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Borderline personality disorder traits and their relationship with dimensions of normative personality: a web-based cohort and twin study

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Abstract

Objective—To describe the structure of genetic and environmental risk factors for four dimensions of borderline personality disorder (BPD) and to understand the source of resemblance of these dimensions and normal personality.

Method—A web-based sample (*n* = 44,112 including 542 twin pairs) completed items from 4 scales of the *Dimensional Assessment of Personality Pathology Basic Questionnaire* and the Big Five Inventory.

Results—A one-factor common pathway model best fits the 4 BPD scales producing a highly heritable latent liability (heritability = 60%) and strong loadings on all 4 dimensions. Affective instability had the lowest trait-specific genetic loading, suggesting that it was a core feature of BPD. A complex pattern of genetic and environmental associations was found between the big five personality traits and BPD dimensions. The strongest genetic correlations with the BPD traits were generally seen for neuroticism (positive), followed by conscientiousness and agreeableness, both negative.

Conclusion—In the general population, these four BPD dimensions reflect one underlying highly heritable factor. The association between normative personality and dimensions of BPD is complex with high degrees of genetic correlation.

Keywords

borderline personality disorder; twins; personality; genetics

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Declaration of interest
None.

Introduction

Borderline personality disorder (BPD) is defined by the *Diagnostic and Statistical Manual for Mental Disorders (DSM-IV)* (1) as a categorical disorder with 9 criteria including affective instability, unstable interpersonal relationships, impulsivity, identity problems (IP), and transient cognitive distortion. Given that only five of these criteria must be present, there are more than 250 ways to fulfill a BPD diagnosis. It is therefore not surprising that exploratory factor analyses give mixed results, often with three (2, 3) or four (4, 5) highly correlated factors. However, confirmatory factor analyses have generally supported a unidimensional structure (6–9).

An alternative dimensional conceptualization of BPD has been proposed by Livesley (10). Based on the *Dimensional Assessment of Personality Pathology – Basic Questionnaire* (*DAPP*) (11, 12), which currently consists of 18 primary traits and four secondary clusters, Livesley hypothesized BPD to be a heterogeneous disorder that encompasses several primary traits and that the genetic architecture of BPD traits is assumed to involve a single common genetic factor that influences all primary traits and multiple trait-specific factors (10).

Previous research indicates that most DSM-IV diagnostic criteria for PDs can be accommodated within the DAPP (13, 14). Furthermore, several authors have argued that normal and abnormal personality can be treated within a single structural framework, and hierarchical models that integrate the two levels of description of personality have been proposed (14). Numerous studies have shown that PDs as defined by the DSM system can be represented by the five-factor model of personality (FFM) (15–17). These results suggest that the traits delineating normal and abnormal personality are continuous and normally distributed and that PDs represent the extremes of these traits (13). The implication of this contention is that the etiological factors underlying normal and abnormal personality are either identical or highly correlated and that the high phenotypic correlation and joint factor structure observed between the two measures should result from common genetic and environmental factors. The prior literature, summarized in a recent meta-analysis by Samuel and Widiger (15), suggests that we should find the strongest (positive) phenotypic associations for our BPD factors with neuroticism (N), followed by negative associations with conscientiousness (C) and agreeableness (A). Would our findings, obtained from a web-based general population sample, mirror these prior results deriving largely from clinical samples?

Aims of the study

This paper has two major goals. The first is to describe the structure of the genetic and environmental risk factors for four major dimensions of borderline personality disorder (BPD) pathology as reported in a general population sample. The second goal is to understand the resemblance between these BPD dimensions and both the underlying BPD factor and the big five normative personality traits as well as to clarify the degree to which these resemblances are the result of genetic vs. environmental factors.

Material and methods

Sample

As outlined in detail elsewhere (18), participants in this study were part of data collected from ‘Twins: an interactive personality test’ from July 1, 2005, to May 1, 2008. This survey, designed as an interactive assessment tool for measures of personality, psychopathology, and substance use and dependence, permits any two people, that is, twins or any other biologic or social relationship, to compare their personalities and behaviours. Participants

could take the survey as individuals. All participants were volunteers and were recruited over the World Wide Web. Potential respondents found out about the site via internet search engines, direct access to its address (http://www.outofservice.com/twins/), or through links from other sites.

Data were collected with automated computerized administration, data entry and scoring, and all participants received individualized feedback after completing the survey. The data described in this article were collected using a non-commercial, advertisement free web site (http://www.outofservice.com) that contains personality measures as well as several games, quizzes, and questionnaires for entertainment purposes. Participants did not provide any identifying information and anonymity was assured. This research obtained exempt ethics approval at Virginia Commonwealth University. Twins were matched to each other through the use of linked passwords. Permission had to be given on the web site to compare their results with their cotwin.

These data contained 44 112 completed questionnaires with unique user codes. This sample was 65.3% women, 85.4% 18 years or older, and 72.0% Caucasian. The mean (SD) of their current ages was 25.2 (11.8). The remaining ethnic breakdown was 6.6% Other, 5.7% Black, 3.5% Latino, 3.0% Indian/Pakistani, 2.8% Chinese, and 1.5% or less the remaining others.

We utilized a variety of quality control measures to assess the amount of duplicate or faked responses, or false twin pairs in the sample (18). These methods included an examination of distributions of our personality measures (which found no excess of extreme scores), an examination of reported year of birth, height, and weight in twin pairs, and a follow-up of the small number of positive responses to an item in our questionnaire about duplicate entries. Consistent with other internet samples (19), these queries suggested low levels of faked or duplicate data.

The 609 twin pairs where zygosity could be determined included 364 female–female monozygotic (MZ) twins, 80 female–female dizygotic (DZ) twins, 77 male–male MZ twins, 21 male–male DZ twins, and 67 opposite sex DZ twins. Of the members of these twin pairs, 84.6% were 18 years or older and 85.1% were White/Caucasian. We restricted our analyses to 542 same-sex pairs because of the very low power to test qualitative sex effects with our modest number of opposite sex DZ twins.

Zygosity was assessed by responses in both twin pairs to the three items found most discriminating when tested against DNA results in the Virginia Adult Twin Study of Psychiatric and Substance Use Disorders (20). When the responses of the two twins were inconsistent, those pairs (n = 9) were eliminated from the study.

Assessments

In consultation with the developer of the Dimensional Assessment of Personality Problems – Basic Questionnaire (DAPP-BQ) scale (Dr. W. J. Livesley) (12), we identified 4 scales that we judged to assess core components of the BPD construct: affective lability (AL), cognitive dysregulation (CD), IP, and IA. Because of space limitations, KSK chose 6 of the original 16 items in each scale on the basis of item content, with the goal of assessing those personality characteristics most representative of the BPD syndrome as seen clinically. (These items are given in Table 1). Using the CORR procedure in SAS (21), Cronbach's alpha for these scales was estimated at AL 0.85, IP 0.65, CD 0.89, and IA 0.85.

Normative personality dimensions were assessed by the Big Five Inventory (BFI) Personality Test (22), a 44-item scale that assesses openness to experience (O), conscientiousness (C), extraversion (E), agreeableness (A), and neuroticism (N). Responses

to these items are recorded by a 5-point Likert scale: i) Strongly disagree, ii) Disagree a little, iii) Neither disagree nor agree, iv) Agree a little, and v) Strongly agree. In the entire sample, these variables were relatively normally distributed with estimates of skewness ranging from −0.50 for A to +0.04 for N. Cronbach's alpha for these scales was as follows: O 0.78, C 0.83, E 0.85, A 0.79, and N 0.83.

Analyses

Exploratory factor analysis, utilizing the varimax rotation, was performed using the SAS procedure FACTOR (21). Linear regression analyses were performed using SAS procedure REG (21).

We conducted two types of multivariate twin analyses in this report, the first of which sought to clarify the genetic and environmental inter-relationships amidst our four BPD scales and the second of which explored the genetic and environmental sources of covariance between our four BPD scales and the five normative personality dimensions, considered one at a time.

These analyses assume that twin resemblance arises from two latent factors: i) additive genes, contributing twice as much to the MZ as to the DZ twin correlation and ii) shared or 'common' environment, which contributes equally to the correlation in MZ and DZ twins. In addition to this 'common' environment, the model also contains individual-specific environment that reflects measurement error and those environmental experiences that make members of a twin pair different. Multivariate twin models can be understood as a combination of factor analysis with the traditional twin model. In our analysis of the four BPD scales, we first examined a single common factor model, in which genetic and environmental effects common to the four scales flow through a single factor. This model also then includes genetic and environmental influences unique to each individual scale. We also examined the fit of an independent pathway model that permitted genetic and environmental effects to flow directly to the individual scales.

The model used to explore the inter-relationship between our four BPD scales and the personality dimensions was somewhat more complex and is depicted in Fig. 2 for genetic factors. In this model, additive genetic factor 1 (or A1) represents the genes specific to the given personality dimension. A6 represents genes that are common to all four of the BPD scales, and A2, A3, A4, and A5 represent genes specific to the individual BPD scales of AI, CD, IP, and IA respectively. The model contains five genetic correlations that reflect the degree of sharing of genetic factors for the two traits in question. The first of these reflects the correlation between the genetic factor for the personality dimension (A1) and the genetic factor common to the 4 BPD scales (A6). Correlations 2 through 5 reflect the genetic relationship between the personality dimensions and genetic effects specific to the individual BPD factor. An important feature of the model is that it provides two pathways through which genetic effects on personality can correlate with genetic effects on individual BPD factors. Let us illustrate this for affective instability in Fig. 2. The genetic correlation between the personality dimension and affective instability could be through correlation 1 via the common genetic factor influencing all BPD scales or through correlation 2 via the genes that are specific in their influence on affective instability. Analyzing four twin-zygosity groups enables us to examine quantitative sex effects – that is, whether the magnitude of genetic effects on the BPD factors and personality is the same in men and women.

Using the software package Mx (23), we fit models by the method of maximum likelihood to data from all same-sex twin pairs. For our model fitting, we polychotomized the distributions of the DAPP and BFI scales into five ordered categories. Prior power analyses

have shown that this approach, which renders analyses much less sensitive to distributional anomalies or influential outliers, maintains nearly all the power of the underlying continuous variable (24).

We used Akaike's information criterion (AIC) (25, 26) for model selection. The *lower* its value, the better the balance between explanatory power and parsimony.

Results

Borderline personality disorder traits

We began with a Varimax rotated factor analysis of the 24 items chosen from the 4 DAPP scales of AI, CD, IP, and IA in our entire sample (n = 44,112). As seen in Table 1, we were able to recover the four factors quite cleanly. Each item loaded most heavily on the scale to which it was assigned. For nearly all items, the item loaded much more strongly on the scale from which it came than on any of the other three scales.

The Pearson correlations between the four BPD traits in our entire sample (n = 44,112) were as follows: AI-CD +0.47; AI-IP +0.48; AI-IA +0.49; CD-IP +0.61; CD-IA +0.41; and IP-IA +0.35. A factor analysis of these four traits produced a single Eigen value above unity (2.40) accounting for 60.1% of the variance. Loadings on this single factor were as follows: AI +0.79; CD +0.81; IP +0.79; and IA +0.71.

Table 2 presents, in the MZ and DZ members of our twin sample, the mean and standard deviation of the four BPD traits as well as the observed polychoric correlations in the complete pairs. Our baseline model to which we compare all subsequent models for the twin analyses of these four BPD traits was a common pathway model with additive genetic, common environmental and individual-specific environmental influences, and separate parameter estimates in men and women (model 1; - 2LL 12,694.8, df = 4276). When we constrained estimates to equality across the sexes, the AIC fit index improved substantially (model 2; $\Delta\chi^2$ = 14.7, Δdf = 18, ΔAIC −21.3). In model 3, we set all shared environmental pathways to zero with a large further improvement in fit ($\Delta\chi^2$ = 18.1, Δdf = 23, ΔAIC −27.9). If, in model 4, we instead set all the additive genetic effects to zero, the model fit deteriorated substantially ($\Delta\chi^2$ = 38.3, Δdf = 23, ΔAIC −7.7). We also fitted independent pathway models (details not shown). The best fit independent pathway model also constrained estimates to equality across the sexes and dropped all shared environmental parameters. However, this model did not fit as well as the best-fit common pathway model ($\Delta\chi^2$ = 13.4, Δdf = 16, ΔAIC −18.6).

The results of the best-fitting model 3 are depicted in Fig. 1. The latent liability to BPD traits was highly heritable (a^2 = 0.60) with the remaining variance in liability resulting from individual- specific environment (e^2 = 0.40). Loadings from the common factor were similar and strong for three of the BPD traits (AI +0.68, CD +0.72 and IP +0.69). The loading for IA was more modest (+0.54).

From this best-fit model, it was possible to calculate the total heritability (or a^2) for each of the four BPD traits and the proportion that resulted from the common factor vs. genetic factors unique to that trait. These results were as follows: AI a^2 = 0.42 (67% common /33% unique); CD a^2 = 0.51 (63% common /37% unique); IP a^2 = 0.53 (55% common /45% unique); and IA a^2 = 0.51 (35% common /65% unique).

Borderline personality disorder traits and normative personality disorder dimensions

As seen in Table 3 controlling for age and sex, in our entire sample (n = 43,799), we predicted the four BPD traits from the 'big five' personality dimensions: O, C, E, A, and N.

The proportion of variance predicted by this model was as follows: AI – 44.8%, CD – 29.1%, IP – 38.9%, and IA – 20.3%. Interestingly, all four BPD traits were predicted by high levels of N and low levels of C. Indeed, for every trait, N was the strongest single predictor. By contrast, the other big five dimensions had variable relationships with the individual BPD traits. O was positively related to AI and CD, uncorrelated with IP, and negatively related to IA. E was positively related to AI and IA and negatively related to CD and IP. A was positively related to IA and negatively related to CD, IP and, quite weakly, AI.

We also calculated the correlation between each individual personality dimension and the common factor obtained from the four BPD traits. As seen in Table 3, of these four phenotypic correlations, N was by far and away most strongly related to the BPD common factor (+0.63), followed by C (−0.38) and A (−0.29).

Table 2 presents, in the MZ and DZ members of our twin sample, the mean and standard deviation of the five personality dimensions and the observed polychoric correlation in the complete pairs. In prior analyses, as we saw for the BPD traits, we found no evidence for shared environmental effects with our five personality dimensions (18). Therefore, to simplify our model fittings to the BPD and OCEAN variables, we examine models containing only additive genetic and individual-specific environmental effects. Table 4 presents the AIC fit indices for each of these models. For four of the five analyses, the best fit was achieved with model II which constrained parameters to equality between men and women but allowed for genetic and environmental correlations between the personality dimension and both the latent risk to BPD and to each of the four specific BPD traits. Only for C did a simpler model, which contained correlations only between C and the latent risk to BPD, fit better. However, to permit comparisons across models, we present the results for model II for all five personality traits in Figs 2a–e. Our discussion here will focus on the genetic results, and we will concentrate on genetic correlations ≥ /0.20/.

With respect to the pattern of genetic correlations, these five best-fit models can be meaningfully divided into three groups (Table 5). Two of the personality dimensions (O and E) had quite modest *negative* correlations with the latent genetic BPD factor: −0.16 and −0.23 respectively. Interestingly, both had additional *positive* genetic correlations with AI, while O had a substantial positive correlation with CD and E a *negative* genetic correlation with IP. Two of the personality dimensions (C and A) had strong *negative* correlations with the latent genetic BPD factor: −0.66 and −0.57, respectively, with relatively modest additional correlations with specific BPD traits. The pattern with N was unique having not only a moderately strong *positive* correlation with the latent BPD genetic factor (+0.44) but also rather robust additional positive correlations with AI and especially IP. The direction of the environmental correlation between the personality dimensions and the latent BPD factor tended to mirror those of the genetic correlations. The environmental correlations tended to be lower than the genetic correlations for C, A, and N and higher for O and E.

Discussion

Borderline personality disorder traits

The covariance between the four main components of BPD: AI, CD, IP, and IA, could be best explained in our sample by one common latent factor, thus supporting the hypothesis proposed by Livesley (10). This latent liability to BPD traits was highly heritable (60%) with the remaining variance in liability resulting from individual-specific environment (40%). This heritability – which reflects a common factor – is not directly comparable to that obtained from a single scale or set of diagnostic criteria. It will be higher because errors

of measurement are ‘taken out’ and go into the specific environmental effects on the individual scales.

Loadings from the common factor were very similar and rather strong for three of the BPD traits (AI +0.68, CD +0.72 and IP +0.69) and substantial but more modest for IA (+0.54). The heritabilities for the four traits were as follows: AI, 42%; CD, 51%; IP, 53%; IA 51%. Specific genetic effects accounted for 33%, 37%, and 45% of the genetic effects in AI, CD, and IP, respectively, and for 65% in IA.

AI had the lowest trait-specific genetic loading, with 67% of the genetic effects coming from the common factor. This is consistent with Livesley’s suggestion that borderline traits are organized around the trait of anxiety and that an important feature of BPD is dysregulation of threat management system leading to pervasive fearfulness and unstable emotions (10). Linehan (27) proposed a theory suggesting that BPD arises primarily from defects in emotional regulation. A large body of empirical evidence supports the importance of AI/emotional dysregulation in BPD (28–30). In a study following patients with BPD over a 2-year period, AI was found to be the most frequent and stable DSM criterion, leading the authors to conclude that it reflects a trait-like symptom in contrast to less stable more reactive criteria (31). Siever (32) has suggested that AI could be a useful endophenotype for genetic studies of BPD.

IA had the highest trait-specific genetic loading with only 35% of the genetic variance coming from the common factor. This trait reflects unstable interpersonal relationships, or ‘interpersonal hypersensitivity,’ a trait postulated by Gunderson to play an important role in the development of BPD (33). Both in a 2-year (31) and 10-year follow-up of patients with BPD (34), AI and IA were the most stable symptoms.

Our results are consistent with findings from a large twin study using data from the Netherlands, Belgium, and Australia (NBA-sample) (35, 36). Although the method of assessment for BPD was different in the two studies, and the BPD traits examined were also not the same, the analytical methods were similar, and the results can be usefully compared. In both studies, a single-factor common pathway model with no sex limitations fitted the data best, indicating that one latent BPD factor best explained the covariance between the four BPD features examined. The variance in this common factor, reflecting liability to BPD, was explained by genetic factors and individual-specific environmental factors alone. The heritability of the latent BPD factor was 51% in the NBA sample study. The loadings from the common factor were somewhat stronger in our study but the genetic effects specific to the components were lower in the study based on the NBA sample (4–20%).

The heritability for the BPD factor in our study was similar to heritability of the BPD latent factor in the above-mentioned study (36). It was, however, considerably higher than for dimensional representations of DSM-IV BPD traits in a population- based sample of young Norwegian twins (37) and for BPD assessed with sum scores in the NBA sample (35, 36). However, an even higher heritability (67%) was found for a latent BPD factor using a measurement model with two different measures at two different time points, thus reducing the variance owing to measurement error (Torgersen et al. in review).

Although the DAPP has changed somewhat over time [e.g. number of primary traits extended from 18 (12) to 30 (13)], and we used a short version of the scales, the heritability estimates for the selected BPD traits in our study were quite similar to those reported by Jang et al. (38) for corresponding traits: AI: 0.42 vs. 0.41, CD: 0.51 vs. 0.49, IP: 0.53 vs. 0.53, IA: 0.51 vs. 0.48.

Borderline personality disorder traits and normative personality disorder dimensions

Our results of the overall association between our BPD common factor and each of the dimensions of the FFM are broadly consistent with the prior literature which has generally found high positive correlations between various measures of BPD and N, substantial negative correlations with A and C, and weaker correlations with E and O. For example, two recent meta-analyses (16, 17) report correlations between BPD (measured by different instruments) and N of 0.49 and 0.54, respectively, between A and BPD, −0.23 and −0.24, and between BPD and C, −0.23 and −0.29 respectively. The phenotypic correlations were −0.09/−0.12 with E and 0.02/0.10 with O.

The proportion of variance in the four BPD traits predicted by the FFM dimensions ranged from 44.8% (AI) to 20.3% (IA). For every trait, N was the strongest single predictor with standardized betas of +0.76 (AI), +0.53 (IA), +0.45 (IP), and +0.39 (CD). These results are in accordance with results reported by Schroeder et al. (39) (e.g. standardized beta N – AI = 0.74). The associations with the other FFM traits were weaker. C showed negative associations with all BPD traits (ranging from −0.11 to −0.30), while A was negatively associated with AI, CD, and IP (−0.02 to −0.13) but positively associated with IA (+0.19). These findings are consistent with the hypothesis that N is a core trait underlying BPD (40).

In this study, the BPD common factor showed a moderate positive genetic correlation with N (+0.44), substantial negative genetic correlations with A (−0.57) and C (−0.66), and modest negative correlations with E (−0.23) and O (−0.16). These results mirror the findings from the phenotypic studies and again suggest that N is closely related to the core trait underlying BPD (40). The magnitude of the genetic correlation between N and the BPD common factor in our study (r_g = +0.44) is much lower than that reported by Distel et al. using data from the NBA sample (36) (r_g=+0.95); these results are not comparable because of differences in the structural models used in the two reports. In our study, the association between N and BPD was captured by a total of five genetic correlations (one to the common factor and four to the individual scales) while the paper by Distel et al. captures this relationship be a single correlation (36). For N in particular, where all genetic correlations between the personality dimension and the individual BPD scales are positive, the total genetic relationship is considerably greater than that captured only by the single correlation to the common factor. The strong negative genetic correlation between our BPD latent factor and A and C is in accordance with a previous finding from our group of a close genetic relationship between BPD and antisocial PD (41).

The environmental correlations between the FFM dimensions and the latent BPD factor were also positive for N and negative for A, C, E, and O. The environmental correlations were lower than the genetic correlations for N, A, and C and higher for E and O. The direction of the environmental correlations in Distel et al.'s study (36) was broadly similar to ours.

The correlations between the genetic factors for the FFM dimensions and the specific genetic factors for the traits not shared with the common BPD factor showed that the strongest genetic correlations were found with AI [E (+0.49), N (+0.44), and O (+0.43)]. The genetic correlation between N and IP was +0.72 and between O and CD +0.45. The rest of the genetic correlations were below 0.35. The overall pattern indicated that N showed the strongest genetic relationship with the specific BPD components, especially AI and IP. The environmental correlations were generally lower, and with a few exceptions, followed the same pattern.

To our knowledge, only one small prior general population twin study, Jang & Livesley (42), calculated genetic and environmental correlations between normal and abnormal

personality measured by NEO-FFI and DAPP. The results indicated that the two scales shared a common broadbased genetic architecture, whereas the environmental influences show greater scale specificity. Genetic correlations were particularly strong with N, ranging from 0.78 (IP) to 0.52 (IA). The four DAPP traits also showed substantial genetic correlations with A (range −0.57 to −0.35) and C (range −0.46 to −0.23). Because we calculated genetic correlation between the FFM dimensions and the specific genetic variance in the DAPP traits not shared with the latent BPD factor, these results cannot, however, be directly compared to our findings.

Taken together, our results indicate a high phenotypic correlation between specific normal personality traits and BPD and suggest that common genetic liability accounts for a substantial part of this association. This supports the conceptualization of BPD as a maladaptive variant of normal personality and suggests that a dimensional classification system should be further explored.

Limitations

These results should be interpreted in the context of at least five potential methodological limitations. First, the web sample is surely not entirely representative of the general population from which it was drawn. As has been noted previously in volunteer twin samples (43), both MZ and female twin pairs were substantially in excess in the twins ascertained from our web site. However, this sample was not highly biased with respect to education (with the median years of education of 12). As detailed elsewhere, on virtually all the wide ranging traits examined in our web study, the sample was quite diverse – contrary to the concern that internet samples would be restricted to highly educated, ‘nerdish’ individuals (18).

Second, the four DAPP scales and the items from those scales that were chosen for inclusion were selected based on clinical and theoretical considerations. If additional scales had been chosen, our results might have been different. For example, inclusion of the DAPP-DQ ‘stimulus seeking’ would likely have produced a greater contribution from the big-five dimension of C.

Third, owing to practical needs of this survey which covered a wide diversity of traits, we had to shorten the four chosen DAPP scales. However, the chosen items did produce, with the possible exception of IP, good alpha levels. Furthermore, we were robustly able to recover the four scales when jointly analyzed.

Fourth, the correlations observed between our DAPP and BFI scales might have been attenuated because the BFI focused on the adaptive aspects of personality. Haigler and Widiger have shown that when personality items were altered to represent more maladaptive aspects of E, O, and A, correlations with PD scales increased dramatically (44).

Finally, power with our modest sized twin sample is limited (45). Our small number of male pairs provided us particularly limited power to detect quantitative sex effects.

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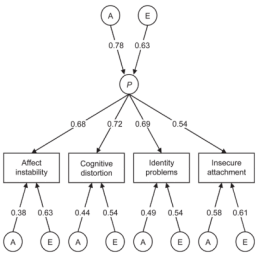

Fig. 1.

Parameter estimates of the best-fit common pathway model for the items selected from four scales of the DAPP: Affective Instability, Cognitive Distortion, Identity Problems, and Insecure Attachment. ‘A’ stands for additive genetic effects and ‘E’ for individual-specific environment.

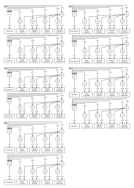

Fig. 2.

(a) Parameter estimates of the best-fit twin model (model II in Table 3) for the inter-relationship between the personality trait of openness and the four DAPP scales of Affective Instability, Cognitive Distortion, Identity Problems (IP), and Insecure Attachment. ‘A’ stands for additive genetic effects and ‘E’ for individual-specific environment. (b) Parameter estimates of the twin model (model II in Table 3) for the inter-relationship between the personality trait of conscientiousness and the four DAPP scales of Affective Instability, Cognitive Distortion, IP, and IA. ‘A’ stands for additive genetic effects and ‘E’ for individual-specific environment. (c) Parameter estimates of the best-fit twin model (model II in Table 3) for the inter-relationship between the personality trait of extroversion and the four DAPP scales of Affective Instability, Cognitive Distortion, IP, and Insecure Attachment. ‘A’ stands for additive genetic effects and ‘E’ for individual-specific environment. (d) Parameter estimates of the best-fit twin model (model II in Table 3) for the inter-relationship between the personality trait of agreeableness and the four DAPP scales of Affective Instability, Cognitive Distortion, IP, and Insecure Attachment. ‘A’ stands for additive genetic effects and ‘E’ for individual-specific environment. (e) Parameter estimates of the best-fit twin model (model II in Table 3) for the inter-relationship between the personality trait of neuroticism and the four DAPP scales of Affective Instability, Cognitive Distortion, IP, and Insecure Attachment. ‘A’ stands for additive genetic effects and ‘E’ for individual-specific environment.

Table 1

Factor loadings for items from the four subscales of the dimensional assessment of personality problems

Scale name	Item entry	Factor1	Factor2	Factor3	Factor4
CD	The world sometimes seems unreal to me	83*	23	9	12
CD	I sometimes wonder whether the things that go on around me are real or imaginary	81*	20	12	9
CD	I have sometimes felt that things were not really happening to me	81*	19	16	12
CD	When I am very stressed I seem to lose touch with reality for a short time	71*	24	17	22
CD	People sometimes tell me that I am not making sense	69*	14	16	14
CD	I often feel as if I am not really there	56*	53*	16	17
IP	I never really enjoy myself	12	77*	6	10
IP	I do not enjoy things like other people do	19	75*	2	11
IP	The feeling of being bored is always with me	23	66*	15	7
IP	I often have moments when I feel very empty	32	63*	15	28
IP	I worry that I will lose a sense of who I am	30	55*	15	19
IP	I have a clear sense of my own identity	−6	−56*	−5	−3
IA	When things go wrong, I need to be with the person who I am especially attached to	4	−2	77*	22
IA	I am only really comfortable when I have someone to keep me company	15	11	76*	4
IA	Worry tends to make me cling to those I am closest to	12	11	74*	23
IA	I become anxious when I have to be alone for any length of time	19	16	72*	8
IA	I hate being separated from someone I love even for a few days	8	2	72*	18
IA	I worry about being abandoned by the person I love	21	26	58*	23
AI	At times my feelings take over and just pour out	14	4	23	77*
AI	I show my feelings very intensely	1	−15	17	75*
AI	I am almost always emotional	14	22	20	74*
AI	I often feel as if I am on an emotional roller-coaster	21	38	16	66*
AI	Little things change my emotions	16	30	16	62*
AI	My moods are very unpredictable	30	32	13	60*

CD, cognitive distortion; IP, identity problems; IA, insecure attachment; AI, affect instability.

Printed values are multiplied by 100 and rounded to the nearest integer. Values >0.38657 are flagged by an *.

Table 2

The mean and standard deviations and polychoric correlations of the four borderline personality disorder traits and five dimensions of the big five inventory in monozygotic and dizygotic (DZ) twin pairs

Zygosity	Variable	Mean*	Standard deviation*	Polychoric correlation†
Monosygotic	Affective instability	2.70	0.91	0.42
	Cognitive distortion	2.02	0.95	0.52
	Identity problems	2.13	0.85	0.52
	Insecure attachment	2.87	1.00	0.51
	Openness	3.61	0.68	0.64
	Conscientiousness	3.58	0.67	0.43
	Extraversion	3.25	0.88	0.48
	Agreeableness	3.82	0.62	0.45
	Neuroticism	2.96	0.76	0.46
DZ	Affective instability	2.80	1.00	0.18
	Cognitive distortion	2.11	1.05	0.29
	Identity problems	2.15	0.87	0.44
	Insecure attachment	2.71	1.01	0.11
	Openness	3.67	0.65	0.16
	Conscientiousness	3.61	0.75	0.29
	Extraversion	3.34	0.93	0.01
	Agreeableness	3.77	0.76	0.07
	Neuroticism	2.91	0.87	0.20

*Mean and standard deviation computed using continuous variables.

†Polychoric correlation computed using ordinal variables.

Table 3

Prediction of borderline personality disorder traits from the big five normal personality disorder dimensions

	Big five dimensions [β (SE)]				
Borderline trait	**Openness**	**Conscientiousness**	**Extroversion**	**Agreeableness**	**Neuroticism**
Affective instability	+0.15 (0.006)**	−0.12 (0.006)**	+0.18 (0.005)**	−0.02 (0.006)*	+0.76 (0.005)**
Cognitive distortion	+0.20 (0.007)**	−0.30 (0.007)**	−0.09 (0.006)**	−0.12 (0.007)**	+0.39 (0.006)**
Identity problems	+0.01 (0.005)	−0.19 (0.005)**	−0.25 (0.004)**	−0.13 (0.005)**	+0.45 (0.005)**
Insecure attachment	–0.09 (0.007)**	−0.11 (0.007)**	+0.16 (0.006)**	+0.19 (0.007)**	+0.53 (0.006)**
Common factor	+0.01	−0.38**	−0.19**	−0.29**	+0.63**

For the common factor, we report a Pearson correlation.

SE, standard error.

* $\rho < 0.05$.

** $\rho < 0.001$.

Table 4

Akaike's information criterion (AIC) values for models examining the genetic and environmental correlations between personality dimensions and borderline personality disorder traits

Twin model		AIC values				
	Description	**Openness**	**Conscientiousness**	**Extroversion**	**Agreeableness**	**Neuroticism**
I	Full – sex effects	–	–	–	–	–
II	Full – no sex effects	−10.8*	−8.5	−5.4*	−11.6*	−18.3*
III	II + no specific A paths	+13.6	−9.2	+24.0	−9.3	+7.7
IV	II + no specific E paths	−8.8	−14.0	+25.8	−8.4	−12.7
V	II + no specific A or E paths	+38.8	−16.0*	+81.2	+7.1	+43.8

*Best-fit model.

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Table 5

Genetic and environmental correlations between personality dimensions and the common factor underlying the borderline personality disorder traits and the specific traits themselves derived from the best-fit twin model*

	A/E	Common factor	Affective instability	Cognitive distortion	Identity problems	Insecure attachment
Openness	A	−0.16	+0.43	+0.45	−0.12	−0.06
	E	−0.31	+0.31	+0.25	+0.14	+0.04
Conscientiousness	A	−0.66	+0.28	−0.06	−0.07	−0.01
	E	−0.33	+0.08	+0.09	+0.17	+0.16
Extroversion	A	−0.23	+0.49	+0.11	−0.32	+0.17
	E	−0.42	+0.29	+0.25	−0.20	+0.14
Agreeableness	A	−0.57	+0.01	+0.31	−0.14	+0.15
	E	−0.36	+0.08	+0.14	+0.09	+0.31
Neuroticism	A	+0.44	+0.44	+0.12	+0.72	+0.26
	E	+0.22	+9.51	+0.16	+0.30	+0.24

*Except for conscientiousness where, for the sake of consistency, we present results from model II instead of model V.

A – refers to additive genetic effects.

E – refers to individual-specific environmental effects.