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Individual Differences in Anterior Brain Asymmetry and Fundamental Dimensions of Emotion

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This research assessed whether individual differences in anterior brain asymmetry are linked to differences in basic dimensions of emotion. In each of 2 experimental sessions, separated by 3 weeks, resting electroencephalogram (EEG) activity was recorded from female adults during 8 60-s baselines. Mean alpha power asymmetry across both sessions was extracted in mid-frontal and anterior temporal sites. Across both regions, groups demonstrating stable and extreme relative left anterior activation reported increased generalized positive affect (PA) and decreased generalized negative affect (NA) compared with groups demonstrating stable and extreme relative right anterior activation. Additional correlational analyses revealed robust relations between anterior asymmetry and PA and NA, particularly among subjects who demonstrated stable patterns of EEG activation over time. Anterior asymmetry was unrelated to individual differences in generalized reactivity.

The present study demonstrates that individual differences in resting electroencephalographic (EEG) brain asymmetry are linked to individual differences in fundamental dimensions of emotion. This investigation is part of a larger program of research that assesses whether resting anterior EEG asymmetry is a biological index that measures important features of affective style. As we have noted previously (e.g., Davidson & Tomarken, 1989; Tomarken, Davidson, & Henriques, 1990), this general line of inquiry emerges from the synthesis of two distinct sets of findings on hemispheric asymmetry that have appeared in recent years.

The first set of findings provides evidence of an association between asymmetrical activation in anterior regions of the cerebral hemispheres and concurrent emotional state. Although this area of research is notable for its divergent perspectives (cf., e.g., Tucker & Williamson, 1984, to Davidson & Tomarken, 1989), there is now substantial evidence from studies using a variety of subject populations (e.g., neurological, psychiatric, and normal) and methods (e.g., lesion and electrophysiological studies) that increased activation of left hemisphere anterior regions is associated with heightened positive affect, decreased negative affect, or both. Conversely, increased relative right hemisphere activation in anterior sites has been linked to heightened negative affect, decreased positive affect, or both (e.g., Ahern & Schwartz, 1985; Davidson, Ekman, Saron, Senulis, & Friesen, 1990; Robinson, Kubos, Starr, Rao, & Price, 1984; for a review, see Davidson & Tomarken, 1989).

The second set of findings provides evidence of individual differences in hemispheric asymmetry that predict performance on cognitive tasks (e.g., Dabbs & Chou, 1980; Levy, Heller, Banich, & Burton, 1983). The results of EEG studies are particularly relevant in the present context. These have shown that resting asymmetry in posterior brain regions (e.g., parietal) predicts individual differences in verbal and nonverbal task performance (e.g., Davidson, Taylor, & Saron, 1979; Glass & Butler, 1977). As would be expected of a marker of individual differences, resting posterior EEG asymmetry also appears stable over time (e.g., Ehrlichman & Wiener, 1979).

Our program of research synthesizes these two sets of observations about hemispheric asymmetry. We examine whether individual differences in anterior EEG asymmetry assessed under resting conditions predicts differences in emotional behavior in the same manner that resting posterior EEG asymmetry predicts differences in verbal and nonverbal abilities. More specifically, consistent with more general theories concerning the biological basis of temperament (for a review, see Derryberry & Rothbart, 1984), we hypothesize that individual differences in anterior asymmetry index differing neural thresholds for affective responses to stimuli and differing mechanisms for the self-regulation of emotion over time (e.g., Davidson & Tomarken, 1989). To test this hypothesis, we have adopted the two interrelated approaches most typically used by researchers to examine whether a specific biological index measures an important affective predisposition or trait. The first approach assesses whether the index predicts emotional reactivity to specific stimuli or situations (e.g., exposure to novelty or threat and maternal separation). The second approach assesses whether the biological index maps onto individual differences in fundamental dimensions or typologies of emotion

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that have been uncovered by prior theory and research (e.g., Gray, 1982; Tellegen, 1985; for a review, see Derryberry & Rothbart, 1984).

Adopting the first approach described above, we have shown in three recent studies that individual differences in resting anterior EEG asymmetry can in fact predict reactivity to emotion elicitors. In two studies, increased relative right frontal EEG activation recorded during initial resting baselines was associated with self-reports of increased negative affect in response to films that subjects were subsequently shown (Tomarken et al., 1990; Wheeler, Davidson, & Tomarken, in press). Wheeler et al. additionally showed that relative left frontal activation is linked to increased positive affective responses to films. Finally, Davidson and Fox (1989) found that resting anterior asymmetry predicted 10-month-old infants' responses to maternal separation. Consistent with earlier findings on the EEG correlates of affective states, EEG asymmetry in posterior sites, recorded at the same time as anterior EEG activity, failed to predict affective reactivity in these studies.

In the present study, we examined the relation between individual differences in resting EEG asymmetry and individual differences in fundamental dimensions of emotion. In particular, we focused on the relation between anterior asymmetry and two sets of individual difference constructs: positive and negative affect (e.g., Watson & Tellegen, 1985) and valence-independent generalized reactivity (e.g., Larsen, Diener, & Emmons, 1986). Although these constructs reflect very different models of emotion, they were both included in the present study because (a) they have received perhaps the greatest attention and most substantial empirical support in contemporary research on the underlying dimensional structure of emotion and (b) we predicted that they would be differentially related to resting anterior asymmetry.

We expected that anterior asymmetry would be strongly linked to the positive and negative affect dimensions that are the core of the model of emotion that has been proposed by Watson, Tellegen, and their colleagues (e.g., Tellegen, 1985; Watson, 1988; Watson & Tellegen, 1985). In this scheme, two orthogonal dimensions, termed *Positive Affect* (PA) and *Negative Affect* (NA), underlie mood self-reports. There were two primary reasons for our decision to assess the relation between anterior asymmetry and this particular dimensional model. First, PA and NA have been found to underlie emotion self-reports across a wide variety of contexts (e.g., Watson, Clark, & Tellegen, 1984; Zevon & Tellegen, 1982) and time frames (e.g., Watson, 1988; for a review, see Watson & Tellegen, 1985). Second, recent conceptualizations of these two dimensions parallel our own previous speculations about the underlying dimensions assessed by measures of anterior brain asymmetry.

Concerning the latter point, Watson and Tellegen and their colleagues (Watson, Clark, & Tellegen, 1988; Watson & Tellegen, 1985) have proposed that Positive Affect is denoted by terms such as *active, interested,* and *enthusiastic* and reflects one's level of pleasurable engagement with the environment. In addition, Depue and Iacono (1989) and Tellegen (1985) have argued that the neurobehavioral substrate of positive affect is a reward-oriented "approach" system that is activated primarily, although not exclusively, by appetitive or other rewarding stimuli. We have previously argued that resting anterior asymmetry indexes a dimension, or dimensions, of approach and withdrawal (e.g., Davidson, 1984; Davidson & Tomarken, 1989; see also Kinsbourne, 1978). In this view, increased relative left frontal activation is associated with heightened approach tendencies that are manifested by greater engagement with tasks or with the environment and heightened appetitive motivation.

Watson, Tellegen, and their colleagues have argued that heightened NA reflects a state of unpleasurable engagement with the environment (e.g., Tellegen, 1985; Watson & Tellegen, 1985). We have hypothesized that heightened relative right frontal activation is associated with increased withdrawal tendencies and with those discrete emotions that are particularly linked to behavioral withdrawal, such as fear and disgust. The parallels between this hypothesis and the Watson–Tellegen conceptualization of NA are evident when one considers that (a) behavioral withdrawal is one of the most likely consequences of the state of unpleasant engagement with the environment that is argued to be characteristic of heightened negative affect (cf. Tellegen, 1985) and (b) specific emotions that are particularly linked to behavioral withdrawal (e.g., fear and shame) are some of the prime markers of Negative Affect in Watson and Tellegen's scheme.

In light of these parallels, we hypothesized that individual differences in resting asymmetry recorded from anterior sites would be linked to individual differences on the general version of the Positive and Negative Affect Schedule (PANAS-GEN), a trait measure of PA and NA that has recently been developed by Watson, Clark, and Tellegen (1988). We predicted that individuals characterized by a stable pattern of increased relative left anterior activation would report that they generally experience greater PA, but lesser NA, when compared with individuals characterized by a pattern of increased relative right anterior activation.

An interesting counterpoint to the Watson–Tellegen (1985) model specifying primary, orthogonal Positive and Negative Affect dimensions is the dimensional model of emotion that has been proposed by Diener, Larsen, and their associates in recent years (e.g., Diener, Larsen, Levine, & Emmons, 1985; Larsen et al., 1986). Focusing on affective reactivity, these authors have found evidence for individual differences in the intensity of affective responses to stimuli that are valence-independent. That is, in their studies, those individuals psychometrically identified as high-intensity responders respond more intensely to both positive and negative affective stimuli.

In the present study, we also examined the relation between anterior EEG activation and valence-independent reactivity. The Affect Intensity Measure (AIM), a scale developed by Larsen (1984; Larsen & Diener, 1987), was used to assess individual differences in generalized reactivity. We should note, however, that in previous studies the nature and direction of the relation between asymmetry and emotion has consistently been what might best be termed *valence-dependent.* Increased relative left frontal activation, or equivalently decreased relative right frontal activation, has been linked to increased PA or decreased NA but not to increases in both or to decreases in both (e.g., Davidson, Ekman et al., 1990; Tomarken et al., 1990). For this reason, we expected that individual differences in resting anterior EEG asymmetry would not predict scores on the AIM. In terms of construct validity, then, the AIM was included in the present study because it helped us assess discriminant as well as convergent relations between anterior asymmetry and fundamental dimensions of emotion (e.g., Campbell & Fiske, 1959).

To test our predictions, we assessed resting EEG asymmetry in mid-frontal (F3 and F4) and anterior temporal (T3 and T4) brain regions. Asymmetries in the mid-frontal region constitute the clear majority of previous EEG evidence indicating linkages between anterior activation and emotion (for a review, see Davidson & Tomarken, 1989). However, some recent evidence has implicated anterior temporal asymmetries in affective responding as well (e.g., Davidson, Ekman et al., 1990). For this reason, although our experimental hypotheses were applicable to both regions, our strongest predictions concerned the affective correlates of frontal asymmetry.

Method

Subjects

Subjects were 90 undergraduate women who were recruited from the introductory psychology pool at the University of Wisconsin—Madison. Subjects ranged in age from 17 to 21 years. As assessed by the Edinburgh Handedness Inventory (Oldfield, 1971), only right-handed subjects were used because of evidence that patterns of hemispheric activation for cognitive, and perhaps affective, functions may differ in left- and right-handed individuals (for a review, see Bryden, 1982). Only female subjects were used because the present investigation was one component of a larger research project, one goal of which was to assess EEG patterning during facial expressions of emotion. We and other researchers (Wagner, MacDonald, & Manstead, 1986) have previously found greater facial expressivity in response to affective stimuli in women relative to men. Because data from only a minority of subjects typically meet the inclusion criteria for analyses assessing the relation between facial expressivity and concurrent EEG (e.g., Davidson, Ekman et al., 1990), we included only women in the present study to maximize the statistical power of these latter analyses. Because of technical problems, the mid-frontal EEG of two subjects and the anterior temporal EEG of one subject were not usable.

Procedure

Overview. Subjects participated in two experimental sessions, approximately 3 weeks apart. In each session, EEG was recorded during a series of resting baselines, after which subjects either completed questionnaires (Session 1) or were exposed to affective film clips (Session 2). Two experimental sessions were held because (a) an additional goal of the broader research project was to assess the test-retest stability of various EEG asymmetry parameters over time (see Tomarken, Davidson, Wheeler, & Kinney, in press), and (b) primary analyses assessing the relation between anterior EEG asymmetry and affective traits were based largely on those subjects who fulfilled specific test-retest stability criteria.

Session 1. All subjects participated individually. When first scheduled for Session 1, subjects were informed that they would be asked to participate in two experimental sessions, separated by about 3 weeks. After arriving at the laboratory, subjects were told that the purpose of the present research was to examine specific parameters of their resting physiology. After electrodes were applied for the measurement of EEG, subjects were informed that all necessary instructions for the experimental procedure would be presented on a video monitor. After the experimenter left the room, the instructions informed subjects that (a) there would be eight 1-min resting baselines, (b) four baselines would be conducted with eyes open and four would be conducted with eyes closed, and (c) during the resting baselines, they should try to minimize eye blinks and movements, but should not be so concerned about doing so that they were distracted. In accordance with previous work in our laboratory (e.g., Tomarken et al., 1990), subjects were not given highly specific instructions concerning the resting baselines. They were told simply to try to be as "restful" as possible. After receiving these instructions, subjects completed a brief questionnaire assessing current mood state.

Two randomly assigned, counterbalanced orders were used for the eyes-open (O) and eyes-closed (C) trials of the resting baselines (O-C-C-O-C-O-O-C and C-O-O-C-O-C-C-O). Subjects heard one tone denoting the beginning of each 60-s baseline and two tones denoting the end of each baseline. There was a 3-min interval between the fourth and fifth baselines. A 45-s interval occurred between all other baselines. Following the eighth and final resting baseline, subjects completed a second questionnaire assessing current mood state, after which electrodes were removed. Then subjects completed the PANAS-GEN, the AIM, and several unrelated measures. Order of administration of questionnaires was randomly varied.

Because the Watson et al. (1988) article describing the PANAS-GEN was published after some subjects had already participated in the first two sessions, this questionnaire could be administered to only 44 subjects during Session 1. An additional 30 subjects were contacted and administered this questionnaire at a later point in time. Most of these additional administrations occurred during a third experimental session during which immunological measures were assessed (Kang, Davidson, Coe, Wheeler, Tomarken, & Ershler, 1991). These later administrations of the PANAS-GEN occurred between 6 and 15 months after Session 1. In the Design and Analysis section, we discuss how this difference in time of administration of the PANAS-GEN was incorporated into the analyses.

Session 2. The procedure for recording resting baselines was identical to that described in Session 1. Following the eight resting baselines, subjects were exposed to nine affective film clips preselected to elicit moderate to high levels of happiness (2 clips), fear (2 clips), and disgust (5 clips). Data from the film epochs are not reported in this article.

EEG Recording and Quantification

EEG was recorded using a lycra stretchable cap (manufactured by Electro-Cap International, Inc., Eaton, OH) that was positioned on the subject's head using known anatomical landmarks (Blom & Anneveldt, 1982). During Session 1, for the first several cohorts of subjects ($n = 46$), EEG was recorded from the left and right mid-frontal (F3 and F4) and anterior temporal (T3 and T4) regions. During recording, all four sites were referenced to vertex (Cz). Two additional channels were recorded to derive an averaged ears reference: Cz-A1 (left earlobe) and Cz-A2 (right earlobe). For the remainder of the subjects, EEG was recorded from the mid-frontal and anterior temporal sites and from 10 additional sites during Session 1: homologous lateral frontal (F7 and F8), central (C3 and C4), posterior temporal (T5 and T6), and parietal (P3 and P4) sites, and mid-line frontal (Fz) and parietal (Pz). In Session 2, for all subjects, recordings were made from the 16 sites noted above.[1]

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[1] Because EEG was recorded from only the mid-frontal and anterior temporal sites in Session 1 for a sizable proportion of subjects, we do not report the relation between EEG asymmetry in other sites and self-reported emotion. However, it should be noted that in previous studies we have failed to find relations between resting asymmetry in more posterior sites and emotion indices (e.g., Davidson & Fox, 1989; Tomarken, Davidson, & Henriques 1990). Similarly, supplementary analyses including only the Session 2 EEG indicated that resting asymmetry in more posterior sites failed to predict PANAS-GEN or AIM scores in the present study.

All electrode impedances were below 5K ohms, and the impedances for homologous sites were within 500 ohms of each other. In both sessions, electrooculograms (EOGs) recording eye movements were additionally used to facilitate artifact scoring of the EEG.

In both sessions, the EEG was amplified with a Grass Model 12 Neurodata System using Model 12A5 preamplifiers (bandpass = 1 and 100 Hz; 60 Hz notch filter in). The amplified signal was passed through antialiasing low-pass filters (Rockland Model 424) set at 65 Hz (roll-off = 24 dB/octave). The EEG was digitized at 200 Hz by a PDP 11/34A minicomputer. The EEG activity for eight channels and the EOG were displayed on a Grass Model 7 polygraph. This paper record was hand-scored to identify portions of the data to be deleted because of eye movements, muscle movements, and other sources of artifact. When artifact occurred on a given channel, data from all channels were removed. Artifact scoring was performed by two of the authors (R. Wheeler and R. Doss). Interrater reliability of artifact scoring computed on 10 subjects' data was assessed as the percentage of seconds of agreement over the total duration of EEG per session (480 s). The percentage agreement was .957, indicating high interrater reliability.

Twenty-five and 50 μv 10 Hz sine waves were recorded on each channel and used to calibrate the digitized EEG. In addition to the original recording montage referenced to vertex, the EEG was recomputed off-line to derive a computer-averaged ears reference. This reference is conceptually equivalent to the more traditional linked ears reference but avoids the potential problem of attenuation of the magnitude of asymmetry due to physical linking of the ears (see Henriques & Davidson, 1990, for a more extended description of the rationale and computational formulae for the computer-averaged ears reference).

Spectral-analytic procedures described by Davidson (1988) and Davidson and Tomarken (1989) were used to derive estimates of the spectral power of the digitized EEG. Briefly, all artifact-free chunks that were 2.05 s in duration were extracted through a Hamming window. A Fast Fourier Transform (FFT) was then used to derive estimates of spectral power (in μv^2) in different 1-Hz frequency bins. These power values were then averaged across each of the artifact-free chunks of a given resting baseline trial. Finally, power values were converted to power density (in μv^2/Hz), which is a measure of the average power within a given frequency band or range.

In the present article, we focused on analyses conducted on measures of power density in the alpha frequency band 8 to 13 Hz. We did so because in previous studies power in this band has been most consistently linked to EEG asymmetries associated with concurrent emotional state (e.g., Davidson, Ekman et al., 1990), with individual differences in affective predispositions (Tomarken et al., 1990), and with verbal and nonverbal performance on cognitive tasks (e.g., Davidson, Chapman, Chapman, & Henriques, 1990). This evidence indicates that decreased alpha power in a given region is associated with increased cortical activation in that region (see Davidson, Chapman et al., 1990, for a more extended discussion). Consistent with our previous evidence that the alpha frequency band typically is associated with the most robust effects, supplementary analyses conducted on other frequency bands (e.g., theta or beta) failed to reveal any effects on the PANAS-GEN scales or on the AIM. Because of space constraints and our a priori predictions concerning alpha power, these latter analyses are not reported in this article.

In the present article, as in other articles that are based on the larger research project (e.g., Wheeler et al., in press), we focus on the analyses conducted using the averaged ears reference. There were two reasons for our decision to do so. First, measures of EEG asymmetry that were derived using the averaged ears reference proved to have higher test–retest stability than that provided by alternative reference montages (e.g., vertex) in both a previous pilot study conducted with an independent sample (Tomarken, Davidson, Baskin, & Angier, 1987) and in the present study (for a complete report, see Tomarken et al., in press). Second, in previous research conducted in our laboratory that examined relations between individual differences in resting posterior EEG asymmetry and verbal and nonverbal task performance, a linked ears reference yielded more robust effects than vertex (e.g., Davidson et al., 1979). This latter effect may possibly be due to the greater stability of ears-referenced, relative to vertex-referenced, EEG.[2]

Several sequential steps were used to compute an overall asymmetry metric for the artifact-free data from the mid-frontal and anterior temporal regions. First, to normalize the data, we log-transformed all power density values. Second, for each experimental session, we computed a weighted average of the power density in each site across each of the eight resting baselines. We weighted by the number of artifact-free 2-s chunks of EEG data available during each baseline trial. Thus, trials with more data than others were given proportionally more weight. In addition, the weighted means for each of the two sessions were averaged to produce a grand average mean.[3]

At the end of this process, then, for each of the four sites of interest (F3, F4, T3, and T4), there were three indices of log power density corresponding to the mean for Session 1, the mean for Session 2, and the grand mean pooled across Sessions 1 and 2. In the final stage, measures of EEG asymmetry were derived. Asymmetry was computed as the difference between log alpha power density in the right (R) hemisphere lead and log alpha power density in the left (L) hemisphere lead (i.e., log R − log L alpha power). Because alpha power is inversely related to activation, higher scores on this index indicate greater relative left hemisphere activation.

Self-Report Emotion Measures

PANAS-GEN. Developed by Watson et al. (1988), the PANAS-GEN contains 20 emotion descriptors, 10 of which assess the high activation pole of Negative Affect (NA; e.g., afraid or distressed) and 10 of which assess the high activation pole of Positive Affect (PA; e.g., enthusiastic or inspired). All descriptors are relatively pure markers of one of these factors (i.e., with a substantial loading on one factor and a near-zero loading on the other). When completing the GEN version of the PANAS, respondents indicate how they feel "in general, that is on the average." The internal consistency reliability of the PA and NA scales of the GEN version is acceptably high (coefficient αs = .88 and .87, respectively). In addition, test–retest reliabilities (.68 for PA and .71 for NA over an 8-week period) indicate that the PANAS–GEN can be used as a trait measure of affect. The two scales are not significantly correlated (e.g., reported *r* by Watson et al., = −.17). Watson et al. have presented evidence indicating that the PA and NA scales validly assess the Positive and Negative Affect constructs.

AIM. The AIM (Larsen & Diener, 1987) is a 40-item questionnaire that assesses the characteristic magnitude or intensity with which an individual experiences emotion (e.g., a sample item is, "The sight of someone who is hurt badly affects me strongly"). The AIM has excellent internal consistency reliability (coefficient αs = .90–.94) and adequate test–retest stability for a measure of individual differences (e.g.,

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[2] Davidson and Fox (1989) and Tomarken, Davidson, and Henriques (1990) reported the results of analyses conducted using a vertex reference simply because an averaged or linked ears reference was not available.

[3] In addition, before computing weighted averages, we imposed a minimal criterion for the inclusion of a given subject's data. According to this criterion, if a subject had less than 10 artifact-free chunks of EEG across the four eyes-open or four eyes-closed baselines of a given session, her data were to be eliminated from the design. Application of these minimal-data criteria resulted in the exclusion of no subjects. Subjects had an average of 43 s of artifact-free data per 60-s baseline.

test–retest *r*s of .81 and .75 over 3 months and 2 years, respectively). In addition, Larsen and his colleagues have shown that scores on the AIM are not contaminated by response bias, and they have presented evidence from several sources documenting the validity of the AIM as a measure of individual differences in affect intensity (for a review, see Larsen & Diener, 1987).

Design and Analysis

Several considerations influenced our choice of a design structure for the analyses. The first of these pertained to the focus on individual differences in EEG asymmetry. Almost by definition, individual difference variables should be stable over time, either in the population as a whole or, at the least, in specific subgroups. An acceptable level of test–rest stability was especially necessary in the present context because our hypotheses concerned linkages between resting asymmetry and measures of individual differences in affective traits that are generally stable over time.

In the present sample as a whole, using the averaged ears reference and pooling over eyes-open and eyes-closed baselines, the test–retest stability of resting anterior asymmetry over the course of 3 weeks was .66 for the mid-frontal site and .73 for the anterior temporal site (both $ps < .0001$). In addition, paired observation t tests indicated no shift in mean values of either frontal or anterior temporal asymmetry over time (both $ts < 1$, $ps > .50$; for a more extensive description of the psychometric properties of resting anterior EEG asymmetry, see Tomarken et al., in press). The test–retest correlations indicate that resting anterior EEG asymmetry certainly has acceptable temporal stability (e.g., Kraemer, 1981). However, given the fairly short time interval used in the present study (3 weeks), these stability correlations, even though deemed acceptable, might not be considered optimal according to the standards that could be applied for a personality measure assessing an affective trait.

Although this issue is by no means definitively resolved at the present time, we believe that EEG asymmetry may well be most analogous to certain measures of emotion or psychopathology that are known to reflect both variations in concurrent state and more stable individual differences (for a more extensive discussion of this point, see Tomarken et al., in press). For instance, it is well-known that the Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) is sensitive to both long-term depressive affect and more transient mood states (Sacco, 1981). As a result of these dual influences, in current research that focuses on more enduring individual differences in depression and that uses the BDI as a basis for subject selection, it has become common practice to (a) administer the BDI on two occasions and (b) select into depressed and nondepressed groups only those subjects who score above or below specific cutpoints (or percentile values) on both occasions (e.g., Blumberg & Hokanson, 1983; Davidson, Schaffer, & Saron, 1985).

We adopted as one of our two primary analytic approaches a method similar to that used in studies of BDI-identified depressives. Using an approach of this sort, we assigned to *relative left mid-frontal* ($n = 12$) and *relative left anterior temporal* ($n = 14$) groups those subjects who scored in the top 25th percentile (i.e., in the direction of greater relative left hemisphere activation) of the distribution of mid-frontal and anterior temporal asymmetry scores in both experimental sessions. Conversely, those subjects who scored in the bottom 25th percentile on both occasions (i.e., in the direction of greater relative right hemisphere activation) were assigned to the *relative right mid-frontal* ($n = 13$) and *relative right anterior temporal* ($n = 14$) groups. Supplementary analyses revealed that the results and conclusions do not differ appreciably if alternative cutpoints are chosen. The mid-frontal and anterior temporal groups were largely independent, with only 8 subjects (3 left anterior and 5 right anterior) who overlap both sets of groups. This low degree of overlap is reflected in the low correlation between mid-frontal and anterior temporal asymmetry overall ($r = .21$).

Planned comparisons assessed the differences between the two sets of left and right anterior groups on the PANAS-GEN and the AIM. To aid in the subsequent interpretation of differences involving these groups, *middle asymmetry* comparison groups were included in omnibus analyses of variance (ANOVAs) conducted separately from the planned analyses. The *middle mid-frontal* ($n = 23$) and *middle anterior temporal* ($n = 29$) groups consisted of those subjects who scored between the 25th and 75th percentile of asymmetry scores in both Session 1 and Session 2.

In contrast to those using an extreme-groups approach, many individual differences researchers treat variables of interest as continua. Because of the general popularity of this approach and because it is presently unclear whether resting anterior EEG asymmetry should optimally be treated as a continuum or a discrete variable, we also conducted correlational analyses. In these analyses, subjects' grand mean anterior asymmetry, pooled over Sessions 1 and 2, was correlated with their PANAS–GEN and AIM scores. We present these correlations for the sample as a whole and for two subgroups of subjects: those who demonstrated stable patterns of asymmetry over time and those who did not. To classify subjects on stability for the correlational analyses, we standardized both the Session 1 and Session 2 asymmetry values and classified as *stable* those subjects whose Session 2 standardized asymmetry values were within one third of a standard deviation of their Session 1 standardized values. The criterion we used was the same as that used in related research from our laboratory (e.g., Wheeler et al., 1991).

Finally, as noted earlier, some subjects completed the PANAS–GEN at a third session between 6 and 15 months after the two experimental sessions during which the EEG was recorded. Preliminary analyses indicated no significant differences in PANAS–GEN scores between subjects who completed it in Session 1 and those who completed it a later point in time ($ps > .10$). Concerning the between-groups analytic strategy, preliminary analyses including time of administration as a factor in the design in addition to asymmetry group failed to reveal any significant main effects or interactions involving time of administration. Indeed, no trends were even evident (all $ps > .35$). For this reason, and because of the relatively small sample sizes involved (see above), we pooled over time of administration when conducting the between-groups analyses. In contrast, initial inspection of the correlations between anterior asymmetry and PANAS–GEN scores revealed that time of administration was a potentially significant factor in certain cases. For this reason, and because of the larger sample sizes involved, in addition to computing correlations for all subjects pooled together, we computed separate correlations for groups who completed the PANAS–GEN in Session 1 and those that completed it in the extra third session.

Results

Between-Groups Analyses

PANAS–GEN scales. We hypothesized that individuals characterized by relative left anterior activation would report that they generally experience more positive affect and less negative affect than individuals characterized by relative right anterior activation. For both the mid-frontal and anterior temporal groups, we directly tested this prediction by conducting a planned Group (left anterior vs. right anterior) × Valence (PANAS PA score vs. PANAS NA score) interaction contrast. This contrast assessed whether left and right anterior groups differed in the relative balance of positive and negative affect.

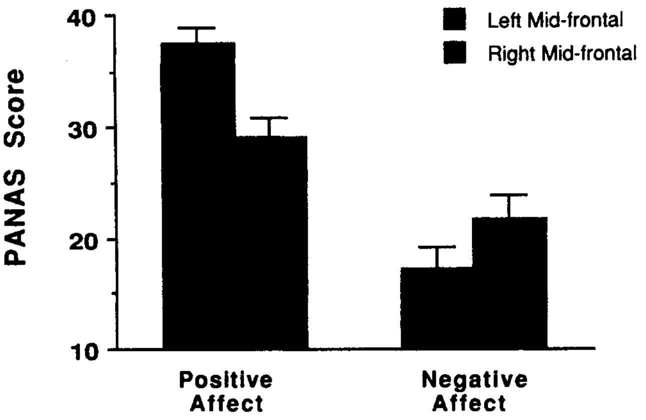

Figure 1. Mean Positive and Negative Affect Schedule (PANAS) Positive Affect and Negative Affect scores for left mid-frontal and right mid-frontal groups. (Error bars denote one half of the standard error of the mean.)

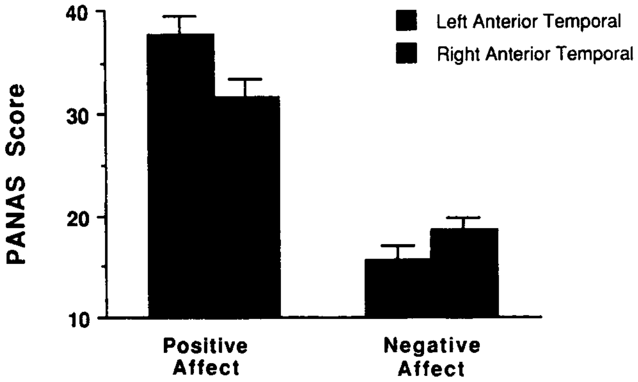

Figure 2. Mean Positive and Negative Affect Schedule (PANAS) Positive Affect and Negative Affect scores for left anterior temporal and right anterior temporal groups. (Error bars denote one half of the standard error of the mean.)

As indicated in Figure 1, the planned contrast performed on the left and right mid-frontal groups indicated a significant interaction, $t(19) = 2.99$, $p < .01$. The form of this interaction was as predicted, with the left mid-frontal group displaying increased PA and decreased NA, relative to the right mid-frontal group (see Figure 1). Follow-up simple effects comparisons indicated highly significant differences between the left and right mid-frontal groups on PA, $t(19) = 3.56$, $p = .002$ ($R^2 = .40$). However, these groups failed to differ on NA, $t(19) = 1.57$, $p > .10$.

The results of an omnibus 3 (left anterior vs. right anterior vs. middle asymmetry) × 2 (PA vs. NA) ANOVA including the middle asymmetry group were consistent with the results of the planned analyses. It yielded significant effects for valence, $F(1, 36) = 100.22$, $p < .0001$, and, most important, a highly significant Group × Valence interaction, $F(2, 36) = 6.15$, $p = .005$. As indicated by the results of subsequent simple effects analyses and post hoc Tukey honestly significant difference (HSD) tests (Spjotvoll–Stoline modification for unequal *n*s, see Kirk, 1982) comparing the middle mid-frontal group to the two extreme asymmetry groups, the middle group had higher PA scores than the right mid-frontal group, simple effects $F(2, 36) = 6.37$, $p < .005$ ($R^2 = .23$, posthoc $p < .01$). However, middle asymmetry subjects failed to differ from the left mid-frontal group ($p > .05$). There were no significant differences among the NA scores (all *p*s > .05).

As indicated by Figure 2, despite the fact that the mid-frontal and anterior temporal groups were largely independent, the patterning of PANAS–GEN scores among subjects in the anterior temporal groups paralleled that among the mid-frontal groups. The results of analyses were consistent with this observation. Specifically, the planned Group (left anterior temporal vs. right anterior temporal) × Valence (PA vs. NA) contrast indicated the predicted significant interaction, $t(23) = 2.20$, $p < .05$. Subsequent simple effects comparisons indicated that the left anterior temporal group reported higher levels of positive affect than the right anterior temporal group, $t(23) = 2.42$, $p < .025$. There were no significant differences between the two groups in NA, $t(23) = 1.14$, $p > .20$.

As well as a significant effect for valence, $F(1, 46) = 160.56$, $p < .0001$, the omnibus 3 × 2 ANOVA including the middle anterior temporal group yielded a significant Groups × Valence interaction, $F(2, 46) = 3.41$, $p < .05$. Although simple effects analyses indicated a significant overall effect on the PA scale, $F(2, 46) = 3.57$, $p < .05$, post hoc comparisons indicated no significant differences between the middle anterior temporal group and the other two groups on this measure (*p*s > .05). Consistent with the results of the planned analyses, follow-up analyses of the NA scale failed to yield significant differences.

AIM. We predicted that asymmetry groups would not differ on the AIM. To test this prediction, for both the mid-frontal and anterior temporal regions, we conducted planned comparisons between the right and left asymmetry groups. We also computed a one-way ANOVA including middle asymmetry groups in the design. In accordance with predictions, neither the planned nor the omnibus analyses of the mid-frontal and anterior temporal sites yielded any significant effects, or even trends, on this measure (all *p*s > .10; see Table 1).

Table 1
AIM Scores for Mid-Frontal and Anterior Temporal Asymmetry Groups

	Asymmetry group		
Site	Left	Middle	Right
Mid-frontal			
M	4.11	4.04	3.90
SD	.42	.54	.37
n	12	23	13
Anterior temporal			
M	3.81	3.91	3.77
SD	.42	.46	.34
n	14	28	13

Note. AIM = Affect Intensity Measure. Potential range of AIM is from 1 to 6.

Correlational Analyses

PANAS–GEN scales. As noted above, for comparative purposes, we also computed correlations between anterior asymmetry and affective traits. More specifically, we computed correlations between subjects' mean asymmetry, pooled over Sessions 1 and 2, and their scores on PA, NA, the difference between scores on the PA and NA scales, and the AIM. The PA–NA difference measure was added because (a) it is algebraically equivalent to the dependent variable used in the planned interaction contrasts to test experimental hypotheses and (b) the difference between PA and NA is itself a meaningful construct in research on the dimensional structure of emotion. This difference is equivalent to the pleasure–displeasure dimension that Russell (1980, 1983) has argued is basic to emotion (e.g., Tomarken et al., 1990).

Table 2 shows the correlations between mean mid-frontal asymmetry, the PANAS–GEN measures, and the AIM. Shown are the overall correlations for the sample as a whole and for two different subclassifications of subjects that were based on time of administration of the PANAS–GEN (Session 1 vs. the extra Session 3) and the stability of EEG asymmetry across Sessions 1 and 2 (stable vs. unstable). When viewing Table 2, the reader should bear in mind that higher scores on the asymmetry index indicate greater relative left frontal activation.

Several features of the results displayed in this table are notable. First, all correlations involving the PANAS–GEN were in the predicted direction; that is, greater relative left frontal activation was consistently associated with increased PA and decreased NA. Second, although none of the correlations for the entire sample were significant, among those subjects who completed the PANAS–GEN at the same time that their EEG was assessed (i.e., during Session 1), the correlation between frontal asymmetry and the PA–NA difference score was significant. In addition, there was a strong correlation in the predicted direction between asymmetry and NA scores among subjects whose EEG was stable over time (*r* = −.47), but not among subjects whose EEG was unstable (*r* = −.02). No correlations involving the PANAS PA scale attained significance.

Table 3 shows the correlations involving resting anterior temporal asymmetry. Similar to Table 2, this table presents the correlations for the sample as whole and for time of administration and EEG stability groups. In addition, the correlations for each of the four Time of Administration × EEG Stability subgroups are included. Because of the overly small sample sizes in certain groups (e.g., *n* = 7), these more fine-grained correlations were not computed for the mid-frontal leads.

As evidenced by Table 3, anterior temporal asymmetry was moderately, although significantly, associated with both PA and the PA–NA difference score overall. Moreover, correlations involving these two affect measures were even more robust among subjects whose asymmetry was stable over time (e.g., stable group *r*s = .49 and .42, respectively). In this regard, the correlations within the Time of Administration × Stability subgroups were revealing, especially the strong correlation between asymmetry and PA among stable Session 3 subjects (*r* = .57). This finding is important, because it indicates that, among subjects whose anterior temporal asymmetry was stable over time, mean EEG asymmetry was able to predict subsequent PA scores obtained between 6 and 15 months after EEG recordings were made. Although correlations involving NA were less affected by EEG stability, this scale and the PA–NA difference score were significantly correlated with anterior temporal asymmetry among all subjects who were administered the PANAS–GEN in Session 1 (see Table 3).

As noted above, examination of Tables 2 and 3 indicated that, in several cases, the magnitude of the correlations between EEG asymmetry and PANAS–GEN measures was conditional on EEG stability or time of administration. To more explicitly test whether these two factors moderated the relation between degree of asymmetry and self-reported emotion, parallelism tests were conducted comparing the slope of the regressions of PANAS–GEN scales on EEG asymmetry for groups differing in time of administration or stability (for the rationale for comparing beta weights rather than correlations, see, e.g., Baron & Kenny, 1986). These parallelism tests are identical to tests of the statistical significance of Asymmetry × Stability or

Table 2
Correlations Between Mid-Frontal Asymmetry and Emotion Scales

	Emotion scale			
Classification	PANAS–GEN PA	PANAS–GEN NA	PA − NA difference	AIM
Overall (*N* = 72)	.20	−.14	.22	.19[a]
Time of PANAS–GEN administration				
Session 1 (*n* = 43)	.25	−.22	.31*	
Session 3 (*n* = 29)	.16	−.06	.14	
EEG stability				
Stable (*n* = 21)	.12	−.47*	.38	−.21[b]
Unstable (*n* = 51)	.23	−.02	.16	.31**

Note. PANAS–GEN = Positive and Negative Affect Schedule (General version); PA = Positive Affect; NA = Negative Affect; AIM = Affect Intensity Measure.

[a] Eighty-four subjects completed the AIM during Session 1. [b] Twenty-two AIM respondents were in the EEG stable group and 62 were in the EEG unstable group.

* $p < .05$. ** $p < .025$.

Table 3
Correlations Between Anterior Temporal Asymmetry and Emotion Scales

	Emotion scale			
Classification	PANAS–GEN PA	PANAS–GEN NA	PA – NA difference	AIM
Overall (N = 73)	.25*	−.19	.28**	−.03[a]
Time of PANAS–GEN administration				
Session 1 (n = 43)	.26	−.41***	.44****	
Session 3 (n = 30)	.22	.18	.02	
EEG stability				
Stable (n = 36)	.49****	−.29	.42**	−.11[b]
Unstable (n = 37)	.14	−.15	.19	.04
Time of PANAS–GEN administration × EEG Stability				
Session 1				
Stable (n = 21)	.52**	−.35	.51**	
Unstable (n = 22)	.16	−.45*	.38	
Session 3				
Stable (n = 15)	.57*	.00	.33	
Unstable (n = 15)	−.15	.27	−.27	

Note. PANAS–GEN = Positive and Negative Affect Schedule (General version); PA = Positive Affect; NA = Negative Affect; AIM = Affect Intensity Measure.
[a] Eighty-four subjects completed the AIM during Session 1. [b] Thirty-seven AIM respondents were in the EEG stable group and 47 were in the EEG unstable group.
* $p < .05$. ** $p < .025$. *** $p < .01$. **** $p < .005$.

Asymmetry × Time of Administration interaction terms included in a hierarchical multiple regression analysis (Marascuilo & Levin, 1983). Such interaction tests have been recommended as the most appropriate procedure for testing whether a given factor serves as a moderator variable in the strict sense of the term (e.g., Paunonen & Jackson, 1985). The only significant effect yielded by the parallelism tests indicated that the relation between anterior temporal asymmetry and the PANAS NA score was moderated by time of administration ($p < .025$). That no other effects were significant necessitates some caution in the interpretation of the differences among subgroups shown in Tables 2 and 3.

AIM. In general, as predicted, the correlations involving the AIM shown in Tables 2 and 3 were nonsignificant. The sole exception was the significant positive correlation between mid-frontal asymmetry and AIM scores among unstable subjects (see Table 2). Oddly, there was a negative, albeit nonsignificant, correlation among stable subjects. Parallelism tests indicated a significant difference in the beta weights associated with these two correlations ($p < .05$). As hypothesized, and consistent with the results of the between-groups analyses, all correlations between anterior temporal asymmetry and AIM scores were nonsignificant and near zero.[4]

Discussion

We hypothesized that relative left hemisphere activation in mid-frontal and anterior temporal sites would be associated with increased PA and decreased NA on the PANAS–GEN. Conversely, we predicted that resting anterior asymmetry would be independent of subjects' scores on the AIM, which assesses generalized reactivity. On the whole, the results of analyses were supportive of these hypotheses, with the qualifications noted below. Between-groups analyses revealed that subjects characterized by relative left hemisphere activation in both the mid-frontal and anterior temporal regions reported increased dispositional PA and decreased dispositional NA, when compared with subjects characterized by relative right hemisphere activation. Follow-up analyses indicated that these differences were significant for PA, but not for NA. The strong parallels between the mid-frontal and anterior temporal results are especially notable given that the two classifications were only weakly correlated in the present context.

Correlational analyses including the entire sample of subjects revealed associations between anterior asymmetry and the PANAS–GEN scales that were consistently in the predicted direction, although generally only moderate in size. When correlations were computed including only those subjects who were administered the PANAS–GEN in Session 1, three of the six correlations between anterior asymmetry measures and the PA, NA, and PA–NA difference score were statistically significant. This observation is important given that subjects excluded from these analyses were administered the PANAS–GEN between 6 and 15 months after EEG recordings. The test–retest correlations reported above (see Method) for both anterior asymmetry and the PANAS–GEN scales suggest that, whereas both sets of measures have adequate stability over time, both are also susceptible to some degree of interindividual change

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[4] Because it might be predicted that increased generalized reactivity would be associated with less stable EEG patterning over time, we conducted t tests comparing stable and unstable EEG groups on AIM scores. For both the mid-frontal and anterior temporal sites, these comparisons were nonsignificant (both $ps > .10$). In addition, total EEG power summed over homologous leads (e.g., F3 and F4) failed to predict AIM or PANAS–GEN scores.

over the course of 6 to 15 months. It is likely that this factor served to attenuate the correlation between anterior asymmetry and PA and NA measures on the combined analyses including both Session 1 and Session 3 subjects. Relatedly, correlations including those subjects whose anterior asymmetry met specific stability criteria across the two experimental sessions offered stronger support for hypotheses than the combined analyses. Particularly notable was the robust correlation between anterior temporal asymmetry and PA among subjects who completed this scale well after Sessions 1 and 2. We address the issue of individual differences in the stability of resting anterior asymmetry in greater detail below.

Analyses of the AIM were also generally consistent with predictions. The ANOVAs indicated that both mid-frontal and anterior temporal asymmetry failed to significantly predict AIM scores. Similarly, correlational analyses revealed that anterior temporal asymmetry generally failed to significantly predict generalized reactivity.

Below, we comment on the implications of our results for conceptualization of the relation between anterior asymmetry and fundamental emotion dimensions, after which we discuss some of the conceptual and methodological issues raised by the present study.

Anterior EEG Asymmetry and Positive and Negative Affect

Previous results from our laboratory demonstrated that individual differences in anterior EEG asymmetry can predict subsequent affective reactivity to stimuli (Davidson & Fox, 1989; Tomarken et al., 1990). The present findings extend these results by showing that individual differences in both mid-frontal and anterior temporal asymmetry are linked to corresponding differences on fundamental dimensions of emotion.

In light of previous findings concerning the affective correlates of anterior asymmetry, the significant effects on the PANAS PA factor yielded by both the between-groups and correlational analyses are especially significant. In some of our previous studies, we have found that anterior asymmetry is more strongly linked to negative affect than to positive affect. For instance, Davidson, Ekman et al. (1990) found that although facial expressions of disgust were associated with significant increases in relative right frontal activation, frontal asymmetry during facial expressions of felt happiness failed to be associated with significant increases in left frontal activation relative to baseline conditions. Similarly, although Tomarken et al. (1990) found that resting frontal asymmetry predicted negative affective responses to film clips, it failed to predict positive affective responses (but see Wheeler et al., in press).

We have speculated previously that the reason for these weaker effects on positive affect measures is that a variety of specific and distinct affective–motivational states may fall under the broad rubric of *positive affect* (e.g., Davidson, Ekman et al., 1990). However, only some of these states may be characterized by strong approach motivation, which is hypothesized to be the critical component linked to relative left anterior activation. In this view, *approach* is denoted by such features as heightened incentive motivation and task engagement. As noted above, Watson, Tellegen, and their colleagues (Tellegen, 1985; Watson & Tellegen, 1985) have argued that pure markers of PA denote strong engagement with tasks or with the environment. In addition, they and others (e.g., Depue & Iacono, 1989) have argued that PA is rooted in a neurobehavioral approach system characterized by heightened appetitive and incentive motivation. That the results of the present study reveal a linkage between PA and anterior asymmetry supports the hypothesis that the latter reflects the relative strength of approach motivation.

Additional Conceptual and Methodological Issues

Four key conceptual and methodological issues are raised by the present study that should be addressed in future research. First, as noted above, it is necessary to examine precisely what affective dimensions particular patterns of brain activation best map onto. For example, as noted above, Russell (1980, 1983) has proposed a model of emotion that is an alternative to that of Watson and Tellegen (1985). In this view, two primary dimensions, denoted as pleasure–displeasure and arousal, underlie mood self-reports. Of particular interest is the pleasure–displeasure dimension that represents the difference between, or relative balance of, positive and negative affect (e.g., Tomarken et al., 1990; Watson, 1988). In this regard, it should be noted that (a) the significant interactions yielded by the planned contrasts directly tested for effects on the difference between PA and NA and (b) there were several significant correlations between anterior asymmetry and the PA–NA difference score, both in the sample as whole and in specific subgroups. Unfortunately, in the present study, unique emotion descriptors that assessed the pleasure–displeasure dimension were not used. More generally, the present study leaves unanswered several additional questions concerning the relation between anterior asymmetry and the dimensional structure of emotion. For instance, the PANAS-GEN assesses only the high activation poles of PA and NA. The PA scale, for example, contains high PA descriptors (e.g., *enthusiastic*) but no low PA descriptors, (e.g., *sluggish*). There is evidence that the high and low activation poles of PA and NA may constitute separate unipolar dimensions under certain conditions (e.g., Burke, Brief, George, Roberson, & Webster, 1989).

In addition to clarifying the relation between anterior asymmetry and different dimensional models of emotion, research is needed to assess the precise mechanisms that account for the relation between anterior asymmetry and individual differences in self-reported affective traits. Although space constraints preclude a more complete discussion of this issue, one possibility is that the linkage between anterior asymmetry and dispositional PA and NA is due to altered neural thresholds for positive and negative reactions to naturalistic emotion elicitors. This possibility is consistent with our prior evidence that individual differences in anterior asymmetry are linked to differences in affective reactivity to emotional stimuli (e.g., Tomarken et al., 1990). In addition, this hypothesis is consistent with evidence that individual differences in personality (e.g., extraversion) are related to differences in reactivity to affective stimuli that, in turn, may contribute to long-term differences in tonic levels of positive or negative affect (e.g., Larsen & Ketelaar, 1991; Tellegen, 1985).

A second conceptual issue raised by the present study concerns the measurement of EEG activation. In the present study, consistent with our previous work and the related work of other investigators, we assessed EEG asymmetry, that is, the difference in activation between homologous right- and left-hemisphere sites. One potential limitation of reliance on a measure of asymmetry is that the same asymmetry score could conceivably reflect several different patterns of brain activity. For example, among those with relative left anterior activation, there could be several subtypes of subjects: those characterized by left anterior hyperactivation, right anterior hypoactivation, or some combination of the two. It would be optimal to arrive at separate indices of site-specific activation rather than to rely on a measure of asymmetry that reflects the difference in activation between two sites. Such indices would be especially optimal in the present context given that we assessed the relation between one variable, anterior EEG asymmetry, and two dimensions, PA and NA, that have previously been shown to be uncorrelated. Perhaps, then, a more fine-grained analysis including site-specific measures would show a more differentiated pattern of results, with EEG activation in certain anterior sites linked predominantly to PA and with activity in other sites linked predominantly to NA (e.g., Davidson & Tomarken, 1989).

Unfortunately, the methodological issues concerning the measurement of individual differences in site-specific resting EEG activation are more complex than they might initially appear. For example, raw power, or power density, in a given site cannot be used as a measure of activation because a major determinant of power values is thickness of the skull (Davidson, 1988). We are currently exploring several methods for assessing site-specific activation and the patterning of activation across multiple sites. All of these require recording of the EEG from multiple scalp regions. Unfortunately, because the EEG was recorded from only five active sites (F3, F4, T3, T4, and Cz) for approximately half the subjects in Session 1, such methods could not be used in the present study.

A third issue raised by the present study is whether it is optimal to treat anterior asymmetry as an underlying continuum and to use predominantly correlational methods of analysis or to focus on extreme groups and to use between-groups analytic methods. Primarily because there is ample precedent for both approaches in previous work on individual differences in emotion, the present study adopted a dual analytic strategy. In general, the results of the two methods of analysis agreed, particularly for the anterior temporal region. In this region, both the between-groups and the stable-subgroup correlational analyses indicated a significant relation between asymmetry and PA and the difference between PA and NA. The mid-frontal findings, however, revealed some inconsistencies, with the between-groups analyses indicating a significant linkage with PA but not with NA, whereas the correlational analyses revealed the reverse pattern, at least among subjects with stable EEG asymmetry.

An examination of scatter plots displaying the regression of PA and NA on mid-frontal asymmetry revealed the likely reason for this discrepancy. NA tended to be linearly related to asymmetry. Conversely, consistent with the results of the omnibus ANOVA reported earlier, the PA scores of subjects with extreme relative right frontal activation tended to be rather discontinuous with scores of subjects with different patterns of asymmetry. These observations are consistent with the common observation that correlational methods are superior when variables are linearly related but that between-groups approaches may be optimal under other circumstances. Clearly, however, the issue goes well beyond that of the relative statistical power afforded by different analytic approaches. In particular, a prime conceptual question is whether variations in patterns of anterior asymmetry reflect differences in kind or differences in degree. We believe that further understanding of the etiology and biological substrates of individual differences in anterior asymmetry will be the critical factor determining whether a typological or dimensional approach is optimal.

The fourth and final issue raised by the present study is that of the stability of anterior EEG asymmetry. Although caution is necessary because of the nonsignificant parallelism results, the correlational analyses indicated that whereas resting asymmetry significantly predicted self-reported affect among subjects whose EEG patterns were stable over time, it typically failed to be a significant predictor among subjects with unstable EEG patterns. In addition, it is important to note that the right and left anterior groups used in the between-groups analyses were composed of subjects who demonstrated both extreme and stable patterns of asymmetry.

That anterior asymmetry demonstrated adequate but not optimal stability and that it failed to significantly predict emotion self-reports in subjects with unstable EEG asymmetry are additional reasons why our results require replication and extension. One question raised by these findings is the nature of individual differences in EEG stability. As reported by Tomarken et al. (in press), estimates of the internal consistency reliability of anterior asymmetry across the eight baselines of a given session indicated high internal consistency in the present sample (e.g., coefficient αs in the .85 to .95 range). This finding suggests that changes in asymmetry over time reflect "true" change rather than measurement error (e.g., Nunnally, 1978). Whether, however, there actually are replicable individual differences in the stability of asymmetry requires an experimental design in which EEG is assessed across multiple recording occasions. Such a design would allow examination of whether those subjects whose asymmetry significantly shifts between Sessions 1 and 2, for example, also are more likely to demonstrate a shift on other occasions.

If there are in fact replicable individual differences in stability of asymmetry, it may prove to be the case that, in the long run, a methodological strategy involving the selection of those subjects who manifest stable asymmetry will yield optimal prediction of affective and other measures. Such a strategy has a strong parallel to one that has been advocated by various personality theorists who have argued that a given trait may only be applicable to those individuals who manifest a consistent pattern of response across items that assess it (e.g., Baumeister & Tice, 1988; Bem & Allen, 1974). However, it should be noted that several commentators have strongly criticized whether selection of stable or consistent responders is actually an optimal strategy for personality assessment (e.g., Paunonen & Jackson, 1985; Tellegen, 1988). Such critics have favored an alternative approach emphasizing aggregation of measures over time or across items and the use of such aggregated measures to predict the behavior of all subjects (e.g., Rushton, Brainerd, & Pressley, 1983). In future studies including multiple assessments of resting asymmetry across time, we intend to evaluate the comparative validity of these differing approaches to the measurement of anterior asymmetry.

Finally, we should note one additional limitation of the present study that warrants attention in future research. Only women served as subjects. Although there is generally weak evidence for sex differences in individual differences related to hemispheric asymmetry (e.g., Hellige, 1990), and although we have recently found no evidence of sex differences in asymmetries related to emotion (e.g., Sobotka, Davidson, & Senulis, 1991), future studies including both sexes are necessary to definitively assess whether the present findings are generalizable to men.

In summary, the present findings indicate that resting anterior EEG asymmetry predicts individual differences in positive and negative affect. These results require replication and extension by studies designed to clarify the precise affective dimensions assessed by EEG asymmetry and the optimal methods for measurement of EEG parameters.

References

Ahern, G. L., & Schwartz, G. E. (1985). Differential lateralization for positive and negative emotion in the human brain: EEG spectral analysis. *Neuropsychologia, 23,* 745–756.

Baron, R. M., & Kenny, D. A. (1986). The moderator-mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology, 51,* 1173–1182.

Baumeister, R. F., & Tice, D. M. (1988). Metatraits. *Journal of Personality, 56,* 571–597.

Beck, A. T., Ward, C. H., Mendelson, M., Mock, J., & Erbaugh, J. (1961). An inventory for measuring depression. *Archives of General Psychiatry, 4,* 561–571.

Bem, D. J., & Allen, A. (1974). On predicting some of the people some of the time: The search for cross-situational consistencies in behavior. *Psychological Review, 81,* 506–520.

Blom, J. L., & Anneveldt, M. (1982). An electrode cap tested. *Electroencephalography and Clinical Neurophysiology, 54,* 591–594.

Blumberg, S. R., & Hokanson, J. E. (1983). The effects of another person's response style on interpersonal behavior in depression. *Journal of Abnormal Psychology, 92,* 196–209.

Bryden, M. P. (1982). *Laterality: Functional asymmetry in the intact brain.* San Diego, CA: Academic Press.

Burke, M. J., Brief, A. P., George, J. M., Roberson, L., & Webster, J. (1989). Measuring affect at work: Confirmatory analyses of competing mood structures with conceptual linkages to cortical regulatory systems. *Journal of Personality and Social Psychology, 57,* 1091–1102.

Campbell, D. T., & Fiske, D. W. (1959). Convergent and discriminant validation by the multitrait–multimethod matrix. *Psychological Bulletin, 56,* 81–105.

Dabbs, J. M., & Chou, G. (1980). Left-right carotid blood flow predicts specialized mental ability. *Neuropsychologia, 18,* 711–713.

Davidson, R. J. (1984). Affect, cognition, and hemispheric specialization. In C. E. Izard, J. Kagan, & R. B. Zajonc (Eds.), *Emotions, cognition, and behavior* (pp. 320–365). Cambridge, England: Cambridge University Press.

Davidson, R. J. (1988). EEG measures of cerebral asymmetry: Conceptual and methodological issues. *International Journal of Neuroscience, 39,* 71–89.

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

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

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

Davidson, R. J., Schaffer, C. E., & Saron, C. D. (1985). Effects of lateralized stimulus presentations on the self-report of emotion and EEG asymmetry in depressed and nondepressed subjects. *Psychophysiology, 22,* 353–364.

Davidson, R. J., Taylor, N., & Saron, C. D. (1979). Hemisphericity and styles of information processing: Individual differences in EEG asymmetry and their relationship to cognitive performance. *Psychophysiology, 16,* 197.

Davidson, R. J., & Tomarken, A. J. (1989). Laterality and emotion: An electrophysiological approach. In F. Boller & J. Grafman (Eds.), *Handbook of neuropsychology* (pp. 419–441). Amsterdam: Elsevier.

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

Derryberry, D., & Rothbart, M. K. (1984). Emotion, attention, and temperament. In C. E. Izard, J. Kagan, & R. Zajonc (Eds.), *Emotions, cognition, and behavior* (pp. 132–166). Cambridge, England: Cambridge University Press.

Diener, E., Larsen, R. J., Levine, S., & Emmons, R. A. (1985). Intensity and frequency: Dimensions underlying positive and negative affect. *Journal of Personality and Social Psychology, 48,* 1253–1265.

Ehrlichman, H., & Wiener, M. S. (1979). Consistency of task-related EEG asymmetries. *Psychophysiology, 16,* 247–252.

Glass, A., & Butler, S. R. (1977). Alpha EEG asymmetry and speed of left hemisphere thinking. *Neuroscience Letters, 4,* 231–235.

Gray, J. A. (1982). *The neuropsychology of anxiety.* New York: Oxford University Press.

Hellige, J. B. (1990). Hemispheric asymmetry. *Annual Review of Psychology, 41,* 55–80.

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

Kang, D. H., Davidson, R. J., Coe, C. L., Wheeler, R. E., Tomarken, A. J., & Ershler, W. B. (1991). Frontal brain asymmetry and immune function. *Behavioral Neuroscience, 105,* 860–869.

Kinsbourne, M. (1978). Evolution of language in relation to lateral action. In M. Kinsbourne (Ed.), *Asymmetrical function of the brain* (pp. 553–556). Cambridge, England: Cambridge University Press.

Kirk, R. E. (1982). *Experimental design: Procedures for the behavioral sciences.* Monterey, CA: Brooks/Cole.

Kraemer, H. C. (1981). Coping strategies in psychiatric clinical research. *Journal of Consulting and Clinical Psychology, 49,* 309–319.

Larsen, R. J. (1984). Theory and measurement of affect response intensity as an individual difference characteristic. *Dissertation Abstracts International, 5,* 2297B. (University Microfilms No. 84-22112)

Larsen, R. J., & Diener, E. (1987). Affect intensity as an individual difference characteristic: A review. *Journal of Research in Personality, 21,* 1–39.

Larsen, R. J., Diener, E., & Emmons, R. A. (1986). Affect intensity and reactions to daily life events. *Journal of Personality and Social Psychology, 51,* 803–814.

Larsen, R. J., & Ketelaar, T. (1991). Personality and susceptibility to positive and negative emotional states. *Journal of Personality and Social Psychology, 61,* 132–140.

Levy, J., Heller, W., Banich, M. T., & Burton, L. A. (1983). Are variations among right-handed individuals in perceptual asymmetries caused by characteristic arousal differences between the hemispheres? *Journal of Experimental Psychology: Human Perception and Performance, 9,* 329–359.

Marascuilo, L. A., & Levin, J. R. (1983). *Multivariate statistics in the social sciences.* Monterey, CA: Brooks/Cole.

Nunnally, J. C. (1978). *Psychometric theory.* New York: McGraw-Hill.

Oldfield, R. C. (1971). The assessment and analysis of handedness: The Edinburgh Inventory. *Neuropsychologia, 9,* 97–113.

Paunonen, S. V., & Jackson, D. N. (1985). Idiographic measurement strategies for personality and prediction: Some unredeemed promissory notes. *Psychological Review, 92,* 486–511.

Robinson, R. G., Kubos, K. L., Starr, L. B., Rao, K., & Price, T. R. (1984). Mood disorders in stroke patients: Importance of location of lesion. *Brain, 107,* 81–93.

Rushton, J. P., Brainerd, C. J., & Pressley, M. (1983). Behavioral development and construct validity: The principle of aggregation. *Psychological Bulletin, 94,* 18–38.

Russell, J. A. (1980). A circumplex model of affect. *Journal of Personality and Social Psychology, 36,* 1152–1168.

Russell, J. A. (1983). Pancultural aspects of the human conceptual organization of emotions. *Journal of Personality and Social Psychology, 45,* 1281–1288.

Sacco, W. P. (1981). Invalid use of the Beck Depression Inventory to identify depressed college students: A methodological comment. *Cognitive Therapy and Research, 5,* 143–147.

Sobotka, S. S., Davidson, R. J., & Senulis, J. A. (1991). *Anterior brain electrical asymmetries in response to reward and punishment.* Manuscript submitted for publication.

Tellegen, A. (1985). Structures of mood and personality and their relevance to assessing anxiety, with an emphasis on self-report. In A. H. Tuma & J. Maser (Eds.), *Anxiety and the anxiety disorders* (pp. 681–706). Hillsdale, NJ: Erlbaum.

Tellegen, A. (1988). The analysis of consistency in personality assessment. *Journal of Personality, 56,* 621–663.

Tomarken, A. J., Davidson, R. J., Baskin, D., & Angier, C. (1987). [Preliminary assessment of the test–retest stability of anterior EEG asymmetry]. Unpublished raw data.

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

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

Tucker, D. M., & Williamson, P. A. (1984). Asymmetric neural control systems in human self-regulation. *Psychological Review, 91,* 185–215.

Wagner, H. L., MacDonald, C. J., & Manstead, A. S. R. (1986). Communication of individual emotions by spontaneous facial expressions. *Journal of Personality and Social Psychology, 50,* 737–743.

Watson, D. (1988). The vicissitudes of mood measurement: Effects of varying descriptors, time frames, and response formats on measures of positive and negative affect. *Journal of Personality and Social Psychology, 55,* 128–141.

Watson, D., Clark, L. A., & Tellegen, A. (1984). Cross-cultural convergence in the structure of mood: A Japanese replication and a comparison with U.S. findings. *Journal of Personality and Social Psychology, 47,* 127–144.

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

Watson, D., & Tellegen, A. (1985). Toward a consensual structure of mood. *Psychological Bulletin, 98,* 219–235.

Wheeler, R. E., Davidson, R. J., & Tomarken, A. J. (in press). Frontal brain asymmetry and emotional reactivity: A biological substrate of affective style. *Psychophysiology.*

Zevon, M. A., & Tellegen, A. (1982). The structure of mood change: An idiographic/nomothetic analysis. *Journal of Personality and Social Psychology, 43,* 111–122.

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