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The genetic and environmental relationship between the interpersonal sensitivity measure (IPSM) and the personality dimensions of Eysenck and Cloninger

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Abstract

A shortened version of the Interpersonal Sensitivity Measure (IPSM) developed to predict depression prone personalities was administered in a self-report questionnaire to a community-based sample of 3269 Australian twin pairs aged 18–28 years, along with Eysenck's EPQ and Cloninger's TPQ. The IPSM included four sub-scales: Separation Anxiety (SEP); Interpersonal Sensitivity (INT); Fragile Inner-Self (FIS); and Timidity (TIM). Univariate analysis revealed that individual differences in the IPSM sub-scale scores were best explained by additive genetic and specific environmental effects. Confirming previous research findings, familial aggregation for the EPQ and TPQ personality dimensions was entirely due to additive genetic effects. In the multivariate case, a model comprising additive genetic and specific environmental effects best explained the covariation between the latent factors for male and female twin pairs alike. The EPQ and TPQ dimensions accounted for moderate to large proportions of the genetic variance (40–76%) in the IPSM sub-scales, while most of the non-shared environment variance was unique to the IPSM sub-scales.

Keywords: Depression; Personality; Genes; Twins

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1. Introduction

Boyce and Parker believe that there is likely no *sui generis* personality type associated with depression, but rather a number of personality traits are likely to dispose individuals to depression (Boyce & Parker, 1989). Based on theoretical speculation and clinical observations they designed the Interpersonal Sensitivity Measure (IPSM) in order to identify "at risk" depression prone personalities (Boyce & Parker).

Initially, a 73-item self-report instrument scored on a four-point Likert scale was completed by 265 general practice attendees (Boyce & Parker, 1989). Principal components analysis reduced the number of items to 36, from which were extracted five interpretable oblique factors: Interpersonal Awareness; Need for Approval; Separation Anxiety; Timidity; and Fragile Inner-Self (Boyce & Parker). Interpersonal Awareness (INT) refers to the way in which individuals appraise and assign meaning to situations. High INT scorers worry about their impact on others, are vigilant, and apprehensive in social settings. The Need for Approval (NA) sub-scale measures the extent to which individuals subordinate personal needs in order to keep others happy so as to avoid rejection and scorn. Separation Anxiety (SEP) measures the level of anxiety that an individual experiences when separated from a significant other. Timidity (TIM) measures a general lack of assertiveness. Finally, Fragile Inner-Self (FIS) assesses an individual's fear of being rejected or ridiculed. An individual's total IPSM scores, based on the five sub-scales, measures the global construct of 'interpersonal sensitivity' (Boyce and Parker, 1989). Inter-correlations between the total IPSM and sub-scales are high and range from 0.26 to 0.47 except for the correlation between FIS and NA ($r = 0.08$; Boyce & Parker) which suggests that the factors are not entirely independent. The total IPSM has satisfactory internal consistency (Cronbach's $\alpha = 0.70$) but shows some sensitivity to depressed mood in samples of depressives assessed during and after depressive episodes (Boyce & Parker, 1989).

The correlation between total IPSM and the Eysenck Personality Inventory (EPI) (Eysenck & Eysenck, 1964) Neuroticism scale in samples of clinically depressed subjects is high (0.61, $P < 0.001$) which suggests that the IPSM and the Neuroticism scale are measuring overlapping constructs. However, Boyce and Parker maintain that neuroticism predicts a global vulnerability towards developing neurotic illnesses rather than depression or depressive sub-types per se (Boyce & Parker, 1989). Several studies have found a significant association between the IPSM and depression (Boyce & Parker, 1989; Boyce, Hickie, Parker, Mitchell, Wilhelm & Brodaty, 1992; Boyce, Hickie, Wilhelm, Brodaty & Mitchell, 1990; Sakado, Sato, Uehara, Sakado, Kuwabara & Someya, 1999). High IPSM scores also predict liability to post-natal depression (Boyce, Hickie & Parker, 1991a; Boyce et al., 1991b) even after antenatal depressives were excluded from the analyses (Boyce et al., 1991b). In terms of depressive subtypes, DSM-III non-melancholics compared to melancholics have higher scores on the total IPSM and sub-scales except for NA and TIM (Boyce et al., 1990).

Although evidence for the genetic contribution to adult personality is compelling (Eaves, Eysenck & Martin, 1989; Loehlin, 1992) most studies on the genetics of personality have employed a core of Extraversion and Neuroticism items from the Eysenckian or equivalent scales. No study to date has examined the heritability of the IPSM sub-scales. Nor has there been a comparison, beyond phenotypic factor analysis (see Boyce et al., 1990), of the IPSM's genetic and environmental covariance with other empirically generated models of personality.

Therefore, our first objective is to estimate the proportion of variance attributable to latent genetic and environmental factors underlying the IPSM and the sub-scales using univariate biometrical analyses. We will then use multivariate analyses to decompose the sources of covariation between the IPSM sub-scales and the dimensions of personality from the Eysenck's Personality Questionnaire (EPQ) (Eysenck, Eysenck & Barrett, 1985), and Cloninger's Tridimensional Personality Questionnaire (TPQ) (Cloninger, Przybeck & Svrakic, 1991, Heath, Cloninger & Martin, 1994) into latent genetic and environmental factors. This will enable us to determine how much of the variance in the IPSM sub-scales can be predicted by the EPQ and TPQ, while at the same time allow us to estimate the proportion of variance which is unique to the IPSM.

2. Method

2.1. Sample

In 1989, an extensive Health and Lifestyle Questionnaire (HLQ) was mailed to 4269 twin pairs born 1964–1971. The HLQ covered a wide range of health issues affecting younger people, including a 12-item self-report version of the IPSM; Boyce & Parker, 1989), as well as shortened versions of the Revised Eysenck Personality Questionnaire (EPQ-R-S) (Eysenck et al., 1985), and TPQ (Cloninger et al., 1991). Most of these twins had been recruited when at school some 10 years earlier, so despite extensive follow-up efforts, we were unable to reestablish contact with exactly 1000 pairs. Those twins who failed to return a completed questionnaire were followed-up by telephone up to five times, at which point they were asked to complete an abbreviated telephone interview to obtain basic demographic information only. Returned questionnaires where all IPSM items were completed were received from 3433 subjects (1355 males and 2078 females) representing an individual response rate of 53% (3433/6538). Returned questionnaires where all EPQ items were completed were received from 2943 subjects (1146 males and 1797 females) representing an individual response rate of 45% (2943/6538). Returned questionnaires where all TPQ items were completed were received from 2871 subjects (1116 males and 1755 females) representing an individual response rate of 44% (2871/6538). The mean age of respondents was 23.2 ± 2.2 years with an age range of 18–28 years.

2.2. Zygosity diagnosis

Zygosity was determined based on twins' responses to standard questions about similarity and the degree to which others confused them. Such procedures have previously demonstrated at least 95% agreement with diagnoses based on extensive blood sampling (Martin, 1975; Ooki, Yamada, Asaka & Hayakawa, 1990). Pairs giving inconsistent responses were further interviewed by telephone for clarification whereupon their zygosity was determined in 80% of cases. If zygosity was still equivocal then twins were asked to send in photographs at several stages of their lives and most were readily assigned with little hesitation by the project staff. Where possible, blood samples were taken from the few remaining uncertain pairs.

2.3. Measures

A shortened 12-item version of the 36-item self-report IPSM (Boyce & Parker, 1989) was included in the HLQ. Our justification for using the shortened version was based on previous findings that showed correlations between the short and full length IPSM of 0.95, 0.90, 0.64, 0.82 and 0.92 for INT, SEP, TIM, FIS, and total IPSM, respectively (Todd, Boyce, Heath & Martin, 1994). Our shortened version contained four of the original five sub-scales: INT (4 items); SEP (4 items); FIS (2 items); and TIM (2 items). In addition, a cumulative measure (full IPSM) was calculated based on all 12 items. The IPSM was originally measured on a four-point scale from "Very unlike me" (1) to "Very like me" (4) (Boyce & Parker, 1989). However, all items in the short version were scored on a three-point scale [Yes/Don't know/No] with "Don't know" responses recorded as missing.

A shortened 54-item version of the TPQ (Cloninger et al., 1991) and 48-item version of the EPQ-R-S (Eysenck et al., 1985) were also included in the questionnaire. The TPQ was designed to assess three higher order personality dimensions as defined by Cloninger's bio-social model of personality: Harm Avoidance (HA 18 items); Novelty Seeking (NS 18 items); and Reward Dependence (RD 18 Items). The EPQ assesses the Eysenckian dimensions of (*N* 12 items), Extraversion (*E* 12 items), Psychoticism (*P* 13 items), and Social Conformity or Lie (*L* 12 items). Both the TPQ and EPQ were scored on the same three-point scale as the IPSM measures. Cumulative scores were then calculated for each of the EPQ, TPQ and IPSM scales: a five-point ordinal scale (0 to 4) for INT and SEP; three-point ordinal scales (0 to 2) for FIS and TIM; 13-point scales (0 to 12) for *N*, *EXT* and *L*; a 14-point scale (0 to 13) for *P*; and 19-point scales (0 to 18) for HA, NS and RD.

2.4. Analysis of raw ordinal data

Analysis of continuous data in recent years has taken advantage of raw data methods using programs such as Mx (Neale, 1999) to make use of complete and incomplete data observations. The extension of raw data methods to ordinal data permits researchers to test hypotheses concerning the equality of threshold distributions within twin pairs, across sex and zygosity. Raw data analysis also allows hypothesis testing of the equality and causes of correlations. In addition, one can obtain Maximum Likelihood tests for the equality of thresholds in complete versus incomplete pairs thereby enabling detection of cooperation bias (Neale & Eaves, 1993).

Raw data methods have the added advantage of increasing the accuracy of the estimation of the thresholds, thereby improving estimation of the polychoric correlations. The major disadvantage is that computational demands are proportional to the number of categories and the current analyses proved no exception. This is because the integration of the multivariate normal distribution becomes extremely time consuming. The distribution of the two- and four-item IPSM sub-scales clearly meant that categorical data analysis was required and since it is hard to obtain stable estimates of polyserial correlations between continuous and ordinal data measures, we therefore chose to treat all of the variables as ordinal and collapsed the larger number of categories in the EPQ and TPQ dimensions. Extensive preliminary analyses revealed that the optimum number of ordinal categories for the EPQ and TPQ dimensions was seven, which balanced the need for minimal information loss and greater computational efficiency. This resulted in no significant change in either the polychoric correlations or the variance of the scales.

2.5. Statistical analysis

The imputation option of PRELIS 2.20 (Jöreskog & Sörbom, 1998) was used to impute missing values using sex and the full number of items within each dimension/sub-scale as matching variables. This approach substitutes values for the missing values from other cases with similar response patterns provided there are (1) no missing values in the matching variables from other cases and that (2) the variance in the values from the other cases is acceptable (Jöreskog & Sörbom, 1992). The total variance of the matching cases by default must be less than half of the total variance of the item to be imputed estimated from all other cases with complete data.

Only 80 IPSM items were imputed, which represents 0.19% of the total number of items. Imputation of missing values increased the total effective sample size to 1388 males (2.4% increase) and 2125 females (2.3% increase). After imputation, both members of 1375 twin pairs had complete responses for the IPSM items, plus a further 763 single twins. A total of 374 EPQ items were imputed (approximately 0.3% of total number of EPQ items), increasing the total effective sample size to 1295 males (13.0% increase) and to 2022 females (12.5% increase). After imputation, both members of 1105 twin pairs had complete responses for the EPQ items, plus a further 917 single twins. A total of 453 TPQ items were imputed (approximately 0.29% of total number of TPQ items), which increased the total effective sample size to 1297 males (16.2% increase) and to 2027 females (15.5% increase). After imputation, both members of 1244 twin pairs had complete responses for the TPQ items, plus a further 836 single twins.

Internal consistencies (Cronbach Alphas) for the full sample for women were 0.51 for *P*, 0.87 for *EXT*, 0.80 for *N*, 0.71 for *L*, 0.83 for HA, 0.72 for NS, 0.60 for RD, 0.52 for SEP, 0.65 for INT, 0.46 for FIS, and 0.33 for TIM. For males, internal consistencies (Cronbach Alphas) were 0.51 for *P*, 0.86 for *E*, 0.80 for *N*, 0.72 for *L*, 0.83 for HA, 0.75 for NS, 0.66 for RD, 0.40 for SEP, 0.63 for INT, 0.50 for FIS, and 0.35 for TIM.

2.6. Genetic analysis

Based on the principles of biometrical genetics (Neale & Cardon, 1992) the degree to which family members are more or less alike can be partitioned into four broad causes of variation: additive genetic influence (*A*), genetic dominance ((*D*) or other genetic non-additivity such as epistasis), common environment (*C*), and specific environment (*E*). In the absence of genotype×environment interaction and genotype×environment correlation effects, which will be confounded with the other parameters in the twin design, the total variance in an observed trait is the sum of *A*, *C*, *E* and *D*. The different patterns of intra-pair correlations between MZ and DZ twins can then be used to indicate the presence of genetic and environmental influences. Current methods use structural equation modeling (SEM) as performed by LISREL (Jöreskog & Sörbom, 1998) or Mx (Neale, 1999) to decide which combination of the four parameters (*A*, *C*, *D*, and *E*) provides the most parsimonious explanation of the observed pattern of MZ and DZ twin correlations (McGregor et al., 1999), while at the same time estimating the size of the genetic and environmental parameters. This task is made easier given that *C* and *D* are entirely negatively confounded in twin studies, so that only one can appear in a given model. Furthermore, detecting dominance is unlikely given the large sample sizes required. It is also

inconceivable for complex behavioural traits to be measured without error, therefore all models must include *E*.

For each of the EPQ, TPQ and IPSM variables, univariate genetic models were fitted separately for males and females, and then jointly to the four same-sex twin pair groups. These models were then extended to include the DZ opposite-sex twin pairs. We could therefore test the heterogeneity of fit for models over sex by adding the separate log likelihood values for the same sex male and female twin pairs and then subtract this value from the log likelihood of the joint fit to males and females (Jardine & Martin, 1984). Our preliminary tests of threshold homogeneity for each of the variables revealed that separate thresholds were required for male and female twin pairs for most variables, while a single set of male/female thresholds was sufficient for FIS and TIM. For HA, there was also marginal within-sex heterogeneity when opposite sex twin pairs were included.

While univariate analysis estimates the contribution of additive and non-additive genetic as well as within family variance in personality scores, it says nothing about the underlying genetic and environmental causes of covariation between personality dimensions. Multivariate analysis takes advantage of the cross-twin cross-trait correlations, and thereby allows us to determine the degree to which separate genetic and environmental factors are responsible for the correlations between variables. In the current study, multivariate analyses were performed with a Cholesky Decomposition (Neale & Cardon, 1992) to partition variances in Mx. The Cholesky Decomposition is a method of triangular decomposition where the first variable is assumed to be caused by a latent variable that can also explain some or all of the variance in the remaining variables (Page & Martin, 1998). The second variable is assumed to be influenced by an additional latent variable that can explain variance in the second as well as remaining variables. This pattern continues until the final observed variable is explained by a latent variable, which is constrained from explaining the variance in any of the previous observed variables (i.e. a factor specific to one variable). The same factor structure is repeated for each source of variance (A, D, C, and E) with the same considerations of parsimony, identification, and categorical cogency as in univariate analysis.

Unfortunately, a Cholesky Triangular Decomposition of all the 11 variables using raw data methods in the multivariate analyses proved too computationally demanding. Models were therefore fitted to 11×11 polychoric correlation matrices based on complete pairs using weighted least squares. Model fit for heterogeneity between the sexes was again assessed by adding the separate log-likelihoods for males and females and then subtracting this value from the log-likelihood of the joint fit to same-sex male and female data (Jardine & Martin, 1984). Heterogeneity of fit was only marginally significant between males and females for the fully saturated ACE model ($\Delta\chi^2_{231} = 280.60$, $P < 0.05$) but significant for the AE model ($\Delta\chi^2_{165} = 245.27$, $P < 0.001$). Therefore, we fitted separate models for male and female twin pairs (results available on request). However, visual inspection of the factor structure revealed no major anomalies between the sexes, so we proceeded with multivariate analysis based on the combined male and female data which included the opposite sex DZ twin pairs.

Since we were interested in determining the extent to which the IPSM assesses new dimensions of genetic and environmental variance we ordered the personality variables such that the seven EPQ and TPQ dimensions preceded the four IPSM sub-scales and then described the analysis in terms of predicting the IPSM from the dimensions of the EPQ and TPQ. Therefore, for each of

the genetic and environmental IPSM sub-scale factors, the sum of the first seven squared loadings will give the total genetic and environmental variance attributable to the EPQ and TPQ scales. The sum of the remaining squared loadings will give the residual genetic and environmental variance attributable to the IPSM sub-scale factors.

3. Results

Polychoric correlations between the EPQ, TPQ, and IPSM variables are shown in Table 1. For female twin pairs, both P and EXT correlated negatively with the full IPSM and INT. *N* correlated highly with the full IPSM, SEP and INT, somewhat modestly with FIS and negatively with TIM. *L* scores correlated positively with TIM. Within the IPSM sub-scales, SEP correlated positively with INT and FIS, which also correlated with each other. HA correlated positively with the full IPSM, SEP, INT, FIS, but not TIM. For male twin pairs, *P* also correlated negatively with the full IPSM and INT, while *EXT* correlated negatively with only the full IPSM. As observed for women, *N* correlated highly with the full IPSM, SEP and INT, and more modestly with FIS, while *L* scores correlated positively with TIM. SEP correlated positively with INT only, and HA correlated positively with the full IPSM, SEP, and INT.

3.1. Univariate analysis

No significant heterogeneity across sex was found for either the ACE or AE models across all variables, with the exception of *P* and NS. For each personality dimension and sub-scale the

Table 1
Polychoric correlations between the EPQ, TPQ, and IPSM variables[a] Females = 2100

	1	2	3	4	5	6	7	8	9	10	11	12	13
1. Age		−0.02	−0.08	−0.08	0.07	0.02	−0.08	0.00	−0.03	0.05	0.02	0.01	0.02
2. Neuroticism	−0.02		**−0.26**	−0.07	−0.15	**0.60**	−0.01	0.00	**0.48**	**0.53**	**0.29**	**−0.20**	**0.50**
3. Extraversion	−0.01	**−0.24**		0.18	−0.11	**−0.60**	0.45	**0.34**	−0.18	**−0.22**	−0.14	−0.02	**−0.23**
4. Psychoticism	−0.01	−0.03	0.11		**−0.21**	**−0.24**	**0.36**	−0.18	0.00	**−0.34**	0.01	−0.15	**−0.22**
5. Lie	0.07	−0.17	−0.08	**−0.21**		−0.03	**−0.37**	0.03	−0.07	−0.11	−0.11	**0.37**	0.01
6. Harm Avoidance	−0.03	**0.58**	**−0.58**	−0.16	−0.07		**−0.28**	−0.13	**0.37**	**0.46**	**0.25**	−0.07	**0.43**
7. Novelty Seeking	−0.09	0.01	**0.42**	**0.37**	**−0.39**	**−0.25**		0.14	0.00	−0.07	0.00	−0.19	−0.10
8. Reward Dependence	−0.03	−0.01	**0.41**	**−0.24**	0.10	**−0.22**	0.12		−0.05	0.17	−0.04	0.09	0.09
9. Separation Anxiety	−0.06	**0.43**	−0.17	0.03	−0.07	**0.33**	0.01	−0.06		**0.38**	**0.28**	0.00	**0.79**
10. Inter Awareness	−0.02	**0.49**	−0.15	**−0.35**	−0.09	**0.44**	−0.08	0.15	**0.24**		**0.22**	0.03	**0.83**
11. Fragile Inner-Self	−0.01	**0.23**	−0.05	0.13	−0.14	0.15	0.12	−0.10	0.18	0.16		−0.09	**0.52**
12.Timidity	0.09	−0.15	−0.12	−0.13	**0.44**	−0.01	**−0.28**	0.05	0.02	0.03	−0.12		**0.39**
13. IPSM	−0.00	**0.48**	**−0.21**	**−0.23**	0.04	**0.42**	−0.12	0.08	**0.68**	**0.83**	**0.44**	**0.42**	

Males≅1250

[a] Correlations > 0.20 are in bold-face. The number of twin pairs is an approximation since correlations are based on pair-wise deletion and sample sizes will vary across variables. EPQ, Eysenck's Personality Questionnaire; TPQ, Tridimensional Personality Questionnaire; IPSM, Interpersonal Sensitivity Measure Inter awareness, Interpersonal awareness.

log-likelihoods associated with the E and AE models deteriorated significantly when compared to the fully saturated ACE model. The AE model did not deteriorate significantly from the full model in terms of the change in log-likelihood values, and was therefore chosen as the best fitting model for each EPQ and TPQ personality dimension and IPSM sub-scale. For FIS, the change in log-likelihood values for the AE ($\Delta\chi^2 = 0.02$, d.f. = 1) or CE ($\Delta\chi^2 = 2.31$, d.f. = 1) models did not deteriorate significantly from the full ACE model, however, the AE model demonstrated the lowest AIC value (−2.00 vs. 0.31) and was chosen over the CE as the best fitting model. Maximum likelihood point estimates for the best fitting univariate results are shown in Table 2.

3.2. Multivariate analyses

Multivariate model fitting results are shown in Table 3. The E model, which predicts no familial aggregation, was firmly rejected when compared to the fully saturated ACE model

Table 2
Maximum Likelihood point estimates for the best fitting univariate models based on raw data analysis[a]

Variable	A	C	E	D	-2LL	d.f.	Δd.f.	Δ-2LL	*P*
N	0.38		0.62		12949.85	3535	1	1.11	0.29
EXT	0.46		0.54		12394.67	3379	1	0.00	–
P	0.40		0.60		10953.81	3431	1	0.00	0.96
L	0.44		0.56		12762.03	3525	1	0.81	0.37
HA	0.44		0.56		12762.03	3525	1	0.81	0.37
NS	0.38		0.62		12519.05	3306	1	1.81	0.18
RD	0.35		0.65		12004.68	3305	1	0.00	0.97
SEP	0.28		0.72		9773.01	3536	1	0.62	0.43
INT	0.36		.64		10356.87	3487	1	0.47	0.49
FIS	0.24		0.76		3623.95	3558	1	0.02	0.92
TIM	0.32		0.68		7562.55	3539	1	0.02	0.88
IPSM	0.27		0.73		9585.10	3414	1	1.83	0.18

[a] N, neuroticism; EXT, extraversion; P, psychoticism; L, lie; HA, harm avoidance; NS, novelty aeeking; RD, reward dependence; SEP, separation anxiety; INT, interpersonal awareness; FIS, fragile inner-self; TIM, timidity; IPSM, total IPSM.

Table 3
Multivariate model fitting results based on weighted least squares (WLS)

Model	χ^2	df	$\Delta\chi^2$	Δd.f.	*P*	AIC
ACE	2038.45	1034				−29.55
ADE	2061.78	1034				−6.22
AE[a]	2112.74	1100	74.29	66	0.22	−87.26
CE	2195.41	1100	156.96	66	***	-4.59
E	2873.28	1166	834.83	132	***	541.28

[a] Best fitting model; Number of complete pairs per twin pedigree: MZFF = 293; MZMM = 154; DZFF = 204; DZMM = 111; DZFM = 197.

($\Delta\chi^2_{132} = 834.83$, $P < 0.001$). The CE model, which predicts familial aggregation arising from common environmental effects also departed significantly from the saturated model ($\Delta\chi^2_{66} = 156.96$, $P < 0.001$). The AE model did not deteriorate significantly from the fully saturated ACE model ($\Delta\chi^2_{66} = 74.29$, $P < 0.22$) and demonstrated the lowest Akaike's Information Criterion (AIC) value of −87.26. It was therefore chosen as the best fitting model. The additive genetic and unique environment factor structures were then simplified by successively dropping non-significant parameters (using goodness-of-fit chi-square to judge whether a parameter, once dropped, results in a significant deterioration in fit). A total of 41 parameter loadings were dropped from the final AE model ($\Delta\chi^2_{41} = 42.83$, $P = 0.39$).

Table 4 summarizes the estimates for the additive genetic and environmental variance within the IPSM sub-scales attributable to the EPQ and TPQ. Multivariate heritability estimates for the IPSM sub-scales were quite similar to the univariate estimates and ranged from 25 to 37%. Only 22% [(10 + 11 + 1)/100] of the total phenotypic variance in SEP was explained by latent additive genetic and environmental factors underlying EPQ and TPQ dimensions. The EPQ explained 40% of the genetic variance in SEP, whereas 60% of the total genetic variance and 88% of the total non-shared environmental variance was explained by genetic and environmental factors unique to SEP.

Latent additive genetic factors underlying the EPQ and TPQ dimensions explained approximately 46% of the total phenotypic variance in INT. Despite a modest additive heritability of 37%, only 24% of the latent genetic effects could be explained by genetic factors unique to INT whereas 70% of the total non-shared environmental variance could be attributable to latent INT environmental factors. For FIS, only 22% of the total phenotypic variance was explained by latent genetic and environmental factors common to the EPQ and TPQ dimensions. A latent genetic FIS factor explained 35% of the genetic variance, while almost all of the specific environmental variance (88%) was attributable to latent environmental effects unique to FIS. The

Table 4
Proportion of total phenotypic variance within IPSM sub-scales attributable to EPQ and TPQ latent additive genetic and environmental factors[a]

Parameter	SEP	INT	FIS	TIM
Genetic effects				
Total genetic variance	25	37	26	33
Explained by EPQ factors 1–4	10	28	7	16
Explained by TPQ factors 5–7	0	0	9	3
Explained by other IPSM factors	-	0	0	8
Unique variance	15	9	9	6
Non-shared environmental effects				
Total environmental variance	75	63	74	67
Explained by EPQ Factors 1-4	11	16	5	6
Explained by TPQ Factors 5-7	1	2	1	0
Explained by other IPSM factors	–	1	3	0
Unique variance	66	44	65	61

[a] IPSM, Interpersonal Sensitivity Measure; EPQ, Eysenck's Personality Questionnaire; TPQ, Tridimensional Personality Questionnaire; SEP, Separation anxiety; INT, Interpersonal awareness; FIS, fragile inner-self; TIM, timidity.

EPQ and TPQ explained approximately 28% of the total phenotypic variance in TIM. Of the total additive genetic variance, 18% was explained by TIM, 24% by other IPSM genetic factors, and the remaining 58% by genetic factors common to the EPQ and TPQ. Again, a very large portion of the total non-shared environmental variance (91%) was attributable to latent environmental effects unique to TIM.

4. Discussion

Univariate model fitting to the IPSM data confirmed a modest genetic influence on the full IPSM (27%), SEP (28%), INT (36%), FIS (24%) and TIM (32%). In each case, non-genetic hypotheses for the total IPSM and IPSM sub-scales were rejected by a chi-square test of goodness-of-fit. For the total IPSM and IPSM sub-scales, there was no evidence of sex differences in the causes of variance as judged by the chi-square tests of goodness-of-fit. Univariate modeling also confirmed previous findings regarding the heritability of the TPQ personality dimensions (Heath et al., 1994; Stallings, Hewitt, Cloninger, Heath & Eaves, 1996) with modest genetic heritability for HA (44%), NS (38%) and RD (35%).

4.1. Underlying genetic structure

The multivariate genetic Cholesky Decomposition allowed us to determine the extent to which the EPQ, TPQ and IPSM assess the same dimensions of genetic and environmental variability. The EPQ and TPQ dimensions explained moderate to large proportions of the genetic variance (40–76%) in the IPSM sub-scales. In terms of the non-shared environment covariance, the four IPSM sub-scales were substantially influenced by environmental factors unique to each sub-scale. Latent EPQ and TPQ non-shared environmental factors explained only 16% of the total environmental covariance in SEP, 29% of the total environmental covariance in INT and only 8 and 9% of the total environmental covariance in FIS and TIM, respectively.

4.2. Limitations

Naturally, the relative proportions of variance in the IPSM attributable to the individual EPQ and TPQ scales will change as a function of their ordering in the Cholesky Decomposition (Loehlin, 1995). We did not reverse the order of the EPQ and TPQ dimensions since our theoretical rationale was to estimate the proportion of variance unique to IPSM only. Variance unique to the IPSM will remain unaltered regardless of the ordering of the EPQ and TPQ scales. It is also important to note that the non-shared environmental variance, in addition to containing variance attributable to aspects of the twin environment not shared with other siblings also contains measurement error. Therefore, one must interpret the IPSM sub-scales with a degree of caution given the sub-scales' low internal consistency vis-a-vis the longer and more internally reliable EPQ and TPQ dimensions. Although the internal reliabilities for the TPQ and EPQ dimensions were almost identical to those found in a previous study (Heath et al., 1994), Cronbach alphas for each of the IPSM sub-scales were lower than those reported in previous analyses (Boyce & Parker, 1989; Todd et al., 1994) despite using a comparable shortened measure of the

IPSM (Todd et al.). It is likely that the shortened version of the IPSM in the current study, and in particular the two-item sub-scales of TIM and FIS have a large measurement error which means that most of the error cannot be attributed to true individual differences per se.

Test–retest correlations were unavailable for the current sample of twins. We were nevertheless able to assess the impact on our heritability estimates after removing measurement error by including the Todd et al. (1994) retest correlations based on a comparable shortened version of the IPSM. After correction, the proportion of the total phenotypic variance due to additive genetic effects for SEP was 0.32 (increase 7%), 0.48 for INT (increase 11%), 0.27 for FIS (increase 1%), and 0.46 for TIM (increase 13%). A critical assumption of this method is that genetic factors that influence the self-report depression prone symptoms are stable over time in their effect throughout adulthood. Gene expression can be quite variable over time, with certain genetic systems "switching" "on" and "off" (Eaves, Long & Heath, 1986) although this is likely to be less of a problem given the tight age range of our sample (18–28).

Even after imputation, the number of twins who provided complete IPSM responses represented 53.7% of contactable subjects, which raises the question of potential sample biases. Social class was obtained for both mail and telephone respondents, whereas telephone respondents to the abbreviated questionnaire ($n = 1198$) were not asked the psychiatric symptom items. Additionally, 171 mail respondents failed to complete the symptom items whereas most answered the social class item. Within the total sample of respondents, therefore, we could test whether the distribution of self-reported social class differed between those responding, and those not responding to the symptoms. No difference was observed ($\chi^2 = 0.57$, d.f. $= 2$, $P = 0.75$). We also compared responders versus non-responders in terms of the level of education attained ranging from (1) "primary school" through to (7) "university postgraduate". There were significant differences in attained education ($\chi^2 = 126.14$, d.f. $= 6$, $P < 0.001$) such that twins with higher education were more likely to respond, yet the correlation between education and total IPSM scores was not significant ($r = -0.02$, $P = 0.31$).

We also tested the possibility that subject participation was correlated with psychiatric symptoms. If this was the case, then to the extent that psychiatric symptomatology aggregates in families, we would expect to find mean scores in single twins (where the cotwin has not responded) to be biased in the direction of non-cooperation, compared with the mean for complete pairs (Neale & Eaves, 1993). No significant threshold liability differences were found between complete and incomplete twin pairs on measures of depression, phobic anxiety or somatic distress and we concluded that cooperation bias was unimportant in our study.

In summary, the EPQ and TPQ dimensions account for a considerable proportion of the genetic variance in IPSM sub-scales. A large proportion of the non-shared environment variance within the IPSM, which is unique to the IPSM and the sub-scales is likely due to measurement error and is not necessarily attributable to true individual differences per se.

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