Normal and Abnormal Personality Traits: Evidence for Genetic and Environmental Relationships in the Minnesota Study of Twins Reared Apart

Kristian E. Markon
Robert F. Krueger
Thomas J. Bouchard, Jr.
Irving I. Gottesman
University of Minnesota, Twin Cities Campus

ABSTRACT Recent studies have demonstrated substantial correlations between normal and abnormal personality traits. Yet little is known about how these correlations are mediated genetically and environmentally: Do normal and abnormal personality traits stem from the same underlying genes and environments? We addressed this question using data from 128 monozygotic and dizygotic twin pairs in the Minnesota Study of Twins Reared Apart (MISTRA). Additive genetic and nonshared environmental correlations between scales of the Minnesota Multiphasic Personality Inventory (MMPI)—an index of abnormal personality—and the Multidimensional Personality Questionnaire (MPQ)—an index of normal personality—were estimated. Results indicated that phenotypic correlations between normal and abnormal personality were mediated by genetic as well

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Correspondence concerning this article may be addressed to Kristian E. Markon or Robert F. Krueger, Department of Psychology, University of Minnesota, Elliott Hall, 75 East River Road, Minneapolis, Minnesota 55455. Electronic mail may be sent to mark0060@tc.umn.edu or krueg038@tc.umn.edu.

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as environmental factors, although the magnitude of genetic mediation tended to be larger overall. Moreover, the patterns of phenotypic, genetic, and environmental relationships among the scales were similar, suggesting that influences on normal and abnormal personality act through systems common to both. It is suggested that future research focus on the neurogenetic substrates of these shared systems and how dysfunction in these systems influences development of disordered personality.

Recently, renewed attention has been focused on the relationship between normal and abnormal personality functioning. Distinctions between adaptive and pathological personality have increasingly been questioned (e.g., Grove & Tellegen, 1991; Strack & Lorr, 1997), with many reevaluating how normal personality relates to personality disorder and other behavioral pathology (e.g., Krueger et al., 1996; Trull & Sher, 1994). The purpose of the present study was to estimate the relative contributions of genetic and environmental influences to covariation between normal and abnormal personality traits, using a sample of twins reared apart. By partitioning correlations among a comprehensive set of normal and abnormal traits into genetic and environmental components, we sought to specify not only how the two forms of personality are related, but also why.

Existing Models of the Relationship Between Normal and Abnormal Personality

In part, a renewed focus on relationships between normal and abnormal personality resulted from the introduction in DSM III (American Psychiatric Association, 1980) of the Axis II personality disorders (Strack & Lorr, 1994, 1997). The introduction of Axis II initiated a number of debates regarding classification and diagnostic structure of personality disorders, focusing not only on the use of categorical versus dimensional schemes, but also on the structure those categories or dimensions might take (e.g., Grove & Tellegen, 1991; Millon & Klerman, 1986). Although these issues have not been completely resolved (e.g., Clark, 1999; Loranger, 1999), there is increasing agreement that abnormal personality is often better modeled as being continuously, rather than categorically, distributed (e.g., Livesley, 1991; Widiger, 1993; Widiger & Frances, 1994), and that the structure of abnormal personality parallels that of normal

personality (e.g., Costa & Widiger, 1994; Eysenck, 1994; Schroeder, Wormworth, & Livesley, 1992). Numerous studies have demonstrated correlations between continuous measures of normal personality traits and personality disorder (e.g., Cloninger & Svrakic, 1994; Costa & McCrae, 1990), as well as joint factor loadings of measures of normal and abnormal personality (DiLalla, Gottesman, Carey, & Vogler, 1993; Schroeder et al., 1994). These relationships are not limited to a few particular domains, but rather have been demonstrated across a number of traits (e.g., Costa & Widiger, 1994) and trait models (e.g., DiLalla, Gottesman, Carey, & Vogler, 1993; Schroeder et al., 1992).

Consensus that normal and abnormal personality are related, however, has not been associated with a similar consensus as to why this is the case (e.g., Widiger, Verheul, & van den Brink, 1999). A number of models have been proposed to explain the relationship between normal and abnormal personality, including hypotheses that normal-range personality traits can influence or cause disorder; that abnormal personality traits can affect other, normal-range, personality traits; and that normality and disorder exist in a spectrum, each representing portions of a continuum (Widiger et al., 1999). There is empirical support for many of these models, and any one or all of them may account for observed correlations between normal and abnormal personality traits to some extent (Widiger et al., 1999).

Models such as these are phenotypic in nature. That is, a given abnormal or normal phenotype is invoked as an explanation for the other, or the two phenotypes are spoken of as existing on a continuum. The emphasis in existing models is on observed variation in normal and abnormal traits, rather than the sources of observed covariation between them. An alternative possibility, therefore, is to model the relationship between normal and abnormal traits in terms of their etiologic antecedents—to ask why, in addition to how, the two types of traits are related. Especially important in this regard is how covariation between normal and abnormal personality traits is genetically and environmentally mediated. Normal and abnormal personality traits may be correlated, for instance, because they are both directly or indirectly influenced by some common set of genes, a common set of environmental factors, or some combination thereof.

One might hypothesize, for example, that environmental traumas exacerbate neuroticism into borderline symptomatology, or that a common set of genes predisposes one toward both social dominance

and psychopathy. Such models make predictions about factors influencing otherwise exogenous phenotypes of interest, and cannot be evaluated in the absence of a genetically informed design, such as a twin or adoption study. Without information about how the magnitude of a phenotypic relationship varies with genetic relatedness, for instance, environmental and genetic effects are not readily disentangled. Moreover, even within a genetically informed design, such hypotheses cannot be evaluated using standard heritability estimates. Although two traits may both show heritable variance, the covariance between the two may be due to some environmentally mediated factor. That is, not all of the variance in a trait is genetic, and that which is may or may not explain covariance with another trait. In fact, genetic and environmental influences on a trait may operate in opposite directions, such that genetic factors tend to induce a positive correlation between the two traits, and environmental factors tend to induce a negative correlation between the two traits, or vice versa.

Joint genetic and environmental effects such as these can, however, be resolved through the estimation of genetic and environmental correlations (Carey, 1988; Carey & DiLalla, 1994; Neale & Cardon, 1992), which reflect the extent to which covariation between two traits can be explained by genetic and environmental effects, respectively. That is, in addition to estimating the genetic and environmental influences underlying variation in a single trait, it is possible to estimate the covariances between genetic influences on two or more traits or covariances between environmental influences on two or more traits. One can say that two traits are genetically correlated to the extent that their genetic influences covary, and that the two traits are environmentally correlated to the extent that their environmental influences covary. Genetic correlation can be thought of as the bivariate analogue of heritability, in that heritability is estimated via the correlation between relatives' standing on the same trait, whereas a genetic correlation or covariance is estimated via the correlation between individuals' standings on one trait and their relatives' standings on a second trait—that is, the cross-correlation. In a twin study, for example, heritability of aggression would be estimated by means of the correlation between aggression in twins and aggression in the cotwins; the genetic correlation between aggression and extraversion, in contrast, would be estimated by means of the correlation between aggression in the twins and extraversion in the cotwins.

Evidence of Genetic and Environmental Covariance Between Normal and Abnormal Behavior

Very few published studies of genetic and environmental relationships between normal and abnormal behavior exist (Carey & DiLalla, 1994). Those that do, moreover, have tended to focus not on abnormal personality per se, but on psychopathological symptomatology more generally, and have largely been limited to the relationships among neuroticism, depression, and anxiety. Eaves, Eysenck, and Martin (1989), for example, estimated the genetic correlation between neuroticism and anxiety to be approximately .9 (.89 among females and .94 among males), and the estimated genetic correlation between neuroticism and depression to be approximately .75 (.76 among females and .73 among males; Jardine, Martin, & Henderson, 1984, present variations of these analyses using data from the same study). Similarly, Roberts and Kendler (1999) estimated the genetic correlation between neuroticism and lifetime diagnosis of depression to be .47. Together, the results of these two studies suggest a common genetic liability toward neuroticism, state symptoms of anxiety, and depression. However, they provide no information about other normal personality traits that have been phenotypically associated with personality pathology, nor about the relationship between normal personality and abnormal personality traits.

The only existing study of genetic and environmental correlations between normal and abnormal personality traits was conducted by Jang and Livesley (1999) using a volunteer sample of twins reared together. Jang and Livesley (1999) estimated genetic and environmental correlations between scores on the Dimensional Assessment of Personality Pathology (DAPP-BQ; Livesley & Jackson, in press)—an inventory derived from DSM-III personality disorder symptomatology—and an abbreviated measure of the five higher-order factors of the NEO-PI, the NEO-FFI (Costa & McCrae, 1988). Substantial genetic and environmental correlations were found for four of the NEO scales—neuroticism, extraversion, agreeableness, and conscientiousness—with those for openness being notably lower. Genetic correlations were larger in magnitude than environmental correlations, but both exhibited similar patterns.

The results of this study are important because they suggest that broad patterns of relationship between sets of normal and abnormal

personality traits can be attributed to genetic influences. Environmental factors, in contrast, may act within these patterns to shape underlying genetic predispositions into distinctive, but correlated, phenotypes. For example, it may be that depressive and anxious personality traits are strongly genetically correlated, but relatively weakly environmentally correlated; one might conclude that depressive and anxious traits emerge from the same genetic sources, but that distinct environmental experiences push the manifestation of these sources toward depression or anxiety in particular (Kendler, Neale, Kessler, Heath, & Eaves, 1992).

Rationale for the Present Study

Studies such as that of Jang and Livesley (1999), utilizing twins reared together, provide vital information about the relative contributions of genetic and environmental effects to covariation between normal and abnormal personality. As has been previously noted, however (e.g., Carey, 1992; DiLalla, Carey, Gottesman, & Bouchard, 1996), reared-together twin studies make certain assumptions about twin relationships that may or may not always be valid. In particular, such study designs assume that reciprocal influences between monozygotic twins, such as imitation, are not greater than those between dizygotic twins. These assumptions are critical to studies of covariation between normal and abnormal personality, where sustained exposure to a cotwin's maladjustment could induce elevated or lowered levels of normal personality traits such as neuroticism, extraversion, or agreeableness. Correlations resulting from indirect influences of one twin's psychopathology on the cotwin, if greater in magnitude among monozygotic than dizygotic twins, could inflate estimates of genetic correlation.

Adoption studies provide powerful controls for the effects of reciprocal interaction, as relatives are raised in separate home environments with no opportunity for contact. The purpose of the present study was to estimate genetic and environmental correlations between measures of normal and abnormal personality in an such a sample, the Minnesota Study of Twins Reared Apart (MISTRA). This sample provides a particularly unusual and powerful opportunity to estimate joint genetic and environmental influences on normal and abnormal personality, as it combines benefits of twin and adoption designs. Essentially, it affords the opportunity to estimate the

correlation between measures of abnormal personality in one individual and normal personality in another individual, each of whom are genetically identical but were separated much or most of their lives. By comparing such correlations to those obtained for other separated twins, sharing half of their genes on average, one obtains an estimate of joint genetic and environmental effects on personality, free from considerations of reciprocal interaction. The mean age of separation in the sample was less than 1 year, and individuals were separated, on average, for over 30 years (DiLalla et al., 1996). Presumably, then, any similarity between their measures of normal and abnormal personality should be due to the genetic material that they share, because shared environmental experiences have been minimal.

Abnormal and normal personality traits were assessed in the current study by scales of the MMPI (Hathaway & McKinley, 1983) and the Multidimensional Personality Questionnaire (MPQ; Tellegen, 2000), respectively. These inventories are well suited for an investigation of genetic and environmental influences on abnormal and normal personality. The MMPI, for example, is the most widely used inventory of personality pathology (Butcher, Graham, & Ben-Porath, 1995), and its scale validities and other psychometric properties have, concomitantly, been thoroughly investigated and documented (e.g., Dahlstrom & Welsh, 1960; Dahlstrom, Welsh, & Dahlstrom, 1972). The psychometric properties of the MPQ, similarly, are excellent, and have been extensively documented in a number of settings (e.g., Krueger et al., 1996; Reise & Waller, 1990; Waller, Tellegen, McDonald, & Lykken, 1996). Finally, MMPI and MPQ primary scales have been shown to be extensively correlated (DiLalla, Gottesman, Carey, & Vogler, 1993). Given that there is a substantial amount of heritable variance underlying these scales (DiLalla et al., 1996; DiLalla, Gottesman, Carey, & Bouchard, 1999; Tellegen et al., 1988), we sought to determine the extent to which genetic variance in each is shared or correlated with the others.

METHODS

Participants

Participants in the present study were adult (mean age = 43, *SD* = 13) monozygotic and dizygotic twin pairs from the Minnesota Study of Twins

Reared Apart (MISTRA) who had been tested by June 1999. Twins, and occasionally their families, were ascertained in various ways, from a number of countries, typically by media accounts of the study and referral by professionals. Zygosity was determined by blood and serum assays and physical similarity measures (estimated probability of misclassification $< .001$). Further details of ascertainment and other procedures have been described in a number of other sources (e.g., Bouchard, 1994; DiLalla et al., 1996; Tellegen et al., 1988).

Data from a total of 119 twin pairs and 3 sets of triplets (2 MZ sets and 1 set with one MZ pair and a DZ member) were available for the present study. Seventy-four monozygotic pairs (45 female, 29 male) comprised 67 MZ twin pairs and 7 pairs created from the three triplet sets by pairing each MZ member of each set with the other MZ members of that set. Fifty-four dizygotic pairs comprised 52 DZ twin pairs (26 female same-sex DZ pairs, 12 male same-sex DZ pairs, and 14 opposite-sex DZ pairs) and two opposite-sex pairs created from the triplet set by pairing the DZ member of the set with the other two members of that set. Including opposite-sex and same-sex DZ pairs together in analyses is consistent with previous published analyses of MISTRA data, which suggest bias introduced by combining the two types of DZ pairs is relatively small (DiLalla et al., 1996). The relatively small sample of opposite-sex DZ pairs, moreover, precludes separate analyses of opposite-sex and same-sex DZ pairs.

Measures

Three sets of MMPI scales were included in our analyses—the first set comprised the 3 validity scales and 10 clinical scales of the MMPI; the second set comprised the Wiggins content scales (Wiggins, 1966), and the third set comprised the Block factor scales (Block, 1965). The clinical scales were not K-corrected. Use of the clinical and Wiggins scales allows comparison between results obtained with a group of empirically keyed scales and a group of scales derived through content and internal consistency considerations and increases the applicability of the present study to the large body of research conducted with those scales. The Wiggins scales, moreover, have no item overlap, and therefore provide a means to control for inflation in scale intercorrelation due to nonexclusive item content. The external validities of the validity, clinical, content, and factor scales of the MMPI are extensively documented (e.g., Dahlstrom & Welsh, 1960; Dahlstrom, Welsh, & Dahlstrom, 1972).

The MPQ is a factor-analytically derived self-report inventory, designed as a comprehensive measure of normal personality functioning (Tellegen, 2000). The 300-item version was used in the present study. The MPQ comprises 11

primary scales (Well-Being, Social Potency, Achievement, Social Closeness, Stress Reaction, Alienation, Aggression, Control, Harm Avoidance, Traditionalism, and Absorption) that can be scored as three higher-order factor scales (Positive Emotionality, Negative Emotionality, and Constraint). The external validity of the MPQ has been demonstrated in a number of settings (e.g., Caspi et al., 1997; Harkness, Tellegen, & Waller, 1995; Krueger et al., 1996; Waller, Binet, & Farney, 1994; White & Depue, 1999), and its psychometric properties are excellent. Scales do not overlap in item content, and reliabilities are good (for Well-Being, $\alpha = .90$; Social Potency, $\alpha = .82$; Achievement, $\alpha = .88$; Social Closeness, $\alpha = .92$; Stress Reaction, $\alpha = .89$; Alienation, $\alpha = .87$; Aggression, $\alpha = .82$; Control, $\alpha = .82$; Harm Avoidance, $\alpha = .88$; Traditionalism, $\alpha = .90$; Absorption, $\alpha = .91$; Tellegen, 1985). Detailed characteristics of the scales and their constituent items are provided by Tellegen and Waller (in press).

Statistical Analyses

To correct for potential biases in model fitting, MMPI and MPQ scale scores were first adjusted for effects of age and sex (McGue & Bouchard, 1984). Each scale was regressed on age, sex, the in-teraction between age and sex, and age squared, using data from family members in addition to twin data ($N = 411$) when available. Residuals from these regressions were used in subsequent analyses and model fitting.

For lower-order scale analyses, MZ and DZ intraclass covariance matrices corresponding to each of the 286 interinventory MMPI-MPQ scale combinations (i.e., 13 MMPI clinical and validity scales * 11 MPQ scales = 143 combinations; 13 Wiggins content scales * 11 MPQ scales = 143 combinations) were calculated using the S+ statistical language (Mathsoft, 1997). Genetic and environmental covariances and correlations were then estimated by fitting bivariate Cholesky decomposition models (Neale & Cardon, 1992) corresponding to each of these 286 scale combinations via maximum likelihood, using the computer program Mx (Neale, Boker, Xie, & Maes, 1999). For higher-order scale analyses involving the MPQ factor scales and MMPI Block scales, MZ and DZ intraclass covariance matrices containing covariances between the three MPQ and two MMPI factor scales were calculated; genetic and environmental correlations between the factor scales were then estimated jointly by fitting a multivariate Cholesky decomposition model to these matrices. In the present study, all genetic effects were assumed to be additive—that is, genes were assumed to act independently of one another, and to "breed true" from generation to generation (Falconer, 1960). Given the reared-apart status of the twins, all environmental effects were assumed

to be nonshared. Environmental factors, in other words, were assumed to be uncorrelated between twins.

The Cholesky model is a multivariate extension of the factor model commonly used in univariate twin analyses of genetic and environmental components of variance (see Fig. 1 for a path diagram of a bivariate Cholesky model). In the univariate case, genetic effects are modeled by assuming two genetic factors, each influencing the trait value of one twin, and correlated to the extent that the twins share genes (.5 for DZ twins and 1.0 for MZ twins). Environmental effects, similarly, are modeled by assuming two environmental factors, each influencing the trait value of one twin, and correlated to the extent that the twins are both exposed to these environmental factors. In twins reared apart, environments are modeled as uncorrelated within twin pairs.

In the bivariate Cholesky model, there are not two genetic factors, one for each twin, but rather, four genetic factors, two factors for each twin. The first

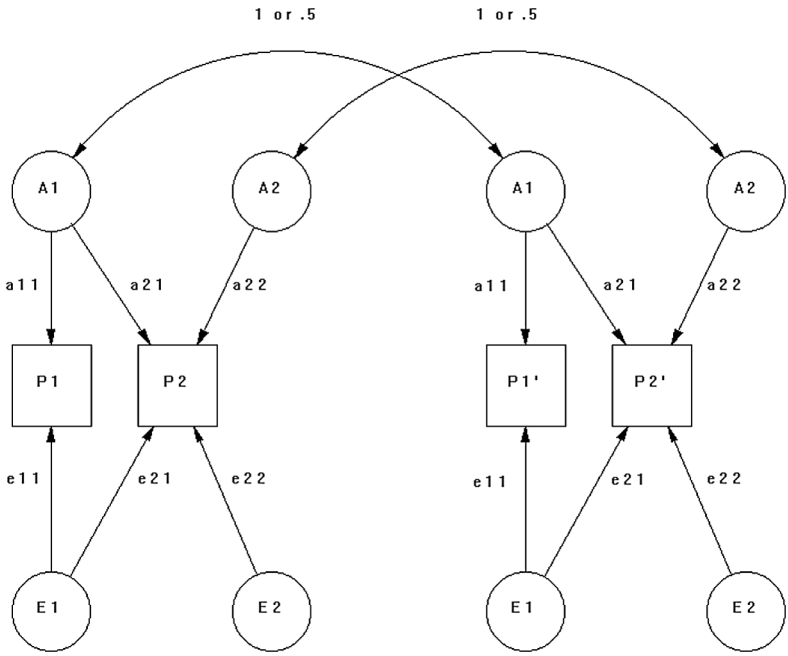

Figure 1
Path model for a bivariate Cholesky decomposition of variance into additive genetic and environmental sources. The two phenotypes are designated as P1 and P2 for the first twin and P1′ and P2′ for the second twin.

factor of each twin is assumed to influence all of the traits measured in that twin, and the second factor is assumed to influence all but the first trait in that twin. The first genetic factor of one twin, moreover, is correlated with the first genetic factor of the second twin to the extent the twins share the same genes (again, .5 for DZ twins and 1.0 for MZ twins); the second genetic factor of one twin is also correlated with the second genetic factor of the second twin to the extent the twins share the same genes on average. Environmental effects in a bivariate Cholesky model are treated analogously (see Figure 1). However, in twins reared apart, these environmental factors are modeled as uncorrelated within twin pairs—that is, the first environmental factor of one twin is not correlated with that of the cotwin, nor are the second environmental factors of each twin correlated.

The Cholesky factor model is particularly useful because it provides a mathematically attractive means of calculating genetic and environmental correlations between traits. The matrix of genetic variances and covariances can be calculated as $A = XX'$, where X is the matrix of loadings of each trait on the genetic Cholesky factors—that is, in Fig. 1, the parameters a_{11}, a_{21}, and a_{22}. The matrix of environmental variances and covariances, similarly, can be calculated as $E = YY'$, where Y is the matrix of loadings of each trait on the environmental factors. Using corresponding genetic and environmental variances, each of the genetic and environmental covariances can be rescaled as correlations. The genetic or environmental correlation between two scales is equal to the genetic or environmental covariance between the scales divided by the square root of the product of the corresponding genetic or environmental variances. These equations are derived from standard tracing rules for path diagrams; one would obtain equivalent results by tracing through coefficients in the Cholesky path diagram; in Fig. 1, for example, the genetic covariance between traits 1 and 2 would be equal to $a_{11}a_{21}$.

For each genetic and environmental correlation 99% and 95% confidence intervals were calculated using the likelihood-based confidence interval routine in Mx. A likelihood-based confidence interval is calculated as the two values, greater and less than the estimate, that decrease the likelihood relative to that of the estimate just by the desired significance level. In the present case, each interval comprised the two values greater and less than the estimated correlation that decrease the likelihood at a significance level of $p = .01$ and $p = .05$ (Neale & Miller, 1997).

Bootstrap confidence intervals (99% and 95%) were also calculated for the phenotypic correlations. Each bootstrap confidence interval was bias-corrected and accelerated, based on 10,000 bootstrap samples with twin pair taken as the sampling unit to correct for dependent observations (Efron & Tibshirani, 1998). Bias-corrected and accelerated bootstrap confidence intervals have been shown to approximate actual coverage well when exact values are known (Efron & Tibshirani, 1998).

RESULTS

MPQ Primary Scales and MMPI Clinical and Validity Scales

Phenotypic Pearson correlations between the MPQ scales and MMPI clinical and validity scales were estimated using sex- and age-corrected scores of individual participating twins and are presented in Table 1. Estimated additive genetic correlations between the MMPI clinical and validity scales and MPQ scales are presented in Table 2; estimated nonshared environmental correlations between the two sets of scales are presented in Table 3.

The phenotypic correlations presented in Table 1, ranging from −.68 to .79 (median absolute r = .18, mean absolute r = .22), are similar to those calculated previously in a high-risk sample (DiLalla, Gottesman, Carey, & Vogler, 1993), although slightly smaller in magnitude. The estimated genetic correlations ranged from −.77 to .88 (median absolute value = .26, mean absolute value = .30), and the estimated environmental correlations ranged from −.64 to .68 (median absolute value = .14, mean absolute value = .18).

All of the MPQ scales exhibited significant genetic correlations with the empirically keyed MMPI scales. However, the distribution of genetic correlations varied widely across MPQ scales, with the Stress Reaction and Alienation scales exhibiting the most significant genetic correlations with MMPI clinical and validity scales. Absorption, Aggression, Harm Avoidance, and Well-Being were also significantly genetically correlated with a number of MMPI scales. The fewest significant genetic correlations were observed with Social Closeness and Traditionalism, each only significantly genetically correlated with one MMPI scale, Si.

The pattern of environmental correlations was similar in that Stress Reaction, Alienation, Aggression, and Well-Being each were significantly environmentally correlated with a number of empirically keyed MMPI scales. In contrast to the pattern of genetic correlations, however, Social Closeness exhibited a number of significant environmental correlations with the MMPI clinical and validity scales, as did Traditionalism to a lesser extent. Moreover, Absorption and Harm Avoidance, which exhibited a number of significant genetic correlations, were significantly environmentally correlated with one and none of the MMPI scales, respectively.

Table 1
Phenotypic Correlations Between MPQ Primary Scales and MMPI Validity and Clinical Scales

	MPQ										
MMPI	wellbe	socpot	achiev	socclos	stress	alien	aggres	control	harmav	tradit	absorp
Lie (L)	**.14**	−.11	.02	.06	***−.26***	***−.18***	***−.40***	***.27***	.09	***.22***	***−.21***
Frequency (F)	***−.23***	.06	.03	***−.29***	***.39***	***.56***	***.28***	***−.25***	***−.28***	***−.17***	***.26***
Correction (K)	***.20***	.04	−.04	**.17**	***−.68***	***−.48***	***−.40***	***.19***	.07	−.11	***−.30***
Hypochondriasis (Hs)	***−.26***	−.07	.06	**−.13**	***.47***	***.35***	.12	−.12	**−.16**	.02	**.18**
Depression (D)	***−.57***	**−.18**	−.10	***−.21***	***.52***	***.29***	.03	−.04	−.03	.05	.00
Hysteria (Hy)	**−.16**	.09	.07	**.15**	.07	−.07	***−.20***	.01	−.04	−.08	.00
Psychopathic Deviate (Pd)	***−.42***	**.20**	.08	−.16	***.40***	***.51***	***.31***	**−.19**	***−.23***	***−.17***	***.20***
Masculinity-Femininity (Mf)	−.08	***.18***	.08	−.01	**.17**	−.10	−.11	.01	.07	***−.28***	***.25***
Paranoia (Pa)	***−.41***	.00	**.14**	−.10	***.46***	***.34***	.12	−.06	**−.18**	−.10	***.20***
Psychasthenia (Pt)	***−.47***	−.09	−.06	***−.22***	***.79***	***.55***	***.36***	**−.15**	−.12	**.14**	***.26***
Schizophrenia (Sc)	***−.37***	−.02	.00	***−.29***	***.64***	***.63***	***.41***	***−.20***	***−.26***	−.03	***.37***
Hypomania (Ma)	.10	***.32***	***.18***	.01	***.27***	***.47***	***.42***	***−.28***	***−.29***	***−.25***	***.44***
Social Introversion (Si)	***−.34***	***−.33***	**−.15**	***−.34***	***.45***	***.40***	***.21***	−.12	−.07	***.22***	.02

Note. Correlations whose 95% confidence intervals do not include zero are shown in bold. Correlations whose 99% confidence intervals do not include zero are shown in bold-faced italics. wellbe = Wellbeing; socpot = Social Potency; achiev = Achievement; socclos = Social Closeness; stress = Stress Reaction; alien = Alienation; aggres = Aggression; control = Control; harmav = Harm Avoidance; tradit = Traditionalism; absorp = Absorption.

Table 2
Genetic Correlations Between MPQ Primary Scales and MMPI Validity and Clinical Scales

	MPQ										
MMPI	wellbe	socpot	achiev	socclos	stress	alien	aggres	control	harmav	tradit	absorp
Lie (L)	.13	−.21	−.11	.01	−.08	−.27	**−.41**	.27	.05	.32	−.27
Frequency (F)	−.31	.25	.37	−.16	**.41**	***.88***	.26	**−.37**	***−.52***	−.07	***.47***
Correction (K)	.21	.02	−.26	.02	***−.77***	***−.75***	***−.56***	.29	.11	−.24	***−.55***
Hypochondriasis (Hs)	−.18	.15	.31	.03	***.72***	***.78***	.25	−.26	−.10	.05	.23
Depression (D)	**−.53**	.04	.05	−.18	**.60**	.34	−.11	−.13	−.14	.14	.01
Hysteria (Hy)	.08	.34	.30	.24	.17	−.03	−.40	.01	.09	−.21	−.05
Psychopathic Deviate (Pd)	***−.48***	**.32**	.33	−.22	***.48***	***.65***	.32	−.28	**−.35**	−.16	**.33**
Masculinity-Femininity (Mf)	−.22	***.62***	.30	.23	.16	−.23	.12	.02	.13	−.22	.23
Paranoia (Pa)	−.32	.33	**.53**	−.25	**.52**	.35	.05	−.02	**−.47**	.07	***.61***
Psychasthenia (Pt)	***−.48***	.01	.22	−.11	***.86***	***.67***	.33	−.14	−.06	.23	***.37***
Schizophrenia (Sc)	**−.35**	.12	.28	−.25	***.68***	***.83***	***.55***	−.31	**−.31**	.06	***.60***
Hypomania (Ma)	.03	.26	**.37**	.06	***.41***	***.78***	***.52***	***−.47***	**−.38**	−.25	***.70***
Social Introversion (Si)	**−.47**	**−.44**	.17	**−.46**	***.61***	***.68***	.19	−.27	−.24	**.39**	−.06

Note. Correlations whose 95% confidence intervals do not include zero are shown in bold. Correlations whose 99% confidence intervals do not include zero are shown in bold-faced italics. wellbe = Wellbeing; socpot = Social Potency; achiev = Achievement; socclos = Social Closeness; stress = Stress Reaction; alien = Alienation; aggres = Aggression; control = Control; tradit = Traditionalism; harmav = Harm Avoidance; absorp = Absorption.

Table 3
Environmental Correlations Between MPQ Primary Scales and MMPI Validity and Clinical Scales

	MPQ										
MMPI	wellbe	socpot	achiev	socclos	stress	alien	aggres	control	harmav	tradit	absorp
Lie (L)	.15	−.03	.08	.11	***−.41***	−.09	***−.36***	**.24**	.12	.12	−.15
Frequency (F)	−.16	−.07	−.18	***−.39***	***.35***	***.32***	***.27***	−.17	−.08	***−.27***	.07
Correction (K)	.09	.00	.12	**.23**	***−.56***	**−.25**	***−.28***	.11	.03	−.02	−.06
Hypochondriasis (Hs)	**−.26**	−.21	−.08	**−.22**	***.31***	.06	.04	−.06	−.18	.01	.14
Depression (D)	***−.64***	***−.29***	−.19	**−.21**	***.50***	***.28***	.09	.01	.03	−.02	−.03
Hysteria (Hy)	***−.32***	−.06	−.04	.10	.07	−.07	−.09	.00	−.10	.00	.03
Psychopathic Deviate (Pd)	***−.33***	.11	−.13	−.11	***.31***	***.34***	***.31***	−.09	−.09	−.18	.06
Masculinity-Femininity (Mf)	−.02	−.11	−.04	−.14	.20	−.02	***−.25***	.00	.02	***−.36***	***.28***
Paranoia (Pa)	***−.47***	−.16	−.03	−.01	***.44***	***.31***	.12	−.03	−.04	**−.22**	−.04
Psychasthenia (Pt)	***−.38***	−.15	***−.33***	***−.31***	***.68***	***.41***	***.39***	−.16	−.14	.06	.14
Schizophrenia (Sc)	***−.31***	−.12	**−.22**	***−.33***	***.58***	***.42***	***.29***	−.09	−.17	−.13	.10
Hypomania (Ma)	***.29***	***.42***	.10	−.04	.07	.14	***.34***	−.12	−.20	**−.22**	.20
Social Introversion (Si)	−.21	**−.21**	***−.32***	**−.25**	***.33***	**.23**	**.23**	−.02	.06	.11	.09

Note. Correlations whose 95% confidence intervals do not include zero are shown in bold. Correlations whose 99% confidence intervals do not include zero are shown in bold-faced italics. wellbe = Wellbeing; socpot = Social Potency; achiev = Achievement; socclos = Social Closeness; stress = Stress Reaction; alien = Alienation; aggres = Aggression; control = Control; tradit = Traditionalism; harmav = Harm Avoidance; absorp = Absorption.

MPQ Primary Scales and MMPI Wiggins Scales

Phenotypic correlations between sex- and age-corrected MPQ scales and MMPI Wiggins scales are presented in Table 4. Estimated additive genetic correlations between the MMPI Wiggins scales and MPQ scales are presented in Table 5; estimated nonshared environmental correlations between the two sets of scales are presented in Table 6.

The phenotypic correlations presented in Table 4 are similar in magnitude to those obtained with the MMPI clinical and validity scales, ranging from −.46 to .75 (median absolute r = .17, mean absolute r = .21). The estimated genetic correlations ranged from −.90 to .96 (median absolute value = .27, mean absolute value = .32), and the estimated environmental correlations ranged from −.50 to .70 (median absolute value = .12, mean absolute value = .16).

Overall, the pattern of genetic correlations between MPQ scales and MMPI content scales was similar to that observed with the empirically keyed MMPI scales. Stress Reaction and Alienation, again, appeared significantly genetically correlated with a number of MMPI content scales, as did Absorption, Aggression, and Well-Being, to a somewhat lesser extent. Social Closeness was again significantly genetically correlated with the fewest number of MMPI scales, being negatively genetically correlated with only the Social Maladjustment scale.

The pattern of environmental correlations between MPQ scales and MMPI content scales was similar to that of the genetic correlations and that of the environmental correlations between the MPQ and empirically keyed MMPI scales. Stress Reaction and Alienation, again, were significantly environmentally correlated with a number of MMPI content scales. Harm Avoidance exhibited the fewest significant number of environmental correlations, not being significantly environmentally correlated with any of the MMPI content scales. Environmental correlations between Social Closeness and MMPI content scales appeared to reach significance slightly more often than the corresponding genetic correlations. In contrast, environmental correlations between Absorption and MMPI content scales were less, and significant less often, than the corresponding genetic correlations.

MPQ Higher-Order Scales and MMPI Block Scales

Phenotypic, genetic, and environmental correlations between sex- and age-corrected MPQ higher-order scales and MMPI Block scales are

Table 4
Phenotypic Correlations Between MPQ Primary Scales and MMPI Wiggins Scales

	MPQ										
MMPI	wellbe	socpot	achiev	socclos	stress	alien	aggres	control	harmav	tradit	absorp
Social Maladjustment	***−.31***	***−.37***	−.03	***−.43***	***.26***	***.21***	.06	.07	.01	**.14**	−.01
Depression	***−.46***	−.03	−.04	**−.17**	***.75***	***.53***	***.32***	***−.19***	−.09	.06	***.28***
Feminine Interests	.09	.06	−.03	***.18***	−.01	−.11	***−.17***	**.16**	***.25***	−.04	.13
Poor Morale	***−.37***	−.12	−.05	**−.15**	***.71***	***.52***	***.33***	−.14	−.10	**.16**	***.28***
Religious Fundamentalism	−.02	**−.15**	−.02	.00	−.03	.04	−.10	.04	.03	***.46***	−.06
Authority Conflict	−.04	.09	−.07	***−.19***	***.28***	***.50***	***.42***	***−.23***	−.08	.06	***.21***
Psychoticism	***−.18***	.03	**.13**	***−.21***	***.57***	***.66***	***.35***	−.10	**−.17**	.00	***.45***
Organic Symptoms	***−.25***	−.06	.02	−.13	***.52***	***.42***	***.22***	***−.19***	***−.21***	.03	**.19**
Family Problems	***−.19***	.14	***.19***	***−.22***	***.36***	***.45***	***.24***	***−.20***	***−.28***	***−.27***	***.31***
Manifest Hostility	***−.23***	**.14**	−.04	−.13	***.56***	***.45***	***.62***	***−.29***	**−.16**	.00	***.30***
Phobias	***−.23***	−.10	.05	−.08	***.58***	***.39***	**.16**	.00	.11	***.20***	***.21***
Hypomania	.07	***.25***	**.16**	.06	***.52***	***.43***	***.35***	***−.26***	−.14	−.05	***.46***
Poor Health	***−.20***	.01	.03	−.02	***.40***	***.27***	.06	**−.14**	−.15	−.07	***.17***

Note. Correlations whose 95% confidence intervals do not include zero are shown in bold. Correlations whose 99% confidence intervals do not include zero are shown in bold-faced italics. wellbe = Wellbeing; socpot = Social Potency; achiev = Achievement; socclos = Social Closeness; stress = Stress Reaction; alien = Alienation; aggres = Aggression; control = Control; tradit = Traditionalism; harmav = Harm Avoidance; absorp = Absorption.

Table 5
Genetic Correlations Between MPQ Primary Scales and MMPI Wiggins Scales

	MPQ										
MMPI	wellbe	socpot	achiev	socclos	stress	alien	aggres	control	harmav	tradit	absorp
Social Maladjustment	−.31	***−.90***	.16	***−.77***	.12	.24	−.22	.19	−.14	**.52**	−.18
Depression	**−.37**	.15	**.43**	−.07	***.78***	***.72***	.32	−.16	−.27	.15	***.47***
Feminine Interests	.08	***.49***	−.08	.34	.08	−.31	−.04	.23	**.40**	.01	.24
Poor Morale	***−.49***	.01	.32	−.12	***.87***	***.88***	**.37**	−.21	−.19	.31	**.37**
Religious Fundamentalism	−.10	**−.36**	−.02	−.11	.09	**.44**	−.10	−.02	.05	***.45***	−.20
Authority Conflict	−.10	.10	.13	−.11	**.56**	***.96***	***.60***	***−.48***	−.19	.05	**.48**
Psychoticism	**−.35**	.13	**.37**	−.26	***.70***	***.85***	**.41**	−.15	−.24	.13	***.59***
Organic Symptoms	−.06	.21	.38	−.06	***.69***	***.69***	**.39**	−.31	−.30	.03	***.48***
Family Problems	−.23	.26	.30	−.20	***.49***	***.66***	***.57***	***−.44***	**−.40**	−.25	***.61***
Manifest Hostility	−.27	.26	.19	.01	***.77***	***.67***	***.73***	−.20	−.20	−.04	***.50***
Phobias	**−.33**	−.05	.17	−.18	***.87***	***.45***	.27	.01	**.30**	.26	**.31**
Hypomania	−.06	.27	.29	.19	***.69***	***.72***	***.51***	***−.55***	−.07	−.02	***.67***
Poor Health	−.13	.34	.31	.21	***.55***	***.65***	.21	−.35	−.12	−.07	.29

Note. Correlations whose 95% confidence intervals do not include zero are shown in bold. Correlations whose 99% confidence intervals do not include zero are shown in bold-faced italics. wellbe = Wellbeing; socpot = Social Potency; achiev = Achievement; socclos = Social Closeness; stress = Stress Reaction; alien = Alienation; aggres = Aggression; control = Control; tradit = Traditionalism; harmav = Harm Avoidance; absorp = Absorption.

Table 6
Environmental Correlations Between MPQ Primary Scales and MMPI Wiggins Scales

	MPQ										
MMPI	wellbe	socpot	achiev	socclos	stress	alien	aggres	control	harmav	tradit	absorp
Social Maladjustment	***−.30***	−.11	−.13	**−.25**	***.30***	.20	.19	.02	.08	−.06	.07
Depression	***−.50***	−.12	***−.36***	−.21	***.70***	***.35***	***.32***	−.20	.07	−.05	.11
Feminine Interests	.02	**−.25**	.01	.07	.01	.04	**−.23**	.07	.14	−.11	.05
Poor Morale	**−.23**	−.19	***−.31***	−.15	***.54***	.20	***.27***	−.07	.00	.04	.18
Religious Fundamentalism	.09	.03	−.04	.08	−.12	**−.26**	−.06	.06	.03	***.47***	.08
Authority Conflict	.07	.10	−.19	***−.28***	.04	.16	***.27***	−.05	.05	.11	−.03
Psychoticism	.12	−.04	−.05	−.16	***.41***	***.46***	***.30***	−.04	−.08	−.15	**.27**
Organic Symptoms	***−.33***	**−.26**	−.20	−.18	***.36***	.18	.10	−.11	−.11	.04	−.06
Family Problems	−.10	.08	.08	−.17	.16	**.26**	−.05	−.03	−.16	**−.27**	−.02
Manifest Hostility	−.09	.08	−.17	**−.23**	***.37***	**.23**	***.54***	***−.34***	−.12	.08	.16
Phobias	−.07	−.13	−.10	.02	**.24**	***.32***	.07	−.04	−.05	.15	.05
Hypomania	***.31***	**.27**	.08	−.04	***.33***	.15	**.24**	−.02	−.18	−.03	**.25**
Poor Health	−.21	−.18	−.11	−.13	***.33***	.05	−.02	−.01	−.17	−.06	.11

Note. Correlations whose 95% confidence intervals do not include zero are shown in bold. Correlations whose 99% confidence intervals do not include zero are shown in bold-faced italics. wellbe = Wellbeing; socpot = Social Potency; achiev = Achievement; socclos = Social Closeness; stress = Stress Reaction; alien = Alienation; aggres = Aggression; control = Control; tradit = Traditionalism; harmav = Harm Avoidance; absorp = Absorption.

presented in Table 7. The pattern of correlations between the Block and MPQ higher-order scales generally reflects and summarizes the patterns of correlations observed between the MPQ primary scales and the MMPI content and clinical scales. The largest phenotypic, genetic, and environmental correlations were observed between MPQ Negative Emotionality and Block Ego Resiliency ($r_p = -.71$, $r_g = -.86$, $r_e = -.53$) and between MPQ Constraint and Block Ego Control ($r_p = .39$, $r_g = .57$, $r_e = .21$). Significant phenotypic and environmental correlations were also observed between MPQ Negative Emotionality and Block Ego Control ($r_p = -.29$, $r_e = -.37$), and a significant genetic correlation was observed between MPQ Positive Emotionality and Block Ego Control ($r_g = -.40$).

DISCUSSION

Although, recently, there have been numerous demonstrations of substantial relationships between normal and abnormal personality traits, such evidence has almost exclusively been limited to phenotypic analyses. The sources of these relationships between normal and abnormal personality, including influences of genetic and environmental factors generally, have largely remained unknown. The results of the present analyses help elucidate the shared genetic and environmental etiologies of normal and abnormal personality and suggest that genetic and environmental factors both account for relationships between normal and abnormal personality to some

Table 7
Correlations Between MPQ Higher-Order Scales and MMPI Block Scales

	MPQ								
	Positive Emotionality			Negative Emotionality			Constraint		
MMPI	r_p	r_g	r_e	r_p	r_g	r_e	r_p	r_g	r_e
Ego Control	−.11	**−.40**	.04	***−.29***	−.18	***−.37***	***.39***	***.57***	.21
Ego Resiliency	**.20**	−.11	***.37***	***−.71***	***−.86***	***−.53***	.04	−.09	.17

Note. Correlations whose 95% confidence intervals do not include zero are shown in bold. Correlations whose 99% confidence intervals do not include zero are shown in bold-faced italics. r_p = phenotypic correlation; r_g = genetic correlation; r_e = environmental correlation.

extent. Three characteristics of our results, in particular, are relevant to the general question of how relationships between normal and abnormal personality are mediated: first, our results suggest that traits related to negative affect and absorption account for many relationships between normal and abnormal personality; second, overall, genetic factors seem to account for much of the covariance between normal and abnormal personality; and finally, phenotypic, genetic, and environmental patterns of relationship between normal and abnormal personality traits seem to be similar.

Major Areas of Relationship Between Normal and Abnormal Personality

The first noteworthy characteristic of our results is that there appeared to be strong phenotypic, genetic, and environmental correlations between MPQ scales reflecting negative affect and many of the MMPI scales. This is demonstrated most concisely in the large higher-order scale correlations between MPQ Negative Emotionality and Block Ego Resiliency, but can also be seen in correlations between the lower-order scales, particularly between the MPQ Stress Reaction and Alienation scales and many of the MMPI scales. Such findings are consistent with previous literature, such as factor analyses (e.g., Block, 1965; Costa, Zonderman, McCrae, & Williams, 1985; Johnson, Butcher, Null, and Johnson, 1984; Waller, 1999) suggesting traits such as general maladjustment, neuroticism, and negative affectivity are major dimensions underlying the MMPI and other measures of abnormal personality (e.g., Costa & Widiger, 1994; Schroeder et al., 1992). Broad associations between the MMPI scales and negative affectivity scales of the MPQ also corroborate numerous findings that neuroticism and related normal personality traits act as general, nonspecific predictors of abnormal personality and other forms of psychopathology (e.g., Krueger, 1999a; Krueger et al., 1996; Widiger et al., 1999). Results of this study extend those general findings, suggesting that genetic and, to a lesser extent, environmental factors, both underlie the negative affectivity that characterizes much of the relationship between normal and abnormal personality.

Absorption was also consistently phenotypically and genetically correlated with a number of the MMPI scales (see Tables 1, 2, 4, and 5). In contrast to negative affective traits such as stress reaction and alienation, however, absorption did not exhibit substantial

environmental correlations with many of the abnormal personality traits as measured by the MMPI. Absorption appears particularly strongly related to psychotic symptomatology, as reflected in the hypomania and psychoticism content scales, and in the Ma, Sc, and Pa clinical scales. Relationships with psychoticism are consistent with the content of absorption items, which reflect the degree to which an individual "is emotionally responsive to engaging sights and sounds. . .thinks in images and has synasthetic and other 'crossmodal' experiences. . .can become absorbed in vivid recollections and imaginings. . .[and] experiences episodes of expanded awareness and other altered states" (Tellegen, 1985). Many of these characteristics, generally reflecting a detachment from experience of external stimuli, aptly summarize the primary symptoms of psychotic phenomena. Previous studies have documented relationships between absorption and psychotic symptomatology (Allen & Coyne, 1995; Kaven, 1992; but see also DiLalla & Gottesman, 1995); our results suggest that covariance between absorption and psychotic symptomatology is due almost entirely to common genetic influences on both.

A third major area of overlap between the MPQ and MMPI suggested by our results is between scales reflecting impulsivity, disinhibition, and antisocial behavior. This is most apparent in the higher-order scale correlations presented in Table 7—specifically, between MMPI Ego Control and MPQ Constraint—but can also be seen to a lesser extent in the lower-order scale correlations, between scales such as the Wiggins Authority Conflict and Family Problems and MPQ Control. This overlap is consistent with recent literature suggesting that disorders such as antisocial personality and substance abuse form a major dimension of psychopathology (Krueger, 1999b) strongly related to disinhibitory personality characteristics (Krueger, 1999a; Krueger et al., 1996). Our results suggest that the covariance between disinhibitory personality characteristics and externalizing symptomatology is at least partially due to substantial correlation between the genetic influences on each.

Relative Strengths of Genetic and Environmental Correlations

A second general characteristic of our results is that, overall, genetic correlations between the MMPI and MPQ scales appear somewhat

larger than the environmental correlations. The trend is somewhat more pronounced for the Wiggins scales than for the clinical and validity scales of the MMPI—perhaps due to item overlap in the latter—but the difference is small: the median absolute values of the genetic and environmental correlations, for example, were .26 and .14, respectively, for the clinical scales (Wilcoxon signed-rank $z = 6.14$; $p < .001$), and .27 and .12 for the content scales (Wilcoxon signed-rank $z = 7.67$; $p < .001$). Greater magnitudes of genetic correlations relative to environmental correlations were also observed in the single previous behavioral genetic study of normal and abnormal personality traits (Jang & Livesley, 1999).

One explanation for the greater size of genetic correlations relative to environmental correlations is that nonshared environmental variance comprises measurement error as well as valid nonshared environmental influences. If measurement errors are assumed to be uncorrelated, they necessarily reduce the percent of nonshared environmental variance in scales available for correlation. Although measurement error also decreases the genetic variance in a set of scales, it does not necessarily reduce the proportion of genetic variance in any given scale that may covary with another. It is therefore possible that measurement error increases the amount of nonshared environmental variance that does not covary between scales, thereby decreasing the magnitude of the nonshared environmental correlations relative to that of the genetic correlations.

Measurement error, however, is indistinguishable from uniqueness in the present study. Thus, another interpretation of the greater magnitude of genetic correlations relative to environmental correlations is that nonshared environmental factors, relative to genetic factors, tend to contribute to what is unique about a given trait. Covariance between normal and abnormal personality would then be attributable more so to shared genetic influences on both. As has been suggested before (Jang & Livesley, 1999; Kendler et al., 1992), covariation between normal and abnormal personality traits may reflect genetic factors that act in a broad sense through temperament or similar processes. Environmental factors, under such a model, would then shape these broadly acting genetic factors into the particular phenotypic forms that are observed (e.g., depression, as opposed to anxiety).

Both interpretations are consistent with the pattern of higher-order scale correlations: although the genetic correlations are slightly larger than the environmental correlations, the difference in magnitude is

much smaller than is the case with the lower-order scales. For example, the mean absolute values of the higher-order scale genetic and environmental correlations were .37 and .28, respectively; the median absolute values were .29 and .29 (Wilcoxon signed-rank $V = 15$; *ns*). Although it is possible that greater reliability of the higher-order scales accounts for the greater similarity in magnitude between the genetic and environmental correlations, it is also possible that the nature of the higher order factors as essential, orthogonal latent influences limits the extent to which different inventories will measure each of them differently. To the extent that the MMPI Psychopathic Deviate and MPQ Control scales each reflect different instantiations of a core disinhibitory process, for example, their environmental uniquenesses may be relatively large, and the environmental correlation between them relatively small; to the extent that MMPI Ego Control and MPQ Constraint both measure the core disinhibitory process, however, their environmental uniquenesses should be smaller, and the environmental correlation between them larger. Clearly more research is needed to resolve this problem.

Parallels Between Patterns of Genetic and Environmental Correlations

A third general trend notable in our results is that the patterns of genetic and environmental correlations largely resemble each other and that of the phenotypic correlations. Although there are some exceptions, generally where phenotypic correlations are relatively large, so too, are the genetic and environmental correlations; when phenotypic correlations are relatively small, the genetic and environmental correlations are, as well. The median absolute difference and Kendall's rank correlation between the phenotypic and genetic correlations, for example, is .11 and .75 ($p < .001$) for the clinical scales and .13 and .76 ($p < .001$) for the content scales. Similarly, the median absolute difference and rank correlation between the phenotypic and environmental correlations is .09 and .71 ($p < .001$) for the clinical scales and .10 and .61 ($p < .001$) for the content scales. The median absolute difference and rank correlation between the genetic and environmental correlations is .20 and .47 ($p < .001$) for the clinical scales and .23 and .38 ($p < .001$) for the content scales. Such similarities are corroborated by other multivariate genetic studies (e.g., Heath, Cloninger, & Martin, 1994; Heath, Madden, Cloninger, & Martin, 1999; Krueger, 2000;

Livesley, Jang, & Vernon, 1998), and are important because they suggest that the domains of genetic and environmental influence are nearly the same as are phenotypically observed. It is important to note that parallels between phenotypic, genetic, and environmental relationships are not mathematically necessary, given a Cholesky model. As was noted earlier, some (e.g., Heath, Cloninger, & Martin, 1994) have suggested that genetic and environmental influences on phenotypic structure may act in different directions. While this may be true for select traits, such effects appear to be the exception, not the rule.

Overall, our results suggest shared systemic substrates of normal and abnormal personality, whose differential functioning in different individuals leads to variation in personality. Consider, for example, that the environmental correlations are, by design, necessarily nonshared in nature: environmental correlations reported here reflect processes whereby environmental factors present for one twin, but not for the cotwin, increase the joint appearance of the two traits in the former. The genetic correlations, in contrast, reflect processes whereby genetic factors present for both twins increase the joint appearance of the two traits in the two twins. The fact that nonshared environmental factors tend to increase covariance between the same sets of traits as the genetic factors is important—the influence of environmental variables tends to act on those structures generated by the genetic background of the individual. Thus, to understand the etiology of abnormal personality, it becomes necessary to understand how genes generate the neural structures that underlie normal and abnormal personality variation, and how these structures process environmental stimuli. Abnormal personality functioning can be seen as the result of abnormalities in the developmental process, due to either genetic or environmental factors, or as the result of abnormal environmental variables impinging on a normally functioning neural substrate. Our results are consistent with the idea that these substrates ultimately are responsible for covariation between normal and abnormal personality and that genetic and environmental variation act through these substrates.

Caveats and Possible Extensions

Approximately 30% of the twins in MISTRA exhibit clinically deviant scores on two or more MMPI scales (cf. Dahlstrom & Welsh, 1960). In this sense, the sample represents a mixture of normal and abnormal variation, requisite for the purposes of estimating genetic and

environmental correlations between normal and abnormal personality traits. It is possible, however, that samples with an increased proportion of individuals exhibiting clinically significant personality variation might illuminate different relationships between normal and abnormal personality traits than were observed here. For example, some traits may not have demonstrated significant environmental correlations with one another in our sample because corresponding environmental influences tend to relate only to more extreme levels of pathological personality variation. Although the phenotypic relationships demonstrated here resemble those found in high risk samples (DiLalla, Gottesman, Carey, & Vogler, 1993; DiLalla, Gottesman, & Carey, 1993), it is important to replicate the findings of the present study using individuals from a more clinically saturated population.

An interesting question for future twin research is how genetic and environmental influences act to produce covariance between personality and diverse acute psychiatric conditions. Increasing evidence suggests that comorbidity among DSM Axis I conditions meaningfully reflects dimensions resembling those of personality (Krueger, Caspi, Moffitt, & Silva, 1998; Krueger, 1999b), and previous studies have demonstrated correlations between personality traits and Axis I conditions in cross-sectional as well as longitudinal contexts (Kendler, Neale, Kessler, Heath, & Eaves 1993; Krueger, 1999a; Krueger et al., 1996; Trull & Sher, 1994). As noted earlier, a few studies have demonstrated genetic and environmental sources of covariance between neuroticism and affective disorder symptomatology, especially major depression (Eaves et al., 1989; Jardine et al., 1984; Roberts and Kendler, 1999). These studies have not, however, addressed other personality traits or Axis I conditions that have demonstrated phenotypic relationships (e.g., constraint and substance use disorders; Krueger et al., 1996; Trull & Sher, 1994). It would be interesting to examine a more comprehensive set of disorders and personality traits to determine whether the present results generalize broadly to more state-like manifestations of psychopathology. It is possible that environmental factors contribute more to the relatively temporary, acute Axis I disorders than to long-standing personality traits such as examined here (cf. McGue et al., 1993).

Another intriguing possibility for future research is to consider the approach taken here in a longitudinal context (Roberts & Kendler, 1999). A genetically informed longitudinal design would allow one to delineate more clearly how the genetic and environmental influences

suggested by our results act over time to affect normal and abnormal personality. In such a design, in addition to estimating the phenotypic cross-trait correlation as in the present study, one would be able to estimate the cross-trait, cross-time correlation as well. Such information could be used to estimate the extent to which genetic factors influencing normal personality at one time are the same as those influencing abnormal personality later, the extent to which environmental factors predispose one to certain normal and abnormal personality traits simultaneously, and so forth. In effect, one could disentangle further the developmental patterns of genetic and environmental influences jointly influencing the two forms of personality. Roberts and Kendler (1999), for example, suggest that the same genetic factors predisposing one to neuroticism at one point in time are largely the same as those predisposing one to major depression at a later point in time. It remains to be seen whether or not such patterns generalize to other traits, and whether or not similar patterns hold generally for environmental influences on normal and abnormal personality. Environmental influences, for example, may tend only to exacerbate certain preexisting forms of normal personality, or otherwise act differently at different points in development (cf. Kendler, Karkowski, Corey, Prescott, & Neale, 1999).

Another question not completely addressed by the present study is why normal and abnormal personality traits differ. The analyses we present here are relevant to the question of why the two forms of personality resemble one another. Normal and abnormal forms of personality, however, are not identical, and the present analyses do not necessarily explain why this is so. It may be, for example, that the two forms of personality differ quantitatively, with each representing forms along a continuum. It may, however, also be that the two forms of personality, or subsets of normal and abnormal traits, are qualitatively distinct in some way (e.g., Meehl, 1992; Widiger & Frances, 1994). Just as our analyses clarify the nature of shared etiologies of normal and abnormal personality traits, it is necessary to determine the sources of variance unique to each. The present study suggests genetic factors seem to account for covariance between normal and abnormal personality somewhat more than environmental factors do; it may be that environmental factors account for what is unique about normal versus abnormal personality.

In spite of these concerns, analyses, such as those presented here, are useful because they help to characterize the sources of phenotypic

covariance between measures of normal and abnormal personality traits. In this regard, they complement and augment existing studies documenting the form of phenotypic relationships between normal and abnormal personality. Phenotypic analyses, useful in describing manifest patterns characterizing the link between the two forms of personality, cannot always elucidate the mechanisms by which these patterns arise. Particularly crucial in this regard is the question of whether the common presence of normal and abnormal traits results from factors arising internal or external to the individual—that is, the genetic and environmental antecedents of both. The results presented here suggest that normal and abnormal traits share genetic and environmental etiologies, acting through substrates common to both forms of personality. Our conclusions echo others who have suggested that abnormal and normal personality variation is etiologically continuous (e.g., Cloninger, 1987, in press; Eysenck, 1994; Widiger et al., 1999), as well as recent approaches to molecular genetic investigation of personality and psychopathology (e.g., Benjamin et al., 1996; Ebstein et al., 1996; Gottesman, in press; Kittles et al., 1999; Kotler et al., 1997). It is important in the future to continue to move from questions of if and how normal and abnormal forms of personality are related to questions of why they are related, in terms of the specific means by which neural systems underlying personality variation develop and operate.

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