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Heritability of MMPI Personality Indicators of Psychopathology in Twins Reared Apart

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This report presents Minnesota Multiphasic Personality Inventory (MMPI) findings from the Minnesota Study of Twins Reared Apart. Data from 65 unique pairs of monozygotic twins reared apart (MZA) and 54 unique pairs of dizygotic twins reared apart (DZA) were analyzed. As in other results from this sample, MZA twins evidenced substantial similarity, highlighting the influence of shared genes. Biometric modeling yielded estimates of heritability for the MMPI's standard validity and clinical scales and for the Wiggins content scales ranging from .26 to .62 (M = .44), echoing previous findings from the twin and adoption literature on personality. The pattern of MZA and DZA correlations suggested nonadditive genetic effects for 3 MMPI scales. Multivariate profile analyses also suggested genetic influence on both profile elevation and shape.

The history of theory and research on psychopathology reveals long-standing interest in the relationship between personality characteristics and mental disorder (Berrios, 1993). Slater and Slater (1944/1971) concluded that the form of neurotic disorder was largely influenced by genetic dispositions associated with personality whereas the actual occurrence of disorder was caused by coaction of genetic diatheses and environmental stressors. Eysenck and Eysenck (1985) theorized that liability toward development of several types of disorders was related to three heritable dimensions of personality: extraversion, neuroticism, and psychoticism. Under this model, high neuroticism confers risk for disorder, with the pattern of extraversion and psychoticism scores related to the actual form of disorder. Similar models have been forwarded by Gray (1985) and Cloninger (1987) with emphases on slightly different personality axes.

Clark, Watson, and Mineka (1994) argued that high negative affectivity is specifically associated with risk for depression and anxiety disorders whereas low positive affectivity is associated only with depression. Consistent with this model, Carey and DiLalla (1994) reported evidence for significant genetic correlations among neuroticism, anxiety, and depression—shared genes accounted for roughly 50% of the variance in observed correlations between neuroticism and these forms of psychopathology. Other investigators have evaluated links between normal personality traits and a range of psychiatric disorders (Berenbaum & Fujita, 1994; DiLalla & Gottesman, 1995; Trull & Sher, 1994; Widiger & Trull, 1992), including personality disorders (Costa & McCrae, 1990; DiLalla, Gottesman, & Carey, 1993; Schroeder, Wormworth, & Livesley, 1992; Trull, 1992).

The nature of associations between personality traits and psychopathology appears to be highly complex. For example, depressed-phase Minnesota Multiphasic Personality Inventory (MMPI) profiles of individuals with bipolar affective disorder differ from profiles of normally functioning individuals and from remission-phase profiles (Lumry, Gottesman, & Tuason, 1982). This suggests change in personality scores as a consequence of psychiatric state effects. MMPI profiles of individuals with severe phobias and obsessions also show strong state-dependent features but are still distinctive 10 years after treatment (Carey & Gottesman, 1981). Similarly, DiLalla and Gottesman (1995) reported strong diagnostic group differences for normal personality characteristics between individuals with schizophrenia and their normally functioning identical cotwins. At the same time, there remained some significant personality correlations between members of these discordant identical twin pairs. The findings suggest a combination of state effects on personality scores and stable personality characteristics associated with disorder.

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Considerable research has been published on the genetics of personality (see e.g., Bouchard, 1994; Heath, Cloninger, & Martin, 1994; Loehlin, 1992; McGue, Bacon, & Lykken, 1993; Tellegen et al., 1988), almost all of which is consistent with the hypothesis of moderate genetic influence (generally accounting for 40–60% of the variance) on personality traits and little or no influence of shared environmental effects. However, the assumptions required in twin studies of personality are not always as clearly met as those required for psychopathology. For example, a critical assumption of the twin method is that identical twins do not imitate each other more than fraternal twins. Although it may be reasonable to assume that one twin would not deliberately imitate an identical twin's psychosis, it is more difficult to assume that one twin might not imitate patterns of cotwin behavior that lead to greater sociability when both are reared together in an average family for the first 18 years of life. Such effects could induce monozygotic (MZ) cotwin similarity that would inflate estimates of heritability under the traditional twin model.

Consequently, the study of adoptees is important. The results of major adoption studies of adolescent (Scarr, Weber, Weinberg, & Wittig, 1981) and adult personality (Loehlin, Horn, & Willerman, 1990) are generally consistent with the results of twin studies, indicating moderate heritability for broad personality dispositions with little influence from shared family environmental factors. Here, we report a study of the genetics of personality correlates of psychopathology with an unusual and important sample of adoptees—monozygotic and dizygotic twins reared apart (MZA and DZA, respectively) since infancy. Among four previous samples of MZA twins (Langenvainio, Kaprio, Koskenvuo, & Lonnqvist, 1984; Newman, Freeman, & Holzinger, 1937; Pedersen, Plomin, McClearn, & Friberg, 1988; Shields, 1962), each demonstrated significant heritability for neuroticism.

Analyses of normal personality characteristics previously have been reported for the sample to be analyzed here (Bouchard, 1994; Bouchard & McGue, 1990; Tellegen et al., 1988; Waller, Kojetin, Bouchard, Lykken, & Tellegen, 1990). Consistent with the literature on twins reared together, these studies reported heritability coefficients ranging from .4 to .6 for broad normal personality traits, with few nonshared environmental effects and some evidence of nonadditive (epistatic) genetic influences.

The present study extends these findings by demonstrating heritability for a range of personality correlates of psychopathology never examined in the previous studies of twins reared apart. The analyses focus on the personality inventory most often used in both clinical and research settings on psychopathology, the MMPI (Hathaway & McKinley, 1983).

Method

Participants

The participants in this study were twin pairs from the Minnesota Study of Twins Reared Apart (MISTRA) who were aged 18 years or older and were tested by August 1995. Compared with previous studies of twins reared apart, the MISTRA series has the lowest age of separation and the latest age of reunion (see Table 1).[1] For several twin pairs, reunion occurred during their participation in the study. Twins were ascertained in a number of ways. First, media accounts of the study led a number of twins or their family members to contact the project. Twins ascertained in this way came from several different countries, although the majority were American. Second, a social worker in England specializing in tracing biological relatives separated by adoption referred a number of twins from the British Isles and present or former British commonwealth countries. Other professionals who became aware of reared-apart twins also referred twins to the project.

After being ascertained and giving informed consent to participate in the study, both twins spent a week at the University of Minnesota undergoing extensive medical, dental, and psychological evaluations. Determination of zygosity was based on analysis of four serum proteins, six red blood cell enzymes, eight blood group systems, and a variety of physical similarity measures. The probability of misdiagnosis of zygosity was less than .001 (Lykken, 1978).

For the present study, data were analyzed from 111 twin pairs and 4 sets of triplets (2 MZ sets and 2 sets with an MZ pair and a DZ member) in which each sibling had completed the MMPI. Sixty-one of the twin pairs were MZ twins; the addition of the MZ individuals from the 4 triplet pairs yielded a total of 65 unique identical sibships (41 female pairs and 24 male pairs) from 132 individuals. Thirty-eight pairs were same-sex dizygotic (DZSS) twins (26 female pairs and 12 male pairs), and 12 pairs were opposite-sex dizygotic twins (DZOS). Four more DZOS pairs were added to the sample by pairing the DZ member from each of the two nonidentical triplet sets with the first MZ member of the set, and then with the second MZ member of the set. This resulted in a total of 54 DZ sibships (38 DZSS pairs and 16 DZOS pairs).[2] The bias introduced by treating the 2 DZ triplets as 4 independent pairs is extremely small: The difference between the standard errors of z-transformed correlations for the 50-sibship case and the 54-sibship case was .004. The inclusion of opposite-sex twin pairs is consistent with other published work on personality from the MISTRA project. However, to assess possible bias introduced by including DZOS pairs, we also carried out the biometric analyses with such pairs removed. See Table 1 for descriptive information about the sample.

Measures

The MMPI was administered independently to each twin. The responses were computer scored for the MMPI's 3 validity scales, 10 standard clinical scales, and the Wiggins content scales. Because heritability of the *K* (Correction) scale confounds interpretation of heritability for *K*-corrected clinical scales, non-*K*-corrected scores were used in this report, with the exception of the mean samplewide MMPI profile presented in Figure 1. The Wiggins content scales have demonstrated content validity and no item overlap and cover a wide range of thoughts, experiences, and behaviors associated with psychopathology (see Greene, 1991, and Wiggins, 1966, for description). These scales are quite similar to the content scales in the revised MMPI (MMPI-2; Butcher, 1990) and also have been used in studies of twins reared together (Goldsmith & Gottesman, 1977).

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[1] The standard deviations and ranges in Table 1 highlight the apparent presence of outliers with respect to time apart before reunion and total contact time. One MZA pair had been in contact for almost 20 years longer than the DZA pair with the most contact time, and one MZA pair was reunited approximately 16 years earlier than was the soonest reunited DZA pair.

[2] Previous reports on the MISTRA project have used slightly different sample sizes based on data available for specific analyses. Bouchard (1994) reported on 59 MZA pairs and 47 DZA pairs; Bouchard and McGue (1990) reported on 45 MZA pairs and 26 DZA pairs; Tellegen et al. (1988) reported on 44 MZA pairs and 27 DZA pairs; and Waller et al. (1990) reported on 53 MZA pairs and 31 DZA pairs.

Table 1
Means, Standard Deviations, and Ranges (in Years) for Age and Three Measures of Contact Time for MZA and DZA Twins

Variable	MZA twins			DZA twins		
	M	*SD*	Range	*M*	*SD*	Range
Age	40.4	12.6	18–68	45.1	13.5	22–77
Time together before separation	0.4	0.7	0–4	0.9	1.2	0.01–4.5
Time apart to first reunion	31.1	15.5	0.5–65.3	41.6	13.7	17–75.2
Total contact time	1.95	3.95	0.02–23.7	0.96	1.2	0.02–4.5

Note. For monozygotic twins reared apart (MZA), $n = 132$ individuals; for dizygotic twins reared apart (DZA), $n = 106$ individuals.

Age and Sex Correction Procedures

Because age and sex effects can bias twin analyses, we corrected MMPI raw scale scores for the linear effects of sex and age and the quadratic effect of age (age^2) using the procedure described by McGue and Bouchard (1984). We computed intraclass correlations from the corrected MMPI scores using the method of Snedecor and Cochran (1980) to derive unbiased estimates of the variance components for an unbalanced analysis of variance (ANOVA). We evaluated potential age differences in the magnitude of heritability estimates by fitting a model in which heritability was dependent on the age of the twin pair. The sample is too small to permit analyses of sex differences in heritability of MMPI scales. For example, the correlation for male and female MZA twins must differ by almost .50 to reach statistical significance.

Biometric Analyses

Although intraclass correlations are presented for descriptive purposes, the statistical models analyzed mean squares for MZA and DZA twins. When MZ and DZ variances differ, the intraclass correlations may be misleading as rough indicators of heritability. A standard biometric model was fit to the mean squares. The model was a simple additive genetic one that assumed no genetic dominance or interactions between genetic loci and no assortative mating for MMPI scales. The full model fit two parameters, V_g (genetic variance, or heritability) and V_u (unique environmental variance). V_c (common environmental variance) was arbitrarily set to zero to reflect reared-apart status. The two parameters were estimated from four mean squares, yielding a chi-square with 2 degrees of freedom as an overall test of the goodness of fit of the model. Comparison of the full model with a nested model that arbitrarily set V_g equal to zero allowed for a chi-square with one degree of freedom as a test of the significance of the heritability estimate.

If the full model fails to fit the data for a given scale, but the test of the heritability parameter indicates significant (nonzero) heritability, this may be the result of nonadditive genetic variance when the DZA correlation is substantially lower than the expectation of one half of the MZA correlation. Alternatively, selective placement or subculture rearing differences may be factors when twice the DZA correlation is substantially higher than the MZA correlation. Biometric models were evaluated with the Davidon–Fletcher–Powell minimizer program (Carey, 1990). The approach to biometric modeling used here is described in detail by Eaves, Eysenck, and Martin (1989). The model fitting answers three questions: (a) Are the data from the MZA pairs consistent with those of DZA pairs according to the model? (b) Can the hypothesis of no genetic effects be rejected? and (c) What is the extent of the genetic effects on the traits?

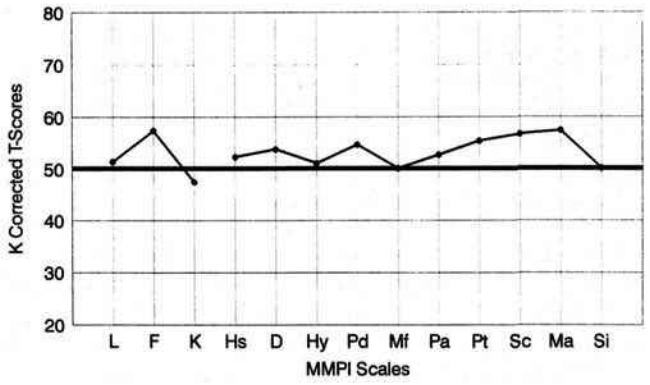

Figure 1. Mean *K*-corrected Minnesota Multiphasic Personality Inventory (MMPI) profile for monozygotic and dizygotic twins reared apart. *T* scores are computed from contemporary MMPI norms (Colligan, Osborne, Swenson, & Offord, 1983). (*L* = Lie; *F* = Frequency; *K* = Correction; *Hs* = Hypochondriasis; *D* = Depression; *Hy* = Hysteria; *Pd* = Psychopathic Deviate; *Mf* = Masculinity–Femininity; *Pa* = Paranoia; *Pt* = Psychasthenia; *Sc* = Schizophrenia; *Ma* = Hypomania; *Si* = Social Introversion.)

Results

Table 2 summarizes non-age-corrected MMPI means and standard deviations subdivided by sex and zygosity. These statistics are based on non-*K*-corrected *T* scores derived from contemporary MMPI norms (Colligan & Offord, 1988; Colligan, Osborne, Swenson, & Offord, 1983). Multivariate analysis of variance (MANOVA) indicated an overall effect for sex on MMPI clinical scale scores, $F(13, 218) = 2.2$, $p < .01$. Men scored higher than women on Masculinity–Femininity (effect size, $d = .38$) and lower than women on the Lie scale ($d = .42$), but these effects were of moderate magnitude. There was no effect for zygosity on MMPI clinical scale scores, $F(13, 220) = 0.84$, *ns*. A *K*-corrected samplewide mean MMPI profile is presented in Figure 1. The clinical scale means are well within normal limits, but the range of individual differences is highlighted by the fact that 18% of the participants had two or more clinical scales above a *T* score of 70. For the Wiggins scales, there was no overall group effect for zygosity, $F(13, 218) = 1.3$, *ns*. There was an overall effect for sex, $F(13, 218) = 2.0$, $p < .05$, with men having a higher average *T* score than women on the Feminine Interests scale ($d = .5$).

Table 2
Mean T Scores for MMPI Clinical Scales (Without K Correction) and Wiggins Content Scales for Twins Reared Apart

	Male				Female			
	MZ (n = 50)		DZ (n = 38)		MZ (n = 82)		DZ (n = 64)	
Scale	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
				Clinical scales				
L	46.8	8.3	50.7	7.2	54.1	10.7	52.1	10.5
F	56.6	10.1	57.7	8.3	56.8	10.7	58.6	8.6
K	46.3	9.3	46.9	10.3	50.5	9.6	45.1	10.4
Hs	51.0	11.2	55.2	10.4	54.1	9.2	54.3	8.0
D	51.1	11.3	55.9	10.5	53.8	10.9	54.5	10.4
Hy	48.9	10.9	51.2	9.6	53.3	12.1	49.9	10.1
Pd	57.4	8.9	53.9	12.4	54.2	11.5	56.2	11.2
Mf	53.6	12.3	52.8	12.0	48.8	8.1	49.7	9.3
Pa	51.4	12.0	54.9	9.0	52.3	11.0	53.0	11.5
Pt	55.2	10.0	55.9	12.8	53.3	10.5	57.1	9.9
Sc	56.3	10.7	56.0	9.0	55.3	10.7	58.7	9.9
Ma	58.6	11.9	54.8	10.7	56.5	11.3	59.2	11.0
Si	49.3	9.2	52.3	10.4	49.8	10.4	52.6	11.2
				Wiggins content scales				
Soc	47.3	9.3	51.2	9.8	48.0	10.0	51.4	10.3
Dep	55.1	13.0	57.8	14.1	52.4	11.5	56.6	12.2
Fem	54.0	12.6	52.6	11.2	48.6	11.2	47.6	9.4
Mor	52.5	8.8	53.4	11.4	48.9	8.9	53.2	9.6
Rel	42.4	10.2	46.4	10.2	44.1	11.6	40.4	12.6
Aut	57.0	10.9	54.1	10.4	52.2	10.7	54.1	12.1
Psy	57.2	13.6	54.0	12.7	53.2	11.8	56.1	11.0
Org	52.5	13.6	55.8	12.7	54.8	12.6	55.1	10.1
Fam	57.8	13.5	57.5	13.8	53.9	14.3	59.2	13.3
Hos	55.3	11.0	51.2	9.3	49.6	10.1	54.7	9.8
Pho	49.3	10.3	48.7	9.6	49.3	8.6	50.6	9.6
Hyp	56.4	11.8	54.6	12.4	54.3	12.1	57.0	12.2
Hea	50.4	9.6	53.6	12.1	52.0	10.4	51.7	8.1

Note. *T* scores derived from contemporary Minnesota Multiphasic Personality Inventory (MMPI) norms (Colligan & Offord, 1988; Colligan et al., 1983). MZ = monozygotic; DZ = dizygotic. MMPI scales: *L* = Lie; *F* = Frequency; *K* = Correction; *Hs* = Hypochondriasis; *D* = Depression; *Hy* = Hysteria; *Pd* = Psychopathic Deviate; *Mf* = Masculinity–Femininity; *Pa* = Paranoia; *Pt* = Psychasthenia; *Sc* = Schizophrenia; *Ma* = Hypomania; *Si* = Social Introversion. Wiggins content scales: *Soc* = Social Maladjustment; *Dep* = Depression; *Fem* = Feminine Interests; *Mor* = Poor Morale; *Rel* = Religious Fundamentalism; *Aut* = Authority Conflict; *Psy* = Psychoticism; *Org* = Organic Symptoms; *Fam* = Family Problems; *Hos* = Manifest Hostility; *Pho* = Phobias; *Hyp* = Hypomania; *Hea* = Poor Health.

The first two columns of Table 3 present intraclass correlations for the MMPI clinical scales. The intraclass correlation for MZA twins is a direct estimate of the broad heritability for each MMPI scale; the heritability estimate from the modeling procedure (V_g) incorporates all of the information from MZA and DZA pairs in our sample.

Degree of DZA resemblance was more variable than MZA similarity. Two factors may account for this. First, the number of DZA pairs is smaller than the number of MZA pairs. Second, the expected value of the DZA correlations is itself low. In a purely additive model, the expected value of the DZA correlation is half the MZA correlation. The expected value is even lower when nonadditive genetic effects are present (Lykken, McGue, Tellegen, & Bouchard, 1992). On both accounts—small sample size and smaller expected value—the DZA correlations will suffer more from sampling error than the MZA correlations.

The remainder of Table 3 presents parameter estimates and chi-square goodness-of-fit tests for the MMPI clinical scales. Do the results of the MZA and DZA samples agree according to a simple genetic model? If not, the fit of the full model will be poor, as indexed by a significant chi-square. All 13 clinical scales fit the additive genetic model well, although the *F* (Frequency) scale had a marginal fit. Within this context, the lower-than-expected DZA correlation for the *F* scale suggests the potential influence of nonadditive genetic factors.

For each of the 13 validity and clinical scales, setting the heritability parameter to zero resulted in a significant worsening of the fit of the model, reflecting significant (nonzero) heritability. The heritability estimates (V_g) in Table 3 provide the best indi-

cator of the extent to which variability in the clinical scales is influenced by genetic factors. As a result of small sample sizes, these estimates have large sampling errors. The heritability estimates ranged from .24 to .61 (M = .43). These are generally consistent with heritability estimates reported for personality inventories in twins reared together (Carey & Rice, 1983; Gottesman, 1963; Gottesman & Goldsmith, 1994; Loehlin & Nichols, 1976).

Table 4 summarizes the intraclass correlations and results of biometric modeling for the Wiggins content scales. Evaluation of the full model indicates that the Religous Fundamentalism scale had a significantly poor fit and the Feminine Interests scale had a marginal fit. All other scales fit the additive genetic model adequately. As was the case for the clinical scales, all of the Wiggins scales had significant heritability as indexed by a significant worsening of model fit when the V_g parameter was set to zero. Heritability estimates ranged from .26 to .62 (M = .44). Again, these estimates are consistent with prior reports on heritability of personality traits. Given the poor or moderately weak fit to the full additive genetic model for the Religous Fundamentalism and Feminine Interests Scales, coupled with evidence of nonzero heritability, the lower than expected DZA correlation for each of these scales suggests the potential influence of nonadditive genetic effects.

To be consistent with previous analyses from this sample, and because of the relatively small sample size, we included DZOS pairs in the analysis sample. As a follow-up analysis, we removed the DZOS pairs and reran our statistical models.[3] For the validity scale and clinical scale analyses, three scales (F, Hysteria, and Hypochondriasis) fit the full additive genetic model poorly ($p < .05$). However, it was not possible to set the heritability parameter to zero for any scale; doing so resulted in a significant worsening of model fit. As above, this suggests the possibility of nonadditive genetic influences on these scales. A nonsignificant trend for DZOS correlations on the MMPI to be higher than DZSS correlations may have influenced the fit of the full model; however, small sample sizes precluded meaningful statistical comparison between DZOS and DZSS twin pairs. Heritability estimates for the reduced sample differed by an average of .02 (range = 0 to .07). Absolute differences between intraclass correlations for DZSS pairs and the full DZA sample averaged .07 (range = 0 to .25). For the Wiggins scales, four scales (Feminine Interests, Religous Fundamentalism, Organic Symptoms, and Poor Health) fit the full model poorly. As for the clinical scales, however, setting the heritability parameter estimate to zero resulted in a significant worsening of model fit.

Table 3
Intraclass Correlations, Parameter Estimates, and Model Tests for Twins Reared Apart: MMPI Clinical Scales Without K Correction

	Intraclass correlation				Model tests (χ^2)	
Scale	MZA	DZA	V_g[a]	V_u[b]	Full[c]	Diff.[d]
L	.44	−.04	.37	.63	3.1	11.2
F	.52	−.02	.45	.55	4.6	19.0
K	.55	.19	.55	.45	1.4	24.8
Hs	.37	.13	.35	.65	0.2	10.5
D	.31	.13	.31	.69	0.6	7.3
Hy	.29	.13	.26	.74	1.9	6.7
Pd	.62	.14	.61	.39	2.7	30.2
Mf	.40	.04	.36	.64	1.6	10.2
Pa	.32	.03	.28	.72	1.2	7.0
Pt	.60	.14	.60	.40	3.9	27.4
Sc	.64	.07	.61	.39	3.6	31.6
Ma	.56	.23	.55	.45	0.2	27.5
Si	.30	.22	.34	.66	1.0	8.6

Note. N = 65 unique pairs of monozygotic twins reared apart (MZA) and 54 unique pairs of dizygotic twins reared apart (DZA). MMPI = Minnesota Multiphasic Personality Inventory; *L* = Lie; *F* = Frequency; *K* = Correction; *Hs* = Hypochondriasis; *D* = Depression; *Hy* = Hysteria; *Pd* = Psychopathic Deviate; *Mf* = Masculinity–Femininity; *Pa* = Paranoia; *Pt* = Psychasthenia; *Sc* = Schizophrenia; *Ma* = Hypomania; *Si* = Social Introversion.
[a] Genetic variance, or heritability. [b] Unique environmental variance. [c] Chi-square (df = 2) for full model. All values are nonsignificant ($p \geq .1$), indicating an adequate fit of the model to the data. Note that this model fixed the parameter for shared environmental variance to zero to reflect rearing status. [d] Chi-square (df = 1) for difference between full model and reduced model with V_g = 0. All values are significant ($p < .01$), indicating significant (nonzero) heritability.

Table 4
Intraclass Correlations, Parameter Estimates, and Model Tests for Twins Reared Apart: MMPI Wiggins Content Scales

	Intraclass correlation				Model tests (χ^2)	
Scale	MZA	DZA	V_g[a]	V_u[b]	Full[c]	Diff.[d]
Soc	.23	.22	.27	.73	0.6	5.8
Dep	.48	.03	.44	.56	3.0	14.3
Fem	.43	−.04	.36	.64	4.1	11.8
Mor	.36	.19	.39	.61	2.4	14.1
Rel	.63	−.12	.57	.43	**11.8**	22.1
Aut	.41	.19	.42	.58	0.4	14.1
Psy	.65	.12	.62	.38	2.2	34.7
Org	.48	.02	.42	.58	2.4	15.2
Fam	.50	.32	.50	.50	1.0	24.1
Hos	.42	.05	.37	.63	1.3	12.0
Pho	.57	.34	.59	.41	0.3	31.7
Hyp	.48	.06	.45	.55	2.5	15.1
Hea	.30	.01	.26	.74	1.2	5.7

Note. N = 65 unique pairs of monozygotic twins reared apart (MZA) and 54 unique pairs of dizygotic twins reared apart (DZA). MMPI = Minnesota Multiphasic Personality Inventory; *Soc* = Social Maladjustment; *Dep* = Depression; *Fem* = Feminine Interests; *Mor* = Poor Morale; *Rel* = Religious Fundamentalism; *Aut* = Authority Conflict; *Psy* = Psychoticism; *Org* = Organic Symptoms; *Fam* = Family Problems; *Hos* = Manifest Hostility; *Pho* = Phobias; *Hyp* = Hypomania; *Hea* = Poor Health.
[a] Genetic variance, or heritability. [b] Unique environmental variance. [c] Chi-square (df = 2) for full model. Value in boldface (*Rel*) is statistically significant ($p < .01$), indicating poor model fit. All other values are nonsignificant ($p \geq .1$), indicating adequate model fit. Note that this model fixed the parameter for shared environmental variance to zero to reflect rearing status. [d] Chi-square (df = 1) for difference between full model and reduced model with V_g = 0. All values are significant ($p < .05$), indicating significant (nonzero) heritability.

[3] Details of these analyses are available from the authors on request.

Heritability estimates for the reduced sample differed from the full sample by an average of .03 (range = 0 to .06), and intraclass correlations for DZSS pairs differed absolutely from the full DZA sample by an average of .11 (range = .02 to .28).

Analysis of Age Effects on Heritability

Although we corrected MMPI scores for the effects of age, age^2, and sex, it is possible given the wide age range in the sample that this does not satisfactorily control for possible age-related changes in heritability. We conducted a follow-up analysis to address this question (see Footnote 3). Using scores corrected for age, age^2, and sex, we fit a model to the twin data in which heritability was dependent on the age of the twin pair. Four scales showed significant effects of age on heritability. Three scales tapping health complaints (Hypochondriasis, Organic Symptoms, and Poor Health) showed decreasing heritability with age, and one scale (Family Problems) showed increased heritability for older twins in the sample. For example, parameter estimates (albeit with large standard errors) suggested that for organic symptoms, heritability is .80 at age 20 and decreases to .16 by age 60. For Family Problems, heritability is .19 at age 20 and increases to .75 at age 60.

Multivariate Profile Analysis

Because MMPI scales are intercorrelated, we conducted a series of multivariate profile analyses (Morrison, 1967) to test for overall pair similarity across the scales and to determine whether overall elevation and shape of the MMPI profile are influenced by genetic factors. MANOVA tests of twin similarity in MMPI profile elevation and shape were conducted separately by zygosity. The analysis of profile elevation is comparable to analyzing each twin's average scale elevation for the MMPI's scales. The analysis of profile shape involved calculation for each twin of the deviation of each MMPI scale from the average. This allows for evaluation of the configural patterning of high and low scales (the peaks and valleys of the MMPI profile), controlling for differences in elevation. In contrast to an analysis of profile scatter, this method allows for assessment of whether twin pairs tend to have elevations and depressions on the same scales. For each zygosity-group analysis, a significant multivariate F indicates that there is more variability between twin pairs than there is within twin pairs. For twins reared apart, such significant within-pair similarity can only be the result of genetic influences; the effect should be stronger for MZA than for DZA twins. Hence, the analysis does not test genetic effects directly by way of a joint MZA vs. DZA comparison, but significant results are suggestive of genetic effects by virtue of the twin groups' rearing status.

In the analysis of profile elevation for the standard validity scales and 10 clinical scales, the multivariate F was significant for MZA pairs, $F(64, 67) = 3.67, p < .001$; there was a nonsignificant trend for DZA pairs, $F(53, 54) = 1.21, p < .10$. For the Wiggins scales, the multivariate test for the elevation effect was significant for MZA pairs, $F(64, 67) = 4.09, p < .001$. There was a smaller but significant effect for DZA pairs, $F(53, 54) = 1.67, p < .05$. Together, these effects suggest significant heritability for general deviance from the norm with respect to MMPI scores. In the analysis of profile shape for the validity and clinical scales, the multivariate F was significant for MZA twins, $F(768, 709) = 1.84, p < .001$. There was a smaller but significant effect for DZA twins, $F(636, 550) = 1.18, p < .05$. For the Wiggins scales, there was a significant profile shape effect for MZA twins, $F(768, 709) = 1.89, p < .001$, and a trend toward significance for the DZA twins, $F(636, 550) = 1.14, p < .10$. The significant effects here suggest that the form of psychopathology is also influenced by genetic factors.

Discussion

These results indicate that roughly 44% of the variance in MMPI clinical and content scales is accounted for by genetic variability, corroborating what is now a well-established finding in the personality literature. The profile analyses also support the notion that genes exert an influence on the level and form of personality traits related to psychiatric disturbance (Dworkin, Burke, Maher, & Gottesman, 1977). The findings are particularly striking given that the twins spent the majority of their formative years out of contact with each other and in different rearing environments. Our analysis of age-related effects on heritability is generally consistent with prior research indicating stability of genetic influences on personality across the life span (cf. Goldsmith, 1983; McGue et al., 1993), although we also found evidence that heritability of MMPI scales tapping health concerns tended to decrease at older ages. This may reflect the increasing salience of uniquely experienced age-related physical problems later in life. The magnitude of nonshared environmental influences on personality dissimilarity was also consistent with previous findings from the twin and adoption literature. Given the reared-apart status of the sample, we did not evaluate models that included shared environmental influences. Although most twins did have some degree of contact with each other prior to reunion, we argue that this small shared environmental influence did not have a demonstrable effect; if such contact were significant, our full additive model (which set the shared environmental variance parameter to zero) would have routinely evidenced a poor fit to the data.

Without large psychiatric samples of twins, adoptees, or affected relatives within families that have been tested with the MMPI (cf. Gottesman & Shields, 1972), we cannot assert with confidence that particular MMPI configurations indicate specific psychiatric diagnoses with greater or lesser roles for genetic variation. However, based on the results of this study, together with clinical experience and research on the relationships between code types and diagnoses, we suggest that code types composed of scales with higher versus lower heritability estimates may provide clues to genetic influences on diagnostic status (Gottesman, 1962). In general, the scales associated with the "psychotic tetrad" and right side of the clinical profile tended to have higher heritabilities (average = .53), whereas the "neurotic" triad scales appeared to be somewhat less heritable (average = .31). Thus, antisocial personality, bipolar disorder, and some of the varieties of schizophrenia might be in the former group, whereas somatoform and dissociative disorders might belong in the latter. Diagnoses associated with "mixed" scale configurations leave inferences about genetic influence in a middle ground. Between-scale differences in heritability were

not directly tested in this study. In addition, because of the influence of relatively small sample size on standard errors of heritability estimates, our interpretation of the scale patterns above should be viewed as tentative.

This sample probably precludes one relationship between psychiatric disorder and personality—that what is inherited is really the psychiatric disorder and that responses to the MMPI items are secondary to the disorder. A small sample such as this could not have ascertained a sufficient number of individuals with specific *DSM–IV* (4th ed. of the *Diagnostic and Statistical Manual of Mental Disorders;* American Psychiatric Association, 1994) disorders to produce twin resemblance on all the MMPI scales associated with these diagnoses (cf. Greene, 1988). By the same logic, our results cannot be used to justify any conclusions, pro or con, about the diagnostic validity of the MMPI or the heritability of overt psychiatric disorder (cf. Gottesman & Shields, 1972; Moldin, Gottesman, Erlenmeyer-Kimling, & Cornblatt, 1990). Hence, we stress that the results apply to heritability of scales that reflect, with varying degrees, combinations of symptom variance and personality trait variance and that are associated with psychiatric disorders.

A number of MMPI scale results invite further comment. There is an apparent discrepancy between the heritability estimates for the MMPI Depression scale ($h^2 = .31$) and the Wiggins Depression scale ($h^2 = .44$); however, the sample size raises concerns about whether this is a statistically reliable difference between heritability estimates for the two scales. Differential heritability, if present, could be due to differing item composition. The MMPI Depression scale was developed empirically. This method may have selected some items that do not reflect complaints specific to depression but that differentiate people in a state of depression from normals—so-called Subtle items. Indicators of depression in the Wiggins scales are concentrated in the Poor Morale and Depression scales, which tap despondency and frank depressive affect, respectively. State effects of depression, as indexed more strongly by the MMPI Depression scale, might show lower heritability because they would require both members of a pair to be depressed (or well) when completing the MMPI. But lasting traits, such as the chronic dysthymia or characterological depression that are tapped by the Wiggins scales, might show stronger genetic effects (cf. Katz & McGuffin, 1993). It is also possible that the greater homogeneity and internal consistency of the Wiggins scales compared with the MMPI clinical scales could result in relatively higher heritability estimates for the Wiggins scales because variance due to error of measurement is accounted for by the unique environmental variance component of standard biometric models.

A confound to the interpretation of our results involves potential effects of the similarity of subcultural rearing environments on within-pair similarity. Subculture differences within Western civilization can result in regional MMPI differences (e.g., Butcher & Pancheri, 1976). Such systematic differences (between Scotland and the southern United States, for example) could induce positive twin resemblance for twin pairs reared within each subculture, and this might be misinterpreted as a genetic effect. However, DZA pairs as well as MZA pairs generally tended to be reared in similar subcultures. Consequently, if subculture rearing effects are inducing within-pair similarity, DZA correlations should be higher than expected under our genetic model and the model should not fit the data. This was not the case, as evidenced by the good model fits in Tables 3 and 4. This suggests that subculture pair similarity in rearing was not sufficiently large to affect the major conclusions about heritability.

What, then, is the effect on MMPI profiles of two twins being reared in highly discrepant cultures? There are two such pairs in this sample, too few for statistical analysis but useful as case studies in profile similarities and differences. For reasons of confidentiality, specific scores and demographic information cannot be provided, but for both pairs the identical twins were reared on different continents, in cultures speaking different languages, and under radically different political systems. Absolute differences for each of the pairs on the MMPI's clinical scales were computed and averaged across the 3 validity scales and 10 clinical scales. Pair 1 had an absolute average difference of 8.5 *T*-score points; Pair 2 had an average difference of 5.1 *T*-score points. For purposes of comparison, the average *T*-score difference for all MZA twin pairs in the sample was 8.6 points. A final point of comparison is provided by the two pairs of MZ twins with the most discrepant MMPI profiles (highest summed absolute difference across the 10 clinical scales and 3 standard validity scales). These twin pairs had averaged absolute *T*-score differences of 16.4 and 17.2 points on the MMPI's validity and clinical scales. The first pair of twins was later to become discordant for a major affective disorder with psychosis. The second pair of twins was discordant for a severe head injury more than 5 years prior to testing, resulting in probable right-side brain damage to the affected twin.

These case study examples are illustrative of our broader findings. Our results indicate that there are significant genetic influences on personality traits and on patterns of traits. This is manifest in similarity among identical twins reared apart, even in the face of large cultural differences. At the same time, our statistical models highlight the importance of nonshared environmental effects that contribute to personality dissimilarity. This type of effect is reflected by the "discrepant" twin pairs described above. Understanding the causal influence of such clear divergence-inducing factors, as well as more subtle ones, will provide crucial insight into important nonshared environmental contributions to personality development (Juel-Nielsen, 1965; Shields, 1962).

The present findings extend previously reported results from the MISTRA project. For all of the MMPI clinical scales and the Wiggins content scales, significant heritability was evident, with suggestions of nonadditive or epistatic genetic effects for three of the MMPI's scales. Despite being separated from each other very early in life and spending childhood and early adulthood living apart, MZ twins were strikingly similar on a variety of personality scales related to the expression of psychopathology—often as similar as previously reported MMPI findings for MZ twins reared in the same household. Pattern of MMPI scales and degree of deviation from the norm were also shown to be influenced by genetic factors. Although the MMPI was used in this study, we expect that the findings should generally be applicable to the MMPI-2. Although caution is clearly warranted when interpreting data from a relatively small and selected sample, the results provide strong evidence for genetic

influences on personality correlates of psychopathology as indexed by the most widely used self-report instrument.

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