Psychophysiology, 40 (2003), 838–848. Blackwell Publishing Inc. Printed in the USA.

DOI: 10.1111/1469-8986.00121

EARLY CAREER AWARD

Clarifying the emotive functions of asymmetrical frontal cortical activity

EDDIE HARMON-JONES
Department of Psychology, University of Wisconsin–Madison, Madison, Wisconsin 53706, USA

Abstract

Asymmetrical activity over the frontal cortex has been implicated in the experience and expression of emotions and motivations. Explanations of the research have suggested that relatively greater left frontal activity is associated with positive affect and/or approach motivation, and that relatively greater right frontal activity is associated with negative affect and/or withdrawal motivation. In past research, affective valence and motivational direction were confounded, as only positive (negative) affects that were associated with approach (withdrawal) motivation were examined. Consequently, this research is unable to address whether asymmetrical frontal activity is associated with affective valence, motivational direction, or some combination of valence and motivation. In this article, I review research on the emotion of anger, a negative emotion often associated with approach motivation, that suggests that asymmetrical frontal cortical activity is due to motivational direction and not affective valence. Methodological and theoretical implications for the study of the frontal asymmetry specifically, and for emotion and motivation more generally, are discussed.

Descriptors: Emotion, Motivation, Anger, Asymmetrical frontal cortical activity

Scientific interest in the neural structures involved in different aspects of emotions has flourished in recent years. A number of methodologies have suggested that the left and right frontal cortical regions are asymmetrically involved in the expression and experience of emotion. Much of this research has suggested that left frontal cortical activity is associated with positive emotions and approach motivation, and right frontal cortical activity is associated with negative emotions and withdrawal motivation. Thus, several scientists have posited that relatively greater activity in the left frontal cortical region is more psychologically and physically healthy than relatively less activity in the same region (e.g., Davidson, 1998; Fox, Henderson, Rubin, Calkins, & Schmidt, 2001). This theoretical model is widely accepted, even among scientists who are not involved in research on frontal cortical asymmetry (e.g., Oatley & Jenkins, 1996; Zajonc & McIntosh, 1992). Indeed, this theoretical conclusion regarding the association of left frontal activity with more positive outcomes has been applied in creating treatment strategies (e.g., biofeedback) for depression (e.g., Baehr, Rosenfeld, & Baehr, 1997; Rosenfeld, Cha, Blair, & Gotlib, 1995).

However, increased left frontal activity may not always be beneficial. Although past research has found left frontal activity to be associated with positive emotions (e.g., Davidson, Ekman, Saron, Senulis, & Friesen, 1990; Tomarken, Davidson, Wheeler, & Doss, 1992), recent research has indicated that these findings resulted because of confounds between approach motivation and positive emotional valence (e.g., Harmon-Jones, in press-a; Harmon-Jones & Allen, 1998; Harmon-Jones & Sigelman, 2001). This recent research has not assumed that approach motivation is always associated with positive emotions. That is, approach motivations can be associated with negative subjective feelings and negative consequences. For example, anger is often associated with approach motivation even though it has a negative valence (i.e., it is experienced negatively and often produces negative consequences). After briefly reviewing research on the relationship between emotion/motivation and asymmetrical frontal cortical activity, I will then describe

This research was funded in part by grants from the National Institute of Mental Health (1 R03 MH60747-01), the National Science Foundation (BCS-9910702), the Wisconsin/Hilldale Undergraduate/Faculty Research Fund, and the University of Wisconsin Graduate School. Thanks to Cindy Harmon-Jones for providing comments on earlier drafts of this article and for helping with several other aspects of the research process. Thanks also to the individuals who assisted with the studies reported in this article: Jon Sigelman, Dirk Wilker, Ziggy Bialzik, Andy Mulder, Greg Kant, John Allen, Kate Vaughn, Sheri Mohr, Amanda Bohlig, Leonard Lee, Lyn Abramson, Mike Hogan, Dave Amodio, Jim Shah, Paige Brazy, Dan Ward, Chris Kolstad, Michael Leggadrio, and Matt Poelzer.

Address reprint requests to: Eddie Harmon-Jones, Department of Psychology, University of Wisconsin, 1202 West Johnson Street, Madison, Wisconsin 53706, USA. E-mail: eharmonj@wisc.edu.

theoretical explanations of the research results. Finally, I will review recent research on anger that supports one theoretical model over the others.

Asymmetrical Frontal Cortical Activity and Emotion

Much of the research on asymmetrical frontal cortical activity has used alpha frequency band activity derived from the electroencephalograph (EEG). Alpha activity correlates inversely with cortical activity (for a review, see Lindsley & Wicke, 1974). More recent research has revealed that alpha power is inversely related to regional cortical activity using hemodynamic measures (Cook, O'Hara, Uijtdehaage, Mandelkern, & Leuchter, 1998) and behavioral tasks (Davidson, Chapman, Chapman, & Henriques, 1990).

Examinations of the Relationship between Indices of Trait Affect/Motivation and Resting EEG

Much research has examined the relationship of baseline, resting frontal cortical activity, and other trait measures of emotion. Research has suggested that resting asymmetrical frontal cortical activity behaves like a trait, in that it demonstrates acceptable test–retest reliability and it is internally consistent within resting baseline sessions (e.g., Tomarken, Davidson, Wheeler, & Kinney, 1992).

Depression. Depression has been found to relate to resting frontal asymmetrical activity, with depressed individuals showing relatively less left than right frontal cortical activity (Gotlib, Ranganath, & Rosenfeld, 1998; Henriques & Davidson, 1991; Jacobs & Snyder, 1996; Schaffer, Davidson, & Saron, 1983). Moreover, relatively less left frontal activity has been found in individuals who were previously clinically depressed but were in remission status (Allen, Iacono, Depue, & Arbisi, 1993; Henriques & Davidson, 1990).

Positive and negative affect. Other research has revealed that trait positive affect is associated with greater left than right frontal cortical activity, whereas trait negative affect is associated with greater right than left frontal cortical activity (e.g., Tomarken, Davidson, Wheeler, & Doss, 1992). In this past research, trait positive affect and negative affect were assessed using the Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988). These affects are best described as activated positive affect and activated negative affect (Watson, Wiese, Vaidya, & Tellegen, 1999), as the measures include words such as interested and active on the positive affect scale, and afraid and distressed on the negative affect scale. As such, the PANAS may be tapping affects that indicate high levels of approach and withdrawal motivation as well (see Watson, 2000, who expressed a similar view).

Behavioral approach. Other research has found that trait behavioral approach sensitivity (BAS) relates to greater left than right frontal cortical activity. For instance, Harmon-Jones and Allen (1997) found that trait BAS related to relative left frontal activity during a baseline recording session (see also Coan & Allen, 2003; Sutton & Davidson, 1997). BAS was assessed using Carver and White's (1994) questionnaire, which assesses the self-reported tendency to respond with drive toward incentives, to be highly responsive to rewards, and the tendency to seek out fun activities. This research was important in that it demonstrated that resting frontal asymmetrical activity related to dispositional approach motivation tendencies. Other, more recent research has conceptually replicated these findings using a lexical decision task to assess approach (and withdrawal) motivational tendencies (Amodio, Shah, Sigelman, Brazy, & Harmon-Jones, in press). However, BAS has also been found to relate to positive affect (e.g., Carver & White, 1994), rendering it difficult to know whether approach motivation or positive affect causes increased left frontal activity.

Examinations of the Relationship between Resting EEG and Responses to Emotion-Eliciting Stimuli

Resting baseline frontal asymmetrical activity has also been found to predict emotional responses. Individuals with relatively greater right than left frontal activity during baseline recording sessions exhibit larger negative affective responses to negative emotion-inducing films (fear and disgust). They also exhibit smaller positive affective responses to positive emotion-inducing films (happiness; Tomarken, Davidson, & Henriques, 1990; Wheeler, Davidson, & Tomarken, 1993). Other research has found that relative right frontal activity at baseline predicts crying in response to maternal separation in 10-month-old infants (Davidson & Fox, 1989).

In addition, relative right frontal activity at baseline predicts attitudinal responses to merely exposed stimuli (Harmon-Jones & Allen, 2001). Compared to individuals with less relative right frontal activity, individuals with greater relative right frontal activity reported more liking of experimentally familiarized photographs of individuals relative to novel individuals. According to Zajonc (1968, 1998), repeated exposure to stimuli may reduce anxiety toward novel stimuli. Thus, individuals with greater right frontal activity may have been more anxious (e.g., Fox et al., 2001) toward the novel stimuli and this anxiety may have been reduced after repeated, nonreinforced exposure, causing the increase in liking of the stimuli.

Providing even stronger causal evidence for the role of asymmetrical frontal cortical activity in emotional responses, another experiment used biofeedback to manipulate left and right frontal activity and then observed the effects of this manipulation on emotional responses (Allen, Harmon-Jones, & Cavender, 2001). In this experiment, participants received biofeedback designed to increase right frontal alpha relative to left frontal alpha or to receive training in the opposite direction. Five consecutive days of biofeedback provided signals of reward (300 Hz reward tone) or nonreward (150 Hz nonreward tone) depending on whether the difference between right and left frontal alpha exceeded a criterion value. Participants were simply asked to try to keep the reward tone playing. Tones were played continuously. Systematic alterations of frontal EEG asymmetry were observed as a function of biofeedback. Moreover, subsequent self-reported affect and facial muscle activity in response to happy, neutral, and sad emotionally evocative film clips were significantly influenced by the direction of biofeedback training. These results suggest that the frontal asymmetry can be altered using biofeedback training and that this alteration can affect emotional responses, with an increase in left frontal cortical activity causing more happy emotional facial expressions and an increase in relative right frontal cortical activity causing more sad emotional facial expressions across all films.

Examinations of EEG Activity during Exposure to Emotionally Evocative Situations

Research has also demonstrated that asymmetrical frontal cortical activity is associated with state emotional responses.

For instance, Davidson and Fox (1982) found that 10-month-old infants exhibited increased left frontal activation in response to a film clip of an actress generating a happy facial expression as compared to a sad facial expression. Frontal cortical activity has been found to relate to facial expressions of positive and negative emotions, as well. For example, Ekman and Davidson (1993) found increased left frontal activation during voluntary facial expressions of smiles of enjoyment as compared to voluntary facial expressions of smiles not associated with enjoyment. More recently, Coan, Allen, and Harmon-Jones (2001) found that voluntary facial expressions of fear, as compared to control expressions, produced relatively less left frontal activity.

Explanations of the Relationship between Asymmetrical Frontal Cortical Activity and Emotion

Three conceptual models have been advanced to explain the observed results. The first model has posited that the left frontal cortical region is involved in the experience and expression of positive emotions and that the right frontal cortical region is involved in the expression and experience of negative emotions (e.g., Ahern & Schwartz, 1985; Gotlib et al., 1998; Heller, 1990; Heller & Nitschke, 1998; Silberman & Weingartner, 1986). Most of the results can be explained with this model, which I refer to as the *valence model*.

A second model has posited that the left frontal cortical region is involved in the experience and expression of approach-related emotions and that the right frontal cortical region is involved in experience and expression of withdrawal-related emotions (Davidson, Jackson, & Kalin, 2000; Fox, 1991; Harmon-Jones & Allen, 1997; Sutton & Davidson, 1997). Again, the obtained results can be accommodated by this model, which I refer to as the *motivational direction model*. That is, the emotions that have been examined in the research are all associated with approach or withdrawal motivation.

A third model has posited that the left frontal cortical region is involved in the expression and experience of positive, approach-related emotions and that the right frontal cortical region is involved in the expression and experience of negative, withdrawal-related emotions (Davidson, 1998; Tomarken & Keener, 1998). The obtained results can also be accommodated by this model, which I refer to as the *valenced motivation model*. That is, the positive affects that have been examined in the research are all associated with approach motivation, and the negative affects that have been examined are all associated with withdrawal motivation.

Because the previously conducted research confounded the valence of the emotion with the direction of motivation, it is unable to address whether the frontal asymmetry reflects the valence of the emotion, the direction of the motivation, or a combination of valence and motivation. On many occasions, positive emotion is associated with approach-related motivation, whereas negative emotion is associated with withdrawal-related motivation. Indeed, most contemporary theories of emotion posit that positive emotion is always associated with approach motivation and that negative emotion is always associated with withdrawal motivation (e.g., Watson, 2000; for a different point of view, see Carver, 2001). However, not all emotions behave in accord with this presumed relationship between the valence of emotion and direction of motivation. Anger is an example of an emotion that does not conform to this relationship. That is, anger is negative in valence (e.g., Lazarus, 1991; Watson et al., 1999), even though it often evokes approach motivation (e.g., Berkowitz, 1999; Darwin, 1872/1965; Plutchik, 1980; Young, 1943).

Anger is considered a negative emotion by most emotion theorists. Why? At least three definitions of emotional valence exist (Lazarus, 1991). Emotions can be regarded as positive or negative (1) *because of the conditions that evoked the emotion*; (2) *because of the emotion's adaptive consequences*; or (3) *because of the emotion's subjective feel*. Anger can be viewed as negative when considering the conditions that evoked the emotion, because anger is evoked by aversive events. Anger could be viewed as either positive or negative when considering its adaptive consequences, depending upon the outcome of a particular situation. Finally, anger could be viewed as either positive or negative when considering the subjective feel or evaluation of the emotion, depending on whether an individual likes or dislikes the subjective experience of anger.

Some scientists focus on the stimulus conditions when defining emotion (e.g., a perceived offense causes anger), whereas others focus on the responses evoked when defining emotion (e.g., anger involves certain physiological changes, behavioral expressions, and subjective feelings). The stimulus-based definitions indicate that the individual's evaluation of the stimulus causing the emotion determines the valence of the emotion (Lazarus, 1991). With these definitions, the beneficial or harmful person–environment relationship is considered the most important and frequent way of distinguishing positive from negative emotions. Thus, according to the stimulus-based definition, anger is a negative emotion.

Response-based definitions indicate that the individual's subjective evaluation of the feeling determines valence. As Jung (1923, p. 544) suggested, "Feeling is also a kind of judging, differing, however, from an intellectual judgment, in that it does not aim at establishing an intellectual connection but is solely concerned with the setting up of a subjective criterion of acceptance or rejection." When anger is examined as a subjective experience, most individuals evaluate it negatively (though there are individual differences in these evaluations; see below).

Because anger is considered a negative but approach-oriented emotion, it provides an optimal testing ground for ascertaining the precise emotional/motivational functions of asymmetrical frontal activity. According to the various models set forth, two competing predictions can be offered for the relationship between anger and asymmetrical frontal activity: (1) If the frontal asymmetry reflects emotional valence, then anger should be associated with increased right frontal activity; (2) if the frontal asymmetry reflects motivational direction, then anger should be associated with increased left frontal activity.

Anger and Approach Motivation

Before reviewing the research on anger and asymmetrical frontal activity, it is important to consider whether anger is associated with approach motivation. Several lines of research suggest that anger elicits behavioral approach or approach motivation tendencies.

Behavioral Evidence

In the animal behavior literature, a distinction has been made between offensive or irritable aggression and defensive aggression (Flynn, Vanegas, Foote, & Edwards, 1970; Moyer, 1976). It has been posited that irritable aggression results from anger and

that pure irritable aggression "involves attack without attempts to escape from the object being attacked" (Moyer, 1976, p. 187). A number of aggression researchers have suggested that offensive aggression is associated with anger, attack, and no attempts to escape, whereas defensive aggression is associated with fear, attempts to escape, and attack only if escape is impossible (Blanchard & Blanchard, 1984; Lagerspetz, 1969; Moyer, 1976). In demonstrating that organisms evidence offensive aggression and that this is an approach behavior, Lagerspetz (1969) found that under certain conditions, mice would even cross an electrified grid to attack another mouse.[1]

Subsequent to frustrating events, anger may maintain and increase task engagement and approach motivation. Consistent with this hypothesis, Lewis, Sullivan, Ramsay, and Alessandri (1992) found that infants who expressed anger during extinction maintained interest during subsequent relearning, whereas infants who expressed sadness during extinction evidenced decreased interest during relearning.

Subjective Evidence

Also supportive of the idea that anger is associated with approach motivation is research testing the conceptual model that integrated reactance theory with learned helplessness theory (Wortman & Brehm, 1975). According to this model, how individuals respond to uncontrollable outcomes depends on their expectation of being able to control the outcome and the importance of the outcome. When an individual expects to be able to control outcomes that are important, and those outcomes are found to be uncontrollable, psychological reactance should be aroused. Thus, for individuals who initially expect control, the first few bouts of uncontrollable outcomes should arouse reactance, a motivational state aimed at restoring control. After several exposures to uncontrollable outcomes, these individuals should become convinced that they cannot control the outcomes and should show decreased motivation (i.e., learned helplessness). In other words, reactance will precede helplessness for individuals who initially expect control. In one study testing this model, individuals who exhibited angry feelings (a manifestation of reactance) in response to one unsolvable problem had better performance and more approach motivation on a subsequent cognitive task than did participants who exhibited less anger (Mikulincer, 1988).

Other research has revealed that state anger relates to high levels of self-assurance, physical strength, and bravery (Izard, 1991), inclinations associated with approach motivation. In addition, Lerner and Keltner (2001) found that anger (both trait and state) is associated with optimistic expectations, whereas fear is associated with pessimistic expectations. Moreover, happiness was associated with optimism, making anger and happiness appear more similar to each other in their relationship with optimism than fear and anger. Although Lerner and Keltner interpreted their findings as being due to the appraisals associated with anger, it seems equally plausible that the approach motivational character of anger may have caused the relationship of anger and optimism. That is, anger creates optimism because anger engages the approach motivational system and produces greater optimistic expectations.

Hormonal and Physiological Evidence

Further evidence supporting the conceptualization of anger as involving approach and not withdrawal comes from research on testosterone, which has been found to be associated with anger and aggression in humans (e.g., Olweus, 1986). In this research, testosterone treatments have been found to decrease withdrawal (fear) responses in a number of species (e.g., Boissy & Bouissou, 1994; Vandenheede & Bouissou, 1993). Other research has demonstrated that damage to the amygdala, a cortical region involved in defensive behavior, has no effect on offensive aggression but reduces reactivity to nonpainful threat stimuli (Blanchard & Takahashi, 1988; Busch & Barfield, 1974).

Individual Differences Evidence

Other evidence consistent with the idea that anger is associated with an approach orientation comes from research on bipolar disorder. The emotions of euphoria and anger often occur during manic phases of bipolar disorder (Cassidy, Forest, Murry, & Carroll, 1998; Depue & Iacono, 1989; Tyrer & Shopsin, 1982). Both euphoria and anger may be approach-oriented processes, and a dysregulated or hyperactive approach system may underlie mania (Depue & Iacono, 1989; Fowles, 1993). Additional research suggests that hypomania/mania involves increased left frontal cortical activity and approach motivational tendencies. That is, individuals who have suffered damage to the right frontal cortex are more likely to evidence mania (for review, see Robinson & Downhill, 1995). Thus, this research is consistent with the view that mania may be associated with increased left frontal activity and increased approach tendencies, because the approach motivation functions of the left frontal cortex are released and not restrained by the withdrawal system in the right frontal cortex. Furthermore, lithium carbonate, a treatment for bipolar disorder, reduces aggression (Malone, Delaney, Luebbert, Cater, & Campbell, 2000), suggesting that anger and aggression correlate with other symptoms of bipolar disorder.

In addition, trait anger has been found to relate to high levels of assertiveness and competitiveness (Buss & Perry, 1992). Finally, in two separate studies, trait anger, as measured by the Buss and Perry anger subscale, related to trait BAS, as measured by Carver and White's (1994) questionnaire (Harmon-Jones, in press-a). Moreover, trait BAS was associated with increased trait aggression. Whereas individuals with relatively low left frontal activation are at risk for deficits in approach motivation and depression, those with relatively high left frontal activation may evidence excessive approach motivation, leading to increased anger and offensive aggression.

Examining the Relationship between Anger and Asymmetrical Frontal Activity

Frontal Asymmetrical Activity and Trait Anger

In all past research on the frontal asymmetry, the valence of the emotion (positive vs. negative) was confounded with the direction of the motivation (approach vs. withdrawal). To address this confound, Harmon-Jones and Allen (1998) assessed the relationship between resting frontal asymmetrical activity and anger, an emotion that is negatively valenced but approach

[1]As this evidence demonstrates, not all forms of aggression are necessarily due to approach motivation. Thus, approach-related aggression needs to be differentiated from other types of aggression. Moreover, it is important to note motivational tendencies may not lead directly to similar behavior (e.g., approach-related anger may not lead directly to approach-related aggression). That is, self-regulation or the environment may impede the behavioral expression of the motivational tendency. However, if there is approach-related aggression, it is fairly safe to assume that anger caused it (Berkowitz, 1993).

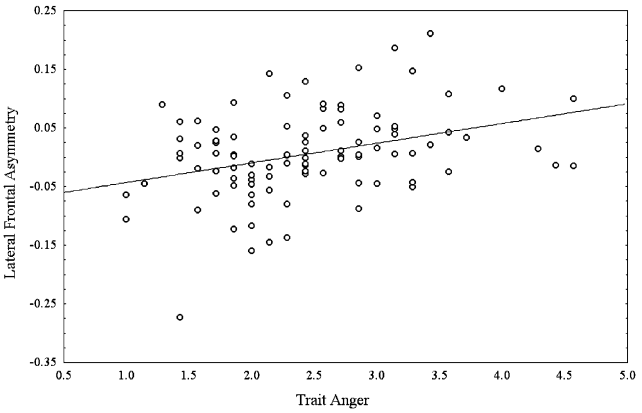

Figure 1. Scatterplot of the relationship between lateral frontal asymmetry and trait anger. Greater scores indicate greater relative left frontal activity and greater trait anger.

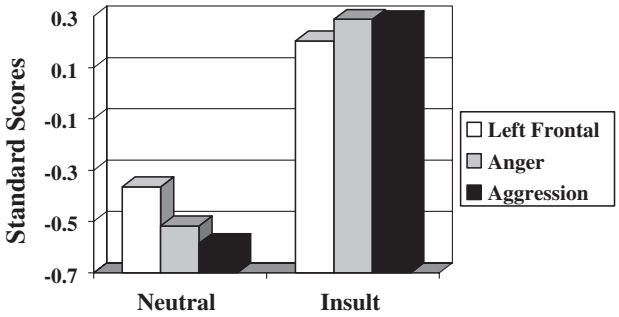

Figure 2. Means (expressed in standardized scores) for relative left frontal activity, self-reported anger, and behavioral aggression as a function of condition. Greater scores indicate greater relative left frontal activity, anger, and aggression. Scores for each of the measures differ significantly between conditions.

related. We found that trait anger related to increased left frontal activity and decreased right frontal activity.

More recently, Harmon-Jones (in press-b) addressed an alternative explanation for the results of Harmon-Jones and Allen (1998). The alternative explanation suggested that persons with high levels of trait anger might regard anger as a positive emotion, and this positive feeling or attitude toward anger could be responsible for anger being associated with relative left frontal activity. Anger might have been regarded positively because of the subjective feel or evaluation of the emotion. After conducting three studies that developed a valid and reliable assessment of attitude toward anger, a study was conducted in which resting baseline asymmetrical activity was related to trait anger and attitude toward anger.

As shown in Figure 1, trait anger related to relative left frontal activity (at mid-frontal and lateral frontal sites). However, relative left frontal activity did not relate to attitude toward anger. Moreover, regression analyses in which the frontal asymmetry was predicted simultaneously by trait anger and attitude toward anger revealed that anger significantly predicted relative left frontal activity, but that attitude toward anger did not. Additional analyses revealed that trait anger related to increased left frontal and decreased right frontal activity, separately, after controlling for whole head alpha power and alpha power at the homologous electrode, an analysis strategy previously suggested by Wheeler et al. (1993). These relationships between anger and left/right frontal activity remained when attitude toward anger was entered into the regression equations, suggesting that attitude toward anger did not mediate the relationship of anger and left or right frontal activity.

Frontal Asymmetrical Activity and State Anger

Although the above-mentioned trait anger research supports the motivational direction model over the other two models, it is based solely on correlational evidence, and is thus subject to all interpretational problems associated with correlational evidence. To address these limitations, research was conducted in which anger was manipulated and its effects on asymmetrical frontal cortical activity were observed.

State anger and frontal cortical activity. Harmon-Jones and Sigelman (2001) conducted an experiment to assess whether situationally induced anger would increase relative left frontal activity. Participants were randomly assigned to a condition in which another person insulted them or to a condition in which another person treated them in a neutral manner. Immediately following the treatment, EEG was collected. As predicted, individuals who were insulted evidenced greater relative left frontal activity than individuals who were not insulted. Moreover, they reported being more angry and behaved more aggressively toward the person who insulted them (see Figure 2). Additionally, regression analyses revealed that relative left-frontal activation was associated with more anger and aggression in the condition in which anger was evoked. This research thus provides the first demonstration of a relationship between state anger and relative left frontal activation, a result predicted by models that posit that the frontal asymmetry reflects motivational direction but not predicted by models that posit that the frontal asymmetry reflects emotional valence.

Frontal asymmetry and cardiovascular reactivity. A recent study was designed to extend the results of the former by examining the relationship between frontal asymmetrical activation and cardiovascular reactivity. Past research has found that anger is related to exaggerated cardiovascular reactivity, that is, elevations in systolic blood pressure (SBP), diastolic blood pressure (DBP), and heart rate (HR). Longitudinal research has found that trait anger predicts coronary heart disease morbidity and mortality (Barefoot, Dodge, Peterson, Dahlstrom, & Williams, 1989), and it has been suggested that the elevated cardiovascular reactivity associated with anger may lead to cardiovascular disease and death (Suarez & Williams, 1989).

Increased relative left frontal activation may be associated with increased cardiovascular reactivity because of an increase in active coping in an attempt to deal with the situation (Obrist, 1981), or because asymmetrical cerebral activation is transmitted through the autonomic nervous system to cause a lateralized imbalance in sympathetic input to the heart, and a significantly lateralized induction could increase cardiovascular reactivity (Lane & Jennings, 1995; Lane & Schwartz, 1987). This latter explanation, the brain–heart laterality hypothesis, proposes that emotional arousal has the potential to cause ventricular fibrillation and sudden cardiac death in individuals with organic heart disease via asymmetrical activation of the cerebral hemispheres. Assessing the relationship between asymmetrical cortical activity and cardiovascular reactivity thus tests a neurophysiological mechanism by which anger relates to cardiovascular reactivity.

In our study, participants were insulted and their BP, HR, and EEG activity were measured after the insult (these variables were also measured at baseline; Harmon-Jones & Sigelman, 2003). Results revealed that insult-related frontal asymmetry related to cardiovascular reactivity while controlling for baseline frontal asymmetry and cardiovascular reactivity. That is, relative left frontal activity was associated with greater cardiovascular reactivity (for HR, $\beta = .55$, $t(7) = 2.02$, $p < .05$, *partial* $r = .61$; for SBP, $\beta = .36$, $t(7) = 1.57$, $p = .08$, *partial* $r = .51$; for DBP, $\beta = .61$, $t(7) = 4.00$, $p < .005$, *partial* $r = .83$).

The effect of coping potential on anger-related left frontal cortical activity. The research presented thus far was based on the assumption that anger is associated with approach motivation. However, this assumption was not fully tested in the past research, as motivational intensity was never experimentally varied. In the past experiments, all participants were provided with approach behavioral opportunities before anger was induced. To assess whether approach motivational intensity causes variations in left frontal activity, we designed an experiment in which approach motivational intensity was manipulated and EEG was assessed. Based on past research that has suggested that the expectation of being able to cope with situational demands influences motivational intensity (e.g., Brehm & Self, 1989; Wright, 1996), we predicted that individuals who expected to be able to rectify an anger-evoking event would evidence greater approach motivational intensity (and hence left frontal activity) than individuals who expected that they could not rectify the same anger-evoking event (Harmon-Jones, Sigelman, Bohlig, & Harmon-Jones, 2003).

In the experiment, first- or second-year university students opposed to a tuition increase were exposed to an editorial that argued for a tuition increase. Before hearing the editorial, participants were informed that the tuition increase would definitely occur in 2 years (*action-impossible condition*) or they were informed that it may occur in two years (*action-possible condition*). Thus, the conditions differed with regard as to whether it was possible for participants to act to change the likelihood that tuition will be increased. In addition, participants in the action-possible condition were told that petitions were being circulated to attempt to prevent the increase. After participants read this condition information and listened to a pilot radio broadcast in which a speaker argued forcefully for a tuition increase, EEG and self-reported emotions were measured. Finally, participants in the action-possible condition were given an opportunity to sign a petition against the tuition increase and take additional petitions on which to collect other signatures.

Results indicated that participants who expected to engage in the approach-related action of signing a petition to ameliorate the tuition-increase situation (action-possible condition) evidenced an increase in left frontal activity and also evidenced greater left frontal activity than participants who expected to be unable to engage in approach-related action (see Figure 3). In addition, within the action-possible condition, participants who evidenced greater left frontal activity in response to the tuition increase message also evidenced greater self-reported anger, providing support for the idea that anger is often an approach-related emotional response. In the condition where action was not possible, greater left frontal activity did not relate to anger, suggesting that some forms of anger may not be related to approach motivation. Finally, within the action-possible condition, participants who evidenced greater left frontal activity in response to the tuition increase message were more likely to engage in behaviors that would reduce the possibility of the tuition increase; that is, they were more likely to sign the petition and to take petitions with them for others to sign. In other words, greater approach motivation was expressed as more action to correct the anger-arousing situation.

This research supports the hypothesis that the left frontal cortical region is involved in approach motivation rather than positive affect. In fact, the results revealed that the stimulus that caused an increase in anger also caused a decrease in happiness, one form of positive affect (see Figure 3). Moreover, the results suggest that the left frontal region is most accurately described as a region sensitive to approach motivational intensity. That is, it was only when anger was associated with an opportunity to behave in a manner to resolve the anger-producing event that participants evidenced the increased relative left frontal activity. It is important to note that the manipulation of coping potential affected relative left frontal cortical activity but did not affect self-reported anger (or other emotions). That is, both experimental conditions showed an increase in anger and a decrease in happiness relative to baseline.

Moderators of the Effects of Anger on Left Frontal Activity

Hypomania and depression. The research reviewed thus far suggests that approach-related anger is associated with increased left frontal activity. However, additional evidence demonstrating that individual differences in approach motivation moderates the effects of anger on relative left frontal activity would further support the connection between approach motivation, anger, and left frontal cortical activity. As reviewed earlier, hypomania/mania has been associated with increases in both anger and

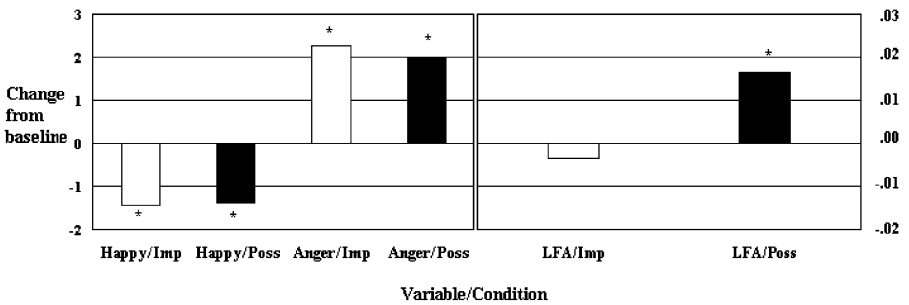

Figure 3. Mean change scores (after the editorial minus baseline) for self-reported happiness, self-reported anger, and relative left frontal activity. Greater scores indicate greater changes in happiness, anger, and relative left frontal activity. Bars with an asterisk over them differ significantly from baseline. Imp: action-impossible condition; Poss: action-possible condition.

euphoria, presumably because mania results from a hyperactive BAS (Depue & Iacono, 1989). Indeed, research has suggested that hypomania/mania is associated with increased self-reported BAS (Meyer, Johnson, & Carver, 1999). Because hypomania/mania is associated with increased activation of the BAS, individuals with hypomania/mania should respond to an anger-evoking situation with greater approach motivation and left frontal activity. In contrast, because unipolar depression is associated with decreased activation of the BAS, individuals with unipolar depression should respond to an anger-evoking situation with reduced approach motivation and left frontal activity. To assess these individual differences characteristics among a sample of unselected undergraduate students, we used the General Behavior Inventory, which was developed to identify individuals who are at risk for developing these disorders (Depue & Klein, 1988; Depue, Krauss, Spoont, & Arbisi, 1989). Participants were exposed to an anger-producing radio editorial and EEG was collected immediately after the editorial. Results indicated that proneness toward hypomania related to increased left frontal activity, and that proneness toward unipolar depression related to decreased left frontal activity (Harmon-Jones et al., 2002). In these analyses, resting, baseline relative left frontal activity was statistically controlled, suggesting that the effects were specific to when anger was aroused.

Empathy reduces anger-related left frontal activity. Although the reviewed evidence links approach anger to relative left frontal activity, it would be important to establish that manipulations that reduce angry approach behaviors also reduce relative left frontal activity. Such evidence would provide further evidence that the observed relationship between approach-related anger and left frontal activity is indeed due to approach motivation and not some other variable. To address this issue, we tested whether empathy would reduce the left frontal activity typically observed during approach-related anger. Past research has suggested that experiencing empathy for another individual can reduce aggression toward that individual (e.g., see review by Miller & Eisenberg, 1988). To assess whether empathy would reduce the left frontal activity typically associated with anger and aggression, college student participants were told that they and another student would be writing essays and evaluating each other based on the essays. Participants then wrote a persuasive essay. Then, the experimenter returned to the participants' room and handed them a folder containing a reading perspective, the other participant's essay, and a questionnaire.

The reading perspective instructions asked participants to remain completely objective (low empathy) or to try to imagine how the other person must feel (high empathy), as in much past empathy research (Batson, 1991, 1998; Harmon-Jones, Peterson, & Vaughn, 2003). The participant then read the essay ostensibly written by the other participant. The essay described the difficulties the other ostensible participant was having with Multiple Sclerosis.

Following the reading of the essay, participants received an evaluation of their essay ostensibly written by the other participant. The evaluation contained either neutral ratings and comments (no insult) or insulting ratings and comments (insult). Immediately after feedback manipulation, EEG was collected. Then, participants completed questionnaires assessing impressions of the other participant and emotions.

In addition to conceptually replicating and extending the results of Harmon-Jones and Sigelman (2001), the experiment revealed that state anger caused an increase in left frontal activity as well as a decrease in right frontal activity (p's $< .05$; see Figure 4). The previous state anger experiment was unable to examine separate estimates of left and right frontal cortical activity because too few electrodes were available for such estimates (e.g., Wheeler et al., 1993). More importantly, the insult did not evoke greater left frontal activity (and lesser right frontal activity) when high levels of empathy were first aroused for the insulting person. These results were revealed in a series of planned comparisons (p's $< .05$). In addition, the low empathy/insult condition produced greater left frontal activity (and lesser right frontal activity) than every other condition (p's $< .001$).

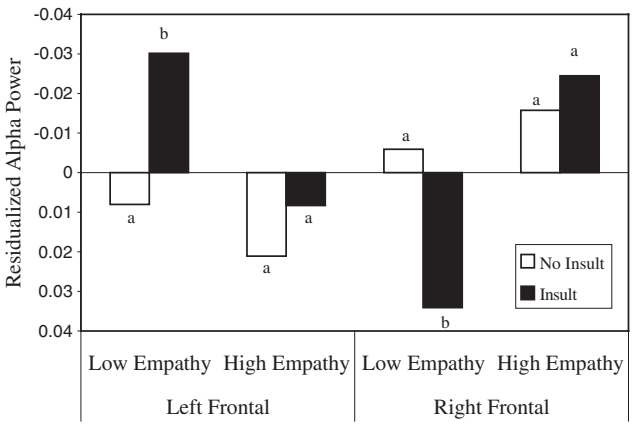

Figure 4. Left and right frontal activity as a function of insult × empathy condition. Scores are the mean residuals derived from regression analyses in which left (right) frontal alpha activity is predicted from whole head alpha power and alpha power at the homologous site. Note the inverted axis (as alpha is related inversely with cortical activity). Within hemisphere, scores with different subscripts differ at $p < .05$.

Moreover, high empathy/insult condition participants expressed less hostile attitudes toward the insulting person than did participants who did not first experience empathy for the insulter ($p < .05$). The experiment thus suggested that the reduction of angry approach inclinations can reduce left frontal activity. However, reported anger was increased in both insult conditions regardless of the level of empathy aroused, again suggesting that there may be types of anger not associated with approach motivation.

Other Evidence Consistent with the Anger-Left Frontal Hypothesis

In addition to the reviewed evidence, other research is consistent with the hypothesis that approach-related anger is associated with left frontal activity. For example, d'Alfonso, van Honk, Hermans, Postma, and de Haan (2000) used slow repetitive transcranial magnetic stimulation to inhibit the left or right prefrontal cortex. They found that inhibition of the right prefrontal cortex caused selective attention toward angry faces, whereas inhibition of the left prefrontal cortex caused selective attention away from angry faces. The increase in left prefrontal activity, which resulted from the inhibition of the right prefrontal cortex, led participants to attentionally approach angry faces, as in an aggressive confrontation. In contrast, the increase in right prefrontal activity led participants to attentionally avoid angry faces, as in a frightening confrontation. The interpretation of these results, which d'Alfonso et al. advanced, concurs with other research that has demonstrated that attention toward angry faces

is associated with high levels of self-reported anger and that attention away from angry faces is associated with high levels of cortisol, which is associated with fear (van Honk, Tuiten, de Haan, van den Hout, & Stam, 2001; van Honk et al., 1998, 1999). This evidence is particularly important because studies that manipulate the brain and observe the manipulation's effects on psychological outcomes provide stronger inferences about brain function than do studies that manipulate psychology and then observe brain function (e.g., Sarter, Berntson, & Cacioppo, 1996).

Questions about Anger and Frontal Asymmetry

Does anger always increase relative left frontal activity? From our recent research, the answer to this question appears to be no. That is, when individuals believed there was nothing they could do to rectify an angering situation, they still reported being angry but did not show an increase in relative left frontal activity (Harmon-Jones, Sigelman, et al., 2003). In addition, in the empathy and anger experiment, when individuals felt high levels of empathy for the insulting person, they reported being angry even though they did not evidence increased left frontal activity (Harmon-Jones, Vaughn, et al., 2003). Moreover, the empathy reduced their aggressive inclinations (i.e., hostile attitudes) toward the insulting person. Considering these studies and others, it would be most accurate to conclude that anger is associated with left frontal activity only when the anger is associated with approach inclinations.

This leads us to a second question: Does anger always involve approach inclinations? In several of the experiments reviewed, participants were given an opportunity to engage in approach-related behavior (e.g., aggression, signing petitions to prevent an injustice), and such opportunities caused an increase in left frontal activity. In contrast, when individuals were not given such opportunities, they did not evidence increased left frontal activity (Harmon-Jones, Sigelman, et al., 2003). Moreover, they did not evidence increased right frontal activity either, suggesting that this type of anger is not associated with increased withdrawal motivation as measured by right frontal activity.

However, it is possible that withdrawal-oriented anger does exist in some individuals in some situations. We have recently created a face-valid trait measure of withdrawal-oriented anger (example item: "I get angry and tend to withdraw from situations and people that make me angry."), and found that it relates positively to trait BIS ($r = .26$, $p < .01$) and negatively to trait BAS ($r = -.21$, $p < .01$), as measured by Carver and White's (1994) BIS/BAS questionnaire (Harmon-Jones, 2003). These findings stand in contrast to typical measures of trait anger, which relate positively to BAS and not to BIS (Harmon-Jones, in press-a). Taken together, these results suggest that typical measures of trait anger relate to approach-oriented dispositions (e.g., BAS), whereas trait withdrawal-oriented anger relates negatively to approach motivation and positively to a measure that may tap withdrawal tendencies (e.g., BIS). We are currently testing whether trait withdrawal anger is associated with increased right frontal activity.

Do individuals need to be in an approach-motivated state for increased left frontal activity to emerge during anger? In other words, is goal-blocking necessary for the increase in anger-related left frontal activity? Such a prediction would follow from the frustration-aggression hypothesis (Dollard, Doob, Miller, Mowrer, & Sears, 1939) and from more recent appraisal views of anger (e.g., Smith & Lazarus, 1993). However, although some of our experiments directly involved goal-blocking (e.g., Harmon-Jones, Sigelman, et al., 2003), others did not (e.g., Harmon-Jones & Sigelman, 2001). Moreover, that trait anger relates to relatively greater left frontal activity at baseline does not seem consistent with a goal-blocking interpretation (Harmon-Jones, in press-b). Although research has not been conducted to directly examine this issue, I would predict that individuals who are quiescent and are not currently approach motivated may become angry and consequently evidence approach-oriented motivation when they are subjected to certain aversive events (even if the event does not block obtaining a specific goal; see Berkowitz & Harmon-Jones, 2003). In other words, the angering event itself can engage the approach motivation system and left frontal cortical activity.

Related to the above issue, does the expectation of approach-related action need to be explicitly present for anger to increase relative left frontal activity? The experimental results of Harmon-Jones, Sigelman, et al. (2003) may suggest such a conclusion. However, it is important to note that the expectation of approach-related action was explicitly manipulated prior to the anger induction. It is possible that during other (or perhaps most) anger inductions, individuals lack awareness of whether or not approach-related action can take place. Under such circumstances, other factors, such as individual differences in learning histories, may determine the likelihood of implicitly "assuming" that approach-related action can take place. In fact, anger may automatically evoke approach motivational tendencies, except when some factor in the environment or person indicates that approach action would be completely futile.

An issue that arises in regional EEG research is the limited spatial resolution provided by EEG. Such limitations make it difficult to ascertain exactly where in the cortex specific effects emerge. However, the consistency of reviewed results across different studies using different methodologies points to a reliable phenomenon occurring in the frontal cortical regions that is worthy of further consideration. Moreover, other methodological approaches have produced results consistent with the reviewed findings suggesting that approach-related anger is associated with left frontal activity. For example, the research by d'Alfonso and colleagues (2000) suggested that manipulations of left frontal activity produced angry responses. Also consistent is the research that suggested that damage to the right frontal region causes mania (e.g., Robinson & Downhill, 1995). Finally, one study using positron emission tomography revealed an increase in the left frontal cortical region when individuals relived angry experiences (Dougherty et al., 1999). However, this research does not diminish the importance of conducting future research using other methods (PET, fMRI, lesions) to ascertain exactly which regions of the frontal cortex are involved in approach-related anger. In doing so, it would be important to attempt to evoke ecologically valid manifestations of anger that evoke action tendencies. Indeed, if asymmetrical frontal cortical activity is associated with motivational direction, as the current evidence strongly suggests, then it is imperative to use experimental manipulations that evoke motivations and actively involve the participant.

However, when one looks to other brain imaging research, one finds little in the way of research on the experience and/or expression of anger. The importance of the emotion of anger cannot be the factor inhibiting research on this emotion, as anger

is involved in much intrapersonal and interpersonal harm. Perhaps the lack of empirical attention to the emotion anger may be due to the difficulty of creating anger in the laboratory and then imaging the brain during this state. The experiments reviewed herein provide examples of methods that could be profitably used to evoke relatively ecologically valid anger in the laboratory. Collaborations between imagers and social psychologists who are experienced with high impact research that evokes emotional responses should facilitate the necessary research. In addition, individualizing the stimuli to create anger, as in Harmon-Jones, Sigelman, et al. (2003), provides another laboratory alternative that may evoke ecologically valid anger and may be of use in imaging studies.

Past models of asymmetrical frontal cortical activity predicted that relatively greater left frontal activity related to the *experience* and *expression* of certain dimensions of emotions or motivations. However, the reviewed research suggests that left frontal activity does not necessarily relate directly to the experience of anger (as measured through self-report). That is, relative left frontal activity relates with experienced anger only when the anger occurs in an approach-oriented setting. In Harmon-Jones, Sigelman, et al. (2003), when individuals believed they had an opportunity to rectify the anger-evoking situation, anger related to relative left frontal activity. However, when individuals believed they did not have an opportunity to rectify the anger-evoking situation, anger did not relate to relative left frontal activity, even though the level of anger was equivalent to that reported in the action-possible condition. These results suggest that the measure of the experience of anger did not differentiate between conditions, whereas the frontal asymmetry did. Perhaps the measure of the experience of anger was insufficiently sensitive to capture the differences in experience. Individuals in the action-possible condition may have felt *hopeful anger*, whereas those in the action-impossible condition may have felt *hopeless anger*. Future studies should refine the measures of self-reported experience to attempt to capture these subtle but important differences in emotional experience.

Conclusion

The research reviewed herein was designed to ascertain the emotive functions of asymmetrical frontal cortical activity. This research found that the frontal asymmetry is responsive to motivational direction and not affective valence. In addition to clarifying the understanding of the emotive functions of asymmetrical frontal cortical activity, the reviewed research points to the importance of considering that approach motivation is not always associated with positive emotions.

REFERENCES

Ahern, G. L., & Schwartz, G. E. (1985). Differential lateralization for positive and negative emotion in the human brain: EEG spectral analysis. *Neuropsychologia*, *23*, 745–755.

Allen, J. J. B., Harmon-Jones, E., & Cavender, J. (2001). Manipulation of frontal EEG asymmetry through biofeedback alters self-reported emotional responses and facial EMG. *Psychophysiology*, *38*, 685–693.

Allen, J. J., Iacono, W. G., Depue, R. A., & Arbisi, P. (1993). Regional electroencephalographic asymmetries in bipolar seasonal affective disorder before and after exposure to bright light. *Biological Psychiatry*, *33*, 642–646.

Amodio, D. M., Shah, J. Y., Sigelman, J. D., Brazy, P. C., & Harmon-Jones, E. (in press). Implicit regulatory focus associated with resting frontal cortical asymmetry. *Journal of Experimental Social Psychology*.

Baehr, E., Rosenfeld, J. P., & Baehr, R. (1997). The clinical use of an alpha asymmetry protocol in the neurofeedback treatment of depression: Two case studies. *Journal of Neurotherapy*, *2*, 10–23.

Barefoot, J. C., Dodge, K. A., Peterson, B. L., Dahlstrom, W. G., & Williams, R. B. (1989). The Cook–Medley hostility scale: Item content and ability to predict survival. *Psychosomatic Medicine*, *51*, 46–57.

Batson, C. D. (1991). *The altruism question: Toward a social-psychological answer*. Hillsdale, NJ: Lawrence Erlbaum.

Batson, C. D. (1998). Altruism and prosocial behavior. In D. T. Gilbert, S. T. Fiske, & G. Lindzey (Eds.), *The handbook of social psychology*, (4th ed., Vol. 2, pp. 282–316). Boston, MA: McGraw-Hill.

Berkowitz, L. (1993). *Aggression: Its causes, consequences, and control*. New York: McGraw-Hill.

Berkowitz, L. (1999). Anger. In T. Dalgleish & M. J. Power (Eds.), *Handbook of cognition and emotion* (pp. 411–428). Chichester, England: John Wiley and Sons.

Berkowitz, L., & Harmon-Jones, E. (2003). *Toward an understanding of the determinants of anger*. Manuscript submitted for publication.

Blanchard, D. C., & Blanchard, R. J. (1984). Affect and aggression: An animal model applied to human behavior. *Advances in the study of aggression*, *1*, 1–62.

Blanchard, D. C., & Takahashi, S. N. (1988). No change in intermale aggression after amygdala lesions which reduce freezing. *Physiology and Behavior*, *42*, 613–616.

Boissy, A., & Bouissou, M. F. (1994). Effects of androgen treatment on behavioral and physiological responses of heifers to fear-eliciting situations. *Hormones and Behavior*, *28*, 66–83.

Brehm, J. W., & Self, E. (1989). The intensity of motivation. In M. R. Rosenzweig & L. W. Porter (Eds.), *Annual review of psychology* (Vol. 40, pp. 109–131). Palo Alto, CA: Annual Reviews, Inc.

Busch, D. E., & Barfield, R. J. (1974). A failure of amygdaloid lesions to alter agonistic behavior in the laboratory rat. *Physiology and Behavior*, *12*, 887–892.

Buss, A. H., & Perry, M. (1992). The aggression questionnaire. *Journal of Personality and Social Psychology*, *63*, 452–459.

Carver, C. S. (2001). Affect and the functional bases of behavior: On the dimensional structure of affective experience. *Personality and Social Psychology Review*, *5*, 345–356.

Carver, C. S., & White, T. L. (1994). Behavioral inhibition, behavioral activation, and affective responses to impending reward and punishment: The BIS/BAS scales. *Journal of Personality and Social Psychology*, *67*, 319–333.

Cassidy, F., Forest, K., Murry, E., & Carroll, B. J. (1998). A factor analysis of the signs and symptoms of mania. *Archives of General Psychiatry*, *55*, 27–32.

Coan, J. A., & Allen, J. J. B. (2003). Frontal EEG asymmetry and the behavioral activation and inhibition systems. *Psychophysiology*, *40*, 106–114.

Coan, J. A., Allen, J. J. B., & Harmon-Jones, E. (2001). Voluntary facial expression and hemispheric asymmetry over the frontal cortex. *Psychophysiology*, *38*, 912–925.

Cook, I. A., O'Hara, R., Uijtdehaage, S. H. J., Mandelkern, M., & Leuchter, A. F. (1998). Assessing the accuracy of topographic EEG mapping for determining local brain function. *Electroencephalography and Clinical Neurophysiology*, *107*, 408–414.

d'Alfonso, A. A. L., van Honk, J., Hermans, E., Postma, A., & de Haan, E. H. F. (2000). Laterality effects in selective attention to threat after repetitive trancranial magnetic stimulation at the prefrontal cortex in female subjects. *Neuroscience Letters*, *280*, 195–198.

Darwin, C. (1872/1965). *The expression of the emotions in man and animals*. Chicago, IL: The University of Chicago Press.

Davidson, R. J. (1998). Anterior electrophysiological asymmetries, emotion, and depression: Conceptual and methodological conundrums. *Psychophysiology*, *35*, 607–614.

Davidson, R. J., Chapman, J. P., Chapman, L. J., & Henriques, J. B. (1990). Asymmetrical brain electrical activity discriminates between psychometrically-matched verbal and spatial cognitive tasks. *Psychophysiology*, *27*, 528–543.

Davidson, R. J., Ekman, P., Saron, C. D., Senulis, J. A., & Friesen, W. V. (1990). Approach–withdrawal and cerebral asymmetry: Emotional expression and brain physiology I. *Journal of Personality and Social Psychology*, *58*, 330–341.

Davidson, R. J., & Fox, N. A. (1982). Asymmetrical brain activity discriminates between positive and negative affective stimuli in human infants. *Science*, *218*, 1235–1236.

Davidson, R. J., & Fox, N. A. (1989). Frontal brain asymmetry predicts infants' response to maternal separation. *Journal of Abnormal Psychology*, *98*, 127–131.

Davidson, R. J., Jackson, D. C., & Kalin, N. H. (2000). Emotion, plasticity, context, and regulation: Perspectives from affective neuroscience. *Psychological Bulletin*, *126*, 890–909.

Depue, R. A., & Iacono, W. G. (1989). Neurobehavioral aspects of affective disorders. *Annual Review of Psychology*, *40*, 457–492.

Depue, R. A., & Klein, D. N. (1988). Identification of unipolar and bipolar affective conditions in nonclinical and clinical populations by the General Behavior Inventory. In D. L. Dunner, E. S. Gershon, & J. E. Barrett (Eds.), *Relatives at risk for mental disorder* (pp. 179–202). New York: Raven Press.

Depue, R. A., Krauss, S., Spoont, M. R., & Arbisi, P. (1989). General behavior inventory identification of unipolar and bipolar affective conditions in a nonclinical university population. *Journal of Abnormal Psychology*, *98*, 117–126.

Dollard, J., Doob, L., Miller, N., Mowrer, O., & Sears, R. (1939). *Frustration and aggression*. New Haven, CT: Yale University Press.

Dougherty, D. D., Shin, L. M., Alpert, N. M., Pitman, R. K., Orr, S. P., Lasko, M., Macklin, M. L., Fischman, A. J., & Rauch, S. L. (1999). Anger in healthy men: A PET study using script-driven imagery. *Biological Psychiatry*, *46*, 466–472.

Ekman, P., & Davidson, R. J. (1993). Voluntary smiling changes regional brain activity. *Psychological Science*, *4*, 342–345.

Flynn, J., Vanegas, H., Foote, W., & Edwards, S. B. (1970). Neural mechanisms involved in a cat's attack on a rat. In R. Whalen, R. F. Thompson, M. Verzeano, & N. Weinberger (Eds.), *The neural control of behavior* (pp. 135–173). New York: Academic Press.

Fowles, D. C. (1993). Behavioral variables in psychopathology: A psychobiological perspective. In P. B. Sutker & H. E. Adams (Eds.), *Comprehensive handbook of psychopathology* (2nd ed., pp. 57–82). New York: Plenum.

Fox, N. A. (1991). If it's not left, it's right. *American Psychologist*, *46*, 863–872.

Fox, N. A., Henderson, H. A., Rubins, K. H., Calkins, S. D., & Schmidt, L. A. (2001). Continuity and discontinuity of behavioral inhibition and exuberance: Psychophysiological and behavioral influences across the first four years of life. *Child Development*, *72*, 1–21.

Gotlib, I. H., Ranganath, C., & Rosenfeld, J. P. (1998). Frontal EEG alpha asymmetry, depression, and cognitive functioning. *Cognition and Emotion*, *12*, 449–478.

Harmon-Jones, E. (2003). *Construction of trait anger-withdrawal scale*. Unpublished raw data.

Harmon-Jones, E. (in press-a). Anger and the behavioural approach system. *Personality and Individual Differences*.

Harmon-Jones, E. (in press-b). On the relationship of anterior brain activity and anger: Examining the role of attitude toward anger. *Cognition and Emotion*.

Harmon-Jones, E., Abramson, L. Y., Sigelman, J., Bohlig, A., Hogan, M. E., & Harmon-Jones, C. (2002). Proneness to hypomania/mania or depression and asymmetrical frontal cortical responses to an anger-evoking event. *Journal of Personality and Social Psychology*, *82*, 610–618.

Harmon-Jones, E., & Allen, J. J. B. (1997). Behavioral activation sensitivity and resting frontal EEG asymmetry: Covariation of putative indicators related to risk for mood disorders. *Journal of Abnormal Psychology*, *106*, 159–163.

Harmon-Jones, E., & Allen, J. J. B. (1998). Anger and prefrontal brain activity: EEG asymmetry consistent with approach motivation despite negative affective valence. *Journal of Personality and Social Psychology*, *74*, 1310–1316.

Harmon-Jones, E., & Allen, J. J. B. (2001). The role of affect in the mere exposure effect: Evidence from psychophysiological and individual differences approaches. *Personality and Social Psychology Bulletin*, *27*, 889–898.

Harmon-Jones, E., Peterson, H., & Vaughn, K. (2003). The dissonance-inducing effects of an inconsistency between experienced empathy and knowledge of past failures to help: Support for the action-based model of dissonance. *Basic and Applied Social Psychology*, *25*, 69–78.

Harmon-Jones, E., & Sigelman, J. (2001). State anger and prefrontal brain activity: Evidence that insult-related relative left prefrontal activation is associated with experienced anger and aggression. *Journal of Personality and Social Psychology*, *80*, 797–803.

Harmon-Jones, E., & Sigelman, J. (2003). *Anger, asymmetrical frontal cortical activity, and cardiovascular reactivity*. Unpublished manuscript.

Harmon-Jones, E., Sigelman, J. D., Bohlig, A., & Harmon-Jones, C. (2003). Anger, coping, and frontal cortical activity: The effect of coping potential on anger-induced left frontal activity. *Cognition and Emotion*, *17*, 1–24.

Heller, W. (1990). The neuropsychology of emotion: Developmental patterns and implications for psychopathology. In N. L. Stein, B. Leventhal, & T. Trabasso (Eds.), *Psychological and biological approaches to emotion* (pp. 167–211). Hillsdale, NJ: Lawrence Erlbaum Associates.

Heller, W., & Nitschke, J. B. (1998). The puzzle of regional brain activity in depression and anxiety: The importance of subtypes and comorbidity. *Cognition and Emotion*, *12*, 421–447.

Henriques, J. B., & Davidson, R. J. (1990). Regional brain electrical asymmetries discriminate between previously depressed and healthy control subjects. *Journal of Abnormal Psychology*, *99*, 22–31.

Henriques, J. B., & Davidson, R. J. (1991). Left frontal hypoactivation in depression. *Journal of Abnormal Psychology*, *100*, 535–545.

Izard, C. E. (1991). *The psychology of emotions*. New York: Plenum Press.

Jacobs, G. D., & Snyder, D. (1996). Frontal brain asymmetry predicts affective style in men. *Behavioral Neuroscience*, *110*, 3–6.

Jung, C. (1923). *Psychological types* [translated by H. Godwin Baynes]. London, U.K.: Paul, Trench, Trubner.

Lagerspetz, K. M. J. (1969). Aggression and aggressiveness in laboratory mice. In S. Garattini & E. B. Sigg (Eds.), *Aggressive behavior* (pp. 77–85). New York: Wiley.

Lane, R. D., & Jennings, J. R. (1995). Hemispheric asymmetry, autonomic asymmetry, and the problem of sudden cardiac death. In R. J. Davidson & K. Hugdahl (Eds.), *Brain asymmetry* (pp. 271–304). Cambridge, MA: Massachusetts Institute of Technology.

Lane, R. D., & Schwartz, G. (1987). Induction of lateralized sympathetic input to the heart by the CNS during emotional arousal: A possible neurophysiologic trigger of sudden cardiac death. *Psychosomatic Medicine*, *49*, 274–284.

Lazarus, R. S. (1991). *Emotion and adaptation*. New York: Oxford.

Lerner, J. S., & Keltner, D. (2001). Fear, anger, and risk. *Journal of Personality and Social Psychology*, *81*, 146–159.

Lewis, M., Sullivan, M. W., Ramsey, D. S., & Alessandri, S. M. (1992). Individual differences in anger and sad expressions during extinction: Antecedents and consequences. *Infant Behavior and Development*, *15*, 443–452.

Lindsley, D. B., & Wicke, J. D. (1974). The electroencephalogram: Autonomous electrical activity in man and animals. In R. Thompson & M. N. Patterson (Eds.), *Bioelectric recording techniques* (pp. 3–79). New York: Academic Press.

Malone, R. P., Delaney, M. A., Luebbert, J. F., Cater, J., & Campbell, M. (2000). A double-blind placebo-controlled study of lithium in hospitalized aggressive children and adolescents with conduct disorder. *Archives of General Psychiatry*, *57*, 649–654.

Meyer, B., Johnson, S. L., & Carver, C. S. (1999). Exploring behavioral activation and inhibition sensitivities among college students at risk for bipolar spectrum symptomatology. *Journal of Psychopathology and Behavioral Assessment*, *21*, 275–292.

Mikulincer, M. (1988). Reactance and helplessness following exposure to unsolvable problems: The effects of attributional style. *Journal of Personality and Social Psychology*, *54*, 679–686.

Miller, P. A., & Eisenberg, N. (1988). The relation of empathy to aggressive and externalizing/antisocial behavior. *Psychological Bulletin*, *103*, 324–344.

Moyer, K. E. (1976). *The psychobiology of aggression*. New York: Harper & Row.

Oatley, K., & Jenkins, J. M. (1996). *Understanding emotions*. Oxford, UK: Blackwell Publishers, Inc.

Obrist, P. A. (1981). *Cardiovascular psychophysiology: A perspective*. New York: Plenum.

Olweus, D. (1986). Aggression and hormones: Behavioral relationship with testosterone and adrenaline. In D. Olweus, J. Block, & M. Radke-Yarrow (Eds.), *Development of antisocial and prosocial behavior: Research, theories, and issues* (pp. 51–72). Orlando, FL: Academic Press.

Plutchik, R. (1980). *Emotion: A psychoevolutionary synthesis*. New York: Harper & Row.

Robinson, R. G., & Downhill, J. E. (1995). Lateralization of psychopathology in response to focal brain injury. In R. J. Davidson & K. Hugdahl (Eds.), *Brain asymmetry* (pp. 693–711). Cambridge, MA: Massachusetts Institute of Technology.

Rosenfeld, J. P., Cha, G., Blair, T., & Gotlib, I. H. (1995). Operant (biofeedback) control of left-right frontal alpha power differences: Potential neurotherapy for affective disorders. *Biofeedback and Self-Regulation*, *20*, 241–258.

Sarter, M., Berntson, G. G., & Cacioppo, J. T. (1996). Brain imaging and cognitive neuroscience: Toward strong inference in attributing function to structure. *American Psychologist*, *51*, 13–21.

Schaffer, C. E., Davidson, R. J., & Saron, C. (1983). Frontal and parietal electroencephalogram asymmetry in depressed and nondepressed subjects. *Biological Psychiatry*, *18*, 753–762.

Silberman, E. K., & Weingartner, H. (1986). Hemispheric lateralization of functions related to emotion. *Brain and Cognition*, *5*, 322–353.

Smith, C. A., & Lazarus, R. S. (1993). Appraisal components, core relational themes, and the emotions. *Cognition and Emotion*, *7*, 233–269.

Suarez, E. C., & Williams, R. B. (1989). Situational determinants of cardiovascular and emotional reactivity in high and low hostile men. *Psychosomatic Medicine*, *51*, 404–418.

Sutton, S. K., & Davidson, R. J. (1997). Prefrontal brain asymmetry: A biological substrate of the behavioral approach and inhibition systems. *Psychological Science*, *8*, 204–210.

Tomarken, A. J., Davidson, R. J., & Henriques, J. B. (1990). Resting frontal brain asymmetry predicts affective responses to films. *Journal of Personality and Social Psychology*, *59*, 791–801.

Tomarken, A. J., Davidson, R. J., Wheeler, R. E., & Doss, R. (1992). Individual differences in anterior brain asymmetry and fundamental dimensions of emotion. *Journal of Personality and Social Psychology*, *62*, 676–687.

Tomarken, A. J., Davidson, R. J., Wheeler, R. E., & Kinney, L. (1992). Psychometric properties of resting anterior EEG asymmetry: Temporal stability and internal consistency. *Psychophysiology*, *29*, 576–592.

Tomarken, A. J., & Keener, A. D. (1998). Frontal brain asymmetry and depression: A self-regulatory perspective. *Cognition and Emotion*, *12*, 387–420.

Tyrer, S., & Shopsin, B. (1982). Symptoms and assessment of mania. In E. S. Paykel (Ed.), *Handbook of affective disorders* (pp. 12–23). New York: Guilford Press.

Vandenheede, M., & Bouissou, M. F. (1993). Effect of androgen treatment on fear reactions in ewes. *Hormones and Behavior*, *27*, 435–448.

van Honk, J., Tuiten, A., de Haan, E., van den Hout, M., & Stam, H. (2001). Attentional biases for angry faces: Relationships to trait anger and anxiety. *Cognition and Emotion*, *15*, 279–297.

van Honk, J., Tuiten, A., van den Hout, M., Koppeschaar, H., Thijssen, J., de Haan, E., & Verbaten, R. (1998). Baseline salivary cortisol levels and preconscious selective attention for threat: A pilot study. *Psychoneuroendocrinology*, *23*, 741–747.

van Honk, J., Tuiten, A., Verbaten, R., van den Hout, M., Koppeschaar, H., Thijssen, J., & de Haan, E. (1999). Correlations among salivary testosterone, mood, and selective attention to threat in humans. *Hormones and Behavior*, *36*, 17–24.

Watson, D. (2000). *Mood and temperament*. New York: Guilford Press.

Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology*, *54*, 1063–1070.

Watson, D., Wiese, D., Vaidya, J., & Tellegen, A. (1999). The two general activation systems of affect: Structural findings, evolutionary considerations, and psychobiological evidence. *Journal of Personality and Social Psychology*, *76*, 820–838.

Wheeler, R. E., Davidson, R. J., & Tomarken, A. J. (1993). Frontal brain asymmetry and emotional reactivity: A biological substrate of affective style. *Psychophysiology*, *30*, 82–89.

Wortman, C. B., & Brehm, J. W. (1975). Responses to uncontrollable outcomes: An integration of reactance theory and the learned helplessness model. In L. Berkowitz (Ed.), *Advances in experimental social psychology* (Vol. 8, pp. 278–336). New York: Academic Press.

Wright, R. A. (1996). Brehm's theory of motivation as a model of effort and cardiovascular response. In P. M. Gollwitzer & J. A. Bargh (Eds.), *The psychology of action: Linking cognition and motivation to behavior* (pp. 424–453). New York: Guilford Press.

Young, P. T. (1943). *Emotion in man and animal: Its nature and relation to attitude and motive*. New York: John Wiley and Sons.

Zajonc, R. B. (1968). Attitudinal effects of mere exposure. *Journal of Personality and Social Psychology*, *9*(2, pt. 2), 1–27.

Zajonc, R. B. (1998). Emotions. In D. T. Gilbert, S. T. Fiske, & G. Lindzey (Eds.), *The handbook of social psychology* (4th ed., Vol. 1, pp. 591–632). New York: McGraw-Hill.

Zajonc, R. B., & McIntosh, D. N. (1992). Emotions research: Some promising questions and some questionable promises. *Psychological Science*, *3*, 70–74.

(Received December 29, 2002; Accepted June 22, 2003)