

EMOTION AND COGNITION: Insights from Studies of the Human Amygdala

Elizabeth A. Phelps

*Department of Psychology, New York University, New York, New York 10003;
email: liz.phelps@nyu.edu*

Key Words emotion regulation, learning, memory, attention, hippocampus

■ **Abstract** Traditional approaches to the study of cognition emphasize an information-processing view that has generally excluded emotion. In contrast, the recent emergence of cognitive neuroscience as an inspiration for understanding human cognition has highlighted its interaction with emotion. This review explores insights into the relations between emotion and cognition that have resulted from studies of the human amygdala. Five topics are explored: emotional learning, emotion and memory, emotion's influence on attention and perception, processing emotion in social stimuli, and changing emotional responses. Investigations into the neural systems underlying human behavior demonstrate that the mechanisms of emotion and cognition are intertwined from early perception to reasoning. These findings suggest that the classic division between the study of emotion and cognition may be unrealistic and that an understanding of human cognition requires the consideration of emotion.

CONTENTS

INTRODUCTION	27
EMOTIONAL LEARNING	29
EMOTION AND MEMORY	33
EMOTION'S INFLUENCE ON ATTENTION AND PERCEPTION	37
PROCESSING EMOTION IN SOCIAL STIMULI	40
CHANGING EMOTIONAL RESPONSES	43
CONCLUSION	46

INTRODUCTION

The emergence of the study of cognition as a discipline within psychology began with the cognitive revolution, which is often characterized as a reaction to the dominance of behaviorism in the middle of the past century (Miller 2003). The cognitive revolution emphasized a view of human cognition as information processing. As a result, a primary goal of cognitive psychology was to explore "the way man collects, stores, modifies, and interprets environmental information or

information already stored internally” (Lachman et al. 1979, p. 7). This approach, inspired in part by the computer metaphor, generally excluded emotion. Instead, emotion was considered a topic of investigation more appropriate for other disciplines of psychology, such as social, personality, or clinical psychology. Although there has been significant debate over the past 30 years about the appropriate role for emotion in the study of cognition (Lazarus 1984, Neisser 1976, Zajonc 1984), until recently these different approaches to the study of human behavior rarely overlapped.

The cognitive revolution provided an important model to aid in the exploration of the nature of mental representation. However, the computer metaphor is no longer the primary inspiration in studies of human cognition. There has been a new revolution inspired by advances in neuroscience and techniques for studying the human brain. Understanding how cognition is linked to neural function is increasingly driving the questions and means of investigation in cognitive psychology. The cognitive neuroscience approach has relied on animal models of neural function as a starting point for studying the representation of cognition in the human brain. Some of these animal models highlight the importance of emotion in understanding cognitive functions (e.g., see LeDoux 1996). These animal models have created a renewed interest in exploring the interaction of emotion and cognition in humans. This emphasis on linking mental processes to neural function has aided in breaking down barriers between traditional psychological disciplines in efforts to understand human behavior. As our understanding of the neural basis of human cognition grows, it has become increasingly apparent that the neural circuitry of emotion and cognition interact from early perception to decision making and reasoning.

In this review, I explore how the cognitive neuroscience approach has informed our understanding of the interaction of emotion and cognition. Animal models of the neural circuitry of emotion have emphasized specific brain structures that appear to be primarily linked to emotional processes, yet interact extensively with other brain systems underlying cognitive function. One of these structures is the amygdala, an almond-shaped structure on the medial temporal lobe that sits adjacent and anterior to the hippocampus (Figure 1). The notion that the amygdala might play a role in emotion first emerged when Kluver & Bucy (1937) demonstrated that medial temporal lobe lesions in monkeys resulted in a range of odd behaviors, including approaching normally feared objects, orally exploring objects, and exhibiting unusual sexual behaviors. Approximately 20 years later, Weiskrantz (1956) demonstrated that it was the amygdala within the medial temporal lobe whose damage resulted in the range of behaviors that came to be known as Kluver-Bucy syndrome. Since that time, the amygdala has been a primary focus of researchers interested in the neural systems of emotion. The amygdala is a structure with extensive connections to brain areas thought to underlie cognitive functions, such as sensory cortices, the hippocampal complex, and the prefrontal cortex (Young et al. 1994). Because of its broad connectivity, the amygdala is ideally situated to influence cognitive functions in reaction to emotional stimuli.

Consistent with this, recent research has suggested that a primary function of the human amygdala is the modulation of neural systems underlying cognitive and social behaviors in response to emotional cues (Anderson & Phelps 2000, Whalen 1998).

This review examines insights into the interaction of emotion and cognition that have emerged from studies of the human amygdala. The amygdala is not the only brain structure that has been identified as important for emotional processes in humans¹, but it is the most thoroughly investigated to date. The cognitive neuroscience research on the human amygdala has drawn from neuroscience studies in nonhuman animals and behavioral paradigms derived from cognitive, social, personality, and clinical psychology. This review highlights five areas of research that demonstrate a role for the human amygdala in the interaction of emotion and cognition: emotional learning, emotion and memory, emotion's influence on attention and perception, processing emotion in social stimuli, and changing emotional responses.

EMOTIONAL LEARNING

Understanding how a stimulus acquires emotional properties is the primary goal in studies of emotional learning. Once a stimulus comes to elicit an emotional reaction, how it is processed may differ from neutral stimuli. Animal models of amygdala function have emphasized its role in emotional learning. This research has primarily examined classical fear conditioning. In a typical study, a neutral stimulus, such as a tone, is paired with an aversive event, such as a footshock. After a few pairings, the animal learns that the tone, the conditioned stimulus (CS), predicts the aversive event, the unconditioned stimulus (US), and the presentation of the tone alone begins to elicit a range of emotional reactions, such as freezing, changes in heart rate and blood pressure, increased startle responses, and stress hormone release. These acquired emotional reactions are conditioned fear responses. Using fear conditioning as a model paradigm, researchers studying nonhuman animals have mapped the pathways for fear learning from stimulus input to response output. These studies have demonstrated that the amygdala is critical for the acquisition, storage, and expression of a conditioned fear response (Davis 1992, Kapp et al. 1992, LeDoux 1996, Maren 2001, but see also Cahill et al. 1999).

¹The current review is limited in that only the role of the amygdala is examined and the representation of emotion in the human brain involves a network of structures, including, but not limited to, the orbitofrontal cortex and the striatum. Limits in neuroscience techniques have generally encouraged the investigations of separate neural structures, but it is obvious that complex circuits of neural mechanisms are important for all aspects of human emotion and cognition. Recent Annual Review chapters that may provide additional information concerning topics discussed in this review include Fanselow & Poulos (2005), McGaugh (2004), Rolls (2000), Schultz (2006), Shors (2006), and Stuss & Levine (2002).

In humans, studies of fear conditioning are consistent with these animal models. Research using functional magnetic resonance imaging (fMRI) has reported increased blood oxygenation level-dependent (BOLD) signal in the amygdala in response to a neutral stimulus paired with an aversive event, called the CS+, compared to another neutral stimulus that did not predict an aversive event, called the CS- (Buchel et al. 1998, LaBar et al. 1998). The conditioned response, measured as a change in skin conductance (an indication of arousal) to the CS+, was correlated with the magnitude of this amygdala activation (LaBar et al. 1998). These results suggest the amygdala is involved in fear conditioning in humans. However, studies of brain imaging only show a correlation between brain activation and stimuli, processes, or behaviors (Cabeza & Kingstone 2001) and do not indicate a critical role for the amygdala. Consistent with animal models, patients with lesions including the right, left, or bilateral amygdala do not demonstrate a conditioned response as measured by skin conductance, even though the response to the US is intact (Bechara et al. 1995, LaBar et al. 1995). These results indicate that the human amygdala plays a critical role in fear conditioning.

The initial findings on fear conditioning in humans were predicted by animal models. However, these studies also demonstrated that the human amygdala's role in fear conditioning is limited to the physiological expression of conditioned fear. Studies in patients with amygdala damage demonstrate that even though they fail to show conditioned fear responses as measured by physiological responses to the CS+, they are able to acquire explicit knowledge about the contingencies between the CS+ and the aversive US. For example, Patient SP, who suffers from bilateral amygdala damage, and normal control subjects were shown a blue square (the CS+) that was paired with a mild shock to the wrist (the US). After a few pairings, normal control subjects showed an increased skin conductance response to the blue square, indicating a conditioned fear response. SP failed to acquire a conditioned response. She was then shown her skin conductance responses indicating a lack of conditioned fear and asked to comment:

"I knew that there was an anticipation that the blue square, at some particular point in time, would bring on one of the volt shocks. But even though I knew that, and I knew that from the very beginning, except for the very first one where I was surprised, that was my response—I knew it was going to happen. I expected that it was going to happen. So I learned from the very beginning that it was going to happen: blue and shock. And it happened. I turned out to be right, it happened!" (Gazzaniga et al. 2002, p. 559).

As her statement clearly indicates, SP had an explicit understanding of the parameters of the fear-conditioning procedure even though she did not show any physiological indication of conditioned fear. This type of explicit knowledge depends on the hippocampal complex for acquisition (Squire & Zola-Morgan 1991), and patients with damage to the hippocampus, whose amygdala are intact, show the opposite pattern; that is, a normal conditioned response as indicated by a physiological measures, but no explicit knowledge of the relation between the CS+ and the US (Bechara et al. 1995). These results highlight the notion that there are

multiple forms of representation and expression for emotional learning and these different forms may rely on different neural substrates.

The normal acquisition of explicit knowledge of the parameters of fear conditioning following amygdala damage raises some questions as to how extensive the human amygdala's role in emotional learning is. Although learning that a stimulus predicts an aversive event through direct personal experience of this event—as in fear conditioning—is a powerful means of emotional learning, it is rather inefficient in that it requires an aversive experience. Humans have developed more efficient, symbolic means of communication, namely language, that allow for the acquisition of the emotional properties of a stimulus without aversive consequences. For instance, one could learn to fear and avoid a neighborhood dog by being bitten, an example of learning through direct aversive experience. However, one could also learn to fear and avoid a neighborhood dog by listening to a neighbor discuss how the dog is mean and dangerous. This type of instructed learning will result in a fear response when the dog is encountered, even though there is only explicit, symbolic knowledge of the dog's emotional properties.

A paradigm called instructed fear was used to determine if the human amygdala plays a role in the expression of symbolically acquired fear responses. In this paradigm, subjects are told that they might receive a mild shock to the wrist paired with one stimulus, such as a blue square (the threat stimulus), while the presentation of another stimulus, such as a yellow square (the safe stimulus), indicates that no shock will be presented. It has been demonstrated that instructed fear results in robust physiological fear responses to the threat stimulus that are similar to responses to a CS+ in fear conditioning (Hugdahl & Ohman 1977). With fMRI, it was found that presentations of the threat stimulus, relative to the safe stimulus, resulted in activation of the left amygdala that was correlated with the magnitude of the skin conductance response to threat versus safe, even though none of the subjects actually received a shock to the wrist (Phelps et al. 2001). A study in patients with right, left, and bilateral amygdala damage found that only those patients whose damage included the left amygdala showed an impairment in the physiological expression of instructed fear (Funayama et al. 2001). These results indicate that the left amygdala is critical for the expression of symbolically acquired fears—fears that are imagined and anticipated, but are never actually experienced.

The results with instructed fear suggest that the neural substrates underlying its expression are similar, but not identical, to those underlying fear conditioning. Although studies have demonstrated instructed fear and conditioned fear are similar in their physiological expression (Hugdahl & Ohman 1977), the slight differences in their neural circuitry suggest there may be subtle differences. In an effort to explore this possibility, the expression of acquired fear responses to subliminally presented stimuli was examined. Previous research has found that when representations of certain natural categories of stimuli, such as pictures of spiders, snakes, and angry faces (called biologically prepared stimuli), are used as a CS+ during fear conditioning, the conditioned response will be expressed through

physiological measures, even if the CS+ is presented subliminally and the subject is unaware of its presentation (see Ohman & Mineka 2001 for a review). In these fear-conditioning paradigms, the CS+ is presented briefly (less than 30 msec) and is immediately followed by a masking stimulus preventing awareness of its presentation as assessed by explicit report (Esteves et al. 1994).

Olsson & Phelps (2004) exposed subjects to classical fear conditioning and instructed fear. In addition, they added a third emotional learning paradigm, observational fear. Learning through social observation is another indirect means for acquiring fear responses. Both humans and nonhuman primates have been shown to learn emotional properties of stimuli vicariously, through observing the emotional reactions of a conspecific (Ohman & Mineka 2001). The procedures for fear conditioning and instructed fear were similar to that described above. The subjects in the observational fear paradigm watched a video of a confederate receiving a mild shock to the wrist paired with some presentations of one stimulus (the observational CS+) and not another (the observational CS-). In all three of the learning groups, the CS+ and CS- were presented supraliminally (with awareness) on some trials, and subliminally (without awareness) on others. Consistent with previous results (Esteves et al. 1994), learning through fear conditioning resulted in expression of conditioned fear with supraliminal and subliminal presentation. In contrast, instructed learning required awareness for expression. This symbolically represented fear was not expressed when the threat stimulus was presented subliminally. Surprisingly, the results for observational fear mirrored those of fear conditioning. With vicarious learning through observation, subjects demonstrated a physiological arousal response to an observational CS+ presented either supraliminally or subliminally.

Instructed and observational fear learning are social means of learning that, unlike fear conditioning, do not require direct aversive experience. However, the similarity in the expression of fears acquired through fear conditioning and social observation suggests a greater degree of overlap in the amygdala's involvement. Research with nonhuman animals has shown that the amygdala is important for the acquisition, storage, and expression of fear conditioning (LeDoux 1996, but see also Cahill et al. 1999). Instructed fear, which depends on language and is unique to humans, most likely relies on the hippocampal complex for acquisition of the episodic knowledge of the link between the neutral and aversive event. It is unlikely the amygdala plays a role in the acquisition of this symbolic, abstract knowledge. In contrast, a recent fMRI study found that observational fear learning results in activation of the bilateral amygdala both during the observation of a confederate undergoing fear conditioning as well as the later test of this fear learning when subjects believed they might receive a shock themselves (Olsson et al. 2004). The magnitude of amygdala activation was similar in both stages, even though subjects knew that there was no possibility they would receive a shock during the observation/learning stage. These results suggest that, like fear conditioning, the bilateral amygdala is important for the acquisition and expression of fears acquired through the vicarious experience of social observation.

Regardless of the means of emotional learning, the amygdala plays an important role in the physiological expression of fear learning and, in some cases, its acquisition as well. More recently, it has been suggested that the amygdala's involvement in the expression of fear learning through social means extends to cultural learning of social group stereotypes based on race and the indirect expression of race bias (Cunningham et al. 2004, Hart et al. 2000, Phelps et al. 2000). Although the current review has focused on fear learning, there is evidence from research with nonhuman animals that the amygdala may also play a role in appetitive conditioning and reward learning (Baxter & Murray 2002, Everitt et al. 2003, Holland & Gallagher 2004). There is some evidence in humans that the amygdala may be involved in learning to associate stimuli with positive outcomes (Johnsrude et al. 2000), but future research will need to explore the extent of the human amygdala's role in appetitive emotional learning.

EMOTION AND MEMORY

As William James wrote in *The Principles of Psychology*, "An impression may be so exciting emotionally as almost to leave a scar upon the cerebral tissues" (James 1890, p. 670). This phrase highlights the commonly held belief that emotion enhances episodic memory. Research on the cognitive neuroscience of emotion and memory has specified a range of means by which emotion can change the formation and recollection of episodic memory. It has been suggested that emotion, through the amygdala's influence, can alter three components of episodic memory: encoding, consolidation, and the subjective sense of remembering. Although episodic memory critically depends on other brain regions, most notably the hippocampal complex (Eichenbaum 2002, Squire & Zola-Morgan 1991), the amygdala may be important for modulating the neural circuitry of episodic memory.

The initial stage of episodic memory formation is encoding. Emotion can influence the encoding of to-be-remembered stimuli through its modulation of attention and perception (Easterbrook 1959). As will be discussed in the next section, emotion, via the amygdala, can influence attention and perception. Previous studies have shown that manipulations of attention will significantly impact memory encoding (e.g., Craik et al. 1996), and these changes in initial stimulus processing with emotion should lead to differences in memory performance. A recent study found that patients with amygdala damage only show an impairment in memory for details of emotional scenes that are central to the event, with intact memory for details that are more peripheral (Adolphs et al. 2005b). This study suggests that the amygdala may be involved in the narrowing of attention around the central emotional details leading to enhanced memory for these details in normal subjects (see also Easterbrook 1959). However, most studies to date have failed to differentiate the amygdala's influence on encoding and consolidation processes. Although a number of brain imaging studies have demonstrated that amygdala activation during encoding can predict later recognition or recall for emotional stimuli (Cahill

et al. 1996, Canli et al. 2000, Hamann et al. 1999), these studies often attribute this effect to emotion's influence on the modulation of memory storage (e.g., Cahill et al. 1996) rather than attention.

The primary neural mechanism that has been explored in an effort to understand emotion's influence on episodic memory is the amygdala's modulation of hippocampal consolidation. Consolidation is a storage process by which memories become more stable over time, and evidence across species suggests that the consolidation of episodic memory critically depends on the hippocampus (Knowlton & Fanselow 1998, Squire & Zola-Morgan 1991). Emotion, specifically arousal, is proposed to enhance hippocampal-dependent consolidation. Using animal models, research by James McGaugh and colleagues has identified the neural systems underlying the effect of arousal on episodic memory consolidation (see McGaugh 2000, 2004 for a review). These studies have shown that physiological arousal results in activation of the beta-adrenergic receptors in the amygdala. The amygdala, in turn, modulates hippocampal processing, resulting in enhanced consolidation or storage for events that elicit an arousal response. Damage to the amygdala does not impair episodic memory for a stimulus, but rather it eliminates any enhancement observed with physiological arousal. Evidence that this effect is due to the modulation of consolidation, as opposed to encoding, comes from studies demonstrating that manipulations of amygdala function *after* stimulus encoding will alter arousal's influence on episodic memory (Packard & Teather 1998). It is suggested that one adaptive function of having a slow consolidation process is to allow for the emotional reaction to a stimulus, which follows its presentation, to influence the memory strength (McGaugh 2004). In this way, events that result in an emotional response, and are more likely to be important for future survival, are less likely to be forgotten.

In humans, a number of studies have reported that arousal enhances the retention of episodic memories (Berlyne 1969, Heuer & Reisberg 1992, Kleinsmith & Kaplan 1963), consistent with the modulation of hippocampal consolidation. Evidence that the human amygdala plays a role in this enhanced memory with arousal comes from studies using a range of cognitive neuroscience techniques. As mentioned above, brain-imaging studies have reported that activation of the amygdala at encoding can predict later retention for emotional stimuli. The amygdala has direct projections to the anterior portion of the hippocampus (Stefanacci et al. 1996). A recent study found that the activation of the amygdala and anterior hippocampus is correlated during the encoding of emotional scenes that were later remembered (Dolcos et al. 2004). In addition, patients with amygdala damage fail to show the normal enhancement of episodic memory with arousal (Cahill et al. 1995). Consistent with a role for the amygdala in modulating storage or consolidation, amygdala damage results in similar forgetting curves for arousing and neutral stimuli, in contrast to normal control subjects who show enhanced retention for arousing stimuli (LaBar & Phelps 1998). Finally, the administration of drugs that block beta-adrenergic receptors also block the impact of arousal on episodic memory (Cahill et al. 1994), consistent with animal models.

Although the effect of arousal on episodic memory is clearly documented, the magnitude of this effect varies depending on the paradigm. It can be quite subtle (e.g., LaBar & Phelps 1998) and is not always observed (e.g., Ochsner 2000). Furthermore, more extreme stress can have an opposite effect; that is, an impairment of hippocampal function and episodic memory (see McEwen & Sapolsky 1995 for a review). Nevertheless, there is robust evidence that the human amygdala, through its modulation of hippocampal consolidation, plays a critical role in situations where physiological arousal leads to enhanced episodic memory.

Until recently, most of the research examining the neural systems underlying the influence of emotion on episodic memory has focused on memory accuracy. However, studies of episodic memory for real life, public, emotional events have suggested that emotion may also influence the subjective experience of memory retrieval, irrespective of memory accuracy. These studies examining “flashbulb” memories have found that for highly emotional, public events the confidence that a memory is accurate and the sense that it is detailed and vivid may not reflect actual accuracy. In one of the first examples of this effect, Neisser & Harsh (1992) examined memory for the Challenger explosion. Within a few days of this tragedy, they asked subjects to record their memory for the circumstances in which they become aware of this event. Two and a half years later, the subjects were asked to report the same memory. Even though the subjects gave detailed recollections and were highly confident in their accuracy, most of these memories were inaccurate. More recently, a study examining memory for the terrorist attack of September 11, 2001 (Talarico & Rubin 2003) found that accuracy for memories of learning about this event did not differ from other, nonemotional events that occurred around the same time. However, memories of the terrorist attacks in comparison with more mundane events were rated as more confident, vivid, detailed, and recollected. Although a study of memory for the O.J. Simpson verdict found that reported emotional arousal at the time of the event is one of the few factors that can predict memory accuracy three years later (Schmolck et al. 2000), all of these studies of “flashbulb” memories suggest that emotion also has an independent effect enhancing the subjective sense of remembering.

One of the difficulties in exploring the neural systems underlying emotion’s impact on the subjective sense of remembering is that most of these studies have examined memories for emotional, public events, which are challenging to investigate using cognitive neuroscience techniques. A recent laboratory study using the remember/know paradigm suggests a similar pattern. Recognition memory judgments are thought to rely on two independent processes: recollection, which includes the retrieval of contextual details, and familiarity, which is a sense that a stimulus is familiar in the absence of contextual details (see Yonelinas 2002 for a review). A subjective measure of these two processes is the remember/know procedure. During recognition judgments, subjects view old and new stimuli and are asked to judge if each stimulus is “new” (not presented before), “known” (familiar, but there is no specific recollection of details for the encoding context), or

“remembered” (recollected with details of the encoding context). Using this procedure, Ochsner (2000) examined recognition for emotional and neutral scenes and found that emotion specifically enhances the proportion of “remember” judgments, even though there was no difference in memory accuracy for emotional and neutral scenes.

A similar paradigm was used to examine the neural mechanisms underlying emotion’s impact on this subjective judgment of recollection (Sharot et al. 2004). As in the Ochsner (2000) study, emotion significantly enhanced the likelihood of “remember” judgments, even though there was no effect of emotion on accuracy. An examination of fMRI responses found that activation patterns in different medial temporal lobe regions were related to the subjective judgment of recollection for emotional and neutral stimuli. Consistent with previous studies (Henson et al. 1999, Wheeler & Buckner 2004), BOLD responses in the posterior parahippocampus differentiated “remember” and “know” judgments for neutral scenes. In contrast, BOLD signals on the amygdala differentiated “remember” and “know” judgments for emotional scenes. The posterior parahippocampus has previously been linked to memory for details of visual scenes (Kohler et al. 2002), which is the type of information that might be expected to result in a judgment of recollection versus familiarity. However, responses in this region were not similarly enhanced for “remembered” emotional stimuli. These results suggest that the neural mechanisms underlying the subjective judgment of recollection differ for emotional and neutral stimuli. The amygdala was specifically linked to judgments of recollection for emotional scenes. For emotional stimuli, judgments of the subjective sense of remembering may be influenced by the emotional qualities of the stimulus with less emphasis on mnemonic details. Consistent with studies of “flashbulb” memories suggesting emotion enhances the feeling of retrieval accuracy irrespective of actual accuracy, these brain-imaging results indicate emotion may alter the neural mechanisms underlying subjective judgments of remembering.

Although there is significant evidence that emotion interacts with episodic memory, and that the amygdala plays an important role in this interaction, an understanding of the complexity of this relationship is just beginning to emerge. Most studies in humans examining the neural systems underlying emotion’s influence on episodic memory have emphasized arousal’s impact on memory consolidation, perhaps inspired by the elegant animal models outlining this mechanism (McGaugh 2000). However, only a few studies have clearly documented arousal’s specific influence on memory consolidation in humans, independent of its effect on encoding (Cahill & Alkire 2003, Cahill et al. 2003, Sharot & Phelps 2004).

Aside from the factors mentioned above, emotion might interact with episodic memory by other means. Emotional stimuli may differ from neutral stimuli in semantic similarity and distinctiveness, factors that have been shown to influence episodic memory (see, e.g., Phelps et al. 1998). Mood at retrieval has also been shown to influence episodic memory (Bower 1981). It is unlikely the amygdala has any role in these other effects of emotion on memory (Kensinger & Corkin 2004, Phelps et al. 1998). An exploration of the underlying neural mechanisms

will aid in determining the complex components of the “scar upon the cerebral tissues” that characterizes emotion’s impact on human episodic memory.

EMOTION’S INFLUENCE ON ATTENTION AND PERCEPTION

Attention and perception are the first stages of stimulus processing, and factors that influence these early processes will also influence downstream cognitive functions, such as memory and reasoning. The importance of emotional salience in attention is well documented (e.g., Niendenthal & Kitayama 1994). An early example of this is the classic “cocktail party effect” described by Cherry (1953), in which an emotionally significant item, such as the subject’s name, was noticed even when it was presented among a stream of unattended stimuli during an attentionally demanding dichotic listening task. This finding and others (e.g., Hansen & Hansen 1998, Ohman et al. 2001) suggests that emotion can facilitate awareness for emotionally salient stimuli in situations where attentional resources are limited.

Recent evidence indicates that the amygdala may mediate the facilitation of attention with emotion (Anderson & Phelps 2001, Morris et al. 1998a). This was investigated using a paradigm that tests the temporal limitations of attention, called the attentional blink (Raymond et al. 1992). In this paradigm, stimuli are presented in rapid succession (e.g., every 100 msec), so quickly it is difficult for subjects to identify any individual stimulus. However, if subjects are told that they can ignore most of the stimuli presented and selectively attend to a few target exemplars, such as those printed in a different color ink, subjects are able to selectively process the target stimuli and later identify them. This ability to selectively attend to specific stimuli in a rapidly presented visual stream is limited by the temporal relation between the different target stimuli. If a second target stimulus is presented a few items after the first target, in what is called the early lag period, subjects will often miss it. It is as if noticing and encoding the first target stimulus results in a temporary refractory period during which time it is difficult to notice and encode a second target. In other words, it is as if attention “blinked” (Chun & Potter 1995).

Using emotional and neutral words as stimuli, Anderson (2005) found that when the second target word is arousing, the attentional blink effect is attenuated. The ability to detect the arousing words was enhanced relative to neutral words when they were presented as the second target in the early lag period. Unlike normal control subjects, patients with left amygdala damage failed to show the normal attenuation of the attentional blink effect with emotion (Anderson & Phelps 2001). These findings indicate that in situations with limited attentional resources, emotional stimuli are more likely to reach awareness, and the amygdala plays a critical role in this facilitation of attention with emotion.

Two mechanisms have been proposed for the amygdala’s facilitation of attentional processing. The first was suggested by Weinberger (1995). In studies with rats, Weinberger demonstrated that sensory cortices (specifically auditory cortex)

may be tuned through fear conditioning to be especially sensitive to stimuli used as a CS+. This sensory tuning with emotional learning depends on the amygdala. This mechanism, which has not been clearly demonstrated in humans, suggests a long-lasting change in perceptual processing for stimuli that have acquired emotional properties through learning.

The second mechanism is a more transient change in attentional thresholds in the presence of emotional stimuli. As seen in Figure 2, anatomical studies have demonstrated that there are reciprocal connections between the amygdala and sensory cortical processing regions, such as the visual cortex (Amaral et al. 2003). The amygdala has been shown to receive input about the emotional significance of a stimulus quickly (Romanski & LeDoux 1992) and prior to awareness (Morris et al. 1998b, Whalen et al. 1998). For instance, robust amygdala activation has been observed in response to faces with fearful versus neutral expressions that are presented subliminally and supraliminally (Whalen et al. 1998). A number of studies indicate that attention and awareness have little impact on the amygdala's response to fearful stimuli (Anderson et al. 2003, Vuilleumier et al. 2001, but see also Pessoa et al. 2002). These results are consistent with previous psychological research indicating that the emotional qualities of stimuli are processed automatically (e.g., Zajonc 1984). It is suggested that this early, automatic amygdala response to fear or threat stimuli is an important factor in its ability to modulate attention and responses to potential danger (Davis & Whalen 2001).

The amygdala's transient facilitation of attention is thought to result from its modulation of sensory cortical regions in the presence of emotional stimuli (Morris et al. 1998a). It is proposed that early in stimulus processing the amygdala receives input about the emotional significance of a stimulus, and through projections to sensory cortical regions, modulates further attentional and perceptual processes (Anderson & Phelps 2001, Vuilleumier et al. 2004). In support of this model, brain imaging studies have demonstrated that visual cortical regions show enhanced activation in response to novel emotional stimuli (Kosslyn et al. 1996). The magnitude of this enhanced visual cortex activation is correlated with amygdala activation in response to these same stimuli (Morris et al. 1998a). Further support that the amygdala is mediating enhanced responses in visual cortical regions for emotional stimuli comes from an fMRI study conducted in patients with medial temporal lobe damage. Vuilleumier and colleagues (2004) presented faces with fear and neutral expressions to three groups of subjects: normal control subjects, patients with damage limited to the hippocampus, and patients with damage to the hippocampus and amygdala. Consistent with earlier studies (Morris et al. 1998a), enhanced activation was observed in visual cortical regions for fear versus neutral faces in normal control subjects. Patients with damage confined to the hippocampus also showed this pattern. However, patients with damage to the amygdala did not show any significant activation for fear versus neutral faces in the visual cortex. These results indicate that the amygdala plays a critical role in mediating the transient changes in visual cortical processing that occur for emotional stimuli.

The anatomical and brain imaging studies supporting a role for the amygdala in the transient modulation of visual cortex have identified regions thought to be important for perceptual functions, including early visual areas such as V1 (Amaral et al. 2003, Vuilleumier et al. 2004). However, the allocation of attention is more often linked to other brain regions, such as the parietal cortex (Corbetta & Shulman 2002). Given this, it is possible that the observed effects of emotion on attention are linked to its effect on perceptual regions. It has been suggested that at least some of the classic effects of attention are the result of the impact of attention on perception (Carrasco 2004), and brain imaging studies have shown that attention leads to enhanced activation in early visual processing regions (Gandhi et al. 1999). The anatomical connectivity between the amygdala and visual cortex suggests that stimuli that lead to amygdala activation, such as fear faces, should enhance perception as well as attention.

In order to examine whether emotion influences perceptual processes that are known to be coded by early visual regions, a psychophysical paradigm was used to examine the effect of fearful face cues on contrast sensitivity (Phelps et al. 2005). The ability to detect contrast is an early perceptual function that has been linked to primary visual cortex. A task that was previously used to demonstrate that covert attention enhances contrast sensitivity (Carrasco et al. 2000) was modified to include cues that were faces with fearful or neutral expressions. It was found that when a face cue directed covert attention, contrast sensitivity was enhanced, replicating the previous effect of covert attention on early perception. In addition, two effects for the emotional expression of the face cue were observed. First, irrespective of whether or not the face cue directed covert attention, a fear face cue enhanced contrast sensitivity. Second, if a fear face cue directed covert attention, contrast sensitivity was enhanced more than would have been predicted by the independent effects of a fear face cue and covert attention on perception. In other words, emotion enhances perception and potentiates the perceptual benefit of attention. These results are consistent with a model in which the emotion, via the amygdala, modulates processing in early visual regions.

The amygdala, through its extensive connectivity with sensory processing regions (Amaral et al. 1992, Freese & Amaral 2005), is ideally situated to influence perception with emotion. The evidence indicates that the amygdala's influence on perception may underlie emotion's facilitation of attention. However, emotion is also proposed to have another effect on attention, which is to capture attention. When attention is captured by emotion, there is impaired processing of nonemotional aspects of the stimulus or event. A number of studies have demonstrated the capture of attention with emotion (Pratto & John 1991), and it is suggested that this effect is due to difficulty disengaging attention from the emotional qualities of a stimulus (Fox et al. 2001). Little is known about the neural systems underlying the capture of attention with emotion, or how this effect may be related to the facilitation of attention and perception observed when processing emotional aspects of a cue or stimulus.

The studies examining emotion's facilitation or capture of attention have primarily used negative, fearful, or threatening stimuli. For some paradigms, it appears arousal is the key component for the facilitation of attention (Anderson 2005), and for others the effects appear to be specific for negative or threatening stimuli (Ohman et al. 2001). Discussions of the adaptive function of emotion's facilitation of attention emphasize the preferential detection of stimuli that signal potential importance or threat (Whalen 1998) and suggest a primary role for the amygdala is the modulation of vigilance in the presence of these stimuli. The current evidence of emotion's influence on attention and perception is largely consistent with this interpretation.

PROCESSING EMOTION IN SOCIAL STIMULI

There is some debate as to whether there are specialized neural mechanisms for processing social stimuli. For instance, some regions of the fusiform gyrus are proposed to be specialized for the recognition of facial identity (Kanwisher et al. 1997). Patients with damage to this region show deficits in recognizing individuals from their faces (Farah 1990). However, it is also proposed that processing in this region may reflect a more general mechanism for the identification of individual exemplars from classes of stimuli for which subjects have developed some level of expertise (Tarr & Gauthier 2000).

Studies of the cognitive mechanisms underlying the recognition of facial identity indicate that faces are not processed like most other objects. Recognizing faces depends on holistic and configural processing to a greater extent (Farah et al. 1998). Although the debate remains open as to whether the neural systems underlying the processing of face stimuli are unique to faces, it is clear that we have developed complex cognitive mechanisms that allow us to quickly distinguish friends from strangers.

Studies on the recognition of facial expression suggest that identifying emotion from face stimuli relies on yet a different set of processes and different neural substrates. This is perhaps not surprising given that the consistent identification of expressions across individuals requires recognizing similarities among different exemplars of face stimuli, whereas the recognition of identity requires recognizing differences among exemplars. It has been proposed that depending on the particular facial expression, different neural mechanisms may be important. In general, these brain regions are not thought to be specialized for recognizing a specific facial expression, but rather are thought to have more general role in the processing of different emotions. For example, it is suggested that insular cortex is important for the recognition of disgust in social and nonsocial stimuli, whereas the basal ganglia play a role in the recognition of anger (Calder et al. 2001, Lawrence et al. 2002). Although brain imaging studies of the amygdala indicate it responds to a range of facial expressions (e.g., Anderson et al. 2003), it appears to play a critical role in perceiving fear. Patients with amygdala damage show impairments in identifying

the intensity of fear in facial expressions (Adolphs et al. 1999). These patients are able to generate normal facial expressions of fear (Anderson & Phelps 2000), but consistently rate expressions of fear in others as less fearful than do normal control subjects. As mentioned earlier, brain-imaging studies show a preferential response to expressions of fear (Breiter et al. 1996), even when presented subliminally (Whalen et al. 1998).

Given that different neural mechanisms may underlie the recognition of facial identity and the recognition of different facial expressions, it would not be surprising that different types of information are necessary to make these judgments. Two recent studies examining the amygdala's role in the identification of fear expressions highlight this point. It is known that recognizing identity from face stimuli depends critically on the specific configuration of different facial features (see Farah et al. 1998 for a review). Subtle changes in this configuration, such as altering the distance between the eyes and mouth, can significantly impair the recognition of facial identity. In contrast, recognizing fear from face stimuli seems to depend critically on a single facial feature—the eyes. A recent imaging study by Whalen and colleagues (2004) demonstrated that amygdala activation was more robust in response to subliminal presentation of the eyes alone for fearful than for happy facial expressions. The magnitude of amygdala activation in response to the eyes of fearful versus happy faces was similar to that observed when the entire face is presented.

Another recent study by Adolphs and colleagues (2005a) used a technique that helped identify which aspects of a face are most important when recognizing expressions. Consistent with the imaging results (Whalen et al. 2004), the identification of fear seemed to depend critically on the eyes. An examination of eye movements when subjects were presented fearful facial expressions showed that normal control subjects predominately fixated on the eyes. A patient with bilateral damage to the amygdala showed a different pattern of eye movements, which suggests she was relying on other facial features in her efforts to identify the emotion. Surprisingly, when this patient was cued to focus on the eyes, her ability to identify fear from facial expressions improved to normal levels; however, she failed to adopt this strategy in the absence of explicit instructions. These results indicate that the amygdala not only responds preferentially to the eyes in face stimuli, but also may be involved in generating behaviors that aid in the identification of facial expressions of fear. Although a unique pattern of facial movements are needed to generate a facial expression of fear (Ekman & Freisen 1976), it seems that not all of these characteristic facial movements have equal importance in communicating this emotion.

The studies identifying the perceptual cues necessary for the identification of fearful facial expressions, which differ for the recognition of identity and, perhaps, other facial expressions, emphasize the complexity in understanding the range of information conveyed in face stimuli and how it is used to communicate information about social interactions. In addition, it has recently been shown that the context of the presentation of a facial expression can significantly alter the amygdala response. Fear and surprise are the two facial expressions that are likely to be

confused due to the similarity in the characteristic facial configurations for these expressions. A recent brain-imaging study found that if a surprise facial expression is preceded by a sentence consistent with a surprise reaction, such as “She just won \$500 dollars,” there is less of a BOLD response in the amygdala than if the sentence is consistent with a fear reaction, such as “He just lost \$500 dollars.” In other words, an emotional context conveyed verbally can alter the amygdala response to a facial expression. In addition, other cues, such as body movements consistent with fear, lead to amygdala activation (de Gelder et al. 2004). These studies suggest that although it is possible to identify the perceptual cues that are most important in evaluating facial expressions of fear, contextual and symbolic information can alter responses in brain regions that mediate the identification of facial expressions from face cues.

When interpreting emotion from facial expression, the amygdala appears to have a specialized role in processing fear. This role may also extend to other facial cues that signal potential threat. For example, two recent studies examined the role of the amygdala in the perception of trust from an individual’s face. Although the facial characteristics that convey trust are not well understood, it was found that this type of judgment seems to depend on the amygdala. When viewing pictures of faces that are rated as more or less “trustworthy,” greater amygdala activation was observed in response to pictures of individuals who were rated as untrustworthy based on their faces (Winston et al. 2002). Consistent with this result, patients with amygdala damage rated pictures of individuals whose faces were deemed untrustworthy by normal controls as both more trustworthy and more approachable (Adolphs et al. 1998).

Although the amygdala may be especially attuned to facial signals of threat or danger, it may also have a broader role in perceiving complex social and emotional signals from both social and nonsocial stimuli. A study by Adolphs and colleagues demonstrated the subtlety of the amygdala’s role in the processing of social cues by showing that it extends to the perception of social and emotional information from nonsocial stimuli. Heberlein & Adolphs (2004) examined the ability to anthropomorphize. It is a natural human tendency to see social and emotional cues and interactions among ambiguous or nonsocial stimuli. This tendency to see human social motives and emotional reactions can extend to inanimate objects. In a classic study, Heider & Simmel (1944) showed subjects a film of different geometric shapes moving around a box. Although these were simple shapes, the nature of the movements resulted in subjects describing the shapes as characters with motives interacting in a complex social situation. Heberlein & Adolphs (2004) showed this video to patients with amygdala damage and found that, unlike normal control subjects, their description of the film emphasized the actual movements of the geometric shapes, devoid of any social or emotional context or motives. These results indicate that the amygdala may play a more general role in perceiving and interpreting emotion from a wide range of stimuli, and the precise perceptual features conveying this information may be less important than the social or emotional content that can be interpreted.

Cognitive psychology research on the processing of social stimuli has primarily focused on understanding the types of cues that are critical to recognize facial identity. The emerging cognitive neuroscience research on the processing of emotion in faces indicates that a different set of processes and cues are required to perceive social, emotional information. The notion that there are different neural substrates that may respond to different facial expressions (Calder et al. 2001) suggests that depending on emotional expression, different types of cues may be important. In addition, the context in which an expression is conveyed can alter the amygdala's response. These results indicate that understanding how faces communicate social or emotional information may depend on the confluence of a range of cues, including, but not limited to, those that signal facial identity, the social context, and the facial expression.

Although the amygdala seems to play a critical role in the processing of fear from facial expressions, it may also play a broader role in perceiving and processing emotion from social and nonsocial cues. As previously mentioned, the amygdala responds to a range of social information, including information about social groups defined by race (Hart et al. 2000, Phelps et al. 2000), bodily cues conveying emotion (de Gelder et al. 2004), other facial expressions (Anderson et al. 2003, Whalen et al. 2001), and interpreting social, emotional information from nonsocial stimuli (Heberlein & Adolphs 2004). It appears that the amygdala is not specialized for detecting fear from faces, even though it is especially attuned to process fearful expressions.

CHANGING EMOTIONAL RESPONSES

A few decades ago, there was debate among psychologists Robert Zajonc and Richard Lazarus concerning the relation between emotion and cognition. The debate, which was highlighted in adjoining articles in the *American Psychologist*, centered around the question of whether the detection of emotion preceded cognitive processing (Zajonc 1984), or whether cognitive functions were a necessary component in the detection and experience of emotion (Lazarus 1984).

Given that the early cognitive neuroscience research on the neural systems of emotion in humans was inspired by studies conducted with nonhuman animals, mostly rats, these studies tended to focus on the primacy of affective responses and not the influence of cognition. Indeed, there is significant evidence that signals of emotion are processed by the amygdala automatically, irrespective of attention (Anderson et al. 2003, Vuilleumier et al. 2001) and awareness (Morris et al. 1998b, Whalen et al. 1998), and that this early detection of emotion can influence a range of cognitive functions, including perception, attention, and memory. Research with nonhuman animals has found that there are specialized subcortical pathways that allow for the early detection of emotion so that the amygdala can perceive potential threat in the environment prior to the completion of standard perceptual functions (Romanski & LeDoux 1992), and there is some evidence that this pathway exists

in humans (de Gelder et al. 1999, but see also Pessoa et al. 2002). These findings strongly support the position that the processing of emotion occurs prior to a complete cognitive analysis (Zajonc 1984).

However, an emerging body of research suggests that a range of cognitive functions can also affect the amygdala and the experience of emotion. These studies indicate that the results of cognitive interpretations are an important factor in the perception of emotion. A few examples of this were described above. For instance, the studies on instructed fear demonstrate that in some circumstances linguistic interpretation and episodic memory are critical for emotional learning. Abstract representations of the emotional properties of a stimulus will influence the amygdala (Phelps et al. 2001), which in turn mediates the physiological expression of fear (Funayama et al. 2001). An emotional context conveyed through language can also alter the amygdala's response to facial expressions, as was demonstrated in the study by Whalen and colleagues (2004) in which the amygdala's response to a surprise face was modulated by the sentence preceding its presentation. In addition, the amygdala's response to social groups defined by race can be modulated by task demands (Wheeler & Fiske 2004). These studies support the position that cognitive functions are a necessary component in understanding the neural systems and processing of emotion (Lazarus 1984).

More recently, there has been a renewed interest in the influence of cognition on emotion inspired by investigations of emotion regulation (see Gross 2002 for a review). The ability to regulate our emotional responses and states is a critical component of normal social function and adaptive interactions with the environment. Although certain stimuli may be prone to evoke an emotional reaction, how those stimuli are processed and interpreted can have a profound impact on both internal states and expressed behaviors and actions. Through conscious strategies and practice, individuals can change their interpretation of specific stimuli, and this can alter emotional reactions. Changing emotional responses through reasoning and strategies emphasizes the impact of cognition on emotion.

Recent studies exploring the neural mechanisms of regulating emotional responses to negative stimuli have suggested one consequence of these conscious regulation strategies is to alter the amygdala response. In a study by Ochsner and colleagues (2002), subjects viewed pictures of emotional and neutral scenes. For some scenes, the subjects were asked to simply attend to their natural emotional reactions. For other scenes, subjects were instructed to reappraise the emotional significance of the situation presented in the scene. For example, if a scene of women crying outside a church is presented, one interpretation is that it is a funeral and the women are crying in grief. However, if instructed to "reappraise" the emotional scene, subjects might instead imagine that the women crying in joy at the wedding of a loved one. Reappraisal is similar to viewing the cup as half full as opposed to half empty. Reappraising the scene can alter the experience of emotion (Gross 2002). Ochsner and colleagues (2002) found that it also diminishes amygdala activation (see Schaefer et al. 2002 for a similar result). A comparison of "reappraise" and "attend" trials for negative scenes showed decreased activation

of the amygdala and increased activation in the middle frontal gyrus of the left, lateral prefrontal cortex (PFC). This lateral PFC region has previously been linked to executive processes of working memory (see, e.g., Smith & Jonides 1999), which suggests this region may be involved in the online processing of the interpretation of the scene. Activation in this left lateral PFC region was correlated with reappraisal success. Those subjects who showed greater activation in this region to “reappraise” versus “attend” trials showed a greater change in their reported emotional response to scenes with reappraisal. In addition, activation in this lateral PFC region was correlated with activation of the amygdala, which suggests a role for this region in inhibiting the amygdala response to these complex scenes.

The finding that brain regions linked to executive function and working memory are correlated with the amygdala response during the conscious reappraisal of scenes suggests one pathway by which complex cognitive manipulations of stimuli might influence the neural circuitry of emotion. However, anatomical connectivity studies of the amygdala and PFC suggest that communication between these regions is not direct. Within the PFC, more ventral and medial regions are thought to be more similar across species, and the amygdala’s connectivity with the PFC is primarily through these regions (McDonald et al. 1996, Stefanacci & Amaral 2002). Studies that have explored the role of the PFC in the inhibition of affect and amygdala function in nonhuman animals have emphasized the involvement of more ventral, medial PFC (vmPFC) areas (Milad & Quirk 2002, Morgan & LeDoux 1995). These studies have primarily examined the extinction of conditioned fear. Once a conditioned fear is acquired, this fear response can be changed through extinction. During a typical extinction procedure, the CS is no longer paired with the US. The animal eventually learns that the CS does not predict the US and the expression of conditioned fear is diminished. A number of studies in nonhuman animals have demonstrated that the vmPFC plays an important role in the retention of extinction learning and inhibiting the amygdala response. This inhibition of the amygdala mediates the diminished expression of conditioned fear with extinction (see Milad & Quirk 2002 for a review). Results from a brain imaging study in humans examining the neural mechanisms of extinction learning were consistent with this animal model (Phelps et al. 2004).

A recent study investigated if the conscious regulation of emotion, which is unique to humans and depends on cognitive strategies, is linked to the mechanisms of extinction learning (Delgado et al. 2004). Both means of changing emotional responses involve interactions between the amygdala and PFC, but the precise region of the PFC seems to vary. In this study, subjects were asked to regulate a conditioned fear response in which the CS’s were colored squares, one of which was paired with a mild shock to the wrist (the US). On the “regulation” trials, subjects were instructed to imagine a soothing scene from nature that incorporated the color of the CS. On the “attend” trials, subjects were instructed to simply attend to their natural feelings and reactions. Consistent with the findings of Ochsner and colleagues (2002), using an emotion regulation strategy diminished the conditioned fear response, resulted in decreased activation of the amygdala, and

enhanced activation in the left lateral PFC. In addition, activation was also observed in the vmPFC region during emotion regulation. The pattern of this activation and its location within the medial PFC mirrored those observed when fear was diminished with extinction learning (Phelps et al. 2004). These results suggest that conscious, emotion regulation strategies, which depend on lateral PFC regions known to be important for executive processes and working memory (Smith & Jonides 1999), may act to diminish negative emotional responses by virtue of their influence on medial PFC regions that have been shown to inhibit the amygdala during extinction. In other words, emotion regulation strategies, which are unique to humans and seem to depend on regions of the PFC that differ in humans, may inhibit fear responses by co-opting the mechanisms of fear extinction that are similar across species.

As our understanding of the influence of higher cognitive functions on emotion processing grows, it has become increasingly apparent that even subcortical neural mechanisms that are preserved across a wide range of species, such as the amygdala, can be significantly influenced by neural systems and behaviors that are uniquely human, such as reasoning and strategies. These higher cognitive functions may influence the amygdala by taking advantage of neural mechanisms that evolved to accomplish more simple tasks across species. These findings highlight the complexity of the debate made famous by Lazarus (1984) and Zajonc (1984) more than 20 years ago. At that time, it seemed reasonable to debate whether emotion or cognition is primary when processing and interpreting stimuli. More recent research from cognitive neuroscience suggests that the answer could not be so simple. The mechanisms of emotion and cognition are intertwined from early perception to complex reasoning. It appears that understanding the separate contributions of emotion and cognition when processing stimuli becomes increasingly difficult as we learn more about the nature of the psychological and neural representations of behaviors typically categorized as either emotion or cognition.

CONCLUSION

As our understanding of the cognitive neuroscience of emotion and cognition grows, it is increasingly apparent that the division of human behavior into emotion and cognition is not as clear as previous philosophical and psychological investigations have suggested. The mechanisms of emotion and cognition appear to be intertwined at all stages of stimulus processing and their distinction can be difficult. It is also apparent that much like the study of cognition divided functions into different domains, such as memory, attention, and reasoning, the concept of emotion has a structural architecture that may be similarly diverse and complex (Russell & Barrett 1999, Scherer 2000). This review has focused on “emotion” overall, but for the different cognitive domains explored the precise characteristics of emotion that influence specific cognitive functions may differ.

As we move forward in the study of the representation of cognition, it is clear that a consideration of emotion is necessary. Examining cognitive functions without an appreciation for the social, emotional, and motivational context will result in an understanding that may be limited in its applicability outside of the research laboratory. The traditional research domains of psychology, such as cognitive, social, and clinical, may help create unified areas of research, but may also diminish our appreciation of the complexity of human behavior by discouraging discussion of their interactions. Adding the complexity of emotion to the study of cognition can be daunting, but investigations of the neural mechanisms underlying these behaviors can help clarify the structure and mechanisms.

ACKNOWLEDGMENTS

The author would like to acknowledge Meridith Carson and Joel Pearson for assistance with the preparation of this manuscript, and the James McKeen Cattell Foundation.

The *Annual Review of Psychology* is online at <http://psych.annualreviews.org>

LITERATURE CITED

- Adolphs R, Gosselin F, Buchanan TW, Tranel D, Schyns P, Damasio AR. 2005a. A mechanism for impaired fear recognition after amygdala damage. *Nature* 433:68–72
- Adolphs R, Tranel D, Buchanan TW. 2005b. Amygdala damage impairs emotional memory for gist but not details of complex stimuli. *Nat. Neurosci.* 8(4):512–18
- Adolphs R, Tranel D, Damasio AR. 1998. The human amygdala in social judgment. *Nature* 393:470–74
- Adolphs R, Tranel D, Hamann S, Young AW, Calder AJ, et al. 1999. Recognition of facial emotion in nine individuals with bilateral amygdala damage. *Neuropsychologia* 37:1111–17
- Amaral DG, Behniea H, Kelly JL. 2003. Topographic organization of projections from the amygdala to the visual cortex in the macaque monkey. *Neuroscience* 118:1099–120
- Amaral DG, Price JL, Pitkanen A, Carmichael ST. 1992. Anatomical organization of the primate amygdaloid complex. In *The Amygdala: Neurobiological Aspects of Emotion, Memory, and Mental Dysfunction*, ed. JP Aggleton, pp. 1–65. New York: Wiley-Liss
- Anderson AK. 2005. Affective influences on the attentional dynamics supporting awareness. *J. Exp. Psychol.: Gen.* 134(2):258–81
- Anderson AK, Christoff K, Panitz D, DeRosa E, Gabrieli JD. 2003. Neural correlates of the automatic processing of threat facial signs. *J. Neurosci.* 23(13):5627–33
- Anderson AK, Phelps EA. 2000a. Perceiving emotion: more than meets the eye. *Curr. Biol.* 10:551–54
- Anderson AK, Phelps EA. 2000b. Expression without recognition: contributions of the human amygdala to emotional communication. *Psychol. Sci.* 11:106–11
- Anderson AK, Phelps EA. 2001. Lesions of the human amygdala impair enhanced perception of emotionally salient events. *Nature* 411:305–9
- Baxter MG, Murray EA. 2002. The amygdala and reward. *Nat. Rev. Neurosci.* 3:563–73
- Bechara A, Tranel D, Damasio H, Adolphs R, Rockland C, Damasio AR. 1995. Double dissociation of conditioning and declarative knowledge relative to the amygdala and hippocampus in humans. *Science* 269:1115–18

- Berlyne DE. 1969. Arousal, reward and learning. *Ann. NY Acad. Sci.* 159(3):1059–70
- Bower GH. 1981. Mood and memory. *Am. Psychol.* 36:129–48
- Breiter HC, Etcoff NL, Whalen PJ, Kennedy WA, Rauch SL, et al. 1996. Response and habituation of the human amygdala during visual processing of facial expression. *Neuron* 17(5):875–87
- Buchel C, Morris J, Dolan RJ, Friston KJ. 1998. Brain systems mediating aversive conditioning: an event-related fMRI study. *Neuron* 20:947–57
- Cabeza R, Kingstone A. 2001. *Handbook of Functional Neuroimaging of Cognition*. Cambridge, MA: MIT Press
- Cahill L, Alkire MT. 2003. Epinephrine enhancement of human memory consolidation: interaction with arousal at encoding. *Neurobiol. Learn. Mem.* 79:194–98
- Cahill L, Babinsky R, Markowitsch HJ, McGaugh JL. 1995. The amygdala and emotional memory. *Nature* 377:295–96
- Cahill L, Gorski L, Le K. 2003. Enhanced human memory consolidation with post-learning stress: interaction with the degree of arousal at encoding. *Learn. Mem.* 10:270–74
- Cahill L, Haier RJ, Fallon J, Alkire MT, Tang C, et al. 1996. Amygdala activity at encoding correlated with long-term, free recall of emotional information. *Proc. Natl. Acad. Sci. USA* 93:8016–21
- Cahill L, Prins B, Weber M, McGaugh JL. 1994. Beta-adrenergic activation and memory for emotional events. *Nature* 371:702–4
- Cahill L, Weinberger NM, Roozendaal B, McGaugh JL. 1999. Is the amygdala a locus of “conditioned fear”? Some questions and caveats. *Neuron* 23:227–28
- Calder AJ, Lawrence AD, Young AW. 2001. Neuropsychology of fear and loathing. *Neuroscience* 2:352–63
- Canli T, Zhao Z, Brewer J, Gabrieli JD, Cahill L. 2000. Event-related activation in the human amygdala associates with later memory for individual emotional experience. *J. Neurosci.* 20:RC99
- Carrasco M. 2004. Covert transient attention increases contrast sensitivity and spatial resolution: support for signal enhancement. In *Neurobiology of Attention*, ed. L Itti, G Rees, J Tsotsos, pp. 442–47. San Diego, CA: Elsevier
- Carrasco M, Penpeci-Talgar C, Eckstein M. 2000. Spatial covert attention increases contrast sensitivity across the CSF: support for signal enhancement. *Vis. Res.* 40:1203–15
- Cherry EC. 1953. Some experiments on the recognition of speech, with one and two ears. *J. Acoust. Soc. Am.* 25:975–79
- Chun MM, Potter MC. 1995. A two-stage model for multiple target detection in rapid serial visual presentation. *J. Exp. Psychol. Hum. Percept. Perform.* 21(1):109–27
- Corbetta M, Shulman GL. 2002. Control of goal-directed and stimulus-driven attention in the brain. *Nat. Rev. Neurosci.* 3:201–15
- Craik FIM, Govoni R, Naveh-Benjamin M, Anderson ND. 1996. The effects of divided attention on encoding and retrieval processes in human memory. *J. Exp. Psychol. Gen.* 125:159–80
- Cunningham WA, Johnson MK, Raye CL, Chris Gatenby J, Gore JC, Banaji MR. 2004. Separable neural components in the processing of black and white faces. *Psychol. Sci.* 15(12):806–13
- Davis M. 1992. The role of the amygdala in conditioned fear. In *The Amygdala: Neurobiological Aspects of Emotion, Memory and Mental Dysfunction*, ed. JP Aggleton, pp. 255–306. New York: Wiley-Liss
- Davis M, Whalen PJ. 2001. The amygdala: vigilance and emotion. *Mol. Psychiatry* 6:13–34
- de Gelder B, Snyder J, Greve D, Gerard G, Hadjikhani N. 2004. Fear fosters flight: a mechanism for fear contagion when perceiving emotion expressed by a whole body. *Proc. Natl. Acad. Sci. USA* 101:16701–6
- de Gelder B, Vroomen J, Pourtois G, Weiskrantz L. 1999. Non-conscious recognition of affect in the absence of striate cortex. *Neuroreport* 10:3759–63

- Delgado MR, Trujillo JL, Holmes B, Nearing KI, LeDoux JE, Phelps EA. 2004. *Emotion regulation of conditioned fear: the contributions of reappraisal*. Presented at Annu. Meet. Cogn. Neurosci. Soc., 11th, San Francisco
- Dolcos F, LaBar KS, Cabeza R. 2004. Interaction between the amygdala and the medial temporal lobe memory system predicts better memory for emotional events. *Neuron* 42:855–63
- Easterbrook JA. 1959. The effect of emotion on cue utilization and the organization of behavior. *Psychol. Rev.* 66(3):183–201
- Eichenbaum H. 2002. *The Cognitive Neuroscience of Memory*. New York: Oxford Univ. Press
- Ekman P, Friesen W. 1976. *Pictures of Facial Affect*. Palo Alto, CA: Consult. Psychol. Press
- Esteves F, Dimberg U, Ohman A. 1994. Automatically elicited fear: conditioned skin conductance responses to masked facial stimuli. *Cogn. Emot.* 8:393–413
- Everitt BJ, Cardinal RN, Parkinson JA, Robbins TW. 2003. Appetitive behavior: impact of amygdala-dependent mechanisms of emotional learning. *Ann. NY Acad. Sci.* 985:233–50
- Fanselow MS, Poulos AM. 2005. The neuroscience of mammalian associative learning. *Annu. Rev. Psychol.* 56:207–34
- Farah M. 1990. *Visual Agnosia: Disorders of Object Recognition and What They Tell Us About Normal Vision*. Cambridge, MA: MIT Press
- Farah MJ, Wilson KD, Drain M, Tanaka JN. 1998. What is “special” about face perception? *Psychol. Rev.* 105(3):482–98
- Fox E, Russo R, Bowles R, Dutton K. 2001. Do threatening stimuli draw or hold visual attention in subclinical anxiety? *J. Exp. Psychol. Gen.* 130(4):681–700
- Freese JL, Amaral DG. 2005. The organization of projections from the amygdala to visual cortical areas TE and V1 in the Macaque monkey. *J. Comp. Neurol.* 486(4):295–317
- Funayama ES, Grillon C, Davis M, Phelps EA. 2001. A double dissociation in the affective modulation of startle in humans: effects of unilateral temporal lobectomy. *J. Cogn. Neurosci.* 13:721–29
- Gandhi SP, Heeger DJ, Boynton GM. 1999. Spatial attention affects brain activity in human primary visual cortex. *Proc. Natl. Acad. Sci. USA* 96:3314–19
- Garcia R. 2002. Stress, synaptic plasticity, and psychopathology. *Rev. Neurosci.* 13:195–208
- Gazzaniga MS, Irvy RB, Mangun GR. 2002. *Cognitive Neuroscience*. New York: Norton. 2nd ed.
- Gross JJ. 2002. Emotion regulation: affective, cognitive, and social consequences. *Psychophysiology* 39:281–91
- Hamann SB, Ely TD, Grafton ST, Kilts CD. 1999. Amygdala activity related to enhanced memory for pleasant and aversive stimuli. *Nat. Neurosci.* 2:289–93
- Hansen CH, Hansen RD. 1988. Finding the face in the crowd: an anger superiority effect. *J. Personal. Soc. Psychol.* 54:917–24
- Hart AJ, Whalen PJ, Shin LM, McInerney SC, Fischer H, Rauch SL. 2000. Differential response in the human amygdala to racial outgroup vs. ingroup face stimuli. *Neuroreport* 11(11):2351–55
- Heberlein AS, Adolphs R. 2004. Impaired spontaneous anthropomorphizing despite intact perception and social knowledge. *Proc. Natl. Acad. Sci. USA* 101:7487–91
- Heider F, Simmel M. 1944. An experimental study of apparent behavior. *Am. J. Psychol.* 57:243–59
- Henson RN, Rugg MD, Shallice T, Josephs O, Dolan RA. 1999. Recollection and familiarity in recognition memory: an event-related functional magnetic resonance imaging study. *J. Neurosci.* 19:3962–72
- Heuer F, Reisberg D. 1992. Emotion, arousal, and memory for detail. In *The Handbook of Emotion and Memory*, ed. S. Christianson, pp. 151–64. Hillsdale, NJ: Erlbaum
- Holland PC, Gallagher M. 2004. Amygdalo-frontal interactions and reward expectancy. *Curr. Opin. Neurobiol.* 14:148–55

- Hugdahl K, Ohman A. 1977. Effects of instruction acquisition and extinction of electrodermal responses to fear-relevant stimuli. *J. Exp. Psychol. Hum. Learn. Mem.* 3:608–18
- James W. 1890. *The Principles of Psychology*. New York: Dover. 670 pp.
- Johnsrude IS, Owen AM, White NM, Zhao WV, Bohbot V. 2000. Impaired preference conditioning after anterior temporal lobe resection in humans. *J. Neurosci.* 20:2649–56
- Kanwisher N, McDermott J, Chun MM. 1997. The fusiform face area: a module in human extrastriate cortex specialized for face perception. *J. Neurosci.* 17(11):4302–11
- Kapp BS, Whalen PJ, Supple WF, Pascoe JP. 1992. Amygdaloid contributions to conditioned arousal and sensory information processing. In *The Amygdala: Neurobiological Aspects of Emotion, Memory, and Mental Dysfunction*, ed. JP Aggleton, pp. 229–54. New York: Wiley-Liss
- Kensinger EA, Corkin S. 2004. Two routes to emotional memory: distinct neural processes for valence and arousal. *Proc. Natl. Acad. Sci. USA* 101(9):3310–15
- Kim H, Somerville LH, Johnstone T, Alexander AL, Whalen PJ. 2003. Inverse amygdala and medial prefrontal cortex responses to surprised faces. *Neuroreport* 14:2317–22
- Kim H, Somerville LH, Johnstone T, Polis S, Alexander AL, et al. 2004. Contextual modulation of amygdala responsivity to surprised faces. *J. Cogn. Neurosci.* 16(10):1730–45
- Kleinsmith LJ, Kaplan S. 1963. Paired-associate learning as a function of arousal and interpolated interval. *J. Exp. Psychol.* 65:190–93
- Kluver H, Bucy PC. 1937. “Psychic blindness” and other symptoms following bilateral temporal lobectomy in rhesus monkeys. *Am. J. Physiol.* 119:352–53
- Knowlton BJ, Fanselow MS. 1998. The hippocampus, consolidation and on-line memory. *Curr. Opin. Neurobiol.* 8(2):293–96
- Kohler S, Crane J, Milner B. 2002. Differential contributions of the parahippocampal place area and the anterior hippocampus to human memory for scenes. *Hippocampus* 12:718–23
- Kosslyn SM, Shin LM, Thompson WL, McNally PJ, Rauch SL, et al. 1996. Neural effects of visualizing and perceiving aversive stimuli: a PET investigation. *Neuroreport* 7:1569–76
- LaBar KS, Gatenby JC, Gore JC, LeDoux JE, Phelps EA. 1998. Human amygdala activation during conditioned fear acquisition and extinction: a mixed-trial fMRI study. *Neuron* 20:937–45
- LaBar KS, LeDoux JE, Spencer DD, Phelps EA. 1995. Impaired fear conditioning following unilateral temporal lobectomy in humans. *J. Neurosci.* 15:6846–55
- LaBar KS, Phelps EA. 1998. Arousal-mediated memory consolidation: role of the medial temporal lobe in humans. *Psychol. Sci.* 9:490–93
- Lachman R, Lachman JL, Butterfield EC. 1979. *Cognitive Psychology and Information Processing: An Introduction*. Hillsdale, NJ: Erlbaum
- Lawrence AD, Calder AJ, McGowan SW, Grasby PM. 2002. Selective disruption of the recognition of facial expressions of anger. *Neuroreport* 13(6):881–84
- Lazarus RS. 1984. On the primacy of cognition. *Am. Psychol.* 39(2):124–29
- LeDoux JE. 1996. *The Emotional Brain*. New York: Simon & Schuster
- Maren S. 2001. Neurobiology of Pavlovian fear conditioning. *Annu. Rev. Neurosci.* 24:897–931
- McDonald AJ, Mascagni F, Guo L. 1996. Projections of the medial and lateral prefrontal cortices to the amygdala: a *Phaseolus vulgaris* leucoagglutinin study in the rat. *Neuroscience* 71:55–75
- McEwen BS, Sapolsky RM. 1995. Stress and cognitive function. *Curr. Opin. Neurobiol.* 5(2):205–16
- McGaugh JL. 2000. Memory—a century of consolidation. *Science* 287:248–51
- McGaugh JL. 2002. Memory consolidation and the amygdala: a systems perspective. *Trends Neurosci.* 25:456

- McGaugh JL. 2004. The amygdala modulates the consolidation of memories of emotionally arousing experiences. *Annu. Rev. Neurosci.* 27:1–28
- Milad MR, Quirk GJ. 2002. Neurons in medial prefrontal cortex signal memory for fear extinction. *Nature* 420:70–74
- Miller GA. 2003. The cognitive revolution: a historical perspective. *Trends Cogn. Sci.* 7:141–44
- Morgan MA, LeDoux JE. 1995. Differential contribution of dorsal and ventral medial prefrontal cortex to the acquisition and extinction of conditioned fear in rats. *Behav. Neurosci.* 109:681–88
- Morgan MA, Romanski LM, LeDoux JE. 1993. Extinction of emotional learning: contribution of medial prefrontal cortex. *Neurosci. Lett.* 163:109–13
- Morris JS, Friston KJ, Buchel C, Frith CD, Young AW, et al. 1998a. A neuromodulatory role for the human amygdala in processing emotional facial expressions. *Brain* 121 (Pt. 1):47–57
- Morris JS, Ohman A, Dolan RJ. 1998b. Conscious and unconscious emotional learning in the human amygdala. *Nature* 393:467–70
- Myers KM, Davis M. 2002. Behavioral and neural analysis of extinction. *Neuron* 36:567–84
- Neisser U. 1976. *Cognition and Reality: Principles and Implications of Cognitive Psychology*. New York: Freeman
- Neisser U, Harsch N. 1992. Phantom flashbulbs: false recollections of hearing news about the Challenger. In *Affect and Accuracy in Recall: Studies of "Flashbulb" Memories*, ed. E Winograd, U Neisser, pp. 9–31. London: Cambridge Univ. Press
- Niendenthal PM, Kitayama S. 1994. *The Heart's Eye: Emotional Influences in Perception and Attention*. San Diego, CA: Academic
- Ochsner KN. 2000. Are affective events richly recollected or simply familiar? The experience and process of recognizing feelings past. *J. Exp. Psychol. Gen.* 129(2):242–61
- Ochsner KN, Bunge SA, Gross JJ, Gabrieli JD. 2002. Rethinking feelings: an fMRI study of the cognitive regulation of emotion. *J. Cogn. Neurosci.* 14:1215–29
- Ohman A, Flykt A, Esteves F. 2001. Emotion drives attention: detecting a snake in the grass. *J. Exp. Psychol.: Gen.* 127:69–82
- Ohman A, Mineka S. 2001. Fears, phobias, and preparedness: toward an evolved module of fear and fear learning. *Psychol. Rev.* 108:483–522
- Olsson A, Nearing K, Zheng J, Phelps EA. 2004. *Learning by observing: neural correlates of fear learning through social observation*. Presented at Annu. Meet. Cogn. Neurosci. Soc., 11th, San Francisco
- Olsson A, Phelps EA. 2004. Learned fear of "unseen" faces after Pavlovian, observational, and instructed fear. *Psychol. Sci.* 15(12):822–28
- Packard MG, Teather LA. 1998. Amygdala modulation of multiple memory systems: hippocampus and caudate-putamen. *Neurobiol. Learn. Mem.* 69:163–203
- Pessoa L, McKenna M, Gutierrez E, Ungerleider LG. 2002. Neural processing of emotional faces requires attention. *Proc. Natl. Acad. Sci. USA* 99:11458–63
- Phelps EA. 2004. The human amygdala and awareness: interaction of the amygdala and hippocampal complex. *Curr. Opin. Neurobiol.* 14:198–202
- Phelps EA, Delgado MR, Nearing KI, LeDoux JE. 2004. Extinction learning in humans: role of the amygdala and vmPFC. *Neuron* 43:897–905
- Phelps EA, LaBar KS, Anderson A, O'Connor KJ, Fulbright RK, Spencer DD. 1998. Specifying the contributions of the human amygdala to emotional memory: a case study. *Neurocase* 4:527–40
- Phelps EA, Ling S, Carrasco M. 2005. Emotion facilitates perception and potentiates the perceptual benefit of attention. *Psychol. Sci.* In press
- Phelps EA, O'Connor KJ, Cunningham WA, Funayama ES, Gatenby JC, et al. 2000.

- Performance on indirect measures of race evaluation predicts amygdala activation. *J. Cogn. Neurosci.* 12:729–38
- Phelps EA, O'Connor KJ, Gatenby JC, Gore JC, Grillon C, Davis M. 2001. Activation of the left amygdala to a cognitive representation of fear. *Nat. Neurosci.* 4:437–41
- Pratto F, John OP. 1991. Automatic vigilance: the attention-grabbing power of negative social information. *J. Personal. Soc. Psychol.* 61(3):380–91
- Quirk GJ, Russo GK, Barron JL, Lebron K. 2000. The role of ventromedial prefrontal cortex in the recovery of extinguished fear. *J. Neurosci.* 20:6225–31
- Raymond JE, Shapiro KL, Arnell KM. 1992. Temporary suppression of visual processing in an RSVP task: an attentional blink? *J. Exp. Psychol.: Hum. Percept. Perform.* 18:849–60
- Rolls ET. 2000. Memory systems in the brain. *Annu. Rev. Psychol.* 51:599–630
- Romanski LM, LeDoux JE. 1992. Equipotentiality of thalamo-amygdala and thalamo-cortico-amygdala circuits in auditory fear conditioning. *J. Neurosci.* 12:4501–9
- Russell JA, Barrett LF. 1999. Core affect, prototypical emotional episodes, and other things called emotion: dissecting the elephant. *J. Personal. Soc. Psychol.* 69:379–99
- Schaefer SM, Jackson DC, Davidson RJ, Aguirre GK, Kimberg DY, Thompson-Schill SL. 2002. Modulation of amygdalar activity by the conscious regulation of negative emotion. *J. Cogn. Neurosci.* 14:913–21
- Scherer KR. 2000. Psychological models of emotion. In *The Neuropsychology of Emotion*. ed. JC Borod, pp. 137–62. New York: Oxford Univ. Press
- Scholck H, Buffalo EA, Squire LR. 2000. Memory for distortions develop over time: recollections of the O.J. Simpson trial verdict after 15 and 32 months. *Psychol. Sci.* 11:39–45
- Schultz W. 2006. Behavioral theories and the neurophysiology of reward. *Annu. Rev. Psychol.* 57:87–115
- Sharot T, Delgado MR, Phelps EA. 2004. How emotion enhances the feeling of remembering. *Nat. Neurosci.* 7:1376–80
- Sharot T, Phelps EA. 2004. How arousal modulates memory: disentangling the effects of attention and retention. *Cogn. Affect. Behav. Neurosci.* 4:294–306
- Shors T. 2006. Stressful experience and learning across the lifespan. *Annu. Rev. Psychol.* 57:55–85
- Smith EE, Jonides J. 1999. Storage and executive processes in the frontal lobes. *Science* 283:1657–61
- Squire LR, Zola-Morgan S. 1991. The medial temporal lobe memory system. *Science* 253(5026):1380–86
- Stefanacci L, Amaral DG. 2002. Some observations in cortical inputs to the macaque monkey amygdala: an anterograde tracing study. *J. Comp. Neurol.* 451:301–23
- Stefanacci L, Suzuki WA, Amaral DG. 1996. Organization of connections between the amygdaloid complex and the perirhinal and parahippocampal cortices in macaque monkeys. *J. Comp. Neurol.* 375(4):552–82
- Stuss DT, Levine B. 2002. Adult clinical neuropsychology: lessons from studies of the frontal lobes. *Annu. Rev. Psychol.* 53:401–33
- Talarico JM, Rubin DC. 2003. Confidence, not consistency, characterizes flashbulb memories. *Psychol. Sci.* 14:455–61
- Tarr MJ, Gauthier I. 2000. FFA: a flexible fusiform area for subordinate-level visual processing automatized by expertise. *Nat. Neurosci.* 3(8):764–69
- Vuilleumier P, Armony JL, Driver J, Dolan RJ. 2001. Effects of attention and emotion on face processing in the human brain: an event-related fMRI study. *Neuron* 30:829–41
- Vuilleumier P, Richardson MP, Armony JL, Driver J, Dolan RJ. 2004. Distant influences of amygdala lesion on visual cortical activation during emotional face processing. *Nat. Neurosci.* 7:1271–78
- Weinberger NM. 1995. Retuning the brain by fear conditioning. In *The Cognitive Neurosciences*, ed. MS Gazzaniga, pp. 1071–90. Cambridge, MA: MIT Press

- Weiskrantz L. 1956. Behavioral changes associated with ablation of the amygdaloid complex in monkeys. *J. Comp. Physiol. Psychol.* 49:381–91
- Whalen PJ. 1998. Fear, vigilance, and ambiguity: initial neuroimaging studies of the human amygdala. *Curr. Dir. Psychol. Sci.* 7:177–88
- Whalen PJ, Kagan J, Cook RG, Davis FC, Kim H, et al. 2004. Human amygdala responsiveness to masked fearful eye whites. *Science* 306:2061
- Whalen PJ, Rauch SL, Etcoff NL, McInerney SC, Lee MB, Jenike MA. 1998. Masked presentations of emotional facial expressions modulate amygdala activity without explicit knowledge. *J. Neurosci.* 18:411–18
- Whalen PJ, Shin LM, McInerney SC, Fischer H, Wright CI, Rauch SL. 2001. A functional MRI study of human amygdala responses to facial expressions of fear versus anger. *Emotion* 1(1):70–83
- Wheeler ME, Buckner RL. 2004. Functional-anatomic correlates of remembering and knowing. *Neuroimage* 21:1337–49
- Wheeler ME, Fiske ST. 2004. Controlling racial prejudice: Social-cognitive goals affect amygdala and stereotype activation. *Psychol. Sci.* 16:56–63
- Winston JS, Strange BA, O’Doherty J, Dolan RJ. 2002. Automatic and intentional brain responses during evaluation of trustworthiness of faces. *Nat. Neurosci.* 5:277–83
- Yonelinas AP. 2002. The nature of recollection and familiarity: a review of 30 years of research. *J. Mem. Lang.* 46:441–517
- Young MP, Scannell JW, Burns GA, Blakemore C. 1994. Analysis of connectivity: neural systems in the cerebral cortex. *Rev. Neurosci.* 5:227–50
- Zajonc RB. 1984. On the primacy of affect. *Am. Psychol.* 39:117–23

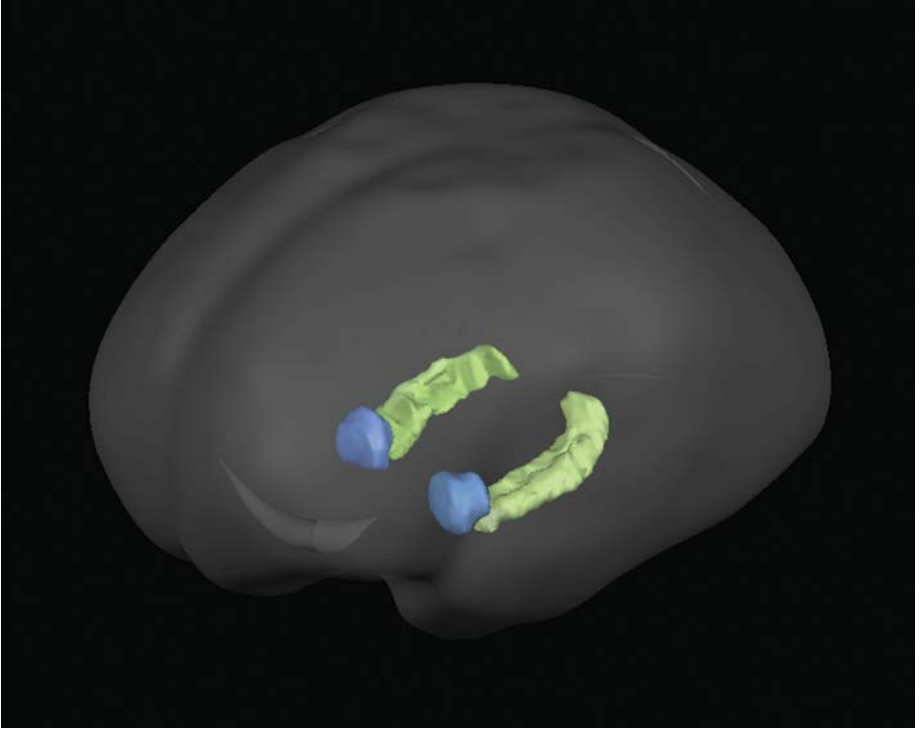


Figure 1 The amygdala (*blue*) and hippocampus (*green*). Reprinted from Phelps (2004).

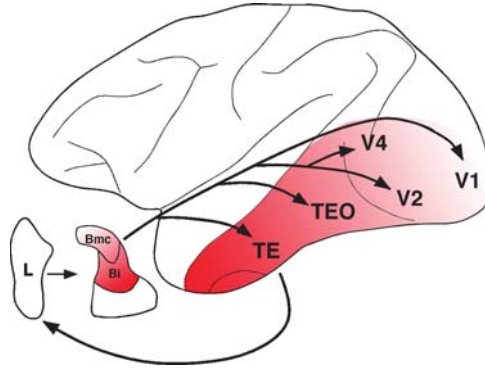


Figure 2 The connectivity of subregions of the amygdala (L, Bi, Bmc) and ventral visual cortical regions (V1, V2, V4, TEO, TE) in the macaque monkey. Reprinted from Freese & Amaral (2005).

CONTENTS

Frontispiece— <i>Herbert C. Kelman</i>	xvi
PREFATORY	
Interests, Relationships, Identities: Three Central Issues for Individuals and Groups in Negotiating Their Social Environment, <i>Herbert C. Kelman</i>	1
BRAIN MECHANISMS AND BEHAVIOR: EMOTION AND MOTIVATION	
Emotion and Cognition: Insights from Studies of the Human Amygdala, <i>Elizabeth A. Phelps</i>	27
STRESS AND NEUROENDOCRINOLOGY	
Stressful Experience and Learning Across the Lifespan, <i>Tracey J. Shors</i>	55
REWARD AND ADDICTION	
Behavioral Theories and the Neurophysiology of Reward, <i>Wolfram Schultz</i>	87
GENETICS OF BEHAVIOR	
Genetics of Affective and Anxiety Disorders, <i>E.D. Leonardo and René Hen</i>	117
SLEEP	
Sleep, Memory, and Plasticity, <i>Matthew P. Walker and Robert Stickgold</i>	139
COMPARATIVE PSYCHOLOGY, ETHOLOGY, AND EVOLUTION	
Neuroecology, <i>David F. Sherry</i>	167
EVOLUTIONARY PSYCHOLOGY	
The Evolutionary Psychology of Facial Beauty, <i>Gillian Rhodes</i>	199
LANGUAGE AND COMMUNICATION	
Explanation and Understanding, <i>Frank C. Keil</i>	227
ADOLESCENCE	
Adolescent Development in Interpersonal and Societal Contexts, <i>Judith G. Smetana, Nicole Campione-Barr, and Aaron Metzger</i>	255
INDIVIDUAL TREATMENT	
Enduring Effects for Cognitive Therapy in the Treatment of Depression and Anxiety, <i>Steven D. Hollon, Michael O. Stewart, and Daniel Strunk</i>	285
	vii

FAMILY/MARITAL THERAPY

- Current Status and Future Directions in Couple Therapy,
Douglas K. Snyder, Angela M. Castellani, and Mark A. Whisman 317

ATTITUDE CHANGE AND PERSUASION

- Attitudes and Persuasion, *William D. Crano and Radmila Prislín* 345

BARGAINING, NEGOTIATION, CONFLICT, SOCIAL JUSTICE

- Psychological Perspectives on Legitimacy and Legitimation, *Tom R. Tyler* 375

INDIVIDUAL DIFFERENCES AND ASSESSMENT

- Personality and the Prediction of Consequential Outcomes, *Daniel J. Ozer
and Verónica Benet-Martínez* 401

ENVIRONMENTAL PSYCHOLOGY

- Child Development and the Physical Environment, *Gary W. Evans* 423

MARKETING AND CONSUMER BEHAVIOR

- Consumer Psychology: Categorization, Inferences, Affect, and Persuasion,
Barbara Loken 453

STRUCTURES AND GOALS OF EDUCATIONAL SETTINGS

- Classroom Goal Structure, Student Motivation, and Academic
Achievement, *Judith L. Meece, Eric M. Anderman,
and Lynley H. Anderman* 487

DATA ANALYSIS

- Analysis of Longitudinal Data: The Integration of Theoretical Model,
Temporal Design, and Statistical Model, *Linda M. Collins* 505

TIMELY TOPICS

- The Internet as Psychological Laboratory, *Linda J. Skitka
and Edward G. Sargis* 529
- Family Violence, *Patrick Tolan, Deborah Gorman-Smith, and David Henry* 557
- Understanding Affirmative Action, *Faye J. Crosby, Aarti Iyer,
and Sirinda Sincharoen* 585

INDEXES

- Subject Index 613
- Cumulative Index of Contributing Authors, Volumes 47–57 637
- Cumulative Index of Chapter Titles, Volumes 47–57 642

ERRATA

An online log of corrections to *Annual Review of Psychology* chapters
may be found at <http://psych.annualreviews.org/errata.shtml>