CHAPTER

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From Experienced Utility to Decision Utility

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INTRODUCTION

s0010

p0110 In the view of neoclassical economics, people make decisions that maximize outcome utility to fit their individual preferences or needs (e.g., see Chapters 1, 2, and 8). But what happens if people's choices are generated by mechanistic processes which have flaws that sometimes distort their outcome choices? The answer to this question requires distinguishing among multiple types of utility, and considering the relation of each utility type to internal psychological and neurobiological mechanisms. In turn, existence of multiple utilities may pose a further question for policy makers. That is, when a person contains several types of utility for the same outcome, and the various types diverge, which utility should be maximized?

EXPERIENCED UTILITY

s0015

p0115 To clarify, start with a hedonic approach to utility. This may be familiar as it arises from Bentham's proposal two centuries ago that people's choices are governed by two sovereign masters: their motives to gain pleasure and to avoid pain (Bentham, 1789). When choices are made between different outcomes, each outcome has its own hedonic consequences, and a good decision in such cases is to choose and pursue the particular outcome that will overall produce the most pleasure and least pain (other considerations being equal). One way to express this in utility terms is to say that a good decision is to maximize the *experienced utility* of the chosen outcome. Experienced utility means the hedonic or pleasurable experience produced

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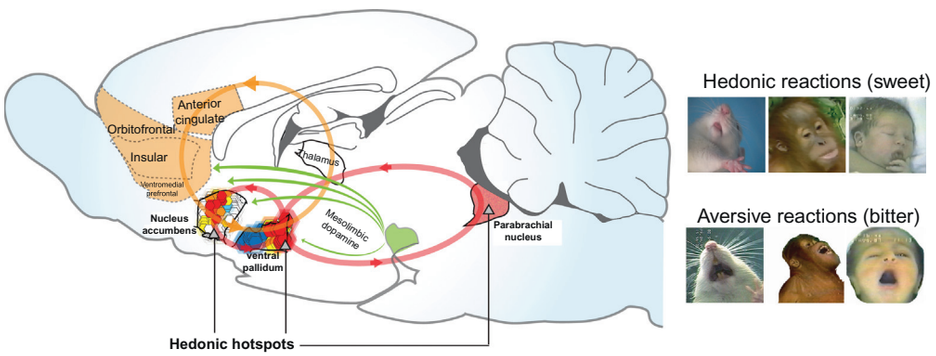
by the outcome when eventually gained (Kahneman *et al.*, 1997). Outcomes that generate a pleasure impact elicit a constellation of objective responses (including affective behavioral reactions, physiological autonomic, and brain limbic reactions) as well as in humans at least, subjective feelings reported as pleasure.

s0020 Brain Mechanisms of Sensory Pleasure: Window into Experienced Utility Generators

p0120 How is experienced utility actually generated within a brain? Our information about how pleasure is generated by neural mechanisms has come so far mostly from studies of sensory pleasures, such as sweet tastes. The brain appears almost surprisingly frugal in mechanisms that generate the pleasure of experienced utility. Pleasure generators are restricted to cubic-centimeter sized "hedonic hotspot" generators within a few brain structrures, such as nucleus accumbens and its target the ventral pallidum, which use particular neurochemical signals to produce intense pleasures (Figure 18.1). The generation of pleasure has been identified primarily by experiments involving manipulations of the brain (it is necessary to alter the causal generation process in order to identify its underlying mechanisms), which for ethical reasons has been done mostly in animal studies involving rats or mice. In animal studies, it is possible to painlessly stimulate a brain system that generates pleasure, and to observe magnification of the hedonic impact for pleasant sensations, for example as evidenced by increases in behavioral "liking" reactions to a sensory pleasure such as sweetness. This approach to discovering pleasure generators is based on Darwin's original description of emotional expressions (Darwin, 1872). For example, anyone who has cared for an infant knows that even a newborn emits facial expressions revealing "liking" for the palatability of a taste. Sweet tastes elicit a contented licking of the lips, whereas bitter tastes are met with gaping mouths, shaking heads, and a vigorous wiping of the mouth. A number of the same expressive responses seen in human infants also occur in rats, mice, and nonhuman primates (Steiner *et al.*, 2001). Experimenters can measure enhancements of those "liking" reactions to the experienced utility of sweetness by painlessly activating a brain hotspot in a rat or mouse. One way of activating the pleasure mechanisms is by a microinjection of a tiny droplet containing drug into the brain hotspot through a previously implanted cannula. The drug microinjection cannot be felt by the animal, but the drug it contains mimics neurotransmitter signals to neurochemically stimulate neurons in the hotspot, thus activating the neural system for experienced utility. Discovering which brain microinjections successfully amplify a sensory pleasure thus can identify the brain hotspot locations and the particular hedonic neurochemical signals within hotspots that generate the pleasure for sensations (Berridge and Kringelbach, 2011; Smith, *et al.*, 2010).

p0125 Mapping of brain pleasure generators in this way has revealed a network of several brain hedonic hotspots that amplify experienced utility expressed as "liking" reactions to sweetness (Mahler *et al.*, 2007; Peciña and Berridge, 2005; Peciña *et al.*, 2006; Smith and Berridge, 2005). The brain hotspots form a chain of "liking"-enhancing islands of brain tissue stretching from front to back of the brain, distributed across several deep structures of the brain below the neocortex. Each brain hotspot is merely a cubic-millimeter or so in volume in the rodent brain (and would be expected to be a cubic-centimeter or so in you, if proportional to the larger human volume of whole brain). A hedonic hotspot is uniquely capable of generating intense enhancements of



f0010 FIGURE 18.1 Experienced utility generators in the brain. Hedonic hotspots that generate experienced utility, as expressed by amplifying pleasure "liking" reactions, are in red and yellow. Mesolimbic dopamine systems of pure decision utility "wanting" are in green. VTA, ventral tegmental area. Right: examples of the hedonic "liking" and "disliking" facial expressions to sweet or bitter taste outcomes have been useful in revealing the brain hedonic hotspots. Activation of the brain hotspots makes sensations seem more pleasant, amplifying their experienced utility.

"liking" reactions to a sensory pleasure when neurochemically stimulated, whereas the rest of the surrounding brain cannot, not even the rest of the same brain structure that contains the hotspot. In normal situations, the neurochemical stimulation that generates pleasure arises naturally from neuronal release of opioid neurotransmitters (natural heroin-like chemicals made by neurons), endocannabinoid neurotransmitters (natural marijuana-like chemicals), and a few other related neurotransmitters able to stimulate neuronal receptors in the hotspot in ways that activate hedonic circuits and amplify a sweet outcome's experienced utility.

p0130 One major hotspot has been found in the nucleus accumbens, a brain structure at the bottom front of the brain. This hotspot when neurochemically stimulated acts to amplify the pleasure of sensations, and makes up about only $1/10^{th}$ of the entire nucleus accumbens. That small 10% ratio reveals how restricted are the mechanisms that generate experienced utility (Peciña and Berridge, 2005). Another related hedonic hotspot lies in the posterior part of the ventral pallidum, the brains structure that receives most outputs from the nucleus accumbens, and which sits near the very bottom center of the forebrain (Peciña and Berridge, 2005; Peciña *et al.*, 2006; Mahler *et al.*, 2007; Smith and Berridge, 2005). The ventral pallidum hotspot may be especially important to experienced utility because it is the only known brain site where damage seems to totally eliminate normal levels of pleasure, abolishing "liking" reactions to a sweet taste and replacing with disgust reactions instead. After ventral pallidum damage a rat gapes to sugary taste as though it were bitter (Smith *et al.*, 2010). Likewise, while rare in humans, patients who have suffered damage to their ventral pallidum on both sides of the brain (usually due to stroke) become apathetic and report that their former favorite pleasures no longer seem worthwhile (Adam *et al.*, 2012; Miller *et al.*, 2006). While it would be premature to claim this evidence proves the ventral pallidum hotspot to be necessary for any and all possible hedonic experiences, the evidence available so far does suggest that the ventral pallidum is needed more than any other known brain structure for normal levels of experienced utility associated with pleasant outcomes. Beyond ventral pallidum and nucleus accumbens, a third hedonic hotspot is located deep in the brainstem (Smith *et al.*, 2010), and additional hotspots might yet still be found, say, perhaps in the prefrontal cortex (Kringelbach, 2010).

p0135 This network of separate but interactive hedonic hotspots acts together as a coordinated single circuit to amplify core pleasure reactions. Activating one hotspot recruits the others within the same hedonic system (Smith and Berridge, 2007; Smith *et al.*, 2011). Unanimous hotspot activation simultaneously appears to be crucial to enhancement of experienced utility. If one hotspot is blocked from activating while another is pharmacologically stimulated, then no pleasure enhancement occurs (Smith and Berridge, 2007; Smith *et al.*, 2010). In other words, a single hotspot "no" vote vetoes other "yes" votes to amplify experienced utility. The network properties reveal a fragile substrate for pleasure enhancement that requires unanimity across the several parts in order to elevate hedonic "liking."

EXPERIENCED UTILITY: NEUROIMAGING BRAIN ACTIVATIONS IN HUMANS

s0025

p0140 The brain appears to have re-used many neural elements that evolved originally for sensory pleasures to also mediate higher pleasures (Kringelbach and Berridge, 2009, 2010; Leknes and Tracey, 2008; Liljeholm and O'Doherty, 2012). For example, the same structures activated by food or drug pleasure also activate to the delight of including seeing a loved one, winning money, listening to favorite music, and even moral and spiritual pleasures (Kringelbach, 2009). In addition, a wider network of human brain structures also activate during most pleasures.

p0145 Studies in humans have examined the neural representation of experienced utility mostly using functional neuroimaging tools such as PET and fMRI (described in detail in Chapter 6). In order to isolate the experienced utility of an event, and separate pleasure from other psychological features of the same event, researchers have focused on finding particular brain sites that show the best positive correlation between neural activation and the intensity of the positive affective response. That correlation becomes especially visible when the reported subjective pleasantness to a particular event is modulated by a manipulation: for example by inducing satiety to make an initially pleasant taste less subjectively pleasant, or by increasing the amount of money won to make the event more rewarding. Then researchers can look for brain sites that alter their activation accordingly to match the change in hedonic evaluation (while ideally other features of the event remain unchanged such as intensity, identity, learning, etc.). For example, when the subjective pleasure or experienced utility of the taste of chocolate, or the odor of bananas, is altered by having a person consume a lot of chocolate or bananas until they would rather not have any more, the orbitofrontal activation evoked by the particular flavor experience of that food declines, more than other flavors and even though the sensory experience otherwise remains unchanged (Kringelbach, 2005; Kringelbach *et al.*, 2003; O'Doherty *et al.*, 2000; Small *et al.*, 2001).

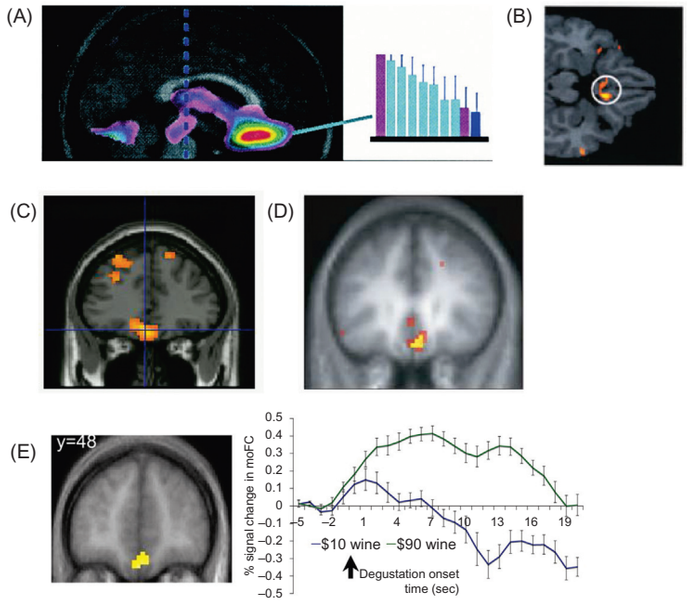


FIGURE 18.2 Experienced utility signals in the human medial orbitofrontal cortex elicited in response to a diverse array of sensory inputs. (A) Responses in this region (measured with PET) show decreasing activity during consumption of a chocolate meal as the reported subjective hedonic ratings for the chocolate decreases from being pleasant to being aversive as satiation develops. From Small *et al.* (2001). (B) Region of medial orbitofrontal cortex showing changes in activation as a function of differences in the reported subjective pleasantness of odor stimuli. From Anderson *et al.* (2003). (C) Region of medial orbitofrontal cortex showing increased activity in response to the presentation of faces reported as high in attractiveness relative to that elicited by faces reported as low in attractiveness. From O'Doherty *et al.* (2003). (D) Region of medial orbitofrontal cortex correlating with the magnitude of points won during performance of a simple decision-making task. From Daw *et al.* (2006). (E) Activity in a similar region of medial orbitofrontal cortex showed differential responding to the receipt of a bolus of wine into the mouth depending on whether that wine (which was in actuality the same wine in both cases) had been labeled as cheap ($10) or expensive ($90). *From Plassmann et al. (2008).* f0015

p0150 In human imaging studies, probably the most robust finding regarding the neural representation of experienced utility is that a particular region of orbitofrontal cortex above the eyes represents pleasure best, namely its anterior medial and adjacent central region (Kringelbach, 2010). Activity in this orbitofrontal cortex region correlates with the subjective pleasantness of a diverse array of different types of stimuli in a number of different sensory modalities (Figure 18.2), including the taste, odor and flavors of food (Anderson *et al.*, 2003; Rolls *et al.*, 2003; Small *et al.*, 2001), auditory stimuli such as musical arrangements (Blood *et al.*, 1999; Vuust and Kringelbach, 2010), visual stimuli such as attractive faces, or infants or even pieces of art (Kirk *et al.*, 2009; Kringelbach, 2010; O'Doherty, Critchley *et al.*, 2003; Parsons *et al.*, 2010). Furthermore, even more abstract rewards not tied to any specific modality such as winning money, obtaining points on a game, or experiencing positive social feedback engage the same region (Breiter *et al.*, 2001; Davey *et al.*, 2010; Knutson, Fong *et al.*, 2001; Lin *et al.*, 2012; O'Doherty *et al.*, 2001; Rilling *et al.*, 2002).

p0155 Experienced utility responses in this region are not only modulated by changes in internal state, such as when going from being hungry to satiated, but such responses can also be changed by top-down cognitive effects. For example, one study presented wine to participants in the fMRI scanner (Plassmann *et al.*, 2008), while telling participants the wine came from either an expensive bottle or a cheap bottle (actually, the wine was the same). Neural responses to the same objective wine stimulus were strongly modulated depending on whether the wine was labeled as coming from the expensive bottle compared to the inexpensive one, such that activity was much stronger for the wine when labeled as expensive. This change in activity also tracked changes in subjective pleasantness for the wines. Likewise, telling people that a pungent odor is cheese induces a very different brain activation pattern to the smell than if people are told the smell is unwashed body odor (de Araujo *et al.*, 2005). These findings indicate that neural representations of experienced utility in the orbitofrontal cortex can be directly modulated by exogeneous changes in context. In addition to orbitofrontal cortex, other regions of prefrontal cortex such as the insula and the anterior cingulate region of cortex also activate during pleasant sensations too (Kringelbach *et al.*, 2003, 2005; O'Doherty *et al.*, 2000; Small *et al.*, 2001).

p0160 Below the cortex, activity in the ventral striatum (nucleus accumbens) is also often found to be present during the receipt of different rewards in humans (Adcock *et al.*, 2006; Breiter and Rosen, 1999; Franklin and Adams, 2011; Knutson *et al.*, 2008; Risinger *et al.*, 2005), although this has been reported less consistently than is the vmPFC (Knutson and Gibbs, 2007; Knutson *et al.*, 2001; O'Doherty *et al.*, 2003a,b). In earliest studies, the ventral striatum was more often found to correlate with anticipated reward than to reward outcomes (Knutson *et al.*, 2001a,b; O'Doherty *et al.*, 2002). More recently, some activity in this region has been found to be better accounted for by a temporal difference reward prediction error signal (see Chapters 15 and 16 of this volume), in which activations at the time of cue presentation resemble an anticipated utility signal, while other activations at the time of outcome AU:1

represent the difference between expected and actual outcomes (as opposed to outcome value *per se*) (McClure *et al*., 2003; Niv *et al*., 2012; O'Doherty *et al*., 2003a,b, 2004). Other subcortical structures, including the ventral pallidum and the midbrain ventral tegmental area, have also been reported to be activated by rewards such as drugs, winning money, or music in some human neuroimaging studies (Boileau *et al*., 2012; Chapin *et al*., 2010; Pessiglione *et al*., 2007).

Relating Rodent to Human Findings: Causing *versus* Coding Experienced Utility

All this raises the question of how the neuroimaging findings in humans relate to the earlier described evidence of a special role for parts of the nucleus accumbens or ventral pallidum in generating experienced utility in rodent brains. One mundane possibility is that fMRI studies measure inputs into a structure and intrinsic processing therein, whereas rodent stimulation studies identify outputs that have actual consequences on hedonic reactions. More substantively, it is possible that some neuroimaging brain activations may reflect experienced utility but are reported as something else, or that some activations reported as experienced utility in fact reflect prediction errors or some other signal such as the sensory properties of an outcome (discussed below).

Perhaps the most important substantive possibility relevant to interpreting results of human neuroimaging of pleasure *versus* animal brain manipulation of pleasure generators is the question of whether neuroimaging activations reflect the *causation* of experienced utility, or rather only the *coding* of experienced utility (activated in the service of mediating some other psychological process). That is, not all brain activations which code for experienced utility need actually help to cause the pleasant experience. Experienced utility representations may also be present in some additional brain areas because the information about the experienced utility is used there to guide learning and updating of other signals needed to guide future choice, such as decision utility and anticipated utility, described later.

For example, evidence for a *causal* role of prefrontal cortex regions (orbitofrontal, insula or anterior cingulate cortex) in eliciting actual experienced utility is mixed. On the one hand, findings in human patients who have damage to the prefrontal cortex that impacts these regions suggest that they may not be critical for experienced utility. While it has long been known that damage to this area results in impairments in decision-making and preference formation, as well as in the elicitation of autonomic responses in anticipation of outcomes, autonomic responses to the receipt of outcomes appear to be largely intact in these patients (Bechara *et al*., 1997; Beer *et al*., 2010; Damasio, 2004; Damasio *et al*., 2012; Kringelbach, 2005; Valenstein, 1986). Furthermore, these patients seem to retain the capacity for essentially normal subjective hedonic experiences in response to the receipt of rewarding outcomes, as far as any outside observer can tell. On the other hand, deep brain stimulation studies of depressed patients have shown that stimulation in ventromedial regions of prefrontal cortex can help elevate mood (Holtzheimer and Mayberg, 2010). Such stimulation studies certainly could be used to support the idea that at least part of the vmPFC (in particular the subgenual cingulate area) may have a causal role in generating changes in affective disposition. Yet it is also possible that downstream neuronal changes in subcortical structures instead mediate the stimulation effects (such as nucleus accumbens), rather than the cortex where the electrode is itself, especially if the electrode primarily activates fibers of passage to those deeper brain structures (Kringelbach *et al*., 2010). Further, such changes in diffuse mood may also heavily involve cognitive appraisals that go beyond the hedonic experienced utility of outcomes. At best, it is clearly the case that more work needs to be done in establishing the extent to which vmPFC is causally involved in generating experienced utility.

BEYOND EXPERIENCED UTILITY

Experienced Utility *versus* Decision Utility

Experienced utility is the endpoint of the decision process. It is the state reached after successful attainment of a particular outcome, pertaining to the hedonic impact and experience of that outcome. However, other signals are needed in order for decisions to actually be made.

As discussed in Chapter 8, *decision utility* is proposed to be the utility signal used at the point of choice to guide decisions about future actions. The decision utility for a given action features an estimate of the expected future utility for a particular outcome reached after choosing that action, weighted by the probability of that outcome occurring and the time at which that outcome is predicted to occur.

How does experienced utility relate to decision-utility? One way to think of this relation is that at the time of decision-making, the decision-making agent will need to compute the decision-utility for available actions on the fly, before engaging in the process of comparing the decision utilities and making a choice (according to models of goal-directed choice; see Chapter 21 and O'Doherty, 2011). Included in this process is knowledge of the

outcomes which can be selected as goals by the agent. These outcomes or goals also have attached to them, learned utility signals stored in memory. Such memory signals representing outcome utility on previous occasions has been called *remembered utility*.

p0195 How do remembered outcomes come to retrieve goal-values? There is evidence that outcomes acquire value representations through an associative learning process by which the experienced utility elicited following delivery of that outcome comes to be associated with the stimulus features of the outcome, a process called incentive learning (see Chapter 15, also Dickinson and Balleine, 2010). Another way of describing remembered utility is as a person's "retrospective reports of the total pleasure or displeasure associated with past outcomes" (p. 376, Kahneman *et al.*, 1997), cognitively reconstructed into declarative memory at the moment of the report as conscious recollections (see Chapter 21 for further discussion of the possible contribution of declarative memory processes in decision-making). That is, remembered utility is the declarative episodic memory of how good a previous reward was in the past. Thus remembered experienced utility becomes attached to the stimulus properties of an outcome (which is state-dependent; for example, one has a different experienced utility of a food outcome depending on whether hungry or sated). This value-attaching process also bears strong similarities to the "somatic marker" mechanism proposed by Damasio, Bechara *et al.* (Bechara *et al.*, 1997, 2005; Damasio, 1996). Using this memory trace of experienced utility, it is then possible for a goal-directed agent to combine this signal with knowledge of the structure of the environment in order to compute a decision-utility.

p0200 Remembered utility typically involves an active reconstruction of memory, rather than veridical recall of actual past pleasures. Reconstruction can introduce some distortions, so that the hedonic memory of the event no longer accurately reflects how good the event truly was at the moment of experience. For example, memory of a hedonic experience can be distorted by memory limitations and be heavily influenced by current beliefs (Gilbert, 2006; Kahneman *et al.*, 1997, 1999; Robinson and Clore, 2002; Wilson, 2002).

p0205 Still, whenever people decide about outcomes they have previously experienced in their past, remembered utility is perhaps the chief factor that is used to compute goal-values. That is, people generally expect future rewards to be about as good as they remember them in the past. In turn, remembered utility about past outcomes can be used to generate predictions for future outcomes, corresponding to future expectations or *predicted utility* that will be gained if the goal is ever obtained again. Of course, in addition to this memory-based incentive learning process, individuals (particularly humans) can also compute the predicted utility or goal-values for some potential future events that have not ever been experienced before. Even less is known about these mechanisms, but candidate processes include generalization (i.e., estimating goal-values based on the degree of perceptual similarity to actual remembered outcomes), making use of knowledge acquired about outcome-values through observing others, and theory-based construction based on inferred or instructed knowledge.

Anticipated or Predicted Utility s0045

p0210 Consideration of future outcome values brings us to the concepts of *anticipated utility* or *predicted utility* (Caplin and Leahy, 2001). One form of anticipated utility has been suggested to be a Pavlovian prediction of reward, a state elicited by a stimulus which through repeated pairings comes to be associated with the subsequent presentation of a reward outcome. The Pavlovian nature of anticipated utility means that it does not feature any associations with actions nor does it directly contribute to the computation of decision utility, it simply indicates how much utility is expected to be experienced in the future independently of actions taken by the organism, based simply on the presence of stimuli that have become associated with the subsequence presentation of a reward outcome.

p0215 Another form of anticipated/predicted utility is the cognitive expectation of reward as a declarative or conscious representation of the future outcome. Predicted utility is the term used by Kahneman *et al.* for a more cognitive construal of future goals: "beliefs about the experienced utility of outcomes" that may occur in future (p. 311, Kahneman *et al.*, 1997). In that, sense predicted utility may be equivalent to what we describe here as a cognitive form of anticipated utility. However, while this form of utility does not contribute directly to the computations underpinning goal-directed choice, this signal does interact with instrumental action-selection in interesting ways, sometimes leading to apparently aberrant choices.

IDENTIFYING WHAT DOES WHAT FOR BRAIN MECHANISMS OF OUTCOME UTILITIES s0050

Brain Mesolimbic Dopamine: Anticipated/Predicted Utility or Pure Decision Utility? s0055

p0220 Perhaps the most famous reward mechanism in the brain is the mesolimbic dopamine system, projecting from midbrain forward to the nucleus accumbens and

related structures. Which form of reward utility does dopamine contribute?

p0225 In past decades, the mesolimbic dopamine was thought by many reward neuroscientists to mediate pleasure or experienced utility itself. But that "dopamine = pleasure" idea began to encounter difficulty about 20 years ago. For example, animals and humans with hardly any dopamine in their brain still seem to have normal "liking" reactions (as described above) to the experienced utility of a pleasant sensation such as a sweet taste (Berridge and Robinson, 1998; Cannon and Palmiter, 2003; Sienkiewicz-Jarosz *et al.*, 2005). Conversely, activating dopamine release through genetic mutation or drugs or a deep brain stimulating electrode or drug stimulation fails to increase "liking" reactions to sweetness although extra mesolimbic dopamine makes animals eat more and seem to "want" rewards more (Berridge, 2012; Smith *et al.*, 2011; Zhang *et al.*, 2009). Similarly, people who have deep brain stimulation electrodes implanted in their brain that activate the dopamine system may come to intensely want to stimulate their electrode, and press a button to do so many thousands of times, yet typically never exclaim "that feels nice" or display any other sign of actual intense pleasure (Berridge and Kringelbach, 2011). The people appear to intensely "want" the electrode stimulation, much more than they actually "like" it.

p0230 Today relatively few neuroscientists still believe dopamine to mediate pleasure or experienced utility. Most who study reward and the brain instead believe that dopamine systems mediate some other form of reward utility. So if dopamine is a faux-pleasure mechanism, what is its real utility role? Some neuroscientists, including one co-author of this chapter (O'Doherty), think dopamine is a prediction-error mechanism of reward learning: that is, remembered utility and anticipated utility (Bayer and Glimcher, 2005; Glimcher, 2011; Niv *et al.*, 2012; O'Doherty *et al.*, 2006; Schultz, 2010; Schultz *et al.*, 1997). Other neuroscientists, including the other co-author of this chapter (Berridge), believe dopamine to mediate a pure form of decision utility: namely *incentive salience* or cue-triggered "wanting" (Berridge, 2012; Berridge and Robinson, 1998).

p0235 The view of dopamine as a prediction error or learning mechanism is explained in detail by other chapters (see Chapters 15 and 16). So here we will consider the pure decision utility or incentive salience alternative as proposed by Berridge and his colleagues, as well as some evidence against the dopamine-learning hypothesis. Finally, we will consider what possibility for convergence exists between the two viewpoints, as well as highlighting any remaining irreducible divergence between these viewpoints.

Berridge's Incentive Salience Theory: Dopamine as Pure Decision Utility s0060

p0240 Incentive salience, or cue-triggered "wanting," is a specific form of Pavlovian-related motivation for rewards that is mediated by mesocorticolimbic brain systems, and is especially modulated by dopamine levels (Figure 18.1) (Berridge, 2007, 2012; Berridge and Robinson, 1998; Robinson and Berridge, 1993). "Wanting" typically gives a felt "oomph" to conscious desires that makes a desire feel more urgent, able to influence choice and produce action. In addicts, excessive "wanting" may produce feelings of urge to take the drug so strong that they border on compulsion.

p0245 Yet the core process of "wanting" can also occur unfelt as a relatively unconscious process. For example, drug addicts in laboratory experiments may work hard to obtain injections containing such low doses of cocaine that the addicts say the injections are empty of any cocaine and even deny that they are working at all (Fischman and Foltin, 1992). Even normal people can have unconscious "wanting," for example induced by subliminally-brief flashes of emotional happy facial expressions or of money, and expressed as behavioral tendencies to ingest more and offer to pay higher prices for a subsequently offered beverage, or work harder for monetary rewards, all the while unaware that they've seen anything, or felt anything, or that their behavior has been changed by what they subliminally saw (Berridge and Winkielman, 2003; Pessiglione *et al.*, 2007; Winkielman *et al.*, 2005). Such results have led to the idea that "wanting" is intrinsically an unconscious process, perhaps because it is mediated chiefly by subcortical brain systems, but can be elaborated into conscious cravings when additional brain systems of awareness are recruited (probably involving the prefrontal cortex regions described above).

p0250 In most cases, "wanting" also typically coheres with "liking" (hedonic impact) for the same reward, but "wanting" and "liking" can be dissociated by some manipulations, especially those that specifically involve dopamine and selectively alter "wanting" (Berridge, 2007; Berridge and Robinson, 1998; Smith *et al.*, 2011). And finally "wanting" can also be distinguished from learning about the same reward (Berridge, 2012; Smith *et al.*, 2011; Zhang *et al.*, 2009). For example, "wanting" triggered by a Pavlovian reward cue can dramatically, even if its previously learned value has not changed (e.g., in hunger, satiety, stress, or drug-related states). AU:2

Incentive Salience "Wanting" *versus* Ordinary Wanting s0065

p0255 Incentive salience as Pavlovian motivation or "wanting" has several neural and psychological features that

distinguish it from more cognitive forms of desire (wanting in the ordinary sense of the word). Ordinary cognitive wanting neurally depends more heavily on cortically-weighted brain circuits, computationally conforms better to model-based systems, and psychologically is more tightly linked to explicit predictions of future value based on declarative remembered previous values in episodic memory (e.g., as conscious episodic memories) (Berridge, 2001; Dickinson and Balleine, 2010; Kringelbach, 2010; Liljeholm *et al.*, 2011). Such cognitive desires are based more firmly on explicit representations of the predicted goodness of future outcome, predictions which in turn are often based on declarative memories of previous pleasure of that outcome (Dickinson and Balleine, 2010). For such cognitive desires, decision utility = predicted utility, and predicted utility = remembered utility. That is, we ordinarily desire an outcome to exactly the same degree that we predict the outcome will be liked, and most predictions about future experienced utility are based on memories of how liked the outcome was in the past.

p0260 Incentive salience is different, and not so rational. For incentive salience, under conditions of dopamine-related stimulation, situations exist where cue-triggered decision utility > remembered utility from the past, and similarly decision utility > predicted utility for future reward value (Berridge and Aldridge, 2008). In other words, it is possible to "want" what is not expected to be liked, nor remembered to be liked, as well as what is not actually liked when obtained. In this framework, incentive salience "wanting" is a pure form of decision utility, which is distinct from other forms of utility and in some conditions can decouple from all the others. That is, "wanting" for an outcome is distinguishable from both experienced utility (hedonic impact or "liking" the outcome), remembered utility of how nice the outcome was in the past, and anticipated or predicted utility of how nice it will be in the future.

p0265 Incentive salience integrates two separate input factors to generate decision utility in the moment of re-encounter with cues for a reward that could potentially be chosen: (i) current physiological/neurobiological state; (ii) previously learned associations about the reward cue, or Pavlovian CS+ (Berridge, 2004; Robinson and Berridge, 1993; Toates, 1986) (Figure 18.1). Sudden encounters with Pavlovian cues for a reward can suddenly trigger pulses of motivation to pursue that reward as a goal. Advertisements that pop up on a web page may prompt the finger to click onto the product. The smell of food as you walk down the street near lunchtime may make you suddenly feel quite hungry, even if you weren't feeling that way moments earlier. And encounters with drug cues can precipitate a recovering drug addict who is trying to stay clean back into addictive relapse. When triggered by learned cues, incentive salience typically occurs as temporary peaks of "wanting", relatively transient and lasting only seconds or minutes, and tied to encounters with the physical reward stimuli. Moments of vivid imagery about the reward and its cues may also serve just as well as actual physical cues to trigger incentive salience.

p0270 A particular reward cue may trigger temptation on some encounters but not on others. Fluctuations of the temptation power for cues helps to illustrate the difference between decision utility and predicted utility. States that alter brain dopamine reactivity can selectively alter decision utility of a reward cue. The same drug cue that potently triggers addictive relapse on a catastrophic occasion, spiraling a recovered addict back into drug taking, may have been successfully resisted on many previous encounters. And for everyone, reward cues vary across hours and days in their ability to evoke desire. Food cues are potent when you are hungry, but not so potent when you have recently eaten. Relevant states of physiological appetite, states of stress, or – for compulsive consumers – trying to take "just one" hit or just one taste of a palatable treat, can all enhance the temptation power of reward cues. Explanations for such fluctuations hinges on the unlearned one-half of inputs that determine whether a cue triggers "wanting": current neurobiological state factors related to dopamine at the moment of cue encounter.

p0275 For example, experiments in the Berridge lab have shown that putting a rat's brain into an elevated state of dopamine activation for about a half-hour, by painlessly giving a microinjection of amphetamine into its nucleus accumbens, causes the rat's next encounter with a previously learned Pavlovian cue for sugary reward to trigger a pulse of desire 50% higher than the cue normally would (and 400% higher than moments before when no cue was present). The pulsed amplification of cue-triggered "wanting" occurs without need of relearning yet only at the moment of cue encounter: the intense "wanting" is temporary, reversible and repeatable whenever elevated dopamine and cue coincide. Such pulses of hyper-"wanting" are expressed behaviorally in amplified bursts of frenzied seeking efforts to obtain the reward, and also evident neurally in amplified bursts of neuronal firing in limbic brain targets of the nucleus accumbens, including the ventral pallidum (Smith *et al.*, 2011; Wyvell and Berridge, 2000, 2001).

p0280 In terms of our utility discussion, the incentive salience thesis is that such amplifications are pure and selective elevations of cue-triggered *decision utility*. Before the cue comes, the dopamine-activated brain of the rat simply wants sugar in the ordinary sense, without necessarily showing any elevation of desire. That

is, the dopamine elevation by itself does not alter the expectation of future reward that is *predicted utility*: the rat neither raises nor lowers its constant level of efforts to obtain reward (expressed during the long periods when the Pavlovian cue is absent), a relatively constant level that reflects its previously learned knowledge that the sugar reward can be earned by pressing the lever. The next moment, when the Pavlovian cue suddenly appears and is physically present to interact with the elevated brain levels of dopamine, the stimulated brain transiently "wants" sugar much more to an unprecedented and exaggerated degree. Upon the cue's arrival, the rat engages in a frenzied burst of efforts to obtain the sugary reward, far above normal or previous levels; simultaneously, neurons in its ventral pallidum suddenly fire in an intense burst at a much higher level than they ever normally would if not dopamine-stimulated or if the cue were absent (Smith *et al.*, 2011; Wyvell and Berridge, 2001). Yet just a few moments after the cue ends, the rat returns to its lower and normal level of "wanting" and neuronal firing. Finally again, moments later still, the cue is re-encountered once more and a new burst of excessive and irrational "wanting" again takes control. It seems unlikely that predicted utility (i.e., stable expectations of future reward based on memories of rewards earned in the past) was ever changed by dopamine flooding, because the flooding lasted the whole half-hour, as would stable learned predictions, whereas desire was amplified only at brief moments of cue encounter (interactively combining with the extra dopamine). Likewise, neural recording studies show that dopamine elevations fail to enhance limbic neural firing signals to Pavlovian cues that carry maximal predicted utility information (i.e., information that a reward is about to occur), but instead powerfully enhance neural or maximal incentive salience (i.e., accompany the highest levels of reward-seeking behavior, but giving no new predictive information) (Smith *et al.*, 2011).

p0285 The selective elevation of pure decision utility thus seems to involve a synergy between (fairly constant) elevated dopamine levels and (phasic) encounters with the Pavlovian cue. We hypothesize that the "wanting" synergy mechanism evolved to allow natural appetite and satiety states to dynamically modulate motivation for learned rewards by modulating the reactivity of brain mesolimbic dopamine systems to relevant cues. But the same synergy mechanism also opens windows of vulnerability to stress and emotional states, addictive drugs and to permanent brain changes associated with drugs that cause addiction, and other factors to usurp decisions by likewise raising the reactivity of mesocorticolimbic brain circuitry.

Computational Modeling of Incentive Salience as Decision Utility

s0070

An initial attempt to computationally model such fluctuations in cue-triggered temptation power was recently made by Jun Zhang *et al.* (2009). This incentive salience is different from learning models such as temporal difference or prediction error in that the Zhang model incorporates a dynamic brain state factor κ (kappa), which can change as rapidly as appetite, satiety or drug-state changes, and which modulates motivation generated from the learned value of a relevant CS for reward (r_t) without requiring any new learning about its UCS value in the new physiological state (Figure 18.3).

p0290

In the Zhang model, the cue-triggered incentive salience or motivational value is defined as $V(s_t)$. Computationally, a multiplicative form of the Zhang equation generates incentive salience as:

p0295

$$\tilde{V}(S_t) = \tilde{r}(r_t * \kappa) + \gamma V(S_{t+1})$$

In this equation, $V(s_t)$ denotes the level of incentive salience ($\tilde{V}$) triggered at the moment of encounter ($_t$) with a learned reward stimulus (S_t); (r_t) denotes the previously learned Pavlovian value of the associated reward when the cue is encountered (i.e., remembered utility, such as a cached memory of accumulated reward values generated by a temporal difference or prediction error learning algorithm on previous occasions with the reward; (κ) denotes the kappa factor or current brain state that can amplify the incentive salience of relevant learned cues, and (γ) denotes a temporal discounting factor that reduces the motivation value of more distantly future cues and rewards relative to ones that occur more immediately.

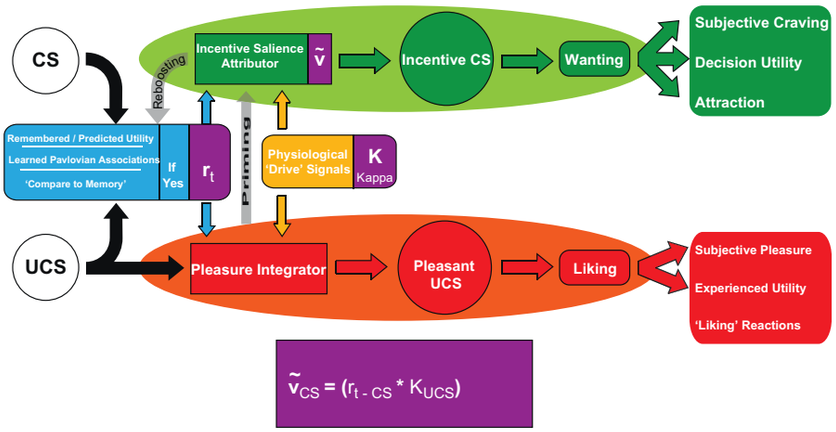
p0300

By this incentive salience model, the important thing is that the decision utility or motivating power of value of a reward cue (r_t) can be raised into higher or lower incentive salience than was learned previously by simply by raising or lowering the κ factor. The change in cue-triggered decision utility would apply instantly to the next encounter of the CS even if the UCS had never been experienced in the new physiological state (Berridge, 2012). That fits the experimental results and human relapse/temptation examples described above. In the new state, the motivation response to the CS would no longer match its previously learned level.

p0305

For convenience, the κ state that held during previous learning trials (i.e., during CS-UCS training) is assumed to be $\kappa = 1$. As long as nothing changes, kappa state can remain 1 and $V(s_t) = (r_t)$. What is most important is the κ state at the subsequent moment of CS re-encounter. Only if $\kappa = 1$ continues to be true at

p0310



f0020 **FIGURE 18.3** Incentive salience distinguishes "wanting," "liking" and learning about the same reward. The remembered utility of a Pavlovian cue's (CS) learned associations with its reward outcome (UCS) is an important input to potentially trigger "wanting" (top), but the decision utility output also involves further computations. Decision utility corresponds to green incentive salience (top) that uses dopamine for generation, whereas experienced utility corresponds to the red "liking" process (lower) that uses hedonic hotspots for generation. Dopamine levels mimic fluctuations of natural appetite or satiety states to act as kappa factor in the Zhang equation (purple, bottom). Dopamine levels, addictive drugs and mesolimbic sensitization in addicts all selectively act to modulate only the incentive salience computation that finally produces decision utility. Thus dopamine elevations can amplify decision utility without changing remembered utility, anticipated/predicted utility or experienced utility. *Figure modified from Berridge (2012), originally based on Robinson and Berridge (1993).*

re-encounter, and physiological state remains essentially unchanged, will "wanting" triggered by the CS match the previously learned value. Any departures of κ from previous value of 1 (i.e., any changes in relevant neurobiological state), will let the level of "wanting" at the moment of CS re-encounter be dynamically modulated. If state declines (e.g., natural satiation state or pathological loss of dopamine), so that $\kappa < 1$, the shift produces a decrement in incentive motivation below the previously learned level. Conversely, if relevant state rises $\kappa > 1$ (e.g., an increase in hunger, an amphetamine microinjection in nucleus accumbens, or an addict taking a priming dose of addictive drug), so that κ the shift enhances CS-triggered levels of motivation above the previously trained amount (Figure 18.3). In these ways, changes in dopamine-related brain state can selectively amplify the decision utility triggered by particular reward-related cues. Some brain states will merely temporarily elevate cue-triggered "wants," such as being intoxicated or hungry or emotionally excited. Other brain states can more permanently render an individual prone to have highly intense cue-triggered "wanting," such as near-permanent brain changes induced by drugs called mesolimbic sensitization that occur in addicts, induced by their history of repeated drug binges. In all cases, the elevations in cue-triggered decision utility can happen without any accompanying elevation in either experienced utility of actual outcomes, remembered utility from past outcomes, or predicted utility of future outcomes.

Applications of Incentive Salience Computation in Economics s0075

The Zhang computational model above has recently p0315 begun to be applied to economic choices and to phenomena such as temporal discounting (in which a smaller and sooner good outcome is chosen over a better but delayed alternative outcome). For example Lade recently suggested that temporal discounting might be better understood by integrating the Zhang incentive salience model with a standard utility function for quasi-hyperbolic discounting (Lade, 2011). As Lade puts it, "cue- triggered 'wanting' increases the motivational value of the immediately obtainable reward, and does not decrease the discounting factor with which future rewards are discounted. Impulsivity can be seen as the desire for immediate gratification on top of the impatience that is already measured by the discount rate δ" (p. 15, Lade, 2011). Such modulations also seem consistent with the *visceral influences hypothesis* of George Loewenstein, a hypothesis which suggests that ordinarily people underestimate the impact that future visceral states such as hunger, emotional or sexual arousal, or even curiosity will have on their future decisions in those states (Loewenstein, 1996; Loewenstein *et al.*, 2003). Related applications have included demonstrations that when people are "jilted" (romantically or socially rejected by another person), or thwarted from obtaining a desired item, they may selectively increase "wanting" for the same item while "liking" it less (Litt *et al.*, 2010), and demonstrations that people's ratings of

incentive values can diverge from their ratings of likeability for the same item (Dai *et al*., 2010).

s0080 ## BERRIDGE'S CRITIQUE OF THE DOPAMINE REWARD-LEARNING HYPOTHESIS

p0320 Proponents of the incentive salience hypothesis above, such as Berridge, also point to empirical evidence suggesting that dopamine does not serve as a mechanism to cause either remembered utility or predicted utility. Such evidence comes from experiments indicating that dopamine actually is not needed for reward learning, and does not causally act as a teaching signal to establish new memories or as a prediction signal to create expectations of future rewards (Berridge, 2012; Cagniard *et al*., 2006; Flagel *et al*., 2011; Robinson *et al*., 2005; Saunders and Robinson, 2012; Shiner *et al*., 2012Smith *et al*., 2011). There are several examples of evidence against the idea that dopamine signals are mechanisms for learning new reward-predictions. One example is evidence that dopamine is simply not needed to learn many kinds of new reward values nor to retrieve previously learned reward values. How can dopamine surges be needed for teaching signals or prediction errors, if many reward values are learned perfectly well without any dopamine? For example, rats that have lost nearly 100% of their brain dopamine (due to microinjections into their brains of a neurotoxin that selectively kills dopamine neurons) remain perfectly able to learn a new dislike for a distinctive sweet taste that they originally liked (through a Pavlovian learning process called taste aversion learning) (Berridge and Robinson, 1998). Likewise a number of new positive reward values are learned quite well without dopamine by mutant mice, which are congenitally unable to make any dopamine because they lack a dopamine synthesis gene: such mice still successfully learn where to find a sugar reward or a cocaine reward (Cannon and Palmiter, 2003; Hnasko *et al*., 2007; Robinson *et al*., 2005). As dopamine-free rats and mice seem to learn those new predictions perfectly well, they seem to have capacity for normal predicted utility values. What they seem unable to do is to "want" the rewards that they "like" and learn about. Without dopamine they would voluntarily starve to death even if surrounded by mountains of tasty food if they were not artificially fed or periodically given dopamine medication.

p0325 Conversely, opposite mutant mice that have *extra dopamine* in their brain synapses seem to "want" rewards more intensely than normal mice, but do not learn any faster or better about rewards (Cagniard *et al*., 2006; Peciña *et al*., 2003). Similarly, boosting dopamine in people who need it may fail to improve learning *per se*, but rather improves performance in a way that suggested the dopamine boost specifically enhanced their attention and motivation to earn reward (Shiner *et al*., 2012; Smittenaar *et al*., 2012). These patterns help bolster the conclusion that dopamine changes most specifically alter decision utility ("wanting") without necessarily altering remembered utility, predicted utility or experienced utility (learning or "liking").

p0330 Proponents of the incentive salience hypothesis also offer an alternative non-learning explanation for why dopamine neuron activations so often obey the prediction error model of reinforcement learning, as described in other chapters of this book: the dopamine neurons are actually coding decision utility as "wanted" value. They suggest that dopamine signals appear to encode pure prediction errors in many studies because those studies have allowed experimental confounds that let Zhang equation decision utility signals mimic prediction error signals (Berridge, 2012).

p0335 Why have prediction error theorists mistaken dopamine as a remembered/anticipated utility mechanism if it actually causes pure decision utility? Because they have relied so heavily on experiments that confounded those utility types together. Remember that when $\kappa = 1$ in the Zhang equation, the incentive salience output mimics a temporal difference model (which provides half the input to incentive salience). That is because the (r_t) associative input is not transformed when $\kappa = 1$, but rather is copied faithfully from cached learning input to become the motivational output. Whenever the physiological state in training is nearly replicated in subsequent testing, $\kappa = 1$. Because the Zhang equation takes prediction error signals as one-half the input, experiments that clamp mesolimbic brain states at a constant level ensure that only the learning half of prediction error inputs will be expressed as Zhang equation outputs in incentive salience. Physiological clamping essentially puts an experimenter's thumb on the "scale" to make sure decision utility always tracks predicted utility. If participants are trained and tested in a constantly clamped physiological state, dopamine neurons will look like they code prediction errors even if they actually code incentive salience. For example, monkeys in classic dopamine neuron recording experiments usually were tested in constant thirst. And most human neuroimaging studies typically test participants in a single constant state. Such studies avoided variation in states that would alter the Zhang equation kappa value (e.g., appetite, satiety, stress, intoxication, withdrawal). Prediction error theorists may have therefore mistaken some mesolimbic decision utility signals for current

value to be pure prediction signals that provide inputs to the computation.

p0340 In real life, physiological states typically fluctuate between appetites and satiety, intoxication and sobriety, stress and relaxation, etc., unlike in state-clamped experiments. Real life fluctuations therefore powerfully modulate the decision utility or temptation power of relevant reward cues. That is why one can easily ignore food cues after dinner, but be riveted and motivated by the same food cues if one hasn't eaten all day, and why an addict who has successfully resisted drug cues many times, may upon a later encounter with the same cues be precipitated back into addictive relapse (for example, in a state of stress, or emotional excitement, or after having just tried to take "just one hit"). These fluctuations in states and the amplified temptation power of the reward cues all act by raising kappa to multiply the decision utility triggered by relevant cues. That is the essence of the incentive-sensitization theory of addiction: drug cues trigger intense "wanting" in addicts who have sensitized brains, especially when encountered in vulnerable states of raised kappa (Robinson and Berridge, 2003; Zhang *et al.*, 2012). Such addicts can "want" their drugs far more intensely than is justified by either their learned values of remembered utility or expectations of future predicted utility for the same drugs.

s0085 O'DOHERTY'S RESPONSE TO THE CRITIQUE OF THE REWARD-LEARNING HYPOTHESIS

p0345 Much primary evidence in favor of the prediction error hypothesis is described in Chapters 15 and 16. In response to the incentive salience critique above, a number of points can also be made in defense of the reward-learning hypothesis presented in those chapters. These replies are suggested here by co-author O'Doherty. First of all, O'Doherty contends that while there is some evidence from the selective dopamine lesion studies and genetic studies described above to indicate that aspects of reward-learning can remain intact without the presence of dopamine, additional evidence by some of the same researchers involved in the studies cited above and others, indicates effects of dopamine manipulations directly on learning (Darvas *et al.*, 2011; Frank *et al.*, 2004; Parker *et al.*, 2011; Robinson *et al.*, 2007). Furthermore, out of the studies that fail to report an effect of dopamine on learning cited above, such studies have typically not been designed to separate out different types of reward-learning such as Pavlovian conditioning and two types of instrumental conditioning: goal-directed and habit learning (Balleine and Dickinson, 1998). Typically in such paradigms, all three of these processes are operating and could contribute to observed behavior. The reason why this is important for the debate is that according to recent proposals regarding the role of dopamine in reinforcement-learning (see Chapter 21) it is suggested that dopamine contributes only to some types of reward-learning but not other types. Specifically, in the domain of instrumental conditioning dopamine is suggested to contribute only to habitual stimulus-response learning but not to learning of goal-directed instrumental associations. If dopamine is not involved in goal-directed instrumental learning but is involved in habitual learning, tasks that confound these processes may not be sensitive enough to detect learning impairments after dopaminergic challenges, because behavior could still in principle be supported by the system left unaffected by the absence of dopamine. In order to definitively address this question it would be necessary to use appropriate tasks and behavioral methods in order to discriminate between different instrumental learning processes such as *over-* and *under-training*, and *reinforcer devaluation* (see Chapter 21) which can discriminate habitual from goal-directed control (Balleine and Dickinson, 1998; Dickinson and Balleine, 2010).

p0350 Furthermore, even within the Pavlovian system there appears to be evidence that some types of learning may be under dopaminergic control while others may not. Recent evidence suggests that Pavlovian conditioned sign-tracking behavior may depend on dopamine and on reward-prediction errors during learning, while another type of Pavlovian conditioned behavior, goal-tracking may not depend on dopamine, and more specifically on reward-prediction errors (Flagel *et al.*, 2011). Consequently, even in a simple Pavlovian conditioning paradigm it may be necessary to distinguish between those reward-learning systems that are dopamine dependent from those which are not. New tools are rapidly coming on line that will enable this question to be more definitively addressed. These include new methods for selectively activating dopamine neurons using optogenetics. It has already been shown using optogenetic techniques that dopamine activity is sufficient to enable reward conditioning to take place (Tsai *et al.*, 2009), although in that particular paradigm the precise cause of the effect could be attributed to either the reward-learning hypothesis or the incentive salience hypothesis.

s0090 INTEGRATION BETWEEN THE TWO VIEWPOINTS?

p0355 Are there any grounds under which these two hypotheses can be reconciled? In the opinion of both

co-authors there is room for a degree of accommodation between the two viewpoints. Due to the remarkable efficiency of brain systems conferred by evolution, dopamine is unlikely to be involved exclusively in one function. Instead, it is entirely feasible that dopamine accommodates multiple functions, from movement to attention, and several of these and other additional functions simultaneously may be involved in choosing and pursuing goals.

p0360 Further, in the opinion of O'Doherty, this multiplicity of dopamine functions may even be enough to reconcile learning *versus* motivation hypotheses of reward utility described above. In other words, dopamine may be necessary for certain types of reward-learning as well as for performance of reward-related behaviors. One way this could occur is through different actions of dopamine in different locations in the brain, on different types of receptors, as well as on different temporal scales. For instance, some computational models of dopamine function propose that phasic and tonic dopamine have different properties, whereas phasic dopamine represents reward-prediction errors, tonic dopamine is proposed to be involved in modulating the vigor of instrumental responding, somewhat similar to that described in the incentive salience hypothesis (Niv *et al.*, 2007).

p0365 However, in the opinion of Berridge, dopamine is not likely to be needed to mediate any type of reward-learning at all, and so not be a component of remembered utility or predicted utility. He would counter that at least some demonstrations where rodents successfully learned-without-dopamine included rather pure Pavlovian learning (e.g., taste-aversion learning in which a taste CS predicts an illness UCS). Others included learning that would be considered either to be Pavlovian or habits, so that either/or both types of learning occurred without dopamine (e.g., dopamine-free mice learn to prefer a drinking spout that contains sugar water over another water spout; this can be viewed either as Pavlovian learning (i.e., spout = CS, sugar = UCS) or a learned stimulus-response habit (i.e., a motor habit comprising a sequence of movements toward the sugar spout). Further, many studies ostensibly finding prediction error encoding by phasic brain activations may be flawed by experimental confounds (e.g., due to clamping of physiological states) which makes their results ambiguous. Finally, Berridge views the special dependence on dopamine of *sign-tracking* (being attracted toward a CS that predicts reward) as occurring precisely because dopamine mediates the incentive salience "wanting" that must be targeted to the CS to make it attractive (not because dopamine was needed to learn that the CS predicts reward), whereas goal-tracking has other mechanisms available to reach its goal (including habit-learning, which again in this case seems not to need dopamine) (Flagel *et al.*, 2011; Saunders and Robinson, 2012). This interpretation seems to be supported by others who study dopamine in sign-tracking, including some involved in the study mentioned by O'Doherty: "we suggest that the role of dopamine in the nucleus accumbens core in stimulus-reward learning is to attribute incentive salience to reward cues" (p.10, Saunders and Robinson, 2012). If the view of those authors and Berridge is correct then the reward-learning hypothesis for dopamine would of course need to be discarded.

p0370 In a further counter-response, O'Doherty would point out that evidence from taste-aversion learning may not be conclusive because according to most formulations of the dopamine-learning hypothesis, aversive conditioning may not depend at all on dopamine reward-prediction error signals, and because taste aversion is a unique form of Pavlovian conditioning in that it requires dramatically less temporal contiguity between CS and UCS in order to take place than other forms of conditioning, therefore suggesting a dependence on very different computational mechanisms than other forms of conditioning.

p0375 Still, both co-authors heartily agree that dopamine is involved in performance factors. This is clearly the case, because of the basic observation that the absence of dopamine results in the inability for animals and humans to generate movements. Thus, both co-authors agree that an integrated theory of dopamine function will need to account for the potential contributions of dopamine to both learning and performance.

CONCLUSION

s0095

p0380 Experienced utility is the essence of rewarding outcomes, but several other types of reward utility also contribute to the decision to choose a particular outcome. This combination of utility types complicates the tasks of economists, psychologists and neuroscientists who wish to understand how decisions are made.

p0385 Here we have suggested that experienced utility is registered in the brain by widespread neural activations in a diffuse circuit network involving many brain structures. By comparison to registration that codes an outcome, the causal generation of its experienced utility may restricted to a much smaller network comprising deep brain hedonic hotspots.

p0390 Beyond experienced utility, other types of anticipated/predicted utility, remembered utility, and decision utility are also involved in choosing and pursuing reward outcomes. The particular brain mechanisms that mediate these additional utility forms are becoming clearer, though debates still continue about important mechanisms, such as the role of dopamine. We

expect that future research will resolve these debates and produce even more agreement. Such developments will further build scientific understanding of how experienced utility arises in the brain, and becomes translated into decision utility.

p0395 Finally, the existence of multiple types of utility must be acknowledged to raise potential quandaries for policy makers, at least in situations when decision utility diverges from experienced utility. In such a situation, a person may choose an outcome with highest decision utility that fails to maximize their actual experienced utility. Conversely, constraining them to accept a different outcome that carries highest experienced utility may force them to forego the one with highest decision utility. Should a policy maker nudge such cases into maximized experienced utility to ensure greatest pleasure and least pain (Kahneman *et al.*, 1997; Thaler and Sunstein, 2009)? Or instead allow unconstrained freedom of choice to maximize decision utility at the expense of bruised hedonic outcomes? It is beyond our scope to answer such questions, but we hope the process of arriving at better answers might be informed by the perspectives on utility sketched here.

s0100 Acknowledgment

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