In: Handbook of Implicit Cognition and Addiction
R.W. Wiers & A.W. Stacy (Eds.), Sage Press, 2006

CHAPTER 31

Automatic Processes in Addiction: A Commentary

KENT C. BERRIDGE AND TERRY E. ROBINSON

Sections 3 through 5 present a fascinating set of chapters on the many different psychological processes that may contribute to pathological drug-seeking behavior in addiction, with an emphasis on implicit automatic processes. We briefly address some highlights.

SPLITTING PSYCHOLOGY OF ADDICTION INTO ITS TRUE COMPONENTS

The idea that addiction may result from dysfunction that alters the balance among multiple different psychological processes, which normally occur simultaneously in encounter with rewards or predictive cues for them, is featured in several chapters. For example, whether an abstinent addict either relapses or continues to abstain at any given point in time may be the result of complex interactions among multiple cognitive and motivational components, all of which may be activated by the same reward-related event. Thus, it is important to know the identity of the real components, differences between them, and the balance that controls behavior at any moment. Yin and Knowlton (Chapter 12) describe the differences between three major categories of psychological process that produce appetitive responses: cognitive act-outcome understanding, simpler S-R (stimulus-response) habits, and middle-level, Pavlovian-triggered appetitive motivational components, which can include attribution of incentive salience to reward-related conditioned stimuli and their neural representations. Yin and Knowlton (Chapter 12) discuss how these three mechanisms operate simultaneously in animals as well as people, and review evidence that they are mediated by different underlying neural systems. Franken et al. (Chapter 13) further survey human neuroimaging studies of reward-related processes, and note the importance of identifying brain activations triggered by reward stimuli that actually mediate reward. These must be distinguished from activation due to other simultaneously activated psychological processes, such as memory and attention. Such efforts will be critical to map psychological components of addiction onto their respective brain mechanisms.

THE ROLE OF S-R HABITS

Ames et al. (Chapter 23) note that the idea that the development of pathological stimulus-response habits contributes to addiction has been prominent ever since William James wrote about habits over a century ago (James, 1890), and there recently has been renewed interest in this idea (Berke, 2003; Cardinal & Everitt, 2004; Everitt et al., 2001; Hyman & Malenka, 2001). Cox et al. (Chapter 17) help unpack the potential role of habits and related implicit goals in addiction. As a methodological issue, Ames et al. (Chapter 23) also point out the need to discriminate automatic habits from controlled cognition. Although habits may contribute to addiction, there are clearly other psychological processes involved too, including motivational ones that can give compulsive force to the act of taking drugs (Robinson & Berridge, 2003).

AFFECTIVE PROCESSES: THE ROLE OF NEGATIVE REINFORCEMENT

One of the most longstanding motivational hypotheses of addiction is the negative-reinforcement idea that addicts take drugs mainly to escape negative affect states of drug withdrawal, or of preexisting life stresses and other unpleasant situations. Curtin et al. (Chapter 16) provide a thoughtful contemporary argument for the negative reinforcement idea here, suggesting that negative affect might be viewed as the most important factor in driving addiction. In their view, implicit mechanisms trigger craving by quickly activating mechanisms of negative affect, while slower cognitive control mechanisms lag behind. For some addicts in some situations, such negative reinforcement explanations are surely plausible. As Wiers et al. (Chapter 22) note in support, "coping drinkers," at least, often may drink to avoid negative affect. Birch et al. (Chapter 18), however, conclude that evidence for the notion that negative reinforcement in general is the primary mechanism for addictive drug-taking is at best inconsistent. Indeed, they point out some studies indicate quite the opposite: Events that cause positive affect may markedly increase craving.

APPETITIVE PROCESSES

For these and other reasons, Wiers et al. (Chapter 22) note that in recent years the dominant research focus has been on implicit processes of positive reinforcement and on positive appetitive reactions underlying addiction. Cox et al. (Chapter 17) point out that people sometimes drink especially when they hold positive expectations. Bechara et al. (Chapter 15) demonstrate that some drug addicts also overrespond to other positive rewards, such as money won in a gambling task. Bechara et al. (Chapter 15) suggest in particular that amygdala-triggered positive reactions, mediated by somatic marker mechanisms (Damasio, 1996), can generally cause exaggerated appetitive response to incentive cues in addicts, most particularly drugs but not necessarily always limited to them. Fascinatingly, these authors show that dopamine manipulations alter money-based gambling decisions most in ambiguous conditions, that is, when players without explicit understanding of the payoff rules must rely on implicit knowledge cues. Among such exaggerated somatic marker reactions, Bechara et al. suggest that the reaction of incentive-wanting may be mediated in part by somatic markers occurring in the supracollosal sector of the anterior cingulate cortex, whereas liking (the hedonic experience following consumption of a reward) may involve somatic markers located in the insula and somatosensory cortex.

Related to distinguishing wanting from liking in addicts, Wiers et al. (Chapter 22)

show that heavy drinkers have higher arousal responses to alcohol-related items in an implicit association task, compared to light drinkers. But heavy drinkers still have equally negative valence as light drinkers toward alcohol items. Wiers et al. (Chapter 22) suggest that this dissociation between positive arousal and negative affective reaction might reflect greater "wanting" for alcohol without greater "liking" for alcohol in heavy drinkers, as predicted by the incentive-sensitization theory (Robinson & Berridge, 1993; Robinson & Berridge, 2003). In support of the idea that incentive salience might be automatically attributed to cause dissociated "wanting" and influence behavior in an implicit manner, other evidence indicates that unconscious "wanting" without insight into accompanying behavior can be produced in both addicts and ordinary undergraduate students (Fischman & Foltin, 1992; Robinson & Berridge, 1993; Winkielman et al., 2005).

Regarding factors that control the strength of positive reactions, Cox et al. (Chapter 17) note that the brain response of heavy drinkers to alcohol cues is actually magnified immediately after they ingest alcohol. It seems noteworthy that the priming-enhanced motivated reaction to alcohol cues occurs even though negative reinforcement withdrawal should be lowest just after a drink. Thus, alcohol on board actually magnifies brain motivation-related responses, rather than medicating them down. Alcohol priming of neural responses perhaps reflects pharmacological activation of brain mesolimbic systems, which in these heavy drinkers generate even stronger incentive salience or "wanting" when under the influence of alcohol. Synergistic potentiation of incentive mechanisms might thus act dangerously to catapult a drinker into sustained alcoholic relapse. This phenomenon may also be related to the reinstatement of drug self-administration produced by drug priming so well described in preclinical studies (McFarland et al., 2004; Shaham et al., 2003; Vezina, 2004).

PERCEPTUAL SALIENCE

The ability of drug-related stimuli to draw the eye and attract attention is a crucial feature of the incentive salience hypothesis of dopamine-related mesocorticolimbic function, and its potentiation in drug users is predicted by incentive-sensitization theory of addiction. This feature is beautifully highlighted in several chapters. The perceptual/motivational interaction is described by Bruce & Jones (Chapter 10) as "salient stimulus properties grabbing attention in a preconscious, automatized and involuntary way," revealed best in spatial visual tasks. Field et al. (Chapter 11) describe how initial orientation and duration of eye fixation measures of drug users looking at drug stimuli correlate with measures of drug craving. Similarly, Waters and Sayette (Chapter 21) describe how smokers pay exaggerated attention to smoking cues, compared to nonsmokers. They also raise the interesting possibility that smokers may be more apt to notice cigarettes or other people smoking when they themselves are attempting to quit than when still smoking. Conceivably deprivation might potentiate mesolimbic brain activation of incentive salience mechanisms in these individuals, raising incentive salience attribution to appropriate cues in a way that is similar to how natural hunger, thirst, sodium depletion, and other deprivation states selectively raise the incentive value of their own appropriate stimuli (Berridge, 2004; Toates, 1986). Enhanced attentional process in addiction may also involve alterations in brain cholinergic systems, which have been shown to exhibit sensitization effects (Arnold et al., 2003).

MODULATION OF REACTIVITY BY CONTEXT, DRIVE STATE, DRUG, AND OTHER ADDICTIONS

Basic appetitive reactions and other automatic processes can be modulated by a variety of factors. Krank and Wall (Chapter 19) show that context is especially powerful as a modulator of addiction processes. They note the famous example of Vietnam veterans who gave up drugs when they returned to postwar civilian life, and suggest that context acts in part to influence addiction by modulating memory processes. Mucha et al. (Chapter 14) describe how basic automatic reactions are also modulated by other factors, including motivational state or drug state. They suggest that basic acoustic startle reflexes, which are modulated by motivational state and drugs, can be used to study mechanisms that overlap with automatic responses involved in addiction. In an interesting extension of modulation of appetitive reactions in a way that suggests cross-talk among different addictions, Zack and Poulos (Chapter 24) show that gamblers/drinkers exhibit facilitated IAT reactions to alcohol-related words immediately after gambling wins. Also, they note amphetamine administration appears to prime gamblers' motivation to gamble, an appetitive facilitation involving mesolimbic activation that may share mechanisms similar to a drug's ability to prime incentive motivation to take more of the same drug. One wonders if these phenomena are related to the cross-sensitization between drugs revealed in animal studies, and to the modulation of sensitization induction and expression by associative context (Anagnostaras et al., 2002; De Vries & Shippenberg, 2002; Robinson & Berridge, 1993).

CONTROLLED COGNITION VERSUS AUTOMATIC APPETITIVE PROCESSES

The complex interaction between automatic appetitive processes versus cognitive control ("willpower") is highlighted in several chapters. Fillmore and Vogel-Sprott (Chapter 20) suggest that cognitive control is especially vulnerable to disruption if a person samples drugs. They show that alcohol specifically impairs cognitive control processes, while leaving automatic processes unaffected. Selective inhibition of controlled cognition needed to abstain thus leaves automatic appetitive mechanisms free to trigger further problematic responses. Bechara et al. (Chapter 15) point out further that damage to ventromedial prefrontal cortex may occur in at least some addicts, and may specifically impair willpower and create special vulnerability to relapse. Finally, in an intriguing and novel twist on the competition between automatic versus controlled processes, Palfai (Chapter 26) suggests it might be possible to harness some automatic reactions to help, instead of hinder, efforts to abstain. Palfai notes that dieters' automatic responses to subliminal diet-related primes can be modified by self-control, and suggests that future therapies might possibly make a self-control reaction itself automatic, using it to aid the cognitive decision to abstain.

Altogether, these chapters help to clarify the nature of the multiple psychological processes that may contribute to addiction, especially implicit or automatic processes. They also reveal specific interactions between basic automatic processes and higher-level regulatory controls. In sum, the authors of this collection of chapters show that recent research on automatic processes has borne valuable fruit, and usefully point the way to research topics for the future.

REFERENCES

Anagnostaras, S. G., Schallert, T., & Robinson, T. E. (2002). Memory processes governing amphetamine-induced psychomotor sensitization. *Neuropsychopharmacology, 26*(6), 703–715.

Arnold, H. M., Nelson, C. L., Sarter, M., & Bruno, J. P. (2003). Sensitization of cortical acetylcholine release by repeated administration of nicotine in rats. *Psychopharmacology* (Berlin), *165*(4), 346–358.

Berke, J. D. (2003). Learning and memory mechanisms involved in compulsive drug use and relapse. In J. Q. Wang (Ed.), *Drugs of abuse: Neurological reviews and protocols* (Vol. 79, pp. 75–101). Totowa, NJ: Humana Press.

Berridge, K. C. (2004). Motivation concepts in behavioral neuroscience. *Physiology and Behavior, 81*(2), 179–209.

Cardinal, R. N., & Everitt, B. J. (2004). Neural and psychological mechanisms underlying appetitive learning: Links to drug addiction. *Current Opinion in Neurobiology, 14*(2), 156–162.

Damasio, A. R. (1996). The somatic marker hypothesis and the possible functions of the prefrontal cortex. *Philosophical Transactions of the Royal Society of London: Series B. Biological Sciences, 351*(1346), 1413–1420.

De Vries, T. J., & Shippenberg, T. S. (2002). Neural systems underlying opiate addiction. *Journal of Neuroscience, 22*(9), 3321–3325.

Everitt, B. J., Dickinson, A., & Robbins, T. W. (2001). The neuropsychological basis of addictive behaviour. *Brain Research Reviews, 36*(2–3), 129–138.

Fischman, M. W., & Foltin, R. W. (1992). Self-administration of cocaine by humans: A laboratory perspective. In G. R. Bock & J. Whelan (Eds.), *Cocaine: Scientific and social dimensions* (Vol. 166, pp. 165–180). Chichester, UK: Wiley.

Hyman, S. E., & Malenka, R. C. (2001). Addiction and the brain: The neurobiology of compulsion and its persistence. *Nature Reviews Neuroscience, 2*(10), 695–703.

James, W. (1890). *Principles of psychology*. New York: Henry Holt.

McFarland, K., Davidge, S. B., Lapish, C. C., & Kalivas, P. W. (2004). Limbic and motor circuitry underlying footshock-induced reinstatement of cocaine-seeking behavior. *Journal of Neuroscience, 24*(7), 1551–1560.

Robinson, T. E., & Berridge, K. C. (1993). The neural basis of drug craving: An incentive-sensitization theory of addiction. *Brain Research Reviews, 18*(3), 247–291.

Robinson, T. E., & Berridge, K. C. (2003). Addiction. *Annual Review of Psychology, 54*(1), 25–53.

Shaham, Y., Shalev, U., Lu, L., de Wit, H., & Stewart, J. (2003). The reinstatement model of drug relapse: History, methodology and major findings. *Psychopharmacology, 168*(1–2), 3–20.

Toates, F. (1986). *Motivational systems*. Cambridge, UK: Cambridge University Press.

Vezina, P. (2004). Sensitization of midbrain dopamine neuron reactivity and the self-administration of psychomotor stimulant drugs. *Neuroscience & Biobehavioral Reviews, 27*(8), 827–839.

Winkielman, P., Berridge, K. C., & Wilbarger, J. L. (2005). Unconscious affective reactions to masked happy versus angry faces influence consumption behavior and judgments of value. *Personality and Social Psychology Bulletin, 31*(1), 121–135.