A Further Validation of the Minnesota Borderline Personality Disorder Scale

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Previous research indicates that borderline personality disorder (BPD) is well conceptualized as a dimensional construct that can be represented using normal personality traits. A previous study successfully developed and validated a BPD measure embedded within a normal trait measure, the Minnesota Borderline Personality Disorder Scale (MBPD). The current study performed a further validation of the MBPD by examining its convergent validity, external correlates, and heritability in a sample of 429 female twins. The MBPD correlated strongly with the Structured Clinical Interview for DSM-IV Axis II Personality Disorders (SCID-II) screener for BPD and moderately with external correlates. Moreover, the MBPD and SCID-II screener exhibited very similar patterns of external correlations. Additionally, results indicated that the genetic and environmental influences on MBPD overlap with the genetic and environmental influences on the SCID-II screener, which suggests that these scales are measuring the same construct. These data provide further evidence for the construct validity of the MBPD.

Keywords: borderline personality disorder, normal personality, nomological network, behavioral genetics

Borderline personality disorder (BPD) is characterized by impulsivity, affective instability, inappropriate anger, self-injury, abandonment fears, unstable relationships, and identity disturbance (American Psychiatric Association, 2000). BPD is highly comorbid with both Axis I and Axis II psychopathology, including depression (Frankenburg & Zanarini, 2004), anxiety disorders (Zanarini et al., 1998), eating disorders (Striegel-Moore, Garvin, Dohm, & Rosenheck, 1999), and substance use disorders (Paris, 1997). Traditionally, BPD was thought of as categorical (yes–no) construct. However, this view has been shifting based on evidence that borderline features fall along a continuum (Edens, Marcus, & Ruiz, 2008; Trull, Widiger, Lynam, & Costa, 2003). For instance, taxometric research suggests that BPD is dimensional (Edens et al., 2008), and continuous measures are consistently and substantially more reliable and valid for psychopathology, in general (Markon, Chmielewski, & Miller, 2011), and personality pathology, in particular (Morey et al., 2007). Therefore, there is a clear need to construct and identify measures that assess BPD in a dimensional manner.

Fortunately, there is a long tradition of dimensional assessment of traits from basic personality theory (e.g., John, Robins, & Pervin, 2008) from which psychopathologists can draw. Indeed, robust associations have been identified between BPD features and normal traits, such as those of the five-factor model (FFM; Costa & McCrae, 1992). For instance, Trull, Widiger, and Burr (2001) found that the trait of neuroticism from FFM of normal personality accounts for a significant amount of variance in BPD features in
both a clinical sample and undergraduate sample, even after controlling for all other personality disorders. Likewise, Morey and Zanarini (2000) found that the neuroticism factor could distinguish BPD and non-BPD individuals, and that the entire FFM model accounted for a significant proportion of variance in BPD diagnosis for both self-report and interview measures. These studies provide considerable evidence that BPD features can be identified using indicators of normal personality traits. Accordingly, researchers have developed a number of methods for assessing BPD using existing personality trait systems (Costa & Widiger, 2002; Mullins-Sweatt et al., 2012). However, until recently, there was no BPD indicator from the extensively validated Multidimensional Personality Questionnaire (MPQ; Patrick, Curtin, & Tellegen, 2002; Tellegen, 1982). Bornovalova, Hicks, Patrick, Iacono, and McGue (2011) developed the Minnesota Borderline Personality Disorder Scale (MBPD) using items from the MPQ pool with cross-validated correlations to other indicators of BPD. In the original validation study, the MBPD was highly and significantly correlated with both diagnostic and self-report measures of BPD, as well as established external correlates such as substance use and depression. The measure also discriminated BPD from antisocial features and provided incremental validity over negative emotionality for predicting BPD diagnostic symptoms, BPD diagnosis, and externalizing behaviors.

The MBPD has considerable potential for research on BPD. For instance, the MBPD can be used to provide an assessment of BPD features in samples in which the MPQ was administered but other BPD measures were not, and allowing for additional research on the MBPD provides insights into the ability of trait instruments to assess PD constructs. However, further validation work in new samples and with novel validation criteria is needed to support the utility of the measure. The purpose of this study was to further evaluate the validity and generalizability of the MBPD in a sample of twin women during their transition from adolescence to adulthood. This sampling approach is valuable in light of the fact that peaks in BPD features occur normatively during transition to adulthood (Mattanah, Becker, Levy, Edell, & McGlashan, 1995). Twin sampling allowed us to conduct exploratory analyses testing whether the MBPD shares etiological influences with other indicators of BPD in the current sample. Previous research has shown that the heritability of BPD in late adolescence is approximately .48 to .50 (Bornovalova, Hicks, Iacono, & McGue, 2013). Other studies have shown similar results regarding heritability for BPD features in late adolescence into early adulthood (Distel et al., 2008, 2011).

Present Study

In the current study, we aimed to conduct a further validation of the MBPD in a large community sample of young female twins. We had three general hypotheses. First, we expected that the MBPD would be strongly related to another validated measure of BPD. Second, we predicted that the MBPD would correlate with theoretically related constructs, although to a lesser degree than with other BPD measures. We selected external variables based on known correlates of BPD, including negative affect (Trull, Sher, Minks-Brown, Durbin, & Burr, 2000), impulsive behavior (Paris, 1997), interpersonal problems (Fonagy & Bateman, 2006), antisocial behaviors (Paris, 1997), eating disorders (Striegel-Moore et al., 1995), major depressive disorder (Fonagy & Bateman, 2006), and alcohol and drug use (Paris, 1997). Third, exploratory biometric models were expected to show that the MBPD shares etiological influences with other measures of BPD in the current sample. This allowed us to test if the same genetic and environmental influences give rise to responses on BPD measures despite a lack of item overlap.

Method

Participants

This study sample was drawn from a larger ongoing project, the Twin Study of Hormones and Behavior across the Menstrual Cycle from the Michigan State University Twin Registry (MSUTR; N = 18,000 twins; see Klump & Burt, 2006, for the description of the study and recruitment procedures). The current sample included 493 young women (238 twin pairs and 17 unpaired twins). Of the twin pairs, 141 were monozygotic (MZ) pairs and 114 were dizygotic (DZ). Participants ranged in age from 16 to 25 years (M = 18.11, SD = 1.93). MSUTR participants are demographically representative of the surrounding region (Burt & Klump, 2012). In the current sample, the ethnicity breakdown was 77% Caucasian, 16% African American, 5% mixed, 1% Asian, 1% American Indian/Alaskan Native—an ethnic distribution representative of the general Michigan area (Burt & Klump, 2012).

Measures

Measures are organized into three categories: (a) the MBPD scale, (b) a convergent validity measure, and (c) external correlates. Diagnostic reliability was calculated from the kappa coefficient and internal consistency was evaluated using Cronbach’s alpha.

MBPD (Bornovalova et al., 2011). The MBPD is a 19-item scale developed using items from the MPQ (Patrick et al., 2002), a well-validated omnibus measure of normal personality. Candidate items were identified in two samples—inner-city drug users (n = 146) and undergraduates (n = 288)—by examining correlations between all MPQ items and diagnostic and self-report measures of BPD. Candidate items that were significantly correlated with BPD measures in both samples were retained for further analyses in a third sample of young adults from the community. Validation analyses were conducted with a special emphasis placed on potential BPD items providing incremental prediction over general negative affect as measured by MPQ Negative Emotionality. The final 19 items were drawn from the MPQ Stress Reaction, Alienation, Control, Aggression, Well-Being, and Absorption scales. MBPD scores were correlated strongly with scores on another personality inventory aimed at assessing BPD, the Personality Assessment Inventory Borderline Features scale (PAIBOR; Morey, 1991) in the undergraduate sample (r = .80), and with an interview-based diagnosis of BPD in the drug-user sample (r = .62). In the current sample, α = .76, and mean interitem correlation was .15.

Convergent validity measure: Structured Clinical Interview for DSM–IV Axis II Personality Disorders (SCID-II) Questionnaire (First, Gibbon, Spitzer, Williams, & Benjamin, 1997). The SCID-II personality questionnaire screener (SCID-II screener) is a self-reported screening tool for personality disorders. The SCID-II screener in this study included 15 (yes–no) items to assess for the nine BPD criteria outlined in the fourth
edition of the *Diagnostic and Statistical Manual of Mental Disorders* (text rev.; *DSM-IV-TR*; American Psychiatric Association, 2000). Wording of items corresponded with the SCID-II diagnostic interview for BPD. Additional items were used as probes for each criterion (e.g., three items assessed identity disturbance, two items assessed for self-harm, two items assessed for impulsivity, three items assessed for inappropriate anger). Symptom counts were calculated by summing the 15 BPD items. In the current sample, \( \alpha = .76 \) and mean interitem correlation was .18.

**External validity measures.**

*Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988).* PANAS scores were collected from participants for 45 consecutive days. Participants were asked to rate the extent to which they experienced daily positive affect (PA; e.g., excited) and negative affect (NA; e.g., scared) on a 5-point scale (1 = *very slightly* and 5 = *very much*) for 20 items. The PANAS-PA was used as a measure of discriminant validity, as positive affect has been found to show a modest negative association with BPD symptoms (e.g., Samuel & Widiger, 2008). The PANAS-NA was used as an external correlate as negative affect is associated with BPD symptoms. Internal consistency was calculated from the item scores at baseline; for PANAS-NA, \( \alpha = .83 \), and for PANAS-PA, \( \alpha = .88 \).

**UPPS-P Impulsive Behavior Scale (UPPS-P; Lynam, Smith, Whiteside, & Cyders, 2006).** The UPPS-P is a 59-item inventory that measures five dimensions of impulsive behavior. The five subscales include Negative Urgency (tendency to engage in rash action in response to negative affect), (lack of) Premeditation (i.e., tendency not to plan or think through consequences of behavior before acting), (lack of) Perseverance (i.e., inability to sustain attention or motivation on a task), Sensation-Seeking (i.e., preference for excitement, stimulation, and danger), and Positive Urgency (i.e., tendency to act rashly in response to strong positive affect). The five subscales include Negative Urgency (tendency to engage in rash action in response to negative affect), (lack of) Premeditation (i.e., tendency not to plan or think through consequences of behavior before acting), (lack of) Perseverance (i.e., inability to sustain attention or motivation on a task), Sensation-Seeking (i.e., preference for excitement, stimulation, and danger), and Positive Urgency (i.e., tendency to act rashly in response to strong positive affect). The subscales have 11, 13, 12, 10, and 14 items, respectively, each of which are calculated by taking the mean of the items. The items have a 4-point Likert scale (1 = *strongly agree* to 4 = *strongly disagree*). Questions assess global lifetime traits. In the current sample, for Negative Urgency, \( \alpha = .85 \); for (lack of) Premeditation, \( \alpha = .86 \); for (lack of) Perseverance, \( \alpha = .83 \); for Sensation-Seeking, \( \alpha = .83 \); and for Positive Urgency, \( \alpha = .91 \).

**Inventory of Interpersonal Problems Circumplex Scales (IIP-C; Alden, Wiggins, & Pincus, 1990).** The IIP-C is a 64-item self-report lifetime assessment of interpersonal difficulties. The inventory assesses interpersonal problems and includes 8 eight-item subscales: Domineering, Vindictive, Cold, Socially Avoidant, Nonassertive, Exploitable, Overly Nurturant, and Intrusive, although items from this scale can also be summed to provide an overall index of interpersonal distress. As our interest in the current study involved the degree to which the MBPD relates to interpersonal problems in general, we focused on the total score. In the current sample, the total score \( \alpha = .94 \).

**Subtypes of Antisocial Behavior Questionnaire (STAB; Burt & Donnellan, 2009).** The STAB is a self-report measure containing 32 items assessing three factors: Physical Aggression (AGG), Rule-Breaking (RB), and Social Aggression (SA). Items were rated on a 5-point scale to assess frequencies of antisocial behaviors (1 = *never* and 5 = *nearly all the time*) across the lifetime. Previous work has demonstrated the ability of the STAB to distinguish college students, community adults, and adjudicated adults (Burt & Donnellan, 2009). In the current sample, \( \alpha = .86 \) for AGG, .85 for RB, and .85 for SA.

**Minnesota Eating Behavior Survey (MEBS; Klump, McGue, & Iacono, 2000; Miller & Pumariaga, 2001; von Ranson, Klump, Iacono, & McGue, 2005).** The MEBS\(^1\) is a 30-item lifetime self-report measure that assesses disordered eating attitudes and behaviors, including body dissatisfaction, weight preoccupation, binge eating, and compensatory behavior. The current study examined the total score of the MEBS (\( \alpha = .88 \)), which indexes higher levels of disordered eating attitudes and behaviors.

**Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I; First, Spitzer, Gibbon, & Williams, 1997).** The SCID-I was used to assess major depressive disorder (MDD), alcohol dependence (AD), and substance dependence (SD). Lifetime symptom counts were calculated for each disorder. Natural log transformations were used to reduce the skew and kurtosis of the symptom counts into an acceptable range, respectively (Chou & Bentler, 1995). For example, MDD, AD, SD exhibited both unacceptable skew (4.68, 6.65, 13.02, respectively) and kurtosis (13.54, 49.20, 194.92, respectively), and log transformation improved skew (2.95, 5.01, 5.01, respectively) and kurtosis (8.35, 26.05, 26.05). The assessment of SD covered amphetamines, cannabis, cocaine, hallucinogens, opioids, and other drugs. The drug class for which a participant reported the most symptoms for was used as their number of SD symptoms. Interrater reliability for diagnostic decisions was: \( \kappa \) for mood disorders = 1.00; \( \kappa \) for alcohol and substance disorders = 1.0.

### Results

**Convergent Validity and External Correlates**

To establish the convergent validity of the MBPD, we examined its relationship with an established BPD measure, the SCID-II screener for BPD via Pearson correlations.\(^2\) Next, we tested the extent to which the MBPD was correlated with other theoretically related external variables. The same correlations were evaluated between the SCID-II screener and the external variables. Finally, we tested if there were significant differences in the magnitude of the relationship of MBPD with external correlates and SCID-II screener with external correlates using a transformation (Williams’s T2 statistic) that accounts for the high correlation (.73) between MBPD and SCID-II screener (Steiger, 1980).

Descriptive statistics for all measures are provided in Table 1. With regard to convergent validity, the MBPD demonstrated a

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\(^{1}\) The Minnesota Eating Behavior Survey (MEBS; previously known as the Minnesota Eating Disorder Inventory [M-EDI]) was adapted and reproduced by special permission of Psychological Assessment Resources, Inc., 16204 North Florida Avenue, Lutz, FL 33549, from the Eating Disorder Inventory (collectively, EDI and EDI-2) by Garner, Olmstead, and Polivy (1983). Copyright, 1983 by Psychological Assessment Resources, Inc. Further reproduction of the MEBS is prohibited without prior permission from Psychological Assessment Resources, Inc.

\(^{2}\) Twins are correlated at higher-than-chance rates, which could possibly inflate the strength of the correlation between our predictor and correlates. We selected two random subsets of twins from the sample (randomly selected one twin from each twin pair) and checked if the correlations differed significantly between the subsets/random halves. In over 95% of the cases, there were no significant differences between random halves.
strong, positive correlation with the SCID-II screener (r = .73, p < .001). The MBPD scores also evidenced moderate correlations with the PANAS-NA; the UPPS subscales of Positive Urgency, Perseverance, and Premeditation; STAB subscales of RB, and SA; the IIP-C; and MDD (rs ranged from .21 to .47). The MBPD evidenced strong correlations with STAB aggression and UPPS Negative Urgency (rs between .60 and .61), and small correlations with Alcohol and Substance Dependence (rs = .15). In support of its discriminant validity, the MBPD was uncorrelated with the UPPS subscale of Sensation-Seeking, negatively correlated with PANAS-PA, and exhibited considerably lower correlations with the STAB subscales of Aggression and Rule-Breaking (indicators of adult antisocial behavior; see Table 1). Moreover, only two external correlations significantly differed between the MBPD and SCID-II—both the IIP-C and STAB Rule-Breaking scales were more strongly related to the SCID-II than MBPD. This suggests that both the SCID-II and the MBPD had very similar nomological networks.

Biometric Modeling

First, using a double-entry method, we estimated intraclass and cross-twin, cross-trait correlations to provide initial estimates of genetic and environmental influences on each phenotype and their association. Genetic influences are inferred if the MZ correlation is greater than the DZ correlation for a given measure. Shared environmental influences are inferred if the DZ correlation is greater than .5 of the MZ correlation. Nonshared environmental influences are inferred when the MZ correlation is less than 1.0. As shown in Table 2, the pattern of twin correlations suggest genetic, shared environmental, and nonshared environmental influences on both MBPD and the SCID-II screener. The cross-twin, cross-trait correlations also suggest an overlap between the two measures that is due to common genetic, shared environmental, and nonshared environmental influences as indicated by (a) the larger cross-twin, cross-trait correlations for MZ relative to DZ pairs, and (b) the nonsignificant difference between the two sets of correlations.

Next, in order to test whether common risk factors give rise to scores on the MBPD and SCID-II (indicating common influences on the phenotypes), we examined the common genetic and environmental influences contributing to the MBPD and SCID-II screener through bivariate Cholesky models using Mx (Neale, Boker, Xie, & Maes, 1999). These models decompose the covariance between pairs of variables instead of just considering the influences on each variable alone (see Figure 1 for visual representation). More specifically, bivariate models allow the genetic, shared, and nonshared environmental influences on the MBPD to correlate with the same influences on SCID-II screener. The magnitude of the genetic, shared, and nonshared environmental correlations between the MBPD with the SCID-II screener signifies the extent to which such influences are common to both measures (Neale & Cardon, 1992).

First, we fit the “full” model that allowed for genetic, shared, and nonshared influences on MBPD to correlate with the influ-

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**Table 1**

**MBPD/SCID-II Screener With External Correlates**

|                  | MBPD (SD) | SCID-II Correlation | Correlations significantly different (z value)
<table>
<thead>
<tr>
<th></th>
<th></th>
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<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>MBPD</strong></td>
<td>24.29 (3.57)</td>
<td>.73**</td>
<td>—</td>
</tr>
<tr>
<td><strong>SCID-II</strong></td>
<td>2.70 (2.77)</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td><strong>Negative affect</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PANAS-NA At baseline</td>
<td>15.03 (3.72)</td>
<td>.37***</td>
<td>.32***</td>
</tr>
<tr>
<td>PANAS-PA At baseline</td>
<td>22.98 (6.10)</td>
<td>—.22***</td>
<td>—.19***</td>
</tr>
<tr>
<td>Impulsivity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>UPPS Premeditation</td>
<td>1.92 (.52)</td>
<td>.29***</td>
<td>.24***</td>
</tr>
<tr>
<td>UPPS Perseverance</td>
<td>1.83 (.48)</td>
<td>.38***</td>
<td>.34***</td>
</tr>
<tr>
<td>UPPS Sensation-Seeking</td>
<td>2.70 (.58)</td>
<td>.05</td>
<td>—.02</td>
</tr>
<tr>
<td>UPPS Negative Urgency</td>
<td>2.05 (.56)</td>
<td>.61**</td>
<td>.60***</td>
</tr>
<tr>
<td>UPPS Positive Urgency</td>
<td>1.69 (.54)</td>
<td>.47**</td>
<td>.46**</td>
</tr>
<tr>
<td>Interpersonal problems</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IIP-C</td>
<td>.79 (.43)</td>
<td>.26***</td>
<td>.38***</td>
</tr>
<tr>
<td>Antisocial behavior</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>STAB Aggression</td>
<td>18.03 (5.62)</td>
<td>.60***</td>
<td>.55***</td>
</tr>
<tr>
<td>STAB Rule Breaking</td>
<td>12.21 (2.95)</td>
<td>.40***</td>
<td>.48***</td>
</tr>
<tr>
<td>STAB Social Aggression</td>
<td>21.45 (5.23)</td>
<td>.40***</td>
<td>.36***</td>
</tr>
<tr>
<td>Eating disorders</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MEBS</td>
<td>5.32 (5.13)</td>
<td>.37***</td>
<td>.40**</td>
</tr>
<tr>
<td>Major depression*</td>
<td>.37 (1.27)</td>
<td>.31***</td>
<td>.34***</td>
</tr>
<tr>
<td>Alcohol dependence*</td>
<td>.14 (.73)</td>
<td>.15**</td>
<td>.15**</td>
</tr>
<tr>
<td>Substance dependence*</td>
<td>.15 (1.28)</td>
<td>.15*</td>
<td>.15*</td>
</tr>
</tbody>
</table>

**Note.** MBPD = Minnesota Borderline Personality Disorder Scale; SCID-II = Structured Interview for DSM–IV Axis II Personality Disorders Screener; PANAS-NA = Positive and Negative Affect Schedule - Negative Affect; PANAS-PA = Positive and Negative Affect Schedule - Positive Affect; UPPS Premeditation = (lack of) premeditation; UPPS Perseverance = (lack of) perseverance; UPPS Sensation Seeking = UPPS Sensation-Seeking; UPPS Negative Urgency = UPPS Negative Urgency; UPPS Positive Urgency = UPPS Positive Urgency; IIP-C = Inventory of Interpersonal Problems Circumplex Scales; STAB = Subtypes of Antisocial Behavior Survey; MEBS = Minnesota Eating Behaviors Survey.

* Means from raw scores of symptoms counts are presented; all analyses used log-transformed, z-scored symptom counts.

* p < .05. ** p < .01. *** p < .001.
ences on SCID-II screener. As indicated in Table 3, the univariate estimates in each model support the results obtained from the cross-twin, cross-trait matrix. In particular, the estimates indicate the presence of all three biometric components (A, C, and E), although the confidence intervals are quite broad and include zero in the full model for A and C. Thus, we fit a series of nested models that progressively dropped the genetic (\( r_A \)), shared (\( r_C \)), and nonshared (\( r_E \)) correlations. Model fit was evaluated using the change in \( -2 \log \text{likelihood} (\Delta -2LL) \); which follows a chi-square distribution) and the Bayesian information criterion (BIC). The BIC is a function of a model’s \( \chi^2 \) value and degrees of freedom (df), and penalizes the model fit for the retention of unnecessary parameters. Lower values (with a difference in BIC > 2) are indicative of better fit (Raftery, 1995). As shown in Table 3, dropping \( r_C \) did not result in a significant different in the \( -2LL(\Delta -2LL = .85, ns) \) but a significant improvement in model fit as indexed by the BIC (\( \Delta \text{BIC} = -2.35 \)); this model was retained as the best-fitting model. However, this model fit only slightly better than the model that dropped \( r_A \), most likely due to the relatively small sample size and power problems with classical

### Table 2

**Intraclass and Cross-Twin, Cross-Trait Correlations for MBPD and SCID-II Screener**

<table>
<thead>
<tr>
<th>Twin correlations</th>
<th>MZ (n = 141 pairs)</th>
<th>DZ (n = 114 pairs)</th>
<th>Z test of equality</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intraclass correlations</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MBPD</td>
<td>.42 (.31, .51)**</td>
<td>.27 (.14, .39)**</td>
<td>1.87</td>
<td>.062</td>
</tr>
<tr>
<td>SCID-II</td>
<td>.35 (.24, .46)**</td>
<td>.27 (.14, .39)**</td>
<td>.97</td>
<td>.332</td>
</tr>
<tr>
<td>Cross-twin, cross-trait correlations</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tw A MBPD, Tw B SCID-II</td>
<td>.35 (.24, .46)**</td>
<td>.24 (.11, .37)**</td>
<td>1.32</td>
<td>.187</td>
</tr>
</tbody>
</table>

* The twin correlation is significantly greater than 0 at \( p < .01 \). ** The twin correlation is significantly greater than 0 at \( p < .001 \).

**Figure 1.** General bivariate model showing genetic (\( r_A \)) and environmental (\( r_E \)) correlations between the MBPD and the SCID-II screener. MBPD = Minnesota Borderline Personality Disorder Scale; SCID-II = Structured Interview for DSM-IV Axis II Personality Disorders Screener; \( A \) = additive genetic effects; \( E \) = nonshared environmental effects.
Parameter estimates from the best-fitting model indicated that there was a large and significant $r_A$ of 1.00 (95% CI = .82, 1.00) between the MBPD and SCID-II screener, suggesting complete overlap in genetic factors for the two phenotypes. The nonshared environmental correlation was .60 (95% CI = .50, .69). This indicates that about 36% of the environmental factors for the MBPD overlap with the environmental factors for the SCID-II screener, whereas the remaining 64% is unique to the MBPD (and vice versa). Interestingly, the univariate estimates changed for the bivariate model that constrained the $r_C$ parameter (see Table 3).

For example, in the best-fitting model that dropped $r_C$, the shared environmental estimates decreased for both measures. Likewise, in the model that dropped $r_A$, the genetic univariate estimates on each measure decreased. These changes are an indication of the overlap between measures, as the overall magnitude of $r_A$ and $r_C$ influences on each measure decreases when the shared genetic and shared environmental effects are constrained to be zero.

### Discussion

In the current study, we aimed to provide further validation of the MBPD in a sample of 493 young women by evaluating evidence for convergent and external validity, as well as evidence that the MBPD is measuring the same etiological influences underlying BPD as a diagnostic BPD measure. Results indicated that the MBPD indeed exhibited strong, positive correlations with other measures of BPD, as well as moderately correlated with external correlates of BPD, such as negative affect, impulsive and antisocial behaviors, interpersonal problems, and Axis I psychopathology. These findings are consistent with previous research on the nomological network of BPD (Goldman, Dangelo, & Demaso, 1993; Trull et al., 2001; Zanarini, Frankenburg, Hennen, & Silk, 2003). Importantly, it should be noted that the SCID-II screener demonstrated similar correlations as the MBPD with all of the external correlates, suggesting that these measures are capturing the same latent construct of BPD.

Second, exploratory biometric models indicated similar etiological influences on the MBPD as previous studies (Distel et al., 2008, 2011). In particular, our study indicates that the MBPD was primarily influenced by genetic and nonshared environmental influences in young women. These effects are similar to that of previous studies (Bornovalova, Hicks, Iacono, & McGue, 2009; Bornovalova et al., 2013; Distel et al., 2008, 2011), with the latter two reports utilizing a different, nonoverlapping measure (PAI-BOR). Next, bivariate models indicated that the genes and unique environmental factors that influence the MBPD also influence and vice versa. Interestingly, the univariate estimates changed for the bivariate model that constrained the $r_C$ parameter (see Table 3). For example, in the best-fitting model that dropped $r_C$, the shared environmental estimates decreased for both measures. Likewise, in the model that dropped $r_A$, the genetic univariate estimates on each measure decreased. These changes are an indication of the overlap between measures, as the overall magnitude of $r_A$ and $r_C$ influences on each measure decreases when the shared genetic and shared environmental effects are constrained to be zero.

This is one of the few studies to focus on the construct validity of trait measures of BPD features in young women sampled from the community (e.g., Bornovalova et al., 2009; Sharp et
al., 2011). Hence, our study contributes to this literature by establishing a reliable and valid dimensional measure of BPD for use in similar samples, as evidenced by the similarity of the MBPD in terms etiology and correlates with a nonoverlapping measure, the SCID-II screener. Moreover, the current study contributes further to the understanding of BPD in this particular age group, both in terms of etiology and external correlates. The potential of the MBPD for assessing BPD in this sample was particularly supported by its similarity, in terms of etiology and correlates, with a nonoverlapping measure, the SCID-II screener.

There are four limitations that should be noted. First, the MBPD should be further validated in multiple, ethnically diverse samples, including those of both female and male twins across different developmental periods, to examine gender differences in convergent validity, external correlates, and heritability. Second, the sample size was relatively small (particularly for a behavioral genetic study). Indeed, with a larger sample size, we would expect to see all three (genetic, shared environmental, and nonshared environmental) parameters to correlate significantly across MBPD and the SCID-II screener. Third, in the current study, the MBPD was only compared with the SCID-II screener. Future studies would benefit from the use of a multiassess-

ment, multi-informant design, as previous work suggests that different assessment methods and informants provide unique information about BPD (Hopwood et al., 2008; Olffmans & Turkheimer, 2009). Finally, despite item content that covers the major diagnostic domains of BPD, in the current study, the MBPD was treated as a unidimensional construct. However, previous work with the DSM-IV-based interviews of BPD consistently reveals a unidimensional factor structure (Clifton & Pilkonis, 2007; Johansen, Karterud, Pedersen, Gude, & Falkum, 2004) or, alternatively, a three-factor structure with factor correlations greater than .85 (Samislow et al., 2002). As such, this indicates that the use of MBPD as a unidimensional construct is appropriate.

Despite these limitations, the current study provided further evidence for the validity of the MBPD, and, in turn, further confidence for the estimation of BPD traits from the MPQ in large, longitudinal, and epidemiological samples such as the Dunedin Multidisciplinary Health Development Study (Caspi et al., 1997) and the Minnesota Twin and Family Study (Iacono, Carlson, Taylor, Elkins, & McGue, 1999), which contain the MPQ. As these studies include developmentally, genetic, and physiological perspectives, the availability of the MBPD opens up important new avenues for research on BPD using existing data. Finally, the MBPD may be useful to clinicians who give the MPQ to screen for personality issues.

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