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Central Theories of Motivation and Emotion

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The concept of emotion has aroused extreme theoretical positions: from Skinner's (1953) denouncement of it as a muddle-minded causal fiction to the view that it is fundamental to the whole of psychology (Panksepp, 1998). Although it is more than 120 years since William James (1884) asked, "What is an emotion?" the question proved so difficult to answer that for a long period the word *emotion* virtually disappeared from psychology textbooks and even from more specialized books on learning or cognition. For those with a strongly behaviorist perspective, there might seem to be no reason to regret this; nor, indeed, to concern yourself with theories, central or otherwise, of emotion and motivation. For those focusing on cognitive processes also, motivation and emotion may seem peripheral. However, we believe that behavioral observations can best be integrated, and cognitive processes best understood, if we see behavior as the result of activation of one or more of a set of distinct hierarchically organized systems in the brain, where each system has evolved under pressure from a different specific class of adaptive requirements. Critically, we believe we can identify the resultant emotion, and associated motivation, with the general adaptive function that defines a class of behaviors even when the specific behaviors produced differ across occasions or species. By this route, we can achieve theoretical integration along the phylogenetic scale. The emotion systems controlling such behaviors, and their interaction with cognitive processes, such as working memory, have now become the subject of intense and detailed study (LeDoux, 1993).

These adaptation-specific (emotional) systems are also connected with two general systems that control approach and avoidance motivations, respectively—as well as a third system that resolves conflicts between these motivations. In this context, *motivation* is an ambiguous term. A motivation (e.g., thirst) is specific and distinct from other motivations (e.g., hunger). But the specificity is most obvious in terms of elicited behavior and, when we talk about motivation rather than emotion, we are most often thinking of it in terms of general approach and avoidance tendencies, or positive and negative reinforcement, ignoring the specific nature of the reinforcer. Further, it is variation in the sensitivities of the systems that control positive and negative affect generally that appears to make the greatest contribution to human personality and to the risk of psychopathology—areas of human psychology where we clearly see the importance, or at least the prominence, of emotion and motivation.

In this chapter, we present emotion as a cluster of reactions, including motivation, that are linked to specific classes of affordances (the aspects of and object or situation that make certain actions available) of stimuli in the world—where both the nature of the external stimulus and the animal's internal state combine to determine the precise affordance at any particular point in time. In the process, it will be necessary to consider neural plasticity resulting from:

- *Simple association:* Where no specialized reinforcer is required to generate plasticity and where behavior undergoes relatively little modification but engages in stimulus substitution;
- *Stimulus-reinforcer pairing:* Where the result will often be observationally classical conditioning, but where response to the conditional stimulus may not be the same as those to the unconditional stimulus, and where the result can also be observationally instrumental conditioning; and
- *Stimulus-response-reinforcer pairings:* Where the result will be observationally instrumental conditioning.

Particularly in this latter case, learning itself is initially associated with strong emotional reactions but well-learned responding need not be. Thus, there is a strong link between emotion and motivation (with the latter apparently embedded in the former). But, emotional reactions have many semi- or actually independent parts and so, at the limit, all that may apparently be left is a motivation. The relation between motivation and emotion, as linguistic terms, may be murky but, as we shall see, the phenomenology, and the use of the terms, can be anchored through central (neurally based) theories.

VALUE OF CENTRAL (NEURALLY BASED) THEORIES

Recently, rather than being the topic that cannot be named, emotion (often without any definition of the term) has become a focus of study of a wide variety of phenomena in behavioral neuroscience. But there is still no consensus as to what an emotion *is* (and, as we shall see, the term may not refer to any single coherent internal entity). Motivation is also not clearly defined. The root of both words implies that the construct being referred to is something that produces movement—and yet most psychologists contrast emotion with motivation. Despite this, it is difficult to think of motivationally significant stimuli that are not characterized by the capacity to elicit emotion. In this chapter, we hope to show that a neuroscientific approach can clarify the nature of emotion, motivation, drive, and related constructs in ways that, if not impossible for a purely behaviorist approach, are at least very difficult if all that is measured is behavior.

The focus of this chapter on *central* theories of motivation and emotion is to a large extent predicated on taking a neuroscientific approach. Behaviorally-based theories of, for example, a *central motivational state* have been proposed in the past (Bindra, 1969). However, the dissection of the parts of which emotional and motivational reactions are composed and the linking of those parts into coherent, predictive theory is very difficult with purely behavioral methodologies. By contrast, a neuroscientist can, often literally, dissect classes of behavior and their control systems. They can also do so without first defining, or even proving the existence of, the higher order entity that they are dissecting. If a particular drug or brain lesion changes one set of behaviors, but not another, then clearly these sets represent different functional classes. That said, proper behavioral analysis will also then be required to determine the functional nature of the classes that have been so separated.

Neurally grounded theories of emotion and motivation have the key advantage, then, that they are anchored in specific anatomically identifiable systems. Their accounts do not depend on the superficial characters of behaviors and, indeed, can treat superficially quite different behaviors in different species as homologous. Neural homology and evolutionary (functional) homology, therefore, go hand in hand. When one is discerned, the other can usually be discovered—and vice versa. Evolutionary (and thus psychological function) become, then, things that must be extracted from the nature of known neural systems. With this approach, the definition of a psychological construct should map to a specific aspect of a coherent neural and functional system. In some cases, achieving this mapping requires elimination of an older psychological term and creation of a new one.

However, neural analysis cannot proceed by itself. While it can anchor and dissect constructs derived from the experimental analysis of behavior and from ethological analysis, the brain is so complex that, without preliminary behavioral analysis, functional systems cannot easily be identified. Neural analysis of circuits that show lateral inhibition, for example, allows explanation of a wide range of sensory illusions—including those where the presence of lateral inhibition in the relevant circuits is inferred rather than directly measured. However, one could not have easily predicted any of these illusions (or any aspect of our experience of "normal" perception) from the simple observation of lateral inhibition at the neural level.

So, central theories of emotion and motivation are the result of continuous interaction between behavioral and neuroscientific approaches. The neuroscientist provides anchors and mechanisms for genuine central (nervous system) theories of motivation and emotion; but, when these theories are properly developed, they are also central theories from a more psychological perspective. The patterns of activity in their higher order neural elements are central cognitive and emotional states. The behavioral neuroscientist, then, can integrate behavioral observations in terms of higher order internal states (something that all but the most radical behaviourist would see as desirable), but does so in terms of direct measures of those central states and so avoids the problems (which drove the development of the radical behaviourist philosophy) of inferring specific complex central states solely from patterns of behavior or, worse, introspection.

Perhaps the most important feature of central theories of motivation and emotion for higher-level psychological analysis is one that is usually implicit rather than explicit at the level of neuroscientific analysis. Central motivational-emotional states need to be viewed at the neural level as complex compounds. This is true in two senses. On the one hand, they are complexes of emotional reactions and motivation: initial elicitation of emotional reactions also generates motivation; but, particularly with well-learned responses, motivation can drive behavior in the absence of major emotional reactions. On the other hand, an emotion can be the result of parallel independent processes rather than of output from a single central control system. It will be seen, later, that current central theories share a tendency to see the critical elements of neural/cognitive processing as "goals." Neither purely cognitive nor purely emotional/motivational attributes are given primacy; and simple stimulus-response reactions are rejected. The key drivers of behavior are seen as cognitive-emotional compounds (Hinde, 1998).

ROAD MAP TO CENTRAL THEORIES OF EMOTION AND MOTIVATION

We start with the esoteric and microscopic. We look at the bits and pieces from which evolution has formed emotions and motivations. We then move to the general, the basic reinforcement systems through which the stimuli that elicit highly specific "fixed action patterns" can, through learning, shape general, flexible, emotion-independent behavior. We then compare and contrast some current central theories of emotion and motivation that amalgamate these specific and general aspects of behavioral control. Finally, we indulge ourselves—and hopefully show that our previous dry, didactic analysis has significant mundane applications—by looking at some possibly unexpected implications of current central theories.

EMOTION, MOTIVATION, AND EVOLUTION

The behavioral neuroscientist thinks in terms of specific neural networks that deliver, often complex, patterns of behavior in response to appropriate environmental circumstance. Such networks cannot appear in evolution or development fully formed. They must result from progressive, incremental changes. In evolution, these changes occur as the result of random mutations interacting with selection pressures. As mentioned earlier, we would equate a specific emotion with the nature of the consistent selection pressure (functional requirement) that has driven the evolution of a set of reactions. But this means that the underlying control of behaviors (and other, e.g., autonomic, reactions) need not map simply to their superficial organization.

Evolution and "Rules of Thumb" (ROT) as a Problem for Behavioral Analysis

The selection pressure driving evolution can be understood in terms of models such as those of optimal foraging theory. These are theoretical analyses that determine the behavioral rules required to maximize such things as the amount of food that an animal can obtain given specific starting assumptions about the environmental constraints (McNamara & Houston, 1980). It should be noted that these analyses are not predictions as to the rules that an animal will use, but define the boundary conditions toward which an animal should evolve if there is sufficient mutation and if selection of advantageous mutations is not blocked in some way.

The important concept here is that the animal can use rules of thumb (ROT) of a relatively simple sort to achieve behavior, under normal ecological conditions, that approaches optimality—but where, in phylogenetically unusual conditions, responding may be suboptimal. For example, the parasite *Nemeritis canescens* "allocates its searching time in relation to host density approximately as predicted by an optimal foraging model [but] the decision rule used by *Nemeritis* . . . is a simple mechanism based on habituation to host scent—a far cry from the Lagrange multipliers and Newton's iterative approximations used by the theorist to solve the problem" (Krebs, Stephens, & Sutherland, 1983, p. 188).

ROT originate because, in the absence of any adaptive behavior, any mutation that results in any increase in adaptive value, however limited, will be selected. A later mutation can then provide a further increase in adaptive value—and so on. The result is that emotional control mechanisms may involve both serial and parallel ROT. In some cases, specific ROT may produce conflicting responses to the same stimulus (freezing and escape when faced with a threat, for example). These present no problem for behavior analysis as the distinct behaviors can be analyzed separately. In other cases, specific ROT may not conflict but may nonetheless fulfill quite different functions (increased blood-clotting factor is only required if escape is not successful). Again, because the responses are different, they can be identified as such and analyzed separately. The critical problem for behavior analysis is that in some cases multiple ROT can deliver essentially the same superficial behavior. They then provide the appearance, but not actuality, of a single generalized pattern of adaptive responding resulting from the application of a single, higher order, functional rule. This is exemplified by the partial reinforcement extinction effect.

The Partial Reinforcement Extinction Effect (PREE) and Serial ROT

The partial reinforcement extinction effect (PREE) is a greater persistence of responding in extinction after prior training on partial (intermittent) reinforcement than after prior training with continuous (consistent) reinforcement. It is one of the more reliable phenomena in behavioral analysis. McNamara and Houston (1980) analyzed the general problem of how long to persist when responses no longer yield rewards. They looked at the specific case (which occurs with extinction of any positively reinforced response) of a number of initial responses that are rewarded with some probability p that are followed by a number of later responses that deliver no reward. The response is assumed to have some cost (e.g., loss of energy in making the response).

Absolute optimality (which cannot be achieved in the real world without precognition) is to cease responding as soon as reward is no longer available. The theoretical

optimality problem is, then, to determine the rule that defines the point when an animal should decide that reward has actually become unavailable rather than the alternative possibility that it is faced with an unusually long run of nonrewarded responses in a sequence with average probability *p*. The precise answer to this question depends on the cost of responding and the value of *p*. Under realistic conditions, the value of *p* is not known and so it must be estimated from the pattern of rewards. Further *p*—reward value and even cost value—are likely to vary from response to response. This presents a highly complex set of adaptive requirements. However, it turns out that "regardless of the exact [values of these parameters], the optimal policy for this sort of problem involves persisting for far more trials in the face of failure if [the original] *p* [of reward] is low. This provides an explanation of the PREE in terms of optimality theory" (McNamara & Houston, 1980, p. 687).

The explanation of the PREE by optimality theory is not a mechanistic explanation. It is, rather, a description of the general functional requirements that provide a background against which any mechanism that results in persistent responding will be selected. It is not a prediction as to how an animal will actually solve the problem. Further, it does not give us any insight into what ROT the animal uses; whether more than one ROT is required; or even whether extinction and resistance to extinction are derived from the same ROT. This is where attempts to determine the central mechanisms underlying the PREE provide some surprising answers.

Behavioral analysis of the PREE suggested that it could depend on simple associative effects (Sutherland, 1966), including those based on conditioning to the after-effects of reward and nonreward (Capaldi, 1967) or, alternatively, could involve more emotionally mediated effects resulting from the generation, by nonreward, of frustration (Amsel, 1992). Consistent with the idea that independent ROT can control apparently similar behavior under different conditions, the PREE is differentially sensitive to drugs. With short inter-trials intervals (when associative explanations appear to explain the behavioral phenomena best) the PREE is not sensitive to anxiolytic drugs; whereas at long inter-trial intervals (when frustration appears to explain the behavioral phenomena best) the PREE can be essentially eliminated by anxiolytic drugs (Feldon, Guillamon, Gray, De Wit, & McNaughton, 1979; Ziff & Capaldi, 1971).

However, if we ask about the psychological nature of the neural systems specifically affected by these drugs, we discover some interesting properties of the processes involved.

Emotional explanations of the PREE have often focused on counterconditioning—the reduction in negative affective value when negative stimuli are paired with positive ones. Anxiolytic drugs do not reduce counterconditioning (McNaughton & Gray, 1983). The drugs appear, instead, to reduce a nonassociative "toughening up" process (McNaughton, 1989b, chap. 7). Further, although the drugs affect both extinction (which could be viewed as dependent on conditioned frustration) and the PREE (which could be viewed as dependent on toughening up to the experience of conditioned frustration) in ways that could seem to depend simply on changes in sensitivity to the emotional experience of conditioned frustration, it turns out that extinction and the PREE depend on quite distinct neural systems and are, in a sense, unrelated to each other (Gray & McNaughton, 2000, appendix 9, table 1). Extinction in continuously reinforced rats is retarded by fiber-sparing lesions of the hippocampus proper, which do not reduce the PREE. Conversely, extinction in continuously reinforced rats is unaffected by lesion of the pathway connecting the subiculum of the hippocampus to the nucleus accumbens but these same lesions abolish the PREE.

Thus, both extinction and the PREE each appear to depend on a number of mechanisms (each one based on a particular ROT) and, in at least some cases, the mechanisms delivering extinction are quite distinct from those delivering the PREE. We thus have evidence for a variety of parallel ROT delivering adaptive extinction responding under a variety of situational circumstances (in particular, varying schedules of reward and reward omission).

Separation Anxiety and Parallel ROT

In one sense, the idea of parallel ROT—that is parallel systems concurrently activated—seems trivial. Autonomic and skeletal reactions, for example, must have evolved separately and are certainly represented in separate parts of the brain once we get "below" command centers such as the periaqueductal grey (Bandler, Keay, Floyd, & Price, 2000; Bandler, Price, & Keay, 2000). However, this issue is only trivial if a single command center controls both aspects of output. At least in the case of separation anxiety, this is not the case.

Separation anxiety is clearly identifiable, both by the means of producing it (removal of the primary caregiver, usually the mother) and by its characteristic pattern of autonomic and behavioral changes. It can be seen, in much the same form, in human children and the young of other mammals, such as rats, dogs, and primates. When the "reaction is beyond that expected for the child's developmental level," it becomes Separation Anxiety Disorder (American Psychiatric Association, 1987).

The behavioral and autonomic components of this emotion give the appearance of joint outputs from a single command center—and, if either output were missing, the

result would not be what is generally recognized as separation anxiety. However, it has been shown that, in rats, the behavioral reactions (locomotion, grooming, defecation, and urination elicited by a novel environment) can be eliminated by the presence of a nonlactating foster mother, whereas the autonomic reaction (a reduction in heart rate) can be eliminated by regular feeding with milk—but not, in either case, vice versa (Hofer, 1972). Thus, the two effector aspects of the one emotion can be doubly dissociated in the laboratory.

It appears that rather than available stimuli each activating a single cognitive center (detecting, say, threat in general), it is possible that each recognizable aspect of an emotion could result from a different aspect of the available stimulus input (Figure 36.1). Each emotion could consist of multiple parallel ROT. As with serial ROT, this does not create a problem for our naming of the phenomena. *Separation anxiety* remains a nameable set of entities that are coherent under normal ecological circumstances and our analysis does not require any change in the everyday use of the term. But, for scientific purposes, we must view the term

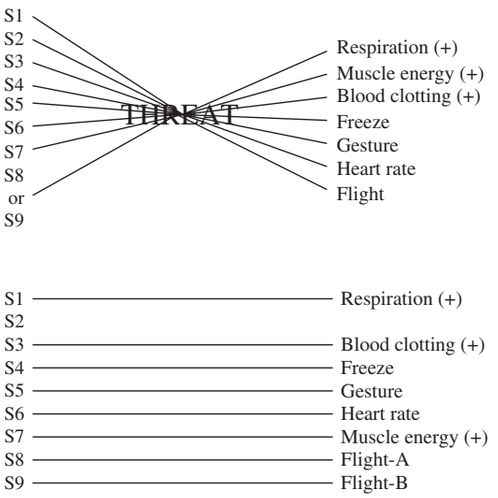

Figure 36.1 The extremes of the possible neural relations that could have evolved to control responses to threat.

Note: The top half of the figure shows the functional relations linking stimuli (S1-S9) to responses where the stimuli are either regular predictors of threat (S1-S7) or where different stimuli are predictive of threat at different times (S8, S9). It can also be viewed as a representation of the simplest view of emotional states, namely that all stimuli activate a single neural representation of threat and this in turn activates the separate response systems. The bottom half of the figure shows, in its most extreme form, the opposite type of neural organization suggested by Hofer's experiments (see text). Here, each response system is under its own private stimulus control. Some stimuli (S2) may have not acquired control over any response system and some stimuli (S8, S9) may have acquired control over a particular response (flight) but only under some circumstances (-A, -B). Redrawn from "Anxiety: One label for many processes". New Zealand Journal of Psychology, 18, Figure 1, p53 by McNaughton, 1989.

as grounded in a particular class of evolutionarily recurring situations (loss of parents) that give rise to a consistent set of adaptive requirements and so a usually consistent effector pattern (behavioral and autonomic) that constitutes a fairly consistent distributed central state—but without the need for a single command center or any other internal link between the components.

Evolution, ROT and Functional Definitions of Emotional Systems

If parts of a functional system can be independent, whether as a result of serial or parallel ROT, how can we understand or define the system—or even refer to it as a system at all? Rather than being a major problem, inverting this question allows us not only a convenient way to refer to, and to distinguish among, central emotional and motivational systems but also as well as a means of dealing with the fact that these systems involve multiple hierarchically organized layers:

> [This] approach to [emotion] stems from analysis of its possible functional significance. This approach is based on the premise that important and pervasive human action tendencies, particularly those which occur across a wide range of cultures and specific learning situations, are very likely to have their origin in the functionally significant behavior patterns of nonhuman animals. . . . This approach, working through the characteristic behavior patterns seen in response to important ecological demands (e.g., feeding, reproduction, defense) when animals are given the rather wide range of behavioral choices typical of most natural habitats, is called ethoexperimental analysis. It involves a view that the functional significance of behavior attributed to anxiety (or other emotions) needs to be taken into account; and that this functional significance reflects the dynamics of that behavior in interaction with the ecological systems in which the species has evolved, implying that these dynamics . . . can be determined far more efficiently when the behavior is studied under conditions typical of life for the particular species. (R. J. Blanchard & Blanchard, 1990b, p. 125)

Detailed ethological analysis of defensive responses obtained under experimentally controlled conditions by the Blanchards has demonstrated a categorical separation of a set of reactions that can be grouped together under the rubric of "fear" from a quite distinct "anxiety" set (R. J. Blanchard & Blanchard, 1988; R. J. Blanchard & Blanchard, 1989, 1990a, 1990b; R. J. Blanchard, Griebel, Henrie, & Blanchard, 1997).

The Blanchards elicited their set of "fear" behaviors with a predator. These behaviors, originally all linked solely through ethology, turn out to be sensitive to drugs that are panicolytic but not to those that are only anxiolytic

(R. J. Blanchard et al., 1997). The Blanchards elicited their set of "anxiety" behaviors (especially risk assessment; see Chapter 49) with stimuli that only suggested the potential presence of a predator. These behaviors, again originally all linked solely through ethology, turn out to be sensitive to anxiolytic drugs. The Blanchards' detailed analysis, and its pharmacological validation, provides a basis for coherent conceptualization of a vast animal literature. For example, their analysis of fear predicts the well-demonstrated insensitivity to anxiolytic drugs of active avoidance in a wide variety of species and of phobia in humans (Sartory, MacDonald, & Gray, 1990). Because of the detailed effects of anxiolytic drugs on operant and other behavior (Gray, 1977), we have argued (Gray & McNaughton, 2000; McNaughton & Corr, 2004) that the key factor distinguishing fear and anxiety is one of "defensive direction." Fear is that set of reactions that have evolved to allow the animal to leave a dangerous situation (predator escape; operant active avoidance); anxiety is that set of reactions that have evolved to allow the animal to enter a dangerous situation (e.g., cautious "risk assessment" approach behavior) or to withhold entrance (passive avoidance).

Evolution, ROT and Hierarchical Organization

With the PREE, we simply accepted the fact that, where there is a single high-level general rule for optimal behavior, there may be multiple ROT that deliver the appropriate behavior under different circumstances. However, when the functional requirement is something as general as "escape," different circumstances may not only require different ROT to produce essentially the same behavior pattern under different circumstances but also require noticeably different behavior patterns to achieve the result.

Here we can link the evolution of serial ROT to the hierarchical organization of emotional systems. At the perceptual level, there are both "quick and dirty" as well as "slow and sophisticated" sensory mechanisms for detecting predators (LeDoux, 1994). There are also simpler and more complex behaviors that can be generated depending on the time available for execution (and other constraints). We can see all these mechanisms as parallel ROT that have evolved to improve survival in the face of threat, each new one filling a gap left by existing mechanisms.

But these ROT have not evolved entirely independently of each other. First, simpler mechanisms will have evolved before more complex ones, providing a substrate for the development of the more complex and also providing a partial solution to the global problem that leaves a gap in adaptive advantage that later ROT must fill. Second, it makes no sense to have available a slow and sophisticated strategy for, say, escape if an evolutionarily older panic reaction takes command of the motor apparatus. When it is activated, a higher and slower mechanism must be capable of inhibiting inconvenient aspects of the lower and faster mechanisms. The result, with defensive behavior, has been the evolution of a hierarchically ordered series of defensive reactions (each appropriate to a particular "defensive distance," see the discussion that follows) that, in turn, map to lower and higher levels of the nervous systems, respectively. While behaviorally and neurally complex, all these reactions fulfill the same basic function and so can all be seen as part of a single "fear system."

The Blanchards developed the concept of defensive distance as part and parcel of their analysis of the differences between fear and anxiety, mentioned earlier. Operationally, with the most basic defensive reactions, it can be viewed as the literal distance between the subject and a predator. It is a dimension controlling the type of defensive behavior observed—that is, specific behaviors appear consistently at particular distances. In the case of defensive avoidance, the smallest defensive distances result in explosive attack, intermediate defensive distances result in freezing and flight, and very great defensive distances (i.e., absence of the predator) result in normal nondefensive behavior. However, defensive distance is not related directly to distance *per se.* It operationalizes an internal cognitive construct of intensity of perceived threat. For a particular individual in a particular situation, defensive distance equates with real distance. But, in a more dangerous situation, a greater real distance will be required to achieve the same defensive distance. Likewise, in the same situation, but with a braver individual, a smaller real distance will be required to achieve the same defensive distance.

This concept can resolve otherwise unexpected findings in, for example, behavioral pharmacology. It is tempting for those who focus on behavior as the thing to be studied in itself, as opposed to being a sign of states within the organism, to expect particular pharmacological interventions to affect specific behaviors in a consistent way. That this is not the case is shown by the effects of anti-anxiety drugs on risk assessment behavior. If perceived intensity of threat is high (small defensive distance), an undrugged rat is likely to remain still. Under these conditions, an anxiolytic drug will increase risk assessment (this will increase approach to the source of threat). But, if perceived threat is medium, an undrugged rat is likely to engage in risk assessment behavior. Under these conditions, an anxiolytic drug will decrease risk assessment (which again increases approach to the source of threat as it releases normal appetitive behavior). Thus, the drug does not alter specific observable risk assessment behaviors consistently but instead produces

changes in behavior that depend on the animal's initial state and are consistent with a pharmacological increase in defensive distance (R. J. Blanchard & Blanchard, 1990; R. J. Blanchard, Blanchard, Tom, & Rodgers, 1990).

This leaves us with a picture of ROT (in this case various levels of defense reaction) that have accumulated hierarchically. Their evolution has been accompanied not only by mechanisms controlling *which* ROT control behavior at any particular moment in time but also by mechanisms that can adjust which *level* of the system is selected by any particular external stimulus configuration (or rather the cognitions engendered by the stimuli).

In the case of the defense system, the hierarchical levels of responding can be mapped to levels of the nervous system and, at least, some of the overall control mechanisms identified. This is shown in Figure 36.2. The precise details contained in the figure are not important for our current argument and are dealt with in detail elsewhere (Gray & McNaughton, 2000; McNaughton & Corr, 2004; see Chapter 36) and are also briefly summarized in the section on specific central theories that follows. The important point is that a central theory of emotion, such as this, can treat different classes of behavior as, in one sense, discrete—each controlled by a particular different part of the brain—but at the same time can show that these different classes contribute to a more generalized functional system with control of the different parts that is at least sometimes integrated.

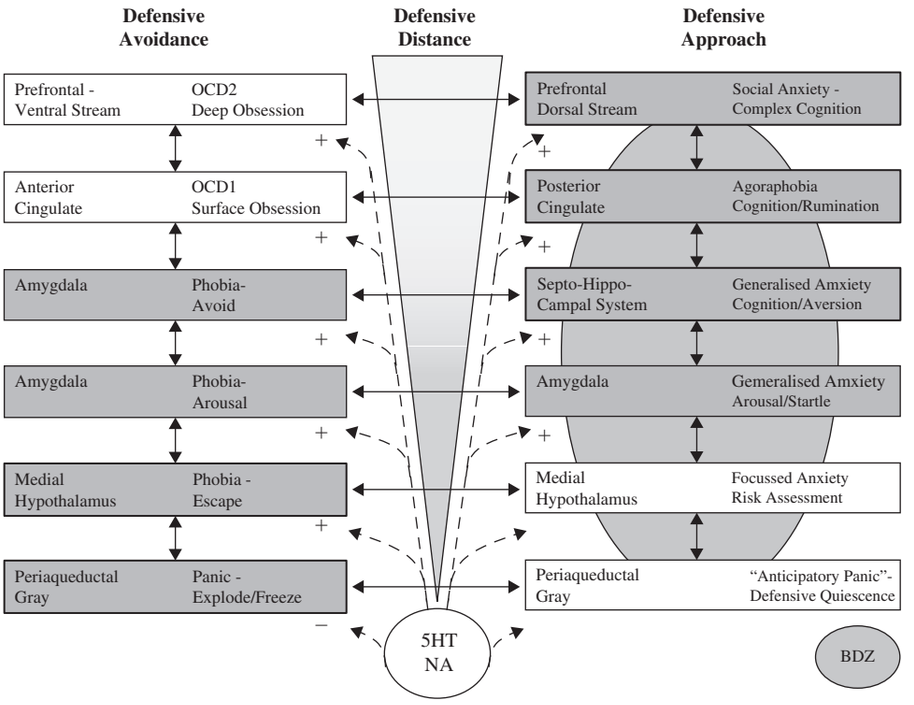

Figure 36.2 The two-dimensional defense system.

Note: The two columns of structures represent subsystems controlling defensive avoidance and defensive approach respectively. Each subsystem is divided, from top to bottom, into a number of hierarchical levels, both with respect to neural level (and cytoarchitectonic complexity) and to functional level (i.e., defensive distance—small at the bottom, large at the top). Each level is associated with specific classes of normal behavior and so, also, symptom and syndrome of abnormal behavior. Each level is interconnected with adjacent levels (vertical arrows shown) and also with higher and lower levels (connections not shown) and these connections allow integrated control of the whole subsystem. The subsystems are also connected with each other (horizontal arrows shown) allowing for control of behavior to pass between one and the other. Superimposed on the levels of each system is input from monoamines systems. The monoamines modulate activity, essentially altering defensive distance generally, and so which level of a subsystem will be in control of behavior at any particular point in time. Endogenous hormones binding to the benzodiazepine receptor (BDZ) can similar alter defensive distance but only in relation to structures in the defensive approach subsystem and to a lesser extent at the highest and lowest levels of the system than at the middle levels (as indicated by the width of the stippled oval as it intersects a structure). NA 5 Noradrenaline; 5HT 5 Serotonin. For details see "A Two-Dimensional Neuropsychology of Defense: Fear/Anxiety and Defensive Distance," by McNaughton and Corr, 2004, *Neuroscience and Biobehavioral Reviews, 28,* pp. 285–305. Adapted with permission from Figure 36.3, p 293.

EMOTION, MOTIVATION, AND LEARNING

Emotional systems have multiple parts that are several and distinct. Each involves a particular proximal form of appetitive or aversive behavior. But emotional stimuli are also reinforcing and, here, there is a surprising functional unity. Before proceeding to a consideration of the link between motivation and emotion, it will be helpful to clarify what modern neuroscience can tell us about the central mechanisms of reinforcement. Much analysis of emotion and motivation in the experimental literature has used learned responses because of their analytical simplicity. This can create problems when we attempt to link emotional concepts developed via ethological analysis with theories of learning and motivation developed via the experimental analysis of behavior.

Association versus Classical Conditioning versus Instrumental Conditioning at the Neural Level

The dominant paradigm for the study of synaptic processes underlying learning and memory is long-term potentiation (LTP), a phenomenon discovered by Bliss and Lomo (Bliss, Gardner-Medwin, & Lomo, 1973; Bliss & Lomo, 1973). Although LTP is usually studied electrophysiologically by high-frequency stimulation of a single neural pathway, its molecular mechanisms can clearly support strengthening of a single synapse that is driven by the coincidence of a previously weak input at that synapse with the firing of the cell produced by a strong input.

The key aspect of this strengthening (which at most junctions depends on a specific receptor, the NMDA receptor) is that it is associative. Only currently active synapses (essentially acting as CS+) are strengthened and other inputs to the same target cell that are not active (CS−) are not strengthened. This strengthening appears, ultimately, to involve structural changes in the synapses and not merely depend on modification of biochemical pathways (see Chapter 27.)

LTP has attracted particular attention because it conforms very tightly to the requirements for memory formation postulated of cortical neural processes by Hebb (1949). Hebb's rule (as it has come to be known) can be summarized as "cells that fire together wire together" and was postulated simply on the basis of psychological findings with no evidence for a matching real neural process until the discovery of LTP.

An important point to note is that Hebb's original example discussed the linking of two stimuli within the visual cortex. His postulated mechanism was, therefore, purely associative, requiring no additional reinforcer to strengthen the connection. A light paired with a light would become associated via connections within the visual cortex, as could a light with a tone—given the existence of "silent" connections between visual and auditory areas (Figure 36.3).

Thus Hebbian learning is best exemplified by what is normally known as "sensory preconditioning." ("Sensory preconditioning" is essentially a misnomer based on the, false, assumptions that learning requires a reinforcer and that without a change in behavior conditioning has not occurred.) The typical sensory preconditioning experiment can be confusing because it requires a reinforcer in order to demonstrate learning that did not itself depend on one. (With humans, we can omit the reinforcer by asking people to report their knowledge verbally.) The typical phases of a sensory preconditioning experiment are:

Phase 1: Stimulus A (a light) is paired with stimulus B (a tone) in a series of classical (Pavlovian) conditioning-like trials. Neither A nor B produces any observable response, before or after the conditioning-like trials.

Phase 2: Stimulus B (the tone) is next paired with a food in a series of conditioning trials. Initially the subject salivates when the food is presented, after a number of trials, they salivate when B is presented.

Phase 3: Stimulus A is now presented to the subject without any previous pairing of A with food. In experiments of this type it is usually found that the subject will salivate when A is presented. Yet, A has never been paired with food.

The conclusion from these results has to be that, during Phase 1, an association was formed between A and B. In Hebb's version of events, there initially exists a weak connection between a cell assembly activated by A and a cell assembly activated by B. When A is presented close in time to B, its weak synapses on the cell assembly encoding B will be activated at the same time that the cell assembly fires and so the connection will be strengthened. On later presentation of A, this connection activates (at least partially) the B cell assembly—and so produces, although perhaps weakly, the neural effects of the presentation of B (Figure 36.3A).

In Phase 2, stimulus B acquires observable consequences. These consequences are therefore likely to follow from the subsequent activation of the A assembly even in the absence of direct input by the B stimulus to the B assembly. This effect of A is demonstrated in Phase 3.

The purely associative process of long-term potentiation can also explain "classical conditioning" involving

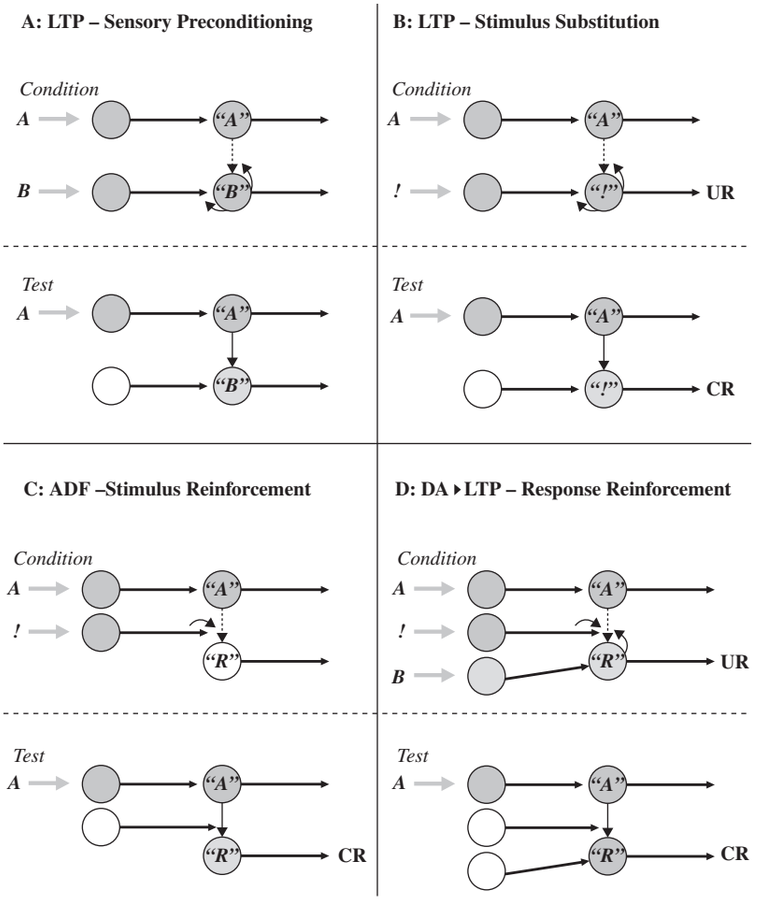

Figure 36.3 Different ways in which neural plasticity can result in associative learning.

Note: (A) Long-term potentiation (LTP) resulting in sensory preconditioning. Pairing of a neutral stimulus A with a second neutral stimulus B strengthens the connection between the representation of A and B such that presentation of A activates the representation of B when B is not physically present. (B) As in A but with the second neutral stimulus (B) substituted by a reinforcer. The unconditioned response (UR) to ! undergoes Pavlovian stimulus substitution with the result that it, or some component of it, appears as the conditioned response (CR) when A is later presented alone. (C) Activity dependent facilitation (ADF) as a basis for reinforced classical conditioning. Pairing of a neutral stimulus with a reinforcer results in strengthening of the connection of A with the neural representation of a response (R), independent of whether the response is currently activated. The result is classical conditioning that can produce a response that was not elicited by the unconditioned stimulus and so need not involve stimulus substitution. (D) Dopamine-dependent-LTP (DA4LTP) as a basis for reinforcement of instrumental responding. A low baseline emission of an operant response R is supported by the presence of an eliciting stimulus B. A conditional stimulus A is paired with the delivery of reinforcement (!) when the response is emitted as a UR. This strengthens the connection between the neural representation of A and the neural center controlling the emission of the response. This results in the response being emitted as a conditioned response when A is presented in future.

Pavlovian stimulus substitution without the need to invoke a specific reinforcement process (Figure 36.3B). If B is a motivationally significant stimulus prior to pairing with A, then activation of its stimulus representation by A will result in the same responses to A as previously occurred to B. This is like sensory preconditioning but with the link between B and an observable response having been established previously by evolution rather than later by an experimenter. In the case of tone-shock conditioning, the specific synaptic junction generating the conditioned fear reaction has been identified as a monosynaptic connection between the thalamus (containing the tone representation) and the amygdala (which is activated by the shock and generates the unconditioned response). Injection of an NMDA antagonist into the amygdala blocks LTP and so acquisition of the conditioned response but has no effect if injected once conditioning is complete (LeDoux, 1994; for a more detailed analysis of fear conditioning see Chapter 39; for a comparison of the neural circuits involved in fear conditioning and eyeblink conditioning see Chapter 26).

Simple LTP-based association can also explain what appears to be instrumental conditioning but is in fact

disguised classical conditioning with stimulus substitution. Pigeons are typically conditioned to peck keys that are lit prior to delivery of the reward. Under these conditions, autoshaping occurs. The pigeon comes to peck the key, essentially because its lit state predicts reward and not because the pecking is instrumentally reinforced. This is shown by two pieces of evidence. First, autoshaping with a superimposed instrumental omission contingency (which pits classical autoshaping against instrumental omission of reward if the pigeon pecks the key) results in behavior cycling between pecking and not pecking. The attractiveness of the lit key overrides any instrumental learning that pecking cancels reward; and the cyclical loss of responding can be attributed to extinction of the classical contingency rather than any effect of the instrumental one. Second, the nature of the key peck is determined by the reinforcer. The pigeon, effectively "drinks" a key paired with water and "eats" a key paired with food (Jenkins & Moore, 1973).

With so much possible with simple LTP-dependent association and its resultant stimulus substitution, we might be inclined to abandon the idea of reinforcement altogether. However, neuroscience provides at least two cases where true reinforcement mechanisms can be invoked.

The first reinforcement mechanism has been demonstrated in classical conditioning in the sea slug *Aplysia californica.* This animal is so simple that specific neurons can be identified and named reliably from animal to animal and be shown to control the same responses in each individual. This has allowed detailed analysis of the entire neural circuit involved in conditioning (Chapter 27; Kandel & Hawkins, 1992). Shock to the tail activates a single neuron that can release transmitter presynaptically onto the terminals connecting sensory neurons with a motor neuron that controls gill withdrawal. Pairing of a light touch to the mantle of *Aplysia* with a shock to the tail can then strengthen the connection between a sensory neuron that detects the touch and the motor neuron—a process referred to as activity dependent facilitation (ADF). The activity dependence of ADF results in a conditioned withdrawal of the gill to subsequent touching of the mantle (the CS+), but not of other sensory inputs, for example, a touch to the siphon. As with LTP, ADF is truly associative in that previous CS− can be conditioned if they are later paired with the shock. An important feature of ADF is that, in contrast to LTP, it allows true reinforcement in the sense of production of a new response that is not elicited by the unconditional stimulus (e.g., freezing to a CS for a shock, in contrast to the movement and vocalization normally produced by the shock).

The second reinforcement mechanism combines features of both standard LTP and ADF (Figure 36.3D). Like LTP, it requires the coincidence of the release of transmitter from the presynaptic neuron with the firing of the postsynaptic cell. However, in addition, LTP only occurs if dopamine is released presynaptically as a result of activation of the brain's "reward system" (Reynolds, Hyland, & Wickens, 2001). Notably the postsynaptic cell controls responding rather than encoding a stimulus. Its initial activation (on which responding and so reward-delivery are dependent) results from the presence in the environment of appropriate eliciting stimuli (unless the response can be spontaneously generated). As discussed next, this allows responses to continue to be produced on some occasions even when reinforcement conditions are changed or when the reinforcer is devalued. That is, a response can be habitual and its cessation will depend on active extinction as a result of negative reinforcement rather than simply fading away in the absence of significant events. (The phasic release of dopamine, relating to reinforcement and tonic release that can be identified with hedonic changes appear to activate different networks; and dopamine may not underlie all rewarding effects, see Chapter 40).

From Emotion to Motivation

Our argument, so far, is that specific ROT (controlled by specific neural mechanisms) have evolved in a not entirely piecemeal fashion so that, in at least some cases, they become organized into functional systems. In the case of defensive avoidance, we have a hierarchically organized system, each part of which can generate appropriate defensive behavior (e.g., freezing, aggression, escape, avoidance) within a specific range of environmental circumstances.

A large part of the theoretical structure of Figure 36.2 is devoted to an account of a fairly large number of particular situation-typical behaviors, which we group together not because of their specific form but because they share the same general function: removing the animal from danger. Aversive stimuli—both natural stimuli, such as a cat, and artificial stimuli, such as presentation of a shock, as well as the omission of expected rewards (frustrative nonreward)—all tend to have similar eliciting properties. Presentation of a cat elicits autonomic arousal, freezing, or attack, if defensive distance is short, and escape where this is possible. Much the same pattern is produced by both presentation of shock and frustrative nonreward: autonomic arousal, attack if there is a conspecific close by to attack, and escape if this is available (Gray, 1987, chap. 10). More general avoidance behavior is appropriate not only for a wide range of dangers, in the sense of things that can cause pain, but also for other stimuli that are merely disgusting, or even simply of no current interest.

Fear conditioning, learned escape, and learned avoidance of the simplest sort can all be viewed, in this context, as

the result of simple Pavlovian stimulus substitution. Pure associative conditioning results in a previously neutral stimulus becoming a signal for an upcoming noxious event and resulting in the class of defensive response appropriate to the level of threat signaled.

Whether the unconditioned stimulus is the presentation of a natural or artificial punishment or the removal of a natural or artificial reward, we can view avoidance behavior in general as resulting from activity in what has been known as the fight-flight system (Gray, 1987) but is probably better called the fight-flight-freeze system (FFFS; Gray & McNaughton, 2000).

It is at this point that we must distinguish between two quite distinct ways in which the words *fear* and *conditioning* can be combined. In the first conjunction of fear and conditioning, *fear conditioning,* a neutral stimulus is paired with a shock and responses such as freezing are conditioned. Critically, the shock is inescapable and so the conditioned form of the previously unconditioned fear responses remains even after many trials. This conditioning is purely associative, as with the learning of a light-tone pairing of the type evidenced in experiments on sensory preconditioning. It is dependent simply on the coincidence of the two critical stimuli that then become associated via the process of long term potentiation (Fanselow & LeDoux, 1999). The stimulus we often refer to as the *reinforcer* is necessary if a response of some type is to be observed but the learned association can be formed even with neutral stimuli and so does not depend on reinforcement in the strict Pavlovian meaning of the term. In the second conjunction of fear and conditioning, *conditioning of avoidance by fear* (with, for example, a lever press as the avoidance response), something quite different happens. In the initial phases of training, there is both a high level of autonomic arousal and the release of the stress hormone corticosterone (Brady, 1975a, 1975b). However, once avoidance is well established, all these signs of emotional reaction disappear and the only obvious difference in behavior, as compared with behavior observed before training, is that the avoidance response is reliably produced.

This leaves us in the apparently odd situation of maintaining that although an avoidance response is being made (as a result of the motivation of fear) the animal is not afraid (in the sense of showing emotional reactions). The commonsense view is that there is no reason for the animal to be afraid because it knows the avoidance response will prevent it from receiving a shock. There are two levels at which we need to take this idea seriously.

The more trivial level at which a learned avoidance response is not driven by fear is that well-learned responses are, in a very real sense, habits. Even with positive reinforcers that are physically present on every trial (such as food for a hungry animal), sufficiently long training results in the animal continuing to respond even when the reinforcer is devalued. The "rewarded response" is made but the reward itself is not consumed (Dickinson, 1980). With successful avoidance responses the reinforcer is never present and so responding can be even more resistant to extinction. The same is true of the conditioned suppression of behavior by anxiety. After extended training, the suppression becomes insensitive to anxiolytic drugs (McNaughton, 1985).

The deeper level at which a learned avoidance response is not driven by fear rests in the fact that, unlike fear conditioning, it is not the presentation of shock that "reinforces" learning: rather it is the omission of shock. Continued responding is driven by relief, not fear. This is not mere semantic quibbling. In the same way that omission of reward has the same reinforcing properties (and many of the same eliciting properties) as the presentation of punishment—the "fear 5 frustration hypothesis" (Gray, 1987)—omission of punishment has the same reinforcing properties as the presentation of reward—the "hope = relief hypothesis" (Gray, 1987). As we shall see, below, we can attribute the learning of new responses to the release of dopamine and, consistent with this, dopamine is involved in avoidance conditioning (Sokolowski, McCullough, & Salamone, 1994; Stark, Bischof, & Scheich, 1999). Omission of punishment is, thus, truly rewarding.

Here we should notice an asymmetry in the types of released behaviors associated with approach reactions compared to those associated with avoidance. Avoidance involves, in general, a hierachically organized set of released action patterns that do not vary much with the specific eliciting stimulus and that vary with "defensive distance"; approach involves, in general, released action patterns only in contact with the eliciting stimulus and then produces stimulus-specific responses. (Avoidance also involves stimulus-specific responses with contacting stimuli: for example attack of a predator is replaced with defensive burying of a shock probe—but these are not as many or various as the stimulus-specific responses produced by contact with appetitive stimuli. Likewise, there is little difference in principle between an appetitive conditioned jaw movement response and an aversive conditioned eyeblink response.)

The specific behaviors observed in the context of active avoidance (when the animal moves away from a localized aversive stimulus) are suprisingly general and depend much more on defensive distance than on the specific nature of the aversive stimulus. Thus, both punishment and frustration will generally increase aggressive responses within and between many species, including humans (Renfrew & Hutchinson, 1983) and, in humans, will even increase aggressive responses directed at completely innocent inanimate objects (Kelly & Hake, 1970). Defensive behaviors,

then, give the appearance of output from a single, fairly homogenous, system—with specific released, as opposed to learned, behaviors varying mainly with the defensive distance.

By contrast, the proximal behaviors required to consummate the approach to an appetitive stimulus are entirely stimulus specific. We eat food and mount a sexual partner, but not vice versa. There has not been reported, however, a hierarchical series of standard behaviors required for approach that varies with "appetitive distance." It may simply be that there has been a lack of appropriate ethological analysis of such approach behavior. However, the behaviors required to approach an appetitive stimulus (other than simple locomotion) are unique to each situation and driven by the specifics of the situation rather than the nature of the appetitive stimulus. Indeed, the most obvious fundamental requirement is the learning of whatever new and, in evolutionary terms, completely arbitrary responses are required to achieve the goal. There are, then, no emotion-general innate reactions that characterize a specific appetitive distance.

This not to say that there is no dimension of appetitive distance. Appetitive goals produce a systematic, distance-related, effect. But the evidence is that variation in distance between an organism and an appetitive goal drives the quantity or intensity of behavior, but not its quality. The intensity with which approach behavior is executed increases the closer an animal is to the goal, as if there is a "goal gradient" (Hull, 1952)—but this is as true of lever pressing on a fixed interval schedule in an operant chamber as of running in runway on a continuous reinforcement schedule.

We have, therefore, two fundamental systems: one that controls the avoidance of specific stimuli (including reward omission) and one that controls approach to specific stimuli (including safety). Each of these is linked to systems that determine the specific aversive (e.g., defensive burying) or appetitive (e.g., eating) behavior that will be released by contact with a motivationally significant stimulus. But each is also more fundamentally a generic system devoted to avoidance or approach, respectively. Because of the asymmetry in functional requirements noted previously, the avoidance system has been named in terms of some common discrete elicited behaviors (fight, flight, freeze); while the approach system has been named generically: the Behavioral Approach System (Gray, 1982) or Behavioral Activation System (Smits & Boeck, 2006)—with the abbreviation, BAS, designating the same appetitive neural system in both cases.

Here we come to the nub of the relationship between the central control of emotion and that of motivation. To a first approximation, when we talk about emotion, we are talking about the elicitation of particular patterns of internal (autonomic) and external (skeletal) behavior; when we talk about motivation, we are talking about the production of generalized approach or avoidance tendencies. Motivation, in this sense, cannot exist without emotion—at least in the initial phases of learning. But, in stable environments, with habitual responses reliably delivering appropriate appetitive stimuli or apparently successfully avoiding aversive stimuli, emotional reactions are minimized.

We need to clear up a common misconception: There can be a tendency to link aversive stimuli and avoidance to emotion and to see them as distinct from appetitive stimuli and an approach that just involves motivation. This tendency results from the fact that the usual way to study aversive stimuli in the laboratory is to deliver electric shock (which requires no prior deprivation of some need for it to be effective); while the usual way to study appetitive stimuli is to deliver food to a hungry animal or water to a thirsty animal. It is common to see the eliciting stimulus of shock as creating the motivational state that drives behavior in the aversive case but to see deprivation, rather than the appetitive stimulus, as driving the motivational state in the appetitive case.

Positive motivation does not, however, require a state of deprivation of some basic need. Female rats can often appear relatively passive during copulation—albeit showing receptive behavior linked to the phase of their ovarian cycle. However, not only does their receptive phase involve permitting the male to mount, it turns out that it involves more active tendencies when appropriate.

> Male (rats) normally pause for a while after intromissions, and for a longer time after intromissions that culminate in ejaculation. . . . Bermant (1961a, 1961b) provided female rats with a lever they could press to produce a male rat. After a mount (regardless of whether it resulted in intromission) the male was removed. The females quickly pressed the lever after the male was removed following a mount (without intromission), paused a bit more after an intromission (without ejaculation), and waited the longest time before summoning a male rat after ejaculation. Thus it appears that male and female rats prefer the same frequency of sexual contact. (Carlson, 1980, p. 333)

Here the reaction of the female rat to the male (albeit approach) is essentially the same class of reaction as that of a rat to a cat (albeit avoidance). The availability of the motivationally significant stimulus—and interactions with it—drives the behavior. One could argue that there is a background level of preparedness on the part of the female rat driven by the ovarian cycle—but there are also variations in fearfulness within rats from time to time and between rats, and the same is true for humans—especially with sexual receptivitiy.

Even with hunger, it should be noted that the normal experience of hunger is linked as much or more to the availability of palatable food, or some other external or temporal cue for eating, than it is related to tissue need or level of deprivation (Pinel, 1997). For example, if a rat is provided for some time with six meals a day that are spaced irregularly but signaled by a buzzer and light stimulus and is then placed on free food so that it is satiated, presentation of the buzzer and light will elicit eating of as much as 20% of their daily food intake (Weingarten, 1983). (The total amount eaten over a day was not changed as later free feeding adjusted for the extra meal.) Likewise, if we see hunger as an essentially emotional rather than homeostatic state, we can understand its links with emotional disorder: the life-threatening reductions in weight that can occur in anorexia nervosa and the health-threatening increases in weight that can occur in depression.

Likewise, simple rewarded responding can depend, like simple fear conditioning, on stimulus substitution. As we noted earlier, in experiments with autoshaped responses, pigeons produce stereotyped responses that show they are effectively drinking the key when they are thirsty and eating the key when they are hungry (Jenkins & Moore, 1973). Further, if the autoshaping schedule (which pairs a lit key with the reward) has added to it an instrumental omission contingency (so that pecking cancels food), the pigeon goes through cycles of responding and nonresponding corresponding to the simple associative contingencies in the situation, unlike a rat that ceases responding and reacts to the reinforcement contingencies (see Millenson & Leslie, 1979).

SOME CURRENT CENTRAL THEORIES OF MOTIVATION AND EMOTION

There are many specific hypotheses currently being advanced by neuroscientists in relation to detailed aspects of the control of specific emotional reactions, motivational control systems, and learning and memory. For the behavioral scientist wanting to enter this field (which can appear like a minefield of novel jargon and mind-boggling detail), it is probably most important to note that the many detailed issues can be dealt with one at a time. You can focus on the detail that pertains to only to the current issue. In essence, one is dealing with the neural specifics of particular ROT. Provided one has been warned about the capacity of ROT to be nested both in serial and parallel, it is not difficult to accept the bits of the jigsaw piece-meal and leave integration until sufficient bits have been obtained to make the overall puzzle worth solving. The most important thing is to not believe that the solution to the puzzle is obvious and to wait for a sufficient number of the pieces to become available.

Partly because they deal with the neural instantiation of ROT, neuroscientists seldom integrate their findings on emotion and motivation into grand overall theoretical schemes. They do use global, apparently integrative, concepts. But these concepts are usually taken directly from behavior analysis and so subsume ROT within what are effectively clusters (such as the PREE and instrumental learning) based on overall evolutionary function. This may give the impression that they are ascribing to ROT a specific source of integrated control but, as we have seen, this need not be the case. Instead, the use value of this approach is to gather together phenomena that may have some, albeit loose, integrated control—or that may have the appearance of control as an emergent property of the interaction of multiple ROT.

There are, nonetheless, neuroscientifically grounded theories that attempt to provide more wholistic, integrated perspectives. In this section, we briefly describe some of these and show how the architecture of each maps to the basic ideas we have presented above.

Gray and McNaughton

We have based a number of the concepts we have already presented on one such theory—the idea, originally proposed by Jeffrey Gray (1982), that behavior is primarily controlled by a Fight-Flight-Freeze System (FFFS) and a Behavioral Approach System (BAS) with, linked to these, and controlling conflict between approach and avoidance, a Behavioral Inhibition System (BIS). This theory has clear links with the idea of multiple ROT, especially in its more recent development (Gray & McNaughton, 2000; McNaughton & Corr, 2004). Multiple ROT are instantiated in the mixture of levels and streams of Figure 36.2, which shows the FFFS and BIS, and in the matching levels of the separate stream of structures controlling the BAS (not shown). It also has at its core the idea that, in general, approach and avoidance behavior are each controlled in fundamentally the same way independent of the specific source of motivation for that approach or avoidance.

Rolls

This latter perspective is presented in perhaps an even stronger way by Edmund Rolls (1990, 2000) in his general theory of the control of emotion and motivation by the brain. He sees evolution as starting with simple ROT in the form of taxes that attract simple animals (including those with no nervous system) toward items that promote

survival and reproduction and that drive them away from items with the opposite consequences. He argues that:

> brains are designed around reward- and punishment-evaluation systems, because this is how genes can build a complex system that will produce appropriate but flexible behavior to increase fitness. . . . If arbitrary responses are to be made by the animals, rather than just preprogrammed movements such as tropisms and taxes, [is] there any alternative to such a reward/punishment based systems in this evolution by natural selection situation? It is not clear that there is, if the genes are efficiently to control behavior. The argument is that genes can specify actions that will increase fitness if they specify the goals for action. It would be very difficult for them in general to specify in advance the particular response to be made to each of a myriad of different stimuli. . . . Outputs of the reward and punishment system must be treated by the action systems as being the goals for action. (Rolls, 2000, pp. 190, 183, 191).

Rolls could, at first blush, appear to be taking an excessively binary view. He states, for example, that "emotions can usefully be defined as states elicited by rewards and punishments, including changes in rewards and punishments" (Rolls 2000, p. 178). He also argues that "the amygdala and orbitofrontal cortex . . . [are] of great importance for emotions, in that they are involved, respectively in the elicitation of learned emotional responses and in the correction or adjustment of these emotional responses as the reinforcing value of the environmental stimuli alters" (Rolls, 1990, p. 161). This perspective seems to force all emotion into either a reward or a punishment box with variation in behavior simply being the results of the learning of arbitrary responses.

However, on closer inspection of the details of Rolls' theory, it is clear that he allows not only for multiple ROT in terms of elements of behavior but also in terms of the separation of, for example, autonomic from behavioral aspects of emotional response. In his view, there are three major, neurally separate, classes of output available for any emotion: there are autonomic and endocrine outputs that optimize the state of the animal for particular types of action; there are *implicit* behavioral responses; and there are *explicit* behavioral responses. Implicit behavioral responses are controlled "via brain systems that have been present . . . for millions of years and can operate without conscious control. These systems include the amygdala and, particularly well developed in primates, the orbitofrontal cortex. They provide information about the possible goals for action based on their decoding of primary reinforcers taking into account the current motivational state, and on their decoding of whether stimuli have been associated by previous learning with reinforcement." This clearly encompasses a wide range of emotion-specific and innately elicited responses. The control of explicit behavioral responses, by contrast, "involves a computation with many 'if . . . then' statements, to implement a plan to obtain a reward or to avoid a punisher." Here the behavior controlled is clearly general in its form and largely based on strategies for simple approach or avoidance. He locates the highest levels of this control in the dorsolateral prefrontal cortex—where they are strongly related to the processing of shortterm (or "active") memory.

Despite Rolls' somewhat different perspective compared to Gray and McNaughton, he is like them in seeing orbitofrontal cortex as, in essence, coding "what" a stimulus is. "What" here has the sense of what specific class of reinforcer such as food, drink, or sex it is that the stimulus represents—and compounds "sensory integration, emotional processing, and hedonic experience" (see Chapter 41). Dorsolateral frontal cortex, by contrast, codes "where" a stimulus is. Thus both theories see a distinction between a reactive and excitatory orbital system and a prospective and inhibitory dorsolateral system.

Critically, in the context of ROT, Rolls (2000) warns that "these three systems do not necessarily act as an integrated whole. Indeed, insofar as the implicit system may be for immediate goals, and the explicit system is computationally appropriate for deferred longer terms goal, they will not always indicate the same action. Similarly, the autonomic system does not use entirely the same neural systems . . . and will not always be an excellent guide to the emotional state of the animal, which the above argument in any case indicates is not unitary" (pp. 188–189).

There is a strong link between emotion and motivation for Rolls, in both their more innate and more conditioned forms. While starting from the position that "emotions can usefully be considered as states produced by reinforcing stimuli" (Rolls, 1990), he sees the particular value of those states as involving elicitation of autonomic and hormonal responses and, in learning experiments, in the production of various conditioned emotional responses. Emotion, viewed in this light, provides a basis for the facilitation of memory storage and for the immediate elicitation of flexible responding when conditions change. The blocking of a learned response by new circumstances leaves intact the conditioned emotional response, which then provides the basis for the development of new behavior. Background autonomic and hormonal reactions provide the basis for the storage of such strategies as then prove successful.

Ledoux

For many years, Joe Ledoux has been developing a theory of fear, and consequentially anxiety, that is more limited in terms of the emotions analyzed but potentially deeper in the picture it presents of the details of the emotional systems. This can be seen as dovetailing to some extent with both the theoretical positions we have described so far.

While Gray and McNaughton focus on hierarchy in terms of the specific elicited behaviors associated with specific defensive distances, Ledoux can be thought of as focusing more on hierarchies of stimulus analysis that are to some extent also selected by defensive distance. He has contrasted "quick and dirty" threat detection systems (operating via the thalamus) with slower and more sophisticated ones operating through sensory cortex (Ledoux, 1994) and more recently (Ledoux, 2002) has laid emphasis on the even slower, and potentially more sophisticated, mechanisms that reside in frontal cortex and are linked to working memory and that form of planning that we can call "worry."

At first, his theory appears to be at total variance with that of Gray and McNaughton. However, when we look at the neural details, we discover that the discrepancy is not great; and we demonstrate a major advantage of a central/neural approach to emotion and motivation. The details of the theories are linked to neural reality very tightly and this allows one to resolve, relatively easily, issues that depend much more on arbitrary linguistic definitions than scientific facts.

Ledoux (2002) argues to some extent that anxiety is really fear but represented differently in consciousness. Thus:

> anxiety, in my view, is a cognitive state in which working memory is monopolized by fretful, worrying thoughts. The difference between an ordinary state of mind (of working memory) and an anxious one is that, in the latter case, systems involved in emotional processing, such as the amygdala, have detected a threatening situation, and are influencing what working memory attends to and processes. This in turn will affect the manner in which executive functions select information from other cortical networks and from memory systems and make decisions about the course of action to take. . . . I believe that the hippocampus is involved in anxiety not because it processes threat, as Gray suggests, but instead because it supplies working memory with information about stimulus relations in the current environmental context, and about past relations stored in explicit memory. . . . When the organism, through working memory, conceives that it is facing a threatening situation and is uncertain about what is going to happen or about the best course of action to take, anxiety occurs. (p. 288)

Ledoux's very influential theory of the neural processing of fear was essentially incorporated into Gray's (1982) original, essentially amygdala-free, theory in its revision by Gray and McNaughton (2000). So, as far as fear goes, there is essentially general agreement among the theories of Ledoux, Gray and McNaughton, and Rolls. It is in dealing with anxiety that he sees the Gray and McNaughton theory as underemphasizing working memory and worry, "in my opinion, it still gives the septum and hippocampus too prominent a role, at the expense of the amygdala and prefrontal cortex" (p. 288).

In resolving the differences, let us first note that Gray and McNaughton's theory is anchored primarily in the effects of anxiolytic drugs. The link between anxiolytic action and effects on hippocampal electrical activity have been ever more firmly established (McNaughton, Kocsis, & Hajós, 2007). However, as Gray and McNaughton noted in their introduction:

> "psychosurgery"—lesions of the cingulate or prefrontal cortex—has been used as a treatment with some degree of success. So these cortical areas could well mediate extreme (Marks, Birley, & Gelder, 1966) or complex forms of anxiety, especially . . . in the case of obsessive-compulsive disorder (Rapoport, 1989). (Gray & McNaughton, 2000, p. 5)

Gray and McNaughton (2000) have a theory of "anxiolytic-sensitive anxiety" that necessarily separates this from the processes of anxiety (or fear or obsession) that are controlled by frontal cortex. What of their view of frontal and cingulate cortex—on which Ledoux focuses:

> We view them . . . as being hierarchically organized areas which deal (in their successively "higher" layers) with progressively higher levels of anticipation of action. . . . In the same way, then, that we distinguished the role of the hippocampus (in resolving concurrent goal-goal conflict) from the role of the defense system and other motor systems in resolving motor program conflicts without goal conflict, so we must distinguish its role from that of prefrontal and cingulate cortex. In our view these cortical areas are involved, quite independently of the hippocampus, in the resolution (i.e., ordering) of conflicts between successive sub-goals in a task. In the case of prefrontal cortex this amounts to saying that it is concerned with plans more than goals as such. However, where (as is common in certain types of working memory task) there is concurrent goal conflict within such a task, both the septo-hippocampal system and the prefrontal cortex are likely to be involved. (p. 5)

This view is not far from that expressed by Ledoux 2 years later, if we do not try and force the word "anxiety" to mean the same thing in the two cases. Gray and McNaughton focus on approach-avoidance conflict; something that can occur as a result of the apposition of two classes of innate releasing stimulus, with no requirement for learning or working memory. Ledoux focuses on "worry," the maintaining of a perception of threat in working memory (with no necessary requirement for anything other than pure avoidance). The two theories are talking about different processes in different structures—and Gray and McNaughton have much the same view of the operations of frontal cortex and of the amygdala as Ledoux. Both theories agree that "the amygdala and hippocampus normally cooperate in the intact brain to store

different components of the fear learning experience" (see Chapter 39).

There is perhaps one area where discrepency may remain and where further experiment (or theoretical analysis) may be required to integrate the theories. Gray and McNaughton see the personality factor of neuroticism as being linked to frontal cortex, and as predisposing to both fear (threat avoidance) and anxiety (threat approach) disorders. Although they do not explicitly do so, they should link this personality factor to worry. For them worry is something that, if excessive, can lead to both pathological fear and pathological anxiety. These two states would appear to not only be conflated in Ledoux's analysis but also to be consequences not causes. Ledoux sees threat, detected in the amygdala, as infecting working memory processes and resulting in worry. There is evidence that worry is not directly aligned with anxiety as measured by standard anxiety scales (Meyer, Miller, Metzger, & Borkovec, 1999) and that worry can result in intrusive negative thoughts (Borkovec, Robinson, Pruzinsky, & DePree, 1983).* This suggests that, provided we use the words "worry" and "anxiety" with sufficiently restricted definitions, we can see Ledoux's theory as being more focused on a cause of pathological anxiety (and fear and depression), and their aetiology, and Gray and McNaughton's as providing a view of state fear and state anxiety that encompasses both normal and pathological examples of these emotions but distinguishes between them.

Many of the differences between these three theories of central emotional and motivational states are more apparent (through variations in the use and meanings of words) than real. Critically, when what each theorist says of the mechanisms and psychological constructs associated with a particular neural structure is compared with the others, their fundamental message is very similar. They all believe that central states are fundamental to emotion and motivation, either in its normal or pathological form.

We would also agree with Ledoux (2002) when he states, "I don't study behavior to understand behavior so much as to understand how processes in the brain work" (p. 209). To this we, personally, would add the coda that we want to understand the processes in the brain because these anchor our understanding of the workings of the mind.

Damasio

While it is not a full theory of emotion, mention should also be made here of Damasio's somatic marker hypothesis (Damasio, 1995, 1996). This is a partial theory of how emotion or motivation can interact with cognition. It is intended to be an account of only one of several ways that affect can influence decision making and focuses primarily on the operation of the ventromedial prefrontal cortex, to the exclusion of other frontal areas. It is of interest here for two reasons: First, its view of emotional influence is different from the theories we have discussed so far. Second, its view of somatic phenomena is broader ranging.

Damasio's theory (Damasio 1995, 1996; for a critical review, see Dunn, Dalgleish, & Lawrence, 2006; also see Chapter 38) originated in an attempt to account for the effects of ventromedial prefrontal damage. His patients showed severe impairments in decision making and in social choices but have intact IQ, learning, and retention of knowledge (including social knowledge) and skills, logical analysis and language skills. They also perform normally on the Wisconsin Card Sorting Test that is normally affected by frontal damage. The abnormal decision making and social choices of these patients were accompanied by abnormalities in emotion and feeling and Damasio postulated that these emotional changes were the cause of the abnormal decision making.

"The somatic marker hypothesis proposes that 'somatic marker' biasing signals from the body are represented and regulated in the emotion circuitry of the brain . . . to help regulate decision-making in situations of complexity and uncertainty" (Dunn et al., 2006, p. 240). The presence of what is, in effect, a somatic image called up by a situation constrains decision making and limits the amount of processing required of cognitive mechanisms either by explicitly labeling a scenario as negative or positive; or implicitly biasing decision mechanisms in a positive or negative direction.

The somatic marker hypothesis differs from the other theories we have discussed in that it keeps the encoding of emotion (or strictly soma, see discussion that follows) distinct from the encoding of the information on which cognitive processes act, even at the prefrontal level. That is, emotional information can supplant, or bias, the processing of other types of information and is only integrated with them by altering their processing. The other theories, by and large, operate in terms of goals—compounds of cognitive (situational) and affective (affordance) information. It remains to be seen (Dunn et al., 2006) how far a somatic marker system in the ventromedial prefrontal cortex can be distinguished from some specific aspect of goal processing and how far it is qualitatively distinct from the other classes of processing that the hypothesis allows occur in other areas of frontal cortex.

The somatic marker hypothesis is also broader ranging than conventional postive/negative valence approaches. Here it departs both from the other theories and from more conventional behaviorist perspectives. Damasio (1996) holds:

> that the results of emotion are primarily represented in the brain in the form of transient changes in the activity patterns

* We thank Rama Ganesan for bringing this literature to our notice.

> of *somato-sensory* structures. I designated the emotional changes under the umbrella term 'somatic state'. Note that by somatic I refer to the musculoskeletal, visceral and internal milieu components of the soma rather than just to the musculoskeletal aspect; and note also that a somatic signal or process, although related to structures which represent the body and its states does not need to originate in the body in every instance. (p. 1412, italics added for emphasis)

Thus, somatic markers are not the simple assignation of valence or even of specific motivation to a stimulus. They are the perception or recall of a quite specific and detailed somatic image. There is no question that we can encode such images, and rehearse in our "mind's eye" the somatic experience of, say, a competition dive. However, to see this image as the basis of a background biasing of a cognitive decision about whether to make a particular bet in Damasio's paradigm task, the Iowa Gambling Task, is a radical departure from most other views of decision making and goal processing.

Central Theories of Emotion and Motivation—Some Broad Conclusions

The details, perspectives, and specific assignment of functions to structures by the theories we have considered differ. However, they all share a picture of the control of behavior by multiple serial and parallel ROT by hierarchically organized systems in the brain. They thus account for (without producing a complete explanation of) the apparent theoretical impenetrability of emotion.

No two emotions need be constructed or controlled in the same way as each other. No single emotion need have a unitary control. Rather, an emotion, as normally identified, may be an emergent structure deriving much of its superficial unity from the evolutionary path that has shaped the various component reactions. That said, the adaptive requirements facing, for example, the autonomic nervous system are sufficiently similar across the different emotions that at the general, as opposed to specific, level they can be seen to have many common features. Critically, neural analysis can determine the similarities and differences in the control of both superficially similar and superficially different reactions.

The theories also share a common picture of a variety of emotions being linked to two broad classes of general behavioral tendency: approach and avoidance. These have their origin, as emphasized by Rolls, in the fundamental properties of taxes—which are defined in terms of their being the result of the simplest stimuli generating, in the simplest way, either approach or avoidance—these ideas follow from Gray's early articulation of the same basic principles. Thus, while affective stimuli will define specific goals (and, with the possible exception of Damasio, the theories all see behavior as goal directed), a behavior such as a lever press can result in food, delivery of a mate, safety from shock, or a variety of other specific results—but in all cases (including relief from nonpunishment) it is reinforced in the same basic way, by the release of dopamine. The control of distal behavior, then, depends on systems fundamentally devoted to approach, in general, and avoidance, in general. To these basic systems, Gray and McNaughton add an additional system that resolves conflict between the approach and avoidance systems—but their view of the basic approach and avoidance systems is essentially similar to that of Rolls and their view of the basic control of avoidance is much the same as that of Ledoux.

FUTURE DIRECTIONS

So far, it might be thought that our analysis has not produced much of an advance, from a behaviorist perspective, beyond confirming the unsurprising conclusions: that different stimuli elicit different proximal behaviors; and that behavioral plasticity can be understood in terms of positive and negative reinforcement. However, there are a number of points where neural analysis provides specific departures from any simple form of these conclusions and where it leads, in extreme cases, to unexpected conclusions.

Beyond the Basics—The Potential for Unexpected Conclusions

Perhaps the most important conclusion that neural analysis allows is that what is paradigmatically conditioning does not necessarily require explicit reinforcement. As we noted, sensory preconditioning and Pavlovian fear conditioning both involve the same basic form of stimulus-stimulus association in which simple long-term potentiation is all that is required for the strengthening of connections. The specific site of this potentiation, for fear conditioning, has been identified as the input from the thalamus (which encodes the conditional stimulus) to the amygdala (which generates the unconditioned, and then conditioned, responses). We have also argued that this purely associative, nonreinforced, type of learning underlies what often appears to be instrumental learning in cases, such as a pigeon pressing a lit key, where the behavior is autoshaping in disguise—although it has not yet been proved that this involves long-term potentiation.

Following on from this conclusion is the fact that true reinforcement in the classic sense intended by Pavlov, while strengthening neural connections, need not reinforce

a previously occurring response. This provides a simple explanation of the fact that, for example, the conditioned response to a stimulus that predicts shock (usually freezing) is not simply the unconditioned response to the shock (vocalization, movement) moved forward in time. Indeed, while there will not be a perfect match between dependence on association rather than reinforcement and the occurrence of stimulus substitution, the neural data suggest that association rather than reinforcement should be suspected whenever the conditioned response (including those that are superficially the result of instrumental conditioning) can be accounted for by stimulus substitution.

A related issue, with instrumental reinforcement, is the demonstration that punishers release dopamine. The broad two-dimensional affective model we presented is, admittedly, derived originally from learning theoretic analysis (Gray, 1975). In this analysis, the omission of expected, or termination of, punishment is functionally equivalent to the presentation of rewarding stimuli; and in a symmetrical manner, the omission of expected, or termination of, reward is functionally equivalent to the presentation of punishment. But the demonstration of a link between punishment and dopamine, and of the role of dopamine in controlling instrumental reinforcement (Reynolds et al., 2001), has two important consequences for this model. First, it means we can be sure that, at a mechanistic level, the effects of reward and punishment omission are identical—they both change behavior by releasing dopamine. It is not the case that they happen to coincidentally produce the same superficial effects on behavior through independent mechanisms. Second, we can link both normal reward and normal punishment omission directly to explanations of addiction—where all addictive drugs (and some addictive-like behavior) have been shown to support continued behavior by the release of dopamine (but see also Chapter 40). We use this fact to provide potential explanations of some behaviors that might not be expected from the perspective of a simple reinforcement theory.

A final point we need to consider before moving on to some specific scenarios is the nature of the interaction between reward and punishment—where we again need to take into account the tendency of evolution to select multiple ROT rather than producing integrated control systems. In terms of simple decision making, for example, reward and punishment systems suppress each other. However, with respect to arousal, and so sometimes the vigor of production of responses, they can summate (Gray & Smith, 1969). These are quite distinct computations and, in terms of the effect of anxiolytic drugs on approach-avoidance conflict, can be shown to be processed in quite different parts of the brain. The inhibitory effect of punishment on rewarded behavior is mediated via the hippocampus, while the excitatory effect of punishment on reward-elicited arousal is mediated via the amygdala and not, in either case, vice versa (Gray & McNaughton, 2000). As a result, the addition of negative reinforcement can increase the levels of behavior generated by a positive reinforcer (e.g., in behavioral contrast). More peripheral theories of emotion and motivation would struggle to account for such findings.

In the sections that follow, we speculatively consider the possible insight that these features of the reward and punishment systems can offer into some of the more perplexing behaviors shown by human beings. (For a higher level view of apparently irrational behavior patterns, see Chapter 37.)

Relief of Nonpunishment: Gambling

We have already considered the complex mechanisms underlying the partial reinforcement extinction effect—where we argued that the phenomena are generally adaptive in that they conform to optimal foraging analyses. Here we consider cases of pathological gambling where persistence in the face of intermittent reinforcement is, in optimal foraging terms, maladaptive.

According to standard behavioral accounts, pathological gambling should not develop very easily and should extinguish fast. That is, engaging in a behavior that provides a high ratio of punishment to reward should led to avoidance behavior, which of course it does in the majority of the population. However, in a significant minority of people, pathological gambling behavior develops. That is, the behavior entails high monetary losses leading to personal, family, and societal problems.

We could attempt to explain this maladaptive behavior using standard learning theory. There is intermittent positive reinforcement, and the ratio and pattern of reward to response are carefully chosen to produce robust conditioning and maximum resistance to extinction. To some extent, this can explain gambling. But it seems not to be a sufficient explanation of normal gambling far less its pathological form. First, in animals simply subjected to intermittent schedules, as we noted earlier, the level of behavior conforms approximately to optimality—with over-responding being present only while information about a new reward density is being gathered. Second, quite apart from the local preponderance of negative reinforcement for the behavior, there is usually additional negative reinforcement in terms of the effect on other aspects of life, and this should produce robust avoidance behavior. Third, there is the brute fact that the majority of people who engage in recreational gambling do not develop pathological gambling behavior. These facts suggest that we must look elsewhere for

a sufficient explanation of this form of counterproductive behavior.

One alternative theory is to assume that people prone to pathological gambling have biased cognitions (e.g., "The more I lose, the more chance of have of winning"). We may suppose that such biases are important in maintaining pathological gambling, but such explanations are high on description but low on powers of explanation, and specifically fail to reveal *why* such cognitive biases exists, let alone *how* they relate to reinforcement sensitivity (which we know is important in gambling behavior). Nor do they explain the intensity of the behavior.

A possibility suggested by our current analysis is that pathological gambling develops as a as a form of self-defeating dopamine-mediated approach behavior. On this view, punishment summates with the expectation of rare, large rewards, to create a high level of arousal. It thus energises and invigorates behavior. Even if the schedule of reinforcement were net positive for the player (as it can be with games such as "21") it involves a background of fairly steady punishment, in the form of loss of the stake and reward omission. This means that when a reward occurs its effect is super-charged by the positive effects of relieving nonpunishment. The resultant physiological arousal acts in the same way as a drug, such as amphetamine, to create an emotional high that produces rapid and resistant conditioning (e.g., to the paraphilia of the gambling context). These emotional 'highs', that are *predicted* by the higher-density of punishments, can become associated with it and so, through counterconditioning, reduce its negative reinforcing value (which is weak at the level of the individual response). The overall picture, as with chemical addiction, is an overriding of background negative stimuli by occasional powerful stimulation of the dopamine system.

As yet these behavioral processes, and the apparently paradoxical fact that punishment in gambling seems to maintain pathological gambling itself, does not make much sense in traditional Skinnerian terms, but it finds a natural explanation within the context of the known properties of the dopamine system—and with the low level of genuine "pleasure" in those addicted to drugs.

Reward-Punishment Mutual Inhibition: Romantic Partner Abuse

A similar process to that seen in pathological gambling may also operate in romantic partners who suffer long-term abuse but who are reluctant to escape their abusing partner (i.e., are reluctant to engage in FFFS-mediated avoidance of the threat stimulus). Putting aside other relevant factors involved in such situations (e.g., children and financial dependence), some abused partners (both males as well as females—here the forms of abuse may differ) repeatedly fail to leave their partners who, on the one hand, they openly declare are abusing them, but, on the other hand, find it difficult to break away (even where there do not exist an financial, or other, objective reason, for doing so).

Partner abuse should be expected to activate the FFFS (as well as the BIS due to the likelihood of conflict) leading to punishment-mediated behaviors (in this case fear, tension, attempts to avoid/escape abuse). When the abusive partner reconciles, the abused partner will not only experience an absence of punishment (itself a good thing in terms of reduced FFFS activity), but also a strong boost to the BAS in the form of release of suppression of the reward system by the punishment (FFFS/BIS) system. As in the case of gambling (see above), relief of nonpunishment processes may also be assumed to operate. This release, and the subsequent rebound effects, would be expected to lead to a heightened BAS activity and an emotional high, which would stamp in, via conditioning, behaviors immediately preceding it, namely the partner's reconciliation behavior and associated stimuli—Konorski (1967) made a similar claim about the rebound effects in romantic 'making-up' behavior.

Once again, the FFFS/BIS-induced arousal would serve further to augment the rebound of the BAS, increasing the subjective intensity of the positive emotional high. (Rebound effects are also suggested by anti-anxiety drugs that are traded illegally for the highs they produce in some people.)

There is a further theoretical twist that would make an additional contribution to this BAS-mediated emotional high and resulting approach behavior (e.g., making up). The mutual inhibition of the reward and punishment systems would mean that the previous negative emotion and behavior associated with the punishment system would now itself be suppressed, making the abused partner, emotionally speaking, to forget (or, at least, attenuate the strength of) the previous punishment delivered by the partner.

Thus, we may predict that one of the major factors contributing to the continuation of abusive relationships is that the abused partner has a strong mutual inhibition between their reward and punishment systems, rendering a supercharged BAS input from the abusive partner's reconciliation behavior. It might be the case that the abusive partner learns how to manipulate the emotions of the abused partner, and this would contribute to the cycle of abuse.

SUMMARY

In our discussion of central states and theories of emotion and motivation, we ranged freely from the exotic, but fairly

well established, theories of the partial reinforcement extinction effect (PREE) to the prosaic, but not clearly understood, behavior of pathological gambling and romantic partner relations. We attempted to show that neural analysis can, and has, generated quite distinct theories that not only have the advantage of being tied to neural and pharmacological reality (and so are less subject to the whims of verbal definition) but also have the advantage of throwing into strong relief some of the less obvious properties of emotional and motivational systems. These properties derive from the fact the emotion and motivation involve multiple serial and parallel ROT, each of which has evolved separately but nonetheless regularly co-occurs with and is often seamlessly integrated with others.

The existence of multiple ROT itself creates an environment in which higher order control mechanisms can evolve. The addition of later, complex, ROT to sets of simpler ones has also tended to produce hierarchical structures with the quickest, dirtiest, and phylogenetically earliests mechanisms located at lower levels of the neuraxis and progressively slower and more sophisticated mechanism located at progressively higher levels.

We considered a number of current central theories of emotion and motivation. These differ in detail and even in their use of terms. But they can all be seen as sharing a fundamentally Hebbian (purely associative, as opposed to reinforced) view of basic memory processes; a picture of two fundamental reinforcement systems—with dopaminergic systems reinforcing specific responses whether these produce reward or relieving nonpunishment; a distinction between ventral ("what") and dorsal ("where") processing streams; a view that behavior results from neural processing of goals (stimulus/response or, better, occasion/affordance compounds); and a view of prefrontal cortex as holding potential or intended goals in mind (i.e., in "working" or "active" memory).

The take-home message is that emotion and motivation are intertwined and each is multifaceted. This is often blindingly obvious at the neural level—but still goes against the grain of our normal use of emotional terms. As we have seen, what is meant by "anxiety" can differ even among neurally driven theorists—making it unclear how far disagreements are about real facts or arbitrary definitions. What is needed, then, is recursive processing of neural and behavioral information. When the resultant "psychological" constructs are also firmly tied down to particular neural instantiations then we will be in a position to say that we truly understand the resultant structure of the behaviors emitted by the organism—and will be on the way to understanding our own minds from an objective standpoint.

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