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THE GENETIC AND ENVIRONMENTAL SOURCES OF RESEMBLANCE BETWEEN NORMATIVE PERSONALITY AND PERSONALITY DISORDER TRAITS

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Recent work has suggested a high level of congruence between normative personality, most typically represented by the "big five" factors, and abnormal personality traits. In 2,293 Norwegian adult twins ascertained from a population-based registry, the authors evaluated the degree of sharing of genetic and environmental influences on normative personality, assessed by the Big Five Inventory (BFI), and personality disorder traits (PDTs), assessed by the Personality Inventory for *DSM-5*-Norwegian Brief Form (PID-5-NBF). For four of the five BFI dimensions, the strongest genetic correlation was observed with the expected PID-5-NBF dimension (e.g., neuroticism with negative affectivity [+], conscientiousness with disinhibition [–]). However, neuroticism, conscientiousness, and agreeableness had substantial genetic correlations with other PID-5-NBF dimensions (e.g., neuroticism with compulsivity [+], agreeableness with detachment [–]). Openness had no substantial genetic correlations with any PID-5-NBF dimension. The proportion of genetic risk factors shared in aggregate between the BFI traits and the PID-5-NBF dimensions was quite high for conscientiousness and neuroticism, relatively robust for extraversion and agreeableness, but quite low for openness. Of the six PID-5-NBF dimensions, three (negative affectivity, detachment, and disinhibition) shared, in aggregate, most of their genetic risk factors with normative personality traits. Genetic factors underlying psychoticism, antagonism, and compulsivity were shared to a lesser extent, suggesting that they are influenced by etiological factors not well indexed by the BFI.

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A broad consensus now suggests that many normative personality characteristics are well conceptualized within a Five Factor Model (FFM) that incorporates overarching dimensions of Extraversion, Agreeableness, Conscientiousness, Neuroticism, and Openness to Experience (John & Srivastava, 1999). These dimensions, as measured by a variety of self-report questionnaires, have been extensively investigated in twin studies, and all have been shown to be partly heritable (Livesley & Jang, 2008; McGue, 2002; South, Reichborn-Kjennerud, Eaton, & Krueger, 2012). In recent decades, there has also been an increasing interest in developing self-report measures for abnormal personality, which we refer to here as personality disorder traits (PDTs) (Clark, 1993; Clark, Vorhies, & McEwen, 1994; Livesley & Jackson, 2009; Krueger, Derringer, Markon, Watson, & Skodol, 2012). One of these measures (the DAPP-BQ; Livesley & Jackson, 2009) has also been studied in twins and found to be moderately heritable (Jang, Livesley, Vernon, & Jackson, 1996; Livesley, Jang, Jackson, & Vernon, 1993).

Several authors have argued that normal personality and abnormal personality have substantial consilience, and models that integrate these personality constructs have been proposed (Markon, Krueger, & Watson, 2005). Numerous studies have shown that personality disorders as defined by the *DSM* system can be represented by the FFM (Costa & Widiger, 2002; Samuel & Widiger, 2008; Saulsman & Page, 2004), arguing that the traits of normal and abnormal personality can be conceptually organized and delineated on a continuum, with PDTs located at more extreme positions (Livesley, 2007). This hypothesis implies that the etiological factors underlying normal and abnormal personality are highly correlated, which should be reflected in high phenotypic correlations, and potentially in even higher genetic correlations between normal personality and PDTs.

This line of research contributed substantially to the alternative *DSM-5* model for personality disorders, which includes five higher-order domains of negative affect, detachment, antagonism, disinhibition, and psychoticism (American Psychiatric Association [APA], 2013). A self-report instrument, the Personality Inventory for *DSM-5* (PID-5), was developed to measure these domains, as well as 25 specific facets of these domains. Four of the five domains that resulted from the initial development studies (Krueger et al., 2012) (negative affect, detachment, antagonism, and disinhibition) were conceptualized as representing maladaptive extremes of the FFM domains of, respectively, neuroticism, extraversion, agreeableness, and conscientiousness. By contrast, the fifth domain of psychoticism, developed to capture unusual cognitive beliefs and perceptions, may be relatively separate from the normative openness domain. For example, PID-5 psychoticism was not highly correlated with openness in a recent study evaluating continuity of normative traits and PDTs (Suzuki, Samuel, Pahlen, & Krueger, 2015).

We are aware of two prior studies that have examined the relationship between genetic risk factors for normative and disordered personality. Markon, Krueger, Bouchard, and Gottesman (2002) examined normative personality as measured by the Multidimensional Personality Questionnaire (MPQ), and PDTs as assessed by the MMPI in 128 monozygotic and dizygotic twin pairs in the Minnesota Study of Twins Reared Apart. They found

widely varying genetic correlations (which, like standard correlations, can vary from –1.00 to +1.00 and reflect the degree of resemblance of the genetic factors influencing two traits) between 11 subscales from the MPQ and 13 scales from the MMPI. However, the findings were not organized by higher-order domains of the FFM. Of greater relevance, Jang and Livesley (1999) modeled responses from 545 volunteer general population twin pairs on the five dimensions of one of the classic FFM inventories, the NEO-FFI, and the 18 dimensions of PDTs from the Dimensional Assessment of Personality Pathology (Livesley & Jackson, 2009). The strongest genetic correlations between the 18 Dimensional Assessment of Personality Pathology dimensions were seen for the FFM trait of neuroticism (range = .05 to .81; median = .48), followed by agreeableness (range = –.65 to .00; median = –.38), conscientiousness (range = –.76 to .52; median = –.31), extraversion (range = –.65 to .33; median = –.28), and openness (range = –.17 to .20; median = –.04). The authors suggested that their results indicated that "these two scales share a common broad-based genetic architecture" (Jang & Livesley, 1999, p. 10).

In this report, we seek to complement these earlier studies by examining, in a population-based sample of Norwegian twins, the relationship between the latent genetic and factors that influence the five dimensions of normative personality as described by the FFM—assessed using the Big Five Inventory (BFI; John, Donahue, & Kentle, 1991) and the dimensions of a shortened version of the PID-5 (which we will refer to as the PID-5-Norwegian Brief Form, or PID-5-NBF) that includes scales to assess the five domains of the *DSM-5* Alternative model plus a scale for compulsivity. In seeking to understand how the genetic and environmental influences on normative and pathological personality interrelate, we address three specific questions:

1. What are the genetic and environmental correlations between the BFI and PID-5-NBF dimensions?
2. To what degree do the dimensions of the BFI in aggregate capture the genetic and environmental risk factors for the individual PID-5-NBF dimensions? That is, how well do all the BFI scales considered together index the genetic (or environmental) influences on the PID-5 scales?
3. To what degree do the dimensions of the PID-5-NBF in aggregate capture the genetic and environmental risk factors for the individual BFI dimensions?

METHODS

PARTICIPANTS

Data for this study came from the second wave of the Norwegian National Institute of Public Health Twin Panel (NIPHTP; Nilsen et al., 2013). Twins were recruited for the NIPHTP from the National Medical Birth Registry of Norway, established in 1967; by mandate, the Registry receives notification of all births in Norway. The first wave of data collection on this sample (born from 1967–1979) consisted of questionnaire data collected in 1998 and structured interview data from 1999 to 2004 (Wave 1) (Kendler et al.,

2008). The data for the current analyses were drawn from a questionnaire completed by participants in 2011 (Wave 2). All participants received the questionnaire by mail, and written informed consent was obtained from all participants. A total of 2,801 twins were invited to participate in Wave 2. Of those, 504 did not complete the BFI items in the Wave 2 questionnaire and one additional twin failed to complete any of the PID-5-NBF items, resulting in a total of 505 unusable individual records. Some missingness was present for individual BFI and PID-5-NBF item responses. However, the majority of this type of missingness was limited to one or two items. Since item aggregate composites were used for these analyses, these records were retained. This left N = 2,296 individual survey participants forming 1,319 twin pairs in the final sample used for the twin modeling: 212 male MZ (five singletons), 116 male DZ (two singletons), 425 female MZ (four singletons), 245 female DZ (four singletons), and 321 opposite-sex DZ (four singletons). These twin pair totals include those where one twin had aggregate score data for the BFI and PID-5-NBF and the other twin did not. The analyses presented here used a two-group MZ(N = 637)/DZ(N = 682) pair data structure. The DZ total includes both same-sex and opposite-sex twin pairs. Zygosity for this sample was determined by the use of questionnaire items (Harris, Magnus, & Tambs, 2006). For 676 of the same-sex pairs, microsatellite markers were collected for a discriminant analysis with the questionnaire, which resulted in an estimated misclassification rate of ~1%.

MEASURES

Twin participants completed the 36-item PID-5-NBF designed to capture the five domains of the *DSM-5* Section III Alternative trait model, which was in development at the time of the study planning, plus a sixth domain with explicit compulsivity content. This subset of items was selected from the full PID-5 (Krueger et al., 2012) battery based on their psychometric quality as indicators of each of the target maladaptive domains. On this inventory, each item is a one-sentence descriptor, and participants are asked to respond to each item on a 1–4 scale: (a) *Very true or often true*, (b) *Sometimes or somewhat true*, (c) *Sometimes or somewhat false*, and (d) *Very false or often false*. Items were not evenly distributed across the trait domains because some domains (e.g., negative affectivity) were expected to be more heavily saturated throughout dimensions of abnormal personality pathology. Therefore, more items were allocated to these domains. A total of 10 items measured the negative affectivity domain, 6 items measured antagonism, and 5 items were created to measure detachment, disinhibition, compulsivity, and psychoticism. Items were randomly numbered on the inventory. The standardized coefficient alphas for these scales in our data were as follows: Negative Emotionality = 0.87, Detachment = 0.78, Antagonism = 0.61, Disinhibition = 0.70, Psychoticism = 0.78, and Compulsivity = 0.81.

Normative personality was assessed by the Big Five Inventory (BFI; John et al., 1991), a 44-item scale that assesses openness to experience (10 items), conscientiousness (9 items), extraversion (8 items), agreeableness (9 items), and neuroticism (8 items). Responses to each item were via a 5-point Likert

scale with the following options: (a) *Strongly disagree*, (b) *Disagree a little*, (c) *Neither disagree nor agree*, (d) *Agree a little*, and (e) *Strongly agree*. The standardized coefficient alphas for these scales in our data were as follows: extraversion, 0.85; neuroticism, 0.84; conscientiousness, 0.77; agreeableness, 0.71; and openness 0.80.

STATISTICAL ANALYSES

To justify the construction of item sum score aggregate variables for each of the BFI and PID-5-NBF target constructs, preliminary exploratory and confirmatory item-level analyses were performed to examine and test for unidimensionality. All individual record item-level analyses were conducted in Mplus version 7.11 (Muthén & Muthén, 2012) using the robust WLSMV estimator for ordinal data in conjunction with the complex option to adjust fit indexes and standard errors for the nonindependence of the twin structure. Due to item responses being coded from 1 (*very true or often true*) to 4 (*very false or often false*) for the PID-5-NBF items, they were all reverse coded for analysis. Although global model fit indexes varied across the 11 separate confirmatory single-factor models fit to the respective construct/trait item indicator sets, there was general support for unidimensionality. That is, the interitem correlations for each BFI and PID-5-NBF construct indicator set were predominantly accounted for by a single common factor.

All bivariate and extended multivariate twin Cholesky decomposition modeling was carried out using OpenMx version 2.0.0.4004 (Neale et al., 2016) in the R environment version 3.1.2 (R Development Core Team, 2014). Due to several considerations, including past experience with this sample and phenotypes, model fitting was restricted to an MZ/DZ two-group data structure. Based on the rationale and assumptions of the classic twin design, the observed BFI and PID-5-NBF scale variation and covariation can be decomposed into three different etiologically informative sources—those due to (a) additive genetic effects (A); (b) shared common environment (C), which reflects aspects of the home and community environment exposure, which makes the twins more similar to one another; and (c) unique environment (E), which reflects environmental experiences that make the twins different from one another as well as random errors of measurement or "noise." We also present genetic and environmental correlations, which can take values from –1.00 to +1.00 and reflect the degree of similarity of, respectively, the genetic and environmental influences of two traits (Carey, 1988; Neale & Cardon, 1992). The Cholesky decomposition analyses employed in this report can be best understood as the twin version of a multiple regression that determines the degree to which genetic and environmental risk factors for the downstream variable (individual scales from the PID-5-NBF or the BFI) are shared with those of the upstream variables (all the scales, respectively, of the BFI and PID-NBF). A raw data full-information maximum likelihood optimization approach was used to obtain parameter estimates and standard errors. All sum score distributions were modeled as quasicontinuous. For the five BFI constructs, aggregate score distributions were reasonably symmetric and normally shaped and so were analyzed without transformation.

However, this was not the case for the PID-5-NBF aggregate distributions. Histograms showed noticeably positive skewness reflecting a disproportionately high frequency of lower values, consistent with the maladaptive nature of these traits. Natural logarithm transformations were applied after incrementing each aggregate sum score by 1 to avoid zero values being set to missing. All of the BFI-PID-5-NBF bivariate models as well as larger BFI-PID-5-NBF multivariate models were submitted to multiple optimizations using different starting values as a check of the stability and convergence of the minimization process. This is done to reduce the chances that the best fit model reflects a local rather than a true global minimum. Model fit was determined by Akaike's Information Criterion (AIC; Akaike, 1987), where a lower value indicated a more optimal balance of parsimony and explanatory power.

RESULTS

DESCRIPTIVE STATISTICS

Table 1 depicts the mean, standard deviation, and phenotypic interscale correlations for the five BFI scales and the six untransformed PID-5-NBF scales. The mean scores for the BFI scales were higher than those seen for the PID-5-NBF, and the variance was generally greater. Noteworthy correlations between the BFI and PID-5-NBF scales include extraversion-detachment (–0.57), neuroticism-negative affectivity (+0.62), conscientiousness-disinhibition (–0.54), and agreeableness-antagonism (–0.32).

GENETIC AND ENVIRONMENTAL CORRELATIONS

Thirty separate bivariate twin analyses between each of the BFI and PID-5-NBF scales were run. For each analysis, the AE model provided a better fit according to AIC than either the full ACE or the CE model. That is, the data were best explained assuming that genetic effects were responsible for all of the observed resemblance among twins. Indeed, for 25 of the 30 models, no change in log likelihood was seen when C parameters were set to zero, and the change was minimal for the remaining five models.

Table 2 presents the estimated genetic correlations between the scales. Six results are noteworthy. First, all but five of these correlations were statistically significant. Second, substantial genetic correlations were seen between extraversion, neuroticism, conscientiousness, and agreeableness, and at least one of the PID-5-NBF dimensions. Third, the genetic correlations were consistently lower with openness than with any of the other four big five factors; the largest genetic correlation observed between openness and any of the PID-5-NBF scales was only +0.25 (for psychoticism). Fourth, for extraversion, neuroticism, conscientiousness, and agreeableness, the strongest genetic correlation was with the predicted PID-5-NBF scale: extraversion with detachment (–0.76), neuroticism with negative affectivity (+0.83), conscientiousness with disinhibition (–0.83), and agreeableness with antagonism (–0.64). Fifth, however, each of the BFI scales also had substantial genetic

TABLE 1. Means, Standard Deviations, and Phenotypic Correlations for the Five Scales From the BFI and the Six Scales From the PID-5-NBF

	BFI					PID-5-NBF					
Variables	Extraversion	Neuroticism	Openness	Conscientiousness	Agreeableness	Negative Affectivity	Detachment	Antagonism	Disinhibition	Psychoticism	Compulsivity
Mean	19.986	11.982	23.144	25.775	26.444	3.175	2.700	1.352	2.830	1.348	1.984
SD	5.117	5.355	5.484	4.208	3.816	4.160	2.680	1.838	2.466	2.155	2.600
BFI											
Extraversion	1.000	−0.427	0.260	0.297	0.249	−0.406	−0.569	0.015	−0.123	−0.226	−0.168
Neuroticism	−0.427	1.000	−0.040	−0.376	−0.351	0.622	0.349	0.081	0.366	0.361	0.321
Openness	0.260	−0.040	1.000	0.006	0.056	−0.036	−0.096	0.075	0.078	0.140	0.004
Conscientiousness	0.297	−0.376	0.006	1.000	0.377	−0.334	−0.287	−0.210	−0.535	−0.299	−0.027
Agreeableness	0.249	−0.351	0.056	0.377	1.000	−0.312	−0.315	−0.324	−0.226	−0.231	−0.173
PID-5-NBF											
Negative Affectivity	−0.406	0.622	−0.036	−0.334	−0.312	1.000	0.580	0.339	0.509	0.624	0.498
Detachment	−0.569	0.349	−0.096	−0.287	−0.315	0.580	1.000	0.298	0.357	0.458	0.379
Antagonism	0.015	0.081	0.075	−0.210	−0.324	0.339	0.298	1.000	0.420	0.445	0.372
Disinhibition	−0.123	0.366	0.078	−0.535	−0.226	0.509	0.357	0.420	1.000	0.506	0.286
Psychoticism	−0.226	0.361	0.140	−0.299	−0.231	0.624	0.458	0.445	0.506	1.000	0.435
Compulsivity	−0.168	0.321	0.004	−0.027	−0.173	0.498	0.379	0.372	0.286	0.435	1.000

TABLE 2. Genetic Correlations (and 95% Confidence Intervals) Between BFI and PID-5-NBF Scales

BFI/PID-5-NBF Variables	Negative Affectivity	Detachment	Antagonism	Disinhibition	Psychoticism	Compulsivity	Mean Genetic Correlation[a]
Extraversion	**−0.53**	**−0.76**	−0.09	**−0.21**	**−0.29**	**−0.31**	−0.40
	[−0.62, −0.42]	**[−0.84, −0.67]**	[−0.21, +0.02]	**[−0.33, −0.08]**	**[−0.41, −0.17]**	**[−0.45, −0.18]**	
Neuroticism	**+0.83**	**+0.49**	**+0.14**	**+0.44**	**+0.53**	**+0.55**	+0.53
	[+0.75, +0.90]	[+0.37, +0.60]	[+0.02, +0.27]	[+0.32, +0.56]	[+0.42, +0.64]	[+0.41, +0.69]	
Openness	+0.03	−0.11	**+0.20**	+0.03	**+0.25**	+0.04	+0.08
	[−0.10, +0.16]	[−0.24, +0.02]	**[+0.07, +0.32]**	[−0.10, +0.16]	**[+0.12, +0.37]**	[−0.11, +0.19]	
Conscientiousness	**−0.58**	**−0.53**	**−0.50**	**−0.83**	**−0.56**	**−0.20**	−0.56
	[−0.70, −0.45]	**[−0.67, −0.40]**	**[−0.63, −0.36]**	**[−0.93, −0.73]**	**[−0.69, −0.43]**	**[−0.38, −0.03]**	
Agreeableness	**−0.53**	**−0.57**	**−0.64**	**−0.37**	**−0.41**	**−0.39**	−0.49
	[−0.69, −0.38]	**[−0.73, −0.41]**	**[−0.79, −0.49]**	**[−0.54, −0.19]**	**[−0.57, −0.24]**	**[−0.59, −0.20]**	

Note. Bold indicates that 95% CIs do not include zero. [a]Correlations were converted to Z-scores, averaged, and then converted back to correlations.

TABLE 3. Individual-Specific Environmental Correlations (and 95% Confidence Intervals) Between BFI and PID-5-NBF Scales

BFI/PID-5-NBF Variables	Negative Affectivity	Detachment	Antagonism	Disinhibition	Psychoticism	Compulsivity	Mean Environmental Correlation[a]
Extraversion	−0.32	−0.41	+0.08	−0.04	−0.18	**−0.10**	−0.17
	[−0.38, −0.24]	**[−0.48, −0.35]**	[0.00, +0.16]	[−0.11, +0.04]	**[−0.26, −0.11]**	**[−0.18, −0.03]**	
Neuroticism	**+0.48**	**+0.24**	+0.04	**+0.29**	**+0.27**	**+0.20**	+0.26
	[+0.43, +0.54]	**[+0.16, +0.31]**	[−0.04, +0.12]	**[+0.22, +0.36]**	**[+0.20, +0.34]**	**[+0.12, +0.27]**	
Openness	**−0.09**	**−0.09**	0.01	**+0.10**	+0.07	−0.02	0.00
	[−0.16, −0.01]	**[−0.17, −0.02]**	[−0.07, +0.09]	**[+0.02, +0.18]**	[−0.01, +0.15]	[−0.09, +0.06]	
Conscientiousness	**−0.19**	**−0.12**	0.00	**−0.32**	**−0.14**	+0.04	−0.12
	[−0.26, −0.17]	**[−0.19, −0.04]**	[−0.08, +0.08]	**[−0.38, −0.25]**	**[−0.21, −0.06]**	[−0.04, +0.12]	
Agreeableness	**−0.24**	**−0.19**	**−0.17**	**−0.15**	**−0.16**	−0.11	−0.17
	[−0.31, −0.17]	**[−0.26, −0.11]**	**[−0.25, −0.10]**	**[−0.22, −0.08]**	**[−0.23, −0.11]**	[−0.18, −0.03]	

Note. Bold indicates that 95% CIs do not include zero. [a]Correlations were converted to Z-scores, averaged, and then converted back to correlations.

correlations (e.g., absolute values > 0.50) with at least one other PID-5-NBF scale, for example, extraversion with negative affectivity, neuroticism with compulsivity and psychoticism, conscientiousness with negative affectivity and psychoticism, and agreeableness with negative affectivity and detachment. Sixth, we calculated the mean genetic correlation between the BFI scales and the PID-5-NBF scales as a general index of the degree to which the individual five factor domains reflect a shared genetic vulnerability to personality pathology. The strongest mean correlation was seen for conscientiousness, followed, in order, by neuroticism, agreeableness, extraversion, and openness.

The estimated individual specific environmental correlations between the BFI and PID-5-NBF scales are seen in Table 3. Four results are of note. First, the environmental correlations are uniformly lower than the parallel genetic correlations, with eight of them not significantly different from zero. Second, the environmental correlations are lower with openness than with any of the other four big five factors. Third, for extraversion, neuroticism, and conscientiousness, the strongest environmental correlation was with the expected PID-5-NBF dimension (detachment, negative affectivity, and disinhibition, respectively). For agreeableness, however, the environmental correlation with negative affectivity exceeded that found for antagonism. Fourth, the mean environmental correlation between the BFI scales and the PID-5-NBF scales, as a broad index of the degree to which the individual five factor domains reflect the environmental risk factors for personality pathology, showed strongest correlations for neuroticism, followed, in order, by agreeableness, extraversion, conscientiousness, and openness.

CHOLESKY DECOMPOSITION: PREDICTION OF PID-5-NBF SCALES FROM BFI

We fitted six separate twin Cholesky decompositions that included all five BFI scales as "predictors," with the distal variable being, in turn, each of the six subscales of the PID-5-NBF. For each of these analyses, the AE model fit better by AIC than the ACE or CE models, again suggesting that genetic factors appeared to be responsible for all the observed twin resemblance. In particular, when setting to zero all the 21 C parameters in these models, the log likelihood fit of the model never deteriorated more than two units, a very small change. In Table 4, we report the a^2 and e^2 estimates from the AE model for the PID-5-NBF scales, but we focus on the percentage of those estimates indexed by the set of BFI scales versus those unique to the PID-5-NBF. Examining genetic effects first, we found that the PID-5-NBF scales formed a spectrum. At one end were negative affectivity, detachment, and disinhibition, where the large preponderance of the genetic effects (81%, 77%, and 71%, respectively) was captured by the aggregated BFI scales. At the other end were compulsivity and psychoticism, where less than half of the genetic variance (37% and 48%, respectively) was assessed by the BFI scales. In between was antagonism, where 51% of the genetic variance was captured by the BFI scales. There is much less sharing of environmental sources of vari-

TABLE 4. Results of Cholesky Decomposition Predicting Genetic and Environmental Effects of All Five BFI Scales on the Individual Dimensions of the PID-5-NBF

	Genetic Effects			Individual-Specific Environmental Effects		
PID-5-NBF Scale	a^2	% Shared with BFI	% Unique	e^2	% Shared with BFI	% Unique
Negative Affectivity	0.36	81	19	0.43	25	75
Detachment	0.34	77	23	0.66	18	82
Antagonism	0.40	51	49	0.60	5	95
Disinhibition	0.35	71	29	0.65	17	83
Psychoticism	0.37	48	52	0.63	10	90
Compulsivity	0.27	37	63	0.73	6	94

ance between the PID-5-NBF scales and the BFI, ranging from a low of 5% (antagonism) to a high of 25% (negative affectivity).

CHOLESKY DECOMPOSITION: PREDICTION OF BFI FROM THE PID-5-NBF SCALES

To complement the BFI-PID-5-NBF multivariate Cholesky modeling results, we also fitted five Cholesky decompositions with all six PID-5-NBF scales included as "predictor variables," and this time with the distal dependent variable being, in turn, each of the five scales from the BFI. For each of these analyses, the AE model provided the better fit by AIC and so we report results from these models in Table 5. We again found no appreciable evidence for C effects; when we set to zero all the 28 C parameters in these models, the log likelihood fit of the model never deteriorated more than five units, a modest change.

These results indicated a wide variation in the degree to which genetic and environmental liabilities to normative personality are captured by the PID-5-NBF scale. Focusing first on genetic effects, at one end of the spectrum is openness, where only a relatively small proportion of the genetic variance (25%) is captured by the PID-5-NBF scales. At the other end of the spectrum are conscientiousness and neuroticism, where a large portion of their genetic effects (87% and 81%, respectively) overlap with the PID-5-NBF. In between these extremes are agreeableness and extraversion, where around two thirds of the genetic effects are captured by the PID-5-NBF. Much less sharing of environmental sources of variance was found between the BFI and the PID-5-NBF scales, ranging from a low of 5% for openness to a high of 25% for neuroticism.

DISCUSSION

The main goal of this article was to examine, in a general population sample of Norwegian twins, the relationships between the genetic and environmental influences on normative personality and on PDTs. We here review our main findings, organized around the three questions we here sought to answer.

TABLE 5. Results of Cholesky Decomposition Predicting Genetic and Environmental Effects of All Six PID-5-NBF Scales on the Individual BFI Scales

BFI Scale	Genetic Effects			Individual-Specific Environmental Effects		
	a^2	% Shared with PID-5-NBF	% Unique	e^2	% Shared with PID-5-NBF	% Unique
Extraversion	51	70	30	49	24	76
Neuroticism	46	81	19	54	26	74
Openness	48	25	75	52	5	95
Conscientiousness	41	87	13	59	14	86
Agreeableness	27	65	35	73	8	92

Our first goal was to examine and clarify the nature of the genetic and environmental correlations between the individual BFI and PID-5-NBF scales. Consistent with the expectations of the PID-5-NBF, four of the five BFI factors had their strongest genetic correlation with the predicted PID-5-NBF scale: extraversion with detachment, neuroticism with negative affectivity, conscientiousness with disinhibition, and agreeableness with antagonism. A similar pattern was seen for the environmental correlations, with the exception that for agreeableness, the environmental correlation with negative affectivity and detachment somewhat exceeded that seen for antagonism. Consistent with prior findings (Jang & Livesley, 1999; Markon et al., 2002), in a general population sample, we observed substantial overlap in the genetic liability to broadly parallel dimensions of normative and pathological personality. As might be expected because the individual-specific environment in standard twin studies includes measurement error, the relationship between environmental influences for normative and pathological personality was weaker, but the overall pattern was similar. That is, for example, we saw a relatively strong relationship between environmental risk factors for neuroticism as measured by the BFI and negative affectivity as assessed by the PID-5-NBF.

However, we found only moderate levels of specificity in the relationship between genetic contributions to normal and abnormal personality. In particular, neuroticism, conscientiousness, and agreeableness had substantial genetic correlations with a majority of the PID-5-NBF scales. One way to interpret this nonspecificity is that underlying the PID-5-NBF scales was a shared dimension of general personality pathology that is best indexed, in the BFI, by elevated levels of neuroticism and low levels of conscientiousness and agreeableness. Also of interest, consistent with prior studies (Saulsman & Page, 2004), openness stood out among the BFI scales in mapping poorly onto PDTs. Based on these findings, this dimension of the FFM seems to have limited relevance for personality pathology (at least as indexed by the *DSM-5* PDTs) (Chmielewski, Bagby, Markon, Ring, & Ryder, 2014).

Our second aim was to obtain estimates of the degree to which the combined dimensions of the BFI jointly share genetic and environmental risk factors with each of the six PDTs as defined by the PID-5-NBF maladaptive traits. For genetic effects, the results were quite variable, suggesting a textured relationship between the individual dimensions of normative and

pathological personality, at least as assessed by the scales we utilized in this study. In particular, genetic risk factors for negative affectivity, detachment, and disinhibition were well indexed by BFI scales. Examination of the results of the Cholesky analyses suggest, as suspected, that this finding was largely driven by the prior strong genetic correlations between these scales and, respectively, neuroticism, extraversion, and conscientiousness. However, for two of the PID-5-NBF maladaptive scales, the normative array of BFI trait scales was able to account for less than half of their total genetic variance. Normative personality as assessed by the BFI does not capture well the genetic risk for compulsivity or for psychoticism, the scales for which were developed to capture unusual beliefs and experiences, eccentricity, and perceptual dysregulation (Krueger et al., 2012). Using the brief measures from the PID-5-NBF, which were focused on the domain level, we found evidence for unique effects on normative versus pathological personality traits. Our findings, which need to be explored further using more comprehensive assessments, raise questions about the position taken by some in the field that normative and pathological personality variations are entirely overlapping (Costa & Widiger, 2002; Livesley, 2007; Samuel & Widiger, 2008; Saulsman & Page, 2004).

The pattern of results found for shared individual-specific environmental risk factors between these normative and maladaptive constructs was more difficult to interpret because of the likely confounding role of measurement error. However, the overall pattern of sharing, albeit at a much lower level, was similar to that seen for genetic risk factors.

The final goal of this article was to reverse the Cholesky model used for the prior analyses and explore how the genetic and environmental liabilities of each of the five normative personality scales were shared with the aggregate effect of the six PDTs. The results were somewhat clearer and more uniform. Genetic risk factors for PDTs did a very good job of predicting genetic effects for neuroticism, extraversion, and conscientiousness, and modestly well for agreeableness. The notable outlier was openness, where genetic risk factors had relatively little overlap with the PID-5-NBF scales.

In summary, in Norwegian adult twins ascertained from a population-based registry, we found that for four of the five BFI dimensions, the strongest genetic correlation was observed with the expected PID-5-NBF dimension (e.g., neuroticism with negative affectivity [+], conscientiousness with disinhibition [–]). However, neuroticism, conscientiousness, and agreeableness had substantial genetic correlations with other PID-5-NBF dimensions, and openness had no substantial genetic correlations with any PID-5-NBF dimension. The proportion of genetic risk factors shared in aggregate between the BFI traits and the PID-5-NBF dimensions was quite high for conscientiousness and neuroticism, relatively robust for extraversion and agreeableness, but quite low for openness. Three of six PDTs (negative affect, detachment, and disinhibition) shared most of their genetic risk factors with normative personality dimensions. This can be interpreted as support for the hypothesis that these traits represent maladaptive extremes of normative personality (APA, 2013). Etiological factors underlying psychoticism, antagonism, and compulsivity are to a lesser extent shared with those of normative personali-

ty, suggesting that they might be located partly on separable continua, rather than representing extremes of normal personality traits.

LIMITATIONS

These results should be interpreted in the context of three potentially important methodological limitations. First and most important, we assessed normative and abnormal personality traits with relatively short instruments: the BFI and a considerably abbreviated version of the PID-5. Our results might have differed appreciably had longer scales been used, likely in the direction of finding stronger and more specific correlations between our normative personality traits and PDTs. In particular, the variance for some of the PID-5 scales was relatively modest, which may have resulted in an attenuation of the phenotypic and genetic correlations observed between them and the BFI scales.

Second, attrition occurred in this twin sample from the original birth registry through to our Wave 2 assessments used in this report. We report detailed analyses of this attrition elsewhere, where we show that cooperation was strongly and consistently predicted by female sex, monozygosity, older age, and higher educational status, but by neither psychiatric symptoms nor psychoactive drug use (Tambs et al., 2009). So some attrition bias in our results is possible. However, the full-information maximum likelihood methods used here are robust to missing data when certain assumptions such as completely missing random are reasonable or other variables related to missingness are in the analysis, which is at least partly the case here.

Third, this sample had inadequate power to detect sex effects on genetic risk factors due to modest numbers of opposite-sex dizygotic twins, who provide the critical information for such analyses. At the personality disorder cluster level in Wave 1 interview data, we tested for and found no evidence for quantitative or qualitative sex effects (Kendler et al., 2006; Reichborn-Kjennerud et al., 2007; Torgersen et al., 2008). Univariate analyses under way with the individual PID-5-NBF scales also suggest no sex effects for four of the six scales, with clear evidence arising only for antagonism. It is therefore unlikely that our results are substantially biased by our focus on models without sex effects.

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