

AFFECTIVE STYLE AND RISK FOR PSYCHOPATHOLOGY

Chapter 9

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In the attempt to identify causal mechanisms of vulnerability to psychopathology, psychophysiological markers are highly valued for the important links they provide among social, behavioral, psychological, and cellular levels of analysis (Anderson & Scott, 1999). The promise of such markers lies in their potential to serve as objectively measurable individual differences related to underlying mechanisms of risk. Often referred to as *endophenotypes* (see Gottesman & Gould, 2003; Iacono, 1998), psychophysiological markers of vulnerability may stem from genetic factors, environmental pressures, or gene \times environment interactions (Gould & Gottesman, 2006). In the history of research on vulnerability to psychopathology, the search for reliable endophenotypes is a common theme. Potential markers such as low urinary levels of the noradrenaline metabolite MHPG (e.g., Fawcett & Maas, 1972), abnormal responses to the dexamethasone suppression test (e.g., Carroll, 1982), and reduced latency to the first period of rapid eye movement (REM) sleep (e.g., Kupfer, 1995) have met with varied success as indicators of vulnerability to diverse forms of psychopathology, yet each comes with shortcomings, suggesting strongly that better markers are needed (Berman, Narasimhan, & Charney, 1997).

One promising candidate for such a marker is the degree of asymmetry in neural activity across the frontal cortex, observed either at rest or during critical emotional challenges. This asymmetry in neural activity is indexed by the difference in brain activation between the right and left hemispheres at any given cortical region. Frontal hemisphere asymmetries in neural activity have—in numerous laboratories and across diverse experimental contexts—been associated with various forms of psychopathology and with more normative expressions of emotion and motivation (Coan & Allen, 2004). The dominant index of such hemispheric differences is *frontal EEG asymmetry*, often a single score representing the disparity in *alpha power* between corresponding left- and right-sided electroencephalographic (EEG) leads placed on the scalp over the frontal cortex. Insofar as individual differences in this measure predict (a)

behavioral patterns of emotional responding and (b) the presence or absence of psychopathology, EEG asymmetry appears to mark the psychological construct of *affective style*, a predisposition to react to the environment with a positive vs. negative emotional valence (see Davidson, 1998a; Hagemann, 2004).

Although EEG asymmetry often corresponds with affective style, which we describe in further detail below, it should not be treated as equivalent to affective style. As a general rule of thumb, neurophysiological markers should never be conflated with psychological constructs. Rather, physiological measures are tools for understanding the neural bases of behavior. Such measures rarely if ever capture the complexity of psychological predispositions and responses (see e.g., Davidson, 2004a)

Affective style describes individual differences in emotional and/or motivational predispositions to respond to environmental events. As instantiated in the “glass-is-half-full vs. glass-is-half-empty” aphorism, some people are predisposed to respond to environmental events with predominantly positive affectivity, whereas others are predisposed to respond to the same environmental events with predominantly negative affectivity. Considerable research over the past decade has identified some of the neural correlates of affective style, including both central dopamine functioning (e.g., Ashby, Isen, & Turken, 1999; Carver, 2004; Laakso et al., 2003) and septo-hippocampal functioning (e.g., Corr, 2004; Gray & McNaughton, 2000). The former system comprises neural structures within the mesocorticolimbic reward pathway (see e.g., Berridge & Robinson, 2003), whereas the latter includes interconnections with the amygdala, a structure implicated in the processing of nearly all emotion cues (see e.g., Davidson, 2002, 2004a). Negative affective style may confer risk for psychopathology, particularly depression (Davidson, 1994, 1998a, 2000), but also anxiety (Davidson, 2002; Heller, Nitschke, Etienne, & Miller, 1997; Nitschke, Heller, Palmieri, & Miller, 1999) hypomania (Harmon-Jones et al., 2002)

and conduct problems (Rybak, Crayton, Young, Herba & Konopka, 2006).

Although frontal EEG asymmetry has been the focus of considerable research on affective style, it is not a sole indicator. Other biological markers of affective style and closely related motivational constructs include cardiac psychophysiology (see Brenner, Beauchaine, & Sylvers, 2005), and certain event-related potential components of the EEG (see Luu, Collins, & Tucker, 2000). Nevertheless, our focus here is on EEG asymmetry, which is the most widely studied marker of affective style.

The empirical association between frontal EEG asymmetry and affective style suggests that the measure may serve as a useful endophenotypic marker of vulnerability for certain forms of psychopathology. This constitutes a major assumption of the chapter that follows in which we (a) explain the association between frontal EEG asymmetry and affective style; (b) discuss the differences and similarities among three types of endophenotypic markers; and (c) review the literature supporting frontal EEG asymmetry as an endophenotypic marker of vulnerability for psychopathology.

CONCEPTUAL MODELS OF AFFECTIVE STYLE

As alluded to above, affective style refers to a trait-like predisposition to respond in emotionally characteristic ways to environmental cues, particularly cues containing affective information (e.g., Davidson, 1992a). The diversity of observations regarding associations among motivational constructs, emotional responses, and frontal EEG asymmetries long ago suggested the need for an organizational model, and to date at least two prominent models of affective style have emerged (Van Honk & Schutter, 2006). The *valence model* references positive and negative affectivity—trait predispositions to engage in positive versus negative affect, respectively (cf., Gray & Watson, 2007). This model suggests that individuals predisposed to relatively greater left frontal brain activity respond to environmental demands with increased probability of positive

affect, or decreased probability of negative affect. By contrast, individuals predisposed to relatively greater right frontal activity are thought to show the reverse pattern, responding to environmental demands with an increased probability of negative affect, and a decreased probability of positive affect. A substantial literature either supports or partially supports the valence model. For example, infants who cry following maternal separation tend toward relatively greater right frontal activity measured at rest than infants who don't cry following separation (Davidson & Fox, 1989; Fox, Bell, & Jones, 1992). In addition, individuals with greater resting right frontal activity tend to respond with greater negative affect when negative film clips are presented, and individuals with greater resting left frontal activity tend to respond with greater positive affect when positive films are presented (Tomarken, Davidson, & Henriques, 1990; Wheeler, Davidson, & Tomarken, 1993).

More recently, a *motivational model* has been proposed (Davidson, 1992b; Harmon-Jones & Allen, 1998) based on accumulating evidence that tendencies toward relatively greater left frontal activity are also associated with (a) trait-like hostility and anger, (b) negatively valenced affective states (Harmon-Jones, 2001; Harmon-Jones & Allen, 1998), and (c) general behavioral activation (reward seeking) tendencies (e.g., Brenner et al., 2005; Coan & Allen, 2003; Harmon-Jones & Allen, 1997; Sutton & Davidson, 1997). These findings are consistent with the motivational model of frontal EEG asymmetry and affective style, which states that individuals predisposed to relatively greater left frontal activity than right respond to environmental demands with an increased probability of approach oriented affect (e.g., both joy and anger), or a decreased probability of withdrawal or passive-avoidance related affect (e.g., fear, sadness). By contrast, individuals predisposed to relatively greater right frontal activity than left are thought to respond to environmental demands with an increased probability of withdrawal or passive-avoidance related affect, or a decreased probability of approach-oriented affect.

The motivational model enjoys a great deal of empirical support, in part because it can accommodate earlier research supporting the valence model. Indeed, several theorists have noted similarities between the motivational model of frontal EEG asymmetry and other motivational models of behavior, particularly that of Gray (1972; 1987), which specifies distinct behavioral inhibition and behavioral activation systems (the BIS and the BAS, respectively; see also Beauchaine & Neuhaus, this volume). According to Gray, the BAS responds to incentives, and guides organisms toward attaining desirable stimuli through approach behaviors, and toward evading undesirable stimuli through active avoidance behaviors. In contrast, the BIS increases arousal and attention and inhibits prepotent approach or avoidance behaviors to resolve goal conflict between competing motivations (Corr, 2004; Gray & McNaughton, 2000). Frontal EEG asymmetries appear to correspond in part with scales developed by Carver and White (1994) to measure dispositional BIS and BAS tendencies in humans. As measured by the Carver and White scales, the BAS and BIS appear to correspond with frontal EEG asymmetries in ways that are highly similar (but not identical) to the motivational model of affective style (Coan & Allen, 2003; Harmon-Jones & Allen, 1997; Sutton & Davidson, 1997). At least three independent investigations confirm that individuals who show a propensity for relatively greater left frontal activity than right while at rest tend to score higher on the BAS scale (Coan & Allen, 2003; Harmon-Jones & Allen, 1997; Sutton & Davidson, 1997).

Attempts to associate right frontal activity with the BIS have been less successful. Although Sutton and Davidson (1997) observed a correspondence between right frontal activity and BIS scores, neither Harmon-Jones and Allen (1997) nor Coan and Allen (2003) were able to do so.

AFFECTIVE STYLE, EEG ASSYMMETRIES, AND PSYCHOPATHOLOGY

The theory of affective style suggests that individual differences in response to environmental challenges (e.g., an argument with a friend or parent, a disappointment) can be

explained in part by emotional reactivity and engagement, and in part by predispositions toward approach vs. withdrawal behavior (which affect emotional reactivity and engagement). Some individuals are more likely to engage actively with or otherwise approach environmental challenges, whereas others are more likely to withdraw from or avoid environmental challenges. Davidson (1998a) has proposed that these differences are probably insufficient to explain the presence or absence of psychopathology. Rather, affective style likely represents a diathesis, or vulnerability, that does not usually manifest as psychopathology without sufficient environmental stress (see also Beauchaine & Hinshaw, this volume). This is a classic moderator formulation (Coan & Allen, 2004) in which affective style alters the effect of environmental challenges on individual functioning. Thus, it is the *interaction* between affective style (vulnerability) and environmental risk that leads either to psychopathology or mental health. Neither affective style nor environmental experience is likely to be a strong determinant of psychopathology in isolation.

Any diathesis/stress model of psychopathology involves a number of assumptions, many of which have not been tested for affective style. Within the model proposed by Davidson (1998a), the first among these is the assumption that affective style is a stable, trait-like phenomenon, and that the markers used to assess it (frontal EEG asymmetry) are stable as well. Apart from this important issue, one may ask what kind of *marker* of psychopathology frontal EEG asymmetry constitutes. Recently, Allen, Urry, Hitt and Coan (2004b) proposed that the extent to which frontal EEG asymmetries represent episode, liability, or genetic vulnerability markers (cf., Iacono & Ficken, 1989) of psychopathology remains uncertain.

If frontal EEG asymmetries serve as *episode markers*, one would expect to see specific patterns of frontal EEG asymmetry in the presence of diagnosable psychopathology, but not when the condition remits. Such a marker would be very useful for delimiting pathology onset or

remission, suggesting when treatment may be needed or no longer needed, or identifying individuals with similar etiologies or prognoses. Episode marker criteria run counter to diathesis/stress models of psychopathology. Indeed, the diathesis/stress model of affective style presupposes that frontal EEG asymmetries function as non-specific *liability markers*—which are present and measurable both prior to and following remission of psychopathology. If frontal EEG asymmetries represent liability markers, they should be relatively independent of situational factors affecting mood and emotion.

Although a large literature has characterized personality traits such as positive and negative affectivity as both stable and moderately to highly heritable (e.g., Finkel & McGue, 1997), the literature to date is relatively silent on the question of genetic explanations for affective style. Stability does not necessarily imply genetic control. A given individual's tendency toward one pattern of affective style versus another may be stable throughout much of his or her lifetime, regardless of whether the predisposition stems from strong genetic influences or early experiences. In either case, a person's affective style and resulting pattern of frontal EEG asymmetry, if a genuine liability marker, would be useful for identifying those who are at greater risk for psychopathology than others in the general population. It remains possible, however, to distinguish liability markers from *genetic vulnerability markers*, which are a special class of liability marker that are entirely under genetic control.

Genetic vulnerability markers satisfy the same criteria as liability markers, with a few additional requirements. If frontal EEG asymmetries mark genetic vulnerability, they should be relatively independent of situational factors, yet characterize most persons with a given disorder (e.g., depression). In addition, they should be present in both depressed and nondepressed individuals, and demonstrate heritability within the normal population. Note, however, that they need not predict future depression, because genetic alleles may be linked to pathology only in the

presence of certain environmental circumstances. In other words, even if frontal EEG asymmetries mark genetic vulnerability, it is not the case that a given individual's affective style is *sufficient* to cause psychopathology.

To date, the degree to which frontal EEG asymmetry functions best as an episode, liability, or genetic vulnerability marker for psychopathology is not yet resolved. Thus far, it is clear that frontal EEG asymmetry does not characterize all psychopathologic individuals (Reid et al., 1998). Nevertheless, many of the extant data are consistent with the proposition that frontal EEG asymmetry indexes vulnerability for psychopathology in at least a subset of individuals who are at risk for certain pathological conditions, including depression and anxiety.

Table 1 provides an overview of studies consistent with frontal EEG asymmetry as an episode, liability, and genetic vulnerability marker of depression. Importantly, none of these studies *confirms* that frontal EEG asymmetry is an episode, liability, or genetic vulnerability marker.

Frontal EEG Asymmetry as an Episode Marker

The efficiency of any variable as an episode marker depends on two factors—*sensitivity* and *specificity*. A sensitive biomarker is present in nearly all people who exhibit a disorder. In contrast, a specific biomarker is present only in people with the disorder, and not in people without the disorder.

Sensitivity. In attempting to classify depressed versus nondepressed individuals, frontal EEG asymmetry has often but not always demonstrated reasonable sensitivity. For example, setting an arbitrarily but objective cutpoint of perfect symmetry in frontal activity (left activity = right activity), relatively greater right-frontal brain activity has characterized 69% (Henriques & Davidson, 1991), 83% (Schaffer, Davidson, & Saron, 1983), and in one study 100% (Allen et al., 1993) of depressed persons. Relatively greater right than left frontal activity also characterizes

individuals suffering from seasonal depression (Allen et al., 1993), college students with high scores on the Beck Depression Inventory (BDI) (Schaffer et al., 1983), and individuals diagnosed with unipolar depression (Debener et al., 2000; Gotlib, Ranganath, & Rosenfeld, 1998; Henriques & Davidson, 1991).

Yet this pattern of moderate to high sensitivity is not always replicated. Reid et al. (1998) analyzed two independent samples (one diagnosed via interview, the other defined psychometrically), and did not find the expected pattern of relatively greater right than left frontal activity among depressed individuals.

Specificity. One can also ask how specific frontal EEG asymmetry is as a marker of psychopathology. Most who study frontal EEG asymmetry and psychopathology attempt to separate depressed individuals from nondepressed controls, but some have sought to distinguish anxious from nonanxious controls (Davidson et al., 2000; Heller, Etienne, & Miller, 1995; Wiedemann et al., 1999). In both cases, relatively greater right (sometimes described as relatively less left) frontal activity corresponds with symptoms of both depression and anxiety. A recent meta-analysis by Thibodeau, Jorgensen, and Kim (2006) revealed that effect sizes concerning both forms of psychopathology lie in the moderate range—indicating substantial overlap in asymmetry scores for depressed/anxious people vs. normal controls—which argues against strong specificity.

To date, no studies have evaluated receiver operating characteristic (ROC) curves for measures of lateral asymmetry. Such reports are needed to establish the sensitivity and specificity of these scores vis-à-vis depression and/or anxiety. Nevertheless, the data presented above suggest that sensitivity is moderate, with insufficient evidence for specificity.

Because relatively greater right frontal brain activity has been associated with anxiety as well as depression (Davidson et al., 2000; Heller et al., 1997; Wiedemann et al., 1999), frontal EEG

asymmetries favoring the right may indicate a general predisposition toward internalizing psychopathology. Some have suggested that the specificity of frontal EEG asymmetries may be improved by taking a more nuanced view of asymmetry patterns. For example, Heller and colleagues (Heller & Nitschke, 1998; Heller et al., 1997) have proposed that more complex patterns of cortical brain function may distinguish depression from anxiety, and even specific forms of anxiety. These authors distinguish between anxious apprehension, which they describe as a primarily worry-based and ruminative concern for the future characterized by negative expectations—and anxious arousal, which they describe as primarily the experience of somatic panic symptoms such as muscle tension and rapid heart rate. In drawing these distinctions, they proposed that anxious apprehension should be characterized by relatively greater *left* frontal activity by virtue of its orientation toward scanning the environment. It should be noted, however, that depression and anxiety (a) share common genetic vulnerability and (b) are often comorbid within individuals (see e.g., Brady & Kendall, 1992; Krueger & Markon, 2006). Thus, efforts to parse lateral asymmetries into depression- and anxiety-specific patterns face a formidable challenge.

Frontal EEG Asymmetry as a Liability Marker

If frontal EEG asymmetry functions as a liability marker, we would expect that—as with episode markers—it demonstrate sensitivity in characterizing depressed persons from nondepressed controls, and from those with other forms of psychopathology. Unlike episode markers, however, one should also expect individual differences in the measure to be stable, not only across time and situations, but also across episodes of psychopathology. That is, one should expect frontal EEG asymmetry to differentiate clinical from nonclinical samples, not only during actual clinical episodes but also during periods of remission. Finally, if frontal EEG asymmetry represents a liability marker, it should be possible to predict later psychopathology in individuals

of nonclinical status who subsequently experience a significant life stressor. In other words, those who carry the liability should be susceptible to psychopathology following adverse events.

In any discussion of the stability of a construct or measure, it should be noted that the term stability has several meanings. One is the preservation of rank order of a trait or measure in a given sample over time. That is, do those high on the measure remain high (and those low remain low)—even if the overall level of the scores rises or falls? Another meaning has to do with the preservation of the actual score across time. We comment on the connotation of stability in the following text.

To date, only two studies have examined the stability of frontal EEG asymmetries during treatment of depression. Allen et al. (1993) found correlations in frontal asymmetry scores over two weeks ranging from .70 to .77 among individuals treated with light therapy for seasonal depression. More recently, Allen, Urry, Hitt and Coan (2004) examined stability in a nonmedicated sample of depressed individuals engaged in a nonpharmacological treatment. Across three measurement occasions separated by four week intervals, intraclass correlations (ICCs) at the frontal sites averaged .56. Across five measurement occasions, the same ICC was .61. Although individuals in this study made substantial improvements in their clinical status, within-sample individual differences were moderately stable. An earlier examination of 15 depressed patients undergoing pharmacotherapy generated no evidence of systematic mood-dependent changes in frontal EEG asymmetry across two weeks of treatment, despite the fact that depressed patients as a group showed the expected pattern of relatively greater right frontal resting activity (Debener et al., 2000). However, most of the patients in this investigation were receiving antidepressants, benzodiazepines, or both, raising the question of whether such medications influenced asymmetry scores. No studies have addressed this question.

Clinical status notwithstanding, the test-retest stabilities of individual differences in frontal

EEG asymmetry range from acceptable to good. For example, Tomarken and colleagues observed an average test-retest correlation across three weeks of .66 in a nonclinical sample of undergraduate university students (Tomarken, Davidson, Wheeler, & Kinney, 1992). In addition, Hagemann and colleagues observed that across four occasions of measurement, approximately 60% of the variance in frontal EEG asymmetry was accounted for by a stable latent trait (Hagemann, Naumann, Thayer, & Bartussek, 2002).

Thus, the limited data suggest that individual differences in frontal EEG asymmetry are relatively independent of clinical status. At the very least, there is little evidence to suggest that occasion-specific variation is related to diagnostic status or even mood. Yet some exceptions have been reported for children and adolescents. For example, in one study of depressed adolescents, music and massage therapy appeared to attenuate the pattern of relatively greater right frontal activity observed in the depressed group (Jones & Field, 1999). Indeed, massage therapy can reduce right frontal asymmetries in one-month old infants (Jones, Field, & Davalos, 1998). Although these findings are striking, it is less clear that the changes in frontal EEG asymmetry actually covaried with alterations in clinical state or mediated the effects of therapy on clinical state (cf., Coan & Allen, 2004).

Differentiation during remission. In three studies, individuals who were formerly depressed continued to demonstrate relatively greater right frontal activity when compared to never depressed controls (Allen et al., 1993; Gotlib et al., 1998; Henriques & Davidson, 1990). For example, Henriques and Davidson (1990) observed, in six normal-mood but previously depressed individuals, evidence of less left than right frontal activity compared to eight never-depressed controls, leading them to suggest that frontal EEG asymmetries provide a state-independent marker of depression risk. In the Allen et al. (1993) study of seasonal affective disorder, individuals suffering from depressive symptoms continued to display a pattern of less

left than right frontal activity compared with controls, even after bright-light-induced remission of symptoms. Later, Gotlib et al. (1998) observed that a group consisting of both currently and previously depressed individuals showed less left than right frontal activity compared with never depressed controls. Moreover, statistical tests of the difference in frontal EEG asymmetry between current and previously depressed individuals were not significant. These authors argued that frontal EEG asymmetry served as a state-independent marker of risk for psychopathology, particularly depression. Interestingly, and contrary to both predictions and past empirical evidence, Gotlib et al. (1998) also observed that frontal EEG asymmetry was not related to susceptibility to a negative mood induction, reports of depressogenic cognitive styles, or attentional biases for negative stimuli.

These last observations do not necessarily invalidate the liability marker hypothesis, but they do appear to run counter to the specific predictions of the diathesis/stress model of affective style and psychopathology, which states that it is the interaction between vulnerabilities reflecting (and possibly supporting) affective style and negative environmental stimuli that results in psychopathology. However, none of these studies measure stressors directly, and under such conditions, no diathesis-stress interaction would be found.

Frontal EEG asymmetry as a moderator of affect. The diathesis/stress model of affective style (Davidson, 1998a) specifies at least two depressogenic elements in producing psychopathology— affective vulnerability and negative environmental challenges. According to this formulation, frontal EEG asymmetries reflect affective dispositions that either enhance or diminish emotional responses preferentially for some but not other classes of stimuli, with consequences for the development of psychopathology. Differential effects of one variable (affective responding) across different levels or types of another variable (eliciting stimuli) is a defining feature of statistical moderation (Baron & Kenny, 1986; Kraemer, Stice, Kazdin,

Offord, & Kupfer, 2001; see also Coan & Allen, 2004).

In early work in this area, Davidson and Fox (1989) observed a greater probability of crying in response to maternal separation among infants who had relatively greater right resting frontal activity. Fox, Bell, and Jones (1992) later reported that this pattern of results was modestly stable over a five month assessment period. Among adults, affective responses to negative emotional film clips are more negative for individuals with a propensity for relatively greater right frontal activity (Tomarken et al., 1990; Wheeler, et al., 1993). Interestingly, responses to *positive* emotional film clips were observed by Wheeler et al. (1993) to be more positive among individuals with a propensity for relatively greater left frontal activity.

Unfortunately, attempts to replicate such observations have not always succeeded, perhaps in part due to methodological inconsistencies across laboratories. This point is exemplified in the work of Hagemann, Naumann, Becker, Maier, and Bartussek (1998), who were not able to replicate any of the findings of Tomarken et al. (1990) or Wheeler et al. (1993). Unlike the earlier studies, which used film clips as affective stimuli, Hagemann et al. (1998) used normed, emotionally charged images. Although they did observe that individuals with a propensity for greater left frontal activity at rest tended to respond more positively to positively-valenced images, this was only observed when using a particular EEG reference point (Cz) with several methodological shortcomings (see below for more information about the EEG referencing; Allen et al., 2004a; Hagemann et al., 2001).

Although the results described above are consistent with a moderator formulation of affective responding, almost no studies have tested the statistical interaction between lateral asymmetry and stimulus type in predicting outcome variables. One exception includes the work of Henderson, Fox, and Rubin (2001), who modeled frontal EEG asymmetries recorded at nine months of age as a moderator of behaviorally rated negative affectivity in predicting social

wariness by the age of four. In this work, infants who showed a high level of negative affect at nine months were more likely to be socially wary at four years *if* at age nine months they also showed relatively greater right frontal activity at rest. Tests of statistical interactions have rarely been applied to more clinically-relevant outcomes. One instance includes work by Bruder and colleagues, who observed that frontal EEG asymmetry moderated treatment response to fluoxetine among individuals suffering from depression (Bruder et al., 2001). That is, individuals with relatively greater left than right frontal activity were more responsive to fluoxetine.

In summarizing the evidence for the status of frontal EEG asymmetry as a liability marker for psychopathology, it is apparent that (1) frontal EEG asymmetries demonstrate trait-like stability across time in depressed participants and non-depressed controls; and (2) variations in frontal EEG asymmetry observed across measurement occasions do not vary with clinical state. Finally, although explicit tests of moderation have been evaluated for affect and social regulation effects that are plausibly related to psychopathology, no explicit tests of the diathesis/stress model have been conducted with regard to the development of diagnosable mental illness.

Frontal EEG Asymmetry as a Marker of Genetic Vulnerability

Weak evidence for frontal EEG asymmetry as a marker of genetic vulnerability derives in part from studies finding relatively greater right than left frontal activity in young infants of depressed mothers (Dawson, Frey, Panagiotides, Osterling, & Hessel, 1997; Dawson et al., 1999b; Jones, Field, Davalos, & Pickens, 1997). For example, Dawson et al. (1997) observed that infants of depressed mothers exhibited a pattern of right frontal asymmetry compared with infants of non-depressed mothers. This pattern of frontal EEG asymmetry distinguished between infants whose mothers were diagnosed with major depression and those whose mothers had sub-threshold symptoms. Dawson and colleagues have also observed infants of depressed mothers to exhibit relatively greater right than left frontal activity during interactions with both their

mothers and familiar experimenters (Dawson et al., 1999a; Dawson et al., 1999b). Field et al. (1995) have observed similar effects, reporting that infants of depressed mothers showed relatively greater right than left frontal activity when compared with those of non-depressed mothers.

Importantly, such studies, although consistent with genetic effects, cannot disentangle genetic predispositions from other heritable (e.g., epigenetic, programming) effects. Moreover, the few empirical investigations of the heritability of frontal EEG asymmetry suggest that despite its trait-like properties, heritability is modest. For example, Coan (2003) found that 22% of the variance in EEG asymmetry was heritable in young adult females, but virtually none of the variance was heritable in young adult males. Similarly, in a study of 73 monozygotic and 50 dizygotic female twins, Anokhin et al. (2006) estimated that approximately 27% of frontal EEG asymmetry over the mid-frontal regions was heritable. In contrast, the authors of another recent study reported heritability coefficients of .61 and .57 for young adult females and males, respectively (Smit, Posthuma, Boomsma, & De Geus, 2007). These estimates are derived from behavior genetics analyses, which detect additive and non-additive heritable effects, but cannot identify interactive effects. Future efforts, with large samples and molecular genetics assessments of specific candidate genes will be needed to examine whether frontal EEG asymmetry reflects the influences of specific alleles.

METHODOLOGICAL ISSUES IN FRONTAL EEG ASYMMETRY RESEARCH

There are many methodological complexities underlying the measurement of frontal EEG asymmetry that are likely to contribute to the aforementioned inconsistencies in observed effects across laboratories. As such, individuals interested in frontal EEG asymmetry may find a brief review of major methodological issues useful. Comprehensive coverage of measurement, data reduction, and data analysis can be found elsewhere (see Allen, Coan, & Nazarian, 2004a).

Alpha power and neural activity/activation. One of the most formidable hurdles for new readers of this literature concerns the inverse relation between neural activity in the *alpha range* (8 to 13 Hz) and cortical processing. A good deal of evidence suggests that when alpha power (i.e., alpha amplitude) is high, active cortical processing is low (Allen et al., 2004a), although not all frequencies in the alpha band show this relationship uniformly (Oakes et al., 2004). Thus, greater left frontal alpha power suggests less left frontal brain activity. It is customary for researchers to assume this inverse relationship and to speak primarily in terms of cortical activity, thereby performing a mental alpha power/cortical activity translation for the reader. Confusingly, however, this is not always the practice among researchers.

Another source of confusion concerns the frequent use of the terms “activity” and “activation” as if they are interchangeable when referring to EEG measures of alpha power. In this chapter, “activity” refers to the inverse of alpha power during tonic (resting) EEG measurement. By contrast, “activation” refers to the difference between two conditions of EEG measurement (e.g., resting vs. task). It is worth noting that the overwhelming majority of studies using EEG are designed to measure cortical activity, not cortical activation.

Asymmetry scores. The most common index of frontal EEG asymmetry is the asymmetry score. As stated above, this is simply the difference in alpha power between the right and left hemispheres at any given cortical region. A closer look at this score, however, reveals several complexities. First, alpha power tends to be positively skewed, and is frequently natural log (ln)-transformed before the difference score is computed. Typically, $\ln(\text{alpha power})$ over the left hemisphere is subtracted from $\ln(\text{alpha power})$ over the right hemisphere. Interpreted in terms of alpha, higher scores on this scale indicate relatively greater right frontal alpha power and lower scores indicate relatively greater left frontal alpha. Expressed in terms of activity, higher scores indicate relatively greater left frontal activity and lower scores indicate relatively greater right

frontal activity. However one wishes to interpret the score, zero indicates symmetry. Because the asymmetry score is so frequently used, it is often difficult to know which hemisphere (or whether one hemisphere more than the other) is responsible for observed hemispheric differences.

Reference scheme. One difficult methodological quandary facing frontal EEG asymmetry research regards the selection of an appropriate *reference site*. Any EEG recording represents the difference in electrical potentials between an active site and a reference. The ideal reference is inactive, providing investigators with estimates of spectral power that reflect activity at the site of interest. One such reference, for example, derives from the average electrical activity of the earlobes, or of the mastoid bones just behind the ear; another involves the average of all EEG recording sites.

Many reference montages exist, and as Reid et al. (1998) and others (Coan, Allen, & McKnight, 2006a; Hagemann, Naumann, & Thayer, 2001) have noted, data derived from one reference scheme often do not correlate well with data derived using another. Thus, reference schemes often do not “agree” about what the brain is doing, raising concerns over the interpretation of findings and about standardization across laboratories. Worse yet, it is difficult to be certain which reference scheme provides the “best” measure of EEG, although empirical and rational arguments tend to conclude that the Cz or vertex (top and middle of the head) may be the most problematic option (Allen et al., 2004a; Hagemann et al., 2001).

Unfortunately, the overwhelming majority of studies in this area depend on the Cz reference scheme (Coan & Allen, 2004). On the one hand, frequent use of the Cz reference could be partially responsible for misleading results and inconsistencies in this literature. On the other hand, the fact that so many significant effects have obtained *despite* this problem may indicate that the size of many effects has been underestimated (Coan et al., 2006a). The issue of the optimal or standard reference scheme in EEG recording awaits resolution. Until that time,

interested readers are advised to be aware of the reference scheme used in any report of frontal EEG asymmetry.

THE FUTURE OF AFFECTIVE STYLE AND FRONTAL EEG ASYMMETRY RESEARCH

The future of research on affective style and frontal EEG asymmetry as a vulnerability marker is naturally somewhat unclear. Nevertheless, in the remainder of this chapter we offer speculations—recommendations perhaps—on what is likely or ought to follow the work that has already been conducted.

The Neural Generators of Frontal EEG Asymmetry.

Identification of the neural structures underlying the manifestation of alpha asymmetries at the scalp is likely to greatly increase our understanding of affective style, as well as of links between affective style and risk for psychopathology. To date, theories of the most likely neural generators of frontal EEG asymmetries have targeted the dorsolateral prefrontal cortex (dlPFC) and the ventrolateral prefrontal cortex (vlPFC) (Craig, 2005; Davidson, 2004b). A large literature now implicates these and other prefrontal (and limbic) regions in depression, anxiety, and conduct problems. For example, components of the right dlPFC may interact with paralimbic structures in linking depressogenic cognitions to negative moods and behaviors (e.g., Mayberg, et al., 1999; Teasdale, et al., 1999), and individuals with unilateral left-hemisphere damage to the prefrontal cortex have long been observed to experience more depressive symptoms than those with damage to the right hemisphere (e.g., Gianotti, 1972).

Dysfunctions of the medial orbitofrontal cortex appear to be common to a variety of anxiety disorders, from simple phobias to obsessive-compulsive disorder and posttraumatic stress disorder (Rauch, Savage, Alpert, Fischman, & Jenike, 1997), and increased activity specifically in left orbitofrontal and ventrolateral prefrontal cortices have been observed in both anticipatory anxiety and anxious apprehension (Chua, Krams, Toni, Passingham & Dolan, 1999; Engels, et

al., 2007). In contrast, converging evidence from a variety of sources implicate the right dlPFC in threat-related vigilance (*anxious arousal*, cf., Engels, et al., 2007) and avoidance motivation (Coan et al., 2006b; Damasio et al., 2000; Kalin, Larson, Shelton, & Davidson, 1998; Kalin, Shelton, Davidson, & Kelley, 2001; Tranel, Bechara, & Denburg, 2002). In one recent study of the neural correlates of cognitive-behavioral therapy (CBT) for spider phobia, researchers reported that spider phobic individuals showed significant activation in the right dlPFC during the presentation of spider films (Paquette et al., 2003). Moreover, these researchers observed decreased activation in right dlPFC during film presentation at a follow-up assessment, presumably as a function of the CBT intervention targeting avoidance motivation.

Prefrontal dysfunctions are now widely implicated in aggressive, impulsive, and even criminal behavior (Raine, et al., 1998), and some evidence suggests this dysfunction may be lateralized. Examples include observations that bilateral or right hemisphere (but not left hemisphere) lesions of the ventromedial prefrontal and orbitofrontal cortices are associated with impulsivity and aggression (Anderson, Bechara, Damasio, Tranel & Damasio, 1999), and that violent criminals show lower ratios of lateral and medial prefrontal to subcortical glucose metabolism in the right hemisphere during continuous performance tasks compared with controls (Raine et al., 1998).

As methods for detailing specific functional relationships between neural structures and affective style grow more sophisticated and affordable, researchers interested in using frontal EEG asymmetry as an index of affective style and risk for psychopathology will do well to link those asymmetries to the neural structures that underlie them.

Emotion Regulation Capabilities vs. Dispositions.

Despite progress in the measurement of frontal EEG asymmetry, noteworthy inconsistencies in findings across laboratories remain (e.g., Allen et al., 2004a; Coan & Allen, 2004; Davidson,

1998b; Hagemann, 2004; Hagemann et al., 1998; Reid et al., 1998). If the potential of frontal EEG asymmetry as a vulnerability marker for psychopathology is to be realized, such inconsistencies must be resolved. One potential reason for these inconsistencies, recently discussed by Coan and colleagues (Coan et al., 2006a), concerns the conditions under which frontal EEG asymmetries are typically recorded.

Individual differences in frontal EEG asymmetry are usually obtained under “resting” conditions, where participants are instructed to relax, rest their eyes, or focus their attention on a fixation point, for the purpose of estimating of their “true” or absolute level of frontal EEG asymmetry. Fundamental individual dispositions are thought to manifest in the absence of strong contextual demands. The currently dominant model of frontal EEG asymmetry and affective style—what Coan and colleagues (Coan, et al., 2006a) have called the *dispositional model*—assumes that all else being equal, individuals have a single true asymmetry score that accurately reflects their affective style and that is independent of situational factors, such as the particular demands of a given emotional situation. According to this perspective, if under optimal resting conditions (or other conditions where situational influences on frontal EEG asymmetry are perfectly controlled or eliminated), a person manifests relatively greater right than left frontal cortical activity, that person is assumed to possess a disposition to respond across most situations with withdrawal-related affect. This withdrawal-related affective predisposition might place such individuals at increased risk for psychopathology.

Coan et al. (2006a) proposed that over-reliance on this traditional dispositional model of frontal EEG asymmetry might be responsible for inconsistencies both within and across laboratories in assessing the role of frontal EEG asymmetry in emotion and psychopathology. This is the case because measures of frontal EEG asymmetry taken at rest are uncontrolled. The uncontrolled nature of such tasks allows for a great deal of variability in participant cognitive

and emotional states—variability that may attenuate meaningful associations between frontal EEG asymmetry, emotion, and psychopathology. Such uncontrolled sources of variance could derive from, for example, the quality of a person's previous night of sleep, level of hunger, alcohol consumption, or relationship status. When uncontrolled sources of variance such as these are allowed to contribute to a given estimate of frontal EEG asymmetry, it may become more difficult to detect the signal of interest, such as relatively greater right prefrontal activity (putatively withdrawal motivation) that may indicate risk for affective psychopathology.

Accordingly, Coan et al. (2006a) argued that the resting condition is not optimal for assessing individual differences in frontal EEG asymmetry that are relevant to affective style and risk for psychopathology. Over-reliance on the resting condition leaves open the question of how individual differences in brain systems implicated in the development of psychopathology actually manifest under stressful conditions—conditions thought to contribute critically to psychopathology in diathesis-stress formulations. For example, although an individual may indeed be at risk for psychopathology, an endophenotypic marker of that risk may only manifest in the presence of relevant stimuli (cf., Allen & Di Parsia, 2002). Put another way, a person at risk for depression may manifest relatively greater right frontal activity during an evaluative social situation, but not during a pleasant reunion with an old friend or, as in the ideal form of the resting task, when in a passive, neutral state.

To deal with this problem, Coan et al (2006a) proposed a *capability model* of frontal EEG asymmetry that conceptualized affective styles as emotion-regulatory abilities instead of more passive emotional predispositions. The practical consequence of this conceptualization is that the optimal measurement of individual differences in frontal EEG asymmetry is likely to require carefully controlled emotional challenges that expose individual capabilities for regulating emotional responses. In support of this position, Coan et al. (2006a) provided empirical evidence

that individual differences in frontal EEG asymmetry are (a) more pronounced during emotional challenges than at “rest,” (b) more resistant to measurement error during emotional challenges than during resting tasks, and (c) more reliable in their association with criterion measures during emotional challenges than at rest.

Coan et al. (2006) noted that a pressing methodological issue related to the capability model concerns the recording conditions under which individual differences in frontal EEG asymmetry are most reliable and “diagnostic” (cf., Mischel, Shoda, & Mendoza-Denton, 2002). That is, the field must identify which classes of stimuli or experimental recording situations, outside of the resting situation, provide access to individual differences in frontal EEG asymmetry most aligned with affective style and risk for psychopathology. There are many possibilities related to this question. Apart from, for example, the simple distinction between the resting task and some emotional task, it may be that the most meaningful and predictive individual differences in frontal EEG asymmetry manifest during the anticipation of emotional stimuli (e.g., Shankman, Klein, Tenke, & Bruder, 2007; Zinser, Fiore, Davidson, & Baker, 1999). Alternatively, the recovery period following an emotional stimulus may be of primary importance. A related point, explored in more detail below, concerns the analysis of *change* in frontal EEG asymmetry.

Frontal EEG Asymmetry and Everyday Experience.

In recent years, great strides have been made in *experience sampling*, the assessment of daily emotional experience and responding. Diary methods are now routinely used to track daily affect, both generally and in specific situations (Bolger, Davis, & Rafaeli, 2003). With such tools, assessments of individual differences in emotional responding have reached unprecedented levels of detail, and have been applied to a wide variety of life situations, from exchanges in the workplace (Conway & Briner, 2002), to marital interactions (Laurenceau, Barrett, & Rovine, 2005), feelings of loneliness (Hawkley, Burleson, Berntson, & Cacioppo, 2003), and responses

to parental demands (Almeida, Wethington, & Chandler, 1999). Moreover, recent developments in experience sampling may ease costs attributable to data collection materials (e.g., hand held computers) and subject burden (e.g., Kahneman, Krueger, Schkade, Schwarz, & Stone, 2004).

To date, no studies have used experience-sampling in testing affective models of frontal EEG asymmetry. Doing so would offer a great deal more insight into the degree to which the emotion-regulatory capabilities frontal EEG asymmetries are thought to index actually manifest in ordinary life situations. In turn, such findings may lead to insights into the role of affective style in the development of psychopathology. Tests of the diathesis-stress model might be optimized with measures of affective responses to specific situations occurring in real time, and relatively free of strict experimental control. For example, individual differences in frontal EEG asymmetry recorded during laboratory emotional challenges emphasizing social evaluation may moderate associations between current social support levels and psychopathology. If individual differences in EEG asymmetry indeed interact with situational demands to influence the likelihood of psychopathology, the circle of measured affective responses and situational demands must be widened to include situations that occur naturally outside of the specific constraints of the laboratory. Experience sampling methods offer this possibility.

Frontal EEG Asymmetry and Time

A related point concerns the analysis of time. Most studies of individual differences in frontal EEG asymmetry related to risk for psychopathology use mean levels of brain activity, often collapsed across eight minute resting conditions. Some studies of state EEG asymmetry appeal to “change” scores between some emotional challenge and a resting condition. Although it is tempting to interpret scores computed in this way as actual measures of change across time, this is only true in instances where one condition directly follows another. Otherwise, such “change” scores are simply difference scores—mean differences in amplitude attributable to different

experimental situations. Davidson (1998a) has outlined the need for including time information in the study of frontal EEG asymmetry as well as other affective measures potentially related to psychopathology. He identified at least three components that need to be better understood, which may lead to a more informed science of the neural markers of risk for psychopathology. These components were referred to as *rise time to peak*, *peak amplitude*, and *recovery time*. Individual differences in any of these parameters could be meaningfully implicated in emotional responding and risk for psychopathology.

The rise time to peak refers to the speed with which a given response, in this instance frontal EEG asymmetry, changes from some initial value to a peak level of activation in response to an experimental situation. At the very least, the computation of the rise time to peak assumes a measured initial value and a peak value. Theoretically, the change score referred to above would provide a measure of rise time to peak if indeed the initial value always preceded the peak value, and some experimental situation was instantiated between the two measurements. Other options for measuring rise time to peak may exist as well, and newer analyses (e.g., multilevel modeling), as well as improvements in data collection hardware and software, make more sophisticated approaches to understanding the rise time to peak tenable. These may include, for example, multiple samples of alpha power throughout the period of stimulus presentation, and the modeling of these multiple samples to include time (e.g., slopes indexing change). To date, no researchers have pursued this strategy.

Individual differences in the peak amplitude of frontal EEG asymmetry—the maximum degree of change in the measure in response to some experimental situation—are likely to be found and likely to be meaningful. This point is closely related to the issue raised by Coan et al. (2006) in their description of the capability model, which states that individual differences in frontal EEG asymmetries are likely to reflect emotion regulation capabilities that may in turn be

optimally meaningful when measured in the context of an emotional challenge. Interest in peak amplitude raises similar questions about the widespread dependence upon resting tasks as well, because resting tasks preclude identification of anything like a peak amplitude measure of frontal EEG asymmetry. Interestingly, Davidson (1998) also described the possibility of measuring individual differences in *activation threshold*—the quantity of stimulus required to cause a given individual to respond. Stimulus thresholds could be similarly framed in terms of a capability model of frontal EEG activity, and may indeed hold significant consequences for our understanding of psychopathology risk. Moreover, and as with peak amplitude, stimulus thresholds are irrelevant in the context of resting measurement.

Finally, recovery times following the peak amplitude of a response are essentially the same as rise times to peak, but in the opposite direction, following rather than preceding the peak amplitude. Although recovery times of EEG asymmetry per se have not been examined, EEG asymmetry has predicted the duration of affective responses assessed via affectively modulated EMG startle (Jackson et al., 2003). As with both rise times and peak amplitudes, recovery times may hold essential information about individual risk for psychopathology. Indeed, it is possible that some of these parameters have more consequences for some forms of psychopathology than others. For example, depressed individuals may ‘hold on’ to particular patterns of activation for longer time periods than controls. All of these parameters could, with sufficient sampling across time, be measured in the context of a single model, using MLM and other data analytic procedures. These possibilities are all quite rich and remain to be examined in future work.

Sociocultural Situations and Factors.

It could be said that the most common context within which emotional behavior occurs, and occurs most intensely, is interpersonal (Roberts, Tsai, & Coan, 2007). There is little doubt that frontal EEG asymmetries are implicated in social responding. Numerous findings now indicate

that relatively greater right frontal activity is associated with greater social inhibition (Fox et al., 1995), lower sociability (Schmidt & Fox, 1994), higher levels of shyness (Schmidt, 1999) and intense social fear (Davidson et al., 2000). Missing from many of these studies, however, is how frontal EEG asymmetries function in social bonding, social support and caretaking, and attachment (e.g., Fox & Davidson, 1991). Increasing evidence suggests that emotional behavior is tightly linked to the formation of attachment bonds and the social regulation of emotion (Coan, Schaefer & Davidson, 2006b; Insel & Fernald, 2004). Increased attention to the role of prefrontal asymmetries in these processes stands to benefit both the study of interpersonal behavior and frontal EEG asymmetry, and, indeed, both are increasingly implicated in the etiology of many forms of psychopathology, some quite severe.

Related to social behavior are sociodemographic factors about which very little is known from the perspective of frontal EEG asymmetry and affective style. An important recent exception to this concerns the relationship between frontal EEG asymmetry and socioeconomic status (Tomarken, Dichter, Garber, & Simien, 2004). In this work, higher SES corresponded with relatively greater left frontal brain activity at rest. Interestingly, SES has long been implicated in the development of psychopathology (Leventhal & Brooks-Gunn, 2000; Truong & Ma, 2006). Tomarken and colleagues (2004) noted that explanations for the relationship between frontal EEG asymmetry and SES may include a variety of stressors associated not only with low SES, but also increased risk for psychopathology, including decreased levels of maternal warmth, peer group instability, fewer opportunities for social support, or decreased cognitive stimulation. We note that many of these additional risk factors—any one or combination of which may be related to frontal EEG asymmetry in determining psychopathology—lie in the interpersonal domain. By expanding attention to a wider variety of social processes and sociodemographic factors, the role of frontal EEG asymmetry in psychopathology might be further clarified.

Concluding Remarks

Frontal EEG asymmetry—a putative measure of affective style—is implicated in emotional responding and psychopathology. Thus far, the status of frontal EEG asymmetries as a marker of risk for psychopathology is uncertain, but an increasing body of evidence suggests it may indeed function as an liability marker of risk for depression and anxiety. Although preliminary evidence suggests it may ultimately serve as a marker of genetically influenced risk for these disorders (i.e., an endophenotype), the probability of this latter possibility does not seem strong. In any case, if frontal EEG asymmetry is ultimately going to offer clinical utility, it will have to be understood in much greater depth, and a large number of questions regarding its optimal measurement conditions will need to be resolved. Among the measurement issues for which resolution may be particularly important concerns the conditions under which individual differences in frontal EEG asymmetry are optimally measured—measurement conditions that constitute “diagnostic situations.” The identification of such situations is going to require researchers in this area to creatively expand their methodological explorations. Several steps may facilitate these goals, including: (a) working toward understanding the neural generators of frontal EEG asymmetries manifest in scalp recordings; (b) conceptualizing individual differences in frontal EEG asymmetry as reflecting capabilities for emotion regulation in specific situations rather than dispositions to respond in a particular way across situations; (c) using experience sampling techniques for the purpose of understanding the role of frontal EEG asymmetries in every day emotional responding, or in response to specific kinds of environmental events; (d) attending to the time course of responding in frontal EEG asymmetry; and (e) paying greater attention to the function of frontal EEG asymmetries in social contexts, including consideration of sociodemographic factors.

When discussing the role of frontal EEG asymmetry as both a measure of affective style and

a marker of risk, a valid objection is that such a marker would have little practical value even if all methodological difficulties were resolved and a frontal EEG asymmetry measure of great specificity and sensitivity resulted. Admittedly, the procedure is far too cumbersome to be used, for example, in some way analogous to tests for scoliosis, given en masse to grade school children across the United States, in some kind of effort to screen for potential depression risk. The costs of simple EEG assessments have, however, decreased dramatically in the last decade, just as computing and analysis power has advanced exponentially. Although frontal EEG asymmetry may never serve practically as a screening instrument to preempt the initial onset of psychopathology, it may yet prove very useful as an assessment of severity, prognosis, or risk for relapse, even in the near future. Moreover, if validated as an indicator of risk, further examinations may discover well-validated correlates of frontal EEG asymmetry that themselves are well-suited to screening and prospective identification of large numbers of at-risk individuals. Finally, the potential of such findings for influencing theory both with regard to normative affective style and to the development of psychopathology is likely in any case to be great. It is with these thoughts in mind that the future of research in this area is anticipated with great enthusiasm.

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Table 1. Characteristics of Psychophysiological Markers as Applied to Frontal EEG Asymmetry and Depression

Episode	Liability	Genetic
<p>Characterizes most depressed persons (sensitivity)^{1,8,9,12,-14,16}</p> <p>Differentiates depressed from nondepressed (specificity)^{1,-6,8,9,10,12,15-17}</p> <p>Changes with variations in clinical state^{-2,-8,15}</p>	<p>Characterizes most depressed persons (sensitivity)^{1,8,9,12,-14,16}</p> <p>Differentiates depressed from nondepressed, not only in episode but in remission as well^{1,9,11}</p> <p>Demonstrates stability in both depressed and nondepressed individuals^{1,2,-8,18}</p> <p>Predicts the future development of depression in individuals currently not depressed^{NA}</p>	<p>Characterizes most depressed persons (sensitivity)^{1,8,9,12,-14,16}</p> <p>Differentiates depressed from nondepressed, not only in episode but in remission as well^{1,9,11}</p> <p>Demonstrates stability in both depressed and nondepressed individuals^{1,2,-8,18}</p> <p>Predicts the future development of depression in individuals currently not depressed^{NA}</p> <p>Is heritable within the normal population^{3, 4, 17}</p> <p>Is more common in depressed persons with a strong family history of depression than those without a such a history^{NA}</p> <p>Is more prevalent in families of depressed individuals than in families of nondepressed individuals^{6,7,12}</p> <p>Identifies those family members at risk for depression^{NA}</p>

¹Allen, Iacono, Depue, & Arbisi, 1993

²Allen, Urry, Hitt, & Coan, 2002

³Anokhin, Heath & Myers, 2006

⁴Coan, 2003

⁵Davidson, Marshall, Tomarken, & Henriques, 2000

⁶Dawson, Frey, Panagiotides, Osterling, & Hessel, 1997

⁷Dawson, Frey, Panagiotides et al., 1999

⁸Debener et al., 2000

⁹Gotlib, Ranganath, & Rosenfeld, 1998

¹⁰Heller, Nitschke, Etienne, & Miller, 1997

¹¹Henriques & Davidson, 1990

¹²Henriques & Davidson, 1991

¹³Jones, Field, Fox, Lundy, & Davalos, 1997

¹⁴Reid, Duke, & Allen, 1998

¹⁵Rosenfeld, Baehr, Baehr, Gotlib, & Ranganath, 1996

¹⁶Schaffer, Davidson, & Saron, 1983

¹⁷Smit, Posthuma, Boomsma & de Geus, 2007

¹⁸Tomarken, Davidson, Wheeler, & Kinney, 1992

¹⁹Wiedemann et al., 1999

^{NA}No Data Currently Available

Note: Numerical superscripts refer to studies listed below. Positive numbers indicate that the study is consistent with the characteristic, and negative numbers indicate the study is inconsistent with the characteristic. NA = None Available. List of characteristics is after that of Iacono & Ficken, (1989).