

Decision utility, incentive salience, and cue-triggered 'wanting'

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"This is good news and bad news for utilitarians: the limbic system reward pathways seem to correspond to a utility pump, but specialized brain circuitry processes experience in ways that are not necessarily consistent with relentless maximization of hedonic experience."

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How do brain representations of the utility of rewards guide decisions about whether to pursue them? Our focus here will be on brain reward 'utility pumps' operating at particular moments in life. Moments such as when you encounter an image, sound, scent or other cue associated in your past with a particular reward; or perhaps just vividly imagine that cue. Such a cue can often trigger a sudden motivational urge to pursue that reward, and sometimes a decision to do so. In drug addicts trying to quit, a cue for the addicted drug might trigger urges that rise to compulsive levels of intensity, despite prior commitments to abstain, leading to the decision to relapse into taking the drug again. Normal or addicted, the urge and decision may well have been lacking immediately before the cue was encountered. The decision to pursue the cued reward might never have happened if the cue had not been encountered. Why can such cues momentarily dominate decision making?

This question has both psychological and neural answers, and it may be useful to consider them together. In particular we think a full psychological answer involves a particular subtype of reward utility. To help make this answer clear in terms of utility pumps, we will draw on a utility taxonomy that distinguishes among subtypes of predicted utility, decision utility, experienced utility, and remembered utility subtypes (Kahneman et al., 1997). We will show how cue-triggered cravings such as the addict's surrender to relapse above can hang on special transformations of one utility subtype, namely, decision utility, and only decision utility, which corresponds to incentive salience. Sudden peak of intensity of incentive salience, by neurobiological mechanisms to be described, can elevate decision utility of a particular reward at the moment its cue occurs. An understanding of what happens at such moments will lead to a better understanding of the mechanisms at work at all decision moments.

Decisions and reward utility types

A good decision is to choose and pursue an outcome that is liked best when it is gained, from among all available options. That is, a good decision maximizes reward utility. However, reward utility is not all of one type. To identify the types of reward utility involved in cue-triggered decisions, we draw here on a 4-type utility framework proposed by Daniel Kahneman and colleagues: predicted utility, decision utility, experienced utility, and remembered utility (Kahneman et al., 1997).

First, predicted utility: predicted utility is the expectation of how much a future reward will be liked. It is based upon cognitive or associative prediction of the rewarding value an outcome will have when it is gained in the future.

Decision utility is the subtype of reward utility most directly connected to an actual decision, but also the most difficult to isolate in psychological terms from other subtypes (especially from predicted utility). As the name suggests, decision utility is the essence

of an actual decision, the valuation of the outcome manifest in choice and pursuit. Most typically, it is revealed by what we decide to do.

Experienced utility is what most people think of the term reward. It is the hedonic impact of the reward that is actually experienced when it is finally gained. It is the affective pleasure components of reward utility. For many, experienced utility is the essence of what reward is all about.

Remembered utility is the memory of how good a previous reward was in the past. It is the reconstructed representation of the hedonic impact carried by the remembered reward. Whenever we decide about outcomes we have previously experienced in our past, remembered utility is perhaps the chief factor determining predicted utility: we generally expect future rewards to be about as good as they have been in the past.

A major contribution of Kahneman's utility taxonomy has been to identify cases where predicted or remembered utility diverges from actual experienced utility (Gilbert and Wilson, 2000; Kahneman et al., 1993; Kahneman et al., 1997). Such divergence can lead to bad decisions, called miswanting by Gilbert and Wilson (Gilbert and Wilson, 2000; Morewedge et al., 2005). If one has a distorted remembered utility, due to memory illusions of various sorts, one will have a distorted predicted utility. Decisions made on the basis of false predicted utility are likely to turn out to fail to maximize eventual experienced utility. Or if predicted utility is distorted for reasons other than faulty memory, such as by inappropriate cognitive theories about what rewards will be like in future, then decisions will again turn out wrong. In either case, predicted utility will fail to match actual experienced utility, and the decision is liable to be wrong.

Thus if decisions are guided principally by expectations about future reward (decision utility = predicted utility), then faulty expectations means that wrong

decisions will be made (decision utility \neq experienced utility). We may thus choose outcomes that we turn out not to like. We choose them because we wrongly expect to like them (and perhaps because we wrongly remember having liked them in the past) – but then we turn out not to like them after all.

The mismatch above captures much of what is discussed under the label of miswanting and decisions that fail to maximize utility. But Kahneman's taxonomy has a further use, which we will exploit here, in understanding another form of miswanting that can occur even when an outcome value is correctly predicted. We suggest it does not necessarily have to be the case that decision utility matches predicted utility (Berridge, 1999, 2003a; Robinson and Berridge, 1993). If decision utility exists as a separate term (with a somewhat separate neurobiological pump), it might sometimes dissociate from predicted utility – just as decision utility (with predicted utility) sometimes dissociates from experienced utility (Berridge, 1999, 2003; Robinson & Berridge, 1993). If at any time decision utility could grow above predicted utility, that could mean choosing an outcome that we actually expected not to like at the moment of decision -- not only that we turned out not to like in the end.

Rational decisions versus irrational decisions

This brings us squarely to the topic of decision rationality. Decision rationality has been defined in various ways, so we wish to be clear about our own definition. First, we do not demand consistency of preference. For psychologists and neuroscientists, there are many good reasons why individual preferences will change from time to time, and so we would not call irrational any mere inconsistency of preference. Second, we'd also suggest the rationality of a decision has nothing at all to do with whether an impartial judge or the majority of other people would like the same outcome. Individual tastes are idiosyncratic. As the adage goes, there is no use disputing about tastes. For the purpose of decision rationality we simply accept

individual tastes for what they are – differences in individual characteristics of experienced utility that make different things liked by different people (or even by the same person at different times).

Further, the rationality of a decision does not even depend on whether or not deciders themselves end up eventually liking their chosen outcome. Deciders can be mistaken about whether they will like an outcome they choose, as in mispredicted miswanting mentioned above. People often choose an outcome they expect to like, but then are disappointed to find they actually don't like it. That is not irrational – in those cases choosers may have done the best they can – they were simply wrong in their expectations of predicted utility. Reasons for being wrong about the predicted utility of an outcome can include ignorance for never-experienced outcomes, incorrect theories about the goodness of a hypothetical outcome or about one's own hedonic tastes, and mistaken memories about having liked something in the past (incorrect remembered utility)(Gilbert and Wilson, 2000; Kahneman et al., 1997). All these can make a decision mistaken, wrong, bad and regrettable – even stupid. But by themselves they do not make a decision irrational, however wrong the decision turns out to be.

We suggest that a decision remains rational as long as one chooses what one expects to like best. That is, as long as decision utility = predicted utility. If predicted utility of an outcome is high, then choosing that outcome is rational by definition. If you believe you will like an outcome, you are rational to choose it, to want it, and to pursue it actively – you should pursue it precisely to the degree you expect to like it. If you turn out not to like the outcome after all, well, blame your theories, memories, or understanding of the world. But decision rationality cannot be held responsible for the eventual unhappy experienced utility, because rationality in this sense cannot be held accountable for the accuracy of your predictions --- only for

the consistency with which you act upon them.

An irrational decision is to choose what you expect not to like. That is, a decision is irrational when its decision utility \neq predicted utility. When decision utility $>$ predicted utility, if that can happen, then one might be said to choose what one does not expect to like – and not only what one actually does not like. To choose what one does not expect to like is to choose in a way that is strongly irrational. For the purpose of identifying irrational decision mechanisms in experiments below, this is the definition we will rely on: that one chooses disproportionately to expectation of liking, so that decision utility $>$ predicted utility. Here, we will describe a mechanism that under specific conditions produces irrational decisions, even by a definition of irrationality that is so restrictive, though we believe it evolved to motivate good decisions in normal life.

Brain and reward utility

Insights into rewards and decisions would be enhanced by an understanding of their brain mechanisms. Affective neuroscience studies of reward have shown that many brain structures are activated by reward utilities (Berridge, 2003b; Davidson et al., 2004; Kringelbach, 2004; Montague et al., 2004; Schultz, 2006; Shizgal, 1999; Shizgal et al., 2001). These include regions of the neocortex, especially prefrontal cortex ventromedial, orbitofrontal, and anterior cingulate areas), and insular cortex (which includes taste sensory representations), and the amygdala.

But what brain mechanisms actually cause reward in the sense of pumping experienced utility? So far, the most potent causal demonstrations for actually causing reward utility have come chiefly from manipulations of brain structures below the cortex: subcortical limbic structures (Berridge, 2003b; Shizgal, 1999). We will focus our analysis of experienced utility and decision utility generation on these subcortical structures, such as mesolimbic dopamine

systems, nucleus accumbens, and ventral pallidum.

But before we focus on details, we must emphasize that neither cortical nor subcortical regions operate on their own, and that massive reciprocal projections link them together. Connections from subcortical to cortical regions are undoubtedly required for translation of 'liking' and other basic utilities generated in subcortical limbic structures into consciousness and cognitive representations. In return, descending projections from cortex to subcortical limbic structures permit cognitive appraisals or voluntary intentions to modulate basic emotional reactions (Davidson, Jackson, & Kalin, 2000). Still, if one has to choose a just few brain mechanisms as causal generators of reward utility, the best candidates for utility pumps come mostly from below the cortex.

Brain utility pumps

Perhaps the most famous subcortical reward generating substrate has been the mesolimbic dopamine system that sends its dopamine-containing fibers up from midbrain, through the lateral hypothalamus, and to the nucleus accumbens and related structures. The nucleus accumbens in turn projects heavily downwards, most densely above all to the ventral pallidum, a relatively little known but highly intriguing limbic structure that sits just in front of the lateral hypothalamus near the bottom of the forebrain. The ventral pallidum projects back upwards into thalamo-cortical circuits that reach orbitofrontal cortex, cingulate cortex and insular cortex, as well as downwards to deeper brain structures. This looping mesolimbic dopamine-accumbens-pallidum-cortical system is a useful brain circuit to turn to in order to tease apart reward utility types.

*Do strongly irrational decisions exist?
(decision utility > predicted utility)*

The point of our subcortical focus is to show how decision utility, especially as cue-triggered 'wanting' might be generated by brain systems in particular circumstances. To start with, you might well wonder, are there really any cases where people irrationally want what they neither like nor expect to like? We think there may be some cases generated by subcortical manipulations, though these cases have not always been recognized for what they are. A good example might be false 'pleasure electrodes', perhaps a case of neuroscientific mistaken identity.

False Pleasure Electrodes – Decision utility without experienced utility?

Pleasure electrodes have been famous since the 1950s, but may generally turn out not to live up to their name. These are stimulation electrodes in the subcortical forebrain, which rats and people would work to stimulate, pressing a lever or button thousands of times in a few hours to activate (Heath, 1972; Olds and Milner, 1954; Shizgal, 1999). What intense pleasure (experienced utility) and expectations of pleasure (predicted utility) must occur in order to motivate such intense wanting to activate the electrode (decision utility)! Or so you might think.

But maybe 'pleasure electrodes' aren't so pleasurable after all. For example, one of the most famous cases ever was "B-19", implanted with stimulation electrodes by Heath and colleagues as a young man in the 1960s (Heath, 1972). B-19 voraciously self-stimulated his electrode, and protested when the stimulation button was taken away. In addition, his electrode caused "feelings of pleasure, alertness, and warmth (goodwill); he had feelings of sexual arousal and described a compulsion to masturbate" (p. 6, Heath, 1972). Still, did B19's electrode really cause an intense pleasure sensation? The answer seems to be no! B-19 was never quoted as saying the sensation was pleasurable in the papers and books written by Heath; not even an exclamation or anything like "Oh -- that feels nice!"

Rather than simple pleasure, stimulation of B19's electrode evoked the desire to

stimulate again and again, along with strong sexual arousal. It never produced actual sexual orgasm or clear evidence of actual pleasure sensation. Clearly the stimulation did not serve as a substitute for sexual acts.

Decades later, another patient showed similar findings, this time a woman with an electrode implanted in deep subcortical forebrain (Portenoy et al., 1986). She stimulated her electrode at home compulsively to the extent that "At its most frequent, the patient self-stimulated throughout the day, neglecting personal hygiene and family commitments" (p. 279, Portenoy et al., 1986). When her electrode was stimulated in the clinic, it produced a strong desire to drink liquids, and some erotic feelings, as well as a continuing desire to stimulate again. Notably, records indicate that "Though sexual arousal was prominent, no orgasm occurred" (p. 279, Portenoy et al., 1986). "She described erotic sensations often intermixed with an undercurrent of anxiety. She also noted extreme thirst, drinking copiously during the session, and alternating generalized hot and cold sensations" (p. 282, Portenoy et al., 1986). Clearly this woman felt a mixture of subjective feelings, but the description's emphasis is on aversive thirst and anxiety. Like patient B19, there is no evidence of distinct pleasure sensations. Although stimulation made B19 want to perform sexual acts and the woman had erotic thoughts, neither patient had orgasmic sensations. In contrast to the failure of these brain stimulating electrodes, stimulating the spinal cord has been shown to actually improve sexual function by enhancing orgasmic performance (Meloy and Southern, 2006).

What could brain stimulation be doing, if not inducing pleasure? This helps pinpoint the idea of incentive salience, a psychological process of reward 'wanting' that is a form of decision utility (Berridge, 2003a; Berridge and Robinson, 1998; Robinson and Berridge, 1993). It is different from reward 'liking' or pleasurable hedonic impact that corresponds to experienced

utility. We suggest that brain stimulation in these patients only evoked intense 'wanting' – but not 'liking'. Brain stimulation caused incentive salience to be attributed to stimuli perceived at surrounding moments, including people (who became more interesting and appealing), the room (which became attractive and 'brightened up'), and most especially the button and the act of pushing it (which became irresistible to do again). The button itself is most closely paired with electrode activation, and so becomes a conditioned stimulus attributed most with incentive salience. If brain stimulation elevated 'wanting' attribution to the button as a form of decision utility without a corresponding increase in experienced utility, a person might well 'want' to activate their electrode again and again, even if it produced no pleasure sensation. That would be mere incentive salience 'wanting' – without hedonic 'liking'.

Does the electrode hijack decision utility alone as we suggest? Or does it also hijack predicted utility as well as decision utility, causing false expectations of future reward? That is, the electrodes might produce a false declarative expectation that the activation will produce an intensely liked pleasure, even though the last one never did. If so, then both predicted utility and decision utility would exceed the eventually experienced utility, or lack of pleasure actually received. We will return to this issue in the animal affective neuroscience experiments below.

One would like to know more about the experience, expectations, and motives of these people with brain self-stimulation. The information available from past studies of patients is frustratingly sparse and crude. It is possible that better information might be gathered in future, now that a revival of deep brain stimulation and electrode implantation appears to be underway (e.g., as experimental treatment for Parkinson's disease). Better information is something to be hoped for.

Animal affective neuroscience experiments: isolating decision utility

Some better information can be gained from affective neuroscience experiments with animals. Our own analyses of reward utility types began over a decade ago in part with an animal equivalent of the electrode patients. In rats as well as people, 'rewarding' brain electrodes typically turn on motivations to eat, drink, have sex, etc. if the electrode stimulation is given freely. Why do rats eat, say, when a reward electrode is turned on? Early hypotheses suggested that they ate more food because stimulation made them like food more (Hoebel, 1988). But a study with Elliot Valenstein led to the different conclusion that the electrode increased the incentive salience or decision utility of food, causing rats to 'want' to eat it, without increasing 'liking' for its hedonic impact or experienced utility (Berridge and Valenstein, 1991).

How can 'wanting' and 'liking' possibly be told apart in rats? We've tackled this by assessing affective reactions that are very specific to hedonic impact 'liking' (Figure 1). They are not influenced by independent changes in 'wanting'. The affective reactions are 'liking' facial expressions that are elicited sweet tastes, of which several expressions are homologous in human infants and many animals including apes, monkeys, and rats (e.g., tongue protrusions). By contrast, nasty bitter tastes elicit 'disliking' expressions (e.g., gapes). Such affective 'liking'/'disliking' reactions provide windows into brain systems that paint a pleasure gloss onto sweet and related taste sensations, because the expressions change when brain manipulations alter the pleasant hedonic impact of those tastes.

Using this approach studies by Susana Peciña, Kyle Smith, Stephen Mahler, Sheila Reynolds, Alexis Faure, Casey Cromwell and others in our laboratory have begun to map neural substrates that generate basic experienced utility for sweetness hedonic impact (Mahler et al., 2004; Pecina and Berridge, 2005; Peciña and Berridge, 2000; Reynolds and Berridge, 2002; Smith and Berridge, 2005). They have identified a

number of neuroanatomical hotspots and neurochemical signals which are able to cause increases in the hedonic impact of sweetness 'liking' (Figure 1). Such experiments use painless microinjections, delivered by previously implanted brain cannulae, to activate a brain substrate. Tiny droplets of morphine-type drugs (called opiate drugs because they activate opioid brain chemicals) are delivered to a hotspot in a brain structure such as nucleus accumbens, where they activate the opioid circuits and cause increased hedonic 'liking' reactions to the sweet taste of sugar. By moving microinjections to different locations in the structure, we can map the boundaries of the hedonic hotspot, and by varying the drug content we can identify the neurochemical systems that paint the pleasure gloss of this basic experienced utility onto sweet sensation.

For opioid neurotransmitters, hedonic hotspots exist in subcortical limbic structures such as the nucleus accumbens and the ventral pallidum (Pecina and Berridge, 2005; Smith and Berridge, 2005). In these hotspots, microinjection of the drug DAMGO activates a type of receptor for natural opioid neurotransmitter chemicals, called mu opioid receptors, on neurons within a millimeter of the microinjection. The neural sphere of stimulating mu opioid activation causes sweet tastes to elicit double or triple the number of positive hedonic 'liking' reactions they normally would. In other words, DAMGO in these hotspots activates an experienced utility pump that magnifies the pleasure impact of sweet tastes to make them more 'liked'. These experiments have helped us identify the places and neurochemical systems in the brain that amplify experienced utility, in the form of basic 'liking' reaction to natural sensory pleasure. At the same time, the microinjections that cause 'liking' also cause greater 'wanting': the rats seek out food and eat three times as much as normal. Thus hedonic hotspot opioid circuits typically amplify experienced utility and decision utility simultaneously together as a combined package.

But by contrast to opioid microinjections that induce both 'liking' and 'wanting' for rewards, deep brain electrode stimulation that makes rats eat more nonetheless fails to increase 'liking' reactions to sweetness (Berridge and Valenstein, 1991). If anything, it caused more 'disliking' reactions (as observed with bitter tastes) were observed instead. In other words, the rats don't seem to eat more because they 'like' food more. Instead, rats eat more despite not 'liking' it more or even in some instances, actually 'disliking' food more. This seems to be a separation among utility types for food reward: increased decision utility ('wanting' and food consumption) without increased experienced utility ('liking' reactions to sugar).

We have observed a number of other similar brain causes that increase motivational 'wanting', but failed to increase pleasure ('liking') for the same reward (Reynolds and Berridge, 2002; Robinson and Berridge, 2003; Wyvell and Berridge, 2000; Wyvell and Berridge, 2001). Many of these brain manipulations that dissociate decision utility from experienced utility have involved the brain dopamine system, which was once thought to cause sensory pleasure. Our work, combined with other neuroscience evidence, has led to the contrary conclusion that dopamine fails to live up to its pleasure neurotransmitter label. Dopamine systems simply seem unable to cause pleasure, as assessed by 'liking' reactions, unless accompanied by other neural events, even though dopamine activation can induce powerful motivation to acquire food and other rewards in animals and humans. We've tried both activating and suppressing dopamine in several ways, but it never alters pleasure reactions (Berridge and Robinson, 1998; Tindell et al., 2005).

So if dopamine is a faux-pleasure, what is its real psychological role? Our studies led us to suggest that modulating reward 'wanting' rather than 'liking', best captures what dopamine does. In particular, by 'wanting' we mean the attribution of

incentive salience to reward stimuli, which makes them be perceived as attractive incentives (Berridge and Robinson, 1998; Tindell et al., 2005). For us in our everyday experience, 'liking' and 'wanting' usually go together for pleasant rewards, as two sides of the same psychological coin. But 'wanting' may be separable in the brain from 'liking', and mesolimbic dopamine systems mediate only 'wanting'. We and our colleagues coined the phrase incentive salience for the particular psychological form of 'wanting' we think is mediated by brain dopamine systems.

What is 'incentive salience'?

"Wanting" is not "liking." "Wanting" is not a sensory pleasure in any sense. And "wanting" cannot increase positive facial reactions to sweet taste, or the hedonic impact of any sensory pleasure. Indeed, incentive salience is essentially nonhedonic in nature, even though it is important to the larger composite of processes that motivate us for reward. Faced with a number of goals (e.g., thirst vs. hunger), "wanting" evolved to serve as a means to make decisions among different types of rewards (e.g., water vs. food). Thus, "wanting" provides common neural currency or a comparison standard (Shizgal, 1997). Usually "liking" and "wanting" for pleasant incentives do go together, virtually as two sides of the same coin. But, "wanting" remains a distinct motivational process.

We believe that brain dopamine systems especially attribute incentive salience to reward representations at moments when a cue is encountered that has been associated with the reward in the past (or perhaps even vividly imagined). Incentive salience is attributed to Pavlovian cues following what have been called Binda-Toates rules of learned incentive motivation (Berridge, 2001; Binda, 1978; Toates, 1986). When a cue is attributed with incentive salience by mesolimbic brain systems, it causes that cue and its reward both to become momentarily more intensely attractive and sought. The cue actually takes on 'motivational magnet' properties of its reward: it becomes almost

ingestible if it is a cue for food reward, drinkable if a cue for water reward, attractive in a drug-related way for cues for drug reward, and so on (and animals have been known to try to eat or drink their incentive cues in studies of what is called Pavlovian 'autoshaping'). The cue also is able to trigger increased 'wanting' for its actual reward, priming the motivational desire in cue-triggered 'wanting'.

Physiological drive states such as hunger or thirst directly modulate the incentive salience attributed to cues relevant to their particular reward. They also modulate the hedonic impact of the rewards themselves. For example, hunger makes food taste better than usual, whereas physiological sodium appetite makes salty tastes 'liked' more, and these physiological states also make learned cues for those rewards instantly attractive and 'wanted'. Multiplicative interactions between reward cues and relevant physiological appetite states are a defining feature of incentive salience 'wanting'.

"Wanting" versus ordinary wanting. The quotation marks around the term "wanting" serve as caveat to acknowledge that incentive salience means something different from the ordinary sense of the word *wanting*. For one thing, "wanting" in the incentive salience sense need not have a conscious goal or declarative target (Berridge, 2001; Winkielman and Berridge, 2004). Wanting in the ordinary sense, on the other hand, nearly always means a conscious desire for an explicitly expected outcome. In the ordinary sense, we consciously and rationally want those things we expect to like. Conscious wanting and core "wanting" differ psychologically and probably also in their brain substrates, with cognitive wanting mediated by cortical structures, and incentive salience 'wanting' mediated more by subcortical systems.

Reward 'wanting' is thus just one type of decision utility. It is only decision utility -- not experienced utility (which is more similar to 'liking'), nor predicted utility (prediction or

expectation of future reward). And it is not all of decision utility --- it can leave out these more cognitive wants. If we are correct in our hypothesis, then this specificity of 'wanting' means that selective activation of mesolimbic dopamine systems produce truly irrational decisions. This mesolimbic utility pump can lead to 'wanting' what is neither 'liked' nor even expected to be liked sufficiently to rationally justify the decision to pursue -- and thus not even wanted in a more abstract cognitive sense.

Cue-triggered 'wanting' as a special form of decision utility

Reward cues are often potent triggers for urges and decisions to pursue and consume those rewards. Why are cues so motivationally potent? The incentive salience hypothesis offers a specific answer because it posits that reward cues are attributed with dopamine-driven incentive salience by mesolimbic circuits.

These conclusions come largely from animal experiments on cue-triggered decision utility, which we will describe now. Such experiments have sometimes used a procedure called Pavlovian-Instrumental Transfer, which for our purposes can be thought of as a way of isolating incentive salience as cue-triggered 'wanting'. In those studies, the rats are first trained to work (press a lever) for the real rewards. Since rewards come only every so often, animals learn to persist in working to earn reward even when sparse. In a separate training session, rats are presented with rewards under conditions where they don't have to work. Besides not having to work for the reward, the significant change here is that each reward is associated with an auditory tone cue 10 to 30 seconds long. Just as with Pavlov's dogs, the cues come to signify reward for the animals, becoming Pavlovian conditioned stimuli (CS+). With these two steps training is complete.

Testing begins after the training is completed. A special experimental feature is employed, namely extinction tests. Rats are tested for their willingness to work for rewards

later under extinction conditions, so-called because the rewards are no longer delivered at all. Since there are no real rewards any longer, the rats have only their expectations of reward to guide them. Naturally, without real rewards to sustain efforts, performance in the extinction test gradually falls. But since the rats originally learned that perseverance pays off, they persist for quite some time in working based largely on their ordinary wanting for reward. The amount of work (number of lever presses) the animals is willing to perform under these conditions of no reward delivery is the measure of 'wanting'. Since no actual rewards are delivered (i.e., extinction), the analysis is not confounded by consumption of rewards.

The crux of the matter to reveal cue-triggered 'wanting' is to test the effects of Pavlovian cues, the tones formerly presented in association with the rewards, in various states of brain mesolimbic activation. These cues are presented once in a while as the rats continue to work, or not as the case may be. During this extinction test, cues come and go while the rats work in order to get reward that is never delivered. Finally, brain mesolimbic activation is manipulated by varying whether or not the rats receive a drug microinjection that causes increases in dopamine release.

Cindy Wyvell used this test in our laboratory and found a form of truly irrational choice that depended on mesolimbic (dopamine) activation (Wyvell and Berridge, 2000; Wyvell and Berridge, 2001). She used amphetamine microinjections into the brain nucleus accumbens to activate mesolimbic dopamine systems. Amphetamine causes dopamine neurons to release their dopamine so that it can reach other neurons. Wyvell found that dopamine activation caused a transient but intense form of irrational pursuit linked to incentive salience (Figure 2). One group of rats received amphetamine microinjections before their behavioral test while another group received saline. During this test, their

baseline performance could be guided only by their expectation of the cognitively wanted sugar, because they received no real sugar rewards. And while they pursued their expected reward, the Pavlovian reward cue (light or sound for 30 seconds) was occasionally presented to them over the course of the half-hour session.

Wyvell's findings were consistent and clear. Amphetamine microinjection enhanced cue-triggered "wanting" for sugar (Figure 2). Animals worked for the rewards and during the presentation of the Pavlovian cue, they showed peaks of dramatically harder work i.e., their level of 'wanting' increased. Amphetamine in their brains selectively raised the height of those 'wanting' peaks, without changing the baseline plateau on which the peaks sat or anything else. It should be noted that there are two types of wanting assessed here: (1) ordinary wanting, where the rat is guided primarily by its cognitive expectation that it will like the worked-for sugar reward, and (2) cue-triggered "wanting," or incentive salience attributed by mesolimbic systems to the representation of sugar reward that is activated by the cue. Dopamine activation selectively quadrupled cue-triggered 'wanting', causing a specific elevation in this particular form of decision utility. A similar specificity, in reverse, has been found for suppressing effects of dopamine blocking drugs on cue triggered 'wanting' (Dickinson et al., 2000).

Even though the dopamine rise in Wyvell's experiments was relatively constant over the half-hour test, the 'hyper-wanting' was not. It required two conditions simultaneously: dopamine activation plus the presence of the cue previously associated with reward. Thus the 'wanting' peak was repeatedly reversible, even over the short span of a 30-minute test session (Wyvell & Berridge, 2000). This cue-triggered "hyper-wanting" phenomenon caused by activating mesolimbic dopamine, demonstrated by Wyvell was irrational and transient.

In a related experiment, Wyvell tested the effect of amphetamine microinjections on the

experienced utility of real sugar, by measuring positive hedonic 'liking' reactions of rats as they received an infusion of sugar solution into their mouths. The amphetamine never increased rats' positive facial reactions elicited by the taste of real sugar, indicating once again that dopamine did not increase "liking" for the sugar reward. Thus, Wyvell found that activation of dopamine neurotransmission in the accumbens did not change ordinary wanting based on cognitive expectation of liking (measured by baseline performance on the lever) nor did it alter 'liking.

In an elevated dopamine state, hyper-wanting is triggered by encounter with reward cues, and at that moment it exerts its irrational effect, disproportionate to the cognitively expected hedonic value of the reward. In other words, we suggest that decision utility diverges from predicted utility during the cue if the brain is dopamine-stimulated by amphetamine. One moment the dopamine-activated brain of the rat simply "wants" sugar in the ordinary sense, although the decision is tempered by the fact that there is no reward presented during extinction. The next moment, when the cue comes, the dopamine-activated brain both wants sugar and "wants" sugar to an exaggerated degree, according to the incentive salience hypothesis (Figure 2). A few moments after the cue ends, it has returned to its rational level of wanting appropriate to its expectation of reward. Moments later still, the cue is reencountered again and excessive and irrational "wanting" again takes control.

The irrational level of pursuit thus has two sources that determine its occurrence and duration: a physiological factor (brain mesolimbic activation) and a psychological factor (reward cue activation). It seems unlikely that mesolimbic activation altered rats' cognitive expectation of how much they would like sugar (which might have rationally increased desire, even though their expectation would be mistaken). That is because amphetamine was present in the nucleus accumbens throughout the entire

session but the intense enhancement of pursuit lasted only while the cue stimulus was actually present.

Human drug addiction as sensitized 'wanting'

Human drug addiction may be a special illustration of irrational "wanting" driven by mesolimbic brain systems (Robinson and Berridge, 1993; Robinson and Berridge, 2003). Addictive drugs not only activate brain dopamine systems when the drug is taken but may also sensitize them afterward. Neural sensitization means that the brain's mesolimbic system is hyper-reactive and therefore more easily activated for a long time and maybe even permanently. The mesolimbic system reacts more strongly than normal if the drug is taken again. This state of hyperactive reactivity is gated by associative cues and contexts that predict the drug. Neural sensitization occurs to different degrees in different individuals. Some individuals are susceptible to sensitization but others are not, depending on many factors ranging from genes to prior experiences, as well as on the drug itself, dose, and so on (Robinson and Berridge, 1993; Robinson and Berridge, 2003).

Efforts to apply these insights gave rise to the incentive-sensitization theory of addiction, developed primarily by Terry Robinson, which specifies the role sensitization of incentive salience may play in driving addicts to compulsively take drugs (Robinson and Berridge, 1993; Robinson and Berridge, 2003). This theory suggests that if an addict's mesolimbic system becomes sensitized after taking drugs, that person may irrationally "want" to take drugs again --- even if they have fully emerged from withdrawal by the time they relapse and even if they decide they don't "like" the drugs very much (or at least like them less than they like the lifestyle they will lose by taking them). This incentive-sensitization theory of addiction thus accounts for why addictive relapse is so often precipitated by encounters with drug cues, which trigger excessive "wanting" for drugs. In a sensitized mesolimbic state, the reward

cues trigger a momentary rise in decision utility that far outstrips any predicted or experienced utility of the drugs. Drug cues are attributed with more incentive salience than other cues because they are associatively paired with strong drugs. Drug cues could trigger irrational "wanting" in an addict whose brain was sensitized even long after withdrawal was over (because sensitization lasts longer), and regardless of expectations of "liking."

Actual evidence that sensitization does indeed cause irrational cue-triggered "wanting" was recently found by Cindy Wyvell in an affective neuroscience animal study of mesolimbic sensitization by drugs similar to the study described above (Wyvell and Berridge, 2001). Rats that had been previously sensitized by amphetamine responded to a sugar cue with excessive "wanting" despite not having had any drug for ten days. Even though the rats were drug-free at the time of testing, sensitization i.e., the brain in a state of permanent mesolimbic activation caused excessively high cue-triggered "wanting" for their reward. For sensitized rats, irrational "wanting" for sugar came and went transiently with the Pavlovian cue associated with the sugar reward, just as if they had received a brain microinjection of drug to immediately activate the mesolimbic system – but they hadn't (Figure 2). Their persisting pattern of cue-triggered irrationality seems consistent with the incentive-sensitization theory of human drug addiction (Robinson & Berridge, 2000). Similarly, neural sensitization by drugs has been found to increase other cue and motivation effects, such as conditioned reinforcement, and the persistence of motivated performance on second-order schedules and instrumental breakpoint in animals (Vanderschuren and Everitt, 2005; Vezina, 2004).

Separating Predicted Utility from Decision Utility?

A crucial question about enhancements of cue-triggered 'wanting' above is whether

the mesolimbic increase applies predicted utility or just decision utility. It is clear that decision utility was elevated in the above experiments by prior sensitization or direct amphetamine effects. But could dopamine elevation also have raised predicted utility too? If so, a sensitized individual might hold mistakenly exaggerated expectations for future reward, expecting eventual experienced utility to be higher than it really will be. If that happened, then decision utility would also elevate and passively trail after predicted utility. After all, if one mistakenly expects a reward to be better than it will be, then one may choose to pursue it more than one otherwise would.

A prediction error interpretation (mistakenly elevated expectation of reward) is highlighted by recent intriguing hypotheses about dopamine and reward learning in computational neuroscience. These have suggested that dopamine neurons may help mediate the associations and predictions involved in reward learning, either via stamping in associations to a UCS prediction error or by modulating the strength of learned predictions or learned habits elicited by a CS (Dayan and Balleine, 2002; McClure et al., 2003; Montague et al., 2004; O'Doherty et al., 2003; Schultz, 2002, 2006; Schultz et al., 1997).

Elegant and influential studies by Wolfram Schultz and colleagues, for example, have indicated that the firing of dopamine neurons impressively obeys predictions generated by computational learning models, in which learning is incrementally grown over trials based on mismatch between predictions elicited by a CS (predicted utility) and a subsequent UCS prediction error (experienced utility). A mismatch leads to a teaching signal that accordingly alters the predicted utility encoding by dopamine neuron firing that the CS will elicit on the next trial (Schultz, 2002, 2006; Schultz et al., 1997).

Current computational models open up several specific ways by which increasing a dopamine-related signal via sensitization or amphetamine might magnify a UCS prediction

error and thereby increase future predicted utility values. For example, dopamine firing has been suggested by learning theorists to approximate the Rescorla-Wagner model of Pavlovian conditioning ($\Delta V = \alpha\beta(\lambda - V)$). That model suggests that sensitization/amphetamine might increase predicted utility (V) to a CS by either elevating the prediction error generated by the hedonic or associative impact of UCS (λ), or else by accelerating the rate parameter (α) that determines speed of learning (ΔV) (Rescorla and Wagner, 1972; Schultz, 2002).

Another related computational model that has been suggested to approximate dopamine firing is the temporal difference model, which adds an important sequential feature to improve the static Rescorla-Wagner rule. Temporal difference models help explain why dopamine fires sometimes appears to literally move forward in time as learning progresses, originally firing only to the reward UCS, but after learning firing to the CS prediction and no longer to the UCS experienced utility (Montague et al., 1996; Schultz, 2002). The expected future reward

$$V(s_t) = \left\langle \sum_{i=0}^{\infty} \gamma^i r_{t+i} \right\rangle$$

is formulated as

$\langle \rangle$ represents the expectation over all future time steps starting from a particular state s , and a factor γ discounts primary rewards r received further in the future. Through learning, animals improve reward estimates based on predicted errors, $\delta(t)$.

This error signal $\delta(t) = r_t + \gamma \hat{V}(s_{t+1}) - \hat{V}(s_t)$ is used to modulate synaptic weights in circuits involved in future predictions, and gradually through successive trials leads to better estimates of future reward value (Montague et al., 1996).

The crucial feature of most temporal difference based computational learning models, when applied to predicted utility of reward and to mesolimbic dopamine function, is that dopamine elevation can

only generate new learning by creating a UCS prediction error if the experienced utility of UCS is greater than CS expected. This feature results from the fact that previously learned values are 'cached', and can be changed only incrementally and only by having further opportunities to learn a changed new relationship between CS and UCS.

For example, if one elevates α , β , λ , or r in Rescorla-Wagner or temporal difference models by a physiological manipulation such as dopamine signal rise, the predicted utility, V or δ carried by a CS+ does not immediately change (Figure 3). Instead, in the next learning trial, the UCS will cause a larger prediction error or faster learning rate, which will be saved until the next trial. Evidence for new learning is postponed until it can be demonstrated in subsequent trials. The new learning is then reflected in a gradually incremented increase in predicted utility generated the next times the CS is encountered. By contrast, the incentive salience hypothesis predicts that dopamine activation will raise incentive salience on the very first re-encounter of the reward cue that was learned before the brain activation occurred.

Thus, a startling feature of Wyvell's behavioral effects of amphetamine and sensitization on cue-triggered 'wanting' described above (and the neural recording experiment described below) is that mesolimbic activation occurred after all learning trials were completed. It didn't need to be relearned. It was immediate upon the first cue presentations in the activated mesolimbic state. Even on the very first trial the next time the cue was encountered CS+ decision utility was elevated. Dopamine activation did not occur before learning so there was no possibility that it could have enhanced subsequent prediction errors (increased predicted utility). In this experiment, mesolimbic activation was delayed until after learning, when it was too late to be able to promote predicted utility via boosting the association between CS+ and UCS. Mesolimbic activation still increased

cue-triggered 'wanting' indicating that it could not have generated increased predicted utility as suggested by these temporal difference based computational models.

It is possible that a future computational model of dopamine and reward learning will escape the computational constraint of cache-based models, and become better able to cope with sudden shifts in value that are not gradually relearned. For example, recent tree-search models have been proposed that exhaustively examine all potential outcomes, pulling up each one for a thorough re-evaluation of its utility values (Daw et al., 2005). So far, such tree-search models have been focused on cortex function and cognitive forms of incentive learning, but conceivably a related future model, if applied to mesolimbic dopamine function, might be able to allow 'instant increases' in CS predicted utility produced by post-learning sensitization or drug administration. If so, mesolimbic dopamine activation could be reinterpreted as having caused excessive general or cue-triggered predicted utility, expressed as over-optimistic expectations about the quality or quantity of upcoming rewards. Can this potential future interpretation be dealt with now?

In plain language, what if dopamine caused a cue to carry higher predicted utility than it ordinarily would, as well as higher decision utility? If so, the elevation of predicted utility by amphetamine or sensitization would become similar to the standard types of wrong decisions or miswanting identified by Kahneman and colleagues, Gilbert and Wilson, and colleagues, and by others (Gilbert and Wilson, 2000; Kahneman et al., 1997; Loewenstein and Schkade, 1999). Those wrong choices are based on wrong expectations. That means that they need not be irrational by the criteria we have adopted – wrong as the decisions remain – so long as the choice's decision utility matches predicted utility.

We believe we can rule out such a possibility for dopamine-based irrational 'wanting' described above. We can tease apart decision utility from predicted utility in an even stronger demonstration. But we have to turn to inside the brain in order to do it. What happens to utility from the brain's point of view when dopamine is released?

Neuronal coding of predicted utility and decision utility

A recent study of neural coding in our laboratory examined changes in predicted utility versus decision utilities related to mesolimbic dopamine activation (Tindell et al., 2005). This dissertation study was conducted primarily by Amy Tindell in the Aldridge laboratory, consisted of a behavioral and neurophysiological investigation of the ventral pallidum, a mesolimbic output structure. She used multiple electrodes to study the firing patterns of neurons, and their relationship to predicted utility, decision utility, and experienced utility of sugar rewards and their Pavlovian cues.

We focused on the ventral pallidum for neural coding of reward utility because it is a 'limbic final common path' for reward signals in mesocorticolimbic circuits (Kalivas and Nakamura, 1999; Oorschot, 1996; Zahm, 2000)(Figure 1). The ventral pallidum integrates reward-related information from the nucleus accumbens (compressed as much as 29:1) and other structures. It especially integrates dopamine influences, because it receives the heaviest projections sent from the nucleus accumbens neurons that most famously get mesolimbic dopamine, and also receives direct mesolimbic dopamine inputs itself. The output of the ventral pallidum is directed back to cortex through the thalamus and also to brainstem nuclei.

Serial cues uncouple predicted versus decision utilities

In order to tease apart predicted utility from decision utility, we used two different cues in series to predict the sugar reward (Tindell et al., 2005). A 10-sec auditory tone cue (CS+1) was followed by a 1-sec auditory

click cue (CS+2), which finally was followed immediately by a sugar pellet (UCS). The two different CSs have very different ratios of predicted utility to decision utility.

The first tone cue predicts everything that follows: it predicts the click cue 10 sec later and the sugar pellet 1 sec after that. Once a rat learns this relationship, which usually only takes a few dozen presentations of the series, the CS+1 tone cue tells the animal everything there is to know about upcoming signals and rewards for the immediate future. By contrast, the second click cue is completely redundant as a predictor. It adds no new information. Rats can easily keep track of the 10 to 11 sec interval between first tone and sugar – they don't need the second cue to tell them sugar is coming. In fact, they begin to hover around the sugar dish a few seconds before it arrives. Thus the CS+1 tone cue carries greatest predicted utility. It sets all expectations for the future. That means that if mesolimbic activation can raise predicted utility, it should best be evident in changes in neuronal firing elicited by the CS+1 tone cue.

But the second click cue still has something the first tone cue doesn't. The CS+2 click carries the greatest decision utility or incentive salience. It occurs at the moment of highest incentive motivation or 'wanting' for sugar, reflected in part by rat's eager hovering around the dish at that moment. If mesolimbic activation causes increases in decision utility that occur without any matching increase in predicted utility, then this should be most evident in changes in neuronal firing elicited by the CS+2 click cue. And it should occur even if there is no change in firing to the CS+1 tone. That profile of activation would indicate that cue-triggered decision utility > predicted utility in the brain of a rat that has an activated mesolimbic dopamine system, setting the stage for the possibility of strongly irrational choice.

Finally, the sugar cue that comes last carries the greatest experienced utility.

The sweet sugary pellet is the event that is actually 'liked' best. It is also the teaching signal event, the reward value that 'stamps in' an association or that instructs a predictive actor in an actor-critic model that a reward event has occurred. One should expect a change in sugar-elicited neuronal firing if mesolimbic activation cause elevations in either hedonic impact, associative stamping in, or UCS prediction errors generated as teaching signals. And just to double check if hedonic impact 'liking' is enhanced by mesolimbic activation, we also examined whether amphetamine or drug sensitization caused any elevation in 'liking' reactions of rats to the taste of sugar.

Sensitization

Rats were trained for two weeks and then some were sensitized while others were treated with a saline placebo over another two week period (Figure 3). One month was allowed to pass before testing because neural sensitization changes require a period of time – preferably without drug -- to "incubate" and develop fully. Brain accumbens and cortical neurons that become sensitized undergo gradual biochemical and even structural changes during the incubation period, for example, some sprouting new dendrite spines (Robinson and Kolb, 2004). In the end, this leaves sensitized neurons structurally changed, and ready to release more dopamine than normal when stimulated by drugs or certain other events. Then all the rats were implanted with recording electrodes in their ventral pallidum, and allowed to recover for another two weeks. Finally, Amy Tindell examined the neuronal coding in relation to CS+1 tone, CS+2 click, and sugar pellet UCS stimuli in the rats, and compared their ordinary neuronal firing to firing when stimulated by extra dopamine caused by injection of amphetamine (in both sensitized rats and normal rats). She found that dopamine activation by amphetamine and by sensitization consistently magnify the firing rate peaks of ventral pallidal neurons whenever the rat heard its maximal incentive stimulus: the CS+2 click immediately before reward (Figure 4). Firing rates were not

similarly enhanced to the maximal predictive CS+1 or maximal hedonic UCS, nor were baseline firing rates ever increased in the absence of a stimulus.

To decode reward utilities in neuronal firing patterns, we used a novel computational technique, 'Profile Analysis', developed by our colleague Jun Zhang, which compares maximal firing of neurons among stimuli, asking whether greatest firing is elicited by either the CS1, the CS+2, or the UCS. This technique allows use to identify how mesolimbic activation changes the stimulus response 'profile' of ventral pallidal neurons.

We found that individual VP neurons fire most often to all three stimuli, but usually not equally to all (Figure 5). Ordinarily, predictive utility seems to dominate neuronal coding in VP, in the sense that the neurons fire most to the CS+1 (next to the CS+2, and only moderately to the sugar). But mesolimbic activation, caused by either amphetamine administration at test or by prior drug-induced neural sensitization, shifts the profiles of VP neural activation toward incentive coding at the expense of prediction coding (Figure 5). The elevations in decision utility appeared to be roughly additive across amphetamine and sensitization treatments. That suggests, for example, that either sensitization or taking an addictive drug could raise the decision utility of taking the drug again. And in an individual already sensitized, the first act of drug consumption would combine with preexisting sensitization to cause an addicted 'hyper-wanting' to take the next dose, a level of decision utility probably unmatched in unsensitized individuals under any known circumstances. Taking the drug directly elevates the mesolimbic activation state, priming cue-triggered decision utility to an extremely high degree and creating a window of vulnerability to further relapse that is greater even than the ordinary sensitized susceptibility to relapse of a drug addict.

It is noteworthy that the shift toward neuronal incentive coding was immediate on the first test trials, and did not require any relearning (Figure 3). That immediate change supports the incentive sensitization hypothesis and stands in contrast to the alternative dopamine-learning hypotheses that require further training trials for an increased reward prediction error ($(\delta)t$) to magnify relearned predictions (V). It appears that in a dopamine-activated or sensitized state, incentive coding by VP neurons might mediate increased cue-triggered 'wanting' and could lead to the compulsive relapse of addiction, especially for drug cues that occur close in time to their reward.

The immediate enhancement of incentive salience by mesolimbic activation comes about because incentive salience or cue-triggered decision utility normally depends on integrating two separate factors: (1) current physiological/neurobiological state; (2) previously learned associations about CS+ (Berridge, 2004; Toates, 1986). Integrating current physiological state with learned cues allows behavior to be guided dynamically by appetite-appropriate stimuli without need of further learning (e.g. Pavlovian cues associated with food are immediately more attractive to a hungry animal). Drug sensitization or acute amphetamine may each 'short circuit' this neurobiological system and directly increase the incentive value attributed to particular conditioned stimuli, triggering greater 'wanting' and pursuit of their reward (Robinson and Berridge, 2003; Tindell et al., 2005).

Finally, these shifts toward VP incentive coding were not due to enhanced UCS hedonic impact ('liking'). Behavioral hedonic 'liking' reactions to sucrose taste remained constant or even diminished slightly with sensitization and amphetamine administration. In other words, mesolimbic activation caused increases in cue-triggered 'wanting' as coded by VP neurons when encountering a CS+ for sugar reward, without any increase in experienced utility or 'liking' for sugar itself.

Explanation for cued hyperbolic temporal discounting?

The shift toward incentive coding suggests how sensitization and addictive drugs may prime motivational behavioral responses of addicts to drug-related stimuli by amplifying the incentive impact of encountering a UCS-proximal drug CS+. Finally, it suggests a mechanism to help explain hyperbolic temporal discounting, at least in cue-triggered decisions. Temporal discounting is well recognized in addicts (Ainslie, 1992), and neuroimaging have shown that mesolimbic systems code immediate rewards (McClure et al., 2004). But temporal discounting is usually just described and accepted as a given. Though it is sometimes posited as a mechanism that drives choices, little is known about the explanatory mechanism for hyperbolic discounting itself. The explanation may be that limbic activation causes circuits involving ventral pallidum to fire more to cues for a temporally close reward, and therefore selectively their incentive salience, causing excessive cue-triggered 'wanting' for the close reward. This also may be why 'visceral states' sometimes exacerbate temporal discounting effects (Loewenstein and Schkade, 1999).

Irrational decision utility in these examples

We suggest that the experiments described above are both examples of decision utility > predicted utility at the same moment. Thus both are examples of irrational 'wanting', defined as 'wanting' something more than one expects to like. In the Wyvell cue-triggered 'wanting' experiments, the elevated decision utility is a peak of frenzied pursuit of the sugar reward, at least for a while. The reward cue causes a momentary irrational desire, during which decision utility > predicted utility (as well as decision utility > experienced utility). In the Tindell neuronal firing experiments, the magnified firing bursts of ventral pallidal neurons at the moment of the cue with most incentive

salience reflects a neural mechanism that may drive irrational 'wanting'. Both cases happen when a reward cue occurs simultaneously with mesolimbic activation, especially dopamine-related activation. Individuals may then "want" what they do not want cognitively. Further, they may not predict associatively in a manner that would justify their 'want'. The decision utility is irrational in the sense that their immediate 'want' exceeds what they know cognitively they will not like (or at least, will not "like" proportionally to their excessive "want").

Importantly, incentive salience attributions are encapsulated and modular in the sense that people may not have direct conscious access to them, and find them difficult to cognitively control (Robinson and Berridge, 1993; Robinson and Berridge, 2003; Winkielman and Berridge, 2004). Cue triggered 'wanting' belongs to the class of automatic reactions that operate by their own rules, under the surface of direct awareness (Bargh and Ferguson, 2000; Dijksterhuis et al., 2006; Gilbert and Wilson, 2000; Wilson et al., 2000; Zajonc, 2000). People are sometimes aware of incentive salience as a product, but never of the underlying process. And without an extra cognitive monitoring step, they may not even be always aware of the product. Sometimes incentive salience can be triggered and control behavior with very little awareness of what has happened. For example, subliminal exposures to happy or angry facial expressions, too brief to see consciously, can cause people later to consume more or less of a beverage – without being at all aware their 'wanting' has been manipulated (Winkielman and Berridge, 2004). Additional monitoring by brain systems of conscious awareness, quite probably cortical structures, are required to bring a basic 'want' into a subjective feeling of wanting.

Applications to human decision making

Although our experiments used drugs and sensitization to manipulate brain dopamine systems in rats, people have brain dopamine systems too. Human mesolimbic

systems can be equally activated by drugs and addiction, but perhaps more relevant to everyday decisions; the same dopamine brain systems are also spontaneously activated by natural appetite states and in many emotional situations.

As a result of all this, an irrational 'want' for something can occur despite cognitively not wanting it, cognitively wanting not to 'want', or cognitively wanting something else. An irrational cue-triggered 'want' may even surprise the person who has it, by its power, suddenness and autonomy. This may explain why some long term drug addicts can proclaim (perhaps even truthfully) to not enjoy their drug as they once did while at the same time they may take part in criminal activity in order to acquire the drug.

Both rewarding and stressful situations activate brain mesolimbic systems. This seems to raise the possibility for decision utility elevations when reward cues occur simultaneously with brain activation at moments requiring a choice. If a person's brain dopamine system were highly activated, and the person encountered a reward cue at that moment, then the person might irrationally elevate the decision utility of the cued outcome, over and above its experienced utility and predicted utility both. That person would be under the control of a decision utility pump. The person might "want" the cued reward just like the rat – even if the person cognitively expected not to like it very much. Such phenomena might not be restricted to basic consumption behavior but could extend interact with more abstract and even economic decisions too (Bernheim and Rangel, 2004; Camerer and Fehr, 2006). Whether hijacked decision utility and irrational "wanting" actually play this role in ordinary human lives and decisions seems to be an intriguing possibility that may deserve further consideration.

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Figure 1.

'Liking' reactions and brain hedonic hotspots. Top: Positive hedonic 'liking' reactions are elicited by sucrose taste from human infant and adult rat (e.g., rhythmic tongue protrusion). By contrast, negative aversive 'disliking' reactions are elicited by bitter quinine taste. Below: Forebrain hedonic hotspots in limbic structures where mu opioid activation causes a brighter pleasure gloss to be painted on sweet sensation. Red/yellow shows hotspots in nucleus accumbens and ventral pallidum where opioid microinjections caused the biggest increases in the number of sweet-elicited 'liking' reactions. Modified from Peciña and Berridge (2005) and Smith and Berridge (2005).

Figure 2.

Irrational cue-triggered "wanting." Transient irrational "wanting" comes and goes with the cue (left). Amphetamine microinjection in nucleus accumbens magnifies "wanting" for sugar reward – but only in presence of reward cue (CS+). Cognitive expectations and ordinary wanting are not altered (reflected in baseline lever pressing in absence of cue and during irrelevant cue, CS-) (right). Modified from Wyvell and Berridge, 2000.

Figure 3.

Decision utility increment happens too fast for relearning. Timeline and alternative outcomes for neuronal firing coding of reward cue after mesolimbic activation of sensitization and/or amphetamine in ventral pallidum recording experiment (Tindell et al., 2005). The incentive salience model predicts that mesolimbic activation dynamically increases the decision utility of a previously learned CS+. The increased incentive salience coding is visible the first time the already-learned cue is presented in the activated mesolimbic state. Learning models by contrast require relearning to elevate learned predicted utilities. They predict merely gradual acceleration if mesolimbic activation increases rate parameters of learning, and gradual acceleration plus asymptote elevation if mesolimbic activation increase prediction errors. Actual data support the incentive salience model. Based on data of (Tindell et al., 2005)

Figure 4

Firing rates in ventral pallidum neurons triggered by the maximal incentive cue (CS+2). Amphetamine magnified firing elicited by CS+2, the click sound which was already predicted by

the earlier CS+1 tone but which was immediately before the sugar reward UCS. Sensitization by drug exposures weeks before similarly magnified firing elicited by CS+2, even if sensitized rats didn't have any drug at the time of testing. Histogram rows show individual action potentials at top of each box (each row of dots is a single trial for a particular rat; different rows are different rats). Firing for the whole group of rats reveals rate peaks, in the cumulative histogram at bottom of each box.

Figure 5

Mesolimbic activation magnifies decision utility coding by neuron firing in ventral pallidum. Population Profile Vector shifts toward incentive coding after amphetamine or sensitization. Amphetamine and sensitization add together to prime the decision utility pump of incentive salience towards CS+2 'incentive-coding' region.. The cue with highest incentive salience, CS+2, increasingly dominates the neuronal Population Profile Vector for all recorded neurons in ventral pallidum as mesolimbic activation increases. Profile analysis shows stimulus preference coded in firing for all 524 ventral pallidum neurons VP (among CS+1, CS+2 and sucrose unconditioned stimulus (UCS). Entire populations are shown by shaded areas. Arrow shows the maximal averaged response of the population. Normal rats (control rats during vehicle tests) have a neuronal profile dominated by prediction utility coding (CS+1 bias), while firing in sensitized animals during amphetamine challenge reveals a profile dominated by decision utility or incentive salience coding (CS+2 bias). (Direction $\theta = \tan^{-1} \left(\frac{\sqrt{(CS1-UCS)/2}}{(2CS2 - CS1 - UCS)/2} \right)$, and Magnitude $r = \sqrt{[(CS1-CS2)^2 + (CS2-UCS)^2 + (UCS-CS1)^2]/2}$). Modified from Fig. 6 and Fig. 7, p. 2628 and 2629 (Tindell et al., 2005)

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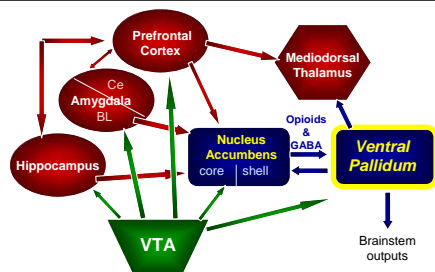
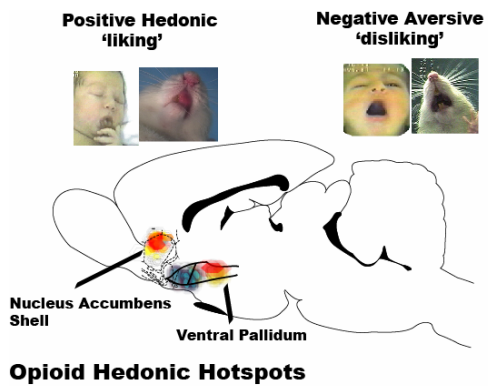


Figure 1

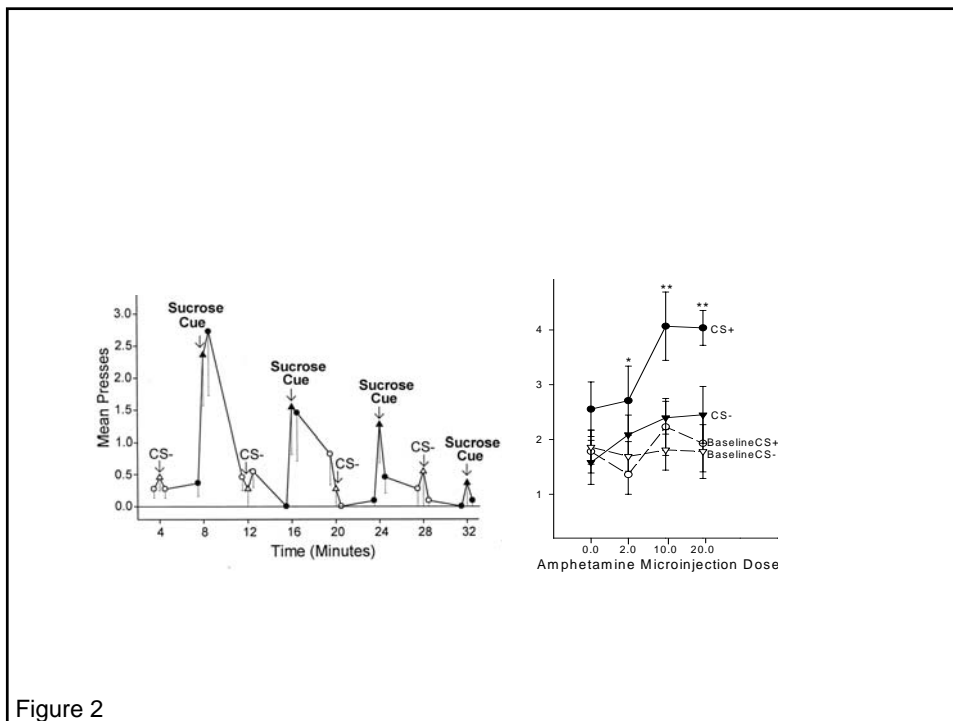


Figure 2

