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The Missing Piece between Learning and Behavior

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Abstract: In the behavioral sciences, it is common to explain behavior in terms of what was learned in a task, as if any subsequent change in performance had to denote a change in learning. However, learning alone cannot account for variability in performance. Instead, incentive motivation plays a direct role (and is more effective) in controlling moment-to-moment changes in an individual's responses than the learning process. After briefly introducing the history of the study of incentive motivation, we explain that incentive motivation consists of a dopamine-dependent process that does not require consciousness to influence responding to a task. We analyze two Pavlovian situations in which incentive motivation can modulate performance, irrespective of additional learning: the instant transformation of disgust into attraction for salt and the invigoration of responses under reward uncertainty. Finally, we consider drug addiction as an example of motivational dysregulation rather than as a consequence of the habit to consume substances of abuse.

Traditionally, motivation is viewed as a conscious goal that leads us to learn to perform specific actions in order to reach need-related, pleasurable rewards. Although this definition may seem intuitive, it fails to capture the subtle relationships that exist between motivation, learning, and behavior. Here, we present the concept of incentive motivation (called the incentive salience hypothesis), showing how motivational processes are produced in the brain, their potential dissociation from pleasure and learning, and the evidence that their computation occurs in the absence of conscious awareness. Basically, incentive motivation is the psychological process that makes specific stimuli (e.g., food, sex, money, games) attractive, approached, and physically contacted. We show how the incentive salience hypothesis can explain specific phenomena that are more problematic for typical reinforcement learning theories. In particular, we discuss how a sudden change in physiological state can transform disgust into attraction without additional learning and the invigorating effect of reward uncertainty on Pavlovian responses – which occur when

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an individual comes to respond to the presentation of a stimulus (e.g., lever, light, sound) that predicts the delivery of reward (e.g., food).

What Is Incentive Motivation?

Incentive motivation is the psychological process that transforms the "cold" memory of stimuli into appetizing incentives (or rewards), such as a glass of fresh water for a thirsty person. This process is responsible for reward attraction, which consists of approaching conditioned cues and unconditioned rewards, and is often referred to as "wanting" (Berridge & Robinson, 1998). The modern incentive interpretation originated from the works of Bolles, Bindra, and Toates (Bindra, 1976; Bolles, 1972; Toates, 1986), and differs from the concept of incentive that was initially formulated within the drive theory (Hull, 1943; Spence, 1956). Here we present a short historical background showing why and how incentive theories have replaced drive theories.

Historical Background

Drive theory, many versions of which were proposed throughout the twentieth century, posits that a need for specific rewards (e.g., food, water, sex) induces a motivational drive that urges organisms to get those rewards. As their consumption satisfies the need in question, need satisfaction is accompanied with drive reduction. Drive concepts all describe motivation as an energizing, homeostatic process. This simple view fits well the intuitive description people have about motivational changes, especially with respect to hunger and thirst. However, despite undeniable successes, only the convenience of its use can explain why this interpretation has been maintained for so long in the scientific literature. A number of empirical data provide evidence against the existence of drive (Archer, 1988; Bodor, Rice, Farley, Swalm, & Rose, 2010; Hinde, 1960; Holst & Saint Paul, 1963; Laumann, Gagnon, Michael, & Michaels, 1994; McFarland, 1969; Robinson, Burghardt, Patterson, Nobile, Akil, et al., 2015b; Valenstein, Cox, & Kakolewski, 1970). For example, drive reduction does not prevent motivation. The intravenous administration of nutrients or the introduction of food and water directly into the stomach by means of a gastric fistula should reduce the drive associated with hunger and thirst. Yet, such treatments are ineffective at reducing appetite in animals and humans (Miller & Kessen, 1952; Myers & Hall, 1998; Turner, Solomon, Stellar, & Wampler, 1975; Wolf & Wolff, 1943).

The accumulation of findings that could not be explained by drive theory suggested the need for a theoretical shift. Bolles (1972) initiated this change, suggesting that motivation originated in incentive expectancies (pleasure anticipation) rather than in drive induction. He emphasized the relevance

of stimulus–stimulus (S–S) associations; drive theories were only focused on stimulus–response (S–R) associations. He argued that a conditioned stimulus (CS) (e.g., a light) acquires its incentive properties due to repeated pairing with a hedonic reward (e.g., food), because it caused the expectancy of that reward. However, it remained unclear how expectancy alone could produce motivation rather than simply a passive anticipation of reward. Therefore Bindra adopted Bolles' incentive prediction approach, while rejecting the idea that expectation was the critical factor for motivation (Bindra, 1976). Instead, he suggested that a CS not only acquires a predictive value, but also the incentive motivational state normally caused by the unconditioned stimulus (UCS) with which it was repeatedly paired. The acquisition of incentive motivation properties can explain why CSs are approached when associated with appetitive UCSs (such as food) and avoided when associated with aversive UCSs (such as shock). However, in Bindra's conceptual framework, the motivational salience of CSs became permanent once acquired; a CS predictive of food should always be attractive whether hungry or sated. Yet it was clear that the internal physiological state of an individual is important to motivation, even if physiological drive alone cannot account for motivation. Accordingly, Toates (1986) proposed and showed that an individual's physiological state modulates the incentive value of a stimulus, whether it is a CS or a UCS. While drive theorists also discussed the incentive property of a stimulus (Hull, 1943; Spence, 1956), to them, incentives were independent of the motivation (drive) for the stimulus. Instead, in the modern incentive perspective (Bindra, 1976; Berridge & Robinson, 1998), the incentive salience of a stimulus directly depends on motivational strength, which possesses a "magnetic" (reward attraction) rather than "energetic" effect (need-triggered behavior). As such, hunger will make the smell of baked goods attractive, rather than simply trigger an increase in sniffing for odorants.

The Bolles–Bindra–Toates model of incentive motivation presupposes that the incentive value of a CS is a consequence of the hedonic impact of the UCS. However, this presumption has revealed itself to be untrue: the incentive salience of a CS ("wanting"), although often influenced and informed by the hedonic reactions ("liking") felt during consumption of the reward UCS or the learning of the CS-UCS association, is a separate psychological and neuroanatomical process (Berridge & Robinson, 1998; Robinson & Berridge, 1993). This raises the following questions: How is incentive motivation controlled in the brain? Does it depend on consciousness? And why is it not equivalent to the anticipated pleasure of the reward or the strength of the learned association between the predictor and the reward? We discuss these questions and then describe their implications for the understanding of behavioral performance and addiction, especially in the context of human gambling (for additional details on the history of motivational theory, see Berridge, 2004; Robinson & Berridge, 2001).

The Role of Dopamine in Motivation

For centuries, it had been believed that motivation and pleasure were two sides of the same coin and, early in the 1980s, some findings suggested that the neurotransmitter dopamine was strongly involved in pleasurable experiences (Wise, 1982). For example, when rats were injected with a dopamine antagonist (which reduces the action of dopamine in the brain), they stopped seeking rewards such as food. The belief was that dopamine antagonists abolished pleasure, thereby causing a loss of motivation for previously pleasurable rewards. In contrast, more thorough studies gave us a new insight of dopamine's role: Reward is not a unitary process, and dopamine only influences the motivational component of reward ("wanting"), not the hedonic reactions ("liking") or the predictive learning component (Berridge & Robinson, 1998; Robinson & Berridge, 1993). Dopamine-deficient (DD) mice – whose brains produce no dopamine – would die of starvation, even if hungry and surrounded by appetizing food, because they simply do not "want" to approach it. But when food is placed directly into their mouths, DD mice enjoy and ingest the same amount of food and learn Pavlovian associations as well as normal mice (Cannon & Bseikri, 2004; Peciña, Cagniard, Berridge, Aldridge, & Zhuang, 2003). In contrast, hyperdopaminergic (DATkd) mice, which overproduce extra-cellular dopamine, exhibit a greater incentive performance for sucrose and proceed more directly to the goal in a runway, but show no enhanced "liking" reactions to sweet tastes (Peciña et al., 2003). These mice do tend to learn the CS-UCS association more rapidly, although this is likely due to high motivation facilitating learning speed – as it does in both animal training and human learning.

Although other neurotransmitters and brain regions are known to play a role in reward (Ikemoto, 2010), mesolimbic dopamine has been shown to be both necessary and sufficient to alter incentive motivation (Berridge, 2007, 2012). However, we recognize that the incentive salience hypothesis is not the only interpretation of dopamine's role in reward (Salamone & Correa, 2002; Schultz, 1998; Wise, 1982). Alternative views of dopamine's role have been extensively discussed in the literature and cannot be presented in detail in the present chapter. One example, known as the prediction error view, sees dopamine as a learning signal (Rescorla & Wagner, 1972; Schultz, 1998). It is based on evidence that phasic dopamine release is high after the delivery of unexpected rewards, but gradually reduces to baseline levels as conditioning and acquisition of a predictive cue progress. This view suggests that dopamine is a prediction error signal used to correct (and learn from) inaccurate predictions (Schultz, 1998; 2010). However, if we assumed that any change in performance necessarily resulted from a change in learning, any immediate shift in performance, as might be adaptive following a sudden physiological change, would be impossible in the absence of new learning. However, recent evidence (explored in further detail later in the chapter) suggests that a dramatic change

in incentive motivation can occur in rats suddenly placed in a state of salt deprivation (Robinson & Berridge, 2013). Indeed, the reward prediction error hypothesis implies that a modulation of performance should only occur if the CS-reward association is gradually relearned in the new motivational state (McClure, Daw, & Montague, 2003). In addition, the prediction error hypothesis has trouble accounting for the enhanced conditioned responding seen under uncertainty, because the predicted higher dopamine release is not assumed to reflect a motivational process. In fact, if dopamine was a teaching or learning signal, animals should perform less under uncertainty, as predicted by the Rescorla-Wagner model of learning (Rescorla & Wagner, 1972).

Yet another theory proposes to interpret dopamine as a neurotransmitter involved in motor control or the exertion of effort (Salamone & Correa, 2002). This hypothesis relies on data showing that low doses of dopamine antagonists (such as haloperidol) reduce lever pressing, running speed, and the propensity of rats to expend additional effort (e.g., climb a barrier) for more palatable food (Cousins, Sokolowski, & Salamone, 1993; Ikemoto & Panksepp, 1996; Salamone, Cousins, & Bucher, 1994). Evidence from patients with Parkinson's disease (characterized by a difficulty in initiating motor movements) who present a degeneration of the substantia nigra (a midbrain nucleus that produces dopamine) lends support to this view. But, overall, it is hard to find evidence for effort-control theory that cannot also be accounted for by incentive motivation theory: 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 addition, an increase in dopamine function tends to promote gambling behavior in Parkinson's patients (Dodd et al., 2005; Voon et al., 2006), which the effort-control theory is unlikely to capture, along with the specific effects described in more detail later (uncertainty and salt depletion).

Incentive Motivation as an Unconscious Process

Contrary to a widespread idea that desire comes only with conscious experience, we can "want" stimuli in the absence of any subjective consciousness. For example, recovering addicts were asked to freely choose between two intravenous injections: one of them contained cocaine (lowest dose: 4 mg; highest dose: 50 mg) and the other was a saline solution (Fischman & Foltin, 1992). Addicts 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 for cocaine than with saline, and no cardiovascular responses were observed. 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." Similarly, irrational cue attraction occurs in crack cocaine addicts who, when found inspecting the floor for a white speck

that is more likely to be an ordinary pebble than crack cocaine, can then be attracted to pick it up, inspect it, and even try to smoke the non-cocaine pebble (Rosse et al., 1993). This type of behavior appears to defy more cognitive forms of motivation; it will occur repeatedly despite the individual's conscious knowledge that, although it possesses some of the reward's properties, the cue is not, in fact, the reward itself. In animals, this can be seen in Pavlovian autoshaping – a procedure in which the presentation of a CS is automatically followed by limited delivery of food – where pigeons might make eating pecks at a keylight (CS) predictive of a food (UCS) and perform drinking pecks when the same CS predicts water (Jenkins & Moore, 1973). Another example is provided by male Japanese quail that, under some circumstances, will approach and copulate with an inanimate object CS that had been previously paired with the opportunity to copulate with a female UCS (Burns & Domjan, 1996; Domjan, O'Vary, & Greene, 1988).

Of course, this is not to say that cognitive processes have no impact on motivation. For example, a learned expectation may magnify the attractiveness of a reward. But an expectation is not a motivation per se; it can be expressed independently of any kind of motivation. Some experiments have shown that young children have some expectations about the world and exhibit surprise when these expectations are violated (Baillargeon, 1987; Woodward, Phillips, & Spelke, 1993). Their reactions depend on their internal model of the world's laws, rather than on their motivation for the manipulated objects. In contrast, "wanting" computations might be necessary to desire something one expects. If I wish to go to a movie, I may have some conscious expectations about a specific movie or about the good time spent doing this activity. As a result, I can infer that my wish to go to a movie is also conscious. But this desire would be unlikely to exist if the deep subcortical structures involved in "wanting" were not activated. 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, Lawrence, Astley-Jones, & Gray, 2009; Kassinove & Schare, 2001; Linnet, Peterson, Doudet, Gjedde, & Møller, 2010). 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 in the ventral tegmental area. To date, no "desire area" has ever been found in the cortical structures, although unconscious "wanting" can naturally alter cortical processing of information (Belayachi et al., 2015).

In this respect, incentive motivation may be related to human interest for specific topics, where this interest could be classified as a psychological state that predisposes individuals to re-engage contents that apply to various contexts of life (Hidi & Renninger, 2006; Renninger & Hidi, 2016). Contrary to traditional reward-directed motivational processes, interest typically occurs in the absence of potential external reward (e.g., money, food); it is a self-reinforcing activity. Interest combines motivational, emotional, attentional, learning, and

cognitive components, in addition to being a conscious state of mind. But current evidence suggests that incentive motivation is the core process controlling its occurrence and development. Panksepp (1998) had initially proposed that interest was related to a "seeking" system: a hypothetical dopamine-dependent brain architecture allowing animals and humans to explore their environment in order to find rewards. Further research has confirmed this general view, as dopamine seems to be involved in each developmental phase of interest (Hidi & Renninger, 2006; Renninger & Hidi, 2016). In addition, although interest is correlated with some knowledge of a topic, knowledge itself is neither necessary nor sufficient to trigger it (Renninger, 2000; Renninger, Ewen, & Lasher, 2002). Some people may come to be interested in something while having little knowledge of it (e.g., the origin of the universe for a non-physicist) or have detailed knowledge of a topic for which they develop no interest at all (e.g., the Highway Code). Even well-developed interests, supported by strong background knowledge, require more than cortical activity – as the brain signature of cognitive processing of information (Panksepp, 1998). In short, despite its complex psychological organization, interest might directly depend on the activation of the reward circuitry in the brain.

Why do we have the strong impression that our motivations are a product of consciousness? One possibility is that human cognition incessantly attempts to rationalize 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," but often they fail to do so accurately (Nisbett & Wilson, 1977). 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 (Litt, Khan, & Shiv, 2010; Robinson & Berridge, 2015). 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.

Learning and Performance

Psychologists have long noted that learning and performance are two distinct components of behavior. Hull (1943), for example, suggested that two major causes of behavior are learned habits and a non-specific motivational drive, which was assumed to provide "energy" for action. Other early theorists criticized the concept of drive (Hinde, 1960; Young, 1961) while recognizing that the explanation of performance requires a strong motivation concept. In human studies, the distinction established between effort and competence (Nicholls, 1984) or between learning goal and performance goal (Dweck & Leggett, 1988) also reflects the emphasis on learning and performance as two different, complementary aspects of behavior.

Surprisingly, however, a number of modern interpretations of behavior tend to forget the role of motivation in controlling performance, presupposing that learning rules are sufficient to capture it. The influential Rescorla-Wagner model of associative learning is at the origin of those interpretations (Rescorla & Wagner, 1972). This model predicts that the association between a CS and an UCS is strengthened from trial to trial (based on an error correction principle) and that the gradual enhancement of conditioned responding to a CS (performance) simply reflects the strength of the CS-UCS association (learning and acquisition). This presumption can be problematic, given that a change in learning can only be inferred from a change in performance (because learning is not measurable directly). In fact, many other processes (e.g., emotions, motivations) can influence performance besides learning, and perhaps more directly. Many current learning models are based on the temporal difference (TD) algorithm, where the difference between what is predicted and the actual outcome is translated into neuronal activity. This neuronal activity is believed to act as an error signal that would help modify future predictions in order to reduce that error. However, current learning models derived or inspired by the TD algorithm reduce performance to learning in quite a similar way to early models (Glimcher, 2011; McClure et al., 2003; Redish, Jensen, & Johnson, 2008; Schultz, Dayan, & Montague, 1997). Error-correction mechanisms are supported by empirical findings that activity of dopamine neurons in the ventral tegmental area correlates with that prediction error signal (for reviews, see Schultz, 1998, 2010). Indeed, dopaminergic activity reaches higher levels than background activity when a reward occurs unexpectedly (positive prediction error), lower levels when a reward does not occur at the expected time (negative prediction error), and remains stable when reward occurrence fits the predicted time (Dreher, Kohn, & Berman, 2006; Fiorillo, Tobler, & Schultz, 2003). Some authors therefore assume that mesolimbic dopamine codes how much learning is required to complete a prediction task rather than being involved in motivation.

However, failing to differentiate between learning and performance has negative implications for our understanding of the mechanisms of behavior. A classic example of how learning alone cannot fully account for the degree of motivation registered through performance is the so-called Crespi effect (Crespi, 1942). In this seminal study, rats from three groups were initially trained to run for 1, 16, or 64 food morsels. At test, all the rats ran for 16 food morsels. Crespi observed that their running speed depended on the contrast between the current amount of reward (16 morsels) and the number of morsels received during training, rather than being based solely on the current learned outcome of the task. Running speed therefore increased for those animals that were initially trained on 1 morsel of food and then tested with 16 food morsels (1-16 group) and decreased in the 64-16 group, while speed remained constant in the 16-16 group. The fact that running speed was related to the reward magnitude obtained on previous trials cannot be the simple consequence of a

learning process, because the rats of all groups received the same amount of training and were given the same amount of learning of the new contingency. These results were later attributed to the incentive properties of rewards by Tolman, Hull, and Spence (Hull, 1943; Spence, 1956; Tolman, 1949).

Two recent illustrations of the necessity to distinguish performance from learning come from our own work. The first one examines the instant shift in Pavlovian responses that follows a physiological change, such as a sudden state of deprivation. It is known that when an animal progressively learns that a particular cue predicts a positive outcome UCS (reward), it will learn to approach and be attracted to that cue. The same is true of avoiding a cue paired with a negative outcome UCS (punishment). However, if the animal learns to associate a cue with an unpleasant outcome, but this outcome then becomes suddenly necessary for its survival, performance can instantly change without having to slowly re-evaluate the association (i.e., without the requirement of any further learning). Robinson and Berridge trained salt non-deprived rats to receive a 9 percent salty solution (three times the concentration of seawater) in their mouths by means of oral cannulas predicted by the presentation of a lever CS (Robinson & Berridge, 2013). These 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 (deoxycorticosterone and furosemide) to produce an intense state of sodium deficiency, and they were placed again in the test chambers. Despite their highly aversive past experience with the lever CS, and having never tasted the salt solution as anything other than disgusting, 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. This result suggests that incentive motivation can instantly transform a learned association from disgust into attraction, independently of the learned value of the CS and without requiring any additional learning of the new contingencies (for a related experiment based on electrophysiological recordings, see Tindell, Smith, Berridge, & Aldridge, 2009).

A second example of how performance and motivation can be independent of learning comes from the invigorating effect of reward uncertainty on Pavlovian responses. Learning models suggest that the stronger the predictive value of a CS regarding its outcome UCS (reward or punishment), the more the animal is inclined to respond to the predictive CS (approach or avoidance). In this view, a CS that predicts UCS delivery with 100 percent probability (certainty) should lead animals to produce stronger conditioned responses than a CS that unreliably predicts UCS delivery (uncertainty: 50 percent probability). However, a number of studies have revealed that an unreliable CS enhances responding compared to a reliable CS (Anselme, Robinson, & Berridge, 2013; Collins, Young, Davies, & Pearce, 1983; Gottlieb, 2004; Robinson, Anselme, Fischer, & Berridge, 2014a). Thus, this effect shows something uncaptured by learning theories. For example, we accustomed rats to obtaining a sucrose

pellet on each trial during three autoshaping sessions, where a trial consisted of one pellet delivered after short presentation of a lever and tone CS. In this procedure, rats spontaneously come to approach, sniff, nibble, and press the available lever – a behavior called sign-tracking (by opposition to the goal-tracking propensity of other individuals, who prefer to approach and interact with the food dish). Sign-tracking behavior is used as a measure of attraction and incentive motivation attributed to a reward-related cue. After three days of training under certain conditions, half of the rats were switched to uncertain conditions, while the other half were maintained on the previous certainty schedule for five additional days. Under uncertainty, animals received nothing on 50 percent of trials and one, two, or three pellets, on a random basis, on the remaining 50 percent of trials, in contrast to certain conditions where animals received one pellet on 100 percent of trials. The number of reward pellets and cue presentations were perfectly matched between the certain and uncertain conditions, so that any differences could not be accounted for by differences in the amount of reinforcement or the number of learning pairings (cue presentations). Although sign-tracking performance was similar in the two groups on training days one through three, rats exposed to uncertain pellets quickly came to approach the lever faster and pressed and nibbled the lever at higher rates than rats exposed to certain conditions (Anselme et al., 2013). The opportunity to receive larger rewards (three pellets) was not the reason for enhanced performance under uncertainty. In fact, receiving one, two, or three pellets per trial at random, without omission of reward delivery, made no difference in the rats' attraction to the lever, compared to a situation of 100 percent chance of only one pellet per trial (Anselme et al., 2013).

Homeostatic mechanisms can readily explain why a state of deprivation would momentarily increase motivation, such as why desire for sweet and salty foods may result from a prolonged period without anything to eat. However, the link between food uncertainty and motivation may seem a bit counterintuitive: Logically, a degraded CS-UCS association should reduce the attractiveness of the CS. Yet, some behavioral findings suggest that invigorated responding under uncertainty results from increased incentive motivation (or "wanting") for the CSs. For example, rats trained under reward uncertainty sign-track toward a lever CS located farther from the food dish than rats trained under reward certainty, suggesting that the CS has acquired a greater capacity to attract attention and motivated behavior (Robinson et al., 2014a). In the same vein, responding decreases toward the end of a fully predictable CS, but does not when the CS is unreliable (Gibbon, Farrell, Locurto, Duncan, & Terrace, 1980). After training the same pigeons in uncertainty versus certainty conditions, it appeared that they chose to peck at the CS previously associated with uncertainty more than that previously associated with certainty (Collins & Pearce, 1985). Finally, reward uncertainty generates a higher number of sign-trackers and stronger sign-tracking responses than reward certainty (Robinson, Anselme, Suchomel, & Berridge, 2015a).

Of course, we are not trying to say that animals prefer uncertainty to certainty, but rather that *unavoidable* uncertainty enhances food-seeking motivation. A hypothesis allowing us to understand the motivational effects of reward uncertainty is that of incentive hope (Anselme, 2015, 2016). To hope something is to "want" it, while having no guarantee that it will be obtained. In this view, organisms "want" uncertain rewards (just as certain rewards), but they also "hope" for their delivery. Incentive hope adds its motivational effect to that of hunger-induced motivation and makes the food reward more attractive when it is eventually obtained. Increased attractiveness of uncertain rewards is unlikely to be learned in the traditional sense but might sensitize brain mechanisms in a similar way to drugs of abuse. For example, we showed that rats initially exposed to high uncertainty maintained more vigorous CS-directed behaviors after uncertainty was dramatically reduced (Robinson et al., 2014a). This somewhat irrational behavior might play a determining role in the problematic attraction to gambling-related cues, particularly in slot-machine gambling (Anselme et al., 2013). However, the motivational effects of reward uncertainty could basically be an adaptive process, allowing animals experiencing unpredictable food access to seek and consume more food items than if those items were easy to find (Anselme, 2013, 2016). A large body of literature in behavioral ecology indicates that, when exposed to unpredictable food in many different conditions (e.g., winter, social subordination, poor foraging capacity), animals (especially small birds) consume more food and become fatter (Cresswell, 2003; Ekman & Hake, 1990; Gosler, 1996; Pravosudov & Grubb, 1997). Higher fat reserves are thought to act as insurance against the risk of starvation, given that more time and energy must be spent when food density is low. The incentive hope hypothesis is a plausible mechanism for explaining *how* increased seeking is possible. Taken together, these findings suggest not only that uncertainty increases reward-seeking motivation, but also that this process was put in place by evolution due to its usefulness for survival.

In support of our claim that uncertainty has motivational properties that enhance "wanting," it is interesting to note that uncertainty processing requires dopamine. Indeed, a number of studies have revealed that midbrain dopamine release is higher when uncertainty of a CS is maximal (de Lafuente & Romo, 2011; Dreher et al., 2006; Fiorillo et al., 2003; Hart, Clark, & Phillips, 2015; Preuschoff, Bossaerts, & Quartz, 2006; Singer, Scott-Railton, & Vezina, 2012; Tan & Bullock, 2008; Zack, Featherstone, Mathewson, & Fletcher, 2014). We found that uncertainty elevates sign-tracking in a similar fashion to amphetamine, a dopamine agonist-like drug known to increase motivation (Robinson, Anselme, Suchomel, & Berridge, 2015a). D'Souza and Duvauchelle (2008) showed that rats exposed to visual and olfactory cues previously associated with uncertain cocaine access (self-administration) exhibited enhanced extracellular dopamine release compared to rats for which those cues fully predicted cocaine access. The incentive hope hypothesis fits well with those results,

because hope can easily be understood as a motivational factor: Hope is what motivates people to seek longer or more intensely. It is therefore unsurprising that it contributes to increasing incentive salience as a dopaminergic process.

Upon the assumption that human interest is basically an incentive motivational process (see Section 1.3), it can be hypothesized that individuals are more likely to develop incentive hope relative to a topic of interest. For example, Costikyan (2013), a game developer, points out that the reason many people like to play games is that the uncertainty associated with the outcome or with the game's path holds their interest. More thorough investigation is needed, but there is some evidence that playing a video game activates the brain reward circuit (Koepp et al., 1998).

In conclusion, considering Pavlovian conditioning from a pure learning perspective is unsatisfactory. Learning is a necessary step, allowing the CS to acquire some predictive value, but this predictive value is not, in itself, what controls the observed performance. Performance is a consequence of the individual's motivation in the task. In particular, incentive motivation is a crucial factor capable of explaining the instant shift and the reward uncertainty effects.

Addictions: The Dysregulation of Motivational Processes

Addiction is characterized by the compulsive pursuit of a specific reward at the detriment of others. It often occurs despite repeated adverse (e.g., health, social, legal) consequences, and is characterized by excessive "wanting" for certain rewards and their cues, often referred to as incentive sensitization. Evidence suggests that addiction, whether for drugs, food, sex or gambling, involves the sensitization of dopamine neurons in subcortical structures that permanently alter an individual's capacity for making appropriate decisions about many aspects of everyday life (Robinson & Berridge, 1993, 2008). The excessive desire and craving that result from sensitized motivational systems is believed to make addiction a pathological problem, followed by frequent relapse, despite repeated intentions to quit. Addiction is often portrayed as a powerful habit (Everitt & Robbins, 2015) that develops as a transition from recreational use to compulsive reward-seeking. Through repeated learning and "stamping-in" of reward associations, neural activity is believed to shift from the ventral to the dorsal striatum. However, although it may be true that reward consumption can become an overly ritualized habitual act, this may be less true of the preceding motivation to obtain the reward. Learned habits alone cannot account for the excessive motivational attraction of rewards and their cues that develops through addiction. The idea that addiction is merely a rigid S–R habit does not account for how motivation imbues the act of drug-taking with characteristic flexibility and innovation of new means of obtaining the reward when required. Nor does it explain the compulsive overtones that cannot be easily overridden by the resolution to abstain. Other extremely well-learned

habits (such as brushing one's teeth or tying one's shoelaces) are not compulsive in the motivational sense. Those habits can easily be left undone or halted midway without the emergence of a compulsive urge to continue. Furthermore, recent evidence suggests that the learning of strong associations does not mean that well-learned predictors carry the most incentive motivation. Instead, as can be seen with gambling, highly uncertain and therefore weak predictors can invigorate motivation and at times sensitize reward pathways (Linnet et al., 2010; Robinson, Anselme, Suchomel, & Berridge, 2015a).

Addiction has also been associated with a loss of cognitive control (Robinson, Fischer, Ahuja, Lesser, & Maniates, 2015c; Robinson, Robinson, & Berridge, 2014b). The excessive motivation for drugs and other rewards may go beyond the ability to use rational thought to influence decisions. In particular, it appears that decisions are no longer linked to learned and experienced outcomes. Whereas addiction is often accompanied by sensitization of "wanting" systems, evidence seems to suggest that the pleasure or "liking" associated with the reward is either reduced (through a process known as tolerance) or stays relatively the same. As a result, addicts often require ever-increasing consumption and abuse of the reward to attain near-equivalent levels of euphoria. In addition, addiction is often accompanied by growing negative outcomes, including loss of family and social connections, loss of employment, increased legal problems, health issues, and (in some cases) powerful withdrawal symptoms. If learning were at the root of motivation, these growing adverse effects would progressively come to outweigh the benefits of the addiction and would attenuate use, mitigating any prior overconsumption. Instead, increased adverse effects fail to deter compulsive use. One report even suggested that within a group of inpatients treated for cocaine rehabilitation, those that reported growing negative side effects (sensitization) over the course of their history of drug use (in this case paranoid psychosis) were most likely to relapse, as indicated by re-hospitalization for addiction treatment (Bartlett, Hallin, Chapman, & Angrist, 1997). These results suggest that negative outcomes no longer seem able to shape the direction of motivation and, in extreme cases in which negative outcomes outweigh the pleasure of the reward, the addict may perpetually struggle to relearn old or learn new healthy behaviors to overcome their addiction.

One reason addicts struggle with using the rise of adverse consequences to overcome addiction is that the desire to pursue the reward has become pathological. Excessive "wanting" for the reward and its cues is believed to result from incentive sensitization, where cues and the reward develop the ability to trigger increasingly intense peaks in craving that make pursuit of the reward almost irresistible. However, evidence initially gathered from animal models of addiction has shown that sensitization of "wanting" is not restricted to the reward of choice. In fact, repeated exposure to a particular reward can result in a sensitized response to other rewards – a phenomenon known as cross-sensitization, which implies a greater response to a treatment, even on

the first exposure, due to prior experience with another treatment. For example, cross-sensitization occurs between drugs of different classes. So pretreatment with either amphetamine or nicotine can accelerate the acquisition of cocaine self-administration. Cross-sensitization also occurs between drugs and stress, where repeated stress can produce greater motivation to consume amphetamine. Finally, cross-sensitization can occur between drugs and natural rewards or between drugs and gambling. For example, amphetamine sensitization can lead to sugar hyperphagia (and the reverse is true of intermittent sugar consumption; Avena & Hoebel, 2003a, 2003b) and, in humans, pathological gamblers show a greater dopamine response to amphetamine, which is correlated with gambling severity (Boileau et al., 2013). Cross-sensitization is further evidence that motivation can occur relatively independently of prior learning. Thus, although addiction may involve disorders of learning and the creation of powerful habits, learning alone is unable to explain the excessive desire and motivation that addicts experience for their rewards and its cues.

Concluding Thoughts

Motivation is directed. For motivation to be more than a sudden peak in activity, a target is required, upon which motivation can be focused. In most cases this target requires learning. Individuals must learn to associate certain cues or actions with a specific reward for it to be imbued with motivation. However, learning alone does not generate motivation. As we have shown here, the weak association between a cue and a reward that results from uncertainty can actually enhance motivation. This is contrary to the predictions that would be made by learning theories. In addition, as we showed with the case of salt depletion, motivation can be generated spontaneously, even out of disgust, without any new learning, simply through a sudden change in physiological state. Finally, although addiction is accompanied by powerful learned habits, these habits cannot account for the excessive "wanting" that develops with addiction and is responsible for the compulsive, and often flexible, pursuit of reward and the craving that can lead to relapse even after years of abstinence.

Outstanding questions include the following:

- What are the subjective feelings of problem gamblers playing their favorite game?
- Which neurotransmitters (other than dopamine) influence gambling behavior?
- Is the preference often shown for variable schedules controlled by the same brain processes as the higher response rates shown in Pavlovian autoshaping?
- Is there a link between the interest in a topic and the hope of obtaining responses to questions related to that topic?
- Small birds exposed to an unpredictable food access come to consume more food, but is this activity correlated with enhanced dopamine release?

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