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REVIEW

# "Wanting," "Liking," and Their Relation to Consciousness

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Most animal and human behaviors emanate from goal-directedness and pleasure seeking, suggesting that they are primarily under conscious control. However, "wanting" and "liking" are believed to be adaptive core subcortical processes working at an unconscious level and responsible for guiding behavior toward appropriate rewards. Here we examine whether "wanting" is an inherent property of conscious goals and "liking" an intrinsic component of conscious feelings. We argue that "wanting" and "liking" depend on mechanisms acting below the level of consciousness, explaining why individuals often struggle to enhance or refrain their motivations and emotions by means of conscious control. In particular, hyperreactivity of subcortical "wanting" systems has been tied to pathological behaviors such as drug addiction and gambling disorder. In addicts, cognitive processes intended to curb drug-seeking wage a constant battle against subcortical urges to take more drug that often ends in relapse following repeated assaults. Nevertheless, we suggest that in nonpathological contexts, "wanting" and "liking" interact with major cognitive processes to guide goal-directed actions.

*Keywords:* "wanting," "liking," reward, addiction, dopamine, consciousness

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## From Drives to Incentives

During many decades, psychologists and ethologists have interpreted animal and human motivations in terms of hypothetical drives capable of energizing behavior (Hogan, 1997; Hughes & Duncan, 1988; Hull, 1943; Lorenz, 1950; Vestergaard, Damm, Abbott, & Bildsø, 1999; Young, 1961). For example, a hungry wolf would come to hunt because a need for food activates a drive that triggers hunting behavior. A period of deprivation was assumed to be the need-inducing factor responsible for the release of the drive energy. Drive reduction was then assumed to follow need satisfaction (e.g., catching and consuming a prey), placing the organism in a state of rest before this drive or another energizes behavior again. This simple interpretation could potentially be used to account for any spontaneous activity, although nobody was able to say how many drives a brain may contain (Hinde, 1960). In addition, as part of a homeostatic mechanism, drive could maintain equilibrium among survival-related parameters—such as blood sugar level and hormonal rates—by inducing positive or negative feedback adjustments after any change in these parameters. Thus, the concept of drive was a comfortable explanation of the reasons why organisms do what they do.

However, the limits of the drive concept were soon suspected. First, some drives clearly do not obey the principles of homeostatic regulation. A prolonged period without exhibiting aggressive behavior does not generate any need for aggression (Archer, 1988), and the same is also true for sexual need. For example, highest levels of masturbation occur most frequently in people with regular partnered sexual activity and a wide range of sexual activities (Laumann, Gagnon, Michael, & Michaels, 1994), while drive theory makes the reverse prediction. Also, some people not devoid of sexual desires lead a happy life of celibacy, contradicting the presupposed existence of an aversive sexual drive (Toates, 2014). Second, drives whose homeostatic nature was unquestioned (such as hunger and thirst) cannot be fully explained in homeostatic terms. A sweet (saccharin) solution is drunk in larger quantities than water despite lacking any nutritional value (Balasko & Cabanac, 1998). A diversity of tastes and flavors leads to greater intake, while access to only one flavor or type of food reduces intake, an effect known as sensory-specific satiety (Berridge, 1991; Rolls, 1986; Rolls, Rolls, Rowe, & Sweeney, 1981). Third, fulfilling the physiological need that triggered a drive for food does not always reduce that drive. So, intravenous feeding and the introduction of food or water directly into the stomach are ineffective at reducing appetite in animals and humans (Miller & Kessen, 1952; Myers & Hall, 1998; Turner, Solomon, Stellar, & Wampler, 1975; Wolf & Wolff, 1943), except with very large loads of 40% or more of normal intake placed in the stomach (Janowitz & Grossman, 1949). Although intake is suppressed in proportion to the duodenal load (e.g., Houpt, Anika, & Houpt, 1979), the evidence that oral ingestion is necessary to reduce

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hunger and thirst is at odds with drive theory. Fourth, if distinct drives existed, distinct brain regions should code them as separate entities. However, no drive-dedicated neurons have been highlighted. Motivation is a relatively nonspecific process that depends on brain regions in which the same neurons may code different motivations according to context as well as the animal's predispositions and experiences (Holst & Saint Paul, 1963; Panksepp, 1998; Valenstein, Cox, & Kakolewski, 1970). For instance, the electrical stimulation of electrodes implanted in the lateral hypothalamus of a rat may elicit eating behavior in the presence of food. But if that food is replaced by a waterspout, the rat comes to drink after a few days, and drinking behavior shows persistence when food is put back in the cage (Valenstein et al., 1970). Finally, the recent dramatic rise in rates of obesity across the United States and the Western world highlight how overconsumption arises with increased diversity, palatability, and availability, and not sudden changes in physiological hunger (Bodor, Rice, Farley, Swalm, & Rose, 2010; Cummins & Macintyre, 2006; M. J. F. Robinson, Burghardt, et al., 2015). In this respect, even "animals often eat, not because of a state of depletion, but because of cues in the situation that evoke ingestion" (Wong, 2000, pp. 140–141).

A radical change in our understanding of motivational concepts occurred with the emergence of incentive theories. Incentive theories see motivation as a psychological process that makes specific stimuli (called rewards) attractive, acting as motivational magnets that are approached and eventually consumed. Bindra first introduced the incentive view as follows: "motivational states influence the production of directed responses, not by a direct influence on motor outflow, but by influencing perceptual processes" (Bindra, 1978, p. 46). According to Bindra, when a conditioned stimulus (CS) is repeatedly paired with an unconditioned stimulus (UCS), the animal not only learns that the CS predicts the UCS delivery, but also transfers the motivation for the UCS to the CS. In other words, the CS comes to acquire the incentive properties of the UCS. A major problem with this interpretation is that it does not relate the incentive value of a CS to the individual's physiological state. After an animal learns a light–food association, the light should acquire incentive properties once and for all. The animal should therefore respond to light as an incentive stimulus, whether or not in the appropriate motivational state (Gallistel, 1978). In this respect, Bindra's view suffered the same difficulty as drive theory, with the difference that the incentive was here acquired rather than innately determined. To remedy this difficulty, Toates suggested that physiological depletion states can enhance the incentive value of UCSs and CSs (Toates, 1986). This was not a return to energy-based theories, but instead insisted on the role of physiological deficits in modulating the incentive value of specific reward sources. For example, the odor of a roast chicken is highly appetizing when hungry but may become totally unattractive after consuming a heavy meal.

The Bindra-Toates model of incentive motivation relied on the assumption that the appetitive value of a stimulus and the pleasure felt during its consumption is the same process, which results from reward learning. In this sense, appetence simply consists of an anticipation of the pleasure from past experiences. This view was mirrored by the findings that dopamine seemed to control pleasure: When rats are injected with a dopamine antagonist (whose effect is to reduce the action of dopamine in the brain), they stop seeking rewards such as food (Wise, 1982). This suggested that the dopamine antagonist abolished pleasure, so that the animal was no longer interested in obtaining what was previously pleasurable.

However, Terry Robinson and Kent Berridge suggested that reward is not a unitary process and is instead composed of three distinguishable components: "wanting" (incentive salience or motivation), "liking" (hedonic reactions), and associative learning (Berridge & Robinson, 1998; T. E. Robinson & Berridge, 1993). Their major discovery is that dopamine in the nucleus accumbens—a mesolimbic region of the brain—controls "wanting," not "liking" or even learning, and hence more than hedonic reactions and pleasure are involved in the expression of motivated behavior. In short, "wanting" (or incentive salience) denotes the motivational attractiveness of a stimulus (whether conditioned or unconditioned), leading animals and humans to approach and work to attain it. Physiological deficits such as hunger or thirst are the most obvious modulators of "wanting," but the simple sight of an appetizing stimulus or the administration of a drug of abuse can have similar effects. Mesolimbic dopamine has been shown to be both necessary and sufficient to transform an animal's mental representations of specific stimuli into rewards (Berridge, 2007; Tindell, Smith, Berridge, & Aldridge, 2009; Wyvell & Berridge, 2000). In contrast, "liking" is the pleasure felt during the consumption of a reward, whether related to food, water, sex, or drug (Berridge, 2000; Berridge & Robinson, 1998). Hedonic reactions can be objectively measured by emotional facial expressions, which exhibit homologies in response to pleasant (sweet) and unpleasant (bitter) tastes in humans, nonhuman primates, and rats (Berridge, 2000; Berridge & Kringelbach, 2008; Grill & Norgren, 1978; Smith, Mahler, Peciña, & Berridge, 2007; Steiner, 1973). The ability to experience "liking" is unrelated to dopamine (Berridge & Kringelbach, 2015; Berridge & Robinson, 1998; Berridge, Robinson, & Aldridge, 2009; Berridge, Venier, & Robinson, 1989) and depends on very specific brain regions (called hedonic hotspots) in areas including the nucleus accumbens and the ventral pallidum. Hedonic hotspots have also recently been discovered in the orbitofrontal and insular cortices (Castro & Berridge, 2015). Rather than dopamine, these hedonic hotspots are sensitive to neurotransmitters acting on opioid and endocannabinoid systems (Castro & Berridge, 2014; Mahler, Smith, & Berridge, 2007; Smith & Berridge, 2007). In the following section, we explain how "liking" and "wanting" components of the incentive salience hypothesis can be double dissociated and examine why they might act under the level of consciousness. We recognize that the incentive salience hypothesis is one interpretation of dopamine's role in reward among others (e.g., Salamone & Correa, 2002; Schultz, 1998; Wise, 1982). These alternative views have been extensively discussed elsewhere and cannot be described in detail in the present article because of space limitations. Nevertheless, some aspects of those theories will be considered and contrasted with the predictions of the incentive salience hypothesis.

## Two Dissociable, Unconscious Processes

"Wanting" and "liking" naturally act in tandem. We tend to "want" what we "like" and "like" what we "want." The pleasure experienced from consuming a reward engages motivational measures that prompt attraction and repeated consumption of that same reward. Yet despite operating as a unified process, "wanting" and "liking" result from distinct brain mechanisms and can therefore

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be dissociated. In a laboratory setting, "liking" without "wanting" is shown in animals with impairments in their ability to produce or use dopamine. For example, rats with extensive damage to their dopamine neurons or pharmacological dopamine blockade, fail to approach ("want") sweet food and develop profound life-threatening aphagia and adipsia, but exhibit normal hedonic "liking" reactions when that food is placed in their mouth (Berridge et al., 1989; Berridge & Robinson, 1998; Peciña, Berridge, & Parker, 1997). Because of a genetic deficiency, dopamine-deficient (DD) mice have virtually no dopamine in their brain. Those mice must be artificially fed and could starve and die while surrounded by appetizing food because they cannot attribute incentive salience to rewards. Nevertheless, when DD mice are forced to eat, they ingest as much food as normal mice, show signs of hedonic "liking" during consumption (Cannon & Bseikri, 2004), and will still prefer sucrose or saccharin over water or consume obtained food rewards because of their hedonic impact (Cannon & Palmiter, 2003; S. Robinson, Sandstrom, Denenberg, & Palmiter, 2005).

In contrast, "wanting" without "liking" is observed during electrical stimulation of the lateral hypothalamus in rats, which increases feeding behaviors but triggers more aversive facial reactions to various tastes (Berridge & Valenstein, 1991). Similarly, hyperdopaminergic mutant mice showing 170% elevations in extracellular dopamine levels when compared with control wild type mice appear to "want" sucrose rewards more than controls in a runway task, but fail to exhibit higher orofacial "liking" reactivity (Cagniard, Balsam, Brunner, & Zhuang, 2006; Peciña, Cagniard, Berridge, Aldridge, & Zhuang, 2003; Yin & Knowlton, 2006). Similar results can be seen in rats following amphetamine or drug-sensitization induced elevation of dopamine release (Tindell, Berridge, Zhang, Peciña, & Aldridge, 2005; Wyvell & Berridge, 2000) or following optogenetic stimulation of the central amygdala, which enhances and focuses "wanting" despite leaving "liking" intact (M. J. F. Robinson, Warlow, & Berridge, 2014). In humans, studies show that dopamine levels are more highly correlated with subjective ratings of "wanting" a reward than with pleasure ratings of that same reward (Leyton et al., 2002; Volkow et al., 2002). However, not all instances of "wanting" without "liking" are restricted to an experimental laboratory setting. Certain highly addictive drugs such as nicotine are exceedingly "wanted" despite producing little to no feelings of pleasure or euphoria. In human drug addicts, prolonged drug consumption induces persistent changes in dopamine neurons that progressively sensitize an organism to the drug as well as to contextual stimuli associated with drug taking (T. E. Robinson & Berridge, 1993, 2001, 2008). This sensitization of the "wanting" system, also known as incentive sensitization, involves neuronal changes in the mesolimbic dopamine system, leading to greater dopamine responses following drug taking or the presentation of drug-associated cues. Accordingly, drug addicts show intense desire for drug, while they often report no increase (and sometimes a decrease) in the pleasure they experience during its consumption.

## Separate Conscious and Unconscious Processes?

In addition to the dissociation that exists between core "wanting" and "liking" (with quotation marks), these processes can be distinguished from conscious wanting and liking (without quotation marks) that we experience in our everyday life. For example, someone wanting to watch a particular film on TV is fully aware of that desire and would consciously arrange things so that he or she can be home before the film begins. In some situations, however, it appears we can "want" and "like" stimuli in the absence of any subjective consciousness. In a study by Fischman and Foltin (1992), recovering addicts could freely choose between two intravenous injections, where one of them contained cocaine (lowest dose: 4 mg; highest dose: 50 mg) and the other was a saline solution. When addicts were allowed to choose between a moderate to high dose of cocaine and saline, they systematically selected the cocaine option, for which subjective feelings and cardiovascular responses were recorded. At the lowest dose of cocaine tested, they also pressed the button that delivered cocaine more often than the button for saline. However, they reported no more subjective feelings than with saline and no cardiovascular responses were recorded. Self-reports from addicts indicated that they thought of sampling both options equally (cocaine and saline). This result suggests that their choice was influenced by unconscious "wanting" (see also Hart, Ward, Haney, Foltin, & Fischman, 2001; Lamb et al., 1991). In a study of the general population, Winkielman and colleagues exposed participants to subliminal pictures of happy or angry human faces and, immediately after, asked them to pour a sweet beverage into a cup, drink it, and rate their subjective feeling about that beverage (Skurnik, Schwarz, & Winkielman, 2000; Winkielman & Berridge, 2003; Winkielman, Berridge, & Wilbarger, 2005). Thirsty participants exposed to the happy face poured and drank more of the beverage and gave it a higher rating than nonthirsty participants. In contrast, exposure to the angry face in thirsty participants reduced these measures on all three tasks. However, conscious reports of feelings were unaffected by exposure to either face, even when thirsty. This result indicates that affective reactions can remain below the level of consciousness, yet interact with and influence incentive motivation, by modifying the value and behavior directed toward a reward.

The unconscious properties of "wanting" may explain why its expression is highly sensitive to physiological changes, irrespective of Pavlovian associations previously formed. For example, Robinson and Berridge trained salt nondeprived rats to receive a 9% salty solution in their mouth by means of oral cannulas predicted by the presentation of a lever CS (M. J. F. Robinson & Berridge, 2013). Those rats developed strong aversion for the CS, actively avoiding it when it was presented. Two days later and in the absence of additional training, the rats were injected with two substances that mimicked sodium deficiency/depletion brain signals (deoxycorticosterone and furosemide) and were placed again in the test chambers. Despite their highly aversive past experience with the lever CS, the rats became avidly and immediately attracted by the lever, showing an instant shift in behavior as soon as the first lever presentation occurred, despite never tasting the salt solution in this new state (see also Tindell et al., 2005, 2009). This means that Pavlovian performance can vary despite learning conditions remaining constant. The ineffectiveness of cognition at altering "wanting" can be noted in very familiar situations. Most smokers know (because of numerous prevention campaigns, TV documentaries, etc.) that smoking is not good for their health, causing pulmonary cancers and respiratory deficiencies. But if prevention against the noxious effects of tobacco may prevent some people from starting to smoke (when their "wanting" for nicotine is not yet present), most of the time, this is insufficient to

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motivate smokers to stop, or frequently results in relapse following repeated attempts to quit.

The prepotent role of dopamine-induced "wanting" in controlling approach behavior is not surprising if we consider its importance for survival (in particular, feeding and reproduction), even in animal species in which consciousness is very unlikely to exist. Dopamine is a phylogenetically ancient neurotransmitter whose function has remained relatively unchanged over biological evolution. Kelley reports that dopamine and its brain receptors "have been found in all species thus far examined, including nematodes, mollusks, crustaceans, insects, and vertebrates" (Kelley, 2005). In the nematode (roundworm) *Caenorhabditis elegans*, dopamine is involved in food seeking and is released in the presence of bacterial food (Sawin, Ranganathan, & Horvitz, 2000). The platyhelminthe (flatworm) *Dugesia japonica* shows a conditioned place preference for an environment paired with the dopamine agonist methamphetamine (Kusayama & Watanabe, 2000). In arthropods, dopamine is also present but, for unknown reasons, it is more involved in aversive than appetitive conditioning (Barron, Søvik, & Cornish, 2010). The study of "wanting" and "liking" as separate components remains difficult in those animals—because their expressionless head capsules prevents the observation of specific orofacial "liking" reactions (Perry & Barron, 2013). However, it is possible that reward attraction and hedonic reactions in invertebrates are governed by distinct neurobiological mechanisms similar to those identified in mammals and birds.

These experimental findings indicate that we are able to "want" and "like" rewards independently of their usual conscious processing, as when one says "I (explicitly) want to eat strawberries because I (explicitly) like how they taste." According to Berridge, it is reasonable to hypothesize the existence of "other more cognitive and predominantly cortically mediated forms of motivational value, which use explicit representations of reward outcome value and representations of act-outcome relationships" (Berridge, 2007, p. 409). However, it must be noted that the existence of a double dissociation between unconscious "wanting"/"liking" and their conscious counterparts has not yet been demonstrated. If unconscious "wanting" and "liking" can be produced in the absence of explicit wanting and liking, there is currently no evidence that the reverse is true. This does not reject the possible existence of such explicit processes, but suggests that current data can be fully explained by means of implicit "wanting" and "liking."

## Beyond Conscious Control: The Case of Addiction

Addiction typically consists of overwhelming involvement with the addictive reward, a loss of control and a narrowing of interests. According to a 2010 survey, less than 10% of the U.S. population met the criteria for chronic alcohol or drug abuse disorder, and an even smaller proportion suffered from chronic addiction (Everitt et al., 2008; Hyman, Malenka, & Nestler, 2006; U.S. Department of Health and Human Services, Substance Abuse and Mental Health Services Administration;). For those who do, addiction can be said to be a chronic relapsing disorder. Even after prolonged periods of withdrawal and abstinence, a high percentage of addicted individuals in treatment programs eventually relapse to drug taking. Three conventional reasons for addiction and relapse are (a) *Drug euphoria*—drug taking driven by intense drug "liking" (Wise, 1982), (b) *Overlearned habits*—drug taking that becomes such a well-entrenched habit that relapse is almost inevitable (Everitt et al., 2008; Hyman et al., 2006; Koob & Volkow, 2010), and finally (c) *Withdrawal escape*—intense withdrawal syndrome that accompanies the cessation of drug intake is so unpleasant an addict would do anything to stop it, so relapse occurs as an escape from withdrawal (Koob, 2013; Koob & Volkow, 2010; Wise & Koob, 2014). All three of these reasons contribute to addiction and relapse. However, only the excessive "wanting" for drugs and their cues that results from incentive sensitization can simultaneously explain (a) compulsive drug taking despite tolerance to the "liking"/euphoria produced by the drug, (b) the flexible acts of drug seeking that addicts will display during craving, and (c) that relapse often occurs in fully "detoxified" addicts months and sometimes years after "recovery." The incentive sensitization theory (T. E. Robinson & Berridge, 1993) proposes that 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 behavior implicitly without need of strong accompanying conscious feelings. The sensitization of mesolimbic brain systems that account for excessive attribution of incentive salience to reward cues in addicts is very long lasting (Castner & Goldman-Rakic, 1999; Paulson, Camp, & Robinson, 1991; Shuster, Webster, & Yu, 1975), which explains how cravings and urges to relapse can persist long after withdrawal symptoms have ceased. 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. At its core, the motivation to take drugs is because of the overattribution of incentive salience ("wanting") to drug-related stimuli. Mere presentation of drug-related cues produces an increase in dopamine release in both healthy individuals (Boileau et al., 2007) and nondependent users (Cox et al., 2009). The same is seen for alcohol cues in heavy social drinkers and abstinent alcoholics when compared with controls (Grüsser et al., 2004; Ihssen, Cox, Wiggett, Fadardi, & Linden, 2011; Myrick et al., 2004; Wrase et al., 2007). Activation of the mesolimbic systems even occurs when drug-related cues are presented outside of conscious awareness (Childress et al., 2008), suggesting that craving and excessive "wanting" for drugs and their cues is the result of mesolimbic dopamine and incentive sensitization and does not require conscious processing to impact behavior.

Unlike "wanting," "liking" does not become sensitized, which is why these two processes can sometimes become unhinged, particularly in cases of addiction. Sensitization fails to occur because "liking" or pleasure has separable, and more restricted brain mechanisms (Berridge & Kringelbach, 2013; Smith et al., 2007). "Liking" may also play only a limited role in determining the addictive properties of a drug, as the addictive potential of a drug does not always correlate well with the amount of pleasure it generates. Morphine, for example, concomitantly generates both positive reinforcing and negative aversive effects yet is intensely "wanted" by addicts (Bechara, Martin, Pridgar, & van der Kooy, 1993; Stolerman, 1985). Thus elevated "wanting" can detach from normal "liking." 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 & Berridge, 2001). Yet sensitization does not increase "liking" reactions that

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reflect the hedonic impact of the reward when it actually arrives; an excessively "wanted" reward may not be excessively "liked." This explains why 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 drug can provoke intense urges to take more, even if the person reports that the dose of drug no longer gives as much pleasure as it initially did. The dissociation between "liking" and "wanting" has for example been shown for alcohol in humans (Hobbs, Remington, & Glautier, 2005; Ostafin, Marlatt, & Troop-Gordon, 2010), and in studies showing tolerance to the euphoric effects of psychostimulant drugs in cocaine-dependent abusers despite enhanced drug seeking (Mendelson, Sholar, Mello, Teoh, & Sholar, 1998; Volkow et al., 1997). There are also reports of increased drug "wanting" but not "liking" in Parkinson's patients with dopamine dysregulation syndrome (DDS), a side effect of dopamine agonist treatment that affects a minority of Parkinson's patients (Evans et al., 2006).

Even beyond addiction, situations may arise in everyday life in which peaks of "wanting" can induce strong urges to pursue or consume an incentive. For example, advertisements or situational factors like being in a store, might increase how much consumer goods are "wanted" while the extent to which they are "liked" remains unchanged (Litt, Khan, & Shiv, 2010). Purchasing a product without actually "liking" the product sufficiently to make the purchase under normal circumstances is likely to induce regret, a situation where "wanting" and "liking" for consumer goods can be driven in opposite directions, almost mimicking reports of drug addiction. Accordingly, Litt and colleagues demonstrate that when people experienced failure in pursuing desired outcomes (e.g., a $5 gift card), they showed increased willingness to pay for this reward, that is, "wanted" the reward more. However, these individuals also happened to "like" the reward less in the sense that they were likely to more often trade it away for an equivalent prize ($5 gift card for a similar store; Litt et al., 2010).

## Pavlovian Learning and Pavlovian Performance

The incentive sensitization hypothesis (T. E. Robinson & Berridge, 1993) suggests that dopamine is the principle neurotransmitter that governs "wanting." However, a number of studies suggest that dopamine does not code reward attraction per se but rather reward learning (Mirenowicz & Schultz, 1994; Montague, Dayan, & Sejnowski, 1996; Schultz, 1998; Steinberg et al., 2013). Indeed, if dopamine responded to a reward's incentive properties, it should be observed each time the reward is delivered throughout training—provided that the animal's deprivation level is held constant. In contrast, Schultz and colleagues showed that phasic dopamine release is high after the delivery of unexpected food and is gradually reduced to baseline levels as conditioning progresses. As such, dopamine release following a fully predictable reward is undistinguishable from regular background activity. Yet phasic dopamine continues to be produced following extensive training in response to the CS presentation and during the interval between the CS and UCS presentation, particularly when its duration varies randomly (Schultz, Apicella, & Ljungberg, 1993). Finally, they also showed that when a reward is omitted at the time predicted by its CS, there is a sudden, short-lasting depression in dopaminergic firing rates (Schultz, Dayan, & Montague, 1997).

These important findings are in agreement with the predictions of the Rescorla-Wagner and Pearce-Hall models of Pavlovian learning (Pearce & Hall, 1980; Rescorla & Wagner, 1972). Briefly, these models posit that the associative strength ($V$) between a CS and a UCS is gradually learned until an asymptotic learning value ($\lambda$) for that specific association is reached. Early in training, the difference between the actual and optimal values is high ($V < \lambda$, hence $\lambda - V > 0$) and becomes nil later in training ($V = \lambda$, hence $\lambda - V = 0$). Thus, dopamine is assumed to be a *teaching signal*, reflecting prediction error ($\lambda - V$) in Pavlovian conditioning. When the CS-UCS association is impossible to learn, because the UCS is or is not delivered on a random basis following the CS, dopamine release is therefore held to a relatively high level despite extensive training. Another computational framework for reward prediction error is the temporal difference (TD) algorithm, which obeys the same logic as described above (Sutton & Barto, 1981).

Although the hypothesis that dopamine is a teaching signal used to correct (and learn from) inaccurate predictions perfectly fits the electrophysiological data, there is only correlative (not causal) evidence linking the reward-induced dopamine signal with learning. There is a tendency to assume that any change in performance necessarily results from a change in learning. Yet what is often overlooked is that a change in learning is only inferred from a change in performance (because it cannot be measured directly), and that many other processes (fear, frustration, motivation, etc.) can influence performance besides learning. Thus, some doubt subsists with respect to the role of dopamine in learning. Steinberg and colleagues have ingeniously attempted to remedy this shortcoming (Steinberg et al., 2013). They trained rats in a blocking procedure, where a CS1 (e.g., light) is repeatedly paired with a reward, before becoming part of a compound CS1-CS2 (e.g., light + tone) paired with the same reward, resulting in only the CS1 producing a conditioned response. The initial CS1-reward training is said to have prevented (blocked) the acquisition of CS2 as a reward predictor. Accordingly, dopamine release is abolished relative to the CS2, and this phenomenon is interpreted as a lack of prediction error signal for that stimulus (Waelti, Dickinson, & Schultz, 2001).

Steinberg and colleagues suggested that artificial stimulation of dopamine neurons (by means of optogenetic tools) at the time of reward delivery should mimic a prediction error signal and facilitate learning about the CS2. Indeed, a significant improvement of performance for the CS2 was observed following optogenetic stimulation of dopamine neurons. The authors interpret this result to indicate that there is a causal relationship between the reward prediction error signal and Pavlovian learning. However, such a conclusion might be premature. If the CS2 was not learned in the traditional blocking paradigm, it would be difficult to explain the recovery from blocking shown following extinction of the CS1-reward association (Blaisdell, Gunther, & Miller, 1999). Several hundreds of extinction trials are often necessary to generate responding to the CS2, but the ability to recover from blocking is a clear indication that the CS2-reward association was learned. Recovery has also been demonstrated in the case of overshadowing, where the debilitated response to an overshadowed stimulus does not consist of a learning failure, but rather of a failure to demonstrate that acquisition in behavior (Matzel, Schachtman, & Miller, 1985). Those effects might result from decreased incentive salience of the CS1, which camouflaged the motivational salience of

the CS2. Finally, reminder treatments (in which the animal is confronted with some portion of the learned situation—CS alone, UCS alone, or context—between training and testing) are also known to increase reactivity to an overshadowed or a blocked stimulus (e.g., Balaz, Gutsin, Cacheiro, & Miller, 1982; Kasprow, Cacheiro, Balaz, & Miller, 1982). Reminder treatments seem to recalibrate the incentive salience of both stimuli, increasing the salience of the CS2 relative to the CS1. In other words, the lack of responsiveness to the CS2 has another origin than the inability to learn the meaning of that stimulus. Given the strong involvement of dopamine in "wanting," we argue that the CS2 is simply disregarded (but nevertheless learned) because it brings no new information about the reward. The CS1 appears as a much more effective predictor because its reliability was already established when the CS2 was not yet present. As a result, animals are not motivated to respond to the CS2, as reflected by the absence of dopamine release for that stimulus (Waelti et al., 2001).

The activity pattern of dopamine neurons highlighted by the proponents of the reward prediction error hypothesis can easily be interpreted in motivational terms. For example, the abolition of dopamine release following fully predictable rewards can be explained by the dullness triggered by certainty, undermining incentive motivation (Anselme, 2013; Anselme, Robinson, & Berridge, 2013). In contrast, the unpredictability of unexpected and uncertain rewards attracts interest and elicits motivation. Finally, the depression in dopamine responses when the expected reward is not delivered may demonstrate motivational disappointment (Berridge, 2012). A number of findings support the view that dopamine responses reflect an animal's motivation (and performance) rather than learning. For example, DD mice (which produce no dopamine) and hyperdopaminergic mutant mice (which overproduce dopamine) learn Pavlovian associations as well as normal mice (Cannon & Bseikri, 2004; Peciña et al., 2003). In autoshaping, it is known that phasic activity of dopamine neurons in response to repeated reward delivery decreases in sign-trackers but remains unchanged in goal-trackers (Flagel et al., 2011). In terms of the reward prediction error hypothesis, this should mean that sign-trackers exhibit more effective learning of the task than goal-trackers. However, this interpretation does not explain why the progression of conditioned response rates is similar in both phenotypes over training (Meyer et al., 2012). Finally, as aforementioned, a fluctuation in internal state or dopamine levels causes an instant shift in an animal's motivation and performance, despite an absence of additional training (M. J. F. Robinson & Berridge, 2013; Tindell et al., 2005, 2009). In contrast, the reward prediction error hypothesis incorrectly posits that a modulation of performance is only possible provided that the CS-reward association is gradually relearned in the new motivational state (McClure, Daw, & Montague, 2003). Performance is controlled by more variables than just learning. Nonetheless, a recent study by Saddoris and colleagues using electrochemical methods to measure dopamine release during a sequential goal-directed task claims that, while dopamine release in the shell portion of the nucleus accumbens was characteristic of incentive salience, the pattern of dopamine signaling in the core may possess signature elements of prediction error coding (Saddoris, Cacciapaglia, Wightman, & Carelli, 2015).

The case of reward uncertainty also provides a good example of the dissociation between Pavlovian learning and Pavlovian performance, highlighting the role of "wanting" and dopamine in the latter. Reward uncertainty is commonly believed to be aversive—that is, humans and nonhumans avoid it when they can. After all, uncertainty about a reward you "want" is unlikely to make you happy or be desirable. Crucially, reward uncertainty can be considered as a degraded form of reward learning. The CS fails to reliably predict the UCS and therefore should be imparted with only limited predictive value. With limited predicted value or associative strength, it might be expected that an uncertain CS would become only mildly attractive. This is based on the premise that the incentive salience or degree to which a CS is "wanted" is determined by its predictive value. Instead, research shows that the predictive value and incentive salience of a CS are independent and that uncertainty may actually promote and recruit incentive salience under several conditions (Anselme et al., 2013; M. J. F. Robinson, Anselme, Fischer, & Berridge, 2014; Tindell et al., 2005). Notably, the parameters under which reward uncertainty promotes the attribution of incentive salience appear to mirror many of the hallmarks of gambling (Anselme et al., 2013; Linnet et al., 2012; Lobo et al., 2010; van Holst, van den Brink, Veltman, & Goudriaan, 2010). In Pavlovian autoshaping, "wanting" or attraction to reward-related cues expressed in the form of conditioned responding is known to reach a higher asymptotic level when reward delivery is uncertain rather than certain (Anselme et al., 2013; Boakes, 1977; Collins, Young, Davies, & Pearce, 1983; Davey, Cleland, & Oakley, 1982; Gibbon, Farrell, Locurto, Duncan, & Terrace, 1980; Gottlieb, 2004; M. J. F. Robinson, Anselme, et al., 2014). It has even been shown that not knowing how hard one must work to be rewarded may even sensitize the brain in ways similar to addictive drugs (Singer, Scott-Railton, & Vezina, 2012). Conversely, sensitizing the mesolimbic reward system with repeated amphetamine injections increases cue "wanting" similarly to reward uncertainty (M. J. F. Robinson, Anselme, Suchomel, & Berridge, 2015).

Alternative interpretations suggest that enhanced responding under uncertainty is a consequence of the animal's frustration caused by the occasional absence of reward (Amsel, 1958; Papini, 2003). Frustration occurs when an expected reward is absent or reduced (in amount or in concentration), such as modeled using the successive negative contrast procedure—where animals trained to obtain a 32% sucrose solution are tested with a 4% sucrose solution (Flaherty, 1996). However, this interpretation is hardly compatible with neurophysiological data showing that mesolimbic dopamine is released in the presence of a CS that predicts an uncertain reward (Dreher, Kohn, & Berman, 2006; Fiorillo, Tobler, & Schultz, 2003). Indeed, successive negative contrast is associated with an attenuation of dopamine release (Genn, Ahn, & Phillips, 2004). Also, it is conceptually unclear why maximal uncertainty (50% chance of reward) should generate frustration since the probabilities of reward and of nonreward are equivalent, resulting in an absence of expectation (Anselme, 2015). Instead, it is likely that reward uncertainty, in Pavlovian situations, enhances the "wanting" for a CS. Of course, this is not to say that uncertain rewards are preferable to certain rewards. One of us has recently suggested that the surge of "wanting" associated with uncertainty might result from the (incentive) hope that a reward will be delivered on the next trial (Anselme, 2015). Incentive hope is assumed to result from the additional "wanting" value associated with a nonguaranteed reward, because obtaining a reward when there is a possibility of missing it makes that reward more attrac-

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tive. The hope-induced "wanting" adds its effect to that of normal "wanting" for that reward, so that uncertain rewards end up becoming more "wanted" than certain rewards. Anselme posits that the motivational qualities of uncertainty are designed to compensate for the high rates of failure organisms experience when seeking resources (Anselme, 2013). Resources are typically randomly grouped and are rarely fully predicted by external cues. If unpredictability were not motivating, the inevitable repeated failure experienced when seeking reward would extinguish behavior. Consequently, this "wanting" caused by uncertainty is likely unconscious and not an independent source of pleasure. However when purposefully programmed or designed as the outcome of a game or slot machine, uncertainty could act as the match to ignite excessive "wanting" that arises beyond our cognitive control, and may promote unhealthy gambling behavior.

In humans, gambling is a potential source of addiction similar to that seen with drugs of abuse. Uncertainty while gambling generates increases in mesolimbic dopamine release (Boileau et al., 2014; Hart, Clark, & Phillips, 2015; Linnet et al., 2012) and this dopaminergic signal appears to promote risk-seeking behavior (Fiorillo, 2011), and correlates with the severity of problem gambling (Joutsa et al., 2012). For example, in a free-choice procedure, pramipexole, a dopamine $D_2/D_3$ receptor agonist, magnifies preference for a variable-ratio schedule (VR50) over a fixed-ratio schedule (FR5) in rats, despite being much less profitable in terms of energy costs (Johnson, Madden, Brewer, Pinkston, & Fowler, 2011). The stimulating effect of reward uncertainty on "wanting" may explain why gambling addicts attend more to their occasional wins and tend to ignore their much more considerable losses (Breen & Zuckerman, 1999). Instead these losses increase dopamine release in the ventral striatum of problem gamblers (Linnet, Peterson, Doudet, Gjedde, & Møller, 2010), particularly near-misses which despite being significantly less pleasant than full misses, trigger a greater urge to play (Clark, Lawrence, Astley-Jones, & Gray, 2009). Thus, although problem gamblers do not enjoy losses, they do find losses motivating, providing further evidence for a dissociation of "liking" and "wanting." There is also evidence to suggest that gamblers may bias their attention toward gambling-related cues as compared with healthy controls, suggesting that these stimuli are more "wanted" in human gamblers (Brevers et al., 2015; Brevers, Koritzky, Bechara, & Noël, 2014).

Given the primary role attributed to learning in conditioning since Pavlov's seminal work (Pavlov, 1927), it is unsurprising that most computational models of Pavlovian conditioning developed until recently were limited to characterizing performance in terms of associative/predictive learning (Mackintosh, 1975; McClure et al., 2003; Pearce & Hall, 1980; Rescorla & Wagner, 1972) or in terms of learning of time intervals (Gallistel & Gibbon, 2000; Gallistel & Balsam, 2014; Gibbon, 1977). Despite their merits, those models do not address important issues related to the effects of motivational processes on performance or to individual differences in responding—in particular, the existence of sign- and goal-trackers. As noted above, the observed variations in conditioned responding reflect differences in acquired incentive salience of the CS rather than differences in associative strength (Flagel et al., 2011; Flagel, Watson, Robinson, & Akil, 2007). For this reason, some researchers call for the development of "performance models" of learning (Meyer et al., 2012). For example, Lesaint and colleagues (Lesaint, Sigaud, Flagel, Robinson, & Khamassi, 2014) have suggested a computer model of performance in autoshaping that can simulate the spectrum of responses found with real animals, from pure sign-tracking to pure goal-tracking (see also Kaveri & Nakahara, 2014). In the model's current version, the proportion of behavioral phenotypes depends on a priori adjustments, but undoubtedly it is a first step in the understanding of individual differences observed in autoshaping. There are also recent attempts to explain how incentive motivation can control responding in Pavlovian contexts (Anselme, 2015; Berridge, 2012; Dayan & Berridge, 2014; Zhang, Berridge, Tindell, Smith, & Aldridge, 2009). Zhang and colleagues proposed a computer model in which the incentive salience of a CS can vary depending on moment-to-moment changes in an individual's physiological state (Zhang et al., 2009). This directly impacts the individual's sign-tracking responses, which can increase (under hunger or with dopamine agonists) or decrease (under satiety or with dopamine antagonists), independently of the opportunity to relearn the altered reward value. Although limited in scope, this model is the first illustration of the feasibility of a Pavlovian mechanism primarily based on motivational rather than learning principles. Finally, one of us developed a more complete Pavlovian model relying on motivational principles (Anselme, 2015). This model explains and predicts the effects of reward uncertainty on responding, as well as other phenomena such as Zhang et al.'s effect. In a revised version, the model is also able to account for individual differences in sign-tracking responses and for stimuli competition such as blocking and overshadowing (Anselme, under review). Currently, models that capture the unconscious nature of "wanting" and "liking" are yet to be built. However, such models are confronted with the difficulty of making a computer endowed with consciousness (e.g., Franklin, 1995; Picard, 1997).

There is also evidence to suggest that dopamine might not control motivation per se, but rather motor control or the exertion of effort. For example, low doses of dopaminergic antagonists such as haloperidol suppress approach behavior but do not alter food consumption (Cousins, Wei, & Salamone, 1994; Koch, Schmid, & Schnitzler, 2000; Ljungberg, 1990; Rusk & Cooper, 1994; Salamone, 1986; Salamone et al., 1996). If rats are given a choice between four food pellets behind a 44-cm barrier and two food pellets freely available, they climb the barrier on most trials to obtain the four pellets. However, after receiving a low dose of haloperidol, they often avoid the barrier and prefer the two-pellet option (Salamone, Cousins, & Bucher, 1994). This suggests that rats with reduced dopamine levels are less prone to exert effort. Beeler and colleagues tested the preference of normal (C57BL/6) mice and of dopamine-transporter knock-down (DATkd) mice for "cheap" food (requiring a low number of lever presses, FR20) or "expensive" food (requiring more and more lever presses, FR40-FR200). DATkd mice are characterized by elevated extracellular dopamine levels and increased tonic dopamine firing activity. They found that DATkd mice pressed more the high-cost lever than C57BL/6 mice, suggesting that dopamine increases the amount of effort the animals are willing to expend (Beeler, Daw, Frazier & Zhuang, 2010).

Without denying the role of dopamine in motor control, the distinction between effort and motivation might be a simple semantic issue (Wise, 2008). Indeed, disentangling a decrease in cost sensitivity from an increase in reward attractiveness is operationally difficult: Nonmotivated animals in a task are also not willing

to deploy effort. It is perhaps equivalent to say that haloperidol-injected rats are content with the two-pellet option because climbing the barrier for more pellets is too costly in terms of effort or because a larger food reward lost its attractiveness. It is also perhaps equivalent to say that DATkd mice accept to press the lever a greater number of times because they are less sensitive to energy cost or because they exhibit a greater attractiveness for food. An important question to ask here is: why would animals modulate their effort in a task if this modulation was not a consequence of the strength with which rewards are "wanted"?

In summary, associative learning is *necessary* to create a link between a CS and a UCS, and enable the attribution of incentive salience to a CS. But associative learning *does not* cause incentive salience attribution itself, nor does it determine the intensity of incentive motivation and behavioral performance in Pavlovian tasks. This is not to deny the existence of learned Pavlovian associations, but rather suggests that incentive salience plays a more direct role in controlling the intensity and flexibility of motivated behavior than associative learning does. Instead, dopamine-induced "wanting" is both necessary and sufficient to drive and elicit motivated behavior. In the same way that incentive motivation is generated and can fluctuate without new associative learning, it also does not require conscious awareness and higher cognitive processes.

## Relationships With Consciousness

The hypothesis that "wanting" and "liking" can exist as unconscious states is sometimes difficult to accept because what we want is typically expressed in the form of conscious goals and what we like is typically associated with pleasure and positive emotion as conscious feelings. Robbins and Everitt wrote that "[a] state of wanting normally requires an object that is desired and it is unclear how this presumably 'subconscious' process of wanting can be linked to representations of such specific goals" (Robbins & Everitt, 2007, p. 435). In addition, Di Chiara noted that suggesting that drug craving results from the unconscious process of incentive attribution is "an assumption that stands in contrast with the fact that craving, being a self-reported measure, is necessarily explicit in nature" (Di Chiara, 2002, p. 80). Similarly, with respect to "liking" as an unconscious emotion, some theorists have argued that "one cannot have an experience that is not experienced" or that "feelings are by definition conscious" (Clore, 1994, p. 285 and 290). Here we use a minimalist definition of consciousness as the ability to experience phenomena or to feel what happens—it is similar to Block's phenomenal consciousness (Block, 1995) or to what philosophers call qualia, which denote individual instances of subjective experience (e.g., Chalmers, 1995). This definition does not presuppose anything with respect to language and self for consciousness to exist. In this section, we present arguments and research that challenge the view that conscious experience is always required for the expression of motivation and emotion. This is not to say that consciousness (and cognition) cannot magnify the expression of "wanting" and "liking" and diversify the situations in which they can be experienced (e.g., an animal that does not possess consciousness is unlikely to be moved by Chopin's piano music). But we argue that consciousness (and cognition) is not responsible for their causation and that, in some cases, we can "want" or "like" specific events without any awareness of that processing.

### Decision-Making: Conscious Goals Are Not Motivations

Earlier, we reviewed evidence that incentive motivation (or "wanting") can exert direct control over behavioral performance, independently of the formation of specific cognitive representations. Animals and humans do not need any representation of food to be hungry, and hunger is sufficient to engage the hungry individual in food seeking. However, this does not address the possible role of cognitive motivations (or conscious goals), and their relationship to subcortical "wanting" processes. Arguments developed here indicate that conscious goals require first the recruitment of unconscious "wanting" for cognitive motivations to exist.

A classic study by Mischel and colleagues (Mischel, Ebbesen, & Zeiss, 1972; Mischel, Shoda, & Rodriguez, 1989) sheds some light on the distinction between cognitive goals and motivation, and more subcortical "wanting." They examined the ability of young preschool aged children to patiently refrain from consuming a tasty marshmallow placed in front of them, when promised that if they could wait, a second marshmallow would additionally be received when the experimenter returned later. Some children succeeded in resisting the tempting marshmallow in order to earn the extra one, while others succumbed to the immediate temptation. The video footage of the children who succumbed to temptation does not suggest a lack of any cognitive intention to refrain from eating the marshmallow placed in front of them. Instead, it highlights a degree of internal conflict, presumably between their cognitive intentions and motivation to wait, and a powerful urge to consume the "wanted" incentive placed in front of them. This impulsive urge to consume the immediate reward is unlikely to emanate from a cognitive origin or it would be just as easy to suppress, as the cognitive intention to wait was easy to generate, based on the instructions given by the experimenter. Instead we suggest that one important psychological process in temptation is incentive salience or "wanting," and it is a process that cognitive self-control must frequently wrestle with. It is interesting that follow-up studies of these children revealed that those who displayed the greatest amount of patience or ability to moderate or suppress their immediate "wants," later went on to have better life outcomes as measured by SAT scores, educational attainment, body mass index (BMI) and other life measures.

In most instances, people are capable to select and sustain behavior aimed at long-term goals that are in line with their reasoned/rational needs. However, hypersensitivity to momentary "wants" that contradict these goals can occur. This can happen to moderate degrees temporarily in ordinary life under conditions of appetite, stress, or emotional arousal. The degree of sensitivity of this unconscious "wanting" system is what makes an individual more vulnerable to the "magnetic" properties of reward cues, such as the smell of brownies or coffee brewing. These cues will trigger powerful "wants" that require the exertion of regular cognitive control to maintain behavior on an appropriate course. In most individuals this may mean merely having a second brownie when you know one was enough, or having another drink at the end of the night. Yet greater degrees of hypersensitivity can occur in more

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extreme conditions, the most dramatic case perhaps being drug addiction, although the same may also be true of other forms of addiction such as gambling disorder. In drug addicts, failure to maintain a cognitively desired goal such as remaining drug free is often the result of the irrepressible "wanting" for the drug (Bartlett, Hallin, Chapman, & Angrist, 1997; Leyton, 2007; Leyton et al., 2002). Drug addicts may be fully aware that taking the drug has deleterious effects on their health, and destroys their family, social, and professional lives. For these reasons, some of them exhibit strong cognitive desires to curtail their consumption or simply quit. Many addicts even voluntarily undergo specific treatments for drug addiction and partake in treatment programs. Nevertheless, 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 to reuse after cessation were approximately 60% within 3 months and at least 75% within 12 months (Hunt, Barnett, & Branch, 1971). For this reason, drug addiction is characterized as a chronic relapsing disorder, where relapse is the rule rather than the exception, and often occurs repeatedly. This suggests that high rates of relapse among addicts occur despite stated cognitive intentions to remain abstinent, in part because of the excessive "wanting" that results from the long-lasting sensitization of dopamine neurons caused by repeated drug administration (T. E. Robinson & Berridge, 1993). For example, addicts who reported sensitization to the psychotic effects of cocaine were shown to relapse with a higher probability (Bartlett et al., 1997). In this case, encounters with drug-related cues (or related vivid imagery) can trigger such powerful "wanting" for the drug that cognitive intentions to abstain may be overcome and the addict acts as if uncontrollably drawn to the drug and drug-taking, resulting in relapse. This excessive "wanting" is experienced as surges of motivation to obtain and consume the drug that can last even beyond the presence of drug-related cues or imagery. At times, this excessive "wanting" can give rise to feelings of craving, and although early evidence suggested that these cravings were infrequent in addicts and often unrelated to drug use (Carter & Tiffany, 1999; Tiffany & Conklin, 2000), more recent studies using ecological momentary assessment techniques (used to capture behavior in a naturalistic environment) show them to correlate strongly with drug use (Preston et al., 2009; Serre, Fatseas, Swendsen, & Auriacombe, 2015). However, excessive "wanting" 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 own behavior astonishing even to him or her. Here, unconscious "wants" would have the ability to override our cognitive pursuit of goals, which explains why addicts often report a sense of loss of control and little insight into their hunger for drugs and their cues (Goldstein et al., 2009). In addition, studies suggest that drug addiction results in compromised cognitive control, due in part to prefrontal cortex dysfunction and changes in glutamatergic transmission (Cornish & Kalivas, 2000; Kalivas, Lalumiere, Knackstedt, & Shen, 2009; Kalivas, Volkow, & Seamans, 2005). Impaired cognitive control would further facilitate the ability of unconscious "wants" to promote drug taking and relapse, and make abstinence harder to maintain.

Kahneman's distinction between "System 1" and "System 2" in his book *Thinking, Fast and Slow* is compatible with this view (Kahneman, 2011). System 1 is fast, affective, and impulsive, and it cannot be switched off, while System 2 is slow, deliberate, effortful, and requires attention (Berns, Laibson, & Loewenstein, 2007; Hoch & Loewenstein, 1991; Kahneman, 2003; Loewenstein, 1996; Strack, Werth, & Deutsch, 2006). Whereas System 2 is more patient and evaluates long-term goals, System 1 lives in the now, and makes a sharp distinction between immediate and future rewards. It is this first system that is believed to account for the choice of immediate rewards over long-term goals, largely because of the influence of motivational states, such as hunger, thirst, sexual arousal and especially drug craving, but also including negative emotions such as pain, exhaustion and stress. These motivational states and their associated cues often overwhelm other more cognitive goals stated by System 2 and thus produce shortsighted, impulsive behavior (Loewenstein, 1996). They activate the mesolimbic dopaminergic circuitry, and are responsible for "wanting" and cue-triggered "wanting," respectively. In consequence, the sight of certain rewards or the cues that predict them may trigger impulsive choice toward immediate rewards, that may often be "wanted" more than they are actually "liked," leading to overconsumption. Lades suggests that in cases of perfect self-control or in the absence of cue-triggered "wanting," there is a matching relationship between the expected pleasure of a reward and the motivation to consume it (Lades, 2011). In such situations, decisions are the product of reflective System 2. However when decisions are influenced by cue-triggered "wanting," this relationship breaks down and impulsive System 1 becomes the dominant driver of decisions. This is not to say that in the complex world we live in, surrounded by many cues at each point in time and driven by various need deprivation states, that System 1 controls all our actions. Instead, the use of willpower and self-control can neutralize those impulsive motivations. Self-control, however, is a resource that can get depleted so that urges tend to influence decisions more frequently (Baumeister, Bratslavsky, Muraven, & Tice, 1998; Vohs & Faber, 2007). This may be particularly the case in the sensitized brain of a drug addict where "wanting" and cue-triggered "wanting" may be particularly strong.

Furthermore, in a number of human decisions, it is difficult to determine whether one's choice will lead to benefit or harm (e.g., which job to apply for, whom to marry, which car to buy, etc.). These are situations where there exists no precedent to provide any remembered value, and where predicted value can be largely inaccurate. When the rewarding/punishing properties of several options cannot easily be assessed and contrasted, emotions have been shown to help us choose the best option. The somatic marker hypothesis (Damasio, 1994) suggests that when the number of factors to consider largely overcome the processing limits of working memory, good and bad affects such as "wanting" and "liking" play a role in guiding decisions through unconscious processing: "The mapping of bodily states at the subcortical level, such as within the mesolimbic dopamine system, occurs in a nonconscious fashion, such that subjects choose the advantageous option without feeling specific feelings of desire for that option or aversion to the disadvantageous option" (Naqvi, Shiv, & Bechara, 2006, p. 261). "Wanting" and "liking" may therefore either interact

with cognitive parameters or act as a default system for guiding decision-making.

Further evidence that "wanting" is an unconscious process that often evades cognitive rationale can be seen when examining the motivation assigned to reward-related cues. For example, detoxified heroin addicts show attentional biases to words related to their addiction on an emotional Stroop test (Franken, Booij, & van den Brink, 2005; Waters, Sayette, Franken, & Schwartz, 2005). Interestingly, the dopamine antagonist haloperidol, reduces that attentional bias, suggesting that "wanting" and particularly sensitized "wanting" in addicts can influence and direct attention. Further studies also show that this attentional bias toward drug-associated visual cues in drug addicts is correlated with self-reported craving (Field, Munafò, & Franken, 2009; Tibboel et al., 2011; Wiers & Stacy, 2006). In some cases, attribution of incentive salience to reward-predicting cues may make the cues "wanted" as much as the reward itself, irrespective of whether the reward constitutes an actual need. Such cues become motivational magnets, sometimes prompting irrational behavior, such as interactions with the cue specific to those previously seen only during interactions with the reward itself (Davey & Cleland, 1982). In animals this can be seen in autoshaping where pigeons might make eating pecks at a key-light CS predictive of a food UCS and perform drinking pecks when the same CS predicts water (Jenkins & Moore, 1973), or in male Japanese quail that under some circumstances will approach and copulate with an inanimate object CS previously paired with the opportunity to copulate with a female UCS (Burns & Domjan, 1996; Domjan, O'Vary, & Greene, 1988; Köksal et al., 2004). In humans, irrational cue attraction occurs in crack cocaine addicts found inspecting the floor for a white speck that is more likely to be an ordinary pebble than crack cocaine, and who can then be attracted to pick it up, inspect and put it in the pipe and even try to light or smoke the noncocaine pebble—a phenomenon that has been called "chasing ghosts" (Rosse et al., 1993). This type of behavior appears to defy more cognitive forms of motivation, because it will occur repeatedly despite the individual's conscious knowledge that although the cue possesses some of the reward's properties, it is not in fact the reward itself.

Goals get their motivational power from recruitment of subcortical brain regions involved in unconscious "wanting." Desires such as successful performance in a video game (Koepp et al., 1998) or the anticipation of possible wins in a gambling task release dopamine in the nucleus accumbens of human participants (Chase & Clark, 2010; Clark et al., 2009; Kassinove & Schare, 2001; Linnet et al., 2010). Of course, cortical regions are also recruited in such complex tasks, because participants are required to reason and make decisions about their behavior. But what motivates them in doing these tasks are the same subcortical processes as those observed with respect to food, sex, and drugs in rats (Gratton & Wise, 1994; Kiyatkin, Wise, & Gratton, 1993; Kosobud, Harris, & Chapin, 1994; Pfaus & Phillips, 1991). As in the case of food and sex, individual preferences may vary according to past experience and current disposition. But cognition accounts for nothing in the attribution of such preferences—"preferences need no inferences" (Zajonc, 1980). The attractiveness of a task, whether simple (seeking food) or complex (playing chess), depends on the ability of specific task-related stimuli to activate dopamine neurons of the ventral tegmental area.

In summary, "knowledge by itself, no matter what kind, is never motivation. Something else is required to translate remembered knowledge into motivation that can actually generate and control behavior" (Berridge, 2012, p. 1124). If our conscious goals, representations, and anticipations usually motivate us, it is because they are somehow interacting with "wanting" processes. Why do we have the strong impression that our motivations are a product of consciousness? One possibility is that human cognition (Kahneman's System 2) incessantly attempts to rationalize our thoughts, beliefs, and actions. Rationalizations are at the origin of the perception of our motivations as conscious goals. Sometimes, they may correctly identify the causes of unconscious "wanting" and "liking," but often they may fail to do so accurately. For example, in a consumerist society, many items such as recent advances in technology are "wanted" far more than they are needed, yet individuals will sometimes justify impulse purchases by arguing that they are needed. In the case of "wanting" and "liking," we argue that correctly identifying the cause of a particular behavior is often epiphenomenal to its occurrence, and does not mean consciousness was required to initially generate the behavior.

## Pleasures Without Conscious Feelings

Feelings are conscious states of the mind, and most theorists see pleasures (and emotions in general) as a subcategory of feelings (Cabanac, 2002; Clore, 1994; Frijda, 1999; James, 1884). Indeed, everyday experience tells us that pleasure is a product of consciousness. A number of works involving human participants have investigated the impact of dopamine on the subjective perception of pleasure (e.g., Laruelle et al., 1995; Sharot, Shiner, Brown, Fan, & Dolan, 2009; Volkow et al., 1995). For example, Drevets and colleagues used the compound [$^{11}$C]raclopride to assess the degree of dopamine binding to D2/D3 receptors under PET scanning and showed that dopamine is released before and after amphetamine injection (Drevets et al., 2001). They concluded that dopamine release in the nucleus accumbens is positively correlated with amphetamine-induced euphoria. However, the conclusions of neuroimaging studies with respect to causal relationships (here, dopamine causes pleasure) must be taken with caution because they provide only correlational results. Also, those studies do not attempt to disentangle the "wanting" from the "liking" components of reward or the conscious from the unconscious parts of reward sensitivity. Wise himself pointed out that "[b]rain imaging studies have indicated that stimulant-induced euphoria is loosely correlated with the degree of drug-induced dopamine release" (Wise, 2004, p. 4). The difficulty of finding strong causal relationships is even worse in the case of assessments based on verbal reports (questionnaires) by participants under the influence of dopaminergic drugs (e.g., Sharot et al., 2009), because the risk of misattribution is elevated—"I want it so I must like it." It is not here the place to discuss what an emotion is or is not, because—contrary to motivation—there is no scientific consensus on its definition (Frijda, 1999). However, the relationship between emotion and consciousness is relevant to this article in that "liking" is typically viewed as an unconscious emotion (Berridge & Winkielman, 2003). Here we would like to briefly review the evidence for why emotions may result from unconscious processes and how these processes might be related to consciousness.

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Primary emotions (disgust, surprise, fear, joy, sadness, and anger) are typically distinguished from secondary emotions (jealousy, shame, empathy, pride, etc.). Secondary emotions are traditionally not believed to exist other than in primates, although dogs and horses might possess a restricted range of them (Morris, Doe, & Godsell, 2008). Although consciousness and emotion come together in humans, it is often difficult to determine if an animal is aware that an emotion is being experienced in phylogenetically distant animal species, such as fish and reptiles. However, we will see that apparent emotions dissociated from conscious experience can be produced in humans under specific conditions, suggesting that being aware of what is felt is unnecessary to be emotionally aroused. Less problematic is the view that emotions originate in brain processing systems inaccessible to consciousness. Even in individuals equipped with cortical structures, there is no reason to believe that those structures are involved in causing emotions. Brain damage to cortical regions is relatively ineffective in abolishing emotional reactions, and their activation fails to produce strong emotional responses (Berridge & Winkielman, 2003). According to Damasio, cortex only conveys the cognitive components of emotion induction and regulation, such as representations, anticipations, and decision strategies (Damasio, 1999). Cabanac posited that emotion is a conscious experience that is felt by mammals, birds, and reptiles because animals from these zoological classes exhibit episodes of stress-induced fever and tachycardia when handled by humans (Cabanac, 2005). In contrast, amphibians and fish do not show such emotional reactions, suggesting that they are not conscious and have no emotion. However, the debate over this question is probably more complex than that. For example, zebrafish (*Danio rerio*) are commonly used as an animal model for the study of fear and anxiety (Blaser, Chadwick, & McGinnis, 2010; Egan et al., 2009). Even octopuses might possess a form of primary consciousness (Mather, 2008). The absence of cortical structures in these animal species certainly means that, contrary to higher vertebrates, they lack the representational components of emotion. Because of that, simpler creatures cannot feel emotions in the way we experience them. But it is likely that they are (unconsciously) *affected* by the presence of specific stimuli in their environment, causing them to approach or avoid those stimuli.

The fact that emotion generally takes place in the field of consciousness does not mean that it is a product of consciousness (Damasio, 1999). We are sometimes unable to say why we feel happy some days and sad other days because we simply failed to consciously identify the stimuli (e.g., the furtive perception of a stimulus previously associated with a pleasant or painful experience) at the origin of these psychological states. Pleasures were not shaped by evolution to embellish our lives; they exist because they allow organisms to distinguish what is good and bad for survival. For example, all mammals and birds like sugar, because it is a potent source of energy. In contrast, acid and sour tastes are typically disliked because, in nature, they are often associated with fermentation and with the presence of toxins. Approach and avoidance behaviors based on what is "liked" and "disliked" are necessary for the survival of all organisms, including those for which consciousness is unlikely to exist. Accumulated evidence suggests that a number of invertebrate species (at least, among arthropods) can experience stress and pain in ways that seem neurochemically similar to vertebrates (Bateson, Desire, Gartside, & Wright, 2011; Elwood, Barr, & Patterson, 2009). Conscious feelings appear to be the perception of core psychological states that emerged from the depth of ancestral brain structures, shaped before the emergence of modern conscious life forms.

Several studies have succeeded in altering emotional states in human participants who remained unaware of the causation of this change (Jiang & He, 2006; Monahan, Murphy, & Zajonc, 2000; Öhman, Flykt, & Lundqvist, 2000). However, the defenders of the conscious emotion hypothesis might argue that an unconscious causation does not mean that emotional reactions are unconscious as well. Yet, as described previously, Winkielman and colleagues showed that both the cause of and the reaction to an emotional stimulus are sometimes unconscious. For example, thirsty participants exposed to subliminal presentations of happy faces pour more juice in their cup, drink more of it, rate its taste higher, and are willing to pay more for it than participants exposed to subliminal presentations of angry faces (Skurnik et al., 2000; Winkielman et al., 2005; Winkielman & Berridge, 2003). It is important that those participants reported no change in their emotional feelings. The suggestion that the absence of emotional changes noted here simply reflects an absence of emotional reactivity cannot explain why the effects of subliminal facial expressions were only observed in thirsty participants, a finding that indicates that basic motivational/emotional processes are involved. This important result means that "subliminally presented emotional faces can cause affective reactions that alter consumption behavior, without eliciting conscious feelings at the moment the affective reactions are caused" (Winkielman & Berridge, 2004, p. 122). Further supporting evidence comes from a case study of a patient who lost the ability to perceive taste as a result of severe bilateral lesions of the insula (Adolphs, Tranel, Koenigs, & Damasio, 2005). The patient in question could not consciously distinguish solutions that contained sugar, salt, or lime juice and drank them indiscriminately. However, when asked to choose among them, the patient showed a strong preference for the sugar solution while remaining unaware of the emotion induced. This supports the idea that a particular reward can be strongly "liked," resulting in it being "wanted" more than the alternatives, despite a lack of any conscious awareness or the ability to verbalize the source or the existence of a preference.

## Conclusion

Although conscious processes influence which rewards we want and like, the evidence presented here highlights the powerful role played by subcortical forms of "wanting" and "liking." These two core processes are involved in guiding our attention, and decision-making despite often remaining below the threshold of consciousness. The limited introspection into their functioning explains why our actions may at times seem foreign even to ourselves. This is especially the case in extreme situations such as drug addiction, where incentive sensitization can lead to excessive "wanting" and incentive salience attribution to reward-related cues, sometimes despite limited expected and reported pleasure. Yet cue-triggered urges to seek out and consume particular rewards are not restricted to pathological situations of addiction and overconsumption. Our behavior can be driven by unconscious processes even in more regular everyday instances, as when trying to resist the temptation sparked by the sight of the dessert menu. As such we propose that "wanting" and "liking" work below the level of consciousness to

generate and direct motivated behavior, which can then be modulated and influenced by conscious goals and pleasures.

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