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Heritability of background EEG across the power spectrum

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Abstract

We estimated the genetic and nongenetic (environmental) contributions to individual differences in the background EEG power spectrum in two age cohorts with mean ages of 26.2 and 49.4 years. Nineteen-lead EEG was recorded with eyes closed from 142 monozygotic and 167 dizygotic twin pairs and their siblings, totaling 760 subjects. We obtained power spectra in 24 bins of 1 Hz ranging from 1.0 to 25.0 Hz. Generally, heritability was highest around the alpha peak frequency and lower in the theta and delta bands. In the beta band heritability gradually decreased with increasing frequency, especially in the temporal regions. Genetic correlations between power in the classical broad bands indicated that half to three-quarters of the genetic variance can be attributed to a common source. We conclude that across the scalp and most of the frequency spectrum, individual differences in adult EEG are largely determined by genetic factors.

Descriptors: Twin study, Temporal stability, Heritability, Genetic correlation

Recordings of resting background EEG show striking interindividual differences (Vogel, 2000). In part, these differences can be described in a qualitative way, for example, the presence or absence of low-voltage EEG, defined as resting EEG without rhythmic activity and with low amplitude that occurs in about 4% of the adult population or, at the other extreme, the presence of continuous alpha waves in an estimated proportion of also about 4% of the adult population (Vogel, 1970). More common, however, is the quantitative description of the individual differences in the EEG traces by the amplitude or power spectrum.

Background EEG power has been linked with various forms of psychopathology. For example, increased theta power and theta/beta ratio is found in Attention Deficit Hyperactivity Disorder (Barry, Clarke, & Johnstone, 2003; Bresnahan & Barry, 2002; Chabot & Serfontein, 1996; Clarke, Barry, McCarthy, & Selikowitz, 2001; Clarke et al., 2003; Jasper, Solomon, & Bradley, 1938; Monastra et al., 1999; Satterfield, Cantwell, Saul, Lesser, & Podosin, 1973), and increased beta power is found in (a predisposition to) alcoholism (Ehlers & Schuckit, 1990, 1991; Gabrielli et al., 1982; Propping, 1977; Rangaswamy et al., 2002; Van Sweden & Niedermeyer, 1999; Vogel, 2000). Therefore, understanding interindividual variance in EEG power could provide clues to the underlying neurobiology of these disorders.

A first step is the partitioning of interindividual variance in EEG power into genetic and environmental parts. This can be done in twin studies that compare the intrapair resemblance between two types of sibling relationships, namely genetically identical (monozygotic twins, MZ) and nonidentical twins (dizygotic twins, DZ). If MZ resemblance for EEG power is higher than DZ resemblance, this constitutes evidence for genetic influences on the EEG. A simple formula by Falconer (1960) computes the relative contribution of genetic influences to the total variance, also called heritability (h^2), as twice the difference in MZ/DZ resemblance:

$$h^2 = 2(r_{MZ} - r_{DZ})$$

where r_{MZ} and r_{DZ} quantify the intrapair resemblance for MZ and DZ twins. Observations from the early years of electroencephalography already have shown that EEG tracings of MZ twins show remarkable resemblance (Davis & Davis, 1936), and more so than those of DZ twins (Lennox, Gibbs, & Gibbs, 1945; Loomis, Harvey, & Hobart, 1936). In more recent approaches, Falconer's formula has given way to maximum likelihood techniques that can use more information than twin correlations alone (Jinks & Fulker, 1970). These models can include data from both types of twins (MZ, DZ) as well as from singleton siblings. By fitting biometric models of sibling resemblance to observed variance–covariance matrices, the relative contribution of genetic and environmental factors can be estimated and the contribution of environmental factors can be further partitioned into factors shared by all siblings and factors unique to a single sibling (Falconer & MacKay, 1996; Neale & Cardon, 1992).

Using EEG power in the classical broad bands (delta, theta, alpha, beta), twin studies have unanimously supported the importance of genetic differences to explain individual differences

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(for an overview, see van Beijsterveldt & Boomsma, 1994). Reliable estimates (where sample sizes have been sufficiently large) have been obtained in children and adolescents. Heritability of absolute power in the broadband frequencies (averaged over leads) ranged from 55% to 90% in 209 pairs of 5-year-old twins (van Baal, De Geus, & Boomsma, 1996), and from 70% to 90% in 213 adolescent twins aged 16 (van Beijsterveldt, Molenaar, de Geus, & Boomsma, 1996). In the adult population, a large number of small-scaled twin studies' correlations have suggested the importance of genetic factors in alpha amplitude measures through Falconer's h^2 calculation. However, studies employing structural equation modeling on large adult samples are still lacking. In an attempt to deal with this, van Beijsterveldt and van Baal (2002) performed a meta-analysis on the twin correlations in five smaller studies with adult samples that had assessed alpha power or similar measures. Although genetic factors significantly contributed to EEG power in each study, it was not possible to equate the results across studies into a single heritability estimate. Therefore, this study will examine a larger sample of twin families to estimate heritability of the adult EEG power spectrum. Heritability of EEG power was estimated in two different age cohorts: young adult twins and their singleton siblings with an age centered around 25 years and middle-aged twins and their singleton siblings with an age centered around 50 years.

An additional issue addressed in this article is whether the different frequencies of the power spectrum have a similar genetic architecture. It has been shown that different frequency bands reflect different cognitive processes (Klimesch, 1999; Ray & Cole, 1985; Rugg & Dickens, 1982; Schacter, 1977). An intriguing question is whether this is reflected in a different heritability for these different frequency bands. As it may be argued that the broad bands lump together sources of information of frequency components, we examined the genetic architecture of the power spectrum in more detail by computing heritability across narrow frequency bins of 1 Hz. By plotting heritability against the frequency of the bin, we obtained the so-called "heritability spectra." This allowed us to investigate whether adjacent frequency bins show sharp discontinuities around the lower and upper frequencies of the broad bands. Second, we calculated the genetic correlations between power in the broad bands, to test how much of the genetic variance across frequencies can be traced to a common source.

Method

Participants

Participants were recruited from the Dutch Twin Registry as part of a large project on the genetics of cognition and adult brain functions (Posthuma, Neale, Boomsma, & de Geus, 2001). Adult twins and their non-twin siblings were asked to participate in a testing protocol lasting 4.5 h. In total, 760 family members from 309 twin families participated in the study. The complete sample consisted of two age cohorts: a younger cohort (mean 26.2 years, *SD* 4.1) and an older cohort (mean 49.4 years, *SD* 7.2). Participating families consisted of one to seven siblings (including twins). On average, 2.5 participants per family participated. Table 1 shows the frequency of families broken down by the number of twins and siblings participating and by zygosity of the twin pair.

Table 1. *Composition of Participating Families*

	Number of families	
Family composition[a]	MZ	DZ
Both twins only	76	72
Both twins+1 sibling	40	54
Both twins+2 or more siblings	10	14
One twin only	3	9
One twin+1 sibling	8	9
One twin+2 or more siblings	—	5
One sibling	2	4
Two or more siblings	3	—

[a]We based family composition on the participating offspring only. For example, a family with "both twins only" could consist of more than two children, but these did not participate in the EEG experiment.

EEG Registration

During one part of the experimental protocol, psychometric intelligence, inspection time, and reaction times were assessed. During the other, EEG was measured at rest and during various reaction time tasks. The order of the two parts of the protocol was randomized across family members. Consequently, half of EEG registration sessions were during morning hours and half were in the afternoon.

Resting background EEG was registered for 3 min under both eyes open and eyes closed instructions, but only results from the eyes closed condition will be reported. Participants were seated in a comfortable reclining chair in a dimly lit, sound-attenuated, and electromagnetically shielded room. They were instructed to relax and minimize eye and body movement.

EEG was recorded with 19 Ag/AgCl electrodes mounted in an electrocap. Signal registration was conducted using an AD amplifier developed by Twente Medical Systems (TMS; Enschede, The Netherlands) for 657 participants and NeuroScan SynAmps 5083 amplifier for 103 participants. Signals were continuously represented online on a Nec multisync 17-in. computer screen using Poly 5.0 software or Neuroscan Acquire 4.2. Standard 10-20 positions were F7, F3, F1, Fz, F2, F4, F8, T7, C3, Cz, C4, T8, P7, P3, Pz, P4, P8, O1, and O2 (American Electroencephalograpic Society, 1991; Jasper, 1958). For NeuroScan participants, Fp1, Fp2, and Oz were also recorded, but not included in the analysis. The vertical electrooculogram (EOG) was recorded bipolarly between two Ag/AgCl electrodes, affixed 1 cm below the right eye and 1 cm above the eyebrow of the right eye. The horizontal EOG was recorded bipolarly between two Ag/AgCl electrodes affixed 1 cm left from the left eye and 1 cm right from the right eye. An Ag/AgCl electrode placed on the forehead was used as a ground electrode. Impedances of all EEG electrodes were kept below 3 kΩ, and impedances of the EOG electrodes were kept below 10 kΩ. The EEG was amplified, digitized at 250 Hz, and stored for off-line processing. Amplifier filter settings for TMS were a single order FIR bandpass filter with cutoff frequencies of 0.05 Hz and 30.0 Hz. NeuroScan filter settings were a lowpass filter at 50.0 Hz. In principle, this suggested 30 Hz as the maximum frequency at which the systems obtained comparable data. Because the filters are not perfect, however, device-specific differences may have been introduced even before the 30.0-Hz frequency used by the TMS system, and the analyses were restricted to an upper level of 25.0 Hz for both systems.

Data Processing

All EEG signals were recalculated with averaged earlobes (A1 and A2) as reference and analyzed using NeuroScan software version 4.2. The 3-min recordings were cut into 43 epochs of 1024

data points (4.096 s). Any linear trend was removed from EEG by fitting and subtracting the regression line for each epoch separately. Next, epochs were excluded per lead when EOG channels showed more than 400 μV and EEG more than 175 μV deviation from ground in either direction. EEG traces were then visually inspected per subject for remaining artifact due to muscle activity, swallowing, eye movement, bad recordings, and externally induced artifacts (e.g., experimenter initiated reset pulses, electrical hum). Only epochs with extreme magnitudes of muscle artifacts and eye movements were excluded. Participants with less than 22 valid epochs after visual inspection were considered unreliable and set to missing (22 epochs ensure at least 1 min 30 s of data per participant). In all instances, however, data were made missing only for the particular lead. For an average lead, 741 participants passed the criteria.

For all remaining epochs, power spectra were calculated with a Hamming window for 5% of the epoch duration at the beginning and end of the epochs. Power spectra were averaged, resulting in a single spectrum with a resolution of about 0.25 Hz (1000/4096 Hz). Power values across the spectrum were aggregated into 1.0-Hz bins, from 1.0 Hz up to but not including 2.0 Hz, from 2.0 Hz up to but not including 3.0 Hz, and so forth up to 25.0 Hz, thus creating twenty-four 1.0-Hz bins. Power in the classical broad bands were defined as follows: theta as the sum of all available data points from 4.0 Hz up to but not including 8.0 Hz, alpha as the sum from 8.0 Hz up to but not including 13.0 Hz, and beta as the sum from 13.0 Hz up to but not including 25.0 Hz.

Statistical Analyses

Statistical genetic analysis of the power spectra was performed using Structural Equation Modeling implemented in the program Mx (Neale, 2003). Extended twin designs provide data characterized by families of variable size. Mx handles such unbalanced data sets via full information maximum likelihood, which uses the observed, raw data instead of variance–covariance matrices. To evaluate how well the specified model fits the observed data, the raw data option in Mx calculates the negative log-likelihood ($-$LL) of the raw data for *each* family (Lange, Westlake, & Spence, 1976) as $-\mathrm{LL} = -k \log(2\pi) + \log|\Sigma| + (y_i - \mu_i)'\Sigma^{-1}(y_i - \mu_i)$, where k ($k = 1, \ldots, p$) denotes the total number of observed variables within a family (and can vary over families), $\Sigma(p \times p)$ is the expected covariance matrix of family members, y_i (for $i = 1, \ldots, p$) is the vector of observed scores, μ_i is the column vector of the expected values of the variables, and $|\Sigma|$ and Σ^{-1} are the determinant and inverse of matrix Σ, respectively.

Twice the difference between two nested models ($-2\{\mathrm{LL}_{full\ model} - \mathrm{LL}_{nested\ model}\}$) is asymptotically distributed as χ^2. A high χ^2 against a low gain of degrees of freedom (Δdf) denotes a worse fit of the second, more restrictive model relative to the first model. By stepwise restricting the number of parameters, the most parsimonious model for the data set can be found. Each nested model is compared to the previous one. Additionally, a linear regression model was employed to include effects of age and sex on the observed scores: $\mu_i = \beta_0 + \beta_1\, age_i + \beta_2\, sex_i$, where μ_i is the expected value of individual i, age_i is the individual's age at the time of measurement, sex_i is the individual's sex (0 denotes female, 1 denotes male), β_0 is the intercept, β_1 is the regression estimate of age, and β_2 is the deviation of males from females. This means model was fitted for the two age cohorts separately.

We tested for sample homogeneity by reduction of the number of parameters, as explained above, between the following groups: twins versus other siblings, zygosity types, sexes, and cohorts. Group homogeneity was tested stepwise in this order. If groups were found not to differ significantly, parameters were equated across those groups, and the next nested model was tested. Given the large number of tests that might be involved (19 leads across 24 frequency bins = 456 tests, and more when any of the groups is found to be heterogeneous), the risk of type I error was greatly increased. Because there is no a priori reason to assume topographic differences in sample homogeneity, we restricted heterogeneity testing to the central lead Cz in four broad bands (delta, theta, alpha, and beta).

Next, the observed interindividual variation in power spectra was decomposed into additive genetic variation (σ_A^2), shared environmental variation (σ_C^2), or nonshared environmental variation (σ_E^2) following Neale and Cardon (1992). Sources of shared environmental variation by definition include all environmental influences that twins and siblings from the same family share, whereas sources of nonshared environmental variation refer to the environmental variation that is unique for an individual and that is typically not shared with other family members. For DZ twin pairs (and sibling pairs if the saturated models indicated no difference in correlation between DZ twin pairs and sibling pairs) correlation between shared environmental influences (C) was fixed at 1 and the correlation between additive genetic influences (A) at 0.5. For MZ twins correlations between additive genetic influences and between shared environmental influences were fixed at 1. Correlation between nonshared environmental influences (E), per definition, is set to 0 for both MZ and DZ twins. Thus, the expectation for the total variance is $\sigma_A^2 + \sigma_C^2 + \sigma_E^2$, the expectation for the covariance between MZ twins is $\sigma_A^2 + \sigma_C^2$, and the expectation for DZ twins/sibling pairs is $0.05 \times \sigma_A^2 + \sigma_C^2$. Heritability is calculated as the proportional contribution of genetic variation to the total, observed variation $\left(\frac{\sigma_A^2}{\sigma_A^2+\sigma_C^2+\sigma_E^2}\right)$. Goodness of fit of the variance decomposition models and significance of estimated parameters was, again, determined by likelihood ratio tests.

Table 2. *Stability of the Frequency Bands over an Average Period of 1.77 Years*

		Frequency band			
Lead	*N*	δ	θ	α	β
F7	27	0.72	0.88	0.89	0.81
F3	28	0.72	0.91	0.91	0.84
F1	27	0.71	0.91	0.93	0.82
FZ	28	0.73	0.91	0.91	0.86
F2	26	0.73	0.91	0.91	0.86
F4	27	0.73	0.90	0.91	0.87
F8	26	0.68	0.84	0.89	0.75
T7	27	0.66	0.84	0.89	0.86
C3	28	0.84	0.95	0.95	0.86
CZ	27	0.11	0.80	0.86	0.52
C4	27	0.39	0.84	0.87	0.68
T8	27	0.68	0.89	0.88	0.81
P7	27	0.87	0.96	0.92	0.90
P3	26	0.55	0.86	0.89	0.75
PZ	27	0.50	0.82	0.84	0.76
P4	28	0.87	0.95	0.96	0.89
P8	26	0.85	0.95	0.93	0.93
O1	26	0.86	0.94	0.94	0.88
O2	26	0.83	0.93	0.93	0.86
Mean		0.69	0.89	0.91	0.82

Results

Temporal Stability

Thirty subjects were retested after an average interval of 674 days ranging from 354 to 1322 days. Twenty-eight had valid EEG data available on any lead. Temporal stability scores (Table 2) are highest for theta and alpha. Stability of beta band power suggests more change over time than alpha and theta, and delta shows lowest stability, varying from .60 to .87 with a few very low scores at Cz and C3, and only moderate scores at Pz and P3.

Sample Homogeneity across Groups

Assumptions of homogeneity across twin/singleton, zygosity, and sex groups were all met. We found evidence for heterogeneity of variance and/or means and/or covariances across the age cohorts (theta: $\chi^2[6] = 14.14$, $p = .028$; alpha: $\chi^2[6] = 20.43$, $p = .002$; beta: $\chi^2[6] = 16.95$, $p = .009$). Therefore, subsequent variance decomposition models will be estimated separately for each cohort.

Broadband Correlations and Variance Decomposition

Table 3 shows the twin correlations for the broad bands as estimated with Mx. These suggest a strong, additive genetic effect as the MZ correlations are high and the DZ correlations are around half the MZ correlation (Falconer & MacKay, 1996). Correlations are generally higher in the alpha band across all leads. They are also higher in the young cohort across all leads and frequencies. There is little evidence for strong topographic differences except for some lower correlations in the temporal

Table 3. *Twin and Sibling Correlations between Identical (MZ) and Any Other, Nonidentical (DZ, SIB) Sibling Pairs, with Heritabilities, of Broadband EEG Power*

	Young adult											
	δ			θ			α			β		
	MZ	DZ SIB	h^2	MZ	DZ SIB	h^2	MZ	DZ SIB	h^2	MZ	DZ SIB	h^2
F7	0.45	0.20	0.43	0.86	0.49	0.86	0.91	0.47	0.91	0.71	0.45	0.73
F3	0.58	0.23	0.55	0.85	0.51	0.85	0.93	0.47	0.93	0.79	0.48	0.80
F1	0.59	0.22	0.54	0.85	0.55	0.85	0.93	0.46	0.93	0.85	0.50	0.85
FZ	0.60	0.23	0.56	0.84	0.51	0.84	0.92	0.45	0.92	0.84	0.50	0.84
F2	0.59	0.22	0.54	0.84	0.52	0.85	0.93	0.46	0.93	0.87	0.50	0.87
F4	0.58	0.18	0.50	0.84	0.51	0.84	0.93	0.46	0.93	0.83	0.52	0.83
F8	0.48	0.21	0.45	0.83	0.48	0.84	0.91	0.43	0.92	0.78	0.46	0.78
T7	0.56	0.25	0.54	0.82	0.46	0.82	0.90	0.48	0.90	0.53	0.20	0.49
C3	0.71	0.32	0.70	0.88	0.48	0.88	0.92	0.44	0.92	0.86	0.46	0.86
CZ	0.78	0.54	0.79	0.87	0.51	0.87	0.93	0.48	0.93	0.88	0.52	0.88
C4	0.76	0.47	0.77	0.85	0.47	0.85	0.91	0.43	0.91	0.85	0.51	0.86
T8	0.69	0.22	0.63	0.84	0.40	0.84	0.90	0.45	0.90	0.27	0.30	0.40
P7	0.59	0.31	0.59	0.84	0.45	0.84	0.88	0.42	0.88	0.80	0.43	0.80
P3	0.74	0.53	0.76	0.88	0.51	0.88	0.89	0.49	0.89	0.88	0.55	0.88
PZ	0.68	0.51	0.71	0.82	0.48	0.82	0.89	0.52	0.89	0.78	0.56	0.80
P4	0.73	0.38	0.74	0.90	0.46	0.90	0.90	0.49	0.90	0.85	0.54	0.86
P8	0.69	0.38	0.70	0.88	0.44	0.88	0.85	0.44	0.85	0.78	0.50	0.79
O1	0.60	0.32	0.60	0.82	0.43	0.82	0.85	0.43	0.85	0.82	0.50	0.83
O2	0.69	0.24	0.66	0.85	0.38	0.85	0.85	0.43	0.85	0.82	0.50	0.83
Mean	0.64	0.31	0.62	0.85	0.48	0.85	0.90	0.46	0.90	0.78	0.47	0.79
	Middle-aged											
	δ			θ			α			β		
	MZ	DZ SIB	h^2	MZ	DZ SIB	h^2	MZ	DZ SIB	h^2	MZ	DZ SIB	h^2
F7	0.45	0.23	0.45	0.66	0.37	0.67	0.83	0.41	0.83	0.67	0.28	0.66
F3	0.37	0.22	0.40	0.73	0.40	0.73	0.87	0.43	0.87	0.81	0.39	0.81
F1	0.35	0.24	0.40	0.74	0.37	0.74	0.84	0.42	0.84	0.81	0.41	0.81
FZ	0.44	0.26	0.46	0.71	0.36	0.71	0.85	0.42	0.85	0.80	0.40	0.80
F2	0.42	0.23	0.43	0.72	0.37	0.72	0.86	0.42	0.86	0.80	0.39	0.80
F4	0.38	0.23	0.41	0.73	0.35	0.73	0.86	0.42	0.86	0.80	0.34	0.80
F8	0.52	0.21	0.50	0.74	0.35	0.74	0.85	0.44	0.85	0.79	0.36	0.79
T7	0.52	0.24	0.51	0.78	0.34	0.77	0.79	0.33	0.79	0.45	0.20	0.43
C3	0.55	0.25	0.53	0.77	0.34	0.76	0.84	0.39	0.84	0.77	0.32	0.77
CZ	0.55	0.26	0.55	0.78	0.29	0.77	0.85	0.40	0.85	0.83	0.39	0.83
C4	0.48	0.23	0.47	0.77	0.32	0.76	0.89	0.37	0.89	0.84	0.37	0.84
T8	0.53	0.19	0.50	0.75	0.31	0.75	0.80	0.38	0.79	0.52	0.32	0.54
P7	0.67	0.22	0.62	0.82	0.33	0.81	0.82	0.36	0.82	0.72	0.29	0.70
P3	0.65	0.25	0.62	0.80	0.32	0.80	0.88	0.39	0.88	0.82	0.37	0.82
PZ	0.69	0.34	0.69	0.79	0.34	0.78	0.87	0.38	0.88	0.82	0.43	0.82
P4	0.68	0.25	0.64	0.74	0.33	0.73	0.89	0.39	0.89	0.78	0.38	0.78
P8	0.66	0.24	0.63	0.76	0.32	0.75	0.90	0.41	0.90	0.78	0.30	0.77
O1	0.73	0.24	0.69	0.75	0.30	0.74	0.83	0.34	0.83	0.73	0.32	0.73
O2	0.70	0.22	0.65	0.75	0.32	0.74	0.87	0.39	0.87	0.70	0.35	0.70
Mean	0.54	0.24	0.53	0.75	0.34	0.75	0.85	0.39	0.85	0.75	0.35	0.75

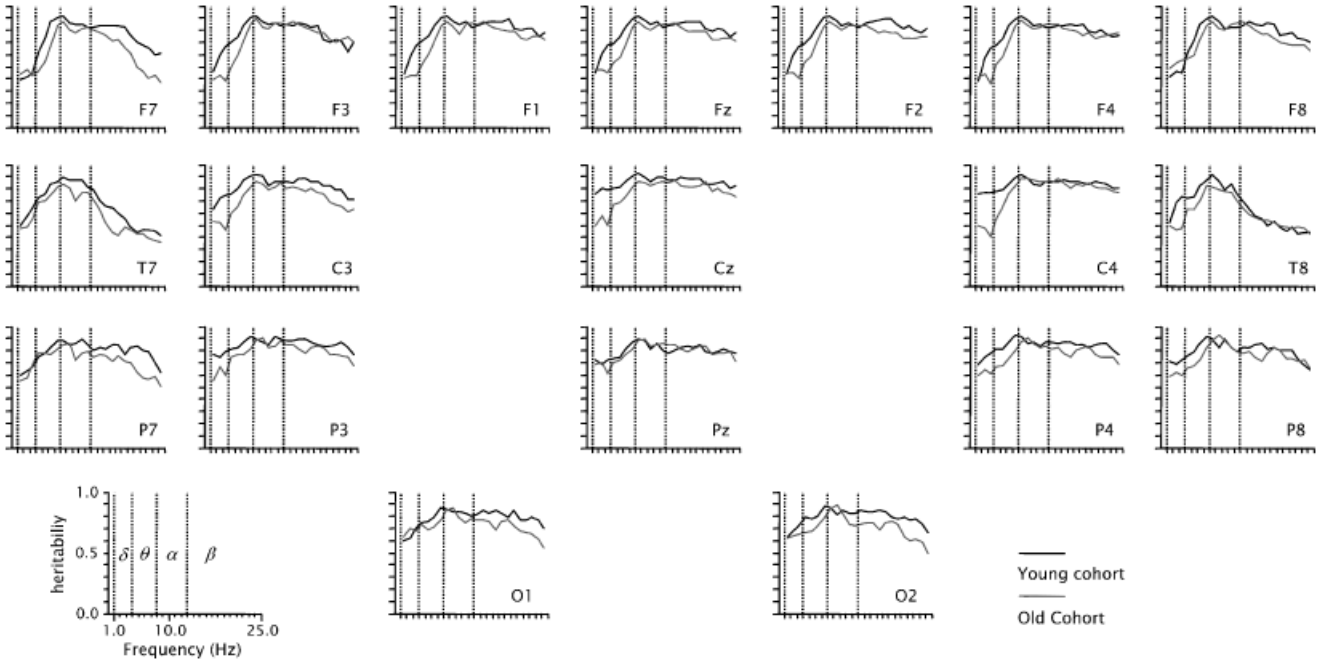

Figure 1. Heritability spectra.

areas in the beta range. The overall pattern of correlations does not suggest a role for common environment in EEG power. The exception may be the correlations for spectra in the young cohort, broadband theta and beta, because the DZ correlation is slightly but systematically over half the MZ correlation. ACE versus AE model fitting, however, did not reach significance for any lead and broadband combination except for delta and beta on Pz and for delta on P3. These significant results did not hold under the Bonferroni alpha level correction for multiple testing. Therefore, in subsequent model fitting an AE model was used for all bands and all leads.

Heritability Spectra Based on 1-Hz Bins

Figure 1 show the heritability spectra for each lead with cohorts plotted separately. Heritability is high for both cohorts, peaking in the alpha range. It drops with decreasing frequency in the theta and delta ranges but remains high with increasing frequency in the beta range, except for the temporal area. Cohort differences systematically showed lower heritability in the older cohort, mainly in the frontocentral regions for theta and delta, and mainly in the left hemisphere for the beta range.

Alignment of Spectra on Individual Alpha Frequency

The boundaries of the alpha band as well as of the other "classical" broad bands are based on population-averaged EEG spectra that only imperfectly reflect the constituent *individual* EEG spectra and, consequently, their genetic determinants. In adult subjects peak alpha frequency ranges from 8 to 13 Hz. Assigning a fixed alpha band to all subjects could easily confound alpha with up to a 3-Hz bin of theta or a 2-Hz bin of beta power depending on whether the individual's peak is high or low within the normal alpha band (Klimesch, 1999). We therefore repeated our genetic analyses on spectra that were aligned on the individual alpha peak. We defined the dominant frequency as the one with maximum attenuation of alpha power by opening of the eyes, following Klimesch. Using this "alpha blocking" definition, we were able to establish the individual alpha frequency for all but 90 subjects.

Alignment did not yield significantly different heritability estimates in the theta, alpha, and beta bands on most leads after examining the 95% confidence intervals. A reduced heritability was only found in two bins surrounding peak alpha in the frontal leads of the young cohort. Overall, we conclude that alignment produces no or marginally different heritability estimates. Because many subjects were lost in the alignment procedure (no clear alpha peak), we proceeded with unaligned spectra from the eyes closed condition.

Genetic Correlations between Frequencies

To get an indication of the extent to which heritable variance of the frequency bins can be traced to a common genetic source, we calculated the genetic correlations between the broadband frequencies, that is, the proportion of genetic variance shared

Table 4. *Phenotypic and Genetic Correlations (Genetic Variation Due to a Common Source) between Broadband Frequencies on Lead CZ*

	Frequency band			
	δ	θ	α	β
Young adult				
δ	—	0.73	0.48	0.55
θ	0.75	—	0.69	0.59
α	0.55	0.74	—	0.55
β	0.62	0.60	0.58	—
Middle-aged				
δ	—	0.58	0.37	0.45
θ	0.63	—	0.69	0.62
α	0.55	0.73	—	0.65
β	0.63	0.65	0.68	—

Note: Upper triangles are phenotypic, lower triangles are genetic correlations.

between any two variables. These were calculated for each cohort separately. The results are shown in Table 4. In both cohorts, 55% to about 75% of the genetic variance overlaps between the bands. The genetic correlations were all significantly different from both zero and unity, suggesting that common as well as unique genetic factors contributed to each of the broad bands.

Discussion

The results show that in adult subjects EEG power at rest is a heritable trait across the entire frequency spectrum. No evidence was found for common environmental influences on the EEG power spectrum. A meaningful contribution of unique environment was limited to the delta frequencies, which showed lower heritabilities down to 40%. This lower delta heritability, together with the lower temporal stability for delta, may be explained in part by larger measurement error for this frequency, due to, for example, residual eye movement artifacts. Measurement error in our modeling will show up as unique environmental influences. Alternatively, we cannot rule out that true environmental factors have more impact on low frequency EEG power than on power in the higher frequency bands. In the upper beta regions, both heritability and stability were somewhat lower in the temporal areas. Again, this might be explained either by larger measurement error or by larger sensitivity to environmental factors. It is hard to explain, however, why unique environmental factors would affect beta frequencies only in these scalp regions.

For the theta and alpha frequencies, our MZ twin correlations were similar to 5-min test–retest correlations reported in the literature (Salinsky, Oken, & Morehead, 1991) as well as the longer term stability over a period of years reported here. Identical twins, therefore, resemble their co-twin about as much as they would themselves over a period of years. Overall, our results establish EEG power to be one of the most heritable complex traits in human subjects. This is in keeping with previous results from smaller studies of twin families (Lykken, Tellegen, Iacono, & McGue, 1998; McGuir, Katsanis, & Iacono, 1982; Christian et al., 1996) and the large adolescent studies (van Beijsterveldt & van Baal, 2002; van Beijsterveldt et al., 1996).

The overarching suggestion of the "heritability spectra" in Figure 1 is that the separation of broad bands on the basis of EEG power has little basis in its genetic architecture. In contrast, the uniformity of the heritability spectra suggests that EEG powers at different frequencies share a common genetic source. We further tested this hypothesis by computing the genetic correlations between the broadband frequencies in a multivariate genetic model. The results indicated that a moderately high to high proportion of genetic variance was shared among the frequency bands. In both cohorts, genetic correlations varied from .55 to about .75. Therefore, a significant proportion of the heritable variance in all frequency bands must be attributed to a common genetic source. This is in concordance with genetic correlations between the broadband frequencies found in adolescents (Anokhin et al., 2001).

Genes common to all frequencies may affect EEG power through "trivial" effects on the conductive properties of the tissues surrounding the cortex. As often observed before, skull and scalp thickness, most likely heritable traits, strongly influence EEG power (Babiloni et al., 1997; Leissner, Lindholm, & Petersen, 1970; Nunez, 1981). A common genetic source for EEG may also reside in nontrivial common influences on cerebral rhythm generators like the central "pacemaker" in the septum for hippocampal slow-wave activity (3–4 Hz) or the thalamocortical and corticocortical generators of cortical alpha rhythmicity (Lopes da Silva, 1991; Steriade, Gloor, Llinas, Lopes da Silva, & Mesulam, 1990). Another possible source could lie in genes directly involved in the bioelectric basis of the EEG signal itself; genes influencing the number of pyramidal cells, the number of dendritic connections, or their orientation with respect to the scalp may directly influence the mass dendritic tree depolarization of pyramidal cells in the cortex that underlies EEG power (Ray, 1990). To resolve the genetic basis of the EEG, a whole genome scan on power in the broad bands followed by positional cloning seems the most rational approach. In view of the high heritability of EEG power, such gene finding is entirely feasible.

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