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Genetic variation of individual alpha frequency (IAF) and alpha power in a large adolescent twin sample

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Abstract

To further clarify the mode of genetic transmission on individual alpha frequency (IAF) and alpha power, the extent to which individual differences in these alpha indices are influenced by genetic factors were examined in a large sample of adolescent twins (237 MZ, 282 DZ pairs; aged 16). EEG was measured at rest (eyes closed) from the right occipital site, and a second EEG recording for 50 twin pairs obtained approximately 3 months after the initial collection, enabled an estimation of measurement error. Analyses confirmed a strong genetic influence on both IAF (h^2=0.81) and alpha power (h^2=0.82), and there was little support for non-additive genetic (dominance) variance. A small but significant negative correlation (−0.18) was found between IAF and alpha power, but genetic influences on IAF and alpha power were largely independent. All non-genetic variance was due to unreliability, with no significant variance attributed to unique environmental factors. Relationships between the alpha and IQ indices were also explored but were generally either non-significant or very low. The findings confirm the high heritability for both IAF and alpha power, they further suggest that the mode of genetic transmission is due to additive genetic factors, that genetic influences on the underlying neural mechanisms of alpha frequency and power are largely specific, and that individual differences in alpha activity are influenced little by developmental plasticity and individual experiences.

Keywords: EEG; Alpha rhythm; IAF; Alpha power; Heritability; Twin study

1. Introduction

The EEG alpha rhythm, an oscillation with a frequency around 10 waves (cycles) per second, dominates the EEG power spectrum recorded from the brain during rest. Consequently it has become the anchor point for quantitative analysis of the EEG, and, due to the continuing interest in understanding brain processes in the resting state, which defines a baseline for brain activity, is one of the most widely studied physiological indices of brain function (Shaw, 2004). Indeed, this spontaneous brain activity, which is maximal with eyes closed and blocked with eyes open, has high intra-individual stability (Binnie et al., 2003; Fernandez et al., 1993), shows a considerable amount of variation among individuals (Klimesch, 1997; Posthuma et al., 2001), and is found to change with age (Li et al., 1996; McEvoy et al., 2001) and mental state (Moretti et al., 2004).

Individual variation in the alpha rhythm has been posited to reflect individual differences in working memory, attentional demands and/or arousal, and cognitive preparedness (Shaw, 2004), although further work is still required to fully clarify its functional significance. Notable is the body of evidence showing alpha frequency, the peak frequency within the spectral alpha band, to have a strong relationship with working memory performance (Klimesch, 1999), faster information processing (Klimesch et al., 1996), and that participants with superior memory performance have an alpha frequency approximately 1 Hz higher than age-matched controls (Klimesch, 1996,1997). This is supported, for example, by the findings that alpha frequency increases from early childhood to adulthood and then decreases with age (e.g. Li et al., 1996; McEvoy et al., 2001) in a similar way to general cognitive performance, and of higher peak frequencies in children with

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higher reading performance compared with age-matched controls (Suldo et al., 2001). These cumulative findings suggest that the alpha rhythm should be related to general cognitive functioning as measured by intelligence tests. However, while alpha frequency has been found to be positively correlated with specific verbal and non-verbal abilities (Anokhin and Vogel, 1996), on balance it does not appear that alpha frequency reflects general intelligence (Anokhin and Vogel, 1996; Jausovec and Jausovec, 2000; Posthuma et al., 2001; Shaw, 2004). Likewise, while a relatively recent study showed a positive association between alpha power and general cognitive ability, the strength of the relationship being dependent on the type of IQ test (Doppelmayr et al., 2002), there are many reports indicating negative or inconsistent findings (e.g. Gaser et al., 1983).

An important generator or modulator of cortical alpha activity is the thalamus, with evidence of a close relationship between thalamic and EEG alpha activity mediated by cortico-thalamic loops (e.g. Lopes da Silva, 1999; Nunez et al., 2001). The occipital cortex is also thought to be important, with the hippocampus and the reticular formation posited to have a more general role. Indeed, several strands of work suggest that alpha oscillations are the result of widespread neuronal activity, and that alpha activity arises from multiple cortical generators (Basar, 1998, 1999). What is recorded at the scalp is thought to be a spatial average of a large number of components (i.e. the interaction of neural firing patterns generated by several circuits), with alpha activity being dependent on which components are the most highly synchronized over the largest area. More specifically, EEG alpha power is thought to reflect the number of neurons that discharge synchronously (Klimesch, 1999), and the higher the number of synchronously active neurons, the higher the amplitude of the alpha rhythm. There is also some indication of an inverse relationship between the amplitude of the alpha rhythm and alpha frequency, such that the higher the amplitude the slower the frequency of the alpha peak (Lopes da Silva et al., 1976; Pfurtscheller and Lopes da Silva, 1999; Singer, 1993), but more recent work suggests that each of these measures may capture different neural processes (Moretti et al., 2004).

Genetic studies, of which there have been a considerable number (reviewed by (Kuhlo, 1976; van Beijsterveldt and Boomsma, 1994; Vogel, 2000), the first study being in 1936 (Davis and Davis, 1936), all indicate extraordinary similarity of the alpha rhythm for MZ twin pairs and, where estimated, high heritability indicating that individual differences in alpha activity are to a large extent mediated by genetic influences. However, it is still not clear whether additive genetic or a combination of additive and non-additive genetic factors play a role in the genetic transmission, with a number of studies indicating a pattern of very low DZ co-twin correlations that are much less than half the corresponding MZ twin correlations (Christian et al., 1996; Lykken et al., 1974; Lykken et al., 1982; Posthuma et al., 2001; Stassen et al., 1999). While there are large differences across studies, especially with respect to age, EEG methodology, and genetic analysis, many of the studies are underpowered, especially for the detection of dominance as a large number of twin pairs are required. More recently, a meta analysis comprising five twin studies that measured individual alpha frequency (IAF), and eleven studies that measured alpha power (inclusion of studies based on overlap of EEG methodology and availability of heritability estimates), attempted to resolve this question of the importance of dominance (van Beijsterveldt and van Baal, 2002). For IAF a robust 'meta' heritability of 81% was indicated, with non-additive genetic factors shown to be important. However, for alpha power where it was not possible to equate estimates across studies, and an averaged heritability of 79% was calculated with no definitive test for dominance, non-additive genetic factors were indicated for adults, but for adolescents and children a purely additive genetic model was shown to be more likely.

The aim of the present study, therefore, was to utilise EEG data from a large sample of adolescent twin pairs, over half of whom were DZ twin pairs and all of the same age (237 MZ, 282 DZ pairs; aged 16), to further examine the extent to which individual differences in both alpha frequency and alpha power were influenced by genetic factors, and to provide additional information on the possible mode of genetic transmission. It also forms one of few studies to examine alpha frequency (i.e. IAF) and alpha power in the same sample, and the first to investigate whether any association between them is due to common genetic or environmental factors, or whether the substantial genetic influences on alpha frequency and alpha power are largely independent of each other. A final aim, since an assessment of psychometric IQ was available, was to explore associations between alpha indices and cognitive ability, and the extent to which any co-variations were genetically mediated.

2. Materials and methods

2.1. Participants

Participants were adolescent twins recruited through South East Queensland primary and secondary schools as part of a study on the genetics of melanoma risk factors (Zhu et al., 1999), and a genetic study of cognition, the Memory, Attention and Problem Solving study (MAPS) (Wright et al., 2001a), of which the recording of resting EEG was a component. The sample consisted of 543 females and 495 males aged 16 years (mean age = 16.24, SD = .35), and included five zygosity groups, 128 MZ (identical) female pairs (MZF), 109 MZ male pairs (MZM), 71 DZ (non-identical) female pairs (DZF), 66 DZ male pairs (DZM) and 145 DZ opposite-sex pairs (DZOS). Zygosity was determined by typing 9 independent polymorphic DNA markers using the AmpFLSTR® Profiler® PCR Amplification Kit and crosschecked with ABO, MN and Rh blood groups and/or phenotypic information (hair, skin and eye colour). Based on this, zygosity was assigned with an extremely low probability of error (less than 10^{-3}).

Twin pairs were excluded from participation if parental report indicated either one had a history of a head injury, neurological or psychiatric illness, substance abuse or dependence, or current use of medication with known effects on the

central nervous system (not including previously concluded short-term treatment). Prior to testing, written informed consent was obtained from all participants and their parent or guardian. Ethics approval for the study was obtained from the Human Research Ethics Committee, Queensland Institute of Medical Research.

2.2. General procedure

The MAPS study protocol included two parallel testing sessions: a psychometric part assessing processing speed and IQ (Luciano et al., 2001) and a psychophysiological part in which 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. While one twin did the psychometric session their co-twin undertook the psychophysiological session, and following a short break, each twin completed the complementary session.

Resting EEG comprised two 4 min recordings, the first with eyes closed followed by eyes open. During the eyes closed condition participants were asked to relax and sit quietly with their eyes closed, to minimize any movement, and were informed that the duration of the recording would be approximately 5 min. During the eyes open condition they were asked to focus on the monitor in front of them, and again to sit quietly, and be relaxed. Recordings took place in a semi-darkened, electrically shielded, and sound-attenuated cubicle.

2.3. EEG recording

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 with tin electrodes 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 and were 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 5 K and all other channels with a factor 20 K by Grass preamplifiers (model P511 K), and recordings were filtered with a band pass filter of 0.01 to 100 Hz (6 dB per octave) and a 50 Hz notch filter.

Software controlling the recording determined that the maximum length of continuously recorded EEG was 12 s with a slight discontinuity of 2 s between successive 12 s blocks. Twenty 12 s blocks were recorded for each of the eyes closed and eyes open condition.

To generate power spectra EEG data were processed for both recordings, with data divided into sixty 4 s 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 with voltages exceeding ±1000 μV for Fp1, Fp2 and EOG, and ±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. EEG was then converted from the time domain to frequency domain (resolution 0.25 Hz) using Fast Fourier Transformation (Niedermeyer and Lopes da Silva, 1999), for a window ranging from 2 to 40 Hz.

2.4. Determination of individual alpha frequency and the alpha power band

IAF was determined as described by Posthuma et al. (2001) following the method of Klimesch (1999). Briefly, in the eyes closed condition an automatic peak picking procedure found the highest peak (maximum alpha power) in a 7 to 14 Hz window. IAF was determined at the point of peak difference in spectral power between the eyes closed and eyes open conditions within the alpha range. As alpha rhythm is generally largest at occipital sites and the IAF correlation between left and right occipital sites was larger than 0.90, IAF was determined at the O2 electrode only. In 26% of cases, visual inspection was required since 1) the frequency of the highest peak in the eyes closed condition did not coincide with the frequency of the highest peak in the eyes open condition, 2) there were fewer than 57 (out of 60) epochs, 3) the highest peak frequency was within 1 Hz of the border (range 7–14 Hz). After inspection, 4% (46 of 1038 individuals) of the sample was eliminated from further analyses, since either no peak could be detected (power <1 μV^2) or the number of epochs was too low.

For alpha power IAF was used as an anchor point to calculate the power in the alpha band that spanned 4 Hz below IAF to 2 Hz above IAF (Klimesch, 1999). Thus for an individual with an IAF of 9.25 Hz the bandwidth for alpha ranged from 5.25 to 11.25 Hz. Alpha band power was computed as the sum of the power frequency bins within this 6 Hz band.

2.5. Intelligence testing

A shortened version of the computerized Multidimensional Aptitude Battery (MAB, (Jackson, 1998)), which consisted of three verbal (Information, Arithmetic and Vocabulary) and two performance (Spatial and Object Assembly) subtests, was used. The verbal subtest Information score provides an estimate of the degree to which an individual has accumulated knowledge about a broad range of topics, while the Arithmetic subtest reflects reasoning and problem solving abilities and Vocabulary provides an indication of the number of words or verbal concepts an individual has learned and stored. The performance subtests measure the ability to visualize abstract objects in different (two-dimensional) positions (Spatial) and how separate parts of an object are assembled (Object Assembly). In addition, the Digit Symbol subtest of the Wechsler Adult Intelligence Scale—Revised (WAIS-R) was administered, which taps mainly figural memory and speed of information processing. Further details regarding the MAB, its Verbal and Performance scales, and subtests, and the Digit Symbol are

Table 1
Mean (SD) and range for individual alpha frequency (IAF) and alpha power at the O2 lead for the full and re-test samples

	Mean (SD)	Range
Full sample		
IAF (N=986)	9.62 (0.81) Hz	7.25–12.00 Hz
Alpha power (N=991)	190.5 (153.2) μV^2	8.8–1217.45 μV^2
Re-test sample		
IAF (N=95)	9.39 (0.89) Hz	7.50–11.75 Hz
Alpha power (N=95)	171.2 (146.7) μV^2	16.8–1009.3 μV^2

provided in previous papers from this laboratory (Luciano et al., 2001; Wainwright et al., 2004).

2.6. Re-testing of sub-sample of twins

Fifty twin pairs (25 MZ, 25 DZ) returned after approximately 3 months for re-testing. This sample size was considered large enough to obtain good estimates of reliablity. The general procedure and processing of IAF and alpha power was identical to the first visit. After inspection of the EEG data five individuals were eliminated from further analyses since no alpha peak frequency could be identified.

2.7. Statistical analysis

Data were analysed with the statistical package MX (Neale et al., 2002) using Maximum Likelihood (ML) estimation procedures. All data were screened for normality, univariate and multivariate outliers prior to analysis. Preliminary testing of basic assumptions concerning the equality of means and variances within twin pairs and across zygosity, and equality of means according to sex were tested prior to genetic modelling. In addition, hypotheses about the covariance structure, including the equality of covariance between MZF and MZM and between DZF and DZM to assess potential differences in the magnitude of genetic effects according to sex, and equality of covariance between same sex DZ twin pairs and opposite sex DZ twin pairs to determine whether different genes were being expressed according to sex, were tested. Successive nested models with increasingly restrictive equality constraints upon means and variances were assessed, with each model being compared to the previous less restrictive model using a χ^2 statistic. A significant change in fit was interpreted as a given constraint hypothesis being unlikely to be true.

Multivariate genetic modelling was conducted to estimate the genetic (A—additive genetic; D—non-additive (dominance)) and non-genetic (unique (E) environmental effects not shared by co-twins plus measurement error) variance of IAF and alpha power, and to explore the relationship between these two indices. The additional information in the cross-correlations (e.g. correlation between IAF for twin 1 with alpha power for twin 2) determines the extent to which genetic influences are shared by the two phenotypes or are phenotypic specific. For MZ twins the co-variance was defined as additive (A) plus dominant (D) factors, and for DZ twins, 0.5A+0.25D (i.e. DZ cotwins share only half their genes on average meaning that only half the additive genetic variance contributes to the dizygotic covariance, and a quarter of the dominant genetic variance). Note that in absence of data from separated twins or half siblings the non-additive genetic (D) and common (C) environmental effects, which are shared within a twin pair, are confounded, so that only one can appear in a given model. Given the importance of dominance indicated in previous studies, and that the DZ correlation for IAF was less than half the magnitude of the MZ correlation, a model including genetic dominance rather than shared (common) environment was fitted to the data. Further, as the pattern of covariances for both IAF and alpha power indicated the size and the aetiology of genetic variation between males and females was similar (i.e. equality of covariances between MZM and MZF, and between DZM, DZF and DZOS), a single ADE model, equating A, D, and E parameters across males and females, was examined, with subsequent nested models tested in which each of the A, D and E parameters were dropped. Re-test data for both IAF and alpha power were included in the model allowing an estimate of the error term, with the assumption of equal variance at test and re-test. Parameter estimations for effects on IAF were equated for test and re-test, and similarly for alpha power. Finally, maximum likelihood (ML) correlations explored the relationship between the alpha indices and the various IQ measures available.

3. Results

3.1. Preliminary analyses

For IAF, one individual was excluded for having an outlying value (±3 SD from mean), and three pair-wise outliers were identified in Mx (%P option provides a likelihood statistic for each pair in the fully saturated model). For alpha power, only one univariate outlier was identified. Significance tests for skewness and kurtosis were used to assess the normality of the sample for IAF and alpha power. IAF values were normally distributed but alpha power was log transformed. Both IAF and alpha power were standardized prior further analyses.

Table 1 shows the descriptive statistics for IAF and alpha power for the total and smaller re-test samples. For IAF, equality of means and variances across birth order and zygosity were indicated, and there was no sex effect on the mean. While a difference in the variances for males (0.81 SD) and females

Table 2
Twin correlations (ML) and their 95% confidence intervals (95% CI) for individual alpha frequency (IAF) and alpha power at the O2 lead for each of the five zygosity groups and MZ and DZ groups

	IAF		Alpha power	
	N (prs)	r (95% CI)	N (prs)	r (95% CI)
MZ females	113	0.85 (0.80, 0.89)	115	0.82 (0.77, 0.86)
MZ males	104	0.88 (0.84, 0.91)	105	0.83 (0.77, 0.87)
DZ females	61	0.56 (0.38, 0.68)	61	0.41 (0.20, 0.57)
DZ males	61	0.28 (0.02, 0.55)	61	0.42 (0.20, 0.58)
DZ opposite sex	131	0.29 (0.13, 0.42)	131	0.41 (0.25, 0.53)
MZ	217	0.86 (0.83, 0.89)	220	0.83 (0.78, 0.86)
DZ	253	0.36 (0.25, 0.47)	253	0.41 (0.30, 0.50)

(1.16 SD) was indicated, this was largely driven by the lower variance in the smaller DZM group and inconsistent with the larger MZM group, which could be equated to the female groups. Consequently all means and variances were equated according to birth order and zygosity in subsequent modelling of residual genetic and environmental effects. For alpha power means and variances were homogeneous across birth order and zygosity, and there were no sex effects.

For both IAF and alpha power, covariances of female and male MZ twin pairs could be equated, as could the covariances for female and male DZ twin pairs for which the 95% confidence intervals overlapped, indicating an equal magnitude of genetic effects for females and males for each of the measures. As such, sex limitation effects were not modelled for any of the variables. Likewise, there were no significant differences for any of the variables between same sex DZ twin

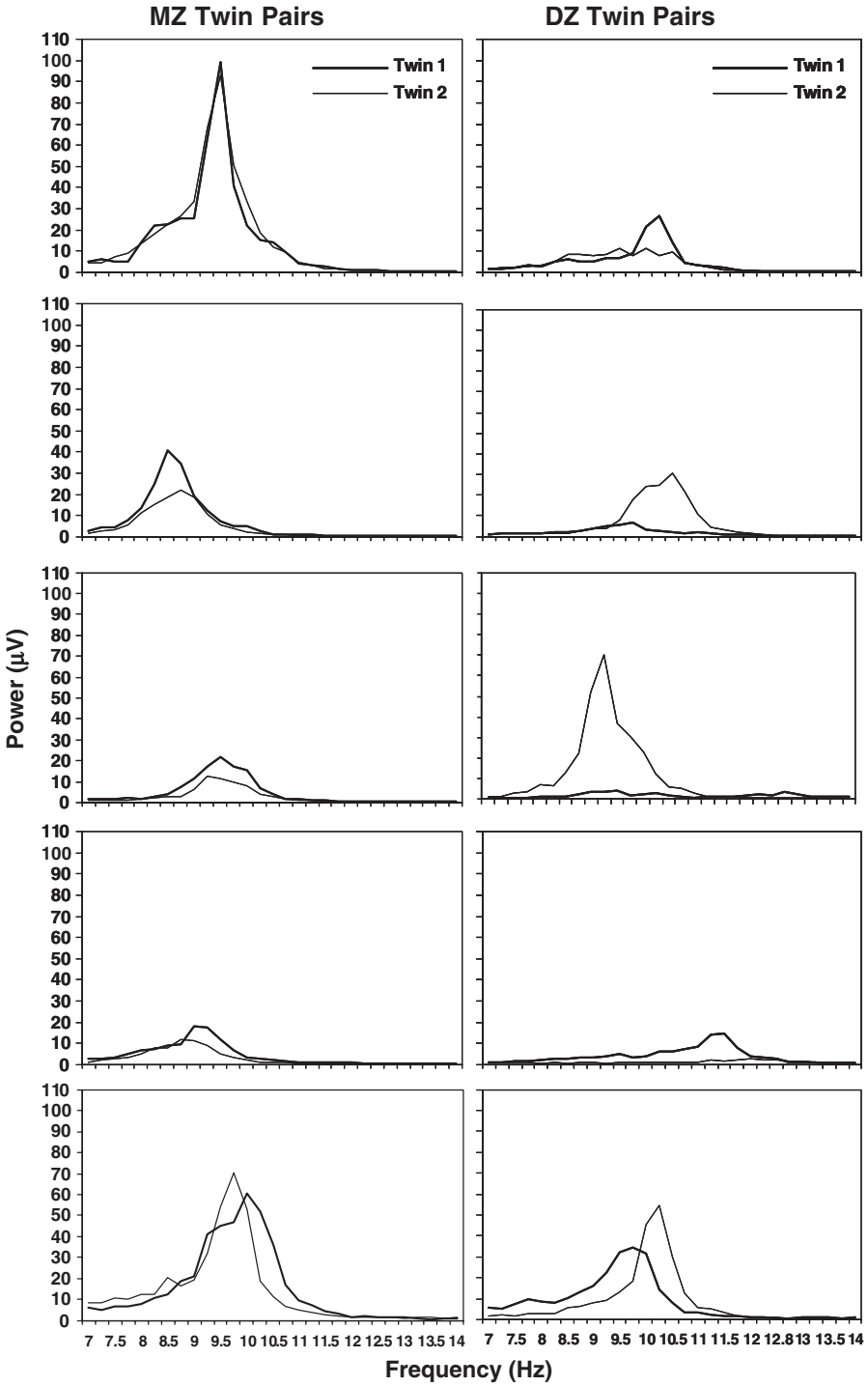

Fig. 1. The occipital (O2) spectral alpha distribution for five randomly chosen MZ (left panel) and five randomly chosen DZ (right panel) co-twins (twin 1 is the first born, twin 2 the second born). MZ pairs show remarkable similarity compared with DZ pairs.

pairs and opposite sex DZ twin pairs indicating that for each measure the same genes were acting on females and males (i.e., no evidence for non-scalar sex limitation). Twin pair correlations are reported in Table 2. While correlations are shown for each of the five zygosity groups, all genetic analyses were run with one MZ and one DZ group. For both IAF and alpha power the MZ twin pair correlations were significantly larger than the DZ correlation.

Fig. 1 illustrates power spectra for five MZ and five DZ twin pairs that were randomly chosen from the sample pool. MZ co-twins show remarkable similarity compared with DZ pairs, and indeed on visual inspection of the spectra zygosity could usually be guessed correctly. The figure also illustrates the large individual variation in both IAF and alpha power.

3.2. Multivariate genetic analysis of IAF and alpha power

At the phenotypic level there was a small but significant correlation between IAF and alpha power ($r=-0.18$ (95% CI: -0.29 to -0.10)). When an ADE model was fitted to the data, the D factor could be dropped from the model ($\Delta\chi^2=1.48$, $\Delta df=3$). Dropping of both additive genetic (A) and non-additive (D) genetic factors provided a poor fit to the data ($\Delta\chi^2=584.19$, $\Delta df=6$). The path diagram in Fig. 2 provides parameter estimates with confidence intervals (ML) for the full AE model (i.e. non-significant paths are shown). The first genetic factor (A1) significantly influenced IAF and alpha power accounting for 81% and 3% of the variances, respectively, (i.e. the majority of the variance in IAF). Similarly, the second genetic factor (A2) accounted for the majority of the variance (79%) in alpha power. Resultant heritability estimates for IAF and alpha power were thus very similar −0.81 for IAF and 0.82 for alpha power. Of the unique environment factors, neither E1 nor E2 had a significant effect on either IAF or alpha power. All estimates for specific and common unique environmental (E) paths shown in Fig. 2 were non-significant. Indeed it was unreliability (*U*: measurement error) that accounted for the remainder of the non-genetic variance, 18% for IAF and 16% for alpha power.

Table 3
ML correlations (95% CI) for IAF and alpha power at O2 with IQ subtests, verbal (VIQ), performance (PIQ) and full scale IQ (FIQ)† ($N=913-987$ individuals)

	IAF	Alpha power
Information	0.06 (−0.01, 0.14)	−0.04 (−0.11, 0.04)
Arithmetic	0.06 (−0.01, 0.13)	−0.03 (−0.10, 0.04)
Vocabulary	**0.09** (0.02, 0.16)	−0.05 (−0.12, 0.02)
Spatial	0.01 (−0.06, 0.08)	−0.02 (−0.09, 0.05)
Object assembly	0.04 (−0.03, 0.11)	**−0.08** (−0.14,−0.01)
Digit symbols	0.07 (−0.01, 0.14)	**−0.09** (−0.16,−0.01)
VIQ	0.07 (0.00, 0.15)	−0.04 (−0.11, 0.03)
PIQ	0.03 (−0.04, 0.10)	−0.05 (−0.12, 0.02)
FIQ	0.06 (−0.01, 0.13)	−0.06 (−0.13, 0.01)

Significant correlations shown in bold.
†FIQ for females=110±13 SD, males=114±13 SD, total=112±13 SD. For detailed genetic analyses focussing on IQ, see Luciano et al., 2001; Wainwright et al., 2004.

Given the low phenotypic correlation between IAF and alpha power, and only a small amount of the genetic variance being shared (3%), the genetic correlation between IAF and alpha power was low ($r_g=-0.19$). However, it is clear from the model that an overwhelming proportion of the covariance between these indices is due to genetic factors.

3.3. ML correlations between alpha and IQ measures

Correlations for both IAF and alpha power with the IQ subtests, verbal and performance IQ scores, and full-scale IQ are shown in Table 3. All correlations were low and for the most part non-significant. Indeed only three correlations reached significance, that between IAF and the verbal subtest Vocabulary (0.09), and a negative correlation between alpha power and the performance subtests Object Assembly

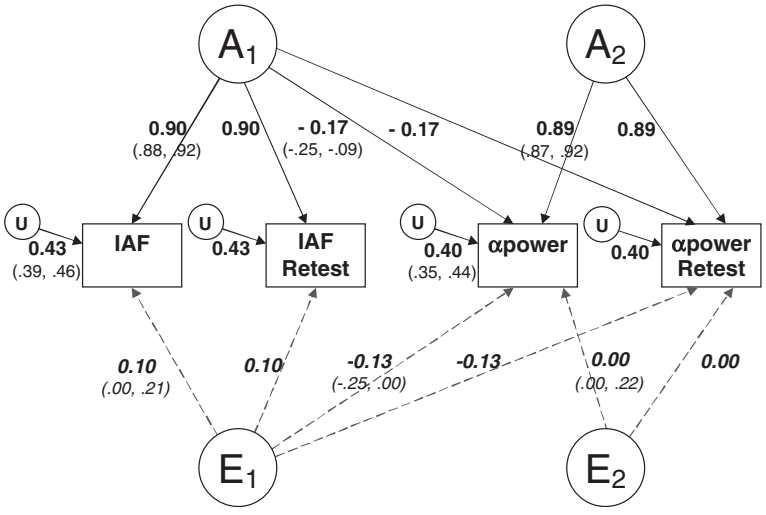

Fig. 2. Path diagram showing additive genetic (A), unique environmental (E), and unreliabity (U) (measurement error) factor loadings with 95% confidence intervals (ML). Paths to respective measures for test and re-test have been constrained to be equal and therefore have the same confidence intervals. Non-significant parameters are shown in italics and with dashed paths. IAF=individual alpha frequency, α power=alpha power.

(−0.08) and Digit Symbols (−0.09). Inter-correlations among the IQ subtest scores were not the focus of this study and we refer the interested reader to our previously published work (Luciano et al., 2001; Wainwright et al., 2004).

4. Discussion

The rationale for this study was to provide a further investigation of individual differences in the alpha rhythm utilising a large and genetically informative adolescent twin sample. The study affirms previous findings, many in smaller samples, showing very high heritability for IAF and alpha power. Heritability estimates were 0.81 for IAF and 0.82 for alpha power, in line with earlier studies and a more recent meta-analysis (van Beijsterveldt and van Baal, 2002). Further, although tempered by power, the pattern of MZ and DZ correlations supported a model incorporating only additive genetic effects with little indication of a role for genetic dominance, and all non-genetic variance was attributed to measurement unreliability rather than unique environmental factors. Findings also revealed that IAF and alpha power may index relatively distinct brain (neural) processes, with very little overlap at either the phenotypic or the genetic level.

While the heritabilities for both EEG alpha frequency and alpha power are, as expected, high and of similar magnitude to that found previously, it is also worth noting that they are considerably higher than the heritability estimates for other psychophysiological indices of brain processing, e.g. P300 amplitude and latency, which we (Wright et al., 2001b) and others (van Beijsterveldt et al., 1998) have found to be moderately heritable. Indeed the heritability of both IAF and alpha power are of the same order as we have found for general cognitive ability (heritability for IQ=0.83) (Luciano et al., 2001; Wainwright et al., 2004), and IQ is recognized as one of the most heritable behavioural phenotypes ever studied (McClearn et al., 1997; Plomin, 1999).

To separate additive from non-additive genetic effects reliably, very large sample sizes and information from many different genetic relationships are required (Martin et al., 1978), and while the size of the sample in the present study was the largest to date, there was still insufficient power to resolve the relative influence of additive vs. non-additive genetic influences. This acknowledged, the data did suggest that, at least in adolescence, additive genetic factors account for most of the (total) genetic variance in both IAF and alpha power, and that non-additive genetic effects are minimal. Means and variances of the MZ and DZ groups were shown to be homogeneous, and the pattern of MZ and DZ correlations did not strongly suggest non-additive genetic effects. Previous studies have reported low DZ correlations, but as many of these included small samples of twins, the low correlations may be attributed to sampling error. Indeed Fig. 1, which showed the alpha spectrum for 5 randomly chosen DZ twin pairs, illustrates how different IAF and alpha power for DZ co-twins can appear with a small sample. As indicated by (van Beijsterveldt and van Baal, 2002), other factors that may have accounted for differences in the DZ twin correlations between studies include the considerable heterogeneity within and across samples (e.g. with respect to age), differences in EEG methodology, and the level of statistical analysis (few used structural equation modelling to estimate genetic and environmental influences).

It was somewhat surprising that all of the non-genetic variance was due to unreliability of the measures, with no significant variance attributed to unique environmental factors. Non-genetic studies have indicated high reliability of alpha activity indices (Binnie et al., 2003; Fernandez et al., 1993), but few twin analyses have included estimates of test–re-test reliability to enable unique environmental effects to be disentangled from measurement error. If all non-genetic variance can indeed be attributed to measurement error, it suggests that at age 16 (years) there is very little plasticity and/or effects of individual experience on the neural mechanisms underlying IAF and alpha power, or more generally, this intrinsic mode of brain activity, which indexes attention and working memory processes. However, a limitation of the study is that IAF and alpha power were only investigated at one electrode position (O2). This was justified on the basis that alpha activity is largest at occipital sites and more easily determined from this site, and that preliminary analyses showed the correlation between left and right occipital sites to be very highly correlated (>0.90). While it appears that, at least for alpha power, the same genes are expressed in all brain areas, the amount of genetic and unique environmental variance does vary from region to region (van Beijsterveldt and Boomsma, 1994).

The new finding that genetic influences on IAF are for the most part independent of alpha power (i.e. largely specific genetic influences) is of some interest. Only 3% of the total variance in IAF was indicated to be in common with alpha power. Non-genetic studies have indicated a small inverse association between the two alpha measures with higher alpha frequency being associated with less alpha power (Lopes da Silva et al., 1976; Pfurtscheller and Lopes da Silva, 1999; Singer, 1993). The phenotypic correlation of −0.18 between alpha frequency and alpha power, found in the present study, is of similar magnitude and in the same direction to that indicated previously (Wieneke et al., 1980). Moreover, recently it was shown that IAF and alpha power may capture different component (neural) processes, with these two indices discriminating between mild Alzheimer's disease and vascular dementia (Moretti et al., 2004). The study reported a reduction in alpha power in Alzheimer's disease that was not associated with a proportional change in alpha frequency, whereas patients with vascular dementia, in which there are diffuse vascular lesions of the white matter, showed a slowing of alpha frequency with no overall change in alpha power. The findings of large specific genetic influences for both IAF and alpha power are in agreement with the interpretation that IAF and alpha power capture different neural processes.

The final aspect of this study was the exploration of a relationship between alpha activity and IQ indices, and the extent to which any association might be attributed to common

genetic factors. The findings did not support an association between alpha activity and IQ, for either IAF or alpha power, with only one correlation (out of a possible 9) for IAF and two for alpha power reaching significance, and all three of these correlations were very low. However it is of interest that IAF had a positive association with the IQ measures while alpha power showed a negative relationship with IQ. Given alpha activity recorded at rest defines a putative neural baseline for the attentional or working memory system, rather than a neural baseline for the whole brain, it is perhaps not surprising that no association was found with measures of general cognitive ability, and only weak associations with sub-sets of abilities. Although these psychometric measures capture distinct cognitive skills they represent higher order cognitive skills from a range of domains and will not exclusively involve the attentional/working memory circuits indexed by alpha activity, and therefore the strength of any association between the overlapping components will be diminished amid noise from the non-contributing factors.

In summary, IAF and alpha power are two highly heritable indices of brain functioning, and it is clear, at least in adolescence, that there is a substantive influence from additive genetic factors on these indices. While there is a small inverse relationship between IAF and alpha power, the substantial genetic influences on IAF and alpha power appear to be largely independent of each other. Given the usefulness of alpha activity as a measure of normal and abnormal brain functioning, and the strength of the genetic influence, the next component of this work is to use an available genome-wide scan to search for quantitative trait loci that harbour genes relevant to variation in these indices of brain function.

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