for coping with the situation and their level of self-efficacy. The theory focuses attention on the personal ‘resources’ at the person’s disposal to deal with motivational pressure to lapse or relapse. A high degree of self-efficacy combined with a strong motivation to remain abstinent may be insufficient if the addict does not have the knowledge or skill to resolve the situation in some way.

Effects of attempts at restraint on future attempts at abstinence

The outcome of the high-risk situation and the way in which the individual interprets it has important consequences for future progress. As noted in an earlier chapter, successfully resisting and continued abstinence contribute to increased self-efficacy and the individual will be more confident in his or her ability to handle future threats successfully. This increase in self-efficacy may increase the likelihood of attempting alternative strategies in high-risk situations and thus improve his long-term chances of abstinence. In contrast, a lapse, either because of unprepared coping strategies or a lack of self-efficacy, then leaves the individual at risk of relapse.

Issues and evaluation

This theory has a great deal in common with simple instrumental learning theories and indeed simple rational choice theories but focuses attention on the concept of ‘personal resources’ that are needed to resist motivational pressures to lapse or relapse. In that regard it has the potential to add value to these approaches.

Associative learning

The theories in this chapter have focused on associative learning mechanisms that operate outside of conscious awareness. It is noteworthy, however, that in most cases they have found it necessary to introduce mentalist concepts such as choice, psychological resources and self-control to provide an explanation of the phenomenon of addiction in humans.

One must always exercise extreme caution when extrapolating from rat and mouse motivation to human motivation, but the fact that addictive drugs can come to control the behaviour of these other species suggests a non-self-conscious mechanism by which they do so in humans. It gets more speculative when one gets into the territory of specific effects on specific neural pathways, and even in rats and mice there is still a great deal of uncertainty about what is going on. However, the experiments suggest some very plausible hypotheses about instrumental and classical conditioning effects in humans that look as though they are relevant.