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Pleasure, Unfelt Affect, and Irrational Desire

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ABSTRACT

An example of unfelt affect is described in which subliminal facial expressions cause unfelt "liking," which influences people's reactions and beverage consumption without causing conscious emotion. Subcortical brain modules for core processes of nonconscious "liking" and "wanting" have been revealed by affective neuroscience studies, based on behavioral affective reactions that occur even in creatures that cannot speak, such as infants or animals. Core "liking" and "wanting" may normally contribute to conscious pleasure and desire. But these core psychological processes are themselves intrinsically inaccessible and provide a basis for unconscious affective reactions. The process of "wanting" also provides insight into cases of truly irrational desire, where one wants what is neither liked nor expected to be liked.

Sweetness tastes nice. The pleasantness of a sweet taste is a gloss on the mere sensation, added by our brains to the sensory quality of sweetness (Frijda, 2001).[1] Sweets need not be nice – there are nasty sweet tastes in this world too (as when aversions are acquired for particular sweet flavors). But humans are evolutionarily and neurobiologically predisposed to find sweet sensation pleasant. This chapter is devoted to the gloss of niceness, especially the niceness of sensory pleasures (for discussion of nonsensory pleasures, see Frijda, 2001). What is the niceness gloss? How is it added to

[1] The idea of pleasure as a "niceness gloss," added to sweetness above and beyond mere sensation, was introduced at the Amsterdam conference by Nico Frijda (in a public discussion with Antonio Damasio). Without fully addressing the interesting point of their contention (the degree to which the affect of sensations is intrinsic to sensory representations or separate from them), I have adopted it as a useful device to link the ideas in this chapter.

I thank Piotr Winkielman and Julie Wilbarger for allowing me to include our unpublished data. I also thank Nico Frijda, Sheila Reynolds, and Piotr Winkielman for helpful discussion of earlier versions of the manuscript.

a sweet sensation by brain systems? And how does it motivate real action – in rational and even irrational ways?

First, I want to examine the possibility that the essence of niceness may be surprisingly nonconscious. Evidence suggests a form of nonconscious "liking" can occur in ordinary human beings without their subjective awareness of it. Nonconscious "liking" may be the core of the niceness gloss that we ordinarily experience as conscious pleasure. But even nonconscious "liking" alone, without subjective feeling, is sufficient to influence behavioral reactions to affect-laden events.

Second, I will briefly address how core "liking" might be organized in the brain. Specifically, opioid synapses in the subcortical forebrain's nucleus accumbens can cause a niceness gloss for sweet tastes, activating a brain circuit for "liking" sweet pleasures.

Third, I will examine a further core incentive process, namely "wanting," which can rather directly translate the pleasure gloss into action. This psychological process and its dopamine brain substrate is only one of several routes to goal-directed action and is not the most complex or interesting psychologically. But under some circumstances "wanting" might cause irrational desires, in a strong sense of irrationality. I define irrational desire here as desire for something that we neither like nor expect to like. In such cases, this "wanting" process becomes interesting indeed.

UNCONSCIOUS EMOTIONAL REACTIONS?

People often may have emotional reactions without knowing why they have them, but they have generally been supposed to know that they have an emotional reaction (Ellsworth & Scherer, 2003; Frijda, 1999). After all, emotions are feelings, and feelings must be felt. Still, let us ask, can there be unfelt affective reactions? And if affect means feeling, this poses a paradox: can there be an unfelt "feeling"?

Two decades of studies have shown convincingly that people can be caused to have emotional reactions by subliminal events they do not consciously perceive (Öhman, Flykt, & Lundqvist, 2000; Zajonc, 1980; Zajonc, 2000). Yet even in such instances of unconscious emotion, it is only the cause and assignment of emotion that proceeds unconsciously. The emotion itself is generally interpreted as being a conscious feeling, detected by asking subjects how they feel.

But a stronger sense of unconscious emotion can be envisioned too. Truly unconscious emotion would mean the emotion itself remains unfelt yet is still evidenced as a valenced reaction in behavior or physiology. This sense of emotion as an unfelt feeling or nonconscious affect seems to turn the ordinary definition of emotion upside down, dropping its most obvious feature, namely, that emotions feel a certain way. But counterintuitive as it is, there are principled reasons from both psychology and affective

neuroscience why a nonconscious emotion might be possible (Berridge & Winkielman, 2003; Frijda, 2001; Kihlstrom, Mulvaney, Tobias, & Tobis, 2000; LeDoux, 2000; Winkielman, Zajonc, & Schwarz, 1997; Zajonc, 2000).

The question can be put as an empirical one. Do subliminal causes of affective reactions always produce a conscious emotion (at least when they succeed in influencing reactions)? Or can people actually remain unconscious of their own emotional reaction that is objectively evident in their later behavior (in addition to being unaware of its subliminal cause)? Piotr Winkielman, together with his student Julie Wilbarger, and I recently conducted a collaborative project that demonstrated unfelt affect (Winkielman, Berridge, & Wilbarger, 2000). That study found nonconscious "liking" – a nonconscious pleasure gloss not sensed by the person at the moment it was caused, but which influenced the response toward an affectively laden stimulus moments later.

Winkielman et al. assessed conscious emotional reactions by asking subjects to rate online their subjective mood immediately after subliminal exposure to emotional facial expression. Subliminal stimuli were happy, neutral, or angry facial expressions (only one-sixtieth of a second duration), followed immediately by a second "masking" photograph of a face with a neutral expression shown long enough to be consciously seen (nearly 1/2 sec, and embedded in a distraction task). Participants were aware only of the second neutral face, and not of the preceding emotional expression. All participants denied later having seen any emotional expressions and were unable to recognize them. Participants also rated their own emotional feelings immediately after subliminal exposure, either on a simple hedonic mood scale, or on a more complex twenty-item mood scale (contentment, irritability, etc.). Finally, their evaluation of subjective emotion was counterbalanced with an opportunity to evaluate a "new fruit beverage" (actually lime Kool-Aid).

For example, participants were given a pitcher of beverage and were asked to pour and drink as much as they wished (the amounts were covertly measured) (Winkielman et al., 2000). In another experiment, participants were given a single sip of the beverage after being shown subliminal faces and asked to rate how much they liked the drink, how much they wanted to consume, and how much they would be willing to pay if it were sold in stores.

Unconscious "liking" was produced best in participants who were thirsty (Winkielman et al., 2000). No change in their subjective emotion was produced by subliminal exposure to happy/angry faces. Yet subliminal exposure to happy facial expressions caused thirsty participants to pour themselves about 50 percent more of the fruit-flavored drink than if they had seen only neutral facial expressions (Winkielman et al., 2000). Conversely, they poured 50 percent less than neutral after seeing angry faces. These participants also consumed or swallowed about 50 percent

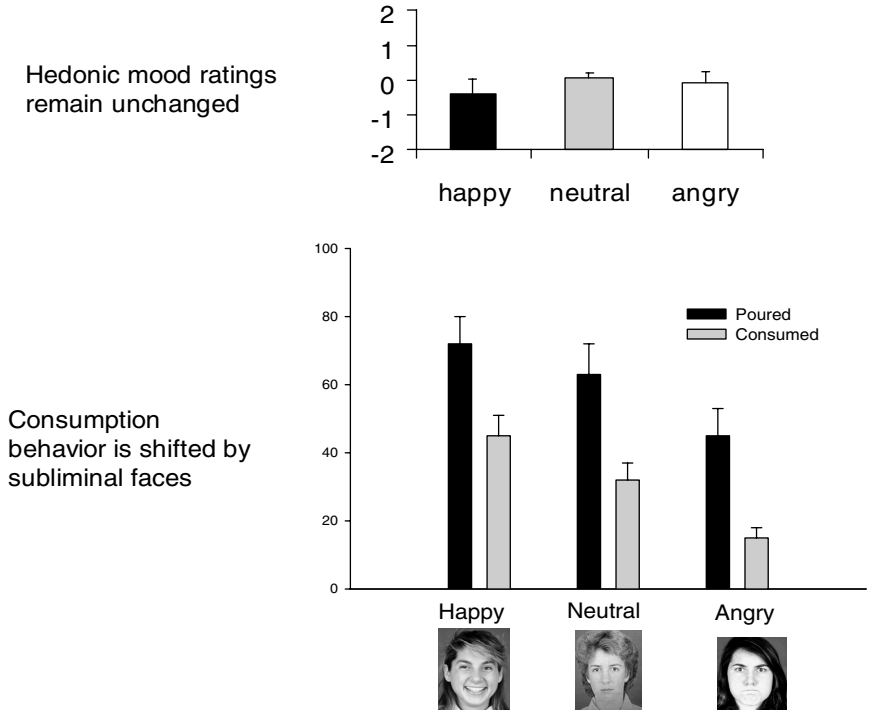

FIGURE 15.1. Unfelt affect controls consumption behavior. *Top:* Ratings of subjective mood were not changed after same subliminal stimuli ("How do you feel right now at this moment?" 10-point hedonic scale). *Bottom:* Pouring and drinking behavior by thirsty men and women was changed after subliminal exposure to happy facial expressions, neutral facial expressions, or angry facial expressions (amount poured of a fruit-flavored drink and amount actually consumed). From Winkielman et al., submitted.

more of what they poured after seeing happy expressions than after neutral expressions (and drank even less after subliminal angry expressions). Their consumption behavior was increased by happy expressions (and decreased by angry expressions) even though they had no conscious awareness of any change in their own mood at the moment subliminal faces are presented (Figure 15.1).

Similarly, the subliminal stimuli altered ratings of liking and wanting given to a sip of the drink and of its monetary value (Winkielman et al., 2000). For example, thirsty participants who were exposed to subliminal happy expressions later gave higher pleasantness ratings and higher answers to the question "How much would you pay for this drink in a store?" They were willing to pay more than twice as much (over 40 cents per can, U.S. currency) after seeing subliminal happy expressions compared to after

seeing subliminal angry expressions (less than 20 cents per can). Again, no conscious emotions or mood changes were reported by these participants after subliminal presentations (Winkielman et al., 2000).

Clearly, happy subliminal faces did not make drinkers feel better in general, since they reported no elevation in subjective emotion at all. Nor did subliminal angry faces make them feel worse. Instead the subliminal stimuli produced no conscious emotion at all when presented. Only later, when an affectively laden drink was encountered, did the implicit affective reaction surface into explicit behavior and ratings.

Thus in normal adults under some conditions, unfelt "liking" reactions can influence a person's consumption behavior later, without reportable subjective awareness of the affective reaction at the moment it was caused. Core "liking" in this sense is consistent with Frijda's notion of the pleasure gloss as "a mental process, itself as unconscious as any other mental process, that changes the tuning for inputs and behavioral inclinations" (Frijda, 2001, p. 77) and a process that requires further processing to become conscious. Although it is possible to try to explain subliminal "liking" effects in terms of unconscious cognition rather than unconscious emotion, Winkielman, Wilbarger, and I believe that such cognitive reinterpretations will not succeed in this case (because cognitive manipulations of attention did not alter the effects, whereas biological thirst state played a determining role; for discussion see Berridge & Winkielman, 2003; Winkielman et al., 2000). If my colleagues and I are correct, this may be an example of truly unconscious emotion.

AFFECTIVE NEUROSCIENCE OF CORE "LIKING" FOR PLEASANT TASTES

What could happen in the brain to generate unfelt "liking"? What brain systems might mediate subliminally caused changes in affective reactions to a drink? We can only speculate, but there are some clues to guide us from the affective neuroscience of taste pleasure.

First it is helpful to note a useful behavioral measure of positive affect that has been employed in some affective neuroscience studies – namely, affective facial expressions to the sensory pleasure of taste, which our laboratory has used to examine brain mechanisms that underlie basic "liking" reactions.

A newborn human infant has two distinct patterns of affective facial reactions to tastes, positive versus negative (Steiner, 1973; Steiner, Glaser, Hawilo, & Berridge, 2001). Sweet elicits positive "liking" facial reactions from newborns (e.g., lip sucking, smiles), whereas bitter elicits negative "disliking" reactions (e.g., gapes, nose wrinkling; Figure 15.2).

Positive facial reactions to sweetness might plausibly be accompanied by conscious feelings of pleasure for normal human infants. But there are

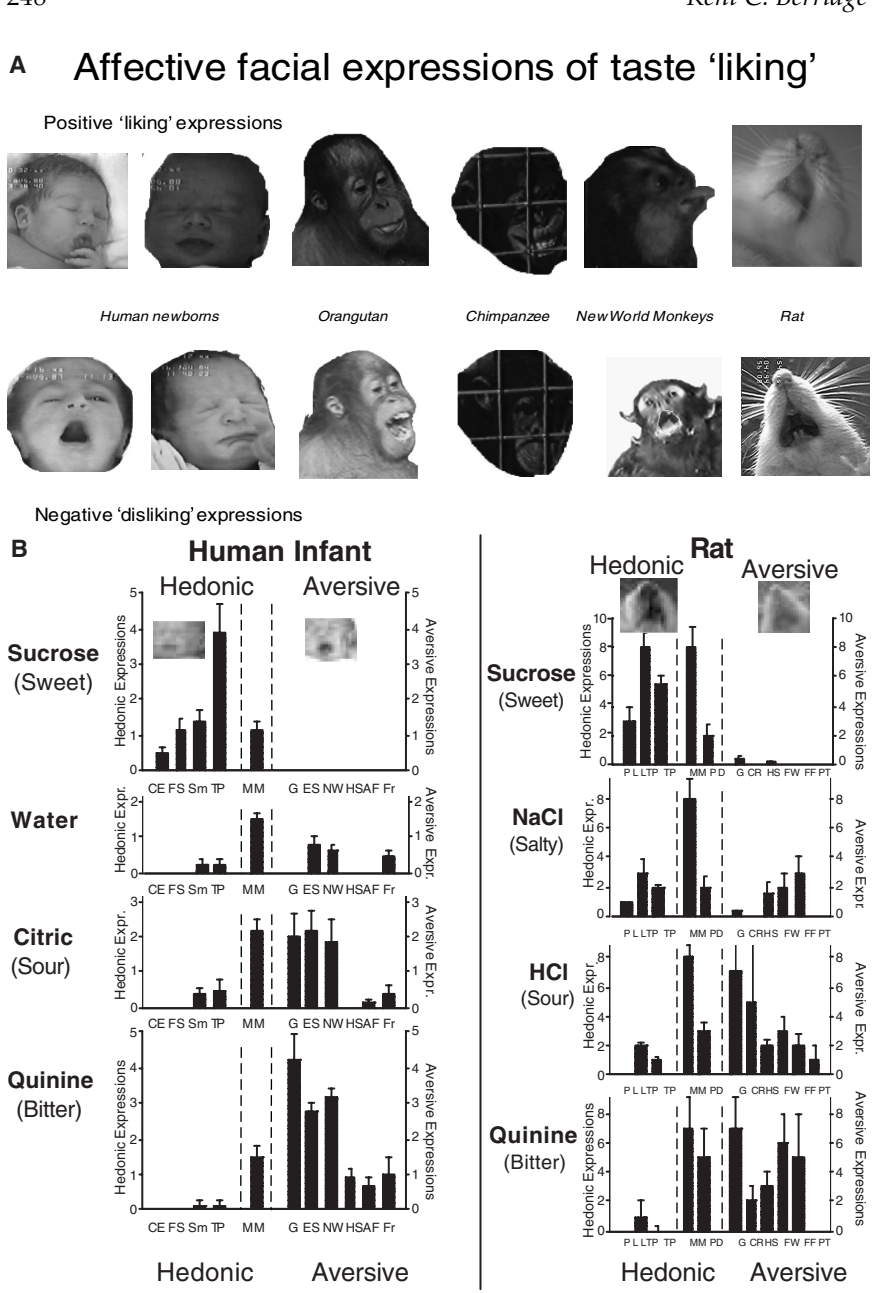

FIGURE 15.2. A. Affective reactions are elicited by sweet tastes from human infants, great apes, monkeys, and rats. B. Human infant affective reactions switch from positive to negative across sweet to bitter tastes (*left*). Affective reactions by rats show a similar gradual switch from positive to negative across tastes (*right*). Modified from Steiner et al., 2001, and Berridge 2000. (*See color insert*)

reasons to think that the facial reaction to pleasure reflects a core process of "liking" rather than the consciousness of the niceness gloss. One reason is that positive affective facial reactions also occur in infants whose consciousness status is more suspect, such as "anencephalic" infants (Steiner, 1973). Anencephalic infants have a brainstem but no cortex, amygdala, or classic limbic system, due to a birth defect that prevents prenatal development of their forebrain. Yet basic tastes elicit normal positive/negative affective reactions from them.

Animals, especially those that prefer sweets, also emit affective facial reactions to sweet versus bitter tastes that can be highly similar to those of human babies (Figure 15.3). Positive facial expressions to sweetness are emitted by chimpanzees, orangutans, and gorillas, various monkeys, and even rats (Berridge, 2000; Steiner et al., 2001). The pattern of positive facial expression becomes increasingly less similar to humans as the taxonomic distance increases between a species and us. But all of these species share some reaction components that are homologous to ours. Those affective reactions share a common evolutionary ancestry and are likely to have similar neural mechanisms (Steiner et al., 2001). This means in practice that one can use mere rats in affective neuroscience studies to examine the brain mechanisms of positive affective reaction to sweetness (Berridge, 2000).

SUBCORTICAL FOREBRAIN SITE CAUSES POSITIVE AFFECTIVE CORE PROCESS: NUCLEUS ACCUMBENS SHELL

The nucleus accumbens is perhaps the most notable brain system of "liking" identified so far (especially its portion named shell; Figure 15.3), because it is a forebrain structure capable of applying a pleasure gloss. It lies at the front base of the brain, just below the prefrontal cortex. Activation of certain neural circuits in the nucleus accumbens causes heightened "liking" for a pleasant taste, as revealed by a recent affective neuroscience experiment conducted by Susana Peciña in our laboratory (Peciña & Berridge, 2000). Nucleus accumbens circuits are one of the few brain systems able to cause an increase in the niceness gloss.

Specifically, selective activation of opioid neurotransmitter receptors on neurons inside the nucleus accumbens of rats causes sweet tastes to elicit extra "liking" reactions. The selective activation was caused by microinjections of morphine (a drug that activates opioid receptors) directly into the nucleus accumbens, made painlessly because the brain cannula had been implanted a week earlier (under anesthesia). Then a few minutes after the morphine microinjection, a bittersweet taste was infused into the rat's mouth while its behavioral affective reactions were videorecorded (Peciña & Berridge, 2000). The rats responded with a distinct shift toward positive affective reactions after the morphine microinjection, indicating they "liked" the taste more (Figure 15.3). Interestingly, morphine

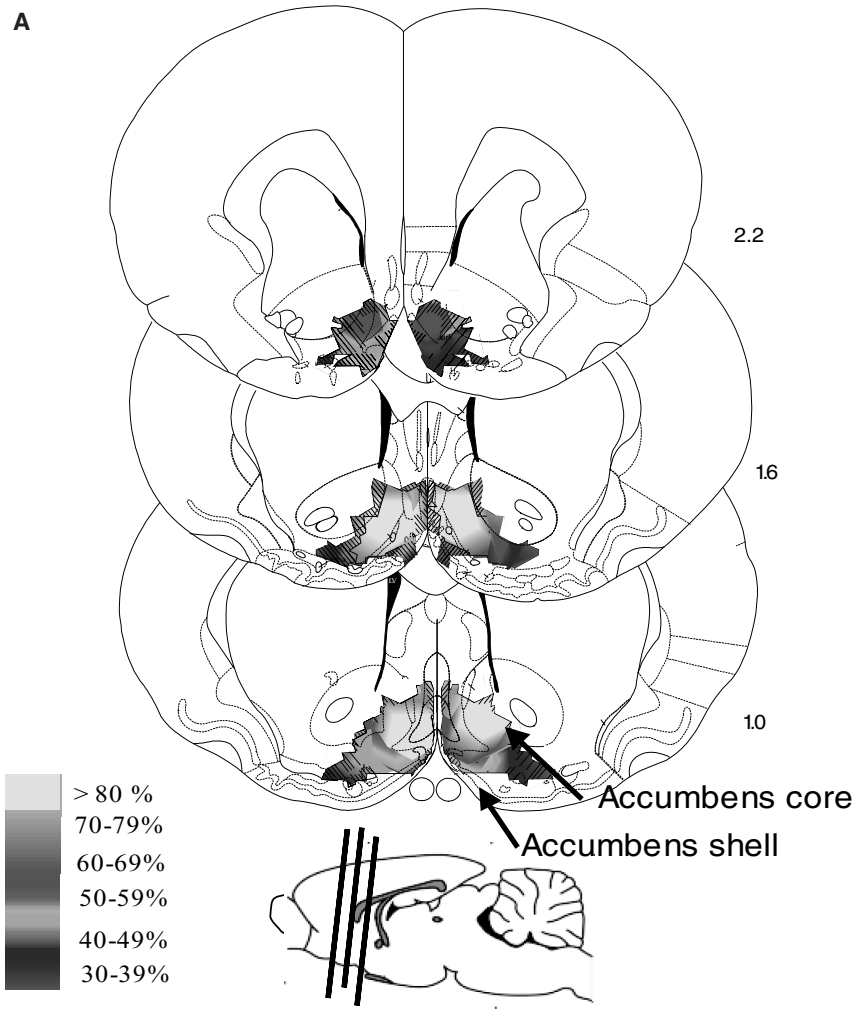

FIGURE 15.3. A. "Liking" and "wanting" site in nucleus accumbens shell where opioid activation causes increased niceness gloss for sweetness (Peciña & Berridge, 2000). Percentages refer to percent increase in appetitive behavior caused by morphine microinjection (100% = twice baseline amount). Coronal brain slices (face on view) are numbered conventionally; their position is shown in profile view below. B. Brain structures of "liking" discussed here (gray), and the dopamine "wanting" system (black). (*See color insert*)

microinjections also caused the rats to eat more of a tasty food than they ordinarily would. This suggests that the accumbens shell activation may increase "wanting" for food, as a consequence of enhancing "liking" for it (Peciña & Berridge, 2000).

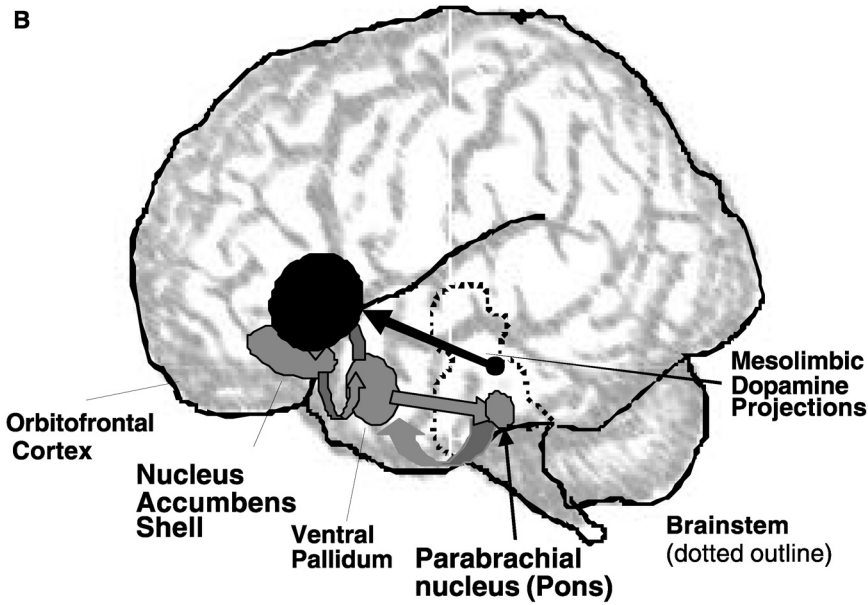

FIGURE 15.3. *(continued).*

The nucleus accumbens is embedded in a larger brain circuit for "liking," which connects to other forebrain structures such as the ventral pallidum, and even to brainstem structures, such as the parabrachial nucleus (Figure 15.3, Berridge, 2003). The ventral pallidum and parabrachial nucleus have been posited by several affective neuroscientists to play important roles in emotion and even in generating a sense of self (Damasio, 1999; Panksepp, 1998). Affective neuroscience studies in our laboratory have found these brain structures share the special ability to cause changes in the niceness gloss, as reflected by "liking" facial reactions to sweet tastes (Berridge, in press).

How do subcortical mechanisms of "liking" reactions to sweet pleasure bear on the subliminal affective reactions to drinks in adult humans found by Winkielman and colleagues? One possibility is that opioid activation patterns in the human nucleus accumbens might be altered by subliminal facial expressions, which activate related brain structures (Morris, Öhman, & Dolan, 1999; Whalen et al., 1998). Altered neuronal activity in the nucleus accumbens (perhaps causing nonconscious "liking") could then change the human affective reaction to a drink, just as morphine microinjection into a rat's accumbens enhances its affective reaction to sweetness and leads to behavioral reaction of greater "liking."

Further, to the degree that conscious feelings of pleasure might be influenced in turn by opioid activation in accumbens, the subjective niceness

gloss could be caused by accumbens-to-cortex signals that are relayed to cortical regions in just a couple of synapses, via the ventral pallidum and thalamus (Zahm, 2000). Opioid activation in the nucleus accumbens is widely thought to partly mediate the intense pleasure of stimuli such as heroin for human drug users (Koob & Le Moal, 2001; Wise, 1998), and similar opioid activation mediates normal human subjective feelings of pleasure caused by tasty food (Yeomans & Gray, 1997). Finally, descending projections from frontal cortex to subcortical "liking" structures might permit cognitive appraisals or voluntary intentions to modulate basic emotional reactions (Davidson, Jackson, & Kalin, 2000).

SURPRISING FALSE CANDIDATE FOR "LIKING": MESOLIMBIC DOPAMINE SYSTEM

As our laboratory has examined various brain mechanisms for the sweet pleasure gloss, one of the findings that surprised us most was failure to modulate "liking" whenever we manipulated the mesolimbic dopamine system. The system of dopamine neurons that stretch from the midbrain to the nucleus accumbens is probably the brain's most famous reward system and is often called the pleasure system. But that may be a case of mistaken identity.

There are several reasons why dopamine neurons have been thought to mediate pleasure. Dopamine neurons in rats are turned on by naturally pleasurable events, such as eating a delicious new food or encountering a sex partner (Ahn & Phillips, 1999; Fiorino, Coury, & Phillips, 1997; Schultz, 1998). Dopamine is also turned on by drugs that many people enjoy, such as cocaine, amphetamine, heroin, or ecstasy (Wise, 1998). Further, if dopamine is blocked by other drugs, all these rewards seem to lose certain rewarding properties. At least, animals no longer seem to "want" rewards when their dopamine systems are suppressed, and it has seemed reasonable to many to infer that it is because they no longer "like" rewards (Gardner, 1997; Hoebel, Rada, Mark, & Pothos, 1999; Shizgal, 1999; Wise, 1985).

We therefore expected dopamine manipulations to powerfully alter affective facial expressions to sweet tastes when we began to manipulate the system and were at first surprised to find that they did not. But to make a long story short, dopamine systems do not seem to mediate the pleasure gloss, at least if "liking" is assessed by behavioral affective reactions to the hedonic impact of sweet tastes. We have turned dopamine systems on with amphetamine that causes dopamine release or with electrical stimulation of intervening brain pathways (e.g., Wyvell & Berridge, 2000) and turned them off with drugs that block dopamine receptors (e.g., Peciña, Berridge, & Parker, 1997). We have even selectively destroyed the dopamine system entirely with chemical neurotoxins that damage only dopamine-containing neurons (e.g., Berridge & Robinson, 1998). All

these manipulations powerfully altered the degree to which rats "want" tasty food, in the sense of their willingness to work for it or consume it. But they never shifted "liking" facial reactions to a sweet taste. The niceness gloss simply persisted unchanged (as does the ability to learn a new "liking"/"disliking" gloss; for review, see Berridge & Robinson, 1998).

Once over our initial surprise, we tried to suggest a psychological function that could masquerade as sensory pleasure in many studies yet still not be a true niceness gloss. Usually "liking" and "wanting" for pleasant incentives do go together, virtually as two sides of the same coin. But our dopamine studies indicate "wanting" may be separable in the brain from "liking." In particular, mesolimbic dopamine systems seem to mediate only "wanting." My colleagues and I have coined the phrase incentive salience for the form of "wanting" we think is mediated by brain dopamine systems. I should acknowledge our suggestion was strongly guided by earlier views of mesolimbic function (Fibiger & Phillips, 1986; Panksepp, 1986; Valenstein, 1976) and of the psychology of incentive motivation (Bindra, 1978; Toates, 1986).

"Wanting" is not "liking." It is not a sensory pleasure in any sense, conscious or nonconscious. It cannot increase positive facial reactions to sweet taste, or the hedonic impact of any sensory pleasure. Instead, incentive salience is essentially nonhedonic in nature, even though we believe it to serve as one component of the larger composite psychological mechanism of reward learning and incentive motivation (Berridge, 2001; Berridge & Robinson, 1998). Originally "wanting" might have evolved as an elementary form of goal directedness to pursue particular innate incentives even in advance of experience, later becoming harnessed to serve learned "likes." In any case, "wanting" remains a distinct process, and we believe that brain dopamine systems especially attribute incentive salience to reward representation whenever a cue for the reward is encountered. Incentive salience causes the cue and its reward to become momentarily more intensely attractive and sought.

The quotation marks around the term "wanting" serve as caveat to acknowledge that incentive salience means something different from the ordinary sense of the word *wanting*. For one thing, "wanting" in the incentive salience sense need not have a conscious goal or declarative target. Wanting in the ordinary sense, on the other hand, nearly always means a conscious desire for an explicitly expected outcome. In the ordinary sense, we consciously and rationally want those things we expect to like. Conscious wanting and core "wanting" differ psychologically and probably also in their brain substrates (Berridge, 2001; Dickinson, Smith, & Mirenowicz, 2000). Incentive salience or "wanting" depends on mesolimbic dopamine systems. Ordinary wanting for consciously explicit targets, by contrast, may depend more strongly on cortical structures, such as prefrontal

cortex and insular cortex (Balleine & Dickinson, 1998; Bechara, Damasio, & Damasio, 2000; Dickinson et al., 2000).

IRRATIONAL DESIRES?

If an outcome is liked, then by rational criteria it should also be wanted. The outcome should be wanted exactly to the degree that it is expected to be liked. "Expected to be liked" is the crucial phrase here. Human expectations of what will be liked can often be wrong – because of ignorance about the outcome, false predictions, or false memory of past outcomes (Elster, 1999; Gilbert & Wilson, 2000; Kahneman, 1999). Expectations are often wrong, but being wrong has nothing to do with what I mean by rational desire. Desires based on wrong expectations are misguided but not irrational. Desire remains rational as long as people still choose what they expect to like. A truly irrational choice would be to choose what you expect not to like.

Does truly irrational choice actually occur? Individuals often choose what they turn out not to like, sometimes based on irrational beliefs (see Elster, 1999). But do individuals ever irrationally choose what they expect not to like? My answer is yes: truly irrational choice can be produced by overactivation of the brain's "wanting" system of mesolimbic dopamine.

ANIMALS AND RATIONAL CHOICE?

It may seem incongruous to turn to animals to distinguish rational versus irrational choice, but the results of animal studies are useful in order to link to our consideration of brain dopamine and mesolimbic function. Let us first lay out what would be needed to make an animal model valid here. A demonstration of irrational pursuit would require: (1) that animals be capable of cognitive expectations of an outcome's hedonic value (wanting in the ordinary rational sense of expected liking); (2) that one be able to assess their rational cognitive wanting; and (3) that one be able to detect when their pursuit deviates from cognitive wanting.

These preconditions may have been met through the work of a leading psychologist of animal learning, Anthony Dickinson of Cambridge University in England, together principally with his former student Bernard Balleine. Dickinson and colleagues developed clever ways to ask a mere rat about its cognitive expectations of reward value, and to detect changes in those expectations (Balleine & Dickinson, 1998; Dickinson & Balleine, in press). They ask rats about their expectations of the value of a food or drink reward in part by testing their willingness to work for the rewards when they must be guided principally by those expectations alone. The rats are first trained to work for the real rewards, which come only every so often, so they learn to persist in working to earn reward.

Then the rats are tested for their willingness to work for these rewards later under so-called extinction conditions, when the rewards no longer come at all. Since there are no longer real rewards, the rats have only their expectations of reward to guide them. Naturally, without real rewards to sustain efforts, performance in the extinction test gradually falls. But since the rats originally learned that perseverance pays off, they persist for quite some time in working based largely on their ordinary wanting for reward.

The issues involved in using a Dickinson-style approach to tease apart ordinary cognitive wanting from cue-triggered "wanting" are rather complex (for more discussion, see Berridge, 2001; Dickinson & Balleine, in press). For our purpose here it is enough to say that these techniques of assessing animals' cognitive expectations of hedonic value can detect when cue-triggered "wanting" suddenly diverges from ordinary cognitive wanting. Combined with appropriate tweaks of the brain, they allow an affective neuroscience of irrational pursuit.

IRRATIONAL PURSUIT: VISCERAL MESOLIMBIC ACTIVATION OF "WANTING" FOR A CUED HYPERINCENTIVE

Truly irrational choice has been produced in our laboratory by the doctoral studies of Cindy Wyvell. She combined brain tweaks, in the form of amphetamine microinjections that activated mesolimbic dopamine systems, with Dickinson's techniques for assessing ordinary cognitive wanting versus cue-triggered "wanting" for a sugar pellet (Wyvell & Berridge, 2000). Dopamine activation caused a transient but intense form of irrational pursuit linked to incentive salience.

In this experiment, Wyvell trained rats first on several days to work for occasional sugar pellet rewards by pressing a lever. On different days, the rats learned a reward cue (CS+) for the sugar pellets, by being exposed to Pavlovian pairings in which sugar was preceded by a light or sound cue. In these cue-learning sessions, rats did not have to work for sugar rewards – instead rewards came freely after each cue. All rats were implanted with microinjection cannulae so that a droplet of amphetamine or of drug-free vehicle solution could be infused into their nucleus accumbens. Finally the rats were tested for work using the Dickinson extinction procedure after they had either received amphetamine microinjections or not. During this test, their performance could be guided only by their expectation of the cognitively wanted sugar, because they received no real sugar rewards. And while they pursued their expected reward, their reward cue (light or sound for 30 seconds) was occasionally presented to them over the course of the half-hour session.

In a related experiment, Wyvell tested the effect of amphetamine microinjections on the niceness gloss of real sugar, by measuring positive hedonic patterns of affective reactions of rats as they received an infusion

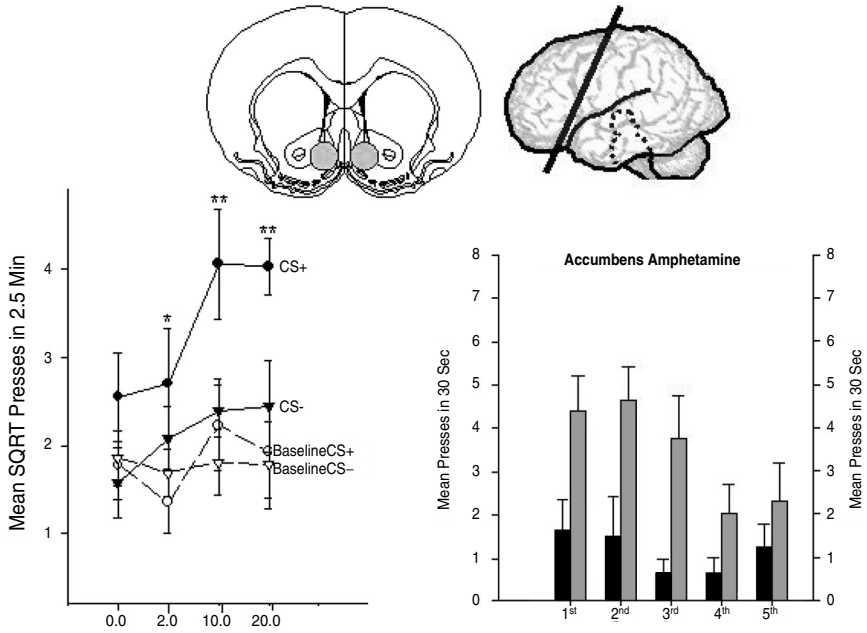

FIGURE 15.4. Irrational cue-triggered "wanting." Amphetamine microinjection in nucleus accumbens magnifies "wanting" for sugar reward – but only in presence of reward cue (CS+). Cognitive expectations and ordinary wanting are not altered (reflected in baseline lever pressing in absence of cue and during irrelevant cue, CS−) (*left*). Transient irrational "wanting" comes and goes with the cue (*right*). Black bars denote work in the absence of the reward cue; gray bars show elevation when cue was present. Modified from Wyvell and Berridge, 2000. (*See color insert*)

of sugar solution into their mouths (Wyvell & Berridge, 2000). In this experiment, amphetamine did not increase positive facial reactions elicited by the taste of real sugar, indicating once again that dopamine did not increase "liking" for the sugar reward.

However, amphetamine microinjection still enhanced "wanting" for sugar in the sense of cue-triggered pursuit of the reward in the first experiment (Figure 15.4). Remember that there are two types of wanting to be assessed here: (1) ordinary wanting, when the rat works guided primarily by its cognitive expectation that it will like the worked-for sugar reward, and (2) cue-triggered "wanting," or incentive salience attributed by mesolimbic systems to the representation of sugar reward that is activated by the cue. What Wyvell found was that activation of dopamine neurotransmission in the accumbens did not change ordinary wanting based on cognitive expectation of liking (measured by baseline performance on the lever). However, amphetamine microinjection dramatically increased cue-triggered "wanting" to more than 400 percent its baseline level.

Cue-triggered "hyper-wanting" is irrational and transient. It is repeatedly reversible, even over the short span of a 30-minute test session (Wyvell & Berridge, 2000). It is triggered by encounter with reward cues, and at that moment it exerts its irrational effect, disproportionate to the cognitively expected hedonic value of the reward. One moment the dopamine-activated brain of the rat simply "wants" sugar in the ordinary sense. The next moment, when the cue comes, the dopamine-activated brain both wants sugar and "wants" sugar to an exaggerated degree, according to the incentive salience hypothesis. A few moments later it has returned to its rational level of wanting appropriate to its expectation of reward. Moments later still, the cue is reencountered, and excessive and irrational "wanting" again takes control.

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

For the brain in a state of mesolimbic activation, the conditioned reward cue becomes a hyperincentive cue, able to trigger an irrational degree of pursuit for the sugar reward, at least for a while. The reward cue causes a momentary irrational desire. Individuals may then "want" what they do not cognitively want – and what they know they will not like (or at least, will not "like" proportionally to their excessive "want").

HUMAN IRRATIONAL CHOICE AND ADDICTION

People have brain dopamine systems too, which may spontaneously activate in many situations. And our brain dopamine systems can be hyperactivated by drugs such as amphetamine or cocaine. If a person's brain dopamine system were highly activated, and the person encountered a reward cue, it seems possible that the person might irrationally "want" the cued reward just like the rat – even if the person cognitively expected not to like it very much.

Human drug addiction may be a special illustration of dopamine irrational "wanting" (Robinson & Berridge, 1993, 2003). Addictive drugs not only activate brain dopamine systems when the drug is taken but may also sensitize them afterward (Robinson, Browman, Crombag, & Badiani, 1998). Neural sensitization means that the brain system becomes hyperreactive for a long time. The system is not constantly hyper active, but it reacts more strongly than normal if the drug is taken again – in a fashion

that is gated by associative context and cues that predict the drug. Neural sensitization occurs to different degrees in different individuals, depending on many factors ranging from genes to prior experiences, as well as on the drug itself, dose, and so on (Robinson & Berridge, 1993; Robinson et al., 1998).

My colleague Terry Robinson and I have suggested that if an addict's mesolimbic system becomes sensitized after taking drugs, that person may irrationally "want" to take drugs again even if they decide they don't "like" them, or like them less than they like the lifestyle they will lose by taking them. This incentive-sensitization theory of addiction thus accounts for why addictive relapse is so often precipitated by encounters with drug cues, which trigger excessive "wanting" for drugs (Robinson & Berridge, 1993, 2003). Cues could trigger irrational "wanting" in an addict whose brain was sensitized even long after withdrawal was over (because sensitization lasts longer), and regardless of expectations of "liking."

Actual evidence that sensitization does indeed cause irrational cue-triggered "wanting" was recently found by Cindy Wyvell in an affective neuroscience animal study similar to the one described above (Wyvell & Berridge, 2001). Rats that had been previously sensitized by amphetamine responded to a sugar cue with excessive "wanting" despite not having had any drug for ten days. Even though they were drug-free at the time of testing, sensitization caused excessively high cue-triggered "wanting" for their reward. For sensitized rats, irrational "wanting" for sugar came and went transiently with the cue, just as if they had received a brain microinjection of drug – but they hadn't. Their persisting pattern of cue-triggered irrationality seems consistent with the incentive-sensitization theory of human drug addiction (Robinson & Berridge, 2000).

IRRATIONALITY IN EVERYDAY LIFE?

Do ordinary people also show irrational cued "wanting" in less extreme situations? Does excessive activation of cue-triggered "wanting" promote irrationally intense pursuit of chocolate, sex, gambling, or other incentives? And can vivid cognitive fantasy ever substitute for cues, triggering in people spontaneous mesolimbic activation and irrational choice? These are intriguing questions about irrational "wanting" in ordinary human lives, but as yet there are no clear answers.

CONCLUSION

Ordinary people seem capable of unconscious emotions in a strong sense of unfelt affective reactions. In normal adults, core "liking" reactions caused by subliminal emotional stimuli may influence a person's "wanting" and

consumption of a beverage later, without the person being able to report his or her own affective reaction at the moment it was caused. To generate the core niceness gloss, there appears to be a subcortical network, including the nucleus accumbens, whose output is reflected in objective core "liking" reactions to sweet pleasures. To directly translate "liking" into action, there appears to be a "wanting" dopamine system, which can influence pursuit of rewards independent of cognitive expectations about them. In extreme cases, excessive "wanting" may produce strongly irrational choice, causing individuals to "want" what they do not cognitively want, and to choose what they do not expect to like.

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