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# Widespread Evidence for Non-Additive Genetic Variation in Cloninger's and Eysenck's Personality Dimensions Using a Twin Plus Sibling Design

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Studies using the classical twin design often conclude that most genetic variation underlying personality is additive in nature. However, studies analyzing only twins are very limited in their ability to detect non-additive genetic variation and are unable to detect sources of variation unique to twins, which can mask non-additive genetic variation. The current study assessed 9672 MZ and DZ twin individuals and 3241 of their siblings to investigate the environmental and genetic architecture underlying eight dimensions of personality: four from Eysenck's Personality Questionnaire and four from Cloninger's Temperament and Character Inventory. Broad-sense heritability estimates from best-fitting models were two to three times greater than the narrow-sense heritability estimates for Harm Avoidance, Novelty Seeking, Reward Dependence, Persistence, Extraversion, and Neuroticism. This genetic non-additivity could be due to dominance, additive-by-additive epistasis, or to additive genetic effects combined with higher-order epistasis. Environmental effects unique to twins were detected for both Lie and Psychoticism but accounted for little overall variation. Our results illustrate the increased sensitivity afforded by extending the classical twin design to include siblings, and may provide clues to the evolutionary origins of genetic variation underlying personality.

**KEY WORDS:** Behavior genetics; dominance; epistasis; non-additive genetic variation; personality.

## INTRODUCTION

One of the most consistent findings in modern personality research has been that unique experiences and genes play important roles in the development of personality differences while shared familial environmental effects are much less influential. Support for this comes from studies of adoptees and their families (e.g., Loehlin *et al.*, 1985), separated twins (e.g., Bouchard *et al.*, 1990), twins reared together (e.g., Eaves *et al.*, 1989), and twins along with other family members (e.g., Eaves *et al.*, 1999). While each methodology has potential confounds individually, most of these confounds do not overlap. Taken together these studies suggest that genetic differences account for one-third to half of the variation in studied personality dimensions while shared environments account for little, if any, of this variation.

Beyond these broad conclusions, there has been little resolving power to describe the genetic architecture underlying personality. Most research on the genetics of personality has come from classical twin studies, which rely on the comparison between MZ (monozygotic, identical) and DZ (dizygotic, fraternal) twins reared together. These studies typically conclude that most of the genetic variation underlying personality is additive in nature; non-additive genetic

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effects are usually non-significant and dropped from the final models (for Eysenck scales, see Eaves *et al*., 1989; Gillespie *et al*., 2003; for Cloninger's scale, see Heath *et al*., 1994b; Heiman *et al*., 2004; Stallings *et al*., 1996). However, classical twin studies usually have too little power to be able to detect non-additive genetic effects due to its high correlation with additive genetic effects (Martin *et al*., 1978), and thus conclusions regarding parameter estimates are usually (and rightfuly) followed by caveats that non-additivity may have existed but have gone undetected.

Extending the classical twin design to include siblings (as well as other relatives) results in a considerable increase in power to detect non-additive genetic or shared environmental effects (Posthuma and Boomsma, 2000). For example, when the population additive genetic variation is twice the dominance genetic variation, using siblings in addition to twins reduces the sample size required to detect dominance variation at 80% power to less than half of what would be required to detect dominance variation using MZ and DZ twins alone. This occurs because of the large number of additional covariance observations that result from including siblings. Whereas adding a twin pair (two individuals) to a twin study provides only one additional observed covariance, adding two siblings of twins provides five additional observed covariances.

Recently, three studies using extended twin-family designs have investigated the genetic and environmental structure of Eysenck's personality dimensions (Eaves *et al*., 1998, 1999; Lake *et al*., 2000). These studies employed twins and their siblings, parents, offspring, and spouses to simultaneously estimate additive and non-additive genetic effects as well as the effects of cultural transmission, assortative mating, shared environments, and unique environments. Consistent with prior classical twin studies, there was little evidence for shared environmental effects, and what little was apparent was attributable to siblings or twins rather than to parental influences. However, unlike many previous studies using the classical twin design, non-additive genetic effects were detectable and pervasive.

In contrast to these studies on the Eysenck scales, no studies with sufficient power to detect non-additive genetic variation have been conducted on the Cloninger personality dimensions to our knowledge. As discussed below, the Cloninger scales measure largely different personality constructs than the Eysenck scales. Moreover, these scales appear to be more highly related to several personality disorders than the Eysenck scales (Mulder and Joyce, 1997). Given this, it is of interested to understand whether non-additive genetic variation is also widespread in the Cloninger scales.

The current study used twins and their siblings to investigate the genetic and environmental architecture underlying the Cloninger and Eysenck personality dimensions. This study assesses whether the findings on the Eysenck personality dimensions from U.S. twins and their relatives replicate in a sample of Australian adults.[5] Moreover, this is the first test that uses siblings in addition to twins to estimate the genetic and environmental determinants of Cloninger's personality dimensions, and thus represents the best opportunity to date to assess the degree of genetic non-additivity underlying these personality dimensions. As we discuss below, non-additive genetic variation may provide clues about traits' evolutionary origins, but it can complicate trait-level and gene-level statistical analyses.

## METHOD

### Participants

Data for the present study were drawn from two cohorts of twins and their relatives. The first cohort was from a follow-up to a 1981 study of twins registered in the voluntary Australian Twin Registry. In 1988, Health and Lifestyle Questionnaires (HLQ) were mailed to 3808 twin pairs who had returned complete data in the original 1981 study. HLQ data were obtained from 5903 (78%) of these twins, who also provided names of relatives (parents, spouses, siblings, and children) who might be willing to fill out similar mailed questionnaires. The second cohort was from a sample of younger Australian Twin Registry twins who were born between 1964 and 1971. In 1989, the HLQ was mailed to 8538 twins from this second cohort, 3769 (44%) of whom completed the questionnaire.[6] These twins also provided names of relatives who might participate in future studies.

The names of 6805 siblings and 12,609 other relatives were obtained from both cohorts. Between 1989 and 1991, the HLQ was mailed to these relatives, and responses were completed by 3667 siblings and 7525 other relatives (response rates of 54 and

[5]With the exception that Lake *et al*. (2000) used both Australian and U.S. samples in their investigation of Neuroticism.

[6]Response rates were much higher for the first cohort because HLQ questionnaires were sent only to those twins who had returned complete data in a previous study. First contact response rates were similar between the two cohorts.

60% respectively). In families in which more than two siblings returned completed data, we randomly selected two siblings in order to reduce the complexity of the statistical models. This reduced our sample of siblings to 3241. Altogether, we analyzed data from 9672 individual twins (46% MZ) and 3241 siblings of twins. Thirty-eight percent of the sample was male.

Ages ranged from 18 to 90 ($\bar{x} = 35.0$, SD = 13.2). Previous studies have found that, although personality tends to change in predictable ways across development (a result replicated in our own findings, see below), once measurement errors are accounted for, the rank orderings of adults on personality traits within age cohorts tend to be quite stable (Watson, 2004). To assess short-term test–retest reliabilities of the scales in this study, a second HLQ was mailed to 500 female and 500 male twins from the first cohort. Completed questionnaires were returned by 451 female and 430 male participants. The average interval between completing the original and repeat HLQ was 2.1 years.

Zygosity of twins was determined based on the twins' responses to standard questions about similarity. Pairs giving inconsistent responses were interviewed further by telephone for clarification. These procedures agree with diagnoses based on blood samples in over 95% of cases (Ooki *et al*., 1990).

## Measures

The HLQ included a short-form 48-item revised Eysenck Personality Questionnaire (EPQ-R) (Eysenck *et al*., 1985) as well as a short-form 54-item version of the Temperament and Character Inventory (TCI) (Cloninger *et al*., 1991). The EPQ-R measures four dimensions of personality: Extraversion (E), Neuroticism (N), the tendency to 'fake good', called Lie (L), and Psychoticism (P). The TCI was originally designed to measure three dimensions of temperament: Harm Avoidance (HA), Novelty Seeking (NS) and Reward Dependence (RD). Subsequent revisions of the model recognized five items that originally contributed to RD made up a separate dimension, Persistence (PS) (Cloninger, 1994). For descriptions of these eight personality dimensions, see Table I.

The TCI and EPQ-R personality dimensions are largely non-redundant descriptions of personality except that HA shares moderate genetic and environmental variation with N and introversion, or the opposite of E (Heath *et al*., 1994a). Both the EPQ-R and, to a greater extent, the TCI personality dimensions are correlated with several personality disorders (Mulder and Joyce, 1997; Mulder *et al*., 1999). N and HA are correlated with avoidant, dependent, and self-defeating personality disorders, while P and NS are correlated with antisocial, borderline, narcissistic, and histrionic personality disorders. Two TCI personality dimensions are more uniquely related to specific personality disorders: RD with schizoid personality disorder and PS with obsessive compulsive disorder.

For each of the EPQ-R and TCI items, participants answered "Yes" (= 1), "No" (= 0), or "I don't know" (= 'missing') to statements about the self. Relevant items were reverse coded. Raw scale scores were the means of the available (non-missing) items on each scale so long as 75% of the participant's values were available on that scale. If more than 25% of a scale's items were missing for a subject, this scale score was treated as missing. Compared to treating scale scores as missing if any of its items were missing, this strategy led to a recovery of 7% of scale scores. We conducted angular transformations (arcsine of the square root) on the raw scores to remove the mean–variance relationship inherent to binomial distributions and to minimize departures from multivariate normality (Eaves *et al*., 1989). All scales were then standardized.

## Genetic Model

Phenotypic variation can be separated into additive genetic ($A$), non-additive genetic ($NA$), common environmental ($C$), and unique environmental ($E$) components. Twins and siblings can be used to estimate three of these four parameters because, although both types of twins as well as siblings share a common familial environment, $C$ (though see below), MZ twins share 100% of their genes while siblings and DZ twins share 50% of their genes. Thus, the degree to which MZ twins' resemblance exceeds DZ twins' or siblings' resemblance estimates genetic variation ($A + NA$). Evidence for $C$ is suggested when DZ twins' or siblings' resemblance is greater than half of the MZ twins' resemblance, while $NA$ is suggested when DZ twins' or siblings' resemblance is less than half of the MZ resemblance.

As is typical in twin analyses, we modeled $NA$ as allelic interactions (dominance; $D$) rather than non-allelic interactions (epistasis) due to an ability to distinguishing the two sources of genetic non-additivity in twin designs (Mather, 1974). It is important to note, however, that evidence for $D$ in a model could be due to dominance, epistasis, or some

**Table I.** Descriptions, Reliabilities, and Descriptive Statistics of Cloninger's and Eysenck's Personality Inventories

| Scale | Description (high vs. low) | # Items | Test–retest *r* | α | $N_F$ | $\bar{x}_F$ | $SD_F$ | $N_M$ | $\bar{x}_M$ | $SD_M$ |
|---|---|---|---|---|---|---|---|---|---|---|
| Cloninger's TCI scales | | | | | | | | | | |
| HA | Fearful, pessimistic vs. carefree, energetic | 18 | 0.79 | 0.84 | 7862 | 0.17 | 0.96 | 4812 | −0.27 | 1.00 |
| NS | Impulsive, curious vs. reflective, uninquiring | 18 | 0.73 | 0.75 | 7862 | −0.04 | 0.97 | 4807 | 0.06 | 1.04 |
| RD | Sensitive, sociable vs. practical, cold | 13 | 0.68 | 0.68 | 7853 | 0.23 | 0.95 | 4807 | −0.38 | 0.96 |
| PS | Industrious, perfectionistic vs. indolent, erratic | 5 | 0.64 | 0.61 | 7834 | −0.04 | 0.99 | 4803 | 0.07 | 1.02 |
| Eysenck's EPQ-R scales | | | | | | | | | | |
| E | Outgoing, active vs. introverted, reserved | 12 | 0.83 | 0.87 | 7745 | 0.02 | 1.00 | 4776 | −0.03 | 1.00 |
| N | Moody, anxious vs. content, stable | 12 | 0.78 | 0.82 | 7873 | 0.14 | 0.96 | 4835 | −0.23 | 1.02 |
| L | Low insight, suggestible vs. honest, open | 12 | 0.77 | 0.76 | 7866 | 0.11 | 0.97 | 4833 | −0.18 | 1.02 |
| P | Machiavellian, tough vs. warm, empathic | 13 | 0.56 | 0.53 | 7887 | −0.16 | 0.98 | 4846 | 0.27 | 0.98 |

*Note*: # Items = number of items used to make scale, α = Cronbach's alpha (pre-imputation), subscripts F, M = female, male, $N$ = number of individual participants used in analysis, $\bar{x}$ = standardized mean, SD = standard deviation.

combination of both. In a similar way, estimates of $C$ and $D$ are negatively confounded in twin studies because precisely the same bit of information (the degree to which DZ twins'/siblings' resemblance is above or below half the MZ twin resemblance) is used to estimate them both, and thus either one can mask the effects of the other. Lack of evidence for $C$, for example, suggests only that shared environmental effects are less powerful than non-additive genetic effects,[7] not that shared environment effects are non-existent. Because we could not estimate $C$ and $D$ simultaneously, we fit ADE rather than ACE models for the eight scales because the average correlation between DZ twins and between other siblings was less than half the MZ twins' correlation in every scale. The absence of shared environmental effects in studies of adopted twins (Bouchard *et al.*, 1990; Tellegen *et al.*, 1988) suggests that a negligible degree of shared environmental effects were missed by not including $C$ parameters in the present analyses. We nevertheless explore the potential amount of $C$ that our analyses missed below.

Gene–environment interactions ($G \times E$) and gene–environment correlations are also possible sources of variation, but as is typical with classical twin designs, these sources of variation are ignored in the present analysis because their estimates are very difficult to extract (Boomsma and Martin, 2002; Eaves *et al.*, 1977). From designs able to detect them (extended twin family designs), there is little evidence that gene–environment correlations explain much if any variation in the EPQ-R dimensions (reviewed in Coventry and Keller, 2005). Evidence for $G \times E$ is more difficult to detect using any design. Without measurements of specific environmental factors, detection of $G \times E$ depends upon high order moments and is generally unreliable (Boomsma and Martin, 2002). In the present analysis, variation due to interactions between genes and unique experiences are included in (i.e., confounded with) the $E$ term, interactions between additive genetic effects and common experiences are included in the $A$ term, and interactions between non-additive genetic effects and common experiences are included in the $D$ term.

A further important assumption of the present analysis is that the shared environmental correlation between MZ twins is essentially the same as that of DZ twins *with respect to the personality dimensions being studied*. The position that greater MZ personality resemblance is due to a special identical twin environment appears untenable: adoption studies of twins reared apart have typically found comparable levels of MZ-DZ differences as have traditional twin analyses (Bouchard *et al.*, 1990; Tellegen *et al.*, 1988). Finally, when DZ twin and sibling covariances are pooled in the present analysis, we are assuming that the shared sibling environment is essentially the same as the shared DZ twin environment. We test this assumption to the degree possible below.

## Statistical Analysis

We transformed age using the natural log to correct for its strong positive skew. This also had the effect of linearizing most of the age–personality relationships, which removed the need to include quadratic age effects. We controlled for the effects of

[7] In general, $C$ masks $D$ more easily than the reverse. Given dominance, $ACE$ models underestimate $\underline{C}$ by $-1/2D$ while $ADE$ models underestimate $\underline{D}$ by $2C$ (Eaves *et al.,* 1977). However, polygenic epistasis can cause this bias to be equally strong in both directions (see Keller and Coventry, 2005).

the log of age (hereafter age), birth order, sex, and their interactions on the mean levels of each of the personality dimensions for every analysis. Failing to account for these effects could lead to underestimations of *D* in the case of age and birth order and overestimations of *D* in the case of sex.

We first ran a series of models that tested assumptions regarding means, variances, and covariances. When these parameters appear to be drawn from the same underlying populations for different sexes or zygosities, it is preferable to pool their estimates for parsimony and accuracy. We used maximum likelihood analysis of individual observations to establish which parameters could be equated. We began with a fully saturated model and constrained successive models, comparing the difference in log likelihoods between successively constrained models to the $\chi^2$ distribution with *df* equal to the parameter differences between models. Parameters shown to be unequal were allowed to vary freely in subsequent models. Our fully saturated model contained a total of 32 freely varying parameters: (a) six slopes for the three covariates and their three interactions, (b) three mean terms (equivalent to intercepts in multiple regression because all variables were standardized) for MZ twins, DZ twins, and siblings, (c) six separate variance terms for MZ female twins, MZ male twins, DZ female twins, DZ male twins, female siblings, and male siblings, (d) nine separate covariance terms for DZ twins and their siblings (three for female–female, male–male, and opposite sex DZ twin-DZ twin pairings, three for female–female, male–male, and opposite sex DZ twin-sibling pairings, and three for female–female, male–male, and opposite sex sibling–sibling pairings), and finally (e) eight covariance terms for MZ twins that parallel the nine covariance terms for DZ twins (the opposite sex MZ twin covariance was not estimated since this pairing is impossible). We set the significance threshold to 0.01 due to the large number of tests conducted and the high sensitivity afforded by our large sample.

*A*, *D*, and *E* parameters were estimated though structural equation modeling using the software package Mx (Neale, 1999) and using a script for general sex limitation inspired by Medland (2004). The saturated (general sex limitation) models for HA, NS, RD, PS, E, and N estimated *A*, *D*, and *E* terms separately for males and females (*Af, Df, Ef, Am, Dm, Em*) and included male-specific additive genetic terms (*A′*) which estimated the degree to which the additive effect of male and female genes overlapped. Beginning with this saturated model, we then dropped or constrained parameters in the following order: (a) dropped *Am*, (b) restored *Am* and dropped *A′*, (c) dropped *Df* and *Dm*, (d) dropped *Am* and *Af*, (e) restored *A* and *D* but constrained *A*, *D*, and *E* to be equal between the sexes, with the total variances differing by some scalar, $\lambda$, (f) fixed $\lambda = 1$, (g) dropped *D*, and finally (h) dropped *A*. In addition to these parameters, for L and P we estimated a special twin environment component of variance, *S*, which was suggested by tests of assumptions.

## RESULTS

### Descriptive Statistics

Scale descriptions, number of items making up each scale, reliability statistics, and descriptive statistics of the eight personality scales are shown in Table I. For consistency with the test–retest statistics, we computed Cronbach's alphas only from twins' data (thus excluding siblings). Table II shows the phenotypic correlations between covariates and between the eight personality scales. The maximum likelihood coefficients and their confidence intervals for 14 correlations between different pairings of twins and siblings are shown in Table III. For all the personality measures except for P and L (see below) the DZ twin correlations were indistinguishable from sibling correlations, suggesting that there was no special twin environment and that DZ twin and sibling correlations could be safely pooled. Table III also shows that the average pooled MZ correlation exceeds twice the average pooled DZ and sibling correlation for each of the eight personality dimensions, implying that non-additive genetic effects are more important than shared environmental effects on these personality dimensions.

### Tests of Assumptions

MZ twins', DZ twins', and siblings' means differed from one another for RD, L, and P, while siblings' means differed from twins' means for PS and E (Table IV, models 1.1 and 1.2). DZ and sibling variances differed significantly for RD and N (model 2.1), while variances differed between the sexes for HA, NS, N, and L (Table IV, model 2.3). The magnitudes of these differences were not large. No means differed by more than 0.15 standard units, and no standard deviation term was less than 90% of the largest term. Nevertheless, we allowed these terms to

**Table II.** Correlations between Six Covariates and Cloninger's (TCI) and Eysenck's (EPQ-R) Personality Dimensions

| | | Covariates | | | | | | TCI dimensions | | | | EPQ-R dimensions | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | S | A | B | A*S | B*S | B*A | HA | NS | RD | PS | E | N | L | P |
| S | Sex (0 = M, 1 = F) | | | | | | | | | | | | | | |
| A | Age | 0.1 | | | | | | | | | | | | | |
| B | Birth order | 0.02 | −0.13 | | | | | | | | | | | | |
| A*S | Age*sex | −0.01 | 0.21 | −0.07 | | | | | | | | | | | |
| B*S | Birth order*sex | 0 | −0.07 | 0.18 | −0.13 | | | | | | | | | | |
| B*A | Birth order*age | −0.05 | −0.28 | −0.2 | −0.06 | 0.03 | | | | | | | | | |
| HA | Harm avoid | 0.21 | 0.03 | 0 | 0.03 | 0.02 | −0.02 | | | | I | | | | |
| NS | Nov. seeking | −0.05 | −0.3 | 0.07 | −0.05 | 0.02 | 0.01 | −0.24 | | | | | | | |
| RD | Reward dep. | 0.3 | −0.03 | 0.01 | 0.01 | 0.06 | 0 | −0.05 | 0.15 | | | | | | |
| PS | Persistence | −0.05 | −0.03 | −0.02 | 0 | −0.02 | 0.03 | −0.12 | 0.01 | 0.04 | | III | | | |
| E | Extraversion | 0.02 | −0.19 | 0.04 | −0.06 | 0.04 | 0 | −0.54 | 0.45 | 0.36 | 0.14 | | | | II |
| N | Neuroticism | 0.17 | −0.09 | 0.02 | −0.01 | 0.02 | 0.01 | 0.63 | 0.02 | 0.05 | 0.07 | −0.19 | | | |
| L | Lie | 0.14 | 0.29 | −0.09 | 0.09 | −0.01 | −0.05 | −0.02 | −0.36 | 0.02 | 0.05 | −0.11 | −0.13 | | |
| P | Psychoticism | −0.22 | −0.21 | 0.05 | −0.04 | −0.02 | 0.02 | −0.22 | 0.34 | −0.24 | −0.02 | 0.13 | −0.09 | −0.22 | |

*Note*: Boxes I & II represent intercorrelations between the TCI and EPQ-R scales, respectively. Box III represents cross-correlations between the TCI and EPQ-R scales.

be estimated independently in subsequent structural equation models. In no cases did MZ variances differ significantly from DZ/sibling pooled variances, suggesting that imitation and competition effects play little role in the variation underlying these traits (Carey, 1986; Eaves, 1976).

Generally, the use of siblings in twin designs relies upon the assumption that covariance terms involving siblings can be equated with covariance terms involving DZ twins (tested by models 3.1–3.3 in Table IV). While this was clearly the case for the first six personality dimensions, this assumption did not hold for L or P from the EPQ-R. This suggests some non-genetic aspect of the MZ and DZ twin environment causes twins to be more similar to one another. Because twins share a twin environment which siblings do not share, this portion of variation can be distinguished separately from the *A*, *D*, and *E* parameters in twin designs that include siblings. We model this special twin environment (*S*) below, and did not pool sibling and DZ covariance terms for L and P. Model 3.5 in Table IV suggests the action of sex-specific genes for HA and RD. That MZ correlations could not be equated with DZ or siblings' correlations for any personality trait provides strong evidence for genetic influences (model 3.6). Finally, the covariances for MZ twins, DZ twins, and siblings could not be set to zero for any variable (model 3.7), demonstrating within-family similarity: twins and siblings within families are more similar to each other than they are to randomly drawn members of the population.

## Structural Equation Modeling of Genetic Hypotheses

The models that best combined parsimony and fit[8] (based upon the Akaike Information Criteria) for each personality dimension are shown in Table V. The most salient result of our study was that we could not drop the *D* parameters for any of the first six personality dimensions (Table V). In the best-fitting models, *D* accounted for between 12% of the variation in male N to 36% of the variation in RD (both sexes), and generally accounted for more phenotypic variation than did *A*.[9] Put another way, the broad-sense heritability estimates for these six personality dimensions were two to three times greater than the narrow-sense heritability estimates. The *A* parameters were non-significant (but nevertheless retained in best-fitting models) for PS, NS, and RD, and were significant for HA, E, and N.

We also found evidence that the genes active in females were different from the genes active for males (modeled as male-specific genes) in HA, RD, and N, although for N there also appeared to be genes that overlapped for both sexes. For the other three dimensions, PS, NS, and E, the relative magnitudes of genetic effects appeared to be similar between the sexes, even

[8]Because interactions are unlikely to exist in the absence of main effects, best-fitting models retained non-significant *A* parameters in the presence of a significant *D* parameters.

[9]There was little resolving power to detect an additional male-specific dominance parameter, *D′*, in addition to *A′*. Thus, evidence for *A′* could be due to both dominant and additive sources of variation.

**Table III.** Correlations between Twins and Siblings along with Lower (0.025) and Upper (0.975) Bounds of 95% Confidence Intervals for the Cloninger and Eysenck Personality Dimensions

| Cloninger (TCI) personality dimensions | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Harm avoidance | | | Novelty seeking | | | Reward dependence | | | Persistence | | |
| | 0.025 | *r* | 0.975 | 0.025 | *r* | 0.975 | 0.025 | *r* | 0.975 | 0.025 | *r* | 0.975 |
| MZF-MZF | 0.39 | **0.43** | 0.47 | 0.36 | **0.40** | 0.44 | 0.34 | **0.39** | 0.43 | 0.31 | **0.35** | 0.36 |
| MZM-MZM | 0.38 | **0.45** | 0.51 | 0.35 | **0.42** | 0.48 | 0.30 | **0.37** | 0.43 | 0.28 | **0.35** | 0.42 |
| MZF-SibF | 0.09 | **0.17** | 0.24 | 0.02 | **0.09** | 0.16 | 0.06 | **0.13** | 0.19 | 0.09 | **0.10** | 0.12 |
| MZM-SibM | 0.09 | **0.20** | 0.31 | 0.05 | **0.16** | 0.27 | −0.03 | **0.08** | 0.19 | −0.07 | **0.05** | 0.16 |
| MZ-Opp. Sex Sib | 0.02 | **0.09** | 0.15 | 0.07 | **0.13** | 0.20 | −0.02 | **0.05** | 0.11 | 0.01 | **0.06** | 0.10 |
| DZF-DZF | 0.06 | **0.13** | 0.19 | 0.05 | **0.12** | 0.19 | 0.05 | **0.13** | 0.20 | 0.08 | **0.14** | 0.21 |
| DZM-DZM | 0.04 | **0.14** | 0.23 | −0.01 | **0.09** | 0.19 | 0.07 | **0.17** | 0.26 | 0.01 | **0.10** | 0.20 |
| DZ-Opp. Sex DZ | 0.03 | **0.09** | 0.16 | 0.06 | **0.12** | 0.19 | −0.05 | **0.01** | 0.08 | 0.04 | **0.10** | 0.17 |
| DZF-SibF | 0.08 | **0.15** | 0.21 | 0.03 | **0.10** | 0.16 | 0.06 | **0.12** | 0.18 | 0.05 | **0.10** | 0.12 |
| DZM-SibM | 0.08 | **0.17** | 0.25 | 0.05 | **0.14** | 0.22 | 0.11 | **0.19** | 0.27 | 0.08 | **0.17** | 0.19 |
| DZ-Opp. Sex Sib | 0.03 | **0.08** | 0.14 | 0.06 | **0.11** | 0.16 | 0.04 | **0.09** | 0.14 | 0.01 | **0.06** | 0.08 |
| SibF-SibF | 0.06 | **0.18** | 0.28 | 0.02 | **0.13** | 0.24 | −0.03 | **0.08** | 0.19 | −0.02 | **0.05** | 0.12 |
| SibM-SibM | 0.06 | **0.22** | 0.36 | 0.04 | **0.19** | 0.33 | 0.06 | **0.20** | 0.33 | −0.05 | **0.10** | 0.24 |
| Sib-Opp. Sex Sib | 0.01 | **0.11** | 0.20 | 0.04 | **0.13** | 0.22 | −0.01 | **0.08** | 0.18 | 0.02 | **0.10** | 0.14 |
| Eysenck (EPQ-R) personality dimensions | | | | | | | | | | | | |
| | Extraversion | | | Neuroticism | | | Lie | | | Psychoticism | | |
| | 0.025 | *r* | 0.975 | 0.025 | *r* | 0.975 | 0.025 | *r* | 0.975 | 0.025 | *r* | 0.975 |
| MZF-MZF | 0.42 | **0.46** | 0.50 | 0.37 | **0.42** | 0.46 | 0.40 | **0.44** | 0.48 | 0.33 | **0.38** | 0.42 |
| MZM-MZM | 0.44 | **0.50** | 0.55 | 0.29 | **0.36** | 0.43 | 0.23 | **0.30** | 0.37 | 0.26 | **0.33** | 0.40 |
| MZF-SibF | 0.11 | **0.18** | 0.24 | 0.08 | **0.16** | 0.22 | 0.04 | **0.11** | 0.18 | 0.05 | **0.12** | 0.19 |
| MZM-SibM | 0.11 | **0.23** | 0.33 | 0.11 | **0.23** | 0.33 | 0.07 | **0.18** | 0.28 | −0.01 | **0.10** | 0.21 |
| MZ-Opp. Sex Sib | 0.09 | **0.15** | 0.22 | 0.03 | **0.10** | 0.16 | 0.07 | **0.13** | 0.19 | 0.01 | **0.08** | 0.14 |
| DZF-DZF | 0.11 | **0.18** | 0.24 | 0.11 | **0.17** | 0.24 | 0.21 | **0.27** | 0.33 | 0.17 | **0.23** | 0.29 |
| DZM-DZM | 0.10 | **0.20** | 0.29 | 0.02 | **0.12** | 0.22 | 0.13 | **0.23** | 0.31 | 0.09 | **0.20** | 0.29 |
| DZ-Opp. Sex DZ | 0.08 | **0.14** | 0.20 | 0.05 | **0.12** | 0.18 | 0.12 | **0.18** | 0.24 | −0.03 | **0.03** | 0.10 |
| DZF-SibF | 0.14 | **0.21** | 0.27 | 0.08 | **0.14** | 0.20 | 0.09 | **0.15** | 0.21 | 0.04 | **0.10** | 0.16 |
| DZM-SibM | 0.10 | **0.19** | 0.27 | 0.07 | **0.16** | 0.24 | 0.03 | **0.12** | 0.21 | 0.07 | **0.16** | 0.24 |
| DZ-Opp. Sex Sib | 0.10 | **0.15** | 0.20 | 0.04 | **0.09** | 0.15 | 0.13 | **0.18** | 0.23 | 0.07 | **0.12** | 0.17 |
| SibF-SibF | 0.13 | **0.24** | 0.34 | 0.05 | **0.17** | 0.27 | 0.12 | **0.23** | 0.33 | −0.01 | **0.10** | 0.21 |
| SibM-SibM | 0.11 | **0.26** | 0.38 | 0.06 | **0.21** | 0.35 | 0.13 | **0.25** | 0.38 | −0.15 | **−0.01** | 0.13 |
| Sib-Opp. Sex Sib | 0.05 | **0.14** | 0.24 | 0.08 | **0.17** | 0.26 | 0.00 | **0.08** | 0.18 | 0.04 | **0.14** | 0.24 |

*Note*: Maximum likelihood correlation coefficient point estimates shown in bold.

though males had higher overall phenotypic variance for PS and NS.

Although the special twin environment was significant for both L and P, its magnitude was relatively small, accounting for 4–11% of the phenotypic variation in these scales. Also unlike the other six personality dimensions analyzed, *D* was non-significant in L and P. This was not due to the inclusion of *S*; its omission changed the point estimates for *A* and *D* very little. Rather, either relatively little non-additive genetic variation underlies these two personality constructs or shared environmental effects mask the genetic non-additivity. Two things regarding this finding should be noted, however. First, although *D* was not statistically significant, its point estimates in the full models of L and P (models 1.1 in Appendix 2) were not always small; for example, *A* and *D* were equal for male Psychoticism. Second, the type of special twin environments detected here would appear as *C* in analyses not including siblings in addition to twins, perhaps explaining why shared environmental effects have been detected in these two dimensions in the past (e.g., Heath *et al*., 1994b).

**Table IV.** Test of Assumptions: Difference in Log Likelihood $\Delta\chi^2$ for Tests on Intercepts, Variances, and Covariances for Eight Personality Dimensions

| | | | TCI dimensions | | | | EPQ-R dimensions | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | $\Delta df$ | HA | NS | RD | PS | E | N | L | P |
| Means models | | | | | | | | | | |
| 1.1 | MZ = DZ | 1 | 0.9 | 1.1 | 8.1* | 0.3 | 0.4 | 1.4 | 22.4** | 12.1** |
| 1.2 | MZ = DZ = Sib | 1 | 2.5 | 2.5 | 4.6 | 8.5* | 8.6* | 0.1 | 25.2** | 18.5** |
| Variance models | | | | | | | | | | |
| 2.1 | DZ = Sib | 2 | 0.4 | 4.6 | 19.3** | 5.1 | 1.2 | 11.5* | 1.5 | 5.9 |
| 2.2 | DZ = Sib = MZ | 2 | 0.9 | 1.9 | 3.9 | 0.6 | 2.1 | 0.2 | 0.7 | 2.1 |
| 2.3 | Sexes equal | 1 | 6.3* | 15.6** | 0.8 | 5.2 | 0.8 | 18.1** | 10.2** | 3.5 |
| Covariance models | | | | | | | | | | |
| 3.1 | MZ-Sib = DZ-Sib | 3 | 1.1 | 0.7 | 2.6 | 2.1 | 1.1 | 0.6 | 3.0 | 0.9 |
| 3.2 | MZ-Sib = DZ-Sib = Sib-Sib | 6 | 3.3 | 2.3 | 3.6 | 2.9 | 4.7 | 12.1 | 1.7 | 5.4 |
| 3.3 | MZ-Sib = DZ-Sib = Sib-Sib = DZ-DZ | 3 | 0.7 | 0.9 | 2.9 | 2.7 | 1.4 | 1.8 | 11.7** | 16.7** |
| 3.4 | Male–Male = Female–Female | 2 | 0.4 | 1.3 | 2.8 | 0.0 | 2.6 | 1.9 | 14.5** | 0.9 |
| 3.5 | Opposite sex = same sex | 1 | 11.0** | 0.0 | 12.4** | 3.6 | 5.0 | 4.7 | 0.9 | 4.2 |
| 3.6 | MZ-MZ = MZ-Sib = DZ-DZ = DZ-Sib | 1 | 183.1** | 161.1** | 143.6** | 133.2** | 185.6** | 144.7** | 13.0** | 91.3** |
| 3.7 | All covariances = 0 | 1 | 329.5** | 289.9** | 222.8** | 197.3** | 517.0** | 347.6** | 445.7** | 239.3** |

*Note*: See text for interpretations of tests.
*$p < 0.01$, **$p < 0.001$.

**Table V.** Heritability and Variance Component Estimates of Best-Fitting Structural Equation Models

| Dimension | $h^2f$ | $h^2m$ | $Af$ | $Df$ | $Sf$ | $Ef$ | $Am$ | $A'$ | $Dm$ | $Sm$ | $Em$ | $\lambda$ |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| TCI dimensions | | | | | | | | | | | | |
| Harm avoidance | 0.53 | 0.57 | 0.15 | 0.27 | | 0.58 | | 0.16 | 0.29 | | 0.55 | Ø |
| Novelty seeking | 0.55 | 0.55 | 0.05 | 0.35 | | 0.60 | 0.05 | | 0.35 | | 0.60 | 1.14 |
| Reward depend.[a, c] | 0.56 | 0.51 | 0.07 | 0.31 | | 0.62 | | 0.24 | 0.11 | | 0.63 | Ø |
| Persistence[b] | 0.55 | 0.55 | 0.00 | 0.35 | | 0.65 | 0.00 | | 0.35 | | 0.65 | 1.09 |
| EPQ-R dimensions | | | | | | | | | | | | |
| Extraversion[b] | 0.57 | 0.57 | 0.23 | 0.24 | | 0.53 | 0.23 | | 0.24 | | 0.53 | 1.00* |
| Neuroticism[c] | 0.54 | 0.49 | 0.19 | 0.23 | | 0.57 | 0.09 | 0.17 | 0.12 | | 0.62 | Ø |
| Lie[a, b] | 0.44 | 0.35 | 0.34 | | 0.09 | 0.57 | 0.27 | | | 0.06 | 0.69 | Ø |
| Psychoticism[a, b] | 0.39 | 0.43 | 0.22 | | 0.11 | 0.68 | 0.24 | | | 0.04 | 0.68 | Ø |

*Note*: Best-fitting models determined by AIC except that $A$ was never dropped in the presence of $D$. Subscripts $f$, $m$ = female, male. $h^2$ = reliability-corrected broad sense heritability = $(A + D)/r$ where $r$ is the test–retest correlation. Variance parameters: $A$ = additive genetic, $A'$ = male specific additive genetic, $D$ = dominant genetic, $S$ = specific twin environment, $E$ = unique environment, $\lambda$ = scalar for male variance (* = $\lambda$ fixed to unity, Ø = male and female $A$, $D$, and $E$ allowed to vary freely). Mean and variance terms were equated across MZ twins, DZ twins, and siblings unless they were found to differ during assumption testing.
[a]MZ and DZ means estimated independently, [b]Twin and sibling means estimated independently, [c]Twin and sibling variances estimated independently.

The effects of sex, age, birth order, and their interactions on the means of the eight personality dimensions are presented in Appendices 1 and 2. As expected, females were much higher than males in HA, RD, and N, and somewhat higher than males in E and L. Males were much higher than females in P and somewhat higher in PS and NS. NS, E, N, and P decreased with age while L increased. We found moderately large age-by-sex interactions for P and NS, predominately driven by the fact that young males are extremely high on these two dimensions. Finally, birth order had consistent though generally very minor effects on personality, and these effects tended not to depend on sex or age.

## DISCUSSION

Corrected for measurement error and short-term fluctuations in responses, genetic effects accounted for about 50% of the phenotypic variation for the

HA, NS, RD, PS, E, and N personality dimensions, and about 40% of the variation in P and L. There was evidence that at least some of the genes that affect variation in HA, RD, and N differ between males and females. Also of note, twins appeared to be more similar to each other on the P and L dimensions than expected from sibling correlations, due ostensibly to some aspect of the twin environment. One plausible interpretation of this source of variation is that twins are more likely to share peer groups than are siblings, perhaps due to being the same age, and that peer groups affect these two dimensions. Activation of certain genes at different ages could also account for some of this variation. Finally, we found no evidence that siblings or twins affect the personality of their co-twins or siblings through imitation or competition, and there was no evidence of common (family) environmental effects.

### Evidence for Non-Additive Genetic Effects

The results of the present study are consistent with three previous extended twin-family design studies (Eaves *et al*., 1998, 1999; Lake *et al*., 2000) that detected ubiquitous genetic non-additivity underlying E and N but little underlying L. The only discrepancy between the studies was in P, for which we found little evidence for non-additive genetic variation in both sexes whereas Eaves *et al*. (1999) reported substantial non-additive genetic variation in females only. Our results indicate that non-additive genetic effects account for an even greater degree of variation in the four Cloninger personality dimensions.

What accounts for the pervasive evidence for non-additive genetic variation underlying these personality dimensions? We identify three reasons why the genetic non-additivity might have been spurious and then turn to possible genetic mechanisms that could account for our observations. First, it is possible that something unique about the MZ environment, such as being treated more similarly by parents or peers, inflated our estimates of total and non-additive genetic variation. While this is an often cited possibility, it enjoys little empirical support (Kendler, 1983). Most damningly, the robust evidence that MZ twins reared apart are about as similar on personality measures as MZ twins reared together (Bouchard *et al*., 1990) makes a 'special MZ environment' an unlikely cause of the high MZ similarity observed in the present analysis.

Second, gene-by-age interactions can also mimic genetic non-additivity because twins are the same age while other relatives, including siblings, differ in age. Eaves *et al*. (1999) noted that such gene-by-age interactions were plausible alternative explanations for the results of their study. However, because sibling-twin age differences were much smaller in the present study than the relative-twin age differences in Eaves *et al*. (1999), and because correlations involving siblings were indistinguishable from correlations involving twins in all cases except for P and L, our results suggest that gene-by-age interactions cannot explain the ubiquitous genetic non-additivity that we detected for the Cloninger scales or for E and N.

Finally, random chance could lead to the spurious detection of genetic non-additivity. Additive and non-additive genetic variance estimates are highly negatively correlated (Martin *et al*., 1978), making point estimates for both $A$ and $D$ imprecise. While the broad-sense heritability estimates shown in Table V were fairly stable across many types of models, the relative contributions of $A$ and $D$ were not (Appendices 1 and 2). Thus, our point estimates of $D$ must be interpreted with some degree of caution, especially in the case of extreme values (e.g., $D = 0.35$ vs. $A = 0.00$ in PS). Nevertheless, the consistency and replicability of the genetic non-additive effects makes chance an unlikely explanation for the totality of our results.

It is likely, therefore, that the non-additive genetic effects detected in the present analysis are real. Given this, several different genetic mechanisms could be implicated. Most simply, the observed non-additivity could be due to genetic dominance at most of the loci that influence variation in personality. However, additive-by-additive epistasis is perfectly confounded with dominance in the current design. Using an extended twin-family design, Eaves *et al*. (1998) argued that some type of additive epistasis (e.g., interactions between the additive effects at two or more different loci) was more likely than dominance to explain their results for E and N because parental correlations were of similar magnitude to sibling and DZ twin correlations.

In some cases, especially with NS and PS, the correlations involving DZ twins or siblings were low enough to make at least some higher-order epistasis a likely interpretation. By this view, higher-order epistatic effects involving two (e.g., dominance-by-dominance epistasis) or more (e.g., additive-by-additive-by-additive epistasis) loci combine with additive genetic effects and/or common environmental effects, leading to the very low DZ or sibling

correlations (see Lykken *et al*., 1992). This would imply that the *A* and *D* parameter estimates in the current study were biased to the degree that such higher-order epistasis affected these eight personality traits. It might also mean that some degree of *C* was missed by assuming that its value was zero.

It is possible to visualize the full set of mathematically equally valid possible parameters estimated from twin designs such as the present one (Keller and Coventry, 2005). In order to estimate parameters, twin designs must assume that (a) either *C* or *NA* (non-additive genetic variation, of which *D* is one form) are equal to zero, and (b) the correlation between DZ twins and/or siblings that is due to genetic non-additivity is $\hat{r} = 0.25$ (which was assumed by modeling genetic non-additivity as dominance in the current study). However, it is perfectly plausible that *C* and *NA* simultaneously affect the phenotype, and it is also possible (likely even) that at least some epistasis causes $\hat{r} < 0.25$. Altering these two assumptions also alters the estimated parameters, allowing for a way to explore all the potential parameters (the *parameter space*) for a given trait.

Figure 1 presents the parameter spaces from the full models (models 1.1 in Appendices 1 and 2) for two personality dimensions, female NS and male P. We do not present the other 14 possible graphs for brevity, but the general point holds for these as well. The figure shows all the possible combinations of parameters *A*, *NA*, and *C* that are possible when $0.125 \leq \hat{r} \leq 0.25$ (values of $\hat{r}<0.125$ are increasingly biologically implausible; see Keller and Coventry, 2005; Eaves, 1988). These parameter spaces for male NS and female P give some indication of the degree of uncertainty that remains in parameter estimates from twin designs, irrespective of statistical power. They show that *A* may have been underestimated (e.g., *A* could be as high as 0.17 in female NS), and they also provide maximum values for how much *C* was missed by fixing it to zero (in general $C < 0.10$ for these 16 dimensions). Nevertheless, the evidence for some type of non-additive genetic variation, *NA*, is pervasive across every personality dimension.

## Implications

By using design better able to detect non-additive genetic variation, the present results call into question previous conclusions that most or all of the genetic variation in EPQ-R and TCI personality dimensions was additive. Our results indicate that much personality is 'genetic' in origin, but nevertheless not transmitted from parents to offspring because the genes that influence personality do so only in combinations with other genes. These combinations are unlikely to be shared between parents and offspring.

Our results could be relevant to gene mapping of personality dimensions. A recent systematic meta-analysis of the associations between candidate genes and personality scales (including the EPQ-R and TCI) found that none of the reported personality-gene associations across the 46 studies were replicated at rates above what would be expected from chance alone (Munafo *et al*., 2003). Variable findings in the literature seem to extend to linkage analysis of personality as well. If many of the genes underlying personality have non-additive effects, as our results suggests, quantitative trait loci (QTL) that have little marginal (i.e., additive) effect could have been missed in linkage and (especially) association analyses (Frankel and Schork, 1996; Purcell and Sham, 2004).

Explicitly modeling dominance and epistasis may therefore be prudent in analyses of personality. Because dominant and additive QTL effects are highly correlated (Mather, 1974), one approach in linkage analyses may be to test a full model that includes both additive and dominance effects against a nested model in which both effect have been dropped (a two degree of freedom test; Dolan *et al*., 1999). Alternatively, Wang and Huang (2002) introduced a score statistic for testing additive and dominant QTL that performs well in the presence of additive genetic effects while increasing power disproportionately in the presence of dominance. Epistatic effects can also be modeled in linkage analyses by fixing the effect of a region showing a large additive effect and including an additional free parameter for the interaction between the candidate region and all other regions (Li and Reich, 2000; Zhu *et al*., 2004). However, there is also doubt about whether it is advisable to model epistasis in linkage analyses even when it is present (Purcell and Sham, 2004).

A better understanding of the genetic architecture underlying personality also provides clues about the evolutionary origins of its underlying genetic variation. Specifically, high levels of non-additive genetic variation suggest that personality has probably not been neutral to selection, because neutral mutation plus random genetic drift tends to result in higher additive than non-additive genetic variation (Falconer and Mackay, 1996). This is seen, for example, in the very high narrow-sense heritability in finger ridge counts (Huntley, 1966).

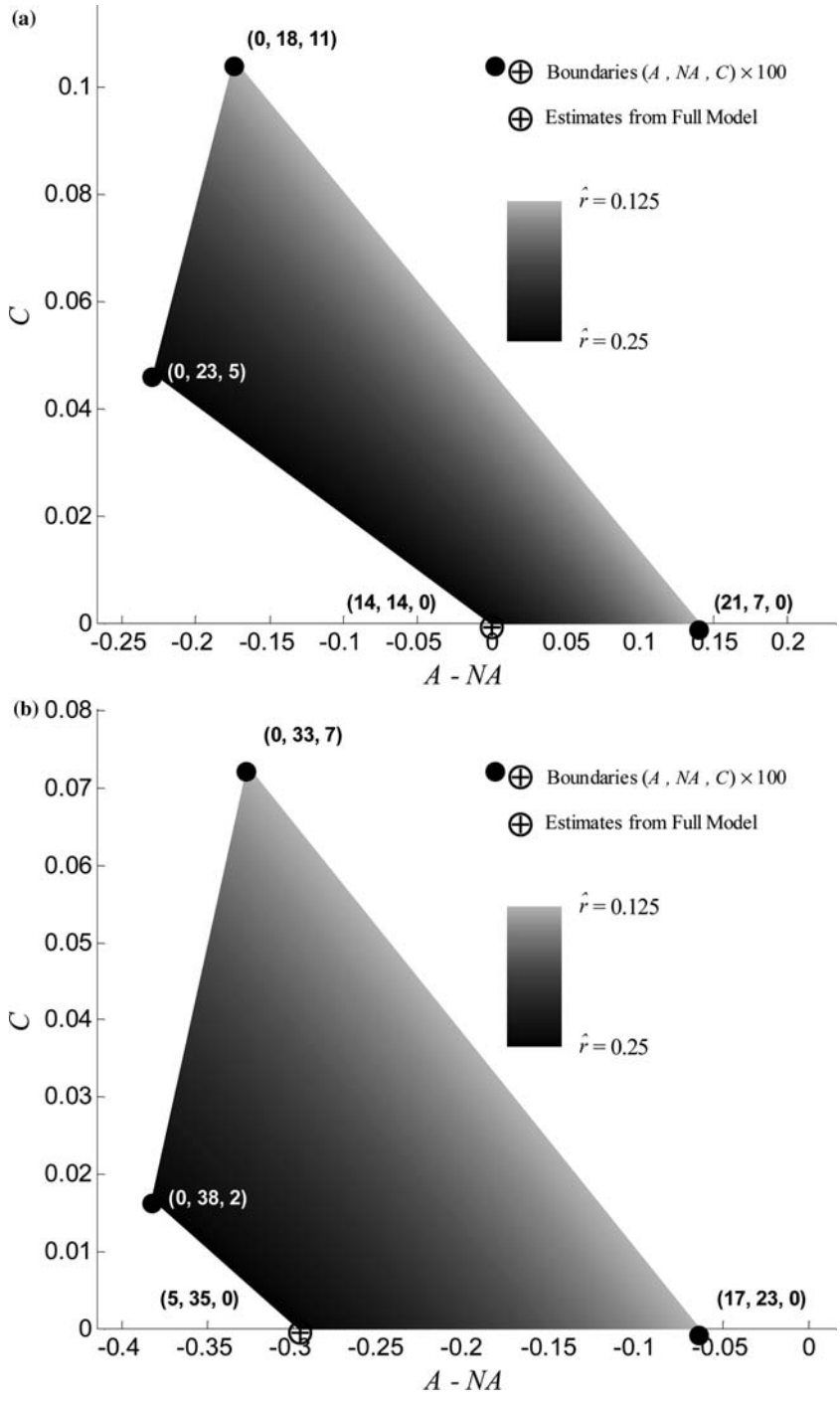


**Fig. 1.** Space of mathematically equally valid parameter values for male Psychoticism (a) and female NS (b).

Unfortunately, high non-additive genetic variation could be the result of two very different types of selection: mutation-selection or balancing selection. Mutation-selection would imply that personality traits are under stabilizing or directional selection, such that deviations away from some optimum are selected against. In this view, genetic variation underlying personality is maintained by a balance between the introduction of new deleterious mutations in the population and their eventual removal

(usually many generations later) by selection. A low ratio of additive to non-additive genetic variation is an expected outcome of strong directional or stabilizing selection due to the faster depletion of additive genetic variation (Fisher, 1930), an expectation corroborated by observations of low narrow-sense and higher broad-sense heritability in traits related to fitness in non-human animals (Merilä and Sheldon, 1999; Roff and Mousseau, 1987). However, little evidence for assortative mating in personality dimensions (Eaves *et al*., 1999) makes stabilizing selection more likely than directional selection.

Certain types of balancing selection, such as heterozygote advantage and antagonistic pleiotropy which leads to heterozygote advantage (see Curtsinger *et al*., 1994), also predict very high levels of non-additive genetic variation. According to these models, two alternative alleles are actively maintained by selection at one or more loci because (a) the marginal fitness effects of the alternative alleles are equal, and (b) the most-fit genotypes cannot 'breed true'. The classic example of this is the genetic polymorphism responsible for sickle-cell anemia. However, heterozygote advantage predicts dominance—not epistatic—variation, which is at odds with findings that parent-offspring correlations are similar to sibling correlations, at least for the EPQ-R personality dimensions (Eaves *et al*., 1998).

## SUMMARY

It is clear that genes play a large role in creating differences in the Cloninger and Eysenck personality dimensions. The studies that have had sufficient power to detect it have concluded that much of this genetic variation is non-additive in nature. However, the precise ways that additive, dominant, and epistatic effects combine to affect the phenotype remain to be elucidated. Given the present results, it is possible that explicitly modeling non-additive QTL effects may increase sensitivity for detecting genes that are associated with differences in human personality. Finally, while the evolutionary forces that have acted upon personality remain unclear, observations of high levels of non-additive genetic variation suggest that personality has not been invisible to natural selection in the ancestral past.

## APPENDIX

**Appendix Table I.** Results of Structural Equation Models of Genetic and Environmental Hypotheses for First Six Personality Dimensions

| Model | Dropped par. or constraint | $\Delta df$ | $\Delta 2LL$ | *Af* | *Df* | *Ef* | *Am* | *Dm* | *Em* | $A'$ or $\lambda$ |
|---|---|---|---|---|---|---|---|---|---|---|
| Harm Avoidance | | | | | | | | | | |
| 1.1 | | | | 0.19 | 0.23 | 0.58 | 0.02 | 0.21 | 0.56 | $A' = 0.21$ |
| **1.2 (vs. 1.1)** | ***Am*** | **1** | **1.1** | **0.15** | **0.27** | **0.58** | | **0.29** | **0.55** | **$A' = 0.16$** |
| 1.3 (vs. 1.1) | *A′* | 1 | 3.9* | 0.19 | 0.22 | 0.58 | 0.00 | 0.45 | 0.55 | |
| 2.1 (vs. 1.3) | *Df,m* | 2 | 44.6*** | 0.37 | | 0.63 | 0.36 | | 0.64 | |
| 2.2 (vs. 1.3) | *Af,m* | 2 | 8.9* | | 0.43 | 0.57 | | 0.44 | 0.56 | |
| 2.3 (vs. 1.3) | *Af,m,Df,m* | 4 | 511.2*** | | | 1.00 | | | 1.00 | |
| 3.1 (vs. 1.3) | $Vf = \lambda\ Vm$ | 2 | 7.3* | 0.06 | 0.36 | 0.58 | 0.06 | 0.36 | 0.58 | $\lambda = 1.10$ |
| 4.1 (vs. 3.1) | $\lambda = 1$ | 1 | 6.5* | 0.06 | 0.36 | 0.57 | 0.06 | 0.36 | 0.57 | |
| 4.2 (vs. 4.1) | *D* | 1 | 38.3*** | 0.36 | | 0.64 | 0.36 | | 0.64 | |
| 4.3 (vs. 4.1) | *A* | 1 | 1.5 | | 0.43 | 0.57 | | 0.43 | 0.57 | |
| 4.4 (vs. 4.1) | *A, D* | 2 | 511.1*** | | | 1.00 | | | 1.00 | |
| $\beta s = 0.42$***; $\beta a = 0.02$*; $\beta o = 0.01$; $\beta as = -0.04$ ; $\beta oa = -0.01$; $\beta os = -0.03$ | | | | | | | | | | |
| Persistence[b] | | | | | | | | | | |
| 1.1 | | | | 0.08 | 0.28 | 0.65 | 0.00 | 0.27 | 0.65 | $A' = 0.09$ |
| 1.2 (vs. 1.1) | *Am* | 1 | 0.0 | 0.08 | 0.28 | 0.65 | | 0.26 | 0.65 | $A' = 0.09$ |
| 1.3 (vs. 1.1) | *A′* | 1 | 0.5 | 0.06 | 0.29 | 0.65 | 0.01 | 0.35 | 0.64 | |
| 2.1 (vs. 1.3) | *Df,m* | 2 | 38.3*** | 0.29 | | 0.71 | 0.26 | | 0.74 | |
| 2.2 (vs. 1.3) | *Af,m* | 2 | 3.1 | | 0.35 | 0.65 | | 0.35 | 0.65 | |
| 2.3 (vs. 1.3) | *Af,m,Df,m* | 4 | 330.5*** | | | 1.00 | | | 1.00 | |
| **3.1 (vs. 1.3)** | ***$Vf = \lambda Vm$*** | **2** | **3.2** | **0.00** | **0.35** | **0.65** | **0.00** | **0.35** | **0.65** | **$\lambda = 1.09$** |
| 4.1 (vs. 3.1) | $\lambda = 1$ | 1 | 5.4* | 0.00 | 0.35 | 0.65 | 0.00 | 0.35 | 0.65 | |
| 4.2 (vs. 4.1) | *D* | 1 | 36.2*** | 0.28 | | 0.72 | 0.28 | | 0.72 | |
| 4.3 (vs. 4.1) | *A* | 1 | 0.0 | | 0.35 | 0.65 | | 0.35 | 0.65 | |
| 4.4 (vs. 4.1) | *A, D* | 2 | 330.5*** | | | 1.00 | | | 1.00 | |
| $\beta s = -0.10$***; $\beta a = -0.03$***; $\beta o = -0.03$***; $\beta as = 0.05$*; $\beta oa = 0.03$*; $\beta os = -0.01$ | | | | | | | | | | |

**Appendix Table 1.** (Continued)

| Model | Dropped par. or constraint | Δ*df* | Δ 2*LL* | *Af* | *Df* | *Ef* | *Am* | *Dm* | *Em* | *A′* or λ |
|---|---|---|---|---|---|---|---|---|---|---|
| Novelty Seeking | | | | | | | | | | |
| 1.1 | | | | 0.05 | 0.35 | 0.60 | 0.10 | 0.30 | 0.58 | *A′* = 0.02 |
| 1.2 (vs. 1.1) | *Am* | 1 | 1.6 | 0.00 | 0.40 | 0.60 | | 0.40 | 0.58 | *A′* = 0.03 |
| 1.3 (vs. 1.1) | *A′* | 1 | 0.9 | 0.03 | 0.37 | 0.60 | 0.12 | 0.30 | 0.58 | |
| 2.1 (vs. 1.3) | *Df,m* | 2 | 35.2*** | 0.33 | | 0.67 | 0.35 | | 0.65 | |
| 2.2 (vs. 1.3) | *A,m* | 2 | 0.9 | | 0.40 | 0.60 | | 0.43 | 0.57 | |
| 2.3 (vs. 1.3) | *Af,m,Df,m* | 4 | 450.3*** | | | 1.00 | | | 1.00 | |
| **3.1 (vs. 1.3)** | ***Vf* = λ*Vm*** | **2** | **0.7** | **0.05** | **0.35** | **0.60** | **0.05** | **0.35** | **0.60** | **λ = 1.14** |
| 4.1 (vs. 3.1) | *λ = 1* | 1 | 14.4*** | 0.05 | 0.35 | 0.59 | 0.05 | 0.35 | 0.59 | |
| 4.2 (vs. 4.1) | *D* | 1 | 36.0*** | 0.34 | | 0.66 | 0.34 | | 0.66 | |
| 4.3 (vs. 4.1) | *A* | 1 | 1.2 | | 0.41 | 0.59 | | 0.41 | 0.59 | |
| 4.4 (vs. 4.1) | *A, D* | 2 | 451.4*** | | | 1.00 | | | 1.00 | |
| βs = −0.07***; βa = −0.32***; βo = −0.00; βas = 0.09*** ; βoa = −0.02; βos = 0.01 | | | | | | | | | | |
| Reward Dependance[a,c] | | | | | | | | | | |
| 1.1 | | | | 0.08 | 0.30 | 0.62 | 0.02 | 0.06 | 0.65 | *A′* = 0.27 |
| **1.2 (vs. 1.1)** | ***Am*** | **1** | **0.4** | **0.07** | **0.31** | **0.62** | | **0.11** | **0.63** | **A′ = 0.24** |
| 1.3 (vs. 1.1) | *A′* | 1 | 0.6 | 0.08 | 0.30 | 0.62 | 0.31 | 0.04 | 0.65 | |
| 2.1 (vs. 1.3) | *Df,m* | 2 | 51.4*** | 0.31 | | 0.69 | 0.27 | | 0.73 | |
| 2.2 (vs. 1.3) | *Af,m* | 2 | 83.2*** | | 0.36 | 0.64 | | 0.30 | 0.70 | |
| 2.3 (vs. 1.3) | *Af,m,Df,m* | 4 | 295.0*** | | | 1.00 | | | 1.00 | |
| 3.1 (vs. 1.3) | *Vf* = λ*Vm* | 2 | 14.5*** | 0.01 | 0.36 | 0.63 | 0.01 | 0.36 | 0.63 | λ = 1.03 |
| 4.1 (vs. 3.1) | *λ = 1* | 1 | 0.7 | 0.01 | 0.36 | 0.63 | 0.01 | 0.36 | 0.63 | |
| 4.2 (vs. 4.1) | *D* | 1 | 37.9*** | 0.30 | | 0.70 | 0.30 | | 0.70 | |
| 4.3 (vs. 4.1) | *A* | 1 | 0.0 | | 0.37 | 0.63 | | 0.37 | 0.63 | |
| 4.4 (vs. 4.1) | *A, D* | 2 | 366.3*** | | | 1.00 | | | 1.00 | |
| βs = 0.60***; βa = −0.04***; βo = 0.00; βas = −0.08*** ; βoa = −0.01; βos = 0.01 | | | | | | | | | | |
| Extraversion[b] | | | | | | | | | | |
| 1.1 | | | | 0.31 | 0.14 | 0.55 | 0.15 | 0.18 | 0.49 | *A′* = 0.17 |
| 1.2 (vs. 1.1) | *Am* | 1 | 17.3*** | 0.14 | 0.32 | 0.54 | | 0.45 | 0.48 | *A′* = 0.07 |
| 1.3 (vs. 1.1) | *A′* | 1 | 3.6 | 0.34 | 0.11 | 0.56 | 0.14 | 0.37 | 0.48 | |
| 2.1 (vs. 1.3) | *Df,m* | 2 | 20.6*** | 0.42 | | 0.58 | 0.45 | | 0.55 | |
| 2.2 (vs. 1.3) | *Af,m* | 2 | 20.5*** | | 0.47 | 0.53 | | 0.52 | 0.48 | |
| 2.3 (vs. 1.3) | *Af,m,Df,m* | 4 | 692.9*** | | | 1.00 | | | 1.00 | |
| 3.1 (vs. 1.3) | *Vf* = λ*Vm* | 2 | 3.8 | 0.22 | 0.24 | 0.53 | 0.22 | 0.24 | 0.53 | λ = 1.03 |
| **4.1 (vs. 3.1)** | ***λ = 1*** | **1** | **0.0** | **0.23** | **0.24** | **0.53** | **0.23** | **0.24** | **0.53** | |
| 4.2 (vs. 4.1) | *D* | 1 | 18.3*** | 0.43 | | 0.57 | 0.43 | | 0.57 | |
| 4.3 (vs. 4.1) | *A* | 1 | 19.9*** | | 0.49 | 0.51 | | 0.49 | 0.51 | |
| 4.4 (vs. 4.1) | *A, D* | 2 | 683.5*** | | | 1.00 | | | 1.00 | |
| βs = 0.06**; βa = −0.20***; βo = −0.01; βas = −0.03 ; βoa = −.03***; βos = 0.04* | | | | | | | | | | |
| Neuroticism[c] | | | | | | | | | | |
| **1.1** | | | | **0.19** | **0.23** | **0.57** | **0.09** | **0.12** | **0.62** | ***A′* = 0.17** |
| 1.2 (vs. 1.1) | *Am* | 1 | 5.9* | 0.12 | 0.32 | 0.56 | | 0.31 | 0.61 | *A′* = 0.08 |
| 1.3 (vs. 1.1) | *A′* | 1 | 4.7* | 0.14 | 0.29 | 0.58 | 0.12 | 0.25 | 0.63 | |
| 2.1 (vs. 1.3) | *Df,m* | 2 | 21.3*** | 0.38 | | 0.62 | 0.31 | | 0.69 | |
| 2.2 (vs. 1.3) | *Af,m* | 2 | 6.6* | | 0.44 | 0.56 | | 0.39 | 0.61 | |
| 2.3 (vs. 1.3) | *Af,m,Df,m* | 4 | 501.5*** | | | 1.00 | | | 1.00 | |
| 3.1 (vs. 1.3) | *Vf* = λ*Vm* | 2 | 4.2 | 0.12 | 0.28 | 0.60 | 0.12 | 0.28 | 0.60 | λ = 1.19 |
| 4.1 (vs. 3.1) | *λ = 1* | 1 | 21.2*** | 0.12 | 0.29 | 0.59 | 0.12 | 0.29 | 0.59 | |
| 4.2 (vs. 4.1) | *D* | 1 | 23.5*** | 0.36 | | 0.64 | 0.36 | | 0.64 | |
| 4.3 (vs. 4.1) | *A* | 1 | 5.8* | | 0.42 | 0.58 | | 0.42 | 0.58 | |
| 4.4 (vs. 4.1) | *A, D* | 2 | 480.7*** | | | 1.00 | | | 1.00 | |
| βs = 0.35***; βa = −0.10**; βo = 0.01; βas = −0.05*; βoa = 0.01; βos = −0.05* | | | | | | | | | | |

*Note*: Best-fitting models by AIC shown in bold (see text). Subscripts *f,m* = female, male. *A* = additive genetic, *D* = dominance genetic, *E* = unique environmental, *A*' = male unique additive genetic, *V* = total variance, λ = scalar for male variance, and βs – βos = standardized beta-coefficients for sex (0 = *m*, 1 = *f*), age, birth order, and their interactions..

[a] MZ and DZ intercepts estimated independently, [b] Twin and sibling intercepts estimated independently, [c] Twin and sibling variances estimated independently..

*$p < 0.05$, **$p < 0.01$, ***$p < 0.001$.

**Appendix Table 2.** Results of Structural Equation Models of Genetic, Environmental, and Special Twin Environment Hypotheses for L and P

| Model | Dropped par. or constraint | $\Delta df$ | $\Delta 2LL$ | *Af* | *Df* | *Sf* | *Ef* | *Am* | *Dm* | *Sm* | *Em* | $A'$ or $\lambda$ |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Lie[a,b] | | | | | | | | | | | | |
| 1.1 | | | | 0.26 | 0.10 | 0.08 | 0.56 | 0.28 | 0.00 | 0.04 | 0.68 | $A'=0.00$ |
| 1.2 (vs. 1.1) | *Am* | 1 | 30.5*** | 0.06 | 0.31 | 0.08 | 0.54 | | 0.29 | 0.04 | 0.65 | $A'=0.00$ |
| 1.3 (vs. 1.1) | $A'$ | 1 | 0.0 | 0.26 | 0.10 | 0.08 | 0.56 | 0.28 | 0.00 | 0.04 | 0.68 | |
| 1.4 (vs. 1.3) | *Sf,m* | 2 | 11.2** | 0.30 | 0.14 | | 0.56 | 0.31 | 0.00 | | 0.68 | |
| **2.1 (vs. 1.3)** | ***Df,m*** | **2** | **2.7** | **0.34** | | **0.09** | **0.57** | **0.27** | | **0.06** | **0.69** | |
| 2.2 (vs. 1.3) | *Af,m* | 2 | 31.6*** | | 0.37 | 0.09 | 0.54 | | 0.29 | 0.06 | 0.65 | |
| 2.3 (vs. 1.3) | *Af,m,Df,m,Sf,m* | 6 | 543.1*** | | | | 1.00 | | | | 1.00 | |
| 3.1 (vs. 1.3) | $Var(f)=\lambda Var(m)$ | 3 | 17.7*** | 0.26 | 0.07 | 0.07 | 0.61 | 0.26 | 0.07 | 0.07 | 0.61 | $\lambda=1.14$ |
| 4.1 (vs. 3.1) | $\lambda=1$ | 1 | 13.6*** | 0.26 | 0.07 | 0.06 | 0.61 | 0.26 | 0.07 | 0.06 | 0.61 | |
| 4.2 (vs. 4.1) | *S* | 1 | 9.8** | 0.30 | 0.09 | | 0.61 | 0.30 | 0.09 | | 0.61 | |
| 4.3 (vs. 4.1) | *D* | 1 | 1.3 | 0.31 | | 0.07 | 0.62 | 0.31 | | 0.07 | 0.62 | |
| 4.4 (vs. 4.1) | *A* | 1 | 28.9*** | | 0.35 | 0.08 | 0.58 | | 0.35 | 0.08 | 0.58 | |
| 4.5 (vs. 4.1) | *A, D, S* | 3 | 519.3*** | | | | 1.00 | | | | 1.00 | |
| $\beta s=0.24$***; $\beta a=0.29$***; $\beta o=-0.02$; $\beta as=-0.03$ ; $\beta oa=-0.02$*; $\beta os=-0.01$ | | | | | | | | | | | | |
| Psychoticism[a, b] | | | | | | | | | | | | |
| 1.1 | | | | 0.19 | 0.04 | 0.10 | 0.67 | 0.14 | 0.14 | 0.06 | 0.66 | $A'=0.00$ |
| 1.2 (vs. 1.1) | *Am* | 1 | 9.9** | 0.06 | 0.19 | 0.09 | 0.65 | | 0.32 | 0.04 | 0.64 | $A'=0.00$ |
| 1.3 (vs. 1.1) | $A'$ | 1 | 0.0 | 0.19 | 0.04 | 0.10 | 0.67 | 0.14 | 0.14 | 0.06 | 0.66 | |
| 1.4 (vs. 1.3) | *Sf,m* | 2 | 22.6*** | 0.24 | 0.07 | | 0.67 | 0.04 | 0.30 | | 0.66 | |
| **2.1 (vs. 1.3)** | ***Df,m*** | **2** | **2.4** | **0.22** | | **0.11** | **0.68** | **0.24** | | **0.04** | **0.68** | |
| 2.2 (vs. 1.3) | *Af,m* | 2 | 11.1** | | 0.25 | 0.10 | 0.65 | | 0.32 | 0.04 | 0.64 | |
| 2.3 (vs. 1.3) | *Af,m,Df,m,Sf,m* | 6 | 348.0*** | | | | 1.00 | | | | 1.00 | |
| 3.1 (vs. 1.3) | $Var(f)=\lambda Var(m)$ | 3 | 21.1*** | 0.10 | 0.18 | 0.05 | 0.67 | 0.10 | 0.18 | 0.05 | 0.67 | $\lambda=0.93$ |
| 4.1 (vs. 3.1) | $\lambda=1$ | 1 | 3.1 | 0.10 | 0.18 | 0.05 | 0.67 | 0.10 | 0.18 | 0.05 | 0.67 | |
| 4.2 (vs. 4.1) | *S* | 1 | 6.6* | 0.12 | 0.21 | | 0.67 | 0.12 | 0.21 | | 0.67 | |
| 4.3 (vs. 4.1) | *D* | 1 | 9.1** | 0.23 | | 0.06 | 0.71 | 0.23 | | 0.06 | 0.71 | |
| 4.4 (vs. 4.1) | *A* | 1 | 4.0* | | 0.29 | 0.06 | 0.66 | | 0.29 | 0.06 | 0.66 | |
| 4.5 (vs. 4.1) | *A, D, S* | 3 | 334.3*** | | | | 1.00 | | | | 1.00 | |
| $\beta s=-0.41$***; $\beta a=-0.21$***; $\beta o=-0.00$; $\beta as=0.10$*** ; $\beta oa=-0.01$; $\beta os=0.01$ | | | | | | | | | | | | |

*Note*: *S* = special twin environment. See Table V for descriptions of other abbreviations.
*$p<0.05$, **$p<0.01$, ***$p<0.001$.

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