Genetic and environmental influences on extreme personality dispositions in adolescent female twins

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Background: The objective was to determine whether the pattern of environmental and genetic influences on deviant personality scores differs from that observed for the normative range of personality, comparing results in adolescent and adult female twins. **Methods:** A sample of 2,796 female adolescent twins ascertained from birth records provided Junior Eysenck Personality Questionnaire data. The average age of the sample was 17.0 years (S.D. 2.3). Genetic analyses of continuous and extreme personality scores were conducted. Results were compared for 3,178 adult female twins. **Results:** Genetic analysis of continuous traits in adolescent female twins were similar to findings in adult female twins, with genetic influences accounting for between 37% and 44% of the variance in Extraversion (Ex), Neuroticism (N), and Social Non-Conformity (SNC), with significant evidence of shared environmental influences (19%) found only for SNC in the adult female twins. Analyses of extreme personality characteristics, defined categorically, in the adolescent data and replicated in the adult female data, yielded estimates for high N and high SNC that deviated substantially ($p < .05$) from those obtained in the continuous trait analyses, and provided suggestive evidence that shared family environment may play a more important role in determining personality deviance than has been previously found when personality is viewed continuously. However, multiple-threshold models that assumed the same genetic and environmental determinants of both normative range variation and extreme scores gave acceptable fits for each personality dimension. **Conclusions:** The hypothesis of differences in genetic or environmental factors responsible for N and SNC among female twins with scores in the extreme versus normative ranges was partially supported, but not for Ex. **Keywords:** Personality, genetic analyses, twin studies, internalizing, externalizing.

Genetic influences on normal personality variation have been consistently demonstrated in twin studies of adult twin pairs reared together (Eaves, Eysenck, & Martin, 1989; Heath, Cloninger, & Martin, 1994) and apart (Tellegen et al., 1988), as well as in adoption samples (Scarr, Webber, Weinberg, & Wittig, 1981). The comparability of genetic influences on personality in adolescence versus adulthood has received relatively less attention (but see Young, Eaves, & Eysenck, 1980). While there are competing theories concerning the number and types of dimensions that best describe personality differences (e.g., Eysenck & Eysenck, 1969, 1975; McCrae & Costa, 1989; Tellegen, 1985), the dimensions of extraversion (Ex), neuroticism (N), psychoticism (P), and social non-conformity (SNC, also known as the 'Lie Scale' with reversed scoring), as proposed by Eysenck and Eysenck (1969), are well known to personality researchers and have been most extensively investigated across both adult (Eaves et al., 1989) and adolescent (Young et al., 1980; Macaskill, Hopper, White, & Hill, 1994) samples.

The facets of personality of interest to many clinical researchers are extreme manifestations of personality traits commonly associated with presenting psychopathology (Cloninger, 1987; Maziade, Caron, Cote, Boutin, & Thivierge, 1990). A common etiological basis between extreme levels of personality and psychopathology is frequently hypothesized. For example, a common genetic liability between measures of personality and psychopathology, such as neuroticism and major depression, has received some support (Jardine, Martin, & Henderson, 1984; Kendler, Neale, Kessler, Heath, & Eaves, 1993). Phenotypic overlap between normal personality dimensions and personality disorders has more extensive support (Widiger & Costa, 1994; Reynolds & Clark, 2001) with evidence that personality disorders may represent extreme personality functioning (Miller, Lyam, Widiger, & Leukefeld, 2001). Genetic covariation between personality disorders and normal personality functioning has been observed in adults (Livesley, Jang, & Vernon, 1998; Markon, Krueger, Bouchard, & Gottesman, 2002). However, the relevance of the study of normal personality variation in adolescence to understanding the causes of psychopathology remains uncertain. Most behavioral genetic studies of normal personality have been conducted within a framework that assumes continuity of genetic and environmental influences on personality variation within the normal range, and on more extreme personality scores.

However, this critical assumption of continuity has remained untested. More specifically, while the underlying genetic and environmental architectures of continuous measures of personality have received much attention in both adolescence and adulthood, investigation of genetic and environmental influences on deviant levels of normal personality is lacking.

Given the possibility of gene–environment correlations (rGE) and gene by environment interactions (G × E) associated with personality differences, there is reason to consider the likelihood that the combined effects of genetic and environmental influences on personality may be nonlinear in scope (Scarr & McCartney, 1983; Eaves, Last, Martin, & Jinks, 1977). Personality may be expressed within the context of three different types of rGE: passive, evocative and active (Scarr & McCartney, 1983). In the case of passive rGE, children inherit both the genotype of their parents in addition to being exposed to the environment provided by their parents (Scarr & McCartney, 1983). For example, more extroverted parents may expose their child (with a genetic tendency towards high extroversion) to more social opportunities, and a positive rGE would result. In the case of an evocative rGE, an individual's genetic constitution elicits a specific pattern of response from their environment (Scarr & McCartney, 1983). For instance, a more highly extroverted child might elicit more social responsiveness from others and thus reinforce their propensity towards socially oriented activity. Finally, individuals are thought to engage in 'niche-building' behavior more and more as they age, in other words active rGEs begin to predominate over passive rGEs (Scarr & McCartney, 1983). During this process, heritability is hypothesized to increase as active rGE increases (Scarr & McCartney, 1983), as most individuals choose environments that are less likely to restrict expression of their genetic propensity. More extroverted young adults may, for example, begin extending their social network as the move away from home. To complicate the matter further, these different forms of rGE are likely to occur in parallel with G × Es. If we take the example of an evocative rGE for the trait of extroversion, genetic and environmental influences might differ in extreme cases compared to scores within the normal range, if individuals at the extremes of extroversion evoke more consistent interpersonal reactions (Eaves et al., 1977). For instance individuals who are extremely extroverted on one hand, or shy on the other hand, may both elicit reliable avoidance behavior in those around them. In this scenario, extreme manifestations of personality may be associated with less environmental variance and proportionality more genetic variability, providing evidence for G × E (Eaves et al., 1977). Taken together, given the possible complex interplay between an individual's personality and the environment, the likelihood of non-linear genetic and environmental influences across the range of personality scores must be considered.

The goals of this study were to determine 1) whether estimates of the importance of genetic factors at the extreme low and at the extreme high (i.e., the more deviant) levels of personality are consistent with those obtained using continuous personality measures, using Eysenckian measures of Ex, N and SNC; 2) whether the assumption of continuity of genetic effects throughout the range of personality variation is supported; and 3) whether the role of genetic factors for these measures of personality is consistent between adolescent and adult twin pairs. Analyses were conducted using self-report data from a large Midwestern US female adolescent twin sample (Heath et al., 1999, 2002), with attempted replication of findings using previously published data from an Australian adult female twin sample (Heath et al., 1994).

Method

Participants

Twins from the Missouri Adolescent Female Twin Study (MOAFTS) were ascertained from state birth records, for an ongoing prospective study of alcohol use disorders and comorbid psychopathology (Heath et al., 1999, 2002). The target sample for the questionnaire phase of the study comprised 4,738 like-sex female twins, born between July 1, 1975 and June 30, 1985 and aged at least 13 years at the time of the study. Using a cohort-sequential design, twins were targeted for assessment at ages 13.5, 15.5, 17 and 19 years. Zygosity was determined using a standardized assessment by parental interview, which shows approximately 95% agreement with genotyping methods (Eaves et al., 1989). Initial interviews were completed with 3,258 female adolescent twins from these cohorts (69% of individual twins targeted). Questionnaire data were obtained from 2,796 twins who were eligible for and completed a one-year follow-up assessment (732 complete MZ pairs, 536 complete DZ pairs, 260 singletons). At the time of the one-year follow-up the age distribution of the female twins was as follows: 13 (.07%), 14 (16.6%), 15 (17.8%), 16 (9.4%), 17 (14.3%), 18 (13.8%), 19 (6.9%), 20 (12.7%), 21 (7.7%), 22 (.7%), 23 (.07%). The average age of the sample at one-year follow-up was 17.1 years (S.D. 2.3). Compared to US 1990 census data for family residence at the time of birth, this sample appears to be largely representative of families in Missouri with twins (Heath et al., 2002).

The personality data provided by this adolescent sample were compared to adult personality data provided by adult females participating in a volunteer twin register maintained by the Australian National Health and Medical Research Council (NH&MRC). A total of 5,967 twin pairs were targeted for a mailed questionnaire survey in 1980–1981 (Martin & Jardine, 1986; Eaves et al., 1989; Heath, Madden, Slutske, & Martin, 1995). In 1989, at ages 25–89, they received a follow-up survey that included some of the same personality measures collected from the Missouri

adolescent twins. We had full personality data on 1,996 MZ (946 complete pairs, 104 singletons) and 1,182 DZ (541 complete pairs, 100 singletons) adult female twins from this sample ($N = 3,178$ individual twins).

Measures

The adolescent questionnaire contained self-report personality scales, in addition to other questions concerning health-related behaviors. Among these personality measures were a subset of items ($n = 30$) from the following scales: Extraversion (Ex), Neuroticism (N), and Social-Non-Conformity (SNC) from the Junior Eysenck Personality Questionnaire (JEPQ; Eysenck & Eysenck, 1975). Extraversion measures levels of sociability, activity and optimism. Introversion is hypothesized to represent the lower tail of Ex's continuum: unsociable, passive and quiet. N assesses emotional instability, restlessness, and low self-regard. Like Ex, N is also viewed as continuous with low levels representing emotional stability, calmness, and reliability. SNC gauges the degree to which individuals are frank, disobedient, and have difficulty responding to social contingencies (this scale, reverse coded, is referred to as the Lie Scale by Eysenck & Eysenck, 1975). The Lie Scale has been reversed to ease interpretation of findings. For instance, Slutske et al. (2002) found in principal components analysis of personality variables that the reversed Lie Scale loaded positively with items measuring behavioral under-control (e.g., novelty seeking item), in addition to being both phenotypically and genetically positively associated with alcohol dependence and conduct disorder. In other studies, the Lie Scale shows consistent negative correlations with attitudes towards substance use (Francis, 1996, 1997) and conduct-related behavior such as being a bully (Mynard & Joseph, 1997). Items measuring Psychoticism (P) were not included in the assessment, based on previous findings that P's phenotypic factor structure differed markedly from the complex underlying genetic factor structure, which led to uninterpretable genetic findings for the P scale score (Heath & Martin, 1990). Phenotypic heterogeneity of the P scale has also been commented on by others (Bishop, 1977; Block, 1977a, 1977b). Such heterogeneity has not been observed for the other scales of the EPQ (Heath & Martin, 1990).

The adult female Australian twin personality data considered in this study were derived from an assessment that was comparable to that used with the adolescents. Data were obtained in a follow-up questionnaire survey conducted in 1989, which included the short-form 48-item revised Eysenck Personality Questionnaire (EPQ-R; Eysenck & Eysenck, 1985). See Heath et al. (1994) for psychometric findings on this sample. Findings comparing the consistency of scores of the JEPQ and EPQ-R in a sample of adolescents found moderate to high reliability estimates (E: .73; N: .78, SNC: .65; Francis & Pearson, 1988).

Statistical approach

Descriptive analyses. In our analyses, each personality variable was first considered as a continuous trait; then, extreme internalizing and externalizing scores were derived. Internalizing traits were defined as low Ex, high N, and low SNC, and externalizing traits were defined as high Ex, low N, and high SNC. Binary variables were created to code for the extreme ranges of the distribution for each measure of personality: below the 25th %tile (No = 0, Yes = 1), and above the 75th %tile (No = 0, Yes = 1). Because of the small number of items for each scale, 25th and 75th %tiles could only be approximated.

Each of the personality scales was subjected to descriptive analyses, including regression diagnostics, exploratory factor analysis, and reliability estimates using SAS (1999). Summary twin pair product moment correlations were computed for continuous scale scores using SAS (1999), and tetrachoric correlations for the binary measures using Mx (Neale, Boker, Xie, & Maes, 2002).

Genetic analyses. Univariate genetic models (Eaves, Last, Young, & Martin, 1978; Neale et al., 2002) were fitted for each continuous and categorical measure by the method of maximum likelihood using the program Mx (Neale et al., 2002). We estimated the proportion of the total variance in each personality measure that could be explained by additive genetic factors (A), environmental influences shared by members of a twin pair (C), and nonshared environmental influences (E). When shared environmental influences were estimated at zero, non-additive genetic influences (D) were estimated. Nonadditive genetic effects and shared environmental effects are confounded in twins reared together, respectively leading to DZ twin correlations less than one half the MZ correlations, and inflation of the DZ correlation to be greater than one half the MZ correlation (Martin, Eaves, Kearsey, & Davies, 1978). As such, nonadditive genetic effects and shared environmental effects could not be included in the same models.

For the categorical measures of personality, estimates were derived under a multifactorial threshold model that assumes that a continuous normal liability distribution underlies the observed binary trait of absence/presence of each extreme personality trait. Under this model, the distributions of twin pairs for these latent liability variables are also assumed to be bivariate normal. The same standard assumptions are used to estimate tetrachoric and polychoric correlations (Tallis, 1962; Joreskog & Sorbom, 1993) and appear plausible for multifactorial personality constructs (Heath et al., 1994). Univariate multiple-threshold models were fitted to 6×6 (<10th %tile, 10th–<25th %tile, 25th %tile–<50th %tile, 50th %tile–<75th %tile, 75th %tile–<90th %tile, ≥90th %tile) contingency tables for each personality trait, in order to examine the fit of a single liability dimension model to the observed twin pair data. This 10th %tile cut-off was the most deviant we could consider without obtaining cells with zero sample size. For binary analyses, the 25th %tile was preferred, since the loss of statistical power was minimized. These analyses were repeated with a model equating off-diagonal elements of the like-sex two-way contingency tables; equivalent to fitting models to the observed numbers of concordant and discordant twins for each combination of trait values. This procedure would maximize our power to detect discontinuity of

genetic and environmental effects, since contributions of within-pair mean differences (i.e., differences in threshold) to the fit of the model are eliminated. A poor fit of the univariate threshold model for a given trait would suggest either discontinuity of underlying genetic and environmental influences, or (less plausibly) violation of the assumption of an underlying normal liability distribution. A multiple-threshold model-fitting approach was applied in place of the DeFries–Fulker (DF) extremes analysis (DeFries & Fulker, 1985, 1988) for two principal reasons: 1) vulnerability of the DF method to artifactual findings due to heteroscedasticity, a particular problem since we are using short scales; 2) the DF extremes analysis does not test whether a single genetic liability dimension exists across the personality range. A model-fitting approach avoids these problems.

Continuous models were fitted to covariance matrices, and multiple threshold models to contingency tables, both providing overall chi-square goodness of fit statistics. Binary models, for extreme personality scores, were fitted to raw data with control for the probit regression of personality variables on age, with the fit of nested models compared by likelihood ratio chi-square test. Likelihood-based 95 percent confidence intervals were computed for estimates of genetic and environmental parameters (Neale et al., 2002). The full ACE models were estimated first and compared against reduced models by likelihood-ratio chi-square, to determine the best-fitting model.

Results

Reliabilities for adolescent data

Regression diagnostics suggested that distributions for the three personality scales were approximately normal (all skewness and kurtosis values ranged between −1.0 and 1.0). Exploratory factor analysis with promax rotation yielded item loadings consistent with the factor structures predicted by Eysenck and Eysenck (1975). Ex items had loadings in the range of .3 to .7 on the Ex factor, with cross-loadings on the other factors being uniformly small (N: .01–.14, SNC: .01–.11). N items had loadings in the range of .42–.64 on the N factor, with cross-loadings on the other factors being small (Ex: .02–.12, SNC: .01–.11). For SNC, the items had loadings in the range of .43–.58 on the SNC factor, with cross-loadings on the other factors also uniformly small (Ex: .03–.06, N: .01–.04). The internal consistency reliabilities as measured by Cronbach's alpha ranged from moderate to good (.76 for Ex, .81 for N, and .64 for SNC).

Intraclass and tetrachoric correlations: adolescent twins

Table 1 shows the adolescent intraclass twin-pair correlations for each of the continuous personality scores, and for the categorical measures. For all of the continuous personality traits, greater MZ versus DZ twin-pair correlations and an approximately 2:1 ratio of MZ to DZ correlations suggest important genetic influences with minimal influences of shared family environment. However, the observed twin correlations for high N (internalizing trait) and high SNC and high Ex (externalizing traits) suggest a possible shared environmental influence.

Continuous trait analyses

Table 2 presents the results of univariate biometrical model-fitting analyses in adolescent females for each continuous scale. Included in the table are standardized maximum likelihood estimates of additive genetic (A), shared environmental (C), and nonshared environmental influences (E) and their 95% confidence intervals. For all continuous personality measures presented in Table 2, the models fit the data well (Ex: $X_3^2 = 3.91$; N: $X_3^2 = 1.11$; SNC: $X_3^2 = 4.36$; $p > .10$ in all cases). The best fitting models were AE models, i.e., C could be dropped (Ex: $X_1^2 = .00$; N: $X_1^2 = .04$; SNC: $X_1^2 = .49$; compared to the full model: $p > .10$ in all cases), but A could not be dropped without a significant worsening of model fit ($p < .05$ in all cases). Non-additive genetic influences were also tested for each personality scale, but the ADE model in no case provided a significantly better fit to the data when compared to the more parsimonious AE model ($p > .10$ in all cases, data not shown). Parallel results for the continuous traits in the Australian adult sample are shown in Table 3. Continuous measures of Ex and N were best fit by

Table 1 Intraclass and tetrachoric correlations (and 95% confidence intervals) for continuous and extreme personality measures, respectively, by zygosity in adolescent female twins

Zygosity	Continuous: Extraversion	Continuous: Neuroticism	Continuous: Social Non-Conformity
MZ (732 pairs)	.37 (.31–.43)	.44 (.38–.49)	.46 (.40–.51)
DZ (536 pairs)	.17 (.09–.25)	.23 (.16–.30)	.24 (.16–.31)
	Internalizing: Low Ex	Internalizing: High N	Internalizing: Low SNC
MZ (732 pairs)	.45(.34–.55)	.44 (.31–.56)	.57 (.48–.65)
DZ (536 pairs)	.24 (.09–.39)	.36 (.21–.49)	.22 (.09–.34)
	Externalizing: High Ex	Externalizing: Low N	Externalizing: High SNC
MZ (732 pairs)	.20 (.04–.36)	.50 (.40–.59)	.52 (.38–.64)
DZ (536 pairs)	.17 (−.03–.36)	.23 (.09–.36)	.40 (.22–.56)

Table 2 Biometrical model fitting results: standardized maximum-likelihood variance estimates (and 95% confidence intervals) for continuous personality scales and for extreme internalizing and externalizing measures in adolescent female twins

	Variance components		
	A	C	E
Continuous:			
Extraversion	.37 (.18–.43)	.00 (.00–.17)	.63 (.57–.69)
Neuroticism	.44 (.26–.51)	.00 (.00–.17)	.54 (.49–.60)
Social Non-Conformity	.40 (.21–.51)	.06 (.00–.22)	.54 (.49–.60)
Internalizing:			
Low Extraversion	.40 (.04–.54)	.04 (.00–.35)	.56 (.46–.67)
High Neuroticism	.20 (.00–.55)	.25 (.00–.48)	.55 (.43–.68)
Low Social Non-Conformity	.55 (.35–.63)	.00 (.00–.17)	.45 (.37–.54)
Externalizing:			
High Extraversion	.07 (.00–.35)	.12 (.00–.30)	.81 (.65–.95)
Low Neuroticism	.49 (.20–.58)	.00 (.00–.24)	.51 (.42–.61)
High Social Non-Conformity	.24 (.00–.62)	.28 (.00–.55)	.48 (.36–.62)

A = additive genetic influences; C = shared environmental influences; E = unique environmental influences.

Table 3 Biometrical model fitting results: maximum-likelihood variance estimates (and 95% confidence intervals) for continuous and for extreme internalizing and externalizing personality measures in same-sex adult female twins

	Variance components		
	A	C	E
Continuous:			
Extraversion	.47 (.35–.52)	.00 (.00–.11)	.53 (.48–.58)
Neuroticism	.45 (.29–.50)	.00 (.00–.15)	.55 (.50–.60)
Social Non-Conformity	.29 (.13–.46)	.19 (.04–.33)	.52 (.47–.57)
Internalizing:			
Low Extraversion	.60 (.44–.68)	.00 (.00–.13)	.40 (.32–.49)
High Neuroticism	.24 (.00–.51)	.18 (.00–.43)	.58 (.49–.68)
Low Social Non-Conformity	.47 (.14–.57)	.00 (.00–.29)	.53 (.43–.65)
Externalizing:			
High Extraversion	.51 (.20–.59)	.00 (.00–.27)	.49 (.41–.59)
Low Neuroticism	.50 (.19–.59)	.00 (.00–.26)	.50 (.42–.59)
High Social Non-Conformity	.06 (.00–.45)	.43 (.10–.57)	.51 (.40–.61)

A = additive genetic influences; C = shared environmental influences; E = unique environmental influences.

AE models ($X_1^2 = .00$, $p > .10$). In contrast to findings for the adolescent sample, SNC was best described by the full ACE model, i.e., estimates of both A and C were significant and could not be dropped without a significant worsening of model fit ($p < .05$ in both cases).

Multiple threshold models, which assume continuity of genetic and environmental influences across the range of personality variation, in all cases gave acceptable fits to the data: goodness-of-fit chi-square (X^2) statistics that were obtained were: adolescents: Ex: $X_{58}^2 = 58.17$, $p = .47$; N: $X_{58}^2 = 55.19$, $p = .58$; SNC: $X_{58}^2 = 60.25$, $p = .39$, and adults: Ex: $X_{58}^2 = 72.27$, $p = .10$; N: $X_{58}^2 = 63.74$, $p = .28$; SNC: $X_{58}^2 = 48.46$, $p = .81$. When we equated off-diagonal elements of the two-way contingency tables, there was only one case where the single liability dimension model failed to fit the data (Ex in the Australian adult women: $X_{28}^2 = 51.86$, $p = .004$). Heritability estimates obtained from the multiple-threshold models (adolescents: Ex: 38%, N: 37%, SNC: 47%; adults: Ex: 48%, N: 42%, SNC: 28%) closely approximated those obtained in the continuous trait analyses.

Extreme (deviant) measures

Tables 2 and 3 also show point estimates and their 95% confidence intervals for categorical measures of deviant personality scores on each of the three hypothesized internalizing and three hypothesized externalizing traits. A lower than anticipated estimate of the genetic variance was obtained for high Extraversion, but this finding was limited to the adolescent sample, and was associated with an extremely high estimate of nonshared environmental variance (81%), suggesting a problem with heteroscedasticity for Ex in this age group. For high and low Neuroticism in the adolescent twin series, and replicated in the Australian adult sample, nonshared environmental variance estimates did not differ from those obtained from the continuous trait analyses (i.e., no evidence for heteroscedasticity was found). For high Neuroticism, however, a substantial point estimate for the shared environmental variance was obtained for each sample (25%; 18%). Point estimates of genetic and shared environmental variances lay completely outside the range predicted by the 95%

confidence interval for the continuous trait analyses. In the adolescent data, the probability of observing a shared environmental variance as high as 25% was $p = .003$ for the continuous trait analysis. Correspondingly, the probability of observing a genetic variance as low as 20% was $p = .01$. For the Australian data, corresponding probabilities of observing a shared environmental variance as high as 18%, and a genetic variance as low as 24%, were $p = .02$ and $p = .01$. A similar pattern was observed for high SNC where substantially higher estimates for shared environmental variance were obtained than would be predicted from the 95% confidence intervals from the continuous trait analysis (adolescent data: 28%, $p < .008$; adult twin data: 43%, $p = .0004$), and the genetic variance in the Australian sample was again unexpectedly low (6%, $p = .005$). A combined analysis, modeling data from both adolescent and adult data sets, confirmed evidence (albeit marginally significant in the case of high N) for shared environmental influences on both high N (22%, 95%CI: 1–42) and high SNC (39%, 95%CI: 15–56) with no evidence for heterogeneity of genetic and environmental parameters across adolescent versus adult samples (high N: $X_3^2 = .36$, $p = .95$; $X_3^2 = .76$, $p = .86$).

Discussion

Continuity of genetic and environmental influences on personality differences throughout the range of personality variation has generally remained an untested assumption of behavioral genetic studies of personality. Yet any discontinuities in the magnitude of genetic or shared environmental influences would be of considerable potential clinical significance. Our results suggest that this assumption needs to be examined more carefully.

When traits were defined as continuous measures, genetic analyses of the Eysenckian personality traits of Extraversion, Neuroticism, and Social Non-Conformity assessed in a cohort of adolescent female twins were similar to findings in adult female twins from Australia (Heath et al., 1994), in that models that included additive genetic and unique environmental effects fit the data best, and there was no evidence for shared environmental influences. In these adolescent girls, additive genetic influences accounted for 37%, 44%, and 40% of the variance in Ex, N, and SNC respectively. Estimated shared environmental variances were small and non-significant (0–6%). The only exception to this pattern was the finding of significant shared environmental influence (19%) on SNC in the like-sex adult Australian female twins. This pattern of moderate heritability estimates with zero shared environmental variance estimated for Ex and N, but suggestive evidence for shared environmental influence on SNC, has been consistent across other studies of personality variation (e.g., Eaves et al., 1989).

However, for two deviant personality measures, high SNC and high N, evidence suggestive of shared environmental variance was obtained in both the adolescent and adult female twin samples. This did not appear to represent a developmental difference, since similar positive point estimates were obtained for high N and high SNC in both samples, and estimates could be equated across the adolescent and adult samples. When we compared the model fit of full models applied to continuous measures of N and SNC versus models that fixed the shared environmental estimates to those derived from deviant score analyses of high N and high SNC, we confirmed that estimates obtained in the deviant score analyses were highly unlikely (p-values in the range .02–.0004).

Several factors could potentially explain this inconsistency. For both high SNC and high N we could exclude the possibility of a problem with differences in error variance at different points on these scales: nonshared environmental variances (which would include error variance) were quite consistent between deviant score and continuous trait analyses. If scores on SNC or N were in fact determined by two more underlying latent factors ('factorial complexity'), having different modes of inheritance, this also might produce the observed differences in results between continuous trait and categorical analyses. However, to the extent that such factorial complexity is operating throughout the range of scale scores, we would expect to have sufficient power in the multiple threshold analysis to be able to reject the hypothesis of a single normal liability dimension. We consider it most likely that there are indeed additional factors contributing to variation in extreme scores that are not operating throughout the full range of personality variation. One limitation of the multiple threshold model for detecting such effects would be the lack of power, with twin-pair cell sizes becoming increasingly small towards the extremes of the distribution. We had 51% power to detect significant shared environmental influences on high SNC in the adolescent females, and 77% power to detect it in the adult females, assuming shared environmental influences accounted for 30% and 40% of the variance respectively in adolescent and adult females, for a trait with an MZ correlation of .50.

A variety of shared environmental experiences might explain our observed findings for high N and high SNC. Shared environmental influences on high SNC could involve parental socio-economic status. In one recent study, conduct disordered and oppositional behaviors decreased in children whose family moved out of poverty (Costello, Compton, Keeler, & Angold, 2003). For high N, the well-known susceptibility of the Eysenckian Neuroticism scale to state effects (e.g., in depressed individuals: Katz & McGuffin, 1987; Carey & DiLalla, 1994) may be a contributing factor. Early traumatic events such as child rape or sexual molestation, which are reported by both members of a female twin-pair far more often

than chance would predict (Dinwiddie et al., 2000; Nelson et al., 2002), are significantly predictive of later depression, and may have long-term effects on personality functioning at the extremes of the personality distribution, or state effects via episodes of major depression.

One possible limitation of this study is that assessment of personality is self-reported. It may be the case that informant ratings of temperament will lead to different conclusions concerning temperament in adolescent girls. Secondly, our binary analyses used the 25th and 75th percentile ranges as cut-off point for categorization. Future studies with larger sample sizes may want to explore more extreme thresholds for categorizing deviant personality types. Although not measured in this study, assessment of personality disorders in future studies would allow for examination of the genetic covariation between normal and abnormal personality functioning in this population in greater detail. In addition, a longitudinal study of changes in environmental and genetic influences on deviant personality beginning in early adolescence into adulthood would allow us to examine more completely how genetic and environmental influences on personality change across time. Finally, inclusion of adolescent male like-sex and male–female opposite-sex twins would allow for investigation of the generalizability of these models across sex.

In conclusion, findings from this study raise the possibility that shared environmental influences were contributing to deviant personality, but not to normal range variation in personality for high neuroticism and high social non-conformity, in both adolescent and adult females. However, because multiple-threshold models fit the data well, the hypothesis of continuity of genetic and environmental influences underlying both normal range and extreme personality variation could not be rejected entirely. The findings for extraversion were in accord with previous findings using continuous personality measures in both adolescents and adults with evidence for additive genetic influences and little or no evidence for contribution from shared environmental influences. Continued attention to deviant manifestations of personality may be important in future studies examining the observed overlap between individual differences and various forms of psychopathology and substance use disorders.

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