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Common and specific genetic influences on EEG power bands delta, theta, alpha, and beta

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Abstract

It is difficult to study the genetic basis of psychological function/dysfunction due to its etiological complexity. Instead, we studied a biological marker, EEG power, which is associated with various psychological phenotypes and is closer to gene function. Previous studies have consistently demonstrated high heritability of EEG band power, but less is known about how common or specific genes influence each power band. For 519 adolescent twin pairs, spectral powers were calculated for delta, theta, alpha, and beta bands at bilateral occipital and frontal sites. All four bands were entered into a multivariate genetic model, with occipital and frontal sites modelled separately. Variance was decomposed into additive (A) and dominant (D) genetic factors, and common (C) and unique (E) environmental factors. Band heritabilities were higher at occipital (0.75–0.86) than frontal sites (0.46–0.80). Both common and specific genetic factors influenced the bands, with common genetic and specific genetic factors having more influence in the occipital and frontal regions, respectively. Non-additive genetic effects on beta power and a common environment effect on delta, theta, and alpha powers were observed in the frontal region.

Keywords: EEG power; Genetic; Environmental; Band-specific; Common factor; Dominance

1. Introduction

The importance of genetic influences has been demonstrated for many psychological traits and disorders (Bouchard and Loehlin, 2001; Deary et al., 2006; Ehringer et al., 2006; Leonardo and Hen, 2006; Preuss et al., 2004). To better understand these phenotypes, it is often advantageous to look at a more basic, underlying, preferably biological trait (or 'endophenotype'), as this will more directly reflect the influence of the genome (Gottesman and Shields, 1972; Gottesman and Gould, 2003). The electroencephalogram (EEG) provides a good endophenotype because it is largely genetically controlled (van Beijsterveldt and van Baal, 2002; Vogel, 2000) and is stable over time (test-retest reliability is around 0.8 for both absolute and relative powers (Pollock et al., 1991; Salinsky et al., 1991)). As noted below, at the phenotypic level it is related to psychiatric disorder (Knott et al., 2001; Lazzaro et al., 1998; Pogarell et al., 2006; Porjesz et al., 1998), as well as personality (Tran et al., 2006) and cognition (IQ, *g*) (Giannitrapani, 1985; Schmid et al., 2002).

One of the most common and straightforward ways of quantifying EEG is to divide the frequency spectrum into discrete ranges (bands), and with a transformation of the data, determine the amplitude or 'power' of each range. EEG band power at a given site reflects the circuitry and function of the underlying pyramidal cells (Schaul, 1998). It varies greatly between individuals (Vogel, 2000), is stable within an individual in a given condition (e.g. at rest with eyes closed) (Williams et al., 2005), and changes according to mental state (Moretti et al., 2004), task demands (Klimesch, 1999), and age (Li et al., 1996; McEvoy et al., 2001). Dividing the frequency spectrum into delta, theta, alpha, and beta waves can be done using fixed bands (e.g. the classical broad bands delta (up to 4 Hz), theta (4–8 Hz), alpha (8–13 Hz), beta (13–25 Hz)) or

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individually aligned bands (ranges for each band are set relative to an individual's peak alpha frequency) (Klimesch, 1999). Smit et al. (2005) have shown that for the purposes of genetic analysis there is little difference between the two, but overall, the disadvantage of excessive data loss with the aligned method outweighs any advantage of using individualised data.

Resting EEG power in specific bands has been associated with various cognitive traits. For example, resting alpha power was found to be positively associated with IQ (Doppelmayr et al., 2002), although a significant relationship is not always found (Gaser et al., 1983; Smit et al., 2006). Aspects of personality have been found to be associated with specific resting EEG band powers. Tran et al. (2006) found that delta and theta powers were positively correlated with extraversion in males and negatively correlated with conscientiousness in females. Alpha and beta correlated negatively with neuroticism in males, although not in posterior sites.

Various forms of psychopathology are associated with abnormal patterns of resting EEG activity in a particular band or bands. EEG is an important endophenotype for alcoholism (see Porjesz et al. (2005) for a review). For example, predisposition to becoming an alcoholic has been associated with elevated resting beta power (Rangaswamy et al., 2004). Increased resting beta power has also been linked to depression (Knott et al., 2001), as has frontal hemispheric asymmetry in resting alpha power (Bruder et al., 2005; Coan and Allen, 2004). Recent findings have suggested that frontal resting EEG asymmetry may also predict future development of anxiety (Blackhart et al., 2006). ADHD has been linked to abnormally high resting theta band activity, particularly in the frontal lobes (Lazzaro et al., 1999; Lazzaro et al., 1998), and this has been linked more specifically to a decrease in attention (Mann et al., 1992). Sufferers of obsessive-compulsive disorder exhibit frontally increased resting delta but decreased alpha and beta powers, and their EEG band powers tend to correlate positively with obsessions and negatively with compulsions (Pogarell et al., 2006).

The summarised evidence indicates that mental disorder and individual differences in cognition are usually associated with unusually high or low power in a specific band or group of bands, rather than a general change across the entire frequency spectrum. This suggests that if resting EEG power is to provide a good endophenotype for studying the genetic basis of psychological function and dysfunction, then its genetic influences should exhibit some band-specificity. A general genetic factor influencing all frequencies uniformly would provide few clues to the genetic basis of a psychopathology which involved only an increase in beta power, for example. Many studies have determined the genetic influences on one band (usually alpha, see van Beijsterveldt and van Baal (2002) for a review), but these do not reveal whether common or specific genes are influencing each band.

A more elaborate study, by van Beijsterveldt et al. (1996), used multivariate modelling to determine to what extent the same genes affected EEG in different parts of the brain. From the analysis it could be concluded that the same genes influenced alpha at all brain regions, and that there were no hemispheric differences in the genetic control of delta, theta, alpha, or beta power. It left unanswered the questions of whether the same genes influenced the different bands, and whether genes controlling delta, theta, and beta differ between specific regions of the brain. Other results of the study are derived from univariate analyses, but they provide a good basis for comparison to the current study: the heritabilities of all four bands were very high and similar to each other (delta 0.76, theta 0.89, alpha 0.89, beta 0.86), there were no mean differences or heritability differences between the sexes, and there was no effect of common environment. It is not known whether the apparent trend of homogeneity of genetic influence across brain regions, power bands, and sexes is a reflection of the true nature of the genetic architecture or simply a lack of power to detect heterogeneity (the sample consisted of 213 twin pairs).

A study by Smit et al. (2005) calculated bi-variate genetic correlations between delta, theta, alpha, and beta powers. These ranged from .55 to .75, indicating that 55% to 75% of the genetic variance overlapped between bands. As the genetic correlations were neither zero nor unity, it suggested both common and specific genetic factors contributed to the power bands. However, the correlations were based on data from one central electrode, so comparisons could not be made between brain regions. A multivariate analysis by Anokhin et al. (2001) also suggested that common as well as specific genetic factors contributed to EEG power bands and event related potentials (ERPs), but beta power was not included and statistical power was low.

Using the largest twin sample to date, the present study aimed to expand on the EEG literature by studying the multivariate architecture of the genetic and environmental influences on the four EEG power bands, determining the extent to which they are influenced by a common factor versus band-specific factors. In doing this we also aimed to disentangle heterogeneity from homogeneity across hemispheres, sex, and age. Specifically, the main objectives were to (1) confirm that each EEG power band is mainly influenced by genetic factors and ascertain the mode of transmission of this influence, and (2) investigate to what extent the same genes influence all four bands, and, conversely, how important band-specific genetic effects are. We focussed on occipital and frontal sites, and while this does not allow us to comprehensively assess variation across brain areas, it allows us to more broadly attribute brain activity to posterior versus anterior regions. Functionally, these brain regions are dramatically different: the occipital lobes perform mainly basic functions in visual perception, whereas frontal lobes perform higher cognitive functions such as executive control, planning, and reasoning. These functional differences may be reflected by differences in the EEG signal and its underlying genetic influences. We applied multivariate structural equation modelling to the classical broad bands delta, theta, alpha, and beta of resting EEG, from homologous left and right sites. Thus, the far more subtle hemispheric functional differences may be contrasted with the lower versus higher order occipito-frontal differences.

2. Methods

2.1. Participants

Participants were adolescent twins recruited through South East Queensland secondary schools as part of a study on the genetics of cognition (Wright et al., 2001a), of which the recording of resting EEG was a component. Twin pairs were excluded from participation if parental report indicated that either twin had a history of head injury, neurological or psychiatric illness, substance abuse or dependence, or current use of medication with central nervous system effects. Prior to testing, written informed consent was obtained from all participants and their parents or guardians. Ethics approval for the study was obtained from the Human Research Ethics Committee, Queensland Institute of Medical Research.

The full sample consisted of 533 females and 505 males between the ages of 15.42 and 18.16 (16.24 ± 0.31). Due to the narrow age range, ages to two decimal places were used for analysis to enhance the possibility of finding any genuine age effects. This sample is almost identical to that reported in Smit et al. (2006), and includes six zygosity groups; 125 monozygotic (MZ; identical) female pairs (MZF), 114 MZ male pairs (MZM), 69 dizygotic (DZ; non-identical) female pairs (DZF), 66 DZ male pairs (DZM), 145 opposite sex DZ pairs (68 female first born (DZFM), 77 male first born (DZMF)). Zygosity was determined by typing 9 independent polymorphic DNA markers using the AmpFLSTR® Profiler® PCR Amplification Kit and crosschecking with ABO, MN, and Rh blood groups and/or phenotypic information (e.g. hair, skin, and eye colour). Based on this, zygosity was assigned with an extremely low probability of error (less than 10^{-4}).

2.2. General procedure

Two parallel testing sessions were used in the Twin Cognition study protocol: a psychometric assessment of processing speed and IQ (e.g. Luciano et al., 2001) and a psychophysiological session where event-related potentials (ERPs) were recorded during a working memory task (Hansell et al., 2001; Wright et al., 2001b) followed by the recording of resting EEG (Smit et al., 2006). As one twin did the psychometric session, the co-twin undertook the psychophysiological session, and after a short break each twin completed the complementary session. Two 4 min recordings comprised the resting EEG, the first with eyes closed and second with eyes open. For the eyes closed condition participants were informed that the duration of the recording would be approximately 5 min, and were asked to relax and sit quietly with their eyes closed, to minimize any movement. For the eyes open condition they were asked to sit quietly and be relaxed, and to focus on the monitor in front of them. Only data from the eyes closed condition are used in this study. Recordings were taken in a semi-darkened, electrically shielded, and sound-attenuated cubicle.

2.3. EEG recording

As described in Smit et al. (2006), EEG was recorded from 15 scalp locations (Fp1, Fp2, Fz, F3, F4, F7, F8, Cz, C3, C4, Pz, P3, P4, O1, O2) using an electrode cap. The tin electrodes were arranged according to the International electrode (10–20) placement system and referenced to physically linked ears, with the ear impedances matched at the beginning of the recording session. The ground lead was located just anterior to the Fz electrode. Ocular potentials (electro-oculogram or EOG) were recorded from single tin electrodes located on the outer canthus and the centre of the supraorbital ridge above the left eye. Impedance readings were all below 5 kΩ. EOG, Fp1, and Fp2 were amplified with a factor 5K and all other channels with a factor 20K by Grass preamplifiers (model P511K). Recordings were filtered with a band pass filter of 0.01 to 30 Hz (6dB per octave) and a 50 Hz notch filter.

Software used for the recording determined that the maximum length of continuously recorded EEG was 12 s with a discontinuity of 2 s between successive 12-second blocks. Twenty 12-second blocks were recorded with eyes open and 20 with eyes closed. To generate power spectra EEG data were divided into sixty 4-second epochs per condition using EEG analysis software EPTOR 1.3.3. (Groot, 1999). Eye movement artefacts were removed from each epoch using a dynamic regression algorithm (Molenaar, 1987), and epochs with abnormal EEG patterns (>25% of amplitude at 0 μV) or extreme voltages (exceeding ±1000 μV for Fp1, Fp2, and EOG; ±250 μV for all other channels) were excluded. The DC component was removed from each epoch and a Hanning window was applied to the first and last 5% of each epoch to prevent spectral leaking. Finally, EEG data were converted to a frequency distribution ranging from 1 to 30 Hz (resolution 0.25 Hz) using Fast Fourier Transformation (Niedermeyer, 1999).

The frequency distribution was divided into the four standard broad bands using fixed bands, such that delta included frequencies ranging from 1.5–4 Hz, theta 4–8 Hz, alpha 8–13 Hz, and beta 13–25 Hz. Band power was computed as the sum of the power frequency bins within each band.

2.4. Statistical analysis

Data were analysed using maximum likelihood (ML) estimation procedures with the statistical package Mx (Neale et al., 2006). Prior to genetic modelling, data were checked for normality and screened for univariate and multivariate outliers. Outlying families were detected and excluded by using the %P option in Mx, which uses a standardised Mahalanobis distance to compute a z-score for each family. Values outside the range of −3 to +3 indicated extreme families (taking into account MZ and DZ similarities) in assumption testing analyses on each power band at each site (univariate outliers) and for the multivariate models prior to model reduction (multivariate outliers). Extreme families were excluded so that results may be generalizable, as maximum-likelihood parameter estimates are biased by outlying families (Tabachnick and Fidell, 2001).

Multivariate genetic modelling partitioned the total variance into that due to genetic factors – additive (A) and non-additive (D, e.g. dominance, epistasis) – and that due to non-genetic factors - common environmental influence shared by co-twins (C), and a combination of measurement error and unique environmental effects not shared by co-twins (E). For MZ twins the co-variance was defined as additive (1.0A) plus non-additive (1.0D) genetic factors plus common environment (1.0C), and for DZ twins, 0.5A + 0.25D + 1.0C. In the absence of data from separated twins or half siblings D and C are negatively confounded, so that only one can be estimated for a given variable in a given model. Which factor to retain was decided independently for each EEG band based on the ratio of MZ and DZ correlations – if the DZ correlation was less than half the MZ correlation, then D was modelled, otherwise C was modelled. For occipital sites (O1, O2) a four-variable Cholesky model was specified in which ACE components of variance were modelled for all four bands. For frontal sites (F3, F4) a four-variable Cholesky model was specified in which ACE components of variance contributed to delta, theta, and alpha, and ADE components of variance contributed to beta. Sex and age were entered into the multivariate models as covariates.

Preliminary multivariate model fitting including all four EEG power bands (delta, theta, alpha, and beta), at each of the frontal and occipital sites separately, indicated a striking concordance between the analogous genetic (A) path loadings in the O1 and O2 models, and likewise in the F3 and F4 models, to the point where the left and right hemisphere models appeared essentially equivalent. There was no such concordance between genetic path loadings in frontal and occipital models, for example, between O1 and F3. Therefore, we combined O1 and O2 into an octovariate Cholesky model (and similarly for F3 and F4), represented schematically in Fig. 1. We tested the equality of left and right hemisphere genetic loadings for each band by equating all analogous left/right loadings (e.g. the A1 loadings on delta left and delta right) at once and comparing model fit. Common environment (C) loadings displayed no pattern suggesting left/right hemisphere equivalence, so their equality was not tested, but as a conservative test of C we tested whether it could be dropped or reduced before reducing the A component further (if C were dropped after fully reducing the A component it would be a less conservative test of C because variance is more constrained). Next, to test for residual hemisphere-specific genetic variance (since all analogous left/right loadings were equated as described above), factors A2, A4, A6, and A8 were dropped. For the frontal model where these could not be dropped we tested whether factors A3, A5, and A7 could be dropped, and then reduced the model further by testing which individual hemispheric specific loadings had to be retained. The ordering of model reduction steps was decided prior to analysis to reduce any bias on significance testing. However, we subsequently checked whether swapping the order of model reduction steps made a substantive difference, and it did not.

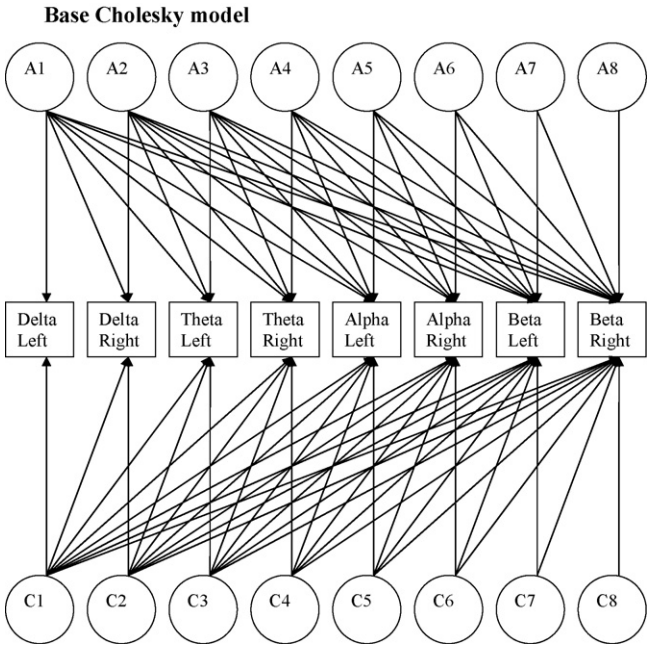


Fig. 1. Schematic representation of the octovariate base Cholesky model for the occipital and frontal models, from which subsequent reduced models were derived. Left and right hemisphere sites are included in a single model (F3 and F4 for the frontal model, and O1 and O2 for the occipital model). Additive genetic ('A' circles) and common environmental ('C' circles) factors explain the variance of EEG power bands delta, theta, alpha, and beta in the left and right hemispheres (squares). Note for the frontal model, non-additive genetic variance (D) was modelled for beta (left and right) instead of common environment (C), i.e. C7 replaced by D1, and C8 replaced by D2). Also, A2, A4, A6, and A8 contain a right hemisphere specific loading, e.g. the A2 loading on delta right. For clarity, unique environmental (E) influences are not shown, but they have the same structure as A and C.

As well as by reducing the Cholesky model, independent pathway models were tested. Smit et al. (2005) suggested that common as well as specific additive genetic factors contributed to each band, so a model reflecting this was tested, consisting of a general factor influencing all bands and four specific factors loading on each band. Other models were based on varimax rotations of factor analyses of the genetic correlation matrices from the occipital and frontal models. These models were compared to each other and the final reduced Cholesky models using the Akaike's Information Criterion (AIC) index.

3. Results

3.1. Preliminary analyses

Mean EEG power and standard deviations for each of the four frequency bands at occipital and frontal sites are presented in Table 1. Distributions of the power bands at each brain region were positively skewed, and thus log transformed (lg $10(x + 1)$) to improve the normality of the distributions. Adding one in this transformation resulted in all values being defined and variables being less skewed and kurtotic. All further analyses are based on transformed data unless otherwise specified. Fifteen twin pairs were identified as univariate outliers at the assumptions testing stage for O1–O2, and 14 for F3–F4. Twenty-one additional pairs were identified as multivariate outliers for O1–O2, and 15 for F3–F4. The composition of the final sample was 483 pairs for O1–O2, and 490 pairs for F3–F4.

Table 1
Descriptives for each EEG power band at each electrode site (from raw data, with outliers excluded)

		Mean (±S.D.) μV^2	Range
Delta	O1	48.76 (±33.75)	7.06–281.67
	O2	63.21 (±45.97)	8.06–347.91
	F3	45.94 (±18.87)	5.00–152.65
	F4	42.55 (±16.92)	4.68–139.02
Theta	O1	36.58 (±32.79)	3.49–371.88
	O2	47.52 (±40.64)	3.57–288.47
	F3	27.02 (±14.40)	2.70–133.07
	F4	27.30 (±15.27)	2.51–134.99
Alpha	O1	114.13 (±101.24)	3.58–826.79
	O2	152.85 (±136.15)	4.40–1055.19
	F3	20.01 (±12.57)	2.48–84.94
	F4	19.50 (±11.95)	2.60–90.78
Beta	O1	10.13 (±6.20)	1.29–45.16
	O2	14.06 (±8.58)	1.87–77.25
	F3	6.55 (±3.68)	0.85–31.97
	F4	7.23 (±4.20)	0.86–34.10

For occipital sites $N = 966$, for frontal $N = 980$.

Homogeneity of sampling was found with equality of means and variances (using an α-level of .01) according to birth order, zygosity group, and sex. The exceptions were for beta at the occipital sites, where the variance of males and females differed at both O1 ($p = .0007$) and O2 ($p = .00008$), with females being more variable than males. These variances were left free to vary between sexes for subsequent analyses. In addition, fixed effects of sex and age significantly influenced some measures. Sex effects were significant for around a third of power bands, but most effects were small. The exception was a strong tendency for females to have higher beta power at the occipital sites. Age effects were stronger and more consistent in size and direction. All age effects were significant except for occipital alpha power, and all were such that EEG power decreased with age.

Twin correlations are presented in Table 2. Covariances of twin pairs did not differ significantly according to sex, or between same sex and opposite sex DZ twin groups for delta, theta, alpha or beta at any site. While there was a trend for DZ male correlations to be greater than DZ females, particularly in frontal areas, 95% confidence intervals were wide, and overlapped. The wider confidence intervals for the same sex DZ groups than the opposite sex DZ group was due to the smaller sample size (i.e. 66 and 69 pairs compared with 145 pairs, respectively). MZ twin correlations/covariances were significantly higher than DZ twin correlations in all four bands at left and right frontal and occipital sites ($\Delta\chi^2_1 = 15.70$ to 114.74, $p < .0001$). For beta power the DZ twin correlations at frontal (left and right) sites were less than half the MZ correlations, but for all other power bands the DZ correlation was half or greater than half of the MZ correlation.

Phenotypic correlations between left and right hemisphere (O1–O2; F3–F4) within each frequency band were very high, ranging from .86 (delta F3–F4) to .95 (theta F3–F4 and theta O1–O2). Phenotypic correlations between occipital and frontal

Table 2
Twin correlations (95% confidence intervals in parentheses) for each zygosity group for delta, theta, alpha, and beta at left and right occipital (O1, O2) and frontal (F3, F4) sites

		MZF (N = 125)	MZM (N = 114)	DZF (N = 69)	DZM (N = 66)	DZOS (N = 145)	MZ (N = 239)	DZ (N = 280)
Delta	O1	0.79 (.71–.83)	0.72 (.63–.78)	0.44 (.24–.58)	0.55 (.31–.70)	0.35 (.18–.48)	0.75 (.70–.80)	0.42 (.32–.51)
	O2	0.80 (.74–.85)	0.74 (.66–.80)	0.41 (.22–.59)	0.53 (.36–.65)	0.28 (.11–.43)	0.77 (.72–.81)	0.39 (.29–.48)
	F3	0.55 (.41–.65)	0.59 (.47–.68)	0.22 (.02–.39)	0.56 (.38–.69)	0.30 (.15–.43)	0.59 (.50–.66)	0.33 (.23–.43)
	F4	0.68 (.57–.75)	0.70 (.61–.77)	0.28 (.09–.45)	0.60 (.43–.71)	0.42 (.27–.54)	0.69 (.62–.75)	0.42 (.32–.50)
Theta	O1	0.86 (.82–.90)	0.86 (.81–.89)	0.56 (.41–.68)	0.61 (.45–.71)	0.37 (.19–.50)	0.86 (.83–.89)	0.49 (.40–.57)
	O2	0.86 (.81–.89)	0.87 (.83–.90)	0.60 (.45–.70)	0.61 (.45–.72)	0.37 (.21–.50)	0.86 (.83–.89)	0.49 (.40–.57)
	F3	0.78 (.80–.83)	0.78 (.70–.83)	0.42 (.23–.57)	0.56 (.39–.68)	0.44 (.29–.56)	0.77 (.72–.81)	0.47 (.72–.81)
	F4	0.83 (.77–.87)	0.83 (.77–.87)	0.44 (.25–.58)	0.61 (.45–.72)	0.47 (.29–.56)	0.82 (.78–.85)	0.49 (.40–.57)
Alpha	O1	0.83 (.78–.87)	0.89 (.85–.92)	0.49 (.31–.62)	0.57 (.40–.69)	0.46 (.31–.57)	0.86 (.82–.88)	0.49 (.40–.57)
	O2	0.85 (.80–.88)	0.89 (.85–.91)	0.48 (.31–.61)	0.51 (.32–.64)	0.42 (.28–.54)	0.86 (.83–.89)	0.46 (.36–.54)
	F3	0.79 (.73–.84)	0.77 (.69–.82)	0.31 (.09–.48)	0.56 (.38–.68)	0.41 (.25–.53)	0.78 (.73–.82)	0.42 (.31–.50)
	F4	0.83 (.78–.87)	0.83 (.78–.87)	0.32 (.12–.48)	0.60 (.44–.71)	0.44 (.29–.56)	0.83 (.79–.86)	0.44 (.34–.52)
Beta	O1	0.78 (.71–.83)	0.78 (.70–.83)	0.32 (.13–48)	0.51 (.30–.65)	0.44 (.29–.56)	0.73[a] (.67–.78)	0.41[a] (.30–.50)
	O2	0.78 (.72–.83)	0.80 (.72–.85)	0.27 (.10–42)	0.52 (.30–.66)	0.45 (.30–.56)	0.78[a] (.73–.82)	0.39[a] (.29–.48)
	F3	0.81 (.75–.86)	0.76 (.68–.82)	0.23 (.01–.42)	0.30 (.10–.47)	0.35 (.20–.48)	0.79 (.74–.82)	0.31 (.20–.40)
	F4	0.80 (.73–.84)	0.82 (.76–.86)	0.28 (.07–.45)	0.25 (.04–.43)	0.41 (.27–.53)	0.81 (.76–.84)	0.33 (.23–.43)

N = maximum number of twin pairs for band and electrode.
[a] Variance equated between zygosities to estimate twin correlation, but left to vary for subsequent analyses.

sites were lower, ranging from 0.54 (delta O1–F3) to 0.73 (alpha O1–F3, alpha O2–F4, and theta O2–F4). In addition, the phenotypic correlations between bands (Table 3) were generally around 0.6, making them suitable for multivariate analysis. Correlations were generally higher at occipital compared with frontal sites, especially in the case of delta–theta correlations (0.85) which were overall the highest in magnitude. Genetic correlations (Table 3) showed a similar pattern, being higher at occipital compared with frontal, and ranged from .48 (alpha–delta at F4) to .88 (theta–delta at O1).

3.2. Variance components modelling of EEG power in four bands

Results of genetic modelling for the occipital and frontal regions are presented in Tables 4 and 5, respectively. Additive genetic influences played a large role in both occipital and frontal models, and, additionally, non-additive genetic effects had a large influence on frontal beta. Corresponding genetic loadings on the left and right hemisphere of each band could be equated without significant loss of fit in both occipital and frontal models (model 2). Dropping the entire C component (model 3) led to a significant loss of fit for both occipital and frontal models, indicating a significant influence of common environment. Sequential dropping of C factors (from lowest to highest) indicated one very small factor loading on the left-hemisphere of all four bands in the occipital model (models 4a to 4g), and one sizable factor loading on the left and right hemisphere of delta, theta, and alpha in the frontal model (models 4a to 4d).

Table 3
Maximum likelihood phenotypic (upper triangle) and genetic (lower triangle) correlations for delta, theta, alpha, and beta at occipital and frontal sites[a]

	Occipital				Frontal			
	Delta	Theta	Alpha	Beta	Delta	Theta	Alpha	Beta
Delta		*0.85*	*0.64*	*0.57*		*0.66*	*0.50*	*0.50*
Theta	0.88		*0.73*	*0.58*	0.62		*0.67*	*0.56*
Alpha	0.67	0.73		*0.64*	0.49	0.63		*0.59*
Beta	0.59	0.58	0.66		0.76	0.75	0.76	

[a] Values given are for left hemisphere. Correlations for right hemisphere are all within ±.03 of values given.

Residual hemisphere-specific genetic loadings were very small, the largest being 0.12. The factors containing these loadings could be dropped in the occipital model (model 5) but not in the frontal model (model 5), indicating that hemisphere-specific effects were only important to the frontal model. Independent assessment of each hemisphere-specific loading in the frontal model (models 7a to 7e) revealed that two were significant: an additive effect on alpha, and a non-additive effect on beta. The final best-fitting occipital and frontal models are shown in Figs. 2 and 3, respectively. For clarity, the unique environmental influences, left in a full Cholesky structure, are left out of the figures and presented in Table 6 instead.

The alternative independent pathway models that were tested did not provide good fits to the data compared to the final reduced Cholesky models. In the occipital region, the model with one general genetic factor and four band specific genetic factors (−2LL = 24317.23, d.f. = 7751, AIC = −10.27) and the two factor varimax-based model (−2LL = 24522.16, d.f. = 7754, AIC = 188.66) both fit poorly compared with the final reduced Cholesky (−2LL = 24249.703, d.f. = 7753, AIC = −81.797). Similarly, in the frontal model the 'general and specific' model (−2LL = 19619.40, d.f. = 7858, AIC = −0.60) and both the two (−2LL = 19777.21, d.f. = 7861, AIC = 151.21) and three (−2LL = 19801.90, d.f. = 7862, AIC = 173.90) factor varimax-based models all fit poorly compared with the final reduced Cholesky (−2LL = 19574.33, d.f. = 7854, AIC = −37.67).

Table 4
Goodness-of-fit statistics for multivariate models of occipital EEG bands delta, theta, alpha, and beta. The best-fitting model is in bold

		vs	−2LL	d.f.	Δ−2LL	Δd.f.	*p*-value
1	ACE Cholesky decomposition[a]		24215.50	7695			
2	Equate additive genetic (A) loadings on left and right hemisphere of each band	1	24221.00	7711	5.51	16	0.993
3	Drop C (omnibus drop)	2	24272.54	7747	51.54	36	0.045
4a	Drop C8, C7, C6, C5 (all loadings zero)	2	24221.00	7721	0.00	10	1.000
4b	Drop C4	4a	24221.66	7726	0.66	5	0.985
4c	Drop C3	4b	24223.57	7732	1.91	6	0.928
4d	Drop C2	4c	24231.56	7739	7.99	7	0.334
4e	Drop C1	4d	24272.54	7747	40.98	8	<0.001
4f	Drop right hemisphere C1 loadings	4d	24233.91	7743	2.35	4	0.672
4g	Drop left hemisphere C1 loadings	4f	24272.54	7747	38.63	4	<0.001
5	**Drop A2, A4, A6, A8***	**4f**	**24249.70**	**7753**	**15.80**	**10**	**0.106**

* A2, A4, A6, and A8 each contain a hemisphere-specific loading (right hemisphere loading without the corresponding left hemisphere loading for that band).
[a] Bands entered into Cholesky in order of frequency with left hemisphere preceding right, i.e. delta left, delta right, theta left, theta right, alpha left, alpha right, beta left, beta right.

A notable feature of both best-fitting models is a common additive genetic factor (A1) that loads strongly on all four EEG bands (occipital: 0.52–0.87, frontal: 0.41–0.68), indicating that in both occipital and frontal regions common genes influence delta, theta, alpha, and beta bands. Loadings from this common genetic factor are substantially higher, accounting for relatively more of the genetic variance, in the occipital model (64% of the influence from all genetic factors) than in the frontal model (43%). Furthermore, a factor analysis on the additive genetic correlation matrix confirmed that one factor accounted for more of the genetic variance in the occipital model (67%) than in the frontal (46%) model, even though this excluded the band-specific non-additive factors in the latter (including these would lower the percentage of *total* genetic influence accounted for by one factor).

The remaining additive genetic factors were essentially specifics (i.e. vertical arrows in Figs. 2 and 3) on theta, alpha, and beta, with some small (2–13% of variance) cross-path loadings (i.e. oblique arrows in Figs. 2 and 3). In the occipital and frontal models, respectively, the 'theta' factor (A3 in occipital, A2 in frontal) accounted for 19% and 39% of the band variance, the 'alpha' factor (A5, A4) 39% and 42%, and the 'beta' factor (A7, A6) 41% and 10%. In the frontal model, a specific non-additive factor accounted for a further 30% of the variance in beta.

Based on the final multivariate models, heritability estimates for the occipital region were delta 0.75, theta 0.85, alpha 0.86, and beta 0.78. For the frontal region, heritabilities tended to be somewhat lower, being 0.46 for delta, 0.64 for theta, and 0.73 for alpha. For beta, broad heritability was 0.80, consisting of 0.50 additive and 0.30 non-additive heritabilities.

Common environmental influences accounted for only 1 to 2% of the total variance in the occipital model. In contrast, common environment was much more influential in the frontal

Table 5
Goodness-of-fit statistics for multivariate models of frontal EEG bands delta, theta, alpha, and beta. The best-fitting model is in bold

		vs	−2LL	d.f.	Δ-2LL	Δd.f.	*p*-value
1	Cholesky decomposition: ACE (delta, theta, alpha), ADE (beta)[a]		19530.01	7813			
2	Equate left and right hemisphere genetic loadings	1	19556.61	7830	26.61	17	0.064
3	Drop C (omnibus drop)[b]	2	19591.85	7851	35.24	21	0.027
4a	Drop C6, C5, C4 (all loadings zero)	2	19556.61	7836	0.00	6	1.000
4b	Drop C2	4a	19557.60	7841	0.99	5	0.963
4c	Drop C3	4b	19562.14	7845	4.53	4	0.339
4d	Drop C1[b]	4c	19591.85	7851	29.72	6	<0.001
5	Drop A2, A4, A6, A8	4c	19603.41	7856	41.27	11	<0.001
6	Drop A3, A5, A7	4c	19574.27	7851	12.14	6	0.059
7a	Drop A8 loading on beta right**	6	19574.27	7852	0.00	1	1.000
7b	Drop A6 loading on alpha right**	6	19591.73	7852	17.46	1	0.000
7c	Drop A4 loading on theta right**	6	19574.28	7852	0.01	1	0.920
7d	Drop A2 loading on delta right**	6	19574.32	7852	0.05	1	0.827
7e	Drop D2 loading on beta right**	7a	19588.06	7853	13.78	1	0.000
8	**retain genetic right-hemisphere loadings on alpha (additive) and beta (non-additive)**	**4c**	**19574.33**	**7854**	**12.197**	**9**	**0.202**

*A2, A4, A6, and A8 each contain a hemisphere-specific loading (right hemisphere loading without the corresponding left hemisphere loading for that band). ** Each hemisphere-specific loading was assessed for significance independently (7a–7d), i.e. to reduce any bias of significance testing. For 7e, the additive (A) and non-additive (D) genetic loadings on beta could not be assessed independently, so the non-additve loading was assessed with the additive loading fixed at zero.
[a] Bands entered into Cholesky in order of frequency with left hemisphere preceding right, i.e. delta left, delta right, theta left, theta right, alpha left, alpha right, beta left, beta right.
[b] C loadings of left and right hemisphere could not be equated ($\Delta-2LL = 23.59$, $\Delta d.f. = 3$, $p < .0001$)

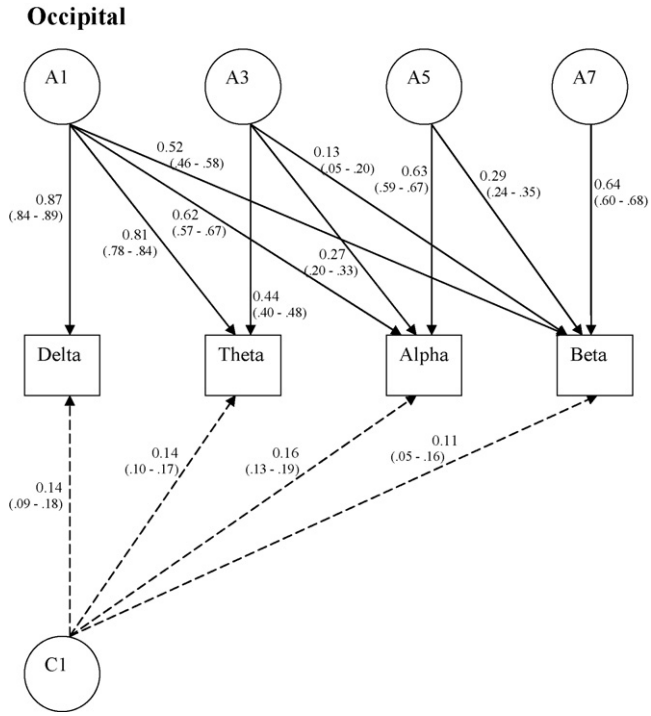
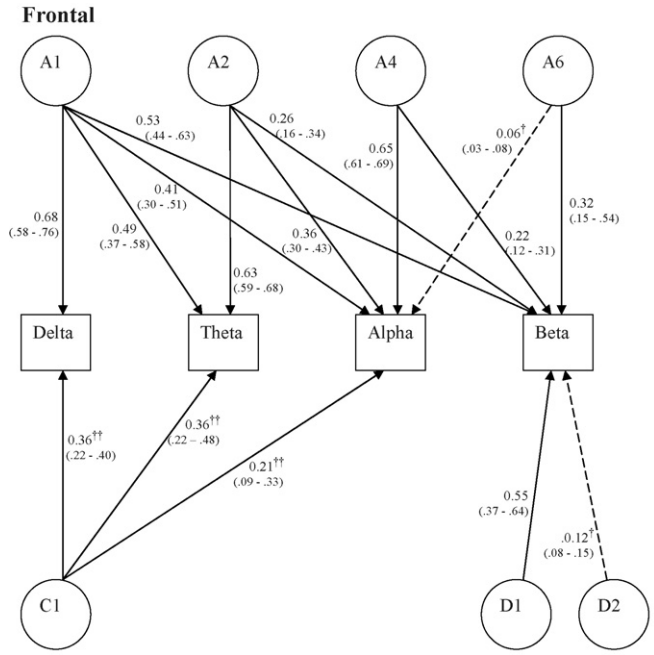


Fig. 2. Structural equation model with additive genetic ('A' circles) and common environmental ('C' circles) factors explaining the variance of EEG power bands delta, theta, alpha, and beta (squares) in the occipital region. Path coefficients are standardised such that the squared path coefficient indicates the percentage of variance accounted for, and 95% confidence intervals are in parentheses. As additive genetic factor loadings on right and left hemispheres of each band were equated, right and left hemisphere EEG power bands are collapsed into one square (e.g. the delta square represents delta left and delta right), but as described in the methods were analysed in an eight-variable Cholesky model. Also for clarity, the unique environmental (E) influences are not shown in this figure but in Table 6.



†The D2 factor loading on beta and the A6 factor loading on alpha only apply to right hemisphere (denoted by dashed lines).

†† C1 loading values shown are for left hemisphere; while of similar magnitude, right hemisphere loadings are consistently higher and therefore could not be equated to left hemisphere loadings. Right hemisphere C1 loadings on delta, theta, and alpha are .42, .39, and .27 respectively.

Fig. 3. Structural equation model with additive genetic ('A' circles), dominant genetic ('D' circles), and common environmental ('C' circles) factors explaining the variance of EEG power bands delta, theta, alpha, and beta (squares) in the frontal region. Path coefficients are standardised such that the squared path coefficient indicates the percentage of variance accounted for, and 95% confidence intervals are in parentheses. As additive genetic factor loadings on right and left hemispheres of each band were equated, right and left hemisphere EEG power bands are collapsed into one square (e.g. the delta square represents delta left and delta right), but as described in the methods were analysed in an eight-variable Cholesky model. Also for clarity, the unique environmental (E) influences are not shown in this figure but in Table 6.

Table 6
Parameter estimates for the influence of unique environment (E) on left and right delta, theta, alpha, and beta EEG bands

Occipital	E1	E2	E3	E4	E5	E6	E7	E8
Delta left	0.48							
Delta right	0.33	0.37						
Theta left	0.27	0.00	0.25					
Theta right	0.18	0.23	0.21	0.15				
Alpha left	0.17	−0.03	0.14	0.01	0.25			
Alpha right	0.09	0.15	0.12	0.09	0.19	0.22		
Beta left	0.22	0.09	0.13	0.04	0.11	0.01	0.35	
Beta right	0.11	0.21	0.11	0.09	0.10	0.11	0.23	0.25

Frontal	E1	E2	E3	E4	E5	E6	E7	E8
Delta left	0.64							
Delta right	0.39	0.46						
Theta left	0.31	0.08	0.36					
Theta right	0.17	0.25	0.26	0.21				
Alpha left	0.22	0.04	0.28	0.04	0.32			
Alpha right	0.16	0.17	0.21	0.08	0.27	0.14		
Beta left	0.21	0.06	0.20	−0.01	0.10	0.00	0.32	
Beta right	0.13	0.20	0.10	0.08	0.07	0.09	0.25	0.21

Loadings greater than $|0.04|$ are significant ($p < .05$).

model, accounting for 13–18% of the variance in delta and theta bands, and 4–7% in alpha.

Unique environment and error accounted for 11 to 23% of the variance at occipital sites, depending on band and hemisphere, and 11 to 40% at frontal, but there were no striking patterns to the variation within this range, aside from delta having a relatively higher E component in both occipital and frontal.

4. Discussion

These results support previous research showing that the human EEG is a highly heritable trait (van Beijsterveldt et al., 1996; van Beijsterveldt and van Baal, 2002; Vogel, 2000). Genetic factors accounted for a substantial amount of the variance in all power bands and brain regions studied. Modelling suggested that this may, at least in part, be explained by common genetic factors influencing all bands in both hemispheres. However, genetic factors specific to bands and brain regions were also shown to be influential: in particular,

non-additive genetic influence was found only for beta power at frontal sites, and in contrast to previous research, a common environment factor was found to influence the power bands at frontal sites. As such, additive genetic (A), non-additive genetic (D), common environment (C), and unique environment (E) factors were all found to have important influences on the human EEG.

To a large degree the same genetic factors were influencing EEG in the left and right hemispheres, but there was not the same concordance for genetic influence in the occipital and frontal lobes of the brain; as such, we refer to occipital and frontal regions, only specifying left or right hemisphere when necessary. In both occipital and frontal regions, multivariate genetic analysis (and a factor analysis) indicated that one common factor could account for a large proportion of the genetic variance in all bands. This general factor may reflect basic structural features such as skull thickness, which would determine the distance from the electrode (on the scalp) to the current generators (underlying pyramidal cells), affecting EEG power over the entire frequency spectrum. Alternatively, it could reflect neural properties or processes that are influenced by the same set of genes and have a broad effect on power across the frequency range. The proportion of genetic variance that this one general factor could account for differed markedly between occipital (64%) and frontal (43%) regions. This can be interpreted as indicating that the genetic architecture underlying EEG has more band-specificity, or is more complex, in anterior regions than in the posterior regions. Such an interpretation would make sense in light of neuroanatomical research which demonstrates that the neural structure of the cerebral cortex is very heterogeneous (Elston, 2003). Pyramidal cells are the most ubiquitous neurons in the cortex (DeFelipe and Farinas, 1992; Nieuwenhuys, 1994; Valverde, 2002), and those in frontal areas are larger, much more structurally complex, and have up to 23 times more spines than those in occipital areas. This means that neuronal circuits are many times more complex and have far greater functional capacity in frontal than occipital areas (Elston, 2003). Since EEG is partly a reflection of the pyramidal cell circuitry in the underlying cortex (Schaul, 1998), a more complex genetic influence on frontal compared with occipital EEG would be expected.

The finding of band-specificity in the genetic effect on EEG fits with the research indicating band-specific effects in psychiatric disorders such as alcoholism (Porjesz et al., 2005), ADHD (Lazzaro et al., 1998), and obsessive/compulsive disorder (Pogarell et al., 2006), thereby enhancing EEG power as a potential endophenotype for these conditions. It also implies that there may be neural substrates or generators whose influence on EEG is specific to certain frequency ranges or bands. Different neural networks (e.g. thalamo-cortical, cortico-cortical) or neurotransmitter systems (e.g. cholinergic, GABAergic, dopaminergic) may be responsible for electrical rhythms of different frequencies. For example, the slow waves delta and theta have been associated with cholinergic systems (Steriade et al., 1990), while the beta range involves the action of $GABA_A$ (Whittington et al., 2000).

A striking example of a band- and site-specific genetic influence on frontal EEG was the large and highly significant non-additive genetic effect on the beta band in the frontal lobe, which was not present at the occipital sites. The results of past research have hinted at genetic dominance in the beta band (van Beijsterveldt et al., 1996), but some more recent research has failed to detect any significant effect (Smit et al., 2005). In our findings, monozygotic twin correlations were more than double the dizygotic twin correlation for beta in both left and right frontal areas, and in separate left and right frontal models, a non-additive genetic effect on beta was large and significant. In the more powerful left–right combined final frontal model, the non-additive factor on beta was highly significant (30% of the variance). Genetic linkage (gene-finding) studies will only be able to accurately determine the sample size required for a certain level of statistical power if non-additive genetic effects are accounted for. From an evolutionary perspective, substantial non-additive genetic effects suggest that the phenotype in consideration was subject to intense selection pressure at some stage (Merila and Sheldon, 1999). In this case that phenotype may be the balance in neural excitation–inhibition homeostasis that beta power seems to reflect (Porjesz et al., 2005).

There are several possible explanations for the non-additive (i.e. D) effect. As EEG power is a complex trait and probably polygenic, D is likely to include dominance relationships between genes at different loci, which is called epistasis (Lykken, 1982). Another polygenic mechanism that can create a D effect, called emergenesis, involves gene effects that combine configurally rather than additively. That is, an emergenic trait relies on the configuration of its component traits, which are themselves influenced by independent genetic effects (Lykken, 1982; Lykken et al., 1992).

Further genetic complexity present in frontal but absent in occipital regions was found in the form of hemisphere-specific genetic loadings on alpha (i.e. dashed arrow from A6) and beta (i.e. dashed arrow from D_{RH}) bands. These loadings were very small, accounting for less than 2% of the variance in each band, but significant. These genetic components may be important when viewed in the context of research suggesting a link between frontal EEG alpha asymmetry and predisposition to depressive disorders (Bruder et al., 2005), and that frontal EEG asymmetry has been shown to have low but significant heritability (Anokhin et al., 2006). It may be that the small lateralised genetic effects on alpha comprise genes that also influence depressive disorders.

A striking result, and a further occipito-frontal difference, is the presence of a highly significant effect of common environment, which is negligible in size in the occipital model but substantial in the frontal model. Aside from a study on infants (Orekhova et al., 2003) which found a C effect that decreased with age, no other study has found a significant influence of common environment on EEG. It therefore seems to be accepted that beyond early childhood the environment plays little or no role in determining the human EEG (particularly as a substantial portion of E may be explained as error, rather than an effect of unique environment). However,

our results suggest that environmental influences may play a more lasting and important role. The large sample size and left–right combined multivariate analysis yielded greater power to detect C than previous studies, and may explain why we have found it where other studies have not. A common environment effect on frontal EEG is consistent with research demonstrating neural plasticity in response to environmental cues, particularly developmental animal research, showing that brains develop more complex circuitry in enriched environments than in impoverished ones (see Renner and Rosenzweig (1987)). Although the circuitry in the visual (occipital) cortex is also susceptible to environmental influence (Wiesel and Hubel, 1963), there is not likely to be a differential between home environments in visual input, so a negligible occipital C effect is unsurprising.

It is important to note that the influence of common environment is confounded with the influence of non-additive genetic effects, so that both D and C cannot be estimated for any single variable. This means that where D is observed (frontal beta power), C may also play a smaller role (hence depressing the measured D effect), and, vice versa, D may play a hidden role where C is observed (frontal delta, theta and alpha) (Neale and Cardon, 1992). A corollary of this is that there is no evidence for band-specificity of the C effect in this study, as the one C factor influencing frontal delta, theta, and alpha powers may also have an unseen influence on beta power. Another methodological consideration is the possibility of experimental testing effects manifesting as a C effect, but this is unlikely given that the same testing conditions and experimenter were used for all twin pairs, and that a substantial C effect was not observed at the occipital sites (a testing effect would have a global impact).

There are sex differences in the structure and function of the human brain (Good et al., 2001), and one might expect EEG data to reflect this. However, finding of sex effects on EEG is mixed; a few studies report a sex effect of some kind, but they are not consistently found (see Vogel (2000) for a review). In the present study, a significant mean effect on sex was found at six of the 16 power bands (at left and right occipital and frontal), with these generally indicating lower power for males (the exception being right frontal delta, which has higher power in males). Females were found to have a larger variance in beta in the occipital sites, but we found no evidence for sex differences in the genetic effects.

Although our adolescent sample had a very narrow age range, we found significant age effects at all sites for all EEG bands, with the exception of occipital alpha. In all cases the direction of the effect was such that older participants tended to have lower EEG band power than younger participants. This is consistent with a study by Martinovic et al. (1998), but other research has found that while slow waves delta and theta decreased through development, alpha, and beta powers actually increased (Clarke et al., 2001; Matousek and Petersen, 1973), and further work suggests that developmental changes in EEG are complex (Klimesch, 1999).

In summary, this study shows that genetic influences are more complex and more heterogeneous across the cortex than previous work suggests. Genetic influences take the form of common and band-specific effects, the former more influential at occipital sites and the latter more influential at frontal sites. Importantly, EEG beta power exhibits non-additive inheritance at frontal but not occipital sites, and the environment plays a greater role than previously thought, with common environment having a significant and substantial impact on frontal EEG power.

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