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Affective Style, Psychopathology, and Resilience: Brain Mechanisms and Plasticity

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The brain circuitry underlying emotion includes several territories of the prefrontal cortex (PFC), the amygdala, hippocampus, anterior cingulate, and related structures. In general, the PFC represents emotion in the absence of immediately present incentives and thus plays a crucial role in the anticipation of the future affective consequences of action, as well as in the persistence of emotion following the offset of an elicitor. The functions of the other structures in this circuit are also considered. Individual differences in this circuitry are reviewed, with an emphasis on asymmetries within the PFC and activation of the amygdala as 2 key components of affective style. These individual differences are related to both behavioral and biological variables associated with affective style and emotion regulation. Plasticity in this circuitry and its implications for transforming emotion and cultivating positive affect and resilience are considered.

Editor's Note

Richard J. Davidson received the Award for Distinguished Scientific Contributions. Award winners are invited to deliver an award address at the APA's annual convention. This award address was delivered at the 108th annual meeting, held August 4–8, 2000, in Washington, DC. Articles based on award addresses are not peer reviewed, as they are the expression of the winners' reflections on the occasion of receiving an award.

One of the most salient characteristics of emotion is the extraordinary heterogeneity in how different individuals respond to the same emotionally provocative challenge. Whereas individual differences can be found at many levels of phylogeny, they are particularly pronounced in primates and probably are most extreme in humans. A number of evolutionary theorists have speculated on the adaptive significance of such individual differences (Wilson, 1994). Although these arguments have never been applied to the domain of emotion and affective style, it is not difficult to develop hypotheses about how such differences might provide advantages to individuals living in groups. However, rather than focus on the distal causes of such individual differences, which are so difficult to subject to rigorous test, I wish only to call attention to the possibility that variability in characteristics such as "fearfulness" or "cheerfulness" might provide some adaptive benefit to individuals living together in groups. Instead, this article examines the proximal mechanisms that underlie such individual differences and describes the nomological network of associations that have been associated with these characteristics. The relevance of affective style for understanding mood and anxiety disorders is illustrated. Plasticity in the underlying brain circuitry that instantiates affective style is described, and its role in promoting resilience is considered.

Affective style refers to consistent individual differences in emotional reactivity and regulation (see Davidson, 1998a; Davidson, Jackson, & Kalin, 2000; Davidson, Putnam, & Larson, 2000). It is a phrase that is meant to capture a broad array of processes that either singly or in combination modulate an individual's response to emotional challenges, dispositional mood, and affect-relevant cognitive processes. *Affective style* can refer to valence-specific features of emotional reactivity or mood or to discrete-emotion-specific features. Both levels of analysis are equally valid, and the choice of level should be dictated by the question posed.

Rapid developments in our understanding of emotion, mood, and affective style have come from the study of the neural substrates of these phenomena. The identification of the brain circuitry responsible for different aspects of affective processing has helped to parse the domain of emotion into more elementary constituents in a manner similar to that found in cognitive neuroscience, where an appeal to the brain has facilitated the rapid development of theory and data on the subcomponents of various cognitive processes (see, e.g., Kosslyn & Koenig, 1992).

This address highlights some of the advances that have been made in our understanding of the brain mechanisms that underlie affective style. These advances have emerged from three major sources: studies of patients with discrete lesions of the brain, neuroimaging studies of normal individuals, and studies of pathologies of brain function in patients with various psychiatric and neurological disorders that involve abnormalities in emotion.

Both lesion and neuroimaging studies provide information primarily on the "where" question, that is, where in the brain are computations related to specific aspects of affective processing occurring? It is important at the outset to consider both the utility of knowing where and how such information can provide insight into the "how" question, that is, how might a particular chunck of tissue instantiate a specific process that is essential to affective style? The brain sciences are now replete with information on the essential nature of specific types of information processing in different regions of the brain. For example, there is evidence to suggest that the dorsolateral prefrontal cortex (DLPFC) is important for maintaining a representation of information on-line in the absence of immediate cues. The neurophysiological basis of this type of information processing is being actively studied in the animal laboratory (e.g., Goldman-Rakic, 1996, 2000). If this region of the brain is activated at certain times in the stream of affective information processing, we can develop hypotheses on the basis of extant work about what this territory of prefrontal cortex (PFC) might be doing during the affective behavior and how it might be doing it.

Conceptual and Methodological Considerations in the Study of Affective Style

Mood and anxiety disorders are generally conceptualized as being caused or at least accompanied by dysfunctions of emotion. However, what specific affective process is dysfunctional is rarely, if ever, delineated, and nosological schemes for categorizing these disorders do not rely on the specific nature of the affective dysfunction in question but rather are based on phenomenological description. Research in my laboratory over the past 15 years has been predicated on the view that more meaningful and rapid progress in understanding the brain bases of mood and anxiety disorders can be achieved if we move to an intermediate level of description that penetrates below the categorical, phenomenologically based classifications of the *Diagnostic and Statistical Manual of Mental Disorders* (American Psychiatric Association, 1994) and seeks to characterize the specific nature of the affective styles that are associated with vulnerability to these forms of psychopathology.

Many of the parameters of affective style, such as the threshold to respond, magnitude of response, latency to peak of response, and recovery function, are features that are often opaque to conscious report, though they may influence the subjective experience of emotion. These parameters of responding can be measured in many different response systems, including both central and peripheral systems. For example, magnitude of response can be measured in a peripheral measure, such as the emotion-modulated startle (Lang, 1995), or in a central measure, such as activation in the amygdala assessed with functional magnetic resonance imaging (fMRI). The extent to which coherence across response systems in these parameters is present has not yet been systematically addressed. In previous work, we have argued that variations in some of these parameters in particular response systems are especially relevant to vulnerability to mood, anxiety, and other disorders (e.g., Davidson, Putnam, & Larson, 2000) and also to resilience (Davidson et al., 2000). One of the important developments in emotion research, in general and in affective neuroscience, in particular, is the capacity to objectively measure these parameters of responding. For example, in several studies, we have used the emotion-modulated startle to capture the time course of valence-specific emotion responding (Jackson, Malmstadt, Larson, & Davidson, 2000; Larson, Sutton, & Davidson, 1998). The startle reflex is controlled by a brain stem circuit that is influenced by activity in forebrain structures. Davis (1992) elegantly dissected the circuitry through which the magnitude of this reflex is modulated during the arousal of fear in rodents. He demonstrated that it is via a descending pathway from the central nucleus of the amygdala to the nucleus pontine reticularis in the brain stem that the magnitude of startle is enhanced in response to a conditioned fear cue. Lesions of the central nucleus of the amygdala abolish the fear potentiation of the startle but do not affect the magnitude of the baseline startle. Vrana, Spence, and Lang (1988) were the first to systematically show that in humans, the same basic phenomenon can be produced. They took advantage of the fact that brief acoustic noise bursts produce the eyeblink component of the startle and little else, thus enabling their presentation as innocuous stimuli in the background. By measuring electromyographic activity from the orbicularis oculi muscle with two miniature electrodes under one eye, they were able to quantify the strength of the blink response and show that the magnitude of the blink was greater when participants were presented with unpleasant pictures in the foreground, compared with the presentation of neutral pictures. Moreover, when participants were exposed to positive stimuli, the magnitude of startle was actually attenuated relative to a neutral condition (Vrana et al., 1988). This same basic effect has now been reported with many different types of foreground stimuli in several modalities (see Lang, 1995, for review).

We have exploited the emotion-modulated startle to begin to characterize the time course of affective responding, or what I have referred to as *affective chronometry* (Davidson, 1998a). By inserting acoustic noise probes at different latencies before and after a critical emotional stimulus is presented, both the anticipatory limb as well as the recovery limb of the response can be measured. And by using paradigms in the MRI scanner that were first studied in the psychophysiology laboratory, the neural circuitry underlying the different phases of affective processing can be interrogated with fMRI. Our current work in this area has emphasized the importance of the recovery function after negative events for vulnerability to certain forms of psychopathology as well as for resilience. We have argued that the failure to rapidly

recover after a negative event can be a crucial ingredient of vulnerability to both anxiety and mood disorders, particularly when such a style is combined with frequent exposure to negative events over a sustained period of time. The failure to adequately recover would result in sustained elevations in multiple systems that are activated in response to negative events. On the other hand, the capacity for rapid recovery after negative events may define an important ingredient of resilience. We have defined *resilience* as the maintenance of high levels of positive affect and well-being in the face of significant adversity. It is not that resilient individuals never experience negative affect, but rather that the negative affect does not persist. Such individuals are able to profit from the information provided by the negative affect, and their capacity for meaning making in response to such events may be part and parcel of their ability to show rapid decrements in various biological systems after exposure to a negative or stressful event (see, e.g., Geise-Davis & Spiegel, in press).

Neural Substrates of Emotion and Affective Style

In this section, a brief overview is provided of core components of the circuitry that instantiates some important aspects of emotion and affective style, with an emphasis on the PFC and the amygdala. It is not meant to be an exhaustive review but rather will present selected highlights to illustrate some of the key advances that have been made in the recent past.

Emotion and affective style is governed by a circuit that includes the following structures and quite likely others as well: DLPFC, ventromedial PFC (vmPFC), orbitofrontal cortex (OFC), amygdala, hippocampus, anterior cingulate cortex (ACC), and insular cortex. It is argued that different subprocesses are instantiated in each of these structures and that they normally work together to process and generate emotional information and emotional behavior.

Prefrontal Cortex

A large corpus of data at both the animal and human levels implicate various sectors of the PFC in emotion. The PFC is not a homogeneous zone of tissue but rather has been differentiated on the basis of both cytoarchitectonic as well as functional considerations. The three subdivisions of the primate PFC that have been consistently distinguished are the DLPFC, vmPFC, and OFC. In addition, there appear to be important functional differences between the left and right sides within some of these sectors.

The case for the differential importance of left and right PFC sectors for emotional processing was first made systematically in a series of studies on patients with unilateral cortical damage (Gainotti, 1972; Robinson, Starr, & Price, 1984; Sackeim et al., 1982). Each of these studies compared the mood of patients with unilateral left- or right-sided brain damage and found a greater incidence of depressive symptoms following left-sided damage. In most cases, the damage was fairly gross, very likely included more than one sector of PFC, and often included other brain regions as well. The general interpretation that has been placed on these studies is that depressive symptoms are increased after left-sided anterior PFC damage because this brain territory participates in certain forms of positive affect and, when damaged, leads to deficits in the capacity to experience positive affect, a hallmark feature of depression (Watson et al., 1995). Note that not all studies support this conclusion. In a recent meta-analysis of lesion studies, Carson et al. (2000) failed to find support for this hypothesis. Davidson (1993) has previously reviewed many of these studies and has addressed a number of critical methodological and conceptual concerns in this literature. The most important of these issues is that according to the diathesis–stress model of anterior activation asymmetry proposed by Davidson (1995, 1998b, 2000) and Henriques and Davidson (1991), individual differences in anterior activation asymmetry, whether lesion induced or functional, represent a diathesis. As such, they alter the probability that specific forms of emotional reactions will occur in response to the requisite environmental challenge. In the absence of such a challenge, the pattern of asymmetric activation will simply reflect a propensity but will not necessarily culminate in differences in mood or symptoms. In a recent study with the largest sample size to date ($N = 193$) for a study of mood sequelae in patients with unilateral lesions, Morris et al. (1996) found that among stroke patients, it was only in those with small lesions that the relation between left PFC damage and depressive symptoms was observed. It is likely that larger lesions intrude on other brain territories and mask the relation between left PFC damage and depression.

A growing corpus of evidence in normal intact humans is consistent with the findings derived from the lesion evidence. Davidson and his colleagues have reported that induced positive and negative affective states shift the asymmetry in prefrontal brain electrical activity in lawful ways. For example, film-induced negative affect increases relative right-sided prefrontal and anterior temporal activation (Davidson, Ekman, Saron, Senulis, & Friesen, 1990), whereas induced positive affect elicits an opposite pattern of asymmetric activation. Similar findings have been obtained by others (e.g., Ahern & Schwartz, 1985; Jones & Fox, 1992). In addition, I review in the next section a body of evidence that supports the conclusion that individual differences in baseline levels of asymmetric activation in these brain regions are lawfully related to variations in dispositional affective style. Using an extended-picture-presentation paradigm designed to evoke longer duration changes in mood (Sutton, Davidson, Donzella, Irwin, & Dottl, 1997), we measured regional glucose metabolism with positron-emission tomography (PET) to ascertain whether similar patterns of anterior asymmetry would be present using this very different and more precise method to assess regional

brain activity (Sutton, Ward, et al., 1997). During the production of negative affect, we observed right-sided increases in metabolic rate in anterior orbital, inferior frontal, middle, and superior frontal gyri, whereas the production of positive affect was associated with a pattern of predominantly left-sided metabolic increases in the pre- and postcentral gyri. Using PET to measure regional cerebral blood flow, Hugdahl et al. (1995) and Hugdahl (1998) reported a widespread zone of increased blood flow in the right PFC, including the OFC, DLPFC, and inferior and superior cortices, during the extinction phase after aversive learning had occurred compared with the habituation phase, before the presentation of the experimental contingencies.

Other investigators have used clinical groups to induce a stronger form of negative affect in the laboratory than is possible with normal controls. One common strategy for evoking anxiety among anxious patients in the laboratory is to present them with specific types of stimuli that are known to provoke their anxiety (e.g., pictures of spiders for spider phobics; making a public speech for social phobics). Davidson, Marshall, Tomarken, and Henrique (2000), in a study using brain electrical activity measures, have recently found that when social phobics anticipate making a public speech, they show large increases in right-sided anterior activation. Pooling across data from three separate anxiety-disordered groups that were studied with PET, Rauch, Savage, Albert, Fishman, and Jenike (1997) found two regions of the PFC that were consistently activated across groups: the right inferior PFC and right medial orbital PFC.

The vmPFC has been implicated in the anticipation of future positive and negative affective consequences. Bechara, Damasio, Damasio, and Anderson (1994) have reported that patients with bilateral lesions of the vmPFC have difficulty anticipating future positive or negative consequences, although immediately available rewards and punishments do influence their behavior. Such patients show decreased levels of electrodermal activity in anticipation of a risky choice compared with controls, whereas controls exhibit such autonomic change before they explicitly know that it is risky choice (Bechara, Damasio, Damasio, & Lee, 1999; Bechara, Damasio, Tranel, & Damasio, 1997; Bechara, Tranel, Damasio, & Damasio, 1996).

The findings from the lesion method when effects of small unilateral lesions are examined and from neuroimaging studies in normal participants and patients with anxiety disorders converge on the conclusion that increases in right-sided activation in various sectors of the PFC are associated with increased negative affect. Less evidence is available for the domain of positive affect, in part because positive affect is much harder to elicit in the laboratory and because of the negativity bias (see Cacioppo & Gardner, 1999; Taylor, 1991). This latter phenomenon refers to the general tendency of organisms to react more strongly to negative compared with positive stimuli, perhaps as a consequence of evolutionary pressures to avoid harm. The findings from Bechara et al. (1997) on the effects of vmPFC lesions on the anticipation of future positive and negative affective consequences are based on studies of patients with bilateral lesions. It will be of great interest in the future to examine patients with unilateral ventromedial lesions to ascertain whether valence-dependent asymmetric effects are present for this sector of the PFC as well, though most lesions in this PFC territory tend to occur bilaterally.

Systematic studies designed to disentangle the specific role played by various sectors of the PFC in emotion are lacking. Many theoretical accounts of emotion assign it an important role in guiding action and organizing behavior toward the acquisition of motivationally significant goals (e.g., Frijda, 1994; Levenson, 1994). This process requires that the organism have some means of representing affect in the absence of immediately present rewards and punishments and other affective incentives. Such a process may be conceptualized as a form of affective working memory. It is likely that the PFC plays a key role in this process (see Watanabe, 1996). Damage to certain sectors of the PFC impairs a person's capacity to anticipate future affective outcomes and consequently results in an inability to guide behavior in an adaptive fashion. Such damage is not likely to disrupt a person's responding to immediate cues for reward and punishment, only the anticipation before and maintenance after an affective cue is presented. This proposal can be tested using current neuroimaging methods (e.g., fMRI) but has not yet been rigorously evaluated. With regard to the different functional roles of the DLPFC, OFC, and vmPFC, Davidson and Irwin (1999) have suggested, on the basis of considering both human and animal studies, that the ventromedial sector is most likely involved in the representation of elementary positive and negative affective states in the absence of immediately present incentives. The orbitofrontal sector has most firmly been linked to rapid learning and unlearning of stimulus–incentive associations and has been particularly implicated in reversal learning (Rolls, 1999). As such, the OFC is likely key to understanding aspects of emotion regulation (see Davidson, Putnam, & Larson, 2000). One critical component of emotion regulation is the relearning of stimulus–incentive associations that might have been previously maladaptive, a process likely requiring the OFC. The DLPFC is most directly involved in the representation of goal states toward which more elementary positive and negative states are directed.

Amygdala

A large corpus of research at the animal, mostly rodent, level has established the importance of the amygdala for emotional processes (Aggleton, 1993; Cahill & McGaugh, 1998; LeDoux, 1996). Because many reviews of the animal literature have appeared recently, a detailed description of these studies is not presented here. LeDoux and his col-

leagues have marshaled a large corpus of compelling evidence to suggest that the amygdala is necessary for the establishment of conditioned fear. Whether the amygdala is necessary for the expression of that fear after learning and whether the amygdala is the actual locus of where the learned information is stored are still a matter of some controversy (see Cahill, Weinberger, Roozendaal, & McGaugh, 1999; Faneslow & LeDoux, 1999). The classic view of amygdala damage in nonhuman primates resulting in major affective disturbances, as expressed in the Kluver–Bucy syndrome, in which the animal exhibits abnormal approach, hyperorality and hypersexuality, and little fear, is now thought to be a function of damage elsewhere in the medial temporal lobe. When very selective excitotoxic lesions of the amygdala are made that preserve fibers of passage, nothing resembling the Kluver–Bucy syndrome is observed (Kalin, Shelton, Kelley, & Davidson, 2000). The upshot of this diverse array of findings is to suggest a more limited role for the amygdala in certain forms of emotional learning, although the human data imply a more heterogeneous contribution.

Although the number of patients with discrete lesions of the amygdala is small, they have provided unique information on the role of this structure in emotional processing. A number of studies have now reported specific impairments in the recognition of facial expressions of fear in patients with restricted amygdala damage (Adolphs, Damasio, Tranel, & Damasio, 1995, 1996; Broks et al., 1998; Calder et al., 1996). Recognition of facial signs of other emotions was found to be intact. In a study that required subjects to make judgments of trustworthiness and approachability of unfamiliar adults from facial photographs, patients with bilateral amygdala damage judged the unfamiliar individuals to be more approachable and trustworthy than did control subjects (Adolphs, Tranel, & Damasio, 1998). Recognition of vocalic signs of fear and anger was found to be impaired in a patient with bilateral amygdalar damage (Scott et al., 1997), suggesting that this deficit is not restricted to facial expressions. Other researchers (Bechara et al., 1995) have demonstrated that aversive autonomic conditioning was impaired in a patient with amygdalar damage despite the fact that the patient showed normal declarative knowledge of the conditioning contingencies. Collectively, these findings from patients with selective bilateral destruction of the amygdala suggest specific impairments on tasks that tap aspects of negative emotion processing. Most of the studies have focused on the perceptual side, in which the data clearly show the amygdala to be important for the recognition of cues of threat or danger. The conditioning data also indicate that the amygdala may be necessary for acquiring new implicit autonomic learning of stimulus–punishment contingencies. In one of the few studies to examine the role of the amygdala in the expression of already learned emotional responses, Angrilli et al. (1996) reported on a patient with a benign tumor of the right amygdala in an emotion-modulated startle study. Among control participants, they observed the well-known effect of startle potentiation during the presentation of aversive stimuli. In the patient with right-amygdalar damage, no startle potentiation was observed in response to aversive versus neutral stimuli. These findings suggest that the amygdala may be necessary for the expression of already learned negative affect.

Since 1995, a growing number of studies using PET and fMRI to investigate the role of the amygdala in emotional processes have begun to appear. Many studies have reported activation of the amygdala detected with either PET or fMRI when anxiety-disordered patients have been exposed to their specific anxiety-provoking stimuli compared with control stimuli (e.g., Breiter et al., 1996; Rauch et al., 1996). When social phobics were exposed to neutral faces, they showed activation of the amygdala comparable to what was observed in both the phobics and controls in response to aversive compared with neutral odors (Birbaumer et al., 1998). In keeping with the human lesion data, a number of studies have now reported activation of the amygdala in response to facial expressions of fear compared with neutral, happy, or disgust control faces (Morris et al., 1996; Phillips et al., 1997). In their fMRI study, Breiter et al. (1996) observed rapid habituation of the amygdala response, which might provide an important clue to the time-limited function of the amygdala in the stream of affective information processing. In a recent study, Whalen, Rauch, et al. (1998) observed activation of the amygdala in response to masked-fear faces that were not consciously perceived. Unpleasant compared with neutral and pleasant pictures also have been found to activate the amygdala (Irwin et al., 1996). Finally, a number of studies have reported activation of the amygdala during early phases of aversive conditioning (Buchel, Morris, Dolan, & Friston, 1998; LaBar, Gatenby, LeDoux, & Phelps, 1998). Amygdalar activation in response to several other experimental procedures for inducing negative affect, including unsolvable anagrams of the sort used to induce learned helplessness (Schneider et al., 1996), aversive olfactory cues (Zald & Pardo, 1997), and aversive gustatory stimuli (Zald, Lee, Fluegel, & Pardo, 1998), has been reported. Other data on individual differences in amygdalar activation and their relation to affective style are treated in the next section. The issues of whether the amygdala responds preferentially to aversive versus appetitive stimuli, is functionally asymmetric, and is required for both the initial learning and subsequent expression of negative emotional associations have not yet been adequately resolved and are considered in detail elsewhere (Davidson & Irwin, 1999). Note that one recent fMRI study (Zalla et al., 2000) found differential activation of the left and right amygdala to winning and losing money, with the left amygdala showing increased activation to winning more money while the right amygdala showed increased

activation in response to the parametric manipulation of losing money. Systematic examination of asymmetries in amygdalar activation and function in appetitive and aversive contexts should be performed in the light of these data.

Hippocampus and Anterior Cingulate Cortex

In this section, brief mention is made of the contributions of the hippocampus and ACC to emotion. More extensive discussion of the contributions of this circuit to emotional processing is contained in several recent reviews (Davidson & Irwin, 1999; Bush, Luu, & Posner, 2000).

The hippocampus has been implicated in various aspects of memory (see, e.g., Zola & Squire, 2000), particularly declarative memory of the sort we experience when we consciously recall an earlier occurring episode. Its role in emotion and affective style has only recently begun to be gleaned from the available corpus of animal studies on the role of the hippocampus in context-dependent memory (Fanselow, 2000). This literature has generally supported a role for the hippocampus in the learning of context. For example, when an animal is exposed to a cue-conditioning procedure where a discrete cue is paired with an aversive outcome, in addition to learning the specific cue–punishment contingency, the animal also learns to associate the context in which the learning occurs with the aversive outcome. Lesions to the hippocampus will abolish this context-dependent form of memory but will have no effect on learning the cue–punishment contingency.

That the hippocampus is a site in the brain with a very high density of glucocortocoid receptors and participates in the regulation of the hypothalamic–pituitary–adrenal axis is particularly germane to the importance of this structure for emotion regulation. Basic research at the animal level has demonstrated the powerful impact of glucocorticoids on hippocampal neurons (Cahill & McGaugh, 1998; McEwen, 1998). Data indicate that exogenous administration of hydrocortisone to humans impairs explicit memory that is presumably hippocamapally dependent (e.g., Kirschbaum, Wolf, May, Wippich, & Hellhammer, 1996), although other data suggest that in more moderate amounts, cortisol may facilitate memory (e.g., Abercrombie, 2000). A number of investigators using MRI-based measures have reported that hippocampal volume is significantly decreased in patients with several stress-related disorders, including posttraumatic stress disorder (PTSD; e.g., Bremner, 1999) and depression (e.g., Sheline, Wang, Gado, Csernansky, & Vannier, 1996; Bremner et al., 2000), although there also have been several failures to replicate (e.g., Vakili et al., 2000). In the studies in which hippocampal atrophy has been found, the implication is that excessively high levels of cortisol associated with the stress-related disorder cause hippocampal cell death and result in hippocampal atrophy as seen on MRI.

Although virtually all of these studies have focused on the implications of hippocampal changes for cognitive function, particularly declarative memory, Davidson et al. (2000) have proposed that the hippocampus plays a key role in the context modulation of emotional behavior. Moreover, we have suggested that the impact of hippocampal involvement in psychopathology may be most apparent in the affective realm. We suggested that in individuals with compromised hippocampal function, the normal context-regulatory role of this brain region would be impaired and individuals would consequently display emotional behavior in inappropriate contexts. This argument holds that what may be particularly abnormal in disorders such as PTSD and depression is not the display of abnormal emotion, but rather the display of perfectly normal emotion in inappropriate contexts. For example, in the case of PTSD, the extreme fear and anxiety were most likely very adaptive in the original traumatic context. This extreme emotional response quite likely plays an important role in facilitating the organism's withdrawal from a threatening situation. However, in PTSD, this response is elicited in inappropriate situations. The patient with PTSD behaves similarly to the animal with a hippocampal lesion in failing to modulate emotional responses in a context-appropriate manner.

These suggestions are only inferential at the present time. Neuroimaging studies are needed to document the role of the hippocampus in this process in normal and disordered populations. In addition, more attention is needed to understand how and why the hippocampus may preferentially extract and process information about context. Finally, some research (e.g., Davis & Lee, 1998) indicates that other structures with direct connections to the hippocampus (e.g., the bed nucleus of the stria terminalis) also play a role similar to the hippocampus. More work is needed to understand the differential contributions of the different components of this circuitry.

Many studies that have used neuroimaging methods to probe patterns of brain activation during the arousal of emotion have reported that the ACC activates in response to emotion. Several investigators (Bush et al., 2000; Whalen, Bush, et al., 1998) have recently distinguished between cognitive and affective subdivisions of the ACC on the basis of where activations lie in response to tasks that are purely cognitive versus those that include aspects of emotion. For example, in response to the classical Stroop task, ACC activation is found consistently more dorsal to the locus of activation observed in response to an emotional Stroop task with emotional words. Still, the question of just what role the more ventral portions of the ACC might be playing in emotion has not been systematically addressed. On the basis of Cohen's model of the role of ACC in conflict monitoring in the cognitive domain (e.g., Carter, Botvinick, & Cohen, 1999), we have proposed that the affective subdivision of the ACC might play a similar role in emotion. When emotion is elicited in the laboratory, it itself presents something of a conflict because social norms dictate certain rules for participant behavior that do not usually include the display of strong

emotion. Thus, the very process of activating emotion in the unfamiliar context of a laboratory environment might activate ACC. Carter et al. have suggested that ACC activation results in a call for further processing by other brain circuits to address the conflict that has been detected. In most people, automatic mechanisms of emotion regulation are likely invoked to dampen strong emotion that may be activated in the laboratory. The initial call for the processes of emotional regulation may result from ACC activation.

Individual Differences in the Prefrontal Cortex and Amygdalar Activations

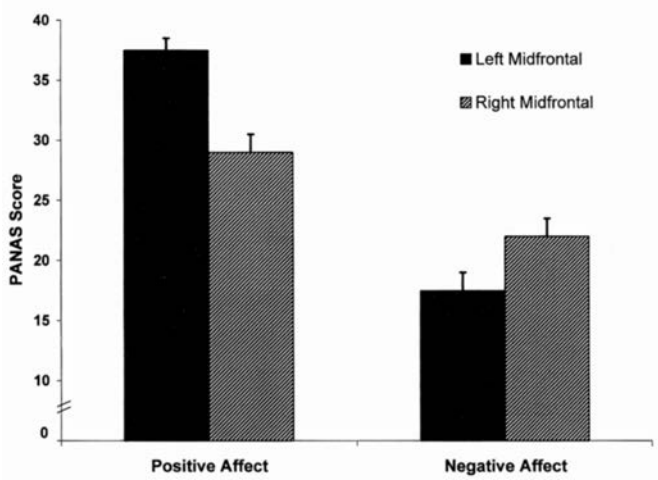
In both infants (Davidson & Fox, 1989) and adults (Davidson & Tomarken, 1989), there are large individual differences in baseline electrophysiological measures of prefrontal activation, and such individual variation is associated with differences in aspects of affective reactivity. In infants, Davidson and Fox (1989) reported that 10-month-old babies who cried in response to maternal separation were more likely to have less left- and greater right-sided prefrontal activation during a preceding resting baseline compared with those infants who did not cry in response to this challenge. In adults, Davidson and Tomarken (1989) first noted that the phasic influence of positive and negative emotion elicitors (e.g., film clips) on measures of prefrontal activation asymmetry appeared to be superimposed on more tonic individual differences in the direction and absolute magnitude of asymmetry.

During our initial explorations of this phenomenon, we needed to determine if baseline electrophysiological measures of prefrontal asymmetry were reliable and stable over time and thus could be used as a traitlike measure. Tomarken, Davidson, Wheeler, and Kinney (1992) recorded baseline brain electrical activity from 90 normal participants on two occasions separately by approximately three weeks. At each testing session, brain activity was recorded during eight one-minute trials, four with eyes open and four with eyes closed, presented in counterbalanced order. The data were visually scored to remove artifact and then Fourier transformed. Our focus was on power in the alpha band (8–13 Hz), though we extracted power in all frequency bands (see Davidson, Chapman, Chapman, & Henriques, 1990, and Davidson, Jackson, & Larson, 2000, for methodological discussion). We computed coefficient alpha as a measure of internal-consistency reliability from the data for each session. The coefficient alphas were quite high, with all values exceeding .85, indicating that the electrophysiological measures of asymmetric activation indeed showed excellent internal-consistency reliability. The test–retest reliability was adequate, with intraclass correlations ranging from .65 to .75 depending on the specific sites and methods of analysis. The major conclusion from this study was the demonstration that measures of activation asymmetry based on power in the alpha band from prefrontal scalp electrodes showed both high internal-consistency reliability and acceptable test–retest reliability to be considered a traitlike index.

On the basis of our prior data and theory, we reasoned that extreme left- and extreme right-frontally activated participants would show systematic differences in dispositional positive and negative affect. We administered the trait version of the Positive and Negative Affect Scales (PANAS; Watson, Clark, & Tellegen, 1988) to examine this question and found that the left-frontally activated participants reported more positive and less negative affect than their right-frontally activated counterparts (Tomarken, Davidson, Wheeler, & Doss, 1992; see Figure 1). More recently Sutton and Davidson (1997) showed that scores on a self-report measure designed to operationalize Gray's (1994) concepts of behavioral inhibition and activation (the Behavioral Inhibition Scale [BIS] and the Behavioral Activation Scale [BAS]; Carver & White, 1994) were even more strongly predicted by electrophysiological measures of prefrontal asymmetry than were scores on the PANAS scales (see Figure 2). Participants with greater left-sided prefrontal activation reported more relative BAS to BIS activity compared with participants exhibiting more right-sided prefrontal activation.

We also hypothesized that our measures of prefrontal asymmetry would predict reactivity to experimental elicitors of emotion. The model that we have developed over the past several years (see Davidson, 1992, 1994, 1995, 1998a, 1998b, for background) features individual differences in prefrontal activation

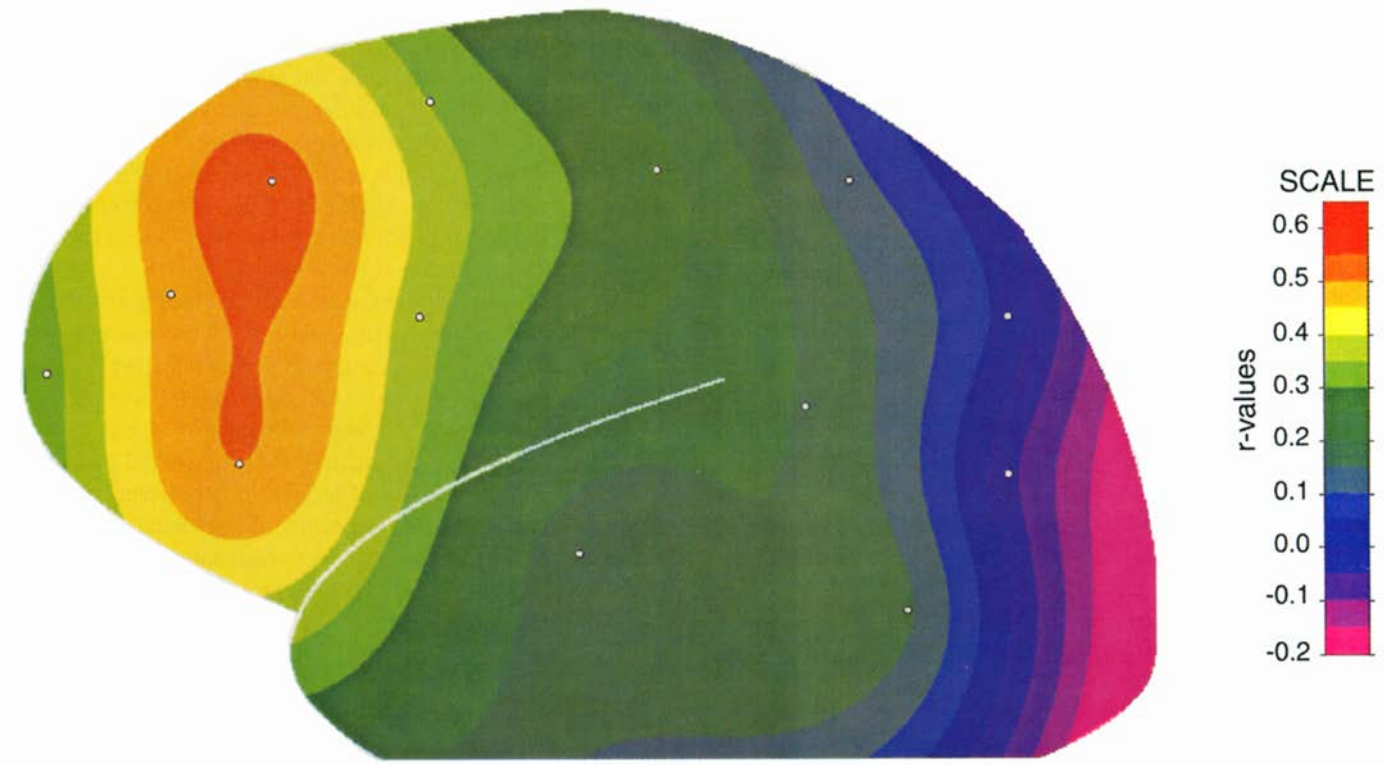
Figure 1
Dispositional Positive and Negative Affect



Note. Dispositional positive and negative affect was measured by the Positive and Negative Affect Scales (PANAS) in participants who were classified as extremely and stably left-frontally active (n = 14) and extremely and stably right-frontally active (n = 13) on the basis of electrophysiological measures of baseline activation asymmetries on two occasions separated by 3 weeks. Error bars reflect the standard error of the mean. From "Individual Differences in Anterior Brain Asymmetry and Fundamental Dimensions of Emotion," by A. J. Tomarken, R. J. Davidson, R. E. Wheeler, and R. C. Doss, 1992, *Journal of Personality and Social Psychology, 62*, p. 681. Copyright 1992 by the American Psychological Association. Reprinted with permission of the authors.

Figure 2

Electrophysiological Measures of Asymmetry and Standardized Scores on the Behavioral Activation Scale (BAS) and the Behavioral Inhibition Scale (BIS)



Note. Relations between electrophysiological measures of asymmetry and the difference between the standardized scores on the BAS and the BIS (Carver & White, 1994; $N = 46$). Electrophysiological data were recorded from each participant on two separate occasions separated by six weeks. The BAS and BIS scales were also administered on these two occasions. Data were averaged across the two time periods before we performed correlation analyses. The topographic map displays the correlations between alpha-power asymmetry (log right minus log left alpha power; higher values denote greater relative left-sided activation) and the difference score between the standardized BAS minus BIS. scales. After correlations were performed for each homologous region, a spline-interpolated map was created. The orange and red values of the scale denote positive correlations. The figure indicates that the correlation between the BAS-BIS difference score and the electrophysiology asymmetry score is highly positive in prefrontal scalp regions, denoting that participants with greater relative left-sided activation report more relative behavioral activation compared with behavioral inhibition tendencies. The relation between asymmetric activation and the BAS and the BIS difference is highly specific to the anterior scalp regions, because the correlation drops off rapidly more posteriorly. The correlation in the prefrontal region is significantly larger than the correlation in the parieto-occipital region. From "Prefrontal Brain Asymmetry: A Biological Substrate of the Behavioral Approach and Inhibition Systems," by S. K. Sutton and R. J. Davidson, 1997, *Psychological Science, 8,* p. 208. Copyright 1997 by Blackwell Publishers. Reprinted with permission.

asymmetry as a reflection of a diathesis that modulates reactivity to emotionally significant events. According to this model, individuals who differ in prefrontal asymmetry will respond differently to an elicitor of positive or negative emotion, even when baseline mood is partialed out. Wheeler, Davidson, and Tomarken (1993; see also Tomarken, Davidson, & Henriques, 1990) performed an experiment to examine this question. We presented short film clips designed to elicit positive or negative emotion. Brain electrical activity was recorded before the presentation of the film clips. Just after the clips were presented, participants were asked to rate their emotional experience during the film clip. In addition, participants completed scales that were designed to reflect their mood at baseline. We found that individual differences in prefrontal asymmetry predicted the emotional response to the films even after measures of baseline mood were statistically removed. Those individuals with more left-sided prefrontal activation at baseline reported more positive affect to the positive film clips, and those with more right-sided prefrontal activation reported more negative affect to the negative film clips. These findings support the idea that individual differences in electrophysiological measures of prefrontal activation asymmetry mark some aspect of vulnerability to positive and negative emotion elicitors. The fact that such relations were obtained after the statistical removal of baseline mood indicates that any difference between left- and right-frontal activation in baseline mood cannot account for the prediction of film-elicited emotion effects that were observed.

The relation between individual differences in brain electrical measures of prefrontal activation asymmetry and depression is a topic that has received extensive treatment in

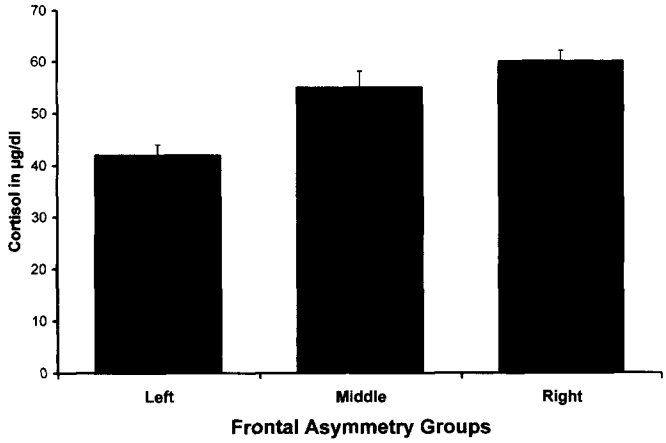
several recent articles. There has been a failure to replicate (Reid, Duke, & Allen, 1998) our initial findings of decreased left prefrontal activation in depression (Henriques & Davidson, 1990, 1991; Schaffer, Davidson, & Saron, 1983), although there have also been several published independent replications or conceptual replications (e.g., Allen, Iacona, Depue, & Arbisi, 1993; Field, Fox, Pickens, & Nawrocki, 1995). Moreover, using PET, Drevets et al., (1997) have reported decreased activation in the left subgenual prefrontal cortex in patients with depression. We interpreted the decrease in left-sided prefrontal activation as a diathesis related to deficits in the approach system and in reward-related responding (Henriques & Davidson, 2000; Henriques, Glowacki, & Davidson, 1994). We also argued that this pattern of left prefrontal hypoactivation would only be found in certain subgroups of mood-disordered patients in the light of the heterogeneity of the disorder (see Davidson, 1998a, for an extended discussion). Most important, we suggested that it is crucial to move beyond descriptive phenomenology and to examine with objective laboratory methods variations in reactivity to emotion elicitors in individuals with this hypothesized diathesis. We proposed that individuals who display left prefrontal hypoactivation would show specific deficits in reactivity to reward, although the need to consider other components of the circuitry with which the prefrontal cortex is interconnected must be underscored in any effort to understand the neural bases of emotion and its disorders.

In addition to the studies described above using self-report and psychophysiological measures of emotion, we have also examined relations between individual differences in electrophysiological measures of prefrontal asymmetry and other biological indices, which in turn have been related to differential reactivity to stressful events. Three recent examples from our laboratory include measures of immune function, cortisol, and corticotropin-releasing hormone. The latter two measures represent key molecules in the activation of a coordinated response to stressful events. Our strategy in each case was to examine relations between individual differences in measures of prefrontal activation asymmetry and these other biological indices. In two separate studies (Davidson, Coe, Dolski, & Donzella, 1999; Kang et al., 1991), we examined relations between the prefrontal activation indices and natural killer (NK) activity because declines in NK activity have been reported in response to stressful, negative events (Kiecolt-Glaser & Glaser, 1981). We predicted that participants with greater right prefrontal activation would exhibit lower NK activity compared with their left-activated counterparts because the former type of participant has been found to report more dispositional negative affect, to show higher relative BIS activity, and to respond more intensely to negative emotional stimuli. In each of the two studies conducted with independent samples, we found that right-frontally activated participants indeed had lower levels of NK activity compared with their left-frontally activated counterparts (Davidson et al., 1999; Kang et al., 1991). We also examined the magnitude of change in NK activity in response to stress and found that participants with greater baseline levels of right prefrontal activation showed the largest magnitude of decline in NK activity compared with other participants (Davidson et al., 1999).

In collaboration with Kalin, our laboratory has been studying similar individual differences in scalp-recorded measures of prefrontal activation asymmetry in Rhesus monkeys (Davidson, Kalin, & Shelton, 1992, 1993). Recently, Kalin, Larson, Shelton, and Davidson (1998) acquired measures of brain electrical activity from a large sample of Rhesus monkeys ($N = 50$). Electroencephalograph measures were obtained during periods of manual restraint. A subsample of 15 of these monkeys was tested on two occasions four months apart. We found that the test–retest correlation for measures of prefrontal asymmetry was .62, suggesting similar stability of this metric in monkey and man. In the group of 50 monkeys, we also obtained measures of plasma cortisol during the early morning. We hypothesized that if individual differences in prefrontal asymmetry were associated with dispositional affective style, such differences would be correlated with cortisol, because individual differences in baseline cortisol have been related to various aspects of trait-related stressful behavior and psychopathology (see, e.g., Gold, Goodwin, & Chrousos, 1988). We found that monkeys with right-sided prefrontal activation had higher levels of baseline cortisol than their left-frontally activated counterparts (see Figure 3). As can be seen from the figure, the left-activated monkeys had particularly low levels of cortisol compared with both middle- and right-activated subjects. Moreover, when blood samples were collected two years after our initial testing, monkeys classified as showing extreme right-sided prefrontal activation at age one year had significantly higher baseline cortisol levels when they were three years old compared with monkeys who were classified at age one year as displaying extreme left-sided prefrontal activation. Similar findings were obtained with cerebrospinal fluid levels of corticotropin-releasing hormone (CRH). Monkeys with greater right-sided prefrontal activation showed higher levels of CRH (Kalin, Shelton, & Davidson, 2000; see Figure 4). These findings indicate that individual differences in prefrontal asymmetry are present in nonhuman primates and that such differences predict biological measures that are related to affective style.

With the advent of neuroimaging, it has become possible to investigate the relation between individual differences in aspects of amygdalar function and measures of affective style. We have used PET with flourodeoxyglucose (FDG) as a tracer to investigate relations between individual differences in glucose metabolism in the amygdala and dispositional negative affect. FDG-PET is well

Figure 3
Basal Morning Plasma Cortisol Levels

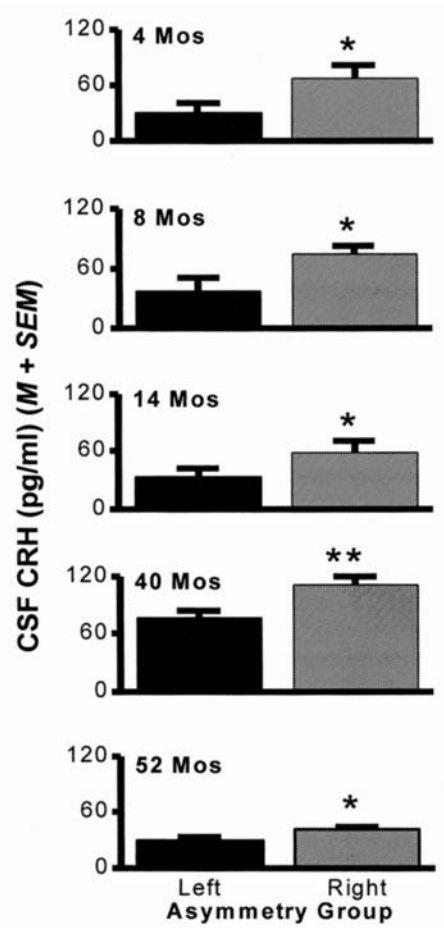


Note. Basal morning plasma cortisol levels are from one-year-old Rhesus monkeys classified as left (n = 12), middle (n = 16) or right (n = 11) frontally activated on the basis of on electrophysiological measurements. Error bars reflect standard error of the mean. From "Asymmetric Frontal Brain Activity, Cortisol, and Behavior Associated With Fearful Temperament in Rhesus Monkeys," by N. H. Kalin, C. L. Larson, S. E. Shelton, and R. J. Davidson, 1998, *Behavioral Neuroscience, 112,* p. 289. Copyright 1998 by the American Psychological Association. Reprinted with permission.

suited to capture traitlike effects because the period of active uptake of tracer in the brain is approximately 30 minutes. Thus, it is inherently more reliable than O^{15} blood flow measures because the FDG data reflect activity aggregated over a 30-minute period. We have used resting FDG-PET to examine individual differences in glucose metabolic rate in the amygdala and its relation to dispositional negative affect in depressed participants (Abercrombie et al., 1998). We acquired a resting FDG-PET scan as well as a structural magnetic resonance (MR) scan for each participant. The structural MR scans are used for anatomical localization by coregistering the two image sets. Thus, for each participant, we used an automated algorithm to fit the MR scan to the PET image. Regions of interest (ROIs) were then drawn on each participant's MR scan to outline the amygdala in each hemisphere. These ROIs were drawn on coronal sections of participants' MR images, and the ROIs were then automatically transferred to the coregistered PET images. Glucose metabolism in the left and right amygdalar ROIs were then extracted. The interrater reliability for the extracted glucose metabolic rate was highly significant with intraclass correlations between two independent raters (≥.97). We found that participants with greater glucose metabolism in the right amygdala report greater dispositional negative affect on the PANAS scales (see Figure 5). These findings indicate that individual differences in resting glucose metabolism in the amygdala are present and that they predict dispositional negative affect among depressed participants.

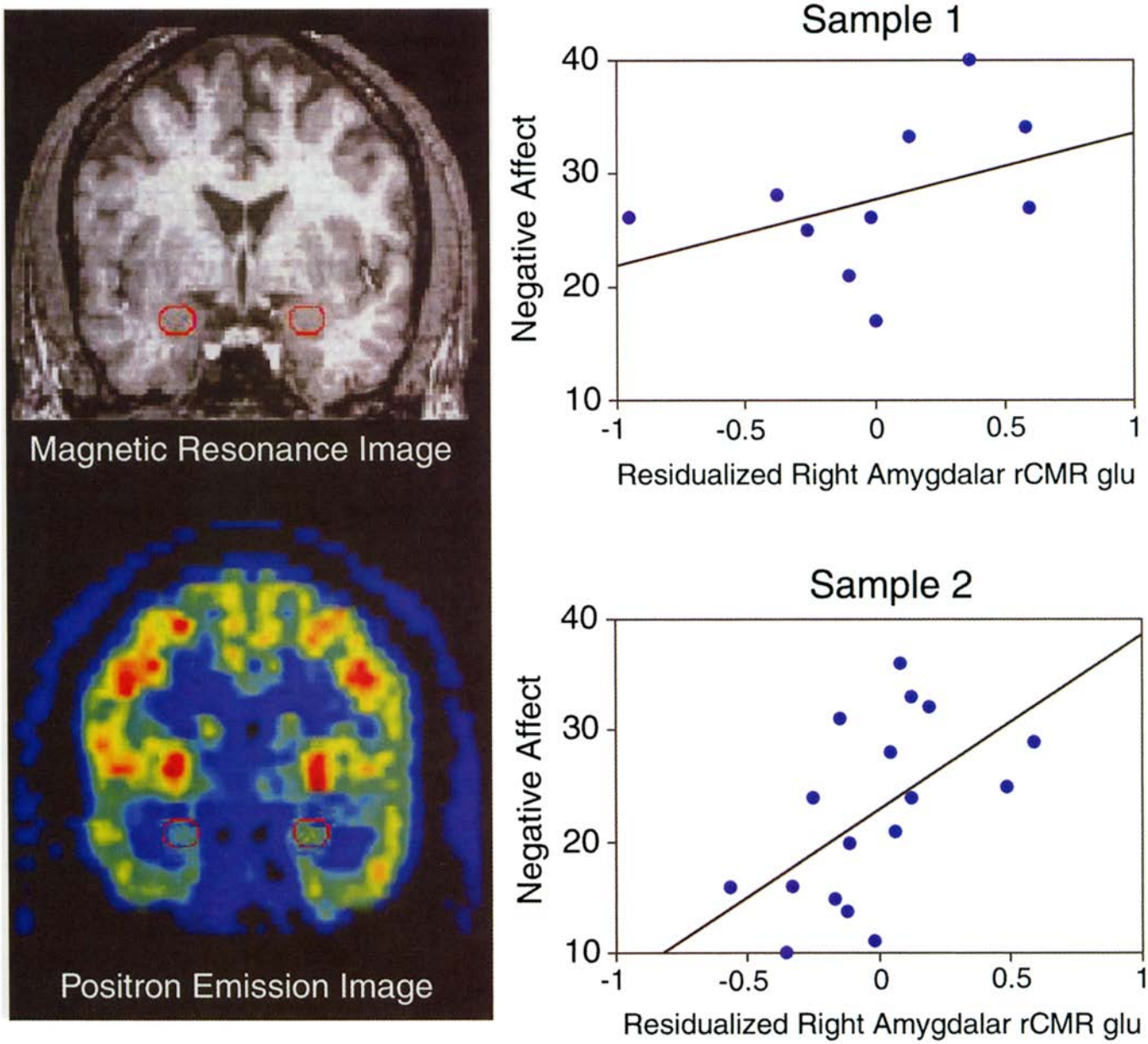
In a small sample of nine normal participants, Irwin, Anderle, Sutton, Kalin, & Davidson (2000) were able to examine the relation between the magnitude of MR signal change in the amygdala in response to aversive compared with neutral pictures and dispositional negative affect on the PANAS. We correlated the average value of the pixels with the maximum Student's t from the left and right amygdala with dispositional negative affect. There was a robust correlation, so that increased signal intensity in the right amygdala was associated with higher levels of negative affect (see Figure 6). A pixel in the fusiform gyrus that revealed robust activation by the

Figure 4
Cerebrospinal Fluid (CSF) Measures of Corticotropin-Releasing Hormone (CRH)



Note. Differences between right (n = 9) and left (n = 10) prefrontally activated Rhesus monkeys in CSF measures of CRH were measured at five different ages. The original classification of the monkeys as extremely right or left activated was performed on the basis of brain electrical activity data collected when they were 13 months old. Error bars reflect standard error of the mean. *$p < .05$. **$p < .01$. From "Cerebrospinal Fluid Corticotropin-Releasing Hormone Levels Are Elevated in Monkeys With Patterns of Brain Activity Associated With Fearful Temperament," N. H. Kalin, S. E. Shelton, and R. J. Davidson, 2000, *Biological Psychiatry, 47,* p. 592. Copyright 2000 by Elsevier Science. Reprinted with permission.

Figure 5
Magnetic-Resonance-Imaging-Coregistered Regions of Interest Around the Amygdala



Note. Images on the left indicate the magnetic resonance (top) and corresponding positron-emission tomography (PET) image (bottom) from one participant, illustrating our method of magnetic-resonance-imaging-coregistered regions of interest (ROI) around the amygdala. ROIs were individually drawn for each participant around the amygdala, and glucose metabolism was then extracted from the PET image. (Right) Scatter plots display the relation between glucose metabolic rate (residualized for variations in whole brain metabolism) in the right amygdala and dispositional negative affect assessed with the Positive and Negative Affect Scales for two independent samples of depressed patients tested on two different PET scanners. The scanner used for the data in the scatter plot on the bottom was one with the better spatial resolution (GE Advance). From "Metabolic Rate in the Right Amygdala Predicts Negative Affect in Depressed Patients," by H. C. Abercrombie, S. M. Schaefer, C. L. Larson, T. R. Oakes, J. E. Holden, S. B. Perlman, D. D. Krahn, R. M. Benca, and R. J. Davidson, 1998, *NeuroReport, 9*, p. 3305. Copyright 1998 by Lippincott, Williams, & Wilkins. Reprinted with permission.

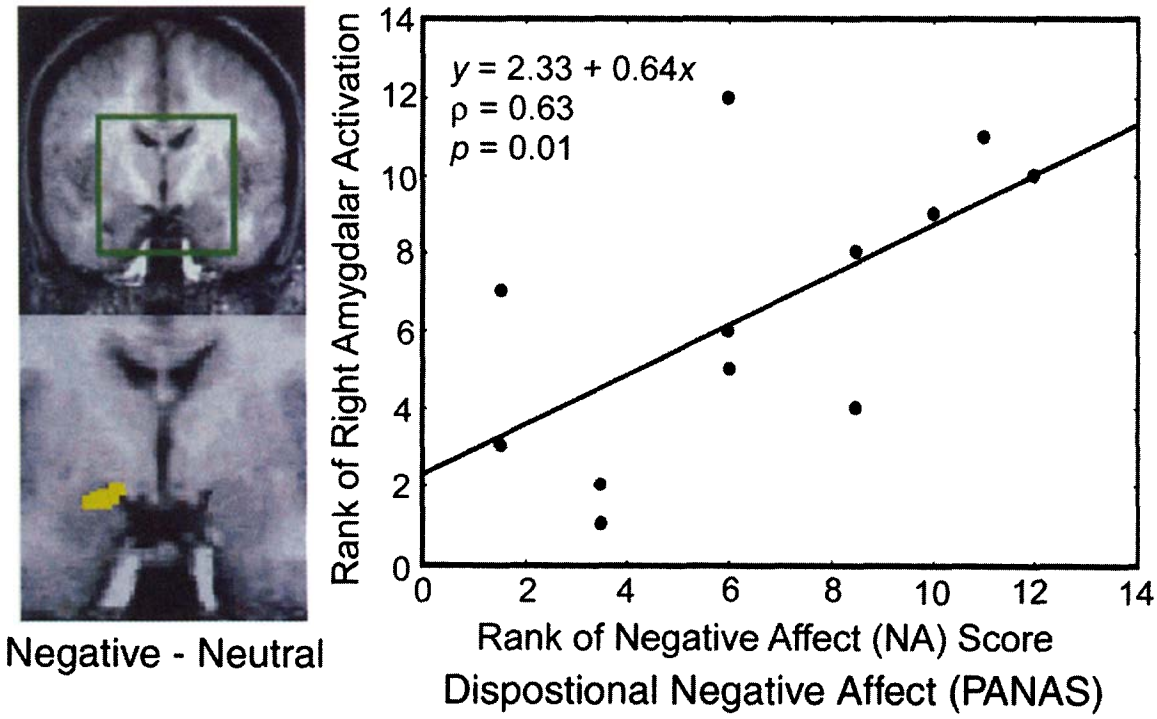
aversive pictures was selected as a control region. We correlated the magnitude of activation in the pixel showing the maximal response in this region to the aversive pictures with dispositional negative affect and found no relation ($p>.5$). Moreover, the correlations in the amygdala and fusiform gyrus were found to be significantly different. The findings from the fMRI and PET studies of amygdalar function indicate that individual differences in both tonic activation and phasic activation in response to aversive stimuli predict the intensity of dispositional negative affect.

Emotion Regulation: A Key Component of Affective Style

One of the key components of affective style is the capacity to regulate negative emotion and specifically to decrease the duration of negative affect once it arises. We have suggested in several recent articles that the connections between the PFC and amygdala play an important role in this regulatory process (Davidson, 1998a; Davidson & Irwin, 1999; Davidson, Putnam, & Larson, 2000).

In a recent study, Larson et al. (1998) examined relations between individual differences in prefrontal activation asymmetry and the emotion-modulated startle. In this study we presented pictures from the *International Affective Picture System* (Lang, Bradley, & Cuthbert, 1995) while acoustic startle probes were presented and the electromyograph-measured blink response from the *orbicularis oculi* muscle region was recorded (see Sutton, Davidson, et al., 1997, for basic methods). Startle probes were presented both during the six-second slide exposure and at various latencies after the offset of the pictures, on separate trials. We interpreted startle magnitude during picture exposure as providing an index related to the peak of emotional response, while startle magnitude after the *offset* of the pictures was taken to reflect the recovery from emotional challenge. Used in this way, startle probe methods can potentially provide new information on the time course of emotional responding. We expected that individual differences during actual picture presentation would be less pronounced than individual differences after picture presentation because an acute emotional stimulus is likely to pull for a normative response across participants, whereas individuals are more likely to differ once the

Figure 6
Coronal Images of the Amygdala



Note. Coronal image in the top left indicates the region that contains the amygdala, and that image is magnified in the bottom left image. The magnified image also contains data showing activation of the right amygdala (right side of the brain is on the left side of the image) in response to unpleasant versus neutral pictures from the International Affective Picture Series (Lang, Bradley, & Cuthbert, 1995). The scatter plot on the right indicates the relation between the magnitude of magnetic resonance signal change in the right amygdala and the rank ordering of dispositional negative affect, as measured by the Positive and Negative Affect Scales (PANAS). (From Irwin et al., 2000.)

stimulus has terminated. Similarly, we predicted that individual differences in prefrontal asymmetry would account for more variance in predicting magnitude of recovery (i.e., startle magnitude poststimulus) than in predicting startle magnitude during the stimulus.

Our findings were consistent with our predictions and indicated that participants with greater right-sided prefrontal activation show a larger blink magnitude after the offset of the negative stimuli, after the variance in blink magnitude *during* the negative stimulus was partialed out. Measures of prefrontal asymmetry did not reliably predict startle magnitude during picture presentation. The findings from this study were consistent with our hypothesis and indicated that individual differences in prefrontal asymmetry are associated with the time course of affective responding, particularly the recovery after emotional challenge.

In a related study, we have found that participants with greater baseline levels of left prefrontal activation are better able to voluntarily suppress negative affect (see Jackson, Burghy, Hanna, Larson, & Davidson, 2000; Jackson, Malmstadt, et al., 2000). Moreover, using fMRI, we have demonstrated that when participants are instructed to voluntarily regulate their negative emotion, reliable changes in amygdala MR signal intensity are found (Schaefer, Jackson, Davidson, Kimberg, & Thompson-Schill, 2000).

The findings from these studies indicate that individual differences in prefrontal activation may play an important role in emotion regulation. Individuals who report greater dispositional negative affect and who show increased reactivity to stressful events may be those individuals who have difficulty regulating negative affect and specifically in modulating the intensity of negative affect once it has been activated.

Plasticity in the Central Circuitry of Emotion

The circuitry that underlies emotion regulation, in particular, the amygdala and prefrontal cortex, have both been targets of intensive study of plasticity (see Davidson et al., 2000, for extensive discussion). In a series of elegant studies in rats, Francis and Meaney (1999) have demonstrated that an early environmental manipulation in rats—frequency of maternal licking–grooming and arched-back nursing—produces a cascade of biological changes in the offspring that shape the central circuitry of emotion and, consequently, alter the animal's behavioral and biological responsivity to stress. For example, the offspring of mothers who display frequent licking and grooming show increased central benzodiazepine receptor densities in various subnuclei of the amygdala as well as in the locus ceruleus (LC), increased α_2 adrenoreceptor density in the LC, and decreased CRH receptor density in the LC (Caldji et al., 1998). Other researchers (Liu et al., 1997; Meaney, Aitken, van Berkel, Bhatnagar, & Sapolsky, 1988; Meaney et al., 1996) have reported that rats exposed to frequently licking–grooming mothers exhibited a permanent increase in concentrations of receptors for glucocorticoids in both the hippocampus and the PFC. All of these changes induced by early maternal licking–grooming and related behavior involve alterations in the central circuitry of emotion that results in decreased responsivity to stress later in life.

These findings in animals raise the possibility that similar effects may transpire in humans. There are clearly short-term changes in brain activation that are observed during voluntary emotion regulation, as noted above. Whether repeated practice in techniques of emotion regulation lead to more enduring changes in patterns of brain activation is a question that has not yet been answered in extant research. There are limited data available that indicate that cognitive–behavioral therapy for certain disorders (e.g., obsessive–compulsive disorder) produces changes in regional brain activity that are comparable with those produced by medication (Baxter et al., 1992). What is absent are data on plastic changes in the brain that might be produced by the practice of methods specifically designed to increase positive affect, such as meditation. The Dalai Lama himself has raised this question in his recent book *The Art of Happiness* (Dalai Lama & Cutler, 1998), where he explains that

> The systematic training of the mind—the cultivation of happiness, the genuine inner transformation by deliberately selecting and focusing on positive mental states and challenging negative mental states—is possible because of the very structure and function of the brain. . . . But the wiring in our brains is not static, not irrevocably fixed. Our brains are also adaptable. (pp. 44–45)

We are now at the point in the development of affective neuroscience where we can rigorously address this question by using neuroimaging methods to probe changes in patterns of activation and transmitter function that might be produced by the systematic practice of techniques like meditation that are designed to promote the cultivation of positive affect. By examining how changes in the central circuitry of emotion might be related to peripheral biology (endocrine, autonomic, and immune function), we can begin to mechanistically examine how emotions and affective style, in particular, can be consequential for our health.

Summary and Conclusions

The circuitry underlying emotion and affective style was reviewed, with an emphasis on the functions of different sectors of the PFC and amygdala. Individual differences in patterns of prefrontal and amygdalar activation are related to behavioral and biological constituents of affective style and emotion regulation. Recent data highlight the role of particular sectors of the PFC in emotion regulation, particularly the regulation of the duration of emotion and the suppression of

negative emotion once it is elicited. These individual differences are conceptualized as diatheses that alter a person's vulnerability or resilience toward developing psychopathology. Recent evidence in animals underscores the extraordinary plasticity of this circuitry and demonstrates that early social experience, in particular, has profound consequences for the developing nervous system. The possibilities of transforming this circuitry in adulthood with specific methods designed to cultivate positive affect were considered. New longitudinal research is needed to address these questions.

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E. Tory Higgins

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