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A quantitative genetic analysis of schizotypal personality traits

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ABSTRACT

Background. Previous twin studies investigating the heritability of schizotypy have often had limited power and have failed to measure the disorganization/social anxiety component.

Method. Seven hundred and thirty-three female twin pairs, drawn from the Institute of Psychiatry Volunteer Twin Register, completed the Oxford–Liverpool Inventory of Feelings and Experiences and the Peters *et al*. Delusions Inventory. Structural equation modelling was carried out on scores for MZ and DZ twin pairs.

Results. The best fitting models for all scales comprised additive genetic and unique environmental effects. Heritability was estimated at approximately 50% for most scales, although it was lower at 37% for the PDI scale. Multivariate structural equation model fitting revealed a best-fitting model in which additive genetic and unique environmental influences act through a single common pathway for Cognitive Disorganization, Unusual Experiences and the PDI, and through a separate common pathway for Cognitive Disorganization and Introvertive Anhedonia.

Conclusions. The various components of schizotypy are moderately heritable. Multivariate model fitting indicates that at least two latent factor structures are required to account for the covariation between the various components of schizotypy. The positive and negative components of schizotypy are relatively genetically independent, although each in turn may be related to Cognitive Disorganization.

INTRODUCTION

Schizotypal personality traits, qualitatively similar to, but quantitatively less severe than, the characteristic symptoms of schizophrenia, may serve as a marker for the predisposition to schizophrenia, while also remaining part of normal individual variation (Kretschmer, 1925; Claridge, 1972, 1987; Gottesman & Shields, 1982). Numerous self-report scales of schizotypy have been developed, including measures that tap general schizotypal traits (e.g. Schizotypal Personality Scale (STA), Claridge & Broks, 1984), and those that tap attenuated psychotic symptoms (e.g. Perceptual Aberration Scale (PerAb), Chapman *et al*. 1978).

Many twin studies of psychosis-proneness have used Eysenck's Psychoticism (P) scale and have either carried out genetic analyses of the entire scale (e.g. Macashill *et al*. 1994), of the individual items (e.g. Neale *et al*. 1986; Heath *et al*. 1989*a*, *b*; Heath & Martin, 1990), or of combinations of items (Kendler *et al*. 1987). These studies have tended to show that individual differences are determined by additive genetic (A) and unique environmental (E) effects, although Heath and colleagues found no evidence for heritable influences on a latent general factor underlying Eysenck's psychoticism items (Heath *et al*. 1989*b*), and an effect of the shared environment for males (Heath *et al*. 1989*a*). Although these studies have the advantage of

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Table 1. *Twin studies of psychosis-proneness*

Study	*N*	Scales	Results	Comments
Kendler *et al.* (1991)	8 MZ and DZ pairs	'Positive trait schizotypy' and 'Trait anhedonia'	Heritability estimated to be >60%	Scales derived from a factor analysis of the STA. Heritability estimates based on twin pair correlations
Claridge & Hewitt (1987)	108 MZ and 102 DZ pairs	STA	AE model best fitting. Heritability estimated at 53%	Biometric model fitting carried out
Kendler & Hewitt (1992)	70 MZ and 63 DZ pairs	Chapman's Social Anhedonia (SocAn), Physical Anhedonia (PhysAn), Non-conformity (NonCon) and Perceptual Aberration (PerAb) scales, STA Paranoid Ideation (STA ParId) and Unusual Experiences (STA UnExp) scales, Eysenck's P scale	AE models best fitting for all scales except STA, UnExp, and PerAb. Heritability estimated at 67%, 57%, 58%, 44%, and 29% for SocAn, PhysAn, NonCon, STA, ParId and P scales, respectively. For STA, UnExp and PerAb, familial resemblance was due solely to common environmental effects	Biometric model fitting carried out. When models were fitted to factors rather than individual scales, no evidence was found for common environmental effects and heritability estimates were between 0·5 and 0·7
Miller (1994)	103 MZ and 73 DZ male twin pairs	Chapman's PhysAn, SocAn, PerAb and Magical Ideation (MagId) scales	MagId and PerAb scales found to be moderately genetically influenced, with heritability estimated at approximately 40%. Familial resemblance attributed solely to common environmental effects for PhysAn and SocAn scales, estimated at 35%	Biometric model fitting carried out
MacDonald *et al.* (2001)	98 MZ and 59 same-sex DZ pairs	Chapman's PerAb, MagId, and SocAn scales and the RISC	Heritability estimated at 32% and 27% for PerAb and SocAn scales, respectively. Familiality due solely to significant common environmental effects for the MagId and RISC scales, estimated at 41% and 30%, respectively	Sample aged 18–27 years. Biometric model fitting carried out
Hay *et al.* (2001)	614 MZ and 720 DZ pairs	Unusual Experiences (Per-Mag; 4 items), Impulsive Non-conformity (Hyp-Imp; 4 items), Social Anhedonia (SAN; 2 items) and Physical Anhedonia (PAN; 2 items)	AE models best fitting for Per-Mag, Hyp-Imp and PAN scales. Heritability estimated at 33%, 28% and 36%, respectively. Heritability estimated at 50% for the SAN scale and attributed solely to dominant genetic effects. No sex specific effects found	Sample aged 18–25 years. Scales derived from a factor analysis of 7 of the Chapman scales (PerAb, MagId, Hypomania, NonCon, SocAn, PhysAn, and Infrequency). Biometric model fitting carried out

STA, Schizotypal Personality Scale (Claridge & Broks, 1984); RISC, Rust Inventory of Schizotypal Cognitions (Rust, 1987).

very large sample sizes, they are severely compromised by the fact that the P scale is not generally considered a very valid measure of psychosis-proneness (Chapman *et al.* 1995, pg. 82).

Table 1 summarizes the twin studies that have been carried out using self-report questionnaires developed specifically for measuring schizotypy or specific components of schizotypy. These studies have tended to report the various components of schizotypy to be influenced by additive genetic effects and to be moderately heritable, although there have been exceptions. For example, Hay *et al.* (2001) found evidence of dominant genetic effects on social anhedonia, and other studies have found common environmental influences on specific positive (Kendler & Hewitt, 1992; MacDonald *et al.* 2001) or negative aspects of schizotypy (Miller, 1994).

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Unfortunately, many of these studies had problems with power due to limited sample sizes, and this has meant that there has been a lack of precision surrounding parameter estimates. For example, although Kendler & Hewitt (1992) reported twin resemblance for the PerAb scale to be due to common environmental effects, a reanalysis by MacDonald *et al.* (2001) showed the lower limit of the 95% confidence intervals to be 0% under the full ACE model, thus indicating that these effects were not significant. Furthermore, MacDonald *et al.* (2001) reported the upper limit of the 95% confidence intervals for the heritability estimate to be 46% under the full model for the PerAb scale, whereas it had been initially suggested that this scale was not heritable. A further limitation of these studies is that they have not included a measure of the cognitive disorganization/social anxiety component of schizotypy that has been identified in many factor analytical studies (e.g. Bentall *et al.* 1989).

Very few studies have carried out multivariate model fitting of schizotypy, and those that exist have also had limited power. For example, MacDonald *et al.* (2001) found that a common schizotypal factor loaded strongly onto the PerAb, Magical Ideation (MagId) (Eckbald & Chapman, 1983), and the Rust Inventory of Schizotypal Cognitions (RISC) (Rust, 1987, 1988, 1989), and less strongly onto the Social Anhedonia (SocAn) (Mishlove & Chapman, 1985) scale. However, while this factor was significantly familial, it was not possible to distinguish between additive genetic and common environmental effects. Similarly, the SocAn scale showed significant specific familial influences, but whether they were due to additive genetic or common environmental effects could not be distinguished.

The present study attempts to further clarify the genetic aetiology of schizotypy by carrying out biometric model fitting on a sample of MZ and DZ twins drawn from the general population. In order to overcome the limitations of previous twin studies of schizotypy, we used a relatively large sample size and well-established questionnaires that cover the main dimensions of schizotypy, including the cognitive disorganization/social anxiety component. A further aim was to explore the possible genetic heterogeneity of the individual components underlying schizotypy by carrying out multivariate model fitting.

METHOD

Measures

We used the Oxford-Liverpool Inventory of Feelings and Experiences (O-LIFE) (Mason *et al.* 1995) and the Peters *et al.* Delusions Inventory (PDI) (Peters *et al.* 1996, 2003). The O-LIFE was derived from factor analytical studies of the Combined Schizotypal Traits Questionnaire (CSTQ) (Bentall *et al.* 1989; Claridge *et al.* 1996), and contains 159 items that require a 'yes' or 'no' response.[1]† It provides scores on four different aspects of psychosis-proneness: Unusual Experiences (UnExp); Cognitive Disorganization (CogDis); Introvertive Anhedonia (IntAn); and Impulsive Non-conformity (ImpNon). Apart from ImpNon, which seems to correspond more closely to the P scale of the EPQ, the other three aspects parallel Liddle's (1987) syndromes of schizophrenia. Mason *et al.* (1995) have reported the internal consistency of the scales to be high (at least 0·7 for the alpha coefficient) and the levels of skewness and kurtosis to be within acceptable limits ($<\pm 1$). The criterion validity of the various scales has been established by findings with patient populations (Mason, 2001, personal correspondence). Further validation of this scale comes from studies showing that individuals with high scores on the positive symptom-like traits perform similarly to psychotic patients on cognitive paradigms (e.g. Steel *et al.* 1996). A longitudinal study has also shown good test–retest reliability for all scales (Burch *et al.* 1998). The O-LIFE also includes full versions of the STA and Eysenck's Extroversion (E) and Social Desirability (SD) scales.

One criticism of the O-LIFE is that items assessing proneness to delusions were taken from Chapman's Magical Ideation scale, which is of a highly skewed nature and has weakened applicability to healthy individuals (Peters *et al.* 1999*a*). Therefore, the 21-item version of the PDI was included in this study as a more adequate measure of delusions. The original 40-item version of the PDI was designed to measure delusional ideation in healthy individuals, using

† The notes will be found on page 814.

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the Present State Examination (Wing *et al*. 1974) as a template (Peters *et al*. 1999*a*). It measures a wide range of delusions and incorporates the multidimensionality of delusions by measuring the distress, preoccupation and conviction with which the belief is held. For each item, the participant scores 1 if the belief is endorsed, and 0 if the belief is not endorsed. If the belief is endorsed, the participant is asked to rate on flanking scales of 1 to 5, the degree of distress, preoccupation and conviction with which the belief is held. The 21-item version of this scale was developed from the 40-item version on the basis of endorsement rates and principal components analysis (Peters *et al*. 1996, 2003). For the 21-item version of the scale, each item was endorsed by approximately 30% of healthy individuals. The scale was reported to have good internal consistency and its construct validity is confirmed by the fact that it correlates significantly with the Delusions Symptoms State Inventory (DSSI) (Foulds & Bedford, 1975). The criterion validity of the scale has been established by the fact that psychotic in-patients have higher scores than healthy controls. Furthermore, individuals with high scores have been shown to exhibit cognitive deficits similar to those shown by clinically deluded groups (Linney *et al*. 1998; Colbert & Peters, 2002).

Participants

Participants were drawn from the Institute of Psychiatry Volunteer Twin Register (IOPVTR), which currently contains about 1600 twin pairs who actively participate in studies. The IOPVTR has been used in a number of studies, many of which have been questionnaire-based (Clifford *et al*. 1984; Rushton *et al*. 1984, 1986; van den Akker *et al*. 1987; Easton *et al*. 1991, 1992; Duffy *et al*. 1992; Thornton & Macdonald, 1999). Zygosity has been assessed using a series of questions that have been found to discriminate MZ from DZ twins with over 95% reliability (Cederlöf *et al*. 1961; Sarna & Kaprio, 1980).

Questionnaires were sent to 3153 twins who were still participating in studies.[2] Pre-paid addressed return envelopes were sent with the questionnaires and the twins were not paid for their participation. Of the total number of questionnaires sent, completed questionnaires were returned by both members of 928 twin pairs, giving a pairwise response rate of 59%. Three hundred and thirty questionnaires were also received from one twin only, giving an individual response rate of 69%. Questionnaires from 15 twins were excluded from the current dataset as they were returned too late (5 in pairs and 10 singles). None of the twins reported having had a psychotic breakdown and all twins were included since other psychiatric problems were not considered relevant. The remaining twin pairs were subdivided into five groups: monzygotic male pairs (MZM), monozygotic female pairs (MZF), dizygotic male pairs (DZM), dizygotic female pairs (DZF) and opposite sex pairs (DZO). Of the pairs where both twins responded, 70 were MZM, 468 were MZF, 29 were DZM, 265 were DZF and 64 were DZO. There were also 40 pairs whose zygosity was either unknown or unclear so these twins were excluded from the current analyses. The age range of the total sample was 17 to 84 years (mean age = 47 years). The female bias reflects the gender bias of the IOPVTR, rather than a differential response bias for males and females; however, since questionnaires were received from so few male twin pairs, the current analyses were restricted to females.

Statistical analyses

Since the total amount of missing data was very small and errors appeared to be random, missing values were imputed. The imputation method used the maximum likelihood prediction from the best available subset of non-missing items from the same scale, thus taking account of individuals' answers on other questions within the same scale as well as the overall endorsement rate of the item for which data is missing. All imputation of missing data was carried out using the statistics package, Stata Release 6.0 (Stata Corp, 1999). If individuals missed out more than 20% of the items for the O-LIFE or the PDI, or missed out an entire page of questions, missing data remained and they were excluded from the analyses of scales to which these items contributed. The total proportion of items imputed for any one scale was $<1\%$.

Quantitative genetic model fitting was carried out on the variance-covariance matrices for MZ and DZ twin pairs using the statistical modelling package, Mx (Neale, 1997). Mx gives the statistical fit of each model in terms of the

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likelihood-ratio chi-square statistic (χ^2) of the model relative to a saturated model. A non-significant χ^2 value means that the model provides a good fit to the data, whereas a significant χ^2 value means that the model does not fit the data adequately. The difference between models can be tested by assessing the change in χ^2 against the change in degrees of freedom (df), provided that one model is nested within the other. In doing this, the significance of the individual parameters is tested. For the following analyses, the 0·05 level of significance was adopted.

For the univariate model fitting, since the MZ correlations were approximately twice the DZ correlations for each of the scales, a full ACE model, in which familial resemblance is due to additive genetic (A) and common environmental effects (C), was fitted to the data. This model also includes variance due to unique environmental effects (E), which accounts for differences between MZ twins and includes measurement error. The ImpNon scale was excluded from the model fitting since it tends to relate more to psychopathy rather than to schizotypy, *per se* (Sharpley & Peters, 1999). Since age correlates significantly with the O-LIFE and PDI scales (Mason *et al.* 1995; Peters *et al.* 2003) and the age range of the twin sample is broad, the model fitting included age (S) as a covariate. Each of the components of this model was then omitted in turn in order to test their significance. Best fitting models were assessed according to Akaike's Information Criteria (AIC) (Akaike, 1987), which takes account of both the goodness of fit and the parsimony of the model (with lower values reflecting a better and more parsimonious fit). Maximum likelihood parameter estimates for the various genetic and environmental effects are reported, for the full and best fitting models, for each of the scales.

In order to investigate why the various dimensions of schizotypy are correlated, multivariate analyses were carried out using Mx. In multivariate genetic analyses, the phenotypic covariances between the scales are divided into genetic, common and unique environmental components. The STA was excluded from the multivariate model fitting since some of the other scales include items from the STA, thus resulting in multicollinearity.

A Cholesky (or triangular) decomposition was fitted initially as it enabled us to inspect the genetic and environmental correlation matrix and to make specific hypotheses of how subsets of variables cluster (factor inspection), which then guided the subsequent model fitting. A Cholesky factorisation decomposes the genetic and environmental covariance matrices into triangular matrices of factor loadings, where the number of factors equals the number of variables. Thus, according to this model, the first factor influences all four scales, the second factor influences all variables except the first, the third factor influences only the third and fourth variables, and the last factor influences only the last variable. Following this, independent and common pathway models were fitted to the data. The independent pathway model specifies that the genetic and environmental factors that are common to the variables have their own (independent) pathways to each of the phenotypic variables; however it differs from the Cholesky in that there are not as many genetic factors as there are variables, and the results are not dependent on the ordering of the variables. The common pathway model is more stringent than both the Cholesky and the independent pathway models, since it specifies that the covariation between the scales is determined by a single phenotypic latent factor, which is itself determined by additive genetic, common environmental and/or unique environmental components. Both the independent and common pathway models specify that each phenotypic variable may also be influenced by specific environmental or genetic factors that are not shared with the other variables.

RESULTS

Assumptions of the twin study

First, we examined a number of the assumptions underlying the classical twin method in relation to the data. Equality of environments between MZ and DZ twin pairs was tested by looking at the relationship between frequency of current contact between twin pairs and similarity between twin pairs in terms of the schizotypy scales, for 217 twin pairs. In agreement with previous studies, we found that MZ female twin pairs had significantly more contact than DZ female twins pairs (Rose & Kaprio, 1988; Treloar, 1992). However, since degree of contact was not correlated with twin pair similarity on

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the scales, the validity of the equal environments assumption was supported. The mean scale scores for our twins were also compared to the norms previously reported for the O-LIFE and PDI scales (Mason *et al*. 1995; Peters *et al*. 1996, 2003; Verdoux *et al*. 1998). Since any differences were minimal (within one standard deviation), it was presumed that our sample of twins does not differ from singletons in terms of schizotypy. Furthermore, there were no significant differences between MZ and DZ twins for the means or variances of any of the scales. Scale transformation was carried out in order to provide a closer approximation to the normal distribution. This also reduced correlations between absolute pair difference and pair sum in MZ twins (i.e. heteroscedasticity), which is said to be indicative of gene–environment interaction (Jinks & Fulker, 1970). Thus, the assumption of independent action of genetic and environmental effects on schizotypy was supported. Lastly, comparisons between twins whose co-twin did and did not return a questionnaire revealed no significant differences for any of the scales for MZ or DZ twin pairs. Thus, it would seem that there was no relationship between levels of schizotypy and whether or not a questionnaire was returned.

Univariate genetic analyses

Table 2 shows the correlations between the various O-LIFE and PDI scales. This shows that there are sizeable correlations between the CogDis, UnExp, and PDI scales. The IntAn scale, on the other hand, correlates moderately with the CogDis scales but does not correlate with any of the other scales.

Table 3 shows intraclass correlations for MZ and DZ twin pairs for each of the schizotypy scales, and the fit functions for the various models that were tested. The results of the model fitting revealed that models with familial resemblance resulting purely from common environment effects (CES models) could be rejected for all aspects of psychosis-proneness. Furthermore, since there was no significant deterioration in fit for any of the scales when the common environmental effect was omitted from the full model, it is unlikely that there are very large common environmental effects on schizotypy.[3] In contrast, the fact that there was a significant deterioration in fit when the additive genetic component was omitted from the full model indicates that there are significant additive genetic effects on schizotypy. This is the case for all aspects of schizotypy except delusion-proneness, for which it was not possible to distinguish between additive genetic and common environmental components. This is evident since the lower limits of the 95% confidence intervals were 0% for both the additive genetic and

Table 2. *Correlations between the various O-LIFE and PDI scales*

	CogDis	UnExp	IntAn	STA
CogDis				
UnExp	0·502**			
IntAn	0·324**	0·034		
STA	0·721**	0·878**	0·174**	
PDIQ	0·486**	0·691**	0·033	0·699**

** $P<0·01$.

Table 3. *Intraclass correlations for MZ and DZ twin pairs and fit functions for univariate models fitted to O-LIFE and PDI scales*

	Correlations			Fit functions			
Scale	MZ	DZ	Model	χ^2	df	P	AIC
UnExp	0·538**	0·278*	ACES	8·31	6	0·22	−3·69
			AES	8·31	7	0·31	−5·69
			CES	27·40	7	0·00	13·40
			ES	173·25	8	0·00	157·25
			AE	47·76	8	0·00	31·76
CogDis	0·549**	0·283*	ACES	2·49	6	0·87	−9·51
			AES	2·49	7	0·93	−11·51
			CES	23·50	7	0·00	9·51
			ES	171·49	8	0·00	155·5
			AE	46·66	8	0·00	30·66
IntAn	0·533**	0·229**	ACES	10·88	6	0·09	−1·12
			AES	10·88	7	0·14	−3·12
			CES	36·30	7	0·00	22·30
			ES	163·97	8	0·00	147·97
			AE	41·06	8	0·00	25·06
STA	0·588**	0·346**	ACES	3·62	6	0·73	−8·38
			AES	3·71	7	0·81	−10·29
			CES	26·90	7	0·00	12·90
			ES	211·22	8	0·00	195·22
			AE	53·60	8	0·00	37·60
PDI questions	0·432**	0·317**	ACES	11·14	6	0·08	−0·86
			AES	12·45	7	0·09	−1·55
			CES	14·25	7	0·05	2·50
			ES	103·04	8	0·00	87·04
			AE	91·86	8	0·00	75·86

A, additive genetic component; C, common environmental component; E, unique environmental component; S, age component.
* $P<0·05$; ** $P<0·01$.

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Table 4. *Parameter estimates for best fitting AES and full ACES models for each of the schizotypy scales (95% confidence intervals in parentheses)*

	ACE model				Best fit model		
Scale	a	c	e	s	a	e	s
UnExp	0·49 (0·28, 0·55)	0·00 (0·00, 0·20)	0·47 (0·41, 0·54)	0·04 (0·02, 0·06)	0·49 (0·43, 0·55)	0·47 (0·41, 0·53)	0·04 (0·02, 0·06)
CogDis	0·51 (0·29, 0·56)	0·00 (0·00, 0·19)	0·45 (0·39, 0·51)	0·04 (0·02, 0·07)	0·51 (0·44, 0·56)	0·45 (0·39, 0·51)	0·04 (0·02, 0·07)
IntAn	0·50 (0·34, 0·56)	0·00 (0·00, 0·13)	0·47 (0·41, 0·54)	0·03 (0·02, 0·05)	0·50 (0·43, 0·56)	0·47 (0·42, 0·54)	0·03 (0·01, 0·05)
STA	0·51 (0·29, 0·60)	0·03 (0·00, 0·23)	0·41 (0·35, 0·47)	0·05 (0·03, 0·08)	0·54 (0·49, 0·60)	0·41 (0·36, 0·46)	0·05 (0·03, 0·08)
PDI	0·22 (0·00, 0·42)	0·14 (0·00, 0·35)	0·57 (0·50, 0·64)	0·07 (0·05, 0·11)	0·37 (0·30, 0·43)	0·56 (0·49, 0·63)	0·07 (0·05, 0·11)

a, Variance due to additive genetic effects; c, variance due to common environmental effects; e, variance due to unique environmental effects; s, variance due to age effects.

unique environmental components, for the PDI. Nevertheless, for all aspects of schizotypy, including delusion proneness, the best fitting model on the basis of the AIC value was the AES model, comprising additive genetic and unique environmental components. The results also show that there was a significant deterioration in the fit of the model when the effects of age were omitted, indicating that these effects are highly significant.

Table 4 shows the parameter estimates and 95% confidence intervals for the best fitting models and for the full ACE models, for each of the scales. Under the best-fitting AES models, heritability estimates were highest for the CogDis, UnExp, IntAn and STA scales, and ranged narrowly from 49% to 54%. For the PDI scale, the heritability estimate was lower at 37%. The confidence intervals for all of these estimates indicate that they are fairly stable and only vary by ± 7 at the most.

Under the full ACES models, the heritability estimates were identical for the IntAn, UnExp and CogDis scales and the estimates of the common environmental effects were zero. However, the upper limits of the 95% confidence intervals indicate that there could be small common environmental effects of not more than 20%. For the STA scale, the common environment was estimated to have a very small effect; the upper limit of the 95% confidence interval indicates that this could be as high as approximately 23%. For the PDI, the effect of the common environment under the full model was estimated to be greater (14%) and the upper limit of the 95% confidence interval indicates that it could be as high as 35%.

Multivariate genetic analyses

Table 5 shows the cross correlations for the remaining four scales, for MZ and DZ twin pairs, with the pairwise twin correlations for the individual scales shown in bold on the diagonal. Significant individual cross scale correlations indicate shared or common aetiological influences between the various components of schizotypy, and whether this is due to shared additive genetic, shared common environmental or shared unique environmental influences is indicated by the ratio of MZ/DZ cross-scale within-pair correlations. For example, significant individual cross scale correlations, together with greater within-pair cross scale correlations for MZ twin pairs compared to DZ twin pairs indicate shared additive genetic influences. On the other hand, significant individual cross scale correlations, together with similar within-pair cross scale correlations for MZ and DZ twin pairs indicate shared common environmental influences. Table 5 shows that there are sizeable individual cross scale correlations for the CogDis, UnExp, and PDI scales, whereas the IntAn scale correlates moderately with the CogDis scale but does not correlate with the PDI and UnExp scales. The ratio of MZ to DZ within-pair cross scale correlations, for scales which are significantly correlated, indicates that there are likely to be shared additive genetic influences.

The results of the multivariate model fitting can be seen in Table 6. A full Cholesky ACES

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Table 5. *Pearson's intraclass cross twin correlations for the Cognitive Disorganization, Unusual Experiences, PDI questions and Introvertive Anhedonia scales*

		MZ twins							
		Twin 1				Twin 2			
		CogDis	UnExp	PDI	IntAn	CogDis	UnExp	PDI	IntAn
Twin 1	CogDis		0·487	0·439	0·276	**0·550**	0·268	0·290	0·240
	UnExp	0·600		0·655	−0·013	0·327	**0·538**	0·428	0·089
	PDI	0·401	0·611		−0·004	0·326	0·395	**0·434**	0·065
	IntAn	0·343	0·109	0·001		0·134	−0·018	−0·050	**0·534**
Twin 2	CogDis	**0·289**	0·250	0·183	0·151		0·495	0·485	0·282
	UnExp	0·192	**0·283**	0·224	0·022	0·462		0·670	0·029
	PDI	0·190	0·249	**0·320**	0·110	0·416	0·707		0·030
	IntAn	0·058	0·049	−0·054	**0·231**	0·361	−0·124	−0·088	
		CogDis	UnExp	PDI	IntAn	CogDis	UnExp	PDI	IntAn
		Twin 1				Twin 2			
		DZ twins							

MZ correlations are in the upper triangle and DZ correlations are in the lower triangle.
The bold type represents the cross-twin correlations for the same scale.

Table 6. *Results of multivariate model fitting (with age as a covariate) carried out on the PDI, Unusual Experiences, Cognitive Disorganization and Introvertive Anhedonia scales (best model in bold type)*

	Fit functions			
Model	χ^2	df	*P*	AIC
1. ACE Cholesky model with age as a covariate	65·808	55	0·151	−44·192
2. AE Cholesky model with age as a covariate	69·491	65	0·329	−60·491
2. AE common pathway model with age as a covariate	276·963	72	0·000	132·96
3. AE independent pathway model with age as a covariate	204·476	69	0·000	66·476
4. AE two-factor common pathway model with age as a covariate	78·919	73	0·297	−67·081
5. Nested AE two-factor common pathway model with age as a covariate	**78·023**	**74**	**0·352**	**−69·977**
6. Nested AE two-factor independent pathway with age as a covariate	78·834	71	0·245	−63·166

model, comprising shared additive genetic, common environmental and unique environmental components provided an adequate fit to the data. However, the shared common environmental component could be omitted from the model without a significant deterioration in fit, and this was preferred on the basis of the AIC value. Dropping either the shared genetic or shared unique environmental path coefficients, or any of the specific genetic or unique environmental path coefficients led to a significant deterioration in the fit of the model.

In order to explore further the genetic architecture of schizotypal traits, independent and common pathway models were fitted to the variance-covariance matrices. The common environmental components were omitted since they were shown not to be important in the univariate or the multivariate Cholesky analyses. Initially, AES independent and common pathway models were fitted, whereby each type of model included both shared and specific additive genetic and unique environmental components, and age as a covariate. However, both

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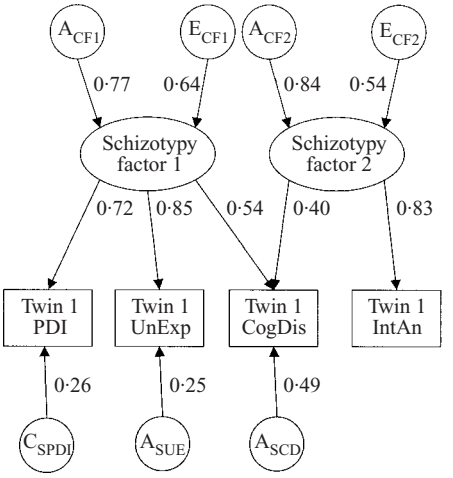


FIG. 1. Standardized path coefficients for best-fitting multivariate AES common pathway model for Twin 1 for the PDI, Unusual Experiences, Cognitive Disorganization and Introvertive Anhedonia scales. (A_{CF1}, additive genetic effect on schizotypy factor 1; E_{CF1}, unique environmental effect on schizotypy factor 1; A_{CF2}, additive genetic effect on schizotypy factor 2; E_{CF2}, unique environmental effect on schizotypy factor 2; A_{SUE}, specific additive genetic component for the Unusual Experiences scale; A_{SCD}, specific additive genetic component for the Cognitive Disorganization scale; C_{SPDI}, specific common environmental component for the PDI scale.) Age and specific unique environmental components are also included in the best-fitting model, although they have been omitted from this diagram.

Table 7. *Genetic (below diagonal) and unique environmental (above diagonal) correlations (95% confidence intervals in parentheses) for the Cognitive Disorganization, Unusual Experiences, PDI and Introvertive Anhedonia scales as estimated from the AES Cholesky and two-factor common pathway models (parameters fixed to zero in parentheses)*

	PDI	UnExp	CogDis	IntAn
AES Cholesky model				
PDI		0·49 (0·43, 0·56)	0·29 (0·21, 0·37)	0·01 (−0·09, 0·08)
UnExp	0·84 (0·77, 0·90)		0·42 (0·35, 0·49)	−0·07 (−0·16, 0·01)
CogDis	0·55 (0·44, 0·66)	0·54 (0·45, 0·63)		0·21 (0·13, 0·30)
IntAn	0·09 (−0·05, 0·23)	0·13 (0·01, 0·25)	0·49 (0·38, 0·58)	
AES two-factor common pathway model				
PDI		0·49 (0·44, 0·54)	0·32 (0·28, 0·37)	(0)
UnExp	0·93 (0·87, 0·99)		0·41 (0·36, 0·46)	(0)
CogDis	0·57 (0·51, 0·63)	0·53 (0·46, 0·59)		0·21 (0·16, 0·27)
IntAn	(0)	(0)	0·46 (0·40, 0·53)	

of these models were incompatible with the data. In order to account for the pattern of inter-scale correlations shown in Table 4, two-factor common and independent pathway models were fitted to the data. The common pathway model had two latent factors, each influenced by additive genetic and unique environmental components, whereby the first latent factor had pathways to the PDI, UnExp and CogDis scales and the second latent factor had pathways to the CogDis and IntAn scales. As with the one-factor common and independent pathway models, this model also included both specific additive genetic and unique environmental components, and age as a covariate.[4] This model provided a good fit to the data, and the importance of the parameters were tested by fitting various nested common pathway models. The best-fitting model was a two-factor AES common pathway model, containing specific unique environmental components for all psychosis-proneness scales, as well as specific additive genetic components for the UnExp and CogDis scales and a specific common environmental component for the PDI scale. To provide a comparison with the two-factor common pathway model, we also fitted a two-factor independent pathway model. However, since the full two-factor independent pathway model was not identified, the equivalent version of the best-fitting two-factor common pathway model was fitted, which allows a direct comparison between the two models. This model has two shared additive genetic and unique environmental components (with the first component having pathways to the PDI, UnExp and CogDis scales and the second component having pathways to the CogDis and IntAn scales), as well as specific unique environmental components for all psychosis-proneness scales, specific additive genetic components for the UnExp and CogDis scales, and a specific common environmental component for the PDI scale. This model provided a good fit to the data, although the equivalent two-factor common pathway model was more parsimonious. Fig. 1 shows the best fitting two-factor common pathway model.

Table 7 shows that, under this model, the CogDis scale has genetic correlations of 53%, 57% and 46% with the UnExp, PDI and IntAn

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Table 8. *Standardized variance components (with 95% confidence intervals) for Cognitive Disorganization, Unusual Experiences, PDI and Introvertive Anhedonia scales as estimated from the AES two-factor common pathway model (parameters fixed to zero in parentheses)*

Scale	Genetic factors			Environmental factors				Age
	Ac	As	h^2	Ec	Es	e^2	c^2	g^2
PDI	0·30 (0·25, 0·35)	(0)	0·30 (0·25, 0·35)	0·21 (0·17, 0·26)	0·34 (0·31, 0·38)	0·55 (0·50, 0·61)	0·07 (0·03, 0·11)	0·07 (0·05, 0·11)
UnExp	0·42 (0·35, 0·49)	0·06 (0·01, 0·12)	0·48 (0·43, 0·54)	0·30 (0·25, 0·35)	0·18 (0·16, 0·20)	0·48 (0·43, 0·55)	(0)	0·04 (0·02, 0·06)
CogDis	0·28 (0·24, 0·32)	0·24 (0·20, 0·29)	0·52 (0·48, 0·56)	0·17 (0·14, 0·20)	0·27 (0·24, 0·30)	0·44 (0·40, 0·47)	(0)	0·04 (0·02, 0·07)
IntAn	0·49 (0·42, 0·55)	(0)	0·49 (0·42, 0·55)	0·20 (0·15, 0·27)	0·28 (0·25, 0·31)	0·48 (0·43, 0·55)	(0)	0·03 (0·01, 0·05)

Ac, common additive genetic effect; As, specific additive genetic effect; h^2, total variance due to additive genetic effects (heritabilty); Ec, common unique environmental effect; Es, specific unique environmental effect; e^2, total variance due to unique environmental effects; c^2, variance due to common environmental effects (specific common environment only); g^2, variance due to age effects.

scales, respectively. The PDI and UnExp scales have a genetic correlation of 93%, and neither of these scales is genetically correlated with the IntAn scale. Table 8 shows that the heritability estimates obtained under this model are almost identical to those obtained under the best-fitting univariate models. The exception to this is the heritability estimate for the PDI, which is slightly lower in the best fitting multivariate model, as a result of the specific common environmental component.

DISCUSSION

The results of the univariate genetic analyses revealed a best-fitting model comprising additive genetic and unique environmental components, for each of the schizotypy scales. These findings are consistent with previous twin studies of schizotypy (Claridge & Hewitt, 1987; Kendler *et al*. 1991; Kendler & Hewitt, 1992; Miller, 1994; Hay *et al*. 2001; MacDonald *et al*. 2001).

The heritability estimates obtained under the best-fitting age-corrected models in this study were approximately 50% for the UnExp, IntAn, CogDis, and STA subscales. For PDI scale, heritability was lower at 37%. The 95% confidence intervals for these estimates indicate that they are quite stable and only vary by $\pm 7\%$ at most. Moreover, the heritability estimates were identical under the full ACES model for the CogDis, UnExp and IntAn scales and only slightly lower for the STA scale.

Although there is some variation between the heritability estimates obtained in different studies, there is considerable overlap between the 95% confidence intervals of the various heritability estimates for different scales (c.f. Claridge & Hewitt, 1987; Kendler *et al*. 1991; Kendler & Hewitt, 1992; Miller, 1994; MacDonald *et al*. 2001; Hay *et al*. 2001). In addition, some degree of variability is to be expected since there are many methodological differences between the studies in terms of schizotypy scales, samples and analyses. Thus, overall, it can be concluded that most schizotypal traits, as measured by self-report questionnaire, have a substantial heritable component in the general population.

The fact that schizotypy appears to be influenced solely by additive genetic and unique environmental effects is consistent with twin and adoption studies of schizophrenia, which have reported similar findings (Cardno *et al*. 1999). This suggests that schizotypy and schizophrenia are influenced by the same broad mechanisms, and provides further support for a continuity model of psychosis. These findings are consistent with a multi-factorial polygenic model of schizophrenia, in which it is postulated that the liability to develop schizophrenia runs on a continuum throughout the general population, and is influenced by both genetic and environmental components, acting in an additive manner (Falconer, 1965; Gottesman & Shields, 1967).

The results of the multivariate model fitting indicate that, although the different components of schizotypy are genetically related, there is not one underlying core dimension or typology that can account for the covariation between them.

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Rather, at least two latent factor structures are required to account for the covariation between the various components, with each influenced by additive genetic and unique environmental effects. In addition, each schizotypal component is influenced by specific unique environmental effects, UnExp and CogDis are influenced by specific additive genetic effects, and delusional ideation is influenced by specific common environmental effects. The results of this model fitting, therefore, suggest that, although the various schizotypal traits have related aetiologies, they are also influenced by somewhat different genetic and environmental influences. This provides further validation for the multidimensional nature of schizotypy. Indeed, the view that the various components of schizotypy may have different underlying aetiological mechanisms is not a new one, and is becoming increasingly substantiated empirically (Reynolds *et al*. 2000).

While the various schizotypal traits are influenced by independent processes to some degree, our findings also show that both positive and negative schizoptypal traits are genetically related to CogDis. One possible interpretation of this is that cognitive disorganization reflects the core genetic liability to schizotypy. Indeed, this is consistent with Meehl's (1990) idea that 'associative loosening' and 'aversive drift' characterize the fundamental deficit in schizophrenia, since these seem equivalent to the disorganization/social anxiety traits that are measured by the CogDis scale (Claridge, 1994). It is also consistent with Bleuler's (1911) view that the underlying disturbance in schizophrenia is a 'breakening of associative threads' in both words and thoughts, which subsequently leads to attentional difficulties.

One interesting question regarding this finding, since the CogDis scale is made up of items relating to both social anxiety and disorganization, is whether different aspects of CogDis are differentially related to positive and negative schizotypal traits. Indeed, one possibility is that social anxiety is genetically associated with negative schizotypal traits, while disorganization is genetically related to positive schizotypal traits. Similarly, it could also be the case that either social anxiety or disorganization reflect the core genetic liability to schizotypy, rather than both together. It has been suggested that extreme social anxiety may be the best marker of a genetic link between schizophrenia and schizotypal personality disorder (Torgersen *et al*. 1993). However, it has also been shown that the disorganization syndrome has the highest familial loading for schizophrenia-related illness and has a significant independent association with familial liability to all psychoses combined (Cardno *et al*. 1997, 2001). In support of this, the disorganization syndrome has also been shown to be associated with a history of multiple past admission and longer lifetime duration of in-patient treatment (Mellers *et al*. 1996).

Also of interest in the multivariate model fitting is the fact that there were significant, albeit small, specific common environmental influences on delusional ideation. Possible cultural influences on PDI scores include socio-economic class and religion since it has been found that delusional ideation is higher among the working classes in Afro-Caribbean populations in the UK, as well as among certain new religious movements, such as Druidism (Peters *et al*. 1999*b*; Sharpley & Peters, 1999). While not focusing on delusional proneness *per se*, a number of studies have reported an association between religious affiliation and belief in the paranormal (Williams *et al*. 1989; Thalbourne *et al*. 1995; Haraldsson & Houtkooper, 1996; Kristensen, 1999; Thalbourne & O'Brien, 1999). Another possible cultural influence is childhood trauma, since it has been shown that a number of traumatic early life events are associated with belief in the paranormal in adulthood (Irwin, 1992, 1994; Lawrence *et al*. 1995).

Our findings must be interpreted in light of the limitations imposed by the sample and the methods we used. One of the main concerns is that the sample was drawn from a volunteer register which may not necessarily be representative of the wider population. While this sample was comparable to the general population in terms of schizotypy scores, it cannot be demonstrated that it is representative of the wider population of twins. In particular, it is worth bearing in mind that the twin pairs included in this study may be more similar than other twin pairs, which may mean that the familiality of schizotypy is overestimated. Furthermore, this problem may be exacerbated by the fact that only pairs where both twins responded were included in the genetic analyses. Another limitation

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of this study is that only one very modest potential index of the equal environments assumption was examined. Thus, although this index was consistent with the equal environment assumptions, it may still be possible that this assumption has been violated, which would mean that the heritability estimates are inflated. Another limitation is that the assessment of negative symptom-like traits was limited to anhedonia. Since anhedonia is not a central negative symptom in schizophrenia, it may be that comparing negative schizotypal traits to negative symptoms in schizophrenia is comparing traits that are not only quantitatively, but also qualitatively, different. Nevertheless, anhedonia has been the central focus of negative schizotypy in previous factor analytical and genetic studies of schizotypy, and its inclusion enables comparisons between studies to be made. The models presented in this study are also oversimplified due to the statistical restrictions imposed on structural equation modelling with observations from only MZ and DZ twin pairs reared together. In order to test more complicated models, it would be necessary to obtain data from twins reared apart or other family members.

NOTES

[1] Although the original version of the questionnaire requires a true–false response on a separate answer sheet, a yes–no response was used in our questionnaire in order to make it more suitable for a postal survey.

[2] This included some single twins since we were attempting to get information for co-twins who had changed address.

[3] Power calculations indicate that, with the sample size used in this study, common environmental effects of approximately 30% would have been detected at the 0·05 level of significance, with 80% power.

[4] The specific unique environmental components were equated in order to make the model identified.

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