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GENETIC AND ENVIRONMENTAL INFLUENCES ON FRONTAL EEG ASYMMETRY: A TWIN STUDY

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Abstract

Research suggests that frontal EEG asymmetry (FA) is a relatively stable trait associated with individual differences in dispositional affect (affective style) and liability to mood disorders. If FA is genetically determined, it can potentially serve as an endophenotype in genetic studies of temperament and mood disorders. The purpose of this study was to assess heritability of FA as well as alpha band EEG power measured at different frontal recording sites. Resting EEG data from a population-based sample of 246 young adult female twins including 73 monozygotic (MZ) and 50 dizygotic (DZ) pairs were analyzed using linear structural equation modeling. FA measured at mid-frontal locations (F3 and F4) showed low but significant heritability, suggesting that 27 % of the observed variance can be accounted for by genetic factors. There was no evidence for genetic influences on FA measured at lateral frontal (F7 and F8) locations. In contrast, alpha band power was highly heritable at all four frontal sites (85-87%). These findings suggest that 1) genetic influences on FA are very modest and therefore FA has a limited utility as an endophenotype for genetic studies of mood disorders and 2) prefrontal neural circuitry underlying individual differences in affective style is characterized by high developmental plasticity.

Keywords

EEG; prefrontal cortex; hemispheric asymmetry; twins; genetics; heritability

INTRODUCTION

A substantial body of literature has been devoted recently to the relationships between frontal hemispheric asymmetry of alpha-band EEG power and different aspects of emotion (see e.g. a recent special issue of Biological Psychology (Allen & Kline, 2004)). Functional interpretation of frontal EEG asymmetry in these studies relies upon a fundamental assumption that EEG alpha band power is an inverse measure of cortical activation underneath the recording electrode. This assumption was initially based on the well-known effects of alpha blocking by sensory stimulation, however, more recent studies combining EEG and hemodynamic imaging methods have shown that cortical alpha activity is negatively correlated with cerebral perfusion in the area beneath the EEG electrode (Cook, O'Hara, Uijtdehaage, Mandelkern, & Leuchter, 1998) and with cerebral glucose metabolism (Oakes et al., 2004). These findings generally support the utility of EEG alpha power as an inverse indicator of regional cortical activation, although the relationships described above are not entirely

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consistent across cortical regions and recording conditions, and more studies are needed to further clarify the functional significance of alpha oscillations.

Frontal EEG asymmetry (FA), which is sometimes referred to as anterior EEG asymmetry, has been extensively studied both as a state-dependent correlate of acute affective response and as a trait-like variable related to stable individual differences in emotional responding (dispositional affect). Experimental factors used to induce emotion (e.g., emotionally evocative film clips, reward and punishment contingencies) produced asymmetric patterns of EEG activation. In these experiments, negative affect was associated with relatively higher right frontal activation, whereas positive emotional states were associated with greater left frontal activation (Davidson, 2003; Tomarken, Davidson, & Henriques, 1990; Wheeler, Davidson, & Tomarken, 1993).

Baseline measure of frontal EEG asymmetry appears to be a relatively stable individual trait with a good internal consistency and test-retest reliability (Tomarken, Davidson, Wheeler, & Kinney, 1992). A structural equation modeling of EEG data obtained across four different sessions (Hagemann, Naumann, Thayer, & Bartussek, 2002) has shown that 60% of the FA variance was due to individual differences on a temporally stable latent trait and 40% of the variance was due to occasion-specific fluctuations.

In normal adults, frontal brain asymmetry has been associated with individual differences in affective style, or dispositional affect. This broad concept encompasses negative or positive bias in affective responding (Jacobs & Snyder, 1996), relative strength of the behavioral activation and inhibition systems as measured by self-report questionnaire (Coan & Allen, 2003; Harmon-Jones & Allen, 1997; Sutton & Davidson, 1997), and repressive vs. defensive coping style (Tomarken & Davidson, 1994). Frontal brain asymmetry has been conceptualized as a biological substrate of the fundamental dimensions of emotion, approach and withdrawal (Davidson, 2001, 2003). The vast literature on the relationships between individual differences in FA, emotion, and behavior has been summarized and discussed in a recent review (Coan & Allen, 2004).

Clinical studies using FA have shown reduced left frontal activation in depressed subjects (Henriques & Davidson, 1991; Schaffer, Davidson, & Saron, 1983) including currently normothymic individuals with prior history of depression (Henriques & Davidson, 1990), suggesting that lateralized deficit of frontal cortical function is associated not only with depressive state itself, but also with increased liability to depression. However, other studies yielded mixed results (Debener et al., 2000; Hagemann, Naumann, Becker, Maier, & Bartussek, 1998; Reid, Duke, & Allen, 1998), prompting a vivid discussion about methodological issues in FA research (Allen, Coan, & Nazarian, 2004; Davidson, 1998; Hagemann, 2004).

Developmental studies have shown that individual differences in FA emerge early in life and are associated with individual differences in infants' behavior along the approach-withdrawal (Davidson, 1992; Fox & Davidson, 1986; Fox et al., 1995; Kagan & Snidman, 1999; McManis, Kagan, Snidman, & Woodward, 2002). Children of depressed mothers exhibit reduced left frontal activation at 1 month of age (Jones, Field, Fox, Lundy, & Davalos, 1997) and at 13-15 months (Dawson et al., 1999; Dawson, Panagiotides, Klinger, & Spieker, 1997). Moreover, FA had a greater sensitivity than behavioral measures in discriminating between infants of depressed and non-depressed mothers (Dawson et al., 1997). Taken together, these findings suggest that that FA can be a biological marker of familial and, possibly, genetic risk for mood disorders, which can potentially have important implications for psychiatric genetic research.

Identification of genes conferring risk for psychopathology, including mood disorders, has been hindered by the complexity and variability of diagnostic phenotypes. An alternative and

increasingly popular approach focuses on “endophenotypes”, or biological markers of complex behavioral phenotypes that index genetically transmitted variability and dysfunction in the basic neurocognitive mechanisms (Gottesman & Gould, 2003). FA meets several important criteria for an endophenotype: 1) it is a relatively stable trait measure; 2) abnormal FA pattern is associated with a history of psychiatric diagnosis (major depression) and, importantly, is also present during remission; 3) FA is associated with continuous indicators of diathesis such as temperamental traits indicating a deficit of approach behaviors and/or greater propensity towards withdrawal behaviors; 4) FA develops early in life and is associated with emotionality in infants and children; 5) finally, FA has clear conceptual links to neurobiological mechanisms of emotion and mood disorders that are corroborated by neuroimaging and brain lesion data. However, another fundamental requirement for an endophenotype for genetic research is its significant heritability. Based on the evidence discussed above, it appears reasonable to hypothesize that FA may reflect genetically transmitted individual differences in neurophysiological substrates of emotion. If heritability of FA is firmly established, FA can potentially serve as a useful endophenotype for genetic studies of mood disorders.

Given the potential significance of frontal brain asymmetry as a biological substrate of affective style and a marker of liability to mood disorders, it is surprising, how little is known about genetic versus environmental origins of individual differences in FA. To the best of our knowledge, the existing literature on the genetic aspects of FA is very scarce and limited to a few conference abstracts. One study reported changes in heritability as a function of the EEG recording time, when each individual minute of a 5-min recording was analyzed separately (Allen, Reiner, Katsanis, & Iacono, 1997). Other studies found a significant familial transmission of FA (Anokhin & Rohrbaugh, 1998) and association between the FA pattern and familial risk for a subtype of alcoholism combined with depression (Anokhin, Rohrbaugh, Vedeniapin, & Sirevaag, 1997). Another study suggested an association of FA with polymorphic variants of genes involved in dopaminergic and serotoninergic neurotransmission (Anokhin, Vedeniapin, Wu, Goate, & Rohrbaugh, 2000). Finally, a recent study(Coan, Allen, Malone, & Iacono, 2003) reported a modest heritability of FA in females and lack of heritability in males. Although these preliminary reports suggest varying degrees of genetic influences on FA, the evidence for heritability remains inconclusive.

The goal of the present study was to assess heritability of FA using a population-based sample of twins. In addition to this main goal, we addressed several ancillary questions. First, we examined whether heritability depends on frontal regions in which FA is typically measured: midfrontal (F3/F4) versus lateral frontal (F7/F8). Second, we examined the contribution of individual alpha sub-bands (lower, medium, and upper alpha) to twin correlations and heritability of FA. Finally, we examined the possibility that intrapair resemblance of MZ twins and, hence, heritability of FA may be influenced by the overall alpha power. We expected that genetic influences on FA would be more distinct in individuals with a well-pronounced frontal alpha as opposed to those with weaker alpha activity in the frontal scalp regions due to potential problems with obtaining reliable measures of asymmetry in the latter group.

METHODS

Subjects

The original sample included 264 female twin subjects including 77 MZ and 55 DZ pairs ascertained from the general population through a twin registry. Subjects were excluded if they had a history of serious head trauma or were using psychoactive medication at the time of testing; there was no exclusion due to depression and/or handedness. Zygosity was determined using a questionnaire administered to both twins. The reliability of zygosity diagnosis by questionnaire has been demonstrated in previous studies (Kasriel & Eaves, 1976). In addition, interviews with parents and blood test data were available for about 15% of the pairs. The study

was approved by Washington University Institutional Review Board and informed consent was obtained from all participants. The subjects were females because the EEG data were collected as part of a larger population-based twin study specifically focusing on risk factors for substance abuse in females. A total of 18 subjects (6.8% of the original sample, including 4 MZ and 5 DZ pairs) were excluded from the present analysis due to persistent artifacts in one or both twins (excessive movements or muscle activity, one or more poorly connected electrodes), signs of drowsiness in the EEG, and subjects' poor compliance with instruction such as opening eyes or talking during the recording. The remaining full pairs (73 MZ and 50 DZ) were included in the final data analysis (age range 17-27; mean age and standard deviation: 21.7±2.7).

Experimental procedure

Two 2-min resting EEG recordings with eyes closed were conducted at the beginning and in the second half of a longer experimental session involving several ERP paradigms, yielding a total of 4 min of the resting EEG. The average interval between the two recordings was 1.5 h. Co-twins were tested on the same day in 53% of the pairs, co-twins from other pairs were tested on different days, most often on consecutive days and at approximately the same time of day. Subjects were instructed to sit quietly with eyes closed, relax, and avoid major body movements and muscle tension. The EEG was recorded using Synamps-Neuroscan bioamplifier (Compumedics USA, Ltd) from 19 scalp locations according to the 10-20 system using an elastic cap with silver-chloride electrodes and a ground electrode on the forehead with a bandpass of 0.05 to 70 Hz. The left mastoid served as reference, and an averaged mastoid reference (A1 +A2) was digitally computed off-line using the right mastoid recording as a separate channel. The use of computer-linked mastoid reference for the measurement of frontal EEG asymmetry has been advocated in recent methodological publications (Allen, Coan et al., 2004; Hagemann, 2004; Hagemann, Naumann, & Thayer, 2001). Electric potentials associated with vertical or horizontal eye movements (EOG) were monitored using additional electrodes placed above and below the left eye and at the outer canthi of both eyes. During the experiment, subjects sat in a comfortable chair in a semi-darkened sound-attenuated booth.

EEG analysis

EEG recordings were divided into 2.048-s epochs and screened for artifacts related to movement, muscle tension, or poor electrode contact. Artifact-free epochs were subjected to fast Fourier transform (FFT), the power spectra for individual epochs were averaged, and the measures of EEG spectral power density in the following frequency bands were obtained: alpha-1 (7.5-9.5 Hz), alpha-2 (9.5-10.5 Hz), alpha-3 (10.5-13.0 Hz), and total alpha (7.5-13.0 Hz). Since the distribution of the power measures was skewed, square root values were used.

Frontal hemispheric asymmetry of the EEG spectral power was calculated as ((L−R)/(L+R)) *100, where L and R are square root power values at the homologous left and right hemisphere sites (F3 and F4, F7 and F8). Normalization by the sum of power at both leads provided an asymmetry measure which is independent of individual variability in the overall spectral power. Asymmetry indices were computed for each of the three alpha bands and for the total alpha power. In addition, we used asymmetry index based on the natural logarithm of spectral power (lnL−lnR) as an alternative measure, since the latter has been used in many recent studies. However, because the normalized and the logarithmic measures have been reported to be almost perfectly correlated (Allen, Coan et al., 2004) and showed very high correlation (r=0.998) in the present data, all results are presented for the normalized measure only.

Statistical analysis

For each of the EEG measures, we computed Pearson correlations between the members of twin pairs (intrapair correlations) separately for MZ and DZ twins. To estimate the relative

contribution of genetic and environmental sources to the total phenotypic variance of FA measures (heritability), we performed a biometrical genetic analysis using a model-fitting approach, which has become a standard in twin genetic research (Neale & Cardon, 1992; Rijsdijk & Sham, 2002). Linear structural equation models were fitted to the variance-covariance matrices obtained from MZ and DZ twins using the Mx package specifically developed to model genetically informative data (Neale, Boker, Xie, & Maes, 2002). These models assume that phenotypic variance arises from the following factors: additive genetic influences (A), non-additive genetic influences (D) or environmental influences shared by family members (C), and individually unique (unshared) environmental influences (E). Path coefficients corresponding to these factors were estimated using a maximum likelihood method and a χ^2 statistic was used to assess the goodness-of-fit of each model, where low χ^2 values indicate a good fit. The significance of individual paths such as A, C, or D was tested by dropping a path and comparing the goodness of fit of the reduced model with the full model (ACE or ADE). If dropping a path significantly reduced the goodness of fit (the χ^2 difference significant), the path was retained in the model, otherwise the more parsimonous model was chosen (i.e. the one that accounted for the variance equally well, but with a fewer number of parameters). In addition, different models were compared using Akaike's information criterion (AIC, computed as $\chi^2 - 2df$) that provides a combined measure of goodness-of-fit and parsimony of a given model at the same time. A model with the lower AIC was considered as better fitting. A detailed description of the model fitting approach can be found elsewhere (Evans, Gillespie, & Martin, 2002; Neale & Cardon, 1992; Rijsdijk & Sham, 2002).

RESULTS

The means and variances of the EEG measures did not differ between MZ and DZ twins, as indicated by one-way ANOVA and Levene's test for homogeneity of variances. There was a significant correlation between FA measures obtained at mid-frontal and lateral frontal locations ($r=.58$, $p<0.001$). Spectral power and FA measures did not correlate significantly with age. Twin correlations and the estimates of genetic and environmental components of variance for the frontal EEG measures are presented in Table 1. There was a modest, but significant MZ correlation for the midfrontal FA measure, whereas the DZ correlation was near-zero and not significant. Genetic model fitting showed that the AE model (additive genetic plus individual environmental influences) shows a good fit, suggesting that 27% of observed individual differences in this measure can be attributed to genetic factors, and the rest 73% to individually specific environmental factors. Although the relative size of the twin correlations suggests the possibility of non-additive genetic effects (MZ correlations are greater than twice the DZ correlations), discriminating statistically between these two sources of genetic variance would require a much larger sample, given the small size of both correlations. Both MZ and DZ correlations for the frontolateral FA measure were low and non-significant, suggesting a lack of familial resemblance with respect to this measure. Model-fitting showed that the environmental “E” model fits the data well (Table 1). Although the “AE” model also showed a good fit ($\chi^2=5.41$, $df=4$, $p=0.25$, $AIC=-2.59$), heritability estimate was non-significant (14%, with the 95% confidence interval including zero). Thus, the “E” model including only individual (non-shared) environmental factors appears to be the most parsimonious one.

In contrast, heritabilities of the alpha band power at all four frontal locations were very high and significant (85-87 %). For all power measures, the AE model showed a very good fit. Since the relative size of twin correlations (DZ correlation is greater than half the MZ correlation) suggested a possibility of shared environmental contribution, we also fitted the ACE model to the data. The ACE model also showed a good fit, however, the variance component estimates for the shared environmental factors were non-significant (confidence interval included zero), and there was no significant improvement in the goodness of fit by adding the shared

environmental component. Therefore, the results obtained using the most parsimonious AE model are presented.

To explore the possibility that genetic influences on mid-frontal FA measure derived from the total alpha power might be due to genetic influences on a specific alpha sub-band, we computed twin correlations for the three alpha sub-bands. MZ correlations were 0.31, 0.28. and 0.25 for alpha-1, alpha-2, and alpha-3, respectively (all significant at $p<0.05$, one tailed), suggesting that all three sub-bands contribute to MZ twin resemblance and heritability of the original FA measure. All DZ correlations were near-zero and non-significant.

Next, to determine whether the timing of the recording within the 2-hour experimental session (at the beginning versus at the end) affects the results, we computed twin correlations separately for the 2 min recordings at the beginning versus at the end of the session. For both halves of the data, the pattern of twin correlations was very similar to that presented in Table 1 (when both halves were collapsed), indicating a high consistency and replicability of the findings within one session.

Finally, we examined the possibility that twin similarity with respect to FA measures may be influenced by the overall EEG power. Since a substantial proportion of the human EEGs (up to 20%) do not show a distinct rhythmic alpha activity in frontal leads even in the eyes closed condition, inclusion of such subjects may introduce some noise into the data, since the estimation of alpha power and, hence, FA may be less reliable in such individuals. We recomputed MZ twin correlation for FA at F3/F4 after removing subjects with low activity using two subsequent cut-offs, the lower 10th and 33th percentiles on the average alpha power at F3 and F4. MZ twin correlation increased from 0.31 to 0.41 and 0.46, respectively, for the two cut-offs, although this increase was non-significant. To further test whether intrapair twin similarity is influenced by the overall alpha power, we computed the correlation between the overall frontal alpha power in a given twin pair (averaged for F3 and F4 and for both twins) and the absolute intrapair difference with respect to the FA index in MZ pairs. There was a modest, but significant negative relationship ($r=-0.29$, $p<0.05$), suggesting that MZ similarity increases with increasing overall alpha power. Finally, we divided our whole sample using median split on frontal alpha power (average of F3 and F4) and computed test-retest correlations between FA measured at the beginning and at the end of the session separately for the lower- and higher-alpha groups. These test-retest correlations were 0.38 and 0.70, respectively, and the difference between them was highly significant ($p<0.01$). Taken together, these results are consistent with our hypothesis that estimation of FA is less reliable in individuals with low alpha activity in frontal regions compared to high-alpha individuals.

DISCUSSION

The main finding of the present study is that frontal EEG asymmetry (FA) shows only a modest degree of heritability in young adult females when assessed at the mid-frontal locations, with less than 30% of the total variance being attributable to genetic factors. No evidence of genetic influences was found for FA assessed at the lateral frontal locations. In contrast, alpha power is highly heritable at all four frontal sites, consistently with previous studies (van Beijsterveldt, Molenaar, de Geus, & Boomsma, 1996). The finding of modest, but significant genetic influences on FA in females is consistent with previous reports of familial transmission and modest heritability (Anokhin & Rohrbaugh, 1998; Coan et al., 2003). Since previous test-retest reliability studies suggest that about 60% of the variance in FA are due to stable individual differences and the remaining 40% are due to occasion-specific factors (Hagemann et al., 2002), the present findings suggest that approximately one-half of the stable inter-individual differences in FA in mid-frontal regions are due to genetic factors, whereas the other half is probably due to individually specific environmental factors. These findings were unaffected

by such factors as sub-band within the alpha band (lower-, medium-, or upper alpha frequency range) and the relative timing of the recording within experimental session.

A major implication of the present findings is that FA has a limited value as an endophenotype for genetic studies of the liability to mood disorders, at least in population-based samples.

Our previous study based on family data (Anokhin & Rohrbaugh, 1998) suggested somewhat higher heritability (0.46, based on parent-offspring correlations). However, these data did not permit to draw definite conclusions regarding heritability, since familial resemblance can result both from genetic influences and common environmental influences. Moreover, the sample in that study included both control families and families selected for multigenerational history of alcohol dependence that were characterized by a high prevalence of psychiatric conditions frequently co-occurring with alcoholism, including major depression. As a result, oversampling families from an extreme end of the liability distribution could lead to some overestimation of familial resemblance on FA.

Individual sub-bands of the alpha frequency band seem to contribute equally to heritability of the FA index derived from the overall alpha band power, although there was a weak insignificant trend towards higher MZ correlations for the lower alpha sub-band compared to the medium and high alpha.

The finding of relatively low heritability of FA, despite the evidence for the association of FA with temperamental traits and liability to mood disorders, points to substantial developmental plasticity of the neural bases of emotion and suggests that stable individual difference in the neural circuitry of emotion, at least its prefrontal cortical components, can be largely shaped by individual experience, rather than by genetic factors. Indeed, the prefrontal areas of the cortex have a longer developmental period which extends into late adolescence and even early adulthood (Casey, Giedd, & Thomas, 2000), which is reflected in the developmental course of some EEG characteristics (Anokhin, Birbaumer, Lutzenberger, Nikolaev, & Vogel, 1996). This prolonged development of prefrtontal cortex results in its greater exposure to environmental influences compared to other cortical areas. It can be speculated that some specific aspects of prefrontal development related to inter-hemispheric relationships may be particularly sensitive to individual experiences in the course of development.

The present results suggest that the degree of twin resemblance with respect to FA may to some extent depend on the overall level of alpha activity in the frontal leads: MZ twins with scarce alpha activity at frontal leads tended to be less similar with respect to FA compared with those pairs who showed a well-pronounced frontal alpha. Given that test-retest reliability theoretically serves as an upper limit for heritability estimates, these results point to the possibility that assessment of FA may be more reliable in individuals with well pronounced alpha activity in the frontal leads. In contrast, the measurement of FA in individuals with weak frontal alpha may be less reliable, leading to low MZ twin resemblance. This possibility can be tested in future test-retest reliability studies, e.g. by regressing within-subject test-retest differences on the overall alpha power.

Some potential limitations of this study should be acknowledged. First, the assessment of FA was based on a relatively short EEG recording (a total of 4 min with eyes closed). Although some earlier studies used longer recordings (e.g. 8 min), it has been demonstrated recently that highly reliable measures of FA can be obtained with considerably shorter recordings, and even a 2 min recording can provide similarly reliable estimates (Allen, Urry, Hitt, & Coan, 2004; Coan, Allen, & Harmon-Jones, 2001). Because an increase in EEG recording length (e.g. from 4 to 6 or 8 min) produces only a very modest gain in reliability of FA measurement (Allen, Urry et al., 2004), the recording length is not likely to affect heritability estimates in any substantial way. Next, due to the modest sample size and relatively broad confidence intervals

for FA measures, the reported point estimates of heritability should be interpreted with caution. It should be also kept in mind that the heritability statistic pertains to a particular population at a particular time, and heritability estimates may be different in other groups, although in practice such differences are modest for most of the studied physiological and behavioral traits. Finally, the present study included female twins only, and there is a possibility that heritability of FA in males may be different. Although previous studies did not find gender effects on heritability of the absolute alpha power (van Beijsterveldt et al., 1996), a recently published abstract suggests a modest heritability of frontal alpha asymmetry in females, but not in males (Coan et al., 2003).

In conclusion, the present findings suggest that genetic influences on FA measure in females are very modest and FA is unlikely to be a useful biological marker (endophenotype) of genetically transmitted liability for mood disorders.

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Table 1

Twin correlations and heritability estimates for frontal EEG asymmetry (FA) and regional alpha power.

EEG measure	Mean±SD	r_{MZ} (n=73)	r_{DZ} (n=50)	a^2 (95% CI)	e^2 (95% CI)	χ^2	df	p	AIC
FA, mid-frontal (F3/F4)	−1.06±4.7	.31**	.03	.27 (.06-.45)	.73 (.55-.94)	7.34	4	.12	−0.66
FA, lateral-frontal (F7/F8)	−0.92±6.2	.15	.08	--	1.00	4.17	5	.52	−5.83
Alpha power, F3	5.26±2.0	.87***	.57***	.87 (.80-.91)	.13 (.09-.20)	5.41	4	.25	−2.59
Alpha power, F4	5.36±2.0	.88***	.55***	.86 (.80-.91)	.14 (.09-.20)	6.87	4	.14	−1.13
Alpha power, F7	4.07±1.6	.87***	.55***	.87 (.80-.91)	.13 (.09-.20)	3.92	4	.42	−4.08
Alpha power, F8	4.10±1.5	.86***	.58***	.85 (.78-.89)	.15 (.11-.22)	3.56	4	.47	−4.44

Notes: Mean amplitude values in microvolts (square root of spectral power density) are presented for the total alpha band (7.5-13 Hz). r_{MZ} and r_{DZ} are intrapair correlations for MZ and DZ twins, respectively. Variance component estimates are based on the best-fitting "AE" model for all variables except lateral-frontal FA and on the "E" model for the latter: a^2 is the proportion of total phenotypic variance explained by genetic factors (heritability); e^2 is the proportion of variance due to environmental factors (95% confidence intervals of the maximum likelihood estimates of the variance components are shown in brackets). Chi-square shows the goodness of fit, with lower values indicating a better fit; AIC is Akaike's Information Criterion. Significance levels:

* p<0.05

** p<0.01

*** p<0.001.