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Frontal Brain Activation in Repressors and Nonrepressors

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We assessed whether resting anterior asymmetry would discriminate individual differences in repressive–defensive coping styles. In 2 sessions, resting electroencephalogram was recorded from female adults during 8 60-s baselines. Subjects were classified as repressors or nonrepressors on the basis of scores on the Marlowe-Crowne Social Desirability Scale (MC), the State-Trait Anxiety Inventory (STAI), and the Beck Depression Inventory (BDI). In midfrontal and lateral frontal sites, repressors demonstrated relative left hemisphere activation when compared with other groups. The MC, but not the STAI or the BDI, contributed unique variance to frontal asymmetry. Relative left frontal activation may be linked to a self-enhancing regulatory style that promotes lowered risk for psychopathology.

A major focus of psychopathology research in recent years has been the identification of factors that promote increased vulnerability to psychopathology. A complementary focus is the identification of protective factors that promote heightened resilience and lowered risk (e.g., Rutter, 1985). In our study, we assessed the relation between two constructs that appear to be linked to differential risk. These constructs are resting anterior electroencephalographic (EEG) asymmetry and repressive–defensive coping. Our findings have implications for understanding the interaction between biological and psychological predispositions linked to decreased vulnerability to psychopathology.

Individual Differences in Anterior EEG Asymmetry: Relations With Emotion and Psychopathology

Recent studies have shown that resting EEG asymmetry in anterior (e.g., prefrontal) regions of the brain is a biological marker of affective style and a potential marker of risk for psychopathology. Relative left hemisphere hypoactivation in anterior regions has been linked to increased negative affect, decreased positive affect, or both, whereas relative left anterior activation (i.e., alpha suppression) has been linked to the opposite pattern (for reviews, see R. J. Davidson, 1992; R. J. Davidson & Tomarken, 1989). Consistent with prior neurological findings indicating a linkage between left anterior lesions and depression (e.g., Robinson, Kubos, Starr, Rao, & Price, 1984), several EEG studies have shown a linkage between left frontal hypoactivation and depression (e.g., Allen, Iacono, Depue, & Arbisi, 1993; Henriques & Davidson, 1991). Perhaps most important, several recent findings indicate that resting frontal asymmetry may be a marker of differential risk for affective or anxiety disorders. Currently euthymic adults who have a prior history of depression demonstrate relative left anterior hypoactivation (Henriques & Davidson, 1990). In addition, behaviorally inhibited children, who may be at heightened risk for anxiety or affective disorders (e.g., Kochanska, 1991), demonstrate relative left anterior hypoactivation when compared with uninhibited children (Davidson, Finman, Rickman, Straus, & Kagan, 1993).

To date, however, relatively few studies have explicitly examined the relation between resting anterior asymmetry and differential risk for psychopathology. In addition, it is presently unclear what processes linked to resting anterior asymmetry may account for its relation to differential risk. One possibility is that relative left anterior hyperactivation is linked to protective processes that promote decreased vulnerability to psychopathology. Concerning the identification of such processes, a variety of findings have converged in recent years to indicate that many nondepressed individuals demonstrate a self-enhancing or self-serving cognitive style. For example, such individuals often make self-serving attributions, overestimate their control over desired outcomes, and demonstrate unrealistic optimism. Evidence suggests that this self-enhancing cognitive style may (a) promote the relatively rapid dampening of negative affective responses to stressors; (b) serve to maintain or enhance self-esteem; and (c) confer lowered risk for psychopathology (for reviews, see Alloy & Abramson, 1988; S. E. Taylor, 1991; S. E. Taylor & Brown, 1988). Conversely, several findings support the assertion that depression is characterized by the breakdown of such "self-deceptive" mechanisms (e.g., Alloy & Abramson, 1988; Sackeim, 1983). To our knowledge, no study has examined whether relative left anterior activation is linked to a self-enhancing cognitive style and other factors that predict lowered risk for psychopathology. Prior findings on the correlates of a repressive coping style suggest its relevance to both issues.

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Individual Differences in Repressive–Defensive Coping Styles

Using criteria first developed by Weinberger, Schwartz, and Davidson (1979), a number of researchers have classified subjects on the basis of responses to the Marlowe-Crowne Social Desirability Scale (MC; Crowne & Marlowe, 1964) and the Taylor Manifest Anxiety Scale (TMAS; J. Taylor, 1953) or measures of related constructs (e.g., neuroticism). Three groups have typically been formed: repressors (low anxiety and high MC scores), low anxious (low anxiety and low MC scores), and high anxious (high anxiety and low MC scores).

Although this typology has yielded a number of correlates, one set of findings is particularly relevant in our context. Repressors, high MC scorers, or both demonstrate a regulatory style that would appear to inhibit the perception of threat and the experience of negative affect and to promote the maintenance of self-esteem. For example, such individuals demonstrate (a) a self-serving attributional style (M. N. Davidson & Weinberger, 1992; Weinberger & Gomes, 1989); (b) a self-serving hindsight bias (Campbell & Tesser, 1983); (c) impaired memory for negative self-relevant feedback (Baumeister & Cairns, 1992) and negatively toned autobiographical events (e.g., Davis, 1990); (d) attentional avoidance of threatening cues unless provoked by strong self-presentational concerns that may render heightened attention adaptive (e.g., Baumeister & Cairns, 1992); (e) a relative inability to consciously perceive negative affective stimuli under specific conditions (for a review, see Schwartz, 1990); and (f) more classically defined defense mechanisms characterized by the inhibition of interpersonal conflict and ambivalent or negative emotions and by selective accentuation of the positive (e.g., Evans, 1979; Sincoff, 1992). It could be predicted that the self-enhancing cognitive style of repressors protects against the development of psychopathology. Consistent with this reasoning, Lane, Merikangas, Schwartz, Huang, and Prusoff (1990) have shown that high MC scores are associated with a decreased lifetime incidence of affective disorders and other manifestations of psychopathology.

Clearly, this evidence is relevant to the questions raised earlier concerning the relation between resting anterior asymmetry and factors that mediate differential risk for psychopathology. If relative left anterior activation is linked to decreased risk and to a self-enhancing cognitive style, one would predict that repressors would demonstrate relative left anterior activation when compared with both high- and low-anxious subjects. We tested this hypothesis in the current study. We recognized, however, that there are alternative predictions that could be offered concerning the relation between the repressive–defensiveness typology and resting anterior asymmetry. First, it is conceivable that resting anterior asymmetry is more strongly linked to individual differences in self-reported emotion than to styles of regulating positive and negative affect. If so, one could predict that repressors and low-anxious individuals, both of whom report low levels of negative affect, would demonstrate relative left anterior activation when compared with high-anxious individuals and would fail to differ significantly from each other.

Several prior findings also suggest a third possible pattern of effects. Repressors report low state anxiety in response to laboratory stressors, yet they demonstrate heightened responsivity on autonomic measures (e.g., Aspendorf & Scherer, 1983; Weinberger et al., 1979). In addition, they demonstrate heightened resting systolic blood pressure (e.g., King, Taylor, Albright, & Haskell, 1990) and heightened salivary cortisol levels (Brown et al., 1993). Increased physiological activation in stress-sensitive response systems has suggested to some commentators an interpretation of repressive–defensiveness that is different from that offered earlier. In this view, repressors demonstrate what Shedler, Mayman, and Manis (1993) have termed the *illusion of mental health.* From this perspective, repressors respond to stressors with heightened distress that they defensively deny but that is revealed by heightened activation on peripheral physiological measures. Although there are alternative interpretations of the physiological activation demonstrated by repressors (see the Discussion section), this particular interpretation may lead to the prediction that repressors would demonstrate relative right anterior activation, particularly when compared with low-anxious subjects. The latter have consistently demonstrated decreased autonomic and neuroendocrine activation when compared with repressors. Consistent with this hypothesis, several studies have shown predominant right hemisphere mediation of autonomic (e.g., Dimond & Farrington, 1977) and neuroendocrine (Wittling & Pflüger, 1990) responses to negative affective stimuli.

Summary and Overview

In sum, prior findings indicate that both relative left anterior activation and a repressive coping style are linked to decreased risk for psychopathology. This evidence leads to the prediction that repressors will demonstrate relative left anterior activation when compared with other groups. However, alternative predictions could be offered. To test these different hypotheses, we recorded resting EEG in college students on two occasions separated by 3 weeks. To assess the repressive–defensive typology, subjects were administered the MC and the trait version State-Trait Anxiety Inventory (STAI; Spielberger, Gorsuch, & Lushene, 1970). In addition, subjects were administered the Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961). In combination with the MC, we used the BDI to classify subjects into groups that paralleled classifications on the basis of the MC and STAI. We included the BDI in addition to the STAI for two reasons. First, several prior studies that have demonstrated self-enhancing cognitive biases among nondepressed individuals have used the BDI to classify subjects (e.g., Alloy & Abramson, 1979). Second, as noted previously, both resting EEG asymmetry and high MC scores have been linked to a lowered lifetime history of depression. We recognized that even high BDI scores among college students are typically associated with depression of only subclinical severity. Recall, however, that a major focus of our study was on lowered vulnerability for depression and on discrimination among groups who report low levels of depression or anxiety (e.g., repressors vs. low anxious).

Method

Subjects

Subjects were 90 right-handed (as assessed by the Edinburgh Handedness Inventory; Oldfield, 1971) women recruited from the introduc-

tory psychology pool at the University of Wisconsin—Madison. Only women were used because (a) one goal of the larger project of which our study was one component was to assess the facial expression of emotion and (b) there is evidence that women are more facially expressive than men (e.g., Wagner, MacDonald, & Manstead, 1986).

Procedure

Session 1. Subjects were run individually and participated in two sessions separated by 3 weeks. The procedure followed that described in several recent publications (e.g., Tomarken, Davidson, Wheeler, & Kinney, 1992). After subjects arrived at the laboratory, we applied electrodes. There were eight 1-min resting baselines (four with eyes open and four with eyes closed). Subjects were simply told to be "restful" during the baselines. Two randomly assigned, counterbalanced orders were used for the 60-s eyes-open and eyes-closed baselines. After the final baseline, subjects completed several personality and emotion measures, including the STAI, the MC, and the BDI. Tomarken, Davidson, Wheeler, and Doss (1992) reported the relation between resting anterior asymmetry and several other personality measures.

Session 2. The resting baseline procedure was identical to that described earlier for Session 1. Following the baselines, subjects completed several self-report measures, including a second BDI. After completing the questionnaires, subjects were exposed to nine affective film clips. Wheeler, Davidson, & Tomarken (1993) reported the relation between resting anterior asymmetry and film responses.

In both experimental sessions, immediately after the resting baselines, subjects indicated their mood during the baselines on seven rating scales assessing discrete positive (e.g., happiness) and negative (e.g., fear) emotions. Consistent with prior results (e.g., Tomarken, Davidson, & Henriques, 1990), there was no relation between resting asymmetry measures and positive or negative affect during the baselines (all *ps* > .20).

EEG Recording and Quantification

The recording and quantification of EEG followed the procedure described in detail by Tomarken, Davidson, Wheeler, and Kinney (1992). During Session 1, for approximately half of the subjects (n = 46), EEG was recorded from the left and right midfrontal (F3 and F4) and anterior temporal (T3 and T4) regions and referenced to vertex (Cz). Two additional channels were recorded: Cz–A1 and Cz–A2 (A1 = left earlobe and A2 = right earlobe). For the remainder of the subjects (n = 44), EEG was recorded from midfrontal and anterior temporal sites and the following 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, as well as midline frontal (Fz) and parietal (Pz). In Session 2, for all subjects, recordings were made from all 16 sites noted earlier. All electrode impedances were under 5 kΩ and impedances for homologous (e.g., F3, F4) sites were within 500 Ω of each other. In both sessions, eye movements (electrooculogram [EOG]) were also recorded.

EEG was amplified with a Grass Model 12 Neurodata System (amplification = 40 K, band-pass = 1–100 Hz) and passed through antialiasing low-pass filters set at 65 Hz (roll-off = 24 dB/octave). EEG was digitized at 200 Hz by a minicomputer. The EEG and EOG signals were visually scored, and portions of the data containing eye movements, muscle tension, and other sources of artifact were deleted. In addition to the original recording montage referenced to vertex, a computer-averaged ears reference was derived off-line (see Tomarken, Davidson, Wheeler, & Kinney, 1992, for the computational formula). All artifact-free chunks that were 2.05 s in duration were overlapped by 75% and extracted through a Hamming window. A fast Fourier transform (FFT) then derived estimates of spectral power (in μV^2) in 1-Hz bins. Power values were then averaged across the chunks of a baseline, after which power density (in μV^2/Hz) was computed in five frequency bands: delta (1–4 Hz), theta (4–7 Hz), alpha (8–13 Hz), beta 1 (13–20 Hz), and beta 2 (20–30 Hz).

All power density values were log transformed to normalize distributions. To generate a measure of EEG asymmetry for a given experimental session, we computed weighted means across the eight baselines of that session using the procedure described by Tomarken, Davidson, Wheeler, and Kinney (1992). Finally, the weighted means per session were averaged to yield grand average log power density for each combination of site and frequency band. EEG asymmetry in the midfrontal (F3 and F4) site was the primary measure used to test the alternative experimental hypotheses. Asymmetries in the midfrontal region constitute the clear majority of previous EEG evidence indicating linkages between anterior asymmetry and emotion. Two other anterior sites, anterior temporal (T3 and T4) and lateral frontal (F7 and F8), were also used to test hypotheses. The experimental hypotheses pertained specifically to anterior asymmetry in the alpha frequency band (8–13 Hz). In previous studies, EEG asymmetry in this band has been most consistently linked to emotion (for a review, see R. J. Davidson, 1992). Because decreased alpha power in a given region is associated with increased cortical activation (e.g., R. J. Davidson, Chapman, Chapman, & Henriques, 1990), increased alpha power in a right hemisphere versus homologous left hemisphere site indicates relative left hemisphere activation.

In this article, as in our other recent articles, we focused on the results of analyses conducted using the averaged ears reference. Measures of EEG asymmetry derived using this reference have higher test–retest stability than that provided by vertex-referenced EEG. As reported by Tomarken, Davidson, Wheeler, and Kinney (1992), for the current cohort, the estimated stability of measures of alpha band anterior asymmetry averaged across both sessions was .79–.84. All analyses were based on log power density values averaged across the two experimental sessions. Because of equipment malfunctions or other technical problems, 4 of the original 90 subjects lacked usable EEG data from either Session 1 or Session 2 in these sites and were dropped from the design. Forty-two subjects had usable EEG data from the lateral frontal and more posterior sites in both experimental sessions.

Self-Report Measures

The STAI. The Trait scale of the STAI (Spielberger et al., 1970) is a 20-item self-report measure that assesses the cognitive and affective components of anxiety. Although the majority of previous repressor studies have used the Taylor Manifest Anxiety Scale (TMAS), we used the STAI because of its generally greater use in contemporary studies of trait anxiety. Prior studies have formed the repressive–defensiveness typology using the STAI (e.g., Anderson, Hall, & Spielberger, 1992) and obtained results that conceptually replicate those of studies using the TMAS. As reported by Spielberger et al. (1970), the STAI Trait scale demonstrates excellent internal consistency reliability (coefficient alphas range from .86 to .92) and levels of test–retest stability that are appropriate for a trait measure (e.g., test–retest correlations across 1–4 months range from .73 to .86).

The BDI. The BDI is a 21-item self-report instrument that assesses the presence and severity of cognitive, motivational, affective, and somatic symptoms of depression. Although there is evidence that the STAI and BDI both tap a broad negative affect dimension (Watson & Clark, 1984), the .65 correlation between these measures in our study suggested significant overlap as well as sufficient unique variance to justify separate presentation of results for the two measures. Because BDI scores can be affected by transient mood states, subjects completed a BDI in each of the two experimental sessions. The test–retest correlation for the Session 1–Session 2 BDI scores was .77. The BDI demonstrates acceptable internal consistency reliability (coefficient alpha = .81; Beck, Steer, & Garbin, 1988). Because of an administrative error, 2

subjects failed to complete the BDI on both occasions and were eliminated from analyses involving this measure.

The MC. The MC (Crowne & Marlowe, 1964) is a 33-item self-report measure that assesses both the tendency to deny negative characteristics that are likely to be common in the general population (e.g., "I have never intensely disliked anyone") and the tendency to ascribe to oneself positive characteristics that are thought to be rare in the general population (e.g., "I never hesitate to go out of my way to help someone in trouble"). Consistent with this dual emphasis, the MC is both moderately negatively correlated with measures of negative affect and moderately positively correlated with measures of positive affect. For example, in our sample, the MC was negatively correlated with the STAI, BDI, and Negative Affect scale of the Positive and Negative Affect Schedule–General (PANAS-GEN; Watson, Clark, & Tellegen, 1988; rs = −.48, −.35, and −.35, respectively, all $ps < .005$) and positively correlated with the PANAS-GEN Positive Affect scale ($r = .33$, $p < .005$). The MC has appropriate internal consistency reliability (KR 20 reliability = .88) and test–retest stability (1-month test–retest correlation = .88) for a trait measure (Crowne & Marlowe, 1964).

Design and Analysis

Three different analytical approaches were used to test predictions. The first approach assessed whether groups of subjects categorized on the basis of patterns of responses to the STAI and MC and to the BDI and MC would differ in EEG asymmetry in anterior and more posterior sites. More specifically, subjects who scored in the top quarter (≥17) of the distribution of MC scores and below the median (38.5) on the STAI were classified as STAI repressors (MC M = 19.92, STAI M = 30.75). Subjects who scored below the top quarter on the distribution of MC scores and above the median on the STAI were classified as the high-anxiety group (MC M = 11.34, STAI M = 47.05), and subjects who scored below the top quarter of the MC and below the median on the STAI were classified as the low-anxiety group (MC M = 12.45, STAI M = 33.68).

To generate BDI classifications, we averaged subjects' BDI scores assessed during the two recording sessions. Paralleling the classifications on the basis of the STAI, subjects who scored in the top quarter of the distribution of MC scores and below the median (5.5) on averaged BDI scores were classified as BDI repressors (MC M = 20.27, BDI M = 2.34). Subjects who scored below the top quarter on the MC scores and above the median on the BDI were classified as high depression (MC M = 11.32, BDI M = 10.00), and subjects who scored below the top quarter of the MC and below the median on the BDI were classified as low depression (MC M = 12.21, BDI M = 3.19).

The percentile cutoff points used to classify subjects into STAI-derived groups corresponded to those used in the original Weinberger et al. (1979) study and several subsequent studies of the repressive–defensive typology (e.g., Wexler, Schwartz, Warrenburg, Servis, & Tarlatizis, 1986). As noted by Weinberger et al., one justification for the requirement that repressors score in the top quarter of the MC is the fact that lowered anxiety per se predicts heightened MC scores (i.e., such criteria allow for better discrimination of repressors and low-anxious subjects). Supplementary analyses of the classifications on the basis of the STAI and BDI indicated that the results and conclusions presented did not change appreciably if alternative cutoff points were used to select subjects. In the case of the BDI, it is particularly noteworthy that the formation of more extreme groups (e.g., requiring high-depression subjects to score 10 or above on the BDI on both occasions) failed to change the pattern of mean differences. Finally, we attempted to form additional groups composed of subjects who scored in the top quarter of the distribution of MC scores and above the median on the STAI or the BDI. Too few subjects met these criteria to be included in the design.

Two-way coping style (STAI repressor, high anxious, low anxious) × Hemisphere (left, right) analyses of variance (ANOVAs) assessed whether the three STAI-derived coping style groups would differ in alpha band log power density in midfrontal (F3 and F4), lateral frontal (F7 and F8), and anterior temporal (T3 and T4) sites. Similarly, Coping Style × Hemisphere ANOVAs assessed whether the three BDI-derived coping style groups would differ in alpha band power in these sites. The Coping Style × Hemisphere interaction was the effect yielded by such analyses that is relevant to the alternative hypotheses noted earlier. This interaction is mathematically identical to the coping style main effect of a one-way coping style ANOVA performed directly on alpha band asymmetry (computed as log right–log left power density). Significant Coping Style × Hemisphere interactions were followed up by pairwise Fisher's least significant differences tests comparing the three coping style groups on asymmetry measures (e.g., Hayter, 1986). Supplementary analyses assessed whether coping style groups would differ on EEG asymmetry in other frequency bands and sites. No differences were predicted.

We also used a second analytical approach that was similar to that used in several of our recent studies on the relation between resting EEG asymmetry and emotion or physiological measures (e.g., Tomarken, Davidson, Wheeler, & Doss, 1992). We formed groups on the basis of subjects' resting asymmetry values for pairs of homologous sites (e.g., F3 and F4). We classified subjects into two groups: those whose asymmetry scores were in top quarter of the distribution of asymmetry scores during both Sessions 1 and 2 (stable and extreme relative left hemisphere activation) and those whose asymmetry scores were in the bottom quarter of the distribution on both occasions (stable and extreme relative right hemisphere activation). Because complete data for the majority of subjects across both experimental sessions were available only from the F3–F4 and T3–T4 regions, we performed such classifications only on the basis of these two sites. We then conducted Asymmetry Group (left, right) × Scale (MC, STAI, BDI) ANOVAs assessing between-groups differences on self-report measures. Because the MC, STAI, and BDI differ in their potential ranges, we performed these analyses on standardized scale scores.

In the first two analytical approaches noted previously, a categorical approach is used. An alternative approach is one that treats both resting anterior asymmetry and self-report measures as continua. For this reason, we also adopted a third analytical approach according to which the relation between anterior asymmetry measures and the MC, STAI, and BDI scales was assessed by Pearson product–moment correlations and multiple regression analyses including the sample as a whole.

Results

Differences Among Coping Style Groups on Anterior Alpha Band Asymmetry

STAI-derived coping style groups. Table 1 shows mean log power density values and asymmetry scores in the three anterior sites for the STAI-derived and BDI-derived coping style groups. The STAI Coping Style × Hemisphere ANOVA on midfrontal (F3 and F4) power yielded a significant main effect for hemisphere, $F(1, 79) = 15.20$, $p < .001$, and a significant Coping Style × Hemisphere interaction, $F(2, 79) = 4.56$, $p < .025$. As indicated by the results of post hoc contrasts following up the significant interaction, STAI repressors had significantly greater relative left frontal activation (i.e., alpha suppression) than did both the high-anxious and low-anxious groups ($ps < .05$ and .01, respectively). The latter two groups failed to differ significantly in midfrontal asymmetry ($p > .30$). Separate contrasts between STAI repressors and other groups on measures of power density in each site failed to yield any significant differ-

Table 1
Mean Alpha Band (8–13 Hz) Log Power Density and Asymmetry in Anterior Sites

	Region								
	Midfrontal			Lateral frontal			Anterior temporal		
Group	F3 LPD	F4 LPD	F3–F4 asym	F7 LPD	F8 LPD	F7–F8 asym	T3 LPD	T4 LPD	T3–T4 asym
STAI derived									
Anxiety repressors									
M	0.89	0.98	0.08	0.05	0.14	0.08	0.13	0.25	0.12
SD	0.80	0.77	0.09	0.99	0.93	0.18	0.85	0.84	0.34
n	23			14			23		
Low anxiety									
M	0.63	0.64	0.01	−0.32	−0.37	−0.05	−0.18	−0.07	0.17
SD	0.99	1.01	0.08	0.85	0.85	0.10	1.08	1.10	0.25
n	26			14			26		
High anxiety									
M	1.11	1.13	0.02	0.23	0.15	−0.09	0.43	0.48	0.05
SD	0.74	0.73	0.09	0.81	0.76	0.20	0.91	0.86	0.28
n	33			12			33		
BDI derived									
Depression repressors									
M	1.05	1.12	0.07	0.13	0.25	0.11	0.31	0.46	0.16
SD	0.81	0.78	0.10	1.05	0.98	0.15	0.85	0.83	0.35
n	20			13			20		
Low depression									
M	0.73	0.75	0.02	−0.15	−0.21	−0.05	−0.03	0.12	0.15
SD	0.97	1.00	0.08	0.75	0.77	0.07	1.05	1.10	0.24
n	27			12			27		
High depression									
M	1.04	1.06	0.02	0.02	−0.07	−0.08	0.32	0.39	0.07
SD	0.79	0.77	0.09	0.97	0.91	0.20	0.99	0.89	0.29
n	32			14			32		

Note. LPD = averaged-ears referenced log-transformed power density in μV^2/Hz (natural log). Asym = asymmetry, calculated as log right minus log left power density. Power density values are means averaged across the two electroencephalographic sessions. Higher asymmetry scores indicate greater relative left hemisphere activation. STAI = State-Trait Anxiety Inventory; BDI = Beck Depression Inventory.

ences ($ps > .10$). Thus, repressors were differentiated from other groups primarily on the *difference* in power between homologous midfrontal sites.

The ANOVA performed on lateral frontal power density also yielded a significant STAI Coping Style × Hemisphere interaction, $F(2, 37) = 4.05$, $p < .05$. Follow-up contrasts indicated a pattern of group differences that mirrored those yielded by analyses of the midfrontal site. Repressors demonstrated relative left frontal activation when compared with both high-anxious and low-anxious subjects ($ps < .025$ and .01, respectively). The latter two groups failed to differ ($p > .40$). Although repressors demonstrated relative left anterior activation, both of the other groups tended to demonstrate relative right anterior activation in the lateral frontal region (see Table 1). The STAI Coping Style × Hemisphere ANOVA performed on anterior temporal log power density yielded only a significant main effect for hemisphere, $F(1, 79) = 12.56$, $p < .001$, because of greater relative left hemisphere activation overall (the probability level for the Coping Style × Hemisphere interaction was .20).

BDI-derived coping style groups. The results of analyses comparing the BDI-derived coping style groups directly paralleled those for the STAI-derived groups. Significant Coping Style × Hemisphere interactions were observed for both the midfrontal and lateral frontal sites ($ps < .05$ and .005, respectively). In both cases, BDI-derived repressors had significantly greater relative left frontal activation than both the low- and high-depression groups ($ps < .05$; see Table 1). The latter two groups failed to differ in midfrontal and lateral frontal asymmetry. No group differences were observed on measures of alpha power density in specific frontal sites (e.g., F3 alone) or on measures of anterior temporal power density or asymmetry ($ps > .20$).

Differences Among Coping Style Groups on EEG Asymmetry in Other Frequency Bands and Regions

For the midfrontal and anterior temporal sites, Coping Style × Hemisphere × Region (midfrontal, anterior temporal) multivariate ANOVAs (MANOVAs) were performed on measures of power density in the delta, theta, beta 1, and beta 2 bands, respectively. Because lateral frontal EEG averaged across both sessions was available from only approximately half of the subjects, we did not include this site in the MANOVAs. These analyses indicated no significant STAI-derived or BDI-derived group

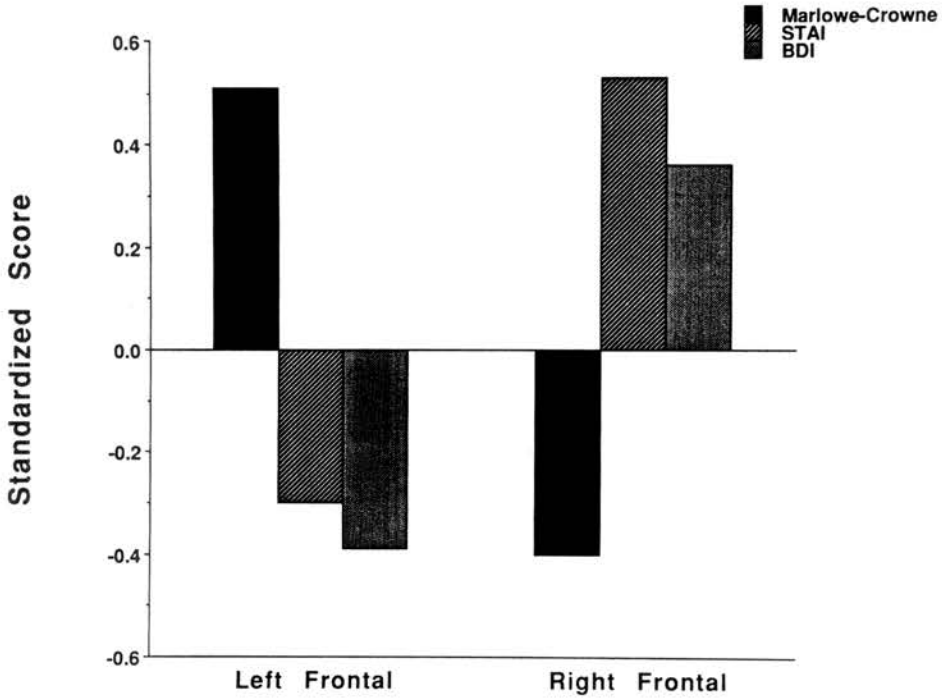

Figure 1. Mean standardized Marlowe-Crowne Social Desirability Scale, State-Trait Anxiety Inventory (STAI) Trait, and Beck Depression Inventory (BDI) scores for the left midfrontal (n = 12) and right midfrontal (n = 13) groups.

differences in asymmetry or overall power in any of the four frequency bands ($ps > .10$). Similarly, Coping Style × Hemisphere ANOVAs performed on measures of lateral frontal power density in each of the four bands revealed no significant main effects or interactions involving coping style factors (all $ps > .20$). In sum, as expected, group differences in anterior EEG asymmetry were evident only in the alpha band.

To assess group differences in more posterior sites, we performed band-specific Coping Style × Hemisphere × Region (central, parietal, posterior temporal) MANOVAs on power density in the delta, theta, alpha, beta 1, and beta 2 bands. These MANOVAs failed to indicate any significant between-groups differences in power or asymmetry (all $ps > .05$).

Differences Between Midfrontal and Anterior Temporal Asymmetry Groups on Self-Report Measures

We also assessed whether groups classified on the basis of stable and extreme patterns of EEG asymmetry in the midfrontal and anterior temporal sites would differ on standardized MC, STAI, and BDI scores. For each region, we conducted a mixed-model 2 (relative left activation, relative right activation) × 3 (MC, STAI, BDI) ANOVA. When subjects were classified on the basis of midfrontal (F3 and F4) asymmetry (ns = 12 and 13 in the left- and right-activated groups, respectively), the ANOVA yielded a significant Asymmetry Group × Scale interaction, $F(2, 46) = 4.18$, $p < .05$. As shown in Figure 1, relative left frontal subjects were characterized by MC scores that were above the mean of the sample and by STAI and BDI scores that were below the mean of the sample. The reverse pattern was characteristic of relative right frontal subjects. The results of a planned contrast comparing the two groups on the difference between standardized MC scores and the average of the BDI and STAI standardized scores was consistent with this observation ($p < .05$). Simple effects contrasts following up the significant Group × Scale interaction indicated that the two midfrontal asymmetry groups differed significantly on MC scores ($p = .05$), with trends evident on the STAI ($p < .07$) and BDI ($p = .10$). No significant effects were yielded by an ANOVA classifying subjects on the basis of anterior temporal asymmetry (all $ps > .05$).

Correlational and Multiple Regression Analyses

Table 2 shows correlations among measures of alpha band asymmetry and the MC, STAI, and BDI. Additional correlations not shown in this table were computed between asymmetry measures and the MC minus STAI and MC minus BDI difference scores. Consistent with the between-groups analyses, the only correlations that were significant involved the midfrontal and lateral frontal sites. Relative left frontal activation in both of these sites was positively and significantly correlated with the MC (see Table 2) and with the standardized MC–STAI (midfrontal $r = .32$, lateral frontal $r = .44$, $ps < .005$) and MC–BDI (midfrontal $r = .26$, $p < .025$, lateral frontal $r = .45$, $p < .005$) difference scores. In addition, relative left frontal activation in the lateral frontal site was significantly inversely corre-

Table 2
Correlations and Hierarchial Regression Analyses With Self-Report Measures as Predictors of Frontal Alpha Band Asymmetry

Dependent variable and predictor	Correlations		Regression	
	r	*p*	*β*	*p*
Midfrontal asymmetry				
MC	.35	<.0001	.33	<.01
STAI	−.20	<.06	−.04	*ns*
MC × STAI	—	—	.29	*ns*
MC	.35	<.0001	.35	<.005
BDI	−.09	*ns*	.03	*ns*
MC × BDI	—	—	.38	*ns*
Lateral frontal asymmetry				
MC	.45	<.005	.40	<.025
STAI	−.31	<.05	−.09	*ns*
MC × STAI	—	—	−.27	*ns*
MC	.45	<.005	.40	<.025
BDI	−.28	<.07	−.13	*ns*
MC × BDI	—	—	−.25	*ns*

Note. *r* = Zero-order Pearson correlation; *β* = standardized beta weight; asymmetry = log right hemisphere power density minus log left hemisphere power density; MC = Marlowe-Crowne Social Desirability Scale; STAI = State-Trait Anxiety Inventory; BDI = Beck Depression Inventory. For the BDI and asymmetry measures, means across the two experimental conditions were used. For all regressions, the two self-report measure (e.g., MC, STAI) were entered simultaneously in an initial step, and the interaction between the two measures (e.g., MC × STAI) was entered in a second step. The sample sizes were 86 (midfrontal STAI), 84 (midfrontal Beck), and 42 (lateral frontal).

lated with the STAI. There were no significant correlations involving other regions or frequency bands, (all *ps* > .05).

To assess the variance in midfrontal and lateral frontal asymmetry measures that was uniquely predicted by the MC, STAI, and BDI, we computed hierarchical multiple regression analyses. Two multiple regression analyses specified the MC and STAI as predictors of midfrontal and lateral frontal asymmetry, respectively, and two specified the MC and BDI as predictors of midfrontal and lateral frontal asymmetry. In each analysis, the two self-report measures were entered simultaneously in an initial step and their unique contributions were tested. Then, in a second step, the interaction between the two measures (i.e., STAI × MC or BDI × MC) was entered and tested. The standardized beta weights corresponding to each predictor are shown in the right columns of Table 2. These four analyses yielded consistent results. The MC scale contributed significant, unique variance to the prediction of both midfrontal and lateral frontal asymmetry. Conversely, the STAI and BDI beta weights were nonsignificant, as were all interaction terms.

Because of their relevance to the broader issue of the predispositions preferentially linked to resting frontal asymmetry, we also conducted supplementary hierarchical multiple regression analyses in which the MC and measures of generalized negative affectivity (NA) and positive affectivity (PA; Watson et al., 1988) were entered as predictors of frontal asymmetry measures. Although several significant relations between anterior asymmetry measures and dispositional PA and NA in our sample have been reported (Tomarken, Davidson, Wheeler, & Doss, 1992), the joint and interactive effects of these measures and the MC scale have not been previously assessed. In all four multiple regression analyses (PA, NA × midfrontal, lateral frontal), the MC scale contributed significant unique variance to midfrontal asymmetry measures (all *ps* < .025), whereas the contribution of PA and NA was nonsignificant (all *ps* > .10). All interaction terms were also nonsignificant (*ps* > .20).

Discussion

Summary of Results

The results support the prediction that a repressive coping style and higher MC scores would be linked to relative left anterior activation. Whether groups were defined on the basis of the STAI and MC, or on the basis of the BDI and MC, repressors demonstrated relative left hemisphere activation in midfrontal and lateral frontal sites when compared with the other groups. When groups were formed on the basis of stable and extreme patterns of anterior asymmetry, subjects with relative left frontal activation demonstrated a pattern of higher MC scores and lower STAI and BDI scores than did subjects with relative right frontal activation. Correlational analyses indicated significant relations between frontal asymmetry measures and MC scores. Furthermore, multiple regression analyses indicated that the MC, but not the STAI or BDI, contributed significant unique variance to midfrontal and lateral frontal asymmetry. All effects were specific to the two frontal sites and to the alpha frequency band.

Implications for Conceptualizations of Resting Anterior EEG Asymmetry

On the basis of prior evidence indicating that MC scores are linked to a lowered lifetime incidence of depression (Lane et al., 1990), our results support the notion that resting left frontal activation may index decreased risk for psychopathology. Given the known correlates of a repressive coping style and of high MC scores, our results also suggest one mechanism by which relative left anterior activation may contribute to decreased risk for psychopathology. As argued earlier, this evidence suggests that relative left anterior activation may promote heightened access to cognitive and other regulatory processes that serve to inhibit or dampen the experience of negative affect and, conversely, to amplify or sustain positive affect and self-esteem. A variety of evidence suggests that such a style may confer decreased risk for depression and perhaps other manifestations of psychopathology (e.g., Alloy & Abramson, 1988; S. E. Taylor & Brown, 1988). This interpretation of our results is also consistent with several additional findings that indicate a linkage between hemispheric asymmetry and a self-enhancing cognitive style. For example, using behavioral paradigms, Drake has shown that increased left hemisphere activation is associated with self-serving attributions (e.g., Drake & Seligman, 1989) and heightened optimism (Drake, 1984).

Relations With the Approach–Withdrawal Conceptualization

R. J. Davidson (e.g., 1992) and others (e.g., Kinsbourne, 1978) have argued that anterior asymmetry reflects fundamen-

tal motivational dimensions of approach and withdrawal. In this view, relative left frontal activation is associated with heightened appetitive or incentive motivation and greater engagement with the environment. Relative right frontal activation is associated with a heightened tendency for withdrawal from potentially threatening stimuli (e.g., novel ones). From this perspective, left frontal hypoactivation may be one biological substrate of the deficits in reward-oriented, appetitive motivation that often appear to be characteristic of depression (Henriques & Davidson, 1991).

One question raised by our findings is the relation between the approach–withdrawal conceptualization of anterior asymmetry and the notion that it is linked to the self-enhancing cognitive and regulatory processes known to be characteristic of repressors. The two perspectives may be complementary. A major component of strong incentive motivation is the ability to sustain goal-directed behavior when the individual is exposed to a significant challenge (e.g., the experience of failure, a novel or complex task). Research in the broad area of behavioral self-regulation has shown that a variety of cognitive processes can facilitate performance and persistence under these circumstances. Such processes include specific types of attributions (e.g., attributing failure to lack of effort rather than to lack of ability), optimistic expectancies and heightened perceptions of control, selective attention to favorable, relative to unfavorable, aspects of one's performance, and heightened allocation of working memory capacity to the processing of task features and the planning of responses (for a review, see Kirschenbaum & Tomarken, 1982).

Although caution is necessary because few studies have explicitly examined the relation between a repressive coping style and goal-directed performance, it is notable that this style has been linked empirically to several of the processes that facilitate performance and persistence in response to failure or other challenges (e.g., self-serving attributions, selective attention to favorable self-aspects). A repressive style may also be linked to several other processes noted previously. For example, several theorists have argued that a combination of heightened task focus and reduced ruminative self-focus is optimal for goal attainment on complex tasks (e.g., Diener & Dweck, 1978; Kuhl, 1985). Both anxious and depressed individuals commonly respond to failure with ruminative self-preoccupation that appears to mediate performance deficits (e.g., Kuhl, 1985). This response style has also been implicated in the onset or maintenance of depressive episodes (Nolen-Hoeksema, 1991). Conversely, the self-descriptions of repressors (e.g., Weinberger et al., 1979) indicate a tendency to respond to challenge with increased planning and other indicators of heightened task focus. In addition, several models of self-regulation (e.g., Kuhl, 1985) specify that the inhibition of negative affect that appears to be characteristic of repressors serves the important function of freeing working memory capacity for processing task-relevant stimuli.

If, in fact, the cognitive style associated with repressive–defensiveness serves to facilitate performance and sustain goal-directed behavior in response to challenge, the finding that repressors demonstrate relative left frontal activation is not inconsistent with the approach–withdrawal interpretation of anterior asymmetry. It is important to add that evidence concerning functions subserved by the prefrontal cortex is consistent with the notion that this region may contribute functionally to approach motivation by mediating cognitive processes that facilitate goal-directed behavior in challenging circumstances. For example, there are strong commonalities between conceptions of the prefrontal cortex and social–cognitive models of self-regulation (compare, e.g., Carver & Scheier, 1981; Stuss & Benson, 1986). In addition, dopaminergic systems known to mediate incentive motivation also project to the prefrontal cortex and modulate higher order cognitive processes supported by this region (e.g., Luciana, Depue, Arbisi, & Leon, 1992). Such processes likely contribute to the successful execution of goal-directed behavior in complex environments (Depue & Zald, 1993). This discussion underscores the need for future studies that assess the linkage among frontal asymmetry, repressive–defensiveness, and various cognitive and behavioral components of self-regulation that may be mediated by the prefrontal cortex.

Relations Among Resting Anterior Asymmetry, Repressive–Defensiveness, and Self-Reported Emotion

In our study, BDI and STAI scores failed to significantly predict frontal asymmetry when MC scores were partialed out. In addition, whereas repressors demonstrated relative left frontal activation compared with all comparison groups, the high-anxiety and high-depression groups failed to differ significantly from the low-anxiety and low-depression groups, respectively. By definition, the high-anxiety and high-depression groups reported heightened negative affect relative to the low-anxiety and low-depression groups. Although one should always regard null findings with caution, the absence of asymmetry differences among these groups suggests that resting frontal asymmetry may have a more preferential relation with dimensions of temperament other than self-reported negative affect. As we have argued, it may be more strongly related to styles of regulating emotion and goal-directed behavior.

This conclusion may initially appear to be inconsistent with the evidence that resting anterior EEG asymmetry has predicted self-reported affective responses to films (e.g., Tomarken et al., 1990) and individual differences in self-reported affective traits (Tomarken, Davidson, Wheeler, & Doss, 1992). In such studies, however, relative left anterior activation might have been associated with heightened repressive–defensiveness, regulatory processes (e.g., selective attention to affective cues), or both that accounted for its relation to self-reported emotion. Consistent with this argument, supplementary analyses combining the MC with measures of generalized positive affect and negative affect revealed that the MC contributed significant, unique variance to midfrontal and lateral frontal asymmetry, whereas positive affect and negative affect did not.

We should caution, however, that the majority of subjects in the high-anxiety and high-depression groups of our study likely did not demonstrate clinically significant psychopathology. If we had restricted these groups to individuals who met diagnostic criteria (e.g., major depression, as defined in the revised third edition of the *Diagnostic and Statistical Manual of Mental Disorders*), we might have found more robust differences in frontal asymmetry between such groups and the low-anxiety and low-

depression groups. However, such a finding would not necessarily be inconsistent with our results because high levels of depression and anxiety often appear to be linked to distinct styles of regulating negative affective responses to stressors (e.g., Nolen-Hoeksema, 1991). Once again, this point underscores the need for direct examination of the relations among anterior asymmetry, psychopathological disorders, and the regulation of emotion and goal-directed behavior.

Possible Inconsistencies Between Our Findings and Previous Repressive–Defensiveness Findings

Although our findings appear to be consistent with several prior repressive–defensiveness findings, it could be argued that they are inconsistent with the autonomic and neuroendocrine differences that have been observed between repressors and other groups. For example, if repressive–defensiveness and relative left anterior activation are linked to lowered risk for affective disorders and the inhibition of negative affect, why do repressors demonstrate heightened activation in peripheral stress-sensitive response systems?

Although space constraints preclude a more extensive discussion of this issue, we should note briefly several possible ways to reconcile these apparently inconsistent correlates of a repressive coping style. First, several commentators have argued that a repressive coping style may be characterized by a "functional disconnection syndrome" between the left hemisphere and other cortical or subcortical regions that may more directly mediate autonomic and neuroendocrine components of affective responsivity (e.g., R. J. Davidson, 1984; Schwartz, 1990). Consistent with this reasoning, R. J. Davidson (1984) reported that repressors demonstrated relative deficits in the interhemispheric transfer of negative affective information from the right to the left hemisphere. In light of this latter finding, one implication of our results is that relative left anterior activation, lowered risk for psychopathology, or both may be linked to reduced cross-callosal transfer of information concerning negative affective stimuli, the individual's responses to such stimuli, or both.

A second perspective is the argument that heightened autonomic and endocrine activation among repressors does not reflect distress per se so much as it reflects the mobilization of processes that serve to inhibit distress, facilitate goal-directed behavior, or both. For example, there is evidence that (a) the inhibition of negative affect is associated with heightened autonomic activity (e.g., Gross & Levenson, 1993; Koriat, Melkman, Averill, & Lazarus, 1972) and (b) via peripheral-to-central feedback loops, such physiological changes may contribute functionally to such inhibition (e.g., Dworkin, Filewich, Miller, Craigmyle, & Pickering, 1979).

Finally, the evidence that the MC loads highly on both Self-Deception and Impression Management factors (e.g., Paulhus, 1989) is relevant. Tomaka, Blascovich, and Kelsey (1992) showed that heightened autonomic activity in response to social stressors is more characteristic of high impression managers. Conversely, heightened self-deception appears to be more strongly related to low levels of self-reported depression and to the self-enhancing cognitive style that, we have argued, may be linked to frontal asymmetry (e.g., Paulhus, 1989). Although it is difficult to make firm predictions here because of the generally weak relation between frontal asymmetry and BDI scores in our sample, this evidence does underscore the need for future studies that directly assess the relation between resting anterior asymmetry and the self- and other-deception dimensions.

Additional Cautions and Limitations

Finally, we should briefly note several limitations of our findings. First, because we only assessed women, it is necessary to assess whether our findings are generalizable across both sexes. Second, in the primary analyses assessing between-groups differences in resting anterior asymmetry, we did not include a defensive high-anxious group (i.e., high MC and low STAI scores) because too few subjects met the criteria that have been used by the majority of prior investigators including this fourth group in the design. The inclusion of such a group is necessary to assess more definitively the relation between anterior asymmetry and the repressive–defensive typology. Third, we should note several potential limitations of the reliance on EEG measures to assess regional brain activation. These include less-than-optimal spatial resolution and the reliance on an asymmetry metric rather than measures of absolute activation in particular regions (see Tomarken, Davidson, Wheeler, & Kinney, 1992, for a more extended discussion). Although findings concerning EEG asymmetry and emotion have generally converged with those of studies using other methods (e.g., Robinson et al., 1984), there is clearly a need to assess whether our findings are similarly generalizable.

Summary and Conclusion

Under resting conditions, repressors demonstrated relative left frontal activation relative to comparison groups. These findings suggest that resting frontal asymmetry may be linked to differential access to cognitive and other mechanisms that serve to regulate affective states and self-esteem and that may mediate differential risk for psychopathology. Future research is necessary to more directly assess the relation between resting frontal asymmetry and such self-regulatory mechanisms and the linkage between our findings and conclusions and (a) the approach–withdrawal model of resting anterior asymmetry and (b) repressors' heightened autonomic and neuroendocrine responses to stressors.

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