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HERITABILITY OF PERSONALITY DISORDERS IN CHILDHOOD: A PRELIMINARY INVESTIGATION

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The heritability of personality disorder features was investigated in 112 child (ages 4–15 years) twin pairs (70 monozygotic and 42 dizygotic pairs). Parents assessed personality disorder features using the Coolidge Personality and Neuropsychological Inventory for Children (CPNI; Coolidge, 1998) that measures 12 personality disorders according to the criteria in the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) (American Psychiatric Association, 1994). Structural equation model-fitting methods indicated that the median heritability coefficient for the 12 scales was .75 (ranging from .81 for the Dependent and Schizotypal Personality Disorder scales to .50 for the Paranoid and Passive-Aggressive Personality Disorder scales). These results suggest that childhood personality disorders have a substantial genetic component and that they are similar to heritability estimates of personality disorder traits in adults and counter hypotheses that only temperaments and higher-order personality disorder traits have significant genetic components (Paris, 1997).

The genetic bases of adult personality disorders, as defined by the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV; American Psychiatric Association, 1994) have received a great deal of attention in recent years (for detailed reviews see McGuffin & Thapar, 1993; Nigg & Goldsmith, 1994; Thapar & McGuffin, 1993). Although a substantial heritable basis for normal personality traits has been found (approximately 45% to 50% of the total variance; Bouchard, 1994; Loehlin, 1992), evidence for the heritability of personality disorders is strong but lies primarily at the trait level or for higher-order dimensions related to personality disorders (Jang, Livesley, Vernon, & Jackson, 1996; Livesley, Jang, Jackson, & Vernon, 1993). The latter estimates of heritability account for approximately 40% to 60% of the total variance. Paris (1997) has argued that heritable traits may determine the form of psychopathology but may be insufficient to determine whether an individual personality disorder itself will develop.

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The issue has also been raised about whether personality traits are stable across the lifespan. There have been suggestions (Costa & McCrae, 1994) that normal personality traits have their greatest stability from the late twenties to middle age, whereas Loehlin (1992) presented evidence that the heritability of personality traits decreases with age. A recent study by Jang, Livesley, and Vernon (1996) of 243 twin pairs from ages 18 to 25 years and 281 twin pairs from ages 30 to 65 years found evidence that genetic contributions to personality disorder traits appear to increase with age.

Research estimating the genetic influence on personality disorders has tended to concentrate mainly on young and middle-aged adults, despite overwhelming evidence that personality disorders may begin in adolescence or earlier (Bemporad, Smith, Hanson, & Cicchetti, 1982; Bernstein, Cohen, Skodol, Bezirganian, & Brook, 1996; Chick, Waterhouse, & Wolff, 1979; Kernberg, 1983; Nagy & Szatmari, 1986; Robins, 1966, 1978; Verhulst, 1984; Wolff, 1993; Wolff & Chick, 1980).

One apparent reason for the paucity of evidence for a genetic basis for personality disorders in childhood may be the lack of standardized interviews or assessment measures for the diagnosis of personality disorders in childhood or adolescence. To address this issue, Coolidge and colleagues (Coolidge, Aksamit, & Becker, 1994; Coolidge et al., 1990; Coolidge, Reilman, Becker, Cass, & Coolidge, 1992; Friedman, 1998; Philbrick, 1990; Reilman, 1993) developed and established the reliability and validity of a parent-as-respondent inventory, in a series of empirical studies, for the diagnosis of personality disorders in childhood and adolescence according to the current DSM.

The purpose of the present study was to assess the heritability of personality disorders in twins from ages 4 to 15 years old using the most recent version of Coolidge's inventory (Coolidge Personality and Neuropsychological Inventory for Children, CPNI; Coolidge, 1998). It was hypothesized that the personality disorder scales in this sample of children would be heritable and similar in magnitude to estimates found for adult personality disorders.

METHOD

PARTICIPANTS

The parents of twins were recruited through students in psychology classes at a midwestern university, through newspaper advertisements, and twin clubs. Participating parents completed a demographic survey, a personality disorder questionnaire, and a zygosity questionnaire.

The final sample consisted of 112 twin pairs, 70 monozygotic (MZ) pairs (34 male pairs and 36 female pairs) and 42 dizygotic (DZ) pairs (14 male pairs, 15 female pairs, and 13 male and female pairs). The mean age of the MZ pairs was 8.6 years (SD = 3.1), and the mean age of the DZ pairs was 8.9 years (SD = 3.1). The mean age of the parents was 38.2 years (SD = 6.4) and 86% of the parents had attained a level of education beyond high school. The mean maternal age at time of birth was 29.0 years (SD = 5.7), and ethnicity was as follows: (a) for MZ twins, Caucasian (82%), Hispanic

(6%), Asian (5%), African American (2%), or other (5%); and (b) for DZ twins, Caucasian (98%) and Asian (2%). Conception followed the use of fertility drugs for 15.2% of the twins. A small proportion of parents reported having exposed the twins to potentially harmful substances before birth, such as alcohol (4%), tobacco (9%), injury (1%), serious illness (1%), and prescription drugs (13%).

Zygosity was diagnosed using a 10-item questionnaire based on a study by Cohen, Dibble, Grawe, and Pollin (1975), and contained items regarding physical similarities (e.g., height, weight, hair and eye color) and confusion of the twins by parents, family, and strangers. Their questionnaire was demonstrated to be approximately 90% reliable (compared with blood typing). A total of 68 mothers and two fathers completed the questionnaires on their MZ twins, and 41 mothers and one father completed the questionnaires on their DZ twins.

MATERIALS

Personality disorders were assessed with the 200-item, parent-as-respondent CPNI for children (Coolidge, 1998). The CPNI was designed with a threefold purpose: (a) to assess the 10 personality disorders on Axis II of the DSM-IV and the two personality disorders in its appendix (depressive and passive-aggressive); (b) to measure neuropsychological dysfunction; and (c) to assess DSM-IV Axis I separation anxiety disorder, oppositional defiant disorder, attention deficit/hyperactivity disorder, and other clinical scales. The CPNI uses a 4-point Likert scale (1 = *strongly false*, 2 = *more false than true*, 3 = *more true than false*, and 4 = *strongly true*) and is designed to be completed by a primary caregiver who is intimately acquainted with the child's behavior. The current normative sample consists of 329 children from ages 5 to 17 years old, and their raw scores were used to establish T scores on the personality disorder scales (for more details see Coolidge, 1998). The 12 personality disorder scales have a median scale reliability of .67 and a median test-retest reliability of .81. Preliminary validity studies support its use in a variety of clinical settings (Coolidge et al., 1994; Coolidge et al., 1990; Coolidge et al., 1992; Friedman, 1998; Philbrick, 1990; Reilman, 1993).

PROCEDURE

The CPNI was mailed to the parents along with the demographic survey containing the zygosity questionnaire and informed consent. The parents were instructed to complete the two forms for their twins on separate days to reduce any contrast effects or effects from repeating the same procedure simultaneously.

STATISTICAL ANALYSES

MZ and DZ twin similarity (Pearson *r*) was estimated for all variables using the computer program PRELIS 2 (Joreskog & Sorbom, 1993a). Structural equation models estimating the magnitude of genetic and environmental influences were then fit to the matrices of MZ and DZ covariances using the computer program LISREL 8 (Joreskog & Sorbom, 1993b). Given the

general population nature of the sample, low endorsement rates of extreme behavior were found on four scales: Narcissistic, Paranoid, Passive-Aggressive, and Schizotypal yielded highly skewed frequency distributions. For these scales, structural equation models estimating the magnitude of genetic and environmental influences were then fit to the matrices of MZ and DZ asymptotically weighted matrices of Pearson correlations by the method of weighted least squares (Neale & Cardon, 1992).

Behavioral genetic designs typically estimate the influence of additive genetic influences (A), environmental influences shared in common (C), and nonshared environmental influences including error (E) on the variance and covariance between variables. Additive genetic influences represent the extent to which genotypes "breed true" from parent to offspring. Shared environmental influences distinguish the general environment of one family from another and influence all children within a family to the same degree (Rowe, 1994). Nonshared environmental factors (Hetherington, Reiss, & Plomin, 1994) include events that have differential effects on individual family members (e.g., pre- and postnatal traumas, differential parental treatment). It should be noted that E is not estimated directly but constitutes the residual variance after the effects of A and C have been removed. As such, this component also contains random error variance.

The first model fit to the data was the "full model" that specified A, C, and E influences. The full model was then systematically modified to test the significance of A, C, and E by fitting a series of "reduced" models. These models systematically removed the effects of (a) additive genetic variance (CE model), (b) shared environmental variance (AE), and (c) additive genetic and shared environmental variance (E only model).

Testing the difference in likelihood ratio χ^2 values assesses the relative fit of the reduced models against the full model. The critical value of χ^2 to test the χ^2 difference is determined by the difference in the number of degrees of freedom (*df*) between the full and reduced model under consideration. The reduced model was rejected whenever χ^2 differences exceeded the critical value of χ^2. Model-fit was also assessed in conjunction with two additional criteria: (a) the principle of parsimony and (b) Akaike's Information Criterion (Akaike, 1987: AIC = $\chi^2 - 2df$). The model reported was the one that did not significantly increase χ^2, that accounted for the variance with the fewest number of parameters, and yielded the smallest value of AIC.

RESULTS

The mean T scores (and SDs) and their ranges for the 12 personality disorder scales for the entire sample of twins are presented in Table 1. These scores establish that there was sufficient variability in the disorders to assess heritability.

The MZ and DZ twin correlations for the 12 scales are presented in Table 2. The MZ correlations were greater than the DZ correlations on all scales, with the greatest ratio on the Conduct Disorder scale. With the exception of the Passive-Aggressive scale, all MZ correlations were significantly greater than the DZ correlations (Fisher's Z test, $p < .05$). All MZ

TABLE 1. Descriptive Statistics for Personality Disorder Scales

Variable	Mean	*SD*	Minimum	Maximum
Avoidant	48.2	10.7	38.2	88.2
Borderline	48.0	10.1	29.4	83.1
Conduct Disorder	47.9	7.9	38.2	97.3
Dependent	50.8	11.8	28.1	84.4
Depressive	46.3	9.8	30.7	78.2
Histrionic	48.4	10.9	35.7	90.1
Narcissistic	46.3	9.1	34.6	85.9
Obsessive-Compulsive	46.1	10.3	30.1	75.3
Paranoid	47.0	10.2	30.8	86.6
Passive-Aggressive	47.9	10.4	32.6	88.6
Schizoid	49.0	9.7	32.2	80.8
Schizotypal	48.2	8.6	33.9	75.0

correlations were significant ($p < .01$), whereas eight of the DZ correlations were significant ($p < .05$).

The fit indices and heritability estimates from the structural equation modeling appear in Table 3. Models that specified nonshared environmental effects (E only model) were eliminated from the table because they did not fit the data. An AE model (without shared environmental effects) best fit the data for nine of the 12 personality disorder scales. For three other scales (Obsessive-Compulsive, Schizoid, and Passive-Aggressive), there was nonsignificant improvement by the addition of shared environmental effects.

The median heritability for the 12 personality disorder scales was .75. Heritability estimates ranged from .81 for the Dependent and Schizotypal Personality Disorder scales to .50 for the Paranoid and Passive-Aggressive scales.

DISCUSSION

Despite the small sample size, these findings suggest that individual differences in personality disorders may have a strong genetic component that

TABLE 2. Correlations for monozygotic (MZ) and dizygotic (DZ) Twins

Scale	r_{MZ}	r_{DZ}
Avoidant	.53	.11
Borderline	.70	.39
Conduct Disorder	.64	.12
Dependent	.82	.40
Depressive	.66	.35
Histrionic	.80	.34
Narcissistic	.66	.34
Obsessive-Compulsive	.76	.44
Paranoid	.51	.25
Passive-Aggressive	.48	.34
Schizoid	.72	.40
Schizotypal	.75	.29

TABLE 3. Fit Indices and Heritability Estimates

Scale	ACE†	AE‡	CE‡	h^2_A	e^2
Avoidant	4.07	4.07	11.32	.61	.39
Borderline	3.62	3.62	15.40	.76	.24
Conduct Disorder	1.64	1.64	9.36	.61	.39
Dependent	1.77	1.77	20.82	.81	.19
Depressive	2.21	2.21	46.07	.76	.24
Histrionic	8.56	8.56	26.37	.79	.21
Narcissistic*	0.00	0.00	4.37	.66	.34
Obsessive-Compulsive	0.45	0.52	12.91	.77	.23
Paranoid*	0.00	0.00	2.20	.50	.50
Passive-Aggressive*	0.00	0.47	0.68	.50	.50
Schizoid	0.59	0.63	10.24	.73	.27
Schizotypal*	3.43	3.43	20.37	.81	.19

Note. A, genetic heritability effects; C, shared environmental effects; E, nonshared environmental effects; h^2_A, additive genetic variance estimates; e^2, environmental effect estimates. AE is underlined because it is the best-fitting model. All models fit were fit to covariance matrices by the method of maximum-likelihood except for those variables denoted by *, which were fit to asymptotically weighted correlation matrices by the method of weighted least squares; †$df = 3$; ‡$df = 4$. Models specifying nonshared environmental effects only (E only model) did not fit the data and are not shown here for brevity.

is present and measurable in childhood. Preliminarily, the results also appear to confirm the hypothesis that personality disorders in children have heritability coefficients that are similar or even greater than those found for adult personality disorder traits and for normal personality traits. Interestingly, Jang et al. (1996) found a .44 median heritability for 66 of 69 personality disorder facet traits in a study of 483 adult twin pairs. If our present findings are substantiated in a larger sample, it may indicate that personality disorder syndromes may even be more heritable than their component traits. It also is possible that contemporaneous parent ratings of behavior yield higher heritability estimates than retrospective ratings and that childhood expressions of personality disorders may yield higher estimates than adult syndromes. The latter finding may be because of a greater period of time that an adult has to adapt to his or her personality dysfunction or to receive treatment.

The finding that the Conduct Disorder scale had a high heritability coefficient (.61) is not without precedent. Jang et al. (1996) found a heritability coefficient of .58 for juvenile antisocial behavior. Cadoret, Leve, and DeVore (1997), in a review of 16 studies, found heritability estimates for CD ranging from .27 to .78 with a median of .48. In the present study, an item analysis of the Conduct Disorder scale between the MZ and DZ twin pairs was performed, because there is some debate (McGuffin & Thapar, 1992) about whether the delinquent component of antisocial behavior is heritable, whereas interpersonal violence is not. Our item analysis revealed that both clusters of items had high heritability estimates. At least one implication of this finding is that strong genetic contributions to antisocial behavior may, as Livesley et al. (1993) have written, "raise questions [for treatment] about the extent to which genetic predisposition imposes limits on the extent to which behavioral change is possible" (p. 1830). Concurrently, animal models involving the neurochemistry and biology of aggressive be-

havior have shown that these behaviors can be significantly altered through modification of particular chemical neurotransmitter systems (Hen, 1996). Perhaps biochemical interventions may be a fruitful avenue of treatment for aggressive behaviors in children or adolescents in the future.

The high heritability estimate for the Borderline Personality Disorder scale (.76) also receives support from the Livesley et al. (1993) finding that identity problems have a high heritability component (.59 in their study). They speculated that labile emotions and an unstable self-concept (core issues of the borderline personality disorder) may be indicators of heritable neuropsychological dysfunction. Furthermore, they speculated that the regulatory and integrative processes associated with neuropsychological functioning may be necessary for the healthy emergence of a stable self-concept.

The present study was limited by the small sample size and generally low rates of psychopathology, although there was sufficient variability in the sample to conclude preliminarily that personality disorders in childhood are measurable and heritable and that their heritability is similar, if not greater, than estimates in adult studies. Future studies may also wish to address the effects of age and gender upon childhood onset of personality disorders, in addition to contrast effects that appear to be present in the parents' ratings of DZ twins. These findings also cautiously support the contention (Jang et al., 1996; Livesley et al., 1993) that personality disorders may be viewed dimensionally, as opposed to categorically, and that personality disorders may be viewed as extreme variations of essentially normal personality traits.

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