include not just family and peers but also school, neighborhood and community, the legal system, the media, and the cultural belief system. All of these sources contribute to socialization and influence the rates and types of adolescent reckless behavior within a given culture.

Why would any culture allow adolescent behavior that disrupts the lives of other people and undermines social order, as reckless behavior often does? The reason is that cultures must accept some kind of trade-off in socialization between promoting individualism and self-expression, on the one hand, and promoting social order, on the other. Cultures characterized by broad socialization promote individualism and self-expression in an effort to produce autonomous, creative children and adolescents who express the full range of their potentialities. One price of promoting these goals is higher rates of adolescent reckless behavior; adolescent potentialities include sensation seeking, egocentrism, and aggressiveness, and if the expression of these tendencies is not tightly controlled by socialization, the result is likely to be high rates of reckless behavior. Cultures characterized by narrow socialization face a similar trade-off. They wish to promote obedience, conformity, respect for authority, and social order, and in doing so they achieve lower rates of disruptive and antisocial adolescent reckless behavior, and a safer, more orderly society. However, in promoting these goals, they run the risk of extinguishing what is brightest, liveliest, and most original in their adolescent children.

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The Mind of an Addicted Brain: Neural Sensitization of Wanting Versus Liking

Kent C. Berridge and Terry E. Robinson

What compels an addict to take a drug like cocaine, heroin, or amphetamine? That is the most important question to be answered about addiction. It is different from "What motivates a person to try a drug in the first place?" or "Why might a nonaddict continue to take drugs occasionally for recreation?" The defining features of addiction are its compulsive nature and persisting susceptibility to relapse. Those are the features we have sought to explain in our biopsychological theory of addiction.[1]

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Most expert explanations of addiction parallel the explanations likely to be given by the lay public: Addicts take drugs for the pleasure they produce, and to avoid the unpleasant consequences of withdrawal.[2] But critical examination shows that these explanations are not sufficient to explain addiction.[1,3] The truth is that addicts continue to seek drugs even when no pleasure can be obtained, and even when no withdrawal exists. For instance, addicts seek drugs when they know those available will be insufficient for pleasure.[1,3] Further, addicts crave drugs again even before withdrawal begins: Craving is often highest immediately after taking a drug.[1,3] And addicts continue to crave drugs long after withdrawal is finished: Relapse remains a potent danger when the addict has reentered normal life, after detoxification and recovery from withdrawal.[1,3] Of course, this is not to say that pleasure and withdrawal play no role in the use of drugs. But after one has accounted for all instances of drug use by addicts motivated by pleasure or withdrawal, a vast amount of compulsive drug use still remains to be explained.

We have offered the incentive-sensitization theory of addiction[1] to explain why addictive drug seeking extends beyond pleasure and withdrawal. The theory has four major tenets:

1. Compulsive drug seeking is the result of a progressive and extremely persistent hypersensitivity of specific neural systems (neural sensitization) induced in susceptible individuals by intermittent drug use. Neural sensitization refers to the persistent increased ability of a drug to elicit an effect from particular neurobehavioral systems, and is produced after the systems have received repeated and intermittent exposure to the drug.[1,4] Neural sensitization can more than double the original effect of the drug on the system. This phenomenon stands in contrast to drug tolerance, or reduced responsiveness. Neural sensitization is more than a simple pharmacological effect. Associative learning exerts a powerful role over neural sensitization, and the expression of sensitized drug effects is controlled by conditioned stimuli (stimuli that have previously signaled administration of the drug, and so may predict its future occurrence).[1,4]

2. The neural systems that are most sensitized by drugs normally mediate a specific motivational process we call "wanting" or, more formally, attribution of incentive salience.[1] This psychological process is not "liking" or pleasure, nor is it directly experienced in conscious awareness. Nonetheless, it causes the perception or representation of an event to become attractive, sought after, and capable of riveting attention. This process normally establishes the motivational value of ordinary incentives. But in addicts, the associative pairing of particular acts and drug stimuli with pharmacological consequences that overactivate the system causes excessive wanting to become focused specifically on drug use. (Note that we have placed "wanting" and "liking" in quotation marks to emphasize that our meaning is different from the conscious, subjective awareness that is often meant by these words. We use the words to denote preconscious psychological processes that can cause conscious desire or pleasure but are not identical to them. Additional cognitive processes are required to transform preconscious "wanting" and "liking" into subjective desire or pleasure. For the remainder of this review, we omit the quotation marks but continue to use wanting and liking to mean the underlying processes.)

3. Repeated drug use sensitizes the neural substrates of wanting but not of liking. With the development of sensitization, addicts come to want drugs more and more even if they like them less and less. In other words, the process of addiction leads to an increasing dissociation between wanting and liking.

4. Finally, as we have alluded to, people do not have direct conscious awareness of either wanting or liking. Rather, activation of the neural substrates of wanting or liking must be translated into subjective awareness by cognitive mechanisms, as are other complex perceptions. Because the basic processes that mediate wanting and liking are not directly accessible to consciousness, people may find themselves wanting particular things without knowing why. Under some circumstances, people may not even know that they want them.

NEURAL SENSITIZATION AND DRUG USE

Many addictive drugs, when taken repeatedly and intermittently (as a developing addict would take them), cause certain neurobehavioral systems to become more and more responsive, or sensitized.[1] In particular, dramatic sensitization effects have been observed in mesotelencephalic dopamine systems.[1,4] The neurons of these systems release dopamine as a neurotransmitter. They originate in the midbrain and project to the neostriatum, nucleus accumbens, amygdala, neocortex, and other areas of the forebrain. Mesotelencephalic dopamine systems show robust sensitization after repeated exposure to drugs such as amphetamine, cocaine, or heroin.[1,4]

Once induced, neural sensitization is extremely persistent. In rats, behavioral effects of sensitization, such as exaggerated hyperactivity to amphetamine, may persist undiminished for more than a year, and perhaps for life.[1] At the very least, sensitization persists much longer than do unconditioned physiological withdrawal symptoms, which decay within weeks after the cessation of drug use.

A crucial point for our hypothesis is that the development and expression of sensitization are powerfully controlled by associative learning, particularly by processes of classical or Pavlovian conditioning that help to establish conditioned reinforcers.[1,3–5] Sensitization effects can be markedly enhanced by stimuli that were previously associated with drug administration. Whether the consequences of sensitization are expressed at a particular place or time is determined to a large extent by whether such conditioned stimuli (including contextual stimuli) are present.[1,3,4] The reason why addicts focus excessive wanting specifically on drug use is primarily this interaction of neural sensitization with associative learning.[1]

NEURAL SYSTEMS OF WANTING ARE SENSITIZED BY DRUGS

The same mesotelencephalic dopamine systems that show promi-

nent sensitization have long been recognized as crucial to incentive motivation. They have commonly been viewed as a major substrate for drug pleasure and for addiction.[1–3,5] But what precisely do dopamine systems contribute to reward? In particular, does their role in wanting a reward differ from their role in liking it? Most existing evidence fails to provide a clear answer, because most evidence has come primarily from animal studies that confound wanting and liking. For measures of instrumental behavior (i.e., work or effort directed at obtaining a reward), preference, goal-directed strategies, and voluntary consumption, changes in wanting look like changes in liking. Instrumental measures most directly reflect the degree to which a reward is wanted. Based on the assumption that rewards are wanted to the degree they are liked, a change in liking or pleasure after dopamine manipulations has often been inferred from altered instrumental performance.[5] It is important to note, however, that in studies of the role of dopamine in reward, there has never been independent evidence for the assumption that changes in performance reflect changes in liking as opposed to changes in wanting alone.

Recent experiments on food reward have examined the relation of liking to wanting directly. The results indicate that instrumental performance does not necessarily reflect liking, at least under certain circumstances.[1,6] A chief line of evidence against the assumption has come from studies in our laboratory.[6] Facial reactions elicited by sweet or bitter tastes can be used to assess the liking (hedonic or aversive affect) elicited by a taste, and are not instrumental in nature.[6] Several studies have shown that dopamine-related manipulations that appear to change the potency of food reward (according to instrumental or voluntary consumption measures of wanting) fail to change the palatability of the same food (as measured by facial reactions).[6] Unlike normal appetite, satiety, or a variety of neural manipulations that increase or decrease the potency of food reward—all of which alter the degree of wanting and liking together—manipulations of dopamine systems appear to change wanting alone.[6] For example, drugs that suppress or activate dopamine systems that alter instrumental measures of food reward failed to change hedonic patterns to sugar. Massive depletion of forebrain dopamine by lesions that destroyed only dopamine-containing neurons eliminated appetite but failed to decrease hedonic reactions to sweet tastes or to increase aversive reactions to bitter tastes. And elicitation of feeding by electrical stimulation of the lateral hypothalamus, which acts in part via activation of dopamine systems and which has been argued to enhance the potency of food reward, failed to increase hedonic reactions or to decrease aversive reactions. Taken together, these observations indicate that dopamine manipulations alter appetite, or wanting, for food rewards but do not alter liking.

Recent neurophysiological evidence also supports the hypothesis that dopamine systems mediate wanting in particular, and not liking. For example, in studies of the neuronal activity of dopamine systems during reward, Schultz and colleagues have shown that monkeys that expected to receive a tasty reward showed maximum electrophysiological activation of dopamine neurons at the moment a conditioned stimulus signaled the reward was about to occur—a moment most relevant to wanting[1]—not at the moment the food was actually received or tasted.[7]

The combination of neurobiological and affective behavioral evidence has led us to suggest that mesotelencephalic dopamine-related systems mediate reward by a psychological process that is separable from sensory pleasure. The process we posit is the attribution of incentive salience to the brain's representations of stimuli and events.[1,6] Incentive salience transforms the representation to which it is attributed, making the event attractive and able to grab attention. Once its representation has been transformed into a salient incentive, an event can elicit approach, instrumental action, goal-directed strategies of cognition, and the conscious experience of desire. Such a transformation by itself is wanting alone—no pleasure need necessarily accompany it.[1,6] Indeed, when a powerful incentive is much desired but not obtainable, as in the myth of Tantalus, the experience becomes unpleasant: To be tantalized but never gratified can be a form of torture.

Incentive salience, or wanting, must be actively attributed to a percept or representation. The active nature of the attribution is critical to understanding the neural bases of motivation and addiction. Although the mesotelencephalic dopamine systems that we suggest mediate the attribution of incentive salience are not primary sensory systems, they nonetheless modulate the brain's responsiveness to sensory stimuli. This modulatory role results from the embedment of mesotelencephalic dopamine systems within larger corticostriatal neural systems that receive extensive high-level, highly processed sensory inputs.

ONLY WANTING—NOT LIKING—IS SENSITIZED BY DRUGS

Addiction is due to sensitized wanting—not to liking. Normally, liking—the pleasure engendered by an encounter with a new incentive—serves as the trigger to activate and direct wanting (via associative learning[1,6]). The degree to which an ordinary incentive becomes wanted

depends essentially on the degree to which it is liked. But in drug addiction, because of neural sensitization of wanting, these two processes become decoupled.

The precise locus of neural sensitization is not known, but changes in mesotelencephalic dopaminergic neurons or the inputs and outputs of these neurons seem to be involved.[1,4] A consequence is that drug wanting increases markedly while liking for a given dose may fade. Because the attribution of incentive salience to particular targets is guided by associative learning, sensitized incentive salience for addicts becomes targeted on drug-associated stimuli and mental representations of drug taking. The act of drug taking and associated stimuli, such as drug paraphernalia, become salient incentives themselves. Crack cocaine addicts who have run out of drug, for example, may compulsively and repeatedly examine every small particle they can find that bears any resemblance to a piece of crack, such as a pebble, bit of plaster, or food crumb, even though they know the search for crack is useless; this phenomenon is sometimes described as chasing ghosts.[8] The generation of incentive salience may sometimes be irrational, but it is no less powerful for that.

PEOPLE ARE NOT DIRECTLY AWARE OF WANTING AND LIKING

Addicts who give up their drug often experience intense subjective craving. But addicts who have unimpeded access to their drug may take it routinely, arranging their lives so they can do so without fail, yet habitually and as a matter of course, without intense conscious craving of any kind. To reconcile such craving-free drug use with our hypothesis of sensitized wanting in addiction, it is important to note that subjective experience can sometimes be a misleading guide to underlying liking and wanting. An important postulate of our hypothesis is that conscious awareness has only indirect access to attributions of incentive salience.

It may seem strange to assert that people are not directly aware of their own likes and wants. After all, whether or not people know much about anything else, don't they know what they like? And wouldn't they know if wanting and liking weren't the same? Perhaps nothing strikes a person with greater immediacy than intense pleasure or pain; nothing seizes the mind more completely than an intense craving. But the intensity of these experiences no more implies that people are directly aware of the activation of the elementary processes that have engendered these emotions than the visual experience of the sun as an extremely intense brightness implies that people are directly aware of the neural or computational processes that mediate visual perception.

Visual perception is perhaps the most telling example to illustrate this point. For instance, in the psychological phenomenon known as blindsight, which occurs after damage to the primary visual cortex, people may retain the ability to report the location, brightness, orientation, and even shape of simple visual stimuli in forced-choice tests—yet be completely unaware of what they are looking at or that they are reporting correctly.[9] The brain of a blindsighted person retains many aspects of visual processing, but the subjective mind is not aware of those visual processes.

Regarding addiction, a consequence of the separation of elementary psychological processes from conscious awareness is that it is not nonsensical to speak of unconscious wanting or of unconscious pleasure, just as it is not nonsensical to speak of implicit knowledge or unconscious perception. People are not directly aware of their own elementary processes of either wanting or liking. These go on independently of consciousness. The transformation of elementary visual processes into conscious visual perception is primarily a change of consciousness, not a change of the fundamental visual processes themselves. Many basic visual processes go on equally in the brains of normal and blindsighted people. Analogously, the translation of incentive salience into conscious craving is primarily a change of consciousness, not of the processes of wanting or liking themselves.

Although this point may be controversial, a variety of examples show that people's conscious awareness of what they want can be dissociated from underlying processes of wanting and liking:

1. Choosing what one most dislikes. In a study by Kahneman et al., people freely chose between two procedures that produced pain by prolonged immersion of the hand in ice-cold water.[10] Subjects often chose the experience that they liked less (in the sense that they said it hurt more) because this procedure entailed a small decrement in pain at the end of the trial.

2. Liking induced by events that one does not perceive consciously. Murphy and Zajonc asked people to rate how much they liked a neutral visual stimulus, such as the sight of an unfamiliar Chinese ideograph. These affective ratings were increased if the ideograph was preceded by a too-brief-to-be-perceived tachistoscopic presentation of a smiling face—even though the subjects never consciously detected the face.[11] In this case, liking was elicited by an unconscious perception, and then was integrated—inappropriately—into the conscious rating of the ideograph.

3. Liking distorted by excessive introspection. Even slow, considered judgments of pleasure can be distorted by the very factor—

painstaking introspection—that might be expected to improve the accuracy of affective evaluation. When ordinary people were asked by Wilson and Schooler to judge the taste pleasure of several strawberry jams, the subjects' immediate judgments roughly paralleled the judgments of experts.[12] However, if subjects were asked to analyze their reasons, their pleasure ratings were different and diverged more strongly from the experts' ratings. In other words, rather than revealing the subtleties of underlying affect to conscious awareness, prolonged introspection buried them deeper under additional layers of cognitive interpretation.

4. Wanting without awareness of liking. Finally, people may want without being aware either that they like or want the object they demonstrably seek. Several studies of addiction itself provide the most compelling demonstrations of this fact. For example, Lamb et al. provided "recovered" heroin addicts the opportunity to press a lever to earn an injection that contained either morphine or saline.[13] The addicts were subsequently asked to rate subjectively how much they liked each injection, how much drug they thought it contained, and how much it would cost on the street. The addicts rated saline as worthless and empty, and after several trials chose not to work for it. Conversely, the intermediate to high doses of morphine were rated as pleasant and drug rich, and the addicts worked at high rates to obtain them. Most important, a remarkable dissociation occurred for the lowest dose of morphine. Every addict rated this injection as empty and worthless, identical to saline in its subjective consequences. But despite their subjective evaluation of the low dose as worthless, four out of five addicts worked for the injection at rates as high as those they showed for the higher morphine doses.

Similar dissociations between subjective evaluation and behavioral indices of wanting have been reported for cocaine. For example, Fischman and colleagues offered recovered addicts the opportunity to earn intravenous infusions of cocaine or saline by pressing either of two levers, after they had received desipramine (which blocks the reuptake of dopamine at synapses).[14] In addition, the subjects were asked to rate their subjective evaluations of the drug they chose. Desipramine significantly suppressed the addicts' subjective craving, measured by agreement with the statement "I want cocaine." Also, desipramine administered with cocaine induced unpleasant subjective states assessed by scales of confusion and anger. But desipramine did not at all reduce the behavioral self-administration of cocaine, measured by lever presses for infusions, despite its marked suppression of the subjective experience of drug craving. Desipramine, by our interpretation, disrupted the translation of sensitized incentive salience into conscious awareness, thus reducing subjective craving, but did not disrupt wanting itself.

Such dissociations between the underlying affective processes that drive behavioral seeking and conscious awareness are inexplicable by the conventional assumption that people seek things because they consciously like them. But these dissociations stand as testimony that people can fail to know the relation between their own wanting and liking. Under some conditions, wanting can powerfully direct human behavior while the person's conscious mind remains unaware of wanting or of the motivated behavior. Because of the inability of cognitive introspection to access underlying wanting or liking processes directly, wanting is sometimes best measured by observing what people actually do.

In order to rise to conscious awareness, a salient incentive, like other events, must be translated into subjective experience by processes of cognitive interpretation. Those processes leave room for significant error about what has transpired, even for highly noticeable events.[15] Questionnaires that purport to measure drug craving by addicts must be interpreted with this in mind. It is quite possible that questions intended to measure drug wanting will in many instances fail to do so because of the accidental masking of these processes by cognitive factors. The occurrence of drug self-administration in the absence of reported subjective craving has led some researchers to suggest that persistent drug use becomes automatic or decoupled from conscious volitional control as a consequence of habitual repetition.[16] We would add that the compulsive quality of automatic habits of drug seeking arises from sensitized incentive salience, acquired through associative pairing of neural hyperactivity with the act of drug use, and not the mere repetition of the act itself.

PHARMACOTHERAPIES FOR ADDICTION?

Finally, the incentive-sensitization theory makes a unique prediction for what any as-yet-to-be-discovered medication must do if it is to cure addiction. The theory predicts that the only pharmacotherapy able to constitute a cure will be one that reverses associatively controlled processes of neural sensitization. Medications that simply prevent sensitization will not work. Addicts would not willingly take them until after they became addicted—when it would be too late. Nor will a cure be found in medications that simply suppress the function of dopaminergic or related neural systems. At low doses of such medications, drug-associated stimuli are still relatively

strong incentives. The higher doses that would be needed to suppress all incentive salience are incapacitating. Still other medications, which suppress drug liking, focus on the wrong component of reward, and can at best be only partly successful. Only reversal of neural sensitization would transform the brain and mind of an addict back into the brain and mind of a nonaddict. As yet, a pharmacological cure for addiction does not exist. But the incentive-sensitization theory tells in advance how to recognize a cure: It will reverse the physiological changes that constitute neural sensitization.

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Listening to Speech in the 1st Year of Life: Experiential Influences on Phoneme Perception

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The use of language to share thoughts, ideas, and feelings is a uniquely human characteristic. And to learn a language is one of the biggest challenges of infancy and early childhood. In order to be successful at this momentous task, the child must break down the speech stream, which consists of highly encoded and overlapping information, into smaller units such as clauses, phrases, and words. A yet smaller unit is the phoneme. Two words may differ by only one phoneme (e.g., *bat* vs. *pat*), yet this difference is enough to convey different meanings. Thus, a critical part of the language acquisition process is the abil-

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