THE HERITABILITY OF TRAIT FRONTAL EEG ASYMMETRY AND NEGATIVE EMOTIONALITY: SEX DIFFERENCES AND GENETIC NONADDITIVITY

by

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The heritability of personality was addressed using a psychophysiological measure, midfrontal EEG asymmetry, and a paper and pencil measure, the Multidimensional Personality Questionnaire (MPQ). The degree to which midfrontal EEG asymmetry was correlated with the scales of the MPQ was assessed. Relatively greater right midfrontal EEG asymmetry was associated with higher Absorption and Negative Emotionality scores in both the Cz and linked mastoid reference schemes in females, but not in males. Relatively greater right midfrontal EEG asymmetry was also associated with higher Traditionalism and Positive Emotionality scores in the Cz reference scheme in females but not in males. Midfrontal EEG asymmetry was found to be modestly heritable in females, but not in males. Further, each of the scales of the MPQ correlated with midfrontal EEG asymmetry demonstrated moderate to high heritability. A bivariate Cholesky model was used to estimate the heritability of the phenotypic correlations between midfrontal EEG asymmetry and each of the scales with which it was related. Only the midfrontal EEG Asymmetry/Negative Emotionality Cholesky model demonstrated sufficient fit the observed data. According to this model, common genetic effects accounted for approximately 40% of the observed phenotypic correlation between midfrontal EEG asymmetry and Negative Emotionality.
INTRODUCTION

It is increasingly difficult to find traits that are not, at least in part, heritable. This is certainly true of personality measures, examples of which include, among a variety of others, Hysterical Personality (Young, Fenton, & Lader, 1971), 'sleepiness' (Toth, 2001), impulsivity (Willcutt, Pennington, & DeFries, 2000) and the 'big five' dimensions of Neuroticism, Extraversion, Openness, Agreeableness, and Conscientiousness (Jang, Livesley, Angleitner, Riemann, & Vernon, 2002; Jang, Livesley, & Vernon, 1996), the latter cluster of which has even been demonstrated to be heritable in chimpanzees (Weiss, King, & Figueredo, 2000). Although genetic variance clearly accounts for variance in personality constructs, the mechanism by which this occurs is not obvious. Ultimately, a truly comprehensive explanation will involve several levels of analysis (cf., Anderson & Scott, 1999) spanning the molecular, cellular, physiological, psychological, and behavioral levels of analysis. Toward this end, psychophysiological investigations can prove particularly useful, for if genetic effects on personality are mediated through effects on a psychophysiological measure, a useful link may be established between macro and micro level explanations of the genetics of temperament and personality (Anderson & Scott, 1999). A potentially good candidate for such a psychophysiological link is frontal EEG alpha asymmetry, a measure which has itself frequently been linked to many of the partially heritable constructs of personality, temperament, and psychopathology (which may represent extreme personality tendencies,
e.g., Coan & Allen, 2003; Fowles, 1994). The nature of the relationship between frontal EEG asymmetry and measures of personality and psychopathology is itself of great theoretical interest. As a measure, frontal EEG asymmetry is thought to index tendencies to approach (indicated by relatively greater left frontal activity) and withdraw (indicated by relatively less left frontal activity). While it has been suggested that frontal EEG asymmetry in turn indexes a predisposition for certain personality styles and risk for affective disorders (Davidson, 1998), this relationship has not been explored causally (Coan & Allen, 2003a; Coan & Allen, 2003c). While behavior genetic models are also entirely correlational, they suggest plausible causal relationships that may serve as useful heuristics for the study of frontal EEG asymmetry and a variety of personality dimensions, including affective style (Davidson, 1998) and risk for psychopathology. Importantly, EEG spectral patterns have been shown to be heritable (Lykken, Tellegen, & Iacono, 1982; Stassen, Bomben, & Hell, 1998; Stassen, Lykken, & Bomben, 1988), suggesting the possibility that EEG asymmetries may be as well.

The present study therefore sought to 1) provide further support for the link between frontal EEG asymmetry and measures of personality, 2) assess the heritability of frontal EEG asymmetry, and 3) use the techniques of behavior genetic modeling to determine the nature of the relationship between frontal EEG asymmetry and personality, as measured by the Multidimensional Personality Questionnaire (MPQ).
Heritability of Personality as Assessed by the Multidimensional Personality Questionnaire

The Multidimensional Personality Questionnaire (MPQ) was developed with the aim of distilling the myriad common personality constructs extant in the personality literature, and with an emphasis on discriminant validity (Tellegen, Lykken, Bouchard, Wilcox, & et al., 1988). While not constructed with a particular higher-order factor structure in mind, the scales of the MPQ, including those intended to measure Well-Being, Social Potency, Achievement, Social Closeness, Absorption, Stress Reactivity, Alienation, Aggression, Control, Harm Avoidance, and Traditionalism, commonly load onto higher order orthogonal factors very similar to others found in the literature (Tellegen et al., 1988). These factors include Positive Emotionality (often compared to Extroversion), Negative Emotionality (often compared to Neuroticism) and Constraint (Tellegen et al., 1988). Investigations have found that these scales and factors have moderate heritabilities (ranging from .39 to .58) for all of the subscales of the MPQ (Tellegen et al., 1988). Other studies corroborate these findings while suggesting that Alienation and Control may be less heritable in women while Absorption may be more so (Finkel & McGue, 1997). At least one study has estimated the heritability of the Well Being scale to be possibly as high as 80% (Lykken & Tellegen, 1996).
Frontal EEG Asymmetry, Personality and Psychopathology

Asymmetrical patterns of Electroencephalographic (EEG) activity recorded over the frontal lobes at rest have proven useful in the prediction of emotion-related traits and behaviors, including affective disorders (see Coan & Allen, 2003a for a comprehensive review). As a trait measure, frontal EEG asymmetry has been linked to depression (Allen, Iacono, Depue, & Arbisi, 1993; Henriques & Davidson, 1990), anxiety (Wiedemann, Pauli, Dengler, Lutzenberger, Birbaumer, & Buchkremer, 1999), trait aggression (Harmon-Jones & Allen, 1998), trait behavioral activation (Coan & Allen, 2003b; Harmon-Jones & Allen, 1997), shyness (Schmidt, 1999), subjective emotional experience reports (Coan & Allen, in press), general "affective style" (Davidson, 1998) and even immune responsivity (Davidson, Coe, Dolski, & Donzella, 1999). It is thought that at its most basic level trait frontal EEG asymmetry, depending on its direction, indexes tendencies to approach (with activity lateralized to the left) or withdraw (with activity lateralized to the right) from novel or affective stimuli (Coan & Allen, 2003a). This approach/withdrawal continuum has itself been proposed as the most fundamental decision-making dimension employed by any organism (Schneirla, 1959).

A number of studies have related frontal EEG asymmetry to trait personality measures, such as behavioral activation (derived from the Behavioral Activation Scale or BAS, Coan & Allen, 2003b; Harmon-Jones & Allen, 1997; Sutton & Davidson, 1997), defensive coping style (Kline, Allen, & Schwartz, 1998; Tomarken & Davidson, 1994), social inhibition
(Fox, Rubin, Calkins, Marshall, Coplan, Porges, Long, & Stewart, 1995; Schmidt & Fox, 1994) and shyness (Schmidt, Fox, Schulkin, & Gold, 1999). Others have found that resting frontal asymmetries lateralized to the left - indicating a propensity to approach - are related to trait measures of aggression and/or angry behavior (Harmon-Jones & Allen, 1998; Harmon-Jones & Sigelman, 2001). Studies of 'affective style' (e.g., Davidson, 1998) have linked frontal EEG asymmetry to global positive and negative affective responses to emotionally evocative films or slides (Wheeler, Davidson, & Tomarken, 1993). In these studies, participants tend to report the intensity of their specific affective responses (e.g., of fear or sadness) in a manner predicted by their resting frontal EEG asymmetry, and in accord with the approach withdrawal model. For example, individuals with greater right frontal activity at rest tend to respond with more intense levels of negative affect to negatively-valenced film clips, particularly those involving fear (Tomarken, Davidson, & Henriques, 1990), and with more intense levels of positive affect in response to positively valenced film clips (Wheeler et al., 1993). Based on these studies and others it has been argued that frontal EEG asymmetry may underlie certain personality styles, and, indeed, may similarly index a diathesis for certain affective disorders (Davidson, 1998). Several studies support the latter notion. In particular, relatively less left frontal EEG activity - indicating a decreased propensity to approach - has generally but not ubiquitously (Reid, Duke, & Allen, 1998) been shown to correlate with depression (e.g., Baehr, Rosenfeld, Baehr, &
Earnest, 1998; Debener, Beauducel, Nessler, Brocke, Heilemann, & Kayser, 2000; Gotlib, Ranganath, & Rosenfeld, 1998; Henriques & Davidson, 1991; Schaffer, Davidson, & Saron, 1983), seasonal depression (Allen, Iacono, Depue, & Arbisi, 1993), and with risk for depression in those with a history of depression who are not currently depressed (Allen et al., 1993; Gotlib et al., 1998; Henriques & Davidson, 1990). Other studies have associated relatively greater right frontal EEG activity with anxiety (Davidson, Marshall, Tomarken, & Henriques, 2000; Heller, Nitschke, Etienne, & Miller, 1997; Nitschke, Heller, Palmieri, & Miller, 1999).

Many authors have speculated that certain forms of psychopathology may arise out of extremes in the continua of normal personality traits (Depue & Collins, 1999; Fowles, 1994; Kring & Bachorowski, 1999). Moreover, like many aspects of personality, psychopathology tends to be rather heritable. Studies have yielded moderate to high heritability estimates for depression (Brown, 1998; Happonen, Pulkkinen, Kaprio, Van der Meere, Viken, & Rose, 2002; Johansson, Jansson, Linner, Yuan, Pedersen, Blackwood, Barden, Kelsoe, & Schalling, 2001; Johnson, McGue, Gaist, Vaupel, & Christensen, 2002; Kendler & Aggen, 2001; Kendler, Gardner, Neale, & Prescott, 2001; Rice, Harold, & Thaper, 2002), anxiety (Fyer, 1993; Gillespie, Zhu, Heath, Hickie, & Martin, 2000; Kendler, Gardner, & Prescott, 2001; Kendler, Neale, Kessler, Heath, & et al., 1992; Knowles, Mannuzza, & Fyer, 1995; Stein, Jang, & Livesley, 1999; Thapar & McGuffin, 1995), and clinical levels of aggression (Cates, Houston, Vavak, Crawford, & et al., 1993;
The relationship between frontal EEG asymmetry and depression, including risk for depression, is increasingly well documented (e.g., Allen et al., 1993; Gotlib et al., 1998; Henriques & Davidson, 1990; Henriques & Davidson, 1991). Researchers are now investigating the role of frontal EEG asymmetry in anxiety disorders (e.g., Davidson et al., 2000; Heller et al., 1997) and in tendencies toward trait aggression (Harmon-Jones & Allen, 1998). Further, it has been suggested that these disorders represent extremes in personality style (e.g., Kring & Bachorowski, 1999). This is particularly true of personality styles related to positive and negative affect, as evidenced by the frequent use of the general rubric "affective disorders" in describing them (e.g., Davidson, 1998). In sum, the links between frontal EEG asymmetry, psychopathology, and personality warrant the inference that frontal EEG asymmetry may underlie, at least in part, important aspects of personality and risk for psychopathology, and, potentially, that heritable aspects of EEG asymmetry contribute to the heritability of personality and risk for psychopathology. This latter conjecture assumes that frontal EEG asymmetry may in fact be heritable, a possibility that has not been fully investigated.
The Heritability of EEG

A number of characteristics attributable to frontal EEG asymmetry suggest that it may be heritable. This likelihood derives from the fact that frontal EEG asymmetries tend to show fairly high inter-individual variation while also showing relatively low intra-individual variation. Investigations into the psychometric properties of frontal EEG asymmetry have yielded very high internal consistency estimates, with Cronbach’s alphas ranging from .81 to .90 (Tomarken, Davidson, Wheeler, & Kinney, 1992). Test-retest estimates of reliability have shown acceptable to high intra-class correlations, ranging from .44 to .71 across 3 weeks (Tomarken et al., 1992) and .50 to .75 across 5 months (Allen, Urry, Hitt, & Coan, 2003). Similarly, Jones, Field, Davalos and Pickens (1997) found that frontal EEG asymmetry recorded at 3 months of age was highly correlated with the same asymmetry at 3 yrs (r = .66, p < .01). More recently, Hagemann, Naumann, Thayer, & Bartussek (2002) determined that across four different measurement occasions, 60% of the explained variance in EEG asymmetry measures was due to individual differences in a temporally stable latent trait, while 40% of the variance of the asymmetry scores was due to occasion-specific fluctuations.

Heritability of EEG spectra at individual sites. With these trait-like qualities attributable to EEG asymmetries, it is perhaps unsurprising that research on the heritability of a variety of patterns of EEG activity derived from numerous laboratories suggest that EEG spectra are moderately to highly heritable (e.g., Lykken et al., 1982;
In particular, Stassen et al. (1998) arrived at a heritability estimate of $h^2 = 0.75$ using two independent analytic methods - one involving analyses of parent-offspring similarity and the other using an analysis of the difference in within-pair similarity between monozygotic and dizygotic twins.

Heritability of EEG asymmetry. In terms of EEG asymmetry specifically, data are more limited, although two conference reports suggest that it too is heritable (MacDhomhail, Allen, Katsanis, & Iacono, 1999; Rickman & Davidson, 1991). In one of these, frontal EEG asymmetry showed that in females, but not in males, there is greater similarity in monozygotic twins than dizygotic twins (MacDhomhail et al., 1999). In the other, a midparent (mean of father and mother) - child correlation of .47 for frontal EEG asymmetry was reported (Rickman & Davidson, 1991). No published reports have yet assessed the heritability of frontal EEG asymmetry. It is noteworthy, however, that infants of depressed mothers show relatively greater right frontal EEG activity similar to that of their mothers (Dawson, Frey, Panagiotides, Yamada, Hessl, & Osterling, 1999; Field, Fox, Pickens, & Nawrocki, 1995). While these studies are consistent with a genetic perspective, they do not address the question of heritability, per se, as these children share environments with their mothers. Nonetheless, although environmental effects (e.g., interactions with the depressed mother) cannot be ruled out, the possibility of genetic transmission - in the form of a potential genetic vulnerability - is worth consideration.
Thus, EEG asymmetries represent possible indices of physiological processes underlying the phenotypic expression of various aspects of personality and psychopathology. To date it has been shown that personality and psychopathology are heritable, and it appears that EEG asymmetry may be as well. These findings, in concert with those of numerous studies linking frontal EEG asymmetry and personality, suggest the possibility that frontal EEG asymmetry and personality are linked to similar genetic and/or environmental origins, and, indeed, that the heritability of personality may in part be mediated by heritability of resting frontal EEG asymmetry. Behavior genetic models in turn suggest the promise of testing these possibilities.

Study Goals and Hypotheses

For this study, it was hypothesized that certain personality attributes are genetically mediated, and that the genes that act on those personality attributes do so in part via their effects on brain mechanisms indexed by activity asymmetries over the frontal cortex. For the purposes of this study, this hypothesis will be referred to as the "Cholesky hypothesis," after the so-called "Cholesky" models frequently employed to evaluate such relationships (e.g., McGue & Lykken, 1992). In order to evaluate the Cholesky hypothesis, several initial criteria need to be evaluated. First, if the Cholesky hypothesis is true, then it must also be true that frontal EEG asymmetry is correlated with the measures of personality that EEG specific genetic effects are thought to underlie. Thus, it was first
hypothesized that frontal EEG asymmetry will be correlated with some or all measures of personality derived from the MPQ. Within this hypothesis, specific hypotheses include 1) relatively greater left frontal resting EEG activity will predict higher scores on the higher order factor of Positive Emotionality and its derivatives, including Well-Being, Social Potency, Achievement, Social Closeness and Absorption; 2) Relatively greater right frontal activity will predict higher scores on the higher order factors of Negative Emotionality and Constraint, in addition to their constituent scales.

Second, if the Cholesky hypothesis is true, then both frontal EEG asymmetry and those dimensions of personality with which it correlates must to some extent be heritable. Current evidence clearly supports this condition in the case of those dimensions of personality measured by the MPQ (e.g., Finkel & McGue, 1997; Krueger, 2000; Patrick, Curtin, & Tellegen, 2002; Tellegen et al., 1988), and suggests that it is likely in the case of frontal EEG asymmetry (Coan & Allen, 2003a; MacDhomhail et al., 1999; Rickman & Davidson, 1991). Thus, it is also hypothesized that both frontal EEG asymmetry and those personality characteristics with which frontal EEG asymmetry is correlated will be partially determined by genetic influences.
Participants and procedure

EEG and personality measures were collected from 197 twin pairs who participated in the Minnesota Twin/Family Study (see Iacono, Carlson, Taylor, Elkins, & McGue, 1999). Because of missing or bad data in EEG, 36 twin pairs were excluded from analysis. An additional 36 twin pairs had to be excluded from analysis due to incomplete MPQ data. The final sample consisted of 125 twin pairs (250 individual participants) comprised of 59 male twin pairs (MZ = 29, DZ = 30) and 66 female twin pairs (MZ = 30, DZ 36). The mean age for the sample was 19 years. In this sample, zygosity was determined using 3 different procedures: 1) Parents filled out a questionnaire of physical similarity; 2) staff rated the physical similarity of twins; and 3) an algorithm was applied based on ponderal index, cephalic index and finger print ridge count. In the case of discrepancies, blood samples were drawn and analyzed with regard to blood group antigens and protein polymorphisms.

EEG Recordings

Five minutes of resting EEG was recorded simultaneously from each twin pair while they sat with eyes closed in adjacent, identically configured laboratories. EEG was recorded from 5 channels (F3, F4, and Cz referenced to linked mastoids [A1 and A2] and two bipolar derivations T5-01, T6-02), each placed according to the standard 10-20 electroencephalographic lead placement scheme. For these analyses data
were additionally re-referenced off-line using a Cz reference scheme, per the recommendations of Reid et al. (1998), who pointed out that the various reference schemes commonly used for EEG recordings do not correlate well. During the original recording of these EEG data, a half-amplitude high pass filter was set at 1 Hz and the half-amplitude high pass filter was set at 30 Hz. Signals were digitized online at a sampling rate of 128 Hz, at a resolution of 12 bits and subsequently partitioned into 117 two-second epochs, overlapping by 1.5 seconds. EEG impedances were kept below 5 kΩ. Eye movements were recorded diagonally with Ag-AgCl electrodes placed on the outer canthus and above one eye. EOG impedances were kept below 10 kΩ.

For the present analyses, all EEG data were visually screened for artifacts, such as can be caused by body movements, muscle activation, eyeblinks, clipped signals, etc., and epochs containing artifacts were omitted. A Fast Fourier Transform (FFT), using a Hamming window, was performed on each epoch, and the average power spectrum across artifact-free epochs for each minute was subsequently obtained. Total power within the Alpha frequency band (8-13 Hz) in the left and right mid-frontal region (F3 and F4, respectively) was then extracted using both Cz and Linked Mastoids reference schemes, and these values were log transformed using the natural log (to normalize the distributions). A measure of EEG hemispheric asymmetry (right hemisphere compared to left hemisphere) was then be derived using the formula \[\ln(F4) - \ln(F3)\]. The use of this asymmetry metric assumes that cortical alpha power represents the inverse of cortical activity. Thus, higher scores
(scores greater than zero) on the left/right difference score indicate relatively less left alpha power and, it is assumed, relatively greater cortical activity. By contrast, lower scores (scores less than zero) on this scale indicate relatively less right frontal alpha power and relatively greater right frontal activity. In the derivation of this scale, alpha power is assumed to represent the inverse of cortical activity (see Allen, Coan, & Nazarian, 2003, for a thorough review of this assumption).

The Multidimensional Personality Questionnaire

The Multidimensional Personality Questionnaire (MPQ) was used to assess relationships between frontal EEG asymmetry and personality. These scales are intended to measure Well Being, Social Potency, Achievement, Social Closeness, Stress Reactivity, Alienation, Aggression, Control, Harm Avoidance, Traditionalism and Absorption. The MPQ also includes three higher order factors similar to many found in the literature. These include Positive Emotionality (related to Extroversion), Negative Emotionality (related to Neuroticism) and Constraint. The scales of the MPQ, as well as the higher order factors associated with it, show relatively low intercorrelations, consistent with its emphasis on discriminant validity.
RESULTS

Results reported here are repeated across two different reference montages. Although rational arguments have been levied in favor of one or another reference scheme (Hagemann, Naumann, & Thayer, 2001; Reid et al., 1998), it remains a somewhat empirical question which reference scheme has the greatest predictive validity with respect to motivational and emotional constructs, as well as in estimating heritability. For this study, all analyses requiring tests of statistical significance will include data derived from both reference schemes. Generally, results that replicate across reference schemes should be considered the most generalizable, being less likely to reflect only the reference-specific "method" variance (cf., Campbell & Fiske, 1959). By contrast, findings that are statistically significant in only one reference montage will not be considered generalizable.

Also, because an earlier conference report (MacDhomhail et al., 1999) suggested a sex difference in EEG heritability, all analyses will be conducted separately for males and females. These subjects were run as an earlier subsample of the sample considered here. In assessing the internal consistency of the EEG data, each of the five minutes of recorded EEG were used as scale "items." With this approach, internal consistency estimates of mid-frontal EEG asymmetry in the Cz and LM reference schemes were .97 and .95, respectively. Mid-frontal EEG asymmetry means ranged from between .002 to .046, depending upon zygosity, sex and reference scheme (see Table 1). In 2X2 (zygosity by sex) ANOVAs conducted for each reference scheme, no mean
In order to determine whether mid-frontal EEG asymmetry was related to any of the scales of the MPQ, a series of zero order correlations was calculated. The results indicated no such relationships in males, but a few in females (see Table 2). In particular, in females, mid-frontal EEG asymmetry was significantly correlated with Absorption ($r_{cz} = -.24; r_{lm} = -.20$) and Negative Emotionality ($r_{cz} = -.19; r_{lm} = -.23$). These negative correlations indicate that individuals who score highly on the Absorption and Negative Emotionality scales were more right mid-frontally active at rest. It is unclear what this means in the case of Absorption, but the correlation with negative emotionality is in line with prevailing theories of the affective components of behavior that frontal EEG asymmetries are thought to index (cf., Coan & Allen, 2003a).

Interestingly, mid-frontal EEG asymmetry using the Cz reference scheme is also negatively correlated with both Traditionalism and Positive Emotionality. While it is worth noting these correlations, it is also apparent that they do not replicate significantly across reference schemes and are thus of dubious generalizability. Such lack of replication suggests that these relationships are not as likely to be

Table 1. Means and standard deviations for mid-frontal EEG asymmetry, by zygosity, sex and reference scheme.

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<th>Females (N = 132)</th>
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<tr>
<td></td>
<td>Cz</td>
<td>LM</td>
</tr>
<tr>
<td>Monozygotic</td>
<td>.03 (.14)</td>
<td>.01 (.06)</td>
</tr>
<tr>
<td>Dizygotic</td>
<td>.05 (.16)</td>
<td>.01 (.07)</td>
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Table 2. Zero order correlations between mid-frontal EEG asymmetry and the primary and higher order scales of the MPQ.

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<td></td>
<td>Cz</td>
<td>LM</td>
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<td>Constraint</td>
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</table>

*p < .05; **p < .01

Positive correlations indicate that higher scores on the scale are associated with relatively greater left mid-frontal activity at rest. Negative correlations indicate that higher scores on the scale are associated with relatively greater right mid-frontal activity at rest.

replicated in future studies. Nevertheless, it is also apparent that the correlations associated with them are not significantly different from those of the other correlated scales. Ultimately, all correlated scales will be considered in subsequent analyses.

Heritability of Frontal EEG Asymmetry

Falconer's estimate. Two statistical methods were used to assess the heritability of frontal EEG asymmetry. The first of these was simply to compute one-way random intra-class correlations among monozygotic (MZ) twins and di-zygotic (DZ) twins separately for use in
calculating a rough estimate of heritability using the Falconer's estimate, \( h^2 = 2(r_{mz} - r_{dz}) \). On occasion, intra-class correlations are estimated to be less than zero. In such instances, the correlation is generally assumed to be zero, and this assumption was made in the analyses discussed here. With this in mind, it was impossible to estimate EEG asymmetry heritability in males using the Cz reference scheme, as both intraclass correlations (MZ and DZ twins) were negative and assumed to be zero. Using the LM reference, intra-class correlations for MZ and DZ male twins were .11 and 0, respectively, yielding a Falconer's estimate of .21. In females, using the Cz reference scheme, intraclass correlations for MZ and DZ twins were .24 and 0, respectively, yielding a Falconer's estimate of .49. Using the LM reference, the intraclass correlations for MZ and DZ twins were .13 and 0 respectively, yielding a Falconer's estimate of .26.

**Latent Variable Modeling.** In addition to Falconer's estimate, a Maximum Likelihood (ML) latent variable modeling approach was applied, using path coefficient estimates to estimate this same heritability parameter mentioned above. In all cases, Mx (Neale, 1997), a statistical software package for latent variable modeling commonly used in behavior genetics research, was used to estimate path coefficients, variance components and model fit statistics.

Because MZ twin intraclass correlations estimated for EEG were in all but one case more than double the magnitude of those estimated for DZ twins, it is likely that the EEG asymmetries are influenced by
Table 3. Fit statistics for ACE and ADE models, by sex and reference scheme.

<table>
<thead>
<tr>
<th></th>
<th>$\chi^2$</th>
<th>df</th>
<th>P-value</th>
<th>AIC</th>
<th>RMSEA</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Males</strong> (N=118)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cz</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$a^2+c^2+e^2$</td>
<td>8.71</td>
<td>3</td>
<td>.03</td>
<td>2.71</td>
<td>.18</td>
</tr>
<tr>
<td>$a^2+d^2+e^2$</td>
<td>8.71</td>
<td>3</td>
<td>.03</td>
<td>2.71</td>
<td>.18</td>
</tr>
<tr>
<td>LM</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$a^2+c^2+e^2$</td>
<td>8.84</td>
<td>3</td>
<td>.03</td>
<td>2.84</td>
<td>.20</td>
</tr>
<tr>
<td>$a^2+d^2+e^2$</td>
<td>8.84</td>
<td>3</td>
<td>.03</td>
<td>2.84</td>
<td>.20</td>
</tr>
<tr>
<td><strong>Females</strong> (N=132)</td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Cz</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$a^2+c^2+e^2$</td>
<td>2.71</td>
<td>3</td>
<td>.44</td>
<td>-3.29</td>
<td>.05</td>
</tr>
<tr>
<td>$a^2+d^2+e^2$</td>
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<td>3</td>
<td>.56</td>
<td>-3.92</td>
<td>.04</td>
</tr>
<tr>
<td>LM</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$a^2+c^2+e^2$</td>
<td>2.80</td>
<td>3</td>
<td>.43</td>
<td>-3.24</td>
<td>.06</td>
</tr>
<tr>
<td>$a^2+d^2+e^2$</td>
<td>2.54</td>
<td>3</td>
<td>.47</td>
<td>-3.46</td>
<td>.06</td>
</tr>
</tbody>
</table>

In this table, $a^2$ represents additive genetic variance, $c^2$ represents common environmental variance, $d^2$ represents non-additive genetic variance, and $e^2$ = unique environmental variance.

Nonadditive genetic effects as much or more so than by additive ones'.

With this in mind, covariance matrices, representing MZ and DZ twins separately, were fit to an ACE and an ADE model. In ACE models, three components of variance thought to influence a given variable are estimated: an 'A' factor, representing additive genetic influences, a

---

1 Additive genetic effects occur when alleles simply 'add up' in the ultimate determination of some trait predisposition or behavior. Nonadditive effects occur when the effects of one or more alleles are dependent upon the presence or absence of other alleles. Such nonadditive effects can result from genetic dominance, or from epistasis. Genetic dominance refers to situations where on allele appears to dominate another at some specific locus. Epistasis refers to the nonadditive interaction of alleles at different loci. In either type of nonadditive genetic influence, MZ twins will be highly similar, whereas DZ twins may be disproportionately dissimilar. Estimates of heritability derived from a combination of additive and nonadditive genetic effects are referred to as estimates of broad heritability.
'C' factor, representing the effects of common environment between siblings, and an 'E' factor representing unique environmental influences. In an ADE model the A and the E factors remain, but the C factor is replaced with a 'D' factor representing non-additive variance. Table 3 shows fit statistics for these models of genetic and environmental influences on mid-frontal EEG asymmetry. Interestingly, male twin data did not fit either model in either reference scheme. Thus, heritability estimates for male twins are not reported here. By contrast, all models fit well to the female twin data, $\chi^2 = 2.80$ (df = 3), $p = .43$, AIC = -3.24, and RMSEA = .06. According to the ACE model, additive genetic effects account for 14% and 4% of the trait variance in Cz and LM derived mid-frontal EEG asymmetry, respectively. By contrast, according to the ADE model, additive genetic effects account for approximately zero percent of the variance in both reference

---

2 In general, latent variable model 'fit' is determined by the degree to which observed data deviates from the specified model. Thus, when the $\chi^2$ statistic is large and statistically significant, the model is thought to fit poorly, because the observed data are significantly different from the model specification. In addition to the $\chi^2$ statistic, two other useful fit statistics are reported here. These are Akaike's Information Criterion (AIC) and the Root Mean Square Error of Approximation (RMSEA). The AIC statistic includes a consideration of the number of unknown parameter estimates in its calculation, providing by extension a consideration of model parsimony. Relatively poor model fit is indicated by higher AIC values while lower values indicate relatively good model fit (Loehlin, 1998). The RMSEA statistic is thought to be relatively independent of sample size by virtue of being a population-based index. It, too, is sensitive to model parsimony. A perfect model fit is indicated by an RMSEA of zero. An RMSEA of less than .05 is thought to indicate an 'excellent' fit to the data, an RMSEA of less than .10 is thought to indicate 'good' fit, and 'one would not want to employ' a model with an RMSEA greater than .10 (Loehlin, 1998, pp. 76-77)
schemes, while nonadditive genetic effects account for approximately 22% and 9% of the trait variance in Cz and Lm derived mid-frontal EEG asymmetry, respectively. Ultimately, the Cz reference ADE model (see Figure 1) was judged the most promising for additional bivariate analyses of those compared. This was because 1) the estimate of heritability increased from the additive ACE to the nonadditive ADE Cz reference scheme models and 2) the Cz ADE model fits nominally (though not significantly statistically) better than its LM counterpart. (This last criterion is relevant only in that it is not inconsistent with a preference for the Cz model.) Thus, in subsequent bivariate genetic analyses, only female data from the Cz reference scheme will be used.

As an additional check on the data reported here, alpha power heritability was estimated for each hemisphere separately (left mid-frontal or F3 and right mid-frontal or F4). Because past research (Stassen et al., 1998; Stassen et al., 1988) has provided very little evidence of effects of common environment on EEG power spectra, and because an ADE model was the optimal model for EEG asymmetry, an ADE model was fit to mid-frontal alpha power for both F3 and F4 separately, independent of sex (both male and female data were included in this model). Both models fit very well, $\chi^2 = 3.82$ (df = 3), $p = .28$, AIC = 2.78, and RMSEA = .06. Further, variance attributable to additive genetic, nonadditive genetic, and nonshared environment were in each case estimated to be .75, 0.00 and .25, respectively. These estimates are virtually identical to those reported elsewhere in the
Figure 1. Heritability of Midfrontal EEG Asymmetry. ADE model path coefficients and variance proportions for mid-frontal EEG asymmetry in females, using the Cz reference scheme. A represents additive genetic effects, D represents nonadditive genetic effects and E represents non-shared environmental effects. This model demonstrated an excellent fit to the observed data, with $\chi^2 = 2.08$ (df = 3), $p = .56$, AIC = -3.92, RMSEA = .04.

![Diagram of ADE model](image)

<table>
<thead>
<tr>
<th>Proportions of Variance</th>
</tr>
</thead>
<tbody>
<tr>
<td>A = .00</td>
</tr>
<tr>
<td>D = .22</td>
</tr>
<tr>
<td>E = .78</td>
</tr>
</tbody>
</table>

literature (Stassen et al., 1998; Stassen et al., 1988). Such replication lends credibility to the data underlying asymmetry estimates reported above.

The Heritability of Absorption, Traditionalism, Negative Emotionality, and Positive Emotionality

To establish whether Positive Emotionality, Negative Emotionality, Traditionalism and Absorption are good candidates for a test of the Cholesky hypothesis, it is necessary that these variables
demonstrate heritability in addition to their established correlational relationship with mid-frontal EEG asymmetry. To this end, and keeping in mind that the relationship between these personality variables and mid-frontal EEG asymmetry was found only in females, covariance matrices among MZ and DZ female twins were for both variables fit to ACE and ADE models, just as with EEG asymmetry. Absorption data did not fit to the models outlined.

In the case of Traditionalism, both the ACE and ADE models demonstrated excellent fits to the observed data, $\chi^2 = 3.34$ (df = 3), $p = .34$, AIC = -2.67, and RMSEA = .04. According to the ACE model, additive genetic effects accounted for approximately 25% of the trait variance, while common environmental effects accounted for the 29% and unique environmental effects accounted for 46% (see figure 2). By contrast, in the ADE model, additive genetic effects accounted for approximately 56% of the trait variance, unique environmental effects accounted for 44%; nonadditive genetic effects accounted for no variance at all.

In the case of Positive Emotionality, the ACE model did not fit, but the ADE model did fit, $\chi^2 = 4.32$ (df = 3), $p = .23$, AIC = -1.68, and RMSEA < .10. According to this ADE model, additive genetic effects accounted for approximately 21% of the trait variance in Positive Emotionality, nonadditive genetic variance accounted for approximately 64% and unique environmental effects accounted for approximately 15% (see Figure 3).
Figure 2, Heritability of Traditionalism. ACE model path coefficients and variance proportions for Traditionalism in females, using the Cz reference scheme. A represents additive genetic effects, C represents common environmental effects and E represents non-shared environmental effects. This model demonstrated an excellent fit to the observed data, with $\chi^2 = 2.57$ ($df = 4$), $p = .46$, $AIC = -3.41$, $RMSEA = .03$.

In the case of Negative Emotionality, the ADE model did not fit, and the ACE model fit well, $\chi^2 = 3.07$ ($df = 3$), $p = .38$, $AIC < -2.93$, and $RMSEA < .07$. According to the ACE model, however, additive genetic effects accounted for approximately 41% of the trait variance in Negative Emotionality, while unique environmental effects accounted for the remaining variance; common environmental effects accounted for no variance at all. Thus, data from this variable were fit to an AE model. The AE Negative Emotionality model demonstrated a substantial
Figure 3, Heritability of Positive Emotionality. ADE model path coefficients and variance proportions for Positive Emotionality in females, using the Cz reference scheme. $A$ represents additive genetic effects, $D$ represents nonadditive genetic effects and $E$ represents non-shared environmental effects. This model demonstrated a good fit to the observed data, with $\chi^2 = 4.32$ (df = 3), $p = .23$, AIC < -1.68, and RMSEA < .10.

Improvement in model fit, $\chi^2 = 4.43$ (df = 4), $p = .35$, AIC = -3.57, RMSEA = .05 (see Figure 4). Thus, it appears that Traditionalism, Positive Emotionality and Negative Emotionality are each candidates for testing the Cholesky hypothesis with mid-frontal EEG asymmetry.

The Cholesky Hypothesis

*Positive Emotionality and Traditionalism.* The phenotypic correlations between midfrontal EEG asymmetry (derived using a Cz
Figure 4, Heritability of Negative Emotionality. AE model path coefficients and variance proportions for Negative Emotionality in females, using the Cz reference scheme. A represents additive genetic effects, and E represents non-shared environmental effects. This model demonstrated an excellent fit to the observed data, with $\chi^2 = 4.43$ ($df = 4$), $p = .35$, AIC = -3.57, RMSEA = .05.

Reference) and both Positive Emotionality and Traditionalism scores were modeled in terms of their genetic and environmental constituents using Cholesky models. In these models, genetic and environmental effects on each of two variables, as well as the relationships between those two variables, can be estimated. Unfortunately, these parameters could not be estimated for frontal EEG asymmetry in the cases of Traditionalism and Positive Emotionality, because in neither case did the bivariate Cholesky model fit the observed data. Models attempted included the ACE, ADE, AE and DE models. Ultimately, the relationships between midfrontal EEG asymmetry and both Traditionalism and Positive Emotionality remain uncertain, both in terms of their reliable phenotypic manifestations (because they did not replicate across reference schemes) and in terms of their bivariate heritabilities.
Negative Emotionality. A test of the Cholesky hypothesis, that the phenotypic correlation between two trait variables is attributable to shared genetic influences, presents some logical difficulties at the outset in the case of mid-frontal EEG asymmetry and Negative Emotionality. A significant phenotypic correlation between these two variables was present in both reference schemes, suggesting that the relationship is reliable, but latent variable models of each individual trait variable are inconsistent in terms of the estimated sources of genetic variance. In the case of mid-frontal EEG asymmetry, genetic effects appear to be primarily if not entirely nonadditive; in the case of Negative Emotionality, genetic effects appear to be almost entirely additive in nature. Both models attribute sizable effects to unique environmental influences and virtually no effects to common environment. Thus, the ADE bivariate model appears to be the most theoretically tenable. Bivariate covariance matrices for both MZ and DZ twins were indeed fit to the ADE model, which demonstrated an excellent fit to the observed data, $\chi^2 = 11.47$ (df = 11), $p = .41$, AIC = -10.53, and RMSEA = .04. According to this model (Figure 5), the proportion of variance attributable to additive genetic effects ($a^2_a$) was .01, while the proportion of variance attributable to nonadditive genetic effects ($d^2_a$) was .21, resulting in a total heritability of mid-frontal EEG asymmetry ($h^2_a$) of .22. The heritability of Negative Emotionality is comprised of additive genetic influences shared with mid-frontal EEG asymmetry ($a^2_{ac} = .39$), additive genetic influences specific to Negative Emotionality ($a^2_{as} = .00$), nonadditive genetic
influences shared with mid-frontal EEG asymmetry ($d^2_{nc} = .02$), and nonadditive genetic influences specific to Negative Emotionality ($d^2_{ns} = .00$), resulting in a total Negative Emotionality heritability ($h^2_n$) of .41. Further, the proportion of variance in trait mid-frontal EEG asymmetry attributable to nonshared environmental effects ($e^2_{e}$) was .78. The same proportion for Negative Emotionality was comprised of nonshared environmental effects common to mid-frontal EEG asymmetry ($e^2_{nc} = .04$), and nonshared environmental effects specific to Negative Emotionality ($e^2_{ns} = .55$), summing to a total estimate of nonshared environmental influence ($e^2_{n}$) of .59.

From these indices, it is further possible to estimate a number of additionally useful proportions (cf., Jockin, McGue, & Lykken, 1996). The proportion of the heritability of Negative Emotionality that can be explained by the additive genetic factors common to mid-frontal EEG asymmetry ($r^2_A$) can be estimated by $(a_{nc} / h_n)^2$. Similarly, the proportion due to nonadditive genetic factors common to mid-frontal EEG asymmetry ($r^2_D$) can be estimated by $(d_{nc} / h_n)^2$, and the proportion of variance in nonshared environment that can be explained by nonshared environmental effects common to mid-frontal EEG asymmetry ($r^2_E$) can be estimated by $(e_{nc} / e_n)^2$. Following these calculations, $r^2_A$ was estimated to be .94, $r^2_D$ was estimated to be .05 and $r^2_E$ was estimated to be .07.

It is further possible to calculate an estimate of the proportion of the covariance between two traits attributable to genetic contributions (Jockin et al., 1996). This proportion of bivariate
Figure 5, Midfrontal EEG Asymmetry/Negative Emotionality Cholesky Model. ADE model path coefficients and variance proportions for Negative Emotionality in females, using the Cz reference scheme. A represents additive genetic effects, D represents nonadditive genetic effects and E represents non-shared environmental effects. For the path coefficients, subscript e represents effects specific to mid-frontal EEG asymmetry, subscript nc represents effects on Negative Emotionality from sources shared with mid-frontal EEG asymmetry, subscript ns represents effects specific to Negative Emotionality, and subscript n represents the total effects (specific and common) on Negative Emotionality. From these estimates it was possible to calculate $r^2_A$, the proportion of heritability in Negative Emotionality attributable to additive genetic factors also underlying mid-frontal EEG asymmetry, $r^2_D$, the proportion attributable to nonadditive genetic factors, and $r^2_E$, the proportion of unique environmental influences on Negative Emotionality attributable to unique environmental influences also underlying mid-frontal EEG asymmetry. It was also possible to calculate $h^2_E$, the proportion of covariance between mid-frontal EEG asymmetry and Negative Emotionality attributable to shared genetic (both additive and nonadditive) influences. This model demonstrated excellent fit to the observed data, with $\chi^2 = 11.47$ ($df = 11$), $p = .41$, AIC = -10.53, RMSEA = .04.

Proportions of Variance

Additive genetic effects

\[
\begin{align*}
    a^2_e & = .01 \\
    a^2_{nc} & = .39 \\
    a^2_{ns} & = .00 \Rightarrow a^2_n = .39
\end{align*}
\]

Nonadditive genetic effects

\[
\begin{align*}
    d^2_e & = .21 \\
    d^2_{nc} & = .02 \\
    d^2_{ns} & = .00 \Rightarrow d^2_n = .02
\end{align*}
\]

Unique environmental effects

\[
\begin{align*}
    e^2_e & = .78 \\
    e^2_{nc} & = .04 \Rightarrow e^2_n = .59
\end{align*}
\]

Correlations

\[
\begin{align*}
    r^2_A & = .94 \quad h^2_A = .22 \\
    r^2_D & = .05 \quad h^2_D = .41 \\
    r^2_E & = .07 \quad h^2_E = .42
\end{align*}
\]
heritability can be estimated by the equation specified in Figure 6. With this equation, a bivariate heritability ($h^2_b$) again, the proportion of the covariance between mid-frontal EEG asymmetry and Negative Emotionality attributable to genetic factors) estimate of .42 was obtained. This estimate suggests that 42% of the relationship between mid-frontal EEG asymmetry and Negative Emotionality is due to underlying genetic factors.

Figure 6, Bivariate Heritability Equation. Equation used for determining the bivariate heritability of the phenotypic relationship between midfrontal EEG asymmetry and Negative Emotionality.

$$h^2_b = \frac{|a_e a_{nc}| + |d_e d_{nc}|}{|a_e a_{nc}| + |d_e d_{nc}| + |e_e e_{nc}|}$$
DISCUSSION

This is the first study to estimate the relative contributions of genetic and environmental influences on trait variance in mid-frontal EEG asymmetry. Further, it is the first study to estimate the degree to which the phenotypic relationship between mid-frontal EEG asymmetry and a paper and pencil personality test, in this case Negative Emotionality as measured by the MPQ, is due to common genetic versus environmental influences. Of initial interest was the fact that males and females appear to differ in the degree to which mid-frontal EEG asymmetry is heritable, with males showing virtually zero heritability and females showing modest heritability estimates (in the range of 20%). Of further interest was that female mid-frontal EEG asymmetry heritability was best accounted for by nonadditive genetic effects, a fact suggested also by the large discrepancies between mono and dizygotic twin intraclass correlation coefficients. What follows is a discussion of the sex differences in the heritability of mid-frontal EEG asymmetry, the relative contributions of additivity and nonadditivity to trait variance in frontal EEG asymmetry, and the interpretation of the phenotypic relationship between mid-frontal EEG asymmetry and Negative Emotionality

Sex Differences in the Heritability of Frontal EEG asymmetry

While no clear hypothesis was stated with regard to sex differences in the heritability of mid-frontal EEG asymmetry, such a difference was not entirely unanticipated. A previous conference
report (MacDhomhail et al., 1999) noted a similar sex difference in a portion of the sample reported on here. The critical question regards the reason that such a sex difference might exist. One possibility is that the difference is the result of epigenetic effects, where the same genes are impacting males and females in different ways or in differing magnitudes. Such effects can result from the interaction between certain genes and sex hormones, for example, or from sex-specific chromosomal differences.

Another possibility is that the genes underlying female mid-frontal EEG asymmetry heritability are expressing themselves no differently than they do in males, but that unique environmental pressures are dominating male trait mid-frontal EEG variance in ways that are relatively unrelated to the expression of mid-frontal EEG asymmetry-relevant genes. While the data under consideration in this study are not sufficient for supporting or refuting any of these possibilities, prevailing stereotypes of social attitudes regarding male and female emotional behavior might be consistent with the later explanation. For example, male emotional behavior is often thought to be more constrained than female emotional behavior, particularly with regard to the emotions associated with right mid-frontal EEG activity, such as fear and sadness (Fabes & Martin, 1991). Unfortunately, this possibility is based on what may be a popular stereotype with little empirical support, and in any case may involve a great deal of complexity in determining the particular aspects of emotion, emotion regulation and emotional personality that may be implicated in reliable
sex differences. For example, though some have reported sex differences in subjective experience reports (e.g., Barrett, Lane, Sechrest, & Schwartz, 2000), the literature on more objectively measured emotional behavior has produced inconsistent results, with some studies finding few or no sex differences (Barrett, Robin, Pietromonaco, & Eyssell, 1998; Gross & Levenson, 1993) and others finding sex differences consistent with many prevailing stereotypes (van der Bolt & Tellegen, 1995). For example, van der Bolt and Tellegen (1995) found evidence that females are generally more "open" to dysphoric emotions than males are. It may be that this difference reflects a cultural (and by extension, environmental) constraint, whereby males are influenced to regulate their emotions more according to environmental cues than by temperamental emotional tendencies. This could, in theory, limit the degree to which male trait mid-frontal EEG asymmetry is controlled by genetic influences. Such questions await further exploration and study.

**Mid-Frontal EEG asymmetry and the MPQ**

In this study, a relationship between mid-frontal trait EEG asymmetry and the Negative Emotionality scale of the MPQ was identified and explored. Negative Emotionality is thought to be high in individuals who have a low general threshold for negative emotions. Such individuals are thought to be highly sensitive to stress, and are likely to view the world as a dangerous or threatening place (Tellegen, 1985). It is interesting that this relationship was significant only
in females in the present sample. In past studies, relatively greater right frontal EEG asymmetry has been associated with Negative Affectivity as measured by the Positive and Negative Affect Schedule (PANAS) in females (Tomarken, Davidson, Wheeler, & Doss, 1992). Interestingly, in tests of this association in males, relatively greater left frontal EEG asymmetry was associated with greater Positive Affectivity, but relatively greater right frontal EEG activity was not associated with greater Negative Affectivity (Jacobs & Snyder, 1996).

In light of past studies, and given the predictions of the approach/withdrawal model of anterior EEG asymmetry and emotion, the association between mid-frontal EEG asymmetry and Negative Emotionality is readily interpretable. Less certain is the association with Absorption, Positive Emotionality and Traditionalism. Because Absorption loads strongly on Positive Emotionality (e.g., Krueger, 2000), it may be tempting to attribute the weak association between mid-frontal EEG asymmetry and Positive Emotionality primarily to the more robust association with Absorption. However, a perusal of the remaining constituents of Positive Emotionality suggests that both Social Potency and Achievement are weakly associated with mid-frontal EEG asymmetry as well. What is difficult about these relationships, weak as they are, is that they are the opposite of what would be predicted by the approach/withdrawal model, and indeed run counter to a large portion of the literature on anterior EEG asymmetry and personality. The same relationship is weakly evident in the case of Traditionalism. While an association between relatively greater right
mid-frontal EEG asymmetry and Traditionalism is not immediately obvious, it does not run counter to the approach/withdrawal model the way absorption and positive emotionality do. Indeed, traditionalism has been associated with the higher order factor of Constraint, and is related to tendencies to control one's environment and avoid harm. Such characteristics are not inconsistent with the trait withdrawal orientation that relatively greater right anterior activity is thought to index.

Nevertheless, the unanticipated relationships between right frontal activity, Absorption and Positive Emotionality merit further consideration. It is possible that these relationships reflect similar sex differences in anterior EEG asymmetry identified elsewhere. For example, in past research, females who are relatively more left frontally active at rest are more temperamentally defensive, while the opposite pattern is true of males (Kline et al., 1998). Adding to the complexity of this sex difference, other studies have found that the female relationship between left frontal activity and high defensiveness is particularly strong when those females are in the presence of males (Kline, Blackhart, & Joiner, 2002). Further, women have been found to differ in men in the pattern of anterior activity associated with negative moods resulting from EEG hookup procedures. In men, individuals who responded to EEG hookup with more negative mood were more right frontally active at rest. The opposite was true of women, who were more left frontal active if they responded to EEG hookup with more negative mood (Blackhart, Kline, Donohue, LaRowe, &
Joiner, 2002). In a study of state changes in frontal EEG asymmetry during different motivational conditions, men were found to become more left frontally active during moments of high expectation, while women were found to become more left frontally active during moments of low expectation (Miller & Tomarken, 2001). It may be critical that in the latter study, the proper interpretation is that "relative left midfrontal asymmetry indexed increases in the likelihood of success in males but [...] decreases in relative left midfrontal asymmetry were related to greater chances of successful outcomes in females" (Miller & Tomarken, 2001, p. 509). These researchers speculated that such sex differences may reflect the interaction between implicated brain regions and different sex hormones or may be related to other sex differences, such as differences in tendencies toward emotional rumination and emotional coping styles (e.g., Finkel & McGue, 1997).

Particularly interesting is the possibility that the sex differences in the relationship between midfrontal EEG asymmetry and these personality characteristics are mediated by the same factors that underlie the sex difference in midfrontal EEG asymmetry heritability. As new research replicates these sex differences and uncovers new ones, it will be increasingly important to identify these mediators.

Interpreting the Cholesky Model. It appears that the relationship between frontal EEG asymmetry and negative emotionality is 1) small, but reliable, 2) approximately 40% attributable to common genetic effects and 3) mediated through common additive genetic effects. This last point is troublesome because additive genetic
effects account for very little overall trait variance in mid-frontal EEG asymmetry per se (probably less than 1%). On the other hand, this may reflect the very small effect size of the phenotypic relationship between midfrontal EEG asymmetry and NEM, which is approximately .04 in the Cz reference scheme. Thus, the proper way to interpret the Cholesky Model may be that common genetic effects exert little influence on mid-frontal EEG per se, but great influence on NEM, and in any case largely account for the relationship between mid-frontal EEG asymmetry and NEM.

Methodological Constraints

This study represents an important "first look" at the heritability of asymmetries in anterior brain activity. Because with these data it was also possible to investigate the heritability of the relationship between midfrontal EEG asymmetry and affective personality measures, it is also of important heuristic value to the field. Future investigators should embed theoretically grounded measures into research of this type in an effort to replicate and extend the field's understanding of sex differences in frontal EEG asymmetry as well as the variables that mediate and moderate those differences. In any case, methodological constraints limit the conclusions that can be made from this study.

The Generalizability of Trait EEG Asymmetry Measurement. First, while frontal EEG asymmetry always shows high internal consistency, it's test-retest reliability has been modest (e.g., Tomarken, Davidson,
Wheeler, & Kinney, 1992). Generalizability analyses of frontal EEG asymmetry suggest that trait measures are highly susceptible to interference attributable to state and occasion sources of variance (Coan & Allen, 2003c). These researchers estimated that this interference could alone account for much of the inconsistency in the literature on frontal EEG asymmetry, and used the Spearman-Browne prophecy formula to estimate that trait variance in frontal EEG asymmetry would be optimally estimated from four separate occasions of measurement (Coan & Allen, 2003c). It is possible that if such a procedure had been used in this case the pattern of heritability, as well as the pattern of relationships with the scales of the MPQ, could look different. In particular, more reliable measurement of trait variance in frontal EEG asymmetry could result in greater heritability estimates and a larger effect size in the relationships between frontal EEG asymmetry and the MPQ scales. This might be particularly true in the case of heritability estimates, since non-trait variance interfering with trait measurement would count as unshared environmental effects.

Another consideration concerns the generalizability of state changes in frontal EEG asymmetry in response to emotional stimuli (e.g., Coan, Allen, & Harmon-Jones, 2001). For example, Coan and Allen (2003c) estimated the generalizability of such state manipulations to be approximately .97, which is extraordinarily high for most psychophysiological measures associated with emotion. In future studies of the heritability of frontal EEG asymmetry, it will be
important to identify the heritability of such state changes, as well as the heritability of the interaction between such state changes and resting trait levels.

The need for more EEG sites. Another limitation of the current study concerns the number of EEG sites from which asymmetry data could be derived. In this case, only the midfrontal region could be assessed, but in previous studies, anterior asymmetry effects are also seen in the lateral frontal, frontal-temporal-central, anterior temporal and frontal-central regions of the scalp. It is possible that the heritability of such anterior asymmetries varies somewhat by particular region. Future researchers should endeavor to acquire data from all anterior regions implicated in this literature.

The acquisition of EEG data from additional sites would confer other advantages as well. For example, it will be important in future studies to compare the heritability of frontal EEG asymmetries to the heritability of asymmetries elsewhere on the head. This will be particularly important with regard to the relationships between such asymmetries and measures of emotion and personality. Further, the acquisition of additional sites will make it possible to estimate asymmetry data using yet another reference scheme, such as an average reference. The addition of such a reference could shed greater light on the generalizability of the heritability estimates derived here, as well as the estimates of the magnitude of the relationship between frontal EEG asymmetry and Negative Emotionality.
Sample size. Finally, a potential limitation of the current study regards the size of the sample involved. By comparison with other studies of frontal EEG asymmetry, the size of the current sample is more than adequate. Indeed, it is substantially larger than most. The models used here in estimating heritabilities, however, ordinarily include larger samples, often numbering in the thousands of participants. This is particularly true when estimating more complex models, such as the Cholesky model described here, in part because the probability of rejecting a complex latent variable model that is in fact wrong is partially a function of statistical power. That is, it is potentially easier to fit a model (according to the $\chi^2$ statistic) with a relatively small sample size. Fortunately, the RMSEA estimate, included as a fit statistic in all models reported here, is, by virtue of being a population based estimate, less sensitive to sample size than other fit indices. In sum, while it would be ideal to have analyzed a larger sample of twins in this case, that ideal does not render the current analyses by default incorrect or untenable (see Loehlin, 1998).

Concluding Remarks

Ultimately, such methodological constraints should inspire caution in interpreting the results reported here. Nevertheless, this study provides a valuable first look at the relative contributions of genetic and environmental effects on the development of trait frontal EEG asymmetries. The relationships reported here between frontal EEG
asymmetry and negative emotionality, as well as the sex differences identified here, should also serve as useful heuristics in the development of future similar studies.

A wide variety of studies implicate frontal EEG asymmetry as an indicator of both trait predispositions to respond in emotionally characteristic ways, and of specific emotional states. As researchers come to understand how the brain regions indicated by these cortical measures both affect and are affected by emotional environmental stimuli, it will become increasingly important to identify the degrees to which patterns of responding associated with these regions are due to genetic versus environmental effects. The vast and growing body of work in the field of EEG asymmetry, as well as the data reported here, should encourage researchers to explicate such patterns and relationships.
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