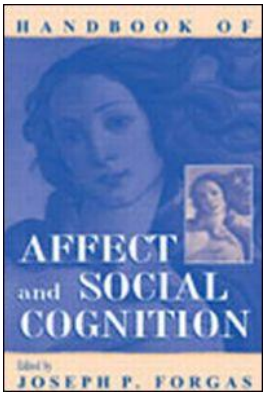

Handbook of Affect and Social Cognition

Joseph P. Forgas

The Interaction of Affect and Cognition: A Neurobiological Perspective

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I

The Relationship Between Affect and Cognition: Fundamental Issues

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The Interaction of Affect and Cognition: A Neurobiological Perspective

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Although emotion and cognition have sometimes been viewed as two distinct components of human psychology (Zajonc, 1980; Zajonc & KunstWilson, 1980), findings from animals and humans strongly support a modified view, in which emotion is an integral attribute of cognition. In fact, there is now good evidence that emotion modulates information processing in domains ranging from memory to reasoning to decision making. Not only does emotion modulate other aspects of cognition, but

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is also in fact properly considered cognitive in its own right, insofar as it constitutes computations over representations: namely, representations of the organism's body state.

In this chapter, we review the influences of emotion on cognition and sketch a theoretical framework that treats affect as thoroughly cognitive. We review recent experimental findings from cognitive neuroscience, with an emphasis on findings from humans that elucidate how affective processing fits into the economy of cognitive processing. Specifically, we discuss three domains for which a role in affective modulation is clearest: memory, judgment, and decision making.

COGNITIVE AFFECT

Emotions color virtually all aspects of our lives. What we pay attention to, what we decide to do, what we remember, and how we interact with other people are all influenced by emotion. Despite the fact that emotions permeate our thoughts and our behavior, the topic of emotion is a relative newcomer to cognitive neuroscience, largely for historical reasons. Recent studies have made substantial progress in understanding how the brain encodes and stores knowledge about emotion, how emotional states influence other cognitive processes, such as memory and decision making, and how emotional states influence behavior.

We can begin by pointing out that affective processing is representational, contrary to what some researchers have proposed. There is now good evidence from neuroanatomy, comparative studies, and neurophysiological and neuropsychological studies that supports the view that emotion concerns representations of the organism's state of the body. In particular, affective representations map the relationship between current or future body states and past or baseline states, with respect to how such changes in body state relate to the organism's survival and well-being. That is, emotion ultimately concerns homeostasis, broadly construed (Damasio, 1994, 1999; Panksepp, 1998).

In addition to representing changes in the global body state of an organism, emotional processing typically represents the relationship between such body state changes and external sensory stimuli. For example, the neural correlates of anger directed at another individual would consist in multiple neural mappings that provide a comprehensive representation of the external stimulus (the sight of the other individual), of the organism's own body state (e.g., readiness to fight), and of the relationship between the two (that the latter is a response toward the former, and that the former may have triggered

the latter). Such a comprehensive set of representations, which comprise the central state "anger," unfold in a complex fashion in time, an issue that has received attention in psychology from componentprocess theory (Scherer, 1984), and for which we can begin to sketch a rough outline of at least some of its anatomic components. First, perception of the external stimulus must in some way trigger relatively fast and automatic components of emotional response (e.g., changes in autonomic tone, heart rate). As these components of emotional response are unfolding in time, parallel components that rely more on retrieval of knowledge from declarative memory and reasoning, and that are more influenced by conscious volition, are triggered as well. Together, then, several different sets of emotional responses are triggered by the stimulus. They result in a dynamic change in somatosensory state of the body, somatovisceral function, endocrine and neuroendocrine function, autonomic tone, and global brain functioning, all adapted to maximize successful behavior in response to the stimulus.

Such an emotional response has several consequences that also unfold in a complex way in time. First, they directly engage, as well as indirectly modulate, the organism's automatic and planned behavior at all levels. Second, they are perceived and represented by the organism's brain as comprehensive changes in body and brain state, a component that results in the conscious experience of the emotion, or "feeling." Third, the organism's emotional behavior may be directed at the stimulus that triggered the emotional response in the first place, and may thus feed back onto the environment in an effort to promote homeostasis, survival, and well-being.

We can begin to sketch a neuroanatomic picture of the component structures involved in some of the above processes. There is now good evidence that the amygdala is critical in the triggering of rapid physiological changes in response to emotionally salient stimuli, as illustrated by its role in conditioned responses. The ventromedial prefrontal cortex, which has extensive anatomic connections with the amygdala and is likely to function in tandem with it, is important in the triggering of physiological changes under more complex circumstances in which behavioral decisions cannot be made solely by conditioned associations or by exhaustive reasoning. Thus, the amygdala and the ventromedial prefrontal cortex may be candidates for the roles of structures that trigger emotional responses very rapidly and after more complex processing, respectively. The physiological changes encompassed by an emotional response are represented in a variety of brainstem, midbrain, and cortical structures that map somatic inputs. For instance, insula and other somatosensory-related cortices, especially in

right hemisphere, have been shown to be important for normal recognition of emotions from external stimuli, for normal awareness of one's own body state, and for normal emotional experience.

AFFECTIVE MODULATION OF MEMORY, ATTENTION, AND DECISION MAKING IN ANIMALS

Before our discussion of affect and cognition in humans, we first briefly review what is known about affective modulation of other cognitive processes from studies in animals.

The largest number of animal experiments have focused on the role of affect in associative memory. Lesion studies in rats have shown that structures such as the amygdala are required for the acquisition of conditioned behavioral responses to stimuli that have been previously paired with an intrinsically aversive event, a paradigm called *fear conditioning* (Davis, 1992a; Gewirtz & Davis, 1997; Le Doux, 1996; LeDoux, Cicchetti, Xagoraris, & Romanski, 1990). In such an experiment, an animal is presented with two different types of stimuli: a stimulus that has intrinsic emotional value to the animal (e.g., a rewarding stimulus, such as food; or an aversive stimulus, such as electric shock), and a stimulus that has no intrinsic value to the animal (e.g., the sound of a tone). When these two stimuli are presented together on several occasions, the animal learns that the presence of one can predict the presence of the other: if the tone sounds, it is likely that the shock (or the food) will also occur. This very basic form of associative emotional memory may be an important substrate for more complex forms of learning and motivated behavior. Although there is extensive processing within the amygdala (Pitkanen, Savander, & LeDoux, 1997), one can describe a rough flow of information from higher-order sensory neocortex into the basolateral amygdala, where stimuli can be associated, and then on to various other structures or nuclei within the amygdala, such as the central nucleus, which serves to link amygdala to emotional effector structures (such as the hypothalamus; see Davis, 1992b). Several different neuroanatomic structures in addition to the amygdala have been shown to be involved in emotionally motivated learning, depending on the details of the task: the amygdala (Davis, 1992a; Le Doux, 1996), the ventral striatum (Everitt & Robbins, 1992), and the orbitofrontal cortex (Gaffan, Murray, & Fabre-Thorpe, 1993; Rolls, 1999) all appear to play important roles and are likely to function as components of a distributed,

large-scale neural system for associating stimuli with their rewarding or punishing contingencies.

Although many of the above structures are implicated in processing both reward and punishment, they appear to be disproportionately important along certain dimensions of emotion. For instance, the amygdala specifically mediates behaviors and responses correlated with arousal and stress, especially emotional arousal pertaining to negatively valenced, aversive situations (Davis, 1992b; Goldstein, Rasmusson, Bunney, & Roth, 1996; Kesner, 1992). A direct dissociation on the basis of arousal has been demonstrated in rats: amygdala lesions interfere with avoidance of water that has been paired with electric shock (a highly arousing, unpleasant stimulus), but do not inferfere with avoidance of water that has been made to taste bitter (an unpleasant but not highly arousing stimulus) (Cahill & McGaugh, 1990). The amygdala thus plays a role in the acquisition of information during emotionally arousing situations, and perhaps especially during situations that are both arousing and unpleasant. Animal studies suggest that the amygdala circuitry underlying the processing of aversive stimuli depends on the central nucleus of the amygdala, whereas processing of stimuli that are rewarding appears to depend on projections from the basolateral nucleus of the amygdala to frontal cortex and ventral striatum.

The detailed role that the amygdala plays in the emotional modulation of other aspects of cognition, such as attention and memory, are rather complicated. Although the human studies we review below speak of "the amygdala" as if it were a homogeneous structure, it is important to bear in mind that the amygdala is in fact a complex collection of nuclei that all subserve somewhat different functions (Swanson & Petrovich, 1998). Furthermore, the amygdala is merely one nodal structure in a very distributed network that can modulate cognition on the basis of affect, and there are multiple neurotransmitter systems within these structures that can carry out different functional roles. The complexity of the systems is illustrated in the effects of emotional processing on modulating motivated learning in animals. A large number of studies (Cahill & McGaugh, 1996; McGaugh, 1989; McGaugh, Cahill, & Roozendaal, 1996) have shown that aversively motivated learning can be modulated by multiple neurotransmitter systems acting within the amygdala. For instance, direct post-training injections into the amygdala of a variety of drugs that modulate GABAergic, noradrenergic, or opiate-mediated neurotransmission influence long-term memory for inhibitory avoidance training (Brioni, Nagahara, & McGaugh, 1989; Gallagher, Kapp, Pascoe, & Rapp, 1981). Recent data suggest that the amygdala can influence memory by modulating consolidation that actually

takes place within other brain structures, such as the hippocampus and basal ganglia. In one set of experiments, reversible pharmacologic lesions of the amygdala with lidocaine showed that the amygdala-mediated enhancement of different types of memory depends on the hippocampus (for memory in a spatial task) or the caudate nucleus (for memory in a cued task). Increasing neural activity within the amygdala by injections of d-amphetamine directly into the amygdala immediately after training enhanced performance on both spatial and cued water-maze tasks, and lidocaine injections into either hippocampus or caudate were found to block the effects of d-amphetamine amygdala injections on the respective task, but leave the amygdala's enhancement of the other task unaffected (Packard, Cahill, & McGaugh, 1994; Packard & leather, 1996). Multiple neurotransmitter systems within the amygdala thus can modulate memory mechanisms in a variety of other structures, providing one mechanism for how changes in neuromodulatory transmitters induced by affective states could also modulate memory neurochemically (see Cahill & McGaugh, 1996, 1998; McGaugh, 1989; McGaugh et al., 1996, for reviews).

In addition to its role in emotional memory, the amygdala, together with a collection of nuclei termed the *basal forebrain,* have been shown to make critical contributions to the effect that emotion has on attentional processes. The amygdala's role in attentional processes has also been investigated in several recent experiments in rats (Holland & Gallagher, 1999). One component of attention, orienting behavior toward cues that have become associated with rewarding contingencies, has been found to rely on a circuit involving the central nucleus of the amygdala and its connections with the substantia nigra and the dorsal striatum (Han, McMahan, Holland, & Gallagher, 1997). Another important component of attention, increased allocation of processing resources toward novel or surprising situations, appears to depend on the integrity of the central nucleus of the amygdala and its connections with cholinergic neurons in the substantia innominata and nucleus basalis, structures in the basal forebrain. Thus, the amygdala could modulate cholinergic neuromodulatory functions of the basal forebrain nuclei, and consequently modulate attention, vigilance, signal-to-noise, and other aspects of information processing that depend on cholinergic modulation of cognition (Everitt & Robbins, 1997). Although this latter function has been studied in animals specifically as an increased ability to learn emotional associations that are unexpected, a more general role has been proposed in humans in regard to general vigilance and in attention to stimuli about which more information could be obtained (Whalen, 1999). Through circuits including components of amygdala, striatum, and basal

forebrain, emotion may thus help to select particular aspects of the stimulus environment for disproportionate allocation of cognitive processing resources; namely, an organism should be designed to preferentially process information about those aspects of its environment that are most salient to its immediate survival and well-being.

Finally, it is clear that structures in close association with the amygdala, notably the bed nucleus of the stria terminalis, are necessary for more prolonged emotional states that can influence information processing in a global and less stimulus-driven fashion. In rats, the bed nucleus of the stria terminalis, together with nuclei within the amygdala proper, appear to be involved in anxiety rather than in fear; moreover, specific neuropeptides, such as corticotropin releasing hormone, have dramatic effects specifically on the bed nucleus of the stria terminalis and on anxiety, but not on fear (Davis, 1992a,b, 1997; Davis, Walker, & Lee, 1997). Although the detailed neural structures involved in fear and anxiety remain to be fully elucidated in humans, the findings from animal studies point toward anatomically and pharmacologically dissociable systems for fear and anxiety, and suggest particular avenues for further research as well as for therapeutic intervention (Davis, 1992b).

AFFECT MODULATES DECLARATIVE MEMORY

The role of the amygdala in fear conditioning as discussed previously is consonant with recent data from humans: subjects with amygdala lesions fail to show conditioned skin-conductance response to stimuli that have been paired with an aversive, loud noise (Bechara et al., 1995; LaBar, LeDoux, Spencer, & Phelps, 1995), and the amygdala, together with other limbic structures, is activated during emotional conditioning paradigms in functional imaging studies (Buechel, Morris, Dolan, & Friston, 1998; LaBar, Gatenby, Gore, LeDoux, & Phelps, 1998). However, the amygdala's most interesting contribution to memory in humans is in the modulation of what we remember as conscious facts—in the modulation of declarative knowledge.

Common experience, as well as a large literature from human cognitive psychology, leave little doubt that declarative memory and emotion are intimately connected (Schacter, 1996). We often remember emotional episodes in our own lives with exceptional vividness and detail. A particularly striking example are so-called flashbulb memories: emotional events, such as president Kennedy's assassination or the explosion of the

Challenger space shuttle, are frequently remembered as highly detailed images (Winograd & Neisser, 1992).

Naturalistic studies of memory for emotional events have suggested that relevant, salient information can often be enhanced by emotional arousal, but that information can also be distorted or suppressed. For instance, subjects giving eyewitness testimony often remember details about a knife or gun while forgetting other information about the scene (the so-called weapon focus effect; Loftus, 1979; Maass, & Koehnken, 1989; Steblay, 1992). Recent studies have attempted to simulate the naturalistic situation in the laboratory. In one series of studies, subjects were shown 12 slides accompanied by a narrative that together told a story (the "Reisberg task"; Burke, Heuer, & Reisberg, 1992; Heuer & Reisberg, 1990). Parts of the story were highly emotional. Subjects remembered the most detail about those parts of the story that were the most emotional. The data provided support for a theory, whereby emotion facilitates memory for information about the most salient features of a stimulus, compared to emotionally neutral stimuli (Burke et al., 1992; Heuer & Reisberg, 1990; Reisberg & Heuer, 1992).

Studies have shown that stimuli are remembered better the more emotionally arousing they are (Bradley, Greenwald, Petry, & Lang, 1992; Hamann, Cahill, & Squire, 1997b). These findings have led to the hypothesis that it is emotional arousal, and not valence, that is the major factor contributing to how well material is encoded into declarative memory. This hypothesis has been directly tested by manipulating arousal, either pharmacologically (Cahill, Prins, Weber, & McGaugh, 1994; O'Carroll, Drysdale, Cahill, Shajahan, & Ebmeier, 1998) or through the use of a specific context (Cahill & McGaugh, 1995) in normal human subjects. Both these manipulations showed that increased emotional arousal resulted in better encoding of material into declarative long-term memory, as assessed by subsequent recall.

The amygdala is the primary structure in humans that has been examined specifically in regard to emotional memory. Emotional memory in humans is impaired by amygdala lesions (Adolphs, Cahill, Schul, & Babinsky, 1997; Cahill, Babinsky, Markowitsch, & McGaugh, 1995; Phelps, LaBar, Anderson, O'Connor et al., 1998), but is not disproportionately impaired by lesions of other structures that can cause amnesia, such as the hippocampus (Hamann, Cahill, McGaugh, & Squire, 1997a; Hamann et al., 1997b). Studies of subjects with bilateral amygdala lesions found specific impairments in declarative memory for emotional material, despite normal declarative memory for neutral material (Fig. 2.1). These studies showed

that the human amygdala enhances the encoding of material into declarative memory when the subject matter is unpleasant and emotionally arousing. Importantly, memory for neutral material was essentially normal in these patients: they just failed to show the normal facilitation when the subject matter was emotional. These findings from lesion studies are, in principle, consistent with two different interpretations. The amygdala may mediate the enhancing effects of emotion on the encoding of information into long-term memory, or it might mediate the enhancing effects of emotion on the subsequent retrieval of such information. Because emotional arousal of the subjects most likely occurred primarily when they first saw the stimuli during encoding, the former possibility is the most plausible. A confirmation has come from studies in normal subjects that used functional imaging during encoding.

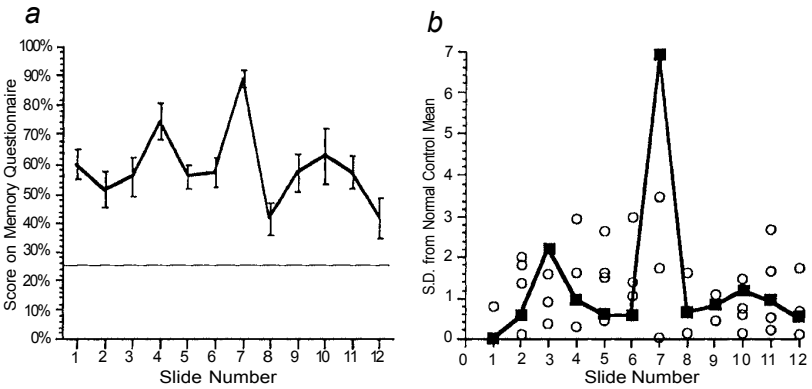

FIG. 2.1. Bilateral damage to the human amygdala impairs declarative memory for emotionally arousing material. Data are from seven normal control subjects, six brain-damaged control subjects who did not have damage to the amygdala, and from subject SM, who has complete bilateral damage to the amygdala. Subjects were shown a series of 12 slides that varied in emotional arousal. Slide 7 was the most arousing slide, showing surgically reattached legs of a car-crash victim, (a) Data from normal controls, showing memory score on a questionnaire about each slide. Chance is at 25% *(dotted line),* (b) Data from brain-damaged controls *(open circles)* and from subject SM *(black squares, solid lines)* plotted as differences from the normal control data. SM differed most from controls on the most emotional slide, an impairment not shown by any of the brain-damaged control subjects. The data suggest that the amygdala is important not for encoding memory in general, but specifically for enhancing the encoding of material into long-term declarative memory when such material is emotionally highly arousing. Modified from Adolphs, R., Cahill, L., Schul, R., & Babinsky, R. (1997). impaired declarative memory for emotional material following bilateral amygdala damage in humans. *Learning and Memory,* 4, 291–300. Copyright © 1997, Cold Spring Harbor Laboratory Press.

A specific role for the amygdala in acquisition of declarative knowledge regarding emotionally arousing stimuli has been reported by recent imaging studies (Cahill et al., 1996; Canli, Zhao, Desmond, Glover, & Gabrieli, 1998; Hamann, Ely, Grafton, & Kilts, 1999). In one study (Cahill et al., 1996), amygdala activation at the time arousing unpleasant emotional stimuli (horror movies) were encoded into memory correlated significantly with how well the stimuli could subsequently be recalled; the authors argued that the amygdala is activated during the encoding of emotionally arousing material into declarative long-term memory. In another study (Hamann et al., 1999), amygdala activation correlated with encoding of either pleasant or unpleasant pictures into memory, as assessed by later recognition of the same stimuli. Taken together, both these imaging studies, as well as the findings from lesion studies, argue that the amygdala plays a critical role in modulating the encoding and the consolidation of knowledge during emotionally arousing circumstances.

It is interesting in this context to note that the human amygdala is also activated during REM sleep (Maquet et al., 1996), a finding consistent with a role for the amygdala in emotional memory because REM sleep typically involves emotional experiences and is likely to serve a function in consolidating emotional memory (Smith & Lapp, 1991). There are now several studies in humans that have confirmed a role for various stages of sleep in reorganizing and consolidating memory (Stickgold, 1999), consistent with single-unit neurophysiological findings from rats that have shown reprocessing of spatial information in the hippocampus during sleep (Wilson & McNaughton, 1994). These findings make it plausible that some of the amygdala's modulation of emotional memory occurs during sleep, and perhaps especially REM sleep.

All the previously discussed findings suggest a role for the amygdala in modulating the encoding and consolidation of long-term memory on the basis of the affective response and experience associated with the material that is being encoded. Of course, structures additional to the amygdala participate in such a process. Also, it remains to be explored how emotion may modulate memory retrieval, an issue that has received considerable attention from cognitive psychologists studying state-dependent memory (Eich, 1995), but of which little is known regarding the neuroanatomic substrates.

A further question concerns the precise mechanisms by which emotional response can modulate declarative memory encoding. It is known that peripheral catecholamines modulate memory in part by neurotransmission of body-state information to central structures, such as the amygdala:

vagotomized rats show a block of the normal potentiation of memory by peripheral catecholamines (Williams & Jensen, 1991), and direct stimulation of vagal afferents modulates memory (Clark et al., 1996). A recent study replicated this finding in humans who had vagal nerve stimulators implanted. Vagal nerve stimulation enhanced the acquisition and consolidation of memory (Clark, Naritoku, Smith, Browning, & Jensen, 1999), providing support for the idea that body-state information modulates cognitive processes, such as memory. The finding suggests that emotional responses to stimuli can modulate cognition not only directly, but also indirectly via the perception of body-state changes that have been triggered by the emotional response. In effect, the finding demonstrates that an emotional physiological response in the body has a direct effect on brain function. We take up this issue in more detail next, with respect to the effect of emotion on decision making.

AFFECT MODULATES DECISION MAKING

As with memory, common experience already suggests that our decisions are strongly influenced by affect. This has often been conceptualized as a case of emotion impairing decision making, but recent treatments have stressed the ecologically adaptive value of an emotional bias in guiding decision making under uncertainty (Damasio, 1994). Although, as one might predict, the amygdala also, at a basic level, participates in the modulation of decision making by emotion (e.g., Bechara, Damasio, Damasio, & Lee, 1999), the best studied structure in this regard is the ventromedial (VM) frontal cortex.

The VM frontal cortices have intimate connections with the amygdala, both directly and via the dorsomedial thalamus, and all these three structures appear to function as components of a distributed neural system for processing the rewarding or punishing contingencies of stimuli in relation to the animal's behavior (Gaffan & Murray, 1990; Gaffan et al., 1993; Schoenbaum, Chiba, & Gallagher, 1998; Tremblay & Schultz, 1999). In humans, the VM frontal cortex stands out as a structure indispensable to making decisions in complex environments, but not necessary for many other cognitive functions, such as those that contribute to working memory or general intelligence.

Clues about the functions of the VM frontal cortex date back to the late 1800s, as shown in the case of Phineas Gage. Gage suffered from a dramatic accident in which an iron rod was shot through his head as a result of an explosion. The injury resulted in a large bilateral lesion of

VM frontal cortex (Damasio, Grabowski, Frank, Galaburda, & Damasio, 1994). It was surprising that he survived at all, but also intriguing was his changed personality after the accident. Whereas Gage had been a diligent, reliable, polite, and socially adept person before his accident, he had subsequently become uncaring, profane, and socially inappropriate in his conduct. This change in his personality remained a mystery until it could be interpreted in light of similar such patients in modern day: Like Gage, other subjects with bilateral damage to the VM frontal lobes show a severely impaired ability to function in society, despite an entirely normal profile on standard neuropsychological measures, such as IQ, language, perception, and memory.

The role of the VM prefrontal cortex in decision making has been explored in a series of studies by Antoine Bechara, who used a task in which subjects had to gamble in order to win money. As with gambling in real life, the task involved probabilistic contingencies that required subjects to make choices based on incomplete information. Normal subjects learned to maximize their profits on the task by building a representation of the statistical contingencies gleaned from prior experiences: In the long run, certain choices tend to pay off better than others. The key ingredient that distinguished the task of Bechara from other tasks of probabilistic reasoning is that subjects discriminated choices by feeling—they developed hunches that certain choices were better than others, and these hunches could be measured both by asking subjects verbally and by measuring autonomic correlates of emotional arousal, such as skin conductance response. Subjects with damage to the VM frontal cortex failed this task (Bechara, Damasio, Damasio, & Anderson, 1994), and they failed it precisely because they were unable to represent choice bias in the form of an emotional hunch (Bechara, Damasio, Tranel, & Damasio, 1997). Not only did subjects with VM frontal damage make poor choices on the task, they also acquired neither any subjective feeling regarding their choices (Bechara et al., 1997), nor any anticipatory autonomic changes (Bechara, Tranel, Damasio, & Damasio, 1996) (Fig. 2.2).

These findings are consonant with prior reports that subjects with VM frontal lobe damage do not trigger a normal emotional response to stimuli, including socially relevant stimuli (Damasio, Tranel, & Damasio, 1990), and support a specific hypothesis that has been put forth by A.R.Damasio (1994, 1995, 1996): the somatic marker hypothesis. According to this hypothesis, the VM frontal cortex is a critical component of the neural systems by which we acquire, represent, and retrieve the values of our actions. This mechanism includes the generation of somatic states, or representations

of somatic states, that correspond to the anticipated future outcome of decisions. Such a mechanism may be of special importance in the social domain, where the enormous complexity of the decision space precludes an exhaustive analysis.

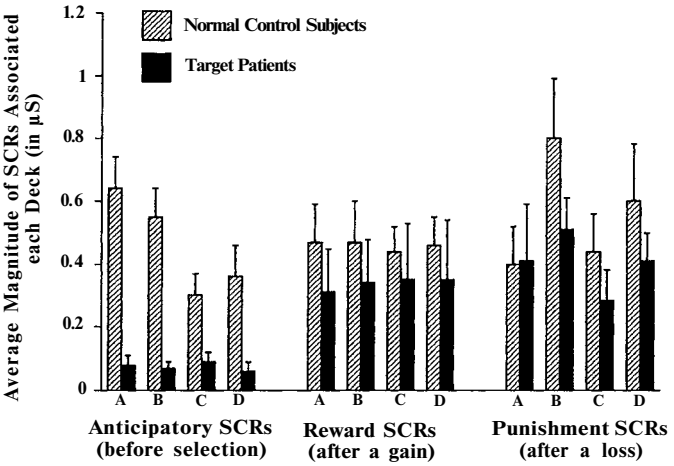

FIG. 2.2. Impaired ability to trigger emotional states during decision making following damage to ventromedial prefrontal cortex. Shown are mean (and SD) skin conductance responses from seven subjects with bilateral damage to the ventromedial (VM) prefrontal cortex *(solid block bars)* and from 12 normal controls *(striped bars)*. Subjects participated in a gambling task (Bechara et al., 1996) in which they were asked to choose from four decks of cards (indicated as A, B, C, D on the x-axis) that were associated with different probabilities of winning and losing money. Decks A and B had the highest risk of losing money, and consequently normal subjects showed large changes in skin-conductance response (SCR) in anticipation as they chose cards from these decks, but subjects with damage to VM prefrontal cortex showed no such anticipatory index of emotional state (*left;* anticipatory SCRs). The discrepancy between control and target subjects became considerably less when examining their SCRs subsequent to having chosen a card from a deck as they were told how much money they had won or lost. Similar to controls, subjects with VM frontal damage did trigger SCRs when they received this news (*middle* and *right:* reward and punishment SCRs). The data thus indicate that the defect following damage to VM frontal lobe is not an inability to respond to reward or punishment per se, but to use the emotional state elicited by reward or punishment to trigger anticipation of future reward or punishment under similar circumstances, and to thus guide decision making on the basis of prior emotional experience. Modified from Bechara, A., Tranel, D., Damasio, H., & Damasio, A.R. (1996). Failure to respond autonomically to anticipated future outcomes following damage to prefrontal cortex. *Cerebral Cortex,* 6, 215–225. Copyright © 1996, Oxford University Press.

AFFECT AND SOCIAL JUDGMENT

Affect and social behavior have long been believed to be related. Our interactions with other people typically involve affective reactions. The close relation between affect and social cognition was already exemplified in the previous section, in which we pointed out that emotion may be disproportionately important to guide decision making in the social domain.

It should come as no surprise then that many of the neural structures involved in processing emotion also play a key role in regulating social behavior. A recent set of studies of the human amygdala bear this out. Studies that have used functional neuroimaging in normal subjects (Breiter et al., 1996; Morris et al., 1996) and studies that have examined patients with damage to the amygdala (Adolphs, Tranel, Damasio, & Damasio, 1994; Adolphs et al., 1999; Young et al., 1995) provide something of a consensus that the amygdala is critical to the recognition of emotions from facial expressions, specifically certain negative emotions, especially fear (Adolphs et al., 1995; Broks et al., 1998; Calder et al., 1996). The findings have been broadly consonant with the amygdala's contribution to social behavior that was suggested by earlier lesion studies in animals (Kluver & Bucy, 1939; Weiskrantz, 1956), as well as with the large number of studies that have investigated the amygdala's role in fear conditioning (Davis, 1992a; Le Doux, 1996). All these different threads of research point to a disproportionately important role for the amygdala in processing stimuli related to danger and threat, both in the environment in general and in the social environment in particular. Given these findings, one might expect that the amygdala would make important contributions also to higher-level social cognition in humans, perhaps especially to those aspects of it that rely on recognizing social information from faces.

We investigated subjects' ability to judge how trustworthy or how approachable other people looked from perceiving their faces. In our study (Adolphs, Tranel, & Damasio, 1998), we found that three subjects who had bilateral amygdala damage all shared the same pattern of impairment: they judged to be abnormally trustworthy and approachable the faces of those people who are normally judged to look the most untrustworthy or most unapproachable (Fig. 2.3). Although the subjects with amygdala damage showed a general positive bias in judging all faces, they showed a disproportionate impairment when judging those faces normally given the most negative ratings. The amygdala's role in processing stimuli related to

potential threat or danger thus appears to extend to the complex judgments on the basis of which we regulate our social behavior.

An issue of further interest is the specificity of the impairment to faces. Follow-up studies revealed that bilateral amygdala damage also impaired judgments for the preferences of nonsocial visual stimuli, such as color patterns or landscapes, although the effect was not as large. In this study (Adolphs & Tranel, 1999), subjects with amygdala damage liked pictures of nonsocial stimuli more than did control subjects. Thus, the amygdala's role does not appear to be entirely restricted to stimuli in the social domain, but may encompass a more general function that is of disproportionate importance to social cognition. A further experiment assessed social judgments that were made about other people on the basis of written descriptions of them. Judgments about people from such lexical stimuli were not impaired by amygdala damage (Adolphs et al., 1998). This latter finding suggests that the lexical stimuli provided sufficient explicit information such that normal task performance could result from reasoning strategies that did not necessarily require the amygdala. However, it is worth noting that there is evidence for the amygdala's importance in processing emotional stimuli that are lexical when such stimuli signal potential threat, danger, or other emotional arousal (Isenberg et al., 1999; Adolphs, Russell, & Tranel, 1999).

One would like also to extend the previously mentioned line of investigations to additional types of stimuli and additional types of social information that can be gleaned from such stimuli. Andrea Heberlein, a graduate student working with us, has begun such an investigation using visual motion cues to provide information about biological and psychological categories. In one experiment, subjects were shown a short video that depicts three geometric shapes moving on a plain, white background (Heider & Simmel, 1944). Although visual motion is the only available cue in this experiment, normal subjects have no difficulty interpreting the motion of the shapes in terms of social categories: The shapes are attributed psychological states, such as goals, beliefs, desires, and emotions, on the basis of their relative motion. By contrast, a subject with selective bilateral amygdala damage did not make such automatic attributions (Heberlein et al., 1998). When shown the same stimulus, she described it in purely geometric terms, lacking the normal, automatic social interpretation. This finding suggests that our interpretation of the world around us is influenced by mechanisms for assigning emotional and social value, an ability that is clearly essential for survival in a complex social environment, but one that may also explain our tendency to anthropomorphize when misapplied.

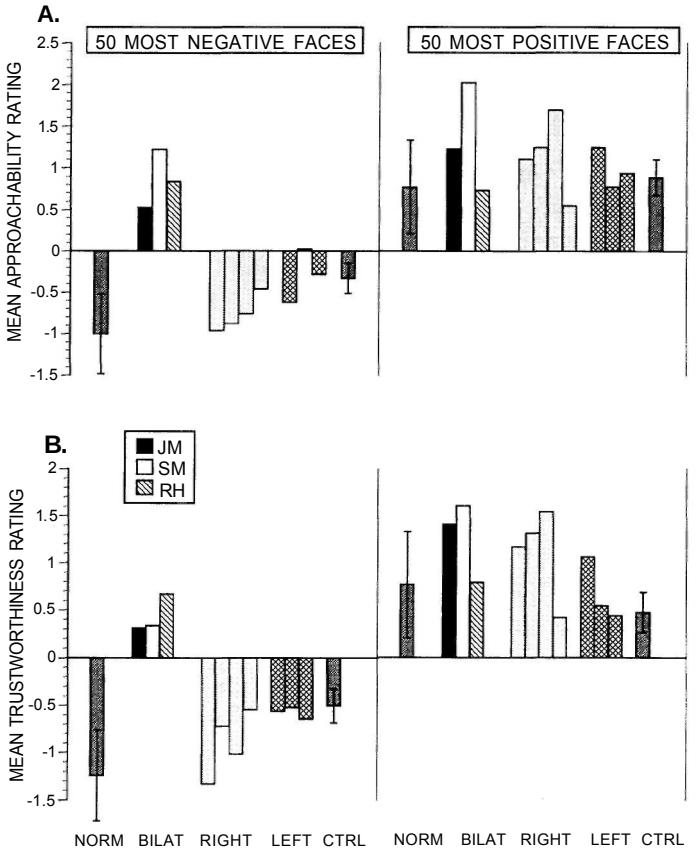

FIG. 2.3. Bilateral damage to the human amygdala impairs social judgment for faces. Shown are mean judgments of (a) approachability and (b) trustworthiness of the faces of 100 unfamiliar people. Data are broken down into those obtained from the 50 faces that received the most negative *(left)* and most positive *(right)* mean ratings from normal controls on each of these attributes. Each face was judged on a scale of −3 (very unapproachable or untrustworthy) to +3 (very approachable or trustworthy). Means and SD are shown for data from 46 normal controls (NORM). Individual means are shown for each of three subjects with bilateral amygdala damage (BILAT), four subjects with unilateral right (RIGHT), and three with unilateral left (LEFT) amygdala damage. Means and SEM are shown for seven brain-damaged controls with no damage to the amygdala (CTRL). Subjects with bilateral amygdala damage differed from all other groups in that they gave abnormally positive ratings to those faces that normally receive the most negative ratings. Modified from Adolphs, R., Tranel, D., & Damasio, A.R. (1998). The human amygdala in social judgment. *Nature, 393,* 470–474. Copyright © 1998, MacMillan Press.

The previously mentioned set of findings supports a role for the amygdala in mediating affective influences on social decision making and social judgment, in addition to a more general role in modulating cognition that may not be specific to the social domain. Similarly, the VM frontal cortices play a role in linking emotional associations to behavioral contingencies that go beyond social decision making. This raises the question of the specificity of affect in regulating social cognition, as compared with cognition in general. We believe that affect plays a disproportionately important role in social cognition, and it seems likely that the highly differentiated affective states of humans evolved to regulate social behavior (see Panksepp, 1998, for a detailed review of some of the neuroscientific support for this). In fact, a whole set of so-called social emotions pertain specifically to social situations (e.g., guilt, embarrassment, shyness, jealousy, shame). One might predict a need for highly differentiated affective responses precisely in guiding cognition and behavior in those domains with the greatest complexity, and surely the social domain is the most complex of all.

CONCLUSIONS

Although, for the sake of brevity, we have highlighted only a few neural structures about which we have clearest experimental evidence for a role in affective modulation of cognition, it should be clear that we envision a highly distributed system consisting of many distinct neuroanatomic components. Such a system would include the whole set of neural structures involved in representing somatic states, from brain stem nuclei, like the nucleus solitarius and the parabrachial nucleus, to neocortical structures, such as somatosensory cortices and insula. In fact, there is good evidence for the role of these structures in both emotion and social cognition. In addition to such representational structures, there are key nodal structures that link affect, or its somatic representation, to cognition and behavior—we have concentrated on the amygdala and VM frontal cortices. However, amygdala and VM frontal cortices are intimately connected with other structures that directly influence cognition: the basal forebrain cholinergic system, which contributes importantly to attention, as well as various neuromodulatory brain stem nuclei and dopaminergic nuclei in ventral tegmentum.

Together, all these neuroanatomic structures and neurochemical systems are in a position to modulate information processing globally in neocortical circuits that subserve perception, recognition, and memory consolidation. There is clear evidence from recent functional imaging studies that emotional states modulate activity in the neocortex globally: many higher sensory

and association neocortices show a decrease in activation during highly emotional states, whereas other regions, such as the VM prefrontal cortices, show an increase (Drevets & Raichle, 1998). Likewise, there is evidence that mood states that are known to engage frontal cortex predominantly on either the left or the right side of the brain (Davidson & Hugdahl, 1995) in turn influence the cognitive functions subserved by that side of the frontal lobe. Thus, positive affect, which engages the left frontal cortex more than the right, facilitates verbal fluency, also a function attributable to the left frontal cortices. By contrast, negative affect, which engages the right frontal cortex more than the left, facilitates figural fluency, a function attributable to the right frontal cortices (Bartolic, Basso, Schefft, Glauser, & Titanic-Schefft, 1999). This affect-state dependence of cognition and its correlation with identified neural structures provide further evidence for the inseparable relation between emotion and other aspects of cognition. Our everyday experience also clearly shows that affect influences essentially all other aspects of cognitive functioning, including memory, attention, and decision making.

Despite such a global interrelation between affect and cognition, it is also the case that particular affective processes disproportionately influence certain types of knowledge structures. For example, as we reviewed previously, social judgments depend on affective circuitry involving the amygdala when such judgments are made on the basis of perceiving other people's faces, but not when they are made on the basis of explicit information provided in language. However, the reasons for this difference should be apparent: explicit, lexical information can bypass substantial information processing and can generate social judgment without requiring some of the intermediate representations that would be required in the case of faces. Subjects with bilateral damage to the amygdala are unable to generate the knowledge from seeing the person's face that person should be avoided; however, if told explicitly that the person is bad or dangerous to approach in some way, the same subjects have no difficulty in using this already supplied knowledge to guide their judgment.

In closing, it is worth considering the evolution of the relationship between affect and cognition, a topic already skirted at the beginning of this chapter. We consider affective processing to be an evolutionary antecedent to more complex forms of information processing; but higher cognition requires the guidance provided by affective processing (Damasio, 1994). The key ingredient offered by affect is a representation that incorporates the value of a stimulus or of an action to the organism. Biological value would provide an important bias not only to relatively fast, hard-wired

stimulus-response processing in situations in which there is a premium on survival (running away from a bear), but would also permeate higher cognition. A key insight is that cognition must include not only representations of external sensory stimuli, but also representations of the organism that is perceiving those stimuli, including a representation of the biological value of those stimuli to the organism.

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