Citation: Berridge, Kent C., "Pleasure, Pain, Desire and Dread: Hidden Core Processes of Emotion," in Well-Being: The Foundations of Hedonic Psychology, edited by Daniel Kahneman et al., New York: Russell Sage Foundation, 1999, pp. 525-557

27 Pleasure, Pain, Desire, and Dread: Hidden Core Processes of Emotion

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Elemental emotional states, such as simple pleasures, pains, desires, and fears, may seem irreducible, but they are not. Each contains dissociable psychological components, or core processes. This chapter explores dissociation of components within elemental emotion, the relations between components, and their embodiment in brain systems. The core processes of emotion and motivation are essentially unconscious and not directly represented in subjective emotional feelings. For example, the subjective experience of an emotion itself may be split apart into dissociable subjective components under a variety of circumstances. Core processes of emotion that underlie subjective experience can be further separated from subjective emotional feelings and occur without conscious awareness under limited conditions. For positive emotional states, core processes of "liking" and "wanting" are psychologically dissociable from each other. "Liking" corresponds to a basic sensory pleasure or hedonic activation. "Wanting" corresponds to a different core process, the attribution of incentive salience to stimuli or events. Core processes of "liking" and "wanting" are mediated by different neural systems in the brain. "Liking" may be activated without "wanting" through brain manipulations. Conversely, "wanting" can be activated without "liking." The phenomenon of "wanting" without "liking" has special relevance for understanding the causes of addiction. Negative emotions involving fear and pain also are dissociable into core processes. Some of the core processes of fear and anxiety may overlap with those of positive desires. In other words, positive and negative emotions may share psychological building blocks (such as incentive salience) even though the final emotions are experienced as opposite.

A COGENT CASE can be made that the quality of life depends partly on the fulfillment of cultural themes of life *meaning*, such as personal goals or relationships (Cantor, Acker, and Cook-Flannagan 1992; Cantor et al. 1991; Ellsworth 1994; Roney, Higgins, and Shah 1995). The quality of life is not reducible to its mere quantity of pleasures and pains but includes purposeful, aesthetic, and moral considerations, too. Life is still a series of pleasures and pains, however, some large and some small, and hedonic states determine at least one important aspect of life's quality. Any appraisal of the quality of life requires consideration of its affective tone. Since cultural appraisals of life meaning are relatively resistant to biopsychological analysis, I restrict myself here to the hedonic analysis of basic emotions.

I argue that even the simplest emotions, as we experience them, are not as elemental or irreducible as they seem. They contain multiple core processes. The nature of these core processes of emotion is not evident to conscious awareness and may not fit into traditional psychological categories. Evidence for these propositions is drawn both from the cognitive and social psychology of subjective emotion (Fischman and Foltin 1992; Hilgard 1986; Kahneman et al. 1993; Murphy and Zajonc 1993; Zajonc 1980) and from the affective neuroscience of emotional processes in the brain (Berridge 1996; Davidson and Sutton 1995; LeDoux 1996; Panksepp 1991).

Dissociation of Emotion into Unconscious Core Components

Our conscious experience of emotion might be likened to the glimmering surface of a pond. We see only the surface of our own emotion. Below the surface lie objects and creatures within the pond—core emotional processes and their antecedents. Cognitive mechanisms of conscious perception must translate an event into active declarative representations in order to be subjectively perceived, representing the event, as the pond's surface represents what is below. What we know of the pond is what we see from above. But the view from above is distorted by ripples in the surface—nuances of the translation process—and by reflected light from above—the modulating influ-

ence of cognitive expectation and appraisal. What is below the surface of our experience of the quality of life?

What defines an emotion, for many psychologists as well as for most other people, is its conscious *feeling*. It is almost impossible to conceive of emotion in any other way. Most would agree with the view expressed by the psychologist Phoebe Ellsworth: "I have always found the idea of unconscious emotions extremely difficult to think about . . . [as] in most definitions of emotion . . . a subjective experience of feeling is an essential component" (1995, 214).

Emotion is nearly unique among psychological categories to the degree that we judge subjective experience of feelings to be an essential component. Unconscious motivations, memories, and even perceptions may be granted, but an unconscious emotion is more difficult to imagine. For memory, we are not conscious of the vast array of declarative memories that may nonetheless be called up at another time, and we have procedural memories that are resistant to introspection. Unconscious perception is somewhat more problematic, and its existence was once a point of contention in psychology (Eriksen 1960). But there have been many demonstrations that events too brief to be consciously perceived may nonetheless have clear psychological consequences, including on subsequent emotional ratings (Kunst-Wilson and Zajonc 1980; Lazarus and McCleary 1951; Moreland and Zajonc 1977; Murphy and Zajonc 1993; Winkielman, Zajonc, and Schwarz 1997; Zajonc 1980). Dramatic demonstrations of unconscious perception have been provided by human patients after neurological damage. For example, a person experiencing the phenomenon of "blindsight" reports no conscious visual sensation in a portion of visual space after damage to the visual area of the occipital cortex. Yet the same person may be able to "guess" the identity of presented visual objects (Gazzaniga, Fendrich, and Wessinger 1994; Weiskrantz 1986, 1996). A similar phenomenon can be reproduced in normal subjects using brief presentations of visual stimuli (Kolb and Braun 1995). But unconscious emotion is less readily accepted. What do we mean by it? How can a process be emotional if it cannot be felt?

Perhaps one reason we find it easier to accept unconscious cognition than unconscious emotion is simply the weight of empirical evidence that forces us to posit unconscious memory or perception; similar phenomena are rare or missing for unconscious emotion. It takes brute demonstrations to compel us to believe in unconscious psychological processes, and we have those in abundance for memory and perception.

Brute demonstrations of unconscious emotion are less common, less forceful, and so less able to compel assent. Most demonstrations of unconscious processes in emotion have focused on showing that people may be unaware of the events that caused their emotion, rather than unaware of the emotions themselves (Murphy and Zajonc 1993; Nisbett and Wilson 1978; Wilson and Schooler 1991; Winkielman et al. 1997; Zajonc 1980). However, demonstrations of unconscious emotional processes do exist, and they are examined later in the chapter.

Dissociation

The concept of "dissociation" among psychological components provides a useful tool for thinking about emotion (Hilgard 1986). Although dissociation as a concept originated far earlier, the work of Hilgard and his colleagues in the 1960s and 1970s may be said to have revived it for mainstream psychology (see Hilgard 1986 for review). Dissociation in this context means the breaking apart of what seems to be an indissoluble whole into components that diverge under special conditions.

Hypnosis provides the most impressive of Hilgard's dissociation examples, which he and his colleagues called "hidden observer" phenomena, reproducible in approximately 5 percent of hypnotically susceptible individuals (Hilgard 1986). "Hidden observer" refers to the "covert" reporting of an event by a person under deep hypnosis even though the person is subjectively unaware of the event (often having received hypnotic instruction not to perceive it). In such incidents, a person may accurately be said both to be completely unaware of the event (to the extent they deny conscious registration of it) and to have cognitively evaluated it (to the extent they can describe it in detail). For example, in one case "hypnotic deafness" was induced by prior suggestion. Afterward the subject reported with apparent sincerity that he heard nothing during this period. Yet when addressed during the hypnotic deafness state with the suggestion that "perhaps there is some part of you that is hearing" and asked to respond if that were true, the subject lifted his hand. Later he reported remembering the action but not having heard the request, and he was unable to explain his own action (Hilgard 1986). In other cases,

hypnotic analgesia has been induced prior to dental or medical procedures that would ordinarily be quite painful. It is difficult to imagine feigning anesthesia in the face of such procedures. Yet although the subjects reported little or no subjective pain, covert behavioral measures using the hidden observer approach revealed normal pain ratings. One seems compelled to conclude that the subjects' reports are massively deceptive, impaired by a memory deficit of a magnitude that defies belief, or instances of cognitively processed pain that fails to reach consciousness. However difficult to conceive, the option of unconscious emotion may become more plausible than the alternatives.

Dissociation of such magnitude, by definition, is a deviation from our ordinary experience. But although this evidence is often startlingly counterintuitive, it comes from an impeccable source and must be considered. If true, it serves to show what is *possible* in the realm of psychological dissociation.

Dissociation Between Emotional Experience and Remembered or Predicted Emotion

Kahneman and his colleagues have drawn on Bentham to apply the term "utility" to depict qualitative outcomes (Kahneman 1994; Kahneman et al. 1993; Kahneman and Snell 1992), and the utility concept has also been used profitably in behavioral neuroscience studies of reward (Shizgal and Conover 1996; Shizgal, this volume). Utility comes in several types. Kahneman (1994), for example, distinguishes between *instant experienced* utility, *decision* utility, and *predicted/retrospective* utility (figure 27.1). Experienced utility is the outcome's *hedonic* value—the degree to which it is *liked* or *disliked*.[1] Decision utility is the degree to which the outcome is *wanted* or *unwanted*, manifest in decisions to get it, lose it, keep it, or avoid it. Predicted utility and retrospective utility are *expectations* or *memories* about the value of an outcome at another time.

For a coherent and rational mind, the three types of utility might be expected to correlate closely. If an event was pleasant, it should be remembered as pleasant, expected to be pleasant, and desired again. But it appears that the three types of utility often diverge for outcomes in real life (Kahneman 1994; Tversky and Kahneman 1986). Decision utility, the degree to which one wants a particular outcome, may be increased or decreased without changing the outcome itself (experienced utility). For example, mere *possession* of a mildly good outcome, such as a free gift mug, increases the decision utility assigned to it: loss has more impact than gain on decision utility, even for the same object (Kahneman, Knetsch, and Thaler 1990). Still, one might argue that this is not necessarily dissociation between wanting and liking (decision utility and experienced utility): perhaps a mug in the hand is not only wanted more but is also *liked* more than one on the shelf?

Dissociation of Belief from Wanting/Liking

Other dissociations cannot be explained away in this manner. Some come from experiments on predicted pleasure and remembered pain in which experienced hedonic utility is assessed and compared to future or past decision utility ratings. For example, Kahneman and Snell (1992) asked subjects to predict what would happen to their liking and

FIGURE 27.1 Reward Value

Expected/ Remembered Utility	Decision Utility	Instant Experienced Utility
Beliefs about value	Wants	Likes

Notes: Three types of utility, corresponding to beliefs about value, wants based on value, and the hedonic value of actual experience. For a given outcome, these three types of utility might be expected to covary together for a rational individual who had experienced the outcome.

wanting ratings for a palatable ice cream if they ate a small portion of it every day for a week. Then the subjects ate the ice cream over a week and rated it each day. Most subjects predicted that their liking/wanting ratings would decline over the week, and they did. But subjects markedly overestimated the magnitude of their decline, doubling it in their predictions (Kahneman and Snell 1992). Their predictions seemed to be guided by the true belief that a monotonous diet reduces preference for a food (LeMagnen 1967; Rolls et al. 1981). But their cognitive belief was apparently stronger than the truth itself, and they misjudged the magnitude of the change. In a second study, Kahneman and Snell asked subjects to taste a spoonful of marginally palatable unflavored yogurt, to rate their liking of it, and to predict how much they would like it and want to eat a full-sized serving of it at home the next day, and then a week later after eating it every day. Again, most subjects predicted a long-term decline over the course of a week. They also predicted they would begin the week (the next day) at the same liking/wanting level they gave in the "spoonful taste test." They were wrong on both counts: they rated each full-sized serving as more unpleasant than they had found the spoonful, and it was their dislike for the unflavored yogurt that declined over time. The inaccuracy of their prediction is striking, since it applies to a relatively commonplace experience: getting used to a new food (figure 27.2).

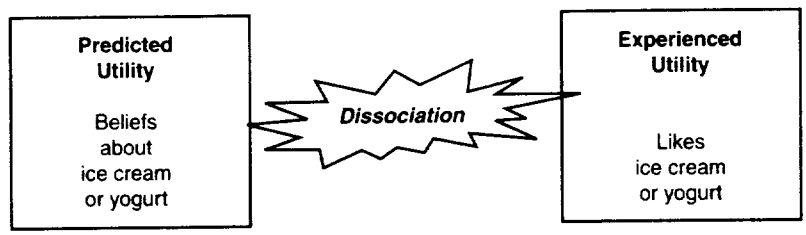
Memories for pleasures and pains are as vulnerable to distortion as predictions. For example, Thomas and Diener (1990) found that subjects' memories of their emotions of the past several weeks tended to overestimate the intensity of emotional events, as compared with their previous daily reports of the events themselves. They also found that emotion frequency tended to be confused with emotion intensity in memory. Recalled intensity ratings for a type of emotion were often biased by its frequency, especially for negative emotions.

Dissociation of Disliking from Decisions

Decision utility (wanting) and predicted utility (beliefs) can be decoupled from experienced utility (liking) on the basis of memory distortion. In a dramatic example, subjects were asked to rate two painful procedures, which induced pain by submersion of the hand in ice-cold water (Kahneman et al. 1993). Later they chose the "least aversive" one to repeat based on their memory of each. Fascinatingly, subjects could be induced to choose the more painful procedure (measured by their own on-line ratings) if that procedure happened to include a decrement at the end (figure 27.3). A similar dissociation between pain reported on-line and remembered later was found in a painful real-life medical procedure (Redelmeier and Kahneman 1996).

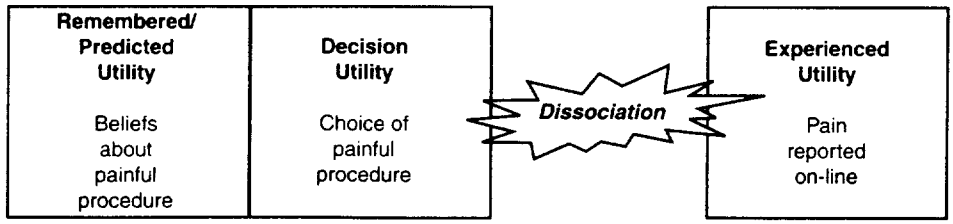
People may be no more accurate in their memories for pleasure than they are in remembering pain. If one asks subjects to report hedonic ratings during a meal, as they pass from hungry to full, they report that the food becomes less palatable. This change of hedonic experience due to physiological factors has been labeled "alliesthesia" (Cabanac 1971). Similar palatability decrements after a meal have been detected in animal studies using electrophysiological or behavioral measures of affective expression to taste, indicating that alliesthesia is shared across species (Berridge 1991; Cabanac and Lafrance 1990; Rolls et al. 1986). Yet despite the fundamental nature of this phenomenon, when people are asked to remember *why* they typically stop eating at the end of a meal, they virtually never report reduced palatability as a reason and may even deny it if it is suggested as a

FIGURE 27.2 Dissociated Reward Value



Notes: Dissociation of predicted utility (beliefs about future emotion) from actual subsequent utility (liking ratings for ice cream or yogurt; based on Kahneman and Snell [1992]).

FIGURE 27.3 Dissociated Pain



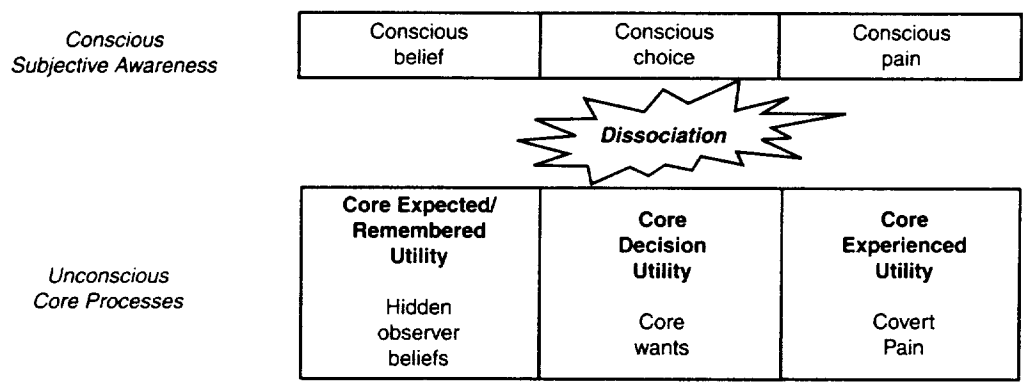
Notes: Dissociation of choice and belief about a painful experience from the actual experience itself, due to the distortion of memory for pain (based on Kahneman et al. 1993). Alliesthesia (change in food palatability as a function of satiety and repetition) provides a similar dissociation for pleasure (based on Mook and Votaw 1992).

potential explanation (Mook and Votaw 1992). Although the hedonic decline is quite apparent in on-line subjective ratings, it seems not to be sufficiently noticeable in memory to be explicitly recalled.

Dissociation of Consciousness from Core Process

The studies above reflect dissociation between the *beliefs* (predictions and memories) people hold about their wants and likes—together with decisions based on those beliefs—from the actual emotional *experience* of events when they occur. As a general rule regarding such dissociations, instant experienced utility (pleasures and pains) seems readily dissociated from remembered utility (memories of pleasure and pain), from expected utility (predictions of future pleasure or pain), and from decision utility (decisions between outcomes that will produce pleasure or pain). Typically, remembered utility, expected utility, and decision utility all cohere together (decisions are usually congruent with past memory and future expectations about particular outcomes), but all of these can be dissociated individually and as a group from experienced utility (the actual experience of the outcome itself).[2] But we can go a step further and actually break apart the instant experienced utility of a particular outcome into dissociated components. It is possible to dissociate *subjective experience* of an event from the underlying *core processes* that constitute wants and likes for that immediate event. The example of hypnotic covert pain from Hilgard, described earlier, is a nice ex-

FIGURE 27.4 Painful Event in Hypnosis



Notes: Hypnotic dissociation of conscious awareness of pain from underlying core processes of the emotion for all types of utility. Hypnotic analgesia reduces the subjective awareness of pain as an experienced event, and therefore decisions and beliefs based on subjective pain. But underlying processes of pain, detected by hidden observer measures, persist. (Description of hypnotic analgesia based on Hilgard [1986]).

emplar of an unconscious emotional core process. It is "pain" in a sense—but not in the usual sense of conscious feeling.

For ordinary people, it may be possible to identify unconscious aspects of emotion even without hypnosis. There are several levels of evidence (in order of increasing strength for unconscious emotion): emotions triggered by unconscious perception; unconscious changes of emotion; and instances where emotion itself is unconscious. The first level consists of demonstrations that it is possible to manipulate conscious aesthetic ratings of a conscious stimulus by prior exposure to events too brief to be consciously perceived, such as by a four-millisecond tachistoscopic presentation of a smiling or scowling face (Murphy, Monahan, and Zajonc 1995; Murphy and Zajonc 1993; Winkielman et al. 1997). These demonstrate unconscious affective processing, at least in the sense that subjects remain unaware of what caused their affective rating. However, it is not clear in such cases whether subjects are completely unaware of the emotion itself or merely unaware of the manipulating event. In support of true unconscious emotion, Winkielman, Zajonc, and Schwarz (1997) found that even when subjects' affective evaluations were altered by prior subliminal presentation of a face, the subjects denied afterward that they had had an emotional experience at the time of the subliminal presentation. Still, it is conceivable that a conscious emotion occurred but was not remembered.

A second, slightly stronger, level of evidence consists of cases in which a conscious emotion is changed, but the *change* in that emotional state seems not to be consciously perceived. For example, in a study of fear, Arntz (1993) asked women who suffered from a spider phobia to perform a series of progressively more difficult actions: to walk toward a spider that was captive in a jar, to touch the jar, to open it, to use a pencil to touch the spider, to dump the spider in an open sink, to touch the spider by hand, and to let it walk on their hand. The women could refuse at any point. They were asked to report their subjective fear or anxiety at each step. Before the test, the women were given either a dose of naltrexone (an opioid antagonist drug that blocks brain receptors for endorphin and enkephalin neurotransmitters) or a placebo, without being told which they received. The high dose of naltrexone markedly inhibited approach to the spider. After receiving it, women refused at an earlier stage (usually before using the pencil to touch the spider) compared to when they received the placebo (usually after touching the spider with the pencil). But subjective reports of fear were *not* significantly increased at *any* stage by naltrexone (though there was a nonsignificant trend toward elevated means). As Arntz notes, this is not entirely conclusive, as a different measure of subjective fear might have detected an effect of the opioid blocker on subjective fear, and the behavioral and subjective measures were obtained in different groups. But oral report is a legitimate measure—and by it these women were not aware of any naltrexone-induced change in fear.

Unconscious Emotion and Motivation

The strongest level of evidence for unconscious emotional processes comes from instances in which the emotion itself—not merely a change in it—is inaccessible to subjective awareness. Not only is unconscious emotion hard to define conceptually, but it is also hard to detect empirically. We can't expect to find lots of instances. But if we find any instances that stand up to close scrutiny, we must recognize them as phenomena of key importance. By their existence, they would radically change the range of psychological entities that must be considered in theories of emotion.

EEG Evidence for Unconscious Emotional Responses

One source of evidence for unconscious emotional responses may come from EEG studies of reaction to subliminal words. For example, Shevrin and colleagues examined classically conditioned fear, instilled by pairing a brief, subliminally presented picture with electric shock (Wong, Shevrin, and Williams 1994). They found that subliminal presentation of the conditioned stimulus came to elicit a distinctive EEG component, even when the subject failed to detect the presentation of anything at all, and they interpreted their results to mean that a conditioned fear response can be elicited entirely outside of conscious awareness. In a related study, Shevrin and colleagues (1992) presented phobic patients with lists of emotional words from various categories. The words were presented tachistoscopically, either too briefly to be perceived consciously (one millisecond) or just long enough to register consciously (thirty to forty milliseconds). One category of emotional words *evoked a more rapid high-frequency EEG response when presented in the subliminal mode* than when the words remained onscreen long enough to register consciously. Words from this emotionally

laden category appeared more effective at evoking an emotional EEG response—in other words, when the patient remained unaware of what had been presented.

Behavioral Evidence of Unconscious Liking and Wanting for Drugs

Evidence for unconscious emotional processes may also be found in human behavior, without using tachistoscopic procedures. A striking example comes from studies of apparently unconscious self-administration by drug addicts (Fischman and 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 and 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 be saline solution without any 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.

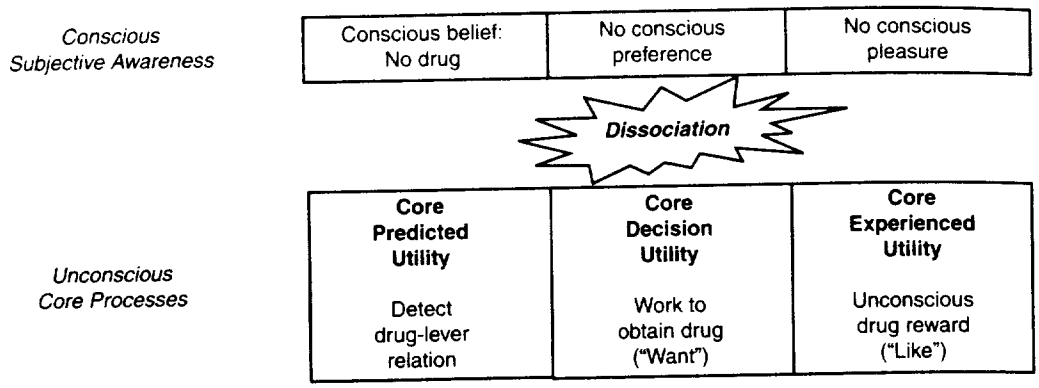
At moderate to high doses (eight to fifty milligrams of cocaine), subjects described the subjective effects as pleasant and typical of cocaine, and they reliably pressed the button that would obtain the highest available dose. But at the lowest dose of cocaine tested (four milligrams), a remarkable dissociation 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 four-milligram dose was not below threshold by all measures. According to the cumulative record of button pushing over the two- to four-hour session, the addicts ***chose and pressed the button that delivered four milligrams of cocaine far more often than the button for saline,*** even while they were unable to detect consciously a difference between the two infusions. As Fischman (1992) recounted: "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]" (179).

If we apply the terminology of Kahneman and his colleagues, Fischman's drug users had zero utility (experienced, decision, and predictive) for the lowest cocaine dose, according to their own subjective report. In ordinary terms, they did not like it, 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," the watered-down drug reward. They worked for it and selectively strove to gain it.

Such stark dissociation between awareness and behavior is rare, but this finding is not a single isolated fluke. Another study by Lamb and his colleagues (1991) of hospitalized addicts who received morphine injections in return for button pressing found similar results. In that study, heroin addicts received an injection of the same dose of morphine or of saline each day, Monday through Friday. The weekly dose changed unpredictably: each Monday brought its own adventure. After each injection, the subjects reported their subjective experience of the drug. Did they like it? How much drug had they been given? How much would they pay for it if it were sold on the street? Lamb and his colleagues were concerned especially with what happened on Thursdays and Fridays, after the addicts had several days' experience with a particular injection. On these days, the subjects had to "earn" their injection, if they wanted it, by pushing a lever three thousand times within forty-five minutes. The subjects did indeed work hard for every injection that contained any morphine at all. For the saline, they refused to work reliably. In their subjective reports, they described the morphine injections as "quite good," "contained drug," "worth paying money for," and so on—all except for the lowest dose of morphine (3.75 milligrams), which they described as equivalent to saline: worthless and empty, despite the fact that they had worked as hard for it as for any of the other morphine doses.

Drug addicts who work to obtain a threshold dose that they cannot subjectively detect, while unaware that they have preferentially chosen it, or who repeatedly press a bar to regain an experience they rate as "worthless," have something in com-

FIGURE 27.5 Unconscious Drug Reward



Notes: Dissociation of conscious drug-related emotion from the underlying unconscious core processes of "liking" and "wanting," based on the descriptions of Fischman and Foltin (1992) and Lamb et al. (1991). The dissociation of awareness from core processes applies to all three types of utility and is revealed in the behavior of addicts seeking a "below threshold" dose of cocaine or morphine. Although they may not be subjectively aware of the drug, they may nonetheless show behavioral evidence that they "like" it, "want" it, and act on their belief of how to get it.

mon with Hilgard's hypnotic subjects. The hypnotic subjects' hidden observers reported covert pain, while the subjects themselves reported they felt no subjective pain. Both cases give behavioral evidence of the existence of an emotional/motivational state that the conscious individuals deny having (figure 27.5).

Unfortunately for those who would study unconscious emotional processes, dramatic dissociations between subjective experience and objective emotional responses so far are limited to special cases such as hypnosis, subliminally brief events, and addicts exposed to threshold doses of drugs. Even these instances provide only sparse information, since investigators are not often theoretically prepared to deal with the dissociations they find between subjective report and behavioral manifestations of emotion. Further studies are needed. But while rare, the instances of unconscious emotional responses collected so far have an important consequence for psychological theory if we accept them as real. They contradict the idea that emotions are necessarily conscious states.

Summary

People often are not accurately aware of what they will like or want in the future, or of what they have liked or wanted in the past. There may even be special moments in which people are unaware of their own "likes," "dislikes," and "wants." These dissociations indicate that emotional core processes are separable from the conscious experience we ordinarily think of as the entire emotion. Even those who believe that emotion is conscious by definition would probably accept that these examples reveal something that is crucial to an understanding of emotion. But although these instances of dissociation point to the existence of unconscious emotional processes, they tell us nothing about the nature of those processes.

Measuring "Liking" and "Wanting" in Creatures That Don't Speak

How can one study emotional core processes that are not accessible to conscious awareness? Psychologists typically measure emotions by asking people to say how they feel. But verbal reports of feelings may miss the very core processes we wish to examine. To explore psychological processes unrevealed in verbal report we also need measures that don't depend on introspection. These measures may be physiological or behavioral. What is necessary is a distinct relation between any measure we choose and underlying emotional processes.

Traditional psychological studies of emotion have focused on adult subjects who can describe their feelings. Human infants prior to language, or animals incapable of language, would be regarded by many to be poor subjects for studying emotion

precisely because they cannot put their feelings into words. But this disadvantage vanishes if we switch our focus of interest to core processes of emotion. Lack of language is not a problem if the process of interest is essentially separate from subjective introspection and verbal report. Infants and animals may in fact be the best subjects for studying some basic aspects of core processes of emotion precisely because of their relative freedom from the many cultural factors that influence construction, expression, or inhibition of adult emotional reactions (Ellsworth 1994; Markus and Kitayama 1991; Ortony and Turner 1990).

Measurement of infant emotion poses a knotty problem to the psychologist but has perhaps been viewed as a more straightforward task, at least in certain respects, by countless generations of parents. Emotional processes find one expression in vocal and behavioral affective reactions. Crying, smiling, and other affective reactions that involve distinct patterns of facial and body responses can often give insight into underlying psychological states (Ekman 1992; James 1884). Even mildly emotional events, such as a pleasantly sweet taste or an unpleasantly strong bitter or salty taste, evoke distinctive affective reactions from human infants (Steiner 1973, 1974; Steiner 1979, figure 6).

The idea that emotions are revealed in behavioral affective reactions as well as in subjective reports is by no means new. Ribot (1897) wrote a century ago: "Every kind of emotion ought to be considered in this way: all that is objectively expressed by movements of the face and body, by vaso-motor, respiratory, and secretory disturbances, is expressed subjectively by correlative states of consciousness, classed by external observations according to their qualities. It is a single occurrence expressed in two languages" (112). Perhaps the reader will have concluded after all that an emotion is not necessarily a single unitary psychological process. But the two languages, objective affective movements/physiological reactions and subjective states/verbal reports, still both express something important about underlying emotional processes (LeDoux 1996; Panksepp 1991, 1992).

It is important to stress the limits of the case being made for the usefulness of affective reactions in the study of emotional processes. There is no implication of a one-to-one relation between affective expression and emotion as an experienced event. Affective expressions can be counterfeited or suppressed, especially by humans. Cultures may construct different configurations of emotional reaction. Many emotions may have no distinctive nonverbal expression. A single emotional reaction may occur in more than one emotional context. But these reservations merely define the border of our interpretive framework for studies of affective reaction. They do not rule out the usefulness of behavioral reactions for the study of emotional core processes.

Affective Reaction and Emotion in Animals

To the extent that we grant emotion to animals, our basis for doing so is likely to be precisely the same as for granting emotion to human infants: their affective reactions strike us in some ways as similar to our own (Darwin 1872; James 1884). Even theorists who believe that human emotions are primarily constructed from social and cultural frameworks still grant that animals share at least some emotions in common with us, based on their facial and other affective reactions to events: "So, for example, one may feel confident in attributing anger and fear to chimpanzees, cats, dogs, and even rats" (Ortony and Turner 1990, 321). The question for a social construction theorist is *which* emotions are shared and which are not.

From a "subcomponents of emotion" perspective, the real question is not *which* emotions we share with animals. Fear itself may not be a unitary phenomenon, to be considered simply present or absent. Even a simple pain or a simple pleasure may be different across species, across different people, and even across different occasions for a single person. This is because it contains multiple core components that may vary in balance. The real questions are: What core processes of emotion, taken as subcomponents, exist in animals, and how similar are they to our own emotional core processes? What is their psychological nature? What are their neural substrates and causes? Core processes of emotion may be shared with creatures that don't speak—infants and animals—even if the final emotion, as a composite event or subjective experience, is quite different in them than in us.

How can a core process of emotion be recognized? *Primarily by features that it shares with ordinary conscious emotion.* Even if unconscious, core processes of emotion must evaluate whether something is good or bad. They must respond qualitatively to the positive or negative nature of an event. They may be specific to a particular emotional category, such as fear, or to a particular

FIGURE 27.6 Facial Display of Hedonic Impact

Notes: Affective expressions of a three-week infant to a sweet taste (left) and to an intensely salty taste. Observations collected by Harris, Booth, and Berridge; photo from Berridge (1996) following Steiner (1973).

type of encounter, such as a loved offspring, a painful injury, or a desired food. That minimal definition is all we have with which to begin. Beyond this, the identifying features of emotional core processes must be established on the basis of evidence. Rather than assert definitions a priori, we must look to experimental results for more detail about the defining features of emotional core processes. That is the goal of the remaining sections.

Conscious Emotion Versus Core Processes of "Emotion"

Despite shared features, there *is* an important difference between emotion as a subjective event and the core processes of emotion that may occur independent of subjective awareness and are revealed through behavioral or physiological measures. One is a conscious emotion, the other is not. It makes sense to distinguish between unconscious emotional core processes and conscious emotional experience, and not to call them by the same name. Perhaps we could agree to use unmodified words such as wants, likes, pains, and fears to denote the subjective emotional feelings. We distinguish these—with quotation marks—from mere "wants," "likes," "fears," "pains," and other unconscious core emotional processes, which, though behavior reveals them to exist and they share features with their subjective states, are not fully worthy of the traditional name (Berridge and Robinson 1995; Berridge 1996).

Core Processes of Positive Emotion in Animals

As conscious entities, we know what we mean by reward: something that is both liked and wanted. In our conscious experience of positive emotions,

we want the things we like, and we like the things we want. Liking and wanting seem so closely entwined that we might almost regard the words as referring to the same basic emotion. But the underlying core processes of positive emotion may not fit into this familiar psychological category of reward. Instead, affective neuroscience studies indicate they can be broken apart into dissociable components. As a first approximation, these core processes can be called "liking" versus "wanting," although we will see that the actual processes may diverge from what is ordinarily meant by these familiar words.

Ever since the discovery that rats would learn to electrically stimulate an electrode implanted in "pleasure centers" in their brains, animal research on the brain substrates for pleasure and reward has been predicated on the postulate that animals, like us, often seek to repeat pleasurable events (Olds 1956; Olds and Milner 1954). The pleasure was inferred, correctly or not, from the observation that animals sought the stimulation. The brain mechanisms of positive emotion have been studied largely by posing questions to animals about the "decision utility" of emotional states. Whether a brain system mediates an animal's "liking" for a reward, in other words, has been approached primarily by answering the related question of whether it "wants" the reward: whether it will work to gain the reward. This approach has revealed many insights into the brain systems whose activation is rewarding in their own right, and that mediate the value of natural rewards such as food or sex (Fibiger, Phillips, and Brown 1992; Gallistel 1994; Hoebel 1988; Olds 1977; Panksepp 1991; Phillips et al. 1992; Shizgal 1997, this volume; Shizgal and Conover 1996; Smith 1995; Valenstein 1976; Wise 1989, 1996; Yeomans 1989). But it rests on the indirect inference of "liking" from "wanting."

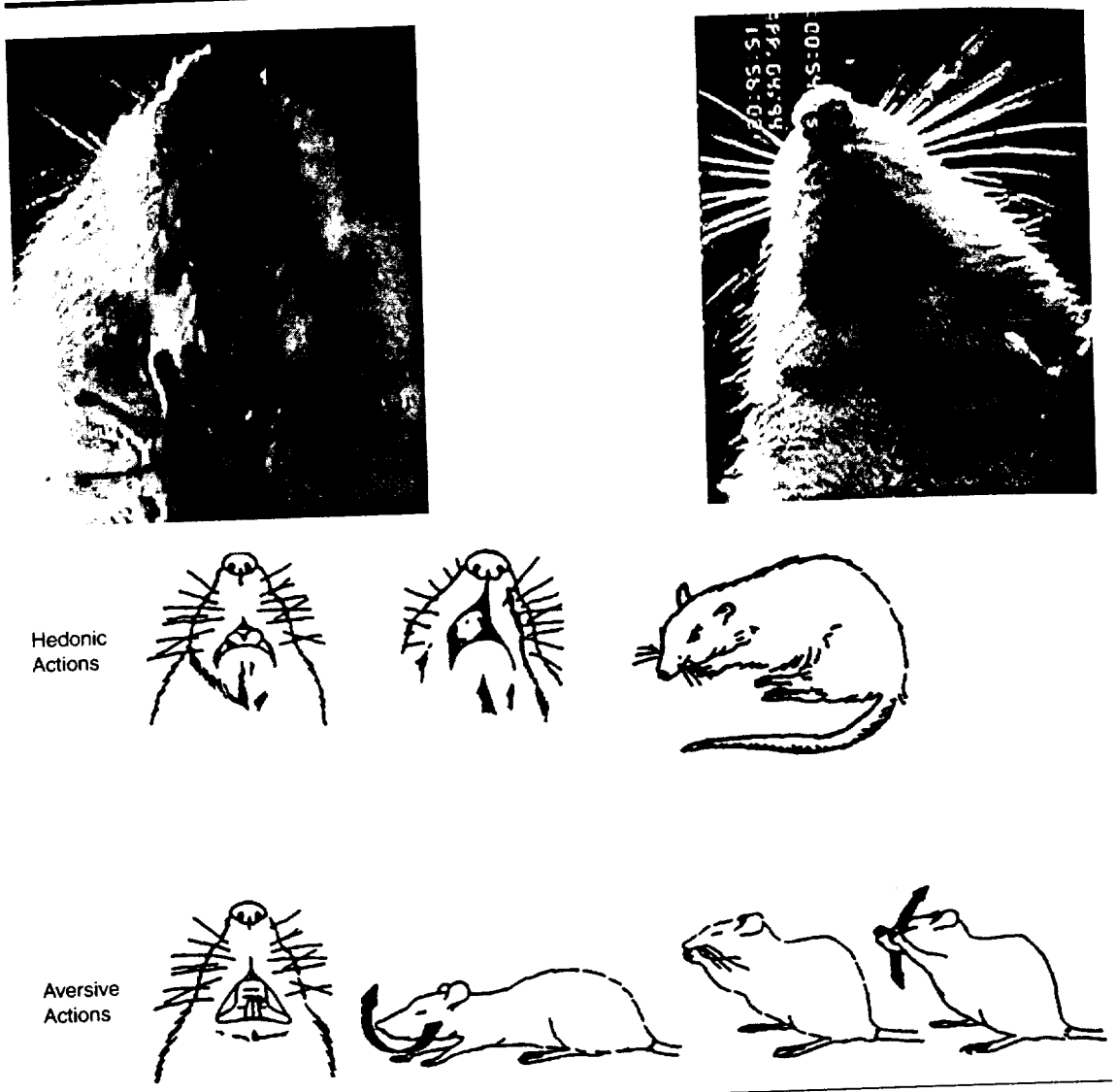
Affective Expression of "Emotion"

A more direct measure of "emotion" such as "liking" is through affective reaction patterns. Animal affective reactions are often less intuitively obvious to us than our own. Yet many can be recognized. Chimpanzees and other primates, for example, show affective expressions to pleasant sweet tastes or unpleasant bitter ones that are in many ways similar to those of a human infant (Steiner and Glaser 1995; Steiner et al. 1999). Given a sweet drink of sugar water, the chimpanzee smacks its lips and reaches for more. Given a bitter drink, it screws up its face into a grimace and gape. Animals other than primates have affective expressions less like ours. But even so apparently unpromising a subject as the rat, an omnivore like us that prefers sweet and fatty foods and dislikes bitter ones, emits distinctive facial and body affective expressions that mirror human and primate affective reactions to tastes (Grill and Berridge 1985; Grill and Norgren 1978b). Sweet tastes elicit a hedonic pattern of reactions such as tongue protrusions—sort of a licking of lips—paw licking, and related movements (figure 27.7). Bitter tastes elicit an aversive pattern of different expressions such as gapes, headshakes, and frantic wiping of the mouth.

There are several reasons to believe that affective reactions of rats to food reflect core processes of emotion similar to those of human pleasure and displeasure (Berridge 1996). One is that they share some expressive movements with equivalent human emotional expressions (such as gape to bitterness). Another is that they fluctuate in similar ways as human subjective pleasure as circumstances change. Human emotional ratings of food pleasure or displeasure can be altered by physiological factors such as hunger or satiety and by psychological factors such as learned aversions or food preferences (Cabanac 1979; Rozin and Schulkin 1990). The affective reaction of rats to taste can be altered by the same sets of physiological and psychological factors. The changes in the animal behavior match the changes in human reports of subjective pleasure (Berridge 1996; Grill and Berridge 1985).

For example, an extremely salty taste such as seawater is ordinarily perceived as unpleasant by humans, and it elicits aversive patterns from rats. But salt elicits hedonic patterns if a rat is in a physiological state of sodium depletion, which produces salt appetite (Berridge et al. 1984; Schulkin 1991), a state in which humans would also find salt to be more pleasant (Beauchamp et al. 1990). A milder enhancement of food palatability similarly accompanies ordinary hunger and is evident in both human reports and animal affective reactions (Berridge 1991; Cabanac 1979; Cabanac and Lafrance 1990). In the opposite direction, pleasure can be changed to distaste. For example, humans often form aversions to the taste of novel foods that have been accompanied by visceral illness (Rozin and Schulkin 1990). They subsequently find the food to be subjectively unpalatable despite having liked it before. Similarly, a rat that emits a hedonic pattern to a novel sweet taste that is followed by visceral illness will switch to

FIGURE 27.7 Animal Display of Hedonic Impact



Notes: Affective expressions of rats to sweet and bitter tastes. Hedonic "liking" patterns include tongue protrusion to a sweet taste (left photograph), lateral tongue protrusion, and paw lick (drawing). Aversive "disliking" patterns include gape (right photograph), headshake, face wash, and forelimb flail (drawing). Drawing after Grill and Norgren (1978a).

aversive expressions to future presentations (Grill and Norgren 1978a).

The correspondence between human subjective pleasure or displeasure and animal/infant affective reactions suggests that they share a core process of hedonic or aversive evaluation (Berridge 1996). This correspondence need not imply that the conscious experience of emotion is also the same. Sometimes it is plausible that they correspond: in the case of an ordinary human infant or adult chimpanzee, many observers might be willing to grant conscious emotions similar to our own. Some might extend this to a rat. But sometimes it is implausible to posit consciousness as an accompaniment to the core emotional processes reflected in affective reactions. For example, anencephalic human infants, who have been born without any forebrain at all, still show both positive and nega-

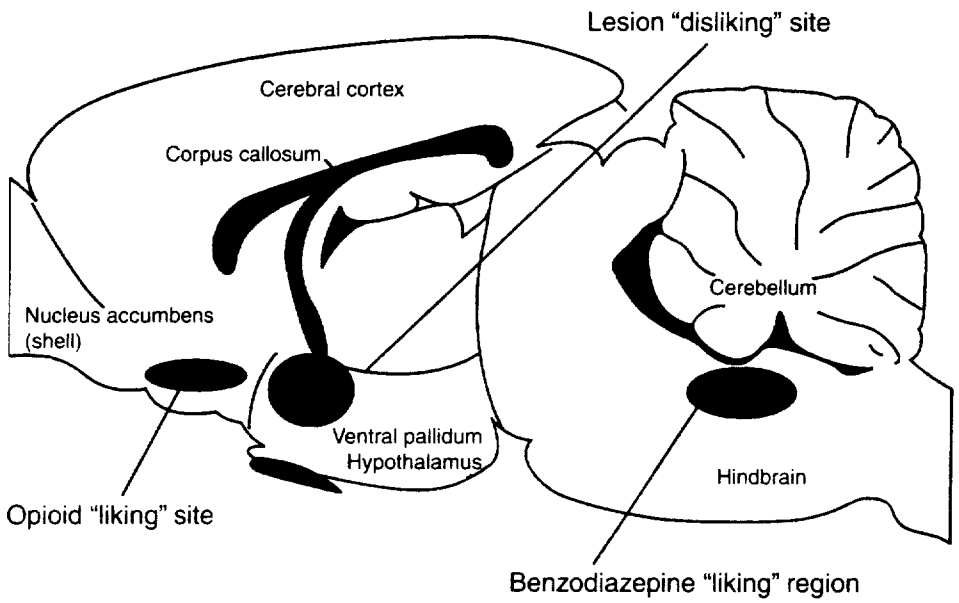
tive affective reactions to presentation of sweet and bitter tastes during their brief lives (Steiner 1973). Similarly, "decerebrate" rats that possess only a brain stem and no forebrain retain a basic capacity to show positive and negative reactions appropriately to sweet and bitter tastes (Grill and Norgren 1978a; Grill and Norgren 1978c). Few would be willing to posit any conscious experience recognizable to us to an anencephalic infant or a decerebrate rat. Still, core processes of evaluation demonstrably exist in such creatures, although they are simpler in behavioral manifestations than the corresponding processes in their full-brained counterparts (Grill and Berridge 1985). Behavioral reactions provide windows into core processes of "wanting" and "liking" that are basically independent of whether the core process is accompanied by subjective emotional experience.

Neural Systems of "Liking" and "Disliking"

Core processes of emotion can be powerfully altered by manipulations of the brain. These can be done painlessly on rats in which the necessary preparations are made while they are anesthetized. Brain manipulations typically change both "liking" and "wanting" core processes of reward together (figure 27.8). One example comes from brain lesions that produce "aphagia"—the pathological failure to eat. The most famous brain region related to appetite and food reward is the lateral hypothalamus and adjacent zones, where lesions have long been known to abolish normal feeding (Teitelbaum and Epstein 1962). Aphagia can lead to starvation unless the individual is artificially fed. (That is always done in contemporary studies.)

Food is not merely "unwanted" after such lesions, it is also "disliked." The aphagia is driven by a dramatic emotional shift. If a normally delicious sweet food is placed in the mouth of the rat, it elicits the complete set of aversive reactions normally reserved for bitter tastes (Cromwell and Berridge 1993; Schallert and Whishaw 1978; Stellar, Brooks, and Mills 1979; Teitelbaum and Epstein 1962). By mapping the brain region involved, my former student Howard Cromwell discovered that the location where neuron destruction produces the emotional shift is actually outside the hypothal-

FIGURE 27.8 Brain Substrates of Food "Liking"



Notes: Brain substrates of food "liking." These include the ventral pallidum site, where damage produces "disliking" or aversion even for sweet tastes; the shell of the nucleus accumbens site, where opioid stimulation by morphine enhances food "liking," and the brain-stem region, where benzodiazepine/GABA stimulation also enhances food liking. Each manipulation of "liking" changes food "wanting" secondarily. See Berridge (1996) for review. Brain atlas based on Paxinos and Watson (1996).

amus, within a structure immediately above and in front of it called the ventral pallidum (Cromwell and Berridge 1993). This is the only site in the brain, as far as I know, where a small lesion can reverse the emotional response to an event from "liking" to "disliking." In a rat brain, it is roughly the size and shape of a dried pea. In the human brain, its size might presumably be closer to that of a larger fresh pea.

Neurotransmitters of "Liking"

Drugs can be potent emotional stimuli. Taking recreational drugs that stimulate brain neurotransmitter systems (cocaine, heroin, etc.) is perhaps the only direct physiological manipulation of the brain that people in substantial numbers are willing to practice on themselves. Pharmacological manipulations of the brain can change "liking" and "wanting" together, just as brain lesions can. But the special feature of some drugs is to shift emotion in the *positive* direction, activating the "liking" and "wanting" components of reward themselves and potentiating the hedonic impact of other events.

Appetite, or "wanting" for food, can be enhanced by a number of drugs, administered either directly to the brain or systemically to the entire body. For example, eating is stimulated by drugs that activate opioid neurotransmitter receptors, such as morphine (Gosnell 1987; Stanley, Lanthier, and Leibowitz 1989). Eating is also stimulated by drugs, such as diazepam (Valium), that facilitate gamma-amino-butyric-acid (GABA) neurotransmission by activating benzodiazepine receptors that are attached to GABA receptors (Berridge and Peciña 1995; Cooper, Higgs, and Clifton 1995). The effects have been best documented in animal studies but appear to occur for humans, too (Drewnowski et al. 1995; Kelly et al. 1992).

Hedonic "liking" for food also can be facilitated by opioid and benzodiazepine drugs. In animals, opioid and benzodiazepine drugs potently enhance hedonic reaction patterns to sweet tastes (Berridge and Treit 1986; Doyle, Berridge, and Gosnell 1993; Gray and Cooper 1995; Parker 1995; Parker et al. 1992). Human subjective palatability ratings are also changed by drugs relevant to opioid neurotransmitter receptors (Drewnowski et al. 1995).

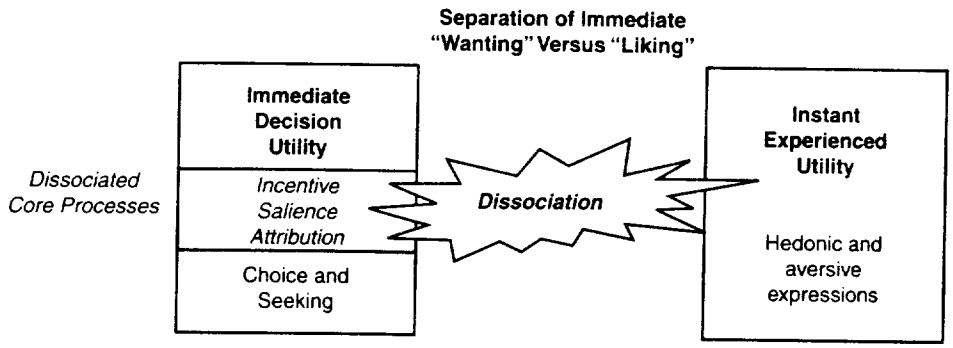
Hedonic enhancement of "liking" occurs even if the opioid or benzodiazepine drugs are delivered directly to the brain of rats by microinjection (Berridge and Peciña 1995; Peciña and Berridge 1995, 1996a). In such a study, the drug is dissolved in a droplet almost too small to be seen by the naked eye, and the tiny droplet flows painlessly into the desired structure of the brain. The droplet is delivered through a permanent microinjection cannula that was implanted on an earlier day while the rat was anesthetized. Minutes after the microinjection, a sweet or other-tasting solution is infused into the rat's mouth, typically for a minute or so, and elicits enhanced hedonic reactions that can be videotaped for later slow-motion analysis (Berridge and Peciña 1995).

Microinjection "mapping" studies have helped identify particular hedonic regions of the brain where benzodiazepine and opioid drugs enhance the emotional response to foods (see figure 27.8). They have showed that neural systems of hedonic processing are distributed throughout the entire extent of the brain, from front to back. For example, an opioid drug like morphine appears to be most effective at enhancing "liking" and "wanting" for food in a small paired region near the base of the front of the brain, called the shell of the nucleus accumbens (Peciña and Berridge 1996b). The left and right members of this pair of sites are shaped a bit like short celery stalks, running lengthwise through the brain (front to back), with their inner concave surfaces facing each other. Microinjections of morphine into the sites enhance hedonic reactions to sweet taste and elicit feeding, whereas injections in nearby surrounding regions fail to do so (Peciña and Berridge 1996b).

By contrast, neural systems responsible for "liking" and consequent "wanting" enhancement by benzodiazepine drugs are contained in the posterior hindbrain. If forebrain systems are disconnected by decerebration, leaving the brain stem to make basic evaluations on its own, a benzodiazepine drug still potentiates positive reactions made by the decerebrate rat to sweet tastes (Berridge 1988). Also, microinjections of a benzodiazepine drug into an ordinary rat's brain elicit more feeding and enhance hedonic reactions to taste more effectively when placed in brain-stem sites than when placed in the forebrain (Higgs and Cooper 1994; Peciña and Berridge 1996a).

Brain systems of food "liking" are thus distributed throughout the entire length of the brain, perhaps as a vertically arranged set of layers. Ordinarily, the layers function together as a single system in a hierarchical fashion (Grill and Berridge 1985). If upper layers are stripped away, the lower ones continue to operate at least to a degree. The core processes of isolated lower substrates still share some evaluative features with ordinary emo-

FIGURE 27.9 Conceptual Dissociation of Decision Utility from Experienced Utility by Manipulation of Dopamine Brain Systems

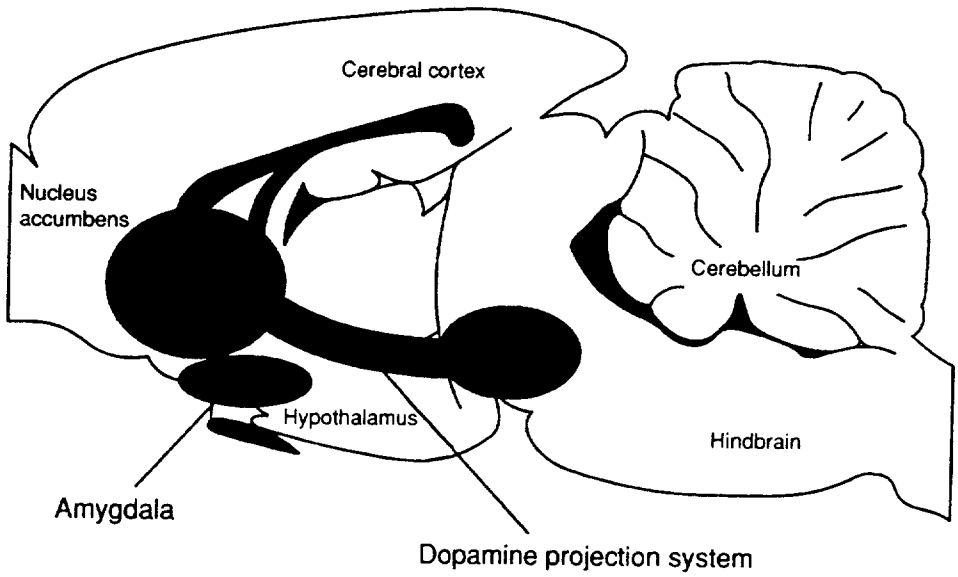


tion, though the features are fewer and less flexible than when all layers are present, and they almost certainly do not include consciousness. If affect can be "free-floating," as pleasure not assigned to a particular object, it may be in part because the neural instantiation of "liking" is distributed widely, including brain-stem systems incapable of representing events as targets. By contrast, "wanting" may always have an object of desire. And "wanting" is mediated by forebrain systems tied to representations of objects and events.

"Wanting" Without "Liking"

In contrast to the brain manipulations that simultaneously change "liking" and "wanting" together, there is a different group of brain manipulations whose effect is selective to "wanting" alone (figures 27.9 and 27.10). These change "wanting" as measured by instrumental goal-directed behavior and voluntary eating—they cause individuals either to seek out food and other rewards and to consume them, or to abandon rewarded tasks and

FIGURE 27.10 Brain Substrates of "Wanting"



Notes: Sites include the ascending dopamine projection from midbrain to nucleus accumbens, where stimulation induces "wanting" without "liking," and where lesions eliminate decision utility without impairing experienced utility or predicted utility, and the amygdala nuclei, where lesions disrupt the elicitation of fear or reward by particular stimuli (see Berridge 1996).

refuse food when given it. But they do *not* change "liking" for that food reward, as measured by hedonic or aversive expressions, in the same individuals. This group of brain manipulations includes hypothalamic stimulation, drugs that act on dopamine receptors or on dopamine release, and 6-hydroxydopamine lesions that selectively destroy neurons that contain dopamine. All of these act on the massive projections of dopamine neurons that stretch from the midbrain to the forebrain, often called the mesolimbic or mesoaccumbens dopamine system (see Hoebel, this volume; Shizgal, this volume). This neural system appears to be a neural common denominator or substrate shared in common by most manipulations that alter "wanting" without changing "liking."

Even hypothalamic stimulation, a potent motivational elicitor, acts in part via this same ascending neural system. Brief pulses of hypothalamic stimulation can be a powerful reward—animals and people will work in order to get them (Hoebel 1976; Olds and Milner 1954; Sem-Jacobsen 1976; Shizgal and Conover 1996; Shizgal, this volume). If stimulation is given freely, especially in sustained bursts, it also has motivating properties. Free stimulation of the lateral hypothalamus can motivate animals or people to eat or to engage in sex, aggression, or another behavior (Glickman and Schiff 1967; Sem-Jacobsen 1976; Valenstein 1976). Which behavior is elicited depends in part on preexisting features of the individual, in part on the electrode placement and other stimulation parameters, and in part on the type of experiences the individual has had previously with hypothalamic stimulation (Valenstein 1976). Dopamine neurons appear to be a crucial link in the chain of causal events responsible for the motivational and rewarding effects of hypothalamic stimulation (Hoebel 1988; Valenstein 1976; Wise 1996; Yeomans 1989).

An early interpretation had been that electrode-induced motivation could be explained by the electrode-induced reward (Hoebel 1988; Olds 1977; Olds and Milner 1954; Wise 1982a). In other words, the pleasure of food, sex, and so on, might be enhanced while the stimulation was on, and that could be a reason the motivated behavior was displayed during stimulation. In a contemporary version of this hypothesis, Shizgal (this volume) suggests that a rewarding electrode evokes high "instantaneous utility," the same psychological concept of instant experienced utility developed by Kahneman and his colleagues (Kahneman 1994; Kahneman, this volume; Kahneman et al. 1997). Instantaneous utility in this sense means the hedonic intensity of the event and also its capacity to generate action to instigate the event (and to resist interruption). Instantaneous utility thus consists of a single global evaluation of current reward intensity, which encompasses both hedonic evaluation and goal-directed action (Shizgal, this volume).

If global enhancement of instant experienced utility were the full story, such brain stimulation should elicit both "wanting" and "liking" for a reward, just as opiate and benzodiazepine microinjections do. But Elliot Valenstein and I have found evidence that "liking" and "wanting" are probably not both activated by brain stimulation of the lateral hypothalamus that evoked eating (Berridge and Valenstein 1991). In our study, rats were allowed to eat or not as they chose during hypothalamic stimulation. Stimulation-induced motivation becomes increasingly stronger with repeated experiences, and after receiving a half-hour of this for several days, many rats became "reliable stimulation-bound eaters." Those rats began to eat as soon as the hypothalamic electrode was turned on, and they stopped eating when the stimulation went off. Once it was clear that stimulation made these rats "want" food, they were tested to see whether the stimulation also made them "like" it more. In order to do that, their affective reactions were videotaped as a sugary solution was infused into their mouths. As the infusion continued, hypothalamic stimulation was turned on for fifteen seconds at a time, interspersed with equal pauses when it was turned off. The sugar infusion remained steady whether stimulation was on or off (since otherwise the rats would have consumed more when the stimulation was on, making it difficult to compare "liking" across the two conditions). Our results surprisingly contradicted the hypothesis that hypothalamic stimulation made rats "want" food by making them "like" it more. Hypothalamic stimulation never enhanced hedonic reactions of rats to sweetness, even though it made the same rats eat avidly. On the contrary, hypothalamic stimulation elevated *aversive* reactions to sweet solutions. Hypothalamic stimulation appeared to make rats "*dislike*" sweetness somewhat rather than "like" it more, but it still made them "want" to eat despite their enhanced "dislike." Cooling the hypothalamus rather than electrically stimulating it can also induce a similar phenomenon of "wanting" food without "liking" it (Berridge and Zajonc 1991; incidentally supporting a hypothesis proposed by Zajonc that focal

brain temperature changes may cause fluctuations in an aspect of positive emotion; see, for example, Zajonc, Murphy, and Inglehart 1989).

Selective Suppression of "Wanting" by Drugs

The mirror image of "wanting" without "liking," namely "liking" without "wanting," can be obtained by suppressing brain dopamine systems. Drugs that block receptors for dopamine, such as haloperidol or pimozide, suppress the incentive value of many rewards (Smith 1995; Wise 1982b, 1994; Yeomans 1989). Such drugs cause animals to refuse to work, whether the reward is food, hypothalamic stimulation, cocaine, or something else. Although the drugs produce motor impairment, too, many investigators believe the reduced willingness to work reflects a distinct motivational deficit. In other words, they reduce the decision utility or "wanting" for such rewards.

But perhaps surprisingly, such drugs do not shift "liking" for the same rewards. Studies of affective taste reactivity aimed at measuring the "liking" for a palatable taste have converged upon the conclusion that dopamine-blocking drugs leave hedonic processes unchanged (Peciña, Berridge, and Parker, 1997; Treit and Berridge 1990). Dopamine receptor blockers fail to shift hedonic expressions to a sweet food or to replace them with aversive reactions, even though they reduce the motivation to eat.

Eliminating "Wanting" for Still "Liked" Rewards

The most dramatic dissociation of "wanting" from "liking" is to eliminate one while preserving the other. Decision utility ("wanting") can be abolished—not merely reduced—while preserving experienced utility ("liking") unchanged, by selective neurochemical lesions that destroy the dopamine system. Many of the aphagia and adipsia symptoms of lateral hypothalamic lesions can be reproduced by microinjections of the neurotoxin 6-hydroxydopamine. In these experiments, the chemical neurotoxin is delivered directly to dopamine neurons while a rat is anesthetized. If particular procedures are followed, the neurotoxin destroys neurons that release dopamine as their neurotransmitter, but it leaves all other neurons healthy. These lesions produce movement deficits, but the aphagia of dopamine disruption cannot be fully explained on the basis of impaired movement alone (Smith 1995). After recovery from their operation, rats that have had 6-hydroxydopamine lesions seem uninterested in food, water, or any other reward. Even if surrounded by mountains of tasty food, they will voluntarily starve to death unless someone regularly feeds them by intubation, even though they seem able to make the limited movements needed to eat.

Such absolute indifference to reward might be explained if the neurotoxin rendered the brain unable to have any reward experience: complete anhedonia. But the rats do not appear to be anhedonic. Despite showing no "wants" whatsoever, rats with 6-hydroxydopamine lesions seem to have normal "likes." We've found that if tastes are infused into their mouths, such rats show normal hedonic reactions to sweet tastes and normal aversive reactions to bitter ones (Berridge and Robinson 1998; Berridge, Venier, and Robinson 1989). These rats are even capable of adjusting their *expected* utility value for particular food by learning about its consequences; that is, they can acquire new "likes" or "dislikes." For example, they learn taste aversions in a normal fashion when a new taste comes to predict visceral illness. If presented with a novel sweet taste, these rats show hedonic reactions, as normal rats do. But if the initial sweet taste is then followed associatively on several occasions by visceral illness, induced by injection of lithium chloride, then rats with dopamine lesions respond to it with aversive reactions on future encounters (Berridge and Robinson, 1998). Taste aversion conditioning switches the *experienced* utility ("liking") of the taste to aversive—reflected by affective expressions—via a change in its *expected* utility or predicted value derived from past associative learning. Both experienced and expected utility apparently remain normal after dopamine loss. Only *decision* utility, or "wanting," appears to be destroyed by the dopamine lesion—reflected by normal intake, preference, instrumental commerce with rewards, and so on—whereas "liking" remains perfectly normal and intact.

The Nature of "Wanting": Attribution of Incentive Salience

I have argued that there is a fracture line of separation between the psychological core processes of "liking" and "wanting." However closely tied together liking and wanting seem in our conscious lives, they are essentially different core processes, with different neural substrates. But what is the *nature* of an unconscious core process such as "wanting"? The word "wanting" is a convenient

label to highlight the dissociation, but it does not tell us much about defining properties.

My colleagues and I have suggested that "wanting" is best viewed as a kind of hybrid core process that combines motivational, associative, and perceptual features. This process transforms the representation within the brain of external stimuli and events. The transformation, which is altered by dopamine manipulations, imbues forebrain representations of objects and events with *incentive salience*. Attribution of incentive salience enables an event to grab attention and to be perceived as attractive, making it a sought-after incentive in its own right (Berridge 1989, 1996; Berridge and Valenstein 1991; Robinson and Berridge 1993).

We posit incentive salience attribution to be one step in a three-step process of reward (figure 27.11), after hedonic activation and the association of an outside event with hedonic activation (Berridge 1996; Berridge and Valenstein 1991; Robinson and Berridge 1993). Incentive salience attribution is mediated in part by neural activity in dopamine-accumbens brain systems, activity that is triggered by subsequent encounters with incentive stimuli or by their cognitive representations. Although stimulus-linked, incentive salience is not merely *sensory* salience. It also enables the attributed stimulus or its representation to elicit approach and to become the target of desire and goal-directed strategies. When translated into conscious awareness, incentive salience may give rise to a subjective experience of wanting or craving. But the core process can occur independently of consciousness—as we saw in the examples described earlier of addicts unconsciously seeking drugs (Berridge 1996; Fischman and Foltin 1992; Lamb et al. 1991). That means its properties may in some ways be studied most directly via behavioral and neurophysiological measures (Berridge 1996; Phillips et al. 1992; Robinson and Berridge 1993).

Perceptual Transformation Aspects of Incentive Salience

The brain structures most strongly linked to incentive salience attributions are those that receive extensive dopamine projections: neostriatum and nucleus accumbens, amygdala, and frontal cortex. These are not primary "sensory" brain structures, but they nonetheless receive massive sensory inputs from the cortex and other sources (Lidsky, Manetto, and Schneider 1985). The motivational effects of hypothalamic stimulation, which act in part via these neurons (Yeomans 1989), depend strongly on the sensory qualities of available targets (Valenstein 1976). The sensory-embedded nature of stimulation-elicited motivation is so marked that a visual signal for food may be effective in motivating behavior when seen through the eye that relays directly to the stimulated side of the brain, but not when seen through the other eye (Beagley and Holley 1977).

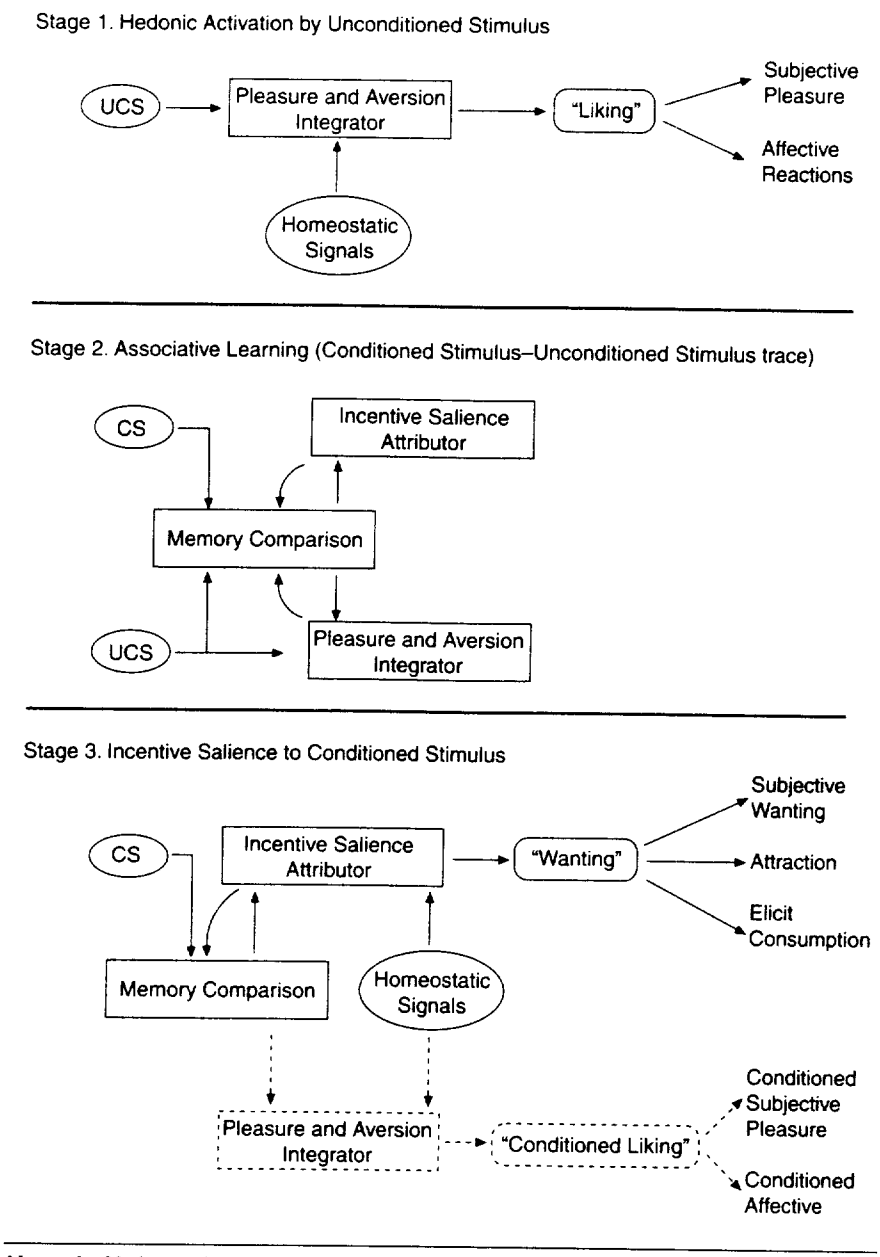
Viewed by the use of neurophysiological techniques that monitor neural activation, many neurons in the nucleus accumbens and neostriatum, as well as some dopamine neurons that project to them, are activated by sensory stimuli—especially if the stimuli have important motivational significance, such as the sight of a tasty morsel that the animal expects to receive (Aosaki, Graybiel, and Kimura 1994; Phillips et al. 1993; Schultz, Dayan, and Montague 1997). I believe the activation of a subset of these neurons in the nucleus accumbens and related sites reflects the attribution of incentive salience to the perceived stimulus. Presumably memories or cognitive representations of incentive events might achieve activation of the same neurons, even in the absence of the physical stimulus. The psychological core process of "wanting" may thus be embodied in identifiable neural events (see Berridge and Robinson [1998] for more discussion of the neural mediation of incentive salience).

It is worth noting that the nature of this core "wanting" process spans traditional psychological categories, such as emotion and perception, and perhaps other categories, too. Incentive salience is a psychological "building block" whose identity would not be anticipated by existing theories of emotion or motivation. Its psychological importance has been revealed by biopsychological manipulations of the brain and observation of their effects on behavior.

Addiction and Sensitization: Dissociated Wanting Run Amok

The hypothesis of incentive salience or "wanting," separate from "liking" and mediated in part by dopamine neural projections, has special application to understanding drug addiction (Berridge and Robinson 1995; Robinson and Berridge 1993). For an addict, drugs of abuse have enormous decision utility. They are powerfully wanted and sought—often at great risk and great cost. The question is, why? Of course, drugs may have been pleasant to an addict in the past, and they can also alleviate withdrawal symptoms. But drug pleasure and withdrawal are probably not sufficient explanations for addiction. Euphoric drugs are sought

FIGURE 27.11 Three Stages of Normal Reward—Incentive Salience Model



Notes: (1) Hedonic pleasure ("liking") acts as the normal trigger for reward. Hedonic neural systems activate the associative and incentive salience steps. "Liking" by itself is free-floating and not sufficient to motivate goal-directed behavior. (2) Associative learning systems are needed to correlate the representation of external objects and events (conditioned stimuli) with hedonic activation. Associative neural systems are separate from those of "liking" and "wanting." (3) Incentive salience is subsequently attributed to conditioned stimuli or their representations by dopamine-related systems, making these stimuli attractive and "wanted." The attributed stimulus acts as an incentive to elicit action and direct motivated behavior.

by addicts even when the available dose is so low or so poor in quality that it can't be expected to give much pleasure. Some addictive drugs, such as nicotine, are not especially euphoric even for people who are addicted. Life for an addict cannot be described as a series of overwhelming pleasures, even when drugs can be had. Hence, pleasure often is not a compelling explanation of addiction.

Similarly, the avoidance of painful withdrawal does not fully explain addiction. Even after an addict has successfully finished withdrawal, addiction often remains a problem. Relapse is a common fate for the graduates of detoxification programs, and addicts who do give up their drug may attest that the struggle did not end with the last pang of withdrawal. Neither pleasure seeking nor pain avoidance provide a fully satisfactory explanation of addiction, even combined (for more discussion of their explanatory inadequacy, see Robinson and Berridge 1993). In other words, the decision utility of a drug for an addict may have strength beyond both its experienced utility or predicted utility. Why?

There may be a straightforward reason. Many addictive drugs, such as cocaine, amphetamine, heroin, and their synthetic derivatives, produce ***neural sensitization*** of brain dopamine systems if the drug is taken repeatedly (Robinson and Becker 1986). The baseline activity rate of a sensitized neural system may be normal, but the neurons are hyper-reactive to a triggering stimulus. Sensitized hyper-responding is elicited by another dose of the drug itself but also appears to involve associative learning about stimuli and contexts that have been paired with the drug (Anagnostaras and Robinson 1996; Robinson and Berridge 1993). The associative focus of sensitized incentive salience on drug stimuli and the act of drug taking may be the reason an addict's intense "wanting" is directed specifically toward drugs. Once induced, neural sensitization lasts for a long time—much longer than withdrawal symptoms—and possibly for a lifetime (Paulson, Camp, and Robinson 1991).

My colleague Terry Robinson and I have suggested an ***incentive-sensitization theory*** of addiction, which incorporates these points. It hypothesizes that neural sensitization of dopamine-related systems occurs in addicts, and that it is responsible for the compulsive nature of drug use in addiction (Berridge and Robinson 1995; Robinson and Berridge 1993). Neural sensitization of incentive salience systems, coupled with associative control of its expression, causes excessive attribution of incentive salience to the act of taking drugs, and to drug-related stimuli and contexts. Drugs become excessively and compulsively "wanted," irrespective of whether they are "liked," and irrespective of whether withdrawal is happening. Addicts, by this view, are much like a rat under hypothalamic stimulation: forced to "want" whether or not they "like" drugs. Paradoxically, if this incentive-sensitization theory of addiction is true, it means that psychological therapies are likely to remain the most effective treatments for addiction for some time. Medication holds less hope, at least for now, because neural sensitization is not reversible by any known physiological or pharmacological means.

Summary

Behavioral affective reactions provide a means of studying core processes of emotion that are not accessible by subjective report. Our biopsychological studies indicate that simple positive emotion has separable core processes of "liking" and "wanting." These psychological components appear to be mediated by different brain systems. The core processes of "wanting" and "liking" typically cohere, but they can be dissociated from each other, as well as from conscious awareness, under some conditions. The compulsive craving of drugs in addiction may be caused by the neural sensitization of the dissociated "wanting" component of incentive salience. For addicts, this may cause drug craving independently of drug pleasure or withdrawal. "Wanting" thus takes on a life of its own and becomes focused on drugs. Whether this account of drug addiction applies also to other types of addictions remains for now an open and unexplored question.

Dissociation of Core Processes of Negative Emotion: Pain, Fear, and Anxiety

Negative states such as pain and fear seem totally different to conscious awareness from positive liking and wanting. But these psychological polar opposites share more in common than one might expect. For example, although we have focused on the role of ascending dopamine systems in positive "wanting," dopamine systems also are needed for at least some types of avoidance behavior motivated by conditioned fear (Salamone 1994).

At the most abstract level, liking versus wanting might be equivalent to a distinction between an

immediate feeling versus doing something about it (and the feeling that accompanies anticipatory doing). Bolles and Fanselow (1980) once suggested that a similar relationship holds between pain and fear. Pain is an affective state, an unconditioned response, a type of experienced utility. Fear or anxiety might be viewed in part as a conditioned or anticipatory response to pain, a type of decision utility similar to the relation of wanting to liking.

In this final section of the chapter, I wish to directly compare positive emotion and negative emotion. My goal is to demonstrate two similarities between them. First, simple negative emotions such as fear can be broken apart into dissociated psychological core processes, just as positive emotions can. This is seen most clearly after brain manipulations. Second, one of the component core processes of negative emotion may overlap to a surprising degree with the core process of "wanting" in positive emotion.

The Anatomy of Fear and Anxiety

A set of fearful affective reactions are evoked from rats by presentation of a sound or other stimulus that predicts a painful event, such as startle, tense freezing, or elevation of heart rate and blood pressure (Davis 1992; LeDoux 1992, this volume; Maren and Fanselow 1996). Conditioned fear reactions are eliminated by several types of brain lesions. Some lesions are relatively uninteresting from the emotional point of view because they simply eliminate sites of auditory processing: the inferior colliculus of the midbrain brain stem, or the medial geniculate nucleus of the thalamus. Other sites in which lesions block conditioned fear are psychologically more interesting, and chief among these is the amygdala.

The neural signal for a sound ascends the brain in two directions relevant to fear from the auditory relay of the thalamus. One goes to the auditory cerebral cortex, and the other to the basolateral complex of the amygdala. Lesions of the auditory neocortex block "old" fears that were conditioned to a sound before the lesion was made (LeDoux 1992). But *new* fearful conditioned reactions to sound can still be acquired based on new learning. One interpretation of this observation has been that fear *ordinarily* is learned through a neocortical pathway but can be diverted to a secondary route after brain damage. A different interpretation would be that cortical sensory lesions change the brain's encoding and *perception* of the stimulus and hence disrupt recognition of sounds previously experienced before the lesion. In either case, fear responses can still be acquired after sensory cortex lesions.

Lesions of the amygdala disrupt the acquisition of new fears, as well as "old" fear to previously trained sounds or places (Davis 1992; Killcross, Robbins, and Everitt 1997; LeDoux 1996, this volume; Maren and Fanselow 1996). They do this even though the neocortex may remain perfectly intact. The same disruption can be produced if the lesion is made smaller and limited to only a portion of the amygdala: either the *basolateral complex* (which receives most of the auditory input from the medial geniculate of the thalamus mentioned earlier) or the *central nucleus* of the amygdala (to which the basolateral nucleus in turn sends its own information). Either lesion disrupts fear conditioning nearly as much as both together. Lesions of the amygdala disrupt many types of fear conditioning in animals and humans (Bechara et al. 1995; LeDoux 1996, this volume; Scott et al. 1997). Thus, at first sight, the amygdala might be taken to mediate the emotion of fear.

Anxiety

Unfamiliar situations can evoke anxiety, which is often viewed as similar to fear, but more diffuse, and is also linked to the amygdala. Ordinary rats and mice, for example, tend to be shy of new places, as expressed by their tendency to avoid elevated and exposed places. These avoidance tendencies are often reduced by amygdala lesions (Davis 1992). Many human children are shy of new situations, too. Individual human children markedly differ in the degree to which unfamiliar situations evoke anxiety and inhibition, and these differences may persist in later life (Kagan and Snidman 1991; Schwartz, Snidman, and Kagan 1996). The persistence of individual human differences in global reactivity, especially to stressful situations, from the earliest months of life through many years later has led Kagan and his colleagues to suggest that these differences may reflect stable underlying neurobiological variations (related to their amygdala) between children.

It has been argued that agitated, or melancholic, depression, which is often accompanied by anxiety, involves a similar brain dysfunction. Jay Schulkin has suggested an explanation that posits the amygdala to be chronically overactivated in such patients, owing to elevation of the glucocorticoid stress hormones that stimulate it (Schulkin 1994). Animal experiments show that cortico-

tropin releasing factor in the amygdala enhances fearful reactions (Schulkin, McEwen, and Gold 1994). Schulkin posits that constant stimulation of this amygdala system in humans who are prone to melancholic depression (for reasons that are not yet clear) may cause their elevated levels of continual vigilance, anxiety, and fear.

Dissociations of Fear: Not a Unitary Emotion?

Taken at face value, the considerations discussed in the preceding section could lead one to conclude that the amygdala generates fearful states as elemental emotions. But it would be too simple to equate amygdala activation with fear. Closer examination shows that fear is not eliminated as a unitary state by amygdala damage. Instead the emotion is fractionated, appearing in some situations but not in others.

For instance, monkeys that fail to show fearful reactions to many frightening events after bilateral amygdala lesions nonetheless still show fear to especially strong stimuli (Kling and Brothers 1992). After the conditioned fear of rats is "eliminated" by amygdala lesions, it can still be reinstated by additional retraining (Kim and Davis 1993). In a striking series of dissociations, Treit and his colleagues found that amygdala lesions suppressed rats' avoidance of a shock-associated object but *failed* to suppress their avoidance of high open platforms (Treit, Pesold, and Rotzinger 1993). To suppress the latter, lesions needed to be made in a different forebrain structure, the septum. A further dissociation between *subtypes of fear* was found when microinjections of a benzodiazepine tranquilizer, midazolam, were placed in particular subnuclei of the amygdala itself. Microinjections limited to the central nucleus suppressed object avoidance but not elevated platform avoidance, whereas basolateral nucleus microinjections produced the opposite pattern of suppression (Pesold and Treit 1995).

Why is fear or anxiety suppressed in some situations but not in others? One possibility is that there are qualitatively distinct subtypes of fear and anxiety, each mediated by its own neural system. Several authors have suggested that such dissociations reflect the elimination of some subtypes of fear but not others, or that they may eliminate essential fear while leaving more elaborate cognitive or behavior reactions to danger (Kagan and Schulkin 1995; Killcross et al. 1997; LeDoux 1996). However, it is difficult to specify the psychological features of the different fear subtypes (beyond merely describing the situations in which fear responses do or do not occur after brain damage) or to say why, for example, fearful freezing or startle should reflect "real fear" more essentially than fearful escape or avoidance measures. An alternative to positing multiple subtypes of fear, and trying to define which is lost after brain damage, is to entertain the possibility that lesions of amygdala nuclei do not result in loss of *any* subtype of fear as a distinct psychological category. ***Instead, they may alter other psychological core processes that are not fear but ordinarily help trigger it in particular situations.*** The nature of these psychological processes, of course, remains to be elucidated, but there are clues about their features in existing evidence.

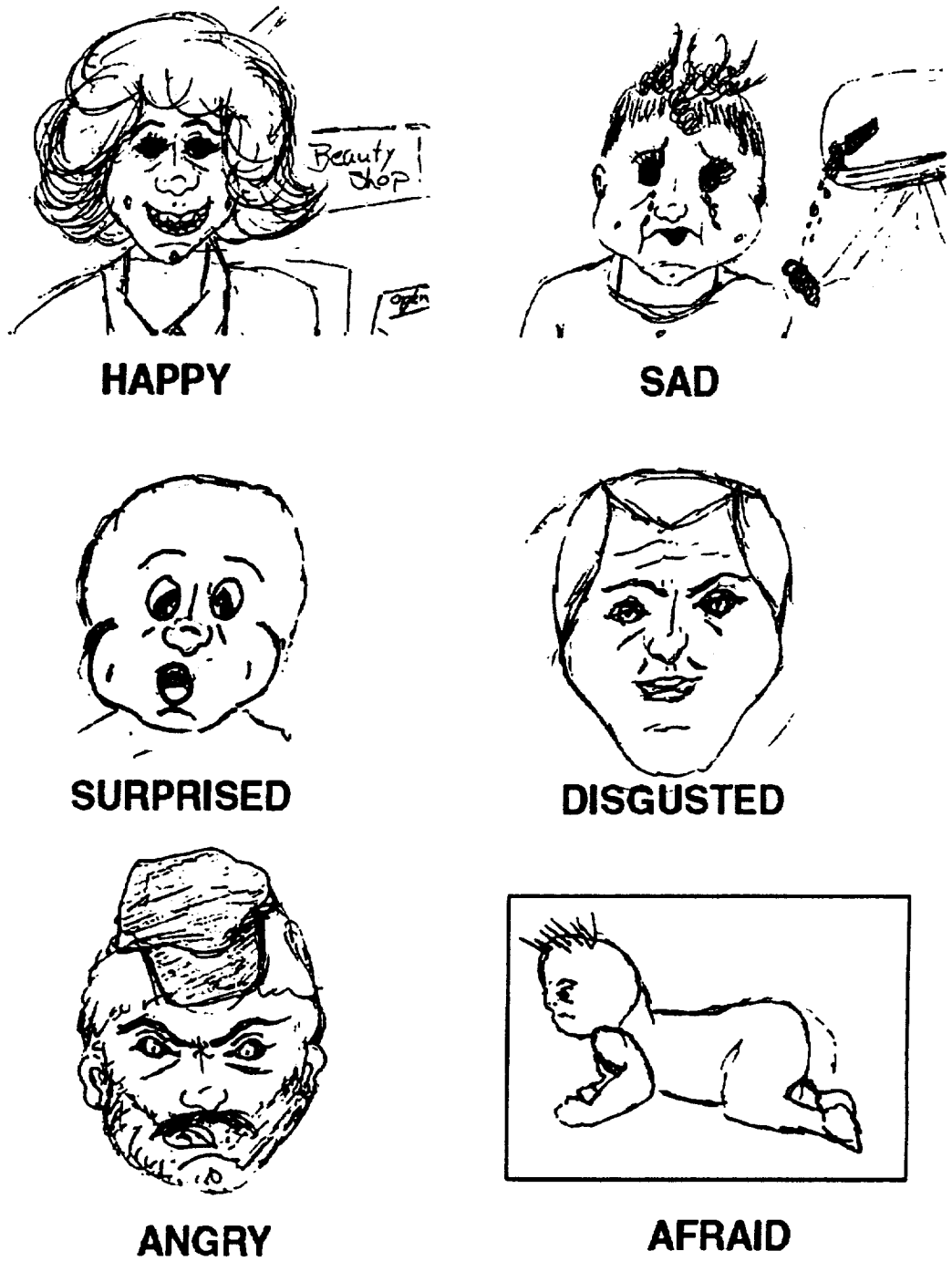
Human Case Study of Fear After Amygdala Lesions

A glimpse into the nature of these psychological processes comes from studies of human patients who have suffered brain damage. Adolphs, Tranel, Damasio, and Damasio (1995), for example, describe the case of a woman who lost most of her right and left amygdala owing to a genetic disease that induced calcification of brain tissue. When asked to identify the emotional expression of facial portraits, she failed to describe fearful ones as "afraid." She successfully identified happy, angry, disgusted, sad, and, to some extent, surprised facial expressions. Expressions that ordinary control subjects considered to be fearful, this woman instead described as surprised or angry. The woman never assigned high ratings of the adjective "afraid" to any faces. (Although she gave her highest ratings in this category to appropriately fearful ones, her fear ratings were far below normal.)

Further insight into her mental condition was provided by her facility at drawing cartoon characters. Her renderings of happy, sad, surprised, disgusted, and angry faces were quite lifelike—so good that most readers could probably correctly assign the intended state to each cartoon (figure 27.12). When asked to draw someone who was afraid, by contrast, she at first failed to comply. When the request was repeated, she drew a figure that conveyed almost no sense at all of a fearful state: a childlike figure crawling on all fours, whose only resemblance to fear was that the figure's hair was stiff and standing on end. Asked about her relatively poor drawing, she replied that "***she did not know what an afraid face would look like***" (5887, original italics).

It seems noteworthy that the artist did not say

FIGURE 27.12 Depiction of Emotion After Amygdala Damage



Source: Reproduced with permission from Adolphs, Tranel, Damasio, and Damasio (1995, 5888).
Notes: Emotional expressions drawn by a woman who had bilateral amygdala lesions due to disease. Note that all emotions are fairly well depicted except for "afraid."

that she did not know what fear was. Fear—as a constructed category of emotion—still made enough sense to her to talk about it. Neither did she claim that she had drawn a good rendering of fear. She was not oblivious to her deficit. Clearly, this deficit was in some way especially related to the recognition and expression of fear. But the possibility that the patient "does not experience fear in a normal way," as the authors suggest (5887), may not be equivalent to a categorical lack of fear. It is certainly not a lack of the construct of fear as a global category of emotional appraisal. Rather, her deficit applies specifically to the interface between fear and the outside world: emotional recognition of the particular events that others would perceive as frightening, expression of fear through facial and other affective reactions, and recognition of fearful affective displays by others.

A related case is that of a woman who lost her amygdala on both sides owing to surgery for epilepsy (Scott et al. 1997). Afterward, she was markedly impaired at recognizing vocal emotion in speech that was deliberately intended to sound fearful, angry, happy, sad, or disgusted. The patient was especially poor at recognizing fearful or angry tones of voice, though she was marginally impaired also at recognizing happy and sad intonations. When asked to identify nonspeech vocal expressions of emotion, such as a fearful scream, a happy laugh, sad sobbing, disgusted retching, or an angry growl, she was often wrong only for the sounds intended to convey fear or anger. However, the patient's difficulty in abstracting information from the sound of a voice was not limited strictly to emotion. She also had trouble recognizing the identity of a voice, indicating an impairment of perceptual recognition that extended beyond emotion. Thus it would be difficult to conclude that her deficit was specific to fear, or even simply to emotion.

Summary

Fearful and anxious emotional reactions to situations unquestionably involve amygdala function. But it appears that the amygdala mediates neither "fear" nor "anxiety" as pure psychological categories. Some aspects of fear and anxiety persist after amygdala damage. Further, some of the fear-related deficits seem to be more closely linked to specific perceptual or representational aspects than to loss of fear as an emotional category.

Beyond Fear: Participation of a Related Core Process in Positive Emotion

Just as fear may not be totally lost as a unitary emotional category after brain damage, but rather distorted, other emotions are often similarly distorted. Amygdala lesions alter many reactions to emotionally *positive* events. Indeed, the original Kluver-Bucy syndrome included "hyper-orality" (attempts to eat atypical objects) and "hypersexuality" (attempts to mate with atypical targets) among the consequences of losing the amygdala and other temporal lobe structures in monkeys (Kluver and Bucy 1939). Animals after amygdala damage sometimes err in the opposite direction. For example, they fail to engage in sexually motivated tasks that an intact rat would perform (Everitt 1990). They also fail to select particular nutrients to eat that would be good for them under particular circumstances, such as salt when they are deficient in body sodium (Schulkin, Marini, and Epstein 1989). Ordinary rats will avidly drink a very salty solution after they are depleted of physiological sodium by a drug. (A similar salt appetite can occur in humans who have lost excessive amounts of sodium in perspiration or who have been on a completely salt-free diet for a prolonged time) (Schulkin 1991). Lesions of the central amygdala block the willingness of rats to ingest salt when they are sodium-deficient (Seeley et al. 1993). They fail to consume salt even though they still seek food and water, and even though they would "like" the taste of salt (they would emit enhanced hedonic reactions) if they did taste it (Galaverna et al. 1993). These observations suggest that even relatively "innate" emotional reactions to positive "key stimuli," such as the taste of salt during sodium appetite, are vulnerable to the same types of amygdala damage that dissociate fear.

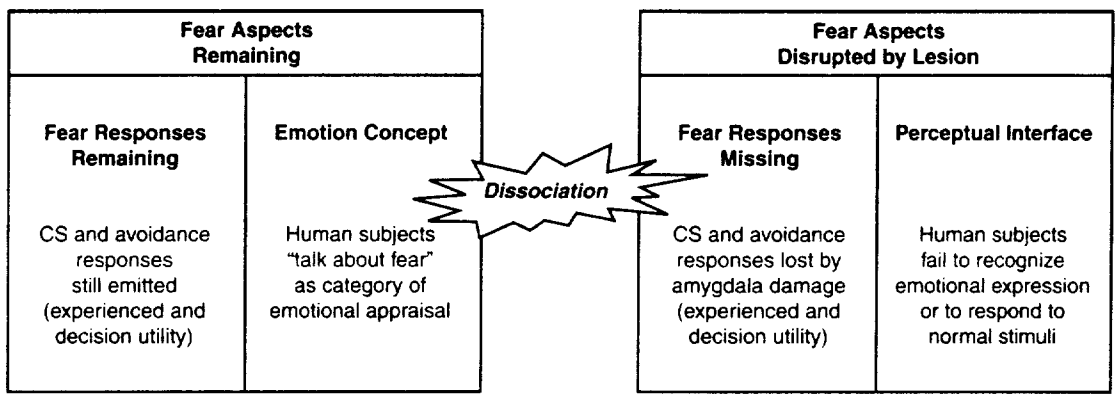
Emotional responses that require explicit ***learning*** appear even more vulnerable to amygdala damage. For example, lateral amygdala lesions prevent a rat from learning to return to a place where it has obtained drug reward before (Hiroi and White 1991). In a "conditioned reinforcement" task, lesions of the basolateral amygdala abolish the value of learned rewards. Ordinary rats will work for conditioned stimulus (a light or sound) that has been paired either with food or with a sexual partner, but amygdala lesions eliminate such conditioned reinforcement (Everitt 1990; Everitt and Robbins 1992). The rats will still work for an unconditioned reward (food or sex itself), but no longer for a learned reward. One

interpretation of this has been that amygdala lesions disrupt an aspect of *reward learning* (Everitt and Robbins 1992). Reward learning, coupled with fear learning, could be expanded to a category of general emotional learning (Davidson and Sutton 1995).

Although emotional learning as a psychological category does seem to bear a special relationship to this brain system, it still does not fit perfectly. The thrust of my argument is that no familiar category will fit. The human failure to recognize facial expression, voice intonation, or voice identity (but retaining the capacity to discuss emotions, which is surely as dependent on learning as the impaired functions are), or the rat failure to drink salt while in a state of sodium depletion (but retaining normal food and water intake and even salt alliesthesia), will not fit without force into the category of emotional learning. Of course, the amygdala nuclei no doubt mediate many functions, and some consequences could be explained by loss of other functions in addition to emotional learning. But most troublesome for an emotional learning hypothesis are the observations that amygdala lesions never really eliminate the capacity for learned emotion at all. Some evidence for this has already been discussed. In addition, even in experiments in which some aspects of emotional learning are lost, as when conditioned reward is disrupted, animals may still show other aspects of having learned an association to the reward. For example, when a sound that has been paired with a food reward is presented to rats that have basolateral amygdala lesions, the rats immediately run to their empty food dish, as normal rats would (Everitt and Robbins 1992; Hatfield et al. 1996). Both the sound and the food dish are merely conditioned stimuli. The rat's response to them in the absence of real food suggests that learned stimuli still trigger a reward association of some sort.

One more traditional psychological category, namely, attention, has been suggested as an amygdala function to explain why animals might retain reward learning yet not show all appropriate behavior to conditioned stimuli (Gallagher and Holland 1994). This idea is based on the finding that central nucleus lesions of the amygdala block orienting or attending reactions to conditioned stimuli for reward, while preserving other reward-related learning. Ordinary rats will turn to look at or listen to a conditioned stimulus light or sound that has been paired with reward, but after being given central amygdala lesions, rats do not (Gallagher, Graham, and Holland 1990; Hatfield et al. 1996). The same lesion disrupts "unblocking," the normal propensity to increase learning about a conditioned stimulus whose predictive value is suddenly enhanced by a change in its paired unconditioned stimulus (Holland and Gallagher 1993). Such failure has been interpreted as a failure to attend to stimulus relations. However, it is difficult to account for many phenomena with an attention deficit hypothesis. For example, although the lesion disrupts unblocking in classical conditioning, it does not disrupt blocking (failing to learn about a conditioned stimulus if the un-

FIGURE 27.13 Dissociation of Fear by Amygdala Damage



Notes: Dissociation of fear by amygdala damage. Many fearful behavioral and autonomic responses of animals are disrupted, but others persist (as may be true for positive emotional responses too). Similarly, humans fail to recognize fearful stimuli or expressions after damage, but can still talk coherently about fear as an emotional category.

conditioned stimulus is already predicted) (Holland and Gallagher 1993). It is difficult to devise a plausible account on that basis for why unblocking but not blocking should be disrupted by an attention deficit. Holland and Gallagher (1993) note that blocking prevents the growth of an association whereas unblocking produces it, but even this difference does not translate immediately into an explanation.

Alternatives to Traditional Psychological Categories

The evidence reviewed earlier suggests that the amygdala nuclei may not, after all, mediate "fear" or "reward" or "emotion" or "emotional learning" or "attention" as distinct psychological categories, or any group of these as traditional psychological categories. This may tell us something important. We need to devise new psychological core processes that will better account for the phenomena. Perhaps the general conclusion to be taken from this discussion is that the real core processes of emotion, revealed by biopsychological studies of brain-behavior relations, simply will not fit into traditional psychological categories. Fortunately, again, the phenomena give clues about the properties of the real core processes.

Overlap of Fear and Anxiety Core Process with "Wanting"

A possibility that seems to me to merit further examination is that some of the emotional deficits related to amygdala damage may reflect an interaction with the incentive salience attribution system of "wanting" discussed earlier. The amygdala receives ascending dopamine projections, and the behavioral effects of amygdala lesions are modulated by dopamine manipulations of the nucleus accumbens. For example, some of the reward learning deficits produced by amygdala lesions are reversed by microinjection of amphetamine into the nucleus accumbens (Cador et al. 1991). Could some of the consequences of amygdala lesions involve changes in the attribution of incentive salience to stimuli? Not in the same way as dopamine lesions do. It is clear that, unlike the dopamine/accumbens system, the amygdala is not needed for the *generation* of incentive salience attributions. Amygdala lesions do not produce global deficits of "wanting" that follow suppression of dopamine neurotransmission. Both humans and rats continue to eat and drink and to seek out many incentives after amygdala damage, seeking some perhaps more than they ordinarily would. But the amygdala might still participate in the *targeted focusing* of incentive salience attributions on particular stimuli rather than on others. Loss of such a function would produce behavioral deficits that combine emotional, motivational, associative learning, and perceptual aspects, as amygdala lesions appear to do. For example, rats that fail to consume salt when they have a sodium deficiency, but eat and drink normally otherwise, might fail specifically to focus incentive salience on salt-related stimuli under appropriate conditions.

"Wanting" Versus "Fear"

Incentive salience by its very name entails a positive state. How could an incentive process be reconciled with an amygdala or dopamine/accumbens role in fear? Despite their differences, there are points of overlap between the core processes of "wanting" and "fear." From the psychological point of view, both are a form of decision utility: they elicit a decision to engage or avoid based on an event's potential for pleasure or pain. From the neural point of view, brain manipulations of the dopamine/accumbens system that alter "wanting" for positive incentives can also alter fearful reactions or avoidance of danger (Robinson and Berridge 1993; Salamone 1994), just as manipulations of the amygdala do.

That positive incentives and negative fear may tap into similar psychological core processes mediated by the same brain systems poses a challenge for the construction of biopsychological hypotheses. What sort of core process could be relevant to both desire and dread? There are several possibilities that could join "wanting" and "fear" together in the same dopamine/amygdala system (Robinson and Berridge 1993). In some cases, a positive incentive could masquerade as an aversive task: individuals in a fearful situation may "want" to escape to safety. But even in a situation in which the motivation is entirely aversive, a process related to incentive salience could be at work. For example, different dopamine/accumbens/amygdala subsystems might mediate "wanting" and "fear" as distinct processes: "incentive salience" versus "aversive salience." Alternatively, the two psychological functions might be combined together into a single "generic" motivational salience attributed by the dopamine/accumbens/amygdala system, while positive/negative valence could be set by other factors. For example, deter-

mination of whether motivational salience would be rewarding or frightening could arise either from within the dopamine/accumbens/amygdala system itself, such as from the intensity of dopamine activation (for example, moderate levels of activation causing attraction, but very high levels becoming frightening), or from outside by the coactivation of separate neural systems. This possibility converges with Kagan's and Schulkin's view of inhibited children and agitated depressed patients as hyper-responsive to events of many kinds owing to a putatively hyperactive amygdala system (Kagan and Snidman 1991; Schulkin 1994).

My speculation concerning potential interaction in positive and negative emotion is not intended as a finished hypothesis but rather is meant simply as a starting point to highlight the kinds of issues involved. The real goal is to identify adequate core psychological processes that can legitimately be mapped onto emotional phenomena and the consequences of brain manipulations.

Summary

Amygdala lesions dissociate aspects of positive emotion in the same way that they dissociate aspects of fear and anxiety, perhaps indicating disruption of a shared underlying core process. In each case, some aspects of the emotion are distorted while others are preserved. Just as the manipulations of brain dopamine systems unexpectedly dissociate emotional reward into "liking" and "wanting" categories, the consequences of amygdala damage seem to cross traditional psychological boundaries of emotion, motivation, learning, perception, and attention, while not entirely disrupting any of the psychological subcategories that have so far been proposed. Reconsideration of our categories of psychological organization may help match the phenomena to their causes.

Conclusion

The dissociations of emotional processes discussed in this chapter illustrate the composite nature of seemingly simple positive and negative emotions. The fracture lines that dissociate core psychological components are not apparent in everyday life, and the nature of those core components is not evident to conscious awareness. Information about psychological core processes comes from studies of unconscious emotional reactions and studies of brain-behavior relations. These suggest that even the simplest emotional experience can be dissociated into components. Some components are essentially unconscious: core processes of "emotion" that may ordinarily help cause subjective emotional feelings but are not themselves accessible to awareness. For positive emotional states, these seem to include separable core processes of "wanting" and of "liking," which are mediated by different brain systems. Particular core processes of positive emotion, such as the incentive salience attribution process denoted by "wanting," may also be components of "fear" and "anxiety."

Dissociation between conscious emotional feelings and unconscious core processes of emotion becomes obvious only under unusual circumstances. It requires hypnosis, addiction, tachistoscopic presentation, or brain manipulations in order to provide compelling demonstrations. The reason for this is that our conscious experience of an emotion and the core processes that constitute it are generally integrated in ordinary life. Experienced utility, decision utility, and predicted/remembered utility do typically covary together. Similarly, hedonic and action-inducing properties of pleasure or pain (instant experienced utility, as conceived by Kahneman [this volume] and Shizgal [this volume]) typically cohere. Most rewards that are liked are also wanted. Most pains are feared. It has been adaptive for these components to work closely together to achieve life goals. But their identity as components allows them to dissociate under some conditions. For psychologists who wish to understand the process and structure underlying hedonic life quality, the dissociation of core components that ***can*** occur is as important as the association that usually ***does***.

More and more we are learning that emotions, like other psychological processes, are actually made up of separate components, many of which are excluded from conscious experience. The perceived unity of an emotional event arises from integration by the systems that generate conscious experience, whose representation of core processes involves distortion and omission. It seems clear that our understanding will be advanced when we parse psychological phenomena into meaningful categories that reflect the nature of evolved components, and when we learn the rules that relate components to each other and to conscious experience.

I thank Phoebe Ellsworth, Daniel Kahneman, Stephen Maren, Susana Peciña, Jay Schulkin, Peter Shizgal, Terry Robinson, Jacob Steiner, and Elliot

Valenstein for their helpful comments on earlier versions of this chapter. I also thank Ralph Adolphs and his colleagues, and the *Journal of Neuroscience*, for permission to reproduce the drawings by their patient.

Notes

1. Instant experienced utility, as it is currently conceived by Kahneman and his colleagues, is slightly more complicated than I have indicated here (Kahneman, this volume; Kahneman, Wakker, and Sarin 1997). Instant experienced utility for Kahneman also includes an action tendency, manifest as action to acquire an immediately available incentive and to resist interruptions of that goal-directed action, in addition to the hedonic experience of the incentive. Decision utility, by contrast, involves a decision to act or choose between more distant incentives, not immediately present. For instant experienced utility, the two connotations of resistance to interruption and hedonic experience are presumed to be different manifestations of the same underlying process. This dual-property sense of instant utility has been adopted for biopsychological analyses of brain stimulation reward by Shizgal (Shizgal 1997; Shizgal, this volume), who views the resistance to interruption by a self-administering rat as tantamount to the hedonic intensity of a brain stimulation reward.

   I focus only on the hedonic value component of instant experienced utility, not on the connotation of action tendency or resistance to interruption included by Kahneman and by Shizgal, because evidence shows the hedonic evaluation of an incentive to be dissociable by brain manipulations from the persistence of the incentive-directed action. These dissociations suggest that the hedonic experience connotation of instant experienced utility may not reflect the same process as the action persistence connotation. In that case, the hedonic evaluation of experienced utility corresponds closely to what I call "liking," whereas the action persistence included by Kahneman and by Shizgal corresponds more closely to "wanting," sharing some properties in common with decision utility.

   To avoid confusion, I use "experienced utility" only to refer to the hedonic evaluation of an incentive ("liking"), and use "decision utility" to refer to decisions to acquire, choose, and persistently pursue the incentive ("wanting"). This is meant to allow transition between the two frameworks. While respecting the definition of these utility terms as given by their authors, it seems reasonable to suppose that they will continue to evolve as concepts in psychology generally do.
2. Although there may be cases in which choice between future outcomes (decision utility) diverges from predictions of future hedonic value (expected utility). For example, people may choose a variety of foods for future consumption that are different from the choices they would make when the occasions arrived; the choices can be made to converge more closely, however, if they are asked to predict explicitly their future likings before making their choice (Kahneman 1994; Simonson 1990). Such cases of dissociation between choice and predictions perhaps occur only when individuals choose without bothering to think through a prediction of future value, or when their choices for the future are guided by criteria that go beyond hedonic maximization (Kahneman, this volume). Both situations differ from the type of dissociation discussed here, in which generated forms of hedonic utility (experienced, decision, expected/remembered) have discrepant values for the same event.

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