

Genetic and Environmental Contributions to Dimensions of Personality Disorder

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Objective: The authors estimated the heritability of the basic dimensions of personality disorder and the relative proportions of the variance attributable to genetic and environmental sources. **Method:** The subjects were 175 volunteer twin pairs (90 monozygotic and 85 dizygotic) from the general population. Each twin completed the Dimensional Assessment of Personality Pathology, a questionnaire that assesses 18 dimensions of personality disorder. The questionnaire was developed on the basis of factor analytic studies that identified a stable structure underlying personality disorders in clinical and nonclinical subjects. Structural equation model-fitting methods were used to estimate the influence of additive genetic, common environmental, and unique environmental effects. **Results:** The estimates of broad heritability ranged from 0%, for conduct problems, to 64%, for narcissism. Behaviors associated with submissiveness and attachment problems had low heritability. For most dimensions, the best-fitting model was one that specified additive genetic and unique environmental effects. **Conclusions:** These results are similar to those reported for normal personality and suggest a continuity between normal and disordered personality.

(Am J Psychiatry 1993; 150:1826–1831)

Biological approaches to personality disorder are receiving increasing attention (1, 2). There is accumulating evidence for the occurrence of genetically determined biological substrates that influence the development of the maladaptive behavioral patterns that constitute personality disorders (2). Although interest in the biological basis of normal personality traits has been an ongoing aspect of the study of personality, the tendency until recently has been to emphasize the role of developmental psychosocial factors in the etiology of personality disorder. Investigation of the biological substrate of personality disorder, however, provides a complementary perspective on etiology that promises to have important implications for classification and treatment.

Evidence from behavioral genetic studies suggests a substantial genetic basis of personality. Twin studies of the genetic contribution to normal personality traits

consistently show heritabilities in the 40%–60% range (3); intraclass correlations for monozygotic twins average about 0.50 across different traits, and correlations for dizygotic twins average about 0.30 (4). Extraversion and neuroticism have received the most investigation. Heritability estimates range from 54% to 74% for extraversion and from 42% to 64% for neuroticism (5, 6). Similar results have been reported for other personality traits (7). The most powerful design for investigating genetic influences compares monozygotic and dizygotic twins who were separated at birth and raised apart. Bouchard et al. (8), reporting on over 100 twins who were reared apart, found comparable heritabilities for traits measured with the California Personality Inventory. Monozygotic twins raised apart were found to be as similar as monozygotic twins raised together.

In contrast to the extensive investigation of normal personality, research on the genetics of personality disorders is sparse. Familial aggregation has been demonstrated for some disorders, especially borderline and schizotypal personality disorders (2, 9–14). Although these studies point to a genetic component, the family study design confounds genetic and environmental factors and provides limited information on the magnitude of genetic and environmental influences. Twin or adoption designs are required for separating and quantifying genetic and environmental effects. A few studies have focused on the heritability of either the categories or the traits of personality disorder; antisocial (15), schizotyp-

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Supported by grant 181(91-2) from the British Columbia Health Care Research Foundation, a National Health and Welfare Canada National Research Scholar Award to Dr. Livesley, and a Senior Research Fellowship from the Ontario Mental Health Foundation to Dr. Jackson.

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al, and borderline personality disorders and some schizotypal traits (16, 17) have a heritable component.

A major development in the field of behavior genetics was the introduction of model-fitting techniques that use structural equations to estimate genetic and environmental components (18–20). The basic structural model designed to estimate heritability has come to be known as the “ACE model.” This model evaluates the effects of additive genetic variance (A), environmental variance attributable to experiences common to twins within each pair, such as family income (C), and environmental variance due to factors not shared by co-twins, such as differential parental care (E) (21–24). The basic ACE model can be modified to test for nonadditive genetic effects attributable to dominance (D), that is, the interaction of alleles at corresponding loci between homologous chromosomes.

In this article we report heritability estimates for the basic dimensions of personality disorder based on data obtained from a volunteer general population study group of twins who were raised together in the same home. Examination of nonclinical subjects was considered justifiable because the pattern of responses of general population subjects to items assessing personality disorder is similar to the pattern for clinical groups (25).

METHOD

Subjects and Procedure

The subjects were 90 monozygotic twin pairs (65 sister pairs and 25 brother pairs) and 85 dizygotic twin pairs (41 sister pairs, 17 brother pairs, 27 sister-brother pairs). The mean ages of the monozygotic and dizygotic twin pairs were 29.07 years ($SD=10.81$, range=16–71) and 28.29 years ($SD=9.38$, range=16–62). The twin pairs were recruited from the Vancouver area of British Columbia, Canada, through newspaper advertisements and media stories. A twin pair was eligible for participation if the twins were aged 16 years or over and had been raised together in the same home. The twin pairs completed the battery of questionnaires at home, a common method for general population twin studies (26). The twins were instructed to complete the questionnaire independently of one another in a nondistracting setting. Zygosity was determined through a questionnaire compiled by Nichols and Bilbro (27). This method has a reported accuracy of about 95% when compared to the results of DNA analysis (28).

Potential biases in estimates of heritability are the effects of gender and age. McGue and Bouchard (29) showed that because monozygotic and same-sex dizygotic pairs share gender and age, any similarity between these twins will be spuriously increased by the existence of gender and age effects on the trait in question. One correction for this possible bias is to conduct gender-by-genotype analyses. This was not possible in the present study because of the small number of opposite-sex dizygotic twins available to us. Instead, the effects of gender and age were removed before the heritability analyses by computing the standardized residual scores from the simultaneous multiple regression of each of the dimensions on age and gender, as suggested by McGue and Bouchard (29). This correction was necessary because the males had significantly higher scores for callousness, conduct problems, narcissism, rejection, restricted expression, stimulus seeking, and suspiciousness ($p>0.003$ in all cases, Bonferroni corrected, two-tailed t tests). Although statistically significant, these effects accounted for only a small proportion of the total variance (adjusted $R^2=0.02$ – 0.07).

Age was positively related to compulsivity and negatively related

to affective instability, anxiety, callousness, cognitive distortion, conduct problems, identity problems, insecure attachment, narcissism, oppositionality, social avoidance, stimulus seeking, and self-harm ($p\leq 0.05$ in all cases, Pearson correlations). As with gender, however, these effects accounted for a negligible proportion of the variance (adjusted $R^2=0.01$ – 0.07).

The numbers of monozygotic and dizygotic pairs in this study group were approximately equal: 51% monozygotic and 49% dizygotic. This ratio is comparable to the 1:1 ratio of monozygotic to dizygotic twins in the general population who survive the first year of life (30). The equal numbers of monozygotic and dizygotic twin pairs were obtained by increasing remuneration to the twin participants to overcome the sampling bias typical of volunteer twin studies, i.e., the tendency for two-thirds more monozygotic than dizygotic twins to volunteer (31).

Critical to the validity of the twin method is the “assumption of equal environments,” that is, the assumption that the common environments of monozygotic twins are not different from the common environments of dizygotic twins. This assumption was tested by comparing the rates of endorsement of items assessing the environments of the same-sex twins, for example, “We spend most of our time together,” “We attend the same school,” “We have the same friends,” “We tend to dress alike,” and “Our parents treat us pretty much the same.” Significant differences were not detected ($p>0.05$ in all cases, two-tailed chi-square tests). Additionally, the monozygotic and dizygotic twins did not report significantly different numbers of serious illnesses ($t=1.04$, $df=348$, $p>0.05$, two-tailed) or separations for more than 1 month ($t=-1.44$, $df=348$, $p>0.05$, two-tailed). These results are consistent with those of other studies, supporting the assumption of equal environments by using different methods (32, 33).

Measure of Dimensions of Personality Disorder

The basic dimensions of personality disorder were assessed with the Dimensional Assessment of Personality Pathology—Basic Questionnaire (34, 35), a 290-item self-report measure that assesses 18 dimensions: affective lability, anxiousness, callousness, cognitive distortion, compulsivity, conduct problems, identity problems, insecure attachment, intimacy problems, narcissism, oppositionality, rejection, restricted expression, self-harm, social avoidance, stimulus seeking, submissiveness, and suspiciousness. Definitions of the dimensions and the facet traits for each dimension are listed in appendix 1. In earlier studies, multivariate statistical procedures were used to identify 18 basic dimensions that provide a systematic representation of the domain of personality disorder. The traits delineating personality disorder diagnoses were identified initially from content analysis of the literature and clinical opinion (36, 37). Self-report scales were developed to assess these traits. The traits were reduced to fewer dimensions by means of factor analysis (34). A factorial structure that was stable across clinical and nonclinical subject groups was identified (25). The results of these analyses were used to define 18 factor-based scales. A self-report inventory was developed to assess each dimension (35). Each dimension is assessed with 16 items, except self-harm and suspiciousness, which have 14 items each. The questionnaire also includes a validity scale.

We used the Dimensional Assessment of Personality Pathology because it provides a systematic assessment of the basic dimensions underlying the overall domain of personality disorder. The instrument has demonstrated factorial validity and satisfactory psychometric properties. Its internal consistency (coefficient alpha) ranges from 0.83 to 0.94. The test-retest reliability over a 3-week period ranges from 0.81 to 0.93. The scales cover a broad range of behaviors and are not reducible to the broad dimensions of neuroticism and extraversion (35). The scales of the Dimensional Assessment of Personality Pathology are also similar to those developed by Clark (38), who used a different procedure to identify the dimensions underlying axis II.

Statistical Analysis

The distributions of raw scores for each scale were examined for departure from normality. Square root or natural logarithmic transformations were performed when necessary to obtain adequate sym-

TABLE 1. Between-Twin Correlations, Adjusted for Age and Gender, of Scores on Dimensional Assessment of Personality Pathology

Dimension	Correlation (r)		Ratio
	Monozygotic (90 pairs)	Dizygotic (85 pairs)	
Affective lability	0.48 ^{a, b}	0.13	3.69
Anxiousness	0.54 ^a	0.33 ^a	1.63
Callousness	0.63 ^{a, b}	0.29 ^a	2.17
Cognitive distortion	0.82 ^{a, b}	0.39 ^a	2.10
Compulsivity	0.42 ^a	0.23 ^a	1.82
Conduct problems	0.52 ^a	0.52 ^a	1.00
Identity problems	0.60 ^{a, b}	0.26 ^a	2.31
Insecure attachment	0.52 ^a	0.38 ^a	1.36
Intimacy problems	0.40 ^{a, b}	-0.01	40.00
Narcissism	0.64 ^{a, b}	0.12	5.33
Oppositionality	0.58 ^{a, b}	0.27 ^a	2.15
Rejection	0.49 ^{a, b}	0.22 ^a	2.23
Restricted expression	0.51 ^{a, b}	0.25 ^a	2.04
Self-harm	0.30 ^a	0.10	3.10
Social avoidance	0.57 ^{a, b}	0.26 ^a	2.19
Stimulus seeking	0.59 ^{a, b}	0.33 ^a	1.78
Submissiveness	0.54 ^a	0.41 ^a	1.32
Suspiciousness	0.49 ^{a, b}	0.25 ^a	1.96

^ap<0.05.^bCorrelation for monozygotic twins significantly greater than that for dizygotic twins (p<0.05, z test, two-tailed).

metry. The relationship between zygosity and each scale of the Dimensional Assessment of Personality Pathology was examined by regressing each of the scales on zygosity. No significant zygosity effects were detected (p>0.003 in all cases, Bonferroni corrected, two-tailed t tests).

Covariances and Pearson correlations between co-twins were computed separately for monozygotic and dizygotic twins by using the computer program PRELIS (39). Overall genetic influence is indicated when the correlation for monozygotic twins is greater than the correlation for dizygotic twins. This general genetic effect can be partitioned into two specific genetic effects: additive genetic effects and nonadditive genetic effects attributable to the genetic dominance effect. The presence of additive genetic effects is indicated when the correlation for monozygotic twins is greater than that for dizygotic twins. When the correlation for monozygotic twins is greater than twice the size of the correlation for dizygotic twins, however, nonadditive genetic effects are also indicated.

Univariate genetic structural equation models (20, 40, 41) were fit to the covariances by using the computer program LISREL VII (42). The purpose of these analyses was to estimate the proportion of the variance for each scale that was accounted for by additive genetic factors, nonadditive genetic factors attributable to dominance (only when the presence of these effects was indicated), common environment, and nonshared environment. Goodness of fit was assessed with chi-square tests and Akaike's information criterion (43, 44), which yields a superior indication of fit in models with a small number of indicators (45). The statistical significance of the effect size accounted for by additive genetic factors (A), nonadditive genetic effects attributable to dominance (D), common environment (C), or nonshared environment (E) was then tested by comparing a number of reduced models that systematically remove components of variance with the full ACE or ADE model. Three reduced models were tested: the AE-only model, which predicts no common environmental effect; the CE-only (or DE-only) model, which predicts no additive genetic effect; and the E-only model, which predicts no family resemblance.

RESULTS

The Pearson correlations obtained for monozygotic and dizygotic twins for each scale of the Dimensional

TABLE 2. Goodness-of-Fit Statistics for Full and Reduced Models of Genetic and Environmental Effects on Scores of Monozygotic and Dizygotic Twins on Dimensional Assessment of Personality Pathology

Dimension	Fit in Chi-Square Units ^a			
	ACE/ADE (df=3)	CE/DE (df=4)	AE (df=4)	E (df=5)
Affective lability ^b	1.21	1.21 ^c	2.35	24.42 ^d
Anxiousness	2.40	6.86	2.45 ^c	38.42 ^d
Callousness ^b	5.16	10.07 ^d	5.16 ^c	44.88 ^d
Cognitive distortion	3.60	7.06	4.02 ^c	43.73 ^d
Compulsivity	1.35	3.43	1.37 ^c	21.27 ^d
Conduct problems	0.65	0.65 ^c	8.93	53.10 ^d
Identity problems ^b	3.90	4.68	4.15 ^c	43.46 ^d
Insecure attachment	0.32 ^c	2.41	40.09 ^d	29.96 ^d
Intimacy problems ^b	0.51	0.51 ^c	2.22	14.01 ^d
Narcissism ^b	2.89	2.89 ^c	6.06	45.31 ^d
Oppositionality ^b	1.05	2.51	1.05 ^c	36.80 ^d
Rejection ^b	0.17	2.08	0.17 ^c	29.98 ^d
Restricted expression ^b	0.36	2.31	0.36 ^c	33.21 ^d
Self-harm ^b	8.95	9.02 ^c	9.06	16.43 ^d
Social avoidance ^b	0.65	1.76	0.70 ^c	36.28 ^d
Stimulus seeking	3.04	7.70	3.25 ^c	45.93 ^d
Submissiveness	1.24	8.10	1.25 ^c	46.02 ^d
Suspiciousness	1.65	6.92	2.65 ^c	29.08 ^d

^aA, additive genetic factors; D, nonadditive genetic effects attributable to dominance; C, common environment; E, nonshared environment.^bADE models were fitted to this dimension; the chi-square differences between full and reduced models are testable with the chi-square critical values of 3.84 (df=1) and 5.99 (df=2) where p<0.05.^cBest fitting by Akaike's criterion (43, 44).^dp<0.05.

Assessment of Personality Pathology and the ratio of the correlations for monozygotic and dizygotic twins are shown in table 1. For all the scales except conduct problems, the correlations for monozygotic twins exceeded those for dizygotic twins, indicating a genetic influence. Dominance effects were indicated for affective lability, callousness, cognitive distortion, identity problems, intimacy problems, narcissism, oppositionality, rejection, restricted expression, self-harm, and social avoidance. Table 2 presents the model-fitting results for the dimensions when an ACE or an ADE model was fitted.

Examination of the model-fitting goodness-of-fit measures for the different models reveals some clear results. First, although the nonshared environmental effects were large, they alone could not satisfactorily account for all the variance. Additive genetic and nonshared environmental effects together accounted for all the variance in anxiousness, cognitive distortion, compulsivity, stimulus seeking, submissiveness, and suspiciousness. Genetic dominance effects and nonshared environmental effects together accounted for all the variance in affective lability, intimacy problems, narcissism, and self-harm. Shared environmental influences appeared to have no practical effect on these dimensions. Shared environmental effects appeared to play a role in insecure attachment. Conduct problems appeared to be wholly environmentally determined.

Heritability estimates computed on the overall ACE and ADE models are presented in table 3. The influence of genetic effects varied widely across dimensions. The

magnitude of additive genetic effects ranged from 0%, for conduct problems, to 56%, for callousness: the median narrow heritability was 40%. Genetic dominance effects were substantial for affective lability (48%), intimacy problems (38%), and narcissism (64%). Genetic dominance effects were present but modest for identity problems, self-harm, and social avoidance. Shared environmental effects were substantial for only two dimensions: conduct problems (53%) and submissiveness (28%). Finally, nonshared effects composed the majority of the effects for most dimensions. These effects ranged from 71%, for self-harm, to 36%, for narcissism (median=47%).

DISCUSSION

Our results are largely consistent with those from investigations of genetic and environmental influences on normal personality traits (5). Twelve of the 18 dimensions that we assessed had heritabilities in the 40%–60% range typically reported for normal personality traits. For most normal traits, common environmental factors had little effect compared to nonshared environmental and additive genetic influences. We also found that common environmental effects were minimal for all dimensions except conduct problems and submissiveness and, to a lesser extent, cognitive distortion and insecure attachment.

The results for the individual scales also converge with those of previous studies. Zuckerman's sensation seeking scale has many substantive similarities to the stimulus seeking scale of the Dimensional Assessment of Personality Pathology, and the heritability estimates for the two scales were 58% (46) and 50%, respectively. The NEO Personality Inventory (47) conscientiousness domain, which correlates highly with the compulsivity scale of the Dimensional Assessment of Personality Pathology (35), had a heritability of 36%, compared to 39% for compulsivity in the present study. Kendler et al. (48) reported that the heritability of suspiciousness, measured by four items from the Eysenck Personality Inventory, was 41%; our estimate was 49%. Anxiousness was 49% heritable; previous studies have typically shown similar values for neuroticism. Our estimate of 41% for the heritability of cognitive distortion is consistent with heritabilities reported for scales assessing schizotypy (16).

Not only are these results consistent with those of other studies, they are also internally coherent. Submissiveness, a scale measuring the submissive, suggestible, and reassurance-seeking behaviors associated with dependent personality disorder, had low heritability (25%). This was similar to the heritability of insecure attachment (36%), a second component of dependent personality disorder (49).

The range of heritabilities estimated for our scales lends support to our earlier contention that the Dimensional Assessment of Personality Pathology scales measure distinct aspects of personality pathology. Loehlin

TABLE 3. Estimates of Genetic and Environmental Influences on Scores of Monozygotic and Dizygotic Twins on Dimensional Assessment of Personality Pathology

Dimension	Estimate of Influence (%)			
	Additive Genetic Factors	Genetic Dominance	Common Environment	Nonshared Environment
Affective lability ^a	1	48	0	51
Anxiousness	49	0	6	45
Callousness ^a	56	0	0	44
Cognitive distortion ^a	41	0	14	45
Compulsivity	39	0	3	58
Conduct problems	0	0	53	47
Identity problems ^a	40	19	0	41
Insecure attachment	35	0	13	52
Intimacy problems ^a	0	38	0	62
Narcissism ^a	0	64	0	36
Oppositionality ^a	52	3	0	45
Rejection ^a	45	0	0	55
Restricted expression ^a	47	0	0	53
Self-harm ^a	14	15	0	71
Social avoidance	47	10	0	43
Stimulus seeking	50	0	9	41
Submissiveness	25	0	28	47
Suspiciousness	48	0	0	52

^aThis dimension was tested for genetic dominance effects. Dominance effects are present when the ratio of the correlations for monozygotic and dizygotic twins is greater than 2.0.

(50) suggested that one reason for the similar heritabilities reported for many personality traits is that they are facets of the higher-order dimensions of neuroticism and extraversion. The differential heritabilities observed suggests that this does apply to our basic dimensions and that several different genetic factors are implicated in personality disorder. Further support is provided by the analysis of genetic dominance effects, which were significant for a few of the dimensions: affective lability, intimacy problems, narcissism, and self-harm.

The estimates of genetic contributions to some dimensions warrant further comment. Statistically significant genetic effects were not observed for several dimensions, including insecure attachment, intimacy problems, and submissiveness. These dimensions describe interpersonal problems relating to close interpersonal relationships. This is a novel finding that requires replication. One might speculate that these scales refer to problems in the way the attachment behavioral system develops and that, although the system is genetically controlled, differential experiences within the family are primarily responsible for its expression and development.

Perhaps the most surprising observation was the high broad heritability of identity problems (59%). Intuitively, it might be expected that identity refers to behaviors and experiences that are largely the product of developmental psychosocial events. The dimensions consist of four traits that consistently factor together: labile or unstable self-concept, chronic feelings of emptiness and boredom, anhedonia that is characterologi-

cal in nature, and pessimism that is also characterological. The scale items sample all traits with a slight weighting toward labile self-concept and chronic feelings of emptiness and boredom. It could be argued that the reason for high heritability is that the scale reflects affective experience. Ten of the 18 scale items, however, refer to other aspects of self-concept and identity. An alternative explanation is that the scale assesses behavioral indicators of neuropsychological dysfunction involving regulatory and integrative processes necessary for the emergence of a stable sense of self.

The results reported suggest a perspective on the etiology of personality disorder that has implications for classification, theories of etiology, and methods of treatment. The similarity between our results and those reported for normal personality is consistent with the hypothesis that personality disorders involve extremes of normal variation and hence a dimensional classification. Most components of personality disorder, except those relating to dependency and attachment, have a substantial genetic component. This observation, if replicated, ought to be reflected in the structure of classification. For most components with a substantive genetic contribution, specific environmental factors have a substantial effect, pointing to the importance of etiological models that account for the way genetic vulnerability influences responses to adverse environmental circumstances. This promises to be an important area of inquiry. The substantial genetic underpinning of many traits also has implications for treatment because it raises questions about the extent to which genetic predisposition imposes limits on the extent to which behavioral change is possible. Genetically influenced traits may also modify responses to various interventions and hence be important factors to consider in treatment planning.

These results should be interpreted in the context of several limitations. First, we studied a group of volunteers from the general population. Although the evidence suggests that the structure of personality disorder is similar in general population and clinical groups, the possibility remains that different results may be obtained from twins with personality disorder. We might also have obtained different results from a representative general population sample. Second, we assessed the traits underlying DSM-III-R diagnoses rather than diagnostic criteria. Although our scales assess traits that psychiatrists consider prototypical of personality disorder, direct assessment of criteria sets might have yielded different results.

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APPENDIX 1. Constituent Scales of the Dimensional Assessment of Personality Pathology

Affective lability: affective instability, affective overreactivity, generalized hypersensitivity, labile anger, irritability.

Anxiousness: guilt proneness, indecisiveness, rumination, trait anxiety.

Callousness: contemptuousness, egocentrism, exploitation, interpersonal irresponsibility, lack of empathy, remorselessness, sadism.

Cognitive distortion: depersonalization, schizotypal cognition, brief stress psychosis.

Compulsivity: orderliness, precision, conscientiousness.

Conduct problems: interpersonal violence, juvenile antisocial behaviors, addictive behaviors, failure to adopt social norms.

Identity problems: anhedonia, chronic feelings of emptiness, labile self-concept, pessimism.

Insecure attachment: separation protest, secure base, proximity seeking, feared loss, intolerance of aloneness.

Intimacy problems: desire for improved attachment, inhibited sexuality, avoidant attachment.

Narcissism: need for adulation, attention seeking, grandiosity, need for approval.

Oppositionality: passivity, oppositionality, lack of organization.

Rejection: rigid cognitive style, judgmental, interpersonal hostility, dominance.

Restricted expression: reluctant self-disclosure, restricted expression of anger, restricted affective expression, restricted expression of positive sentiments, self-reliance.

Self-harm: ideas of self-harm, self-damaging acts.

Social avoidance: low affiliation, defective social skills, social apprehensiveness, fear of interpersonal hurt, desire for improved affiliative relationships.

Stimulus seeking: sensation seeking, recklessness, impulsivity.

Submissiveness: subservience, suggestibility, need for advice.

Suspiciousness: hypervigilance, suspiciousness.