

Addiction Motivation Reformulated: An Affective Processing Model of Negative Reinforcement

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This article offers a reformulation of the negative reinforcement model of drug addiction and proposes that the escape and avoidance of negative affect is the prepotent motive for addictive drug use. The authors posit that negative affect is the motivational core of the withdrawal syndrome and argue that, through repeated cycles of drug use and withdrawal, addicted organisms learn to detect interoceptive cues of negative affect preconsciously. Thus, the motivational basis of much drug use is opaque and tends not to reflect cognitive control. When either stressors or abstinence causes negative affect to grow and enter consciousness, increasing negative affect biases information processing in ways that promote renewed drug administration. After explicating their model, the authors address previous critiques of negative reinforcement models in light of their reformulation and review predictions generated by their model.

Despite negative health, economic, social, and functional consequences, the addicted individual uses drug frequently and in large amounts and, if drug use is interrupted, will likely return to drug use. Why do individuals persist in taking addictive drugs in the face of the high ultimate costs? According to early models of drug motivation (e.g., Wikler, 1948), negative reinforcement is a key motive for drug use. That is, addicted individuals take drugs to escape or avoid aversive states such as withdrawal or stress. Interestingly, in contrast to these early views, much recent research suggests that negative reinforcement is not, in fact, a potent influence on drug motivation.

In the present article, we attempt to show that when properly construed, cardinal features of addiction¹ do indeed reflect the powerful motivational impacts of negative reinforcement. We attempt to accomplish this by reformulating negative reinforcement accounts such that escape or avoidance of *negative affect* is the principal motive for addictive drug use. Our reformulation has been guided by data that elucidate the nature of affective processing. We believe that the properties of affective processing not only allow us to account for

cardinal features of addictive behavior but, in addition, allow us to reconcile our negative reinforcement account with criticisms that have been lodged against such accounts.

Negative Reinforcement

Among motivational accounts of addiction, negative reinforcement ranks as one of the earliest and most venerable (Jellinek, 1960; Lindesmith, 1947; Wikler, 1948). For instance, half a century ago Wikler (1948) observed that addictive drugs such as opiates can produce physical dependence after a very small number of uses (see also Wikler, 1980). That is, after a few uses (perhaps after only a single use), falling levels of drug in the body precipitate an aversive withdrawal syndrome. Wikler proposed that the addicted organism takes the drug, primarily, to stave off or alleviate this syndrome. Moreover, Wikler believed that withdrawal can be elicited by means of associative mechanisms, which can account for relapses occurring long after discontinuation of drug use.

Other negative reinforcement theories have been proffered. For instance, Solomon's opponent-process model holds that the initial effects of addictive drugs are appetitive, but that these effects violate an affective homeostasis (Solomon, 1977; Solomon & Corbit, 1974). Therefore, intrinsic homeostatic mechanisms are recruited automatically in response to drug-induced euphoria or pleasure. These homeostatic mechanisms yield responses that counter appetitive drug effects, eventually neutralizing these effects (i.e., producing tolerance) and, when drug levels decline, persistent homeostatic responses will be unopposed, resulting in

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¹ We use the term *addiction* instead of the term *drug dependence*. We do this for several reasons. First, "dependence" has lost any meaning over and above "addiction." Both now refer to patterns of drug use that impose some significant cost on the individual; are difficult to interrupt; are likely to recur following interruption; and are characterized by tolerance, withdrawal symptoms, and so on. Use of the term *dependence* may lead to confusion with the more specific term *physical dependence*, a state in which reduced drug levels in the body elicit withdrawal symptoms.

frank, aversive withdrawal symptoms (see Siegel's, 1983, similar model of "compensatory responses" that emphasizes the role of associative processes in eliciting homeostatic adjustments). Like Wikler, Solomon concluded that, for the inveterate drug user, negative reinforcement is far and away the most potent motivational influence on drug use.

Numerous observations accord with the notion that the motivational basis of addictive drug use is the reduction or avoidance of aversive internal states. For instance, aversive drug-withdrawal symptoms are often associated with increases in addicted individuals' self-reported urges and intentions to take drugs (Baker, Morse, & Sherman, 1987; O'Brien, 1976; Wikler, 1980). In addition, addicted individuals rate coping with negative affect as the prepotent motive for drug use (Goldman, Brown, & Christiansen, 1987; Wetter et al., 1994). Despite such supporting observations and findings, over the past 10 to 15 years, negative reinforcement accounts of addiction motivation have fallen into disfavor.

Critiques of Negative Reinforcement

The decline in sway of negative reinforcement accounts can be attributed to two major factors: (a) Over the past 15 years, researchers have produced substantial evidence that supports alternative models of addiction motivation that focus on the appetitive (Stewart, de Wit, & Eikelboom, 1984; Stewart & Wise, 1992) or incentive-sensitization (T. E. Robinson & Berridge, 1993, 2003) impacts of drugs (particularly psychomotor stimulants) and (b) Researchers have noted or uncovered phenomena that appear anomalous or incongruent with negative reinforcement accounts.

With regard to the former, incentive models are supported by research showing that reinstatement of drug self-administration in animals is accomplished by exposing the animal to a small "priming" dose of the addictive drug (e.g., Stewart & Wise, 1992). A great deal of research suggests that, after self-administration of an addictive drug has been extinguished, exposure either to drug or to drug cues powerfully prods renewed self-administration (e.g., Stewart et al., 1984). Also, some evidence shows that addicted individuals may report higher levels of drug craving while taking a drug or immediately after taking a drug than they report when they are drug deprived (e.g., Meyer, 1988). In sum, there is substantial evidence that the direct actions of drugs have motivational potency independent of withdrawal relief.

Reviewers have also pointed to evidence suggesting that the motivational impacts of negative reinforcement are weak and unable to account for significant addiction phenomena. For instance, reviewers have claimed that addicted individuals often relapse when withdrawal symptoms should have abated (T. E. Robinson & Berridge, 1993) and have noted that addicted individuals rarely attribute relapse to frank signs of withdrawal (e.g., autonomic signs of withdrawal; McAuliffe, 1982). These observations seem incongruous with a model in which withdrawal misery serves as a principal setting event for self-administration.² Such evidence has led a recent reviewer to conclude, "In summary, then, the withdrawal-relief theory of opiate addiction championed by Wikler, and once so widely accepted, is clearly not supported by most recent evidence" (Lyvers, 1998, p. 113). T. E. Robinson and Berridge (1993) concluded, "Escape from distress cannot explain the defining characteristics of addiction, craving and relapse" (p.

252). Other recent reviews have echoed these sentiments (e.g., van Ree, Gerrits, & Vanderschuren, 1999).

We contend, however, that an integration of animal and human psychopharmacologic research with data and theory from cognitive and affective science supports a reformulated model of negative reinforcement. This model is an extension of Baker et al.'s (1987) earlier dual-valence model of drug motivation that integrated both positive and negative reinforcement processes. It is important to note that many of the current model's features are also predated in the drug motivation and affective-processing literatures. For instance, some features reflect ideas previously expressed by Wikler (1977) and Solomon (1977). In addition, we borrow heavily from Metcalfe and Mischel's (1999) characterization of "hot" and "cool" memory systems, and we incorporate Tiffany's (1990) notion that automatization is relevant to drug self-administration. Finally, although we stress the importance of negative reinforcement mechanisms in addiction motivation, we do not view such mechanisms as sole determinants of such motivation. We do believe, however, that recent accounts have inappropriately undervalued negative reinforcement influences on drug motivation. This has occurred, we believe, because theorists have sufficiently appreciated neither the affective basis of drug motivation nor the resulting implication: that addicted drug users sustain their drug use largely to manage their misery. In this article, we first sketch the model in broad strokes, without reference to the empirical literature. After introducing the major tenets of the model, we review supporting evidence and then account for findings that have been viewed as anomalous or conflicting with negative reinforcement accounts.

An Affective Model of Drug Motivation: A Synopsis

We believe that negative affect is the prototypic setting event for drug use and relapse in the addicted drug user. It becomes so because of the regularity with which withdrawal occurs following discontinuation of drug use and the rapidity and efficiency with which drug use ameliorates the aversive withdrawal syndrome.

Withdrawal symptoms arise very early in the course of addictive drug use, and their magnitude increases as addiction becomes more severe. Different addictive drugs produce different sorts of withdrawal syndromes, but negative affect is common to the withdrawal syndromes of all addictive agents. Negative affect is not only a universal element of withdrawal, but evidence suggests that it is also the motivationally prepotent element. That is, it indexes the aversiveness of the withdrawal syndrome, it prompts drug self-administration, and it is rapidly and efficiently relieved by drugs.

Drug absorption, distribution, and elimination kinetics produce innumerable spikes and troughs in drug levels in the body over the course of addictive drug use. This provides countless opportunities for the addicted individual to acquire a proceduralized drug motivational processing routine. Specifically, the addicted individual learns to detect the interoceptive (internal) cues of the negative

² Negative reinforcement models, including the present one, do accord some role to appetitive motivation. Solomon (1977), for instance, assumed that appetitive motivation was especially important early in the course of drug use and at the beginning of a binge (after b-processes producing withdrawal symptoms from a previous binge had abated).

affect that occur whenever drug levels begin to fall in the body. Early in the course of addiction, the individual learns to detect such cues when they are nascent and to respond with drug self-administration. Typically, the preconscious detection of affective cues biases response options and prompts drug-use routines. Therefore, during ongoing drug administration, the individual may be aware of wanting to take drugs and may be aware of the act of drug self-administration, but he or she is typically unaware of the motivational impetus. In sum, for the addicted individual with ready access to drugs, the motivational processing routine leading up to drug use tends to occur without awareness and occurs in the service of the escape and avoidance of negative affect caused by falling levels of the drug. See Figure 1.

The proceduralized motivational processing routine described earlier is not always effective in forestalling negative affect (e.g., sometimes drug is unavailable). Interruptions in drug use or the occurrence of significant stressors will both produce strong negative affect in the addicted individual. As negative affect mounts, it enters consciousness and influences information processing in ways that foster renewed drug use (see Figure 2). For instance, mounting negative affect tends to produce hot information processing, which biases attentional and response selection processes. Thus, the individual's attention tends to focus on perceived threats and negative affect per se. In addition, response biasing makes avoiding and/or escaping negative affect the primary motivational concern. The individual is biased toward response options that have most efficiently ameliorated negative affect in the past (i.e., drug use), and at very high levels of negative affect, the individual may execute such responses reflexively. In turn, other response options less tightly linked with reduction of negative affect are devalued.

Strong negative affect not only increases levels of hot information processing but also decreases the amount and influence of

cool information processing. This prevents cognitive control resources from being applied to the process of affective coping and regulation. Specifically, the influence of declarative knowledge is reduced and, with it, the ability to forestall immediate relief in favor of long-term benefit. When cool processing prevails, it is possible to interrupt prepotent motivational processing sequences and enact less well-trained, more effortful responses to situations that would normally elicit drug use. Thus, an implicit message of the model is that cognitive control resources are most likely to be applied at moderate levels of negative affect. At very low levels of negative affect, the individual is unaware of the affect and drug motivational processing is proceduralized (see Figure 1). The probability of awareness of negative affect increases in a roughly linear fashion with duration of withdrawal. Thus, at very high levels of negative affect, the individual is aware of his or her affect (see Figure 3), but the affect so increases the incentive value of drug use that drug self-administration occurs fairly reflexively (see Figure 2).

These processes do not exhaust the motivational influences that may influence a particular drug-use decision. For instance, the individual may be influenced by an awareness that he or she will not be able to use drugs for an extended period, by expectancies (e.g., that drug use may boost performance), or by social factors. However, we view these influences as modulators of addictive drug use, not as the fundamental motivational substrata that are responsible for the core features of addictive behavior as it occurs in nonhuman species as well as in humans. Moreover, to the extent that attitudes and expectancies influence decision making through controlled processing, their influence is most pronounced at moderate levels of negative affect. At very low levels of negative affect drug use tends to be proceduralized, and at very high levels of negative affect hot information processing precludes cognitive control.

Prototypic drug motivational processing in addiction at low levels of affect

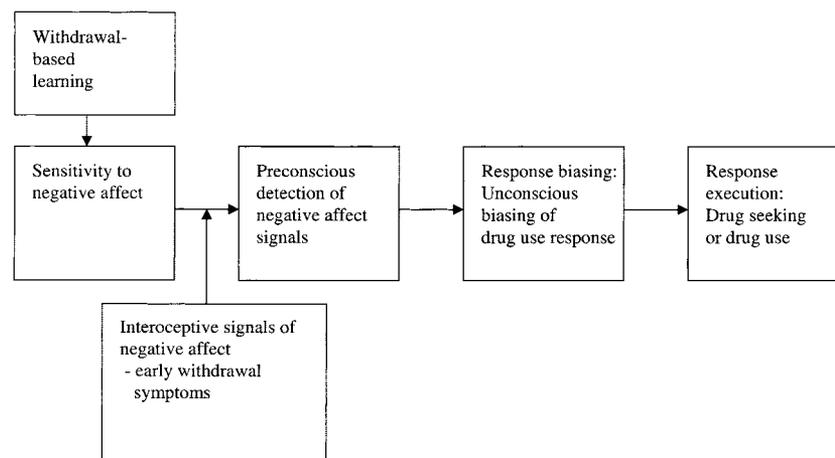


Figure 1. Through repeated withdrawal/drug-use cycles, the addicted individual becomes sensitive to external or internal stimuli that signal negative affect. At prototypic low levels of negative affect, the detection of these signals occurs outside conscious awareness and frequently results in the biasing of response options (i.e., primes previously reinforced operants, increasing the likelihood that these responses will be performed and will have increased hedonic tone).

Prototypic drug motivational processing in addiction at high levels of affect

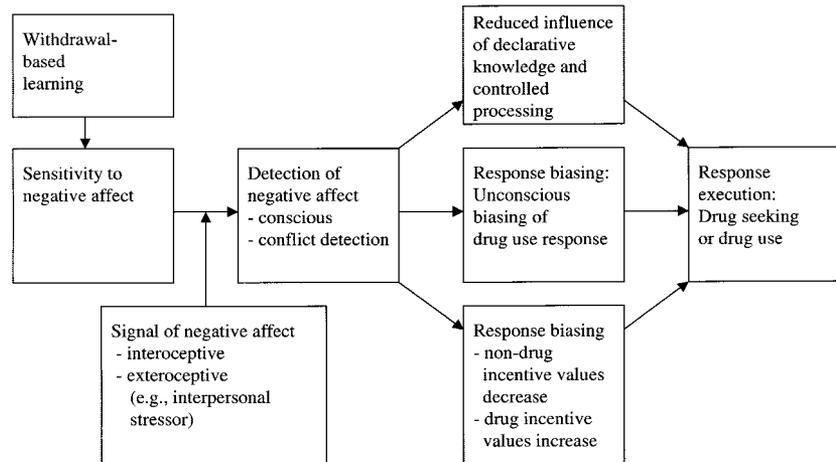


Figure 2. High levels of negative affect tend to produce awareness of the affect as well as to produce three types of response biasing. First, as described in Figure 1, previously reinforced responses can be primed and have increased hedonic tone. Second, the high levels of negative affect may shift the balance of the hot and cool information-processing systems in favor of the hot system, making well-learned, automatic affective-ameliorating responses more likely and reducing the influence of declarative knowledge and controlled cognitive processing. Third, strong negative affect will likely lead to a devaluing of alternative reinforcers (e.g., food) and an increase in the incentive value of a drug.

Our reformulated model of negative reinforcement yields several predictions regarding drug use by addicted individuals. For instance, in contrast to the conclusions of recent reviews (e.g., Lyvers, 1998; T. E. Robinson & Berridge, 2003; van Ree et al., 1999), we believe that a careful review of the relevant literature reveals the following:

Drug use, especially drug use that occurs after a significant period of interruption, tends to be reinitiated in the context of negative affect and stress. Thus, drug relapse tends to occur in the context of negative affect.

Withdrawal severity, and especially the affective elements of withdrawal, is an accurate index of relapse vulnerability.

Treatments that ameliorate the affective elements of the withdrawal syndrome are efficacious in reducing relapse to drug use.

The resumption of drug self-administration by animals is prompted not only by “priming” treatments in which animals are exposed to drug and drug cues but also by stressful events that produce internal states associated with negative affect.

Negative affect moderates the relation between cognitive control and drug use. Cognitive control resources are not recruited at low levels of negative affect, typically, and at very high levels of negative affect, such resources are relatively unable to influence drug outcomes.

We now turn to a more in-depth exposition of our model, present empirical support for its tenets, discuss the ways in which

our model fits with current data, and adumbrate novel predictions that it suggests.

Formative Influence of Withdrawal

We contend that (a) negative affect is a central component of the drug withdrawal syndrome; (b) among the elements of this syndrome, negative affect has the greatest motivational impact on drug use among addicted users; (c) physical dependence (and hence, the tendency to undergo withdrawal) starts early in the course of addiction; (d) withdrawal symptoms, including negative affect, can be detected on the basis of interoceptive cues; and (e) the contingencies between drug use and/or disuse and the aversive withdrawal symptoms are ideal for fostering learning by means of negative reinforcement. Evidence for these assertions is discussed in the following text.

Withdrawal: Universality of negative affect. All addictive drugs produce withdrawal syndromes, and these syndromes vary greatly across different drugs. For instance, whereas the alcohol withdrawal syndrome may involve tremor, auditory hallucinations, and convulsions and may be life threatening, the withdrawal syndromes produced by cocaine, opiate, or tobacco use are only unpleasant. Despite the heterogeneity in withdrawal symptoms across different drugs, all the syndromes have in common the fact that they comprise negative affect (manifest as specific emotions such as anxiety, irritability, or sadness, e.g., Gold, Washton, & Dackis, 1985; Kosman & Unna, 1968; Mansky, 1978). Therefore, if one were to postulate a universal element of withdrawal that constitutes the aversive setting event for further drug self-administration or relapse, it would be negative affect.

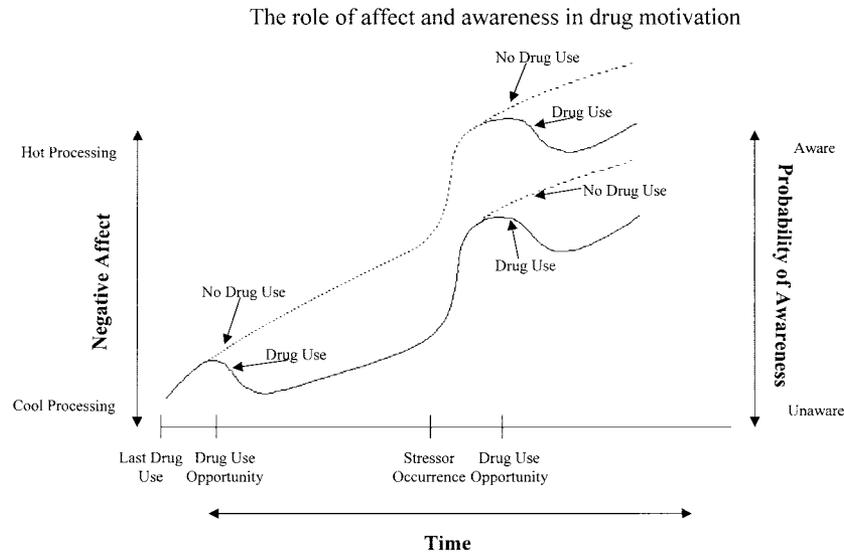


Figure 3. The horizontal axis represents time since last drug use, and the vertical axis represents intensity of the affective response. Affect increases in direct proportion to the amount of time since last drug use. As affect grows, the probability of the affect being consciously available grows as well. Also, as the affect escalates, information processing begins to be dominated by the hot system rather than the cool system. If the drug is used optimally, nascent negative affect will be quelled before it becomes available to consciousness. If drug use is impeded at this point, however, affect may become conscious, and the addicted individual may be aware that negative affect decreases following renewed drug use. Negative affect spurred by exteroceptive stressors can become conscious as well, and may be relieved by drug use.

Negative affect: A readout of withdrawal-based motivation. A variety of evidence points to negative affect as being the motivationally prepotent element of the withdrawal syndrome. Although nonaffective signs of withdrawal (e.g., tremor) are sometimes cited by physically dependent individuals as precipitating drug use or relapse (e.g., O'Brien, 1976; Rankin, Stockwell, & Hodgson, 1982; Wikler, 1977), addicted individuals more frequently cite negative affect as a crucial setting event for drug use or relapse (e.g., Brandon, Tiffany, Obremski, & Baker, 1990; Marlatt & Gordon, 1980).

Studies that have explicitly contrasted affective and nonaffective withdrawal signs and symptoms on their ability to model relapse vulnerability have shown affective symptoms to yield superior predictions (e.g., Kenford et al., 2002; Piasecki et al., 2000). This is consistent with the vast body of information that shows that measures of negative affect, and the tendency to use drugs in response to negative affect, sensitively index a variety of outcomes relevant to addiction motivation, such as self-administration, relapse, and urge self-reports (e.g., Brandon, 1994; Holahan, Moos, Holahan, Cronkite, & Randall, 2001; Shiffman, Paty, Gnys, Kassel, & Hickcox, 1996). These data are congruent with Solomon's (1977) notion that *affective* responses should index drug motivational processing.

The centrality of negative affect in drug motivation is also supported by basic psychopharmacologic research with animals that suggests that the somatic signs of withdrawal are not central to the aversive quality of withdrawal. For instance, this research shows that the nonaffective, somatic signs of withdrawal (e.g., diarrhea, shaking, writhing) are not responsible for the place aversions acquired when a distinctive context is paired with opiate

withdrawal (Hand, Koob, Steinus, & LeMoal, 1988; Mucha, 1987). In addition, brain research has shown that the amygdala, a crucial component of the affective processing system (LeDoux, 1996), seems to be necessarily involved in the aversive aspects of opiate withdrawal (Kelsey & Arnold, 1994; van Ree et al., 1999). The involvement of the amygdala in both withdrawal and negative affect supports the supposition that the motivational components of withdrawal have an affective basis. Both the human and animal research reviewed previously suggest that negative affect may be the best synthetic "readout" of withdrawal aversiveness. Our contention is that withdrawal symptoms such as rhinorrhea, lacrimation, and myoclonus exert relatively little impact on drug motivation as long as the addicted individual is content.

Physical dependence begins early in the course of addiction. If the relief of withdrawal-induced negative affect serves as a formative experience in the development of addiction, the capacity to experience withdrawal must occur early in the course of addictive disorders. There is copious evidence that physical dependence, the potential to undergo withdrawal upon drug abstinence, arises very early in the course of addictive drug use. Both human and animal laboratory research has shown that withdrawal symptoms and signs can be observed in organisms after just a handful of drug exposures (e.g., Heischman, Stitzer, Bigelow, & Liebson, 1989). The early onset of physical dependence is also suggested by field research on clinical populations, which has shown that most of those who regularly use addictive drugs report withdrawal symptoms quite early in their drug-use careers (e.g., Centers for Disease Control and Prevention, 1999; Hesselbrock, Segal, & Hesselbrock, 2000). Thus, although social context and incentive properties of

drugs may motivate early drug use, over the course of addiction, the management of withdrawal becomes an omnipresent concern.

Signals of incipient withdrawal-induced negative affect are detected interoceptively. Because the addicted organism experiences repeated bouts of withdrawal exacerbations on a daily basis, withdrawal can become associated with external cues and contexts (O'Brien, 1976; Wikler, 1965). However, we contend that this sort of external association is frequently overshadowed by *interoceptive* signals, which have a superior contingency with the affective components of the withdrawal syndrome. Indeed, early effects of falling levels of a drug in the body are ideally suited to signal the incipient growth of the affective elements of the withdrawal syndrome. The consequences of falling drug levels need not themselves be formally classified as substrata of either withdrawal or affective responses. For instance, in the case of tobacco smoking, inhaled smoke produces a "throat scratch" sensation and paralysis of pulmonary cilia (e.g., Rose, Westman, Behm, Johnson, & Goldberg, 1999). Lessening of either in response to falling blood nicotine levels could signal increases in withdrawal and/or negative affect. The key is that these consequences reflect falling drug levels and, therefore, signal negative affect.

A sizable literature attests to the ability of interoceptive cues to function as conditioned stimuli (CSs). Razran (1961) noted that learning based on interoceptive CSs is particularly durable (e.g., resistant to extinction). More recently, Lal and his colleagues (e.g., Spencer, Yaden, & Lal, 1988) have shown that animals can use internal cues of hypotension as discriminative stimuli that guide instrumental behaviors. Observations of organisms' exquisite sensitivity to interoceptive cues, and the capacity of such cues to signal internal events, lend support to the notion that internal events may signal negative affects and may also serve as discriminative stimuli for instrumental behaviors such as drug self-administration (e.g., Spencer et al., 1988). There is also evidence in humans that panic disorder patients are sensitive to chemoreceptor signals (e.g., sensitivity to alterations in blood gases) or other subtle physical cues of incipient fear (Barlow, Chorpita, & Torovsky, 1996; Bouton, Mineka, & Barlow, 2001).

The contention that organisms can detect and act on interoceptive cues has received direct support from drug-discrimination research that showed that animals can use internal cues of withdrawal to guide instrumental responding (Gauvin, Carl, Goulden, & Holloway, 1993). In drug-discrimination training, organisms are trained to emit one instrumental response while under one drug state and to emit a different instrumental response under a different drug state (or a placebo condition). The only cues available to the organism to guide its responding are the interoceptive cues produced by the drug or drug state. Both the direct effects of addictive drugs and their withdrawal syndromes serve as effective drug-discrimination cues (Gauvin et al., 1993; Gauvin, Harland, & Holloway, 1989).

One may make inferences about the subjective or phenomenologic quality of the withdrawal state on the basis of the agents or drugs that substitute for it (i.e., elicit the same instrumental response as does withdrawal). For example, if an animal is trained to press a lever in response to withdrawal, then that animal will also press that same lever when given an anxiogenic drug or exposed to a nonpharmacologic stressor such as a cat or shock (Gauvin et al., 1993). In addition, drugs that produce elevated anxiety reports in humans (e.g., pentylenetetrazole, FG7142, DMCM, yohimbine)

readily substitute for withdrawal states produced by a variety of addictive agents (Gauvin et al., 1993; Wood, Laraby, & Lal, 1989). This body of work suggests a stimulus equivalence between the interoceptive cues of negative affect and withdrawal.

Our model holds not only that animals learn to detect withdrawal signals interoceptively but also that they detect and respond to inklings of negative affect or withdrawal when such signals are unavailable to conscious awareness. Evidence shows that a fledging response can indeed effectively signal subsequent growth in that same response. Siegel and his colleagues (Kim, Siegel, & Patenall, 1999; Sokolowska, Siegel, & Kim, 2002) have recently compared interoceptive and exteroceptive cues on their ability to serve as effective CSs for a drug unconditioned stimulus (US). Kim et al. (1999) found that when a drug (morphine) was infused quickly (14–17 s), an infusion-paired exteroceptive cue (a context) served as an effective CS (i.e., one capable of eliciting associative drug responses). However, when drug was infused *slowly* (over 25–30 min), the exteroceptive cue was an ineffective signal; animals appeared to show associative drug effects without a contingent exteroceptive cue. Presumably, slow infusion produced sufficiently salient internal cues and cues that emerged gradually enough to possess good signaling properties, so that exteroceptive cues were rendered redundant and, hence, were overshadowed. This research suggests the following: (a) that initial effects of an internal reaction can effectively signal the later elements of that same reaction (termed *homoreflexive associations* by Dworkin, 1993), (b) that interoceptive cues may overshadow contingent exteroceptive cues, and (c) that gradually emergent internal events are more likely to yield homoreflexive associations than are precipitous events. These features support the plausibility of the internal cueing of withdrawal elements such as affective responses.

Not only does it appear likely that homoreflexive associations are ubiquitous (Dworkin, 1993), but also it appears likely that exteroceptive cues are *not* strongly contingent with withdrawal exacerbation in the addicted individual (because few exteroceptive cues are uniquely contingent with falling drug levels). Moreover, withdrawal symptoms are gradually emergent, with full-blown withdrawal not manifest for hours after drug abstinence (e.g., Mansky, 1978), which may further compromise contingencies with exteroceptive cues.³ In summary, research suggests that incipient growth in negative affect may be signaled by the first inklings of negative affect responses per se (e.g., responses producing anxiety) or by any internal response to falling drug levels that is a reliable harbinger of negative affect (e.g., mild tremor).

Contingencies between drug use and withdrawal. Contingencies between drug use or misuse and withdrawal are ideal for producing behavior change by means of negative reinforcement. First, withdrawal-induced negative affect is produced by *declines* of the drug in the body (presumably at central nervous system and

³ It is possible that exteroceptive or interoceptive cues paired with drug delivery elicit compensatory responses, as suggested by Siegel (1983), and that these manifest as withdrawal symptoms. Such associations could interfere with the association of interoceptive cues with withdrawal onset. However, as Kim et al.'s (1999) research suggests, we believe that because of temporal contiguity and perhaps because of "belongingness" (Öhman & Mineka, 2001), the initial manifestations of withdrawal and negative affect are the most informative signal of exacerbations in those states.

peripheral sites of action), not necessarily by the *absence* of drug. Thus, withdrawal symptoms may occur even when drug levels in the body are quite high—just so long as they have declined from a higher level (e.g., Isbell, Fraser, Wikler, Belleville, & Eisenman, 1955; Mello & Mendelson, 1970). This means that the physically dependent drug user experiences numerous bouts of withdrawal-induced negative affect every day despite having significant levels of a drug in the body. Thus, each drug-taking episode produces a rise and fall in drug levels (Parrott, 1999; Wikler, 1977), and negative affect is entrained to these cycles.

The periodicity of the affective sine wave precipitated by withdrawal and its relief naturally varies as a function of such factors as the metabolic and distributional half-lives of the drug (e.g., Adler & Geller, 1984). A basic property of the affective manifestations of the withdrawal syndrome, however, is that there be a roughly linear relation between the duration of drug abstinence and the magnitude of affective disturbance, at least over the first minutes, hours, and days of withdrawal (see Figure 3). Indeed, considerable research in different addicted populations has shown a fairly linear growth in withdrawal symptoms that starts minutes after discontinuation of a drug and builds steadily over days (e.g., Jarvik et al., 2000; Mansky, 1978; Schuh & Stützer, 1995; Zinser, Baker, Sherman, & Cannon, 1992). For instance, Jarvik et al. (2000) reported a tight temporal association between declining blood levels of nicotine and escalation in levels of the self-reported withdrawal symptom of craving. Moreover, psychophysiological indices of affective processing also link falling drug levels and withdrawal with increased negative affectivity (e.g., Fendt & Mucha, 2001).

The reverse side of the contingency between drug use and withdrawal is that addictive drugs rapidly and effectively alleviate the negative affect engendered by withdrawal.⁴ It is important to note that we are not simply asserting that addicted individuals believe that drug use reduces negative affect, although we do agree that such beliefs and expectations do influence drug-use decisions (Goldman et al., 1987; Wetter et al., 1994). Rather, numerous studies (Mello & Mendelson, 1970; Parrott, 1999; Wikler, 1977, 1980; Zinser et al., 1992) have shown that addictive agents very quickly and efficiently reduce negative affect along with other elements of the withdrawal syndrome.

Basic Research in Cognitive and Affective Processing: Implications for Addiction

Basic research on cognitive and affective processing supports the detection and processing of affective cues outside of conscious awareness. This research suggests that affect detected without conscious awareness may influence affective processing. The “mere exposure” effect (Zajonc, 1968) refers to the fact that simple exposure to a stimulus (e.g., a shape, photograph, nonsense word) reliably affects individuals’ liking or positive affective reactions to that stimulus. Remarkably, stimulus exposures too brief to produce conscious recognition reliably enhance individuals’ affective reactions to the stimulus (e.g., Murphy, Monahan, & Zajonc, 1995). Moreover, some evidence has shown that mere exposure effects are stronger when exposure latencies are brief, defying conscious awareness, than they are when they are prolonged (i.e., permitting conscious awareness; Bornstein & D’Agostino, 1992).

The results of mere exposure, priming, and neuropsychological research support an information-processing model in which affective information is afforded priority in the stimulus evaluation or information-processing cascade (see Murphy & Zajonc, 1993; Öhman & Mineka, 2001). Moreover, affective primes do not appear to convey significant information about specific emotions but rather to impart effects consistent with the broad hedonic tone of preconscious primes (Murphy et al., 1995). Later stages of information processing incorporate nonaffective information into processing, and this may blunt or dilute the impact of the affective signal. In sum, there is a wealth of evidence that “people classify their experience as either good or bad and do so immediately, unintentionally, and without awareness that they are doing it” (Bargh & Chartrand, 1999, p. 474).

In addition to influencing affective processing, there are experimental and neuropsychological data (e.g., Bargh, Gollwitzer, Lee-Chai, Bardollar, & Roman, 2001; Bechara, Damasio, Tranel, & Damasio, 1997; Damasio, 1994) attesting to the impact of affect on behavior and decision making when the individual is unaware of either the affective prod per se or its contingency with behavior. Research findings also indicate that brief, preconscious exposures seem to affect behavior particularly when the exposure involves *affectively* tinged information (Murphy & Zajonc, 1993; and preconscious influences are not restricted to self-report measures; Öhman & Mineka, 2001). These findings are consistent with evidence that unconscious appraisal processes are especially focused on affectively valenced stimuli or events (Öhman & Mineka, 2001). Taken together, this body of research demonstrates that unconsciously processed information not only affects the hedonic evaluation of a cue but also influences motivated behavior—including motivated behavior that is less automatic than is drug self-administration for the inveterate drug user (e.g., arranging letter tiles into words; Bargh et al., 2001).

The foregoing research and theorizing may have significant implications for drug motivation. First, it suggests that affective cues may have effects on preferences, attitudes, and behaviors despite the fact that the affective prod occurs outside of awareness (see Figure 1). Second, it suggests that the first preconscious inklings of negative affect exert effects that are largely restricted to hedonic valence per se. Third, it suggests that humans’ information-processing systems are specifically organized so as to extract immediately, affective information from stimuli and stimulus arrays. These findings are consistent with the notion that addicted individuals learn, through iterative trials, to extract immediately the affective meaning of inchoate interoceptive cues and then dampen them by drug use. Unless drug use fails to occur, all this may transpire with little or no conscious awareness (see Figure 3).

We recognize, however, that not all affective influences on drug use occur outside of awareness. Drug doses are often separated by lengthy intervals causing sustained and noticeable withdrawal symptoms (e.g., after overnight abstinence); this is, in fact, responsible for addicted individuals’ nearly universal awareness of the contingency between drug use and diminished negative affect

⁴ To the extent that addiction yields chronic elevations of negative affect (see, Parrott, 1999; Solomon, 1977; Wikler, 1980), drug use may produce *reductions* in negative affect, but not an absence of negative affect.

(e.g., Goldman et al., 1987; Wetter et al., 1994). Therefore, we acknowledge that addicted individuals are often aware of negative affect that cues drug use. However, we believe the foregoing evidence supports the plausibility of addicted individuals learning to detect and respond to negative affect in an efficient manner not requiring the allocation of significant cognitive resources or mental workspace.

A variety of processing mechanisms might underlie the exquisite sensitivity of addicted organisms to affective signals. In a network model, for example, the heightened reactivity to relevant affect nodes could be attributed to chronically high levels of activation caused by their repeated stimulation (Metcalf & Jacobs, 1998; Metcalf & Mischel, 1999). This high level of chronic activation would decrease the threshold for future activation based on similar inputs. It is also possible that the countless episodes in which internal cues signal worsening affect foster the acquisition of automatic interoceptive cue-search strategies (e.g., M. D. Robinson, 1998).

Responding to Negative Affect Arising From Nonpharmacologic Sources

We believe that a crucial formative experience for addicted individuals is their learning to respond to withdrawal-induced negative affect with drug self-administration. However, over the course of the development of addiction, the organism also learns to respond to nonpharmacologic stressors with responses indicative of heightened drug motivation. Thus, stressors (e.g., arguments, work stress, time pressure) and negative affect arising from these nonpharmacologic sources tend to spur self-administration and relapse, prompt craving, and so on (e.g., Drobles & Tiffany, 1997; Marlatt & Gordon, 1980). Additionally, not only external stressors but also internal evaluative processes such as negative self-appraisals that increase negative affect may lead to increased drug motivation (Baumeister, Heatherton, & Tice, 1994). What supports the association between nonpharmacologic stress and drug use?

One possibility is that addictive drugs actually do quell negative affects arising from nonpharmacologic sources. There is certainly evidence that drugs can reduce some stress effects (Curtin, Lang, Patrick, & Stritzke, 1998; Sayette, Martin, Perrott, Wertz, & Hufford, 2001). However, there is also evidence that use of an addictive drug does not consistently ameliorate stress-induced distress or negative affect (e.g., Kassel, Stroud, & Paronis, 2003). We believe that the weight of evidence indicates that drugs can alleviate negative affects caused by (nonpharmacologic) stressful events, but only under certain conditions. Stress relief, at least in some cases, appears to depend on factors such as the timing of drug use, the match between the type of negative affect and the drug, and the environmental and cognitive context of drug use (e.g., there is evidence of greater stress relief due to alcohol use when alcohol is administered concomitant with a task or event that constrains mental work space and when it displaces worry or rumination; Curtin et al., 1998; Steele & Josephs, 1988). It is important to note that we do not assume that the amelioration of stress-induced negative affect, when it does occur, occurs by the same mechanisms that reduce withdrawal-induced negative affect.

Despite an inconsistency in stress reduction, stressors do become effective motivational prods for the addicted individual (Brandon et al., 1990; Kassel et al., 2003; Marlatt & Gordon, 1980;

Shiffman et al., 1996). We believe that this occurs not only because the drug can, under some circumstances, alleviate non-pharmacologic negative affect, but also because as the drug discrimination research previously discussed (Gauvin et al., 1993; Wood et al., 1989) shows, animals readily generalize across withdrawal, on the one hand, and negative affective states elicited by stressors, on the other. Thus, animal research suggests that the internal cues produced by withdrawal and those produced by stressful events are similar and, therefore, either can serve as control stimuli for drug self-administration. This supposition is consistent with basic animal research that shows that instrumental responses may be discriminated not on the external event (US) that gives rise to them, but instead on the affective state elicited. For example, an animal trained to emit a particular response to avoid or escape shock will spontaneously use that same response to avoid or escape a different sort of aversive stimulus (e.g., a loud noise generated by a klaxon; Mineka, 1985). Such generalization may be especially likely in addiction where there is no salient, external cause of withdrawal distress.

A final reason that stressors may motivate drug use is that the addicted organism is so frequently in a state of mild or moderate withdrawal that a stressor and withdrawal may summate to produce a particularly high level of negative affect. That is, stress effects are likely often superimposed on withdrawal effects because addicted individuals experience iterative withdrawal/drug-use cycles. In such circumstances, drug use yields a net reduction in negative affect even if the effect is primarily due to withdrawal relief.

To review briefly: We believe that the addicted organism shows heightened drug motivation in response to stressors, just as it does to withdrawal because (a) drugs can ameliorate stress effects, under certain conditions; (b) there is generalization between negative affect associated with drug withdrawal and the affective reactions to stressors; and (c) negative affect induced by stress and withdrawal may sum to occasion negative reinforcement by means of drug use.

Signaled Escape and Avoidance: The Look of Automatized Drug Motivational Processing

In this section, we discuss a behavioral phenomenon that we believe exemplifies the sort of proceduralized motivational process that we are invoking and therefore supports the plausibility of our suppositions. If negative reinforcement plays a strong causal role in the acquisition and maintenance of addictive behavior, then should not the addicted individual appear perpetually fearful, depressed, and/or angry? Should not we observe strong correlations between negative affect and the instrumental response that results in escape or avoidance (drug self-administration), as critics of negative reinforcement models suggest (e.g., Jaffe, 1992; Lyvers, 1998)?

These questions led us to examine behavior that is maintained solely on the basis of negative reinforcement. It is important to note that we are invoking signaled avoidance learning as a primary motivational process to account for addictive behavior. We use signaled avoidance learning as a model because we posit that nascent withdrawal cues or other interoceptive events associated with them serve as signals. Signaled avoidance learning has typically been studied by presenting an organism with a signal (CS)

that is followed by shock or some other aversive stimulus (US), unless the organism emits an avoidance response. In the present account of addiction, the CS might be cues of falling levels of drug, (e.g., nonaffective withdrawal signs or symptoms, or nascent negative affect per se), and the US is severe negative affect. The avoidance response is, of course, drug intake.

Much of the argument that follows could also be derived from studies of Sidman avoidance learning in which no explicit CSs are presented (Sidman, 1966). Rather, shocks or some other aversive USs occur at regular intervals, which the organism can avoid by making avoidance responses at a rate that exceeds the intershock interval. Here, the passage of time is the CS. We believe that the passage of time may also constitute an effective CS for drug users, but one that is typically overshadowed by signals of negative affect or withdrawal.

One intriguing observation about avoidance performance is that, despite the fact that negative reinforcement is indisputably the causal mechanism, the well-trained animal appears to perform the avoidance response fearlessly. The animal appears “nonchalant” (Mineka, 1985). In fact, tests designed to gauge the animal’s fear of the CS reveal that animals show decreased fear of the CS over conditioning trials (e.g., Cook, Mineka, & Trumble, 1987; Starr & Mineka, 1977). In addition, there is desynchrony between physiological indicants of fear processing and avoidance responding (e.g., Black, 1959; see also, Rachman & Hodgson, 1974). Thus, while avoidance and/or escape from an aversive US (or fear engendered by the US) is the sole ultimate precipitant of avoidance behavior, the organism’s apparently fearless behavior belies this. The organism is somewhat fearful (no one has demonstrated avoidance responding in the absence of fear), but the organism does not seem fearful. In fact, if one could productively ask the well-trained animal whether it is fearful, our surmise is that it would say “no”—just as the well-trained addicted individual would similarly deny significant negative affect between routine drug doses (e.g., Shiffman et al., 2002). This denial would hold just so long as the effective avoidance response was available.

There is not complete consensus on the factors that are responsible for the apparent desynchrony between avoidance performance and fear, but considerable evidence implicates feedback that the organism gains through performance of the avoidance response. In a typical avoidance paradigm, the organism performs the avoidance response, and this not only delays the aversive US but also terminates the CS (the “warning stimulus”). Research has suggested that termination of the CS provides feedback that is largely responsible for the decrease in fear that accrues over the course of avoidance performance (e.g., Cook et al., 1987). Similarly, in escape paradigms, fear of an aversive US is reduced to the extent that the organism has control over US duration (Mineka, Cook, & Miller, 1984). Therefore, in the context of addiction, we would expect to see little evidence of negative affect in the freely using addicted individual, to the extent that drug use reduces or avoids the US (exacerbated negative affect) and attenuates or eliminates the CS (signals of falling levels of drug, etc.). Moreover, one would expect to see no, or only modest, relations between overt negative affect and drug use measures to the extent that drug use is available (e.g., Zinser et al., 1992). In fact, the sensations of direct drug actions (e.g., feelings of intoxication) would serve as ideal “safety signals,” indicating that the avoidance response was efficacious in quelling negative affect. Thus, direct

drug effects may be reinforcing (as conditioned inhibitors) in that they signal a period of relatively low negative affect.

Although ongoing avoidance performance may mask the causal relation between fear and the instrumental response, response prevention is a very effective way to unmask this relation. That is, failure to perform the avoidance response, or its unavailability, significantly increases the animal’s fear and fear of the CS (e.g., Solomon, Kamin, & Wynne, 1953). This suggests that associations between negative affect and drug use and motivation may be minimal until the addicted individual loses access to drug. Then, relations between drug use/motivation and negative affectivity should become more apparent. For example, studies with smokers have shown that drug withdrawal increases the concordance between negative affect and drug self-administration and drug urges (Sayette et al., 2003; Zinser et al., 1992).

One other important characteristic of avoidance behavior is its remarkable resilience—its refractoriness to extinction (Solomon et al., 1953). This seems to be a function of the fact that during the typical extinction paradigm, the avoidance response continues to terminate the CS, and the US no longer occurs (in keeping with the extinction manipulation; Mineka, 1979). Even toxic effects of drug that may arise from long-term use might not effectively discourage continued drug use. An extensive literature shows paradoxical impacts of punishment of an avoidance response (i.e., punishment of a well-entrenched avoidance response may actually lead to a vicious cycle in which the avoidance response is strengthened rather than weakened; Mineka, 1985). This may occur because punishment of the avoidance response increases negative affect and fear and this, in turn, increases the likelihood that the organism will emit the overlearned avoidance response.

The avoidance response is not only slow to extinguish but also extremely susceptible to reinstatement once extinguished. In support of this, Bouton (2000) and others have shown that conditioned fear can easily be renewed in response to subtle environmental change. The relevance of this to addiction is that the return of avoidance responding (i.e., relapse to drug use) would be a common, if not expected, consequence if the organism (the addicted individual) was exposed to environmental change after cues for drug use had been extinguished. Even if an addicted individual has learned not to respond to affective signals in an environment, subtle change in that environment could renew their motivational value and foster resumption of drug use.

Selection and Execution of the Proceduralized Avoidance and Escape Response

We have not discussed at length the automatization or proceduralization of the addicted individual’s drug-use behaviors. However, we agree with Tiffany’s (1990) assertion that, like other stereotypic motor acts, some aspects of the drug-use ritual are susceptible to automatization. That is, the addicted individual may use a drug in such a manner that the supportive information processing does not demand significant working-memory resources, can occur without awareness, and may be difficult to modify or regulate (see Jastrow, 1906; Shiffrin & Schneider, 1977). However, we do not view the automatization of the drug-use ritual per se to be the most interesting or significant aspect of self-administration, at least from a motivational perspective. We believe that in the vast majority of cases the addicted individual is

aware to a great degree of “deciding” to use drug and of various components of the self-administration ritual; this is, no doubt, especially true of elaborate drug-use rituals that involve “cooking-up” drugs, tying off veins, and so on. Rather, it is the automatization of the information processing that precedes self-administration decisions (referred to by Tiffany, 1990, as “stimulus configurations necessary for the elicitation of component action,” p. 154) that accounts for the seeming anomalies in addictive drug use. It is the unconscious impact of negative affect that accounts for apparent incongruities such as taking a drug without noticeable urge, discomfort, or even intention to do so (contrast with T. E. Robinson & Berridge, 2003; Tiffany, 1990). Moreover, it is clear that the automatization of the drug-use ritual and its information-processing precursors can be separate phenomena. Individuals may certainly be aware of engaging in an instrumental act but unaware of its motivational origins (Bargh & Chartrand, 1999).

Thus far, we have focused on the fact that signals of negative affect arising out of stressors or withdrawal set the stage for continued addictive drug use. However, affective responses may influence drug-use in another way. The prior history of negative reinforcement may impart a positive affective valence on drug cues and the drug use ritual. Just as is the case with negative affect, such positive affective responses may influence instrumental drug-use behaviors outside of conscious awareness. Consistent with this construction, there is evidence that drug users mount anticipatory affective responses prior to drug use opportunities. For instance, smokers show significantly increased relative activation of the left-frontal cortices and significantly increased suppression of acoustically elicited startle, reflecting increased positive affect, when anticipating a drug-use opportunity (e.g., Zinser, Fiore, Davidson, & Baker, 1999; cf. Breiter et al., 1997). Moreover, such anticipatory responses are greatest in the presence of withdrawal-induced negative affect—when the potential for negative reinforcement is optimal. In essence, such positive affective responses may reflect the “safety signaling” properties of drug cues. Such responses may also arise from appetitive or incentive effects of drug (Stewart et al., 1984). Our contention is that such affective signaling may guide drug-use decision making on a preconscious basis (Bechara et al., 1997).

In summary, in the great majority of cases the drug user is aware that he or she is tying off a vein or picking up a glass of alcohol. However, the likelihood of drug use is guided by affective reactions that influence the incentive value of such behavior, and these reactions typically occur beyond awareness. Thus, the act of drug use is typically transparent, whereas its affective prods are not.

When Affect Grows

It is easy to imagine how the addicted individual may become trapped in a cycle of withdrawal and self-administration when unconscious instigators trigger and guide automatic drug-use behaviors. However, it is clear that addicted individuals are often aware that they are experiencing stress or negative affect and are aware that they want to take a drug to alleviate this distress (e.g., Brandon, 1994). At first blush, it seems that an addicted individual who is motivated to cease drug use would be more likely to quit successfully if the provocation for use were to become known or conscious. For instance, if the addicted individual realizes that he

or she is experiencing negative affect, that individual could use higher level cognitive control resources to problem solve and identify an alternative affective coping strategy. Thus, if negative affect becomes severe and noticeable, because of abstinence or a stressor, this should draw the addicted individual’s attention to the need for affective coping and permit controlled, planful information processing directed to that end. However, data suggest that addicted individuals are often unlikely to refrain from drug use when they are aware of their negative affect, especially when it is severe (e.g., Brandon et al., 1990; Shiffman et al., 1996). One reason for this is that addicted individuals may have an impoverished array of alternative coping responses, perhaps because of an overreliance on drug use as a way to influence affect. In addition, severe negative affect may influence information processing in ways that further promote addictive drug use.

The Nature of Emotional or Hot Information Processing

Affects can be viewed as response or action tendencies (e.g., Lang, Bradley, & Cuthbert, 1990; Öhman & Mineka, 2001). Therefore, the occurrence of a strong affective response has profound implications for the types of responses that an organism is likely to emit. Among the response processes influenced by affect are those involving attentional and stimulus appraisal responses; the fearful organism searches the environment for signs of threat, the angry or hostile organism seeks an opportunity to aggress.

Recent reviews of cognitive neuroscience research on stress and emotions (e.g., Metcalfe & Jacobs, 1998) have provided insights into how emotions may influence both attentional processes as well as dispositions to respond. These reviews indicate that “emotional” and “nonemotional” information processing are qualitatively different. The distinction between emotional (hot) and nonemotional (cool) information processing could have important implications for addiction (Metcalfe & Mischel, 1999).

Evidence implicates the hippocampus in a cool memory- and information-processing system. The cool system predominates when the organism operates close to an affectively neutral state. It supports processing of spatial-temporal features that constitute the characteristic elements of episodic memories, it supports integration of declarative knowledge, and it may facilitate generation of a narrative story line regarding a remembered event. It is cognitive, integrative, and reflective. Conversely, high levels of stress and associated negative affect activate a hot information-processing system that involves structures such as the locus coeruleus and the amygdala and is implicated in conditioning and implicit memory.

The hot system has features of special relevance to addiction. For instance, there is some encapsulation of hot, or affective, memories (see Öhman & Mineka, 2001). That is, hot processing is not readily modifiable by declarative knowledge that may provide a broader interpretive context, allow for nuanced evaluation, or foster a long-term perspective. Hot information processing tends to be bottom-up in nature and relatively uninformed by preexisting declarative knowledge (Schwarz, 2000).

Attentional and Response Biasing: Impact of Hot Emotional Processing

One key feature of the hot processing system is a strong attentional bias that directs attention to stimuli or events that are

associated with stress or emotional activation (the association may be intrinsic or acquired). Thus, in the context of hot processing, attentional processes are narrowly directed at cues such as threat cues, startling events, or cues that signal these (e.g., Metcalfe & Jacobs, 1998; Yee & Miller, 1994). The impact of negative affect on attention allocation may produce reciprocal effects such that an individual cannot disengage his/her attention from distressing material (e.g., a stressor or from mounting craving/dysphoria itself), and this may foster greater subsequent negative affect (Compton, 2000; Derryberry & Reed, 2002).

Strong negative affect also biases processing of response options, promoting either intrinsic response dispositions or previously rewarded responses (see Figure 2). As an example of an intrinsic, genetically determined, response bias, mammalian organisms are primed by negative affect to respond to startling stimuli with an exaggerated motor burst (e.g., Lang et al., 1990). However, high levels of negative affect also, no doubt, foster associative processes that permit the organism to deal with certain sorts of perceived threats. For instance, there is evidence that adrenergic agonists that activate the amygdala facilitate the acquisition of fear avoidance and escape responding (e.g., McGaugh et al., 1993). Moreover, as negative affect increases, there is a tendency to respond in a reflexive, rapid manner. Thus, according to this formulation, high levels of negative affect produce a strong end-based bias in terms of the organism's information processing. The organism is primed to attend to signals of threat and, to some extent, it develops "tunnel vision" with respect to response options. Thus, high levels of negative affect produce two kinds of attentional bias. There is an initial inability to disengage attention from threat cues, but once safety signals are perceived, the organism is similarly unable to divert attention from potential avoidance and escape options (Derryberry & Reed, 2002). In the case of addiction, we believe that to the extent that the hot system predominates, the organism focuses on response options that are associated with rapid and efficient affective control and reduction (i.e., drug use; Metcalfe & Mischel, 1999).

One important implication of hot processing is that at high levels of negative affect, cognitive control resources have relatively little impact on decision making and behavior. The modulation of cognitive control can be understood by means of an analysis of how negative affect resets incentive values of drug, and nondrug, instrumental acts and the implications of this for the detection of conflict.

Resetting Incentive Values

We posit that drug deprivation, and its attendant distress, renders salient response options that previously repleted the deprivation, just as occurs with other sorts of deprivation-induced distress (e.g., animals do not acquire a strong tendency to approach water cues in response to deprivation [of water] unless they have consumed water while thirsty; e.g., Hall, Arnold, & Myers, 2000). Evidence suggests that signals of dehydration act as "conditional modulators" (e.g., Hall et al., 2000; Rescorla, 1991). We assert that signals of negative affect may come to "set the stage" or modulate the initiation of behaviors that are associated most strongly with affective repletion. That is, deprivation powerfully inflates the incentive value of cues associated with repletion.

The powerful impact of deprivation on incentive value can be seen in organisms exposed to severe food deprivation (e.g., prisoner-of-war camps). After severe food deprivation, such individuals, when given free access to food, not only eat beyond metabolic requirements, but they show extraordinary preoccupation with food (e.g., hoarding it, dreaming about it, etc.). These effects may persist for years and testify to the powerful impact of deprivation on incentive properties (Crow & Eckert, 2000; Polivy, Zeitlin, Herman, & Beal, 1994).

If negative affect modulates incentive value, how might this occur? It is possible that incentive value is inflated by means of dynamic interactions among the prefrontal cortex, the amygdala, the bed nucleus of the stria terminalis, and the shell of the nucleus accumbens (NAC) in the regulation of goal-directed behavior, especially as it is influenced by conditioned cues (e.g., Baldwin, Holahan, Sadeghian, & Kelley, 2000; Floresco, Blaha, Yang, & Phillips, 2001; Schoenbaum, Chiba, & Gallagher, 2000). For example, the amygdala, which is centrally involved in the processing of negative affect (e.g., LeDoux, 1996), enervates the NAC, which is centrally involved in imparting incentive value (Baldwin et al., 2000; Floresco et al., 2001). These structures appear to be crucially involved in pharmacologic motivational processes just as they are involved in nonpharmacologic motivational phenomena. Interestingly, drug withdrawal can influence glucose utilization in both the basolateral amygdala (BLA) and the NAC (Pratt, Brett, & Laurie, 1998), and these structures appear to mediate the motivational potency of drug cues (e.g., Kruzich & See, 2001). In addition, the BLA appears to be crucial to both the acquisition and expression of the associative incentive properties of drug cues (Kruzich & See, 2001). A study by LaBar et al. (2001) found an interaction between food cues and deprivation, such that the greatest amygdalar activity occurred in the context of cues plus deprivation. This is consistent with the amygdala playing a role in mediating the increased salience of cues of repletion in the context of deprivation. Finally, it should be noted that elements of this circuitry are involved in imparting incentive value on cues associated with reward (Everitt et al., 1999), so it is by no means specific to negative reinforcement.

Drug signals may not only modulate the salience and impact of drug cues but also decrease the salience and impact of nondrug cues (cues for operants that do not yield maximally efficient affective repletion). The consequence of this narrowing or winnowing of sources of reinforcement is to render the organism insensitive to nonrepleting incentives and dependent on a narrow range of operants that result in maximal repletion of the ongoing deprivation. There is evidence that the withdrawn organism does, indeed, face an impoverished range of attractive response options. When animals have been trained to respond instrumentally for a variety of reinforcers, they will tend to distribute their responses as a lawful function of contingencies and reinforcer magnitude. However, drug withdrawal strongly suppresses responding for other, nondrug, incentives (e.g., water), as do cues associated with withdrawal (e.g., Goldberg, 1976). The fact that withdrawal, like clinical depression, produces diminished interest and pleasure in response to a variety of appetitive stimuli may be due to the fact that withdrawal elevates thresholds for incentive processing (e.g., Harrison, Liem, & Markou, 2001). Interestingly, an administered drug repletes the deprivation and permits the organism to respond once again for nonpharmacologic incentives. (Antidepressants

such as selective serotonin reuptake inhibitors also reverse the anhedonia of withdrawal [and depression] and restore the incentive value of stimuli; e.g., Harrison et al., 2001.) Thus, the addictive drug (or other agents that ameliorate negative affect; Harrison et al., 2001) not only ameliorates negative affect but also restores the incentive value of nonpharmacologic incentives; it fills the world with other potential reinforcers. Thus, the addicted individual suffering from severe negative affect relapses not only because the incentive value of drug is enhanced (due, in part, to safety signaling; e.g., Zinser et al., 1999) but also because the incentive value of nondrug stimuli is suppressed (see Figure 2).

Modulation of Cognitive Control

Implicit in the model just presented is the notion that the impact of cognitive control resources is blunted at both high and low levels of negative affect. To understand why this is so, it is vital to understand how negative affect and conflict regarding alternative responses to the negative affect influence the recruitment of cognitive control resources.

We adopt the notion of Botvinick, Braver, Barch, Carter, and Cohen (2001) that cognitive control resources, which manage controlled perceptual-attentional processes and bias behavior in service of goals, are recruited in response to conflict detection. In the case of motivational processing in addiction, we assume that a conflict monitoring system comprising the anterior cingulate cortex (ACC), and possibly the insula (Botvinick et al., 2001; cf. Volkow & Fowler, 2000; Weinstein et al., 1998) detects significant negative affect and recruits cognitive resources to cope with the affect. We contend that ACC activity increases to the extent that negative affect increases and conflicting motivation constrains or counters drug use. It is well established that the ACC is especially likely to be engaged when a well-learned response, such as addictive drug use, is no longer functional, feasible, or acceptable, and the organism is striving to abandon or replace that response (Botvinick et al., 2001). Moreover, the ACC may also be activated by significant aversive events, such as pain and negative affect (Jones, Brown, Friston, Qi, & Frackowiak, 1991).

Although ACC involvement may be directly related to negative affectivity, its ability to engage cognitive control resources is not so related. If someone attempting to quit smoking experiences moderate negative affect, the ACC would indeed recruit cognitive control resources to resolve the conflict. Thus, declarative memory and controlled processing could be brought to bear in deciding on a course of action (abstain vs. smoke). However, as affect mounts, it inflates the incentive value of cues associated with addictive drug use (and decreases the value of nondrug cues; Harrison et al., 2001), thereby tending to short-circuit the influence of cognitive control processes. That is, by inflating the incentive value of drug cues, relative to other response options, negative affect can reduce the conflict among response alternatives.⁵ Thus, the organism is led to use the drug in response to this incentive imbalance and, therefore, resolves the conflict. These relations may explain why subjectively experienced urges to use drugs, associated with response conflict and ACC activation (Weinstein et al., 1998), are not always related to drug-use likelihood (Tiffany, 1990); at very low and very high levels of negative affect, drug use is likely, but conflict, and hence urge persistence, may be minimal. The present formulation assumes, however, that when conflict is recurrent, it

will give rise to repetitive, troublesome urges, and those, in turn, will motivate a resumption of drug use.

In sum, the addict who is striving to resist drug use tends to relapse, in part, because the relative incentive value of drug-use versus other response options reduces conflict and hence reduces the call for cognitive control. It may also be the case that even when conflict is detected, the relative inaccessibility of declarative memory (because of hot information processing) may reduce access to information that would raise the incentive value of nondrug operants. A lack of cognitive control resources, in turn, has a number of consequential specific impacts associated with hot processing (e.g., further inaccessibility of declarative memory, a truncated temporal perspective).

The Affective Imperative: Enlisting Cognitive Control

The model presented above suggests that cognitive control resources are most likely to influence drug use at moderate levels of negative affect. For instance, expectations that smoking will disturb others are unlikely to come to mind when an individual is smoking automatically and are unlikely to deter smoking if the individual is experiencing profound distress. However, it would be a mistake to assume that cognitive control resources necessarily have the effect of deterring drug use. In fact, even for the person attempting to maintain abstinence, declarative memory and controlled processes may often be “corrupted” in service of promoting or rationalizing drug use. This will occur, according to the present model, because negative affect, even at moderate levels, is aversive and intrinsically primes escape and avoidance strategies when they are available.

Tice, Bratslavsky, and Baumeister (2001) recently provided an example of the motivational force of even moderate levels of negative affect and of how declarative knowledge (specifically, expectancies of controllability) influenced coping with that affect. Tice et al. found that individuals faced with moderate stress (i.e., imagining involvement in a distressing accident) tended *not* to engage in “impulsive” coping behavior (e.g., eating fattening snacks, seeking immediate gratification) when they believed that their negative affect was fixed (i.e., intransigent). However, when individuals perceived that their negative affect was malleable, they readily engaged in behaviors associated with relief. The Tice et al. research indicated that when individuals believe that affective relief is available, even at moderate levels of negative affect,

⁵ T. E. Robinson and Berridge (1993) also speculated that deprivation and drug withdrawal can influence the incentive value of drug cues (cf. their Figure 3). The current formulation differs from theirs in several important ways. First, we implicate negative affect, per se, rather than withdrawal as the crucial setting event that inflates the motivational value of drug cues. Moreover, we stress that the presence or anticipation of negative affect and not appetitive drug actions has a predominant impact on drug motivation. Also, we believe that anticipatory excitement engendered by drug cues does not necessarily derive from the appetitive effects of the drug. Rather, these may reflect the value of the drug as a safety signal. Having said this, it is important to note that addictive drugs can have appetitive effects that cannot be attributed to negative reinforcement (e.g., positive reinforcement produced in an initial drug response), and these effects, no doubt, influence drug motivation (e.g., when an individual is already experiencing a strong negative affect; cf. Baker et al., 1987).

affective coping leaps up the hierarchy of response alternatives. The authors noted, "When people feel acutely bad, they generally wish to feel better, and this wish is often urgent" (p. 54).

Tice et al.'s (2001) study conveys two points that we believe are relevant to drug use: (a) Even modest levels of negative affect can be powerfully motivating. That is, there is an "affective imperative" that causes the motive of affective coping to assume processing priority (Baker, 1998). (b) Cognitive control resources (e.g., declarative memory) are not necessarily enlisted to counter immediate gratification (in the person trying to abstain). For instance, in the Tice et al. study, the belief that affect was controllable led to the ascendancy of short-term versus long-term goals. Similarly, in the case of addiction, there is a great deal of evidence that attitudes and expectancies encoded in declarative memory may actually promote drug use and relapse (Baumeister et al., 1994; Goldman et al., 1987; Leventhal & Cleary, 1980).

Thus, although moderate levels of negative affect may permit the application of cognitive control resources to drug-use decision making, these resources do not necessarily support abstinence outcomes (even if that is an ostensible goal). This is because of the imperative of reducing even moderate levels of negative affect and because the content of the addicted individual's declarative memory may impede rather than promote abstinence attainment (e.g., strong expectations and conviction that drug use will provide relief—relief that is rapid, reliable, and repeatable; e.g., Goldman et al., 1987; Leventhal & Cleary, 1980; Wetter et al., 1994).

In summary, as negative affect grows and enters consciousness, the addicted individual is increasingly likely to focus on response options for dealing with the threat of impending, worsening negative affect. Negative affect that is not immediately followed by drug use constitutes a conflict for the addicted individual. The existence of a conflict results in recruitment of cognitive control resources that might be used to delay or avoid drug use. However, even at moderate levels of negative affect, the incentive value of drug may be so strong that cognitive control is co-opted to rationalize or defend drug use. If drug use does not occur, and negative affect persists or builds, then attention is increasingly drawn to responses that offer the prospect of rapid, reliable escape from distress. To the extent that the organism remains in conflict over drug use, urges will persist and cognitive control resources will be enlisted. Recurrent, persistent, troublesome urges may themselves contribute to drug motivation. Moreover, once negative affect becomes severe, the incentive value of drug use becomes so great as to reduce conflict, and this results in characteristic features of hot information processing (Metcalf & Mischel, 1999). Then, the individual is likely to focus on well-rehearsed, efficacious coping options, and he or she will tend *not* to engage in reflective information processing that involves nuanced or distal considerations. The individual will be transfixed by the prospect of immediate delivery from the spiral of escalating negative affect and will not consider the prospects of long-term deleterious consequences. Consistent with this, the addicted individual experiencing heightened affect will discount long-term payoffs in favor of immediate gain (e.g., Madden, Petry, Badger, & Bickel, 1997), which may be due, in part, to the inability of the individual to process the high-level construct features of distal goals (Trope & Liberman, 2000). In short, the individual is very likely to relapse back to drug use.

When Affect Grows: Implications for Relapse

The foregoing material suggests that the prototypic relapse context should be characterized by negative affect produced by either withdrawal or stress. Yet, previous reviews have questioned the link between withdrawal- or stress-induced negative affect, on the one hand, and relapse, on the other hand. For instance, reviewers (e.g., T. E. Robinson & Berridge, 2003) have noted that relapses may occur long after withdrawal should have abated and that withdrawal severity is not well correlated with relapse. Moreover, they have noted that initial, appetitive effects of drug, not aversive withdrawal effects, seem to be uniquely effective in instigating resumption of drug use in animals (i.e., relapse).

Our appraisal of the literature suggests that negative states, whether produced by withdrawal or stress, are highly determinant of relapse. For instance, we have found that if tobacco withdrawal is assessed in a sensitive manner that captures its dimensionality (e.g., shape, volatility/scatter), each dimension yields an informative estimate of relapse vulnerability (e.g., Piasecki, Jorenby, Smith, Fiore, & Baker, 2003a, 2003b). Moreover, *timing* of relapse is also consistent with a withdrawal model as smokers, and those addicted to other types of drugs, tend to relapse when withdrawal is at its peak (e.g., Kenford et al., 1994). In addition, recent research has suggested that withdrawal may frequently be more prolonged than once thought (Piasecki, Fiore, & Baker, 1998; Piasecki, Kenford, Smith, Fiore, & Baker, 1997). Research with drugs other than nicotine also has shown consistent relations between negative affect, stress, and withdrawal, on the one hand, and relapse, on the other hand. This holds for both retrospective and prospective studies (e.g., Brown et al., 1990; Hussong, Hicks, Levy, & Curran, 2001; Marlatt & Gordon, 1980; McKay, 1999; McKay, Maisto, & O'Farrell, 1996).

As noted previously, some authors (e.g., Lyvers, 1998; T. E. Robinson & Berridge, 2003) have questioned the role of negative reinforcement in relapse because resumption of drug use in animals seems to be uniquely prompted by direct drug effects (presumed to be appetitive; e.g., Stewart et al., 1984; Stewart & Wise, 1992). However, recent data show that aversive states induced through food deprivation or stress also powerfully prompt renewed self-administration among animals (Campbell & Carroll, 2000; Shaham, 1996; Shaham, Erb, & Stewart, 2000). In fact, Stewart's findings suggest that stress-induced reinstatement effects are stronger than those produced by priming injections of drugs (Shaham et al., 2000).⁶ Moreover, naturally occurring withdrawal also has the capacity to reinstate drug self-administration (Shaham, Rajabi, & Stewart, 1996).⁷ In sum, these findings suggest that negative states

⁶ Stress-induced reinstatement effects appear to be associative in the sense that they are bound to the context of prior drug self-administration; as such they may serve as "occasion setters" (Shaham et al., 2000). Yet, associative elicitation of fear does not appear to be effective in reinstating drug responding (Shaham et al., 2000). This may occur because of species-specific defense reactions, such as freezing, that are elicited by fear cues.

⁷ Naturally occurring versus antagonist-elicited withdrawal may be more effective in reinstating drug use (Shaham et al., 1996). This may be because the stimulus properties of precipitated withdrawal differ from those produced by naturally occurring withdrawal that is more relevant to the organism's prior learning history.

produced by a variety of instigators significantly prompt resumption of drug taking in both humans and animals.

The Affective Motivational Model: Fit With Addiction Phenomena

Criticisms of Negative Reinforcement Models: Relevance to the Affective Processing Model

Numerous criticisms have been leveled against negative reinforcement models. Some of these have already been addressed in our presentation of the reformulated model (e.g., the relation between withdrawal and relapse). In the following section we address briefly the ability of the reformulated negative reinforcement model to account for additional criticisms.

Some Drugs Are Highly Addictive but Do Not Produce Severe Withdrawal Syndromes: If Relief of Aversive Withdrawal Symptoms Is an Important Determinant of Addiction, There Should Be a Relation Between a Drug's Tendency to Support Addiction and the Aversiveness or Severity of Its Withdrawal Syndrome

Cocaine, nicotine, and buprenorphine are offered as examples of drugs that produce few or only mild withdrawal symptoms and yet are highly addictive (e.g., Jaffe, 1992; Lyvers, 1998). On the basis of the evidence, Jaffe (1992) concluded, "there is little correlation between the visibility or physiological seriousness of withdrawal signs and their motivational force" (p. 9).

Our motivational model suggests that the affective components of withdrawal are most motivationally influential and that dramatic somatic signs are not crucial determinants of drug motivation. Relevant to this, nicotine, cocaine, and buprenorphine (all addictive drugs) produce withdrawal syndromes comprising negative affect as prominent symptoms (e.g., Coffey, Dansky, Carrigan, & Brady, 2001; Fudala, Jaffe, Dax, & Johnson, 1990; Jorenby et al., 1996; Lago & Kosten, 1994).

Some authors have contended that the severity of the affective distress in withdrawal is insufficient to exert potent motivational effects (Jaffe, 1992; Lyvers, 1998). These authors are correct that the affective changes seen during nicotine and cocaine withdrawal are not necessarily large in magnitude (e.g., Jorenby et al., 1996; Piasecki et al., 1998). However, it is difficult to gauge the motivational significance of an affective reaction based on a reviewer's subjective evaluation of its apparent severity. What is crucial is the extent to which variance in affective events organizes available motivational data (e.g., Kenford et al., 2002). It is also important to bear in mind that self-report provides an incomplete index of affective processing, as our model makes clear. Finally, it is important to note that modest increases in misery might predict drug use because addicted individuals have learned that they foreshadow a relentlessly increasing level of distress.

Drugs That Produce Withdrawal Symptoms Do Not Necessarily Support Addictive Behavior

The other side of the poor-correlation coin is that some agents that produce withdrawal do not appear to support chronically high levels of self-administration indicative of addiction. If withdrawal

is a prepotent motivator of addictive behavior, should not all agents that produce withdrawal also support addictive behaviors? Prominently mentioned among these agents are certain tricyclic antidepressants (imipramine, amitriptyline), anticholinergics, and κ -opioid agonists (e.g., ethylketazocine, bremazocine, ketacyclazocine, benzomorphan ligands; e.g., Jaffe, 1992).

In our view, the central issue concerns whether the withdrawal syndromes of such agents comprise negative affect and whether the drugs in question reverse that negative affect in a timely and efficient manner. Most of the agents mentioned above can produce a variety of somatic signs of withdrawal. However, some of these agents (e.g., tricyclic antidepressants) do not appear to produce significant negative affect that rapidly follows discontinuation of the agent, and that is rapidly reversed by the same agent (e.g., Bialos, Giller, Jatlow, Docherty, & Harkness, 1982; Davidson, 1998). Moreover, some of these agents such as the κ -opioids, ethylketocyclazocine and ketocyclazocine, and anticholinergics, such as scopolamine, can indeed support self-administration and even abuse (Brown, 1990; Stanilla & Simpson, 1995). Additionally, some of these agents can produce aversive direct effects at fairly low dosages (e.g., the κ -agonist U50,488H; Bechara & Van der Kooy, 1987; anticholinergics; Brown, 1990), which could limit their self-administration regardless of a withdrawal syndrome. In sum, it is difficult to draw firm conclusions on the basis of gross characterizations of classes of drugs because drug self-administration and withdrawal occur on a continuum that defies simple dichotomization, because aversive agonist effects may render the presence or absence of withdrawal moot, and because of inadequate information on the affective consequences of withdrawal and its amelioration by specific drug classes.

Treating Withdrawal Distress Alone Is Generally Regarded as Minimally Effective in Overturning Addiction

Reviewers have noted that pharmacotherapies that maintain a stable level of addictive drug in the body (e.g., methadone maintenance, nicotine replacement therapy) can reduce withdrawal symptoms, but they are not consistently effective in quelling drug urges or in preventing relapse (Jaffe, 1992; T. E. Robinson & Berridge, 1993; Wise & Bozarth, 1987). Some reviewers have argued that if withdrawal suppression does not constitute a sufficient intervention, this suggests that addicted individuals are seeking reinforcement from drug use other than withdrawal escape and avoidance (e.g., elation).

It is true that drug replacement therapies such as methadone maintenance and nicotine replacement therapies are only partly efficacious in treating addictive disorders. Yet, it is striking that most of the efficacious pharmacotherapies for addictive disorders have in common the amelioration of withdrawal symptoms, including negative affect (e.g., all of the nicotine replacement products; Killen et al., 2001; West & Shiffman, 2001), bupropion SR (Jorenby et al., 1999), methadone (Strain, Stitzer, Liebson, & Bigelow, 1996), buprenorphine (O'Connor et al., 1997), and so on. Thus, one could safely conclude that the most consistently efficacious treatment strategy used in addiction is that of administering an agent that ameliorates negative affect as well as other withdrawal symptoms. This suggests that suppression of negative affect is a cornerstone of the efficacious treatment of addiction.

It must also be borne in mind that replacement agents are efficacious even though patients continue to experience some withdrawal despite taking them. These agents are often used at dosages too low to produce optimal effects (as they tend to produce lower peak levels of drug in the body than addicted individuals typically self-administer; e.g., Dale et al., 1995), and these agents certainly do not wholly suppress affective reactions to stressors that patients encounter in their daily lives. In addition, none of these pharmacotherapies provides rapid amelioration of a phasic affective event.

Model Predictions

In addition to the model's ability to account for discrepant findings, our model also suggests specific, testable hypotheses:

Some individuals, often referred to as *chippers*, can use addictive drugs extensively without developing signs of significant addiction. Chippers can be thought of as addiction-resistant individuals as they do not develop addictive behavior despite high levels of drug exposure. According to the present model, chippers are distinct from addiction-susceptible individuals because chippers' negative affect is not significantly entrained to drug use. Thus, abstinence will increase various measures of negative affectivity (e.g., startle probe response) among addicted individuals, but not among chippers (see Shiffman, Paty, Gnys, Kassel, & Elash, 1995). Moreover, drug use by abstaining chippers should produce smaller decreases in negative affect than among abstaining addicted individuals.

In an affective priming experimental strategy, exposing addicted individuals to negative affective primes versus neutral primes should increase drug motivation (e.g., urges to use drug, work to obtain drug, and decreased drug use latencies). This manipulation should not produce awareness of any contingency among primes, affect, and motivation. Chippers' motivation to use drug should be relatively unaffected by negative affective primes. In addition, individuals differing in dependence level (heavy drug users vs. chippers) should differ in their susceptibilities to stressors in terms of the stressor's ability to produce response conflict over drug versus nondrug response options and also in the commitment of cognitive control resources to the response conflict.

Drug abstinence should, as a linear function of abstinence duration, increase addicted individuals' attention to cues of negative affect (at least until the time when withdrawal wanes). This should be evident using a number of information processing paradigms. For instance, the severely withdrawn individual should be relatively less able to ignore threat stimuli than when he or she is not withdrawn.

Among highly addicted organisms faced with drug and rewarding nondrug response options, one should observe the following to occur as a function of withdrawal duration (and resultant negative affect): greater evidence of ACC activation (e.g., with fMRI, P450-evoked responses, urge self-reports) and greater constraints on cognitive control resources. For instance, the performance of a trained animal faced with response options leading to drug infusion versus a set level of rewarding brain stimulation will show a quadratic relation between ACC activation and duration of withdrawal. Little response conflict will occur immediately after a prior drug infusion, but ACC activation will grow with increased withdrawal duration. This growth of activation will be reversed when

the incentive value of drug is sufficiently great so as to reduce conflict at high levels of withdrawal. Moreover, one should see a requirement for greater payoffs of nondrug response options in order to activate the ACC as a function of withdrawal duration/negative affect.

Novel Model Elements

Although the reformulated model has much in common with prior negative reinforcement models of addiction motivation, it also comprises novel elements. For instance, the prototypic instigators of addictive drug use are interoceptive cues, especially interoceptive cues of negative affect or internal events contingent with negative affect. This can be contrasted with other associative models where exteroceptive cues are given primary weight. In addition, according to the present model, addicted organisms have acquired a proceduralized motivational-processing sequence in which interoceptive signals of negative affect engage drug self-administration response sequences and may induce awareness of the desire or urge to use a drug without awareness of the affective origins or setting events for the desire.

The model also returns to the somewhat discredited notion that withdrawal and stress increase the likelihood of drug self-administration. However, the current formulation distinguishes between distress due to withdrawal versus distress due to external stressors. Although addictive drugs relieve the aversive withdrawal symptoms to which they give rise, they less consistently alleviate stress-induced distress. Stress-induced drug use is due, in substantial part, to the fact that avoidance and escape responding (drug use) is largely discriminated on affective cues per se, and this accounts for generalization of drug self-administration across stress and withdrawal.

The current model highlights the similarities and differences between ongoing drug use versus drug use after prolonged abstinence (i.e., relapse). Although ongoing drug use and relapse differ in some notable respects (e.g., strength of affective correlates), both are typically motivated by escape from or avoidance of negative affect. Negative affect modulates the nature of drug motivational processing. When the addicted individual is either at very high or very low levels of negative affect, drug motivational processing typically occurs in such a way as to subvert cognitive control. When drug use occurs freely in response to automatized motivational processing, negative affect tends not to build significantly, and therefore cognitive control resources are never recruited. At high levels of negative affect, drug use has great incentive value, which reduces conflict over response options, and therefore cognitive control resources are relatively unavailable to guide decision making.

Conclusion

In the tradition of Wikler and Solomon (Solomon & Corbit, 1974; Wikler, 1965), we have presented a negative reinforcement model of addiction reformulated in light of contemporary basic science in the fields of affect and information processing. This model emphasizes the pivotal role of negative affect in motivating drug use. We suggest that this model can organize extant data in the literature as well as generate novel, specific hypotheses for future investigation. We hope that our attempts to integrate tradi-

tional drug motivation theories with advances in basic research will reinvigorate research on the pivotal role of negative reinforcement in the maintenance of addiction.

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