

Individual Differences in EEG Spectral Power Reflect Genetic Variance in Gray and White Matter Volumes

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The human electroencephalogram (EEG) consists of oscillations that reflect the summation of postsynaptic potentials at the dendritic tree of cortical neurons. The strength of the oscillations (EEG power) is a highly genetic trait that has been related to individual differences in many phenotypes, including intelligence and liability for psychopathology. Here, we investigated whether brain anatomy underlies these EEG power differences by correlating it to gray and white matter volumes (GMV, WMV), and additionally investigated whether this association can be attributed to genes or environmental factors. EEG was measured in a sample of 405 young adult twins and their siblings, and power in the theta (~4 Hz), alpha (~10 Hz), and beta (~20 Hz) frequency bands determined. A subset of 121 subjects were also scanned in a 1.5 T MRI scanner, and gray and white matter volumes defined as the total of cortical and subcortical volumes, excluding cerebellum. Both MRI-based volumes and EEG power spectra were highly heritable. GMV and WMV correlated .25 to .29 with EEG power for the slower oscillations (theta, alpha). Moreover, these phenotypic correlations largely reflected genetic covariation, irrespective of oscillation frequency and volume type. Genetic correlations ($.31 < r_A < .43$) revealed that only moderate proportions of the heritable variance overlapped between MRI volumes and EEG power. The results suggest that MRI volumes and EEG power share genetic sources of variation, which may reflect such processes as myelination, synaptic density, and dendritic outgrowth.

■ **Keywords:** Gray matter volume, white matter volume, electroencephalography, endophenotype, MRI, development

Electroencephalographic (EEG) recordings of electrical brain activity during rest and task conditions have been a prime target to serve as endophenotypes of brain function, cognition, and brain disorder. EEG consists of oscillations that reflect the summation of postsynaptic potentials at the dendritic tree of cortical neurons. These constituent oscillations include alpha oscillations of about 10 Hz that reflect the inactivity of the cortical area producing the rhythm (Niedermeyer, 1999a). Theta rhythms of 4 Hz to about 6 Hz, represent working memory processing in adults (Gevins et al., 1997), but may reflect the relative immaturity of the brain (Niedermeyer, 1999b; Smit et al., 2011) or drowsiness (Niedermeyer, 1999c). Beta oscillations of about 15 to 25 Hz reflect active processing or movement-related oscillations (Niedermeyer, 1999c).

A large body of research has linked EEG parameters to cognitive ability (Anokhin & Vogel, 1996; Jaušovec & Jaušovec, 2000; Onton et al., 2005; Tesche & Karhu, 2000; Thatcher et al., 2008) and various psychopathologies including ADHD, depression, alcoholism, and autism (Barry et al., 2003; Cameron et al., 2003; Kemner et al., 1999; Linkenkaer-Hansen et al., 2005; Porjesz & Begleiter, 2003). Many EEG parameters are influenced by genetic factors. For example, the strength of EEG oscillation (EEG power),

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connectivity between brain areas (measured with EEG coherence or synchronization likelihood), and more complex measures (network efficiency, dynamical signal complexity, and the decay in autocorrelation) all show moderate to high heritability (Almasy et al., 1999; Anokhin et al., 2001; Posthuma et al., 2005; Smit et al., 2005, 2008, 2010; van Baal et al., 1996; van Beijsterveldt et al., 1996, 1998a, b; Zietsch et al., 2007). As such, these EEG-derived parameters fulfill two important criteria of the endophenotype concept (de Geus, 2002): they are related to the (heritable) phenotype, and they are heritable traits.

Increased use of structural and functional magnetic resonance imaging (MRI) scans in cognitive and clinical neuroscience also has produced a large body of evidence linking MRI volumetric measures to brain function, cognition, and brain disorder. As with the EEG parameters, MRI volumetric brain measures tend to be highly heritable, both in childhood and later in life. This is seen for whole-brain measures of cerebral gray and white matter volumes (GMV and WMV), cortical thickness, and volumes of subcortical structures such as hippocampal volume (Hulshoff Pol et al., 2006; Peper et al., 2007; Posthuma et al., 2002, 2003; van Leeuwen et al., 2009).

Variation in both EEG power and MRI volumes has often been related to the same phenotypes. For example, EEG power and MRI volumes show correlations with cognitive performance across normal and patient subject groups. Both WMV and GMV show correlations with intelligence ranging from .25 to .40 (Luders et al., 2009; Posthuma et al., 2002). EEG power of various frequencies has also been found to be significantly correlated with IQ in several studies, including 10 Hz alpha (Thatcher et al., 2005) and 6 Hz theta in elderly subjects (Finnigan et al., 2011), albeit with slightly smaller effect sizes (about $r = 0.20$). Schizophrenia may serve as another example. Here, a triad of effects has been reported between gray matter changes, IQ deterioration, and ~10 Hz alpha power reduction (Leeson et al., 2011; Sponheim et al., 1994; Sun et al., 2009). In sum, for many phenotypes that have correlations with brain volume, correlations with EEG power have also been reported.

In sum, MRI volumes and EEG-derived parameters show substantial overlap in their relation with brain function, cognition, and brain disorder. This overlap in the individual differences in EEG and MRI may perhaps be explained by the same neurodevelopmental processes, notably including myelination and synaptic formation and pruning (Casey et al., 2005; Huttenlocher, 1979; Huttenlocher & Dabholkar, 1997; Lenroot & Giedd, 2006; Paus, 2010). If so, they would be expected to share a common etiology, including genetic and environmental determinants. Here we will focus on the relation between EEG power and MRI volumes. Whitford et al. (2007) showed that EEG power and MRI volumes are indeed correlated (in the range $r = .24$ to $r = .30$ for alpha oscillations). We aim to replicate these findings in a sample of healthy adults and test for significance of phenotypic

correlations between EEG power and MRI volumes. Next, we aim to make use of the extended twin design of monozygotic (MZ) and dizygotic (DZ) twins (and their siblings) to investigate through multivariate genetic modeling whether MRI volumes and EEG indeed share part of their genetic and environmental determinants.

Methods

Subjects

Subjects were recruited from The Netherlands Twin Registry as part of projects on the genetics of cognition and adult brain functions. Adult twins and their non-twin siblings were asked to participate in a testing protocol lasting 4.5 hours. In total, 405 twins and siblings (213 female; 192 male) from 160 twin families participated in the study. The sample consisted of a young adult age cohort ($M = 26.2$ years, $SD = 4.1$). The sample included 61 MZ and 77 DZ complete twin pairs, and 114 siblings. Of these, 121 subjects (average age 27.4 years) also took part in an MRI study (48 male, 73 female; 20 complete MZ pairs, 23 complete DZ twin pairs, and 25 siblings).

EEG Acquisition and Analysis

A detailed description of EEG acquisition can be found elsewhere (Smit et al., 2005). In short, we obtained 3-minute eyes closed background EEG on 19 scalp positions (F7, F3, F1, Fz, F2, F4, F8, T7, C3, Cz, C4, T8, P7, P3, Pz, P4, P8, O1, and O2) using Ag/AgCl electrodes mounted in an electrocap. Vertical and horizontal eye movement was measured using bipolar derivations. The EEG was amplified, digitized at 250 Hz, and stored for offline processing.

All EEG signals were re-referenced to average earlobes. After visual inspection, Independent Components Analysis was performed and components reflecting eye movements removed. A minimum of 100 s of data was required. Power was calculated using Thomson's multitaper method as implemented in MATLAB. Finally, the resulting power values were $10 \times \log_{10}$ transformed averaged into the theta (3.0–5.4 Hz), alpha (6.0–13.0 Hz) and beta (15.0–25.0 Hz) frequency bins.

Structural MRI Assessment

For the 121 subjects, MRI scans were acquired using a Philips 1.5 T Intera scanner (Philips, Best, The Netherlands) at the University Medical Center Utrecht. For a detailed description of the acquisition and processing of the scans of this sample, see Baaré et al. (2001). In short, the T1-weighted images (voxel size $1 \times 1 \times 1.2 \text{ mm}^3$) were transformed into Talairach orientation (no scaling; Talairach & Tournoux, 1988) and corrected for magnetic field inhomogeneities (Sled et al., 1998). Segments of gray and white matter of the cerebrum were obtained by an automated method validated earlier (Schnack et al., 2001), from which tissue volumes were calculated.

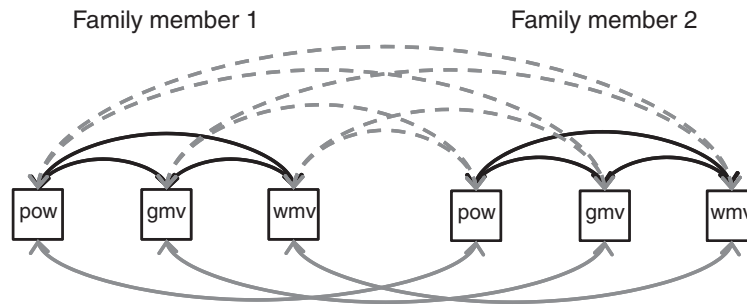


FIGURE 1

Trivariate saturated model used in the statistical analysis of EEG power (pow), gray matter volume (gmv), and white matter volume (wmv). The model estimates phenotypic correlations (black arrows), twin correlations (gray arrows), and cross-twin-cross-trait correlations (CTCT; dashed gray arrows). Family members can be monozygous (MZ) twins, dizygous (DZ) twins, or twin-sibling pairs with separate CTCT and twin correlations. Correlations between DZ twin pairs and twin-sibling pairs were fixed to be equal. All models used age-fixed and sex-fixed effects on the means. Separate analyses were performed for EEG oscillation power in the three frequency bands and are listed as Model 1 in Table 4.

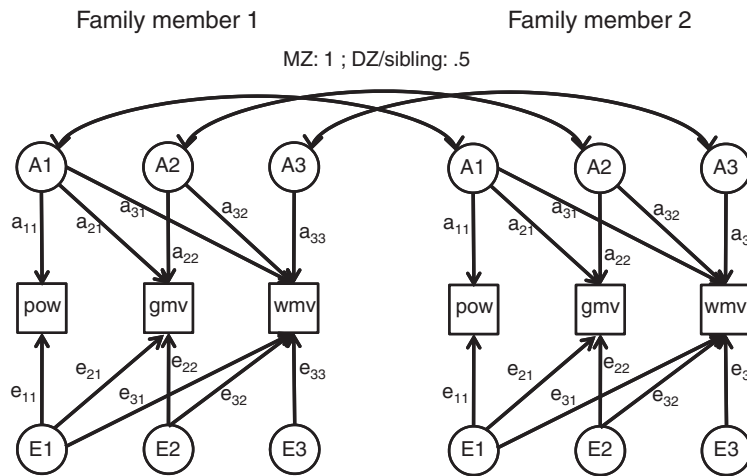


FIGURE 2

Trivariate path model used in the statistical analysis of EEG power (pow), gray matter volume (gmv), and white matter volume (wmv). Family members can be monozygous (MZ) twins, dizygous (DZ) twins, or twin sibling pairs. The path model describes the relation between pairs of family members, but can easily be expanded to include > 2 family members. Additive genetic factors (A) are correlated 1 between MZ twins, 0.5 between DZ twins and siblings, and 0 between unrelated subjects. Unique environmental factors (E) are uncorrelated across family members, but may still mediate phenotypic correlation between the variables. Separate analyses were performed for EEG oscillation power in the three frequency bands and are listed as Model 5 in Table 4. Model 10 in Table 4 (theta and alpha oscillations) is the same model with path loadings e21 and e31 removed. Model 10 (beta oscillations) has path loadings e21, e31, a21, and a31 removed.

Statistical Methods

Phenotypic correlations between MRI volumes and EEG power, and correlations between MZ and DZ/Sib pairs for all traits, as well as cross-twin-cross-trait correlations among MRI and EEG traits, were estimated in a so-called saturated model in trivariate models with EEG power, GMV, and WMV, which specifies correlations among relatives, but does not put any constraints on the correlational structure. In addition, variances are freely estimated for each variable. Previous results had suggested no sex differences in correlation structure for EEG power (Smit et al., 2005; Zietsch et al., 2007) or for MRI volumes

(Baaré, 2001), and we therefore did not model this effect. Note, however, that all models include sex and age as linear fixed effects (covariates). Figure 1 shows the saturated model.

These trivariate saturated models formed the basis for the trivariate genetic models that were applied to the data. As previous studies found no empirical evidence that resemblance among family members for EEG or MRI characteristics is influenced by common environment shared by family members, the genetic models included two types of latent (unobserved) factors that explained trait variance and covariance among twins and siblings. Additive genetic

TABLE 1
Heritabilities and Twin Correlations of EEG Power and MRI Volumes

Variable	h^2	r_{MZ}	r_{DZ}
EEG power			
theta	0.76 [0.66, 0.84]	0.77 [0.67, 0.84]	0.35 [0.21, 0.48]
alpha	0.93 [0.89, 0.95]	0.93 [0.89, 0.95]	0.51 [0.38, 0.61]
beta	0.85 [0.78, 0.89]	0.85 [0.77, 0.90]	0.56 [0.45, 0.66]
GMV	0.73 [0.56, 0.84]	0.71 [0.49, 0.84]	0.36 [0.09, 0.59]
WMV	0.90 [0.77, 0.95]	0.89 [0.76, 0.94]	0.27 [0.00, 0.51]

Note: GMV = gray matter volume, WMV = white matter volume; values in brackets are 95% Confidence Intervals; EEG power from oscillations in the theta (3.0–5.6 Hz), alpha (6.0–13.0 Hz) and beta (15.0–25.0 Hz); heritability from multivariate AE models; twin correlations from saturated models.

(A) factors can explain resemblance among family members; they correlate 1 between MZ twins and, on average, .5 between DZ twins and siblings. Unique environmental factors (E) cause differences between family members, including between MZ twins. The effect of these latent factors on the observed, univariate, or multivariate phenotypes can be estimated by genetic structural equation modeling. Figure 2 shows the path model used. Data were analyzed in a series of trivariate models including GMV, WMV, and EEG power (separate models for the alpha, beta, and theta frequency bands). The model included three latent A and three latent E factors; the first A and E factors influence all 3 traits, the second A and E factors influence only 2 traits and the last A and E factor only influences one trait. Through this parameterization, the phenotypic covariance matrix is decomposed into a genetic and an environmental part ($\Sigma_P = \Sigma_A + \Sigma_E$). Standardization of each of these matrices provides phenotypic, genetic, and environmental correlations among traits. Note that the correlations do not necessarily add up ($r_P \neq r_A + r_E$) due to each covariance being standardized to different variances.

Statistical modeling was done in Mx version 1.54 (Neale et al., 2003). Mx computes the likelihood of a model based on the observed and expected (modeled) covariance matrices, then maximizes the likelihood by stepwise changing of the model parameters. After fitting parameters for the full saturated model, we applied constraints; for example, by constraining the values of parameters to each other or by dropping them to zero. This results in a nested sub-model with a different likelihood. Twice the difference in likelihood between the full model and a nested sub-model with restricted number of parameters is asymptotically chi-square distributed, with the number of constrained parameters as degrees of freedom. If the test is significant, this indicates that the parameter significantly contributes to the model and needs to be retained.

Specifically, after fitting the saturated model we tested for the significance of the phenotypic correlation by setting it to zero. If significant, we tested whether the AE model provided a nonsignificant deterioration of model fit. In this model we tested whether the correlation between GMV and EEG

TABLE 2
Phenotypic Correlations Between EEG Power and MRI Volumes

EEG power frequency	MRI volume	
	GMV	WMV
<i>Phenotypic r</i>		
theta	.28 [.09, .45]	.27 [.07, .44]
alpha	.29 [.06, .47]	.25 [.03, .45]
beta	.01 [−.08, .23]	.04 [−.05, .26]
<i>Genetic r</i>		
theta	.43 [.14, .69]	.31 [.04, .54]
alpha	.41 [.16, .61]	.33 [.10, .52]
beta	ns	ns
<i>Environmental r</i>		
theta	−.08 [−.43, .32]	.21 [−.26, .59]
alpha	−.02 [−.36, .34]	.27 [−.14, .59]
beta	ns	ns
<i>Proportion genetic</i>		
theta	1.07 [.66, 1.62]	.88 [.28, 1.17]
alpha	1.01 [.80, 1.23]	.93 [.67, 1.04]
beta	ns	ns

Note: GMV = gray matter volume, WMV = white matter volume; values in brackets are 95% Confidence Intervals; EEG power in theta (3.0–5.6 Hz), alpha (6.0–13.0 Hz) and beta (15.0–25.0 Hz) frequency bands. Correlations were estimated in the saturated model. *Genetic r* represents the correlation between additive genetic factors and, thus, the proportion of heritable variance shared between the two phenotypes; *Environmental r* represents the correlation between the environmental factors and, thus, the proportion shared environmental variance between the two phenotypes; *Proportion genetic* represents the proportion of the phenotypic correlation that can be attributed to genes; values > 1 indicate opposite sign in environmental and genetic correlations. ns = no significant phenotypic correlation.

power was mediated by genetic or environmental factors by dropping path loadings e21 and a21 respectively (see Figure 2). Similarly, the etiology of the relation between WMV and EEG power was tested by dropping e31 and a31. Nonsignificant path loadings were removed. Finally, we tested the significance of the genetic effects on each of the variables EEG power, GMV, and WMV by dropping all path loadings from the variables to A.

Results

The twin correlations from the saturated models are shown in Table 1. These show very high MZ twin correlations for all measures. Moreover, the DZ twin/sibling correlations are around half the MZ correlation, suggesting that non-additive genetic effects or common environment do not play a significant role. Phenotypic correlations are shown in Table 2, and cross-twin-cross-trait correlations (CTCT) are shown in Table 3. These indicate that there are small to moderate correlations between EEG power and MRI volumes for alpha and theta oscillations. The MZ CTCT correlations are about the same size as the phenotypic correlation, indicating a genetic etiology for this relationship. Genetic correlations and the proportion of the phenotypic correlation attributable to genetic effects are also shown in Table 2. These reveal that, although the phenotypic correlations are 100% genetic, EEG power and MRI volumes show only moderate overlap (31% to 43%) in genetic variance.

TABLE 3
Cross-Twin-Cross-Trait Twin Correlations Between EEG Power and MRI Volumes

EEG power frequency	GMV		WMV	
	CTCT _{MZ}	CTCT _{DZ}	CTCT _{MZ}	CTCT _{DZ}
theta	.28 [.07, .47]	.15 [−.04, .33]	.25 [.04, .44]	.00 [−.20, .19]
alpha	.29 [.06, .48]	.05 [−.17, .27]	.23 [.01, .43]	−.06 [−.28, .17]
beta	.01 [−.19, .24]	−.09 [−.25, .05]	.05 [.00, .26]	−.12 [−.30, .11]

Note: GMV = gray matter volume, WMV = white matter volume, CTCT = cross-twin-cross-trait twin correlations, MZ = monozygous, DZ = dizygous; values in brackets are 95% Confidence Intervals; EEG power from oscillations in the theta (3.0–5.6 Hz), alpha (6.0–13.0 Hz) and beta (15.0–25.0 Hz); estimated in the saturated model.

TABLE 4
Multivariate Model Fit Between EEG Power and MRI Volumes

Model description	Model	Tested against	Oscillation frequency																
			theta (~4 Hz)				alpha (~10 Hz)				beta (~20 Hz)								
			-2LL	df	χ^2	df p	-2LL	df	χ^2	df p	-2LL	df	χ^2	df p					
Saturated	1		1920.37	609					2024.7	609					1816.21	609			
Significance R(Pow, GMV)	2	1	1928.36	610	8	1	.005		2030.8	610	6.1	1	.013		1816.22	610	0.01	1	.933
Significance R(Pow, WMV)	3	1	1927.14	610	6.77	1	.009		2029.67	610	4.97	1	.026		1816.4	610	0.19	1	.666
Significance R(GMV, WMV)	4	1	1962.01	610	41.65	1	<.001												
AE	5	1	1925.86	615	5.49	6	.482		2032.05	615	7.35	6	.29		1827.84	615	11.63	6	.071
R(Pow, GMV) 100% A	6	5	1926	616	0.14	1	.709		2032.06	616	0.01	1	.92	ns					
R(Pow, WMV) 100% A	7	5	1926.65	616	0.79	1	.373		2033.73	616	1.68	1	.195	ns					
R(Pow, GMV) 100% E	8	5	1934.23	616	8.37	1	.004		2041.56	616	9.51	1	.002	ns					
R(Pow, WMV) 100% E	9	5	1930.83	616	4.97	1	.026		2039.45	616	7.4	1	.007	ns					
Constraints from 6–9	10		1935.3	619					2043.06	619					1828.98	620			
Drop A Pow	11	10	2021.08	620	85.78	1	<.001		2222.11	620	179.05	1	<.001		1973.3	621	144.32	1	<.001
Drop A GMV	12	10	2009.47	621	74.17	2	<.001												
Drop A WMV	13	10	2008.86	621	73.56	2	<.001												

Note: Pow = EEG power, GMV = cerebral grey matter volume, WMV = cerebral white matter volume; EEG power from oscillations in the theta (3.0–5.6 Hz), alpha (6.0–13.0 Hz) and beta (15.0–25.0 Hz); model fit from a trivariate SEM model with sex and age as covariates on all variables. Model 5 is the AE model (Figure 2) and is equivalent to a saturated model with all r_{DZ} and $CTCT_{DZ}$ constrained to .5 times their MZ counterparts, and therefore nested in the saturated model.

Table 4 shows the results from the formal testing of these correlations for each of the frequency bands. For both alpha and theta oscillations, the phenotypic correlations were significant for both WMV and GMV, $\chi^2(1) > 4.97$, $p < .05$. Next, we observed that the saturated model could be constrained to an AE model, $\chi^2(6) < 11.6$, $p > .05$. Note that this model is equivalent to constraining all DZ correlations (including DZ cross-twin cross-trait correlation) to half the MZ equivalent, and is therefore nested in the saturated model. This test provides further evidence that non-additive genetic effects and effects of common environment are absent for both EEG power and MRI volumes. Next, we tested the source of the phenotypic correlations in the AE model. Path loadings e21 and e31 could both be dropped, $\chi^2(1) < 1.59$, but not path loadings a21 and a31, $\chi^2(1) > 4.97$, $p < .05$. Thus, for alpha and theta band EEG power, the phenotypic correlations with GMV and WMV were entirely explained by additive genetic factors. The heritabilities for power and MRI volumes were highly significant and are shown in Table 1.

Table 5 shows the effects of the covariates on the mean. The effect of sex on MRI volumes was quite large and highly significant, whereas the effect of age was much weaker — although a significant yearly decrease was found for GMV (see

e.g., Walhovd et al., 2005). EEG power showed well-known effects of higher power in females and a strong decrease in theta power (Smit et al., 2011).

Discussion

We have shown that the power of EEG alpha and theta oscillations was significantly correlated with MRI-derived WM and GM volumes in a sample of young adult subjects. We therefore fully replicated the results reported by Whitford et al. (2007), who found similar correlations ($.24 < r < .30$ for alpha oscillations, $.21 < r < .36$ for combined delta-theta oscillations). In addition, we have shown for the first time that these correlations are entirely genetically mediated.

The results show that beta band power did not reflect variance in either GMV or WMV. This discrepancy between theta/alpha and beta EEG power may seem surprising because significant phenotypic and genetic overlap between alpha and beta EEG power has been observed. Genetic and phenotypic correlations between beta and the lower frequencies (alpha and theta) were all around .60 (Smit et al., 2005). Clearly, this overlap between beta and alpha/theta oscillations does not include variance related to MRI volumes and may help elucidating what factors underlie the relationship between EEG power and MRI volumes.

TABLE 5
Means and Effects of Covariates

Variable	M	b _{age}	b _{sex}
EEG power			
alpha (dB)	4.1	-.04	-.68*
beta (dB)	-4.8	-.01	-.84***
theta (dB)	3.5	-.13***	-.65**
MRI volumes			
GMV (ml)	653.1	-2.8*	56.7***
WMV (ml)	461.2	2.0	77.8***

Note: Age covariate as increase in dB per year (EEG power) or ml per year (MRI volumes); sex covariate coded as males relative to females; GMV = cerebral grey matter volume, WMV = cerebral white matter volume.

* $p < .05$, ** $p < .01$, *** $p < .001$.

EEG power reflected only a subset of the genes causing variation in MRI volumes. This is consistent with the view that MRI volumes reflect multiple processes — pruning and dendritic outgrowth, and increased axonal myelination causing increases in WMV and decreases in GMV (e.g., Casey et al., 2004; Lenroot & Giedd, 2006). EEG power, on the other hand, is likely to reflect more directly the postsynaptic potentials of the apical dendrites of cortical pyramidal cells, and to reflect the synaptic density and the tendency of the thalamo-cortical loops to oscillate. Nevertheless, EEG power and MRI volumes pick up shared genetic factors that influence individual differences in two — at first sight — rather diverse brain measures.

What is the nature of these genetic factors and how do they cause the association between EEG power and MRI volumes? Formally, three explanations exist, which may also co-occur. First, the shared genetic factor observed in this study may cause variation in the volumetric measures, in turn causing variance in the EEG power spectrum. For example, there may be GMV-related alterations in conductive properties of the neural tissue, and WMV could be related to EEG power as the result of increased effectiveness in cortico-cortical and thalamo-cortical connectivity, both important aspects in oscillation generation (Steriade, 1999). Second, the correlated activity reflected in EEG power could actually cause volumes to increase because increased activity may cause extended dendritic branching (Peng et al., 2009; Sin et al., 2002; Yu & Malenka, 2003). Third, both EEG power and MRI volumes may show individual differences as a result of underlying differences in neurohistological variables such as dendritic arborization, expansion, synaptic density, and myelination. A possible design to tackle the core question of causality is the longitudinal assessment of both types of parameters in large twin samples (De Moor et al., 2008).

Although EEG power reflected only part of the (genetic) variation in MRI volumes, other — and perhaps more complex — derivations of the EEG signal than EEG power may provide additional explanatory power. Such variables have often been shown to be heritable, including dynamic com-

plexity (Anokhin et al., 1999), connectivity (van Baal et al., 2001; Posthuma et al., 2005; Smit et al., 2010), decay in temporal autocorrelations (Linkenkaer-Hansen et al., 2007), and graph theoretical analysis of the brain network (Smit et al., 2010; Smit et al., in press). It was also observed that these measures are, to at least some degree, not correlated with EEG power. For example, the power-law decay in autocorrelations is largely unrelated to EEG power (Linkenkaer-Hansen, 2007). Also, connectivity was, to some degree, genetically uncorrelated with EEG power (Smit et al., 2010).

MRI volumes, and especially GMV, have well-documented changes in psychopathological states such as ADHD (Casey et al., 1997; Castellanos et al., 2002; Durston et al., 2004; Rubia et al., 1999) and schizophrenia (Chua et al., 2007; Gur, Cowell, et al., 2000; Gur, Turetsky, et al., 2000; Honea et al., 2005; Hulshoff Pol et al., 2001; Wright et al., 2000). The current results suggest that EEG could serve as an endophenotype for the structural brain changes characterizing these disease states. Its usefulness may benefit from source localization of EEG activity to the implied cortical structures (Wright et al., 2000), compared to the whole-brain approach used in the current study. Future investigations may reveal whether these brain-projected EEG power values predict — as one would expect — cortical volumes of the same brain areas.

In sum, these results show that simple, inexpensive, and widely available EEG recordings reflect genetic structural brain parameters. Studies that require large numbers of observations — such as genome-wide studies — may benefit from the more widespread availability of EEG apparatus when exploring the genetics of structural brain parameters. Replication of loci with significant genome-wide association could include cohorts with resting EEG power data. Rather than focusing on complex compound measures, focusing on the covariation between EEG power and MRI volumes may isolate specific processes such as synaptic density that underlie both traits.

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