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CHAPTER 12

A Liking Versus Wanting Perspective on Emotion and the Brain

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Abstract

Reward, pleasure, threat, fear, and disgust are emotional labels that we often use with confidence, as if we knew the identity of their corresponding psychological processes. Those psychological processes of emotion are quite real and deeply grounded in brain systems shared by humans with many animals. But, the identity of fundamental psychological components within emotion are sometimes mistaken because only the final products are experienced, losing the identity of important psychological components that arise en route. Some of those components can have counterintuitive psychological features. For example, the experience of pleasant rewards actually contains distinct psychological processes of "liking" (hedonic impact) and "wanting" (incentive salience). Experience of fear-evoking threats hides distinct psychological components of passive reaction and an actively coping form of fearful salience. Perhaps most counterintuitively, the component of "fear" salience in threat shares a hidden psychological and neural relationship to that of "wanting" for rewards. These psychological components have implications both for ordinary emotions and for pathological disorders ranging from addiction to paranoia. Affective neuroscience studies in this way can produce surprises and insights into the psychological structure of emotions.

Keywords emotion, affect, brain studies, wanting, liking, subjective feelings, emotional reactions

What Is an Emotion?

I believe the defining feature of an emotion is that it has *affect*: a quality of pleasure versus displeasure. That is not just my idea, of course. The proposition that affect defines emotion dates back at least a century of psychology to Wilhelm Wundt, who proposed that all emotions could be plotted along one dimension of affect (from positive to negative) and another of arousal (from low to high) (Wundt, 1904), and the dimensional idea of emotion has been endorsed by many psychologists ever since (Barrett, 2006; Russell, 2003). Wundt and these psychologists typically have viewed the affect dimension as a single continuum stretching from strong pleasure through neutrality to strong displeasure. This can be called a one-dimensional (1D) view of affect in emotion. An alternative two-dimensional (2D) view is that pleasure and displeasure are really separable from each other, with distinct brain mechanisms, and are better described as two different and orthogonal dimensions that can change independently of each other (Berridge & Grill, 1984; Cacioppo, Gardner, & Berntson, 1997; Gray & McNaughton, 1996; Lang, 1995; Larsen, McGraw, & Cacioppo, 2001; Norris, Gollan, Berntson, & Cacioppo, 2010). These views are theoretically quite distinct, but experimentally, it has proven quite difficult to find evidence that clearly favors either the 1D or the 2D view. This difficulty arises partly because even 2D advocates must concede that positive and negative mechanisms often exert reciprocal inhibition on each other, so that in practice they do not operate independently, even if their underlying generating mechanisms are different. Perhaps that is why debate about the dimensional nature of emotional affect has persisted for over 100 years.

Whether as 1D or 2D, positing affect as the defining feature of emotion helps to distinguish

emotions from other psychological processes that also are evaluative, such as cognitive appraisals. For example, cognitive appraisals of value can also be characterized as positive or negative, such as the sense of being useful or beneficial versus disadvantageous or detrimental to the individual's goals (Ellsworth & Scherer, 2003). But, cognitive appraisals are not necessarily also emotional reactions, by the affective definition, unless those appraisals also involve affect that is actively generated in the moment.

Of course, defining emotion as affect sets only a minimalist criterion for emotion and is not a sufficient definition of any particular type of emotion (e.g., anger, fear, etc.). Still, the emotion-as-affect definition helps highlight a minimal basic feature needed for distinguishing an emotion from other psychological processes and experiences and guides a search for emotion in the brain by specifying that we look for affective mechanisms.

What Do Brain Studies of Emotion Offer to Psychology?

What use are brain studies when it comes to emotion? Obviously, affective neuroscience studies can tell us about the brain, but their primary value, in my view, is that they can also tell us new things about the *psychology* of emotion. That is, studying the brain can help us to understand the psychological essence of emotional processes, such as by showing us surprising phenomena that simply should not exist according to the ways we have thought psychologically about emotion in the past. Such surprises sometimes force us to rethink the psychological nature of emotional processes to explain better the phenomenon we have just seen revealed by the brain manipulation.

My colleagues and I have encountered several surprising emotional psychology features in our own affective neuroscience research, and I describe some of these examples here. One was the finding that liking a reward and wanting that reward are two very different processes from the brain's point of view, with implications for both normal and pathological psychological processes (Berridge & Kringelbach, 2015; Berridge & Robinson, 2011). Another was the finding that the affectively opposite emotions of desire and dread actually share neural underpinnings and some psychological features and can sometimes even be converted back and forth from the same generating events in the brain.

Wanting Versus Liking

Ordinarily in life, when you say you like something, it is natural to expect that you also want that thing. Liking and wanting seem to be two sides of the same coin, and the two words are almost synonyms in daily use. But they turn out to have underlying core processes that are separable. Here, I will use these terms in quotation marks to refer to the underlying core processes of "liking" (hedonic impact) and "wanting" (incentive salience), which have objective as well as subjective features, and which in some situations can occur objectively without conscious awareness (Berridge & Winkielman, 2003; Winkielman, Berridge, & Wilbarger, 2005). This use distinguishes these core processes from the subjective experiences of liking and wanting, meant by the same words without quotation marks.

In academic psychology as well, the concept of the incentive value of reward was used for decades to represent both liking and wanting. Indeed, several psychological theories of incentive value treat these two aspects as interchangeable and essentially identical. And, this was once also my view—until we were surprised.

Our finding that there are separate brain systems for "liking" and "wanting" a single pleasant reward came from a set of, in a sense, failed experiments. The goal of those experiments was to confirm the pleasure role of a brain mechanism that was widely thought to mediate liking for rewards: the mesolimbic dopamine system. Most previous behavioral neuroscience studies that concluded dopamine caused pleasure had inferred that dopamine manipulations changed how much a reward was "liked" (hedonic impact) by observing changes in how much the reward was "wanted" (preferred, pursued, worked for, or consumed) (Wise, 1985). Our approach was a bit different in that we tried to assess dopamine-caused changes in hedonic impact more specifically, without mediation by "wanting," by measuring facial "liking" reactions to sweet taste reward. We did this by focusing on positive hedonic facial expressions of taste pleasure, such as those that are elicited from human infants by sweet tastes (e.g., lip licking). Infant facial expressions of disgust, by contrast, are elicited by bitter tastes (e.g., gapes and headshakes). Apes and monkeys, and even rats, also display similar positive "liking" expressions to sweetness, versus negative "disgust" expressions to bitterness (Fox & Davidson, 1986; Grill & Norgren, 1978; Steiner, 1973; Steiner, Glaser, Hawilo, & Berridge, 2001), and we used "liking" reactions of rats because they were the most feasible way to ethically manipulate brain systems.

Pleasure is never merely a sensation alone: Affect is always actively generated by the brain (Berridge & Kringelbach, 2015). Correspondingly, the facial affective expressions are not simply functions of taste sensation; "liking" expressions also can be shifted by physiological or psychological manipulations of the taste's hedonic impact, such as by hunger/satiety shifts or by inducing learned preferences or aversions—or by brain manipulations that alter pleasure circuitry. Applying our "liking" expression measure to dopamine, then, we expected to find that drugs that suppressed dopamine systems would reduce "liking" reactions to sweetness. This prediction was supported by many experiments that had found such drugs reduced the incentive value of sweet food or other rewards when using "wanting" measures, so we were quite surprised to find in our first experiments that the drugs caused no change in "liking" facial expressions elicited by a sweet reward.

At first, we thought we had done something wrong as most affective neuroscientists of the time, after all, were pretty sure that mesolimbic dopamine was the brain's chief pleasure neurotransmitter. So, we followed up over a 20-year period with various related and stronger manipulations: dopamine brain lesions, electrical stimulation, direct brain microinjections, and others. But, we always found that "liking" for sweetness remained unchanged, even though these brain dopamine manipulations powerfully changed "wanting" for the same reward (Berridge & Robinson, 1998; Berridge, Venier, & Robinson, 1989). Because we could successfully change "liking" by manipulating other brain limbic systems, the failure of dopamine manipulations to alter the hedonic impact of sensory pleasure stood in stark contrast to opioid manipulations that enhanced "liking" reactions, or to ventral pallidum lesions that abolished normal "liking" for sweetness (Berridge & Kringelbach, 2015). What dopamine loss actually seemed to do was to selectively abolish "wanting" for the reward while leaving its liking intact.

Subsequent brain studies confirmed that increasing dopamine also did not enhance pleasure "liking," though extra dopamine boosted wanting. After about 10 years, dopamine suppression studies in humans also began to confirm that manipulating dopamine release in the brain changed subjective wanting ratings for drug or food rewards but did not change liking ratings for the same rewards when those ratings were teased apart (Boileau et al., 2016; Brauer & De Wit, 1997; Leyton, 2010; Sienkiewicz-Jarosz et al., 2013). Dopamine-related incentive salience or "wanting" typically gives a felt "oomph" to a conscious desire that makes it urgent, allowing it to control choice and produce action.

Based on the wanting/liking distinction, and on the discovery that addictive drugs can sensitize as well as stimulate dopamine systems, my colleague Terry Robinson and I suggested that excessive wanting may help explain what happens in some cases of compulsive addiction (T. E. Robinson & Berridge, 1993). In addicts, excessive "wanting" may produce urges to take the drug so strong that they border on compulsion. Fortunately for most people, both "liking" and "wanting" usually happen together for rewards in life. But, some addicts may have sensitized brains, which involve persistent neural changes caused by addictive drugs in dopamine-related mesocorticolimbic systems. Brain sensitization is nearly the opposite of brain tolerance to drugs: Both can happen simultaneously in the same brain, but sensitization lasts longer. The result of sensitization may be a bit like too much dopamine: excessive wanting even if the drug is not particularly "liked" and even if withdrawal symptoms are long gone.

More recently, the separation of wanting from liking has also been suggested to apply to other behavioral addictions, ranging from binge eating and other eating disorders to compulsive gambling, at least for some individuals (Boileau et al., 2014; Cowdrey, Finlayson, & Park, 2013; Davis et al., 2009; Voon et al., 2014). These individuals show a similar hyperreactivity to their addiction-related cues, suggesting that their mesolimbic systems have become similarly sensitized, even without drugs. It has long been known that some nondrug experiences can induce sensitization similarly to drugs (e.g., severe stresses, salt appetite induction, etc.), so it is not at all inconceivable that sensitization-type brain changes might endogenously arise in some individuals through particular experiences to produce behavioral addictions.

Pleasure Generators: Fragile Network of Small Hedonic Hotspots

If not dopamine, then what is the neural generator of "liking" or pleasure itself? A much more restricted brain circuit appears to mediate "liking" than incentive motivation "wanting" (Berridge & Kringelbach, 2015). The generation of pleasure "liking" is more restricted *neurochemically*: Opioid stimulation, but not dopamine stimulation, in some limbic structures can enhance "liking" (whereas "wanting" is enhanced by both). "Liking" is also more restricted *anatomically*: enhanced by opioid "hotspots" but not by the rest of the same limbic structures (even if the entire

structure can enhance "wanting"). And. "liking" generation is also more restricted as a brain *circuit*, requiring unanimous activation of multiple hotspots simultaneously (whereas "wanting" can be enhanced by a single hotspot). In short, enhancement of pleasure "liking" is restricted and fragile, and brain pleasure systems are relatively recalcitrant to activation compared to "wanting" systems. Consequently, our brain limbic mechanisms may consign us more often to states of desire than of pleasure.

My colleagues and I have called these anatomically small pleasure-generating islands of brain tissue contained within the larger sea of a limbic structure, such as nucleus accumbens or ventral pallidum, "hedonic hotspots." The size of each hotspot discovered so far is only a cubic millimeter in volume of the brain of a rat. In the brain of a person, a hotspot would be expected to be about a cubic centimeter in volume, extrapolating on the basis of the difference between rats and humans in whole-brain size.

A signature feature of these hedonic hotspots is that they can generate increases in pleasure "liking" reactions to sweetness when stimulated with appropriate neurochemical microinjections. For example, pleasure-enhancing neurochemicals stimulate opioid receptors that detect heroin-like neurochemicals in the hotspots or endocannabinoid receptors that detect marijuana-like neurochemicals. Bu, no "liking" enhancement occurs if the same drug microinjections are moved outside the boundaries of the hedonic hotspots, even if they are administered within the same structure. Outside the hotspots in these structures, the microinjections instead stimulate intense "wanting."

Several of these hedonic hotspots have been found, scattered across the brain from cortex to brainstem; they are functionally interconnected like an archipelago of interacting islands. Hotspots are found in limbic prefrontal cortex, nucleus accumbens, ventral pallidum (the chief target of nucleus accumbens), and the brainstem pons. The entire network may need to activate together as a single integrated circuit to amplify sensory pleasures. This integration involves cooperation between the various hotspots. For example, activation of one hotspot by an opioid microinjection automatically recruits activation in other hotspots in different brain structures (Smith & Berridge, 2007). Pleasure magnification requires unanimity among all opioid hotspots in the nucleus accumbens and ventral pallidum (Smith & Berridge, 2007). Pleasure will not be enhanced by a hotspot opioid activation if unanimity is prevented by simultaneously suppressing another hotspot with an opioid-opposing drug. Although opioid stimulation of either hotspot would usually be sufficient to enhance "liking," it cannot do so if the larger circuit is prevented from joining in the activation. However, stimulation of "wanting" persists after either hotspot is activated, even if "liking" enhancement has been prevented. Thus, partial activation of the limbic circuit is enough to generate intense desire, whereas intense pleasure generation requires the whole.

Sharing Between "Wanting" and "Fear"

Another surprise that has arisen in our affective neuroscience studies is the surprising overlap in mesolimbic mechanisms between positive reward "wanting" and an active form of "fear." An enormous psychological divide appears to exist between these motivations: One is positive in valence and the other negative. But, they paradoxically share motivational mechanisms, which can be seen by manipulating the nucleus accumbens, especially the portion called the medial shell, near the midline (Reynolds & Berridge, 2008). Of course, several other brain structures, from amygdala to hypothalamus, are also known to mediate various aversive emotional reactions, including fear. The amygdala is especially well known in rats for fear learning of protective responses to threats, such as passive freezing to a Pavlovian cue that predicts foot shock (LeDoux, 1996). The nucleus accumbens, by comparison, produces a more active coping form of fearful reaction.

Either "wanting" or "fear" can be activated in rats by tapping an appropriate emotional key in the nucleus accumbens shell with a drug microinjection. This part of the nucleus accumbens appears functionally arranged as a sort of emotional keyboard that organizes generators of emotion in a valenced pattern from front to back (Reynolds & Berridge, 2008). Tapping keys in the front of this structure elicits strong desires, reflected as large increases in eating behavior (doubling or quadrupling food intake), or creating a desire to return to the place where the key was tapped (i.e., establishing a conditioned place preference for the location where the drug microinjection previously occurred). Tapping keys in the back of the nucleus accumbens shell with the same drug microinjection conversely elicits strong fear in an actively coping form. For example, a rat may emit defensive antipredator reactions toward people seen in the room, similar to reactions that rodents ordinarily emit toward threats, such as when a mother ground squirrel defends the pups in her burrow against an approaching rattlesnake, using forepaws to throw sand toward the perceived threat

(Coss & Owings, 1978). This action is sometimes followed by fearful squealing, escape attempts, or even biting of the approaching hand if a person gently attempts to pick up the normally tame rat at the end of a session. All of these reactions can be produced by microinjections of a drug such as DNQX, a glutamate receptor-blocking drug that prevents nucleus accumbens neurons from becoming excited. This blockade stops nucleus accumbens neurons from exerting their normal inhibitory influence on downstream neurons in output targets, and excites or disinhibits those target neurons to generate the intense "wanting" or "fear" reactions.

The shared mechanism of desire and dread takes the form of valenced motivational salience generated by neural events in mesocortical circuitry sending signals to the nucleus accumbens. The affective valence of the generated intense emotion can be flipped back and forth between positive and negative by psychological factors, such as the emotional ambience of the outside environment. Even in the same individual and in the same hour, both valences can be activated together by a brain manipulation, and it is possible to convert one into the other (Reynolds & Berridge, 2008; Richard & Berridge, 2011). Incentive salience makes a reward-related stimulus sought out, attention grabbing and attractive and able to elicit approach. Applied to fearful salience, a similar process may make a frightening stimulus attention grabbing and compel a motivated response of avoidance or repulsion, almost as an incentive stimulus grabs attention and compels approach, but the threat is perceived as negative, requiring a coping response and perhaps even attack.

Desire and dread are opposites in valence, but we have found that taps in the middle of the accumbens keyboard do not cancel each other out but reliably elicit both motivations together in the same rat. This overlap made us wonder whether a shared mechanism of motivational salience that has relatively plastic valence might contribute to generating both states. If so, we hoped it might be possible to flip an intermediate neural key in the accumbens back and forth between generating fear and generating desire as a flexible psychological building block, depending on the psychological context in which it was activated.

We first examined the shared mechanism possibility by testing the effects of the microinjections in situations where the ambience could be flipped: either a soothing environment (the rat's own home: dark, quiet, and familiar) or a stressful environment (a room with bright lights [rats avoid bright places] and where a loud discordant soundtrack played [by the musician Iggy Pop]). The rats much preferred their home to Iggy Pop if given a choice (Reynolds & Berridge, 2008). But if not given a choice, and simply plopped in one or the other environment after a brain microinjection, the environment determined what the brain building block built. We found that many middle keys in the accumbens could switch between desire and dread, depending on which environment the rat was in as the microinjection acted. Some accumbens sites even flipped valence completely, changing from pure fear generators to pure desire generators or vice versa.

It turns out that both the intense desire and the intense fear depend on mesolimbic dopamine reaching the nucleus accumbens (although acting there in slightly different ways) (Richard & Berridge, 2011). The involvement of dopamine in both fear and desire is further evidence that they mechanistically share something in common. This role of dopamine in fearful salience has been suggested as related to the ability of antidopamine drugs to treat fearful paranoia in schizophrenia. For example, nearly all traditional antipsychotic drugs block dopamine receptors, and even newer "atypical" antipsychotics that also affect serotonin or other receptors may still owe most of their clinical efficacy to blocking dopamine D2 receptors (Howes & Kapur, 2009). Overall, such demonstrations reveal a surprising degree of overlap in the functional architecture that can be shared across positive and negative emotions.

Unconscious Emotion as Pure Example of Objective Emotional Reaction, Separable From Subjective Emotional Feelings

Measuring "liking" expressions to sweetness, or pursuit or consumption behavior to assess "wanting," taps into objective behavioral responses, not subjective reports of feelings. So, can objective measures truly reflect emotional or motivational processes? This question can be rephrased by asking, Are emotions necessarily subjective, always consciously felt? Psychologists are familiar with many psychological processes that are not consciously experienced: implicit perceptions, implicit memories, implicit cognition, and so on (Kihlstrom, Mulvaney, Tobias, & Tobis, 2000; Schacter, 1994; Wiers & Stacy, 2006). So, cannot emotions be implicit or unconscious as well? Many would draw a line when it comes to emotions because for some people subjective feeling seems to be the essence of emotion. After all, just like wanting and liking, emotion and feeling are words often used almost interchangeably in daily life.

In my view, evidence indicates that unconscious emotional reactions can occur in ordinary people, at least in particular situations (Berridge & Winkielman, 2003; Winkielman, Berridge, & Wilbarger, 2005). For example, affective valence can be unconsciously triggered as a positive or negative hedonic reaction and, once triggered, can go on to unconsciously influence a person's behavior without the person becoming aware of that affective reaction. Examples of unconscious core reward effects have been demonstrated in people ranging from drug addicts to ordinary college students. Such findings have important implications, suggesting that emotional reactions have an objective identity independent of their manifestation in subjective feelings.

As an experimental example of unconscious emotional reaction, Piotr Winkielman, in an experiment carried out in collaboration with Julia Wilbarger and me several years ago, asked college students to view a computer screen on which they were told faces would be flashed for a half second; the student's task was to identify the gender of the face as woman or man (Winkielman et al., 2005). Half a second is long enough to consciously see the face but not long enough to closely inspect it. It was not too hard to judge the gender of the photographs presented, but uncertainty or a mistake also can occur when one has only a half second to view. Unbeknown to the students, subliminally fast flashes of other faces with happy or angry emotional expressions were also sometimes flashed on the screen (1/60th second each), just before the half-second face they were asked to identify. Either a series of happy faces was flashed during the session, a series of angry faces, or a series of emotionally neutral or blank-expression faces. The participants reported seeing no emotional expressions, and they could not pick the emotional faces they saw out of a lineup of other faces later, indicating that their conscious experience of the emotional faces had been avoided by the subliminal procedure. Finally, the students were also told they would subsequently be asked to judge a new fruit-flavored drink that was being brought to market by a beverage company, and they were given a pitcher of the drink to pour, taste, and rate before the end of the session.

All the students reported their own hedonic mood and emotional feelings just before they started and again after viewing the faces (Winkielman et al., 2005). Their emotional reports were not shifted at all by either happy or angry faces they had subliminally "seen," compared to their ratings before they started or compared to people who had seen a different emotional set of flashes. That is, when students subliminally viewed happy faces, they did not feel any increase in positive mood or in feelings of contentment. Instead, their hedonic mood continued to be rated at baseline levels. Similarly, after seeing angry faces, they did not feel any increase in negative mood or irritability. In other words, when assessed by conscious emotional feelings, the flashed emotional faces either had not been seen or had no emotional impact if seen.

Yet, when presented a few minutes subsequently with the novel beverage, the participants found the drink 50 percent more attractive if they had just seen subliminal happy face photos than if they had seen angry face photos: They poured and drank more and rated the drink more highly. Further, they expressed willingness to pay four times more for the drink if it were sold when asked after the happy faces than after the angry faces.

Winkielman and colleagues interpreted this result to mean that the subliminal happy faces activated incentive mesolimbic circuits of positive emotional reaction (both "liking" and "wanting") in the brains of students who viewed them, which persisted for some minutes undetected as students evaluated their own mood (Winkielman et al., 2005). The "wanting" surfaced only when an appropriate target was finally presented in the form of a hedonically laden sweet beverage that they could taste and choose to ingest or not. Further, the emotionally induced changes in drink ratings and behavior were strongest if the student had begun the session feeling at least a bit thirsty. Interaction of internal states with perceived incentives is a signature feature of the "wanting" process that my colleagues and I call *incentive salience*. This process is mediated by brain mesolimbic systems that use dopamine as a main neurotransmitter and seems able to occur either as conscious feelings of wanting or as unconscious "wanting," as seemed to be the case here.

Evidence for unconscious emotional processes have also been found in people without using brief visual flashes. A striking example comes from studies of apparently unconscious self-administration by drug addicts (Fischman & Foltin, 1992; Lamb et al., 1991). For example, in a study by Fischman and her colleagues, recovering addicts were invited to the laboratory, where they were comfortably seated (Fischman, 1989; Fischman & Foltin, 1992). Intravenous lines were inserted into their veins. The subjects could obtain either of two intravenous infusions, depending on which of two buttons they pressed. On a particular day, one intravenous

infusion might contain a high dose of cocaine while the other contained a low dose of cocaine. On another day, one might contain saline solution without drug while the other contained cocaine. Or—for all the subjects knew—both lines might contain merely saline. Each time the addict pressed one of the buttons, it turned on a light and delivered a pulse of its particular infusion. The subjects were free to try the solutions and to administer each to themselves as they chose.

At moderate-to-high doses (8–50 mg cocaine), subjects described the subjective effects as typical of cocaine, and they reliably pressed the button that would obtain the highest available dose. But, at the lowest dose of cocaine tested (4 mg), a remarkable dissociation occurred between self-administration and subjective effects. At this very low dose, the subjects reported that they had received only saline, and that the solution contained no cocaine. Indeed, no cardiovascular responses were observed, supporting the subjects' mistaken contention that the infusion was drug free. But, the 4-mg dose was not below threshold by all measures. According to the cumulative record of button pushing over the 2- to 4-hour session, the addicts chose and pressed the button that delivered 4 mg cocaine far more often than for saline, even while they were unable to detect consciously a difference between the two infusions. As the lead author, Marian Fischman recounted later: "If you want to know what the subjects say about their self-administration of these low doses, they tell me that they were not choosing cocaine over placebo. They often insist that they were sampling equally from each of the two choice options and both were placebo. On the other hand if you look at the data from that session you see that they were choosing the low dose (over saline)" (Fischman & Foltin, 1992, p. 179). In terms of their subjective emotional feelings, those addicts did not like their diluted cocaine infusion, did not want it, and did not even believe that cocaine was available. But, there was another sense, manifest in their actions, in which they simultaneously did "want," and perhaps "liked," their watered-down drug reward. They worked for it and selectively strove to gain it.

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What is not yet known is whether such unconscious emotions are limited to simple positive–negative affective reactions, as in the studies discussed, or whether they also occur as a distinct qualitative emotion such as unconscious fear, anger, disgust, joy, and so on? That still needs to be resolved. Also, it should be noted that to say unconscious emotional reactions are possible does not imply that conscious feelings are not functionally important on their own. Clearly, conscious feelings are important in determining well-being, in shaping the content of daily life, and as goals that motivate human behavior. Conscious emotions take on a special importance in clinical affective disorders, for which they may become the source of dysfunction. But, the lesson to draw from unconscious emotion is that objective emotional reactions are separable from subjective emotional feelings. Subjective feelings and objective emotional reactions ordinarily do occur together, but cases like these show they can equally sometimes exist separately from each other.

For these reasons, I believe it is important to recognize that objective emotional reactions can exist whether or not subjective emotional feelings accompany them. This is why example, my colleagues and I often use "liking" to refer to objective hedonic reactions that underlie conscious pleasure experiences when brought into conscious awareness, but remain detectable in behavior or in brain responses even when they are unconscious. Similarly, we use "wanting," in quotation marks, to describe the objective motivational process of incentive salience, which has a number of signature features in behavior and in brain mesolimbic reactions that can be measured, whether or not a subjective feeling of wanting also occurs at the same moment. This distinction helps to keep track of objective emotional reactions from the ordinary sense of liking or wanting as conscious feelings. To describe emotional reactions as objective does not deny that a subjective feeling of liking or wanting usually also occurs. It simply recognizes that "liking" can occur as an objective hedonic reaction, and "wanting" can occur as an objective motivational reaction, whether or not the conscious feeling of pleasure or desire is also present.

An Alternative View: Only Conscious Feelings Are Emotional?

The question of subjective feelings versus objective emotional reactions takes on special importance when studying animals, human infants, or brain-damaged human adults who are unable to give subjective ratings of their emotional feelings. Do those studies actually study emotion when subjective ratings are unavailable? LeDoux suggested that the answer is no (LeDoux, 2014). In discussing his laboratory's distinguished studies on the role of brain amygdala circuitry in fear learning (LeDoux, 2014), LeDoux wrote, "I and others have called the brain system that detects and responds to threat the

fear system. This was a mistake that has led to much confusion" (p. 2871). He suggested his previous mistake was to infer fear" when his rats froze to the sound of a tone that predicted an imminent foot shock. Rather than calling this reaction "fear," he suggested that such a reaction be called "threat detection" when it occurs in the absence of a subjective report (i.e., in animals, human babies, or patients with brain damage). For example, cringing from the sound of impending shock or at the sight of a syringe needle are threat detection responses but do not necessarily qualify as fear. So, rather than regarding the amygdala as part of a fear circuit, LeDoux suggested instead that it is part of "a neural circuit that underlies the expression of defense response elicited by conditioned and unconditioned (presumably innate) threats" can be called a "defensive survival circuit."

LeDoux's proposal to talk about threat-rather-than-fear systems in animals or human infants can be recognized as similar to early behaviorist proscriptions against using emotional terms (e.g., fear, liking, wanting, etc.) and behaviorist exhortations to speak instead only of objective observations (e.g., threat stimuli, defense responses, neural circuits). It is just that for LeDoux the restriction was applied specifically to infants, animals, or patients with brain damage because they were unable to speak of their emotional feelings. Similarly, to speak of a "defensive survival circuit" echoes reductionist ambitions of the last century to replace psychological terms with neural terms presuming a one-to-one correspondence between them or that the essence of function could be mapped to a specific anatomical circuit, such as the amygdala.

To qualify as having an actual emotion LeDoux suggested requires an emotional verbal report, such as saying we enjoy the sunset. Of course, strictly speaking, even verbal reports may not quite be proof of feelings because what people say often decouples from what they feel even when trying to make an honest report, and underlying processes may not always be accessible to introspection (Nisbett & Wilson, 1978; Schooler & Mauss, 2010; Wilson & Schooler, 1991).

Conversely, hearing a verbal report is no proof that a corresponding feeling exists. For example, after typing the preceding sentence, I just now asked my iPhone's assistant, Siri, "Do you like sunsets?" Siri replied, "I'd rather not say." Yet, I do not take this response as meaning that my phone (or its software resources in the cloud) feels any true emotional reluctance to tell me whether it enjoys sunsets. If it answered yes, I would not have believed a true emotional enjoyment actually was behind the assertion. Its answers arise from a programmed script for dealing with verbal inquiries. Misleading answers can also come from other people. Even if you hear me talk about my feelings, saying, for example, "I really love this fantastic sunset," you do not know if I really have those emotions. I might be lying—or be a talking zombie that does not actually have feelings—or saying the phrase while looking at a blank wall in my office. I also never have proof of your feelings. The point is, demanding "proof" is too stringent a standard, and verbal reports are simply not the stuff that proof is made of. If we adhered strictly to need for proof, we would all be in a solipsist's quandary, each being sure only of our own individual subjective feelings but in doubt about everyone else's. So, in fact, none of us adheres to such a demanding standard for proof. Even LeDoux, during every human interaction, in accepting assertions about enjoyed sunsets, temporarily dropped any professional insistence on having proof. What is important is that we acknowledge this inconsistency and not pretend that we have a consistent standard for proof. Incremental evidence, not proof, is the currency of most daily life and even of most science. So, incremental evidence is what we must look for regarding emotional processes, recognizing that evidence by its nature is always partial and tentative and requires close examination.

There actually is not any other choice. Even if we wished to stick with "defense circuits" rather than "fear circuits" in brain studies of emotion, there are serious problems inherent in that approach that will render it unsatisfactory. Neither external threats nor defensive brain systems will suffice as alternatives to emotional concepts for explaining how brains generate emotion-related behavior First, consider external threats as trigger stimuli in the world; while an auditory tone that predicts foot shock may reliably trigger defensive reactions in rats, we cannot use those threat stimuli in the world as a proxy for the reaction. First, the auditory tone is not actually a threat; it is merely an activation of a small loudspeaker. The threat comes from an experimenter programming the equipment to deliver the foot shock, but the tone's predictive relation is entirely arbitrary and not inherent or causal to the threat.

Conversely, very serious threats can also occur without eliciting any fear reaction. For example, people have no fear response to inhaling carbon monoxide gas from a faulty furnace or when stuck in a car tunnel or closed garage; they cannot smell

or feel it and may die of poisoning with smiles on their face. Threat does not necessarily imply fear or defensive response. And, oppositely, intense fears such as in free-floating anxiety, panic attacks, and post-traumatic stress disorder can occur without any identifiable corresponding threat stimulus in the outside world. Defensive fear responses do not necessarily imply a threat. The connection between threat stimuli and defensive fear is evolved but merely correlative and is often broken. When we travel, we may worry about being blown up by terrorists, while ignoring the statistically much greater threat of dying in a car accident. What this means is that we cannot outsource fear to external threats even if we call the emotional response a defensive reaction. We would be relying on a mere correlation that is not a trustworthy marker for the emotional reaction we want to understand.

Next, although one might try to alternatively reduce fear to an underlying defensive neural system or node, such as the amygdala, this approach also meets difficulties. The amygdala is indeed famous for mediating learned fear reactions, such as when a rat freezes to a sound that predicts a foot shock, and its fame is largely to LeDoux's own outstanding contributions. However, the amygdala also mediates many reward reactions (Janak & Tye, 2015; O'Doherty, 2012; M. J. Robinson, Warlow, & Berridge, 2014). For example, my laboratory has shown that optogenetic stimulation of neurons in the central nucleus of the amygdala, which has often been called the output circuit for fear, causes appetitive motivation in the form of addiction-like intense and narrowly focused desires for an associated reward (M. J. Robinson et al., 2014). Optogenetic stimulation uses a laser light to excite amygdala neurons after a virus has been previously microinjected to transfer a gene that codes a photoreceptor molecule. Subsequently, the infected neurons make the photoreceptor themselves, and a laser shone into the brain via a permanently implanted optic microfiber causes those neurons to fire. We find that pairing laser stimulation of central nucleus neurons in rats with earning a particular sugary reward causes those rats to develop intense "wanting" in an addiction-like fashion (M. J. Robinson et al., 2014). Similarly, pairing the amygdala stimulation with earning an intravenous cocaine reward causes the rats to develop intense "wanting" for that cocaine option, while ignoring an equally good cocaine option that was not associated with amygdala stimulation (Warlow, Robinson, & Berridge, 2017). So, this "defensive output circuit" can actually function equally well as an opposite "appetitive output circuit." At best, a "dedicated fear circuit" theorist could hope that some tiny population of neurons in the central amygdala is mysteriously invulnerable to our virus and laser stimulation and dedicated to fear. But, if so, that neural population is vanishingly small, composed of only a relatively few neurons. And, there is no clear evidence yet that convinces me that such a subset of dedicated defensive neurons exists at all.

Similarly regarding reward, the nucleus accumbens, with its dopamine input projections, is famously identified with appetitive functions of "wanting," "liking," and learning about rewards. Yet, as described previously, my laboratory and others have found that nucleus accumbens neurons and their dopamine inputs can also reversibly generate actively coping "fear" reactions, in which perceived threatening stimuli are defended against with antipredator reactions (e.g., rodents tossing sand toward rattlesnakes), frantic escape attempts, or reactive aggression (e.g., biting a human hand that attempts to pick up the rat).

Finally, even categories like "defensive" reactions still need additional psychological labels. In our laboratory, if we change the drug microinjection in the posterior nucleus accumbens from a glutamate antagonist to a gamma-aminobutyric acid (GABA) agonist (which more strongly hyperpolarizes the neurons), it adds excessive "disgust" to the excessive "fear," so that if the rat now encounters a sweet sucrose taste, it emits "disgust" gapes as though the taste were instead bitter (Faure, Richard, & Berridge, 2010; Reynolds & Berridge, 2002). While both "disgust" and "fear" are defensive, it seems important to recognize the difference. Calling such reactions (or nucleus accumbens or amygdala) "defensive" is not enough to capture their psychological natures. I think that difference will always require emotion-related concepts such as "disgust" and "fear," even if sometimes only in an objective sense.

So, just like for the amygdala, nucleus accumbens circuitry also has multiple modes of motivation function that generate quite different emotional reactions. There may be few, if any, circuits in the brain permanently dedicated to generating only one emotion or even one affective valence (Lindquist, Wager, Bliss-Moreau, Kober, & Barrett, 2012).

Reconciling Views of Emotional Reactions

Fortunately, I think a point of agreement can be found between my view that "fear," "wanting," "liking" and other emotional/motivational processes can be

studied as objective reactions as well as subjective feelings and LeDoux's recent position that emotion is purely subjective and that objective reactions are therefore nonemotional. Those objective reactions are key, with their signature identifying features, and recognizing their nature is important. We both agree that objective defensive/threat/"fear" reactions can occur either with or without subjective feelings of fear, and that it is important to clearly speak in terms that differentiate between objective versus subjective emotional reactions. Further, we both agree that relating objective reactions to underlying neural circuitry can reveal important insights that often apply to emotional feelings.

By Their Fruits You Shall Know Them

In the end, I suggest the best way to judge the validity of each of these scientific approaches is by their products: What new insights or conclusions does each provide? For example, fruits of the "wanting"/"liking" distinction can be found in its clinical applications, from addiction to aspects of schizophrenia and depression and to Parkinson's disease. Regarding addiction, the incentive sensitization addiction hypothesis originally suggested drug addiction results from growth of "wanting" or incentive salience, but not "liking" for drugs. That idea was based originally on rat experiments, but its implications directly applied to people who are addicts and even often to their subjective feelings. So far, that application has survived two decades (Berridge & Robinson, 2016). It has now also extended to include a number of other behavioral addictions, ranging from compulsive gambling, to sex, and so on, all based on the same animal origin notion that sensitized dopamine-related mesolimbic brain reactions mediate intense "wanting" for addictive targets (Callesen, Scheel-Kruger, Kringelbach, & Moller, 2013; Friedman & Chang, 2013; Ondo & Lai, 2008; O'Sullivan, Evans, & Lees, 2009; Politis et al., 2013). It also has been applied to some human eating disorders, such as extreme binge eating with obesity (Davis et al., 2009).

New clinical evidence has even come from recent findings that some patients with Parkinson disease develop what is called dopamine dysregulation syndrome (DDS) when treated with newer dopamine-stimulating medications that directly activate their brain dopamine receptors (i.e., direct agonist medications). These patients with DDS can develop intense addiction-like compulsive motivations to pursue gambling, shopping, sex, Internet use, excessive hobbies, or similar activities, and some patients with DDS also addictively overconsume their medications (Callesen et al., 2013; Friedman & Chang, 2013; Ondo & Lai, 2008; O'Sullivan et al., 2009; Politis et al., 2013). The addictive pursuits are not typically experienced as very pleasant, and virtually all addictive symptoms usually fade if dopamine-stimulating medications are stopped (Pettorruso et al., 2016). This pattern is further support for the idea that overstimulation of mesolimbic dopamine is the likely culprit behind intense "wanting" in a host of addictive compulsions. Thus, one fruit has been a comprehensive account of a number of human addictions.

Another clinical application of "wanting" versus "liking" has been the reinterpretation of anhedonia symptoms of schizophrenia, depression, and untreated Parkinson disease. Originally conceived as a lack of pleasure or liking, it has become clear that many people retain many pleasures intact, but no longer value them (Barch, Treadway, & Schoen, 2014; Hardman, Herbert, Brunstrom, Munafo, & Rogers, 2012; Luking, Pagliaccio, Luby, & Barch, 2016; Sienkiewicz-Jarosz et al., 2013; Treadway & Zald, 2013). Consequently, the "anhedonia" for many patients has been suggested to be better seen as a lack of incentive motivation or wanting (i.e., "avolition") rather than a loss of pleasure (Barch et al., 2014; Treadway & Zald, 2013). Conversely, enhanced "wanting" without necessarily "liking" has been suggested to apply to hypomania and poor impulse control, in which extroversion traits may become excessively exaggerated (Kirkland, Gruber, & Cunningham, 2015). So, another fruit has been the insight that human affective disorders can involve dissociation of "wanting" from "liking," and that sometimes alteration of "wanting" has masqueraded as alteration of "liking."

Finally, the conclusion that active "fear salience" generated by mesolimbic dopamine-related mechanisms makes percepts become attention riveting, similar to incentive salience but with a negative threatening valence rather than positive attraction, has been suggested to contribute to paranoid symptoms in schizophrenia (Barch et al., 2014; Heinz & Schlagenhauf, 2010; Howes & Kapur, 2009) and in addictive drug users who experience psychostimulant-induced psychosis (Cicero, Docherty, Becker, Martin, & Kerns, 2015). All of these concepts came originally from studies of objective "liking" and "wanting" reactions in animals but have turned out also to apply to human emotions and clinical disorders—extending to their disordered subjective feelings as well as to their objective behavioral

symptoms. In this way, studies of brain emotional systems, even in animals, can sometimes produce psychological surprises, with useful implications for normal emotional function in people and for their disorders.

These conclusions were possible because incentive salience has signature features that allowed us to distinguish "wanting" from hedonic "liking" in studying objective emotional reactions of animals. But, while objective, those reactions remain as psychological as their subjective cousins. That relationship allowed our conclusions to carry testable implications for corresponding human psychological processes, including subjective feelings. In the future, similar progress can be made by identifying signature features of objective "fear," "disgust," and other emotional reactions and examining their relations to underlying brain mechanisms. From these efforts, new testable predictions about human emotion are likely to emerge and may well bear successful fruits.

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Author Queries

AQ 1: Use square brackets if the parenthetical material is not in the original.