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## Structure of Genetic and Environmental Risk Factors for Symptoms of *DSM-IV* Borderline Personality Disorder

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### Abstract

**IMPORTANCE**—Previous studies have indicated that the psychopathological dimensions of borderline personality disorder (BPD) are influenced by a unitary liability factor. However, to our knowledge, the underlying etiological nature of the individual criteria for BPD as defined by the *DSM-IV* has not been explored.

**OBJECTIVE**—To determine the structure of genetic and environmental risk factors for the symptoms of BPD.

**DESIGN, SETTING, AND PARTICIPANTS**—Multivariate twin study with BPD criteria assessed by personal interview within a general community setting. Participants included 2794 young adults from the Norwegian Institute of Public Health Twin Panel.

**MAIN OUTCOMES AND MEASURES**—The 9 criteria for BPD assessed by the Structured Interview for *DSM-IV* Personality.

**RESULTS**—A common pathway model dominated by 1 highly heritable (55%) general BPD factor that strongly influenced all 9 BPD criteria (standardized path coefficients, 0.53–0.79) fit the data best. The model also included 2 additional common liability factors, mainly influencing

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criteria reflecting the affective and interpersonal dimensions. Both of these were mostly influenced by environmental liability factors (heritability, 29.3% and 2.2%). With 1 exception (criterion 2, unstable and intense relationships), the specific criteria were strongly influenced by environmental factors. Five of the 9 criterion-specific genetic effects were either 0 or negligible.

**CONCLUSIONS AND RELEVANCE**—These results indicate that most of the genetic effects on the individual BPD criteria derive from 1 highly heritable general BPD factor, whereas the environmental influences were mostly criterion specific.

Borderline personality disorder (BPD) is a severe psychiatric disorder associated with high rates of suicide, functional impairment, intensive use of treatment, and high costs to society.<sup>1</sup> Its etiology is not well understood. Familial aggregation of BPD has been demonstrated in several studies,<sup>2,3</sup> and twin studies indicate that this is due to genetic factors.<sup>4-7</sup> The *DSM-IV* specifies 9 criteria for BPD (Table 1) and describes the essential feature of BPD as “a pervasive pattern of instability of interpersonal relationships, self-image and affects in addition to marked impulsivity.”<sup>8(p650)</sup> Although these 4 areas are commonly regarded as the major psychopathological dimensions or sectors of BPD,<sup>3,9,10</sup> the main hypothesis regarding the etiology of BPD is that a single underlying unitary psychopathological factor (influenced by genetic and environmental factors) gives rise to all 4 dimensions as well as the 9 lower-order diagnostic criteria.<sup>3</sup> An alternative hypothesis conceptualizes BPD as a personality disorder emerging from the interaction or co-occurrence of multiple underlying genetically based traits of measurable characteristics that each reflect an underlying genotype and represent the primary psychopathological entities of BPD.<sup>11</sup>

The empirical evidence from factor analytic studies of the *DSM-IV* criteria for BPD does not provide clear support for either of these etiological models. Several studies have supported a unidimensional structure.<sup>12-14</sup> However, 3 latent factors<sup>12,15,16</sup> and 4 latent factors<sup>17</sup> have also been reported. In addition to the heterogeneity of the BPD diagnosis, the lack of consistency in these studies might be due to differences between samples (clinical vs population based), diagnostic instruments, or type of assessment (interview vs questionnaire). The structure of the etiological factors for BPD can also be tested in family or twin studies. Multivariate models specifying 1 or more common latent factor influenced by genetic and environmental factors (common pathway models) can be compared with each other or with models directly specifying common genetic and environmental factors (independent pathway models).

Family studies indicate that the 4 major psychopathological dimensions or sectors of BPD are elevated or aggregate in families of probands with BPD.<sup>2,3</sup> Results from 2 recent twin studies (based on non-*DSM-IV* questionnaire data from different instruments) suggest that genetic factors and not common environmental factors account for the familial aggregation of the dimensions or traits assessed by these instruments.<sup>3,6</sup> One of the family studies and both twin studies also found that the higher-order BPD dimensions or traits could best be accounted for by a unitary liability factor (a common pathway model fit better than an independent factor model) with a high degree of heritability.<sup>3,6,7</sup> All studies used only 4 BPD dimensions in their models and not the 9 lower-order *DSM-IV* criteria. It was therefore not possible to test more complex multivariate models with several common etiological factors (common and independent pathway).

To our knowledge, no study has yet fully explored the structure of the etiological factors for BPD analyzed at the criterion level. To address the limitations in the previous studies, we used interview data from a large population-based twin study of Axis I and II psychiatric disorders in Norwegian twins. By including all 9 criteria in our models, we were able to test a larger number of alternative multivariate models. The main aim of the study was to clarify

the structure of genetic and environmental risk factors for *DSM-IV* diagnostic criteria for BPD.

## Methods

### Participants

Data for the current investigation come from a population-based study of psychiatric disorders in Norwegian twins recruited from the Norwegian Institute of Public Health Twin Panel.<sup>18</sup> Between 1999 and 2004, Axis I and II psychiatric disorders were assessed at interview in 2801 twins (43.5% of those eligible) born between 1967 and 1979. The mean age of participants was 28.2 years (range, 19–36 years). Zygosity was determined by a combination of questionnaire items and genotyping. The misclassification rate was estimated to be less than 1.0%, which is unlikely to substantially bias results.<sup>19</sup> Several articles describing details of the sample and the measures used in this report have been published.<sup>5,20,21</sup>

### Procedures

A Norwegian version of the Structured Interview for *DSM-IV* Personality<sup>22</sup> was used to assess personality disorders. This instrument is a comprehensive semistructured diagnostic interview for the assessment of all *DSM-IV* Axis II diagnoses, and it includes nonpejorative questions organized into topical sections rather than by disorders. This allows for a more natural flow of the interview and increases the likelihood that useful information from related questions may be considered when rating related criteria within that section. The specific *DSM-IV* criterion associated with each set of questions is rated according to the following scoring guidelines: 0 indicates that the criterion is not present or is limited to rare isolated examples; 1, subthreshold (some evidence of the trait, but not sufficiently pervasive for the criterion to be considered present); 2, present (criterion clearly present for most of the last 5 years); and 3, strongly present (criterion is associated with subjective distress or some impairment in social or occupational functioning or intimate relationships). The Structured Interview for *DSM-IV* Personality is conducted after the Axis I interview, which helps the interviewer distinguish long-standing behavior reported by the subject from temporary states due to an episodic psychiatric disorder.

Most of the interviewers were psychology students in their final part of training or experienced psychiatric nurses. They were trained by professionals (1 psychiatrist and 2 psychologists) who had extensive previous experience with the instrument, and they were closely followed up individually during the entire data collection period. Most of the interviews were conducted face to face, but for practical reasons, 231 (8.3%) were obtained by telephone. Each twin in a pair was interviewed by a different interviewer.

Interrater reliability was assessed based on 2 raters' scoring of 70 audiotaped interviews. Intraclass correlations for the number of endorsed BPD criteria at the subthreshold ( 1) and threshold ( 2) level were 0.93 and 0.92, respectively. The poly-choric correlation was 0.94.

Approval was received from The Norwegian Data Inspectorate and the Regional Ethical Committee, and written informed consent was obtained from all participants after complete description of the study.

### Statistical Analysis

Previous studies, in our sample and others, have failed to find either quantitative or qualitative sex differences in genetic and environmental influences on BPD.<sup>4,5</sup> Our models were therefore run with equal parameters specified for male and female subjects. The

phenotype under study here is not BPD at the diagnostic level but the BPD criteria. Data used in the analyses thus came from all participants in the sample regardless of whether they fulfilled diagnostic criteria for BPD or other personality disorders. Using multiple threshold tests, we have shown elsewhere that the 0 to 3 coding of the BPD criteria represents different degrees of severity on a single continuous liability dimension.<sup>5</sup> Owing to low endorsement, the 2 upper categories (2 and 3) were collapsed in the current study. We thus used an ordinal variable with 3 categories: 0 indicates not present; 1, subthreshold; and 2, present or strongly present.

In the classic twin model, individual differences in liability are assumed to arise from 3 latent sources: additive genetic (A), that is, genetic effects that combine additively; common or shared environment (C), which includes all environmental exposures that are shared by the twins and contribute to their similarity; and individual-specific or unique environment (E), which includes all environmental factors that contribute to differences between the twins plus measurement error. Because monozygotic (MZ) twins share all their genes and dizygotic (DZ) twins share on average 50% of their segregating genes, A contributes twice as much to the resemblance in MZ as to that in DZ twins for a particular trait or disorder. Both MZ and DZ twins are assumed to share all their C factors and none of their E factors.

Model fitting was performed using the raw data option in the software package Mx,<sup>23</sup> which allows for full information maximum likelihood estimation of parameters using incomplete data, such as the unmatched “singleton” twins in our sample. A Cholesky decomposition is the simplest way to decompose the covariance matrix between the 9 BPD criteria into genetic and environmental components. This is a fully parameterized descriptive model that imposes no restrictions on the underlying structure of the genetic and environmental influences.

The independent pathway model specifies direct paths from 1 or more common genetic and environmental factor to all criteria as well as paths from specific genetic and environmental factors for each criterion. In the simplest version of this model, 3 common factors (A, C, and E) are specified, in addition to specific factors (A, C, and E) for each criterion. The number of common factors can be extended to test more complex models.

The common pathway model is a more restricted version of the independent pathway model wherein the covariation between the criteria is accounted for by 1 or more common latent factor influenced by genetic and environmental factors. The simplest form of this model (a single-factor common pathway) specifies 1 common latent factor influencing all 9 BPD criteria and the genetic and environmental factors influencing it (A, C, and E). The number of latent factors can be extended depending on the hypothesis to be tested.

The independent and common pathways are nested submodels of the full Cholesky decomposition with fewer parameters. In this study, the full A, C, E Cholesky decomposition therefore serves as the baseline model against which the more restricted models are tested. The fit of the alternative submodels can be compared by using the difference in twice the log likelihood ( $2\ln L$ ), which, under certain regularity conditions, is asymptotically distributed as  $\chi^2$  with degrees of freedom ( $df$ ) equal to the difference in number of parameters ( $\Delta\chi^2$  test). According to the principle of parsimony, models with fewer parameters are preferable if they do not result in a significant deterioration of fit. A commonly used index of parsimony is the Akaike information criterion (AIC), calculated as  $-2\ln L - 2 df$ .<sup>24</sup> A lower AIC value indicates a superior fit.

We started by fitting a full Cholesky decomposition, specifying A, C, and E latent factors. Subsequently, genetic or shared environmental factors were eliminated to test whether a

more parsimonious model fit the data better than the full model. In a second set, independent pathway models specifying different numbers of genetic and environmental factors were run in several steps. With 9 observed variables, a maximum of 4 factors can be estimated. Accordingly, a model with 4 genetic and 4 environmental factors was fit in the first step. In subsequent models, the number of factors was reduced by dropping 1 from each of the biometric components (A and E) in each step. Finally, common pathway models were tested, with 1 to 4 latent factors influenced by genetic and environmental factors and by genetic and environmental factors specific for each criterion.

After we identified the best-fitting independent and common pathway models, we tested for the significance of item-specific genetic factors. To obtain decent starting values for each step in the model reduction, we performed exploratory factor analysis with varimax rotation on the expected covariance matrices (ie, genetic and environmental covariances for independent pathway models and phenotypic covariances for common pathway models). All models were fit to 2 zygosity groups without sex stratification.

## Results

Of the 2801 participants in the study, 2794 (1022 male and 1772 female) had valid data on the BPD criteria. The final sample consisted of 221 MZ male pairs, 116 DZ male pairs, 448 MZ female pairs, 261 DZ female pairs, 340 DZ opposite-sex pairs, and 22 single responders.

Table 1 shows the prevalence of endorsed criteria (criterion score,  $\geq 1$ ). Affective instability (criterion 6) was most commonly reported (20.4%), followed by impulsive potentially damaging self-harm (criterion 4) and inappropriate or intense anger (criterion 8) (18.8% and 17.8%, respectively). The criteria comprising the self-image disturbance dimension (criteria 3 and 9) were least frequently endorsed (2.3% and 5.5%, respectively). Table 1 also shows sex differences in endorsement. Impulsive self-harm (criterion 4) was reported more frequently by male participants, whereas unstable relationships, suicidal behavior, affective instability, feeling of emptiness, and inappropriate anger (criteria 2, 5, 6, 7, and 8) were reported significantly more frequently by female participants. Based on endorsed criteria above threshold (criterion score,  $\geq 2$ ), the prevalence of BPD in the sample was 0.4%.

The phenotypic correlations between the criteria are shown in eTable 1 in the Supplement. Of the 36 correlations, 10 were 0.50 or higher and 25 were 0.40 or higher. The twin correlations are presented in Table 2. All MZ correlations were less than 2 times the corresponding DZ correlation, suggesting absence of nonadditive genetic effects.

The model-fitting results are shown in eTable 2 in the Supplement. Given that the twin correlations indicated no non-additive genetic effects, the full Cholesky decomposition (model 1), against which all other models were compared, included all 9 criteria and specified genetic, shared environmental, and unique environmental effects. Models 2 and 3 are more parsimonious versions in which shared environmental or genetic effects, respectively, were set to 0. Model 2, specifying only genetic and unique environmental effects, fit the data best. Subsequent models were therefore fit without shared environmental effects.

We fit 12 independent factor models with varying numbers of genetic and environmental risk factors (models 4 through 15 in eTable 2 in the Supplement). We started with 4 genetic and 4 environmental factors (model 4). In steps 1 through 4, we were able to reduce the 8-factor model to a model with 1 genetic and 3 environmental factors (model 11). Further reduction resulted in a deterioration of fit compared with model 11. We then sought to remove the item-specific genetic factors, but this was not possible without a significant reduction in fit (model 15). The best-fitting independent pathway model was therefore

model 11, which included 1 genetic factor and 3 environmental factors (AIC,  $-12.34$ ). The genetic factor substantially influenced all 9 criteria (standardized path coefficients, 0.31–0.57). The 3 environmental factors included 1 general factor that influenced all criteria, 1 factor influencing the criteria making up the affective dimension, and 1 factor influencing the 2 criteria making up the interpersonal dimension (results not shown in eTable 2).

Models 16 through 20 are common pathway models with 1, 2, 3, or 4 latent factors. The 3-factor common pathway model (model 18) fit the data best (AIC,  $-120.39$ ) and fit better than all the independent pathway models. We were not able to remove the item-specific genetic factors without a significant reduction in fit (model 20).

The Figure. shows the best-fitting common pathway model with parameter estimates. The squared path coefficients give us the amount of variance explained by each latent factor. Paths shown in color are those with parameter estimates of 0.32 or greater (so they explain 10% or more of the phenotypic variance). The first latent factor seems to be a general BPD factor substantially influencing all 9 criteria, with standardized path coefficients ranging from 0.53 to 0.79. It is strongly influenced by genetic factors, with a heritability of 55% ( $0.74^2$ ). The remaining 45.0% of the variance is explained by unique environmental factors. This suggests that all the BPD criteria are strongly influenced by common genetic and environmental factors that increase the risk for all the dimensions and criteria of BPD. We labeled this the *BPD factor*.

The second liability factor strongly influenced criterion 2, unstable relationships (standardized path coefficient, 0.74) but also had some effect on criterion 1, avoidance of abandonment, the other criterion making up the interpersonal dimension of BPD. This factor, which we labeled the *interpersonal factor*, was mostly (97.8%) influenced by environmental factors (heritability, 2.2%).

The third factor mainly influenced the 3 criteria that make up the affective dimension of BPD, that is, criteria 6, affective instability, 7, feelings of emptiness, and 8, intense anger. The affective instability criterion was most strongly influenced by this factor (standardized path coefficient, 0.63), which we labeled the *affective factor*. Its influences on most of the other criteria were negligible. Unique environmental influences accounted for 70.7% of the variance in this factor, and its heritability was 29.3%.

Specific genetic effects on the criterion level were found for the impulsivity dimension (criteria 4 and 5; standardized path coefficients, 0.52 and 0.38, respectively) and 2 criteria in the affective dimension (criteria 7 and 8; standardized path coefficients, 0.42 and 0.30). Self-image (criteria 3 and 9) and the interpersonal dimension (criteria 1 and 2) had negligible specific genetic influence, and almost all the genetic effects on these criteria came from the common latent BPD factor.

Table 3 shows the total influence of genetic factors on the 9 DSM-IV criteria for BPD and the specific sources of genetic influences as predicted by our best-fit model. The 2 criteria making up the impulsivity dimension of BPD (criteria 4 and 5) had the highest heritability, with genetic influences coming from the general BPD factor and criterion-specific factors. Genetic risk factors for the criteria making up the interpersonal dimension (criteria 1 and 2) and the self-image dimension (3 and 9) came almost exclusively from the general BPD factor and showed the lowest heritability. The criteria making up the affective dimension (6, 7, and 8) were influenced by genetic factors associated with the general BPD factor as well as the affective and criterion-specific factors.

Sources of environmental influence are shown in Table 4. All criteria were influenced by environmental factors through the general BPD factor but to a lesser extent than for the

genetic factors. The interpersonal factor accounted for 70.1% of the environmental influence on the unstable relationships criterion (criterion 2) and 7.3% of the environmental influence on avoidance of abandonment (criterion 1). The other criteria showed negligible influence from this factor (0.1%–1.8%), indicating that the environmental influence from this factor is specific to the interpersonal dimension of BPD. The environmental influence through the affective factor seems to be relatively specific to the affective dimension of BPD (criteria 6, 7, and 8), with low effect (0%–6.3%) on the other criteria.

Substantial specific unique environmental effects were found for all criteria (except criterion 2, unstable relationships), perhaps because measurement error is included in these estimates. The low specific effect on criterion 2 is probably due to the very strong loading of this criterion on the interpersonal factor (standardized path coefficient, 0.74), which is essentially an environmental liability factor.

## Discussion

To our knowledge, ours is the first study to fully explore the structure of genetic and environmental risk factors for DSM-IV BPD by fitting a large series of models to data for all 9 BPD criteria in a population-based sample of young adult twins. The best-fitting model was dominated by 1 highly heritable general BPD factor, which strongly influences all 9 BPD criteria, and 2 factors more or less specific for the interpersonal and the affective dimension, respectively. Both of these were mainly influenced by environmental factors. With 1 exception (criterion 2, unstable and intense relationships), the specific criteria were strongly influenced by environmental factors, whereas 5 of the 9 criterion-specific genetic effects were either 0 or negligible.

Our identification of a highly heritable general BPD liability factor parallels results from 2 recent twin studies based on questionnaire data, which found that 1 common liability factor best accounted for the relationship between the higher-order BPD traits.<sup>6,7</sup> It is also in accordance with results from a family study of BPD, which showed that a model in which the 4 psychopathological sectors represent manifestations of a single latent BPD construct fit the data best.<sup>3</sup>

The general BPD factor was substantially influenced by genetic factors, with a heritability of 55%. This finding is similar to the heritability estimates in the above-mentioned twin studies, which were 60%<sup>7</sup> and 51%,<sup>6</sup> and in the family study by Gunderson et al<sup>3</sup> (43.9% or 58.6%, depending on method of assessment). The heritability estimates in all of these studies are higher than what has been reported from studies using dimensional representations of *DSM-IV* personality disorders<sup>5</sup> or sum scores of questionnaire items.<sup>4</sup> This difference is probably due to the fact that the effects of measurement error are largely eliminated by examination of a common factor.

The general BPD factor accounted for most of the genetic influence on all criteria except for the impulsive self-harm criterion (criterion 4), which received almost two-thirds of its genetic influence from criterion-specific factors. This is especially interesting in light of the proposal by the American Psychiatric Association to add nonsuicidal self-injury as a psychiatric disorder in the *DSM-5*.<sup>25</sup> The feeling of emptiness criterion (criterion 7) received equal genetic influence from the general BPD factor and criterion-specific genetic factors. The common factor that specifically influenced the affective dimension accounted for about one-third of the genetic influence on the affective instability criterion (criterion 6), and the general BPD factor explained the rest of the genetic effects.

Taken together, these findings support the hypothesis put forth by Livesley<sup>26</sup> that BPD is a heterogeneous disorder encompassing several primary traits but that the genetic architecture is assumed to involve a single common genetic factor that influences all the primary traits in addition to multiple trait-specific genetic factors.

All the individual BPD criteria, and thus all 4 higher-order dimensions or sectors, were significantly influenced by genetic factors. This result is in accordance with findings of previous family<sup>2,3</sup> and twin<sup>6,7,27,28</sup> studies. Our findings are also in accordance with those of more recent brain imaging studies of patients with BPD compared with normal controls. Differences have been found in structural changes<sup>29-31</sup> and functional responses<sup>30,31</sup> in brain regions involved in emotional regulation,<sup>32-35</sup> impulsivity,<sup>36,37</sup> and interpersonal relationships.<sup>38,39</sup>

The environmental risk factors for BPD were mostly criterion specific. In contrast to what was found for genetic factors, the common general BPD factor accounted for only about one-quarter of the unique environmental influence on all criteria except for the suicidal behavior criterion (criterion 5), where it accounted for 54.2%. The interpersonal factor accounted for 70.1% of unique environmental influences on the unstable relationships criterion (criterion 2), suggesting that specific environmental factors account for much of the liability to unstable relationships. Gunderson and Lyons-Ruth<sup>40</sup> pointed out parents' contributions to early attachment and development of interpersonal hypersensitivity, and Fonagy and Luyten<sup>41</sup> have emphasized the importance of the childhood attachment environment for the development of mental properties relevant to BPD. The affective factor accounted for 42.2% of the affective instability criterion (criterion 6), but the rest of the criteria were influenced mainly by criterion-specific factors.

There is a vast literature documenting an association between BPD and traumatic life events, including childhood physical and sexual abuse.<sup>42-45</sup> However, the extent to which these factors have a causal effect on BPD is unclear.<sup>46</sup>

The best-fitting models included only A and E factors. Given our moderate sample size, our power to detect C effects is limited. According to the twin correlations shown in Table 3, there is evidence only of low-magnitude C effects.

## Implications

The results from this study indicate that most of the genetic effects on the BPD criteria were concentrated in a single general BPD factor. This suggests that as a first approach for gene-finding studies, it might be advised to concentrate on an aggregate measure of risk for BPD rather than on specific subdimensions.

Although the BPD construct has undergone minimal changes from its introduction in 1980,<sup>47,48</sup> our findings indicate that it has good psychometric properties and that, from a genetic perspective, the *DSM-IV* criteria for BPD seem to reflect a single dimension of liability. This indicates that caution should be shown when revising the diagnosis for *DSM-5*.

The close etiological connection between the psychopathological dimensions in BPD supports the recommendation that treatment for BPD should target all dimensions of the disorder rather than be directed at individual sectors.<sup>3</sup> It is not possible to tell how this should be done from our findings, but, in theory, treatment that can target the common etiological factor would thereby influence all the BPD symptoms. From a clinical viewpoint, it could also be helpful to know that impulsivity levels of patients with BPD are highly heritable and likely to reflect stable temperamental features. By contrast, self-image and the



interpersonal dimension are much more likely to have been influenced by prior life experiences, with considerably more modest genetic contributions.

### Strengths and Limitations

The current study has 3 main methodological strengths compared with previous studies. First, it was performed in a large population-based sample. Second, the participants were mostly interviewed face to face with a interview wherein all items were assessed in all participants. Third, all 9 *DSM-IV* criteria were assessed in all subjects, giving us the opportunity to explore a large number of models, which has not been possible in previous studies.

Four methodological limitations should be considered when the results are interpreted. First, in this population-based study, few subjects endorsed many of the criteria. Most of the information therefore comes from subthreshold levels of BPD symptoms. However, previous analyses have indicated that different numbers of endorsed criteria represent different degrees of severity on the same continuum.<sup>5</sup>

Second, although previous studies indicate no sex differences in the genetic and environmental influence on BPD,<sup>4,5</sup> they may exist. However, our statistical power to detect such effects is limited.<sup>49</sup> Given the size of our sample and the computational challenges of the analyses, we did not test whether any significant sex effects were present on the criterion level.

Third, these results were obtained from a particular population, young adult Norwegian twins, and may not extrapolate to other cultural, ethnic, and age groups. The prevalence of personality disorders varies across studies. In our sample, estimates for *DSM-IV* personality disorders are similar to rates recently reported across 10 countries.<sup>50</sup> Our prevalence for BPD was similar to previous estimates from the United Kingdom<sup>51</sup> but lower than in recent studies from the United States.<sup>52</sup>

Fourth, substantial attrition was observed in this sample from the birth registry through 3 waves of contact. We reported elsewhere<sup>53</sup> detailed analyses of the predictors of non-response across waves. Briefly, cooperation was strongly predicted by female sex, monozygosity, and higher educational status but not by symptoms of psychiatric disorders. A series of analyses did not show any evidence of changes in the genetic and environmental covariance structure due to recruitment bias for a broad range of mental health indicators in the second questionnaire. Although we cannot be certain that our sample was psychopathologically representative, these findings suggest that a substantial bias is unlikely.

Finally, there are inherent limitations in all twin studies using phenotypic symptom-based data. The methods used can offer only a coarse approximation of the likely true underlying genetic and environmental structure. For example, the method assumes no gene  $\times$  environment interaction or assortative mating, and it is also dealing with latent liability factors rather than measurable factors.

### Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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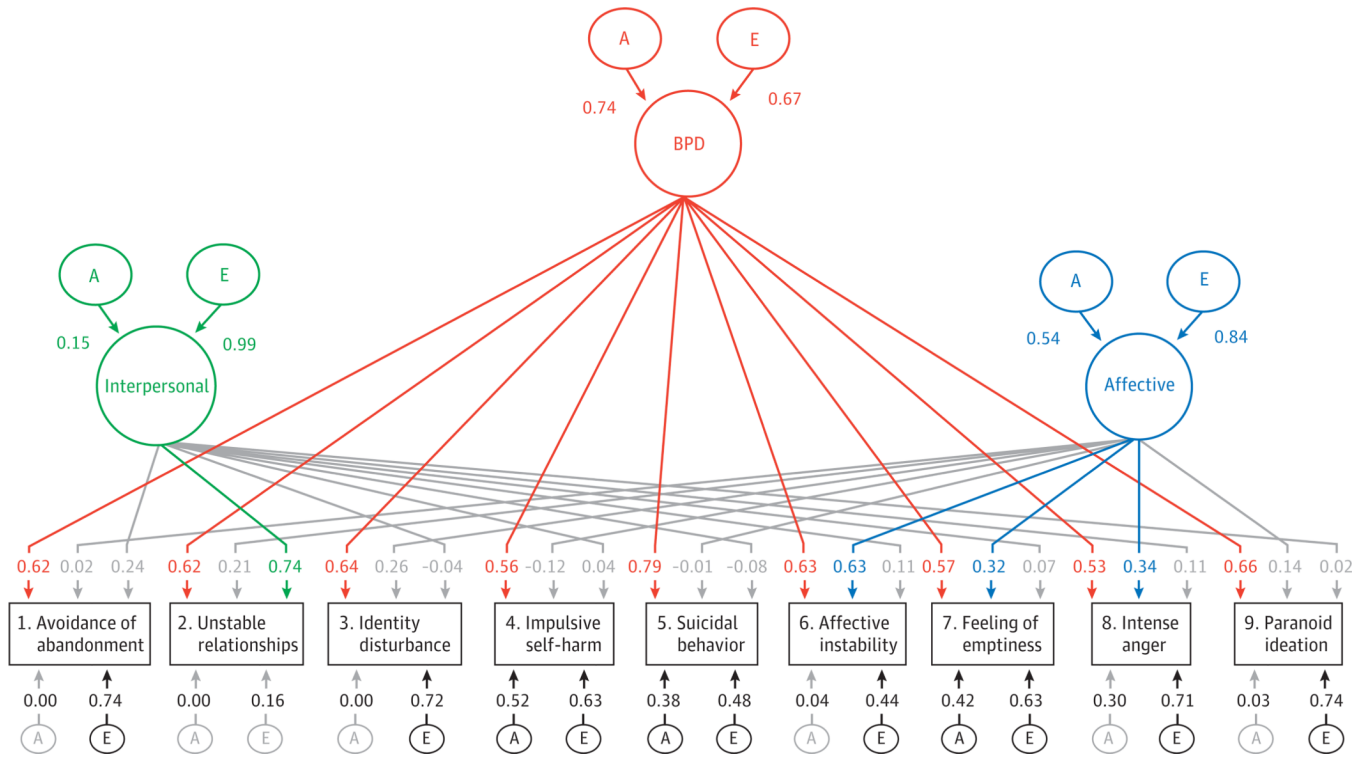
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**Figure. Genetic and Nonshared Environmental Parameter Estimates From the Best-Fitting Model for the 9 DSM-IV Criteria for BPD**

Values represent path coefficients, which need to be squared to calculate the proportion of variance in liability accounted for by the latent variables. Paths with values of 0.32 or greater (accounting for 10% or more of the phenotypic variance) from the 3 common latent factors are shown in green, red, and blue. Criterion-specific paths are shown in black. Paths not exceeding 0.32 are depicted in gray. A indicates additive genetic effects; BPD, borderline personality disorder; E, nonshared environmental effects.

**Table 1**Prevalence of *DSM-IV* Criteria (Score 1) and Dimensions for Borderline Personality Disorder

Dimension	Criterion	Participants, No. (%)		
		Total (N = 2794)	Female (n = 1772)	Male (n = 1022)
Interpersonal	1. Frantic efforts to avoid real or imagined abandonment	225 (8.1)	156 (8.8)	69 (6.8)
	2. A pattern of unstable and intense interpersonal relationships characterized by alternating between extremes of idealization and devaluation	334 (12.0)	230 (13.0)	104 (10.2) <sup>a</sup>
Impulsivity	4. Impulsivity in at least 2 areas that are potentially self-damaging	526 (18.8)	254 (14.3)	272 (26.6) <sup>a</sup>
	5. Recurrent suicidal behavior, gestures, or threats or self-mutilating behavior	162 (5.8)	121 (6.8)	41 (4.0) <sup>a</sup>
Affective	6. Affective instability due to a marked reactivity of mood	571 (20.4)	418 (23.6)	153 (15.0) <sup>a</sup>
	7. Chronic feelings of emptiness	304 (10.9)	224 (12.6)	80 (7.8) <sup>a</sup>
	8. Inappropriate, intense anger or difficulty controlling anger	498 (17.8)	361 (20.4)	137 (13.4) <sup>a</sup>
Self-image	3. Identity disturbance: markedly and persistently unstable self-image or sense of self	65 (2.3)	46 (2.6)	19 (1.9)
	9. Transient, stress-related paranoid ideation or severe dissociative symptoms	154 (5.5)	102 (5.8)	52 (5.1)

<sup>a</sup>Significant association ( $P < .05$ ).

**Table 2**Twin Correlations for *DSM-IV* Criteria for Borderline Personality Disorder<sup>a</sup>

Criterion	Criterion No.								
	1	2	3	4	5	6	7	8	9
1. Avoidance of abandonment									
MZ twins	0.21	0.20	0.19	0.20	0.15	0.16	0.19	0.12	0.18
DZ twins	0.14								
2. Unstable relationships									
MZ twins	0.23	0.23	0.17	0.23	0.23	0.22	0.25	0.14	0.20
DZ twins	0.14	0.17							
3. Identity disturbance									
MZ twins		0.24	0.17	0.22	0.23	0.26	0.13	0.19	
DZ twins	0.14	0.16	0.17						
4. Impulsive self-harm									
MZ twins			0.44	0.24	0.12	0.18	0.14	0.15	
DZ twins	0.14	0.14	0.13	0.27					
5. Suicidal behavior									
MZ twins				0.50	0.30	0.26	0.17	0.23	
DZ twins	0.14	0.20	0.18	0.21	0.42				
6. Affective instability									
MZ twins					0.32	0.22	0.15	0.23	
DZ twins	0.11	0.15	0.15	0.10	0.23	0.21			
7. Feeling of emptiness									
MZ twins						0.39	0.12	0.19	
DZ twins	0.16	0.20	0.21	0.16	0.23	0.16	0.34		
8. Intense anger									
MZ twins								0.21	0.18
DZ twins	0.10	0.11	0.11	0.12	0.17	0.13	0.12	0.15	
9. Paranoid ideation									
MZ twins									0.29

	Criterion No.								
Criterion	1	2	3	4	5	6	7	8	9
DZ twins	0.14	0.16	0.15	0.14	0.23	0.19	0.16	0.16	0.25

Abbreviations: DZ, dizygotic; MZ, monozygotic.

<sup>a</sup>Data represent polychoric correlations based on the full A, C, E Cholesky decomposition (model 1; eTable 2 in the Supplement); see the Statistical Analysis subsection in the Methods section for a full description of the model. MZ twin correlations are in bold.



**Table 3**Sources of Genetic Influence on *DSM-IV* Criteria for Borderline Personality Disorder

Criterion	Genetic Influence, %				
	Total Genetic Influence	Borderline Personality Disorder Factor	Affective Instability Factor	Interpersonal Factor	Criterion-Specific Factors
1. Avoidance of abandonment	21.2	99.3	0.1	0.6	0
2. Unstable relationships	23.6	89.3	5.5	5.2	0
3. Identity disturbance	24.4	91.9	8.1	0	0
4. Impulsive self-harm	44.6	38.5	0.9	0	60.6
5. Suicidal behavior	48.6	70.3	0	0	29.7
6. Affective instability	33.5	64.9	34.6	0.1	0.5
7. Feeling of emptiness	38.4	46.3	7.8	0	45.9
8. Intense anger	27.8	55.4	12.1	0.1	32.4
9. Paranoid ideation	24.5	97.3	2.3	0	0.4

**Table 4**  
Sources of Environmental Influence on *DSM-IV* Criteria for Borderline Personality Disorder

Criterion	Environmental Influence, %				
	Total Environmental Influence	Borderline Personality Disorder Factor	Affective Instability Factor	Interpersonal Factor	Criterion-Specific Factors
1. Avoidance of abandonment	77.7	22.2	0	7.3	70.5
2. Unstable relationships	76.6	22.5	4.1	70.1	3.3
3. Identity disturbance	75.2	24.5	6.3	0.2	69.0
4. Impulsive self-harm	54.9	25.6	1.8	0.3	72.2
5. Suicidal behavior	51.7	54.2	0	1.2	44.6
6. Affective instability	66.4	26.8	42.2	1.8	29.2
7. Feeling of emptiness	62.0	23.5	11.7	0.8	64.0
8. Intense anger	72.4	17.4	11.3	1.6	69.7
9. Paranoid ideation	75.7	25.8	1.8	0.1	72.3