

10 Incentive Saliency in Addiction and Over-Consumption

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Drug Addiction, Recovery, and Relapse

Most adults have used a potentially addictive drug at least once, if caffeine, alcohol, and nicotine are included in addition to illicit drugs. In some cases, contact with a substance is so frequent and socially accepted that many people fail to recognize it as a “drug.” However, even among those who have used such potent drugs as cocaine or heroin, relatively few develop formal addiction. Addiction is characterized by compulsive drug seeking, impairment of social and psychological functions, and/or damage to health. Typically it involves overwhelming involvement with the addictive reward, loss of control, and narrowing of interests. According to a 2010 survey, less than 10 percent of the Americans met the criteria for chronic alcohol-abuse or drug-abuse disorder, and an even smaller proportion suffered from chronic addiction (SAMHSA 2011). Yet the worldwide monetary and social costs associated with addiction are enormous.

A chief problem in treating addiction is chronic or repeated relapse among those who are trying to quit. Even after prolonged periods of withdrawal and abstinence, a high percentage of addicted individuals in treatment programs eventually relapse to drug taking. For example, in the case of a study of heroin users, relapse rates after cessation were approximately 60 percent within 3 months and at least 75 percent within 12 months (Hunt, Barnett, and Branch 1971). For this reason, drug addiction is characterized as a chronic relapsing disorder; relapse is the rule rather than the exception, and often occurs repeatedly.

There are three conventional reasons often suggested in addiction neuroscience to explain relapse in drug addiction:

Drug euphoria Addicts resume drug taking to experience the intense pleasure (euphoria) they remember the drug producing.

Over-learning of habits or predictions Drug taking becomes such a well-entrenched habit that relapse is almost inevitable, or learning becomes distorted in other ways to create false predictions about drugs' rewards.

Withdrawal escape The withdrawal syndrome that accompanies the cessation of drug intake is so unpleasant that an addict would do anything to stop it, and so relapse occurs as an escape from withdrawal.

Each of these three explanations certainly plays a role in relapse. However, we believe, for several reasons, that these explanations are insufficient to explain the central problem underlying relapse in addiction.

First, drug pleasure or euphoria certainly accounts for the initial pattern of drug use and abuse, but it may have more difficulty accounting for relapse as tolerance can develop to the pleasure. Even addicts who no longer find their drugs particularly pleasant may experience increases in drug craving that persist for a long time. (See also the chapter by Kringelbach.)

It also has been suggested by some learning-oriented scientists that the repeated use of drugs creates a learning disorder, such as making drug taking an overly ritualized habitual act or creating false expectations of exaggerated reward. Ritualization may be true of the act of drug taking, but cannot explain the preceding flexible acts of drug seeking during craving. And there is little reason to believe that addicts mispredict the reward value of their drugs or the consequences of their actions. Learned habits or mispredictions alone cannot account for the excessive motivational attraction of addiction.

Many addictive drugs surely induce tolerance (when the drug is present) and withdrawal (when the drug is absent). Withdrawal is typically described as an intense negative emotional state accompanied by dysphoria, anxiety, and irritability, and may indeed be a potent reason why many addicts relapse and take drugs, at least while the withdrawal lasts. Yet withdrawal is a relatively short-lived phenomenon; it decays substantially within days to weeks. By contrast, relapse often occurs even after withdrawal is no longer reported, and even in fully "detoxified" addicts months after "recovery."

In contrast to these suggestions, the incentive sensitization theory (Robinson and Berridge 1993) proposes an alternative explanation that can

account for the persistence of relapse and the independence of addiction from pleasure, withdrawal, or faulty expectations. It may also have applications to some addictions that extend beyond drugs. The incentive sensitization theory proposes that relapse often occurs as a result of brain changes that lead to intense incentive motivation for drugs. These brain changes generate pulses of incentive salience or “wanting,” often triggered by encountering drug cues, which may be experienced as feelings of drug craving or may even control one’s behavior implicitly without need of strong conscious feelings. Craving occurs when the process of incentive salience (or core “wanting”), mediated primarily by subcortical mesolimbic brain systems that use dopamine as an important neurotransmitter, is translated into conscious awareness. Prior to any conscious awareness of drug craving, the motivation to take drugs is due to the over-attribution of incentive salience to drug-related stimuli. It is important to note that incentive salience is a distinct psychological process from withdrawal and drug pleasure. In some cases, attribution of incentive salience to reward-predicting cues may make the cues as “wanted” as the reward itself. Such cues become motivational magnets, sometimes prompting irrational behaviors, such as interactions with cues specific to those previously seen only during interactions with the reward itself (Davey and Cleland 1982), as when a person addicted to crack cocaine scans the floor for a white speck (which is more likely to be an ordinary pebble than crack cocaine), picks it up, inspects it, puts it in a pipe, and tries to light it and smoke it—a phenomenon that has been called “chasing ghosts” (Rosse et al. 1993).

Incentive Salience and Utility

In this section we consider the aforementioned explanation of addiction in terms of forms of reward utility that are important to decision making: predicted utility, decision utility, experienced utility, and remembered utility (Kahneman, Wakker, and Sarin 1997; see also the chapter by Knutson and Karmarkar and the chapter by Plassmann and Wager). Predicted utility is an expectation of how much a future drug reward will be liked. Decision utility is the valuation of the drug manifest in choice and pursuit. Experienced utility is how much the pleasant drug is liked when actually taken. Remembered utility is the memory of how pleasant the drug was in the past. Experienced utility is considered the end point of the decision process.

It is the state reached after successful attainment of a particular outcome, pertaining to the hedonic evaluation of that outcome. Experienced utility informs both remembered and predicted utility to some degree. However, other signals are needed in order for decisions to actually be made. The incentive-sensitization theory suggests that only one of these—decision utility, of which incentive salience is one constituent—need be distorted to create a compulsive addiction.

Typically, decision utility is determined by predicted and remembered utility. However, predicted and remembered utility may fail to be perfect representations of experienced utility, because hedonic memories can become distorted, as when peak-end averages of a hedonic experience outweigh in memory the actual amount of pain or pleasure that was experienced (Kahneman, Wakker, and Sarin 1997). Drug addicts are also believed to often fail to accurately translate experienced utility into decisions (Bechara 2005). In general, any distortion in memory or prediction that leads to faulty predictive utility will likewise affect decision utility, producing decisions that fail to maximize the experienced utility of chosen outcomes.

However, addicts may continue to have problems in excessive decision utility even when their remembered utility and predicted utility for a drug's consequence are quite accurate. Incentive salience has a special role in this. Incentive salience, or cue-triggered "wanting," is a specific form of Pavlovian-related motivation for rewards (Berridge and Robinson 1998; Berridge 2012). Incentive salience is mediated by mesocorticolimbic brain systems, and is especially modulated by dopamine levels. (See also the chapter by Kringelbach.) "Wanting" typically coheres with "liking" (hedonic impact) for the same reward, but "wanting" and "liking" can be dissociated in certain circumstances and by some manipulations, especially those that specifically involve dopamine. Finally, "wanting" can also be distinguished from learning about the same reward. For example, "wanting" triggered by a Pavlovian reward cue can dramatically increase motivation for the reward, even if its previously learned value has not changed (e.g., in hunger, satiety, stress, or drug-related states) (Robinson and Berridge 2013). Abstinence from smoking for only 24 hours can dramatically potentiate neural responses to smoking-related cues (McClernon et al. 2009).

In this framework, incentive salience "wanting" is a pure form of decision utility, distinct from other forms of utility, and in some conditions it

can decouple from all the others (Berridge and Aldridge 2008). That is, “wanting” an outcome is distinguishable from experienced utility (hedonic impact, or “liking” the outcome), from the remembered utility of how nice the outcome was in the past, and from the anticipated or predicted utility of how nice it will be in the future. For incentive salience, under conditions of dopamine-related stimulation, situations exist in which cue-triggered decision utility exceeds remembered utility from the past and, similarly, decision utility exceeds predicted utility for future reward value. In other words, it is possible to addictively “want” something that is not expected to be liked, or remembered to be liked, as well as something that is not actually liked when obtained.

Such addictive “wants” may be especially triggered on particular encounters with addictive cues but not on other encounters with the same cues. According to the incentive sensitization theory of addiction (Robinson and Berridge 1993), attribution of incentive salience to a reward cue becomes exaggerated in addicts as a result of long-lasting mesolimbic brain changes (Paulson, Camp, and Robinson 1991). Exaggeration can happen because incentive salience, which makes up part of decision utility, always results from the synergy between two sources: previously learned associations about the reward cue and the current brain state at the time the cue is encountered (Berridge 2012; Zhang et al. 2009).

Fluctuations in the temptation power of cues, which illustrate the difference between decision utility and predicted utility, hinge on the current neurobiological state factors related to dopamine at the moment the cue is encountered. In particular, incentive sensitization suggests that craving and relapse are magnified by a sensitized neural system (mesocorticolimbic dopamine and related systems), which can flip into a super-reactive mode under several conditions: when the person is under stress, or when a person tries to “just take one” hit of his or her addictive drug (which primes mesolimbic systems to react more powerfully to cues), or during other emotional states that heighten mesolimbic reactivity. At such moments, ordinary stimuli, such as cues associated with rewards, are transformed into potent incentive stimuli, making such cues attractive and able to trigger an urge to pursue and consume their associated reward.

This type of synergistic modulation of “wanting” is not limited to addicts. Most people have experienced at least moderate pulses of incentive salience generated by similar rules. For example, advertisements that

pop up on a Web page may prompt clicking. The smell of food as you walk down the street may make you suddenly feel quite hungry, even if you weren't feeling that way moments earlier. But the smell of food as a cue is not constant in its temptation power: if you really haven't eaten all day you might find the aroma extremely tempting, whereas you won't if you have recently eaten. The essence of incentive sensitization suggests that addicts encounter fluctuations like this in the temptation power of their drug-associated cues, but that, because of the enduring sensitization of their mesolimbic systems, their maximal peaks of temptation are much higher than those that other people are likely to experience in daily life.

The basic mechanisms of the excessive attribution of incentive salience to drugs and drug-related stimuli can even occur as a mostly automatic and unconscious process, creating urges to take drugs whether or not a strong subjective feeling of craving is simultaneously present. Such dissociation between acted-on motivation and confusing subjective feelings is what often renders the compulsive quality of an addict's behavior astonishing even to the addict.

Only "wanting," and not "liking," becomes sensitized, and consequently more intense on its own, as addiction develops. That is because "liking" has separable and more restricted brain mechanisms. In animal studies, sensitization increases neuronal firing in pathways that code incentive salience as well as the behavioral ability of reward cues to trigger frenzied bursts of effort to obtain the reward (Tindell et al. 2005; Wyvell and Berridge 2001). Yet sensitization does not increase "liking" reactions that reflect the hedonic impact of the reward when it actually arrives. Similarly, in humans who are becoming drug-tolerant addicts, incentive motivation to take the drug can grow as they become addicted, so that a single hit of the drug can provoke intense urges to take more even if the person reports the dose of drug no longer gives as much pleasure as initially. Beyond drug addiction, consequent incentive sensitization may also manifest itself in food bingeing, pathological gambling, hypersexuality, and other compulsive motivations.

The neuroadaptations responsible for the sensitization of incentive salience are long lasting if not permanent, potentially persisting for years after the individual stops taking drugs. For example, neurochemically, sensitization leads to an enhanced dopamine elevation produced by an

addictive drug in the synapses of the nucleus accumbens in the face of a drug challenge (Vezina 1993; Kalivas and Duffy 1990). Anatomically, there are also persistent changes in the brain cells and circuits of the mesolimbic system that respond to drugs and control incentive salience (Robinson and Kolb 1997). These include structural changes in the morphology of neurons in brain structures of the nucleus accumbens and prefrontal cortex, increased release of dopamine, and increased sensitivity of dopamine D1 receptors. There have also been reports that cocaine causes an increase in the subpopulation of dopamine D2 receptors that are in a high-affinity state, which may occur even in spite of a reduction in overall D2 receptors and which may result in dopamine supersensitivity in addicts (Seeman et al. 2005; Flagel et al. 2010). This, we suggest, is why relapse is prevalent and persistent despite recovery, and regardless of withdrawal, even when strong pleasure is not to be expected from taking a drug.

Liking and Wanting Things Other Than Drugs

Over-eating is a chief cause of obesity. Could exaggerated “wanting” or “liking” play a role in some individuals over-eating? Excessive hedonic reactions to food would magnify both “liking” and “wanting” above that of a regular individual, thus contributing to binge eating and obesity (Berridge, Robinson, and Aldridge 2009; Davis et al. 2009). Alternatively, changes in “wanting” alone could be responsible for over-eating. Sensitization of mesolimbic dopamine systems by exposure to cycles of bingeing and dieting has been suggested to occur (Avena and Hoebel 2003a,b). Enhanced sensitivity of the mesolimbic reward system could attribute high levels of incentive motivation to the sights and smells related to food, and could drive excessive consumption, without necessarily producing comparable levels of “liking,” when the food is consumed. (See also the chapter by Todd and Minard.)

A different set of problems may face individuals who have been suggested to have elevated “liking” as well as elevated “wanting”—individuals who may deserve to be called food addicts (Davis and Carter 2009; Davis et al. 2009). Notably, Davis and colleagues found that certain individuals who are both obese and binge eaters are far more likely to carry both an allele for a gene that codes a gain of function for μ -opioids and an allele for a gene that may be associated with higher binding for the dopamine

D2 receptor. Together these genetic traits have been suggested by Davis and colleagues to combine to simultaneously increase “liking” and “wanting” for foods in a manner that strongly promotes binge eating and gives rise to addiction-like features, including loss of control and relapse. Similarly, it has been suggested that individuals who carry genes promoting elevated dopamine function may experience stronger cue-triggered urges in response to food cues, which may make them more liable to develop obesity (Campbell and Eisenberg 2007).

Conversely, it has been suggested that anorexia nervosa is related to a reward dysfunction that suppresses the “wanting” for food despite leaving the “liking” portion intact (as well as developing an abnormal “wanting” for body perceptions of self as thin) (Keating et al. 2012).

Gambling may also involve special recruitment of incentive salience brain systems. Uncertainty may especially promote incentive salience under conditions that mirror many of the hallmarks of gambling. (For more on the role of uncertainty in material acquisitiveness, see the chapter by Preston and Vickers.) This may produce a further example of the dissociation between experienced or remembered utility and decision utility. Individuals sometimes seem driven by cues to gamble, in all cases at a global monetary loss, for only a moderate experienced utility. Compulsive gamblers may also show other addictions (Zhang et al. 2009).

Other Consumer Behaviors

Even beyond addiction, situations may arise in everyday life in which incentive salience becomes particularly high, inducing moments of strong decision utility for pursuing or consuming an incentive. (See also the chapter by Plassmann and Wager, the chapter by Preston and Vickers, and the chapter by Knutson and Karmarkar.) Consumer goods can similarly be strong incentives influenced by powerful cues. Some situational factors, such as being in a store, may increase how much consumer goods are “wanted” while the extent to which they are “liked” remains unchanged (Litt, Khan, and Shiv 2010). Purchasing a product without actually “liking” the product sufficiently to make the purchase under normal circumstances is likely to induce regret. Accordingly, Litt et al. (2010) suggested that “wanting” and “liking” consumer goods can be driven in opposite directions, almost mimicking reports of drug addiction. When people

experienced failure while pursuing a desired outcome (e.g., a \$5 gift card), they showed increased willingness to pay for the reward—that is, they “wanted” the reward more. However, these individuals also happened to “like” the reward less in the sense that they were more likely to trade it away for an equivalent but different prize (a \$5 gift card for a similar store).

Incentive Salience in Temporal Discounting?

Individuals are often faced with having to make the choice between smaller rewards received sooner and larger ones received later. Such dynamic inconsistencies are central to temporal-discounting models. (See the chapter by Rick, the chapter by Lea, and the chapter by Preston and Vickers.) A recent interpretation of temporal discounting by Leonhard Lades (2011) suggests that incentive salience may be a factor in temporal discounting, particularly by driving up the value of immediate goals and making them “wanted” more than they may be “liked.” As described by Lades, drawing on other decision theorists, the incentive-salience model of intertemporal choice originates from intertemporal-discounting models that posit two decision systems, one (System 1) affective, impulsive and “in the now” and the other (System 2) more patient, cognitive, and analytical (Berns, Laibson, and Loewenstein 2007; Hoch and Loewenstein 1991; Kahneman 2003; Strack, Werth, and Deutsch 2006; Loewenstein 1996). Lades suggests that in cases of perfect self-control, or in the absence of cue-triggered “wanting,” there is a direct correlation between the expected pleasure of a reward and the motivation to consume it (Lades 2011). In such situations, decisions are products of the reflective System 2. When decisions are influenced by cues that trigger “wanting,” however, the relationship breaks down and the impulsive System 1 becomes the prevalent decision maker.

Similar dissociations between “liking” and “wanting” have been applied to social incentives. Dai, Brendl, and Ariely (2010) have suggested that there are two types of impulsive preferences toward identical human faces—face likability and face incentive value—which they believe to correspond to “liking” and “wanting” respectively. Dai et al. find that under certain conditions there may be a disconnection between “wanting” to look at a face and “liking” the face. “Liking” reactions are independent of the viewers’ gender, whereas males “want” to visually consume attractive

female faces much more than females “want” to look at attractive male faces. (On evolved mate-selection preferences in males, see also the chapter by Saad and the chapter by Griskevicius, Redden, and Ackerman.)

Summary

The incentive-sensitization theory helps explain why the development of addiction is a gradual and incremental process, but also why addiction is a persistent problem once established. Essentially, the magnification of decision utility can create a sense of compulsive motivation without altering the predicted utility, the experienced utility, or the remembered utility of the drug. Exaggerated discounting of other rewards relative to the drug will result, as will a probabilistic form of compulsion in which the addict remains in principle capable of resisting temptation on any single trial but in practice is likely to succumb to relapse if required to encounter a series of repeated temptations.

Powerful “wanting,” often in the absence of equivalent “liking,” is not restricted to addictive drugs. The incentive-sensitization theory may provide an explanation for why consumer goods and certain foods can also become excessively compelling incentives. In turn, cues such as those contained in advertisements can trigger potent “wanting” peaks that propel susceptible individuals toward over-consumption of material goods and toward addiction-like disorders such as binge eating. However, there is a tremendous amount of individual variation in sensitization and in the functioning of the mesolimbic dopamine system. Some individuals, owing to their genes, their hormones, their life experiences, and other factors, are highly susceptible to sensitization. Other individuals are relatively resistant and less likely to develop the neurobiological changes in brain dopamine systems that underlie sensitization. This may explain why relatively few people who take drugs or over-indulge in other forms of reward actually develop compulsive levels of “wanting,” and why certain individuals may be better equipped to resist temptation than others.

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