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Review

# Frontal EEG asymmetry as a moderator and mediator of emotion

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**Abstract**

Frontal EEG asymmetry appears to serve as (1) an individual difference variable related to emotional responding and emotional disorders, and (2) a state-dependent concomitant of emotional responding. Such findings, highlighted in this review, suggest that frontal EEG asymmetry may serve as both a moderator and a mediator of emotion- and motivation-related constructs. Unequivocal evidence supporting frontal EEG asymmetry as a moderator and/or mediator of emotion is lacking, as insufficient attention has been given to analyzing the frontal EEG asymmetries in terms of moderators and mediators. The present report reviews the frontal EEG asymmetry literature from the framework of moderators and mediators, and overviews data analytic strategies that would support claims of moderation and mediation.


*Keywords:* Frontal EEG Asymmetry; Emotion; Mediators; Moderators

## 1. Introduction

Over 70 published studies have now examined the relationship between emotion or emotion-related constructs and asymmetries in electroencephalographic (EEG) activity over the frontal cortex. A review of these studies suggests asymmetries in frontal EEG activity—including resting levels of activity as well as state-related activation—are ubiquitous and involved in both trait predispositions to respond to emotional stimuli and changes in emotional state (Coan and Allen, 2003a).

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Studies of asymmetry can be classified as one of four types: (1) studies examining frontal EEG asymmetry as an individual difference that is related to other traits or trait-like measures; (2) Studies examining frontal EEG asymmetry as an individual difference that can predict state-related emotional changes and responses; (3) Studies examining frontal EEG asymmetry as an individual difference that is related to psychopathology or risk for psychopathology, especially depression and anxiety; and (4) Studies examining state-related change in asymmetry as a function of state changes in emotion. The first three types of studies explicitly assume that frontal EEG asymmetry has trait-like properties, whereas the fourth type of study assumes that state-related changes in EEG asymmetry can be elicited and observed.

Such findings suggest the possibility that the brain systems tapped by frontal EEG asymmetries may moderate, in the case of activity, and mediate, in the case of activation, emotional responding. In particular, Davidson (e.g., Davidson, 1993) has proposed that frontal EEG asymmetries reflect the activity of brain systems that moderate trait tendencies to approach, and withdraw from, novel stimuli, and mediate approach and withdrawal-motivational tendencies that underlie state emotional responding. Nevertheless, definitive analyses of frontal EEG asymmetry as a moderator and mediator of emotion[1] are rare, possibly due to insufficient attention paid to the precise differences between moderators and mediators, as well as to the data analytic demands that the differentiation of moderators and mediators entail. This article reviews the large and growing literature on frontal EEG asymmetry, emphasizing its potential as an important moderator and mediator of emotional responding. Additionally, conceptual and statistical considerations in assessing the extent to which frontal EEG asymmetry functions as both a moderator and mediator of emotional responding are reviewed, and recommendations are made for future investigations.

## 2. Moderators, mediators and frontal EEG asymmetry

Conceptually, moderators and mediators have been confused in the literature at large (Baron and Kenny, 1986). This may be particularly true of the literature on frontal EEG asymmetry, which frequently implicates moderators and mediators—sometimes both—conceptually, but making use of neither term, nor the special statistical considerations that optimize the specification of moderators and mediators. Though important statistical considerations will be discussed in detail later, it will be useful to review moderators and mediators conceptually before continuing.

*Moderators*: Moderators are essentially third variables that represent conditions under which some independent variable becomes maximally potent or effective. For example, in many people, images of poisonous snakes (indeed, any snakes) elicit fear responses, while, by contrast, more neutral images (such as, for example, images of elephants), do

[1] To be precise, we would seldom be interested in whether frontal asymmetries in electrical activity per se represent moderators or mediators of emotional responding, but rather would like to know whether activity in the system(s) tapped by frontal EEG asymmetries serve as a moderators or mediators of emotional responding (see Kline et al., 2003). For brevity and ease of reading, in the present discussion, we will talk about frontal EEG asymmetry as a moderator and/or a mediator.

not. If one were to argue that resting frontal EEG activity functions as a moderator of such responses, one might draw from the literature to formulate the following argument: Since resting asymmetries characterized by greater right-than-left frontal activity seem to be associated with traits and behaviors indicative of withdrawal behaviors (e.g., Wheeler et al., 1993), and since the emotion of fear is considered by many to be an emotion with withdrawal-related motivational properties (e.g., Coan et al., 2001), one might predict that individuals who possess a greater trait tendency toward relatively greater right frontal resting EEG activity would be more sensitive to fear cues, or, at the very least, would react more strongly to fear-related cues. Using the emerging example of snakes versus elephants, such a prediction would be borne out by evidence that (1) individuals normatively respond with more fear to images of snakes than to images of elephants and (2) that individuals possessing relatively greater right frontal activity at rest would respond to images of snakes with still more fear than is otherwise normative. This type of sensible prediction has indeed been made by Davidson (1998a,b), who has referred to trait capacities to respond affectively in characteristic ways as *affective style*. Davidson (1998a,b) has argued that an individual's affective style is in part moderated by asymmetries in frontal cortical activity, and his model enjoys substantial, though not unequivocal, empirical support.[2]

*Mediators*: Mediators, by contrast, are third variables that represent the mechanism through which (or partially through which) the effect of a given independent variable is made manifest. For example, if one of the components of an ordinary fear experience is a motivational tendency to withdraw, then eliciting that component of fear might require activity in the brain systems tapped by frontal EEG asymmetries. This needn't necessarily mean that cortical activation asymmetries are always involved as a third variable mediator of fear. It might mean, however, that whenever fear is characterized by a withdrawal-oriented motivational component (and some would no doubt argue that this is *always* the case), that component is *dependent* upon systems that are tapped by frontal EEG asymmetries. To the extent that this is true, frontal EEG asymmetry would then function as a mediator of emotional responses.

## 3. The measurement of frontal EEG asymmetry

Various issues surrounding the measurement and analysis of cortical EEG asymmetries can make apprehending results in this area challenging. Thus, some discussion of measurement and data analytic issues is warranted up front, though many of these issues are addressed in greater detail elsewhere in this volume (see Allen et al., this volume). First, evidence suggests that activity within the alpha range (typically 8–13 Hz) may be inversely related to underlying cortical processing, since decreases in alpha tend to be observed when underlying cortical systems engage in active processing. With this in mind, all results reviewed here will be reported in terms of theoretically assumed cortical activity rather than alpha power per se.

[2] An equally plausible prediction, though one not as frequently or explicitly encountered in the research literature, is that the effects of frontal EEG asymmetry on, for example, depression, are themselves moderated by other psychological traits (for a discussion of this possibility, see Minnix et al., this volume).

Second, in reviewing the literature, one will variously encounter references to frontal EEG *activity* and frontal EEG *activation*. At first blush, insistence upon this distinction may appear pedantic, but in fact, strict attention here can significantly enhance understanding of research in this area, and is relevant to making inferences concerning the distinction between frontal EEG asymmetry as a moderator or a mediator. For the purposes of this article, and as a general recommendation, *activity* will refer to a tonic recording of cortical processes as measured by EEG, while *activation* will refer to the change in EEG activity in response to a provocation, such as the presentation of an emotional stimulus. For example, one may be interested in measuring an individual's asymmetry in frontal EEG *activity* at rest as well as that same individual's asymmetrical frontal EEG *activation* in response to an experimental manipulation, such as the presentation of an image of a venomous snake. While this implies that activity refers only to resting or baseline measures, this is not necessarily the case. Indeed, one could measure an individual's resting activity both at baseline and following a stimulus presentation. The difference between those post-stimulus and baseline activity measures, however, would represent that individual's *inferred activation in response to the stimulus*. This distinction is important for maintaining conceptual clarity when reviewing the literature on frontal EEG asymmetry. Thus, in reviewing the literature, one of the functions of this article will be to make clear this distinction in past literature as well as in the arguments and data presented here.

Finally, an occasional difficulty in reviewing the literature on frontal EEG asymmetry involves discerning precisely what specific hemispheric effects are responsible for observed asymmetries. This difficulty frequently results from the widespread practice of computing and analyzing an asymmetry index, typically a difference score, rather than analyzing hemisphere as a two-level factor. The most commonly reported of these indexes is computed by subtracting the natural log of left hemisphere alpha power from the natural log of right hemisphere alpha power (ln[right alpha] − ln[left alpha]). This approach results in a unidimensional scale representing the relative activity of the right and left hemispheres, with the middle point of the scale equaling zero or symmetrical activity. In interpreting this scale, higher scores indicate relatively greater left frontal activity whereas lower scores indicate relatively greater right frontal activity (again keeping in mind that higher scores result from relatively greater right frontal alpha power—the putative inverse of activity). A limitation of this particular metric—and other similar metrics—is that the metric provides no information regarding the extent to which each hemisphere is contributing to the observed difference score (e.g., Jones et al., 1998).

It is not necessarily the case, however, that the use of asymmetry metrics is either unwarranted or particularly problematic. Asymmetry scores have some distinct advantages, not least of which is their capacity for controlling individual differences in skull thickness that might produce artifactual and non-neurogenic individual differences in recorded power values. Asymmetry metrics can also make statistical tests more sensitive by reducing the number of contrasts in a particular model and increasing statistical power. When such difference score based asymmetry effects are followed up by specific hemisphere analyses, such an approach is not only warranted but may be the most efficient data analytic approach to these kinds of data (cf. Coan and Allen, 2003b). Asymmetry scores also conceptually simplify certain analyses, such as those involving correlations between frontal asymmetries (as a difference score) and other individual difference measures (e.g., behavioral activation;

Coan and Allen, 2003b; Harmon-Jones and Allen, 1997; Sutton and Davidson, 1997). Additionally, difference scores based on alpha power asymmetries tend to show high internal consistency and acceptable test-retest reliability, dispelling fears about reduced reliability attributable to difference scores per se (Allen et al., this issue; Jones et al., 1997; Tomarken et al., 1992b). Nevertheless, reports of specific hemispheric effects will be important for the ultimate understanding of the precise nature of cortical asymmetries in emotional responding. Thus, tables presented in this article include a column labeled "Hem" that specifies whether a given report investigated specific hemispheric effects ("yes") or used an asymmetry metric only ("no").

## 4. What frontal EEG asymmetry measures: evidence from its associations with other traits

Resting measures of asymmetrical frontal EEG activity have been associated with other trait measures. Such relationships suggest the functional properties of the underlying cortical systems that give rise to frontal EEG asymmetry. Findings from numerous studies reflect an emerging consensus that relatively greater trait left frontal activity is associated with trait tendencies toward a general appetitive, approach, or behavioral activation motivational system, and that relatively greater trait right frontal activity is associated with trait tendencies toward a general avoidance or withdrawal system (Coan and Allen, 2003a; Davidson, 1993). This has resulted in Davidson's highly influential approach/withdrawal motivational model of emotion (Davidson, 1993; Davidson, 1998a,b). According to this model, left frontal activity, either as a state or a trait, indicates a propensity to approach or engage a stimulus, while relatively greater right frontal activity indicates a propensity to withdraw or disengage from a stimulus. A comprehensive tabular summary of the literature associating trait frontal EEG asymmetry with other trait measures can be found in Table 1. Though no attempt to review every report in this literature will be made within the body of this article, a number of particularly noteworthy studies will be highlighted in the remainder of this section.

An obvious paper and pencil measure for assessing Davidson's approach-withdrawal model of frontal EEG asymmetry in emotion might be Carver and White's (1994) BIS/BAS scales, which purport to measure Gray's (1972, 1987) behavioral inhibition and activation systems (BIS and BAS, respectively) as human traits. According to Gray, the BIS initially inhibits action, increases arousal and attention and subsequently guides behavior toward removing or avoiding an undesirable stimulus. The BAS essentially functions in the opposite manner, responding to incentives, and guiding organisms toward attaining a desirable stimulus, including negative reinforcers. Researchers have identified relationships between these systems and frontal EEG asymmetry (Coan and Allen, 2003b; Harmon-Jones and Allen, 1997; Sutton and Davidson, 1997). In particular, Sutton and Davidson (1997) proposed that the BIS and BAS should map closely onto withdrawal and approach tendencies, respectively, arguing that these constructs are functionally identical. They found that relatively greater left frontal activity was associated with both higher BAS scores and greater BAS-BIS difference scores. Further, they found that relatively greater right frontal activity was associated with higher BIS scores (Sutton and Davidson, 1997). Work by Harmon-Jones and Allen (1997) and Coan and Allen (2003b), however, suggest that the relationship is

Table 1
Trait frontal EEG asymmetry and other trait-like measures

| Citation | *N* | Age | Handedness | Reference scheme | Independent variable | Dependent variable | Hem | Results summary |
|---|---|---|---|---|---|---|---|---|
| Buss et al., 2003 | From 85 infants samples sizes for specific analyses vary | 6 months | NA | AR, Cz | Extreme right (ER), intermediate (I) and extreme Left (EL) groups from EEG (5–9 Hz) at FP1/2, F3/4, F7/8; baseline (B) vs. stranger (Str.) groups | Cortisol level (C), fear (F) and sadness (S) ratings | Yes | ER, ↑ C<br>ER and Str., ↑ F<br>ER and Str., ↑ S |
| Coan, 2003 | 250 (132 female) | Mean = 19 years | No information | Cz, LM | Sex (M vs. F); positive emotionality (PEM) and negative emotionality (NEM) and others from the multidimensional personality questionnaire (MPQ); monozygotic twins (MT) and dizygotic twins (DT); twins used to assess heritability ($h^2$) | EEG at F3/4; | No | In M, no significant findings<br>In F, ↑ RFA, ↑ PEM<br>In F, ↑ RFA, ↑ NEM<br>In F, $h^2$ of F3/4 ≈ 0.22<br>In F, $h^2$ of bivariate phenotypic correlation between F3/4 and NEM ≈ 0.42 |
| Coan and Allen, 2003 | 32 (26 female) | 17–24 years | R | AR, LM | EEG at FP1/2, F3/4, F7/8, FTC1/2, C3/4, T3/4, TCP1/2, T5/6, P3/4, O12 | BIS/BAS | Yes | ↑ LFA, ↑ BAS |
| Davidson et al., 1999 | 24 (9 female) | 17–21 years | R | LM | EEG at F3/4, F7/8 and T3/4 | Natural killer (NK) cell activity at rest, before exam, and following pos and neg film clips | No | ↓ RFA, ↓ NK (rest)<br>↓ RFA, ↓ NK (exam)<br>↑ LFA, ↑ NK (pos film clip) |
| Ehlers et al., 2001 | 134 (68 female) | 7–13 years | Both | No information | Handedness (L vs. R); strongly native American (SNA) vs. native American (NA); sex (M vs. F); positive family history of alcohol abuse (FHP) vs. no such history (FHN) | EEG at F3/4, C3/4, P3/4, O1/2 | No | SNA, ↓ LFA |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Field et al., 2002 | 48 infants (29 female) | newborn | NA | Cz | Infant EEG at F3/4, P3/4 | Maternal serotonin (MS), maternal postnatal cortisol (MPC), maternal EEG at F3/4, P3/4, maternal vagal tone (VT), infant cortisol (IC), infant sleep state changes (SS), infant Brazelton assessment (BA) | No | ↑ infant RFA, ↓ MS<br>↑ infant RFA, ↓ MPC<br>↑ infant RFA, ↑ maternal RFA<br>↑ infant RFA, ↓ maternal VT<br>↑ infant RFA, ↑ IC<br>↑ infant RFA, ↓ BA |
| Fox et al., 1995 | 48 (28 female) | 49–62 months | No information | Cz | EEG at F3/4, P3/4 and O1/2 | Social competence (SC) | Yes | ↑ RFA, ↓ SC<br>↑ LFA, ↑ SC |
| Hagemann et al., 1999 | 36 (24 female) | Mean = 24.7 | R | Cz | Positive affectivity (PA), negative affectivity (NA), extroversion (E), and neuroticism (N) | EEG at F3/4, T3/4, C3/4, P3/4 A1/2 | Yes | ↑ NA, ↑ LATA |
| Harmon-Jones and Allen, 1997 | 37 females | No information | R | Cz | EEG at F3/4 and P3/4 | BIS/BAS | No | ↑ LFA, ↑ BAS |
| Harmon-Jones and Allen, 1998 | 26 (11 female) | Mean = 13 years | R | Cz | EEG at FP1/2, F3/4, C3/4, P3/4, T5/6, T3/4, O1/2 | Dispositional anger (A) | No | ↑ LFA, ↑ A |
| Jacobs and Snyder, 1996 | 40 males | 18–53 years | R | LM | EEG at F3/4, P3/4 | PANAS (PA and NA); BDI | No | ↑ LFA, ↓ NA score<br>↑ LFA, ↓ BDI score |
| Jackson et al., 2003 | 47 (30 female) | 57–60 years | R | LE | Unpleasant (U), pleasant (P) and neutral (N) pictures; startle probe during early viewing (A), later during viewing (B), and after viewing (C) | EEG at FPF1/2, FP1/2, F3/4, F7/8, FC3/4, FC/8, C3/4, CP3/4, CP5/6, T3/4, T5/6, P3/4, PO3/4 | No | ↑ RFA, ↑ C negativity |

Table 1 (*Continued*)

| Citation | *N* | Age | Handedness | Reference scheme | Independent variable | Dependent variable | Hem | Results summary |
|---|---|---|---|---|---|---|---|---|
| Jones et al., 1997 | 87 (infants) | No information | No information | Cz | Baby groups: overstimulating (O) vs. understimulating (U) mothers | EEG at F3/4 and P3/4, various physio and beh. measures | Yes | O babies, ↑ LFA<br>U babies, ↑ RFA<br>(Mothers showed the same pattern as infants) |
| Kang et al., 1991 | 20 females | 17–20 years | R | Cz, LM | Extreme LFA and RFA groups | Natural killer (NK) cell, lymphocyte and T-cell activity | No | RFA group, ↓ NK activity |
| Kalin et al., 2000 | 49 (23 female; rhesus monkeys) | Longitudinal data at 4, 8, 14, 40 and 52 months | No information | No information | Extreme LFA and RFA groups in monkeys | Cerebrospinal fluid CRH | No | ↑ RFA, ↑ CRH |
| Kline et al., 1998 | 85 (60 females) | 17–33 years | R | Linked ears (LE) | Defensive coping (EPQ-L scale) | EEG at F3/4, FP1/2, F7/8, C3/4, T3/4, T5/6, P3/4, O1/2 | Yes | For women, ↑ LFA ↑ defensiveness<br>For men, ↑ LFA ↓ defensiveness |
| Kline et al., in press-a, in press-b | 72 (42 female) | Mean = 20.4 | R | No information | Defensiveness (D), parental caring (PC) | EEG at FP1/2, T3/4, F7/8, and F3/4 | Yes | ↑ defensiveness, ↑ LFA |
| Kline et al., in press-a, in press-b | 235 (141 females) | Mean = 20.4 | R | No information | High (HD) vs. low (LD) defensiveness groups; experimenter gender: same vs. opposite | EEG at F3/4, FP1/2, F7/8, C3/4, T3/4, T5/6, P3/4, O1/2 | Yes | HD, ↑ LFA in presence of opposite sex. |
| McManis et al., 2002 | 166 children | 10–12 years | R | LM | Low fear (LF), moderate fear (MF) and high fear (HF) at 2 years old; low reactivity (LR) vs. high reactivity (HR) at 2 years old | EEG at F3/4, P3/4, A1/2 | No | HF and HR, ↑ RFA |
| Moss et al., 1985 | 12 (Japanese), 12 (Western), all Female | J, mean = 32.6 years; W, mean = 29.1 years | R | Cz | Cultural group (J vs. W) | EEG at T3/4 and P3/4 | Yes | W = ↑ LPA |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Merckelbach et al., 1996 | 29 females | 22–38 years | No information | A1 | L vs. R hemisphere preference (questionnaire) | EEG at F3/4 and P3/4 | No | ↑ LHP, ↑LFA |
| Schmidt, 1999 | 40 females (extreme scorers selected from among 271) | M = 20.97 years | R | Cz | Low shy vs. high shy; low soc vs. high soc | EEG at F3/4, P3/4 and O1/2 | Yes | ↑ shyness, ↑RFA<br>↑ soc, ↑ LFA<br>High shy, high soc had ↑ LFA than high shy, low soc |
| Schmidt and Fox, 1994 | 40 females (extreme scorers selected from among 282) | No information | R | Cz | Low shy vs. high shy; low soc vs. high soc | EEG at F3/4, P3/4, A1/2 and O1/2 | Yes | ↓ soc, ↑ RFA<br>Low shy, high soc = ↑ RPA<br>Low shy, low soc = ↑ LPA |
| Stough et al., 2001 | 16 (11 females) | 20–30 years | No information | No information | Measures of openness (O), agreeableness (A) and conscientiousness (C) | EEG at FP1/2, F3/4, F7/8, T3/4, T5/6, C3/4, O1/2. | Yes | No effects in alpha band. |
| Sutton and Davidson, 1997 | 46 (23 female) | 18–22 years | R | LM | EEG at F3/4 7 and P3/4 | BIS/BAS, BAS–BIS diff score | No | ↑ LFA, ↑ BAS<br>↑ RFA, ↑ BIS<br>↑ LFA, ↑ BAS-BIS diff |
| Tomarken and Davidson, 1994 | 90 females | No information | R | Cz | High defensive (HD) vs. low defensive (LD) | EEG at F3/4, F7/8, T3/4, C3/4 and P3/4 | Yes | HD = ↑ LFA in F3/4 and F7/8 |
| Tomarken et al., 1992a | 90 females | 17–21 years | R | Cz, LM | EEG at F3/4, F7/8, T3/4, P3/4, C3/4 | General positive and negative affect (PA and NA) | No | ↑ LFA, ↑ PA<br>↑ LFA, ↓ NA |
| Urry et al., in press | 84 | 57–60 years | R | AR, LM | EEG at 29 sites, including FPF1/2, FC3/4, FP1/2, F7/8, F3/4, and FC7/8 | Eudaimonic well-being (EWB), hedonic well-being (HWB) | | ↑ LFA in FC3/4, ↑ EWB<br>↑ LFA in FC3/4, ↑ HWB |

RFA: right frontal activity, LFA: left frontal activity; RATA: right anterior temporal activity, LATA: left anterior temporal activity; RPA: right parietal activity, LPA: left parietal activity.

robust for BAS, but not so robust for BIS. While both Harmon-Jones and Allen and Coan and Allen found associations between relative left frontal activity and higher BAS scores, neither study detected a strong association between relative right frontal activity and BIS scores (though Coan and Allen did detect a statistical trend suggestive of this relationship). In both reports, it was suggested that the theoretical association between withdrawal motivations and the BIS is more complex than that between approach motivations and the BAS (Coan and Allen, 2003b; Harmon-Jones and Allen, 1997). Theoretically, Davidson's (1998a) withdrawal construct may be more general than that of the BIS. For example, the hypothetical systems underlying Davidson's withdrawal construct are thought to motivate or predispose organisms to *withdraw* from sources of aversive stimulation, whereas the BIS has been described as a system that, among other things, *interrupts* behavior, increases arousal and increases attention (Gray, 1994), none of which are necessarily involved in withdrawal behaviors or predispositions. Indeed, Davidson's withdrawal construct could overlap with functions included in any of the motivational systems described by Gray (for a more detailed discussion, see Coan and Allen, 2003b). By contrast, Davidson's approach and Gray's BAS constructs (as well as the similar construct of the behavioral facilitation system; Depue and Collins, 1999; Depue and Iacono, 1989) may share substantial overlap.

The rather robust relationship between frontal EEG asymmetry and the BAS is further bolstered from findings designed to test the primarily motivational approach-withdrawal model of frontal asymmetry against a valence model (cf. Heller and Nitschke, 1997). For example, Harmon-Jones and Allen (1998), and more recently Harmon-Jones (2000), found that left frontal activity was associated with trait anger, a negatively-valenced but approach-related emotion conceivably related to BAS functions. Indeed, in at least one study, BAS sensitivity as measured by Carver and White's (1994) BAS scale was associated with a greater likelihood of aggressive behavior (Wingrove and Bond, 1998).

While the relationship between the self-reported BIS scores and trait withdrawal propensities remains uncertain, other evidence that trait frontal EEG asymmetries are related to both behavioral propensities to approach and withdraw is available. One source of this evidence derives from measures of social behavior. For example, Fox et al. (1995) found evidence that children with greater relative right frontal activity at rest were generally more inhibited socially, and scored lower on measures of social competency (Fox et al., 1995). Moreover, children were more sociable and more socially competent to the extent they had relatively greater left frontal activity. Schmidt and colleagues (Schmidt, 1999; Schmidt and Fox, 1994) investigated the relationship between frontal EEG asymmetry and measures of sociability in adults and found that individuals scoring low on measures of sociability showed relatively greater right frontal activity at rest (Schmidt and Fox, 1994). Further, Schmidt (1999) found that shyness was associated with relatively greater right frontal activity, while sociability was associated with relatively greater left frontal activity. Interestingly, Schmidt (1999) also found that shy individuals who nevertheless scored high on measures of sociability possessed greater left frontal activity than other shy individuals with low sociability scores. More recently, Kalin et al. (2000) have investigated the relationship between frontal EEG asymmetry and other physiological traits that may *underlie* processes related to those reviewed above. For example, they have reported a positive relationship between extreme right frontal EEG activity and high cerebrospinal fluid concentrations of corticotrophin-releasing hormone (CRH) in rhesus monkeys (Kalin et al., 2000). CRH is

itself thought to mediate stress responses, as well as fear, anxiety and depression. According to this work, higher CRH levels are associated with higher levels of stress (De Souza, 1995; Kalin et al., 2000). Further, Kalin and colleagues have supplied evidence that rhesus monkeys showing extreme relative right frontal EEG activity have both elevated cortisol levels and exaggerated defensive behaviors (Kline et al., 1998).

## 5. Frontal EEG asymmetry as a possible moderator of emotional responding

When considering how resting frontal EEG asymmetry may serve as a predictor of subsequent emotional responses, two non-mutually exclusive relationships are possible.

1. Resting frontal EEG asymmetry taps an individual difference that may facilitate or diminish an emotional response (e.g., happiness) across many classes of stimuli (e.g., happy, sad, angry, fearful, and disgusting stimuli). For example, greater relative left frontal activity might be expected to be associated with greater self-reported happiness, for positively- as well as negatively-valenced stimuli.
2. Resting frontal EEG asymmetry taps an individual difference that may facilitate or diminish emotional responses preferentially for some but not other classes of stimuli. For example, greater relative right frontal activity might be expected to potentiate startle in response to negatively-valenced stimuli, but not to positively-valenced or neutral stimuli.

Strictly speaking, it is only the second case that qualifies as a moderator variable (cf. Baron and Kenny, 1986), as will be detailed below. The first instance is nonetheless a theoretically useful and interesting finding that frontal EEG asymmetry may serve as an individual difference variable that predicts emotional responses.

Davidson (1998a) has proposed that trait EEG asymmetries index propensities for reacting in predictable ways to emotionally evocative stimuli. Davidson (1998a) has called this propensity "affective style," and he proposes that frontal EEG asymmetry indexes a system that may have emotion-specific or valence-specific (p. 309) moderating influences, with implications for risk for psychopathology. The intent of the following section is therefore to review the literature suggestive of frontal EEG asymmetry as a possible moderator of emotional responding. The question of whether and to what extent this evidence unequivocally implicates frontal EEG asymmetry as such a moderator, however, depends upon a deeper consideration of what is meant, not only conceptually but also statistically, by the term *moderator*.

### 5.1. Modeling frontal EEG asymmetry as a moderator

Before describing the data analytic identification of a moderator variable, it is important to understand precisely what a moderator variable should look like in terms of statistical effects. Fig. 1 depicts a general moderator model that will serve as a useful guide to such a discussion. For the purposes of the illustration, assume that frontal EEG asymmetry may moderate the intensity rating of a fear experience following the stimulus presentation of an image of a poisonous snake versus some more affectively neutral image, such as that of an

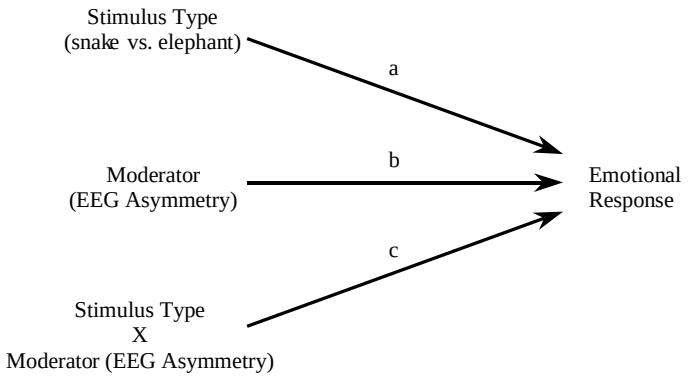


Fig. 1. Frontal EEG asymmetry as a moderator. Model adapted from Baron and Kenney (1986).

elephant. In this example, we might expect fear ratings in response to images of a poisonous snake to be relatively high and those to images of an elephant to be relatively low, with ratings in response to one, the other, or both images varying as a function of frontal EEG asymmetry.

According to this model, there are three causal paths to the emotional response—the rating of fear intensity according to our example. The first of these paths, path a, represents the causal influence of the independent variable, in this case, image type (poisonous snake verses an elephant). Path b would represent the unique contribution of trait frontal EEG activity. Path c represents the causal influence of the interaction between the type of image presented and an individual's trait pattern of resting frontal EEG asymmetry.

At its simplest, the moderator hypothesis states that path c is significant. Baron and Kenny (1986) note, however, that a significant contribution of this path alone does not necessarily present a strong case for a moderator effect. While a main effect of the independent variable is possible in this model, and in the case of this example even likely, it is preferred that the moderator variable be uncorrelated with both the stimulus and emotional response variables, if the moderator is specifically to be identified as a moderator only (i.e. not simultaneously a moderator *and* mediator). It is easy to see that a correlation would not exist between frontal EEG asymmetry and the likelihood of encountering an image of a poisonous snake versus an elephant (at least in the experimental context). Less certain, however, is that frontal EEG asymmetry is unrelated to continuous measures of affective intensity, regardless of the particular stimulus being rated. If frontal EEG activity predicted fear intensity ratings independent of the eliciting stimulus, it could be somewhat difficult to disambiguate the moderating influence (path c) of frontal EEG asymmetry on our specific construct of interest—emotion responsivity—from its main effect (path b) of predicting intensity. Such a condition would not preclude the identification of a moderator, however. A statistical method for dealing with this problem is outlined below.

Moderator models of frontal EEG asymmetry assume that frontal EEG asymmetry itself is most likely a continuous variable. This is true, even if hemispheres act independently of each other to achieve their moderating effects. Raw EEG is always measured as a continuous variable, and while frontal EEG asymmetry can be artificially dichotomized into categorical

variable, such an approach is not recommended, in part because doing so reduces statistical power and may result in artificially obtained "groups" that distort the underlying continuous relationships. Following from Davidson's diathesis/stress theory of psychopathology and affective style, it makes the most sense to analyze frontal EEG asymmetry as a continuous moderator, and the following examples adhere to this. This data analytic approach is perhaps best illustrated with the snake/fear example, because the diathesis/stress theory models "stress" as an environmental event beyond an individual's direct control. Recall that this example assumes that any given individual's trait pattern of frontal EEG asymmetry will serve to either attenuate or amplify a fear response to the visual stimuli.

Baron and Kenney (1986) present three possible models representing the interaction between an independent variable (e.g., snake versus elephant) and a continuous moderator variable (e.g., resting frontal EEG asymmetry). These are (1) the *linear model*, (2) the *non-linear model*, and (3) the *step–function model*. In the linear model, one would expect to find a gradual, linear change in the effect of the independent variable on the outcome criterion as a function of the moderator. In terms of the example, the relationship between the interaction term (the snake/elephant by EEG asymmetry interaction) and fear ratings would be gradual and steady. That is, frontal EEG asymmetry should both attenuate and amplify the effect of the fear-relevant (but not fear-irrelevant) stimulus, depending on the relative difference between the left and right hemispheres. This model would then predict that individuals with relatively greater right frontal EEG asymmetry would be expected to show an amplified fear response, and individuals with relatively greater left frontal activity should show an equal and opposite attenuation in their fear response, with these effects specific to the snake stimulus.

In the non-linear model, a quadratic equation would best exemplify the relationship between the interaction term and the outcome criterion. Following from the example, it might be expected that relative right frontal activity would correspond with a more intense fear rating in response to the snake image presentation, while finding that relative left frontal activity was not different from symmetrical activity in terms of influencing fear ratings,[3] with effects again specific to the snake and not the elephant stimulus.

In the step–function model, some critical level of the moderator variable is assumed. In terms of the example, this model would state that fear responses are at a clearly defined normative level until a particular threshold in relative right frontal EEG asymmetry is crossed, the result of which would be a large jump in the intensity ratings of fear in response to the snake but not elephant image presentation. There is no evidence to suggest such a model is likely.

In data analytic terms, each of these models can be tested with simple regression equations per the recommendations of Cohen and Cohen (1983) and Aiken and West (1986). This recommendation holds if the independent variable is continuous as well. Further, standard

[3] The potentially independent contributions of the left and right frontal regions to the observed interaction would be important to examine in subsequent analyses. Although parsimonious to assume relatively comparable magnitude of effects from the left and right hemisphere in the linear model, this need not be the case, as a single hemisphere could be responsible for a majority of the effect, with the other hemisphere contributing little. In the case of the nonlinear model, it obviously cannot be assumed that both hemispheres contribute equally across the whole range of asymmetry scores, and underscores the desirability of follow-up analyses involving hemisphere (cf. Allen et al., this issue).

general linear model (GLM) approaches are essentially equivalent to regression approaches, and their flexibility makes them in many cases more desirable, especially with regard to certain common statistical packages, such as SPSS, etc. In cases where the moderator variable is independently correlated with the criterion variable, it may be desirable to calculate one's general linear model using a type 1 sum of squares in order to control for multicolinearity with the moderator by independent variable interaction term. This is easily accomplished in most popular statistical packages.

### *5.2. Empirical support for frontal EEG asymmetry as a moderator*

Thumbnail reviews of a large number of studies relevant to this section can be found in Table 2. Early studies suggest that frontal EEG asymmetry may serve as a moderator involved in predictions of emotional reactions in infants. For example, Davidson and Fox (1989) found that infants who cried in response to maternal separation had greater right frontal activity at rest than those who did not. This result was replicated by Fox et al. (1992), who also found that this effect was modestly stable over 5 months.

Similar findings have been obtained in adults. For example, when asked by Tomarken et al. (1990) to report affective responses to emotional film clips, individuals with greater right frontal activity at rest responded with more intense levels of negative affect to negatively-valenced film clips, particularly those involving fear (Tomarken et al., 1990). In a similar report, Wheeler et al. (1993) found that individuals with greater right frontal activity responded with more intense negative affect to negatively valenced films, and that individuals with greater left frontal activity responded with more intense positive affect to positively valenced films. These studies suggest that individual differences in emotional responding are in part a function of individual differences in trait frontal EEG asymmetry. Indeed, Davidson (1998a) has cited these findings to argue that one's affective style—as indicated by one's trait frontal EEG asymmetry—may in part determine one's risk for certain affective disorders such as depression and anxiety, a proposal that clearly identifies frontal EEG asymmetry as a moderator of emotion and related affective processes. While neither of these studies explicitly assessed the moderator hypothesis in terms of the recommendations of Baron and Kenny (1986), each adopted a similar approach. Each study can be conceptualized in terms of Fig. 1 as follows: (a) the stimulus type is the film type (positive or negative); (b) resting frontal EEG asymmetry is the potential moderator; and (c) intensity rating would be the emotional response. In the end, both Wheeler et al. (1993) and Tomarken et al. (1990) provide compelling—though not definitive—evidence that resting frontal EEG asymmetry is a genuine moderator of emotional responding.

Recently, Henderson et al. (2001) explicitly tested frontal EEG asymmetry recorded at 9 months of age as a moderator of the relationship between negative affectivity, also measured at 9 months, and social wariness at approximately 4 years of age. In this work, 9-month-old negative emotionality predicted social wariness at 4-year follow-up in infants who'd shown relatively greater right frontal EEG activity at 9 months. No relationship was found between social wariness behaviors at follow-up and infant frontal EEG asymmetries favoring the left. It is noteworthy that these results are consistent with Baron and Kenny's (1989) *non-linear* moderator model. More recently, Coan and Allen (2003c) conducted a moderator analysis of resting frontal EEG asymmetry and emotional experience using a

Table 2
Trait frontal asymmetry as a predictor of state dependent changes

| Citation | *N* | Age | Handedness | Reference scheme | Independent variable | Dependent variable | Hem | Results summary |
|---|---|---|---|---|---|---|---|---|
| Davidson and Fox, 1989 | 13 infant females | 10 months | R (parents) | Cz | EEG at F3/4, P3/4 | Infant response to maternal separation (crying vs. not-crying) | Yes | Criers, ↑ RFA non-criers, ↑ LFA |
| Fox et al., 1992 | (1) 33 infants (17 female), (2) 13 infants (7 females) | (1) 14–24 months, cross-sectional, (2) 7–12 months, longitud-inal | R/L | Cz | EEG at F3/4, P3/4 | Infant response to maternal separation (crying vs. not-crying) | Yes | Criers, ↑ RFA effects consistent over time |
| Hagemann et al., 1998 | 37 (22 female) | 19–44 years | R | Cz, LM | EEG at F3/4, T3/4, C3.4, P3/4, A1/2 | Positive affect (PA), negative affect (NA), affective bias (AB), and generalized reactivity (GR), all in response to affective slides | No | Cz, 8 min resting: ↑ LFA, ↑ PA<br>Cz, 4 min eyes closed: ↑ LFA, ↑ PA<br>LM reference: no effects ↑ R T3/4, ↑ NA<br>Note: overall results equivocal with regard to A/W model |
| Henderson et al., 2001 | 97 infants (51 females) | Longitud-inal; time 1, 9 months; time 2, 48 months | No information | AR | EEG at F3/4, C3/4, P3/4 and O1/2; negative reactivity (NR) | Sociability (S) and social wariness (SW) | No | ↑ RFA and ↑ NR, ↑ SW |
| Tomarken et al., 1990 | 32 females | 17–41 years | R | Cz | EEG at F3/4, T3/4, P3/4, C3/4 | Reported positive affect (PA) and negative affect (NA) following film clips | No | ↑ RFA, ↑ NA<br>↑ RFA, ↑ PA-NA difference<br>↑ RFA, ↑ fear report |
| Wheeler et al., 1993 | 26 females with stable asymmetry across sessions | 17–21 years | R | Cz | EEG at F3/4, T3/4, P3/4, C3/4 | Reported positive affect (PA) and negative affect (NA) following film clips | No | ↑ LFA, ↑ PA<br>↑ RFA, ↑ NA |

RFA: right frontal activity, LFA: left frontal activity; RATA: right anterior temporal activity, LATA: left anterior temporal activity; RPA: right parietal activity, LPA: left parietal activity.

general linear model approach similar to that used by Henderson et al. (2001). Using data previously reported on in Coan et al. (2001); Coan and Allen (2003c) sought to assess the extent to which trait frontal EEG asymmetries moderated self-reported emotional experience in response to posed emotional facial expressions that depicted anger, disgust, fear, joy and sadness. To explicitly test a moderator hypothesis, Coan and Allen (2003c) constructed a single general linear model where emotion type, trait frontal EEG asymmetry, and an emotion by trait EEG asymmetry interaction were used to predict emotional experience. This model was able to accommodate both the categorical (emotion) and continuous (trait frontal EEG asymmetry) predictors, as well as their interaction, easily. Additionally, these predictors were interacted with the other categorical factors of reference scheme (average versus linked mastoid) and particular frontal region (F3/4, F7/8 and FTC1/2) to assess the degree to which these additional factors influenced the effects of interest. If frontal EEG asymmetry moderates emotional experience and intensity, then this model would result in a significant emotion by frontal EEG asymmetry interaction. Coan and Allen (2003c) found that relatively greater left frontal activity at rest predicted an increased likelihood of reporting an experience of anger, joy, disgust, but not sadness and fear (trait EEG asymmetry by emotion interaction, $F(4, 865) = 3.53$, $P < 0.01$) independently of reference scheme (average and linked mastoid) or specific frontal region (F7/8, F3/4 and FTC1/2). This moderator effect was, however, only partially consistent with predictions of the approach/withdrawal model of frontal brain asymmetry. Positive correlations between frontal EEG asymmetry and experience were found, as predicted, in the cases of joy and anger, but the predicted negative relationships between trait frontal EEG asymmetry and disgust, in addition to fear and sadness, were not. These findings may in part be due to methodological idiosyncrasies such as the use of voluntary emotional facial expressions as emotional stimuli, but suggest that the role of frontal EEG asymmetry as a moderator of emotional response may only partially support of the approach/withdrawal model.

Other studies suggest the same. Using normed emotion eliciting images, Hagemann et al. (1998) essentially failed to replicate earlier findings. In their study, Hagemann et al. (1998) did indeed find that individuals with relatively greater left frontal activity at rest tended to respond more positively to positively valenced images, but only when using a Cz reference (arguably the most problematic of all reference montages; see Hagemann et al., 2001). The effect did not generalize to frontal EEG recorded using a linked mastoids reference montage. Other results reported by Hagemann et al. (1998) were generally inconsistent with the approach/withdrawal model advocated by Davidson and colleagues. For example, Hagemann et al. (1998) found that relatively greater left anterior temporal activity at rest was associated with more intense experience reports associated with *negative* affect, whereas the approach/withdrawal model would predict precisely the opposite. Such discrepant findings have prompted Davidson (1998b) to suggest that that Hagemann et al.'s (1998) study was methodologically inconsistent with earlier ones supportive of the approach-withdrawal model, pointing out that in Wheeler et al.'s (1993) study, only data from individuals with highly stable frontal EEG asymmetry patterns (across 3 weeks of measurement) were analyzed. Both authors have noted the need to resolve these inconsistencies with further research.

Inconsistencies aside, if the possibility of frontal EEG activity as a moderator of emotional responses is ultimately borne out, there are likely to be consequences for affectively

based psychological disorders. Indeed, Davidson's theory of frontal EEG asymmetry as a partial determinant of affective style proposes explicitly that certain affective styles serve as diatheses that, in response to the appropriate stimuli, can increase risk for certain forms of psychopathology. Thus, evidence that frontal EEG asymmetry may moderate risk for psychopathology merits consideration.

## 6. Frontal EEG asymmetry as a risk factor and possible moderator of psychopathology

Resting frontal EEG asymmetry may serve as an index of risk for a variety of emotion-related disorders, including depression and anxiety. Whether it indexes a *general* propensity to respond in ways consistent with depression or anxiety, or a *specific* tendency to do so in some emotionally evocative situations and not others, remains an empirical question. Yet evidence of frontal EEG asymmetry's role in moderating emotional responsivity suggests a similar role in the development of various disorders involving affect.

Researchers have identified a link between relatively greater right frontal resting activity—or relatively lower left frontal resting activity—and depression (e.g., Henriques and Davidson, 1990, 1991; Schaffer et al., 1983) including seasonal depression (Allen et al., 1993). In the first report of this type, Schaffer et al. (1983) found that higher scores on the Beck Depression Inventory (BDI) were associated with relatively greater right frontal resting activity in their sample of undergraduate research participants (see Table 3). Subsequent studies have identified the same relationship in clinically diagnosed participants (Allen et al., 1993; Gotlib et al., 1998; Henriques and Davidson, 1991). In some of these studies, participant samples have included euthymic individuals who have suffered previous bouts of depression (e.g., Gotlib et al., 1998; Henriques and Davidson, 1990). Some researchers, however, have failed to replicate the relationship between resting frontal EEG asymmetry and depression (Reid et al., 1998). For example, despite the use of two separate and large samples, Reid et al. (1998) were unable to detect such a relationship. In interpreting the findings, Reid et al. (1998) suggested that given the heterogeneity of depression, frontal EEG asymmetry might tap only one of several risk trajectories associated with depression. Reid et al. also speculated that some unidentified qualities of their research environment may have been to blame, noting that traits may interact in particular ways within particular environments and that these interactions may have masked preexisting asymmetries in their research participants.

Evidence that infants of depressed mothers show relatively greater right frontal resting activity further suggests a link between frontal EEG asymmetry and depression (e.g., Dawson et al., 1999a; Field et al., 1995). For example, Dawson et al. (1997) found that infants of depressed mothers showed less left frontal activity than those of non-depressed mothers and that such lower left frontal activity discriminated infants whose mothers were diagnosed with major depression from those with mothers whose symptoms were sub-threshold. More recently, Dawson and colleagues have found that infants of depressed mothers are less affectionate and show evidence of relatively less left frontal activity not only at rest, but also while interacting with their mothers and while interacting with familiar strangers (Dawson et al., 1999b). In independent investigations, Field et al. (1995) have achieved similar ef-

Table 3
Trait frontal EEG asymmetry and measures of psychopathology

| Citation | *N* | Age | Handedness | Reference scheme | Independent variable | Dependent variable | Hem | Results summary |
|---|---|---|---|---|---|---|---|---|
| Allen et al., 1993 | 8 females (4 with seasonal affective disorder; SAD) | No information | R | Cz | SAD (S) vs. control (C) groups; pre–post bright light treatment | EEG at F3/4, P3/4 | Yes | S ↓ LFA Unchanged by treatment |
| Allen et al., in press | 30 depressed females | 18–45 | R | Cz, LM, AR | Time of assessment to examine stability of asymmetry over time in depression | EEG at F3/4, F7/8, FTC1/2, others | No | Asymmetry stable across 3–5 monthly assessments, median intraclass correlation = 0.61 |
| Baehr et al., 1998 | 24 (13 depressed; no sex information) | 43–57 years | No information | Cz | Depressed (D) and non-depressed (ND) groups (BDI median split) | Percent time spent with RFA vs. LFA in F3/4 | No | D, ↑ pct time with RFA |
| Bruder et al., 2001 | 53 (28 females) | 18–65 | No information | Nose | Resting EEG at F3/4, FP1/2, F7/8, FC5/6, FT9/10, C3/4, T7/8, CP5/6, TP9/10, P3/4, P7/8, P9/10, O1/2 | Recovery vs. non-recovery from depression following SSRI (fluoxetine) treatment | Yes | In women: non-responders, ↑ RFA |
| Davidson et al., 2000 | 28 (no sex information) | 19–68 years | R | LM | Social phobics vs. controls—anticipating public speech | EEG at AF1/2, F3/4, F7/8, T3/4, P3/4, C3/4, Cz, and Fz in alpha 1 (8–10 Hz); and alpha 2 (10–13 Hz) | Yes | Alpha 1: phobics, ↑ RFA/RATA |
| Davidson et al., 1985 | 20 (10 depressed; 7 females in each group) | 18–23 years | R | Cz | Happy, sad and neutral face pictures, depressed vs. non-depressed, left visual field (LVF) vs. right visual field (RVF) | EEG at F3/4, P3/4 | Yes | Group differences in frontal asymmetry between RVF and LVF presentations appears to account for group differences in self-report ratings of happiness in response to lateralized picture presentations |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Dawson et al., 1997 | 117 infants (52 females) | 13–15 months | No information | LM | Depressed (D) vs. non-depressed (ND) mothers; major depression (MD) vs. sub-depression (SD) | Infant EEG at F3/4 and P3/4 | Yes | D, ↓ LFA<br>MD, ↓ LFA compared to SD |
| Dawson et al., 1999a,b | 99 infants (60 females) | 13–15 months | R/L | LM | Depressed (D) vs. non-depressed (ND) mothers; interaction with mother vs. familiar adult | Infant EEG at F3/4 and P3/4 | Yes | D, ↓ LFA (across other conditions) |
| Dawson et al., 1999a,b | 117 infants (52 females) | 13–15 months | No information | LM | Depressed (D) vs. non-depressed (ND) mothers | Infant EEG at F3/4 and P3/4; affection behaviors (AB) | Yes | D, ↓ AB (D and ↓AB), ↓ LFA |
| Dawson et al., 1997 | 30 infants (21 females) | 11–17 months | No information | Cz | Emotional faces during emotional stimuli; depressed (D) vs. non-depressed (ND) mothers | Infant EEG at F3/4 and P3/4 | Yes | Bilateral ↓ in activation during negative faces in D group |
| Dawson et al., 1992 | 27 infants (no sex information) | 11–17 months | No information | Cz | Emotion conditions (play w/mother (P), stranger approach (SA), maternal separation (MS); depressed (D) vs. non-depressed (ND) mothers; secure (S) vs. insecure (IS) attachment | Infant EEG at F3/4 and P3/4 | Yes | S: If D, ↓ LFA during P<br>D, ↓ RFA |
| Debener et al., 2000 | 37 (25 female) | 23–64 years | L/R (most R) | Linked earlobes | Depressed (D) vs. non-depressed (ND) groups | EEG at Fp1/2, F3/4, F7/8, C3/4, T3/4, T5/6, P3/4, O1/2 | Yes | D, ↓ LFA, but not stable over time<br>C, ↑ temporal stability in asymmetry |
| Earnest, 1999 | 1 female (case study) | 14 years | No information | Cz | Pre and post biofeedback treatment to ↑ LFA | BDI score | No | ↓ BDI score post treatment |
| Field et al., 1995 | 32 (16 female) | 3–6 months | R (mothers) | Cz | Depressed (D) vs. non-depressed (ND) mothers | Infant and mother EEG at F3/4 and P3/4 | Yes | D, ↑ RFA (mothers and infants) |

Table 3 (*Continued*)

| Citation | *N* | Age | Handedness | Reference scheme | Independent variable | Dependent variable | Hem | Results summary |
|---|---|---|---|---|---|---|---|---|
| Field et al., 2000 | 160 depressed, 100 non-depressed, all females | Mean = 17.8 | No information | Cz | Depressed (D) vs. non-depressed (ND) mothers and their respective infants | Infant and mother EEG at F3/4 and P3/4 | Yes | D, ↑ RFA (mothers and infants) |
| Fox et al., 1996 | 96 (56 female) | 46–62 months | No information | Cz | EEG at F3/4, P3/4 and O1/2 | Sociability (S), externalizing (E) and internalizing (I) | Yes | (↑ S and ↑RFA), ↑ E<br>(↓ S and ↑RFA), ↑ I |
| Gilbert et al., 1999 | 50 male smokers | Mean = 28.1 | R | | Smoking (S) and non-smoking (NS) conditions, NEO personality inventory (NEO-PI), fagerstrom TOLERANCE questionnaire (FTQ), negative mood (NM) | EEG at F3/4 and P3/4 | No | ↑ NM, ↑RFA<br>NS, ↑RFA |
| Gotlib et al., 1998 | Study 1: 77, study 2: 59 all female | No information | R | Cz | Previously depressed (PD), depressed (D) and never depressed (Nev) groups | EEG at F3/4, mood/cognitive measures | Yes | PD, D, ↓ LFA<br>No other effects, suggesting no cognitive mediation |
| Henriques and Davidson, 1990 | 14 (6 previously depressed) | $D_{mean}$: 37.4 years, $C_{mean}$: 34.7 years | R | Cz, LM | Never depressed (Nev) and previously depressed (PD) groups | EEG at F3/4, F7/8, T3/4, T5/6, P3/4, C3/4 | Yes | D, ↓ LFA<br>D, ↑ RPA |
| Henriques and Davidson, 1991 | 28 (18 female) | 31–57 years | R | Cz, LM, AR | Depressed (D) vs. non-depressed (ND) groups | EEG at F3/4, F7/8, T3/4, T5/6, P3/4, C3/4 | Yes | Cz: D, ↑ RFA<br>AR: D, ↑ RFA<br>LM: no effects |
| Heller et al., 1997 | 40 (22 female) | No information | R | LM | Anxious (A) and control (C) groups; anxious arousal (Ar) vs. worry (W) tasks | EEG at F3/4, A1/2, P3/4 | Yes | A, ↑ LFA (A and Ar), ↑ RPA |
| Jones and Field, 1999 | 30 (no sex information) | Mean = 18.8 years | No information | Cz | Music (Mu) vs. massage (Ma) therapy; pre, during and post tests | Depression measures and EEG at F3/4, P3/4 | No | ↑ LFA from pre to during, and from during to post in both Mu and Ma |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Jones et al., 1998 | 25 infants (25 female) | 1 month | No information | Cz | Pre, during and post massage | Infant EEG at F3/4 and P3/4 | No | ↓ RFA from pre to during and from during to post |
| Jones et al., 1998 | 63 infants (no sex information) | 1 week | No information | Cz | Depressed (D) vs. non-depressed (ND) mothers | Infant EEG at F3/4 and P3/4, vagal tone | Yes | D, ↓ LFA<br>D, ↓ vagal tone |
| Jones et al., 1997 | 44 infants (no sex information) | 1 month, 3 months (longitudinal) | No information | Cz | Depressed (D) vs. non-depressed (ND) mothers | Infant EEG at F3/4 and P3/4 | Yes | D, ↑ RFA<br>↑ RFA, ↑ Neg affect pattern stable |
| Kentgen et al., 2000 | 25 females | 12.2–18.8 years | R | Nose | Depressed (D) vs. non-depressed (ND) adolescents, anxiety comorbidity (A), no anxiety | EEG at F3/4, F7/8, C3/4, T7/8, P3/4, P7/8 | Yes | No frontal relationships.<br>D, ↓ RPA |
| Miller et al., 2002 | 110 (66 female) | Mean = 26.6 | R | AR | Males (M) vs. females (F), history of childhood depression (D) vs. No depression history (ND) | EEG at F3/4, AF3/4, F7/8, FC1/2, FC5/6, C3/4, T7/8, P3/4, P7/8, O1/2 | Yes | D and M, ↑ LFA<br>D and F, ↑ RFA |
| Minnix et al., this volume | 12 (6 females) | 19–52 years | R | LE | Reassurance seeking (RS); Beck Depression Inventory (BDI) | EEG at F3/4, FP1/2, F7/8, C3/4, T3/4, T5/6, P3/4, O1/2 | No | ↑ RS and ↑ BDI, ↑ LFA<br>↓ RS and ↑ BDI, ↑ RFA |
| Nitschke et al., 1999 | 67 (39 female) | 17–20 years | R | LM | Anxious apprehension (AAp), anxious arousal (AAr), depressed (D), co-morbid (CM) and control groups (C) | EEG at F3/4, F7/8, T3/4, T5/6, P3/4 | Yes | AAr, ↓ LFA<br>Aap no ↓ LFA |
| Papousek and Schulter, 2001 | 50 (25 females) | No information | R | Nose | Resting EEG and electrodermal activity (EDA) at two time points (T1 and T2); depression (D) and anxiety (A) measured | EEG at FP1/2, F3/4, P3/4 | No | If ↑ RFA AND ↑ anxiety, ↑ EDA<br>If ↑ LFA and ↑ depression, ↑ EDA |

Table 3 (*Continued*)

| Citation | *N* | Age | Handedness | Reference scheme | Independent variable | Dependent variable | Hem | Results summary |
|---|---|---|---|---|---|---|---|---|
| Papousek and Schulter, 2002 | Study 1: 56 (30 female), study 2: 128 (68 female) | Study 1: 18–36 years, study 2: 18–31 years | R | Nose | Study 1: anxious tension (AT); negative mood (NM); Time 1 (T1) vs. Time 2 (T2), study 2: anxious tension (AT); depression (D); anxiety; Time 1 (T1) vs. Time 2 (T2) | EEG atFP1/2, F3/4, T3/4, P3/4 for both Alpha 1 ($\alpha$1) and Alpha 2 ($\alpha$2) | No | In both studies, $\Delta$ AT covaries with $\Delta$ $\alpha$2 asymmetry in the fronto-polar region |
| Petruzzello and Landers, 1994 | 19 males | Mean = 22.7 years | R | LM | Pre and post rigorous exercise conditions | EEG at F3/4, T3/4; state measures of anxiety level | Yes | ↓ Anxiety post-exercise<br>↑ LFA post-exercise |
| Reid et al., 1998 | Study1: 36 (17 depressed), study 2: 27 (13 depressed), all female | Study 1: mean = 18.53 years, study 2: mean = 27.54 years | R | Cz, LM, AR | Study 1: depressed (D) vs. non-depressed (ND) groups (BDI); study 2: depressed (D) vs. non-depressed (ND) groups (SCID) | EEG at Fp1/2, F3/4, F7/8, T3/4, T5/6, P3/4, C3/4, O1/2, A1/2, FTC1/2, TCP1/2, PO1/2 | No | D and ND not different (both studies)<br>↑ left anterior temporal activation in depressed (study 2, in LM reference, trend in AR reference) |
| Rosenfeld et al., 1996 | 5 (4 female) | No information | R | Cz | EEG at F3/4 | Pre and post therapy session reports of affect; affect change (AC) score | No | Subjects with ↑ LFA at beginning of session show ↑ change from neg to pos affect |
| Schaffer et al., 1983 | 15 (10 female) | No information | R | Cz | High vs. low BDI scores | EEG at F3/4, P3/4 | Yes | ↑ BDI, ↑ RFA |
| Silva et al., 2002 | 55 females | No information | R | AR | Restrained eaters (RE) vs. unrestrained eaters (UE) | EEG at F3/4, P3/4 | Yes | RE, ↑ RFA |
| Tomarken et al., this volume | 38 (20 female) | Mean = 13 years | R | Cz, LE, AR | Family history of depression (F) vs. no family history (NF); socio-economic status (SES) | EEG at F3/4 | No | F: ↓ LFA<br>↓ SES, ↓ LFA |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Wiedemann et al., 1999 | 48 (39 female) | Mean = 36.55 years | R | Cz | Panic (P) vs. control groups (C); conditions: rest (R), neutral stim (N), panic stim (Pn), anxiety stim (A), emotional stim (E), motor task (M) | EEG at F3/4, P3/4 | Yes | Rest: P, ↑ RFA<br><br>A: P, ↑ RFA<br>P, ↓ LFA when shown erotic pictures |

RFA: right frontal activity, LFA: left frontal activity; RATA: right anterior temporal activity, LATA: left anterior temporal activity; RPA: right parietal activity, LPA: left parietal activity.

fects, reporting more right frontal activity (not less left frontal activity) in depressed versus non-depressed mothers, and correspondingly similar differences in their respective infants. Infant studies such as these are important not only because they add to the growing literature on frontal EEG asymmetry and depression, but because they hold the potential for suggesting the origin of trait frontal EEG asymmetries. EEG spectra are highly heritable (see Lykken et al., 1982), and though it is impossible to tell from these reports the degree to which infant frontal EEG asymmetries derive from genetic versus environmental influences, the finding of mother/infant asymmetry similarities points to the need for research on the heritability of frontal EEG asymmetry, in addition to research designed to identify which maternal behaviors or other environmental factors may influence infant trait asymmetries. To date, there is limited evidence bearing on whether frontal EEG asymmetries are heritable (Coan, 2003; MacDhomhail et al., 1999). For example, in a small sample of female mono- and di-zygotic twins, Coan (2003) estimated that genetic influences may account for as much as 22% of the variance in resting mid-frontal EEG asymmetry, and that most of this genetic influence is non-additive. Other designs would also prove informative in addressing this question, including an adoption design comparing infants of depressed versus non-depressed adoptive mothers.

If frontal EEG asymmetry were indeed a moderator of risk for psychopathology, one would expect trait levels of frontal EEG asymmetry to be relatively stable, even as episodes of depression come and go. As a moderator, trait (stable) frontal EEG asymmetry would interact with other environmental variables to cause depressive episodes; environmental variables, which are potentially less consistent, would serve as the co-determinants of any particular episode. If, by contrast, frontal EEG asymmetry changes to a significant degree with the presence or absence of an affective disorder, one would conclude that frontal EEG asymmetry potentially acts as a mediator of psychopathology. (And a more complex possibility exists, such that asymmetry has some trait-like predictive value in the face of episode-dependent changes, thus potentially serving as moderator *and* mediator.) A review of the literature suggests a somewhat inconsistent picture regarding the stability of trait frontal EEG asymmetry in relationship to affective psychopathology. For example, Jones and Field (1999) have found that music or massage therapy applied to depressed adolescents resulted in the attenuation of their group level relative right frontal activity, suggesting that relative decreases in the magnitude of psychopathology can result in corresponding decreases in relative right frontal resting activity. Further, Jones et al. (1998) have demonstrated that applying massage therapy can produce alterations in frontal EEG asymmetry in infants. In this work, applying massage therapy to 1-month-old infants decreased relative right frontal EEG activity from pre-test to mid-treatment and from mid-treatment to post-test (Jones et al., 1998).

Two studies offer some insight into the stability of trait frontal EEG asymmetry across time in adults undergoing treatment for depression. Debener et al. (2000) examined 15 medicated depressed patients on two occasions separated by 2 weeks, they found (1) that control subjects showed relatively greater left frontal resting activity than did subjects who had depression (this finding was anticipated) and (2) although depressed subjects exhibited lower test–retest stability of frontal EEG asymmetry than did controls, no systematic mood-dependent changes in asymmetry occurred across sessions in these depressed patients. Patients in this study received a variety of antidepressant compounds, most initiated

prior to the first EEG assessment, and 11 of the 15 additionally received benzodiazepines, thus raising the possibility that the variability in the depressed patients could reflect the acute effects of the initiation of a trial of medication; no evaluation of this possibility was performed.

One study examined the stability of frontal EEG asymmetry in a non-medicated sample of depressed patients as they progressed through a non-pharmacological treatment (Allen et al., 2004; reported in preliminary form in Urry et al., 1999). Allen et al. (2004) found evidence of adequate stability in resting frontal EEG asymmetry in depressed patients. Depression and frontal EEG asymmetry were assessed at 4-week interals. Across three occasions of measurement the median ICC at frontal sites (across three reference schemes) was 0.56, across five assessments the comparable value was 0.61. This stability was apparent despite rather substantial improvements in clinical status over the same interval. These values are comparable to those reported by Debener et al. (2000) for control subjects, and by Tomarken et al. (1992b) in unselected college students. Interestingly, just as with Debener et al., Allen et al. found that changes in frontal asymmetry over assessments was not related patient's clinical status or mood. The most obvious difference between the two studies is that patients in Allen et al. (2004) received no medication, whereas those in the study of Debener et al. (2000) received a variety of antidepressant compounds and most additionally received benzodiazepines. Clearly a systematic investigation of the impact of antidepressant and antianxiety medications on frontal EEG asymmetry would be desirable, both in terms of understanding the extent to which they may alter asymmetry, and also in terms of informing questions related to mechanism of action.

In aggregate, these studies tentatively suggest some trait-like stability in frontal EEG asymmetry across time in depressed subjects, but provide no evidence to suggest that variation in frontal EEG asymmetry across measurement occasions is due to changes in clinical status. From a moderator perspective, it would be expected that test–retest be fairly high even in clinical populations as symptoms wax and wane. While the evidence for this is strong on one study, but not another, it is consistently the case that occasion-related variance in resting frontal activity is *not* linked to clinical state.

Most of the work on frontal EEG asymmetry and psychopathology has concerned depression, but several studies have also examined anxiety disorders as well. Relatively greater right frontal activity has been associated with panic disorder (Wiedemann et al., 1999) and social phobia (Davidson et al., 2000), while anxious apprehension has been associated with relatively greater left frontal activity (Heller et al., 1997). Although the general pattern is consistent with the model suggested by Davidson (1998a,b), inconsistencies have prompted Heller and colleagues (e.g., Heller and Nitschke, 1998) to propose a revised valence model of frontal EEG asymmetry and anxiety; i.e., anxiety is comprised of two distinct though related processes, anxious apprehension and anxious arousal, and these processes are reflected in frontal EEG asymmetries as relatively greater left frontal and right parietal activity, respectively (see also Nitschke et al., 1999). Although there are data supportive of this position (Heller et al., 1997; Nitschke et al., 1999), it is hard to reconcile with other findings that place all forms, or nearly all forms, of anxiety squarely in the domain of relatively greater right frontal activity. In an attempt to resolve this discrepancy, Heller and Nitschke (1998) have argued that affective valence may account for the difference between their findings and those of Davidson et al. (2000) and Wiedemann et al. (1999). In particular, Heller and

Nitschke (1998) have argued anxiety coupled with negative affective valence should be associated with the pattern of right frontal activity that other researchers have found in their studies of frontal EEG asymmetry and anxiety. Thus, ultimately Heller and colleagues argue that anxiety can become manifest in frontal EEG asymmetry as either relatively greater left frontal, relatively greater right frontal, or relatively greater right parietal activity, depending on the relative presence or absence of anxious apprehension, anxious arousal and negative affect.

## 7. Frontal EEG asymmetry as a possible mediator of emotional responding

Systematic alterations in frontal EEG asymmetry in response to specific emotional stimuli suggest the possibility that the systems tapped by frontal EEG asymmetry may also fulfill a mediating role in emotional responding. While this need not be true of all emotional responding, it may well be true of the motivational properties that form one or more of the components of emotional responding.

Modeling frontal EEG asymmetry as a mediator. Just as with modeling frontal EEG asymmetry as a moderator, it will be useful to review the data analytic and statistical considerations underlying the specification of a mediator. Fig. 2 represents a mediational model of frontal EEG asymmetry in emotional responding, also adapted from Baron and Kenny (1986). For this illustration, consider again the independent variable example discussed previously, where subjects are presented with an image of either a poisonous snake (the fear stimulus) or an elephant (a neutral stimulus).

Frontal EEG asymmetry could be said to be a mediator of emotional responding if a measurable change in it is necessary—at least in part—for the relationship between some emotion-eliciting stimulus and the subsequent emotional response. That is, it could be said to mediate emotional responses to the degree that it serves as, or is highly correlated with, the mechanism by which emotional stimuli have their effects. In the case of the example, arguing that frontal EEG asymmetry is a mediator of fear responses is tantamount to saying that the fear response would not occur, or would occur differently, or would occur at a lower level of self reported intensity, if there was no change in frontal EEG asymmetry in the direction of increased relative right activity (in accord with the approach/withdrawal model). In this way, the mediational models refer specifically to frontal EEG *activation* as opposed to *activity*.

In Fig. 2, this situation is represented by three causal pathways, identified as paths a, b and c. In path a, changes in the type of stimulus (in the example, the presentation of a

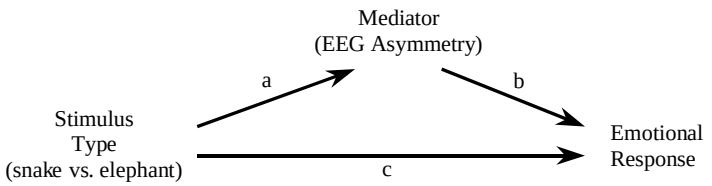


Fig. 2. Frontal EEG asymmetry as a mediator. Model adapted from Baron and Kenney (1986).

Table 4
Frontal EEG activation asymmetry as a state measure

| Citation | *N* | Age | Handedness | reference scheme | Independent variable | Dependent variable | Hem | Results summary |
|---|---|---|---|---|---|---|---|---|
| Allen et al., 2001 | 18 females | 18–38 years | R | Cz | Biofeedback training to move asymmetry towards greater left or right activity | Self report emotion, facial EMG | Yes | ↑ RFA caused ↓ positive affect, ↓ zygomatic, and ↑ corrugator activity |
| Benca et al., 1999 | 17 (4 female) | 35–63 years | R | LM | Wakefulness vs. various sleep stages (REM, StgII, SWS) | EEG at F3/4, F7/8, T3/4, T5/6, P3/4 O1/2 | Yes | Waking EEG correlated with sleep (notably REM) in frontal and temporal regions |
| Blackhart et al., in press | 77 (41 female) | 16–39 years | R | LM | Pre (PRE) and post (POST) EEG hook-up mood ratings | EEG at F3/4, FP1/2, F7/8, T3/4, T5/6, C3/4, P3/4, O1/2 | No | Women: ↓ POST mood, ↑ LFA<br>Men: ↓ POST mood, ↑ RFA |
| Coan and Allen, in press | Resting EEG: 30; state EEG:31 | No information | R | AR, LM | Resting EEG at F3/4, F7/8, FTC1/2; state EEG at F3/4, F7/8, and FTC1/2 during voluntary facial expressions of anger (A), disgust (D), fear (F), joy (J) and sadness (S). | Emotional intensity reports (IR) following voluntary facial expressions of anger (IR-A), disgust (IR-D), fear (IR-F), joy (IR-J) and sadness (IR-S) | No | ↑ resting LFA, ↑ IR-A<br><br>↑ resting LFA, ↑ IR-D<br>↑ resting LFA, ↑ IR-J<br>↑ LFA during A, ↑ IR-A<br>↑ LFA during J, ↑ IR-J<br>↑ RFA during F, ↑ IR-F |
| Coan et al., 2001 | 36 (26 female) | 17–24 years | R | Cz, LM, AR | Voluntary emotional facial expressions grouped according to approach (A), withdrawal (W) and control (C) conditions | EEG at F3/4, F7/8, FTC1/2, P3/4 | Yes | W, ↓ LFA<br>A = C |
| Collet and Duclaux, 1986 | 24 (13 female) | 18–45 years | R | AR | Emotional expression during emotional films; happy (H), sad (S) and neutral (N) | EEG at F3/4, T1/2, T3/4, T5/6, C3/4, P3/4 O1/2 | No | No effects |
| Davidson et al., 1990 | 11 females | 17–41 years | R | Cz | Emotional facial expressions during emotional film clips | EEG at F3/4, C3/4, T3/4, P3/4 | Yes | No effect of films.<br>Disgust (face), ↑ RATA<br>Joy (face), ↑ LATA |
| Davidson and Fox, 1982 | 24 infant females | ∼10 months | No information | Cz | Films of an actress performing happy vs. sad faces | EEG at F3/4, P3/4 | Yes | Happy, ↑ LFA |
| Davidson et al., 2003 | 41 (29 female) | 23–56 years | R | LE | Meditation (M) vs. control (C); pre-treatment (T1), post-treatment (T2), 4 month follow-up (T3); antibody titers to the influenza vaccine (HIA) | EEG at F3/4, FC7/8, T3/4, C3/4 | No | No frontal effects.<br>At T2 and T3, ↑ baseline activity in left central region (C3/4) in M<br>In M, greater relative left activity in C3/4 from T1 to T2 corresponded with greater HIA. |

Table 4 (*Continued*)

| Citation | *N* | Age | Handedness | reference scheme | Independent variable | Dependent variable | Hem | Results summary |
|---|---|---|---|---|---|---|---|---|
| Davidson et al., 1992 | 9 rhesus monkeys (4 female) | ~12 months | No information | LM | Benzodiazepam shot vs. vehicle | EEG at F3/4, P3/4 | Yes | Benzodiazepam, ↑ LFA |
| Davidson et al., 1993 | 9 rhesus monkeys (4 female) | ~12 months | No information | LM | Benzodiazepam shot vs. vehicle | EEG at F3/4, P3/4, freezing time in response to challenge | No | Those with most ↑ LFA to Benzodiazepam showed longest duration freezing behavior |
| Dawson et al., 1992 | 21 infants | 21 months | No information | Cz | Baseline (B) vs. "mother out" (MO) conditions | EEG at F3/4, P3/4 | No | ↑ overall frontal activation during MO |
| Ekman and Davidson, 1993 | 14 | No information | No information | LM | Duchenne (D) vs. unfelt smiles (U) vs. anger face (A) | EEG at F3/4, F7/8, C3/4, T3/4, T5/6, P3/4, O1/2 | Yes | D ↑ LFA, LATA<br>D ↑ LFA than A |
| Ekman et al., 1990 | 31 females | 17–41 years | R | Cz | Emotional facial expressions during emotional film clips; Duchenne (D) vs. unfelt (U) smiles | EEG at F3/4, C3/4, T3/4, P3/4 | Yes | D, ↑ LATA<br>U, ↑ RATA |
| Fox and Davidson, 1986 | 35 infant females | ~10 months | R (parents) | Cz | Stranger approach (SA), mother approach (MA), maternal separation (MS) condition | EEG at F3/4, P3/4 | Yes | MA, ↑ LFA (mother reach sub-condition); (↑ LFA if vocalizing)<br>MS + crying, ↑ RFA (↑ LFA if vocalizing) |
| Fox and Davidson, 1987 | 35 infant females | ~10 months | R (parents) | Cz | Stranger approach (SA) vs. mother approach (MA); facial expressions of joy (J), anger (A), sadness (S); Duchenne (D) vs. unfelt (U) smiles | EEG at F3/4, P3/4 | Yes | D, ↑ LFA than U<br>A and S (no crying), ↑ LFA<br>A and S (crying), ↑ RFA |
| Fox and Davidson, 1988 | 16 infant (no sex information) | 2–3 days | R (parents) | Cz | Emotional facial expressions during taste conditions (sucrose [S], citric acid [CA], $H_2O$) | EEG at F3/4, P3/4 | Yes | 1–3 Hz band: $H_2O$, ↑ RFA<br>S, ↑ LFA<br>6–12 Hz band: $H_2O$, ↑ RFA<br>S, ↑ LFA |
| Gilbert et al., 1994 | 32 (16 female) | 21–35 years | R | No information | Various self-report measures | EEG at F3/4, T3/4, P3/4 (others) | Yes | ↑ BDI, ↑ RFA (in normals) |
| Hagemann and Naumann, 2001 | 31 (19 female) | 19–36 | No information | Cz | Occular artifacts vs. no occular artifacts in EEG recordings | EEG at FP1/2, F3/4, F7/8, T3/4, C3/4, T5/6, P3/4, O1/2 | Yes | No effects of occular artifact in the alpha range |
| Harmon-Jones et al., 2002 | 67 (33 female) | No information | R | LE | Anger manipulation (AM) vs. no anger (NA), symptoms associated with depression (D) and hypomania-plus-biphasia (HB) | EEG at F3/4, F7/8, FT7/8 | Yes | ↑ LFA during AM<br>During AM, if ↑ HB, then ↑ LFA<br>During AM, if ↑ D, then ↑ RFA |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Harmon-Jones and Sigelman, 2001 | 42 males | No information | R | LM | Baseline (B), insult (I), no-insult (NI) conditions | Self-reported anger (A) and aggression (AG); EEG at F3/4, F7/8, P3/4 | Yes | I produced, ↑ A, ↑ AG, ↑ LFA<br>LFA correlated with anger in I, not NI<br>LFA correlated with Aggression in I, not NI |
| Jones and Fox, 1992 | 23 females | 18–22 years | R | Cz | Emotional facial expressions during videos of anger (A), happiness (H), disgust (D), and sadness (S); positive (P) vs. negative (N) affectivity groups | EEG at F3/4, T3/4, P3/4 | Yes | H, ↑ LFA<br>S, ↑ RFA<br>D, ↑ RFA<br>P, ↑ LFA during H<br>N, ↑ RFA during H |
| Kline et al., 2000 | 49 females | Mean = 64.2 years | No information | No information | Odor conditions; vanilla (V), neutral (N), valarian (VN). | EEG at Fp1/2, F3/4, F7/8, O1/2, P3/4, T5/6 | Yes | V, ↑ LFA |
| Miller and Tomarken, 2001 | 60 (30 female) | Mean = 19 | L and R | Cz | Incentive levels: large reward (LR), reward (R), no reward (NR), punish (P), large punish (LP); expectancy levels: high (HE), medium (ME) and low (LE); response levels: active (A) and passive (PS); hand response levels: left (LT) and right (RT) | EEG at F3/4, C3/4, P3/4, AF3/4 | Yes | ↑ R, ↑ LFA<br>Men: HE, ↑ LFA<br>Women: LE, ↑ LFA<br>LT, ↑ RFA<br>RT, ↑ LFA |
| Reeves et al., 1989 | 16 (7 female) | 20–50 years | R | LM | TV segments depicting positive (P) and negative (N) scenes | EEG at F3/4, O1/2 | Yes | P, ↑ LFA<br>N, ↑ RFA |
| Sabotka et al., 1992 | 15 (8 female) | 18–25 years | R | LM | Reward (R) vs. punishment (P) conditions | Ratings of happiness (H) vs. sadness (S) during conditions; EEG at F3/4, F7/8, T3/4, C3/4, O1/2, TP3/4; Approach (finger press; FP) vs. withdrawal (finger lift; FL) responses | Yes | R, ↑ LFA<br>P, ↑ RFA |
| Sanders et al., 2002 | Study 1: 39 (29 female), study 2: 26 infants | Study 1: mean = 31 years | No information | Cz | Studies 1 and 2: lavender odor (L) vs. rosemary odor (R); baseline left frontal (LF) vs. baseline right frontal (RF) | EEG at F3/4, P3 | No | Study 1: L, ↑ LFA<br>Study 2: no effects |
| Schmidt et al., 1999 | 24 males | 18–38 years | R | Cz | Prednisone (P) vs. control groups; pre-treatment (T1) vs. post-treatment (T2) | EEG at F3/4, C3/4, P3/4, O1/2 | No | T2 and P, ↑ RFA |
| Tucker and Dawson, 1984 | 9 method actors (5 female) | No information | R | LM | Imagination condition; depressed (D) vs. sexually aroused (S) | EEG at F3/4, C3/4, P3/4, O1/2 | Yes | S ↑ RFA, compared to D |

Table 4 (*Continued*)

| Citation | *N* | Age | Handedness | reference scheme | Independent variable | Dependent variable | Hem | Results summary |
|---|---|---|---|---|---|---|---|---|
| Waldstein et al., 2000 | 30 (18 female) | Mean = 24 years | R | Cz | Imagination and film conditions; happiness (H) vs. anger (A) | EEG at F3/4, C3/4, P3/4, O1/2 | Yes | H ↑ LFA compared to A |
| Zinser et al., 1999 | 72 (no sex information) | Mean = 26.3 | R | Cz | Cigarette deprivation (D) and control (C) groups by 1 cigarette "anticipation" (A) and 2 cigarette "no wait" (N) groups (2 × 2 factorial) | EEG at F3/4 | No | D,A ↑ LFA<br>Smoking itself, ↓ LFA |

RFA: right frontal activation, LFA: left frontal activation; RATA: right anterior temporal activation, LATA: left anterior temporal activation; RPA: right parietal activation, LPA: left parietal activation.

snake versus an elephant) correspond to different changes in frontal EEG activity (frontal EEG *activation*). In path b, frontal EEG activation corresponds to changes in emotional responding. In path c, changes in levels of the independent variable (snake versus elephant) correspond to changes in emotional responding. Frontal EEG asymmetry can be said to be a mediator of emotional responding to the extent that path c—the direct relationship between emotional stimuli and emotional responding—is attenuated when paths a and b are statistically adjusted for. By this reasoning, frontal EEG asymmetry would be a perfect mediator of emotional responding if statistically adjusting for paths a and b resulted in a residual of zero for path c (thus identifying frontal EEG activation as a necessary condition for emotional responding). This idealized scenario is unlikely. Rather, frontal EEG asymmetry is likely to represent, if anything, one of several mediating processes in emotional responding. If this is the case, then it would be expected that paths a and b improve the overall fit of the model, and that the residual in path c is significantly decreased when paths a and b are statistically adjusted for. Such a finding would suggest that, while not necessary, frontal EEG activation may be a sufficient condition for generating emotional responses, an idea that could in theory be tested.

In applying data analysis to this mediational model, one can start by conceptualizing each path (a, b and c) as separate regression equations. That is, one could regress frontal EEG activation (the mediator) on the independent variable, the emotional response (the criterion variable) on the independent variable, and the emotional response on frontal EEG activation. Baron and Kenny (1986) point out that coefficients from each equation can alone be used to suggest (or, more strongly, rule out) a mediating relationship. Using the example, by this method one would expect to find (1) that frontal EEG activation is related to the differential presentation of a fearful or non-fearful stimulus (represented in Fig. 2 as path a), (2) that the intensity of a fear related response will be related to the presentation of a fearful versus a non-fearful stimulus (represented in Fig. 2 as path c), and (3) that the intensity of the fear related response will be related to frontal EEG activation (represented in Fig. 2 as path b). If any of these three conditions are not obtained, then the possibility of EEG activation serving a mediating role in the observed emotional responding can effectively be ruled out. If these conditions are obtained, however, then mediation has not been ruled out until controlling paths a and b show an observed impact on path c in Fig. 2.[4]

Another possible method of testing mediational models of this sort is to utilize structural equation modeling (SEM). The SEM approach is increasing in popularity, an indirect measure of which is its conspicuous appearance as a new component of various popular statistical programs, such as SAS, Statistica, etc. (Also useful are SEM dedicated programs

[4] It should be noted that while it may be tempting to conclude, from analyses such as those described here, that mediation can be ruled in, this is in fact not generally true. With the mediational analyses described here, true mediation can at best only *fail to be ruled out*, unless statistically adjusting for paths a and b do indeed result in a residual of zero, which is exceedingly unlikely (Patrick McKnight, personal communication). Moreover, the Baron and Kenney method of addressing the status of a hypothesized mediator may suffer from unrealistic type I error rates and low statistical power with small or even medium sample sizes. The length and scope of this article precludes a detailed discussion of the many complexities involved in the determination of statistical mediation, such as issues of sample size, attempts to model multiple mediators, etc. Thus, our treatment of these data analytic issues are somewhat idealized. For additional information on mediation analyses, the reader is enthusiastically referred to Kenny et al. (1998) and MacKinnon et al. (2002).

such as LISREL and EQS. EQS is particularly recommended.) SEM approaches essentially reconstruct the three regression equations listed above as one model, where coefficients are estimated within the context of the entire covariance matrix of the model's variables. This allows one to estimate the overall "fit" of the model one has constructed, using various indices (for a thorough review of SEM approaches, see Loehlin, 1998).

### *7.1. Empirical support for frontal EEG asymmetry as a mediator*

A number of studies provide oblique evidence that frontal EEG asymmetry may indeed mediate emotional responses (see Table 4). Most of the evidence reviewed below simply demonstrates that frontal EEG asymmetry can be altered by emotional or quasi-emotional stimuli. Few studies measure other emotional responses as a correlate of changes in frontal EEG asymmetry, where each is in response to some emotional stimulus. No studies to date have explicitly tested the mediational hypothesis.

The approach/withdrawal model of frontal EEG asymmetry accommodates state changes as well as individual differences in trait propensities. According to the model, stimuli intended to elicit approach-oriented responses should result in an observed relative left frontal EEG activation, while stimuli intended to elicit a withdrawal-oriented response should result in an observed relative right frontal EEG activation. Indeed, there is evidence to support this prediction (e.g., Coan et al., 2001; Ekman et al., 1990; Davidson and Fox, 1982).

Some of the earliest work in this area was done by Davidson and Fox (1982), who found that 10–12-month-old infants showed evidence of increased left frontal EEG activity in response to films of an actress performing happy faces. Subsequently, Fox and Davidson (1986) found that infants as young as 2–3 days exhibited an increase in left frontal activity in response to drops of sugar water deposited on their tongues, while exhibiting more right frontal activity in response to neutrally flavored drops (of water). Still later, Fox and Davidson (1987) found that 10 month-old infants who reached for their mothers during a mother approach task showed more concomitant left frontal activity than infants who did not, and that babies who cried in response to maternal separation showed a similar right frontal activity effect compared to other infants. Moreover, Fox and Davidson (1988) found that anger and sadness in response to maternal separation corresponded with relatively greater left frontal activity, unless the infants were crying, in which case both anger and sadness corresponded with relatively greater right frontal activity. (This last finding presents some interesting difficulties for both the approach/withdrawal and valence models of EEG asymmetry, but nevertheless serves to illustrate the potential mediating role of processes indicated by anterior EEG alpha asymmetries.)

Similar results have been obtained in studies of adults. Using emotional films to investigate the relationship between emotional experience and frontal EEG asymmetry, Davidson et al. (1990) found that, although frontal EEG recordings averaged across the entire period of viewing emotional films did not show evidence of differences in hemispheric activity, important differences did emerge during facially expressive emotional reactions to those films. In particular, disgust films elicited relatively greater right anterior temporal activity relative to baseline, while happy films elicited more left anterior temporal activity (Davidson et al., 1990). In a reanalysis of the same data set, Ekman and colleagues (Ekman et al., 1990) found that individuals who exhibited Duchenne smiles (smiles involving activation of the

orbicularis pars lateralis muscle) in response to happy films showed more concomitant left anterior temporal activity than did individuals who exhibited "unfelt" smiles. Subsequently, Ekman and Davidson (1993) asked participants to voluntarily perform Duchenne versus unfelt smiles and again found that Duchenne smiles resulted in greater left anterior temporal, as well as left frontal, activity.

Thus, lateralized brain activity *seems* to be a potentially important element in the collection of properties that comprise at least some emotions or emotion families. This idea is borne out further by other work (e.g., Coan et al., 2001; Harmon-Jones and Sigelman, 2001; Jones and Fox, 1992). For example, Coan et al. (2001) used a voluntary directed facial action task (cf., Levenson et al., 1990) to elicit approach (joy and anger) and withdrawal (disgust, fear and sadness) related emotions, hypothesizing that approach-related emotions should result in left lateralized frontal activation while withdrawal-related emotions resulted in right lateralized frontal activation. Their predictions were partially confirmed; withdrawal-related emotions, particularly fear and sadness, did result in the expected relative right frontal activation compared to a control condition, but approach-related emotions did not result in a comparable relative left frontal activation (Coan et al., 2001). While Coan et al. (2001) did measure other emotional responses, such as subjective emotional experience reports, they did not test an explicit mediational model of frontal EEG asymmetry and emotion.

Recently, Coan and Allen (2003c) used the extant data reported in Coan et al. (2001) to assess the relationship between state frontal EEG asymmetry and emotional experience during voluntary emotional facial expressions. They found that anger and, marginally, joy were more likely to be reported if their concomitant state EEG asymmetries involved greater left activity, and that fear was more likely to be reported if its concomitant state EEG asymmetry involved greater right activity (state EEG asymmetry by emotion interaction, $F(4, 895) = 8.17$, $P < 0.001$). These results proved to be independent of reference scheme or specific frontal region. Unfortunately, this data set did not include a single dependent measure that was recorded following each emotion task, and the lack of such an outcome measure precluded a proper mediational analysis. Had each participant been asked, for example, to rate the degree to which they experienced *fear* following each emotion induction, a mediational analysis, where emotion type would be the independent variable, frontal EEG asymmetry would be the mediator and *fear experience* would be the outcome measure, would be readily forthcoming.

Further evidence of the relationship between emotional states and concomitant changes in frontal EEG asymmetry can be found in a series of studies conducted by Harmon-Jones and co-workers (Harmon-Jones and Sigelman, 2001; Harmon-Jones et al., 2003). In an investigation that probably comes closest to testing a true mediational model of frontal EEG asymmetry and emotion, Harmon-Jones and Sigelman (2001) observed that individuals who showed relative left frontal *activation* (again, change in frontal EEG asymmetry from rest) in response to an insult were more likely to report experiencing anger. Similarly, Harmon-Jones et al. (2003) observed that individuals showing left frontal activation under a different anger induction procedure also displayed more aggressive and retaliatory behavior. Interestingly, Harmon-Jones et al. (2003), also found that left frontal activation only occurred in response to an anger elicitation when coping or retaliatory responses were possible. In their study, college students were confronted either with a bogus radio broadcast confirming that a tuition increase was certain or with one that suggested that such an increase was merely

under consideration. Those subjects who were led to believe that the increase was merely under consideration (1) showed greater left frontal activation than those who believed the increase was certain and (2) were more likely to engage in coping actions such as signing and taking petitions (Harmon-Jones et al., 2003). Taken together, these studies suggest that frontal EEG asymmetry, or by inference the activity of the brain systems it measures, may mediate emotional responding, and that these systems are likely to be particularly motivational in nature, as predicted by the approach/withdrawal model.

## 8. Frontal EEG asymmetry as a trait-like moderator and a state-related mediator

One important question regarding frontal EEG asymmetry's role as either a moderator or a mediator is its relative robustness as one or the other. That is, one could ask how robust trait-like individual differences in baseline frontal EEG asymmetry are across different emotional states? An equally important question could be asked of state dependent frontal EEG asymmetries: how robust are state frontal EEG asymmetries across individuals? Generalizability theory, or *g*-theory (Cronbach et al., 1972), provides a method of estimating the reliability of particular facets of any given measure across other facets of that measure. In *g*-theory, variance components are estimated and used to calculate specific intraclass coefficients, in this context referred to as generalizability or "*g*" coefficients. Thus, *g*-theory can be applied to answer the precise questions articulated above. Indeed, for this article, *g*-theory was applied to an extant data set described in Coan et al. (2001).

In this data set, frontal EEG asymmetries were recorded from 36 research participants both at rest and during a voluntary directed facial action task wherein they were asked to perform voluntary facial expressions denoting anger, disgust, fear, joy and sadness (see Coan et al., 2001 for a detailed description of the methods used in this study). These emotions were then grouped according to the approach/withdrawal motivational model of emotion. By this scheme, frontal EEG asymmetries during anger and joy were arithmetically averaged into an approach condition and disgust, fear and sadness were arithmetically averaged into a withdrawal condition. An additional control condition was also employed.

Generalizability theory allowed for analyses of these data such that the following questions could be evaluated empirically: (1) were state changes in frontal EEG asymmetry resulting from the emotional manipulation task reliably elicited in all subjects? And (2) were trait predispositions in frontal EEG asymmetry preserved within emotional state conditions? Results indicated first that trait, state and the trait by state interaction accounted for approximately 8, 10 and 11% of the variance in frontal EEG asymmetry during state manipulations, respectively (see Table 5). These results suggest that variations in frontal EEG asymmetry attributable to traits, states and trait by state interactions are approximately equal—around 10%. In addition to this information, trait and state *g*-coefficients were 0.42 and 0.97, respectively. Thus, the answers to questions 1 and 2 above appear to be yes and somewhat, respectively. Indeed, it appears that state changes were strikingly robust to individual differences in this study. Put another way, to the extent that state changes in frontal EEG asymmetry occurred at all in response to the emotional manipulation, they occurred in nearly all of the subjects, regardless of their trait predispositions. By contrast, trait predispositions were only moderately preserved within state manipulations. Ultimately, these

Table 5
Results of a state manipulated frontal EEG asymmetry generalizability study

| | *N* | Variance component | % variance |
|---|---|---|---|
| Trait | 36 | 0.003 | 8.23 |
| State | 3 | 0.004 | 9.79 |
| Trait × state | | 0.005 | 11.42 |
| Residual | | 0.030 | 70.57 |
| | | Relative error variance | Generalizability coefficient |
| Trait | | 0.002 | 0.42 |
| State | | 0.0001 | 0.97 |

results raise the possibility that frontal EEG asymmetry may function more robustly as a mediator of emotional responses than as a moderator of emotional responses, although different results might be obtained if one assessed frontal EEG asymmetry across several occasions to provide a better and more stable estimate of trait asymmetry. Indeed, the Spearman–Brown Prophecy Formula suggests that the generalizability of trait frontal EEG asymmetry across emotional states would increase from 0.42 to 0.74 if averaged across 4 occasions of measurement. Such an approach may make it easier to assess the degree to which trait predispositions in frontal EEG asymmetry affect state changes in the same.

## 9. Concluding remarks

While there is a great deal of evidence suggesting that frontal EEG asymmetry may function as both a moderator and a mediator in various aspects of emotion, it is striking how few studies provide explicit evidence of either moderation or mediation. Indeed, in the case of mediation, explicit tests are entirely lacking, although extant data sets could conceivably be used to explicitly test for both mediation and moderation. In this paper, recommendations have been made for the rigorous evaluation of frontal EEG asymmetry as either a moderator or a mediator.

### *9.1. Frontal EEG asymmetry as a moderator of emotion*

Thus far, compelling evidence suggests a moderating role for frontal EEG asymmetry in at least some emotions, supporting Davidson's (1998a,b) theory of affective style. Nevertheless, generalizability analyses presented here suggest that while frontal EEG asymmetry may function as a moderator, its influence may only be modestly reliable across emotional states, and it is emotional states that trait frontal EEG asymmetries are supposed to be moderating. Others have obliquely pointed this out in various ways. For example, Davidson and colleagues (e.g., Davidson, 1998b; Henriques and Davidson, 1991; Tomarken et al., 1990; Wheeler et al., 1993) have long advocated for the use of multiple measures of resting frontal EEG asymmetry in its measurement as a robust trait. This group of researchers have taken different approaches to this, from averaging values across multiple sessions (thereby increasing the measure's reliability; e.g. Sutton and Davidson, 1997) to selecting

only individuals whose resting frontal EEG asymmetry remains stable over 2 or >2 weeks (e.g., Wheeler et al., 1993). Such practices have many psychometric virtues. Indeed, the Spearman–Brown prophecy formula would predict that with four occasions of measurement, the reliability of trait frontal EEG asymmetry, even across emotional states, should increase to a quite respectable range.

### *9.2. Frontal EEG asymmetry as a moderator of psychopathology*

To the extent that trait frontal EEG asymmetry moderates emotional responses, it may do the same for psychopathology. That is, as Davidson (1998a) has argued, an individual's particular affective style may create a predisposition to risk for psychopathology. If this is true, the empirical data to date remain mixed in their support this proposition. While several studies have suggested a relationship between frontal EEG asymmetry and psychopathology (Allen et al., 1993; Bruder et al., 1997; Henriques and Davidson, 1990, 1991; Gotlib et al., 1998; Debener et al., 2000; Davidson et al., 2000; Wiedemann et al., 1999), some have suggested otherwise (Reid et al., 1998), and none have attempted to rigorously model frontal EEG asymmetry as a moderator in the explicit fashion recommended by Baron and Kenny (1986). Importantly, however, establishing a moderating effect of frontal EEG asymmetry will require that data sets include measures of risk-related events or traits that frontal EEG asymmetry can moderate. So for example, if it is found that a particular cognitive vulnerability (for example, hopelessness; Abramson et al., 2002) is related to depression, then one could ask if that relationship is especially strong for those lowest in left frontal activity. Ideally, of course, one would obtain prospective data, such that hopeless cognitions and frontal EEG were assessed in a relatively high-risk but euthymic population, and the development of depression at a later timepoint would be assessed.

One might object to this last remark on the grounds that modeling moderator effects for psychopathology is not possible given the difficulty in implementing the kind of large, prospective studies that such an approach would require, but such declarations are probably premature. There may be ways to model frontal EEG asymmetry as a genuine moderator of psychopathology that do not require such costly commitments. For example, Allen et al. (1993) studied participants suffering intermittently from seasonal affective disorder (SAD)—a psychological difficulty whose course, treatment and eliciting environmental stimulus is relatively well understood, and moreover, relatively manipulable by investigators (via techniques such as phototherapy and/or waiting for the seasons to change). In studying psychopathology with an episodic course, of which SAD is a prime example, one could construct a GLM that would straightforwardly test the moderator model such as the following: SAD = season + trait frontal EEG asymmetry + season × trait frontal EEG asymmetry. One could also substitute a phototherapy manipulation for the Season variable in the case of SAD, or a psychotherapy treatment variable in the case of non-seasonal depression.

Other possibilities no doubt exist. For example, various negative life events have been associated with depression (e.g., Goodman, 2002). One could imagine that individuals who have just experienced the death of a close loved one may be at greater risk for depression than would those who have not. One could imagine further, indeed Davidson's diathesis/stress model predicts, that individuals with relatively greater right frontal EEG asymmetry would

be particularly likely to become depressed following the death of a close loved one. As a general linear model, one could state this as follows. Depression = loss status (yes/no) + trait frontal EEG asymmetry + loss × trait frontal EEG asymmetry. Similarly, adjustment disorders with depressed features frequently strike freshmen undergraduate students away from home for the first time. The approach/withdrawal diathesis/stress model would predict that individuals who show relatively greater right frontal EEG activity at rest are at greater risk for such adjustment disorders during their freshman year of college.

### *9.3. Frontal EEG asymmetry as a mediator*

While no study to date has explicitly modeled frontal EEG asymmetry as a mediator of emotional responses, several studies provide evidence that this is at least a distinct possibility, and the generalizability analyses reported here suggest that if frontal EEG asymmetry does indeed function as a mediator, its effects are quite robust to individual differences in trait frontal EEG asymmetry.

As for modeling frontal EEG asymmetry as a mediator, it will be important for individuals to record measures of emotional response other than frontal EEG asymmetry per se. That is, many studies of state changes in frontal EEG asymmetry regard frontal EEG asymmetry as a dependent variable only. In order to assess its function as a possible mediator, some other criterion of emotional response must be established. A good example of this, though also one in which frontal EEG asymmetry is not explicitly modeled as a mediator, was conducted by Davidson et al. (2000). These researchers identified state changes in frontal EEG asymmetry in social phobics as a function of anticipating giving a speech. In addition to demonstrating changes in frontal EEG asymmetry, other measures of physiological and self-reported distress were obtained, allowing at least the possibility of an explicit mediational model. Similarly, Harmon-Jones et al. (2003), in identifying state changes in frontal EEG asymmetry resulting from bogus anger inducing radio broadcasts, also measured coping behaviors, such as signing and taking petitions, again allowing for the possibility of an explicitly mediational model. Ultimately, if frontal EEG asymmetries prove to satisfy criteria as a mediator of emotion, then activity in the brain systems tapped by frontal EEG asymmetry may not simply be an output of emotion, but rather may be something that *facilitates* the emotional response.

### *9.4. Future directions*

As a sub-field of emotion and motivation, the study of frontal EEG asymmetry holds substantial promise. In terms of basic science, this sub-field promises to inform us regarding the fundamental properties of emotion, both in terms of how emotions occur and what properties they entail. In more applied settings, the possibility yet remains that frontal EEG asymmetry may serve as a useful liability marker for depression and anxiety. Regardless of the application of this measure, it is increasingly important that theoretical predictions surrounding frontal EEG asymmetry are put to more rigorous tests, especially those provided by testing explicit mediational and moderational models. In the absence of such explicit tests, the field will remain a collection of studies merely *suggestive* of moderating and mediating influences, around which much exciting and potentially important speculation

and theory is generated. It is possible to more explicitly test that speculation and theory with models that more adequately can support or refute such inferences.

The results of these explicit tests will guide the field in investigating whether and how frontal EEG asymmetry may serve as a risk marker for psychopathology, and in investigating what underlying physiological systems influence and are influenced by emotion. Gaining a deeper understanding of the fundamental properties of emotion will require clear thinking in terms of how the various components of emotion and emotional experience are related. It is in this spirit that the present remarks are offered.

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