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A Multiprocess Perspective
on the Neuroscience of Emotion

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During the past century, neuroscientists and psychologists have viewed emotion through different lenses. According to many contemporary psychologists our emotions are a product of the way in which we interpret the world. In this view, the way we think about, or appraise, the significance of an event determines whether it will make us happy or sad, angry or glad. The same stimulus, such as your brother punching you in the arm, will have an entirely different meaning depending upon whether his action seems deliberately harmful or playfully affectionate. How you respond to his punch will be determined by how you interpret its meaning. The goal of this research is to identify how appraisal patterns give rise to complexities of emotional experience, expression, and regulation (see, e.g., Frijda, 1986; Lazarus, 1991).

In contrast, neuroscientists have viewed emotions as expressions of inherited programs for action in specific situations that have been of importance to humans and related species for millions of years (see, e.g., Panksepp, 1998). In this view, complex emotions are learned responses to primary reinforcers that have been built on top of these simple and prepotent response tendencies (see, e.g., Rolls, 1999). The goal of research is to identify the neural systems responsible for the basic responses of fear, rage, disgust, affiliation, and so on. Although some researchers acknowledge that

neural systems carry out some simple forms of appraisal (e.g., for fear, see LeDoux, 1996), by and large, neuroscience theories simply do not speak to the issue of how complex person–situation relationships determine what feelings will be elicited.

Which view is correct? Are emotions the product of complex cognitive appraisals, or are they the product of simple programs embedded in our genes and brains? This is the crux of the conflict between psychology and neuroscience as it traditionally has been understood, and debates over this and related issues have been the source of much consideration (for discussion, see Ekman & Davidson, 1994; LeDoux, 1996). A complete account of emotion, however, should make reference to all levels of analysis, ranging from the feelings and behaviors associated with emotion to how they are computed at the neural level of brain structures and systems.

The purpose of this chapter is to begin sketching a theoretical framework that bridges these levels. We begin with the view that this conflict is more apparent than real by arguing that psychological and neuroscience approaches are asking complementary questions about emotion that are couched at different levels of analysis. In the following section we outline the basic elements of our framework, specifying two kinds of processes that are used to generate and regulate emotions. In this theory, emotion is the product of an interaction between simple, nonconscious, automatic processes and deliberative, conscious, and controlled processes. Then, in subsequent sections we use data from multiple fields to support and develop this theory, describing how the functions of specific brain regions can be understood in terms of their role in automatic or controlled emotion processing. Finally, we briefly consider how our theory can begin to foster a rapprochement between neuroscience and psychological approaches to emotion.[1]

AUTOMATIC AND CONTROLLED PROCESSING IN EMOTION

In recent years there has been an explosion of interest in questions concerning the nature of emotional experience, both in the scientific disciplines (see, e.g., Ekman & Davidson, 1994; Lewis & Haviland, 1993) and in lay domains (see, e.g., Damasio, 1994; Goleman, 1995; LeDoux, 1996). Many studies have been directed at determining what kinds of emotions people generate and when they report feeling them (see, e.g., Feldman, 1995; Feldman Barrett,1998). Studies examining the kinds of emotion regulatory strategies people use are receiving increasing attention as well (see, e.g., Gross & Levenson, 1993). Most of this research has been descriptive rather than causal in its analysis, however, leaving unexplored issues concerning the information processing mechanisms used to generate and regulate the emotional responses in question. Our theory is aimed at specifying the in-

formation processing mechanisms involved in emotion generation and regulation, identifying their neural substrates, and seeking to ultimately understand the factors that determine when and how effectively they are used. The present chapter tackles the first two of these three goals.

Many mental phenomena have been well modeled as the product of a quick and automatic process that sets the stage for a slower and more deliberative processes which modify and/or monitor ongoing activity (Chaiken & Trope, 1999). We hypothesize that emotion generation and regulation are no different. Considerable evidence suggests that the automatic processes associated with an emotional response both quickly and effortlessly classify people, objects, and events as positive or negative (Quigley & Feldman Barrett, 1999; Robinson, 1998). This *automatic emotion processing* is consistent with what has been called primary appraisal (Lazarus, 1991). It is also consistent with automatic evaluations of environment features (Bargh, 1990; Bargh, Chaiken, Govender, & Pratto, 1992; Bargh, Chaiken, Raymond, & Hymes, 1996; Chaiken & Bargh, 1993; Chartrand & Bargh, 1996; Fazio, Sanbonmatsu, Powell, & Kardes, 1986), various kinds of affective conditioning (sometimes with subliminally presented stimuli; see, e.g., Ohman, 1988), and the inability to ignore emotionally relevant information (as in the emotional Stroop Task; see MacLeod, 1991). An important aspect of automatic emotion processing is that the rapid detection of potential threats or possible rewards, and the accessing of associated information, can initiate appropriate approach or avoidance behaviors (e.g., fleeing a threat or approaching a reward). More complex emotion knowledge (in the form of discrete emotion scripts or mental representations; see, e.g., Fehr & Russell, 1984; Shaver, Schwartz, Kirson, & O'Connor, 1987) are thought to be deployed during the generation of an emotional response, either because they are chronically accessible or because it is directly primed or preconsciously activated by the mere presence of features in the environment (for a discussion, see G. A. Bonanno, Chapter 8, this volume). Such occurrences also constitute automatic emotion processing.

But emotions are only partly the result of processes that interpret the significance of events in an automatic, or bottom–up, fashion. We also consciously direct attention to internal sensations and thoughts or external people and objects, search for and retrieve information from memory, construct a representation of our experience, and select or inhibit our actions. Collectively, the use of directed, effort-demanding processes in the generation and regulation of emotion can be termed *controlled emotion processing*. Examples of controlled emotion processing abound in the clinical and experimental social psychology literatures and include the following:

- Studies of pain perception demonstrating that deliberately attending to and describing painful physical sensations can lessen the psychological experience of them as painful (Cioffi, 1993)

- Studies relying on self-reports of emotional experience (see, e.g., Feldman Barrett, 1998)
- Studies of emotion disclosure demonstrating that retrieving and recounting past personal traumas can lessen negative affect accompanying their recollection, and even improve one's physical health (see, e.g., Pennebaker, 1997)
- Studies of decision making demonstrating that emotions may sometimes help (see, e.g., Damasio, 1994) or bias judgments (Forgas, 1995)
- Studies of emotion regulation demonstrating our abilities to inhibit or alter ongoing emotional responses (Gross & Levenson, 1993)

By deliberately monitoring, activating, and processing emotions, one may consciously reconstrue the meaning of an experience and respond differently.

Automatic and controlled emotion processing can configure in a number of ways to produce emotional experience and expression. For example, consider the emotion generation process in an individual whose emotional reactions are complex and subtle. On the one hand, the fine texture of her experience could result from the automatic activation of a rich network of semantic and affective schemas (composed of both linguistic labels and organized personal experiences) that are easily accessible due to repeated use. In addition, past painful or rewarding experiences may have stamped in certain action tendencies and physiological responses that also are elicited automatically. In this way, her highly differentiated emotional response is mediated by a complex knowledge base without effort or intent. On the other hand, her automatic and quick responses could have been simple and undifferentiated, with the complexity of her experience and behavior arising only after she attempts to describe and understand her feelings. She might possess a rich and consciously accessible vocabulary specialized for doing so. But, unlike the first case, complex emotional responses would take shape slowly, requiring effort and concentration to apply emotion knowledge in the description and regulation of her feelings. Both of these examples stand in contrast to individuals who do not parse their emotional responses with much granularity or precision and instead rely upon global judgments of hedonic tone. They might simply note, "I feel good" or "I feel bad," either because they lack the knowledge, motivation, or executive capacity to construe their feelings otherwise.

Unfortunately, current understanding of the brain structures involved in emotion is still a long way from providing the precise neural dynamics underlying the complexities of everyday examples of emotion such as these. However, as elaborated in the section that follows, neuroscience data support the general theory that automatic and controlled processes are involved in emotion, and suggest further that each type of processing may be carried out by a number of separate neural systems.

NEURAL SYSTEMS FOR EMOTION GENERATION AND REGULATION

Evidence from multiple domains suggests that automatic and controlled emotion processes are carried out by at least five distinct neural systems. Each system plays a different but essential functional role in the generation and regulation of emotion. Each function is carried out by mechanisms that operate with differing degrees of deliberative control and are identified in the top row of Figure 2.1.

Considerable evidence suggests that the first three systems can operate automatically and comprise three distinct kinds of automatic emotion processing. Separate processes detect potential threats (Function 1) and possible rewards (Function 2), as well as acquire and execute appropriate approach or avoidance behaviors (e.g., fleeing a threat or approaching a reward). A third system (Function 3) adds complexity to these responses through the automatic activation of semantic emotion knowledge. This system oversees retrieval from memory of complex emotion knowledge that is used to form more discrete emotional experiences, attribute an emotional quality to a stimulus, and devise strategies to cope with emotional states and emotionally evocative stimuli.

The extent to which one deliberately differentiates and regulates this initial response is determined by the third, fourth, and fifth functions. The deployment of complex emotion knowledge (Function 3) can also occur under conscious direction. One can deliberately look up information in semantic memory about how to understand or regulate an emotional response, as well as to decide whether to alter that response. The fourth system determines whether it is necessary to seek greater understanding or control over emotional responses (Function 4) by detecting discrepancies between competing response tendencies or consciously held plans. When a discrepancy is detected, one can deliberately use emotion knowledge to alter or regulate the emotional response (i.e., a return to Function 3). In addition, we need to evaluate the current affective meaning of an external stimulus or behavioral response so that one can make the decisions or take the actions necessary to make these changes (Function 5). The distinctions between these different functions and between automatic and controlled emotion processing is supported by various types of neuroscience data, reviewed below.

The Amygdala: Detecting and Responding to Potential Threats

Currently, more is known about the function of the amygdala in emotion than any other brain structure. In the past few years, data from various domains have provided converging evidence that the amygdala might be best characterized as a preattentive analyzer of the environment that looks for

BRAIN STRUCTURE	Amygdala	Basal Ganglia	Lateral Prefrontal and Association Cortices	Anterior Cingulate Cortex	Orbital and Ventromedial Frontal Cortex
FUNCTION	Detecting & Learning about Potential Threats	Registering Rewards & Acquiring Habits	Retrieving and Storing Semantic Emotion Knowledge	Conflict Monitoring	Context-dependent action selection
USE AND APPLICATION	Detect arousing, potentially threatening stimuli and associate them with corresponding physiological responses and appropriate actions	Automatize sequences of behavior and thought that have proved consistently reinforcing	Identify stimuli & differentiate feeling states; attribute emotional qualities to stimuli; repository of regulatory strategies & lay emotion knowledge	Monitor on-going behavior and determine whether change is necessary	Inhibit ongoing emotional responses based on analyses of context; generate affective reactions based on these analyses that guide further regulatory judgments
TYPE OF PROCESS	Automatic	Automatic, but requires attention	Retrieval can be Automatic or Controlled	Conflicts detected automatically, but making changes takes control	Controlled

FIGURE 2.1. Chart showing the brain structures thought to be important for emotion processing, their functional role, how they are used and applied to processing emotional information, and the type of processing they carry out (whether it is automatic, controlled, or both).

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significant information that should be encoded into memory (Holland & Gallagher, 1999; LeDoux, 1996; Whalen, 1998). It would make sense for a system performing this function to be biased toward the early detection of ambiguous but evocative stimuli, even though these objects may ultimately prove to be either threatening or rewarding. For both kinds of stimuli, the amygdala would code the association between the appearance of stimulus and the affective response evoked. But if the stimulus proves to be rewarding over time, other areas may be more important for promoting the long-term reinforcement of approach behaviors that the amygdala is not designed to perform (such as the basal ganglia, discussed in the following subsection). In this way, the amygdala would still play a role in encoding the significance of rewarding/positive stimuli but would play a different role in mediating behavior toward those stimuli later on.

The anatomy of the amygdala is consistent with this conclusion (for location see Figure 2.2A). Information about the identity of a stimulus can reach the amygdala by one of two routes: the first is a cortically based system used to recognize stimuli on the basis of distinct perceptual features; the second consists of more direct connections to sensory organs via the thalamus, bypassing the longer cortical route (Aggleton, 1992). In a series of experiments conducted with rats, LeDoux and colleagues have shown that each input pathway supports a different kind of emotional learning. The cortical route allows the discrimination of stimuli on the basis of complex analyses of their distinctive features, as well as the acquisition of differential conditioned responses to them. In contrast, the subcortical pathway by itself can support rapid leaning of conditioned responses to crude, coarsely defined perceptual stimuli (LeDoux, 1996; LeDoux, Romanski, & Xagoraris, 1989). On the basis of these results, LeDoux suggested that the subcortical pathway provides a quick analysis of the affective properties of stimuli that serves as an initial template for subsequent processing.

Some recent results in humans and animals have corroborated LeDoux and colleagues' findings. Neuroimaging studies have shown amygdala activity while subjects are learning to associate aversive noise with neutral tones (LaBar, Gatenby, Gore, LeDoux, & Phelps, 1998), and neuropsychological studies have shown that amygdala lesions block the acquisition of such responses (LaBar, LeDoux, Spencer, & Phelps, 1995). In addition to its role in these implicit forms of memory, the amygdala also has been shown to influence the consolidation of explicitly accessible, episodic memories for emotional events (for further discussion see Chapter 3). For example, recall of the emotional elements of a negative story is correlated with amygdala activity during encoding (Cahill et al., 1996), and degenerative decay of the amygdala due to disease eliminates this memory advantage (Cahill, Babinsky, Markowitsch, & McGaugh, 1995; Markowitsch et al., 1998). Studies in animals have indicated that modulation of
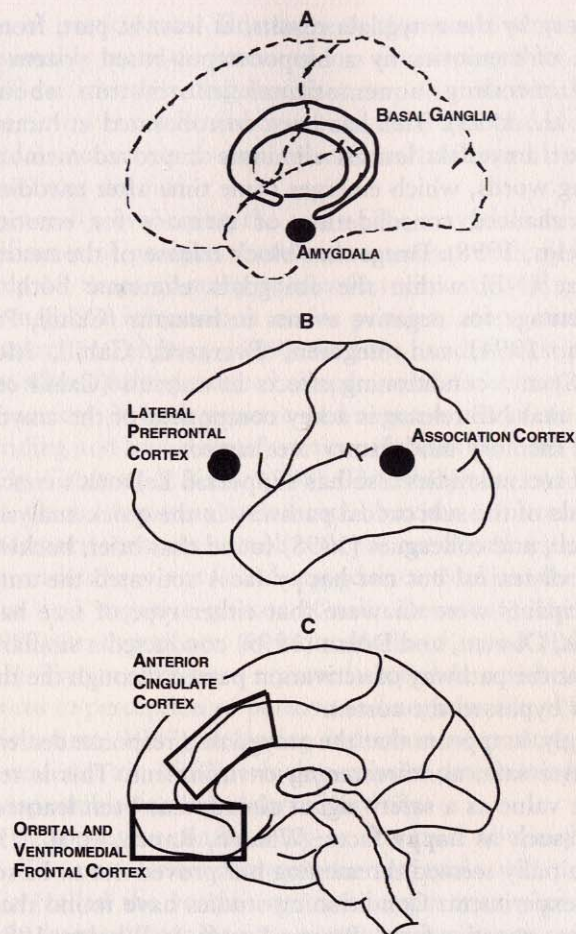
FIGURE 2.2. Anatomical locations of the brain structures discussed in this chapter. Figure 2.2A depicts two subcortical structures shown with dark (solid) lines beneath a transparent (dashed line) lateral view of the left hemisphere. The basal ganglia are important for encoding sequences of thought and behavior that have proved rewarding over time. The amygdala is important for detecting threats and encoding relationships between the sensory characteristics of stimuli and the internal affective states elicited by them. Figure 2.2B depicts two cortical areas superimposed on a lateral view of the left hemisphere. The left prefrontal cortex is important for looking up abstract semantic and associative knowledge in a posterior area that stores semantic information. Figure 2.2C depicts two cortical areas superimposed on a medial view of the right hemisphere. The anterior cingulate cortex (ACC) is used to track the extent to which current behavior is failing to achieve a desired outcome. Signals from the ACC are used to indicate errors, conflicts, pain, uncertainty, anxiety, and violations of expectation. The ventromedial frontal cortex (VMFC) and orbital frontal cortex (OFC) are used to represent the current affective value of a stimulus in the context of current goals. This subsystem enables an individual to alter responses to a stimulus on the basis of its shifting motivational significance.

explicit memory by the amygdala results, at least in part, from enhancing consolidation of memories by a hippocampus-based system that is specialized for encoding nonemotional information about episodes (McGaugh et al., 1995). This has been corroborated in humans by demonstrating that amygdala lesions eliminate improved memory for negatively arousing words, which emerges some time after encoding and is attributed to enhanced consolidation of memory for emotional stimuli (LaBar & Phelps, 1998). Drugs that block release of the neurotransmitter norepinephrine (NE) within the amygdala eliminate both the explicit memory advantage for negative events in humans (Cahill, Prins, Weber, & McGaugh, 1994; van Stegeren, Everaerd, Cahill, McGaugh, & Gooren, 1998) and conditioning effects in animals (Cahill et al., 1995). This suggests that NE release is a key component of the amydala's affect-encoding and memory modulatory mechanism.

A pair of recent studies also has supported LeDoux's conclusions concerning the role of the subcortical pathway in the quick analysis of stimuli. Whalen, Rauch, and colleagues (1998) found that brief, backward-masked presentations of fearful but not happy faces activated the amygdala even though participants were unaware that either type of face had been presented. Morris, Ohman, and Dolan (1999) conducted a similar experiment and found that the pathway of activation passed through the thalamus and amygdala but bypassed the cortex.

Intriguingly, it appears that the amygdala's response decreases to stimuli that signal a safe, nonthreatening environment. This is true both for stimuli whose value as a safety signal already has been learned before the study begins (such as happy faces; Whalen, Rauch, et al., 1998), or for stimuli that initially seemed threatening but proved not to be so during the course of an experiment. Conditioning studies have found that the amygdala response to negative faces (Breiter, Etcoff, & Whalen, 1996; Morris et al., 1996) or aversive conditioned stimuli (LaBar et al., 1998) decreases and habituates with repeated presentation, although activity in other cortical areas does not decrease. An important question for future research will be to determine whether these decreases in amygdala activity are due to passive habituation or active inhibition by other areas.

A system sensitive to potential threats[2] should be activated by positive stimuli that are relatively unfamiliar or novel. This was demonstrated recently by a neuroimaging study investigating perception of unfamiliar faces of black and white individuals by African American and European American participants (Hart, Whalen, Shin, McInerney, & Rauch, 1999). For African American individuals, black faces should presumably be more positive and less threatening than are white faces, whereas the opposite should be true for European American participants. However, during an initial block of trials, amygdala activation was observed for both types of faces in

both groups of participants. This suggests that novel same-race faces, although presumably more positive and less threatening than are other-race faces, are still sufficiently ambiguous that they elicit an initial amygdala response. This view was confirmed by results from a second block of trials in which amygdala activation to same-race faces habituated but activation to different-race faces did not.

More generally, evaluating the role of the amygdala in coding the affective significance of positive stimuli is somewhat difficult at present because roughly 10 times as many studies have investigated perception and memory for negative information as for positive information (based on a *PsycINFO* search in February 1999). And the handful of studies that have used positive stimuli have produced mixed results. One neuroimaging study has related memory of both positive and negative stimuli to amygdala activity at encoding and found significant correlations for both stimulus types (Hamann, Ely, Grafton, & Kilts, 1998). This study also included a control condition in which interesting and unusual but not emotional stimuli were presented. Memory for these stimuli was not correlated with amygdala activity. Other studies have not found amygdala activation when participants view positive and negative photos (Canli, Desmond, Zhao, Glover, & Gabrieli, 1998) or experience an induced elated mood (Baker, Frith, & Dolan, 1997). Two studies have reported amygdala activation when averaging responses to perception of positive and negative films (Lane, Reiman, Ahern, & Schwartz, 1997) or during induction of positive and negative moods (Schneider et al., 1997), which precludes determining which stimulus type is responsible for the observed activity.

Studies of appetitive conditioning in rats have obtained more consistent results. The connection between the amygdala and portions of the basal ganglia (called the ventral striatum) appears to play a key role in these animals learning to associate neutral stimuli with appetitive (e.g., food or sex) rewards (Everitt & Robbins, 1992; Schultz, 1998). Lesions of the basal ganglia impair the acquisition of such associations, whereas lesions to the striatum impair the transformation of such associations into habitual responses to the reinforced stimulus (Everitt & Robbins, 1992; MacDonald & White, 1993). Single-unit recording studies in animals show that once learned, cells in the basal ganglia can signal the positive or negative reward value of a stimulus (Rolls, 1999). Although the amygdala may store associations between the appearance of a rewarding stimulus and the physiological responses it elicits, other brain regions such as the ventral striatum or medial and orbital frontal cortex seem to be more important than the amygdala for the perception of stimuli that already have acquired positive/reward value (see Adolphs, 1999; Lane, Reiman, Bradley, et al., 1997; Rolls, 1999). These brain regions are discussed in the subsections that follow.

Summary

Animal and human studies are generally consistent with the claim that the amygdala functions to determine whether incoming stimuli are threatening and, if so, to rapidly associate perception of those stimuli with the appropriate responses. Any novel or ambiguous stimulus may initially seem threatening and thus warrant a response from the amygdala.

The Basal Ganglia: Learning to Skillfully Attain Rewards

Situations that elicit positive and negative affect seem to require very different kinds of responses. On the one hand, it behooves us to learn very quickly and rapidly that something (or someone) engenders fear, anger, or disgust so that we can respond immediately and appropriately the next time we encounter it. On the other hand, it makes sense to stamp in behaviors and thoughts that have led to a desirable end only if they continue to do so reliably. The old aphorism, "Fool me once, shame on you, fool me twice, shame on me," suggests that it is advisable to be sure that rewards are due neither to chance nor deception. Whereas the amygdala is especially well suited for the former function (as discussed above), the basal ganglia are especially well suited for the latter. The basal ganglia are designed to slowly encode sequences of behavior that, over time, have been repeated and rewarded—or at least not punished (Lieberman, 2000). The representations thus encoded support not only the execution of habitual behaviors but the prediction of what comes next in a sequence of thoughts or actions.[3]

Anatomically, the basal ganglia are well suited for making habitual the patterns of action or thought that repeatedly have led to a desired or positive outcome. They lie in the center of the brain underneath the cortex, receive inputs from areas of the parietal and temporal lobes that code the spatial and physical characteristics of a stimulus, and send outputs to various motor control centers (see Figure 2.2A). The basal ganglia also participate in a number of functional control circuits that link it with areas of the frontal lobe and other cortical regions (Alexander, Crutcher, & DeLong, 1990). Each circuit has a specific functional domain, including spatial and object working memory, and motor control. One of these circuits connects four of the structures for emotion discussed in this chapter: the amygdala, ventral portions of the basal ganglia, the anterior cingulate cortex, and the orbital frontal cortex.

The basal ganglia are composed of two main parts, the caudate and putamen, which are involved with habitual cognition and action, respectively (Alexander et al., 1990; Houk, Davis, & Beiser, 1995; Lieberman, 2000). Lesions to the caudate, either as a result of stroke or degenerative

disease (e.g., Huntington's chorea) impair perception of emotion conveyed through facial expression and tone of voice (see, e.g., Cohen, Riccio, & Flannery, 1994; Speedie, Wertman, Ta'ir, & Heilman, 1993). Importantly, the perception of vocal prosody, which requires integration of changes in vocal tone across time, is impaired by damage to the basal ganglia but not to the amygdala (Anderson & Phelps, 1998). In contrast, damage to the putamen impairs the production of nonverbal behavior, including emotional intonation and the production of voluntary facial expressions (see, e.g., Van Lancker & Pachana, 1995).

Many streams of animal and human research demonstrate that the sequencing and habit-forming function of the basal ganglia play a special role in positive emotion. For example, basal ganglia damage in rats eliminates the potentiation or rapid repetition of responses to rewards that increases with repeated receipt of them (Everitt & Robbins, 1992) and eliminates the ability to learn simple stimulus–reward associations that are repeated over time (Mishkin & Appenzeller, 1987; Packard, Hirsh & White, 1989). In humans, selective left basal ganglia lesions often cause depression, as do left prefrontal lesions (Robinson & Paradiso, 1996), and depression is common among patients with either Huntingtons's chorea (Hopkins, 1994) or Parkinson's disease (McPherson & Cummings, 1996), both of which involve basal ganglia degeneration. Neuroimaging results dovetail with and extend these neuropsychological findings. Basal ganglia activation has been found during the subconscious registration of positive faces (Morris et al., 1996), during the experience of positive but not negative emotion elicited by films or recall of personal experiences (Lane, Reiman, Bradley, et al., 1997), and during cocaine-induced euphoria (London et al., 1990). Selective caudate activation has been observed during the presentation of emotional words (Beauregard et al., 1997) and positive pictures (Canli et al., 1998). Some investigators have found basal ganglia activation during recall of sad but not happy memories (George et al., 1995; Lane, Reiman, Ahern, & Schwartz, 1997); however, the exact reason for these findings is unclear at present.

A problem with many of these studies is that they do not make clear whether basal ganglia involvement in positive affect is associated with the experience of positive affect per se, with the activation of learned response sequences that promote movement toward a reward, or both. Some other evidence suggests that the basal ganglia may be especially important for the approach-related behaviors associated with various kinds of emotions (but are particularly characteristic of positive emotional states). Berridge and Robinson (1998) have dissociated the processes involved in approach-related behaviors and the experience of reward. Their studies show that dopamine release in the basal ganglia changes how much a rat works to get a reward but not how it responds once that reward is received. They suggest that dopamine in the basal ganglia mediates "wanting," or the motivation

to seek out and approach a reward or outcome, but not the phenomenal "liking" of that reward as it is experienced (see Breiter & Rosen, 1999).

Summary

The basal ganglia are important for encoding the temporally patterned stimulus–stimulus and stimulus–response relationships that underlie implicit cognitive and motor skills. These implicit skills are essential because they allow us to make automatic the sequences of thought and action that lead to the attainment of goals and receipt of rewards of various kinds.

The Lateral Prefrontal and Association Cortex: Using Complex Emotion Knowledge

Much of the knowledge that we use to assess the emotional relevance of stimuli and events is stored in the form of organized knowledge structures that specify the meaningful relationships among different stimuli (Fiske & Taylor, 1991). These emotion concepts or schemas may represent several things (Fehr & Russell, 1984; Mesquita & Fridja, 1992; Shaver et al., 1987; Shweder, 1993) including:

- The abstract cause of an experience
- The meaning of a situation to the individual and his or her immediate goals
- Bodily sensations
- Expressive modes (i.e., display rules for expression)
- How the emotion functions interpersonally
- Sequences of action to take to enhance or reduce the experience (i.e., plans of emotion management)

These kinds of emotion knowledge may function like culturally constructed internal guides or working models of emotional episodes (Saarni, 1993). This knowledge may be learned episodically, through experience. For example, children rapidly learn the type of psychological events and abstract situations that are associated with particular emotion labels (fear, sadness, happiness, anger, guilt, and so forth; see, e.g., Harris, Olthof, Meerum, Terwogt, & Hardman, 1987). They are also aware of the typical actions and expressions that are supposed to accompany a particular emotional state (Trabasso, Stein, & Johnson, 1981). Over time and repeated use, however, this episodic knowledge may become instantiated as semantic representations of the possible objects that can cause an emotional experience, the relational contexts associated with the experience, and the behavioral

repertoire that exists for dealing with the experience and the larger situation.

Currently, we know much more about the structure and function of semantic memory than we do about its neural locus. Classic models of semantic memory depicted it as a system of linked information nodes, where the number of links between nodes corresponds to the conceptual distance between two pieces of information in the associative network (Bower & Forgas, 2000). Thus, the concept "doctor" and "nurse" are separated by fewer links than are "doctor" and "horse." The system of associations for a given concept has links or pointers to visual, auditory, and other representations of that concept in separate, modality-specific processing/memory systems (Kosslyn & Koenig, 1992; Ochsner & Kosslyn, 1999). More recent connectionist models of semantic memory describe sets of subsymbolic or subconceptual nodes, which become active in different combinations to represent higher-order conceptual information (Rumelhart, 1989). These models readily explain how existing schematic knowledge can automatically facilitate encoding and retrieval by filling in missing information and guiding the interpretation of ambiguous stimuli (see, e.g., McClelland, 1995).

Although semantic memory involves widespread connections throughout the entire brain, neuropsychological and neuroimaging studies suggest that the left temporal–parietal–occipital junction may play a special role in storing semantic information. Such studies also suggest that the left inferior prefrontal cortex may be important for retrieving semantic (especially verbal) information (see Figure 2.2B; see also Kosslyn & Koenig, 1992; Tulving, Kapur, Craik, Moscovitch, & Houle, 1994). Storage and access to semantic information about the emotional connotations of verbal material might well depend more on these structures in the right than in the left hemisphere (Borod, 1992). Both areas receive highly processed information from all sensory modalities.

Semantic memory plays a role in emotion in several different ways: First, semantic memory is a repository of schematized knowledge about the origins, evolution, and sequelae of our emotions. It includes our implicit or explicit theories about what emotions are, when we feel them, why we feel them, and what we should do when we feel them. People may differ in their degree of emotion knowledge and the way they use it to cope, and such differences can have a profound impact on the emotions they experience and their ability to cope with them (Lane & Schwartz, 1987). When semantic knowledge is activated without accompanying activation of the amygdala or basal ganglia, it can be used to tell us something about an emotion. But when semantic knowledge is activated in conjunction with either or both of these systems, the activated knowledge becomes part of an emotion. That is, a discrete emotional episode can emerge in the context of complex emotion knowledge that allows us to differentiate, label, and even draw inferences about our emotional states.

Second, repeated use of semantic knowledge "greases the wheels" of accessibility and over time can lead to the automatic activation of chronically accessed knowledge in the presence of appropriate cues. For instance, depressives tend to evaluate themselves negatively and view the future with great pessimism. Repetition of these thoughts over time may make access to them so automatic that it is not impeded by performing another task at the same time this information is being retrieved (Andersen, Spielman, & Bargh, 1992; Bargh & Tota, 1988). Highly accessible and schematized emotion knowledge also can guide the construal of ambiguous events. For example, depressives will interpret neutral sentences as negative and remember them that way later on (Williams, Watts, MacLeod, & Mathews, 1990). Interestingly, depressives show decreased activation of the left inferior prefrontal cortex, an area important for effortfully retrieving semantic information (Drevets & Raichle, 1998). The hypofunctionality of this area could belie depressives' tendencies to rely on automatically accessible negative information.

A third way in which semantic memory plays a role in emotion is by providing a link between stimuli with similar valence. This role was advocated strongly by Bower (see Bower & Forgas, 2000; see also Bradley, 1994), who proposed that there are nodes for different emotions in semantic memory. In this model, words, objects, visual images, and so on, gain emotional meaning through their common connections with these emotion nodes. This model can account for many of the effects of moods or affective states on judgment and memory, in terms of the spread of activation between items that share links with a given emotion node (for a review, see Bower & Forgas, 2000, although this model has been heavily criticized and supplanted by more elaborate views of emotion and memory—see Chapter 3). Repeated use of these affective associations can also make the activation and spread among them automatic. Similarly valenced concepts will thus tend to activate each other by virtue of their semantic, emotional association (Bargh et al., 1992; Fazio et al., 1986; Hermans, De Houwer, & Eelen, 1994).

A fourth and final point about semantic emotion knowledge concerns its relationship with the associations stored by the amygdala and the amygdala's role in consolidating episodic memories that become the basis for semantic memory. Semantic knowledge is derived from regularities in our daily episodic experience, and many different kinds of experience contribute to the emotional meaning of people, places, events, and so on. Undoubtedly, much of this knowledge comes from the norms for emotional expression specified by our familial and national cultures (see, e.g., Markus & Kitayama, 1991). Importantly, this emotion knowledge is stored separately from the affective associations stored by the amygdala, and it can influence behavior in different ways. Recent research has shown that patients with amygdala lesions can rate the emotional valence and the degree of

arousal elicited by photos (see, e.g., van Stegneer et al., 1998) in the same way as control subjects do. This is despite the fact that they fail to show the boost in episodic recall for the emotionally evocative information that controls exhibit (Cahill et al., 1994). Similarly, patients with amygdala lesions may fail to show fear conditioning, even though they possess explicit knowledge about the relationship between the conditioned and unconditioned stimuli (see, e.g., Bechara, Tranel, Damasio, & Adolphs, 1995). Data such as these suggest that explicit judgments of the emotional meaning of stimuli may be guided by explicitly accessible semantic knowledge independent of the associations coded by the amygdala. Furthermore, these data support the division between processes that automatically detect potential threats (Function 1) and processes that learn more complex semantic information about them.

Although each type of knowledge is accessed in different ways, the systems do interact. Their interaction allows the amygdalar response to significant and arousing stimuli to influence the development and consolidation of semantic knowledge. This has been shown strikingly by the contrast between patients who have suffered amygdala damage in adulthood and patients who suffered amygdala damage early in life. The former judge emotional faces to be as arousing as controls do (Adolphs, Russell, & Tranel, 1999, cited in Adolphs, 1999), whereas the latter judge the faces to be much less arousing (Adolphs, Lee, Tranel, & Damasio, 1997). It is possible that loss of the amygdala early in life keeps an individual from learning the significance of certain facial expressions because they could not become aroused when seeing them.

Summary

Semantic emotion knowledge is intimately involved with the generation of distinct emotional experiences. It is the repository of emotion concepts and theories, and links diverse and discrete memories together that share a common emotional association. We can draw upon this database automatically during the generation of an emotional state, or when we consciously represent or label emotional states to draw inferences about the emotions we are experiencing. Although it is not yet known exactly which brain structures mediate automatic use of this knowledge, it is clear that effortful access involves areas of the lateral prefrontal cortex specialized for this function (Tulving et al., 1994). It also is not yet determined definitively whether these lateral prefrontal brain regions alone are responsible for the labeling and attribution of emotional states. The emotional contents of semantic memory have been influenced by the amygdala, which facilitates consolidation of episodic memories for significant, arousing events. Research on the relationship between episodic and semantic memory has shown that over

time the important regularities of our episodic memories slowly become incorporated into our database of semantic knowledge. This suggests that the amygdala contributes to the development of semantic knowledge by influencing what information is incorporated into long-term semantic memory.

Anterior Cingulate Cortex: Evaluating the Need for Controlled Processing

Regulation or conscious modification of emotional responses requires that one know such intervention might be necessary. Evaluating the need for regulation is the function of the anterior cingulate cortex (ACC), and this evaluative function is an essential part of many types of controlled processing. At a broad level, the ACC can be seen as part of an executive system used to regulate behavior in many domains (Ochsner et al., 2000; Posner & DiGirolamo, 1998; Shallice, 1994; Stuss, Eskes, & Foster, 1994).

The evaluative role of the ACC is supported by its rich connectivity with many brain areas. The ACC is a large heterogeneous area on the medial wall of each hemisphere just behind the frontal lobes (see Figure 2.2C), and different subregions within the ACC have connections with different parts of the brain. Like the orbital frontal cortex (OFC) and ventromedial frontal cortex (VMFC), the more anterior parts of the ACC have connections with other frontal areas as well as with subcortical areas involved in emotion, such as the amygdala, hypothalamus, and striatum. More posterior areas of the ACC have interconnections with frontal, parietal, and subcortical areas involved in attention. Separate middle and posterior subregions of the ACC have connections with cortical and subcortical areas involved in motor control and pain, respectively (Devinsky, Morrell, & Vogt, 1995; Dum & Strick, 1993). A large fiber tract, the cingulum bundle, courses through the center of the cingulate gyrus connecting the different subregions and may facilitate communication between them and functional integration among them (Ballantine, Flanagan, Cassidy, & Marino, 1967).

Data from animal and human studies has implicated the ACC in various kinds of behaviors that involve monitoring and evaluation of one's behavioral performance, one's internal state, or the presence of external rewards. The ACC also monitors for the occurrence of events (such as uncertainty, conflict, or expectancy violation) that signal the need for a deliberate change in behavior. A clear example of such monitoring comes from neuroimaging and event-related-potential studies in humans (Carter et al., 1998; Deheane, Posner, & Tucker, 1994; Gehring, Goss, Coles, Meyer, & Donhin, 1993) and single-unit recording studies in monkeys (Brooks, 1986). These studies have shown that the ACC emits a signal whenever participants make errors in simple reaction time tasks. Importantly, this signal is larger when participants are more motivated to respond correctly. The signal occurs whether participants become aware that they have re-

sponded incorrectly on their own (Gehring et al., 1993) or when error feed-back has been provided (Badgaiyan & Posner, 1997). The signal also occurs when the correct response has been made but an expected reward is withheld (Brooks, 1986).

More generally, the ability to monitor both for errors and for the correct execution of desired responses is necessary whenever sensory input or behavioral performance is being closely monitored to ensure optimal performance.[4] Such monitoring is necessary during the learning of new skills, when uncertainty regarding performance parameters is high. Neuroimaging studies have shown that the ACC is active during the learning of motor sequences (Rauch, Savage, Brown, et al., 1995) or word pairs (Raichle, 1997), but this activity drops away when such tasks have been well learned, performance has become habitual, and monitoring is no longer necessary. Physical pain is another important signal that current behavior is not meeting desired ends. Neuroimaging studies have shown that ACC activity is correlated with the degree of painful stimulation experienced (Davis, Taylor, Crawley, Wood, & Mikulis, 1997; Porro, Cettolo, Francescato, & Baraldi, 1998; Rainville, Duncan, Price, Carrier, & Bushnell, 1997; Talbot et al., 1991) even if it is illusory (i.e., not due to direct physical stimulation; see Craig, Reiman, Evans, & Bushnell, 1996). ACC lesions (for cingulotomy) lessen the psychological experience of pain but not the ability to discriminate differences in the amount of painful stimulation (Hebben, 1985).

The evaluative process sensitive to uncertainty, conflict, pain, or expectancy violation may play an important role in both the generation and regulation of emotion. For example, an ACC signal may be involved with the transformation of an initial affective response into a discrete emotional episode. When a response is initiated, the affective feeling can be attributed to an object via the deliberate use of emotion knowledge, resulting in the initiation of an emotional episode. ACC activation is probably a necessary component of this emotion generation process. After an emotion is generated, an ACC signal that the current course of behavior is in need of change could also initiate searches for more appropriate responses and the causes of conflict. The experience of conflict or pain accompanying ACC activation may thus be part of more complex emotional responses that emerge as other emotion systems become activated. In this way ACC activation can be the trigger for a cascade of responses that brings experience and behavior in line with expectations or situational requirements.

These speculations are supported by recent studies showing that monitoring one's current emotional state while viewing emotionally evocative photographs activates portions of the very same region of the ACC responsive to pain (Lane, Fink, Chau, & Dolan, 1997; Lane et al., 1998). Moreover, studies with both animals and humans have shown that monitoring

the affective states elicited by painful and rewarding stimuli is important for learning about their significance. In rabbits ACC lesions impair the ability to discriminate reinforced from unreinforced stimuli (Gabriel, 1993), and in humans ACC activity is correlated with the acquisition and expression of conditioned skin conductance responses (Fredrikson, Wik, Fischer, & Andersson, 1995; Fredrikson et al., 1998). Interestingly, ACC lesions eliminate the distress call emitted by infant monkeys when separated from their mothers, which also could be due to an inability to evaluate and experience the pain associated with separation (von Cramon & Jurgens, 1983).

The ACC also seems important for mediating some of the physiological changes associated with monitoring and learning about significant stimuli. ACC lesions eliminate both the anticipatory slowing of the heart rate that precedes presentation of a conditioned stimulus in rabbits (Buchanan & Powell, 1993), as well as the gastrointestinal distress produced by learned helplessness in monkeys (Henke, 1982). And electrical stimulation of the ACC causes changes in the heart rate and respiration in both animals and humans (Buchanan & Powell, 1993; Pool & Ransohoff, 1949).

A final and important point is that it is not yet clear whether ACC activation is associated merely with the occurrence of events over which control should be exerted or whether it plays a direct role in implementing this control as well. The data reviewed above suggest that the ACC represents the conscious correlates of pain, uncertainty, conflict, emotional experience, and expectancy violation, signaling that behavioral change and reorientation of attention may be necessary. But the data necessary to indicate that the ACC helps implement these changes is somewhat ambiguous. Lesion studies of ACC function sometimes show deficits in emotional behavior (see, e.g., Damasio & Van Hoesen, 1986) or executive control (see, e.g., Cohen, Kaplan, Moser, Jenkins, & Wilkinson, 1998; Ochsner et al., 2000) but not always (see, e.g., Corkin, 1980). Part of the reason for these discrepancies may be that the patients studied often have lesions in other brain areas as well (see, e.g., Damasio & Van Hoesen, 1986) or that the studies have involved psychiatric populations whose brain function may have been abnormal before ACC damage occurred (see, e.g., Corkin, 1980).

Studies of psychiatric populations may shed unexpected light on ACC function, however. Psychosurgical lesions to the ACC have been used as a treatment for mood-related disorders such as obsessive–compulsive disorder (OCD), chronic pain syndrome, and depression (Baer et al., 1995; Ballantine, Bouckons, Thomas, & Giriunas, 1987; Ballantine et al., 1967). When such cingulotomies are effective, patients report a lessening of the anxiety associated with their symptoms even though the frequency of symptoms does not immediately decline. The conflict and uncertainty signaled by the ACC may be an essential ingredient of anxiety, which would explain the efficacy of cingulotomy for treating anxiety disorders.

However, cingulotomy results in either no deficit or minor and short-lived deficits in performance of various cognitive tasks, including many that activate precisely the ACC area that has been surgically removed (Cohen, Kaplan, Meadows, & Wilkinson, 1994; Corkin, 1980; Janer & Pardo, 1991; Ochsner et al., 2000). If the ACC is necessary for implementing regulatory behavioral changes, then deficits in task performance should have been observed. But if the ACC is only responsible for signaling the need for control, then we would expect performance deficits only if one is trying to use this conscious signal but is unable to do so. Thus it is possible that cingulotomy patients, who experience a great deal of uncertainty and conflict before the operation is performed, have become quite skilled at *ignoring* the signal that the ACC generates. Because they are quite good at performing tasks without using their anxiety as an indicator of performance accuracy, when the ACC is lesioned they suffer no performance decrement whatsoever.

Summary

The ACC has many subregions that together seem to serve a similar function in different domains. Together they enable the ACC to evaluate the "congruence" of feelings that one is experiencing by signaling uncertainty, conflict, or pain. The ACC also may be important for determining whether a stimulus will generate threat or pain in the future. This evaluation is represented in consciousness and can be used by other components of an executive system responsible for self-monitoring and regulation.

The Orbital and Ventromedial Frontal Cortex: Selecting and Implementing Regulatory Actions

Our bottom–up emotional responses are not always appropriate for every situation, and effective emotion regulation involves the active modification of these prepotent responses as well as the active use of emotional responses to guide judgment and decision making. Data from both animal and human studies indicate that the OFC and VMFC (see Figure 2.2C) are important for selecting and implementing these regulatory actions (Rolls, 1999; Stuss et al., 1993). The ability to deliberatively deploy regulatory responses based on an analysis of the current context requires integrating many different kinds of information, including bottom–up analyses of the affective value of a stimulus and information about situational factors that might indicate a change in those values. Anatomically, these areas are well suited to integrate these kinds of information: they receive input from every sensory modality, have reciprocal connections with subcortical and

brainstem nuclei involved in emotion, and receive input from other areas of the frontal and temporal lobes that integrate and associate information from many modalities (Vogt, 1986).

Selecting and implementing the appropriate means of regulation requires the ability to determine the motivational relevance of an object (be it a person, place, thing, or thought), and studies in humans and animals suggest that OFC/VMFC is involved in this process. Neurophysiological recording studies in monkeys have shown that OFC areas are sensitive to stimuli with reward value, including faces, tastes, and smells only if such stimuli are relevant to current goals or needs. Thus, food-sensitive neurons will respond only when an animal is hungry (Rolls, 1999). OFC neurons also are capable of rapidly learning to associate a novel stimulus with a reward, firing whenever the newly reinforced stimulus is presented. Importantly, these neurons will cease firing to that stimulus soon after its presentation is no longer reinforced, even though neurons in subcortical areas (such as the amygdala or basal ganglia) may continue to fire when that stimulus is shown (Rolls, 1999). This suggests that whereas subcortical areas continue to represent information about the past reinforcement properties of a stimulus, the OFC/VMFC tracks the current affective value stimuli. And, when necessary, the OFC/VMFC changes their value when a stimulus–reward pairing changes (see Bouton, 1994).

Neuroimaging experiments in humans also support the OFC/VMFC's role in computing the motivational value of external stimuli. OFC activation has been found during the perception of both primary reinforcers, including pleasantly experienced touches, odors, and tastes (Francis et al., 1999), and secondary reinforcers including happy or fearful faces (Morris et al., 1998), negatively valenced words (Beauregard et al., 1997), and visual mental images of aversive scenes (Shin et al., 1997). Similarly, VMFC activation has been found for negative photos (Canli et al., 1998), anxiety elicited by anticipation of painful electric shock (Drevets, Videen, Price, & Preskorn, 1992), and the experience of sadness elicited by the combined recollection of sad personal memories and viewing of photos of sad faces (Drevets et al., 1992). A limitation of these studies is that they have not been designed to test whether OFC activity is due to the experience of emotion, the learning, inhibition, or remapping of affect–response relationships, or some other reason.

Beyond computing the significance of external stimuli based on the current context, the OFC/VMFC is important for acting on the basis of these computations. In general, OFC/VMFC lesions in animals impair the ability to change stimulus–reinforcement associations and cause responses to perseverate in old patterns (Bechara, Damasio, Tranel, & Damasio, 1996; Stuss et al., 1994). Thus VMFC lesions in rats increase the time it takes to extinguish conditioned fear responses (Morgan & LeDoux, 1995).

OFC lesions also can lead to feeding, drinking, and sexual behavior that is no longer sensitive to external cues indicating the availability of food, water, or sexual partners (Rolls, 1999).

Data from human studies generally support the findings from animal research: OFC/VMFC lesions impair the ability to change stimulus–reward relationships and cause perseveration of previously learned or prepotent responses (Freedman, Black, Ebert, & Binns, 1998; Rolls, Hornak, Wade, & McGrath, 1994). This inability to use information about the current value of stimuli to control the expression of previously learned behavioral or experiential responses can have serious social and interpersonal consequences.[5] Various kinds of personality and emotional changes have been reported following OFC and VMFC lesions, including apathy, violence, and the exhibition of socially inappropriate behavior and language (Damasio, 1994; Saver & Damasio, 1991; Rolls, 1999). To an observer, some patients may seem to lack affect and can speak without passion about experiences that should evoke emotion (Damasio, 1994). Other patients might seem unpredictable, suddenly violent, exhibiting outbursts of sudden anger or sexual attraction, punctuated by periods of apathy and indifference (Rolls, 1999). Although the experience and expression of emotion clearly seems to be dysregulated in these patients,[6] at least some of these problems may be due to impairments in understanding the emotions conveyed through the facial and vocal expressions of others, which also is impaired by OFC lesions (Hornak, Rolls, & Wade, 1996). This apparent perceptual deficit could reflect an inability to evaluate the meaning of external cues that normally provide regulatory feedback.

In everyday life, the OFC/VMFC may help encode and represent information about the shifts and changes in affective or emotional responses that are part and parcel of complex human social life. When we reason using our feelings, we may feel that we are "guessing" which response might be correct because we can't explicitly verbalize the criteria that guide our decisions. Such guessing has recently been shown to activate the VMFC (Elliott, Rees, & Dolan, 1999). Without the OFC/VMFC, the ability to represent affective/emotional responses that constrain the application of social knowledge is missing, leaving an individual adrift in a sea of knowledge without his emotions to anchor him.

Finally, it is important to note that the affective representations mediating OFC/VMFC function are distinct from those stored in semantic memory in that semantic memory is important for knowing how to behave but OFC/VMFC is important for being able to act accordingly. This has been demonstrated most clearly in studies of stimulus–reward reversal in patients with OFC/VMFC lesions. On these tasks patients often report knowing that the stimulus–reward relationship has changed but are unable to alter their behavior accordingly (Rolls et al., 1994; Saver & Damasio, 1991).

Similarly, Damasio (1994) has described a patient with OFC/VMFC lesions who performed normally on laboratory tasks tapping explicit knowledge of appropriate emotional responses to a variety of simple and complex social cues, but was completely unable to make use of this knowledge to make decisions or guide behavior in his everyday life. In both of these cases, the failure to represent the current affective value of a stimulus or choice is necessary to constrain the application of semantic knowledge. This fact was demonstrated elegantly by means of a simple laboratory-based gambling task. Patients with OFC/VMFC lesions didn't win money because they failed to generate anticipatory changes in skin conductance that signaled an impending choice would likely result in monetary loss (Bechara et al., 1996). In essence, these patients could not generate affective reactions to analyses of their response options, and were unable to judge which actions would be most appropriate to take.

Summary

Taken together, the human and animal data suggest that the OFC and VMFC (a) represent the current, contextually specified, emotional/motivational value of an external stimulus and (b) bring response tendencies (whether learned or innate) and the judgment process under the control of this emotional/motivational information. Together, these functions allow us both to alter our emotional responses based on analyses of the current context and to generate affective responses based on these analyses. These two functions form the foundation for the active regulation of emotion and emotion-guided behavior.

SUMMARY AND FUTURE APPLICATIONS OF THE THEORY

Based on our brief review, there is evidence that the distinction between automatic and controlled emotion processing is useful for understanding the way in which different brain systems contribute to the generation and regulation of emotion. In most cases, as depicted in Figure 2.3, automatic emotion processing starts the ball rolling as the amygdala and basal ganglia analyze internal and external inputs for the presence of threats and rewards. If a stimulus is threatening, the amygdala quickly associates the stimulus's perceptual characteristics (shape, sound, etc.) with appropriate (e.g., avoidance) responses. If a stimulus proves rewarding, the basal ganglia is essential for encoding and storing the sequences of thought and action that made that reward possible. These types of activation are likely ongoing and ubiquitous within an individual, constituting the core affective life of that individual (Russell & Feldman Barrett, 1999). Core affect varies in intensity,

and a person is always in some state of core affect. Core affect becomes directed into an emotional episode when the automatic activation of semantic emotion knowledge attributes this affect to an object. In a bottom–up fashion, complex emotion knowledge stored in semantic memory provides information about the identity of the attributed object, its meaning, its associates, and other possible responses to it.

Controlled emotion processing begins when the anterior cingulate signals the presence of a discrepancy (e.g., between competing approach and avoidance tendencies), a degree of uncertainty, or a violated expectancy. These signals indicate that emotion knowledge may need to be deployed to consciously transform core affect into an emotion or that the trajectory of an ongoing emotional response (initiated via automatic knowledge activation) may be in need of regulation or alteration. The OFC and VMFC are essential for computing the current affective value of an external stimulus, taking into account changing reinforcement contingencies and situational contexts that may dictate that the affective meaning of that stimulus has changed (e.g., what once was positive may now be aversive). These compu-
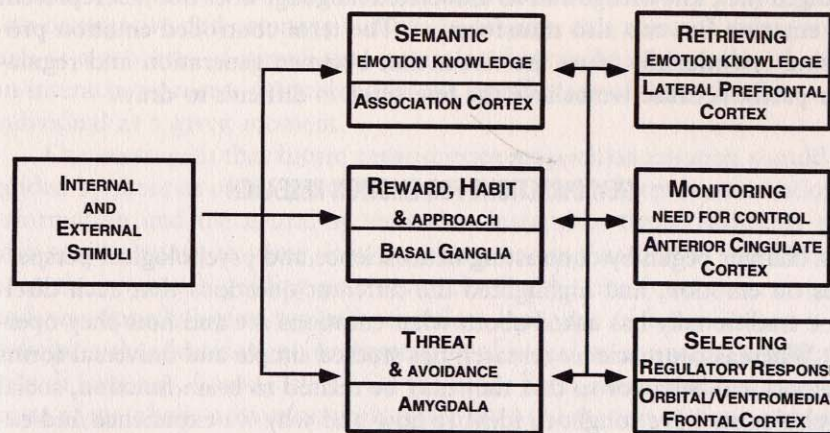
FIGURE 2.3. Flowchart showing the flow of information between different brain structures used for emotion processing. Internal (e.g., body sensations, images, and thoughts) and external (faces, tones of voice, words, actions, etc.) stimuli are processed by three systems that each process a specific type of emotion-related information. Once representations in each system are activated they can influence behavior and experience automatically. The responses generated by these activated representations can influence deliberative, consciously guided behavior through the ACC, OFC, and VMFC. These systems enable an individual to guide choices or direct attention using his or her current feelings about an object or stimulus. See text for details.

tations are essential for guiding decision making, inhibiting prepotent emotional responses, and activating appropriate physiological responses.

Also important for controlled emotion processing is deliberate access to semantic memory and searches of it. Retrieval of emotion knowledge aids in labeling and redirecting emotional responses, making it easier to select emotion regulatory strategies. The effortful application of emotion knowledge may thus serve a regulatory function because the way in which we interpret and draw inferences about the meaning of our current affective responses may change them. Although areas of lateral prefrontal cortex are used to retrieve and manipulate semantic information online, it is not yet clear how those brain regions or others mediate the application of this knowledge to the understanding of affective states. Recent studies have suggested that related regions on the medial surface of the frontal lobes may be important for making such attributions about internal states (Lane, Fink, et al., 1997; Lane, Reiman, Ahern, & Schwartz, 1997).

It is important to note that controlled emotion processing influences both emotion generation and regulation. Any act of regulation necessarily generates a new emotional response. Each time an emotion is inhibited, labeled, or reappraised, the input to the network of emotion processing systems changes and the emotional response that emerges therefore also is changed (i.e., knowledge and its associated language does not just represent the emotion but can also transform it). The term controlled emotion processing intentionally blurs the distinction between generation and regulation partly because we believe the line often is difficult to draw.

NEW DIRECTIONS FOR EMOTION RESEARCH

This chapter began by contrasting neuroscience and psychological perspectives on emotion, and highlighted the different questions that each discipline traditionally has asked about what emotions are and how they operate. Whereas neuroscience research has studied simple and universal forms of emotional behavior so that they may be related to brain function, social psychologists have sought to identify how and why we experience and express more complex blends of emotions in situations more like those we encounter every day. The primary aim of this chapter has been to show how the gap between these two approaches can be bridged by a theory that focuses on the processes that give rise to emotion. The level of information processing is a natural bridging point for low-level analyses of neural systems and high-level analyses of emotional experience and behavior.

We believe that currently available neuroscience data support our account of emotion generation and regulation in terms of the interaction between automatic and controlled processing. However, our review falls short of explaining exactly how the neural systems that instantiate these pro-

cesses interact to produce many of the phenomena that interest psychologists the most. Indeed, our theory posits that automatic and controlled processes give rise to phenomena such as emotion suppression and reappraisal, the differentiation of blends of emotion, and the interaction of emotion and cognition (Forgas, 1995; Schwarz & Clore, 1988). Unfortunately, we provide little hard data that the neural systems we identify as automatic or controlled emotion processors are involved in these behaviors. The reason for this shortcoming is clear: until very recently, investigations of the brain bases for emotion have been conducted exclusively by neuroscientists pursuing a research agenda that does not cater directly to the tastes of psychologists.

A consequence of the neuroscience approach is that the vast majority of research on the neural basis of emotion has employed simple stimuli (e.g., affective pictures and words or pleasant or unpleasant physical stimulation) in carefully controlled situations. In these studies the appraisal of emotional significance is predetermined and controlled by the experimenter and does not model the way in which emotion appraisals are determined and modulated by context and the meaning of a stimulus to an individual. By limiting the range and kinds of motivational and social relevance of the stimuli used to study emotion, this research has provided information about only a limited class of emotion appraisals that involve clearly positive or negative and arousing stimuli. In essence, this research treats emotion as a property of stimuli that is perceived by individuals, rather than as an interactive process determined by the significance of that stimulus to an individual at a given moment.

Our message is that future neuroscience research on emotion should be guided by theories of the processes used to encode and represent emotional information and the neural systems that instantiate them. Although this may seem obvious to some, it is important to emphasize that this has not been the approach guiding past research. On the one hand, neurologists and neuropsychologists typically correlate locations of brain lesions with deficits in the identification or expression of verbal or nonverbal emotional stimuli (see, e.g., Borod, 1992). On the other hand, neuroimagers correlate areas of metabolic activation with either the presentation of similar stimuli or the presence of emotion disorders (see, e.g., George et al., 1995; Rauch et al., 1996). The problem with this approach is that these correlations don't tell us very much about *why* particular systems are active and what this activation means.

Testing and Applying Theory

We propose that the distinction between automatic and controlled processes used here can serve as a foundation for research with a strong theo-

retical orientation. From this perspective, research should investigate not just what kinds of stimuli activate different brain systems but what processes each system carries out, in what situations those processes are used, and how different processes interact. To illustrate the way in which our theory can facilitate this process and be tested at the same time, we now consider the way in which it can illuminate four domains of emotion research.

Emotion Generation

An enduring question about the generation of emotional responses is why different individuals experience different emotions. Our theory suggests that this question can be addressed at multiple levels of analysis. As discussed earlier, one could determine whether individuals who experience complex blends of emotions do so because of automatic processes that effortlessly initiate a nuanced response, controlled processes that deliberatively build a complex response, or some combination of the two. By knowing which types of processing are associated with which neural systems, neuroscience data can be used to address this question. Neuroimaging studies could, for example, indicate whether the amygdala and basal ganglia or the anterior cingulate and prefrontal cortices are activated when individuals report emotional experiences of differing complexity. Such studies could also address related questions, such as determining whether individual differences in the ability to articulate emotions are characterized by dysfunction in one or more brain systems.

A first step in this direction was taken in a recent study that showed the ability to complexly express and understand emotional states verbally is correlated with activation of a rostral region of the ACC (Lane et al., 1998). The ACC-based evaluation system likely interacts with other prefrontal brain areas to control the conscious use of information about our emotional states, but the exact way in which this happens remains to be elucidated. By designing studies to test theoretical predictions about the neural systems used to generate emotion, research offers a twofold benefit: it simultaneously allows us to test theories about the processes involved in emotion and informs us about the behaviors in which particular neural systems participate.

Emotion Regulation

The study of emotion regulation is concerned with how, why, and when we change what we do and think in order to change the way we feel. Current interest centers on the efficacy and consequences of adopting particular regulatory strategies, such as emotion suppression, reappraisal, or the retrieval of mood-congruent or -incongruent memories (for a review, see Gross,

1998). From our perspective, research on the neurofunctional bases of these processes should not be limited to drawing inferences based on studies of related phenomena such as extinction or reversal learning. Rather, studies should directly investigate the neural systems involved in these phenomena, taking into account the theoretical perspective advanced here.

For example, neuroimaging studies or studies of lesion patients could determine whether emotion suppression involves the ACC, OFC, and VMFC, as our theory would predict. Other regulatory strategies, such as reappraising the meaning of an emotionally evocative stimulus, should involve lateral frontal areas used to access information in semantic memory. The study of emotion regulation also brings to the fore the question of how controlled emotion processing influences automatic emotion processing. Suppression and reappraisal might exert their effects at a low level by "turning off" automatic processing systems such as those involving the amygdala and basal ganglia, or they might exert their effects at a high level, without influencing automatic processing systems at all. Future work will undoubtedly address these issues.

Relation between Emotion and Cognition

Many contemporary theorists make a distinction between emotion and cognition. From our perspective, the separation of cognition and emotion may depend upon one's level of analysis. The emotion–cognition distinction seems quite useful for understanding high-level phenomena that involve descriptions of phenomenal experience or behavior that can be clearly labeled as emotional (e.g., feeling and acting afraid) or nonemotional (e.g., giving the meaning of a word). But as we move down levels of analysis, the distinction begins to blur. At the level of information processing mechanisms, it often is not clear whether a given process serves a purely emotional or cognitive function because it may be involved in phenomena that may be labeled emotional or cognitive at a higher level. The evaluative/monitoring function carried out by the ACC is a good example: whether one is retrieving a word meaning or running from a bear, it is important to determine whether the current course of action needs to be changed. The blurring and separation of affect and cognition continues at the neural level of analysis. Some brain structures, such as the ACC, carry out computational processes that may be important for all forms of behavior, whereas others, such as the amygdala, seem to be important only for analyzing affective stimuli.

This discussion highlights the crux of the problem: what criteria do we use to distinguish emotion and cognition? Is the distinction experiential, behavioral, computational, or neural? Is the presence of affective feelings or behavior enough even though activity of putatively emotion processing

brain systems cannot be detected? Alternatively, should the activation of the amygdala be sufficient to indicate emotion even if the individual is not consciously experiencing emotional feelings? These questions may not have clear answers, and many definitions of emotion resolve the issue by describing a set of criteria all or some of which are necessary constituents of an affective state or emotion episode. It is interesting to note that psychologists seldom debate definitions of cognition, which may be similarly difficult to provide. Indeed, definitions of cognition often turn upon the use of mental representations to mediate the link between responses and behavior. By that definition, many instances of emotion are also instances of cognition.

Definitions of Emotion

Our theoretical approach also may speak to controversies about the proper way to distinguish emotions and moods, or between different kinds of emotional states. The two most prominent proposals posit either a discrete set of distinct, universal affects that are innate, language free, and defined by physiology (see, e.g., Ekman, 1993; Izard, 1977) or a constructed set of social categories that are culturally relative (see, e.g., Averill, 1980; Shweder, 1993). The apparent conflict in this distinction, which has roiled emotion research for decades, may come from the fact that most psychologists formulate emotion from one level of analysis while failing to take the others into consideration. It seems likely that a given neural structure may be involved in more than one kind of emotional response, and it is unlikely that there will be simple one-to-one mappings between a single brain system and any one emotion or component of emotion (i.e., there are no centers for happiness, sadness, or anger in the brain). Furthermore, while emotion concepts and their associated semantic knowledge are important, they are probably not sufficient in and of themselves to constitute an emotional response (although it is conceivable that an emotional response could begin with the activation of this knowledge). Core affect associated with the more automatic processes of the amygdala and basal ganglia are probably necessary for the emergence of a true emotional response.

By integrating theory and research across multiple levels of analysis, our theory suggests that neither perspective is precisely correct. In the broadest context, emotional experiences are not just pure cognitive constructions; nor are they biological universals. Rather, they emerge from a constellation of neurophysiological events across different computational systems differing in degrees of automaticity or controlled processing. The phenomena we typically identify as emotions, that is, anger, sadness, fear, guilt, and so on, are mediated by culture in that discrete emotion concepts and associated knowledge are imposed, either automatically or deliberately, on the more basic elements of pleasant or unpleasant core affect.

A Social Cognitive Neuroscience Approach

In closing, we note that the integration of the neural, information-processing, and behavioral–phenomenological levels of analysis in the study of emotion is an example of an emerging *social cognitive neuroscience approach*. This approach emphasizes that information about the structure and function of the brain systems used for emotion is particularly useful for constraining theories about the relationships between the cognitive/process and behavioral/phenomenological levels of analysis (see Lieberman, 2000; Lieberman, Ochsner, Gilbert, & Schacter, in press; Ochsner & Kosslyn, 1999; Ochsner & Lieberman, 2000; Ochsner & Schacter, 2000). Any theory of emotion should make sense of the complexity of experience and expression observed at the social–interpersonal level in terms of the neural information-processing mechanisms that give rise to these behaviors and experiences. This approach has been our implicit guide throughout this chapter.

CONCLUSIONS

The interaction of automatic and deliberative processing can account for a wide range of emotional phenomena. We humans experience emotion and alter our behavior not only in the pursuit of basic needs to eat, have sex, make friends, or avoid pain. We use our feelings en route to making all kinds of decisions never faced by our ancestors or biological cousins, and perhaps more importantly, we have the ability to deliberately reason about the nature and meaning of our feelings as we make each choice. Because our feelings don't come with explanations, the outcome of this controlled process can have profound consequences in the short and long term. Indeed, in many circumstances it behooves us to figure out whether we're angry or guilty, whether we're sad or frightened, and whether we'll continue to feel that way in the future.

It is our contention that neuroscience research on emotion should address these abilities in full. Only by testing hypotheses about the relationships between socially relevant emotion appraisals, their mediating processes, and their neural substrates will future research close the gap between psychological approaches to emotion and neuroscience descriptions of the brain.

ACKNOWLEDGMENTS

NOTES

1. For comprehensive reviews of the neuroscience literature on emotion, see Gray and McNaughton (1995), LeDoux (1996), Panksepp (1998), and Rolls (1999).

2. More generally, studies have shown that a variety of stimuli with acquired threat value activate the amygdala, including presentation of disgust or fear faces (Morris et al., 1996, 1998), vocal expressions of fear and disgust (Phillips et al., 1998), aversive odors (Zald & Pardo, 1997), negatively valenced photos (Irwin et al., 1996; Lane, Fink, et al., 1997; Taylor et al., 1998), and stimuli that evoke symptoms in psychiatric populations (see, e.g., Birbaumer et al., 1998; Rauch et al., 1995; Shin et al., 1997). A few studies have failed to show amygdala activation during perception of negative stimuli, although the reasons why may differ in each case and are not entirely clear (see, e.g., Canli et al., 1998; see also Adolphs, 1999, for a discussion). Neuropsychological studies also support the importance of the amygdala in the perception of threat-related facial expressions by demonstrating that amygdala lesions will disrupt recognition of expressions of disgust, surprise, and especially fear but will not disrupt perception of facial identity (Adolphs et al., 1994; Calder, Young, Rowland, & Perrett, 1996; Young, Aggleton, Hellawell, & Johnson, 1995).

3. For example, damage to basal ganglia can disrupt the normal sequence of grooming in a rat (Berridge & Whishaw, 1992) or the ability to know what comes next when we are making dinner or doing our job (Caine, Hunt, Weingartner, & Ebert, 1978).

4. The evaluative monitoring function of ACC is required any time controlled or executive processing is needed for task performance. This means that ACC activation should be found anytime a task requires (a) withholding a prepotent response or mediating competition between multiple possible responses, (b) selecting novel or difficult cognitive or behavioral responses, (c) planning and decision making, or (d) correcting erroneous responses (Botvinick, Braver, Carter, Barch, & Cohen, 1999; Posner & DiGirolamo, 1998; Shallice & Burgess, 1993). ACC activation has been observed in all of the following situations (Posner & DiGirolamo, 1998): when one's attention must be divided among many stimuli (Corbetta, Miezin, Dobmeyer, Shulman, & Petersen, 1991); when one is deciding if named animals are dangerous (Petersen, Fox, Posner, Mintun, & Raichle, 1988); when one is generating multipart mental images (Kosslyn et al., 1993); when one is generating words from single letter cues (Phelps, Hyder, Blamire, & Shulman, 1997) or word stems (Buckner et al., 1995); when one is generating word meanings (Petersen, Fox, Posner, Mintun, & Raichle, 1990; Tulving et al., 1994); when one is generating hypotheses (Elliott & Dolan, 1998); when one is spontaneously generating random finger or hand movements (Dieber et al., 1991); while one is holding information in mind during working memory tasks (Petit, Courtney, Ungerleider, & Haxby, 1998); during recognition tasks (Taylor et al., 1998); when one is perceiving stimuli that have been degraded perceptually (Barch et al., 1997); and when a prepared response must be withheld, as in the Stroop task (Bench, Frith, Grasby, & Friston, 1993).

5. The sensitivity of OFC to changes in reinforcement contingencies may be quite general: a recent fMRI study showed OFC activity when an expected visual stim-

ulus failed to appear in a cued location (Nobre, Coull, Frith, & Mesulam, 1999). This result suggests that OFC neurons are sensitive not just to the relationship of stimuli to external rewards but to their relationship with internally generated expectations. OFC activation following violations of expectation could be due to the elicitation of a negative affective response to the violation; it could be due to inhibition of a prepared response to the expected target (Krams, Rushworth, Deiber, Frackowiak, & Passingham, 1998), or it could be due to interference tasks that require inhibition of habitual responses (e.g., the Stroop task; Beauregard et al., 1997). Withholding inappropriate responses could also induce activation of lateral parts of the OFC during retrieval of memories of words (Paradiso et al., 1997).

6. OFC/VMFC also has been associated with emotion dysregulation in psychiatric patients. For example, heightened OFC/VMFC activity has been found during symptom provocation in patients with anxiety disorders, including posttraumatic stress disorder (PTSD), obsessive–compulsive disorder (OCD), and simple phobia (Breiter et al., 1996; Rauch, Savage, Alpert, Fischman, & Jenike, 1997; Rauch, Savage, Alpert, Miguel et al., 1995; Rauch, van der Kolk, Fisler, & Alpert, 1996; Shin et al., 1997). Recently, a posterior area of the VMFC immediately inferior to the genu of the corpus callosum has been associated with unipolar and bipolar depression, showing heightened activity during episodes of mania and decreases during bouts of depression (Drevets, Price, Simpson, & Todd, 1997). The abnormal activity normalizes when symptoms remit, and similar findings have been obtained in OCD patients whose elevated OFC activity normalizes following successful drug or behavior therapy (see, e.g., Saxena et al., 1999). Although consistent with the involvement of OFC and VMFC in emotion, the data from just about every one of these studies is ambiguous. It is not clear whether the symptom-related neural activity indicates an abnormal neural system whose dysfunction produces the disorder, on the one hand, or the use of a normally functioning system to regulate abnormal behavior and experience generated by some other system, on the other.

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