

# Toward a Biology of Personality and Emotion

RICHARD J. DAVIDSON

*Department of Psychology, University of Wisconsin-Madison, Madison,  
Wisconsin 53706, USA*

**ABSTRACT:** For most of this past century, scholarship on the topics of personality and emotion has emerged from the humanities and social sciences. In the past decade, a remarkable change has occurred in the influence of neuroscience on the conceptualization and study of these phenomena. This article argues that the categories that have emerged from psychiatric nosology and descriptive personality theory may be inadequate, and that new categories and dimensions derived from neuroscience research may produce a more tractable parsing of this complex domain. The article concludes by noting that the discovery of these biological differences among individuals does not imply that the origins of these differences lie in heritable influences. Experiential shaping of the brain circuitry underlying emotion is powerful. The neural architecture provides the final common pathway through which culture, social factors, and genetics all operate together.

**KEYWORDS:** Personality; Emotion; Psychiatric nosology; Descriptive personality theory; Affective neuroscience; Prefrontal cortex; Amygdala; Neuroimaging studies

For most of the last century, the domains of personality and emotion were mostly the province of literature and philosophy. The social sciences and psychiatry began to address these phenomena in the latter half of the 20th century with mixed success. The categories that are widely used in the study of both personality and emotion are those that first emerged in the earliest eras of scholarship in these arenas. Traditional personality theory and the descriptive nosology of psychiatry were important influences on the development of these constructs.

In the past 10 years, a remarkable change has occurred in our conceptualization of these topics and in our ability to study them in intact human beings. The change represents the emergence of new areas of interdisciplinary research that represent the melding of basic neuroscience with the psychological study of personality and emotion. Termed affective neuroscience,<sup>1,2</sup> this new interdisciplinary area is having a major impact on our conception of personality, emotion, and disorders of emotion. This article will consider some of these promising new trends and illustrate some of the key changes that these new developments are suggesting in how we conceptualize these topics.

Address for correspondence: Richard J. Davidson, Laboratory for Affective Neuroscience, Department of Psychology, University of Wisconsin, 1202 West Johnson Street, Madison, WI 53706. Voice: 608-262-8972; fax: 608-265-2875.  
rjdavids@facstaff.wisc.edu

## THE ROLE OF THE NEUROSCIENCES IN THE STUDY OF PERSONALITY AND EMOTION

Before the advent of neuroimaging to study human brain function, most of the neuroscientific research on emotion was confined to the study of animals, mostly rodents, in whom lesions were made in various subcortical nuclei, and the behavioral consequences of these lesions were measured. This work has had and continues to have enormous impact and has been of great importance to the development of more sophisticated models of brain function and emotion in humans.<sup>2</sup> Most of this work is performed in rodents, and rodents have a relatively small cerebral cortex. In humans, it is undoubtedly the case that the cortex plays a very important role in emotion,<sup>3</sup> and it was not until studies in both non-human primates and humans that the role of various cortical territories in emotion was widely appreciated.

Human research has highlighted the role of various territories of the prefrontal cortex (PFC) in particular that are crucial to different aspects of emotion.<sup>4</sup> The PFC is especially important to emotion regulation and individual differences in affective style, as will be described below. Emotion regulation refers to processes that enhance, suppress, or maintain an emotional response while affective style refers to consistent individual differences in basic parameters of emotional reactivity and emotion regulation. Many of the regulatory processes implemented in the PFC operate relatively automatically and are thus opaque to direct self-report and are represented only indirectly in self-report measures. For this reason, the neuroscientific work on affective style and emotion regulation has uncovered certain components of emotion, emotion regulation, and individual differences that could not have been discovered through self-report methods. Moreover, the identification of the mechanisms of emotional reactivity provide a more direct path to understanding how both genetic and experiential factors may operate synergistically on a final common pathway to shape personality and emotion.

## THE CENTRAL CIRCUITRY OF EMOTION

### *The Prefrontal Cortex*

Though approaching the topic from very different perspectives, a growing body of literature is converging on the idea that there exist two fundamental systems that underlie approach and withdrawal-related emotion and motivation, or positive and negative affect.<sup>4-8</sup> The precise description of these systems differs somewhat across investigators as does the anatomical circuitry that is featured, but the essential elements are quite similar in each of these different proposals. The approach system has been described by Davidson and Irwin<sup>4</sup> as facilitating appetitive behavior and generating particular types of positive affect that are approach-related, such as the emotion occurring as an organism moves closer toward a desired goal. The withdrawal system, on the other hand, facilitates the withdrawal of an organism from sources of aversive stimulation and/or organizes appropriate responses to cues of threat. This system also generates withdrawal-related negative emotions such as disgust and fear. A variety of evidence indicates that these systems are implemented in partially sep-

arable circuits, and it is to this evidence that we now turn. Our focus will be on two key components of this circuitry—the prefrontal cortex (PFC) and the amygdala. For more extensive discussion of this entire circuitry including other regions not considered here, see Davidson and Irwin.<sup>4</sup>

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 include the dorsolateral, ventromedial, and orbitofrontal sectors. In addition, there appear to be important functional differences between the left and right sides within each 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.<sup>9–11</sup> 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 and likely included more than one sector of PFC and often included other brain regions as well. The general interpretation that has been placed upon these studies is that depressive symptoms are increased following 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.<sup>12</sup> Though most of the extant lesion data are consistent with this general picture (see Robinson and Downhill<sup>13</sup> for a review), some inconsistencies have also appeared (e.g., Refs. 14,15). Davidson<sup>16</sup> has reviewed these studies in detail 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 and colleagues (e.g., Refs. 1,17,18), 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.*<sup>19</sup> found that among stroke patients, it was only in those with small-sized 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,<sup>20</sup> whereas induced positive affect elicits an opposite pattern of asymmetric activation. Similar findings have been obtained by others (e.g., Refs. 21–23). In addition, I will review in the next section a body of evidence that supports the conclusion that individuals who vary in their baseline levels of asymmetric activation in these brain regions differ in their dispositional affective style. Using an extended picture presentation par-

adigm designed to evoke longer-duration changes in mood,<sup>24</sup> 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.<sup>25</sup> During the production of negative affect, we observed right-sided increases in metabolic rate in anterior orbital, inferior frontal, and middle and superior frontal gyri, while the production of positive affect was associated with a pattern of predominantly left-sided metabolic increases in the pre- and post-central gyri. Using PET to measure regional cerebral blood flow, Hugdahl and his colleagues<sup>26,27</sup> reported a widespread zone of increased blood flow in the right PFC including the orbitofrontal and dorsolateral cortices and inferior and superior cortices during the extinction phase after 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 *et al.*,<sup>28</sup> 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, Rauch *et al.*<sup>29</sup> found two regions of the PFC that were consistently activated across groups: the right inferior PFC and right medial orbital PFC.

The ventromedial PFC has been implicated in the anticipation of future positive and negative affective consequences. Bechara and his colleagues<sup>30</sup> have reported that patients with bilateral lesions of the ventromedial PFC 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, while controls exhibit such autonomic change before they explicitly know that it is a risky choice.<sup>31-33</sup>

The findings from the lesion method when effects of small unilateral lesions are examined and from neuroimaging studies in normal subjects and patients with anxiety disorders converge on the conclusion that an increase in right-sided activation in various sectors of the PFC is 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.<sup>5,34</sup> 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.* on the effects of ventromedial PFC 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 PFC as well.

Systematic studies designed to disentangle the specific roles played by various sectors of the PFC in emotion are lacking. Many theoretical accounts of emotion as-

sign it an important role in guiding action and organizing behavior toward the acquisition of motivationally significant goals (e.g., see Refs. 35,36). 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 likened to a form of affective working memory. It is likely that the PFC plays a key role in this process.<sup>37</sup> Damage to certain sectors of the PFC impair an individual's capacity to anticipate future affective outcomes and consequently result in an inability to guide behavior in an adaptive fashion. Such damage is not likely to disrupt an individual's responding to immediate cues for reward and punishment; only the anticipation before and sustainment after an affective cue is presented. This proposal can be tested using current neuroimaging methods (e.g., functional magnetic resonance imaging [fMRI]) but has not yet been rigorously evaluated. With regard to the different functional roles of the dorsolateral and ventromedial sectors of the PFC, Davidson and Irwin<sup>4</sup> suggested, on the basis of considering both human and animal studies, that the latter sector is most likely involved in the representation of elementary positive and negative affective states in the absence of immediately present incentives, while the former sector is most directly involved in the representation of goal states toward which these more elementary positive and negative states are directed.

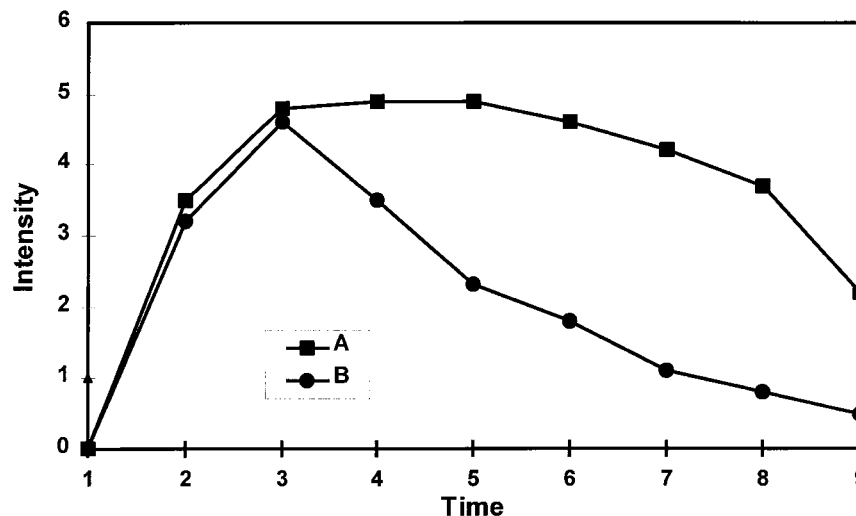
### *The Amygdala*

A large corpus of research at the animal—mostly rodent—level has established the importance of the amygdala for emotional processes.<sup>38–40</sup> Because many reviews of the animal literature have appeared recently, a detailed description of these studies will not be presented here. LeDoux and his colleagues 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 following learning and whether the amygdala is the actual locus of where the learned information is stored is still a matter of some controversy (see Refs. 41,42). Also not resolved is the extent to which the amygdala participates in all learning of stimulus–incentive associations, both negative and positive, and whether there are functional differences between the left and right amygdala.<sup>4</sup> 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, hyper-orality and sexuality, 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.<sup>43,44</sup> The upshot of this diverse array of findings is to suggest a more limited role for the amygdala in certain forms of emotional learning, though 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.<sup>45–48</sup> 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.<sup>49</sup> Recognition of vocalic signs of fear and anger was found to be impaired in a patient with bilateral amygdala damage,<sup>50</sup> suggesting that this deficit is not restricted to facial expressions. Other researchers<sup>51</sup> have demonstrated that aversive autonomic conditioning is impaired in a patient with amygdala 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 where 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 and colleagues<sup>52</sup> reported on a patient with a benign tumor of the right amygdala in an emotion-modulated startle study. Among control subjects, they observed the well-known effect of startle potentiation during the presentation of aversive stimuli. In the patient with right amygdala damage, no startle potentiation was observed in response to aversive versus neutral stimuli. These findings suggest that the amygdala might 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., Refs. 53,54). When social phobics were exposed to neutral faces, they showed activation of the amygdala comparable to that observed in both the phobics and controls in response to aversive compared with neutral odors.<sup>55</sup> Consistent 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 disgusted control faces.<sup>56–59</sup> In the Breiter *et al.*<sup>57</sup> fMRI study, they observed rapid habituation of the amygdala response, which may provide an important clue to the time-limited function of the amygdala in the stream of affective information processing. In a recent study, Whalen and his colleagues<sup>59</sup> observed activation of the amygdala in response to masked fear faces that were not consciously perceived. Unpleasant compared with neutral and pleasant pictures have also been found to activate the amygdala.<sup>60,61</sup> Finally, a number of studies have reported activation of the amygdala during early phases of aversive conditioning.<sup>62–64</sup> Amygdala activation in response to several other experimental procedures for inducing negative affect has been reported, including unsolvable anagrams of the sort used to induce learned helplessness,<sup>65</sup> aversive olfactory cues,<sup>66</sup> and aversive gustatory stimuli.<sup>67</sup> Other data on individual differences in amygdala activation and their relation to affective style will be 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 are considered in detail elsewhere.<sup>4</sup>



**FIGURE 1.** Hypothetical time courses of two individuals in response to an emotionally arousing stimulus. Both individuals show comparable initial reactivity but differ in the recovery. Individual A shows a prolonged recovery, whereas individual B recovers more rapidly.

### AFFECTIVE STYLE

Davidson<sup>17,68</sup> has used the term affective style to refer to the broad range of individual differences in different subcomponents of affective reactivity and dispositional mood. This is a very global term, and it is imperative to specify with more precision which particular system one is measuring affective reactivity in and which subcomponent of reactivity is being targeted for study. For example, one could measure affective reactivity in different response systems by using startle magnitude, MR signal change in the amygdala, or ratings on a self-report scale as the measure. Each of these obviously reflects activity in very different systems, and activation in these systems will not necessarily cohere. What is meant by subcomponent of reactivity has been articulated in detail in Davidson<sup>17</sup> and includes the following parameters: tonic level, threshold to respond, peak or amplitude of response, rise time to peak of response, and recovery time (see FIG. 1). These are not meant to necessarily reflect an exhaustive list of subcomponents, they are merely offered as examples. Each of these subcomponents can potentially be studied in different response systems, leading to many parameters of affective style. We know virtually nothing about the psychometric characteristics of measures of these different parameters, except for self-report measures (for two recent efforts examining different subcomponents of affective style in two different physiological response systems see Refs. 69,70), though this information is crucial if we are to develop rigorous measures of these constructs. In this section, we review data on the contributions of individual differences in prefrontal and amygdala function to affective style.

In two decades of previous research, we have performed a large number of studies designed to examine the role of activation asymmetries in prefrontal cortex and other anterior cortical zones in aspects of affective style. This work has been reviewed recently,<sup>1,17</sup> and only highlights will be presented here. Using measures of scalp-recorded brain electrical activity, we found that indices of activation asymmetry based on power spectral measures were stable over time and exhibited excellent internal consistency reliability,<sup>69,71</sup> thus fulfilling a number of important psychometric criteria for an index of a trait-like construct. In a series of studies, we found that there are large individual differences in the magnitude and direction of baseline asymmetric activation in brain electrical activity measures obtained from prefrontal scalp regions in both infants<sup>72</sup> and adults.<sup>73</sup> In 10-month-old infants, we found that those with greater relative right-sided prefrontal activation in prefrontal scalp regions were more likely to cry in response to a brief period of maternal separation compared with their left-activated counterparts.<sup>74</sup> In toddlers and young children, we have observed that those individuals with greater relative right-sided prefrontal activation show more behavioral inhibition and wariness measured through laboratory-based behavioral observation.<sup>72</sup> In adults, we have found that individual differences in such measures predict dispositional mood,<sup>71</sup> self-report measures of behavioral activation and inhibition,<sup>75</sup> repressive defensiveness,<sup>76</sup> reactivity to positive and negative emotion elicitors,<sup>77-78</sup> baseline immune function,<sup>79</sup> and reactivity of the immune system to emotional challenge.<sup>80</sup> In very recent work<sup>81</sup> we found that individual differences in electrophysiological measures of prefrontal asymmetry predicted the magnitude of recovery following a negative affective stimulus. These data suggest that the prefrontal cortex may play a role in regulating the time course of emotional responding and/or in the active inhibition of negative affect. We will return to these issues later in the article.

We have also found that individual differences in these brain electrical measures of anterior asymmetry are associated with mood and anxiety disorders. In particular, we have found that depressed subjects and persons who are currently euthymic but have a history of past depression exhibit less left prefrontal activation compared with never-depressed controls.<sup>18,82</sup> We have also found that when social phobics anticipate making a public speech, they show large increases in right-sided prefrontal activation though they do not differ from controls at baseline.<sup>28</sup>

In a series of studies with Kalin,<sup>83-85</sup> we have demonstrated that similar activation asymmetries can be measured in rhesus monkeys and that they predict similar types of behavior and biology as we observe in humans. In a recent effort of this kind, we<sup>85</sup> found that animals with greater relative right-sided prefrontal activation exhibit higher basal levels of the stress hormone cortisol. Similar data have recently been reported in humans.<sup>86</sup>

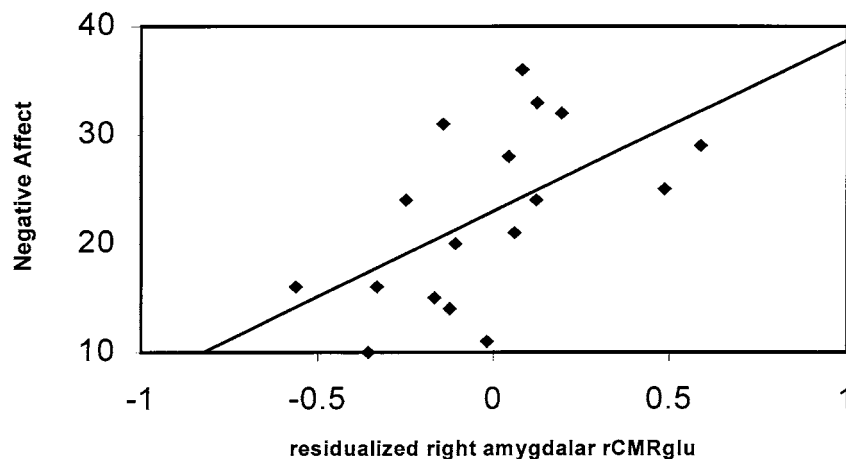
A number of our original EEG observations have now been independently replicated by others,<sup>21,87-92</sup> though a few studies have appeared reporting only partial replications of aspects of our original findings.<sup>93,94</sup> Davidson<sup>95</sup> has called attention to a number of crucial methodological and conceptual issues in these replication attempts and suggests that the difficulties in replication are mostly a function of significant methodological limitations. Moreover, few studies using neuroimaging to address the role of prefrontal asymmetries in affective processes have appeared. As noted by Davidson and Irwin,<sup>4</sup> only a very small handful of studies using PET or

fMRI have conducted the proper statistical comparison to uncover asymmetry effects in their data. They comment on the complexity of performing these analyses. Because the structural anatomy is not symmetrical, particularly for cortical tissue, it is very difficult to extract homologous regions for asymmetry analyses. The size of the regions may differ on the two sides of the brain, the anatomical homologue may not be in exactly the same location in each hemisphere, and the shape of the cortical territory on each side of the brain is often different. These facts present formidable methodological obstacles when using neuroimaging to make inferences about patterns of asymmetric activation.

The data from the Larson *et al.*<sup>81</sup> study referred to previously indicated that persons with greater relative left-sided prefrontal activation at baseline have greater recovery of startle potentiation following the offset of a negative stimulus. Moreover, the measure of asymmetric prefrontal activation accounted for more variance in the magnitude of startle post-negative-stimulus offset (i.e., startle recovery) than it did during the stimulus. These findings imply that individual differences in prefrontal activation asymmetry may play a role in regulating the time course of emotional responding and that those individuals with more left-sided prefrontal activation may recover more quickly from negative affect or stress than their right-activated counterparts.

A clue to the mechanism that may underlie this consequence of left prefrontal activation is provided by a study from LeDoux's laboratory where they found that rats with lesions of the medial prefrontal cortex show dramatically slower extinction of a learned aversive response compared with sham-operated controls.<sup>96</sup> These findings imply that there is a descending pathway between the medial PFC and the amygdala<sup>97</sup> that is inhibitory and thus represents an active component of extinction. In the absence of this normal inhibitory input, the amygdala remains unchecked and continues to remain activated. Whether this inhibitory input from the medial PFC is an important component of the prominent habituation observed in the amygdala remains to be clarified. Davidson<sup>17</sup> has suggested that in humans and possibly other primates, the major inhibitory influence on the amygdala may derive from the left prefrontal cortex. Consistent with this idea, recent PET findings suggest that in normal human subjects, glucose metabolism in the left medial and lateral prefrontal cortex is reciprocally coupled to metabolic activity in the amygdala, such that those subjects with increased left prefrontal metabolic rate have decreased metabolic rate in the amygdala.<sup>98</sup> We propose that this mechanism may be responsible for the dampening of negative affect and the shortening of its time course in those persons who appear to be more resilient. Such an affective style may also facilitate the maintenance of approach-related positive affect.

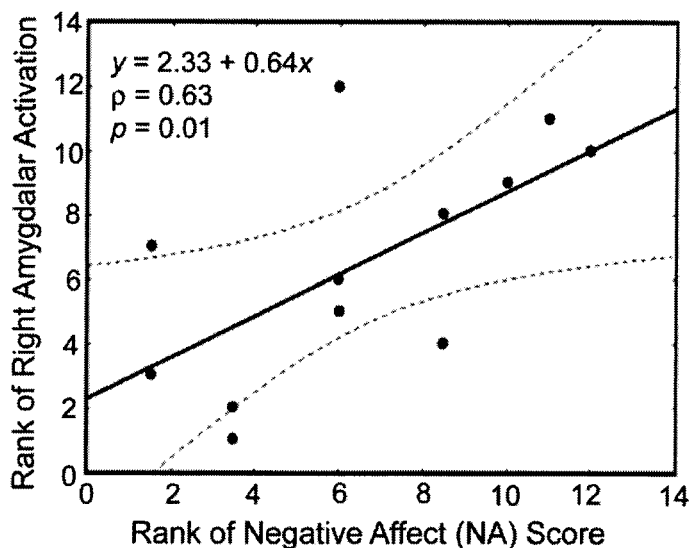
The two key features of the circuitry underlying positive and negative affect highlighted herein are the prefrontal cortex and the amygdala. In the previous section, we detailed studies on the basic function of the amygdala in affective behavior. Here we ask the question about individual differences in amygdala function and its relation to affective style. Although most research on the amygdala has emphasized its phasic function, there is a tonic level of activation in the amygdala that can be assessed with PET measures of regional glucose metabolism. Using MRI-based coregistration, we can draw regions of interest around the amygdala on an MR scan coregistered to the PET image and extract metabolic activity in such small regions without using any spatial filtering of the PET image. This provides higher resolution than could ordi-



**FIGURE 2.** Scatter plot of the relation between metabolic rate in the right amygdala and dispositional negative affect. Metabolic rate in the amygdala was obtained by coregistering MRI and PET images and then drawing regions of interest (ROIs) on the MRIs around the amygdala. These ROIs were then automatically transferred to the PET images, and glucose metabolic rate for these regions was determined. (From Abercrombie *et al.*<sup>101</sup>)

rarily be achieved using conventional cross-subject aggregation methods that require spatial smoothing of the images (see Ref. 99). Using such procedures, we have found that individual differences in metabolic activity in the right amygdala, in particular, predict dispositional negative affect on the Positive and Negative Affect Schedule (PANAS)<sup>100</sup> in a group of depressed patients (see FIG. 2).<sup>101</sup> Using the same measure of negative affect, we<sup>102</sup> have also found MR signal change in the amygdala in response to negative versus neutral stimuli accounts for a substantial amount of variance in PANAS trait negative affect scores ( $r = 0.63$ ; see FIG. 3). Other researchers have found that individual differences in right amygdala glucose metabolic rate in response to emotional films predicts the recall of negative emotional films assessed three weeks following the PET procedure. Those persons with higher levels of glucose metabolism in the right amygdala recalled more of the negative film clips.<sup>103</sup> Other investigators using both PET<sup>104</sup> and fMRI<sup>63</sup> reported that those subjects with greater activation in the amygdala during classical aversive conditioning showed greater evidence of electrodermal conditioning. Ketter *et al.*,<sup>105</sup> using the anesthetic procaine as a pharmacological challenge, reported that those individuals who had a dysphoric response to the drug had significantly greater activation of the amygdala compared with subjects exhibiting a euphoric response. Moreover, amygdala blood flow correlated positively with fear and negatively with euphoria on self-report measures of emotional intensity.

Some of the data reviewed above on relations between amygdala activation and dispositional negative affect appear at least on the surface to be inconsistent with the animal and human neuroimaging data, implying that the amygdala is important only in the initial learning of stimulus–threat associations,<sup>62,63</sup> but not in the expression



**FIGURE 3.** Scatter plot of the relation between MR signal change in the right amygdala in response to unpleasant versus neutral pictures assessed with functional MRI and dispositional negative affect assessed with the Positive and Negative Affect Schedule. (From Irwin *et al.*<sup>102</sup>)

of pre-existing temperamental variation such as behavioral inhibition. For example, in our own data using PET-derived measures of glucose metabolism in the amygdala,<sup>101</sup> we found that subjects with greater metabolic rate in the right amygdala report higher levels of dispositional negative affect as assessed by the PANAS. A similar association was found using the identical affect measure with fMRI where subjects showing larger MR signal increases in the amygdala in response to negative versus neutral pictures reported higher levels of dispositional negative affect. The PANAS requires subjects to rate a series of single-word adjectives on a 1–5-point scale to indicate the extent to which that emotion is present during their daily life. Thus, in these experiments, it appears that activation levels in the amygdala are associated with the expression of a pre-existing affective style. We believe the key to resolving this apparent inconsistency among these findings lies in a more in-depth understanding of the strategies people use to respond to questionnaires like the PANAS. When subjects are asked to make global inferences about the affective dispositions that are extended in time, they are not veridical integrators of the momentary affective states that unfolded over the period in question. Rather, as a number of commentators have forcefully argued, they exhibit systematic heuristic biases that reflect the information that is accessible at the time.<sup>106,107</sup> In particular, in a series of elegant studies Kahneman<sup>106</sup> has demonstrated that individuals tend to adopt what he refers to as the “peak-end” rule for forming these retrospective affective evaluations. Thus, although an individual might be asked to rate how “nervous” he was during the past month, he is likely to weight excessively information about the peak episode of ner-

vousness during this period, as well as his level of nervousness very recently. The peak intensity of the emotion in question may be especially related to amygdala activation since it is likely to represent a response to a particularly threatening or novel episode. Such complexities in measuring subjective aspects of emotion underscores the need to develop more objective measures that do not depend upon self-report and that can better capture the time course of emotional responding, or what Davidson<sup>17</sup> has referred to as affective chronometry.

The fact that there exist reliable individual differences in baseline metabolic rate in the amygdala also requires comment in light of the earlier discussion about the amygdala's role in phasic affective processes. There is clearly intrinsic neural activity in the amygdala, even during sleep.<sup>108</sup> As a number of studies have now shown, baseline nontask ("resting") levels of activation in the amygdala are associated with dispositional negative affect<sup>101</sup> and depression.<sup>109</sup> Whether these baseline differences in amygdala activation reflect activation in response to the PET environment or whether such differences predict the magnitude of task-induced activation in the amygdala in response to emotion elicitors are questions that must be addressed in future research. We believe that when PET is used to measure baseline differences in amygdala activation, at least for the right amygdala, it likely reflects an important influence of the experimental situation itself. This claim is made on the basis of the fact that our recent evidence<sup>99</sup> using MR-coregistration to extract glucose metabolic rate in several subcortical regions revealed that test-retest reliability over a 6-month period is excellent for all subcortical regions we examined (hippocampus, caudate, thalamus, left amygdala) except for the right amygdala. These findings are consistent with the idea that situational influences are important in modulating activation in the right amygdala.

### IMPLICATIONS AND CONCLUSIONS

The concepts and findings reviewed in this article underscore the utility of the modern melding between neuroscience and studies of personality and emotion. Certain aspects of automatic emotional processes, such as the automatic regulation of the time course of emotional responding, are opaque to consciousness and therefore will not be directly revealed in self-reports. The study of the underlying brain mechanisms associated with basic parameters of affective style and emotion regulation will provide us with new and better methods to parse the domains of emotion and personality.

A critical issue not addressed above concerns the distal causes of the individual differences in affective style that have been featured. As I have detailed extensively elsewhere,<sup>110</sup> the fact of biological differences among individuals says nothing about the origins of those differences. A large corpus of neuroscience research over the past decade has underscored the importance of experiential determinants of the structure and function of the circuitry that has been featured here. Social influences on brain structure, activation patterns, neurogenesis, and even gene expression have all been demonstrated (see Davidson *et al.*<sup>110</sup> for review). Although heritable influences surely occur, environmental influences, particularly when they occur repetitively over time, can be extremely powerful and produce lasting changes in the brain.

The fact that such experiential influences occur provides an impetus for the development of neurally inspired training programs to transform dysfunctional affective styles into ones that may be more adaptive (see Ref. 111). This is only a promissory note at the present time and requires much additional study and validation.

#### ACKNOWLEDGMENTS

The research from the Laboratory for Affective Neuroscience and the Wisconsin Center for Affective Science reported in this article was supported by National Institute of Mental Health Grants MH43454, MH40747, and P50-MH52354; by Research Scientist Award K05-MH00875; and by a grant from the Research Network on Mind–Body Interaction of the John D. and Catherine T. MacArthur Foundation. I wish to thank the members of the Laboratory for Affective Neuroscience and the members of the Wisconsin Center for Affective Science for their sustained contributions to this research program.

#### REFERENCES

1. DAVIDSON, R.J. & S.K. SUTTON. 1995. Affective neuroscience: the emergence of a discipline. *Curr. Opin. Neurobiol.* **5**: 217–224.
2. PANKSEPP, J. 1998. *Affective Neuroscience*. Oxford University Press. New York.
3. RÖLLS, E.T. 1999. *The Brain and Emotion*. Oxford University Press. New York.
4. DAVIDSON, R.J. & W. IRWIN. 1999. The functional neuroanatomy of emotion and affective style. *Trends Cogn. Sci.* **3**: 11–21.
5. CACIOPPO, J.T. & W.L. GARDNER. 1999. Emotion. *Annu. Rev. Psychol.* **50**: 191–214.
6. GRAY, J.A. 1994. Three fundamental emotion systems. *In The Nature of Emotion: Fundamental Questions*. P. Ekman & R.J. Davidson, Eds.: 243–247. Oxford University Press. New York.
7. LANG, P.J., M.M. BRADLEY & B.N. CUTHBERT. 1990. Emotion, attention and the startle reflex. *Psychol. Rev.* **97**: 377–398.
8. SCHNEIRLA, T.C. 1959. An evolutionary and developmental theory of biphasic processes underlying approach and withdrawal. *In Nebraska Symposium on Motivation*. M.R. Jones, Ed.: 1–42. University of Nebraska Press. Lincoln, NE.
9. GAINOTTI, G. 1972. Emotional behavior and hemispheric side of lesion. *Cortex* **8**: 41–55.
10. SACKEIM, H.A., *et al.* 1982. Pathological laughter and crying: functional brain asymmetry in the expression of positive and negative emotions. *Arch. Neurol.* **39**: 210–218.
11. ROBINSON, R.G., L.B. STARR & T.R. PRICE. 1984. A two year longitudinal study of mood disorders following stroke: prevalence and duration at six month follow-up. *Br. J. Psychiatry* **144**: 256–262.
12. WATSON, D., *et al.* 1995. Testing a tripartite model: I. Evaluating the convergent and discriminant validity of anxiety and depression symptom scales. *J. Abnorm. Psychol.* **104**: 3–14.
13. ROBINSON, R.G. & J.E. DOWNHILL. 1995. Lateralization of psychopathology in response to focal brain injury. *In Brain Asymmetry*. R.J. Davidson & K. Hugdahl, Eds.: 693–711. MIT Press. Cambridge, MA.
14. HOUSE, A., *et al.* 1990. Life events and difficulties preceding stroke. *J. Neurol. Neurosurg. Psychiatry* **53**: 1024–1028.
15. GAINOTTI, G., C. CALTAGIRONE & P. ZOCCOLOTTI. 1993. Left/right and cortical/subcortical dichotomies in the neuropsychological study of human emotions. *Cogn. Emotion* **7**: 71–93.

16. DAVIDSON, R.J. 1993. Cerebral asymmetry and emotion: conceptual and methodological conundrums. *Cogn. Emotion* **7**: 115–138.
17. DAVIDSON, R.J. 1998. Affective style and affective disorders: perspectives from affective neuroscience. *Cogn. Emotion* **12**: 307–330.
18. HENRIQUES, J.B. & R.J. DAVIDSON. 1991. Left frontal hypoactivation in depression. *J. Abnorm. Psychol.* **100**: 535–545.
19. MORRIS, P.L.P., *et al.* 1996. Lesion characteristics and depressed mood in the stroke data bank study. *J. Neuropsychiatry* **8**: 153–159.
20. DAVIDSON, R.J., *et al.* 1990. Approach/withdrawal and cerebral asymmetry: emotional expression and brain physiology. *J. Pers. Soc. Psychol.* **58**: 330–341.
21. AHERN, G.L. & G.E. SCHWARTZ. 1985. Differential lateralization for positive and negative emotion in the human brain: EEG spectral analysis. *Neuropsychologia* **23**: 745–755.
22. JONES, N.A. & N.A. FOX. 1992. Electroencephalogram asymmetry during emotionally evocative films and its reaction to positive and negative affectivity. *Brain Cogn.* **20**: 280–299.
23. TUCKER, D.M., *et al.* 1981. Right frontal lobe activation and right hemisphere performance decrement during a depressed mood. *Arch. Gen. Psychiatry* **38**: 169–174.
24. SUTTON, S.K., *et al.* 1997. Manipulating affective state using extended picture presentation. *Psychophysiology* **34**: 217–226.
25. SUTTON, S.K., *et al.* 1997. Asymmetry in prefrontal glucose metabolism during appetitive and aversive emotional states: an FDG-PET study. *Psychophysiology* **34**: S89.
26. HUGDAHL, K., *et al.* 1995. Brain mechanisms in human classical conditioning: a PET blood flow study. *Neuroreport* **6**: 1723–1728.
27. HUGDAHL, K. 1998. Cortical control of human classical conditioning: autonomic and positron emission tomography data. *Psychophysiology* **35**: 170–178.
28. DAVIDSON, R.J., *et al.* 2000. While a phobic waits: regional brain electrical and autonomic activity in social phobics during anticipation of public speaking. *Biol. Psychiatry* **47**: 85–95.
29. RAUCH, S.L., *et al.* 1997. The functional neuroanatomy of anxiety: a study of three disorders using positron emission tomography and symptom provocation. *Biol. Psychiatry* **42**: 446–452.
30. BECHARA, A., *et al.* 1994. Insensitivity to future consequences following damage to human prefrontal cortex. *Cognition* **50**: 7–15.
31. BECHARA, A., *et al.* 1996. Failure to respond autonomically to anticipated future outcomes following damage to prefrontal cortex. *Cereb. Cortex* **6**: 215–225.
32. BECHARA, A., *et al.* 1997. Deciding advantageously before knowing the advantageous strategy. *Science* **275**: 1293–1295.
33. BECHARA, A., *et al.* 1999. Different contributions of the human amygdala and ventromedial prefrontal cortex to decision-making. *J. Neurosci.* **19**: 5473–5481.
34. TAYLOR, S.E. 1991. Asymmetrical effects of positive and negative events: the mobilization-minimization hypothesis. *Psychol. Bull.* **110**: 67–85.
35. FRIJDA, N.H. 1994. Emotions are functional, most of the time. *In* *The Nature of Emotion: Fundamental Questions*. P. Ekman & R.J. Davidson, Eds.: 112–122. Oxford University Press. New York.
36. LEVENSON, R.W. 1994. Human emotion: a functional view. *In* *The Nature of Emotion: Fundamental Questions*. P. Ekman & R.J. Davidson, Eds.: 123–126. Oxford University Press. New York.
37. WATANABE, M. 1996. Reward expectancy in primate prefrontal neurons. *Nature* **382**: 629–632.
38. LEDOUX, J.E. 1996. *The Emotional Brain: The Mysterious Underpinnings of Emotional Life*. Simon & Schuster. New York.
39. CAHILL, L. & J.L. MCGAUGH. 1998. Mechanisms of emotional arousal and lasting declarative memory. *Trends Neurosci.* **21**: 273–313.
40. AGGLETON, J.P. 1993. The contribution of the amygdala to normal and abnormal emotional states. *Trends Neurosci.* **16**: 328–333.

41. CAHILL, L., *et al.* 1999. Is the amygdala a locus of “conditioned fear”? Some questions and caveats. *Neuron* **23**: 227–228.
42. FANSELOW, M.S. & J.E. LEDOUX. 1999. Why we think plasticity underlying Pavlovian fear conditioning occurs in the basolateral amygdala. *Neuron* **23**: 229–232.
43. KALIN, N.H., S.E. SHELTON, R.J. DAVIDSON & A.E. KELLEY. 2001. The primate amygdala mediates acute fear but not the behavioral and physiological components of anxious temperament. *J. Neurosci.* In press.
44. MEUNIER, M. *et al.* 2000. Effects of aspiration versus neurotoxic lesions of the amygdala on emotional responses in monkeys. *Eur. J. Neurosci.* **11**: 4403–4418.
45. ADOLPHS, *et al.* 1995. Fear and the human amygdala. *J. Neurosci.* **15**: 5879–5891.
46. ADOLPHS, R., *et al.* 1996. Cortical systems for the recognition of emotion in facial expressions. *J. Neurosci.* **16**: 7678–7687.
47. BROKS, P., *et al.* 1998. Face processing impairments after encephalitis: amygdala damage and recognition of fear. *Neuropsychologia* **36**: 59–70.
48. CALDER, A.J., *et al.* 1996. Facial emotion recognition after bilateral amygdala damage: differentially severe impairment of fear. *Cogn. Neuropsychol.* **13**: 699–745.
49. ADOLPHS, R., D. TRANEL & A.R. DAMASIO. 1998. The human amygdala in social judgment. *Nature* **393**: 470–474.
50. SCOTT, S.K., *et al.* 1997. Impaired auditory recognition of fear and anger following bilateral amygdala lesions. *Nature* **385**: 254–257.
51. BECHARA, A., D. TRANEL, H. DAMASIO, *et al.* 1995. Double dissociation of conditioning and declarative knowledge relative to the amygdala and hippocampus in humans. *Science* **269**: 1115–1118.
52. ANGRILLI, A., *et al.* 1996. Startle reflex and emotion modulation impairment after a right amygdala lesion. *Brain* **119**: 1991–2000.
53. BREITER, H.C., *et al.* 1996. Functional magnetic resonance imaging of symptom provocation in obsessive-compulsive disorder. *Arch. Gen. Psychiatry* **53**: 595–606.
54. RAUCH, S.L., *et al.* 1996. A symptom provocation study of posttraumatic stress disorder using positron emission tomography and script-driven imagery. *Arch. Gen. Psychiatry* **53**: 380–387.
55. BIRBAUMER, N., *et al.* 1998. fMRI reveals amygdala activation to human faces in social phobics. *Neuroreport* **9**: 1223–1226.
56. MORRIS, J.S., *et al.* 1996. A differential neural response in the human amygdala to fearful and happy facial expressions. *Nature* **383**: 812–815.
57. BREITER, H.C., *et al.* 1996. Response and habituation of the human amygdala during visual processing of facial expression. *Neuron* **17**: 875–887.
58. PHILLIPS, M.L., *et al.* 1997. A specific neural substrate for perceiving facial expressions of disgust. *Nature* **389**: 495–498.
59. WHALEN, P.J., *et al.* 1998. Masked presentations of emotional facial expressions modulate amygdala activity without explicit knowledge. *J. Neurosci.* **18**: 411–418.
60. IRWIN, W., *et al.* 1996. Human amygdala activation detected with echo-planar functional magnetic resonance imaging. *Neuroreport* **7**: 1765–1769.
61. LANE, R.D., *et al.* 1997. Neuroanatomical correlates of pleasant and unpleasant emotion. *Neuropsychologia* **35**: 1437–1444.
62. BUCHEL, C., *et al.* 1998. Brain systems mediating aversive conditioning: an event-related fMRI study. *Neuron* **20**: 947–957.
63. LABAR, K.S., *et al.* 1998. Human amygdala activation during conditioned fear acquisition and extinction—a mixed-trial fMRI study. *Neuron* **20**: 937–945.
64. MORRIS, J.S., A. OHMAN & R.J. DOLAN. 1998. Modulation of amygdala activity by masking of aversively conditioned visual stimuli. *Neuroimage* **7**: S52.
65. SCHNEIDER, F., *et al.* 1996. Cerebral blood flow changes in limbic regions induced by unsolvable anagram tasks. *Am. J. Psychiatry* **153**: 206–212.
66. ZALD, D.H. & J.V. PARDO. 1997. Emotion, olfaction and the human amygdala: amygdala activation during aversive olfactory stimulation. *Proc. Natl. Acad. Sci. USA* **94**: 4119–4124.
67. ZALD, D.H., J.T. LEE, K.W. FLUEGEL & J.V. PARDO. 1998. Aversive gustatory stimulation activates limbic circuits in humans. *Brain* **121**: 1143–1154.

68. DAVIDSON, R.J. 1992. Emotion and affective style: hemispheric substrates. *Psychol. Sci.* **3**: 39–43.
69. TOMARKEN, A.J., *et al.* 1992. Psychometric properties of resting anterior EEG asymmetry: temporal stability and internal consistency. *Psychophysiology* **29**: 576–592.
70. LARSON, C.L., *et al.* 2000. Temporal stability of the emotion-modulated startle response. *Psychophysiology* **37**: 92–101.
71. TOMARKEN, A.J., R.J. DAVIDSON, R.E. WHEELER & R.C. DOSS. 1992. Individual differences in anterior brain asymmetry and fundamental dimensions of emotion. *J. Pers. Soc. Psychol.* **62**: 676–687.
72. DAVIDSON, R.J. & M.D. RICKMAN. 1999. Behavioral inhibition and the emotional circuitry of the brain: stability and plasticity during the early childhood years. *In* *Extreme Fear, Shyness, and Social Phobia: Origins, Biological Mechanisms, and Clinical Outcomes*. L.A. Schmidt & J. Schulkin, Eds.: 67–87. Oxford University Press. New York.
73. DAVIDSON, R.J. & A.J. TOMARKEN. 1989. Laterality and emotion: an electrophysiological approach. *In* *Handbook of Neuropsychology*, Vol. 3. F. Boller & J. Grafman, Eds.: 419–441. Elsevier. Amsterdam.
74. DAVIDSON, R.J. & N.A. FOX. 1989. Frontal brain asymmetry predicts infants' response to maternal separation. *J. Abnorm. Psychol.* **98**: 127–131.
75. SUTTON, S.K. & R.J. DAVIDSON. 1997. Prefrontal brain asymmetry: a biological substrate of the behavioral approach and inhibition systems. *Psychol. Sci.* **8**: 204–210.
76. TOMARKEN, A.J. & R.J. DAVIDSON. 1994. Frontal brain activation in repressors and non-repressors. *J. Abnorm. Psychol.* **103**: 339–349.
77. TOMARKEN, A.J., R.J. DAVIDSON, R.W. WHEELER & R.C. DOSS. 1990. Individual differences in anterior brain asymmetry and fundamental dimensions of emotion [abstract]. *Psychophysiology* **27**: S70.
78. WHEELER, R.E., R.J. DAVIDSON & A.J. TOMARKEN. 1993. Frontal brain asymmetry and emotional reactivity: a biological substrate of affective style. *Psychophysiology* **30**: 82–89.
79. KANG, D.H., *et al.* 1991. Frontal brain asymmetry and immune function. *Behav. Neurosci.* **105**: 860–869.
80. DAVIDSON, R.J., *et al.* 1999. Individual differences in prefrontal activation asymmetry predict natural killer cell activity at rest and in response to challenge. *Brain Behav. Immun.* **13**: 93–108.
81. LARSON, C.L., S.K. SUTTON & R.J. DAVIDSON. 1998. Affective style, frontal EEG asymmetry and the time course of the emotion-modulated startle [abstract]. *Psychophysiology* **35**: S52.
82. HENRIQUES, J.B. & R.J. DAVIDSON. 1990. Regional brain electrical asymmetries discriminate between previously depressed subjects and healthy controls. *J. Abnorm. Psychol.* **99**: 22–31.
83. DAVIDSON, R.J., N.H. KALIN & S.E. SHELTON. 1992. Lateralized effects of diazepam on frontal brain electrical asymmetries in rhesus monkeys. *Biol. Psychiatry* **32**: 438–451.
84. DAVIDSON, R.J., N.H. KALIN & S.E. SHELTON. 1993. Lateralized response to diazepam predicts temperamental style in rhesus monkeys. *Behav. Neurosci.* **107**: 1106–1110.
85. KALIN, N.H., *et al.* 1998. Asymmetric frontal brain activity, cortisol, and behavior associated with fearful temperament in rhesus monkeys. *Behav. Neurosci.* **112**: 286–292.
86. BUSS, K., *et al.* 1997. EEG asymmetry, salivary cortisol, and affect expression: an infant twin study. *Psychophysiology* **34**: S25 Abstract.
87. ALLEN, J.J., *et al.* 1993. Regional electroencephalographic asymmetries in bipolar seasonal affective disorder before and after exposure to bright light. *Biol. Psychiatry* **33**: 642–646.
88. DAWSON, G., *et al.* 1992. Frontal lobe activity and affective behavior of infants of mothers with depressive symptoms. *Child Dev.* **63**: 725–737.
89. FOX, N.A. 1991. If it's not left, it's right: electroencephalograph asymmetry and the development of emotion. *Am. Psychol.* **46**: 863–872.

90. JACOBS, G.D. & D. SNYDER. 1996. Frontal brain asymmetry predicts affective style in men. *Behav. Neurosci.* **110**: 36.
91. HARMON-JONES, E. & J.J.B. ALLEN. 1997. Behavioral activation sensitivity and resting frontal EEG asymmetry: covariation of putative indicators related to risk for mood disorders. *J. Abnorm. Psychol.* **106**: 159–163.
92. WIEDEMANN, G., *et al.* 1999. Frontal brain asymmetry as a biological substrate of emotions in patients with panic disorders. *Arch. Gen. Psychiatry* **56**: 78–84.
93. HAGEMANN, D., *et al.* 1998. Frontal brain asymmetry and affective style: a conceptual replication. *Psychophysiology* **35**: 372–388.
94. REID, S.A., L.M. DUKE & J.J. ALLEN. 1998. Resting frontal electroencephalographic asymmetry in depression: inconsistencies suggest the need to identify mediating factors. *Psychophys* **35**: 389–404.
95. DAVIDSON, R.J. 1998. Anterior electrophysiological asymmetries, emotion and depression: conceptual and methodological conundrums. *Psychophysiology* **35**: 607–614.
96. MORGAN, M.A., L. ROMANSKI & J.E. LEDOUX. 1993. Extinction of emotional learning: contribution of medial prefrontal cortex. *Neurosci. Lett.* **163**: 109–113.
97. AMARAL, D.G., *et al.* 1992. Anatomical organization of the primate amygdaloid complex. *In* *The Amygdala: Neurobiological Aspects of Emotion, Memory and Mental Dysfunction*. J.P. Aggleton, Ed.: 1–66. Wiley-Liss. New York.
98. ABERCROMBIE, H.C., *et al.* 1996. Medial prefrontal and amygdalar glucose metabolism in depressed and control subjects: an FDG-PET study. *Psychophysiology* **33**: S17.
99. SCHAEFER, S.M., *et al.* 2000. Six-month test-retest reliability of MRI-defined PET measures of regional cerebral glucose metabolic rate in selected subcortical structures. *Hum. Brain Mapp.* **10**: 1–9.
100. WATSON, D., L.A. CLARK & A. TELLEGEN. 1988. Development and validation of brief measures of positive and negative affect: the PANAS scales. *J. Pers. Soc. Psychol.* **54**: 1063–1070.
101. ABERCROMBIE, H.C., *et al.* 1998. Metabolic rate in the right amygdala predicts negative affect in depressed patients. *Neuroreport* **9**: 3301–3307.
102. IRWIN, W., *et al.* 1998. Ratings of affective stimulus characteristics and measures of affective reactivity predict MR signal change in the human amygdala. *NeuroImage* **7**: S908.
103. CAHILL, L., *et al.* 1996. Amygdala activity at encoding correlated with long-term, free recall of emotional information. *Proc. Natl. Acad. Sci. USA* **93**: 8016–8021.
104. FURMARK, T., *et al.* 1997. The amygdala and individual differences in fear conditioning. *Neuroreport* **8**: 3957–3960.
105. KETTER, T.A., *et al.* 1996. Anterior paralimbic mediation of procaine-induced emotional and psychosensory experiences. *Arch. Gen. Psychiatry* **53**: 59–69.
106. KAHNEMAN, D. 1999. Objective happiness. *In* *Well-being: The Foundations of Hedonic Psychology*. E. Kahneman, E. Diener & N. Schwartz, Eds.: 3–25. Russell Sage Foundation. New York.
107. SCHWARTZ, N. & F. STRACK. 1999. Reports of subjective well-being: judgmental processes and their methodological implications. *In* *Well-being: The Foundations of Hedonic Psychology*. D. Kahneman, E. Diener & N. Schwartz, Eds.: 61–84. Russell Sage Foundation. New York.
108. MAQUET, P., *et al.* 1996. Functional neuroanatomy of human rapid-eye-movement sleep and dreaming. *Nature* **383**: 163–166.
109. DREVETS, W.C., *et al.* 1992. A functional anatomical study of unipolar depression. *J. Neurosci.* **12**: 3628–3641.
110. DAVIDSON, R.J., D.C. JACKSON & N.H. KALIN. 2001. Emotion, plasticity, context and regulation. *Psychol. Bull.* **126**: 890–906.
111. DAVIDSON, R.J., K.M. PUTNAM & C.L. LARSON. 2000. The neurobiology of emotion and emotion regulation in relation to violence. *Science* **289**: 591–594.