CONCEPTUALIZING ADDICTION

Theories of drug craving, ancient and modern

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Abstract

This paper reviews the principal theoretical models of drug craving and provides some directions for future research. The main models are classified broadly into three categories: (1) phenomenological models; based on clinical observation and description; these have been influential in classification systems of addictive disorders and in the development of pharmacological therapies; (2) conditioning models: based on conditioning theory; these have been influential in the development of cue exposure treatments; (3) cognitive theories; based on cognitive social learning theory: these have been influential in the development of cognitive therapies of addiction. It is concluded that no one specific theory provides a complete explanation of the phenomenon of craving. However, theories of craving grounded in general theories of human behaviour offer greatest promise, and generate more specific and testable research hypotheses. Theories that do not require craving to be present for relapse to occur have more empirical support than those that provide simplistic causal explanations. The cue–reactivity model shows promise in the exploration of the relationship between craving and relapse. However, further attention to the phenomenology of craving could help to advise the future measurement and study of drug craving, particularly in the context of research in which drugs are available to human subjects, with adequate ethical safeguards. There is a need for further study of the temporal dynamics of craving and consensus in the field on the most appropriate methods of measurement. Finally, new psychotherapies such as cue exposure and pharmacotherapies that aim to attenuate drinking behaviour, such as naltrexone and acamprosate, provide opportunities to improve understanding of the nature and significance of craving. However, the relatively uncritical assumption that craving is the underlying basis of addiction and represents the most appropriate target for treatment is challenged.

Introduction

The concept of craving has endured mixed fortunes over the years. In antiquity craving was seen as the key substrate of addiction and the driving force behind continued use of a drug in spite of increasingly severe consequences. Lettsom (1789), for example, observed that for the ‘inebriate’ alcohol:

... becomes as necessary as food ... neither threats nor persuasions are powerful enough to overcome [the desire for alcohol], and the miserable sufferer is so infatuated, as in spite of locks and keys, to bribe by high rewards the dependent nurse, privately to procure the fatal draught (p. 157).

Kerr (1889) refers to craving for alcohol as ‘Pathological depravity of the appetite centre’ (p. 221) and to ‘the dire drink crave, as in the punishment of Tanatalus provokes the thirst it can never quench’. In this century, Jellinek...
(1960) regarded craving for alcohol and relapse (or ‘loss of control’) as being intimately related. In Jellinek’s view, craving was in part due to a true physical demand for alcohol as a result of changes in cellular metabolism.

There is no doubt that such beliefs in the central role of craving as the ‘cause’ of addiction had their roots in careful clinical observations: what alcoholics told the physician or scientist about their experiences. For example, the following account of Jack London (1913) conveys the centrality of craving in the relapse process on encountering a reminder of past drinking:

Benicia showed before me ... where in the old days I had lived and drunk deep. I had no intention of stopping at Benicia ... The tide favoured, the wind was fair and howling—glorious sailing for a sailor.. And yet, when I laid eyes on those fishing arks lying in the waterfront tules, without debate, on the instant, I put down the tiller, came in on the sheet, and headed for shore. On the instant, out of the profound of my brain-fog, I knew what I wanted. I wanted to drink. I wanted to get drunk.

Similarly, the centrality of craving forms the basis of many accounts of Alcoholics Anonymous members.

However, even as Jellinek formulated his disease concept of alcoholism, the seeds of doubt about the concept of craving were being sown. With the rise of behavioural science in the addictions, questions began to be asked about the utility of the craving concept. Mello (1972), for example, argued that craving is a tautological concept because craving was most often defined by subsequent drinking behaviour: if a person drank, it was because they were craving. Later, Marlatt (1978) suggested that craving is an epiphenomenon, a cognitive rationalization used by drinkers to explain relapse, but one which is neither necessary nor sufficient to cause relapse.

Nevertheless, craving found its way into international classification systems as a key symptom of alcoholism and later alcohol dependence. A WHO panel (Jellinek et al., 1955) regarded craving as the underlying basis of the onset of addiction, excessive drinking, loss of control and relapse. Craving subsequently entered the International Classification of Diseases (ICD) as a key symptom of alcoholism. Later, Edwards & Gross (1976) described the ‘subjective awareness of the compulsion to drink’ as one of the seven elements of the dependence syndrome, although they noted that craving need not be present in order to establish a diagnosis. A ‘strong desire or compulsion’ to use a drug remains one of the diagnostic criteria of ICD-10 (although it does not now feature in DSM-IV).

As more research on craving was conducted in the 1970s and 1980s, it became apparent that many addicts did not experience craving, that relapse is not invariably preceded by craving, and that craving does not inevitably lead to relapse (Tiffany, 1990). The term ‘craving’ had been used in many different ways by different researchers, with different operational definitions or sometimes little in the way of definition at all. This led Kozlowski & Wilkinson (1987) to call for more precise terminology applied to craving research. A meeting on craving organized in 1991 by the US National Institute of Drug Abuse (Pickens & Johanson, 1992) came to an essentially pessimistic view about the future of craving research. It was not possible for the group of scientists involved to reach a consensus about the nature or relevance of the craving concept. However, it did help to generate some new research directions which will be described in this paper. So by the end of the 1980s, the concept of craving had, so to speak, fallen on hard times.

Then came a growing interest in the development of new drugs aimed at attenuating drinking behaviour (e.g. naltrexone, acamprosate). Many of the studies carried out to investigate the effects of these drugs in human alcoholic subjects adopted the relatively uncritical assumption that they might achieve their effects through reductions in craving. This assumption has been challenged (Lowman et al., 2000) on the basis that medication-reduced craving may not translate into the prevention of relapse, because craving and relapse tend to be at best poorly coupled, and at worst, unrelated. Some have suggested that the target of medications development in this area might be addiction itself rather than craving (Moncrieff & Drummond, 1997). Nevertheless, the development of and interest in such medications has led to what has been described as a ‘renaissance’ in the concept of craving, both from a theoretical perspective, and in relation to...
the development of instruments to measure craving (Anton, 1999).

Although craving is enjoying something of a comeback, there remains a lack of consensus in the field about the nature of craving, theoretical models that best characterize it, and the most appropriate ways to measure it. Some of the theoretical models have been developed exclusively in animals. Because animals are unable to provide researchers access to their thoughts and feelings, one must rely on the observation of their behaviour in controlled conditions and make assumptions about the kind of processes that underlie the behaviours (Li, 2000). In this sense, animal models of ‘craving’ may, in the end, tell us more about the nature of addiction than providing insights into human craving per se. Thus some of the animal derived theories of craving tend to be underspecified or even silent on what the craving experience may be like, or its significance to human craving, although they may be of help in understanding addiction itself.

In a similar way some of the craving theories derived from human research do not assign a central role for craving in the process of addiction or relapse. Contrary to earlier more simplistic clinical models, several modern theories of craving adopt the view that conscious (cognitive–symbolic) craving need not be present for relapse to occur (e.g. Drummond et al., 2000). This does not negate the importance or relevance of craving in human addictive behaviour, a position that would clearly be contrary to the experience of addicts and clinicians. Rather, it poses a challenge for research to define the conditions under which craving occurs and in which it is related to drug-taking behaviour.

This paper describes the main theories of craving, drawing on previous reviews (Pickens & Johanson, 1992; Singleton & Gorelick, 1998) and a forthcoming issue of Addiction on ‘Research perspectives on alcohol craving’ particularly one review (Lowman et al., 2000) that explores these theories in more detail. The aim of the paper is to take stock of theories of craving at the beginning of a new millennium and to explore directions for future research.

What is craving?
In some ways it is perhaps easier to say what craving is not rather than what it is. As Singelton & Gorelick (1998) point out, ‘There is often a discrepancy between the standard dictionary definition of craving as “a strong desire” and how persons with alcohol-related problems use the word to mean “any desire or urge, even a weak one, to use that substance”’ (p. 178). Similarly, researchers and clinicians use the term ‘craving’ to mean various things including liking, wanting, urges, desires, need, intention or compulsion to use (Kozlowski & Wilkinson, 1987; Drummond et al., 2000). Sometimes the autonomic and behavioural correlates of subjective craving have been included in definitions of craving. In general, however, craving is taken to be ‘the conscious experience of a desire to take a drug’. As will be revealed, some theories of craving provide more precise predictions about the nature of craving, and the conditions under which it might occur, than do others. Therefore, some theoretical models lend themselves more to investigation in humans where a precise set of experimental conditions can be created to elicit and measure craving, and its antecedents and consequences. Other theoretical models are less well specified with regard to craving and lend themselves more to the study of addiction than they do for craving itself. Models derived solely from animal research are mentioned here only in so far as they might shed light on the human experience of craving consistent with the operational definition provided above.

Phenomenological models
Phenomenological models of craving are essentially descriptive rather than explanatory, and are derived from the interview and observation of clinical addict populations. Such models have their roots deep in the history of the field, and some examples were given in the introduction (e.g. Jellinek, 1960; Edwards & Gross, 1976). Craving is taken to be a symptom of an underlying addictive disorder, much as negative hedonic mood states are taken as symptoms of a depressive disorder. Isbell (1955) made a distinction between physical (or ‘non-symbolic’) and ‘symbolic’ craving, based on his clinical observations, the former being principally a manifestation of drug withdrawal, and the latter being a precipitant of relapse after a prolonged period of abstinence, long after physiological withdrawal had subsided.

More recently, Modell and colleagues (1992) have highlighted the symptomatic similarities be-
tween addiction and obsessive compulsive disorder. Subjective craving for drugs or alcohol has been characterized as having obsessive elements, and the use of drugs within this model may represent compulsive behaviour in addicts. Modell et al. also point to the potential similarities in underlying neural pathways implicated in the two disorders, suggesting that they may share a similar aetiology.

The strength of phenomenological models is in their attention to the human experience. Their popularity in the field may in part be related to the simplicity of having a ‘symptom’ for addiction (i.e. craving) which is readily elicited and can form the basis or part of a diagnostic interview. However, therein also lies a danger in assuming that craving is essential to make a diagnosis of addiction, or that craving should be the primary target for treatment interventions. As stated earlier, this may account for the loss of confidence amongst researchers in phenomenological models when empirical research failed to find a direct connection between craving and drug use. Further, looking more closely at individual phenomenological models, one can see that most, if not all, make assumptions or predictions about the underlying processes that may account for the craving experience. In Jellinek’s case there was an assumption about changes in cellular activity. In Isbell’s work one can see the beginnings of conditioning theories of craving and relapse. Thus, although phenomenological models are useful in helping to generate hypotheses about craving, they offer few specific pointers as to how to test hypotheses. This requires recourse to more specific general theories of behaviour.

Conditioning theories

1. Conditioned withdrawal model

Wikler (1948) provided a formulation of craving, or more specifically relapse, based on conditioning theory. He proposed that neutral stimuli in the environment can, over the course of many pairings with drug taking, come to elicit conditioned responses through a process of conditioned learning. He proposed that the conditioned responses elicited by cues (e.g. the sight of a needle and syringe, or a place where drugs had previously been injected) would resemble drug withdrawal. This conditioned withdrawal would, in turn, lead to relapse to drug taking through the addict’s desire to relieve the unpleasant conditioned withdrawal experience.

This conditioned withdrawal model was later expanded by Drummond, Cooper & Glaubi (1990) and is displayed in Fig. 1. A cue (conditioned stimulus, CS), such as the sight and smell of a favourite drink, is repeatedly paired with a falling blood alcohol level (unconditioned stimulus, US) the morning after a heavy drinking session when the alcohol dependent drinker is in a state of alcohol withdrawal (the unconditioned response, UR). Following a period of abstinence, once withdrawal has subsided, the CS can elicit a conditioned withdrawal response (CR) which resembles alcohol withdrawal. As craving is part of the withdrawal syndrome, conditioned craving within this model is elicited as part of the conditioned withdrawal response. Craving is relieved by alcohol consumption and reinstatement occurs through a process of negative reinforcement. Craving in the context of conditioned withdrawal is similar to Isbell’s (1955) ‘symbolic craving’. Drummond (2000) draws attention to the distinction between ‘cue-elicited craving’ (craving in response to cues) and ‘withdrawal-related craving’ (craving taking place in the context of unconditioned drug withdrawal). Within a conditioned withdrawal model, these two forms of craving are believed to represent the CR and the UR respectively. Drummond (2000) has proposed that although these two forms of craving are likely to be correlated and at times co-exist, cue-elicited craving is likely to be more predictive of relapse than withdrawal-related craving. This is because cue-elicited craving occurs, by definition, in the presence of cues, including access to the drug itself, whereas withdrawal craving occurs mostly in the absence of the drug. Relapse can only occur when the drug is available.

Wikler (e.g. Ludwig, Wikler & Stark, 1974), regarded craving as a negative and dysphoric mood state, and as the key element of conditioned withdrawal that drives the relapse process. This has subsequently been challenged on the basis that craving is often reported by many drug addicts to be a pleasant and positively hedonic mood state, and that cues are equally likely to be associated with pleasurable drug effects during a conditioning history (e.g. Lyvers, 1998; also see conditioned drug-like model below).
2. Conditioned opponent process model
Siegel (1989) has put forward a similar conditioning model that draws on Solomon & Corbitt’s (1974) opponent process theory. Over the course of a drinking career the body develops opponent processes that are homeostatic responses which counteract the drug’s effect. Thus if a drug causes a positive hedonic state (pleasure), the homeostatic response is a negative hedonic state (displeasure). The combination of the two is essentially to move from a positive state to a neutral hedonic state. Gradually the opponent process increases in size and duration and, according to Siegel, this could account for the development of drug tolerance, as it will oppose and ‘cancel out’ the drug effect, and so lead to tolerance. However, as the opponent process is slower in onset and longer in duration than the drug effect, with increasing use the user experiences dysphoria (withdrawal) after the initial drug effect has worn off.

Importantly in terms of conditioning and relapse, Siegel’s model predicts that the CR produced by a drug cue will be a conditioned opponent process that will be negatively hedonic. Thereafter, the prediction of this model in terms of subsequent drug taking will be essentially the same as Wilder’s model (i.e. the addict will take more drug to overcome the negative hedonic craving state).

3. Conditioned drug-like model
Stewart, de Wit & Eikelboom (1984) describe a conditioning model alternative to the previous two. They note that the experience of craving can often be pleasurable rather than withdrawal-like, particularly in relation to stimulant drugs such as amphetamines. They proposed that environmental cues, following repeated pairings with drug taking and the pleasurable (unconditioned) drug effects, can come to elicit drug-like CRs. These positively hedonic CRs (including craving) then prime the individual to take more drug through a process of positive reinforcement.

4. Two process theory
Glautier & Remington (1995) point out some of the failings of such “monistic” models of CR form (i.e. models that propose CRs to drug cues will be unidirectional). They show that the empirical data do not definitively support either a drug-like or a withdrawal-like response. Further, drawing on the broader conditioning literature, they note that one cannot assume the motivational
significance of cues or responses to cues based on their presumed affective valence. In other words, one cannot assume that a positively- or negatively-hedonic conditioned craving state will necessarily lead to drug use.

Tiffany (1995a) takes this analysis further and examines modern theories of the relationship between operant and classical conditioning. You will recall that it was assumed in Wikler’s model, for example, that conditioned withdrawal would lead to drug taking through a process of negative reinforcement. Citing the work of Rescorla & Solomon (1967), Tiffany notes that conditioned stimuli elicit a ‘central emotional state’ that functions to motivate or modulate instrumental behaviour. Depending on the conditioning history of the animal and the central emotional state a CS elicits, the effects of exposure to a CS may, paradoxically, decrease rather than increase subsequent drug taking. Thus one cannot assume, as do simple monistic models, that negatively hedonic conditioned craving, for example, will automatically lead to relapse. Similarly, extinction of conditioned responding (including conditioned craving) through repeated exposure to cues without drug taking (as in cue exposure treatment) may theoretically, and paradoxically, increase rather than decrease drug taking (Drummond et al., 1990). Although this does not appear to be the case empirically, there is a need to develop more sophisticated research paradigms that take account of the effect of cues on central emotional states to study the significance of craving and conditioned responses in humans.

5. Incentive sensitization theory
This is the subject of another paper in this issue (Robinson & Berridge, 2001) but will be dealt with here as it relates to the issue of craving theories. Incentive sensitization theory as described originally by Robinson & Berridge (1993) is more a model of addiction and addictive behaviour rather than craving per se. However, it has relevance to theories of craving in that certain predictions are made regarding the nature of craving within this model. Citing a wide array of research findings principally from animal literature on the effects of stimulant drugs they propose that the neural system (mesolimbic dopaminergic pathway) responsible for drug seeking and drug taking becomes sensitized by repeated drug use. This system is responsible for incentive motivation and reward and is separate from systems that mediate the pleasurable effects of drugs. Thus Robinson & Berridge make a distinction between ‘liking’ a drug and ‘wanting’ a drug. They argue that ‘wanting’ is associated with the sensitized incentive motivational system, and is different from ‘liking’ which is synonymous with craving. Further, they add that wanting may not always be conscious and hence relapse may occur without conscious awareness (Robinson & Berridge, 2001). The expression of sensitization is said to be ‘context-specific’ and is hence mediated by conditioning processes. Thus incentive properties of drugs may be strongest in the contexts in which they were normally taken in the past.

The model as currently formulated provides an essentially pessimistic view of craving (liking), namely that it is epiphenomenal to the hypothesized unconscious process (wanting) driving addiction and relapse. Further, there is no clear way that one could begin to alter sensitization processes either pharmacologically or psychologically as they are a biological neuroadaptive consequence of prolonged drug taking. However, it should be noted that most of the evidence for this model is based on animal research with stimulant drugs and it cannot be assumed in advance of the appropriate research that this applies to alcohol. Further, as London’s (1913) earlier quotation highlights, ‘wanting’ alcohol may be highly conscious.

On the other hand, incentive sensitization may offer some insights into the ‘priming effect’ of alcohol in which the incentive salience of, and craving for, alcohol is primed by small doses in alcohol dependent individuals. It would be possible to study the influence of priming doses on craving, as has been carried out in the 1970s (e.g. Rankin, Hodgson & Stockwell, 1983), providing appropriate ethical safeguards for alcohol dependent subjects being exposed to alcohol can be ensured.

6. Cue-reactivity model
Because of the conflicting findings of empirical studies examining the nature and significance of CRs to drug cues, Drummond and colleagues (1995) proposed a cue-reactivity model as a means to conceptualize and study responsivity to drug cues. Within this model cue-reactivity can
be autonomic (e.g. increased skin conductance, heart rate, salivation), cognitive-symbolic (e.g. subjective craving) or behavioural (e.g. drug-seeking behaviour). The model does not assume that autonomic or subjective craving responses to drug cues are intervening variables in the cue–drug-seeking relationship. In other words, cue-elicited craving is not considered to be a necessary condition for drug seeking or relapse to drug taking. Further, the cue–reactivity model does not assume an underlying conditioning mechanism, and thus provides a bridge between conditioning and cognitive theories. Cognitive-symbolic cue–reactivity could include changes in outcome and efficacy expectancies (see below), which may be important in subsequent drug-seeking behaviour. Further, it has been argued that cue reactivity could represent variously an orienting response, frustration or generalized arousal, rather than being a conditioned response (e.g. Drummond et al., 1990). Again the cue–reactivity approach can incorporate such models in experimental design rather than assuming an underlying conditioning mechanism. Importantly, the cue–reactivity paradigm, in which individuals are exposed to drug-relevant cues in an experimental setting, provides an important means of studying craving and other cue-elicited phenomena and their relevance to drug seeking and relapse.

The cue–reactivity model has given rise to psychological treatment approaches that encompass both conditioning and cognitive theories. Cue exposure treatment aims to reduce relapse liability by altering cue reactivity (Monti et al., 1993; Drummond & Glautier, 1994). Such an approach can be conceptualized both in terms of conditioning theory (e.g. the extinction of CRs to drug cues following repeated unreinforced exposure to drug cues) or cognitive theory (e.g. the enhancement of self-efficacy (see below) by rehearsal of coping strategies in a ‘safe’ environment) (Drummond et al., 1995). Thus cue exposure treatment does not necessarily assume any particular theoretical model of drug craving, but rather provides an empirical treatment approach in which craving theories can be tested.

Cognitive theories

1. Cognitive social learning theory

Marlatt & Gordon (1985) have developed a cognitive social learning theory (CSLT) of relapse and relapse prevention which has been adopted widely by the treatment community as a basis for psychological treatment. Although this is predominantly a theory of relapse, it has relevance to understanding craving and its role in relapse. Central to CSLT is the proposition that in a given ‘high-risk situation’ (one in which the abstinent drinker is faced with the choice of drinking or not drinking) the likelihood of relapse will depend on their expectations. Efficacy expectations are the individual’s confidence in his/her ability to resist the temptation to drink (Bandura, 1977). Outcome expectations are the individual’s beliefs about the consequences of drinking or not drinking. A positive outcome expectancy would be the belief that alcohol would have a positive effect (e.g. lead to pleasure, relieve pain), and a negative expectancy would be that drinking alcohol would have a negative effect (e.g. lead to hangover or loss of a job). The outcome will depend on an interaction between these factors. For example, in the presence of positive outcome expectations and low self-efficacy, relapse is more likely.

Marlatt (1985) views the role of craving in relapse as being mediated by the anticipated effect of taking the drug, i.e. the expectancies. In this sense craving could be seen as a ‘desire for positive drug effects’. However, he also invokes conditioning theory to explain craving suggesting that craving may be ‘... a conditioned response elicited by stimuli associated with past gratification’ (p. 49). Marlatt also regards craving and self-efficacy to be reciprocally related, such that high craving should undermine self-efficacy as it challenges the addict’s coping skills.

Beck et al. (1993) extended Marlatt’s theory by describing four main types of craving: (1), craving in response to withdrawal symptoms (craving is the ‘need to feel well again’); (2) response to lack of pleasure (attempts to improve mood); (3) ‘conditioned’ response to drug cues; and (4) response to hedonic desires (e.g. ‘the habit of combining drugs and sex as a way to magnify sexual experience’) (p. 158). Beck et al. also regard craving as having a cognitive basis in dysfunctional beliefs about the perceived need for a drug, thus in their view, confusing ‘urge’ with ‘need’. The cognitive restructuring of such beliefs therefore becomes a focus for cognitive therapy.

Marlatt & Gordon (1985) and later Lowman
et al. (1996) have provided evidence that a variety of situations and affective states are described by addicts as relapse precipitants. Most often cited are negative mood states and social pressures to drink, with craving and responses to cues as relatively unimportant (Marlatt, 1996). However, Drummond et al. (1995) have pointed out that high-risk situations including social pressure and negative affect are themselves cues, and that a wide range of cues including alcohol itself will be present in high risk social situations. Thus it would be difficult for an individual to identify which single factor amongst the whole array of cues present in a situation of social pressure to drink, was responsible for relapse. It is more likely to be a combination (or cluster) of cues, both interoceptive and exteroceptive, that can contribute to relapse (Drummond, 2000). In this sense, it is perhaps more useful to regard conditioning and cognitive accounts of relapse as being different levels of analysis rather than being contradictory (Drummond et al., 1995).

2. Cognitive labelling model
Several researchers have suggested that craving represents the cognitive labelling of physiological processes, such as those arising from drug withdrawal or conditioned responses to cues (Cooney et al., 1984; Kozlowski & Wilkinson, 1987; Drummond et al., 1990; Tiffany, 1990). This model is based on Schachter & Singer’s (1962) cognition–arousal theory of emotion, and applied to drug craving. Cognition–arousal theory posits that the emotional state will depend on an interaction between the physiological state of arousal and the cognition appropriate to that arousal. The cognitive labelling of this physiological state is subject to external situational factors and the individual context. Thus, as Tiffany (1995b) points out, an addict may interpret the internal physiological effects of exposure to cues associated in the past with drug use, as craving for the drug. Thus craving during drug withdrawal may be a different phenomenon from craving in response to a drug cue, but may be interpreted (cognitively) by the addict as the same (see Drummond, 2000, for a more extensive description of the distinction between cue-elicited and withdrawal-related craving). Further, West & Schneider (1987) proposed that craving for cigarettes arises from the smoker’s interpretation of the physiological changes caused by nicotine withdrawal as a need for a cigarette. The potential importance of this model is that the addict’s craving responses to cues could be, theoretically at least, diminished through therapeutic interventions aimed at cognitive reframing of craving. However, Tiffany has noted that the cognitive labelling model has not been clearly specified in addictions, and there is only limited evidence in support of the model. Further, Schachter & Singer’s (1962) cognition–arousal model has not in itself been well supported by research (e.g. Reisenzein, 1983).

3. Dual affect model
Baker, Morse & Sherman (1987) proposed that drug taking is regulated by complex affective processing systems. Within this model craving (or urges) may be elicited by either an appetitive response to drug cues (‘positive affect urges’) or a withdrawal based response (‘negative affect urges’). Thus within one individual different cues could elicit either positive or negative urges. However, importantly, the two systems are hypothesized to interact with each other such that stimulation of the positive affect urge system will inhibit the negative affect system. Further, they propose that priming doses of a drug will increase the salience of a drug cue, which is consistent with some empirical evidence. This theory provides an explanation for the apparently conflicting evidence that both positive and negative mood states can be associated with craving and relapse.

Tiffany (1995b), however, has criticized the model on the basis that positively- and negatively-hedonic urges empirically tend to be positively correlated with each other rather than mutually inhibitory. Nevertheless, the model has several attractions, particularly in relation to understanding the effect of the prevailing mood state on cue reactivity.

4. Dynamic regulatory model
Niaura and colleagues (1988) developed a dynamic regulatory model of addiction that incorporates several of the theoretical models already described. They argued that craving arises from a combination of conditioned responses to cues, and positive and negative affect. Subsequent drug use and relapse is then mediated by coping
5. Cognitive processing model
Tiffany's (1990) cognitive processing model of cue reactivity posits that drug use is, rather like driving a car, essentially an automatic process, and is therefore carried out without conscious awareness or effort most of the time. Thus craving will not occur during a typical drug use sequence, although there will be accompanying physiological processes required to initiate and carry out drug consumption (Fig. 3). However, if the addict's normal process of drug taking is impeded by, for example, access to the drug being denied, a non-automatic, effortful cognitive process is elicited. In this model craving is conceptualized as 'constellations of verbal, somato visceral and behavioural responses supported by non-automatic cognitive processes' (p. 156). Thus, in his example, the alcoholic who is denied access to alcohol may be frustrated, annoyed, craving for alcohol, and engaged in cognitive problem solving and behavioural responses (e.g. running to another pub) to gain access to an alternative supply of alcohol. An alcoholic who is attempting to abstain from alcohol and is confronted by an alcohol cue in cue exposure may experience craving as a result of the elicitation of non-automatic cognitive processes. Such responses would be very different from the alcoholic's response to cues during a drinking phase before a commitment to abstinence has been made. This is clearly a very different conceptualization from the conditioned...
response models described earlier in which relatively simplistic assumptions are made about the relationship between cues and CRs. Tiffany argues that this could explain the lack of a reliable association between cues and craving, and the lack of predictive power of craving in relapse.

Tiffany’s theory is often interpreted as being essentially pessimistic about the role of craving in relapse. However, closer examination reveals that subjective craving, and associated physiological and behavioural responses to cues could, under certain conditions, be predictive of relapse within this model. However, this theory needs to be empirically tested further in clinical addict populations, as most of the work has been so far carried out in smokers.

Conclusions
What can be concluded from this review of craving theories? Has any progress been made since early phenomenological models posited a role for craving in addiction and relapse? What are the implications of such advances for future research and treatment? It would be a brave researcher who would choose one particular theory as representing the complete explanation of craving over all others, given the state of existing evidence. Although research has advanced particularly in the past two decades, most often the key experiments to compare one theory with another have not yet been conducted. However, some pointers can be identified.

It is clear from this review that more specific theoretical models with a firm basis in general theories of behaviour (e.g. conditioning and cognitive theories) allow more accurate specification of the nature and expected antecedents and consequences of craving when compared with earlier more simplistic phenomenological models. However, basic conditioning models are inadequate to explain fully the role of craving and the relapse process. Craving research would be well served by taking account of recent advances in general conditioning theory, including two process learning theory concerning the hypothesized interaction between operant and classical conditioning, rather than assuming simple causal relationships. The role of central emotional states is particularly important to study in this respect. Further, there is some evidence that cognitions such as efficacy and outcome expectancies may have a
role in the craving–relapse relationship. This needs further exploration, and the cue–reactivity paradigm offers promise in elucidating these relationships.

Overall, theoretical models that assume a direct causal relationship between craving and relapse are not supported by the research evidence. There is a need to develop theories of craving that do not require craving to be present for relapse to occur. This does not mean that craving is simply an epiphenomenon, but rather opens the way for the study of the conditions under which craving may be related to drug taking. The issue of the predictive validity of craving measures has been discussed in more detail elsewhere (e.g. Tiffany, 1992; Drummond et al., 2000). It is possible that craving has a causal role in relapse, but a measure of craving may have little or no predictive value. This may be because we have imperfect measures of craving, and therefore need to develop improved methods of measurement. Alternatively, if craving varies considerably over hours or days, it may be that existing craving measures cannot capture the craving occurrences relevant to relapse. For example, measures that aggregate long periods of time (e.g. assess average craving over the past week), or measure craving at a time point distant from the relapse event (e.g. at the beginning of a residential treatment programme), may have little or no predictive power.

Little is known of the ‘temporal dynamics’ of craving (Drummond et al., 2000). Several of the models described here make different predictions about the likely temporal relationships between craving and relapse. Short-lived, cue-elicited responses may be more predictive of relapse than withdrawal-related or background levels of craving. However, little attention has been paid to such temporal issues in the development of craving measures with some exceptions (e.g. ecological momentary assessment of craving; Shiffman et al., 1997; Shiffman, 2000). Most often craving measures have been unidimensional, single-item questionnaires of unknown validity and reliability (Tiffany, 1992; Sayette et al., 2000). There is a need for the field to reach consensus on the most appropriate methods to measure craving, not only the subjective elements, but also the autonomic and behavioural correlates.

This review does not aim to dismiss the relevance of phenomenology in the study of craving. As has been argued previously (Drummond et al., 2000), little attention has been paid to research on the ‘domains of craving’ (i.e. the precise study of the phenomenology of the human experience of craving). This includes further study of the ‘ecology of craving’, including the study of the human craving experience in the natural drinking environment as opposed to a laboratory setting: most craving research has so far been in the latter setting. Further, it has been suggested that experimental paradigms where access to drugs or alcohol is permitted, could allow more direct observation of the relationship between craving and drug taking (Meyer, 2000), providing adequate safeguards to human subjects can be assured (Dolinsky & Babor, 1997). Similarly, there has been relatively little interest in autonomic and behavioural correlates of craving compared with the cognitive–symbolic elements. The development of human brain imaging paradigms may further expand the opportunities to study and conceptualize craving.

In relation to treatment, it can be concluded that we are some way from finding a direct application for craving research in the treatment arena. However, treatment approaches that assume a simplistic causal relationship between craving and relapse are, on the basis of existing knowledge, likely to prove inadequate. However, recently developed psychological and pharmacological treatments could offer opportunities to further elucidate the nature and relevance of craving itself. For example, cue exposure treatment allows a method to study the occurrence of subjective craving and its autonomic and behavioural correlates in a laboratory setting, and its relevance to relapse (Drummond et al., 1995). If cue exposure treatment is able to extinguish conditioned responses to drug cues in a laboratory setting, does this translate into a reduced likelihood of relapse? There is some limited empirical evidence in support of this in relation to alcohol dependence, but it appears to be much more complex than a simplistic conditioning model would suggest (Monti et al., 1993; Drummond & Glaudier, 1994). Does the combination of cue exposure and cognitive therapy aid relapse prevention? Again, there is some evidence in support of this notion (Rohsenow et al., 1995). Equivalent research needs to be conducted in the opiate, cocaine and nicotine fields.

The development of medications to attenuate
drinking behaviour and/or prevent relapse has been both a stimulus to the ‘renaissance’ of craving research, and a potential threat to its future. The relatively uncritical assumption that craving represents both an underlying cause of addiction and the appropriate target for relapse prevention has been questioned. Indeed, little is known about the precise mechanism of effect of drugs such as naltrexone and acamprosate in the attenuation of drinking behaviour (Lhuintre et al., 1990; O’Malley et al., 1992; Volpicelli et al., 1992; Littleton, 1995; Swift, 1995; Sass, 1996; Whitworth et al., 1996; Monti et al., 1999). Even so, these drugs are often referred to as ‘anti-craving’ medications in advance of clear evidence that the drug effects are mediated by effects on craving (e.g. Davidson, Swift & Fitz, 1996; Spanagel & Zieglsangberger, 1997; Kratzer & Schmidt, 1998). Therefore, it would be unfortunate if failure to demonstrate the positive effects of such medications on craving turned attention away from promising, and otherwise beneficial, addiction treatments.

There is a need to study the effects of medications such as naltrexone and acamprosate on craving, including their effects on cue-elicited craving, and autonomic and behavioural reactivity. Some progress has been made in this area in relation to understanding the effects of medications on the reinforcing effects of alcohol, and on cue reactivity (e.g. Littleton, 1995; Swift, 1995; Monti et al., 1999). It is possible that this research could help to elucidate the neurobiological substrates of craving through a variety of research paradigms including cue reactivity and neuroimaging. As these medications have specific effects on neurotransmitters, it may be possible to study craving through the influence of specific receptor blockade and/or agonist effects (e.g. Littleton, 2000; Drummond, 2000).

In conclusion, craving remains an enduring and fascinating phenomenon for basic through to clinical research. It also continues to be perceived as a major practical problem for both clinicians and their drug-addicted patients. It has eluded complete explanation for several centuries. However, recent theoretical advances may help to shed further light on the nature and relevance of craving and hopefully to develop new treatment approaches to prevent relapse and alleviate the suffering associated with addiction to drugs in the next century.

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