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BRIEF REPORT

Predicting Domains and Rates of Change in Borderline Personality Disorder

Mark F. Lenzenweger
State University of New York at Binghamton and Weill Cornell Medical College

John F. Clarkin
Weill Cornell Medical College

Kenneth N. Levy
Pennsylvania State University and Weill Cornell Medical College

Frank E. Yeomans and Otto F. Kernberg
Weill Cornell Medical College

What changes and how quickly these changes occur as a result of therapy in borderline personality disorder (BPD) is an important ongoing question. The features of BPD patients that are most predictive of *rates of change* in such patients remain largely unknown. Using the Cornell Personality Disorders Institute (CPDI) randomized controlled trial data, we sought to determine (a) the number and nature of broad domains underlying a large number of rate of change (slope) measures across many psychological, psychiatric, and psychosocial indexes, and (b) which baseline individual difference psychological features of the BPD patients correlated with these rate of change domains. We examined the latent structure of slope (rate of change) measures gleaned from individual growth curves for each subject, studied in multiwave perspective, on separate measures of anger, aggression, impulsivity, depression, global functioning, and social adjustment. Three broad domains of change rate could be discerned. These domains were reflected in factors that are described as (a) anger/aggression change ("aggressive dyscontrol"), (b) global functioning/social adjustment change ("social adjustment/self-acceptance"), and (c) anxiety/depression/impulsivity change ("conflict tolerance/behavioral control"). Factor scores were computed for each change domain and baseline measures of personality and psychodynamic features, selected a priori, were correlated with these factor scores. Multiple regression analyses revealed (a) baseline negative affectivity and aggression predicted the aggressive dyscontrol change domain, (b) baseline identity diffusion predicted the social adjustment/self-acceptance change domain, and (c) baseline social potency predicted the conflict tolerance/behavioral control change domain. These baseline predictors suggest potential research foci for understanding those aspects of BPD that change at comparable rates over time.

Keywords: borderline personality disorder, prediction, rate of change, individual growth curve

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Mark F. Lenzenweger, Department of Psychology, State University of New York at Binghamton and Department of Psychiatry and Personality Disorders Institute, Weill Cornell Medical College, John F. Clarkin, Department of Psychiatry and Personality Disorders Institute, Weill Cornell Medical College, Kenneth N. Levy, Department of Psychology, The Pennsylvania State University and Department of Psychiatry and Personality Disorders Institute, Weill Cornell Medical College, Frank E. Yeomans and Otto F. Kernberg, Department of Psychiatry and Personality Disorders Institute, Weill Cornell Medical College.

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Correspondence concerning this article should be addressed to Dr. Mark F. Lenzenweger, Department of Psychology, Science IV, The State University of New York at Binghamton, Binghamton, NY 13902-6000. E-mail: mlenzen@binghamton.edu

Borderline personality disorder (BPD), which affects about three in every 200 people (Lenzenweger, 2008), once thought immutable and unlikely to change over time, has been shown through recent empirical study to demonstrate considerable plasticity. Evidence for this comes from symptom declines among subjects in both prospective longitudinal study of nonclinical (Lenzenweger, 1999b; Johnson et al., 2000) and diagnosed patient samples (Shea et al., 2002; Zanarini, Frankenburg, Hennen, & Silk, 2003). BPD improves across a wide range of domains (e.g., social functioning, aggression, affect) with a variety of psychotherapeutic interventions (e.g., Clarkin, Levy, Lenzenweger, & Kernberg, 2007; Bateman & Fonagy, 2009). Understanding the predictors of such malleability and treatment responsivity represents an important scientific and clinical goal. Moreover, the precise nature of psychological and psychosocial functioning change among BPD patients and the rate at which these changes occur as in the treatment of BPD represent important ongoing questions.

One way to examine predictors of change in BPD is to focus on end-point/follow-up outcomes. For example, emotional underinvolvment (Hooley & Hoffman, 1999), initial severity of BPD, childhood trauma, and quality current relationships (Skodol et al., 2007), and extraversion (Hopwood & Zanarini, 2010) have all been associated with either BPD symptom level or global psychosocial functioning at follow-up. Such bivariate associations fail to capture the *dynamic process* of change in BPD as they derive from static cross-sectional assessments (i.e., baseline x endpoint variable correlation; see Singer & Willett, 2003). The *rate of change* as outcome variable captures the dynamic nature of change. Lenzenweger and Castro (2005) found baseline agentic positive emotion, anxiety, fear, and nonaffective constraint were significantly (though differently) predictive of the rate of change in BPD features over time. However, rates of change in *normal* personality systems (e.g., agentic extraversion, affiliation, constraint, negative emotion) over time were not found to predict rates of change in Cluster B pathology (which include BPD) features over time (Lenzenweger & Willett, 2007).

Here we examine baseline psychological predictors in relation to rates of change with BPD across a wide range of domains, such as suicidality, aggression, impulsivity, depression, and social adjustment. We adopted a model-guided approach to select baseline predictors for this study. We selected potential predictors of change based on two viable theoretical models relevant to the pathogenesis of borderline pathology, a neurobehavioral model (Depue & Lenzenweger, 2005, 2006) and a psychodynamic/object relations model (Kernberg, 1984; Kernberg & Caligor, 2005). The primary neurobehavioral systems identified by Depue and Lenzenweger (2005) are manifested in the following phenotypic personality traits: (a) agentic extraversion; (b) affiliation (close interpersonal bonds); (c) negative emotion (i.e., anxiety, anger); (d) fear (often termed harm avoidance; for escape from explicit danger); and (e) nonaffective constraint. These traits obviously do *not* directly or simplistically reflect neurobiological activity, rather they are personality traits that are phenotypic indicators of the human behaviors associated with or linked to underlying neurobehavioral processes. An extensive animal and human neuroscience literature supports this conjecture regarding these personality traits and underlying neurobehavioral systems (see Depue & Collins, 1999; Depue & Lenzenweger, 2005; Depue & Morrone-Strupinsky, 2005; Spoont, 1992). Predictions from this neurobehavioral model for BPD have received empirical support (Lenzenweger, Johnson, & Willett, 2004; Silbersweig et al., 2007). We represented the principal theoretical constructs of the psychodynamic/object relations (Clarkin, Lenzenweger, Yeomans, Levy, & Kernberg, 2007; Kernberg, 1984; Kernberg & Caligor, 2005; Stern et al., 2010) model of borderline personality pathology (i.e., identity diffusion, primitive psychological defenses, reality testing [social empathy] impairments, and aggression) in our selection of baseline measures. The neurobehavioral (Depue & Lenzenweger, 2005, 2006) and psychodynamic/object relations (Kernberg, 1984; Kernberg & Caligor, 2005) models are complementary, though differing in level of analysis (Clarkin, Lenzenweger, et al., 2007). There are other viable dimensional models of PD relevant to BPD, however, the focus of our group is specifically on neurobehavioral systems and psychodynamic processes. Clearly, no single study or single research group can test all possible theoretical models. We have selected the neurobehavioral model as well as the psychodynamic/object re-

lations model as the theoretical foundation for this work as we have been principally involved in their development for many years and we have conducted many empirical studies using the constructs from these models in a wide domain of investigations.

We use data from the Cornell Personality Disorder Institute (CPDI) BPD treatment study (Clarkin, Levy, et al., 2007), which includes rich baseline assessments of BPD patients as well as a large of array of individual growth curve data, to conduct this study.

Method

Subjects

The CPDI BPD treatment study design has been described in extensive detail elsewhere (Clarkin, Levy, Lenzenweger, & Kernberg, 2004; Clarkin, Levy, et al., 2007). There were 58 men and women between the ages of 18 and 50 who met *DSM–IV* (American Psychiatric Association, 1994) criteria for BPD in this study. BPD was diagnosed in our patients using the *International Personality Disorder Examination* (IPDE, Loranger, 1999). BPD symptom assessments were reliable (intraclass correlation = .83) as were categorical diagnoses (κ = .64). Axis I psychopathology was diagnosed using the Structured Clinical Interview for *DSM–IV* (SCID; First et al., 1996). The sample was a heterogeneous group of BPD cases as is seen in community settings (see Korfine & Hooley, 2009).

Assessment Instruments: Diagnosis of Axis I and Axis II Disorders

International Personality Disorder Examination (IPDE). The IPDE was used to assess both *DSM* and *ICD-10* PD features (Loranger, 1999).

Structured Clinical Interview for *DSM–IV* (SCID). The SCID was used to assess Axis I disorders (First, Gibbon, Spitzer, & Williams, 1996).

Assessment Instruments: Baseline Predictor Measure Instruments

We selected specific indexes from the following measures as baseline predictors of rates of change. All of these indexes were drawn from established quantitative measures possessing good to excellent psychometric characteristics (i.e., reliability, validity).

Avoidant Personality Disorder features. Avoidant PD features were assessed using the IPDE and the dimensional score for these features was used in these analyses.

Multidimensional Personality Questionnaire (MPQ). MPQ (Tellegen, 1982; Tellegen & Waller, 2008) assesses 11 primary personality dimensions and three higher-order traits (positive emotion, negative emotion, constraint). We used the following MPQ measures as baseline predictors: harm-avoidance, social closeness, social potency, aggression, negative emotion, and alienation.

Inventory of Personality Organization (IPO). The IPO (Clarkin, Foelsch, & Kernberg, 2001; Lenzenweger, Clarkin, Kernberg, & Foelsch, 2001) assesses five clinical dimensions relevant to the diagnosis of borderline personality organization (Kernberg, 1984), namely identity diffusion, primitive psychological defenses, reality testing impairments, aggression, and moral values. We used the identity diffusion, primitive defenses, reality testing, and aggression IPO subscales as baseline predictors.

Psychopathic Personality Inventory (PPI). The PPI (Lilienfeld & Andrews, 1996) assesses personality—based psychopathy in nonincarcerated individuals (Lilienfeld & Andrews, 1996). We used the PPI blame externalization subscale a baseline predictor that tapped the "self versus other" perception facet of the psychodynamic model.

Adult Temperament Questionnaire (ATQ). The ATQ (Evans & Rothbart, 2007; Rothbart, Ahadi, & Evans, 2000) is a self-report inventory that assesses 18 facets of adult temperament. We used the ATQ effortful control factor (activation control, effortful attention, inhibitory control) as an additional measure of nonaffective constraint.

Assessment Instruments: Measures for Rate of Change Data

At study baseline and each of the subsequent assessments occurring at 3 month intervals, each subject was evaluated by an experienced clinician. The principal domains assessed (and the instruments used to asses them) were sui-

cidality (Overt Aggression Scale [Modified]; Coccarro et al., 1991), aggression (Anger, Irritability, and Assault Questionnaire; Coccarro & Kaoussi, 1995), impulsivity (Barratt Impulsiveness Scale-II; Patton, Stanford, & Barratt, 1995), anxiety (Brief Symptom Inventory; Derogatis, 1993), depression (Beck Depression Inventory-II; Beck, Steer, & Brown, 1996), social functioning (Global Assessment of Functioning; American Psychiatric Association, 1994), and psychosocial adjustment/functioning (Social Adjustment Scale; Weissman & Bothwell, 1976). Each of these measures is associated with good to excellent psychometric properties. We calculated an IGC for each subject for each of the 11 indexes noted in the first column of Table 1. Each IGC, which defines change as a linear function of time, generates an intercept and slope value. The intercept value in the IGC refers to the *initial level* value for the subject, whereas the slope value defines the *rate of change*. The data for all patients were combined irrespective of treatment condition in the original RCT. We retained the slope values for these subjects as dependent variables, which explicitly define our interest in how quickly (or not) change was happening in the subjects over time.

Statistical analysis. The primary data for these analyses were the rate of change estimates (slopes), which were retained from ordinary least squares analysis of the unconditional IGCs. A principal components analysis (PCA) was performed on the rate of change values for 11 dimensional measures in the first column in Table 1. Our sample size was smaller than those seen in many factor analytic studies. However, we note we used PCA primarily as a data reduction technique. This was *not* a latent structure/model testing exercise, an attempt at recovery of a population factor pattern, or a scale revision effort (see Reise et al., 2000). Our subject to variable ratio for the PCA was 5.2:1[1] and well within the range of values recommended by methodologists (Gorsuch, 1983; cf., MacCallum, Widaman, Zhang, & Hong, 1999). PCA components were retained, as is common, if they had an eigenvalue greater than 1.0. A Varimax (orthogonal) rotation was used to facilitate interpretation of the obtained factor solution. Variables were assigned to specific factors if the factor loaded the variable at .50 or higher. Factor scores were calculated for each of the retained factors using a regression approach. Associations between the baseline predictors and the factor scores were examined with Pearson correlations. Forward stepping multiple linear regression was used to determine which baseline predictor(s) showed the strongest association with each of the factor scores. The relative importance of baseline predictors in the regressions was assessed with the part (semipartial) correlation coefficient (Darlington, 1990). Our use of the regression approach allowed us to evaluate the relations of multiple predictors to the factor scores, thus, taking into account both multicollinearity and multiple tests. Therefore, we did not adjust the alpha level for the zero order correlations reported below. Differences in group means were tested using one-way analysis of variance (ANOVA). To control for initial treatment group, partial correlations were computed between the baseline predictors and the retained factor scores using two dummy variables, which coded original treatment group, as covariates.

Results

The sample consisted of 58 subjects (54 female) with an average age of 30.5 ($SD = 6.92$). Forty subjects were Caucasian, five African American, three Hispanic, five Asian American, and five Other/Mixed Ethnicity. Seven subjects were married, 40 were divorced, 11 cohabiting, and 21 in a relationship (not cohabiting). Most of the subjects ($n = 33$) had completed college and/or graduate study, four had an Associate's degree, 18 had some college, two were high school graduates, and one had less than high school education. The mean WAIS Vocabulary Scaled score was 13.80 ($SD = 2.71$). The mean GAF score was 48.63 ($SD = 8.74$), suggesting considerable functional impairment. In terms of Axis I psychopathology (lifetime): Major depression = 44.8%, Anxiety disorder (any) = 44.8%, Eating disorder (any) = 29.3%, and Drug or Al-

[1] No definitive rule exists for the optimal or required subject to variable ratio; nor would such a rule make sense in all applications. Further, there are some who suggest firm guidelines for sample size in factor analysis; however, it is well known that common, universal "rules of thumb" regarding sample size in factor analysis are far from universal and generally not altogether useful for researchers (see MacCallum, Widaman, Zhang, & Hong, 1999; Preacher & MacCallum, 2003).

Table 1
*Principal Components Analysis Solution (With Varimax Rotation) for Rate of Change Across 11 Assessment Measures in Borderline Personality Disorder Patients (*N = 58)

	Factor 1	Factor 2	Factor 3
Rate of change measure	Aggressive dyscontrol change rate	Social adjustment/Self-acceptance change rate	Conflict tolerance/Behavioral control change rate
Anger (past week)	.90		
Irritability	.85		
Verbal Assault	.83		
Direct Assault	.70		
Suicidality		.80	
Social Adjustment		.66	
Depression		.61	.50
Global Functioning		.60	
Anxiety			.79
Impulsivity-III "coping instability"			.78
Impulsivity-II "poor planning"			.34

Note. GAF reversed. Barratt Factor I omitted from analysis it revealed insignificant change over time in Clarkin et al. (2007) study. Loadings smaller than .30 are omitted for clarity.

cohol Abuse/Dependence = 31%. The mean number of Axis II disorders was 2.48 (*SD* = 1.06; Low = 1/High = 5). Many subjects displayed either lifetime suicidal behavior (75%) or prior parasuicidal behavior (80%).

The PCA yielded evidence for three factors underlying the 11 measures of rate of change (slope) for the outcome dimensions in these BPD patients (see Table 1). The clustering of DV's seen on each of the three factors suggested relatively clear substantive interpretations. The three PCA accounted for 61% of the variance in the slope measures and represent: (a) aggressive dyscontrol change, (b) psychosocial change (that is, global functioning/social adjustment change), and (c) conflict tolerance (anxiety/depression) and impulsivity change. Principal axis factoring yielded highly similar results to those obtained with the PCA. We calculated factor scores for the sample and each subject received a factor score for each of the three factors. All results reported in this article for the factor scores derived from the principal components analysis were highly similar to those obtained using principal axis factoring as the data reduction method.

In this context, we note we were clearly aware that these BPD subjects were each originally assigned randomly to one of three treatments and received that treatment during the study period. To determine if initial treatment group was related to levels on the three factor scores we conducted one-way ANOVA's on the three factor scores. These ANOVA's revealed no significant differences as a function of original treatment group membership (i.e., TFP vs. DBT vs. SPT) (multivariate one-way ANOVA, $p > .60$; all univariate one-way ANOVA's $p \geq .40$), which supported combination of subjects across treatment cells for these analyses. These results are consistent with those reported by Clarkin et al. (2007), wherein contrasts for individual slopes for each domain did not reveal significant *between groups* treatment effects.

The zero-order/bivariate correlations between each of the 13 baseline individual difference characteristics and the factors scores obtained from the three factor PCA solution are in Table 2.[2] We then used multiple regression analysis (MRA) to determine which variable(s) would be significantly related to each of the factors scores when the predictors were considered jointly. Regression of Factor 1 scores on the 10 variables with significant bivariate associations (see Table 2) revealed on two signifi-

[2] We evaluated all of the pairwise associations reported in Table 2 (the correlations between the baseline predictors and the factor scores), using partial correlation wherein original treatment group was dummy coded and treated as a covariate. Results of these partial correlation (control analyses) were essentially identical to those in Table 2.

Table 2
*Bivariate Correlations Between Baseline Predictor Variables and Domains of Change Factors (*N = *58)*

Behavioral feature	Aggressive dyscontrol change rate	Social adjustment/Self-acceptance change rate	Conflict tolerance/Behavioral control change rate
Avoidant PD (IPDE)	.20	−.18	−.27*
Harm-Avoidance (MPQ)	−.15	.09	−.08
Social closeness (MPQ)	−.30*	.13	.14
Social potency (MPQ)	.02	.17	.32*
Aggression (MPQ)	.44**	−.26†	.01
Negative Affect (MPQ)	.48*	−.25†	−.05
Alienation (MPQ)	.36*	−.07	.02
Identity Diffusion (IPO)	.30*	−.36**	−.09
Primitive Defenses (IPO)	.28*	−.30	−.05
Reality Testing (IPO)	.31*	−.21	−.05
Aggression (IPO)	.32*	−.23†	.00
Blame externalization (PPI)	.29*	−.10	.07
Effortful Control (ATQ)	−.28*	.18	.07

Note. Values are Pearson product-moment correlation coefficients; two-tailed test of statistical significance. IPDE = International Personality Disorder Examination; MPQ = Multidimensional Personality Questionnaire; IPO = Inventory of Personality Organization; PPI = Psychopathic Personality Inventory; and ATQ = Adult Temperament Questionnaire.
† $p < .10$. * $p < .05$. ** $p < .01$.

cant predictors (Model Multiple $R = .54$), both from the MPQ, negative affect (*Beta* = .34, semipartial $r = .30$, $t = 2.53$, $p = .014$) and aggression (*Beta* = .29, semipartial $r = .25$, $t = 2.17$, $p = .035$). The semipartial correlations indicate that both negative affect and aggression make relatively unique contributions to the prediction model. Regression of Factor 2 scores on primitive defenses and identity diffusion revealed only identity diffusion (Model Multiple $R = .36$) was a significant predictor (*Beta* = −.36, semipartial $r = -.36$, $t = -2.84$, $p = .006$). Finally, regression of Factor 3 scores on social potency and avoidant personality disorder features revealed only social potency (Model Multiple $R = .32$) was a significant predictor (*Beta* = .32, semipartial $r = .32$, $t = 2.55$, $p = .013$). In summary, the MRA revealed (a) baseline negative affectivity and aggression predicted the aggressive dyscontrol change domain (Factor 1); (b) baseline identity diffusion predicted the social adjustment/self-acceptance change domain (Factor 2); and (c) baseline social potency predicted the conflict tolerance/ behavioral control change domain (Factor 3). To reiterate, the relations described here involve the prediction of domains of change rates over time as predicted by cross-sectional baseline individual difference variables.

Discussion

In tapping the rate of change data extracted from the CPDI-BPD study, we were able to probe the multidimensional array of change as it revealed itself in these BPD patients over time. Our results clearly indicate that treated BPD patients are showing change over time in numerous dimensions simultaneously. However, it is important to note that similar rates of change cluster together and these clusterings fall into several broad domains. Thus, there are broad areas of functioning and/or psychological features that change at comparable rates and there is some consistency in content among those features changing at comparable rates. Specifically, the three factors that coalesced, accounting for 61% of the variance across a wide range of rate of change measures, represent: (a) anger/aggression change ("aggressive dyscontrol"); (b) global functioning/social adjustment change ("social adjustment/self-acceptance"); and (c) affective dyscontrol related to anxiety/depression/impulsivity change ("conflict tolerance/behavioral control"). Our results suggest these domains are not merely cohering due to phenotypic similarity of the measured variables, but because the variables within these domains are changing at comparable rates, which drives their covariation. This *multidimensionality* has

important clinical implications as it suggests that different areas of functioning and symptomatology that are impaired in BPD will change at different rates *and* certain sets of variables will change, broadly speaking, in step (i.e., as a domain). The multidimensionality also raises the interesting possibility that different mechanisms may be at work in relation to change in each of the domains (see Clarkin & Levy, 2006). These data raise the interesting possibility that patient-level features (variables) may work in concert with treatment and this process may relate to particular domains of change. For example, consider the baseline level of identity diffusion of a patient interacts with treatment and a by-product of this interaction is an improvement in social functioning over time. Finally, the multidimensionality in change rates we observed may be reflective, in part, of the heterogeneity known to characterize BPD patients (Clarkin, Hull, & Hurt, 1993; Lenzenweger, Clarkin, Yeomans, Kernberg, & Levy, 2008) and may provide insights to potential endophenotypes for BPD (Lenzenweger, 1999a; Gottesman & Gould, 2003).

This report extends our understanding of the CPDI BPD data in important ways beyond the Clarkin, Levy, et al. (2007) findings. We note that in the Clarkin, Levy, et al. (2007) study the patients were randomly assigned to one of three treatment groups and rates of change were assessed on each of 12 outcome variables taken individually. This article extends that report by providing a more succinct summary of the change patterns harbored within the 12 individual domains described in Clarkin, Levy, et al. (2007) . It does so by reducing them through a principled data reduction approach (PCA) to the three broad domains we identified here. Moreover, as substantial differences in rates of change did *not* exist among the three different treatments described by Clarkin, Levy, et al. (2007) , we collapsed across those treatment groups and we can broadly speak to BPD patients in treatment. Thus, the present report provides a streamlined picture of change in BPD. Finally, this report helps to reveal those baseline individual difference variables predictive of change. The original Clarkin, Levy, et al. (2007) report did not address predictors of change beyond assigned treatment group.

To gain leverage on the possibility that relatively distinct mechanisms might be at play in terms of the different domains of change, we examined the relations between baseline psychological characteristics (predictors) and scores for each of these domains. In short, if different variables at baseline predicted different domains of change rates, then those variables might offer some initial insights into potential change mechanisms. We adopted a judicious stance in the selection of predictor variables to ensure the baseline predictors were theoretically relevant to BPD and were not excessive in number. We discovered that (a) baseline negative affectivity and aggression predicted the aggressive dyscontrol change domain, (b) baseline identity diffusion predicted the global social adjustment/self-acceptance change domain, and (c) baseline social potency predicted the conflict tolerance (i.e., affective/impulsivity) change domain. Bearing in mind the direction of the rates of change (slopes) associated with clinical improvement, these associations show that (a) lower initial levels of negative affect and aggression were associated with more rapid clinical improvement in the anger/aggression change domain, (b) higher baseline identity diffusion was associated with more rapid rates of clinical improvement in the global functioning/social adjustment domain, and (c) lower initial levels of social potency (i.e., dominance) were associated with more rapid rates of clinical improvement in anxiety and depression as well as impulsivity change domain (termed "conflict tolerance/behavioral control"). *This profile may be clinically useful in identifying those BPD patients likely to show the most rapid rates of change in therapy.*

Each of these predictors points to important systems of relevance to BPD. The negative affect (anxiety, distress, irritability, and depression) and constraint (indirectly indexed by aggressive behavioral dyscontrol) systems are both strongly related to the rate at which change occurs across an array of angry, aggressive behaviors, consistent with primary tenets of the neurobehavioral modeling of BPD (Depue & Lenzenweger, 2005). The relative importance of both negative affect *and* aggression is underscored by the fact that each made *relatively unique* contributions to the prediction of change in the regression analysis, despite themselves being correlated with one another ($r = .48$). That baseline social potency, which taps interpersonal dominance and forceful social visibil-

ity, is related to the rate of change across the domain of impulsivity and depression raises the interesting question of the degree to which social dominance, which is related to activity in the incentive reward/motivation system, (a) impacts effective engagement with the external world offering rewards (including therapy); and (b) how certain levels of such connection may serve to impact order with respect to anxiety, conflict, and impulse control. Finally, the identity diffusion feature, which is a critical component of Kernberg's model (1984; Kernberg & Caligor, 2005; Stern et al., 2010) of BPD, emerged as a predictor of rates of change in psychosocial functioning. Thus, not only does identity diffusion represent a potentially important predictor of rate of change in social adjustment, the association supports the foundational role in underpinning social functioning as argued by Kernberg (1984; Kernberg & Caligor, 2005). From the clinical standpoint, identity diffusion may be a focus for enhanced treatment efforts as part of the pathway to improved psychosocial functioning, an area of improvement that is critical for continued psychological health (see also Zanarini, 2008).

There are some limitations to be kept in mind when considering these results. First, the sample consisted of 58 subjects. This sample size is lower than we might like for a multivariate study drawing upon factor analytic methods; however, this concern must be viewed in light of the multiwave prospective design embodied in these data. Even though we used PCA as a data reduction as opposed to a model testing technique, we stress a future study should have a larger sample size. Second, the patients we report on in this study were followed for a period of one year. It is conceivable that over a longer follow-up period, with more waves of assessment, that other predictors may have emerged as useful in understanding those correlates of the rate of change domains. Indeed, more assessment waves would yield more stable estimates of slope for all measures examined in multiwave perspective and confer even greater power to the study. Third, we chose our baseline predictors based on two viable *dimensional* models of BPD, Depue and Lenzenweger's neurobehavioral model and Kernberg's psychodynamic/object relations model. As noted, other dimensional models of PD with relevance to BPD have been proposed (e.g., Cloninger's (1993)—Siever's (Siever & Davis, 1991), Linehan's (1993; Crowell, Beauchaine, & Linehan, 2009), Millon's (Millon & Grossman, 2005); and Beck's (Beck, Freeman, Davis, & Associates, 2004)—and those models might very well potentially direct one to a different set of predictors. One could also consider descriptive accounts of BPD as ensconced in *atheoretical* approaches to personality such as embodied in the five factor approach and other psychometric conceptualizations (e.g., Widiger, Clark, & Watson, 2009). However, no one research group can test all possible models of a form of psychopathology and we selected our models based on our prior clinical and research experience. Fourth, we note that each the three treatments that were administered originally to these patients have different foci and it is conceivable that these treatments might have had some degree of differential impact on the studied outcome dimensions. However, the empirical results reported by Clarkin, Levy, et al. (2007) suggested that this was essentially not the case. Thus, despite the treatments having markedly different foci, these different foci did not translate into markedly different change rates across the treatments. This feature of the dataset provides a nontrivial degree of assurance that combination of the BPD patients into one sample for this study was defensible. It is conceivable that our findings would not generalize to nontreated BPD individuals. Finally, most of the patients in this study were women. Although BPD affects men and women equally in the general population (Lenzenweger et al., 2007), women still outnumber men in clinical practice settings. Thus, the current findings have considerable relevance to the modal BPD-affected individual seen clinical practice.

In summary, our data suggest that there are differentiable domains of change in BPD and each of these domains is predicted by relatively unique baseline personality/psychological factors. These predictors suggest not only a theoretical focus in the search for mechanisms of change in BPD, but also foci for clinical intervention. We present these results not as definitive or confirmatory, but rather we emphasize their heuristic value viewed within a context of discovery.

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Correction to Marcus, Fulton, and Edens (2011)

In the article, "The two-factor model of psychopathic personality: Evidence from the Psychopathic Personality Inventory," by David K. Marcus, Jessica J. Fulton, and John F. Edens, (*Personality Disorders: Theory, Research, and Practice*, Advance online publication, October 10, 2011. doi:10.1037/a0025282) an asterisk was misplaced in the reference section. Data from Blonigen, Hicks, Krueger, Patrick, and Iacono (2006) were not included in the meta-analysis because that study did not use the Psychopathic Personality Inventory (PPI). The asterisk should have been placed next to Blonigen et al. (2010), which used the PPI and was included in the meta-analysis.

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