A Twin Study of Normative Personality and DSM-IV Personality Disorder Criterion Counts: Evidence for Separate Genetic Influences

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Objective: Both normative personality and DSM-IV personality disorders have been found to be heritable. However, there is limited knowledge about the extent to which the genetic and environmental influences underlying DSM personality disorders are shared with those of normative personality. The aims of this study were to assess the phenotypic similarity between normative and pathological personality and to investigate the extent to which genetic and environmental influences underlying individual differences in normative personality account for symptom variance across DSM-IV personality disorders.

Method: A large population-based sample of adult twins was assessed for DSM-IV personality disorder criteria with structured interviews at two waves spanning a 10-year interval. At the second assessment, participants also completed the Big Five Inventory, a self-report instrument assessing the five-factor normative personality model. The proportion of genetic and environmental liabilities unique to the individual personality disorder measures, and hence not shared with the five Big Five Inventory domains,

Models of normative personality strive to achieve the most parsimonious way of describing individual differences in characteristic patterns of thinking, feeling, and behaving. Consensus has converged on a model with five dimensional factors that provides an adequate representation of normative personality (1). According to the "Big Five," the main features of normative personality can be summarized by scores on the five primary domains of extraversion, agreeableness, conscientiousness, neuroticism, and openness to experience (2).

According to DSM (3, 4), personality disorders constitute an enduring pattern of inner experience and behavior that deviates markedly from the expectations of the individual's culture and is manifested in at least two of the following domains: cognition, affectivity, interpersonal functioning, and impulse control. DSM-IV and DSM-5 list criteria sets for the same 10 distinct and categorical personality disorders, for which diagnosis were estimated by means of multivariate Cholesky twin decompositions.

Results: The median percentage of genetic liability to the 10 DSM-IV personality disorders assessed at wave 1 that was not shared with the Big Five domains was 64%, whereas for the six personality disorders that were assessed concurrently at wave 2, the median was 39%. Conversely, the median proportions of unique environmental liability in the personality disorders for wave 1 and wave 2 were 97% and 96%, respectively.

Conclusions: The results indicate that a moderate-to-sizable proportion of the genetic influence underlying DSM-IV personality disorders is not shared with the domain constructs of the Big Five model of normative personality. Caution should be exercised in assuming that normative personality measures can serve as proxies for DSM personality disorders when investigating the etiology of these disorders.

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requires a specific number of criteria to be endorsed. Numerous studies have concluded that DSM personality disorders can be characterized with the five-factor model of personality both conceptually and empirically (5, 6). Although the DSM constructs constitute the personality disorder measures most widely used by both clinicians and researchers, 18 different dimensional models of pathological personality have been published (7). Among the more widely used is the self-report Dimensional Assessment of Personality Pathology (8). Psychometric studies comparing the factor structure of this instrument with that of normative personality have found strong similarities with respect to the number of underlying domains and their content (9), the notable exceptions being a lack of evidence for an "openness to experience" dimension in pathological personality (10) and a psychoticism dimension in normative personality (11).

See related feature: Editorial by Dr. Skodol (p.590)

Normative personality traits were among the first psychological phenotypes to be studied using genetically informative samples, such as twins, and it is well established that these traits are moderately heritable, with genetic influences accounting for some 40%-60% of individual differences across the Big Five domains (12, 13). Only more recently have personality disorders been investigated using genetically informative samples, and results have demonstrated that the heritability of personality disorders as defined by the DSM criteria is similar in magnitude to that of normative personality (14, 15). Similarities in the extent of genetic influences have fueled speculation that largely the same etiological factors may underlie both normal personality and personality disorders (16). Shared genetic influences are plausible, given the large number of studies that have found overlapping genetic influences between neuroticism and axis I disorders, such as mood and anxiety disorders (17), and the substantial comorbidity typically observed between axis I disorders and personality disorders (18) attributable to shared genetic risk factors (19). However, to our knowledge, no study has had the appropriate data for directly assessing the extent of overlapping genetic etiology in normative personality and DSM personality disorders. The only study to investigate the extent of shared genetic influences between normative personality and a wide set of pathological personality domains (as indexed by the Dimensional Assessment of Personality Pathology Inventory) was performed by Jang and Livesley (16), and they concluded that there was evidence in favor of a common, broadly based genetic architecture. Two studies have estimated the amount of genetic liability to borderline personality disorder that was unique to this construct and not shared with the five domains of normative personality. Distel et al. (20) analyzed a large twin sample and found no unique genetic liability in the Personality Assessment Inventory-Borderline Features scale. More recently, Kendler et al. (21) estimated genetic correlations between the Big Five domains and the four subscales of the Dimensional Assessment of Personality Pathology that were judged to assess the core components of borderline personality disorder. They found substantial genetic correlations between borderline personality disorder and neuroticism, conscientiousness, and agreeableness. In summary, there are strong phenotypic associations between the Big Five domains and pathological personality traits, and the limited empirical evidence available suggests that this association may be largely due to shared genetic influences.

The aim of this study was twofold: first, to assess the phenotypic similarity between normative and pathological personality, and second, to investigate the extent to which genetic and environmental influences underlying individual differences in normative personality account for symptom variance across all 10 personality disorders in DSM-IV.

METHOD

Participants

Data for the study were drawn from two waves of a longitudinal population-based study of mental disorders among Norwegian twins, from which a sample was recruited from the Norwegian Institute of Public Health Twin Panel (22, 23). The first wave of data collection was carried out between 1999 and 2004, at which time 2,801 adult twins (44% of those eligible) born between 1967 and 1979 were assessed for DSM-IV axis I and axis II disorders. The sample consisted of 2,793 twins with valid data for DSM-IV personality disorders: 220 monozygotic male twins, 117 dizygotic male twins, 449 monozygotic female twins, 259 dizygotic female twins, and 340 dizygotic opposite-sex twin pairs in addition to 23 single twins.

The second wave of data collection was conducted in 2010 and 2011, and to limit the length of interviews, and thus maximize participation, the twins were reassessed only on a subset of the disorders from wave 1. Of the twins who participated in the first wave, 17 had withdrawn their consent to participate in further research, 14 had unknown addresses, and 12 had died, leaving 2,758 eligible twins who were invited to participate in a follow-up study. After two written reminders and a final telephone contact to nonresponders, 2,284 twins were interviewed in wave 2 (82.8% of those eligible). The distribution of zygosity groups for the pairs with complete personality disorder data at wave 2 was 154 monozygotic male twins, 76 dizygotic male twins, 358 monozygotic female twins, 179 dizygotic female twins, and 219 dizygotic opposite-sex twin pairs, comprising 986 twin pairs and 312 single twins who participated in the personality disorder interviews.

Zygosity was determined by a combination of questionnaire items and genotyping, and the misclassification rate has been estimated to be less than 1.0%, an error rate unlikely to be a source of bias.

Measures

In both waves, personality disorders were assessed using a Norwegian version of the comprehensive Structured Interview for DSM-IV Personality (SIDP-IV) (24). The specific DSM-IV criterion associated with each set of questions is rated using the following scoring format: 0=not present, 1=subthreshold, 2=present, and 3=strongly present. Behaviors, cognitions, and feelings that were prominent for most of the past 5 years are thought to be representative of an individual's long-term personality. At wave 1, all 10 of the DSM-IV personality disorders were assessed, whereas at wave 2, only six personality disorders (two from each DSM-IV cluster) were reassessed: paranoid, schizotypal, antisocial, borderline, avoidant, and obsessive-compulsive.

In wave 1, all but 231 (8.3%) of the interviews were conducted face-to-face, and the remainder were obtained by telephone. In wave 2, all interviews were conducted over the telephone. Interviewers at both waves were mainly senior clinical psychology graduate students or experienced psychiatric nurses, although some were clinical psychologists. Each twin in a pair was interviewed by a different interviewer.

The endorsement rates for the individual personality disorder criteria were in general too low for twin models to be fitted to DSM-derived categorical personality disorder diagnostic status. We therefore adopted a dimensional approach, in which we analyzed variables defined as the counts of positively endorsed criteria for each personality disorder. To improve statistical power, we treated criteria endorsed at the subclinical level (i.e., SIDP-IV criteria scored 1 or greater) as being positive. Finally, to lessen the impact of empty cells in the twin contingency tables during model estimation, symptom counts above 3 for each of the personality disorder variables were collapsed. For all personality disorders, this resulted in variable values ranging from 0 to 3. In the first publications from wave 1 (25), we investigated whether subthreshold endorsement of individual criteria on the SIDP-IV interview (i.e., a score of 1) should be considered qualitatively different from scores at a clinical level (rated 2 or 3) and whether the subthreshold count of endorsed criteria (a count less than the clinical threshold as given in DSM for the various personality disorders), should be treated qualitatively differently from scores above the clinical threshold. Results from multiplethreshold tests supported the notion that scores below and above the clinical threshold, both for individual criteria and for their counts, represent different levels of severity on the same liability dimension.

The interscorer reliability of the SIDP-IV interview was assessed at both waves. At wave 1, 70 interviews were recorded and scored by a second interviewer, and at wave 2, 95 interviews were recorded and scored by two additional interviewers. We calculated intraclass and polychoric correlations between subthreshold personality disorder counts, as judged by the different reviewers. At wave 1, intraclass correlations across the personality disorders ranged from 0.81 to 0.96 (polychoric correlations=0.80–0.99), while at wave 2, intraclass correlations ranged from 0.68 to 0.85 (polychoric correlations=0.81–091).

Normative personality was assessed by the Big Five Inventory (26), a self-report instrument completed by participants at wave 2. The Big Five Inventory, a self-report instrument developed to measure the five prominent domains of normative personality, consists of 44 items each scored on a 5-point scale. Extraversion is represented by eight items (Cronbach's alpha=0.85), agreeableness by nine items (alpha=0.71), conscientiousness by nine items (alpha=0.75), neuroticism by eight items (alpha=0.84), and openness by 10 items (alpha=0.79). The ordinal response options on these items were summed for each of the five domains, resulting in variables that were reasonably normally distributed, and in all subsequent analyses the Big Five Inventory variables were treated as continuous variables.

Statistical Analysis

To determine the degree of phenotypic association between the five Big Five Inventory domain sum scores and the subthreshold DSM-IV personality disorder criterion counts, we estimated polyserial correlations. Polyserial correlations are well suited to quantifying the association between a continuous variable and an ordinal variable and are less prone than Pearson correlations to underestimating this association if the ordinal variable is skewed or contains few categories (27).

The extent of shared genetic variance underlying normative and pathological personality was investigated using a series of multivariate twin models. Twin models allow the variance of an observed phenotype to be partitioned into three sources. The influence of additive genetic factors (referred to as A) can be inferred by the extent to which the correlation between monozygotic twins is twice as large as the correlation between dizygotic twins. Common environmental influences (referred to as C) are those that can be inferred if the correlation between monozygotic twins is equal in magnitude to the correlation between dizygotic twins. Any remaining variance in the phenotypes that cannot be accounted for by A or C is attributed to a unique environmental component (referred to as E), representing factors that contribute to making individuals within both monozygotic and dizygotic twin pairs dissimilar. Analogous to the way in which the variance in a phenotype can be partitioned into A, C, and E, the covariance between variables can be decomposed similarly by using a multivariate twin model. The extent of genetic and environmental overlap between the Big Five domains and each of the DSM-IV personality disorders was estimated by fitting a series of six-variate Cholesky twin decompositions to the Big Five Inventory domains and each of the 10 personality disorders measured at wave 1 and the six personality disorders measured at wave 2. The Cholesky decomposition is one of the most widely used multivariate twin analyses and contains as many latent A, C, and E factors as there are observed variables (28). The first five factors orthogonally contribute to the variance of a given personality disorder that is shared with the Big Five domains, and the last factor contributes variance that is unique to each personality disorder. Because of the large number of twin pairs required to estimate sex-specific effects, Cholesky path coefficients were constrained to be equal across sex, but separate thresholds and means were estimated for male twins and female twins, because there are systematic differences in the mean levels of Big Five traits and endorsement of personality disorder criteria across sex that may otherwise add an unwanted confounder to the interpretation of results from the biometric analyses if not taken into account.

The best-fitting models were selected on the basis of the lowest value for Akaike's information criterion, a fit statistic that jointly expresses the parsimony and explanatory power of a model (29).

For each personality disorder, we report the total genetic variance, the proportion that is unique, and the proportion that is shared with each of the Big Five domain constructs. Genetic and environmental correlations were also calculated and reported. The genetic correlation quantifies the extent to which the genetic variance in two phenotypes is shared.

All statistical analyses were performed in R, version 3.1.2 (30), and twin analyses were carried out using the free R-based

FIGURE 1. Polyserial Phenotypic Correlations Between Big Five Inventory Sum Scores and Personality Disorder Symptom Count Variables at Waves 1 and 2

A. Wave 1		Patan	oid schill	oid schitt	phypal Antisc	borde Borde	Hine Histin	Narcies	sistic Avoid	ant Depe	ndent obsee	Sive	- 1
Extra	aversion	-0.16	-0.29	-0.21	o	-0.1	0.15	0	-0.54	-0.27	-0.06		- 0.8
Agreeableness		-0.18	-0.14	-0.12	-0.24	-0.19	-0.11	-0.17	-0.13	-0.01	-0.14		- 0.4
Conscientiousness		-0.14	-0.15	-0.16	-0.28	-0.2	-0.14	-0.14	-0.2	-0.18	-0.05		- 0.2
Neuroticism		0.28	0.15	0.27	0.14	0.35	0.14	0.08	0.36	0.31	0.15		0.2
Openness		0.05	-0.04	0.06	0.12	0.09	0.15	0.15	-0.18	-0.12	0.1		0.8
	B. Wave a	2		Patanc	and schize	opal Antisc	bords Bould	etline Avoi	dart Op	sessive			
		Extraversion			-0.16	0.01	-0.17	-0.6	-0.0	8).8).6		
	Agreeableness			-0.26	-0.09	-0.2	-0.24	-0.2	-0.	2	0.8 0.4 0.2		
	Conscientiousness		isness	-0.16	-0.13	-0.31	-0.31	-0.22	2 -0.0).2		

0.51

0.1

0.44

-0.16

0.21

0.08

OpenMx structural equation package (31), an R extension developed to analyze twin and family data. Model parameters were estimated by means of full information maximum likelihood, an approach that makes use of all observed data. If missing data are considered missing at random, this method returns asymptotically accurate parameter estimates.

Neuroticism

Openness

0.36

0.03

0.32

0.1

0.13

0.14

RESULTS

Phenotypic Correlations

Phenotypic correlations between the Big Five domains and the personality disorder symptom counts assessed as wave 1 and wave 2 are provided in Figure 1. At wave 1, the Big Five domain construct with the largest absolute-valued median correlation across all 10 personality disorders was neuroticism, with a median correlation (r_m) of 0.21 (range=0.08 to 0.36). This was followed by conscientiousness (r_m =-0.15, range=-0.28 to -0.05), agreeableness (r_m =-0.14, range=-0.24 to -0.01), extraversion (r_m =-0.13, range=-0.54 to 0.15), and openness to experience (r_m =0.08, range=-0.18 to 0.15).

For the wave 2 data, the Big Five domain with the largest absolute-valued median correlation across the six personality disorders assessed concurrently was again neuroticism (r_m =0.34, range=0.13 to 0.51), followed in decreasing order by agreeableness (r_m =-0.20, range=-0.26 to -0.09), conscientiousness (r_m =-0.19, range=-0.31 to -0.06), extraversion (r_m =-0.16, range=-0.61 to 0.01), and openness to experience (r_m =0.09, range=-0.16 to 0.14).

Heritability, Unique to Each Personality Disorder and Shared With Big Five Inventory

For all the twin decompositions, dropping all the common environmental parameters (C) resulted in a more parsimonious solution than did the full ACE or CE models, as indicated by a lower Akaike's information criterion value. Results from the Cholesky AE models are summarized in Table 1. For the wave 1 data, the percentage of genetic variance in the personality disorder traits not shared with the Big Five domains ranged from 22% (avoidant) to 79% (schizotypal), with a median of 64%. Conversely, the percentage of

unique environmental variance in the personality disorders not shared with the Big Five domains ranged from 89% (avoidant) to 99% (schizoid), with a median of 97%.

Across the six personality disorders assessed at wave 2, the percentage of genetic variance not shared with the Big Five domains ranged from 18% (avoidant) to 58% (obsessive), with a median of 42%. The percentage of unique environmental variance at wave 2 ranged from 79% (avoidant) to 98% (obsessive), with a median of 96%. On average, the percentage of genetic variance that was unique to the six personality disorder traits assessed at both waves was 59% at wave 1 and 39% at wave 2, when the personality disorders and Big Five were measured concurrently.

To further facilitate comparisons across the personality disorder traits, we also present additive genetic and environmental variances unique to each personality disorder and the portions shared with the five factors of normative personality, in the form of stacked bar charts, in Figure 2.

Genetic Correlations

-0.2

-0.4

-0.6

-0.8

_1

Genetic correlations between the domains of normative personality and the personality disorder traits are presented in Figure 3. For the wave 1 data, the Big Five domain with the highest absolute-valued median genetic correlation across the 10 personality disorder traits was agreeableness (r=-0.40), followed by conscientiousness (r=-0.38), neuroticism (r=0.36), openness (r=0.20), and extraversion (r=-0.18). At wave 2, the median genetic correlations across the six personality disorders were, in order of decreasing absolute magnitude, neuroticism (r=0.56), conscientiousness (r=-0.54), agreeableness (r=-0.46), extraversion (r=-0.28), and openness (r=0.19).

DISCUSSION

Phenotypic Correlations

The pattern of correlations between DSM-IV personality disorder criterion counts and the Big Five domains

was largely consistent with that of previous meta-analyses (5, 32). The Big Five domain construct of neuroticism had the strongest association with personality disorder, and as did Samuel and Widiger (32), we observed the highest correlations between neuroticism and borderline, avoidant, and dependent personality disorder criterion counts, whereas the lowest correlations were found for antisocial, narcissistic, histrionic, and obsessive personality disorder criterion counts. The same pattern was also evident for the wave 2 data. Of the Big Five domains, openness to experience displayed the weakest association with personality disorder criterion counts, a finding consistent with results from both previous meta-analyses of DSM personality disorders and normative personality, in which no significant correlations were reported between personality disorders and openness to experience (5, 32). The weak association is also consistent with the lack of an openness factor reported by psychometric studies of the Dimensional Assessment of Personality Pathology, arguably because of a lack of indicators of openness (10).

Although we found that correlations between the Big Five domains and the personality disorder criterion counts were higher when assessed concurrently at wave 2, overall the difference was modest, considering that up to 10 years separated the waves. A reduction in the strength of association over longer intervals is to be expected, for although a high level of temporal stability is commonly reported for normative personality traits (33), the stability for personality disorders is typically found to be lower (34). Any age-specific genetic or transient environmental influence operating at wave 1 will

		Genetic Effects		Individual Environmental Effects				
Study Wave and Personality Disorder	a ²	% Shared With Big Five Inventory	% Unique	e ²	% Shared With Big Five Inventory	% Unique		
Wave 1								
Paranoid	0.20	41.5	58.5	0.80	3.5	96.5		
Schizoid	0.27	46.8	53.2	0.73	1.2	98.8		
Schizotypal	0.27	21.2	78.8	0.73	5.5	94.5		
Antisocial	0.41	30.7	69.3	0.59	3.4	96.6		
Borderline	0.36	48.3	51.7	0.64	2.3	97.7		
Histrionic	0.32	32.6	67.4	0.68	3.4	96.6		
Narcissistic	0.24	33.2	66.8	0.76	1.3	98.7		
Avoidant	0.35	78.4	21.6	0.65	10.6	89.4		
Dependent	0.30	39.3	60.7	0.70	3.7	96.3		
Obsessive- compulsive	0.26	23.7	76.3	0.74	1.4	98.6		
Wave 2								
Paranoid	0.19	79.4	20.6	0.81	3.2	96.8		
Schizotypal	0.29	47.3	52.7	0.71	4.3	95.7		
Antisocial	0.37	55.1	44.9	0.63	4.3	95.7		
Borderline	0.32	61.1	38.9	0.68	10.8	89.2		
Avoidant	0.28	81.8	18.2	0.72	20.9	79.1		
Obsessive- compulsive	0.22	42.1	57.9	0.78	2.4	97.6		

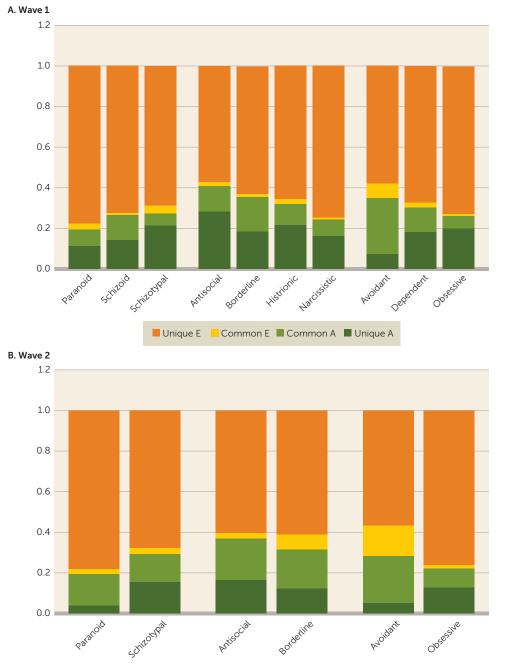
TABLE 1. Heritability (a^2) and Proportion of Unique Environmental Variance (e^2) Within Each Personality Disorder, Including the Proportions of Genetic and Environmental Variance Explained by the Big 5 Personality Domains

not contribute to shared genetic or environmental variance across the time points.

Shared and Unique Genetic Variance

For the personality disorders assessed at wave 2, the average genetic variance unique to the personality disorder criterion counts was 38.9%, suggesting a moderate influence of genetic factors specific to DSM-IV personality disorders. Although to our knowledge this is the first study to estimate the proportion of genetic liability in DSM personality disorders that is shared with normative personality, the limited evidence available from analyses of data based on dimensional models of pathological personality suggests that only modest genetic liability is specific to pathological personality (19, 35). Borderline personality disorder is the only personality disorder for which the shared genetic variance with the five-factor model of personality has been studied more extensively, but not by using the DSM criteria. In a large, extended twin sample, Distel et al. (20) found that all genetic variance in the Personality Assessment Inventory-Borderline Features scale was shared with normative personality, as measured by the NEO Five-Factor Inventory. In contrast, our twin Cholesky decomposition results indicated that approximately 39% of the genetic variance was unique to the DSM-IV borderline personality disorder criterion count and hence not shared with the Big Five domains. Further differences between our results and those of Distel et al. were evident in the genetic correlations between borderline personality disorder and the normative personality domains. Overall, the genetic correlations between borderline personality disorder and the Big

FIGURE 2. Stacked Bar Plots Displaying, for Each Personality Disorder, the Proportion of Genetic and Individual Specific Environmental Variances at Waves 1 and 2 That Are Shared With the Big Five Inventory Factors and Unique to Each Personality Disorder^a



^a A=additive genetic influence; E=environmental influence.

Five domains were lower in our sample, and this was especially pronounced for agreeableness and extraversion. Whereas Distel et al. observed genetic correlations of 0.81 with agreeableness and 0.62 with extraversion, the absolute-valued estimates in our sample were 0.44 and 0.25, respectively.

This difference could be due in part to the measures of normative personality used. The Big Five (26) and the fivefactor model (33) are taxonomies of personality traits derived through factor analysis, both positing that individual variance in personality disorder traits was approximately 50% lower at wave 2. The lower level of nonshared genetic influences at wave 2 is most consistent with the influence of time-specific genetic effects in one or both traits and is somewhat at odds with the longitudinal stability observed in the phenotypic correlations. Although the mean level of personality disorder symptoms is known to decline over time (36), the limited empirical evidence suggests that the underlying genetic influences are relatively stable. For example,

differences can be attributed to variability on five broad domains. However, whereas the Big Five is rooted in the lexical approach and is based on the investigation of descriptive terms embedded in natural language, the five-factor model is based on analyses of questionnaire data. The associated measurement instruments-the Big Five Inventory for the Big Five and the NEO Personality Inventory-Revised for the fivefactor model-may therefore differ in genetic or environmental correlation with personality disorders.

The genetic correlations between normative personality and borderline personality disorder in our sample are more in agreement with those reported by Kendler et al. (21), for whom the correlations between Big Five domains and the core features of borderline personality disorder as measured by the Dimensional Assessment of Personality Pathology were, in decreasing order, neuroticism, conscientiousness, and agreeableness. The modest genetic correlations found in our sample between openness and all personality disorder criteria counts are consistent with those found between the 18 subscales of the Dimensional Assessment of Personality Pathology-Basic Questionnaire and the NEO Five-Factor Inventory domains (16).

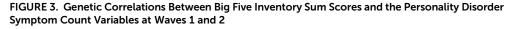
Across measurement waves, the genetic correlations were largely similar, but the proportion of unique genetic Bornovalova et al. (37) found no time-specific genetic influences on borderline personality disorder across four waves spanning the ages of 14 to 24 years. In conclusion, our results indicate that the etiology underlying DSM-IV personality disorders is not well captured by Big Five normative personality measures. This is in contrast to the Personality Inventory for DSM-5-Norwegian Brief Form, a short form of the Personality Inventory for DSM-5 (38) dimensional model of personality pathology designed to cover all the maladaptive trait features of DSM-IV-TR personality disorders. A recent publication based on the sample we used in the present study found that the Personality Inventory for DSM-5-Norwegian Brief Form at an aggregate level tapped the same genetic risk factors as the DSM-5 section II classification for most of the personality disorders (39). Although Wright et al. (35) reported an overlap in both

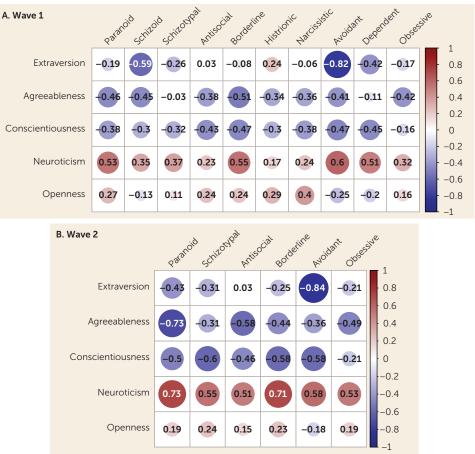
phenotypic and genetic correlations between normative personality and the Personality Inventory for DSM-5, we believe that a reasonable conclusion that follows from our results here and those of Reichborn-Kjennerud et al. (39) is that the Personality Inventory for DSM-5 is a better trait representation of DSM personality disorders than is the Big Five Inventory.

In conclusion, our results suggest that although the observed association between DSM personality disorder criteria and normative personality is largely due to common genetic influences rather than environmental influences, a substantial proportion of the genetic risk underlying the endorsement of personality disorder criteria appears not to be shared with normative personality.

Limitations

The interpretation of results presented in this study should be considered in light of several possible limitations. First, because of the low prevalence of endorsed criteria, we were unable to analyze categorical personality disorder diagnoses. In previous publications we examined whether the personality disorder criterion count variables are in accordance with an underlying continuous liability to increasing levels of endorsements of the personality disorder criteria and found this assumption to be satisfied empirically (25). Second, the





sample consists of Norwegian twins in a limited age range of adulthood (ages 30-44), and the results may not generalize to other populations. Third, only a subset of DSM-IV personality disorder traits was assessed at wave 2. so we were unable to replicate wave 1 results for all disorders. The median summaries of nonoverlapping genetic variance differences may be due in part to four personality disorders not assessed at wave 2. However, the results for the six personality disorder symptom counts assessed at both waves were very similar. Fourth, although there was evidence of selective attrition from wave 1 to wave 2, this was modest. The full information maximum likelihood estimation approach used in the twin analyses is robust against biases because of common types of missing data (40), so the attrition is unlikely to affect the estimates from our analyses. A final limitation concerns the lack of more explicit modeling of sex differences. Sex-limited twin models of ordinal data require very large samples to attain sufficient power. However, previous twin studies have failed to find either quantitative or qualitative gender differences for DSM-IV personality disorders and personality traits (13, 14).

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Dr. Krueger is a coauthor of the Personality Inventory for DSM-5 and provides consulting services to aid users of the instrument in the interpretation of test scores. (The Personality Inventory for DSM-5 is the intellectual property of the American Psychiatric Association, and Dr. Krueger does not receive royalties or any other compensation from publication or administration of the inventory.) The other authors report no financial relationships with commercial interests.

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