REWARD LEARNING: Reinforcement, Incentives, and Expectations

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How rewards are learned, and how they guide behavior are questions that have occupied psychology since its first days as an experimental science. Many answers have been suggested during the past 100 years. The discussion continues today because the answer to these questions is more complex than it first appears, and also because there may be more than one correct answer.

I. Reinforcement Theories

Certain ideas in intellectual history have emerged occasionally on the scene in so clear and compelling a form that they seem as though they *must* be true (even if they are false). Self-evident to those who hold them, they have a tenacious foothold on intellectual life that is impervious to mere evidence against them. They may be wrong, and may be repeatedly shown to be wrong. Yet they will not die. Among this class of ideas is *reinforcement:* the idea that reward learning consists primarily of a process by which behavior is directly strengthened or weakened by the consequence that follows it.

Reinforcement is alive and strong as a concept in psychology. Covering the last 5 years of the 20th century, the PsycINFO database listed for each year over 500 published articles that include *reinforcement* as a key word.

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A. Descriptive (Atheoretical) Forms of the Reinforcement Concept: Skinner's Radical Behaviorism

The radical behaviorist sense of reinforcement was the simplest, though not chronologically the first version of the reinforcement concept. Reinforcement in a behaviorist sense, as used by Skinner, was merely a description of the relation between a change in a behavior caused by following it with a stimulus such as food. No explanation was offered. As Skinner put it,

> The Law of Effect is no theory. It simply specifies a procedure for altering the probability of a chosen response. But when we try to say *why* [italics in original] reinforcement has this effect, theories arise. Learning is said to take place because the reinforcement is pleasant, satisfying, tension reducing, and so on. (Skinner, 1950, p. 78).

Skinner took a dim view of theoretical attempts to explain the "why" of reinforcement: "Theories are fun. But it is possible that the most rapid progress toward an understanding of learning may be made by research which is not designed to test theories" (Skinner, 1950, p. 99).

In the first half of the 20th century, such an explicitly antitheoretical account had two forms of appeal. First, it offered a purely objective alternative way of talking about behavior that could be claimed to escape the theoretical disagreements of earlier mentalistic psychology. Second, at the time Skinner wrote it seemed to many possible to believe that his hope for "the most rapid progress" might actually be true. One could be impressed in 1938, when Skinner's (1938) first major book appeared, by the clarity of the early studies of operant performance curves, of the smooth versus scalloped differences in their shapes produced by fixed ratio versus fixed interval reinforcement schedules, and so on. Such precision in behavioral description had never before been achieved, let alone brought under experimental control. One could still hope then that the second half of the 20th century would be filled with many powerful demonstrations of new and useful behaviorist principles for predicting and controlling behavior.

But the Skinnerian hope turned out to be unfulfilled. Very few new principles of use were produced by behaviorist studies over the next 50 years. However objective a behaviorist description might be, it generated very little for predicting or control of behavior beyond the original law of effect, and a few straightforward applications of that law. And as Skinner himself acknowledged, the radical behaviorist concept of reinforcement offered nothing at all for understanding the "why" of reward's effect on behavior. It had no theory and no explanatory power.

Today there remain a few calls for a return to atheoretical behaviorism (Staddon, 1999; Uttal, 2000). They reflect an almost aesthetic preference held by these authors for a purely atheoretical behavioral description, which

avoids any inference about mediating process that could turn out to be wrong, even at the cost of explanatory power. But contemporary behaviorists draw mostly on the old evidence for support, and simply reassert the original Skinnerian faith that good progress might be made in an atheoretical fashion. That faith, once plausible, now has nearly died away from psychology for lack of fruition. Most important to the demise of behaviorism has been a consensus in psychology that the behaviorist fear of theory and psychological constructs was misplaced. It has proven eminently possible to test theories grounded in cognitive, associative, and motivational hypotheses over the past 50 years. Many such theoretical constructs have been disproven, others confirmed, and still others modified on the basis of experimental results. Contemporary psychology and neuroscience of reward learning are both concerned almost entirely with accounts of why reward works. We will therefore turn to theoretical answers to the why of reward and reinforcement.

B. Theoretical Versions of Reinforcement Concept: S–R Habits, Hedonic Reinforcers, and Drive Reduction

1. Thorndike's Original Law of Effect: Satisfiers and Annoyers

The Law of Effect was formulated first by Thorndike (Thorndike, 1898) in remarkably hedonic-sounding language, that is in terms of *satisfaction* or *annoyance.* Thorndike asked regarding animals, What do they *feel?,* a question familiar to today's psychologists of emotion and to affective neuroscience. In Thorndike's words, the initial goal of his experimental program with animals was to "not only tell more accurately *what they do* [italics in original], and give the much-needed information *how they do it,* [italics in original] but also inform us *what they feel* [italics in original] while they act,"(Thorndike, 1998, p. 1126). Regarding such feelings, Thorndike's law of effect stated originally that behavior that was followed by *satisfying* effects would be repeated more often in the future (Galef, 1998). Behavior followed by *annoying* effects, conversely, would be less likely to be repeated. Satisfaction and annoyance are essentially hedonic terms. They specify feelings or affective states of pleasure and displeasure. This use of a hedonic sense of the reinforcement concept soon diminished only to grow again over the intervening century.

Later formulations of the law of effect by Thorndike dropped the explicit postulation of affective states and adopted the more behaviorist formulation that led eventually to Skinner's position (Moss & Thorndike, 1934). In later versions, positive reinforcement became whatever property of a stimulus increased the frequency of the behavior it was contingent upon. No substitute for hedonic satisfaction, however, was offered to replace it until the emergence of drive reduction theories. Hedonic pleasure has always re-

mained the primary candidate, at least implicitly, for a specific psychological state to mediate reinforcement, when such a state is considered at all. As indicated by Skinner's quote above, this is often true even for behaviorists. But as behaviorists no longer tried explicitly to identify the nature of positive reinforcement states, processes of a different nature for "stamping in" behavior had to come from other sources, such as from associationist accounts of reinforcement.

2. *Stimulus–Response Association: Reinforcement by Strengthening of S-R Habits*

If reinforcement of a behavioral response means any specific psychological process at all, aside from hedonic pleasure, it must mean strengthening of a stimulus–response (S-R) habit. The idea that the impact of a reward on behavior might be mediated by the strength of a specific association was a stark alternative to hedonic accounts. An S-R association is essentially a *learning* process, not a motivational, emotional, or affectivexs one. Associationist behaviorism grew with the later works of Thorndike, John Watson (1913), and with subsequent generations of associationist behaviorist psychologists who evolved alongside the radical behaviorists, and who gave rise to Thorndike-based concepts of reinforcement that can be found still today in psychology and behavioral neuroscience (McFarland & Ettenberg, 1998; White, 1989).

Associationist behaviorists were not radical or atheoretical behaviorists as was Skinner, because they postulated specific psychological processes in order to explain their observations. In this case, the specific process was the strength of the learned association between two particular events that had been paired together in an individual's history. But they were behaviorists still because they restricted themselves to a few relationships, S-R associations, stimulus–stimulus (S-S) associations, and little else, which dealt entirely with events that could be observed objectively: physical stimuli, behavioral responses, and the measurable relation among them.

Associationist accounts of reinforcement allowed S-R theorists to drop hedonic satisfaction from the arena of postulated psychological events, yet still have a complete explanation for why reinforcers strengthened behavior. The explanation was simply that the occurrence of a reinforcer such as food could cause a direct increase in the strength of S-R association that preceded it. Whatever behavior had just been emitted was the strengthened response. Whatever stimulus had just been present when that behavior occurred was the stimulus that now became associated with the response. The strengthening of the association created something new: a habit. It imbued the stimulus with the ability to elicit again the response, in a direct,

essentially reflexive fashion. When presented a second time, the stimulus was now more likely to elicit the response. If reinforced a second time, the habit would be strengthened further. The apparent simplicity of this explanation of reinforcement was stunning. It allowed one to explain—not merely describe as Skinner could—why a behavior became more and more frequent and persistent when rewards were given contingent upon it. And it allowed explanation without ever for a moment requiring one to invoke more difficult or complex psychological processes.

The explanation was simple. No "expectation" of reward was required. The subject didn't have to expect the reward or anticipate or represent its occurrence in any fashion at all. The subject simply responded to the eliciting stimulus with the associated habit. No hedonic impact, no affective or emotional state of any sort, was posited. The subject didn't need to have a satisfaction or any other affective feeling at all. Instead the strengthening of association could be done directly. A three-neuron circuit would suffice. Simply let a neuron representing "food occurred" potentiate the connection between a neuron carrying "stimulus" to a neuron generating "response." Complicate this circuit further with millions of additional neurons, as real-life brains certainly do, there still appeared no need for more than the three basic functions. For example, many of the basic features of stimulus-response reinforcement appear to be captured by an extremely basic neural arc circuit in the spinal cord, which is capable of elementary operant conditioning of a motor stretch reflex (Wolpaw, 1997).

One might imagine that the S-R habit explanation of reward learning described above is the sort of explanation that only a behaviorist could love. It seems to leave out every sort of psychological process that we experience ourselves regarding reward and substitutes rigid and reflexive S-R habits that are entirely alien to the notion of rewards. But there was actually *evidence* to support the S-R reinforcement theory. A vivid example is the early "kerplunk" experiment by the archetype S-R theorist John Watson (Carr & Watson, 1908). In this experiment, rats were trained to run in a maze for a food reward. As the rats learned the maze very well, they began to run very quickly through each length and turn. They became routine and accomplished performers on the task. Then, one day, the length of the runway was changed: either lengthened or shortened. The question was, what would the rats do on their first trial in the longer or shorter runway? The answer was, if the new length were longer, the rats ran smoothly and quickly for their customary distance—as far as they used to in the old runway. Then they paused—even though they had not yet reached the end. If they had reached the spot where food would have been before, they stopped and sniffed at that spot, even if that meant ignoring food further away at the new end. It was as though the rats had learned simply

to run through a motor habit: a certain number of steps, performed almost reflexively once triggered at the beginning. Conversely, what if the runway were suddenly shortened rather than lengthened, so that the food and wall were moved closer to the starting place? On their first trial in the shorter runway, the rats ran full speed—right over the nearer food—and right into the wall at the end, ignoring the clear warning sight of it in front of their faces—kerplunk! Again, it appears that the rats' well-learned performance had become a routine motor habit, a series of running steps linked together as a pure S-R routine.

C. Hullian S-R Reinforcement and Drive Reduction

Findings such as the "kerplunk" phenomenon gave credibility to S-R interpretations that rewards worked simply by strengthening the tendency to emit a particular habitual motor action when in the presence of a particular stimulus. But it was not immediately apparent why different rewards (food, water, drug, or access to a sex partner, social partner, etc.) all should strengthen habits. What did they have in common? Nor was it clear how an individual could learn long sequences of responses, which were unlikely to originally be emitted by chance, and so could not easily be strengthened all at once. Nor again was it clear how individuals learned different responses to obtain different goals. How is it that one learned to go to a drinking fountain for a cold drink when thirsty, but to a food dish when hungry? If these were simply habits, why did't they get mixed up? Why didn't one equally drink when hungry or eat when thirsty?

Hullian learning theory, and similar formulations by other psychologists of the time, represented a principled attempt to explain such complexities within the constraints of an S-R framework (Hull, 1943). First of all, Hull specified the hypothetical property supposed to be shared by all reinforcers that gave them the ability to reinforce behavior: they all *reduced drive.* When we are hungry, Hull argued, we are in a state of drive that activates us. Food, when eaten, reduces that hunger drive. It was the reduction of drive, Hull argued, that was the source of reinforcement. Drive reduction, in essence, was reward. Similarly, when we have been a long time without a drink, or if we are out in the hot sun, thirst activates drive. A sip of water reduces the drive of thirst, and so becomes a reinforcer. Many of the physical stimuli that serve as rewards (food, water, drugs for individuals in drug withdrawal) can be conceived as drive reducers. Social rewards are perhaps a bit more tricky to construe as reducers of drive, but if one grants the possibility of a sexual drive, then the act of engaging in sex can be viewed as possibly reducing that drive. Similarly, if a mother has a maternal drive, then presentation of her infant might reduce that drive by providing

an outlet. If individuals have a social drive to mingle and interact with others, then access to social partners or social approval might in turn reduce the relevant drives. If these were all allowed to be considered as drives, then drive reduction was at least in principle a parsimonious way to account for a wide variety of reinforcers. Behavior was finally a simple consequence of the level of drive at the moment and the ability of stimuli present to trigger S-R habits, which had previously been created by drive reduction. This simple relationship could be expressed as an equation:

$$_{S}E_{R} = {}_{S}H_{R} \times D,$$

where $_{S}E_{R}$ was the actual strength of a behavioral response in the presence of a particular stimulus, $_{S}H_{R}$ was the associative strength of the learned habit within the individual, and D was the level of drive at the moment. Hull added other factors, too, but this equation captures the essence of his model.

Drive reduction, if it was the mechanism for S-R reinforcement, also provided an associationist way to explain why we seek food when hungry but water when thirsty. The answer was the sensations that accompany hunger or thirst were themselves part of the stimulus in a particular S-R link. If a rat felt hungry and perceived a food lever in an operant chamber, then pressing the bar in the presence of that compound stimulus composed of the see-lever-and-feel-hungry combination would produce food, and result in reduction of the drive. But if the rat felt thirsty instead of hungry and saw the same food lever, then the food which a press would produce would *fail* to reduce its thirst drive. Pressing the food lever after seeing the bar while feeling a parched throat, and so on, would be a habit that would not get strengthened. Thus hunger and thirst were not merely drives, but were also cue sensations or discriminative stimuli in themselves, which could combine with drives to determine whether a habit would be triggered. It was the entire stimulus combination that triggered the S-R habit, according to Hullian theory. Specific drives acted as discriminative stimuli, as well as to energize behavior, which served to gate the ability of external stimuli to trigger habits that were related to a particular drive but not to others.

Finally, the Hullian account of reward learning offered one more mechanism to help explain how complex *chains* of responses become learned. The mechanism was Pavlovian associations among pairs of stimuli, and the explanation was that classical conditioning allowed stimuli encountered when distant from the goal to trigger incremental or partial responses. After all, most behavior is not a single act and therefore cannot be reinforced as a simple habit. Behavior is a stream of acts. Complex habits grow out of smaller ones. Even a simple rat that has learned to walk down a T-maze

runway and turn the corner to find food must learn a sequence of responses, not just one. Hullian learning theory suggested that rats did learn a sequence: specifically they learned the sequence backwards, starting with what to do at the food dish, and building gradually back to the beginning of the maze.

For Hull, a hungry rat on its first time in a T-maze must wander aimlessly. It does not know what to do. But eventually, if it wanders enough, it will find the food dish and food within it. If it eats the food, it will have experienced an S-S pairing: the sight of the food dish, followed by the stimulus of food itself. In the parlance of Pavlovian conditioning, the sight of the food dish is a conditioned stimulus (CS). The food itself is an unconditioned stimulus (UCS). So there has been a CS-UCS pairing, and an association between the two stimuli is formed. The next time the rat is put in the maze, it still does not know what to do. But if it wanders down to the end of the runway, it may see the food dish around the corner. Originally the food dish was meaningless: just another sight to see in the maze. But now the sight of the dish is a Pavlovian CS for food, which can elicit at least a weak food-appropriate response. Normally the rat, if it saw real food, would walk toward the food and then eat it. It cannot eat the food dish, especially not from a distance, but it can walk toward the dish just as it would walk toward the food itself. And this is what the rat should do by Hullian theory: emit to the CS whatever fractions it can of the response that it would ordinarily emit to real food. Such *fractional responses* will most often include approach, for various rewards, and so the rat directly approaches the dish. Once it reaches the dish, it can eat the real food, and when it does that, the entire preceding set of responses will be reinforced by drive reduction. The rat has just learned to walk toward the dish as soon as it is seen.

To modern psychologists, the Hullian account of reward learning seems strained, especially given the plausibility of cognitive alternatives. For example, the learning of a sequential maze could be explained by positing that the rat has formed a cognitive representation of the spatial location of food, of its own spatial location, and of the contours of the maze. An alternative explanation is that the rat finds its way to food by following this cognitive map. Several authors have provided excellent discussions of the relation between such cognitive processes and the simpler S-R processes conceived by Hullian learning theory and of evidence relevant to both (Dickinson, 1989; Dickinson & Balleine, 1994; Holland, 1993; Toates, 1986, 1998). My focus here is specifically on the *reward* learning aspect of Hullian theory, that is, the strengthening of associations by *drive reduction,* rather than on the cognitive versus S-R contents of what is learned. I thus wish to restrict our present consideration to the reward concepts of reinforcement and drive reduction versus alternative concepts of reward. Of what

use are they in understanding reward learning, and what evidence has led to the replacement of reinforcement concepts with new concepts of reward? That will be the topic for the rest of this chapter.

D. Role of Drive Reduction Theory in Human Motivation and Social Psychology

The notion that rewards work via mechanisms of reinforcement and that reinforcement is gated by drives proved to have long-lasting consequences in psychology. Reverberations can be found in fields far removed from S-R behaviorism, such as in human social and motivational psychology (McClelland, 1987). Although no longer conceived in terms of S-R habits, of course, reward is sometimes still discussed as working via reinforcement states or goal states that follow performance of the motivated behavior. For example, in a major text on human motivation McClelland (1987) adopted an almost Thorndikian notion of hedonic reinforcement (though he allowed the reinforced response to be as abstract as power seeking) when he wrote,

> This method also led to a working definition of a motive as a recurrent concern for a goal state based on a natural incentive—a concern that energizes, orients, and selects behavior. . . . The fact that the concern is about a goal state has the important implication that the means of getting to the goal is not part of the definition of a motive. The goal state may be defined as the outcome of certain acts. (McClelland, 1987, pp. 590, 591)

McClelland went further in conceiving the reinforcement mechanism to be gated by drive and discussed motives, such as the power motive, as though they were a form of drive. For example, regarding the power motive, he speculates,

> It might be inferred that brain norepinephrine represents a physiological reward system . . . specifically for the power motive. To put it in everyday language, suppose brain norepinephrine turnover . . . represents the extent to which power stimuli "turn on: subjects, or make them "feel good". Clearly, the pictures used in this experiment would have more turn-on value for some subjects than others. So these results mean that subjects who are high in Power will learn power-related materials faster if the materials have turn-on value for them than if they do not; in particular, they will learn the power-related materials faster than subjects low in n Power, for whom the stimuli have no turn on value. The situation is analogous to putting hungry rats in a maze with food at the end that they either like or do not like. The rats who are hungry and like the food will learn fastest, and those who are not hungry and dislike the food will learn slowest. (McClelland, 1987, pp. 279–280)

McClelland's description of how the power motive might interact with reward seems similar to reinforcement and drive concepts we have seen.

Although McClelland's view of human motivational psychology is different in many ways from early learning theories, it shares in common at least a reinforcement mechanism. In this case reinforcement acts through a hedonic state a bit like Thorndike's original satisfying state, which is "turned-on" by appropriate outcomes for individuals who are considered high in a power motive drive state.

E. Role of Drive Reduction Theory in Physiological Psychology

The Hullian idea that reward consisted of reducing drives, and that reward learning was the learning of responses that accomplished drive reduction, was tremendously powerful as well in physiological psychology and behavioral neuroscience. The physiological study of motivation became largely the study of stimuli and brain mechanisms of deficit states, such as blood glucose deficits, liver cues, and neuronal energy metabolism for hunger drive and for hormonal and other physiological stimuli for thirst, sex, aggression, and other drives.

This drive reduction orientation began to change slowly with the discovery by Olds and Milner of electrical brain stimulation reward (Olds & Milner, 1954), the phenomenon in which both animals and humans would learn a response and work in order to deliver depolarizing electrical stimulation via implanted electrodes in their lateral hypothalamus, nucleus accumbens, or associated sites. Brain stimulation reward apparently did not need a special drive to be effective, and James Olds wrote of "pleasure centers in the brain" in terms that were not strictly limited to drive reduction. But it was still possible to conceive of brain stimulation reward as reducing natural ongoing drive states. After all, there is always one drive or another that can be reduced.

Given the predominance of drive reduction theories, this interpretation seemed tenable even after it was discovered that brain stimulation also could *evoke motivated behavior* such as eating. According to drive reduction theory, it was at first expected that sites where stimulation would reduce eating (presumedly by reducing drive) would also be sites where stimulation was rewarding (again presumedly by reducing drive). Conversely, this view led investigators to expect, as Olds wrote later, that an "electrical simulation which caused the animal to respond as if it were very hungry might have been a drive-inducing stimulus and might therefore have been expected to have aversive properties" (Olds, 1973, p. 89). The hope that drive reduction would prevail as the explanation of reward was beautifully captured in retrospect by Neal Miller as he described his own attempts to find the neural basis of drive reduction reward, guided by drive reduction theory:

> If I could find an area of the brain where electrical stimulation had the other properties of normal hunger, would sudden termination of that stimulation function as a reward? If I could find such an area, perhaps recording from it would provide a way of measuring hunger which would allow me to see the effects of a small nibble of food that is large enough to serve as a reward but not large enough to produce complete satiation. Would such a nibble produce a prompt, appreciable reduction in hunger, as demanded by the drive-reduction hypothesis? (Miller, 1973, pp. 54–55)

In other words, if one found a "hunger drive" neuron, then one would have found not only hunger but also reward. Increases in the neuron's activity, caused by stimulation, would produce hunger drive and eating behavior. Decreases in the same neuron's activity, caused by a nibble of food, would produce drive reduction and psychological reward. Reductions in such a neuron's activity would be tantamount to reward itself, and would be the neural basis for reward learning regarding food.

Drive reduction was simple and understandable as a theory of reinforcement. Its parsimony made it attractive. Drive reduction *explanations* of reward were seriously damaged, however, on the rocks of unexpected experimental findings that accumulated fast and furious during the late 1960s and early 1970s. A major destructive finding came from observations in brain stimulation reward and electrode-evoked hunger studies themselves. Contrary to expectations, it turned out that the brain sites where stimulation caused eating behavior were almost always the same sites where stimulation was rewarding (Valenstein, 1976; Valenstein, Cox, & Kakolewski, 1970). Rather than be opposite, the two effects were identical, or at least, had identical causes. The realization that drive and reinforcement might reflect the same state, rather than opposites, was a major shock to physiological psychology (and continues to cause aftershocks today). It led in part to the rise of alternative hedonic frameworks of affective neuroscience (for example, Panksepp, 1998).

The reinforcement concept is by no means dead yet in contemporary physiological psychology or behavioral neuroscience. Many behavioral neuroscience investigators still invoke a Thorndikian reinforcement mechanism as a key concept in their accounts of reward learning (Kelley, 1999; McFarland & Ettenberg, 1998; Rolls, 1999; White, 1989). But the *explanation* of reinforcement by *drive reduction* has largely been abandoned, due to the failure of experimental results to confirm the expectations described by Olds and by Miller. Instead, the reinforcement term is used typically today in behavioral neuroscience either in an implicit hedonic sense, or simply as a term to label a behavioral reward effect without requiring any effort by the author to specify a precise mechanism or explanation. Any reward given after successful performance is often called a reinforcer, regardless of the psychological mechanism through which it works to influence behavior.

Despite its longevity, reinforcement may not after all be a very valuable concept for understanding reward. Reinforcement has been largely replaced as an explanatory concept, or at least greatly diminished in importance, by alternative *incentive* concepts of motivation that better explain reward phenomena. Quite a number of motivational theorists in biopsychology have argued that incentive concepts may be much more useful than reinforcement concepts for understanding most reward processes and most motivated behavior (Berridge & Robinson, 1998; Bindra, 1978; Blackburn, Pfaus, & Phillips, 1992; Bolles, 1972; Depue & Collins, 1999; Dickinson & Balleine, 1994; Flaherty, 1996; Panksepp, 1998; Phillips, Blaha, Pfaus, & Blackburn, 1992; Stewart, de Wit, & Eikelboom, 1984; Toates, 1986; Toates, 1994; Tomie, 1996). As they have been quite successful in this argument, in my opinion, I shall focus on incentive processes for the remainder of this chapter.

II. Bases of Modern Incentive Theory: Bolles–Bindra–Toates

A major shift from drive reduction and reinforcement or "stamping in" theories began to occur in the early 1970s, as incentive theories of motivation began to rise. Most important to understanding the biopsychology of reward and motivation may be a particular type of incentive mechanism that I will call the Bolles–Bindra–Toates theory, in recognition of the three psychologists who elaborated its basic tenets, Robert Bolles, Dalbir Bindra, and Frederick Toates (Bindra, 1974, 1978; Bolles, 1972; Toates, 1986, 1994). It is worth mentioning that current biopsychological incentive theory also owes much to the work of many other psychologists of learning and motivation, for example, J. Konorski, P. T. Young, R. L. Solomon, R. A. Rescorla, and A. J. Dickinson (Dickinson & Balleine, 1995; Dickinson et al., in press; Konorski, 1967; Rescorla & Solomon, 1967c; Young, 1966). But I think it fair to say that the specific theory of reward and incentive motivation discussed below was developed chiefly by these three theorists.

A. Bolles: S-S Reward, Not S-R Reinforcement

A major attack on drive reduction, together with an alternative approach, was mounted by Robert Bolles in 1972 (Bolles, 1972). Bolles surveyed a number of phenomena, which had been identified in the late 1960s and early 1970s, that defied explanation in terms of drive reduction. These were, first, operant *stereotypies* of behavior that developed in operant conditioning, sometimes called "misbehavior" (Breland & Breland, 1961). For example, a raccoon that was being trained to put a coin-like object into the slot

of a piggy bank for food reward obstinately refused to release the coin object from its paw when it had slid into the slot, but instead pulled it out again and made "washing" movements with the coin, dipping it and wiping it again, as though the coin were food. Second, *autoshaping* of what looked like operant behavior under conditions where there was actually no reinforcement of responses by drive reduction. For example, a pigeon that received presentations of a signal light followed by free food reward, after many pairings came to peck the signal light whenever it was turned on, even though it had never been explicitly reinforced to do so. The pigeon had "shaped itself," hence the term *autoshaping*. Third, bizarre "adjunct" behaviors occurred, such as *schedule-induced polydipsia,* in which animals drank exorbitantly large amounts of water for no apparent reason when put into a training schedule. For example, a hungry rat presented with tiny food rewards interspersed with delays of a minute or so begins to engage in copious amounts of drinking behavior, and may consume up to one-half of its body weight in water in a mere 3-hr session (Falk, 1971).

Bolles argued that these phenomena demonstrated that reward learning could not operate by the response reinforcement and drive reduction principles that had reigned for nearly half a century. First of all, the phenomena were happening in the absence of response reinforcement. Reinforcement, whether drive reduction or hedonic satisfaction or something else, had not stamped in a misbehavior/autoshaped/adjunct response to a stimulus, since none of these responses had been explicitly reinforced by the experimenter. The animals had instead learned something else, and reinforcers were causing some other kind of process to occur. Bolles argued that the animals had learned S-S* associations, that is, associations between a CS (S) and a subsequent hedonic stimulus that caused pleasure (S*). The first S did not elicit a response, he argued. Instead it elicited an expectation, an expectation specifically of the second S*. The animals had also learned an R-S* association, Bolles suggested, namely that their own response was a predictor of the emotionally laden S*. At the moment they engaged in these strange behaviors, such as treating a keylight or a coin as though it were food, he suggested, they did so because they were seized by an overwhelming expectation of food itself (Bolles, 1972).

Bolles's S-S* expectancy account provided the seed of an explanation of why animals sometimes behaved as though they had reward when they did not yet have it. But expectancy by itself was not a full explanation. It is not clear why an individual who expects food should engage in bizarre behavior. Why not simply sit back and enjoy the expectancy, knowing that the real food is about to arrive? More explanation was needed to account for the behavioral phenomena.

B. Bindra: A Conditioned Stimulus Becomes the Incentive

In 1978, Dalbir Bindra provided an extension of the incentive account that helped fill the explanatory gap (Bindra, 1974, 1978). He adopted the S-S* framework of Bolles, but sidestepped the idea that it was an expectancy alone that caused irrational behavior. He suggested instead a specific motivational process was evoked. The CS or S, he suggested, does not simply cause the subject to expect S*, but also evokes a *central motivational state of the hedonic S**, which in turn causes the animal to *perceive* the S as an S*, to the extent that perception could be supported by the stimulus properties of the signal stimulus. The S, in other words, takes on specific motivational properties that normally belong to the S* itself. These motivational properties are incentive properties: they attract the animal and elicit goal-directed behavior and even consumption.

By Bindra's account, food is normally an attractive incentive. Animals seek it out. They eat it if they can. If they are raccoons, they may wash it before they eat it. When they do eat it, they enjoy it. The incentive properties of food allow it to produce all these effects. Bindra made the startling claim that the CS—the S—gained the same properties as a function of its association with the S*. An animal, he suggested, approaches the CS for a reward. It finds the signal attractive. If the CS is food, the animal wants to eat it. If it is an S for a tasty food S*, the animal may enjoy its attempt to eat the CS.

Bindra's assertion that a CS in a sense becomes an incentive to the animal, the same incentive as the S* stimulus, is rather starlingly unintuitive. It seems almost a step back from Bolles's expectancy account. It asserts that signals are taking on motivational and hedonic properties as a consequence of their Pavlovian association with rewards. This is not entirely in congruence with our own sense of how we respond to motives—drive reduction was almost easier to accept by comparison. But what if Bindra were right? The raccoon washes its coin, according to this, because the coin has become "food-like" to the animal. The pigeon pecks the keylight signal for food in autoshaping because the light has become a glowing piece of food. Suddenly there is a psychological explanation for these phenomena.

Still at face value, Bindra could not possibly be right, at least not unless something more is added. Critics noted that if CSs simply became incentives, then one would always respond to them, whether hungry or thirsty or not (Gallistel, 1978). There was not a clear way to explain how incentives and CSs would interact with drive states. Yet clearly drive state is important, and, in addition, true expectancy may exist and guide behavior in its own way.

C. Toates: Conditioned Incentives, Hedonic Modulation, and Drive Cues

In a book published in 1986, Frederick Toates adopted the Bolles–Bindra notions of S-S* associations and hedonic transfer of incentive properties to the CS, yet in a new synthesis escaped the rigid predictive difficulties (Toates, 1986). First, he posited that both cognitive expectancy and more basic reward processes might occur simultaneously within an individual, and influence behavior in different ways. Then he addressed how basic reward processes could guide normal behavior via incentive cues and also grab control to produce even irrational behavior.

1. Alliesthesia: Changing Hedonic Value

Toates began with the idea that hedonic incentives were the target of motivation. Hedonic incentives were stimuli that directly produced experiences that felt good. Tasty food, refreshing drinks, sexual partners, drugs that caused pleasant feeling, social rewards were all hedonic incentives. Toates also drew on the work of the psychologist and physiologist Michel Cabanac (Cabanac, 1979, 1992), to note that the ability of these incentives to produce pleasure was themselves in many cases modulated by drive states. Physiological consequences of eating, etc., were posited by Toates to feed back to influence motivation—but not directly. Rather, for Toates, drive states modulate the value of incentive stimuli. Pleasure is not an invariant property of even tasty food, Cabanac had shown. A sweet taste may be delicious when we are hungry; it may be ambrosia if we are starving; yet if we are stuffed after large meal, it may retain only a ghost of its pleasure producing properties. Our palate can become jaded, and no longer take pleasure from what it once did. In laboratory demonstrations, for example, Cabanac showed that human subjects gave higher subjective ratings of pleasure to a sugar solution when they were hungry than when they had recently eaten. The pleasure of the sensation had changed with their physiological state, even though the sensory quality of the sweetness was no more or less than it was before. Cabanac called this changed hedonic quality of the sensory experience "alliesthesia," which means essentially a change in sensation (though to be fully accurate, the phenomenon is a change only in the *pleasure* of the sensation), and argued that alliesthesia is a basic property of most hedonic sensations. A hot bath feels delightful if we are cold, but may seem positively unpleasant on a hot day—when a cold plunge into cool pool seems much more pleasant. In another example, the saltiness of seawater is unpleasantly intense to most individuals. But in a phenomenon known as salt appetite (Schulkin, 1991), the taste of salt

becomes more pleasant if we lose our normal body levels of sodium chloride, either by going on an entirely salt-free diet for several weeks or by taking drugs that fool physiological mechanisms into reducing sodium stores (Beauchamp, Bertino, Burke, & Engelman, 1990).

Alliesthesia shifts in the hedonic impact of stimuli such as sweet or salty tastes can be detected in animals or in human infants too, even without the use of subjective ratings of pleasure, by measuring affective reactions. Certain behavioral affective reactions made by human infants, such as facial expression, reflect the hedonic impact of a taste stimulus (Berridge, 2000; Steiner, 1979; Steiner, Glaser, Hawilo, & Berridge, 1999). If a sweet taste is placed in the mouth of a human infant, a chimpanzee, or a monkey, certain hedonic reaction patterns are elicited, such as rhythmic tongue protrusions or finger sucking (Figure 1). Even in a rat, a sweet taste elicits behavioral affective reactions (e.g., rhythmic tongue protrusions, licking of lips and paws). Alliesthesia can be observed in the change in these hedonic reactions to sweetness; they are increased by hunger and decreased after a meal (Berridge, 1991; Cabanac & Lafrance, 1990). Similarly, the salty

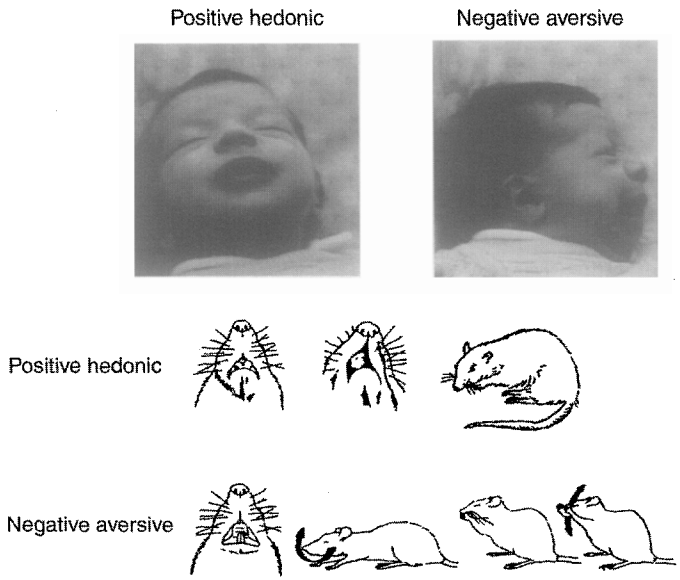

Fig. 1. Hedonic impact or "liking" reflected in behavioral affective reactions elicited by tastes from human infant and by rats. Positive hedonic reaction of a 3-week-old infant to a sweet sucrose solution versus negative aversive reaction to an unpleasantly salty solution is shown at top. Drawing at bottom shows positive hedonic and negative aversive reactions elicited by tastes from rats. (From Berridge, 1996.)

taste of seawater elicits hedonic reactions during physiological states of sodium depletion, but elicits a bitter-like aversive response (e.g., gapes, arm flails, headshakes) during states of normal sodium balance (Berridge, Flynn, Schulkin, & Grill, 1984). Alliesthesia, or shifts in the hedonic impact of incentive stimuli caused by changed physiological states, may thus be a common occurrence in the lives of both humans and other animals.

III. Consequences for the Incentive Model of Motivation and Reward

Toates drew upon phenomena such as alliesthesia to modify the Bolles–Bindra theory, and to allow physiological drive states to play a role. Hunger, thirst, and other drive states, he posited, served to potentiate the hedonic value of primary rewards. Rather than produce reinforcement by being lessened, as drive reduction hypotheses had always assumed, drives interacted with incentive stimuli such as the taste of food to multiply or potentiate the hedonic value of food or of another incentive stimulus. Food tastes better during hunger, water is more delicious during thirst, saltiness becomes more pleasant during sodium deficiency, and so on. Hedonic modulation by physiological drives made incentive stimuli more attractive and so more able to control behavior in a Bindra–Bolles fashion.

But the multiplicative interaction between drives (e.g., hunger) and incentive stimuli (a tasty morsel) works both ways, according to Toates. Not only do drives such as hunger potentiate the incentive value of food, but the reverse is also true. An external incentive stimulus, such as the presentation of a morsel of food to an individual who is hardly at all deprived, can potentiate appetite for more food as effectively as an increase in physiological need. One bite of food can seemingly intensify hunger in an individual who moments before was not at all thinking of dinner. The French expression puts it, "*l'appetit vient en mangeant*" (loosely translated, appetite comes in the act of eating). You may have experienced this as the "cocktail peanut" phenomenon: after taking one tidbit without desire and merely to be polite, you suddenly find you want to eat a few more. Cornell, Rodin, and Weingarten (1989) found that human subjects who had been fed to satiety, if asked to take a bite of either pizza or ice cream, would subsequently choose to eat more of whichever of these two foods they had just been given.

In animal laboratory settings the ability of a reward stimulus to increase motivation for itself is known as priming. A rat, for example, may seem reluctant to begin work when placed at the beginning of the day's session back in the chamber where lever presses will earn food, but it can often

be suddenly energized by simply giving it one free food pellet. After ingesting its free reward, the rat is likely to show renewed interest in working for more food. Priming is also well known in studies of brain stimulation reward and of addictive drug reward, where animals earn activation of the stimulation electrode or a small intravenous pulse of rewarding drug by performing a response. Responding may be low in the beginning of a session—unless a free reward is given or until the first reward is finally earned—and then the animal sets to work. This is very much in keeping with the Toates model of incentive motivation.

And how does reward learning play a role according to this Toatesian scheme incentive–drive interaction? Precisely as Bolles and Bindra had specified: CS for primary rewards, such as the sight of a signal for food, acquire the incentive properties of their primary UCS, such as the taste of food, via Pavlovian associations produced by their pairings. So, for example, the mere presentation of an auditory CS that has previously been paired with food reward causes rats that are not physiologically hungry to begin eating again (Weingarten, 1983; Weingarten & Martin, 1989). Priming of appetite can thus be done by a Pavlovian cue as well as by the actual taste of food. Presentation of an auditory CS for sucrose reward appears to elicit a *hedonic* state appropriate to sweetness too (Figure 2). For example, if a tone that has been paired with sucrose is played to a rat while an infusion of water is squirted into its mouth, the rat increases its number of positive hedonic reactions (e.g., lip licking) (Delamater, LoLordo, & Berridge, 1986). Conversely, if a different tone that has been paired with quinine is presented during the oral infusion of water, the rat suppresses its positive hedonic reaction patterns and instead increases its aversive reaction patterns (e.g., gapes).

Toates added the additional stipulation that the incentive properties of such conditioned reward stimuli were modulated by drive states such as hunger in exactly the same way as the incentive properties of food itself. In effect, Toates argued, a CS light that predicts food to a hungry pigeon or rat becomes an attractive, potentially edible, and possibly even tasty food-like object. It elicits approach and even consummatory behavior that would ordinarily be directed to the food itself. But when no longer hungry, the same light for the animal is just a predictive signal, devoid of motivational properties.

The associative link that allows a CS to acquire hedonic value from its unconditioned partner can be quite powerful, according to Toates, powerful enough to overcome any existing hedonic value the signal stimulus may already have. An example of this is taste aversion conditioning. In taste aversion conditioning, a normally palatable food is paired associatively with an unpleasant physiological consequence such as nausea and related upper

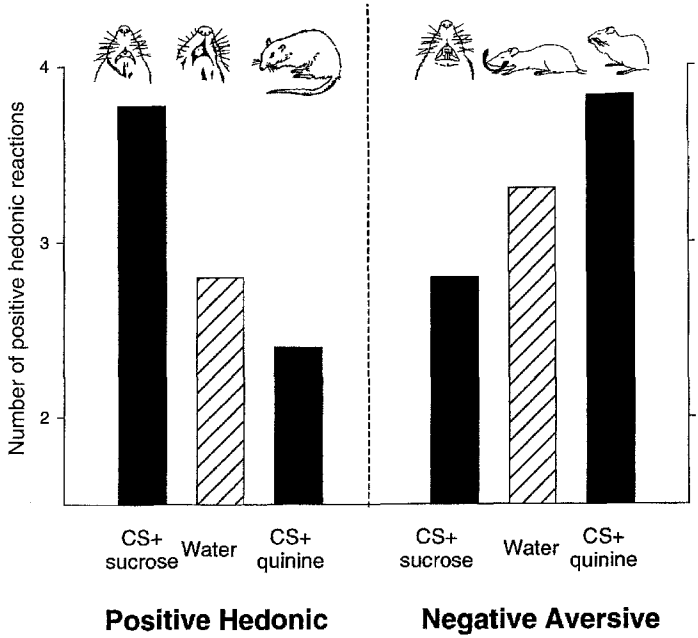

Fig. 2. Conditioned stimuli (CS) elicit hedonic states. The affective reaction pattern elicited from rats by the taste of water is modulated by two tone CS that were either paired with the sweet taste of sucrose (CS+ sucrose) or the bitter taste of quinine (CS+ quinine). Positive hedonic reactions were increased by the sucrose cue and decreased by the quinine cue. Negative aversive reactions were conversely increased by the quinine cue but decreased by the sucrose cue. Each auditory CS appeared to evoke an affective state similar to that of the sweet/bitter taste it signalled. (Figure modified from Delamater et al., 1986.)

gastrointestinal distress of the sort produced by food poisoning or by a drug (or excessive alcohol) that mimics aspects of such illness (Garcia, McGowan, Ervin, & Koelling, 1968; Rozin, 1999). The first time the food is encountered, the illness has not yet occurred. The individual enjoys the palatable food and finds the experience of consuming it pleasant. Then the illness follows, which is of course unpleasant. The taste aversion is not revealed, however, until the food is once again encountered. The important point is that the taste signal does not simply call up a representation of UCS state of illness. It may or may not call up an explicit re-representation of the nausea state. But the taste itself is always perceived as reduced in pleasantness after aversion conditioning—even if one doesn't realize why. The same taste that before seemed so pleasant may evoke quite a different reaction and be perceived as disgusting. It simply tastes bad. Animals too, if they have had a pairing with drug-induced ills after ingesting sweet taste

that evoked positive affective reactions, switch their reaction when the taste is next presented: now they respond with aversive reactions as though the taste were bitter (Berridge, Grill, & Norgren, 1981; Grill & Norgren, 1978). Taste aversion learning obeys all the rules of Pavlovian learning and can be strengthened, weakened, or even prevented by proper application of those rules. For example, one can protect against taste aversion learning via Pavlovian *latent inhibition,* which means that a very familiar CS, one that has been experienced many times before, will resist entering into the new Pavlovian association. In other words, if a food is already familiar when the illness first occurs, then a conditioned aversion is not likely to follow. That is why, if several foods are eaten at a meal hours before a visceral illness occurs, but only one of the foods is new, then the individual may afterwards intensely dislike that new food but not feel differently about the other foods (Rozin, 1999).

The power of S-S* associations was dramatically illustrated in a special demonstration of taste aversion conditioning by Peter Holland, in which an auditory tone S (Pavlovian CS) was made to literally stand in for its food S* (Holland, 1990). In one experiment, Holland presented one tone to rats as they drank a wintergreen-flavored sucrose solution and a different tone as they drank a peppermint sucrose solution. Then later, he presented *one* of the tones by itself (without any sucrose or flavor) to the rats as they were made ill by lithium chloride (injection of LiCl causes nausea). Finally, the rats were allowed to choose between the peppermint sucrose and the wintergreen sucrose (without any tones present). Holland found that the rats drank considerably less of the particular solution whose tone had previously been paired with illness. They had developed a conditioned aversion for the flavor, apparently mediated by the auditory CS. In this case, no food was paired with illness, only its CS tone had been paired. Holland concluded that the tone had evoked a sensory representation of its taste—a taste memory—when it had been paired with the illness, and that the rats had learned an aversion to the *remembered* taste, called to mind by the tone. The CS had fully substituted for its taste in the learning of this new aversive reward value: after S-S* associations, the S can stand in for the S*.

Toates's use of S-S* associations in formulating an extension of Bindra's theory of incentive motivation is remarkable, for it entails several implications, some of which are quite unintuitive (Figure 3A). The theory is all the more interesting and important if these implications are true.

First it means, as we have said already, that the incentive value and even hedonic value of stimuli rises and falls with changes in relevant physiological deficit or drive states. This should be true regarding food reward, drink reward, sex reward, drug reward, brain stimulation reward, and perhaps

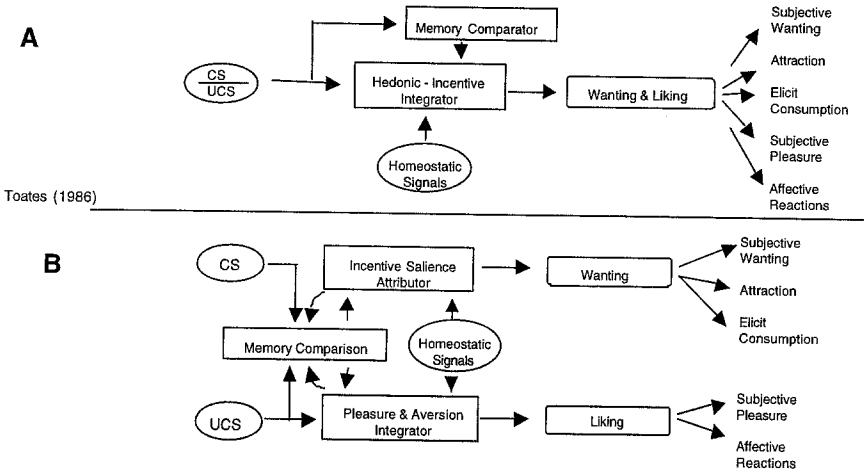

Fig. 3. (A) The Bindra–Toates model of conditioned incentive motivation in which "wanting" and "liking" are identical, based on Toates (1986). (B) Incentive salience model or modified Bindra–Toates model in which "liking" and "wanting" for rewards and Pavlovian reward cues are separate, from Berridge (1996; modified from Robinson & Berridge, 1993). Some brain manipulations, such as impairment of dopamine systems, disrupt Pavlovian incentive salience, selectively suppressing "wanting" for a food reward (but leaving "liking" alone). Other brain manipulations, such as microinjection of amphetamine into the nucleus accumbens, selectively potentiates the ability of a Pavlovian conditioned stimulus (CS) to elicit "wanting" for its food reward (without potentiating "liking" for that food reward). In human drug addiction, too, neural systems of incentive salience may be selectively sensitized, leading addicts to "want" to take drugs even if they don't particularly "like" them. (From Robinson & Berridge, 1993.) UCS = unconditioned stimulus (such as food reward or drug reward).

many other types of reward. A bite of sweetness is not fixed in its hedonic value, and neither is a given dose of cocaine. Both may shift predictably according to this model.

Second, it means that drive states will themselves be often potentiated by a sudden encounter with a relevant incentive or even with a CS for that incentive. The smell of fresh-baked bread or of sizzling meat are potent triggers of appetite as mealtime approaches. Even the mere sight of the food or a realistic image may make the mouth water and arouse appetite.

Third, Toates suggested, the multiplicative outcome of the interaction between external incentive stimuli and internal physiological cues for drive states is not only sufficient to trigger motivation but may be *necessary* for substantial motivation to occur. In a multiplicative relationship, there is no output if either element is zero. Incentive stimuli and drive cues both need the other to create a motivational state. If either category is totally missing, then even relatively strong intensities of the other category may have little

effect on motivation or behavior. In other words, drive reduction, which was so important to earlier theories of reinforcement, is almost powerless by itself to cause reward. Could this counterintuitive assertion possibly be true? The answer appears to be yes.

A. Drive Reduction: Insufficient for Strong Reward

In retrospect, evidence to support Toates can be found going back decades. In an early demonstration of the importance of simultaneous co-occurrence of oral incentive stimuli together with drive reduction, Miller and Kessen (1952) trained rats to run in a T maze for one of two possible rewards. One reward was intragastric milk that was pumped directly into the stomach, which reduced hunger drive alone. The other reward was the chance to lap milk normally, which both reduced drive and provided an oral incentive stimulus comprising taste/tactile/consummatory act components. This experiment was predicated on the demonstration by many earlier studies that rats would run faster down a runway to obtain a larger reward (e.g., more food pellets) than for a smaller reward, and similarly would run faster for any food that they preferred. In other words, running speed during a test was proportional to the "goodness" of the reward that had been given during training, and better rewards produce faster running. The rats approached the "pure drive reduction" goal only slowly, essentially walking hesitantly toward the intragastric reward. Intragastric milk appeared to be only a very weak reward, as the rats would choose it over nothing, but never eagerly. By contrast, rats ran swiftly toward the natural milk drink reward, which combined oral incentive plus drive reduction, even though the same amount of milk ended up in their stomachs in both cases.

In a similar demonstration, McFarland showed that drive reduction was not a sufficient reward for thirst (McFarland, 1969). McFarland trained doves to peck an illuminated keylight in order to receive a sip of water to drink. Once the doves had learned this task, McFarland switched them to a new task, which came in one of two versions. In one version, the second task was the same as the first: pecking on a new key light simply earned again the chance to drink. In the other version of the new task, pecking on the new key light earned a direct infusion of water into the dove's crop (a pouch within the esophagus where doves normally store small quantities of water or food before passing it on to the stomach). The amount of water was the same in both cases, and thus provided about the same degree of physiological need reduction. McFarland found that pigeons who were switched to the natural drinking task simply maintained their performance at high rates. They immediately learned that the new key provided water reward, just as the old key had, and they kept pecking at it. By contrast,

the doves who were switched to the crop infusion of water initially pecked a number of times at the new key, just as they had at the old key, but then gradually ceased to peck at all. Even though they received just as much water into their bodies as before, the water was no longer able to reinforce or maintain their behavior. It appeared no longer to be a reward. The results of McFarland's switched task experiment are important because they show that drive reduction by itself is not only insufficient to train a powerful new level of motivated behavior, but that drive reduction alone is also an insufficient reward to maintain motivation even in a rewarded task that has already been well learned (McFarland, 1969).

B. Drive: Insufficient to Cause Motivation

The Bindra/Bolles/Toates theory posits that motivation as well as reward is incentive based, not drive-based. Just as drive reduction by itself turns out to be insufficient for reward, so physiological drive by itself should not be necessary or sufficient to cause motivation. In an early demonstration that physiological drive was unnecessary for motivation, Solomon and Stellar and their colleagues asked whether appetite for food would be suppressed by intravenous feeding of the average daily intake of calories and nutrients (Turner, Solomon, Stellar, & Wampler, 1975). They provided dogs with intravenous meals, solutions of glucose, and other nutrients delivered directly into the bloodstream through a vein catheter, giving as many calories as the dogs would ordinarily eat in a day. The question was whether this intravenous feeding would satisfy their appetites. The answer was no: if the dogs were given the chance to eat ordinary food, they ate almost as much as they would have ordinarily without the extra calories. As a result of the combined intravenous feeding and voluntary meals, the dogs became quickly overweight. But still they continued to eat.

In a more sophisticated demonstration of this principle, Bedard and Weingarten (1989) precisely parceled out the role of oral incentive cues versus physiological drive reduction in turning off appetite, and found an interactive effect precisely as Toates would have predicted. Bedard and Weingarten used a sham feeding paradigm, in which rats were first implanted with a gastric fistula—a permanent tube into their stomach. If the gastric fistula were open, then what the rat ate or drank would leak out of the tube after it were swallowed—hence the name sham feeding. On the other hand, the open gastric fistula also allowed Bedard and Weingarten to give an intragastric meal directly, by simply passing liquefied food through the tube into the stomach. Bedard and Weingarten took hungry rats and simply asked, what was the crucial event that made them stop being hungry? Was it the physiological effects of food in the stomach and

its absorption into the body? If so, then a gastric infusion of liquid food ought to suffice to satisfy the hungry rats. Or was it the opportunity to experience the oral stimuli of biting, tasting, chewing, and swallowing the food? If so, then sham feeding, with the gastric fistula open, ought to suppress a subsequent meal even though the food from the first meal had leaked out of the stomach. Or was a Toatesian interaction required between physiological and incentive stimuli? The answer was that neither oral stimulation nor drive reduction alone were able to suppress appetite. Bedard and Weingarten found that appetite for a second meal was suppressed only if rats first received the combination of sham feeding and a nutrient infusion (or the chance to simply eat an ordinary meal first, which also combined both oral and physiological aspects of eating). Similarly, it is difficult to satiate thirst by drive reduction alone. Thirsty rats continue to drink even after they are given intragastric infusions of water unless they are also allowed to experience oral stimulation related to the act of licking and drinking (Rowland & Nicolaidis, 1976). According to Toates's view, motivated behavior exists only when both types of external incentive and internal drive stimuli are present. Motivation is terminated only when the individual's requirements for both are satisfied. And reward learning, of the sort needed to produce vigorous behavior directed toward that reward in the future, occurs only when both are delivered interactively to the individual. Though counterintuitive at first glance, Toates's notion of multiplicative interaction between incentive and drive stimuli appears to be crucial in order to properly understand basic incentive motivation and reward.

IV. Do Conditioned Incentive Stimuli Take on the Motivational Properties of Primary Incentive Stimuli?

Let's consider a few other surprising implications of the Bolles/Bindra/Toates incentive process. Toates's theory makes the startling claim that when a cue, or CS, is associatively paired with a primary hedonic reward (tasty food, pleasant drug), the result is not only *learning* of a Pavlovian association (and perhaps a cognitive expectation too) but also a *motivational* transformation. The claim is that, to the individual who has learned the association, the cue actually takes on a number of the incentive motivational properties originally possessed only by that primary hedonic event. The conditioned cue becomes attractive and valuable to the individual, in the same way that the primary reward is valuable, and may even elicit some of the same hedonic impact, or pleasure, elicited by the unconditioned event. The individual may try to interact with the cue in some of the ways normally reserved for interaction with the primary hedonic reward. If the

primary reward were tasty food, for example, then after Pavlovian conditioning, the hungry individual might find the cue alone to be somewhat tantalizing, pleasant, and even perhaps edible.

An edible cue seems to stretch credibility, but cases of edible cues produced by classical conditioning appear to exist. We have encountered one already—autoshaping—which now deserves a closer look.

A. Autoshaping

Autoshaping takes its name from a superficial similarity to operant conditioning or shaping, in which an animal's response such as lever pressing or key pecking is gradually increased by an experimenter who awards reinforcement only after the animal shows an approximation of that response. But in reality autoshaping is a pure incentive process with no response reinforcement at all. It is caused by Pavlovian associations, rather than operant contingencies. It creates an attractive and even edible Bindra–Toates-style conditioned incentive out of a mere Pavlovian cue stimulus.

In autoshaping, originally discovered by Brown and Jenkins (1968) and by Williams and Williams (1969), a pigeon or rat is placed in an operant chamber where there is a signal light above a lever to press (for rats) or a signal light embedded in a key or button to peck (for pigeons). The signal light is occasionally illuminated and followed by presentation of food reward. In other words, the signal is a Pavlovian CS for food. The lever can be pressed (or key pecked for pigeon subjects) as the animal chooses. The crucial feature that distinguishes autoshaping from ordinary shaping is that in autoshaping there is absolutely no contingency between pressing the lever or pecking the key and food reward. Food and its CS signal are presented intermittently regardless of whether or not the animal performs the instrumental response. Under these conditions there is no rational reason for the animal to work. There is no response reinforcement. There is no shaping because food reward is not selectively given when the animal performs the response. The reward is free. Yet, amazingly, under these conditions, as a trial continues, pigeons begin to peck robustly at the key when it is illuminated, and rats begin reliably to press the lever (often biting the light itself as well).

When autoshaping was first discovered, early attempts were made to explain it in traditional reinforcement terms, by suggesting that perhaps reinforcement was occurring fortuitously. If the animal had simply happened to peck the key or push the lever by chance before the food was delivered, reinforcement would occur accidentally, and if they responded again, they would be again accidentally reinforced by another food reward, even though there was no outside contingency between their response

and the reinforcer. In effect, the animals would have created their own response–reinforcer contingency. Skinner had many years earlier advanced a similar explanation of superstitious behavior, that is, particular behavioral patterns that some animals developed when put into an operant chamber, even though the experimenter did not intend to reinforce those behaviors (Skinner, 1948).

A moderate difficulty for the accidental reinforcement explanation is that *any* response should be equally likely to be strengthened, whereas in autoshaping the response is usually pecking or biting of the cue or lever pressing. Clearly something else is needed to explain the directedness of this behavior, something that draws upon the predictive relation between the cue and its particular reward. Staddon and Simmelhag (Staddon & Simmelhag, 1970) offered the first explanation that drew upon this predictive, Pavlovian relationship. They suggested that the cue light became a Pavlovian CS for food or water and elicited a few Pavlovian CRs, similar to the responses that ordinary food or water would elicit as UCs. Then once a few Pavlovian CRs had been predictably elicited by the cue, they suggested, those reward-appropriate responses were particularly and especially reinforced in ordinary fashion by the arrival of a food or water reward.

But even this degree of reinforcement-based explanation was demolished by later experiments that showed autoshaped subjects apparently did not need any kind of response reinforcement, even accidental reinforcement, to develop autoshaping (Schwartz & Williams, 1972). This was shown by adding a *negative* contingency between responses and reward (an omission contingency) to the autoshaping procedure. If the animal responded by pecking the keylight then food was withheld and not delivered, even if the cue had just occurred. If the animal did not respond when the cue was illuminated, then the food reward came as usual. The animal ought to learn to simply sit and wait for signaled food, or at least to refrain from approaching the lever or key, under these conditions. But instead it was found that autoshaping still developed—albeit somewhat more slowly—under an omission contingency. The subjects continued to develop and maintain approach and consummatory-like responses directed toward the food cue—even though it cost them a real food reward every time they did so. This rules out a reinforcement explanation.

B. Consumption of Conditioned Stimuli

Further observations supported yet another aspect of the Bolles/Bindra/Toates interpretation, that is, that CSs take on the natural incentive properties of the reward they represent. It was discovered that autoshaped subjects often attempt to "eat" their reward's CS. Rats, for instance, may gnaw away

at the plastic cover that protects the cue lightbulb, sometimes destroying the cue entirely. Pigeons have a distinctive movement pattern of the beak used in feeding pecks, in which they bring the top and bottom halves of the beak rapidly together as they encounter their normal food, such as a speck of grain. In autoshaping, the same feeding peck is directed towards the keylight itself if it has signaled food. By comparison, pigeons that have been autoshaped while thirsty using a cue-water association show a drinking peck, a different movement pattern in which the lower beak is extended longer to scoop up water. Thus in a literal sense, animals appear sometimes to try to eat or drink their CS cue light, depending on whether their primary hedonic reward was food or water. They treat the CS object as though it were edible food or potable water, and try to consume it in the appropriate fashion (Allan & Zeigler, 1994; Jenkins & Moore, 1973).

Autoshaped eating of a keylight is certainly consistent with the proposition that CSs for a reward take on the properties of that reward, and are treated almost as though they *were* the reward. But an alternative interpretation was also possible, one also based on Pavlovian sensorimotor conditioning. It is conceivable that what has been associated with the cue is not so much an incentive motivational status of food as it is the sensorimotor response pattern that is usually elicited by food. We have already mentioned that the autoshaped keypeck uses a movement pattern very similar to the movement used in real eating or drinking. It has long been known that motor responses, or simple movement patterns, can easily be conditioned by Pavlovian training. Eyeblink movements, for example, elicited by a wind-like puff of air, are also elicited by a tone CS for the airpuff. Could autoshaping too be merely a conditioned motor response? If so, the phenomenon would be of much less motivational interest.

C. Pure Incentive Motivation Elicited by Pavlovian Cues

Fortunately there are ways to show that the sensorimotor conditioning explantation is insufficient, and that a CS indeed takes on motivational properties as the incentive conditioning model suggests. One procedure that has been used to accomplish this uses separate Pavlovian pairings outside of the instrumental training situation, which are then used to influence a different instrumental response that involves a different movement from any Pavlovian CR. This procedure has been given various names, such as the conditioned emotional response or Pavlovian instrumental transfer paradigm. It essentially measures the effect of a Pavlovian *incentive cue* upon motivation reflected through instrumental performance to obtain a reward.

For example, Lovibond (Lovibond, 1983) trained rabbits to perform an instrumental response for sucrose reward and then separately trained them

in a Pavlovian cue–reward association. Instrumentally, the rabbits learned to push a lever to obtain a sucrose squirt when they were hungry. Quite separately, they got free sucrose during training of a Pavlovian cue, a clicking sound. The sound came on for 10 sec, and then the sucrose solution was delivered. No response was necessary during this Pavlovian portion of the training, and the rabbits had no opportunity to press the lever during the cue training. Finally, the lever was returned, and the rabbits were allowed to work again to obtain the sucrose. Now the clicker cue was occasionally presented. In this case, the clicker could elicit no conditioned motor response relative to the lever, but it could still elicit an incentive motivational state. The incentive state could be expressed only through the instrumental lever-pressing response, not through any Pavlovian motor response. And indeed, the rabbits pressed the lever that had earned sucrose more during the period when the clicker cue was presented than during the period when there was no Pavlovian cue. In other words, the cue had clearly potentiated instrumental responding, even though the motor response needed to perform the instrumental task was unrelated to any Pavlovian motor responses. This indicates that a heightened motivation for the reward was truly produced by the cue. (Aversive motivation may work in a similar way: if a Pavlovian cue is paired with shock, that cue can increase the level of instrumental performance of animals that are performing a response to avoid the shock (Rescorla & Lolordo, 1965). In such cases, the cue elicits what has been called a conditioned *emotion,* such as fear of shock or desire for sucrose, that is appropriate to its paired UCS.

This demonstrates that the Pavlovian cue can elicit a motivational state. But does it show that the cue itself is the target of the motivational state? Does the individual merely want the unconditioned reward? Or does it want the CS as well as the UCS, as Toates's model would predict. That is, does the cue's association cause it to become an incentive itself, as well as being able to potentiate incentive motivation for the unconditioned reward?

D. Sensory Preconditioning of Incentive Value

Evidence that the cue itself actually becomes a target for incentive motivation comes from an experimental procedure called *sensory preconditioning.* In sensory preconditioning, a neutral cue is first paired with another neutral cue. Neither stimulus has motivational value at this first stage. They are simply presented together again and again, so that the subject learns an association between them. Then one of the neutral stimuli is given new motivational value. The motivational value may be given by ordinary Pavlovian conditioning, by pairing it with a reward or with a punishment. Alternatively, one of the neutral stimuli may be given motivational value

by performing a physiological manipulation upon the individual that is relevant to that particular stimulus.

For example, in a salt appetite experiment using sensory preconditioning Fudim (1978) combined pairs of mixed flavors for rats: for some rats, banana flavor was combined with salt into a salty banana mix, and almond flavor was combined with sucrose to make a sweet almond mix. For other rats, the pairs were salty almond and sweet banana. Then Fudim induced a physiological state of sodium deficiency in the rats, by giving them an injection of formalin (which causes sodium-containing body fluids to move out of their normal physiological compartments, producing a physiological sodium deficiency). The sodium deficiency would be expected to produce a salt appetite in which salty tastes would be sought out (Schulkin, 1991). While the rats were sodium-deficient, Fudim presented them with a drinking tube that contained pure banana flavor by itself and with another tube that contained pure almond-flavored solution. No salt or sugar was now present in either tube; instead, there was merely a flavor that had previously been paired with salt or sugar. Impressively (from the point of view of the Bindra–Toates model), the sodium-deficient rats drank much more of the banana or almond solution that had been paired with salt than they did of the flavor that had been paired with sucrose. When the rats were not sodium-deficient, on the other hand, they did not show this preference to consume the salt-paired solution. In other words, the drive state caused the rats to seek out and actually ingest the CS itself—precisely as the Bindra/Toates model would suggest.

E. Sensory Preconditioning of Hedonic Value

One last objection could be raised at this point to the Toates–Bindra interpretation. Perhaps the rats in Fudim's experiment did not like the CS flavor they drank when sodium depleted, but rather drank it solely for its association with postingestive effects of salt. The rat might drink the flavor in a way similar to why you might drink an unpleasant tasting medicine: not because you like the medicine's flavor, but because you hope to feel better afterwards. The Toates–Bindra model specifies that conditioned incentives actually become *liked.*

In order to find out whether the Bindra–Toates prediction was literally true, and whether the CS actually gained *hedonic value* itself, Jay Schulkin and I modified Fudim's sensory preconditioning experiment and added an additional test of the flavor's hedonic impact: the taste reactivity measure of hedonic and aversive patterns of behavioral reactions that are elicited by tastes (Berridge & Schulkin, 1989). Similar to Fudim's experiments, our rats first received two flavors that were paired either with salt or with

sucrose. We used bitter-tasting quinine and sour-tasting citric acid as our CS flavors. Bitter and sour were chosen as cues because we wanted to be sure that the CS flavors had no positive hedonic value to begin with (so to be able to tell if positive hedonic value were suddenly acquired). Rats ordinarily respond to bitter or sour tastes with strongly aversive reactions (gapes, etc.). Some of our rats received a salty bitter mixture and a sweet sour mixture. Other rats received a salty sour mixture and a bittersweet mixture for conditioning of the taste–taste association. Later the rats were made to develop a salt appetite by injecting them with furosemide (a drug that disrupts the kidney's hormonal regulation of physiological sodium balance). Once they were sodium deficient, the rats were allowed to drink if they wished either a sour citric acid solution or a bitter quinine solution—but no salt was present in either solution. As Fudim (1978) had found, our rats drank more of the CS flavor that had been paired with salt, and only when they were sodium deficient. Finally, we squirted into the mouths of the rats (through oral cannulae) either the purely sour solution, or the purely bitter solution, and measured their hedonic and aversive affective reactions. Ordinarily the sour or bitter tastes elicited mostly aversive reactions, such as gapes, headshakes, and forelimb flails and this remained true when the rats were tested in a normal physiological state. But when the rats were in a sodium-deficient state, the sour or bitter CS flavor elicited positive hedonic reactions, such as tongue protrusions and paw licking (Figure 3). Only the salt-paired conditioned flavor elicited hedonic reactions, and only when the rats were sodium deficient. The other bitter or sour flavor (which had not been paired with salt) elicited only aversive reactions at all times (Figure 4). In other words, sodium-deficient rats actually "like" a sour or bitter taste if the flavor is a CS for saltiness and if they have a physiological salt appetite at the moment. The rats not only wanted to consume these flavors—they also "liked" them, as Toates' model suggests—even though they had never "liked" concentrated salt itself when they had tasted it before in the taste mixtures (because they had not had a salt appetite then). An integration of associative and physiological information had occurred, passing the alliesthesia increase in hedonic palatability directly to the CS, and giving it new incentive and hedonic value.

V. Splitting Pavlovian Incentives: "Liking" versus "Wanting"

The Bindra–Toates model suggests that Pavlovian incentives become both "liked" and "wanted" as a consequence of reward learning. Conditioned incentive value is equivalent to conditioned hedonic value according to the original Bindra–Toates model (Toates, 1986). Individuals literally move

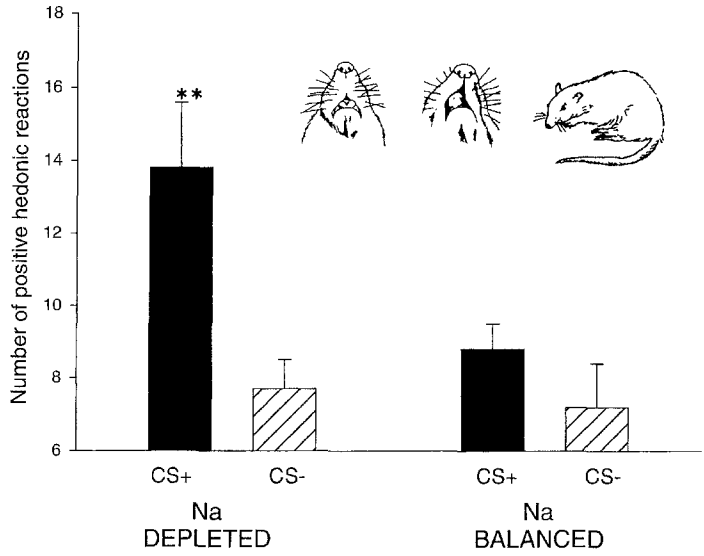

Fig. 4. Alliesthesia of a sour–bitter conditioned stimulus (CS) in salt (NaCl) appetite. Hedonic reactions patterns (lip and paw licking) elicited by a sour (citric acid) or bitter (quinine) taste is modulated by Pavlovian conditioning and physiological deficit state of sodium depletion. The CS+ is the sour/bitter taste that was previously paired with the taste of concentrated NaCl. The CS− is the other sour/bitter taste that was paired with sucrose. Citric acid was the CS+ for some rats, whereas quinine was the CS+ for other rats. The sour/bitter CS+ taste specifically elicited many more hedonic reactions than the CS− when the rats had a salt appetite induced by physiological sodium depletion (NaCl depleted). Neither taste elicited hedonic reactions when the rats were in a normal physiological state of sodium balance. (Figure modified from Berridge & Schulkin, 1989.)

along a gradient of conditioned hedonic stimuli as they get closer to the goal. But recent evidence has led my colleagues and I to suggest there are two modifications that need to be attached to this conclusion. The result is what I have called a modified Bindra–Toates model or the incentive salience model (Figure 3B; Berridge, 1996; Berridge & Robinson, 1998; Berridge & Valenstein, 1991; Robinson & Berridge, 1993).

The first issue concerns how the core processes of incentive value that we've been discussing relate to subjective experience, that is, to conscious pleasure and desire. To many, the term *reward* is often used as a surrogate for conscious pleasure, that is, something that is consciously liked, and indeed one dictionary definition of reward is "a pleasant stimulus" (p. 2584, *Shorter Oxford Dictionary*)(Brown, 1993). But the psychological reality is more complex than the dictionary would suggest. Conscious pleasure may not always be required for reward phenomena. First, a number of studies of

humans indicate that hedonic affective states and the motivational processes triggered by both Pavlovian incentives and unconditioned incentives can sometimes be produced *without* conscious awareness: for example, people may be trained to perform responses for a 'pleasant' drug they are not aware of receiving, or may be made to have an emotional reaction by an event that they do not consciously notice (Fischman & Foltin, 1992; Lamb et al., 1991; Winkielman, Zajonc, & Schwarz, 1997). In other words, unconscious pleasures and desires may exist as unconscious psychological core processes (Berridge, 1999). If so, we need to be able to distinguish conscious pleasures from unconscious core processes of reward. My colleagues and I have adopted the terms "liking"—in quotation marks—to refer to unconscious core processes underlying conscious liking, since the core process is not really liking in the usual sense. Similarly, we've used the term "wanting" in quotation marks to refer to the core process of desire elicited by an incentive, which may occur even unconsciously. It is outside the scope of this chapter to say much more about unconscious core processes of reward or about their relationship to real conscious pleasures and desires, but I refer the interested reader to another chapter that focuses on unconscious affective core processes (Berridge, 1999).

Second and more relevant to the Bindra–Toates model of Pavlovian incentive learning is a *split* that may occur between the incentive processes of "liking" and "wanting." Typically these processes go together, and both obey Bindra–Toates rules. But in a number of affective neuroscience experiments my colleagues and I have found that in some instances "liking" and "wanting" separate dramatically, and we have therefore proposed that the Bindra–Toates model of incentive motivation be modified to allow separate control of these two incentive processes (Berridge, 1996; Berridge & Valenstein, 1991; Berridge, Venier, & Robinson, 1989; Robinson & Berridge, 1993). Separation of "wanting" from "liking" happens especially in cases involving certain neural or psychopharmacological manipulations that can occur in animal experiments and even in real-life human predicaments, such as drug addiction. In such cases, incentive motivation can reflect Bindra–Toates rules applied to "wanting" alone (Figure 3B).

A. "Liking" without "Wanting"

A number of studies from the past 10 years, mostly involving brain manipulations, have shown that it is possible to have "liking" without "wanting" for an incentive, or vice versa, "wanting" without "liking." For example, "liking" without "wanting" appears to be the result of extensive damage to brain dopamine systems. Such damage leaves individuals apparently without motivation for any incentive, conditioned or unconditioned (Fibi-

ger & Phillips, 1986; Marshall, Richardson, & Teitelbaum, 1974; Ungerstedt, 1971), but "liking" for the same incentives remains normal, at least for the hedonic impact of food rewards. For example, in studies of taste-elicited affective reactions, Robinson and I have found that positive hedonic reaction patterns to sweet tastes remain essentially normal in animals that have lost nearly all of their mesolimbic dopamine neurons that project from midbrain to forebrain structures (Berridge & Robinson, 1998; Berridge et al., 1989).

B. "Wanting" without "Liking": Pure Pavlovian Incentive Salience

Conversely, "wanting" without "liking" can be produced by several manipulations in the laboratory and probably in real human life. In the laboratory, electrical stimulation of the lateral hypothalamus in rats triggers a number of motivated behaviors, such as eating. Increased appetite is normally accompanied by increased hedonic appreciation of food. This "alliesthesia" or hedonic enhancement is also caused by many other manipulations that cause eating, such as drugs and conditioned appetites. But Elliot Valenstein and I found eating caused by electrical stimulation of the lateral hypothalamus was not accompanied by enhanced hedonic reactions to the taste of food. If anything, rats responded more aversively to a sweet taste, as though it became bitter, when their hypothalamus was stimulated.

Similarly, Cindy Wyvell, working in our laboratory, has recently found that a related "wanting" without "liking" can be triggered by microinjections of amphetamine that activate dopamine neurons in the nucleus accumbens (Figure 5). The presentation of a Pavlovian CS for food causes rats to work even harder than normal in order to obtain food. Microinjection of amphetamine into the accumbens specifically potentiates the conditioned incentive motivation properties of the food cue and increases the ability of the cue to trigger "wanting" for the food reward. But the enhanced "wanting" for food is not accompanied by increased "liking," as evidenced by the microinjection's failure to increase positive hedonic patterns of behavior elicited from the rats by a sweet taste (Wyvell & Berridge, 2000). Accumbens amphetamine magnifies the conditioned incentive ability of a food cue to trigger "wanting" for food, but has no effect on "liking" for food.

A related "wanting" without "liking" phenomenon for a food UCS itself, very similar to the effects of hypothalamic stimulation, has been found recently by Sheila Reynolds in our laboratory to be produced by accumbens microinjections of muscimol, a drug that stimulates γ-aminobutyric acid (GABA) receptors. GABA neurons in the accumbens receive dopamine inputs. These neurons may be the next link in the neural chain of "wanting,"

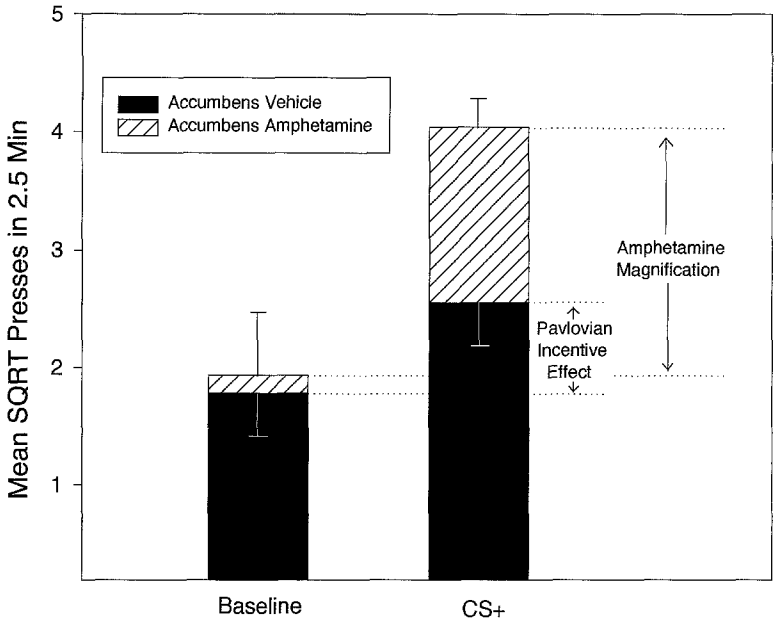

Fig. 5. Magnification of a Pavlovian food cue's incentive properties by a microinjection of amphetamine into the nucleus accumbens (20-μg dose). Bar pressing by rats on a bar that previously earned them sucrose pellets (measured under extinction conditions when no food pellets were actually delivered). Presentation of a Pavlovian conditioned stimulus (CS) for food reward increases bar pressing for the reward. Facilitation of mesolimbic dopamine neurotransmission markedly magnifies the incentive salience attributed to the Pavlovian reward cue, thus increasing its ability to trigger "wanting" for the food reward (without much affecting baseline rate of pressing in the absence of the food cue). (From Wyvell & Berridge, 2000.)

as modulation of these GABA neurons has been suggested to be a shared output mechanism for all mesolimbic reward (Carlezon & Wise, 1996). Muscimol microinjections in the accumbens are known to cause rats to eat intensely, much as LH stimulation does (Stratford & Kelley, 1997). Yet even though this drug microinjection makes the rats avidly seek and consume food, it does not at all increase their hedonic affective reactions elicited by a sweet taste (Reynolds & Berridge, 2000). The GABA agonist in the accumbens makes rats "want" food—and so they eat avidly—but does not make them "like" any more the food they "want."

Finally, human drug addicts apparently may often crave drugs even when they don't derive much pleasure from them. For example, some drugs, such as nicotine, generally fail to produce substantial pleasure at all in most people, but can still be quite addictive. There is reason to believe that

the same dopamine-accumbens neural system may become sensitized or hyperresponsive to drugs and CSs in the brains of drug addicts (Robinson & Berridge, 1993). If so, Terry Robinson and I have suggested that this may cause heightened incentive salience to be attributed to drug cues, causing addicts to "want" to take drugs even if they don't particularly "like" the drugs.

Incentive salience attribution, operating by Bindra–Toates rules, may thus produce "irrational" pursuit of goals and sometimes cause pathological behavior in human daily life as well as in animal laboratories. Incentive salience in short is the property of a perceived reward stimulus that makes it attractive, attention grabbing, "wanted," and a target for goal-directed strategies (Berridge, 1996; Berridge & Robinson, 1998; Robinson & Berridge, 1993). When attributed to a specific stimulus, incentive salience can make an autoshaping cue light appear "food-like" to the autoshaped pigeon or rat that perceives it. When attributed to the smell emanating from a bakery, incentive salience can rivet a person's attention and trigger sudden thoughts of lunch. When attributed in an excessive or sensitized fashion to the sight of drug paraphernalia or drug-associated settings, incentive salience can trigger sudden and compulsive "wanting" in an abstinent heroin addict to take the drug again. Or if attributed to the sight of small white chunks of powder, incentive salience may lead crack cocaine addicts to rummage desperately about on the floor "chasing ghosts"—even if they know cognitively that the white chunks they find there are more likely to be table sugar than cocaine—a not-quite-rational act more than a little reminiscent of autoshaping in animals.

Addiction is a pathological case, but incentive salience attribution operates by Bindra–Toates rules, and is crucial to normal reward learning. Reward in the full sense cannot happen without it. "Liking" by itself is not true reward any more than is "wanting" by itself: nothing need be reward*ed* by hedonic activation alone. Pleasure is not itself goal directed or necessarily associated with objects or events. Pleasure by itself is simply a *triggered affective state*—there need be no object of desire. It is the process of incentive salience attribution that makes a specific associated stimulus or action the object of desire. "Liking" and "wanting" are needed together for full reward (Figure 4).

VI. From Pavlovian Incentives to Incentive Expectations: Dickinson–Balleine

My discussion has stressed the evidence for the modified Bindra–Toates model of incentive motivation and explored at some length its implications.

By this model, incentive motivation is guided by Pavlovian cues for rewards. The cues become attributed with incentive salience (and sometimes with hedonic properties as well), and so the Pavlovian reward cues become beacons, triggers for motivation, enablers for the arousing effects of physiological drive states, and almost partial substitutes for the rewards they represent.

I have emphasized the evidence for this Pavlovian incentive process in part because it is counterintuitive that the incentive salience of Pavlovian reward cues, rather than drive reduction learning or cognitive expectation, is a chief mechanism for motivation and reward learning. The conclusion is unlikely to be accepted without emphasis and evidence. But evidence such as that reviewed above demonstrates that incentive salience attribution to Pavlovian reward cues is a very powerful mechanism of reward learning in animals, and may be also be influential aspects of human reward and motivation (e.g., addiction).

Still, incentive salience mechanisms operating by Bindra–Toates rules do not work alone. Cognitive expectation of a reward for goal-oriented action is surely another important mechanism for human incentive motivation, and quite possibly for animals too, and is far more accessible to the inner eye of human introspection. Cognitive expectation of a future reward is the form of incentive learning that comes naturally to mind for one who is asked, What does it mean to work for an incentive? The commonsense or *rational* meaning is that the incentive is known in advance, that the incentive and its hedonic value is held explicitly in mind as a declarative cognitive representation by the seeker during the work, and that the action is coordinated specifically in order to attain the hoped for goal. Incentive motivation in this highly cognitive sense clearly happens in us many times each day. Cognitive incentive learning may happen in animals also, but has been less tractable to verification or experimental analysis in animals for the obvious reason that we have no access to animal introspection and must rely on nonverbal tests of cognition. Yet the study of cognitive forms of incentive motivation can still be approached indirectly in animals, and thus its basic mechanisms can be probed with rigorous experimental tools. Foremost in attempts to examine the psychological properties and brain substrates of cognitive incentive learning in animals has been the efforts of Anthony Dickinson and his colleagues, especially Bernard Balleine.

A. Cognitive Expectation of Incentive Value Based on Past Hedonic Experience

Building on a scheme sketched originally by Tolman (Tolman, 1949), Dickinson (1989) defined cognitive incentive learning by several features that

can be demonstrated in goal-directed behavior. He and his colleagues have argued that if one learns cognitively how to gain a particular incentive, then this must entail understanding of the causal relationship between working (as a cause) and gain of the incentive reward (as a consequence). In other words, in the cognitive sense of incentive motivation, one knows that one's effort can produce the incentive. Dickinson uses the term *"act–outcome representation"* to denote cognitive understanding of the causal relationship. Once one has learned to represent the act–outcome relationship between a particular action and the incentive it produces, then it may be the case that when one engages specifically in that incentive-related action, one does so with the *expectation* of earning the reward. Whether this description is true or not can be tested experimentally by probing whether the individual takes into account new information regarding the value of the reward. An experimenter can ascertain under what conditions expected reward value equals the reward's true value.

A cognitive expectation of reward, according to Dickinson and Balleine (1995), is what gives the representation of a stimulus *incentive value* in the everyday and fullest sense of the term. The representation of a stimulus has these properties if "instrumental behavior is mediated not only by a representation of the action-outcome relation, but also by a representation of the incentive value of the outcome, or what in common parlance would be referred to as the desire for the outcome" (Dickinson & Balleine, 1995, p. 163). This is a cognitive sense of incentive value, not a Pavlovian or Bindra–Toates sense, and is more complex than incentive salience "wanting."

Dickinson's work and related studies have shown that animals seem to have the rational sense of incentive learning too. Even rats can be shown to expect particular outcomes, and to govern their instrumental performance based on their expectation. One remarkable thing is that the explicit cognitive expectation sense of incentive learning (responding with cognitive expectation to gain the reward) and the Pavlovian incentive salience process (directly "wanting" and pursuing the conditioned cues of reward) both seem to exist simultaneously. They usually guide behavior together in the same direction: towards the goal. But under certain conditions they guide behavior in different directions, and in those situations the two processes can be pitted against each other to better study them. Sometimes one wins, sometimes the other. Surprisingly, Dickinson and colleagues have shown that the rational sense of incentive learning is in one way *stupider* than the Pavlovian form of incentive learning. Or rather, to put a more positive gloss on it, the cognitive mechanism is rational: it expects an incentive always to be as good as it has been in the past. Sometimes this rational expectation is wrong. It is wrong chiefly when the hedonic value of the

incentive is suddenly changed. Under those conditions, the Pavlovian mechanism may have an advantage.

B. Pulling Apart Cognitive Expectations from Pavlovian Incentive Salience

In a typical experiment, Dickinson and Balleine and colleagues have assessed whether an animal has a cognitive representation of a reward by asking whether it can act appropriately when it is suddenly placed in a new motivational state. For example, if a rat had learned while hungry to perform two instrumental responses, one for sucrose solution and another for food pellets it might be tested in a state of thirst or in a state of caloric satiety, or after one food had been associatively paired (in a different setting) with LiCl illness to induce a taste aversion. Any of these manipulations would change the relative hedonic values of the foods. Two features of the new state test are particularly important in these experiments in order to reveal the role of the cognitive act–outcome expectation. First, the rat is tested in extinction, so that operant responses no longer earn the actual food rewards. The rat must therefore choose to work based solely on its *representation* or expectation of the rewards, because the food reward itself does not arrive, and the rat never gets to *experience* its altered hedonic value. In many experiments, the rat's expectation of a food's hedonic value appears to be based directly on the explicit hedonic experiences it has had of the food in the past. Second, Dickinson and Balleine take pains to arrange the experimental situation so that potential Pavlovian CSs predict both foods equally well. This obviates control of behavior by Pavlovian-related motivational processes, which otherwise could guide behavior to one goal or the other (and which would be more related to the sense of incentive salience and to the Bindra–Toates sense of incentive learning).

Under these conditions, Dickinson and Balleine have shown that rats quite often fail to appropriately modify their instrumental behavior for a newly revalued food, when the rats are forced to act on the basis of their expectations. Instead they continue mistakenly to expect the food to have the same hedonic value it had before. If a food had high hedonic value when it was experienced during training (e.g., while hungry), then the rat often continues to work hard in the test—even if the food would now have reduced hedonic value (e.g., either because now the rat is sated or because food has subsequently received one conditioned aversion pairing with LiCl) (Balleine & Dickinson, 1991; Dickinson & Balleine, 1994). If a rat had not worked vigorously before, because the food did not have great value during training, it still does not work harder during the test, even though the food would now have enhanced hedonic value (e.g., because physiological

deprivation is now higher, or because a benzodiazepine drug has been given that increases food palatability) (Balleine, 1992; Balleine, Ball, & Dickinson, 1994; Dickinson & Balleine, 1994). The rat's expectation of reward apparently is that the reward will be the same as before—even though now it won't. In these situations a rat can be said to have a cognitive expectation of reward in one sense, but the expectation is an incorrect one. The rat has not yet fully learned about the new hedonic value of the food (its cognitive representation is not updated), and its behavior is not guided by an accurate cognitive representation of the food at that moment.

Dickinson and colleagues have found a crucial way in which a rat can be provided with information sufficient to correct its mistake, allowing it to change its expectation-driven instrumental behavior appropriately to the new incentive value of the food or water reward. What the rat needs is to actually *experience the new hedonic value* of the reward *while it is in the changed state* of thirst, hunger, drug-state, or after a single taste-LiCl pairing (Balleine & Dickinson, 1998; Dickinson & Balleine, 1994). This would ordinarily happen naturally if the test were not conducted under extinction conditions. Then the rat would simply sample both rewards, quickly experience which one was better in the new state, and modify its behavior accordingly. But it is also sufficient, Dickinson and Balleine show, simply to allow a rat to experience one taste of the new hedonic value of the food anyplace else, such as in its home cage (as long as the rat is in the same physiological state as to be tested later). If the rat has experienced the new hedonic value prior to the instrumental extinction test, it can later employ the now known value of the food in the cognitive expectation it has of the food's value, and of the food's causal relationship to the two different actions. It then modifies its instrumental response in the extinction test appropriate to the revalued food. Other instrumental responses, used to obtain another reward that was not revalued, are not altered. For Dickinson and colleagues, this selective and intelligent change in behavior demonstrates true incentive learning in the common sense or rational meaning of the phrase. It demonstrates that a rat knows in a cognitive sense what it is working for and how to get it.

C. Semipermeable Boundary between Cognitive Expectation and Pavlovian Incentive Salience

The distinction described above between cognitive expectation of incentive value and Pavlovian incentive salience of CS has emerged quite clearly in many experiments conducted by Dickinson and his colleagues. Cognitive expectation appears to be based squarely on past experience of the incentive value. Incentive salience attributed to Pavlovian CS, on the other hand,

follows Bindra–Toates rules and can integrate new physiological states with associative information even in advance of explicit experience of a new hedonic value.

However, it should probably be acknowledged that the division between the two forms of control is not absolute. In a number of other experiments, leakage between the processes seems to have occurred, and integration in advance has been observed even in behavior apparently guided by the cognitive mechanism of act–outcome representations (Rescorla, 1994a, 1994b; Shipley & Colwill, 1996). For example, in contrast to the findings of Dickinson and his colleagues (Balleine & Dickinson, 1991, 1992; Lopez, Balleine, & Dickinson, 1992), Rescorla (1992, 1994b) reported that rats did suppress their instrumental performance for a food (under extinction conditions) directly after they had had a single pairing of a taste with LiCl illness (i.e., *before* they had ever had a chance to experience the newly aversive palatability of that food, which would have required a second food presentation). Similarly, in a hunger-to-thirst transfer, Shipley and Colwill (1996) found that when rats that had learned while hungry to work for sucrose solution and for food pellets were later tested in extinction while thirsty, they would directly work more for the solution—before experiencing the new hedonic values of pellets and solution in the thirsty state. That is again different from the hunger-to-thirst results of Dickinson and colleagues (Dickinson & Dawson, 1987). Yet even Dickinson and colleagues themselves find that expectation-guided behavior in a few conditions is able to integrate in advance, without need of the usual new hedonic experience, for example after shifts from thirst to hunger (Dickinson & Balleine, 1990), though not after shifts from hunger to thirst (Dickinson & Dawson, 1987).

Thus there appears to be at least some ability even in rats for the cognitive mechanism to skip over the usual rationalist procedural assumption that the future will reflect the past, and instead to integrate associative and physiological information in advance of experiencing the new hedonic value, as the Bindra–Toates conditioned incentive salience mechanism automatically can do. This does not mean that the distinction is not real between cognitive expectation of reward and attribution of incentive salience to Pavlovian CS. As Shipley and Colwill (1996) noted regarding the difference between their result and Dickinson's, "it is not impossible to reconcile our finding of a direct effect of outcome revaluation on instrumental performance with the general thesis proposed by . . . Dickinson and his colleagues" (p. 65). There are several reasons why the cognitive mechanism might sometimes be able to make special use of information "in advance" yet still be distinct from other psychological incentive mechanisms, as Dickinson suggests.

First, as Shipley and Colwill (1996) point out, in some cases the cognitive expectation might not have to be readjusted in advance after all, but rather might make use of past experiences that were not intended by the experimenter. For example, their rats may have previously been thirsty and have experienced the increased hedonic value of the taste of water when drunk in a thirsty state. If so, they might have generalized that previously gained information about hedonic experience to the sucrose solution when they were thirsty.

Second, if a manipulation of incentive value produces an explicit hedonic experience by itself, then that hedonic experience should revalue the cognitive expectation of future hedonic experience. For example, when Rescorla (1992) found that a single pairing of a food with an injection of hypertonic LiCl reduced subsequent performance for that food (even before the rats were able to taste the now-aversive food), Balleine and Dickinson (1992) argued that the hypertonic injection had produced pain, a direct unpleasant experience (hypertonic solutions are more concentrated than body fluids and irritate pain receptors; isotonic solutions do not produce this irritation pain and so are usually used for injection instead—as Rescorla [1994b] did in a later replication). Pairing of the food with pain would have given the new (negative) hedonic experience needed by the cognitive mechanism to revalue its act-outcome expectation. It is probably worth mentioning that ordinary taste aversion learning produced by isotonic LiCl works because it makes the next experience of the food taste bad, according to Dickinson and colleagues, not because the rat carries an explicit association of the unpleasant nausea state. Presumably the nausea is too delayed to enter into an explicit cognitive representation with the idea of the food (even though it enters into a Pavlovian association). The disgusting experience of the unpleasant taste, when next encountered, is the immediate and explicit (negative) hedonic event that is usually needed to reduce the cognitively labeled incentive value of the taste.

Third, if Pavlovian cues are present in a way that can selectively guide a response, then Bindra–Toates incentive salience should be attributed to those reward cues, to control behavior in an incentive-appropriate fashion even in advance of experiencing the new consequences. As we saw earlier, the Bindra–Toates incentive conditioning mechanism can integrate physiological state to appropriately change "wanting" and "liking" for reward cues as soon as the physiological shift occurs, without need of experiencing the new hedonic value of the S* reward. That is enough in many cases to guide goal-directed behavior. So for example, Krieckhaus and Wolf (1968) found that rats that had learned that a concentrated salty solution was to be found from a certain spout when later made sodium deficient went directly to obtain the solution from that spout. Because attribution of

incentive salience to Pavlovian cues, such as a salt spout, can always be expected to follow integrative Bindra–Toates rules, any situation that has such conditioned incentive cues available will be able to guide behavior, and perhaps overrule or influence the cognitively expected hedonic value.

In general, the cognitive incentive expectation and Bindra–Toates incentive salience attribution mechanisms usually act to guide behavior in the same direction, not different directions. It is only in special cases, as when hedonic value is suddenly shifted and not yet re-experienced, that divergence between the mechanisms can be expected to happen at all. The two processes operate ordinarily in close association, and information leakage or communication between them might sometimes occur. The anticipatory integration of information that ordinarily drives the Bindra–Toates condition incentive salience attribution may under some conditions—not yet identified—be able to permeate the boundary and to affect the cognitive expectation even before the incentive is encountered again. We must accept for the moment that we do not yet understand what factors gate the access of the cognitive expectation to anticipatory integration. Presumably, cognitive access to Bindra–Toates integration might occur more often in humans than in rats, but there is surprisingly little evidence on the matter. When can cognitive expectation integrate in advance? When does it remain tied to the rationalist prediction that future value will equal past value? The answers to these questions remain for future research. For now, the best conclusion may be that there is a fundamental difference between these two mechanisms of reward learning, but that the divide between them can occasionally be bridged.

VII. Brain Mechanisms of Reward Learning

It is not my purpose to provide here a review of brain mechanisms of reward learning (for excellent reviews see Everitt et al., 1999, in press; Robbins & Everitt, 1996; Rolls, 1999; Schultz, 1998; Shizgal, 1999). Instead I only wish to touch on a few studies of brain substrates that are especially relevant to the distinction between cognitive incentive expectation to and the Bindra–Toates attribution of incentive salience to CS. Recent evidence indicates that these two processes differ not only psychologically but also in terms of their brain mechanisms, giving further justification for considering them as distinct. Incentive salience 'wants' for Pavlovian reward cues appear to be mediated primarily by the mesolimbic dopamine system and its forebrain targets (Balleine & Killcross, 1994; Dickinson, Smith, & Mirenowicz, 2000; Wyvell & Berridge, 2000). Cognitive act–outcome expectancy appears to be more directly mediated by specific cortical regions, such as the insular cortex and the prefrontal cortex (Balleine & Dickinson, 1998).

A. Mesolimbic Dopamine Systems Mediate Bindra–Toates Incentive Salience Attribution

Regarding reward learning, the brain system that has received the most attention in the past decade is the mesolimbic dopamine system, or dense projection of dopamine neurons from the midbrain's tegmentum to the forebrain's nucleus accumbens (and prefrontal cortex and amygdala to a lesser degree) (Beninger & Miller, 1998; Braver & Cohen, 1998; Montague, Dayan, & Sejnowski, 1996; Phillips et al., 1992; Schultz, 1998; Schultz, Dayan, & Montague, 1997; Vaccarino, Schiff, & Glickman, 1989). Mesolimbic dopamine neurons are activated by a variety of hedonic rewards, including palatable foods, sexual copulation, and drugs such as cocaine, amphetamine, or heroin, in a way that seems related to the incentive value of the reward (Di Chiara, 1998; Koob, 1996; Phillips et al., 1992; Wise, 1985, 1998). More relevant to reward *learning,* mesolimbic dopamine neurons and their accumbens targets are also activated by Pavlovian CSs for rewards (Di Ciano, Blaha, & Phillips, 1998; Fiorino, Coury, & Phillips, 1997; Schultz, Tremblay, & Hollerman, 1998). In many instances where predictive Pavlovian cues exist, the dopamine and accumbens neurons are activated before the hedonic reward occurs.

The predictive or anticipatory nature of mesolimbic dopamine responses has led to suggestions that this is a specialized brain substrate for reward learning (Montague et al., 1996; Schultz, 1998). These models have typically proposed that dopamine neurons play two roles in reward learning. First, the models suggest that dopamine neurons mediate the predictive *expectation* of a reward triggered by a CS. Second, they suggest that dopamine neurons play a role in *"teaching" new predictions* by signaling the occurrence of outcomes that deviate from the expected. Generally these models have not attempted to choose between the Pavlovian incentive type versus cognitive incentive type of predictions, or to at all specify the precise psychological nature of the predictive expectation they invoke. However, the results of several studies indicate that the specific psychological nature of the expectation mediated by mesolimbic dopamine systems best corresponds to the Bindra–Toates sense of incentive salience attribution to CS—and not the cognitive sense of act–outcome expectation (Balleine & Killcross, 1994; Dickinson et al., 1999; Wyvell & Berridge, 2000).

B. Mesolimbic Dopamine Substrates of Pavlovian Incentive Salience

In one study, Dickinson et al. (2000) trained rats in the Dickinsonian paradigm that allows separation of cognitive incentive learning from Pavlovian incentive learning. They then tested the effects of drugs that suppress the neurotransmission of mesolimbic dopamine systems. They gave rats a

dopamine antagonist drug that blocks neural receptors for dopamine (either pimozide or alpha-flupenthixol). Dopamine suppression blocked both the acquisition and expression of Pavlovian incentive learning. If the rats received the dopamine antagonist just before their Pavlovian training trials, in which a Pavlovian CS was paired in a predictive fashion with food reward, they later behaved as though they never had received the Pavlovian training. When the CS was later presented to them as they bar pressed for food under extinction conditions in an undrugged state, they pressed no more than before the CS arrived. By contrast, rats that had been given vehicle during the Pavlovian training trials pressed harder when presented with the Pavlovian CS, as expected. In other words, the dopamine antagonist appeared to have blocked the acquisition by the Pavlovian CS of the conditioned incentive salience that would ordinarily have increased "wanting" for food.

In a follow-up experiment, Dickinson and colleagues (2000) found that the dopamine antagonist similarly prevented the *expression* of conditioned incentive salience attribution to a CS when the drug was given during the final test rather than during Pavlovian training. In this condition, all the rats received the CS–food Pavlovian training in an undrugged state. Later the hungry rats were tested for bar pressing under extinction conditions, while the CS was occasionally presented to them, after half the rats had received a dopamine antagonist drug and the others had not. The rats that were tested in a suppressed state of dopamine transmission failed to show any enhancement for bar pressing when the CS was presented, whereas the undrugged rats did press more during the CS. In other words, dopamine suppression blocked the expression of Pavlovian incentive value that had previously been learned while undrugged. The dopamine antagonists appeared to block the attribution of incentive salience to the CS that would ordinarily have caused cue-elicited "wanting" for the food.

A recent study by Cindy Wyvell in our laboratory, mentioned earlier, further found that conditioned incentive salience can be *enhanced* if mesoaccumbens dopamine systems are stimulated by amphetamine microinjection rather than suppressed (Wyvell & Berridge, 2000). Wyvell found that activation of dopamine neurotransmission in the accumbens, caused by microinjections of amphetamine directly into that brain structure, magnified the incentive salience attributed to a Pavlovian CS (Figure 5). In this study, as in the Dickinson experiments, rats learned to press a bar for food and separately learned an association between a predictive Pavlovian CS and food reward. A microinjection of amphetamine was made into the nucleus accumbens of the rats just before they were tested while bar pressing in extinction. Amphetamine microinjection dramatically magnified the increase in bar pressing caused by presentations of the Pavlovian food CS,

without much changing baseline bar pressing when the CS was not present. In other words, the activation of accumbens dopamine system apparently magnified the ability of the Pavlovian CS to trigger "wanting" for a food reward in a specific conditioned incentive fashion. It seemed to magnify the attribution of incentive salience specifically to the Pavlovian food cue (as the modified Bindra–Toates model of incentive salience would suggest).

The mediation of Pavlovian incentive learning by mesolimbic dopamine systems appears to be restricted to Pavlovian based "wanting" rather than to "liking." Pavlovian conditioning can still cause shifts in the hedonic reaction pattern elicited by a CS even after the mesolimbic dopamine system is removed. For example, Berridge and Robinson found that changes in "liking" caused by a conditioned taste aversion could still be learned by rats that had lost their mesolimbic dopamine system due to lesions caused by a neurotoxin, 6-hydroxydopamine, which destroyed 98–99% of their mesolimbic dopamine neurons (Berridge & Robinson, 1998). We found that rats that had lost their dopamine systems still were perfectly capable of this learned suppression of incentive "liking" caused by Pavlovian conditioning. Rats with dopamine lesions switched their reaction to a sweet polycose-saccharin taste from a positive hedonic pattern (licking of their lips and paws) to an aversive pattern (gapes, arm flails, headshakes, etc.) just as normal rats did. In other words, it is Pavlovian incentive learning, not Pavlovian hedonic learning, that is mediated by mesoaccumbens dopamine systems. Dopamine in the accumbens is needed to "want" a conditioned incentive, but not necessarily to "like" it or to learn new "likes" and "dislikes."

C. Brain Substrates of Cognitive Incentive Learning

Less is known about the neural substrate for the cognitive expectation form of incentive learning than about conditioned incentive forms of reward learning. However, recent studies by Balleine and Dickinson and colleagues suggest that cognitive act–outcome representations of incentive value appear *not* to depend on the mesolimbic dopamine system. Instead, the prefrontal cortex and the insular cortex appear to be more important.

D. Mesolimbic Dopamine Does Not Mediate Cognitive Act–Outcome Expectation

At least two studies indicate that *cognitive representations* of act–outcome relations are *not* mediated by dopamine projections to the nucleus accumbens or by the nucleus accumbens itself. In one study, Balleine and Killcross (1994) found that accumbens lesions did not impair a rat's ability to adjust its level of instrumental response based on a change in reward value.

They concluded that accumbens lesions "do not influence sensitivity to the instrumental contingency" (p. 191) and that the role of the nucleus accumbens is "dissociated from the control of performance mediated by the act-outcome relation" (p. 181). The lesions did impair classically conditioned approach responses directed toward the food cup, consistent with an interpretation that there was reduced incentive salience attributed to reward-related stimuli such as the food cup, and Balleine and Killcross accordingly interpreted their accumbens lesion deficit as reduced "conditioned affective arousal produced by classical conditioning" (p. 191).

Second, in the dopamine antagonist study described earlier, Dickinson and colleagues (2000) tested the effects suppressing dopamine projection systems on cognitive incentive value versus Pavlovian incentive value. They gave rats dopamine-suppressing drugs that suppressed Pavlovian incentive salience. In this experiment they gave the drug under two conditions that would be expected to determine the rats' experienced-based expectation of the food's hedonic value. First, they let some rats eat the food while under the influence of the drug (but while outside of the instrumental test chamber), which allowed them to experience any hedonic consequences of the drug on food reward. Dopamine blockade failed to suppress the cognitive incentive value, or act–outcome representation, of the food for the rats. When the rats were allowed to eat food under the influence of the dopamine-blocking drug and thus gain an explicit representation of the remembered hedonic impact that food had in the drugged state, that memory did not reduce their bar pressing for the food later when they were tested under extinction conditions. In these conditions, food was no longer delivered, and so the rats had to operate entirely on the basis of their memory of the past food. Apparently, the incentive expectation based on that memory had not been diminished by the dopamine-blocking drug.

Dickinson and colleagues gave other rats in the same experiment the dopamine antagonist while they were being tested on the instrumental task under extinction conditions—that is, while they had to rely on their expectation of incentive value to guide their behavior. Again the dopamine antagonists failed to suppress the cognitive incentive value. Even when the rats were tested directly under the drug's influence (Dickinson et al., 2000), they still pressed at a normal rate under extinction conditions. Even though the dopamine antagonist would have suppressed bar pressing if the food were actually earned by a bar press (when it could disrupt the reboosting of incentive salience to its conditioned representation), it did not suppress the purely cognitive *expectation* of incentive value.

In other words, dopamine blockade suppresses the incentive motivation of rats to work for real food, if they experience the food as they are working, and also suppresses the Pavlovian incentive ability of cues to stimulate

motivation. But dopamine blockade apparently does not suppress the cognitive incentive expectation of the same food reward, which is based upon explicit *memory* of the food's hedonic value and on a cognitive representation of the act–outcome causal relation. As Dickinson and colleagues put it, "the absence of the effect of DA [dopamine] antagonists on incentive learning suggests that the incentive values of food rewards that control instrumental responding through a representation of the action-outcome contingency are mediated by a system that is not strongly modulated by a DA input" (Dickinson et al., 2000). That is, cognitive expectations of incentive value must be mediated by other brain systems.

E. Prefrontal Cortex and Insular Cortex Mediate Cognitive Expectation of Incentive Value

The brain systems that mediate cognitive incentive value are just beginning to be understood. So far, Balleine and Dickinson's studies have implicated structures such as prefrontal cortex and insular cortex in cognitive incentive expectation (and neurochemical substrates such as benzodiazepine and cholecystokinin systems that may mediate the experienced hedonic value of a food, which forms the explicit memory basis of expected hedonic value) (Balleine et al., 1994; Balleine & Dickinson, 1998).

The human and primate prefrontal cortex contains a dorsolateral region that has been especially implicated in act–outcome planning and related executive functions (Damasio, 1996; Niedermeyer, 1998; Robbins, 1996; Smith & Jonides, 1999), and in rats the corresponding region of prefrontal cortex is called the prelimbic area. Balleine and Dickinson made prelimbic lesions in the prefrontal cortex of rats that learned to perform two responses (press a bar, pull a chain), each of which earned its own specific food reward (Balleine & Dickinson, 1998). Then the rats were allowed to eat their fill of one of the foods elsewhere, before being tested on the two responses under extinction conditions (remember again, when neither response produced real food, so the rats were forced to operate solely on the basis of their expected incentive values). Eating a food to satiety is well known in humans to cause a temporary decrease in appetite and palatability ratings for that particular food, a phenomenon called *sensory-specific satiety* (Hetherington, 1996; Rolls, 1986). Rats also show a sensory-specific satiety decrease in hedonic behavioral affective reactions to a sweet taste just after they have consumed a large meal of it (Berridge, 1991). Thus, Dickinson and Balleine could expect that their rats would experience reduced hedonic value of the particular food they were sated upon by the end of their large meal of it. When tested immediately afterwards, normal rats did not work much on the response associated with the food they had just become sated

on, but they still continued to work robustly on the response associated with the other food. Rats that had prelimbic prefrontal cortex lesions, by contrast, failed to work hard at either response (Balleine & Dickinson, 1998). Damage to the prefrontal cortex appeared to impair the ability of rats to generate selective goal-directed behavior, when faced with multiple tasks carrying multiple outcomes, based on their expectations of relative hedonic value. It is interesting to note that human patients with damage to the prefrontal cortex have been to reported to lack an anticipatory galvanic skin response that normal people show when faced with a risky choice in certain situations (Bechara, Damasio, Tranel, & Damasio, 1997). This could be construed as a failure by these humans to generate an appropriate predictive representation of the causal relation between an act and its outcome's affective value. Conceivably there could be a link between prefrontal cortex mediation of the anticipatory prediction of emotional outcomes in humans, reflected in human anticipatory autonomic responses, and the type of outcome value expectation posited by Dickinson and his colleagues for rats (though it should be acknowledged that the brain lesions, tasks, and response measures, as well as the species involved, are all quite different in the two experimental paradigms).

The insular cortex, by contrast, is a cortical region involved in gustatory processing and has been shown to be important in mediating learned taste preference and avoidance (Dunn & Everitt, 1988). When Balleine and Dickinson (1998) made lesions in this area and tested rats in a similar experiment, rats with insular cortex lesions still worked under extinction conditions, apparently guided by an intact expectation of reward. However, after eating one food to satiety, they continued to work robustly on both responses under extinction conditions, and did not reduce responding on the task associated with the pre-fed food (Balleine & Dickinson, 1998). This was not due to an inability to experience sensory-specific satiety, as Balleine and Dickinson found that rats with insular cortex lesions showed a normal selective decline in responding after prefeeding if the two responses actually still earned the two foods (i.e., not extinction). The insular cortex lesions only prevented the *satiety devaluation* when tested under conditions that forced the rats to rely solely on *expectations* regarding the two foods. Balleine and Dickinson concluded that "what is impaired, however, is the ability to store and retain information about the changed value" (Balleine & Dickinson, 1998, p. 415).

Although much remains to be done to clarify the brain systems that mediate cognitive forms of incentive learning, these results suggest that prefrontal and insular cortical regions may play special roles. Balleine and Dickinson suggest that, in particular, the prelimbic prefrontal area may mediate learning about the causal contingency between act and outcome,

whereas the insular area may be needed to mediate cognitive expectation of the hedonic value of a particular food reward that is based on previous hedonic experience.

VIII. Conclusion

Many processes have been considered over the years as potential psychological mechanisms for reward learning. First of all, *reinforcement* concepts have been advanced in several forms. These include most notably stamping in accounts of a S-R habit via a mechanism triggered either by a hedonic satisfier or by drive reduction. Reinforcement concepts still have detectable influence on research and theory today, in fields that range from behavioral neuroscience to human motivational psychology. Next, *conditioned incentive* concepts of reward learning and motivation have become viewed as increasingly important, from the S-S* associations posited by Bolles (1972), so important in guiding goal-directed behavior, to the acquisition by CS of flexible and state-dependent incentive properties posited by Bindra and Toates, and finally to the splitting of "wanting" from "liking" and the attribution of conditioned "wanting" to cues posited by the incentive salience account. Finally, we have seen *cognitive expectation* mechanisms of hedonic-laden incentive outcomes. These concepts posit motivated behavior to be guided via representations of a causal relation between an act and its expected hedonic consequences, which has been constructed on the basis of explicit past experiences.

This diversity of potential mechanisms reflects more than changes in the theoretical orientation of psychologists over the decades and more than the existence of multiple schools of thought. It reflects a real diversity in the underlying psychological phenomena. Regarding S-R reinforcement, many experimental demonstrations, beginning with Watson's "kerplunk" experiment, leave no room to doubt that S-R habits indeed exist. Similarly, many demonstrations show that Pavlovian CS for rewards take on powerful incentive properties. Pavlovian reward cues and their representations elicit desire and are often needed for motivation. Conditioned cues themselves become the target of "wanting," and become triggers for conditioned "liking." They are sought after and even sometimes consumed. These Bindra–Toates incentive properties are by no means restricted to animals: from the ability of a taste of food to prime appetite for that food in normal humans to the intense, irrational, and overpowering cravings of drug addiction, there are numerous phenomena in daily life to testify to the power of incentive conditioning processes in the control of human motivation and behavior. Finally, because cognitive expectations are of all these processes the most

accessible to human introspection, as well as because of the focus of contemporary psychology on cognition, we need little to convince ourselves that cognitive incentive expectations form the basis of much of our daily behavior and of nearly all the motivated plans of which we are aware.

The point is that all these processes of reward learning and motivation exist simultaneously as psychological processes. They are all within each of us, operating in parallel and usually in cooperation. They diverge from one another under limited circumstances, and when that happens they may compete for control of behavior. Sometimes one process wins, sometimes another. We should not aim to dissolve all reward learning into one type of explanation, whether it be cognitive or associative, or reinforcement or incentive in nature. Future progress in the psychology of reward learning and of appetitive motivation will instead be best gained by a clearer understanding of the nature and features of each of these psychological processes, their relation to brain mechanisms, and the rules that govern interactions among them.

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