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Genetic and Environmental Influences on Frontal EEG Asymmetry and Alpha Power in 9–10 Year Old Twins

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Abstract

Modest genetic influences on frontal EEG asymmetry have been found in adults, but little is known about its genetic origins in children. Resting frontal asymmetry and alpha power were examined in 951 9–10-year-old twins. Results showed that in both males and females: (1) a modest but significant amount of variance in frontal asymmetry was accounted for by genetic factors (11–27%) with the remainder accounted for by non-shared environmental influences, and (2) alpha power were highly heritable, with 70–85% of the variance accounted for by genetic factors. Results suggest that the genetic architecture of frontal asymmetry and alpha power in late childhood are similar to that in adulthood and that the high non-shared environmental influences on frontal asymmetry may reflect environmentally-influenced individual differences in the maturation of frontal cortex as well as state-dependent influences on specific measurements.

Descriptors

Frontal asymmetry; EEG; Alpha; Genetic; Child; Twins

The alpha rhythm is a waveform with a characteristic 8–13 Hz frequency and ranging in amplitude from 10 and 150 microvolts. It is usually observed when the individual is at rest and is attenuated or blocked when one is engaged in a cognitive task. In this context, alpha power is believed to be an inverse measure of regional cortical activation, and attenuated alpha power over one hemisphere (i.e., frontal asymmetry) has traditionally been used as an index of hemispheric differences in task performance (Shagass, 1972). Moreover, the pattern of frontal asymmetry at rest has been reported to appear early and is modestly preserved from infancy through the school-age years (Fox, Henderson, Rubin, Calkins, & Schmidt, 2001).

Davidson and colleagues published the first paper linking positive and negative affect to frontal asymmetry (Davidson, Schwartz, Sharon, Bennett, & Goleman, 1979). Given the advantages of being noninvasive, less expensive, fast time resolution, and more widely available than many neuroimaging modalities, frontal asymmetry has been widely used as a measure of underlying approach- or withdrawal-related behavioral tendencies and affective style in children and adults (Coan & Allen, 2004; Davidson, 1992; Harmon-Jones& Allen, 1997; Shankman, Tenke, Bruder, Durbin, Hayden, & Klein, 2005). In general, relatively greater left frontal activity (i.e.,

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relatively reduced left alpha power) is suggested to be associated with positive affect and/or approach motivation and behavioral patterns, whereas relatively greater right frontal activity (i.e., relatively reduced right alpha power) is related to negative affect and/or withdrawal motivation and behavioral patterns (Harmon-Jones & Allen, 1997; Sutton & Davidson, 1997).

Atypical frontal asymmetries have been found in children with attention-deficit/hyperactivity disorder (Baving, Laucht, & Schmidt, 1999), oppositional defiant disorder (Baving, Laucht, & Schmidt, 2000), and other forms of externalizing behavior (Santesso, Reker, Schmidt, & Segalowitz, 2006). For example, Santesso et al. (2006) linked frontal asymmetry with externalizing behavior in a sample of non-clinical 10-year-olds, finding relatively greater right frontal activity in children with higher reported externalizing behavior. Abnormal frontal asymmetry has also been found to be related to affective disorders such as anxiety and depression (Baving, Laucht, & Schmidt, 2002; Forbes, Shaw, Fox, Cohn, Silk, & Kovacs, 2006). For example, it has been found that infants of depressed mothers exhibit relatively greater right frontal activity, which is presumed to be a trait characteristic of risk for depression (Dawson et al., 1999). In a study comparing children of mothers with childhood-onset depression and normal controls, relatively greater left frontal activity has been found to be associated with anxious/depressed problems in the childhood-onset depression group but not in the normal controls (Forbes et al., 2006).

Despite the significance of frontal asymmetry as a biological correlate of behavioral tendencies and affective style and as a marker of liability to psychopathology, little is known about the genetic influences underpinning frontal asymmetry. To the authors' knowledge there are only two published behavior genetic studies in which the genetic and environmental origins of individual differences in frontal asymmetry were examined (Anokhin, Heath, & Myers, 2006; Smit, Posthuma, Boomsma, & de Geus, 2007). Anokhin et al. (2006) collected resting EEG data from a sample of 246 female twins at mid-frontal (F3 and F4) and lateral-frontal (F7 and F8) locations. Genetic influences were found to explain a significant but small portion of the total variance (27%) in EEG asymmetry at mid-frontal locations, whereas no significant genetic influences on frontal asymmetry at lateral-frontal sites were found. Consequently, the authors concluded that frontal asymmetry may not be a reliable biological marker for psychopathology. In another study, Smit et al. (2007) found that frontal asymmetry was heritable in young adulthood (under 35 years old) and genetic influences accounted for 37% and 32% of the variance for females and males, respectively. Furthermore, one unpublished thesis on frontal asymmetry by Coan (2003) on 125 twin pairs aged 19 reported a heritability estimate of 22% in females, but a non-fitting model in males precluding heritability estimation.

To date no study has been conducted investigating the heritability of frontal asymmetry in children. Furthermore, the question of whether genetic influences on frontal asymmetry are moderated by sex of the individual remains unsolved. For example, in Anokhin et al.'s (2006) study only females were included. In the other two studies in which males were included (Coan, 2003; Smit et al., 2007), genetic influences on frontal asymmetry were examined separately for males and females but no direct comparisons were conducted between the two sex groups. The current study included both male and female twins aged 9–10-years in order to assess whether genetic and/or environmental influences on EEG measures are equal across the two sexes.

Another important question concerns the reliability of frontal asymmetry. Baseline measurement of frontal asymmetry appears to be a relatively stable individual trait with moderate rest-retest reliability (Hagemann, Naumann, Thayer, & Bartussek, 2002; Sutton & Davidson, 1997; Tomarken, Davidson Wheeler, & Kinney, 1992). For example, Hagemann et al. (2002) measured EEG data across four different sessions and reported that 60% of the

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asymmetry variance was accounted for by a temporally stable trait whereas 40% was accounted for by occasion-specific fluctuation. In contrast, most studies on frontal asymmetry either base measurement on one time-point or alternatively two different time points in the same experimental session. In the present study EEG alpha power and frontal asymmetry were assessed at two rest sessions on the same day in order to examine the reliability of within-session alpha measures as well as to obtain a more reliable measure of asymmetry used in the majority of published EEG alpha asymmetry studies.

In contrast to frontal asymmetry studies, genetic studies on EEG alpha power have consistently shown that in adult populations individual differences are largely determined by genetic factors and that there are no sex differences in heritability (Anokhin et al., 2006; Smit, Posthuma, Boomsma, & de Geus, 2005; Tang et al., 2007; van Beijsterveldt & van Baal, 2002). For example, Anokhin et al. (2006) reported that alpha power was highly heritable at frontal sites, with heritability ranging from 85% to 87%. Relatively fewer twin studies have been conducted to examine the genetic origins of alpha power in children and adolescents. In one study, alpha power data were collected from 213 16-year-old twin pairs, and the heritability estimate was reported to be high (89%) with no sex differences in heritability (van Beijsterveldt, Molenaar, de Geus, & Boomsma, 1996). In another study by van Baal, de Geus, and Boomsma (1996), high heritabilities of 70–83% and 63–82% for alpha 1 and alpha 2, respectively, were found at frontal locations in 209 5-year-old twin pairs. Similarly, no sex differences were found.

In this study the heritabilities of frontal asymmetry and alpha power are examined using a community sample of 9–10-year-old male and female twins. Following Anokhin et al.'s study (2006) EEG data were collected over both frontal hemispheres at two same-day rest sessions. A central goal was to determine whether children display heritabilities of frontal asymmetry and alpha power similar to those found in adult populations, and assess whether there are differential effects across boys and girls. Based on previous findings, it was expected that high genetic influences would be found for alpha power in both boys and girls. Furthermore, it was hypothesized that in children: (1) frontal asymmetry would show a modest but significant heritability, and (2) genetic and environmental influences on asymmetry would not differ across males and females.

Method

Participants

The sample consisted of twins participating in the University of Southern California (USC) Twin Study of Risk Factors for Antisocial Behavior. Ascertainment procedures for the study have been described in detail recently (Baker, Barton, Lozano, Raine, & Fowler, 2006; Baker, Jacobson, Raine, Lozano, & Bezdjian, 2007). In brief, the Twin Study of Risk Factors for Antisocial Behavior is an ongoing prospective longitudinal study of the interplay of genetic, environmental, social, and biological factors on the development of antisocial behavior from childhood to adolescence. The twins were evaluated using an extensive protocol, including cognitive, behavioral, psychosocial, and psychophysiological measures. The twins and their parents from 605 families (N = 1,219 children) were recruited from the Los Angeles community and the sample is representative of the ethnic and socio-economic diversity of the greater Los Angeles area (Baker et al., 2007).

The participants were invited to the USC laboratory for a 6–8 hour assessment. Caregiver participation was primarily (> 92%) the biological mothers. Zygosity determination was based on DNA microsatellite analysis (> 7 concordant and zero discordant markers = monozygotic (MZ); one or more discordant markers = dizygotic (DZ)) for 87% of the same-sex twin pairs. For the remaining same-sex twin pairs, zygosity was established by questionnaire items about the twins' physical similarity and the frequency with which people confuse them. The

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questionnaire was used only when DNA samples were insufficient for one or both twins. When both questionnaire and DNA results were available, there was a 90% agreement between the two (Baker et al., 2007).

The current study includes data from the first wave of assessment in 2000–2004, when the children were 9–10 years old (mean age = 9.6, *SD* = 0.58). All left-handed subjects were excluded. Only twins (1) who came to the USC laboratory for an assessment, (2) where zygosity could be diagnosed, and (3) with valid data on EEG measures were included in the current study (n = 951). Logistic regression analyses were conducted to examine whether the participants (n = 951) and non-participants (n = 268) differed on sex, age, and socioeconomic status, based on the Hollingshead Four-Factor index of social status (Hollingshead, 1979). Results revealed that odds-ratios (OR) was not significant for family socioeconomic status, (OR = 1.01, 95% confidence interval (CI): 0.71 – 1.76), and sex (OR = 1.12, 95% CI: 0.71 – 1.76). However, the two groups differed on age (OR = 0.35, 95% CI: 0.20 – 0.60), indicating that younger children tended not to complete the whole EEG session.

Experimental Procedures

The assessment was divided into two sessions, separated by a one-hour lunch break. The psychophysiological tasks were administered individually to each child while the co-twin and the primary caregiver were being interviewed in other rooms. While the electrodes were attached to the child, the interviewer conversed with the child to help the child relax (approximately 10–20 minutes). Once the electrodes were attached to the child the interviewer left the room. Before administrating psychophysiological tasks, baseline assessments of EEG were obtained for three minutes (rest 1) in which children were instructed to sit still with eyes open, and for another three minutes (rest 2) after all the tasks were completed (approximately 1.5 hours later). Because many 9–10-year-old children have difficulties following the instruction to keep eyes closed for over three minutes, all children were asked to keep eyes open and focus on the blue cross on the computer screen during the rest sessions.

EEG Recording and Quantification

EEG data were recorded from 32 scalp sites and the left and right mastoids using a lycra Electro-Cap based on the 10–20 system, using James Long Inc. amplification system (Caroga Lake, NY). An anterior midline site (AFz) served as the ground electrode and Cz as reference site. EEG data was re-referenced offline to an average mastoids configuration. Recording sites were prepared by gently abrading each site with a conductive abrasive. Impedances were kept below 10 KΩ with the majority of impedances being less than 5 KΩ. The scalp EEG was amplified by a factor of 5000 with a sampling rate of 512 Hz. The hardware filter settings for the EEG channels were 0.1-Hz high pass and 100-Hz low pass. One bipolar electrooculogram (EOG) channel was recorded from above and below the supra- and infra-orbital ridges of the left eye using the same bandpass settings. Automated regression-based algorithms were used to minimize blink artifacts in the EEG (Lins, Picton, Berg, & Scherg, 1993) and epochs confounded by eye or body movements were excluded.

After correcting for artifacts, epochs were subjected to Fast Fourier transform (FFT), and measures of EEG power for the alpha band (8–13 Hz) were obtained from mid-frontal (F3 and F4) and lateral-frontal (F7 and F8) sites. For each rest session EEG power was averaged over the entire 180 seconds period, giving one average power value for each electrode site. Frontal asymmetry was calculated as L – R, where L and R are power values at the homologous left and right hemisphere sites (F3 and F4, F7 and F8), such that positive scores indicate higher right than left frontal activity. A total of six variables were used in the following analyses: alpha power at F3, F4, F7, F8, and frontal asymmetry at mid-frontal (F3–F4) and lateral-frontal (F7–F8). Analyses were first conducted for rest 1 and rest 2 separately and then for the EEG

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measures averaged across the two sessions. Initially the log transformation measure of EEG asymmetry traditionally used was calculated, but ultimately the univariate genetic model did not converge. Consequently, prior to inferential analyses, the EEG power and asymmetry scores were ranked (PROC RANK using the BLOM option) and normalized (PROC STANDARD) to reduce the positive skew in their distributions (van den Oord, Simonoff, Eaves, Pickles, Sildberg, & Maes, 2000) using the statistical software SAS 9.1.3.

Statistical Analyses

Twin methodology uses the differences in the proportion of genes shared between MZ twin pairs, who share 100% of their genes, and DZ twin pairs who share 50% of their segregating genes, to estimate the genetic (additive (A)/dominant (D)), shared environmental (C), and non-shared environmental (E) contributions to variance within phenotypes. The additive genetic component is the independent effect of individual genes summed over loci, if acting alone, makes MZ intraclass correlations twice the DZ intraclass correlations. A DZ intraclass correlation less than half an MZ intraclass correlation indicates dominance (non-additive genetic effects between alleles at the same locus) or epistasis (interaction of alleles at different loci). In contrast, a DZ intraclass correlation more than half a MZ intraclass correlation indicates the presence of genetic and shared environmental effects. The shared environmental effect indexes environmental effects common to both members of a twin pair (e.g., living in the same neighbourhood, having the same teacher) that act to increase their similarity. It should be noted that dominant genetic and shared environmental influences are negatively confounded, and cannot be estimated simultaneously in a study of MZ and DZ twins reared together. Non-shared environmental effects are environmental factors that are unique to each twin, that is, those effects that make siblings dissimilar. Heritability is the proportion of the phenotypic variance that is attributable to genetic influences (Neale & Cardon, 1992; Posthuma et al., 2003).

Univariate models, which estimate the relative contribution of additive genetic factors (A), dominant genetic effects (D) or shared environmental factors (C), and non-shared environmental factors (E) to individual differences, were first fit for each EEG measure individually. Saturated models, which estimate variances, covariances, and means of frontal asymmetry and power at each site, were used as the baseline. A series of models were fit to the data and compared: ACE, CE, AE, E, ADE, and DE. Since MZ twins are genetically identical, additive genetic factors are correlated 1.0 in the model. For DZ twins the genetic factors are correlated 0.5 as they on average share 50% of their segregating genes. Since DZ twins share only one fourth of the dominant genetic effect shared by MZ twins, the correlations between dominant effects are 1.0 and .25 for MZ and DZ twins, respectively. Shared environmental influences are assumed to contribute equally to similarity in MZ and DZ twins, and thus shared environmental factors correlate 1.0 in both MZ and DZ twins. There is no correlation between twins for the non-shared environmental effects by definition, and this effects also include measurement error (Neale & Cardon, 1992; Plomin, DeFries, McClearn, & McGuffin, 2001; Rijsdijk & Sham, 2002). To test for sex differences, a model in which the magnitudes of genetic and environmental effects were allowed to differ between males and females was compared against a model in which the estimates were constrained to be equal (Neale & Cardon, 1992).

All genetic models were fit to data using Mx (Meale et al., 2003) with a maximum likelihood estimation procedure. This method allows the inclusion of singletons, where information from only one twin in a pair is available, and/or the inclusion of twins with missing data, ultimately increasing power in the analyses. Goodness of fit of models was assessed by a likelihood-ratio χ^2–test, which is the difference between −2 log likelihood (−2LL) of the full model and that of the nested model. This difference has a χ^2distribution, with the degrees of freedom (*df*) equal

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to the difference between the number of estimated parameters in the two models. For the comparisons between non-nested models, Akaike's information criterion (AIC = $\chi^2 - 2\times df$, Akaike, 1987) and the Bayesian information criterion (BIC = −2LL + *df ln* N) which includes a parsimony adjustment were computed. Small values of AIC and BIC indicate a good-fitting, parsimonious model. Specifically, differences in BIC larger than 10 provide very strong evidence in favor of the model with the smaller value (Raftery, 1995).

Results

Descriptive Statistics and Age Effects

Means, standard deviations, and number of participants for mid- and lateral- frontal asymmetry indices and alpha power at each site at each rest session are presented in Table 1. No significant mean or variance differences were found between two members of a twin pair, nor were there any mean or variance differences across zygosity groups for any of the measures (results available upon request). However, significant mean differences between boys and girls were found for alpha power at F3 rest 1 ($\chi^2 = 21.834$, $df = 9$, $p = .009$) and F4 rest 1 ($\chi^2 = 17.908$, $df = 9$, $p < .04$), with girls showing slightly higher alpha power values than boys. Finally, age expressed in months was not significantly correlated with asymmetry, $r = -.05$ to $-.003$, ps > .12. In contrast, EEG power scores were significantly correlated with age, $r = -.16$ to $-.07$, ps < .05.

Intraclass Correlations

Intraclass correlations for the mid- and lateral- frontal asymmetry indices and alpha power at F3, F4, F7, and F8 are presented in Table 2. All MZ intraclass correlations were higher than DZ intraclass correlations, suggesting genetic influences on these measures. For example, the intraclass correlations for F3 rest 1 were .85 for MZ boys and .86 for MZ girls, whereas the corresponding values for DZ twins were .32 and .42 for boys and girls, respectively. All MZ intraclass correlations were less than 1, suggesting the influences of non-shared environmental factors. Furthermore, evidence for genetic dominance or epistasis is indicated when an MZ intraclass correlation exceeds twice the values of the DZ intraclass correlation for the same-sex twin pairs. This was particularly evident for alpha power at F3, F4, and F7 in males at rest 2.

Univariate Model Fitting

To further examine the etiological patterns suggested by the intraclass correlations, univariate modelling was carried out. The results from the univariate model-fitting are displayed in Table 3.

For mid-frontal asymmetry at rest 1, a model estimating genetic (A), shared environmental (C) and non-shared environmental (E) components separately in boys and girls (Model 2) fit the data better compared to the saturated model (Model 1) ($\Delta\chi^2 = 7.57$, $df = 9$, $p = .58$). Next, to test for sex differences, the model estimating A, C, and E components separately in boys and girls (Model 2) was compared to a model constraining variance components to be equal across sexes (Model 3). Findings indicated that Model 3 had a better fit, suggesting equality between boys and girls ($\Delta\chi^2 = 2.37$, $df = 3$, $p = .50$). Then, an AE model, in which C was set to zero (Model 4) was compared to the ACE model (Model 3), and showed a better fit ($\Delta\chi^2 = 2.55$, $df = 1$, $p = .11$). In addition, a CE model, in which A was set to zero (Model 5) was compared to the ACE model (Model 3) ($\Delta\chi^2 = .04$, $df = 1$, $p = .83$) and also showed a relatively better fit. Though both the AE model (Model 4) and the CE model (Model 5) were better than the ACE model (Model 3), the CE model (Model 5) had slightly smaller AIC and BIC values than the AE model, indicating a better fit. A model only estimating E (Model 6) was next compared to the better fitting CE model (Model 5). This model did not provide a better fit to the data ($\Delta\chi^2$

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= 30.19, $df = 1$, $p < .001$). Finally, we compared an ADE model (Model 7) with a DE model (Model 8). The DE model (Model 8) did not fit the data better than the ADE model ($\Delta\chi^2 = 3.98$, $df = 1$, $p = .046$). Also, the CE model (Model 5) had smaller AIC and BIC values than the ADE model (Model 7) and the AD model (Model 8), suggesting no dominant genetic influences for mid-frontal asymmetry at rest 1. Thus, the best-fitting model was the CE model (Model 5), constraining estimates to be equal across sexes.

For lateral-frontal asymmetry at rest 1 and for mid-frontal asymmetry at rest 2, the best fitting model was an AE model (Model 4), equating estimates across sexes. There was no indication of dominant genetic influences for either lateral-frontal asymmetry at rest 1 or for mid-frontal asymmetry at rest 2. However, for lateral-frontal asymmetry at rest 2, the best fitting model was a DE model (Model 4), equating estimates across sexes.

For F3, F4, F7 and F8 at rest 1, the best fitting models were AE models (Model 4), equating estimates across sexes, with no indications of dominant genetic influences. For F3, F7, and F8 at rest 2, the best fitting model was an AE model (Model 4), equating estimates across sexes, with no indication of dominant genetic influences. For F4 at rest 2, the best fitting model was also an AE model (Model 4), however, estimates could not be constrained to be equal in males and females (Table 3).

The parameter estimates from the univariate modelling are presented in Table 4. For mid-frontal asymmetry at rest 1, 28% of the variance was attributable to shared environmental effects (ps < .05), and 72% was attributable to non-shared environmental factors (ps < .05). For mid-frontal asymmetry at rest 2, 21% of the variance was due to additive genetic effects (ps < .05), and 79% was due to non-shared environmental factors (ps < .05). For lateral-frontal asymmetry during at rest 1, 11% (ps < .05) of the variance was explained by genetic influences, and 89% (ps < .05) was due to non-shared environmental factors (ps < .05). However, for lateral-frontal asymmetry at rest 2, 27% (ps < .05) of the variance was explained by dominant genetic influences, and 73% (ps < .05) was due to non-shared environmental factors (ps < .05).

Regarding alpha power, at rest 1 genetic effects accounted for 79–85% (ps < .05) of the total variance, with non-shared environmental factors accounting for the remaining 15–21% (ps < .05) of the variance. At rest 2, genetic factors accounted for 70–82% (ps < .05) of the variance, while 18–30% (ps < .05) of the variance was due to non-shared environmental influences. No shared environmental influence was found. For F4 at rest 2 estimates could not be equated across males and females, and the heritability was slightly higher in girls (82%) than in boys (70%) (Table 4).

Stability of Frontal Asymmetry and Alpha Power

Since some participants finished the first rest session only ($n = 785$), the slight decrease in heritability from rest 1 to rest 2 is possibly due to an increase in measurement error, which is incorporated into non-shared environmental influences. The phenotypic stability across the two rest sessions for alpha power at F3, F4, F7, and F8 was fairly strong in this sample, ranging from $r = 0.69$ to 0.82, ps < .001 in boys, and $r = 0.71$ to 0.79, ps < .001 in girls. Regarding asymmetry measures, the stability across the two rest sessions ranged from $r = 0.43$ to 0.67 for boys and $r = 0.48$ to 0.70 for girls, all ps < .05.

Frontal asymmetry indices and alpha power were averaged across the two rest sessions and univariate models were again fit to the data. For both asymmetry and power measures the results of model fitting and parameter estimates were very similar to those when the two sessions were separately fit. The models constraining variance components to be equal for boys and girls and dropping the shared environmental component proved to be the best-fitting models. For mid-frontal asymmetry 24% ($p < .05$) of the variance was accounted for by genetic

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factors, whereas 76% ($p < .05$) was due to non-shared environmental factors. For lateral-frontal asymmetry, 15% of the variance was due to genetic factors and 85% ($p < .05$) to non-shared environmental influences. For alpha power, 71 to 80% ($ps < .05$) of the total variance was due to genetic factors while non-shared environmental factors accounted for the remaining 20 to 29% ($ps < .05$) of the variance.

Discussion

In the current study, the heritability of frontal asymmetry and alpha power was examined in male and female twins in late childhood. Key findings are as follows: (1) frontal asymmetry showed a modest degree of heritability at both the mid-frontal and lateral-frontal locations, with 11–27% of the total variance being attributable to genetic factors and 72–89% attributable to non-shared environmental factors, (2) alpha power are highly heritable at all four frontal sites, with 70–85% of the variance accounted for by the genetic factors, (3) there were no shared environmental influences on any EEG measure, and (4) the genetic architecture of frontal asymmetry and alpha power is similar for boys and girls. To the authors' knowledge, this constitutes the first twin study to investigate the heritability of frontal asymmetry in children.

Consistent with the prior literature, EEG alpha power at all four frontal sites are highly heritable in children and there are no sex differences in heritability (except F4 at rest 2). This high heritability is comparable with the heritability found in other studies. Together with the findings of high heritability in 5-year-olds (van Baal et al., 1996) and 16-year-olds (van Beijsterveldt et al., 1996), it is suggested that alpha power is a highly stable and heritable trait across childhood and adolescence. Findings support the argument that alpha activation is associated with a spectral fingerprint of the individual brain (Nunez, 1981).

Regarding frontal asymmetry, in line with prior findings in adult populations (Anokhin et al., 2006; Coan, 2003; Smit et al., 2007), non-shared environmental influences were high, suggesting possibly similar genetic mechanisms in frontal asymmetry in late childhood and adulthood. Genetic influences were low, and no shared environmental influences were observed for most of the measures. The genetic mechanisms underlying frontal asymmetry are similar in males and females. Environmental influences were less in evidence for alpha power, but still accounted for 16–36% of the variance (see Table 4). The predominance of environmental influences in our child sample may come from a number of sources. One source may be the influence of the environment on individual differences in brain development.

Research has shown that the development of frontal areas extends into late adolescence and even early adulthood, which makes this brain region more sensitive to environmental influences compared to other brain areas, and there are substantial individual differences in the rate of development (Casey, Giedd, & Thomas, 2000.; Hudspeth & Pribram, 1990; Thatcher, Walker, Giudice, 1987). In this context, environmental differences may partly account for the large individual differences in the rate of maturation of frontal cortex, especially in the development of inter-hemispheric relationships in this age range, which in turn may contribute to the small twin correlations and hence low heritability in frontal asymmetry. In addition, in the current study participants were asked to keep their eyes open during the rest sessions. The high non-shared environmental influences may be partly due to the variation induced by eye movements. Furthermore, the eyes open condition could lead to greater environmental variability in visual stimulation across subjects.

Although the age range of the current sample is narrow (9–10 years), we nevertheless examined the effect of age on EEG measures. Results showed that age expressed in months was not significantly correlated with frontal asymmetry. In contrast, age was associated with alpha power, with older children showing decreasing power scores. This finding is in line with the

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prior literature showing maturation reductions in EEG power with increasing age (e.g., McGuire, Katsanis, Iacono, & McGue, 1998),

There have been very few experimental studies in children that identify specific components of the environment that alter EEG. One longitudinal study demonstrated that children randomly assigned to an environmental enrichment group from ages 3–5 years (better nutrition, more physical exercise, and cognitive stimulation) showed a significant reduction in slow-wave EEG both at rest and during a sustained attention task at age 11 compared to matched controls (Raine et al., 2001). On the other hand, studies on posttraumatic stress disorder (PTSD) have failed to find frontal asymmetry differences between individuals with PTSD and normal controls (Rabe, Beauducel, Zollner, Maercker, & Karl, 2006; Shankman et al., 2008), suggesting that trauma may not be an environmental factor affecting EEG asymmetries. At the same time, Rabe, Zoellner, Beauducel, Maercker, and Karl (2008) recently found that cognitive behavioral therapy can change frontal asymmetries (reduced right frontal activation) in individuals with PTSD. Identifying which specific environmental influences permanently shape EEG power and asymmetries reflects a significant gap in our knowledge, and remains an important next step for future studies.

Compared to alpha power, frontal asymmetry measures showed lower stability across the two rest sessions in the current study. This is consistent with prior studies showing relatively low to modest stability of frontal asymmetry and high stability of alpha power (Vuga, Fox, Cohn, Kovacs, & George, 2008). Specifically, Hagemann et al. (2002) recorded EEG data from 59 individuals on four occasions of measurements each separated by 4 weeks and in each occasion six baselines were collected. Using latent state-trait change model, they found that 40% of the variance of the asymmetry measure was due to state-dependent fluctuations whereas the rest 60% of the variance was due to a temporally-stable latent trait. It has been argued that the observed frontal asymmetry represents a superimposition of a trait-like activation asymmetry with substantial state-dependent fluctuations (Davidson, 1992; Hagemann et al., 2002; Tomarken et al., 1992).

Although aggregation across the two rest sessions in the current study was conducted to derive a more reliable measure of EEG asymmetry, it has been argued that this procedure cannot entirely eliminate state-dependent fluctuations (Steyer & Schmitt, 1990). Indeed, Davidson (1998) has argued that repeated testing across several weeks is required to derive a reliable asymmetry measure. Despite this, most studies derive asymmetry scores from either one rest period or two rest periods in a same-day assessment (Hagemann et al., 2002; Shankman et al., 2008). The relatively low heritability of frontal asymmetry measures observed in the current study may be partly due to the situational effects or person-situation interactions (Davidson, 1992). However, when frontal asymmetry indices were averaged across the two rest sessions to increase measurement reliability, results similar to those presented in Table 3 and Table 4 were obtained. These findings suggest that the relatively high non-shared environmental effects may not be entirely due to short-term effects, such as measurement error. It is also worth noting that specific conditions of experiment, time-of-day, mood state, and temperature may contribute to non-shared environmental influences. Consequently it cannot be concluded from current findings that *trait* frontal asymmetry is not highly heritable, since state-dependent specificity rather than trait specificity was more likely measured in the current study. In future studies, genetic modeling would benefit from assessing resting asymmetry at multiple time-points in which measurement error, state-dependent fluctuations, and trait specificity are statistically separated (Hagemann et al., 2002), so that the heritability of the frontal asymmetry as a trait can be more reliably examined.

A few potential limitations of the current study should be noted. First, only mid-frontal and lateral-frontal regions were included in the current study. The analyses were restricted to the

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alpha band at frontal locations because it is the dominant frequency in resting subjects and a replication of Anokhin et al.'s study (2006) was intended. Future research could examine the genetic origins of frontal asymmetry acquired from other anterior regions, e.g., frontal-temporal-central, anterior temporal, and frontal-central regions.

Secondly, asymmetry indices were calculated as L-R in the current study. Given the fact that difference scores between correlated measures are inherently less reliable than the individual measures contributing to them, it may be possible that environmental effects on asymmetry are artifactually inflated, as reliability constrains heritability, and the sum of genetic and environmental influences must equal 100% of the phenotypic variance in AE models. Furthermore, the current study is not strictly comparable to other laterality studies which use a log transformation (e.g., Anokhin et al., 2006; Smit et al., 2007). To the authors' knowledge there has been at least one prior failure to fit genetic models to frontal asymmetry data using the traditionally log transformation (Coan, 2003). Although for comparability purposes model-fitting based on the traditional asymmetry measure would have been preferable, the current study documents an alternative approach for asymmetry data modeling in future studies when log transformations fail to fit.

A third limitation concerns the low intraclass correlations in male DZ twins at F3 and F4 at rest 2. It is possible that this is due to dominant genetic influences. However, a model including dominant genetic effects did not describe the data better than a model with additive genetic influences. Although the low DZ male intraclass correlation might suggest dominant genetic influences in the boys, the moderate resemblance among DZ opposite sex pairs would suggest otherwise. When examining the data for outliers, no influential points were found.

Finally, there are several factors that may affect psychophysiological recording, such as temperature, humidity, experimenter characteristics, and season of testing (Bouscein, 1992), which may contribute to part of the shared environmental components. Therefore, it is possible that the impact of genetic influences on the etiology of all EEG measures, especially mid-frontal asymmetry, is underestimated in our univariate analyses.

In conclusion, the present study extends the previous findings on adult populations and has shown no or only modest genetic influences on frontal asymmetry measured at mid-frontal and lateral-frontal locations in 9- to 10- year-old children. In contrast, EEG alpha power is highly heritable in children, comparable to what is found in older adolescents and adults. Males and females show similar genetic architecture in the majority of these EEG measures. It is suggested that the high non-shared environmental influences on frontal asymmetry in children may be partly due to both state influences and also environmental influences on individual differences in rate of maturation of the frontal cortex.

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Table 1

Means (Standard Deviations) and Sample Sizes for Frontal Asymmetry and Alpha Power at Rest 1 and Rest 2, by Sex and Zygosity (Untransformed Data)

	Males		Females		DZ opposite sex	
	MZ	DZ	MZ	DZ	Males	Females
Mid asymmetry rest 1	−.02 (1.05) n=212	.32 (1.06) n=128	−.11 (.92) N=234	.11 (2.22) n=146	.01 (.83) n=113	.15 (.71) n=110
Lateral asymmetry rest 1	−.16 (1.05) n=212	.07 (1.26) n=128	−.10 (1.45) N=234	−.41 (3.18) n=146	−.09 (1.08) n=113	−.14 (1.20) n=110
Mid asymmetry rest 2	−.10 (.99) n=162	.14 (1.00) n=94	.12 (1.62) N=212	.41 (2.82) n=121	−.01 (.96) n=92	.07 (1.02) n=91
Lateral asymmetry rest 2	−.17 (1.28) n=162	.01 (1.21) n=94	−.15 (1.58) N=212	−.12 (1.04) n=121	−.08 (1.02) n=92	.03 (1.34) n=91
F3 rest 1	13.43 (5.69) n=212	14.70 (7.01) n=128	15.02 (8.58) N=234	14.70 (7.36) n=146	13.38 (6.30) n=113	15.52 (8.89) n=110
F4 rest 1	13.48 (5.59) n=212	14.06 (6.16) n=128	14.80 (8.32) N=234	14.48 (7.68) n=146	13.36 (6.38) n=113	15.22 (9.06) n=110
F7 rest 1	9.50 (4.16) n=212	10.13 (4.65) n=128	10.06 (6.05) N=234	9.04 (5.45) n=146	9.18 (4.34) n=113	10.33 (6.86) n=110
F8 rest 1	9.81 (4.23) n=212	9.98 (4.07) n=128	10.25 (5.16) N=234	10.85 (8.07) n=146	9.36 (4.69) n=113	10.60 (5.89) n=110
F3 rest 2	14.27 (6.51) n=162	14.52 (6.29) n=94	15.29 (8.38) n=212	14.85 (8.97) n=121	13.78 (7.03) n=92	16.06 (10.62) n=91
F4 rest 2	14.47 (6.64) n=162	14.24 (6.09) n=94	15.04 (7.78) n=212	14.02 (7.16) n=121	13.80 (6.88) n=92	15.91 (11.63) n=91
F7 rest 2	10.07 (4.62) n=162	10.17 (4.65) n=94	10.32 (5.94) n=212	9.83 (5.15) n=121	9.50 (4.60) n=92	11.16 (8.18) n=91
F8 rest 2	10.42 (4.77) n=162	10.14 (4.63) n=94	10.62 (5.51) n=212	10.07 (4.94) n=121	9.66 (5.04) n=92	11.10 (6.99) n=91

Note. MZ = Monozygotic, DZ = Dizygotic, Mid asymmetry = Mid-frontal asymmetry (F3–F4), Lateral asymmetry = Lateral-frontal asymmetry (F7–F8).

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Table 2

Intraclass Correlations for Frontal Asymmetry and Alpha Power at Rest 1 and Rest 2

	Males		Females		
	MZ	**DZ**	**MZ**	**DZ**	**DZ opposite sex**
Mid asymmetry rest 1	.35*	.21*	.23*	.19*	.29*
Lateral asymmetry rest 1	.12	.08	.09	−.01	.04
Mid asymmetry rest 2	.27*	−.05	.16*	.12	.06
Lateral asymmetry rest 2	.29*	.16	.25*	−.37*	−.25*
F3 rest 1	.85*	.32*	.86*	.42*	.41*
F4 rest 1	.80*	.28*	.86*	.30*	.41*
F7 rest 1	.81*	.38*	.80*	.33*	.44*
F8 rest 1	.74*	.45*	.82*	.38*	.34*
F3 rest 2	.71*	.06	.81*	.35*	.31*
F4 rest 2	.69*	.02	.82*	.64*	.28*
F7 rest 2	.62*	.25	.77*	.39*	.31*
F8 rest 2	.62*	.29*	.75*	.45*	.34*

Note. MZ = Monozygotic, DZ = Dizygotic, Mid asymmetry = Mid-frontal asymmetry (F3–F4), Lateral asymmetry = Lateral-frontal asymmetry (F7–F8).

* $p < .05$.

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Table 3

Univariate Model-Fitting Results for Frontal Asymmetry and Alpha Power at Rest 1 and Rest 2

			Overall fit				Model comparison test						
	Model		***−2LL***	***df***	***AIC***	***BIC***	χ^2	***df***	***p***	$\Delta\chi^2$	***Δdf***	***p***	**Comparison model**
Mid asymmetry rest 1	1	Saturated	2633.16	926	781.16	−1600.69							
	2	ACE Boy≠Girl	2640.73	935	770.73	−1625.25	7.57	9	.58				
	3	ACE Boy=Girl	2643.10	938	767.10	−1633.52	9.94	12	.62	2.37	3	.50	Model 2
	4	AE Boy=Girl	2645.65	939	767.65	−1635.39	12.50	13	.49	2.55	1	.11	Model 3
	5	**CE Boy=Girl**	**2643.15**	**939**	**765.15**	**−1636.65**	**9.99**	**13**	**.69**	**0.04**	**1**	**.83**	**Model 3**
	6	E Boy=Girl	2673.34	940	793.34	−1624.70	40.18	14	< .001	30.19	1	< .01	Model 5
	7	ADE Boy=Girl	2647.81	937	773.81	−1628.02	14.65	11	.20				
	8	DE Boy=Girl	2651.79	938	775.79	−1629.18	18.63	12	.10	3.98	1	.05	Model 7
Lateral asymmetry rest 1	1	Saturated	2669.25	927	815.25	−1585.79							
	2	ACE Boy≠Girl	2671.78	936	799.78	−1612.88	2.53	9	.98				
	3	ACE Boy=Girl	2672.46	939	794.46	−1621.99	3.21	12	.99	0.68	3	.88	Model 2
	4	**AE Boy=Girl**	**2672.46**	**940**	**792.46**	**−1625.14**	**3.21**	**13**	**1.00**	**0.00**	**1**	**1.00**	**Model 3**
	5	CE Boy=Girl	2672.81	940	792.81	−1624.96	3.56	13	.99	0.35	1	.55	Model 3
	6	E Boy=Girl	2675.12	941	793.12	−1626.96	5.87	14	.97	2.66	1	.10	Model 4
	7	ADE Boy=Girl	2672.43	937	798.43	−1615.70	3.18	10	.98				
	8	DE Boy=Girl	2672.53	938	796.53	−1618.80	3.28	11	.99	0.10	1	.75	Model 7
Mid asymmetry rest 2	1	Saturated	2177.94	756	665.94	−1250.19							
	2	ACE Boy≠Girl	2181.49	765	651.49	−1276.27	3.547	9	.94				
	3	ACE Boy=Girl	2182.49	768	646.49	−1285.05	4.544	12	.97	0.99	3	.80	Model 2
	4	**AE Boy=Girl**	**2182.49**	**769**	**644.49**	**−1288.14**	**4.544**	**13**	**.98**	**0.00**	**1**	**1.00**	**Model 3**
	5	CE Boy=Girl	2183.83	769	645.83	−1287.47	5.890	13	.95	1.35	1	.25	Model 3
	6	E Boy=Girl	2189.84	770	649.84	−1287.56	11.897	14	.62	7.35	1	< .01	Model 4
	7	ADE Boy=Girl	2182.26	766	650.26	−1278.97	4.319	10	.93				
	8	DE Boy=Girl	2182.31	767	648.31	−1282.05	4.365	11	.96	0.05	1	.83	Model 7

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			Overall fit							Model comparison test			
	Model		***−2LL***	***df***	***AIC***	***BIC***	χ^2	***df***	***p***	$\Delta\chi^2$	***Δdf***	***p***	**Comparison model**
Lateral asymmetry rest 2	1	Saturated	2163.06	756	651.06	−1257.64							
	2	ACE Boy≠Girl	2177.38	765	647.38	−1278.32	14.33	9	.11				
	3	ACE Boy=Girl	2184.51	768	648.51	−1284.02	21.45	12	.04	7.13	3	.07	Model 2
	4	AE Boy=Girl	2183.72	767	649.72	−1281.34	20.66	11	.04	0.79	1	.37	Model 3
	5	CE Boy=Girl	2178.52	767	644.52	−1283.94	15.47	11	.16	5.99	1	.01	Model 3
	6	E Boy=Girl	2189.84	769	651.84	−1284.47	26.78	13	.01	11.32	1	< .01	Model 4
	7	ADE Boy=Girl	2178.10	766	646.10	−1281.05	15.05	10	.13				
	8	**DE Boy=Girl**	**2178.10**	**767**	**644.10**	**−1284.15**	**15.05**	**11**	**.18**	**0.00**	**1**	**1.00**	**Model 7**
F3 rest 1	1	Saturated	2364.85	926	512.85	−1734.84							
	2	ACE Boy≠Girl	2369.73	935	499.73	−1760.75	4.88	9	.85				
	3	ACE Boy=Girl	2370.35	938	494.35	−1769.90	5.50	12	.94	0.62	3	.89	Model 2
	4	**AE Boy=Girl**	**2370.45**	**939**	**492.45**	**−1772.99**	**5.59**	**13**	**.96**	**0.10**	**1**	**.75**	**Model 3**
	5	CE Boy=Girl	2453.44	939	575.44	−1731.50	88.59	13	< .001	82.10	1	< .01	Model 3
	6	E Boy=Girl	2669.26	940	789.26	−1626.74	304.41	14	< .001	298.82	1	< .01	Model 4
	7	ADE Boy=Girl	2381.77	937	507.77	−1761.03	16.92	11	.11				
	8	DE Boy=Girl	2390.49	938	514.49	−1759.83	25.64	12	.01	8.72	1	< .01	Model 7
F4 rest 1	1	Saturated	2398.59	926	546.59	−1717.97							
	2	ACE Boy≠Girl	2405.53	935	535.53	−1742.85	6.93	9	.64				
	3	ACE Boy=Girl	2406.58	938	530.58	−1751.78	7.99	12	.79	1.06	3	.79	Model 2
	4	**AE Boy=Girl**	**2406.58**	**939**	**528.58**	**−1754.93**	**7.99**	**13**	**.84**	**0.00**	**1**	**1.00**	**Model 3**
	5	CE Boy=Girl	2482.63	939	604.63	−1716.90	84.04	13	< .001	76.12	1	< .01	Model 3
	6	E Boy=Girl	2671.00	940	791.00	−1625.87	272.41	14	< .001	264.42	1	< .01	Model 4
	7	ADE Boy=Girl	2413.97	937	539.97	−1744.93	15.38	11	.17				
	8	DE Boy=Girl	2419.63	938	543.63	−1745.25	21.04	12	.05	5.66	1	.02	Model 7
F7 rest 1	1	Saturated	2424.08	927	570.08	−1708.38							
	2	ACE Boy≠Girl	2430.62	936	558.62	−1733.46	6.54	9	.69				
	3	ACE Boy=Girl	2433.34	939	555.35	−1741.55	9.27	12	.68	2.73	3	.44	Model 2
	4	**AE Boy=Girl**	**2433.37**	**940**	**553.37**	**−1744.69**	**9.29**	**13**	**.75**	**0.02**	**1**	**.88**	**Model 3**
	5	CE Boy=Girl	2495.41	940	615.41	−1713.67	71.33	13	< .001	62.06	1	< .01	Model 3

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	Model		Overall fit −2LL	df	AIC	BIC	χ^2	df	p	Model comparison test $\Delta\chi^2$	Δdf	p	Comparison model
	6	E Boy=Girl	2675.12	941	793.12	−1626.96	251.04	14	< .001	241.75	1	< .01	Model 4
	7	ADE Boy=Girl	2433.37	937	559.37	−1735.23	9.29	10	.51				
	8	DE Boy=Girl	2443.83	938	567.83	−1733.15	19.75	11	.05	10.46	1	< .01	Model 7
F8 rest 1	1	Saturated	2443.64	927	589.64	−1698.59							
	2	ACE Boy≠Girl	2452.42	936	580.42	−1722.56	8.78	9	.46				
	3	ACE Boy=Girl	2458.10	939	580.10	−1729.17	14.46	12	.27	5.70	3	.13	Model 2
	4	**AE Boy=Girl**	**2458.10**	**940**	**578.10**	**−1732.32**	**14.46**	**13**	**.34**	**0.00**	**1**	**1.00**	**Model 3**
	5	CE Boy=Girl	2515.65	940	635.65	−1703.54	72.01	13	< .001	57.60	1	< .01	Model 3
	6	E Boy=Girl	2675.12	941	793.12	−1626.96	231.48	14	< .001	217.00	1	< .01	Model 4
	7	ADE Boy=Girl	2456.96	937	582.96	−1723.44	13.32	10	.21				
	8	DE Boy=Girl	2465.62	938	589.62	−1722.26	21.98	11	.03	8.66	1	< .01	Model 7
F3 rest 2	1	Saturated	2035.95	756	523.95	−1321.19							
	2	ACE Boy≠Girl	2043.89	765	513.89	−1345.07	7.94	9	.54				
	3	ACE Boy=Girl	2045.92	768	509.92	−1353.33	9.98	12	.62	2.04	3	.57	Model 2
	4	**AE Boy=Girl**	**2045.92**	**769**	**507.92**	**−1356.43**	**9.98**	**13**	**.70**	**0.00**	**1**	**1.00**	**Model 3**
	5	CE Boy=Girl	2091.02	769	553.02	−1333.88	55.07	13	< .001	45.10	1	< .01	Model 3
	6	E Boy=Girl	2189.84	770	649.84	−1287.56	153.89	14	< .001	143.92	1	< .01	Model 4
	7	ADE Boy=Girl	2044.80	766	512.80	−1347.70	8.86	10	.55				
	8	DE Boy=Girl	2046.42	767	512.42	−1349.99	10.48	11	.49	1.62	1	.20	Model 7
F4 rest 2	1	Saturated	2010.56	756	498.56	−1333.89							
	2	ACE Boy≠Girl	2022.87	765	492.87	−1355.58	12.31	9	.20				
	3	ACE Boy=Girl	2035.12	768	499.12	−1358.73	24.56	12	.02	12.25	3	< .01	Model 2
	4	**AE Boy≠Girl**	**2031.81**	**767**	**497.81**	**−1357.30**	**21.25**	**11**	**.03**	**8.94**	**2**	**< .01**	**Model 2**
	5	CE Boy≠Girl	2046.17	767	512.17	−1350.12	35.61	11	< .001	23.30	2	< .01	Model 2
	6	E Boy≠Girl	2189.00	769	651.00	−1284.89	178.44	13	< .001	157.19	2	< .01	Model 4
	7	ADE Boy≠Girl	2030.97	763	504.97	−1345.40	20.41	7	.01				
	8	DE Boy≠Girl	2039.16	765	509.16	−1347.43	28.60	9	< .001	8.195	2	.02	Model 7
F7 rest 2	1	Saturated	2056.86	756	544.86	−1310.73							
	2	ACE Boy≠Girl	2065.16	765	535.16	−1334.43	8.30	9	.50				
	3	ACE Boy=Girl	2067.78	768	531.78	−1342.40	10.92	12	.54	2.62	3	.45	Model 2
	4	**AE Boy=Girl**	**2067.78**	**769**	**529.78**	**−1345.50**	**10.92**	**13**	**.62**	**0.00**	**1**	**1.00**	**Model 3**

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	Model		Overall fit −2LL	df	AIC	BIC	χ^2	df	p	Model comparison test $\Delta\chi^2$	Δdf	p	Comparison model
	5	CE Boy=Girl	2095.09	769	557.09	−1331.84	38.23	13	< .001	27.31	1	< .01	Model 3
	6	E Boy=Girl	2189.84	770	649.84	−1287.56	132.98	14	< .001	122.06	1	< .01	Model 4
	7	ADE Boy=Girl	2067.49	766	535.49	−1336.36	10.63	10	.39				
	8	DE Boy=Girl	2071.98	767	537.98	−1337.21	15.11	11	.18	4.48	1	.03	Model 7
F8 rest 2	1	Saturated	2058.68	756	546.68	−1309.82							
	2	ACE Boy≠Girl	2066.28	765	536.28	−1333.87	7.60	9	.57				
	3	ACE Boy=Girl	2068.94	768	532.94	−1341.83	10.26	12	.59	2.65	3	.45	Model 2
	4	**AE Boy=Girl**	**2069.50**	**769**	**531.50**	**−1344.64**	**10.82**	**13**	**.63**	**0.56**	**1**	**.45**	**Model 3**
	5	CE Boy=Girl	2085.92	769	547.92	−1336.43	27.24	13	.01	16.98	1	< .01	Model 3
	6	E Boy=Girl	2189.84	770	649.84	−1287.56	131.16	14	< .001	120.23	1	< .01	Model 4
	7	ADE Boy=Girl	2069.39	766	537.39	−1335.41	10.71	10	.38				
	8	DE Boy=Girl	2076.20	767	542.20	−1335.10	17.52	11	.09	6.81	1	< .01	Model 7

Note. A = Additive genetic effects, C = Shared environmental effects, D = Dominant genetic effects, E = Non-shared environmental effects, Mid asymmetry = Mid-frontal asymmetry (F3–F4), Lateral asymmetry = Lateral-frontal asymmetry (F7–F8), −2LL = −2 (log - likelihood), AIC = Akaike's information criterion, BIC = Bayesian information criterion, χ^2 = Difference in log likelihoods between nested models, *df* = Change in degrees of freedom. Model in bold is the best fitting model.

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Table 4

Results from Best-Fitting Models for Frontal Asymmetry and Alpha Power at Rest 1 and Rest 2

	A	C/D	E	A	C/D	E
	Parameter estimates (95 % confidence interval)					
	Males			Females		
Mid asymmetry rest 1		.28 (C) (.18 – .37)	.72 (.63 – .82)		Same as male	
Lateral asymmetry rest 1	.11 (.00 – .24)		.89 (.76 – 1.00)		Same as male	
Mid asymmetry rest 2	.21 (.06 – .34)		.79 (.66 – .94)		Same as male	
Lateral asymmetry rest 2		.27 (D) (.12 – .41)	.73 (.59 – .88)		Same as male	
F3 rest 1	.85 (.82 – .88)		.15 (.12 – .18)		Same as male	
F4 rest 1	.83 (.79 – .86)		.17 (.14 – .21)		Same as male	
F7 rest 1	.81 (.77 – .85)		.19 (.15 – .23)		Same as male	
F8 rest 1	.79 (.73 – .83)		.21 (.17 – .27)		Same as male	
F3 rest 2	.78 (.72 – .83)		.22 (.17 – .28)		Same as male	
F4 rest 2	.70 (.56 – .80)		.30 (.20 – .44)	.82 (.75 – .86)		.18 (.14 – .25)
F7 rest 2	.73 (.66 – .79)		.27 (.21 – .34)		Same as male	
F8 rest 2	.71 (.63 – .77)		.29 (.23 – .37)		Same as male	

Note. A = Additive genetic effects, C = Shared environmental effects, D = Dominant genetic effects, E = Non-shared environmental effects, Mid asymmetry = Mid-frontal asymmetry (F3–F4), Lateral asymmetry = Lateral-frontal asymmetry (F7–F8).

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