Cognitive social learning and related perspectives on drug craving

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Abstract

Many modern theories of drug use and dependence assign central prominence to the role of craving in drug use and relapse. However, some continue to debate whether drug craving has any motivational significance in terms of drug-using behaviors. Cognitive social learning theory adds additional perspective by imbedding craving within a network of cognitive processes that, as they inter-relate, influence drug use and relapse. This paper reviews tenets of cognitive social learning theory as they relate to craving, focusing on theoretical models that have attempted to explain how craving fits our understanding of the process of drug relapse. In addition, cognitive social learning theory complements theories that emphasize more biological or information processing aspects of addictive behaviors. Studies of addiction that have assessed elements of social learning theory suggest the following: (1) there exists an inverse relationship between efficacy and craving; (2) there appears to be an association between affect and craving, but the precise nature and strength of this association is unclear; (3) the relationship between outcome expectations and craving is largely unknown; and (4) correspondingly little is known about relationships between coping and craving. More empirical study is needed to address the strengths and limitations of cognitive social learning theory as it relates to our understanding the motivational significance of drug craving.

Introduction

The purpose of this paper is to review aspects of cognitive social learning theory (CSLT) as they may relate to understanding the nature and function of craving for drugs. At the outset some caveats are in order. Although there is a need to understand alcohol craving, most of the relevant research has to do with craving for nicotine or other drugs. Therefore, the discussion that follows, and the underlying theoretical principles, apply in large measure to all drugs of abuse. Additionally, focus will be placed mainly on theory and less on detailed results of research studies. The reason for this is that the literature on cognitive social learning correlates of drug use and relapse has burgeoned in the past decade, and findings are mixed. The status of CSLT, in terms of understanding addictive behaviors, still remains unclear except that circumstances are more complicated than they seemed at first, which is usually the case. Be that as it may, the plethora of research on CSLT as applied to addictive disorders, although presenting mixed findings, nevertheless suggests that there is wheat...
among the chaff. For example, increasingly, evidence supports the motivational significance of drug craving in relation to drug use (e.g. 1,2). The challenge that faces us is a heuristic one—revising and refining our understanding of how exactly CSLT principles play out in the arena of substance use and abuse.

A brief overview of CSLT

Let us briefly review some of the central tenets of CSLT as they apply to addictive behaviors. According to Albert Bandura, the father of modern CLST,3 as human beings we live in a social and cognitive world. Behavior is largely, but not exclusively, associated with our ability to manipulate cognitively and store symbolic representations of our environment, which is an elaborate way of saying that what and how we think affects the way we behave; but how we behave also affects how we think, and so on, which is referred to as reciprocal determinism.4,5 This may not come as a surprise, but it has implications for understanding addictive behaviors because it emphasizes active cognitive processing and, more important, conscious reasoning and decision-making in using drugs, quitting drugs and relapsing to drug use.

Efficacy expectations form the cornerstone of human agency.5,6 That is, effortful behavior is thought to be mediated to a significant degree by the confidence we have in our ability to do something: drive a car, write a paper, resist the temptation to use a drug. Cognitive social learning theory therefore assigns central prominence to efficacy expectations in explaining effortful behaviors. However, there are different kinds of expectations that influence efficacy expectations to determine the probability that a given behavior will be executed. Outcome expectations, the subjectively assessed probability that a given action will produce the intended consequences, can influence the effect of efficacy expectations on behavior. For example: a particular outcome is expected after executing a particular response (i.e. an “If–then” framework; e.g. “If I smoke a cigarette, then I will relax”). A corresponding self-efficacy expectancy is the level of confidence to execute a particular behavior (e.g. “I am not confident that I can resist smoking when I am stressed”). Put together: “I am not confident that I can resist smoking when I am stressed; I know that if I smoke, I will relax”. Thus the probability of smoking in a stressful situation is compounded by strong outcome expectations for the effect of the drug, and weak efficacy expectations concerning the ability to execute the skills necessary to resist smoking in that situation. The probability of smoking would be weaker if efficacy expectations were strong, in spite of strong outcome expectations. Two points deserve emphasis: (1) the probability of behavior is a result of the reciprocal influence of efficacy and outcome expectations; and (2) to be meaningful and practical, this theoretical analysis has to be specific in terms of defining thoughts and behaviors that are directly tied to the situation at hand. Research on efficacy and outcome determinants of drug use requires this very micro level of analysis which has generally been lacking (but see Haaga and colleagues’ work7).

What is the ongoing dynamic process by which efficacy and outcome expectations are acquired and shaped? Typically, the most powerful teacher is direct experience—gradually, continued effort with partial success will feed back and change efficacy and outcome expectations.8 This represents an interactive process, with success breeding success and failure breeding further failure. Although an oversimplification, it serves to make the point. Importantly, learning can occur through numerous channels: vicariously by observing others engaging in a given behavior; through verbal means; and by attending to physiological signals in a particular situation. Thus an individual, for example, could learn to use drugs and to stop using drugs by observing others using and not using drugs. For the addict, watching someone use drugs can influence outcome expectations (“drug use will lead to a pleasurable experience”) and efficacy expectations (“I will not be able to refuse the offer of drugs”), which then increases the probability that drug use will occur. It is also important to note that CSLT recognizes the importance of associative and other learning processes as determinants of behavior.9

CSLT and addictive behaviors

A little over a decade ago, Alan Marlatt proposed a social learning model of drug relapse which has shaped a great deal of research and is still highly regarded.10 Marlatt stressed the importance of cognitive factors, for example, in terms of covert antecedents of relapse. In this model, urges or
cravings for a drug influence risk for relapse (high-risk situation) and other cognitive processes (rationalization and denial) which in turn set the stage for relapse. However, in CSLT urges or cravings themselves are believed to be mediated by expectancies regarding the immediate effects of the substance, usually anticipated positive effects of the drug.

Marlatt expanded CSLT by appealing to associative learning theory with a cognitive slant, i.e. the conditioned stimulus (CS) evokes cognitive processes which represent the conditioned response (CR). Referring to these cognitive processes as covert mediators, he made an important distinction—between (1) anticipated positive effects of drug use coupled with desire, which he called the craving response, and (2) the intention to use the drug, which he called urge.

The distinction between desire and intention is key and has not been studied enough, because it is often assumed they are one and the same. However, it is entirely possible that these constructs may be uncoupled under certain circumstances, for example when an addict is confronted by drug cues, and contingencies are such that he or she knows that drug use is impossible. This has been demonstrated in several studies where manipulation of anticipated drug use influences report of craving during drug cue exposure. Whether or not we refer to desire as craving and intention to use as urge, however, is less important to us than it is that we are clear about what we ask subjects to report when we do our studies (our research subjects probably do not keep up with the research literature, so we need to be sure that they understand what it is that we want them to tell us about).

Marlatt went on to describe how an encounter with a high-risk situation does or does not lead to drug relapse. Remember that craving and urges already feed into and help to define the high-risk situation. What is key subsequently is the importance of coping responses which, if lacking or weak, are associated with decreased confidence in ability to navigate the high-risk situation, and are coupled with positive outcome expectations regarding initial use of the substance. If the substance is used, Marlatt predicted the outcome would be the abstinence violation effect (AVE), i.e. a sense of guilt for having transgressed, that would lead to collapse of all resolve to stay abstinent, thereby increasing the probability of relapse to a chronic and dependent pattern of drug use. However, it should be noted that, although theoretically appealing, currently little evidence supports this formulation of the AVE.

**Dynamic regulatory model of drug relapse**

CSLT figured prominently in our own theory of relapse. Our thinking also was shaped by the work of Tim Baker and colleagues, on the dual affect model of cue reactivity, and by Jane Stewart’s work on conditioned incentive, or appetitive motivational, models of addiction. Figure 1 presents a dynamic regulatory model of drug use and relapse that attempts to describe associations among exposure to drug-related cues, affective responding and cognitive social learning factors. Rather than merely sketch out associations among various variables and constructs, this model makes some predictions concerning the direction of relationships, and specifies positive and negative feedback cognitive–behavioral processes.

The model postulates that relapse precipitants can include an affective imbalance from a neutral state (either positive or negative), contextual cues previously paired with drug use and its effects, or both. In the model contextual cues, including drug-related stimuli, can precipitate a reaction alone, but they also can combine with affective precipitants, so their effects could be summative. This prediction is supported by results of a recent study by Cooney and colleagues who found additive effects of alcohol cue exposure and induced negative affect on urges to drink.

In this model, cues are thought to trigger a series of responses including urges to consume the drug, positive outcome expectations for drug use, that is, for initial effects of the drug. Depending on which affective circuit is activated, a somewhat different pattern of responses will emerge. For example, if negative affect is the precipitant, this is likely to trigger outcome expectations concerning drug-induced relief from distress. Physiological responses may include conditioned withdrawal symptoms or arousal associated with escape motivation. If positive affect is the precipitant, then outcome expectations are likely to involve anticipation of pleasurable experiences. The pattern of physiological adjustments may also prepare the individual to obtain the substance, and may reflect attentional processes.
being directed toward substance-use cues, as Tiffany has also noted in his theoretical model of drug urges.\textsuperscript{19} There may also be evidence of conditioned isodirectional physiological responses. This suggests that measurement of psychophysiological responses, or at least peripheral responses, is probably not useful for testing theoretical models related to drug urges, because of the potential competing influences on these responses. However, underlying patterns of brain activity, if they could be specified with sufficient precision to allow real-time measurement of activity in neural circuits thought to be associated with addiction and reward, may be more closely linked to hypothesized constructs in this and similar models.

Reactions to antecedent stimuli can serve as positive feedback to affective states. For example, an urge response and positive outcome expectations (e.g. drug-induced relief of distress) to a negative affective stimulus might amplify negative affect because this reaction might be perceived as aversive: for example, as a threat to abstinence. It is also possible that negative affect increases the salience of immediate positive outcome expectations regarding drug use. Cognitive reactions (positive outcome expectations) to positive affective states are also thought to amplify positive affect in so far as the reactions trigger further anticipation of reward (this is compatible with an appetitive motivational, and possibly even an incentive–sensitization model of drug urges, assuming wanting and liking are not uncoupled\textsuperscript{20}).

Once either of the positive or negative affect circuits is activated, we hypothesize that there will occur an interaction with cognitive–behavioral coping efforts and attributions. So for example, if the cluster of urge, outcome and physiological reactions is intense, then this will undermine existing coping skills and contribute to attributions that this state is uncontrollable, stable and caused by personal factors (e.g. personal weakness). However, to the extent that cognitive–behavioral coping can be brought to bear, and to the extent that causal attributions for the state are congruent with personal control, then the system will be dampened and the probability of drug use will decrease. Self-efficacy is thought to be centrally interactive with reactions to cues and coping efforts, such that efficacy inhibits urges and outcome expectations and increases the likelihood of coping and, reciprocally, urges and outcome expectations decrease confidence.

If the system is energized sufficiently efficacy and coping skills are overwhelmed, and a lapse occurs. This may then feed back to increase positive affective responses and urges, which is consistent with sensitization models of drug use.
A lapse may also increase negative affect because of attributions made about the lapse and dissonance between perceptions of oneself as an abstainer, which represents Marlatt’s abstinence violation effect.

What are some problems with this theoretical formulation? Tiffany recently pointed up several potential weaknesses. First of all, this model does not adequately deal with unconscious or pre-conscious processes in terms of their relation to increasing the probability of drug use either among non-abstinent users or among abstainers. Tiffany has made a very important distinction between automatic and non-automatic processes related to drug use. For example, it is only under conditions where drug use is impeded that urges are likely to occur, and where they may have some motivational significance. In agreement with this formulation, and in response, our theoretical model really applies to the case where abstinent addicts encounter drug cues and negative affect in a high-risk for relapse situation, and where they are motivated to stay abstinent. However, it is entirely possible that all of the cognitive and other processing that our model refers to can occur very quickly and perhaps with varying degrees of automaticity. For example, reactions to drug use cues may be very intense initially, and thereby very quickly knock out positive efficacy outcome expectations and coping behaviors, leading straight to a lapse. We know very little about the time course of these reactions in real time.

A second concern raised by Tiffany has to do with the inherent instability of such a model from a systems engineering point of view—once energized the system will literally explode, because of the activity of some of the positive feedback loops. In all honesty, we did not attend to the integrity of the model from a systems engineering point of view; rather, this was, and still is, primarily a heuristic model, designed to stimulate further thought and hypothesis testing. However, it is also true that there are some brakes built into the system. Efficacy and coping are the braking mechanisms for the affective/urge circuits. We also speculated that relapse, or perhaps more accurately a return to chronic use, would itself temporarily stabilize the system. It is also possible that positive and negative urge/outcome expectancy circuits, once activated, reverberate, but that the intensity of reverberation is constrained by biological limits.

Another concern is the central position given efficacy expectations in the model; everything is funneled through this point. Whether or not this is true remains to be demonstrated. This proposition flows directly from CSLT, which posits that efficacy expectations are central in determining effortful behaviors. Remember, this is a model of cognitive processing that occurs in the abstinent addict who is trying to remain abstinent during an encounter with a high-risk situation. Note, however, that the position that efficacy holds could easily be exchanged for urge in the model. If we assume that urges measure intention to use a drug, then the theories of reasoned action and planned behavior would support the notion that urges will be the immediate determinant of behavior. Data from a smoking cue-reactivity study, presented below, address the potential implications of such an exchange.

Smokers motivated to quit \(n = 46\), and who were abstinent overnight, were presented with a high-risk scenario in the laboratory. This consisted of a role-play in which the smoker interacted with a confederate who pretended to be waiting for their car to be repaired in an auto shop. Unbeknownst to the subject, at a predetermined interval the opposite sex confederate took out a pack of the subject’s favorite brand of cigarette and proceeded to light and smoke it. The high-risk situation is complex, involving drug use paraphernalia, modeling of smoking behavior, the sight and smell of the cigarette, the potential mix of affective reactions associated with performing an unfamiliar exercise with some unknown person under the watchful eyes of the experimenters and the uncertainty of what is required of the subject. After cue exposure, subjects rated their urge to smoke, efficacy at being able to resist smoking in such a situation and anxiety. Their ability to cope with the situation in terms of reducing risk of smoking was rated by blind reviewers. We then evaluated their smoking status at 6 months.

We re-analyzed the results reported previously, performing path analyses to test specifically the mediating role of efficacy vs. urges in response to cue exposure and in relation to 6-month smoking rate. In the first model (Fig. 2a), although the zero order correlation coefficient between urge and outcome was significant, in the multivariate model the effect of urge was no longer significant when efficacy, skill (the measure of coping) and anxiety were en-
Figure 2. (a) Full path model linking efficacy, anxiety and behavioral coping skills to urge and smoking rate, in response to a smoking cue exposure paradigm. Numbers in parentheses represent zero-order correlations. Numbers outside parentheses are path coefficients. $R^2$ is variance accounted for at each step. (b) Reduced path model linking efficacy, anxiety and behavioral coping skills to urge and smoking rate. (c) Full path model linking urge, anxiety, and behavioral coping skills to efficacy and smoking rate. (d) Reduced path model linking urge, anxiety, and behavioral coping skills to efficacy and smoking rate. *$p < 0.05$; **$p < 0.01$; ***$p < 0.001$. 
tered. When effects of the other variables on smoking outcome were removed, however, it appeared that urge represented a final common pathway to outcomes (Fig. 2b). However, this model represents a poor fit to the data ($Q = 0.400; W = 38.45; p < 0.05$), and so cannot be considered tenable. (The closer the $Q$ statistic is to 1, the better the model fit. $W$ is a chi-square value that permits statistical evaluation of the goodness-of-fit of the model; $p$ values should be non-significant for the model to be judged as adequately fitting the data$^{24}$).

Next we switched things around, putting efficacy in the model as the intermediate intervening variable. Notice that in the full model (Fig. 2c), both efficacy and urges have significant direct effects on smoking outcomes. Moreover, urge has an indirect effect on outcome through efficacy. When we reduce the model, however, so that efficacy represents the final common pathway to outcome (Fig. 2d), then the model fits nearly as well as the full model ($Q = 0.818; W(42) = 8.43; p > 0.05$) and we cannot reject this more parsimonious explanation. So we offer this as tentative support for a central role of efficacy expectations in mediating response to a high risk situation and outcomes. Parenthetically, note the failure of the associations between skill (or coping), anxiety and efficacy to reach significance (power may have been lacking due to the small sample size), although these were in the predicted direction.

**CSLT models: problems and prospects**

One problem, common to most of CSLT and related hybrid theoretical models, is determining the best methods for evaluation. The usual approach is to break things up into smaller pieces, and then to adapt the experimental or measurement paradigms that one is most comfortable with: for example, the laboratory-based cue exposure paradigm, or the real-world self-monitoring approach. It is fairly typical to concoct one’s own set of measures or adapt those already developed to fit the particular circumstances of the study at hand. Practicality deems this necessary, but it makes it difficult to integrate results across diverse studies. Is there a way to test a model such as ours in its entirety? Approaches such as covariance structure modeling bring us closer to a full test, but they do not capture the dynamic aspects of theoretical models, not only our own, but those such as Tiffany has also proposed.$^{21}$

Another problem has to do with specification of temporal sequencing. We are used to thinking in terms of simple cause and effect paradigms and in terms of linear relationships. It is difficult to evaluate models that incorporate feedback and feedforward pathways.

It would be useful at this point to comment on the status of what we know concerning association between various cognitive social learning elements in relation to drug cravings or urges. First, it appears that there is evidence to support the notion of an inverse relationship between efficacy and urges, although we cannot yet say which comes first.$^{23,25}$

Secondly, there appears to be an association between affect and urges, but there is controversy here. The evidence is weighing in heavily that there is an association between negative affective states and urges,$^{26–28}$ especially in studies that involve experimental manipulations of mood.$^{19,29}$ However, we should not yet be ready to disregard the role of positive affect. In a recent study reported by Tim Baker’s group, negative affect was found to be associated with smoking urges during deprivation, but positive mood was associated with urges when smokers were non-deprived.$^{30}$ Therefore, things may depend to a significant degree on how one looks at them, e.g. in the context of regular use or deprivation; during laboratory manipulations vs. assessment in the real world.

Thirdly, there appears to be some association between efficacy and affective response, but the precise nature and strength of this association is not known.

Fourthly, what is the relationship between outcome expectations and urge? Much has been written concerning the role of outcome expectations for drug effects in terms of their relationship with measures of drug use and treatment outcome.$^{9,31,32}$ There is still debate concerning the importance of specifying global vs. specific outcome expectations, and the dimensional structure of these expectations.$^{9}$ However, very few laboratory cue exposure studies have assessed outcome expectancies so that their relationships with urges and other measures of reactivity could be assessed. Indeed, we are guilty of that in our own studies.

Fifthly, similar to outcome expectancies for effects of drugs, there is a growing literature on the importance of coping with high-risk situa-
Distinctions have been made between behavioral and cognitive coping, and these have been tested for their relative importance in terms of preventing relapse. More sophisticated distinctions have also been made between initial coping with a high-risk situation versus so-called restorative coping after an initial lapse and probability of relapse. Very little is known, however, about relationships between coping and urges.

Where should we concentrate our research efforts? We should concentrate on defining more carefully what we mean by terms such as craving and urge. It is important to maintain a distinction between the concepts of being attracted to the idea of using a drug, and actual intention to use. If we are clear about what we understand these terms to mean, we can then be clear in making sure that our research subjects understand these terms and provide us with valid reports.

As noted before, we need to define and develop better ways to evaluate expectancies for effects of drugs to evaluate their ultimate motivational importance. The issue of common pathways to lapse or relapse deserves more attention, perhaps not to determine if there really is one but, rather, to determine if this is a blind alley for investigation. In terms of social learning, we must remember that much of drug use and drug relapse takes place in social contexts where there are other users. How important are factors such as observational learning, both in terms of risk and in terms of reducing risk for substance use? Cognitive manipulations also deserve more attention. For example, manipulating the expectancy that one will be able to use a drug in the context of a cue-reactivity experiment can have profound consequences on urge to use the drug but see also work by Sayette and colleagues. This has obvious implications in terms of understanding the importance of cognitive factors related to drug urges. Other studies have shown that actively suppressing the urge to use drugs has a rebound effect, increasing drug urges subsequently, compared to not suppressing urges.

Should we pursue investigations attempting to find physiological correlates of self-reported urges in human beings? To reiterate, if we focus on peripheral responses we will probably waste our time. However, recent data on the relationship of functional brain imaging to cocaine craving suggests that this type of approach may prove fruitful in our quest to understand the nature of craving.

Finally, if we are to make real and steady progress in understanding drug craving, its motivational significance, and ultimately what drives drug use and relapse, we should set up scientific task forces whose charge it is to integrate and construct newer and better theoretical models, with testable propositions, along with task forces whose charge it is to evaluate existing methodologies for investigating theories, and to suggest new approaches whenever warranted by the evidence.

Acknowledgments
This study was supported in part by US Public Health Service grant HL32318. I wish to thank David Abrams, William Shadel, Peter Monti and Damaris Rohsenow for their comments and insights.

References


