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Associations Between Changes in Normal Personality Traits and Borderline Personality Disorder Symptoms over 16 years

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Abstract

There has been significant movement toward conceptualizing borderline personality disorder (BPD) with normal personality traits. However one critical assumption underlying this transition, that longitudinal trajectories of BPD symptoms and normal traits track together, has not been tested. We evaluated the prospective longitudinal associations of changes in five-factor model traits and BPD symptoms over the course of 16 years using parallel process latent growth curve models in 362 patients with BPD (*N*=290) or other PDs (*N*=72). Moderate to strong cross-sectional and longitudinal associations were observed between BPD symptoms and Neuroticism, Extraversion, Agreeableness, and Conscientiousness. This study is the first to demonstrate a longitudinal link between changes in BPD symptoms and changes in traits over an extended interval in a clinical sample. These findings imply that changes in BPD symptoms occur in concert with changes in normal traits, and support the proposed transition to conceptualizing BPD, at least in part, with trait dimensions.

Keywords

Borderline Personality Disorder; Personality Traits; Five Factor Model; Personality Development; Latent Growth Curve Modeling

A growing body of evidence indicates that borderline personality disorder (BPD) can be conceptualized using normal personality traits (Miller et al., 2012; Mullins-Sweatt et al., 2012; Samuel et al., 2012; Trull & Brown, 2012; Trull et al., 2003; Widiger, 2005). Doing so would solve a number of problems with the polythetic categorical approach of the Diagnostic and Statistical Manual of Mental Disorders (DSM; American Psychiatric Association, 2013). For instance, one problem that has been widely cited involves diagnostic co-occurrence. Dimensional trait models provide an explanation for the co-occurrence that exists between BPD and other disorders, insofar as this co-occurrence can be explained in

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terms of shared trait tendencies. Trait approaches also solve the problem of diagnostic heterogeneity. The current DSM formulation in which a patient needs 5 of 9 possible criteria creates the potential for 256 different combinations of criteria above the diagnostic threshold, all of which nevertheless receive the same diagnosis. In contrast, trait models provide a means of articulating specifically how patients might differ from one another in terms of continuously graded personality features. Third, a major advantage of this approach involves the potential to integrate the diagnosis of personality pathology with basic personality science, a broad literature with potentially useful insights regarding the etiology, development, and manifestation of BPD (Vachon et al., 2013; Widiger & Trull, 2007; Wright, Pincus, & Lenzenweger, 2013).

The most widely discussed trait model for reformulating PDs has been the Five-Factor Model (FFM: Extraversion, Agreeableness, Conscientiousness, Neuroticism, and Openness). In meta-analyses on the cross-sectional associations between FFM traits and BPD symptoms (Samuel & Widiger, 2008; Saulsman & Page, 2005), BPD is consistently associated with high Neuroticism, low Agreeableness, and low Conscientiousness. In these studies, correlations tend to range from −.20 to −.30 for Agreeableness and Conscientiousness and . 50 to .60 for Neuroticism. Some aspects of low Extraversion such as low warmth and low positive emotions are also associated with BPD (Samuel & Widiger, 2008). Traits furthermore predict future levels of BPD (Morey et al., 2007) and more generally can be used to account for differential rates in personality pathology over the life span (e.g., Vachon et al., 2013). Behavior genetic research suggests that the heritable components of BPD overlap entirely with the heritable aspects of normal traits (Distel et al., 2009). Finally, some research indicates that borderline symptoms can be conceptualized as extreme variants of normal personality, and particularly Neuroticism, from an Item Response Theory perspective (Samuel et al., 2013).

Nevertheless, it is also true that borderline symptoms provide incremental information over traits in predicting patient dysfunction (Hopwood & Zanarini, 2010), perhaps indicating that traits do not capture all that is important about BPD. Importantly, this has been the case even for pathological traits (Morey et al., 2012), which tend to correlate more strongly than normal traits to BPD (e.g., Hopwood et al., 2012), so this pattern of results apparently cannot be fully explained by the amount of maladaptive content in trait measures.

This pattern of findings seems to support the hybrid diagnostic model of personality disorder classification that is listed in Section III (Emerging Models and Measures) of the DSM-5 (Skodol, 2012; Krueger et al., 2012). In this model, BPD will be indicated, in Criterion A, by specific forms of self and interpersonal dysfunction and in Criterion B by traits involving maladaptive Negative Affectivity (i.e., high Neuroticism, including emotional lability, anxiousness, separation insecurity, and depressivity), Disinhibition (i.e., low Conscientiousness, including impulsivity and risk taking) and Antagonism (i.e., low Agreeableness, hostility). Initial research on the trait (Criterion B) portion of this model has been promising. Several studies have shown strong convergence and relatively specific associations between BPD-linked DSM-5 traits and severity on BPD symptoms (Andersen et al., 2014; Few et al., 2013; Fossati et al., in press; Hopwood et al., 2012; Morey & Skodol, 2013). Miller and colleagues (2012) extended these findings by showing that

estimates of BPD severity based on DSM-5 Section III traits and traditionally defined criteria have highly similar patterns of associations with external correlates.

However in contrast to robust evidence for cross-sectional associations between BPD symptoms and normal traits, little is known about longitudinal relationships between traits and BPD symptoms. There is no statistical requirement that cross-sectional associations reflect correspondence in rates of change over time; these are quantitatively dissociable processes. This is particularly the case when cross-sectional associations are only moderate, as is the case with FFM-BPD correlations in meta-analytic work. For instance, the conditions of a particular assessment method (Widiger & Anderson, 2003) or other third variables could explain cross-sectional correlations between traits and BPD. However, it is rather unlikely that two types of variables, such as personality traits and BPD symptoms, would be longitudinally associated if they were not capturing aspects of the same underlying construct. Demonstrating the association of trajectories in personality traits and BPD symptoms over time would therefore bolster the claim that BPD can be represented, at least in part, with trait criteria as had been articulated in the DSM-5 and would provide strong evidence in favor of employing basic personality research to understand psychopathology.

No existing study has evaluated the longitudinal association of the trajectories of changes in normal traits and BPD symptoms in a clinical sample over a long-term period of follow-up. Perhaps the study with most similar aims was conducted by Warner and colleagues (2004) using data from the Collaborative Longitudinal Personality Disorder Study. In that study, cross-lagged models were used to evaluate the stability of severity on an interview-based BPD diagnosis, levels of traits hypothesized to characterize BPD (see Lynam & Widiger, 2001), and the cross-lags between these domains. In general, this study revealed that both BPD and BPD-linked FFM traits are relatively rank-order stable over 1 year, and that there was some limited evidence that traits and BPD symptoms could predict one another after controlling for initial values in either system.

However, although Warner et al. (2004) conceptualized their study as evaluating whether change in traits accounted for change in disorders, the cross-lagged model used in that study is not amenable to evaluating associations between longitudinal trajectories in traits and longitudinal trajectories in BPD features (see e.g., Lenzenweger & Willett, 2007). This is because a standard cross-lagged panel model estimates differential, or rank-order, stability between two waves using autoregressive paths (e.g., traits at Time 2 regressed on Traits at Time 1), and thus the cross-lagged paths (e.g., Disorder at Time 2 regressed on Traits at Time 1) predict changes in rank order from one variable to the next (i.e., the residuals after controlling for previous time point). Therefore, these paths do not test whether individual change in one variable is associated with individual change in another, and instead answer a different question. Importantly, rank-order stability may be relatively high even when there is significant individual variability in the direction and rate of change trajectories. Furthermore, with two waves of data only initial starting value and ending value are considered, but not the shape of change over the interim. An additional consideration is that, Warner et al. did not test all FFM trait domains, but rather focused on FFM facets thought to be specific to BPD as a group (i.e., five of the NEO Personality Inventory Neuroticism facets, and one Conscientiousness facet). Finally, their study was limited to 1-year intervals.

When the question of interest is whether individual differences in the shape and rate of within-person change over time in one variable (e.g., traits) is associated with shape and rate of change in another variable (e.g., BPD), then the most appropriate model is a latent growth curve model (LGM). Growth curve models can be estimated using structural equation models (SEM) that parameterize the sample average and individual differences in trajectories of change in a variable (e.g., BPD or traits) over time as a function of growth factors. Growth factors are just like other factors estimated in a confirmatory factor analysis framework, but they (a) use as observed data variables that are related to each other longitudinally (e.g., Time 1, Time 2, Time 3), and (b) code change over time in the factor loadings. In a typical and very basic model there would be a factor that estimated the level of the growth trajectories, and a factor that estimated the slope, or rate of change in the trajectories—together, a level and slope would provide the minimum necessary information for a longitudinal trajectory. In this type of model the average level of the sample's trajectories is accounted for by an intercept factor mean, with individual differences reflected in the intercept factor variance, whereas rate of change is accounted for by a slope factor mean and variance. Statistically significant variances for the individual and slope factors suggest that individual heterogeneity in trajectories can be associated with or predicted by other variables. In the case of parallel process models, which are a multivariate extension of a standard LGM analogous intercept and slope terms depict change in another trait or PD feature within the same structure. Thus, changes in traits can be correlated with changes in PD symptom severity over time. For example, Wright, Pincus, & Lenzenweger (2013) recently applied parallel process LGM to examine the association between trajectories of change over time in avoidant PD and normal personality traits. They found that increases in trait Dominance and Affiliation and decreases in Neuroticism were associated with more rapid remission of avoidant PD symptoms.

In the current study we use parallel process latent growth curve modeling (Figure 1 to evaluate the longitudinal relationship between FFM traits and BPD symptoms over 16 years in a sample of individuals with significant rates of baseline BPD symptoms (Zanarini et al., 2005). This approach allowed us to test cross-sectional associations between BPD symptoms and trait levels as well as the relationship between changes in traits and changes in BPD symptoms over time. Significant correlations between trait and BPD intercept levels would be consistent with previous findings regarding cross-sectional associations between personality traits, particularly high Neuroticism and low Agreeableness and Conscientiousness with BPD. Given that previous studies in this particular sample have shown significant negative correlations between Extraversion and BPD (Hopwood, Donnellan, & Zanarini, 2010) and that some aspects of Extraversion correlate negatively with BPD in meta-analyses, we also expected significant longitudinal correlations between Extraversion and BPD symptoms. Significant slope (i.e., change) correlations would suggest that rates of change in traits are linked with rates of change in BPD symptoms. By additionally modeling cross-sectional correlations among the observed variables, parallel process growth curves allow for a very stringent test of associations of the trajectories of change even after controlling for within time associations.

Method

Participants were 362 individuals enrolled in the McLean Study of Adult Development (MSAD; Zanarini et al., 2005) after being screened for a) age between 18 and 35, b) known or estimated IQ > 70, c) absence of historical or current symptoms of schizophrenia, schizoaffective disorder, bipolar I disorder, or an organic condition that could cause serious psychiatric symptoms, d) English fluency and e) meeting interview criteria for BPD or another PD. At baseline, 77.1% (*n* = 279) of participants were women, 87% (*n* = 315) were Caucasian, the mean age was 27 years (*SD* = 6.3). The average Global Assessment of Functioning score was 39.8 (*SD* = 7.8) and 290 met diagnostic criteria for BPD. Of the original 362 participants, 79.8% (*n* = 289) were followed through 16 years, with 28 (7.7%) having died and the other 45 (12.4%) lost to attrition.

Assessments

For the purposes of this study, we used the total score (*range* = 0–44) of the *Revised Diagnostic Interview for Borderli*nes (DIB-R; Zanarini et al., 1989) to assess BPD symptoms. We used the *NEO Five-Factor Inventory* (NEO-FFI; Costa & McCrae, 1992) to assess normal personality traits. Assessments occurred at baseline and every two years for 16 years (9 assessments total). As has been reported previously (Zanarini et al., 2012), symptoms declined precipitously over the course of the study, with the majority of change occurring in the first half of the study.

Analyses

We first evaluated the shape of average change on BPD symptoms and each of the five traits in the MSAD sample over 16 years. We constructed six individual LGMs in Mplus 7.11 (Muthén & Muthén, 2012), one for BPD symptoms and one for each FFM trait. Missing data were handled with Full Information Maximum Likelihood estimation. LGMs offer a highly flexible approach to estimating the mean shape (i.e., level and rate) of change, and individual heterogeneity around the average trajectory (i.e., growth factor variances), in a given psychological system (e.g., BPD, personality traits), using specific parameterizations of latent factors. In a standard approach, latent intercept and slope factors capture the level and linear rate of change in a given construct, respectively. However, additional factors can be added to estimate non-linear rates of change (e.g., quadratic trends) and stark shifts in the rate of change (i.e., piecewise models). Thus, through the combination of the appropriate growth factors the pattern of change can be closely fitted using LGMs. Individual heterogeneity in the shape and rate of change can be ascertained by testing for significant variances in the estimated growth factors. Initially we fitted linear growth models, although we expected these to be poorly fitting given a previously established dampening in the rate of change in BPD symptoms in this sample after 8 years. As such, we then tested different approaches to modeling this shift, fitting models that either included a quadratic rate of change, or a clearly defined shift at 8 years.

As described above, LGMs can also be estimated in a multivariate fashion, capturing growth in more than one psychological system simultaneously (i.e., parallel processes), and assessing the associations (i.e., covariances) between growth factors across systems. Parallel

process (alternatively multivariate or associative) LGMs can therefore directly test whether the trajectories in BPD symptoms track with the trajectories in normal personality traits, offering a stringent test of study hypotheses. Figure 1 provides a diagram of these models as they were fitted in this study.

Given that past research indicates that levels of personality (Roberts, Walton, & Viechtbauer, 2006) and BPD symptoms (McGlashan, 1986) are associated with age and that there was age variability in this sample, age was controlled in final models by regressing growth factors on age at the start of the study.[1] We similarly controlled for sex. Additionally, because this sample was recruited from a clinical setting and many of the patients either were in treatment at the outset or sought treatment over the subsequent years, we included treatment level since the prior assessment as a time-varying covariate. Treatment level was defined as the sum of treatment types (e.g., medication, outpatient psychotherapy, inpatient treatment) an individual engaged in between assessment points. This variable was treated as a time-varying covariate such that the observed variables (i.e., traits and BPD) for each wave were regressed on treatment level since the previous assessment.

Model fit was evaluated using multiple fit statistics. Due to the extreme sensitivity of the overall model chi-square to modest departures from fit, and the difficulty with exactly fitting growth in 9 waves of assessment, we relied primarily on the Root Mean Square Error of Approximation (RMSEA; Browne & Cudeck, 1992), which provides a test of "close" fit of the model to the data. By convention, RMSEA values of .05 or lower indicate close fit, which can be further ascertained with 90% confidence intervals (CIs) and a *p*-value. Further, we considered the values of the Comparative (CFI) and Tucker-Lewis (TLI) fit indices, for which values of close to or exceeding .95 have been determined to indicate excellent fit of the model to the data (Hu & Bentler, 1999). To compare non-nested models, we relied on the sample size (i.e., second order bias) corrected Akaike Information Criterion (AIC_C; Sagiura, 1978). The AIC_C is a relative fit index, which balances the increase of model fit associated with adding parameters with the loss of parsimony by imposing a weighted penalty.

Results

In the current study, model estimation proceeded as follows. Our first step involved fitting a univariate LGM to the observed DIB-R BPD scores, as they are the primary focus of this study. As a point of departure, we initially estimated a simple linear LGM, although it was expected to be poorly fitting based on the observed patterns of means (see top panel of Figure 2). As expected, the model was a poor fit to the data ($\chi^2_{40} = 759.74$, $p < .001$; RMSEA = .22, RMSEA 90% CI = .21-.24, RMSEA $p < .01$; CFI =.63; TLI = .67; AIC_C = 16305.8). In an effort to capture the marked shift in the average trajectory at year 8

[1]We alternatively modeled age as a separate growth process in addition to elapsed time in the study (e.g., Ferrer et al., 2004), however no substantive differences in results were observed despite considerable increases in model complexity. Futhermore, we considered modeling growth centered on youngest age in the sample. However, given the MSAD sampling strategy that selected for individuals elevated in PD features, the most salient time point in terms of BPD features was the initial assessment, regarldess of age, and stratifying by age created a distorted mean trajectory over time. Therfore, we elected to model growth in time since assessment and use age as a linear covariate of growth factors.

discussed above and observable in Figure 2, we next estimated a LGM with a quadratic growth factor which improved fit significantly based on the $\Delta\chi^2$ and AIC_C, although it remained outside of acceptable ranges (χ^2_{36} = 225.24, $p < .001$; RMSEA = .12, RMSEA 90% CI = .11-.14, RMSEA $p < .01$; CFI =.90; TLI = .90; AIC_C = 15780.0). Based on an examination of the residuals, we next fit a piecewise LGM with an intercept factor and two linear slope variables. The first linear slope variable captured linear change from baseline to year 8, and the second reflected linear changes from years 8 to 16. In other words, linear slopes were estimated for the first and second half of the study for BPD symptoms (see Figure 1, piece 1 and 2). However, model fit was poor and comparable to the quadratic model (χ^2_{36} = 235.03, p < .001; RMSEA = .12, RMSEA 90% CI = .11-.14, RMSEA $p < .01$; CFI =.90; TLI = .90; AIC_C = 15789.8). Based on an analysis of residuals at each time point in this model, we included a quadratic term for the first half of the study. This model, with latent variables reflecting the intercept, piece 1 (years 0–8) linear slope, piece 2 (years 8–16) linear slope, and the additional piece 1 quadratic slope, provided an excellent fit to the data (χ^2_{31} = 61.41, p = .0004; RMSEA = .06, RMSEA 90% CI = .04-.07, RMSEA $p = .32$; CFI =.98; TLI = .98; AIC_C = 15630.5). Overall, this model indicates that initial changes in BPD symptoms were steepest in the beginning of the study, but the rate of change declined as time progressed (i.e., rate of change leveled off to some degree). Figure 2 shows that the changes in BPD symptoms estimated by this model tracked very closely with the observed changes in the variable over time.

We next evaluated growth models for each of the normal range traits. In the interest of space, we do not report detailed modeling steps and fit here; a comprehensive summary of fit is provided in Supplementary Table 1. To briefly summarize, the complex growth model with a piecewise and quadratic term for the first piece was the best fitting model in an absolute sense (i.e., lowest χ^2 value) for each of the five traits. This is the case despite the fact that in order to stabilize model estimation (i.e., avoid a non-positive definite covariance matrix), the residuals for the initial assessment wave were fixed to zero. In the case of Agreeableness and Neuroticism, the AIC_C clearly favored the same growth structure as BPD features, whereas for Extraversion, Conscientiousness, and Openness the AIC_C very slightly (AIC_C differences < 3.0) preferred the piecewise model without the quadratic term describing changes from baseline to 8 years. It can be concluded from these results that the same complex piecewise growth structure observed in BPD was a good approximation for each trait, although for some traits the addition of the quadratic growth parameter did not improve model fit sufficiently to overcome the AIC_C's penalty for the added complexity.

Third, in order to directly estimate the associations in the change between BPD symptoms and each of the normal traits, bivariate parallel process LGMs were fitted. We report here the results of models where the same pattern of growth was fitted to BPD and each of the traits when controlling for age, sex, and treatment between waves. Neither age nor sex predicted BPD growth parameters. Women were modestly higher on Agreeableness (β = .16, 95% CI = .06, .26, $p = .002$), Conscientiousness (β = .13, 95% CI = .03, .23, $p = .015$), and Neuroticism (β = .15, 95% CI = .05, .25, $p = .004$). Age was predictive of lower Extraversion (β = −.22, 95% CI = −.32, −.12, $p < .001$) and higher Neuroticism (β = .18, 95% CI = .07, .28, $p = .001$) intercepts and Extraversion piece 2 slope (β = .19, 95% CI = .

02, .36, p = .031). Total level of treatment between assessments was always a significant negative predictor of BPD scores, was generally negatively predictive of Neuroticism, and was never predictive of Agreeableness and Openness. This variable was generally not predictive of Conscientiousness and Extraversion scores, although it was significant for a few waves in each. Despite the fact that treatment level significantly accounted for level of BPD symptoms and Neuroticism at each wave, the main study results related to patterns of growth in BPD and traits are substantively similar, and the interpretations identical, to those in the unconditional models and we report these in Supplementary Tables 2. Furthermore, even when the structure of growth is different, using instead best fitting models for Extraversion, Conscientiousness, and Openness (i.e., linear peicewise models), the conclusions are identical (See Supplementary Table 3). In all estimated models, residuals associated with BPD symptoms and trait scores were correlated within each time point to control for any effects particular to a specific assessment wave. See Figure 3 for a depiction of the rich heterogeneity in rates and shape of change in BPD and each trait. Although the mean trajectories presented in Figure 2 are clearly discernable in the Figure 3 panels, what is most dramatic is the extreme variability in initial starting value and rates and shape of change across the study waves when Figure 3 is scrutinized. It is this heterogeneity in trajectories that we hypothesized to be significantly related across systems.

Model fit values and relevant parameter estimates for the parallel process models are shown in Table 1. As described above, the model distinguished between the first and second half “pieces”, which correspond to the first and second halves of the study, to account for different trajectories of change during those time periods (Figure 1). We modeled each trait in the same fashion as the BPD symptom LGM in order to directly associate the trajectories of change across systems. The baseline means of the DIB-R score for BPD and all five traits significantly differed from zero, as did the mean of the linear slope values (i.e., mean rates of change) for BPD symptoms in the initial half of the study. Otherwise, no slope significantly differed from zero after controlling for covariates (with the exception of the second linear slope for Extraversion).[2] Importantly, although generally only the initial linear slopes for BPD were significant, all intercepts and slopes had significant variances suggestive of rich individual heterogeneity in the rate and shape of change, which can then be associated across variables (i.e., BPD and traits).

BPD and trait growth factors were allowed to freely covary within each system. The only two BPD growth factors that were correlated within the final models were the piece 1 linear and quadratic slopes. There was a strong negative correlation between these latent variables suggesting that some individuals started the study with a dramatic drop whose rate then slowed across waves (i.e., upward facing curve), whereas others initially were more stable but their rate of decline increased dramatically as waves progressed (i.e., downward facing curve). The strong correlation suggests that this inverse relationship was nearly uniform across individuals. The same pattern was observed for each of the traits, although additionally intercepts were negatively related to piece 1 linear slope and positively related to quadratic slopes. Due to differences in the average rate of change in the slopes of the

[2]Readers are encouraged to consult Supplementary Table 2 for unconditional model parameters.

traits, these patterns of correlation take on slightly different meanings. For instance, in a negative average slope, a negative slope/intercept covariance usually suggests that those individuals with higher scores decline more rapidly, while those with lower scores are relatively more stable. The opposite is true for a model with a positive average slope. When the slope is neutral, it may suggest a general regression to the mean, or a shuffling of rank order. The interested reader is encouraged to consult Supplementary Tables 2 and 3, which provide estimates of growth without covariates and therefore a better approximation of patterns of significant change.[3]

Table 1 contains the correlations of growth factors across domains. The correlations between corresponding latent intercept and slope factors across traits and BPD symptoms (e.g., the correlations between linear slopes in piece 1) were of central interest for the study. These values are presented in bold in the table. Intercept correlations were consistent with previous research in indicating a strong positive correlation between BPD symptoms and Neuroticism, moderate negative correlations between BPD symptoms and Extraversion and Agreeableness, and a small negative correlation with Conscientiousness. The intercept correlation with Openness was non-significant. The consistency of these results with previous reports from MSAD studies (Hopwood et al., 2010) provides confidence in the rest of the solution. In terms of associated change, the correlations between the piece 1 linear and quadratic slopes were significant for Neuroticism, Agreeableness, and Conscientiousness with effect sizes that ranged from modest ($r = -.26$, Agreeableness) to strong ($r = .62$, Neuroticism). A similar pattern of significance was observed for the links among acceleration of change (i.e., quadratic growth factors). For change in the second half of the study, the correlations between BPD change and trait change were somewhat stronger, ranging from moderate ($r = -.34$, Extraversion) to strong ($r = .72$, Neuroticism) effects, which now include a significant association with change in Extraversion. Taken together, these results suggest that decreases in BPD symptoms during the study were significantly associated with decreasing Neuroticism and increasing Agreeableness, Conscientiousness, and to some degree Extraversion.

Discussion

This is the first study in a clinical sample followed for an extended interval to document that changes in normal personality traits are associated with concurrent changes in BPD symptoms. Specifically, in addition to the well-established cross-sectional associations between BPD symptoms and normal traits (e.g., Samuel & Widiger, 2008), this study showed that reduction in BPD symptoms is robustly associated with decreasing Neuroticism and increasing Agreeableness, and Conscientiousness. Less consistent longitudinal associations were also observed between decreases is BPD and increases in Extraversion. Of interest is that the longitudinal associations observed in this study were appreciably larger than those observed in meta-analytic research on cross-sectional associations, despite the

[3]Some methodologists have noted that covariation among intercept and slopes can reflect regression to the mean, and therefore correlations among slopes across variables merely reflect co-regression to the mean as opposed to substantively interesting processes. To guard against this interpretation, we reran all models regressing out intercepts from slope factors to test their robustness to regression to the mean effects. All significant slope correlations remained significant in these models, lending confidence to our interpretations of capturing meaningful processes of linked change across BPD and traits.

fact that this study employed different methods for the assessment of traits and BPD (i.e., self-report and diagnostic interview, respectively). These data provide strong and novel evidence for a critical assumption of the view that normative traits underlie phenotypic variance in BPD, which is instantiated in the proposed transition to a dimensional model for PD assessment in Section III of the DSM-5.

The findings from this study suggest several areas for further research. Of interest, particularly in light of the DSM-5 Section III "hybrid" model, is the degree to which traits can fully account for BPD symptomatology. Although traits demonstrate negative outcomes in their own right (Cuipers et al., 2010; Ozer & Benet-Martinez, 2006; Roberts et al., 2007), some research suggests that traits and symptoms provide incremental information about clinical outcomes (Morey et al., 2012). Furthermore, findings from this study that the rates of change in BPD are relatively steeper (i.e., less stable on average) than for normal traits (see Figure 3) parallel recent research suggesting that PD symptoms are also less rank-order stable than traits (Hopwood et al., 2013). Evidence for the differential mean-level and rank-order stability as well as incremental validity of BPD symptoms and associated personality traits supports the DSM-5 "hybrid" proposal to conceptualize BPD using both traits and indicators of symptomatic dysfunction (Skodol, 2012).

However, it is also possible that the observed differential stability and incremental validity is due to assessment method differences rather than assessment content differences *per se*, suggesting that traits fully account for variation in BPD symptoms. If so, hybrid models may be unnecessarily complex. Relatedly, recent research suggests that measures intended to capture general personality impairment provide modest incremental validity over basic (Bastiaansen et al., 2013) or pathological traits (Berghuis et al., 2014; Few et al., 2013; Hopwood et al., 2012) when predicting BPD symptoms. At the same time, the extant research is limited by shared method variance (Bastiaansen et al., 2013; Berghuis et al., 2014; Hopwood et al., 2012) or poor reliability of measurement (Few et al., 2013) beckoning further research. Further demonstrations of the clinical relevance in unshared variance in traits would bolster the justification for mutual consideration of BPD symptoms and trait ratings in the diagnostic manual.

Two specific directions would be useful for future research on this issue. Both of these directions have to do with differences between the trait instrument used in this study and the DSM-5 trait model (which also conforms to a five-factor structure; Gore & Widiger, 2013; Thomas et al., 2013; Wright et al., 2012). First, whereas the DSM-5 model focuses on pathological traits, the NEO-FFI measures traits in the normal range. One would expect stronger correlations with DSM-5 traits than with a measure of normal range FFM traits. Second, like some other FFM measures but unlike the NEO-FFI, the DSM-5 model includes both higher order domains and lower order facets. The lack of facet markers in this study precluded analyses of the specific components of broad FFM traits that track with BPD over time. This is particularly important given that facet-level correlations with BPD vary within FFM traits (Samuel & Widiger, 2008), and because the DSM-5 Section III model BPD is conceptualized using facets, not domains. A related and complicating issue for future studies is that, unlike the notable consensus across trait theories in terms of the domains that

describe personality, there is greater variability across trait models in terms of the composition of facets.

Second, Neuroticism, which is the trait that is most strongly associated with BPD symptoms, reflects a common risk factor for or associated feature of psychiatric disorders in general as opposed to a specific indicator of BPD (Lahey, 2009). More generally, meta-analytic evidence suggests that most personality disorders (Samuel & Widiger, 2008) and other forms of psychopathology are positively correlated with Neuroticism and negatively correlated with Extraversion, Agreeableness, and Conscientiousness (Kotov et al., 2010). These findings imply that the results of this study are unlikely to be specific to BPD, and are likely to extend to a broad range of personality disorders and clinical syndromes that are associated with these basic domains of personality (e.g., major depression, anxiety, substance abuse, etc.). For instance, changes in Neuroticism and Extraversion are associated with not only changes in BPD symptoms as observed here, but have also been shown to relate to changes in avoidant PD symptoms (Wright et al., 2013). Thus, extrapolating beyond these findings suggests that changes or maturation in personality domains undoubtedly reflect more general processes that cut across diagnoses, and have implications for general patterns of psychopathology development and remission.

Furthermore, it is well known that BPD is strongly associated (i.e., “comorbid”) with the other personality disorders listed in the DSM. This has led some investigators to conclude that BPD features “comprise the traits that define the universe of PDs” (Turkheimer et al., 2008; p. 1615). Indeed, some theorists treat borderline pathology as the core dimension of personality pathology (Kernberg, 1984); BPD is also strongly associated with a range of other internalizing and externalizing disorders (Eaton et al., 2012; Kotov et al., 2011; Sharp et al., 2014). Accordingly, these patterns of trait change *and* symptom change may reflect general processes of increasing mental health. Overall, future research is needed to articulate the cross-sectional and longitudinal boundaries between normal traits, BPD, and other disorders, toward an optimally efficient but comprehensive diagnostic system.

Third, basic personality research suggests a role for both heritable and contextual factors in both the stability and change in personality traits (Hopwood et al., 2011), raising several possibilities regarding the mechanism of the longitudinal associations between normal traits and BPD symptoms observed in this study. For instance, changes may relate to genetically disposed endogenous personality maturation, the effects of environmental influences such as psychotherapy or stabilization of social supports, epigenetic factors, some combination of the above, or other influences. In this study we controlled for the level of psychiatric treatment between assessments, and while BPD was negatively associated with treatment between assessments, this clearly did not fully account for change. However, there are likely nuances in this variable that were missed given the nature of our measurement approach, as well as a host of other unmeasured but potentially important environmental factors. Moving forward it would be useful to employ sensitive designs that could establish the degree to which linked changes in traits and BPD symptoms are mediated by heritable factors, life events, or other environmental influences.

Finally, research is needed to address specific limitations of the current study, such as the use of a brief measure of normal traits that may have led, for instance, to an attenuated cross-sectional association between BPD symptoms and Conscientiousness relative to what has been observed in previous studies (see Samuel & Widiger, 2008, for an empirical review of both trait-BPD associations and the moderating impacts of measurement tool on these associations). This may especially be the case because the NEO-FFI items for the Conscientiousness domain do not sample equally from all facets, with a notable absence of Deliberation items, which clinicians and researchers both indicate is highly prototypical of the construct (Lynam & Widiger, 2001; Samuel & Widiger, 2004). The use of a brief measure also precluded a facet-level analysis of trait-BPD relations as described above. Another limitation had to do with the somewhat small sample size relative to what is needed for complex statistical modeling. For instance, this issue made it difficult to evaluate the incremental value of different traits, such as the degree to which changes in traits other than Neuroticism relate to changes in BPD with Neuroticism controlled. Indeed, the correlation between Extraversion and Neuroticism may explain the association between Extraversion and BPD in this study, which runs somewhat counter to meta-analytic research. Future research with larger samples is needed to more carefully evaluate the specific longitudinal associations between normal traits and BPD. Finally, our use of the DIB-R to conceptualize BPD symptoms in this study raises issues about generalizability to other indicators of BPD. In particular, although the DIB-R is well-validated, it offers a more detailed and stringent conceptualization of BPD than other instruments designed to measure BPD; the degree to which the current results extend to such instruments remains an open question.

In summary, these results provide compelling support for the view that normal personality represents a critical substrate for BPD. One could view these results as suggesting that normal personality traits and BPD symptoms reflect elements of a common underlying system. However, the current state of evidence does not rule out the possibility that other features of temperament or environment are incrementally influential on BPD symptoms (Zanarini & Frankenburg, 2007). In any case, results underscore the value of integrating two large bodies of research that have heretofore been somewhat separate: research from personality psychology on the etiology, course, predictive validity, and measurement of traits, and research from clinical psychology and psychiatry on the etiology, course, predictive validity, and measurement of BPD. Given the weight of existing evidence, eschewing basic models of personality in conceptualizing BPD and other forms of psychopathology in diagnostic taxonomies is difficult to justify on empirical grounds. In contrast, synthesizing these literatures and considering further the links between normal traits and BPD symptoms is likely to lead to a better understanding of personality pathology and, ultimately, better methods for the assessment and treatment of this common and severe condition and likely other forms of psychopathology.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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References

American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, 5th Edition (DSM-5). Washington, DC: APA; 2013.

Anderson J, Snider SK, Sellbom M, Krueger RF, Hopwood CJ. A comparison of the DSM-5 Section II and Section III personality disorder structures. Psychiatry Research. in press.

Bastiaansen L, De Fruyt F, Rossi G, Schotte C, Hofmans J. Personality disorder dysfunction versus traits: Structural and conceptual issues. Personality Disorders: Theory, Research, and Treatment. 2013; 4(4):293.

Berghuis H, Kamphuis JH, Verheul R. Specific Personality Traits and General Personality Dysfunction as Predictors of the Presence and Severity of Personality Disorders in a Clinical Sample. Journal of personality assessment. 2013

Costa, PT., Jr.; McCrae, RR. Professional Manual for the Revised NEO Personality Inventory (NEO-PI-R) and NEO Five-Factor Inventory (NEO-FFI). Odessa, FL: Psychological Assessment Resources; 1992.

Cuipers P, Smit F, Penninx BW, de Graaf R, ten Have M, Beekman AT. Economic costs of neuroticism: A population-based study. Archives of General Psychiatry. 67:1086–1093.

Distel MA, Trull TJ, Willemsen G, Vink JM, Derom CA, Lynskey MM, Nicholas G, Boomsma DI. The five-factor model of personality and borderline personality disorder: A genetic analysis of comorbidity. Biological Psychiatry. 2009; 66:1131–1138. [PubMed: 19748081]

Eaton NR, Krueger RF, Keyes KM, Skodol AE, Markon KE, Grant BF, Hasin DS. Borderline personality disorder co-morbidity: Relationship to the internalizing-externalizing structure of common mental disorders. Psychological medicine. 2011; 41(5):1041–1050. [PubMed: 20836905]

Few LR, Miller JD, Rothbaum AO, Meller S, Maples J, Terry DP, Collins B, MacKillop J. Examination of the section III DSM-5 diagnostic system for personality disorders in an outpatient clinical sample. Journal of Abnormal Psychology. 2013; 122:1057–1069. [PubMed: 24364607]

Fossati A, Krueger RF, Markon KE, Borroni S, Maffei C. Reliability and Validity of the Personality Inventory for DSM-5 (PID-5): Predicting DSM-IV Personality Disorders and Psychopathy in Community-Dwelling Italian Adults. Assessment. in press.

Hopwood CJ, Donnellan MB, Blonigen DM, Krueger RF, McGue M, Iacono WG, Burt SA. Genetic and environmental influences on personality trait stability and growth during the transition to adulthood: A three wave longitudinal study. Journal of Personality and Social Psychology. 2011; 100:545–556. [PubMed: 21244174]

Hopwood CJ, Donnellan MB, Zanarini MC. Temperamental and acute symptoms of borderline personality disorder: associations with normal personality traits and dynamic relations over time. Psychological Medicine. 2010; 40:1871–1878. [PubMed: 20018125]

Hopwood CJ, Morey LC, Donnellan MB, Samuel DB, Grilo CM, McGlashan TH, Shea MT, Zanarini MC, Gunderson JG, Skodol AE. Ten year rank order stability of personality traits and disorders in a clinical sample. Journal of Personality. 2013; 81:335–344. [PubMed: 22812532]

Hopwood CJ, Thomas KM, Markon KE, Wright AGC, Krueger RF. DSM-5 traits and DSM-IV personality disorders. Journal of Abnormal Psychology. 2012; 121:424–432. [PubMed: 22250660]

Kernberg, OF. Severe personality disorders. New Haven, CT: Yale University Press; 1984.

Kotov R, Gamez W, Schmidt F, Watson D. Linking 'big' personality traits to anxiety, depressive, and substance use disorders: A meta-analysis. Psychological Bulletin. 2010; 136:768–821. [PubMed: 20804236]

Kotov R, Ruggero CJ, Krueger RF, Watson D, Yuan Q, Zimmerman M. New dimensions in the quantitative classification of mental illness. Archives of General Psychiatry. 2011; 68(10):1003–1011. [PubMed: 21969458]

Krueger RF, Derringer J, Markon KE, Watson D, Skodol AE. Initial construction of a maladaptive personality trait model and inventory for DSM-5. Psychological Medicine. 2012; 42:1879–1890. [PubMed: 22153017]

Lahey BB. Public health significance of neuroticism. American Psychologist. 2009; 64:241–256. [PubMed: 19449983]

Lenzenweger MF, Willett JB. Modeling individual change in personality disorder features as a function of simultaneous individual change in personality dimensions linked to neurobehavioral systems: The longitudinal study of personality disorders. Journal of Abnormal Psychology. 2007; 116:684–700. [PubMed: 18020716]

McGlashan TH. The Chestnut Lodge follow-up study III: Long-term outcome of borderline personalities. Archives of General Psychiatry. 1986; 43:20–30. [PubMed: 3942471]

Miller JD, Morse JQ, Nolf K, Stepp SD, Pilkonis PA. Can DSM-IV borderline personality disorder be assessed via dimensional personality traits? Implications for the DSM-5 personality disorder proposal. Journal of Abnormal Psychology. 2012; 121:944–950. [PubMed: 22428791]

Morey LC, Hopwood CJ, Markowitz JC, Gunderson JG, Grilo CM, McGlashan TH, Shea MT, Yen S, Sanislow CA, Ansell EB, Skodol AE. Comparison of alternative models for personality disorders II: 6-, 8-, and 10-year follow-up. Psychological Medicine. 2012; 42:1705–1713. [PubMed: 22132840]

Morey LC, Skodol AE. Convergence between DSM-IV-TR and DSM-5 diagnostic models for personality disorder: Evaluation of strategies for establishing diagnostic thresholds. Journal of Psychiatric Practice. 2013; 19:179–193. [PubMed: 23653075]

Mullins-Sweatt SN, Edmundson M, Sauer-Savala S, Lynam DR, Miller JD, Widiger TA. .Five-factor measure of borderline personality traits. Journal of Personality Assessment. 2012; 94:475–487. [PubMed: 22489634]

Muthén, LK.; Muthén, BO. Mplus user's guide (6th edition). Los Angeles, CA: Muthén & Muthén; 2010.

Ozer DJ, Benet-Martinez V. Personality and the prediction of consequential outcomes. Annual Review of Psychology. 2006; 57:401–421.

Roberts BW, Kuncel NR, Shiner R, Caspi A, Goldberg LR. The power of personality: Comparative validity of personality traits, socioeconomic status, and cognitive ability for predicting important life outcomes. Perspectives on Psychological Science. 2007; 2:313–345.

Roberts BW, Walton KE, Viechtbauer W. Patterns of mean-level change in personality traits across the life-course: A meta-analysis of longitudinal studies. Psychological Bulletin. 2006; 132:1–25. [PubMed: 16435954]

Samuel DB, Carroll KM, Rounsaville BJ, Ball SA. Personality disorders as maladaptive, extreme variants of normal personality: Borderline personality disorder and neuroticism in a substance using sample. Journal of personality disorders. 2013; 27(5):625–635. [PubMed: 24044664]

Samuel DB, Miller JD, Widiger TA, Lynam DR, Pilkonis PA, Ball SA. Conceptual changes to the definition of borderline personality disorder proposed for DSM-5. Journal of Abnormal Psychology. 2012; 121:467–476. [PubMed: 21875165]

Samuel DB, Widiger TA. A meta-analytic review of the relationships between the five-factor model and DSM-IV-TR personality disorders: A facet level analysis. Clinical Psychology Review. 2008; 28:1326–1342. [PubMed: 18708274]

Sharp C, Wright AGC, Fowler JC, Freuh C, Allen JG, Oldham J, Clark LA. Borderline personality pathology as the 'g' factor of personality disorder. Manuscript submitted for publication. 2014

Skodol AE. Personality disorders in the DSM-5. Annual Review of Clinical Psychology. 2012; 8:317–344.

Trull, TJ.; Brown, WC. Borderline personality disorder: a five-factor perspective. In: Widiger, TA.; Costa, PT., editors. Personality disorders and the five-factor model. 3rd ed.. Washington, DC: American Psychological Association; 2012. p. 119-132.

Trull TJ, Widiger TA, Lynam DR, Costa PT Jr. Borderline personality disorder from the perspective of general personality functioning. Journal of Abnormal Psychology. 112:193–202. [PubMed: 12784828]

Turkheimer E, Ford DC, Oltmanns TF. Regional analysis of self-reported personality disorder criteria. Journal of Personality. 2008; 76:1587–1622. [PubMed: 19012659]

Vachon DD, Lynam DR, Widiger TA, Miller JD, McCrae RR, Costa PT Jr. Basic traits predict the prevalence of personality disorder across the life span: The example of psychopathy. Psychological Science. 2013; 24:698–705. [PubMed: 23528790]

Warner MB, Morey LC, Finch JF, Gunderson JG, Skodol AE, Sanislow CA, Shea MT, McGlashan TH, Grilo CM. The longitudinal relationship of personality traits and disorder. Journal of Abnormal Psychology. 2004; 113:217–227. [PubMed: 15122942]

Widiger, TA. A temperament model of borderline personality disorder. In: Zanarini, M., editor. Borderline personality disorder. Boca Raton, Florida: Taylor & Francis; 2005. p. 63-81.

Widiger TA, Anderson KG. Personality and depression in women. Journal of Affective Disorders. 2003; 74:59–66. [PubMed: 12646299]

Wright AGC, Thomas KM, Hopwood CJ, Markon KE, Pincus AL, Krueger RF. The hierarchical structure of DSM-5 pathological personality traits. Journal of Abnormal Psychology. 2012; 121(4):951–957. [PubMed: 22448740]

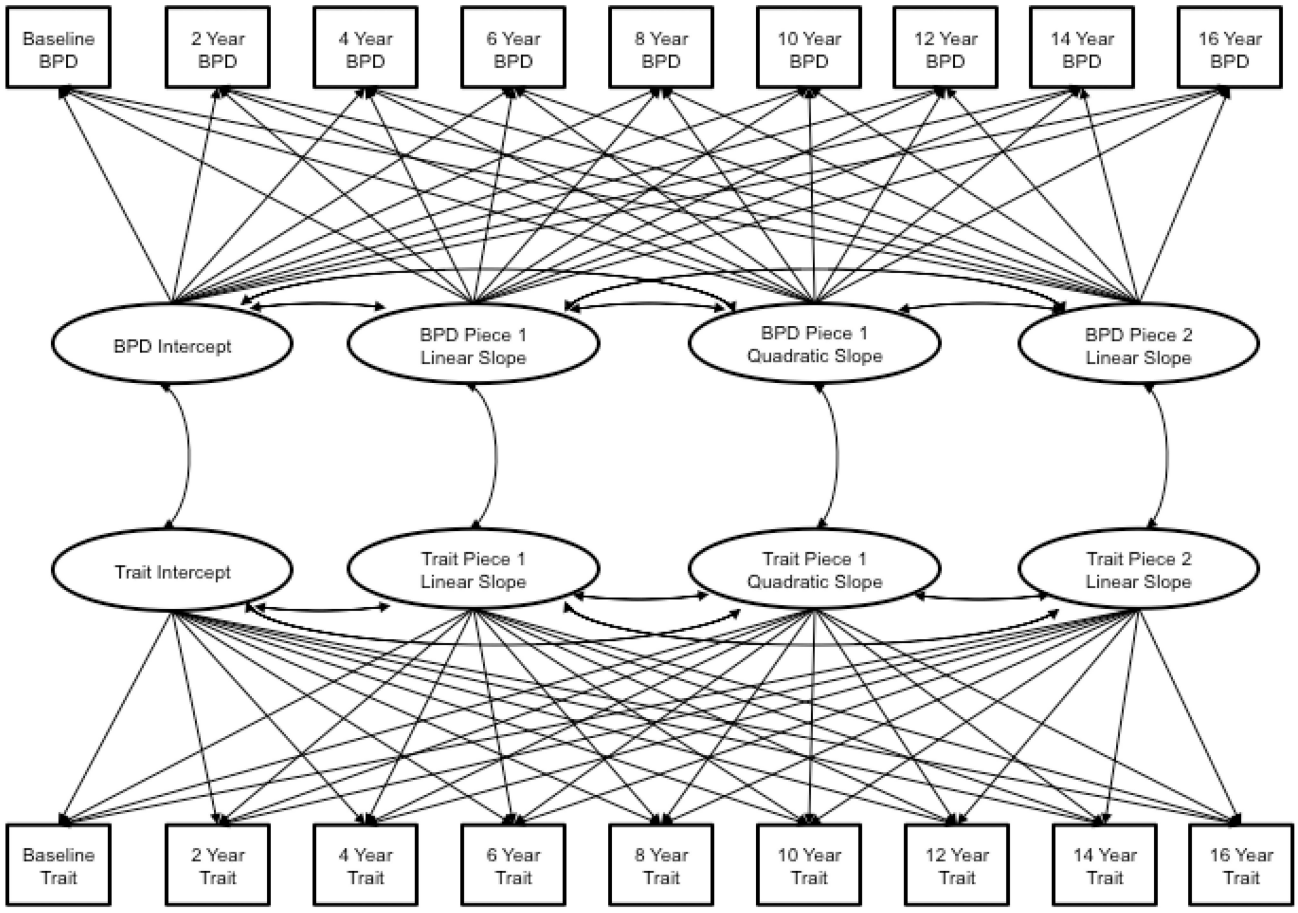
Wright AGC, Pincus AL, Lenzenweger MF. A parallel process growth model of avoidant personality disorder symptoms and personality traits. Personality Disorders: Theory, Research, and Treatment. 2013; 4(3):230–238.

Zanarini MC, Frankenburg FR. Pathways to the development of borderline personality disorder. Journal of Personal Disorders. 1997; 11:93–104.

Zanarini MC, Frankenburg FG, Hennen J, Reich DB, Silk KR. The McLean Study of Adult Development: Overview and implications of the first six years of prospective follow-up. Journal of Personality Disorders. 2005; 19:505–523. [PubMed: 16274279]

Zanarini MC, Frankenburg FR, Reich DB, Fitzmaurice G. Attainment and stability of sustained symptomatic remission and recovery among patients with borderline personality disorder and axis II comparison subjects: a 16-year prospective follow-up study. American Journal of Psychiatry. 2012; 169:476–483. [PubMed: 22737693]

Zanarini MC, Gunderson JG, Frankenburg FR, Chauncey DL. The Revised Diagnostic Interview for Borderlines: Discriminating BPD from other Axis II Disorders. Journal of Personality Disorders. 1989; 3:10–18.



Paths from latent to measured variables were constrained as follows:

Year	0	2	4	6	8	10	12	14	16
Intercept	1	1	1	1	1	1	1	1	1
Piece 1 Linear Slope	0	2	4	6	8	8	8	8	8
Piece 1 Quadratic Slope	0	4	16	36	64	64	64	64	64
Piece 2 Linear Slope	0	0	0	0	0	2	4	6	8

Figure 1.

Piecewise Parallel Process Latent Growth Curve Model of the Longitudinal Relationship between Personality Traits and Borderline Personality Disorder.

Note. Piece 1 = year 0 thru year 8, Piece 2 = year 8 thru year 16. BPD = Borderline Personality Disorder. Growth factor covariances, reflected by curved arrows, were all freely estimated. Although only those covariances of primary interest are depicted here for simplicity and accessibility, study models allowed all growth factors to freely covary.

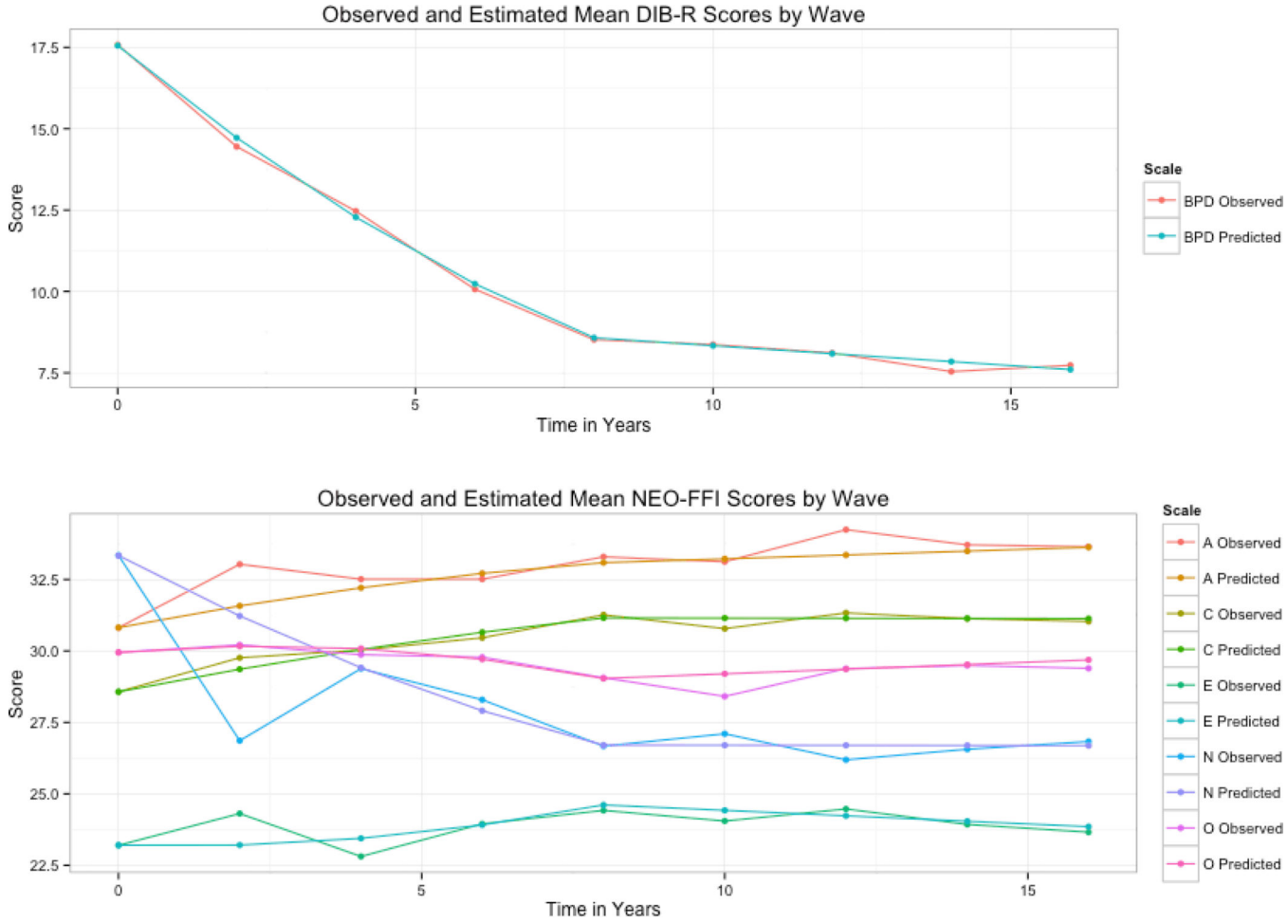


Figure 2.

Observed and estimated study variable means over 16 years.

Note. Data were collected every two years. DIB-R = Diagnostic Interview for Borderline Personality Disorder – Revised; BPD = Borderline personality disorder; NEO-FFI = NEO Five Factor Inventory; A = Agreeableness; C = Conscientiousness; E = Extraversion, N = Neuroticism; O = Openness.

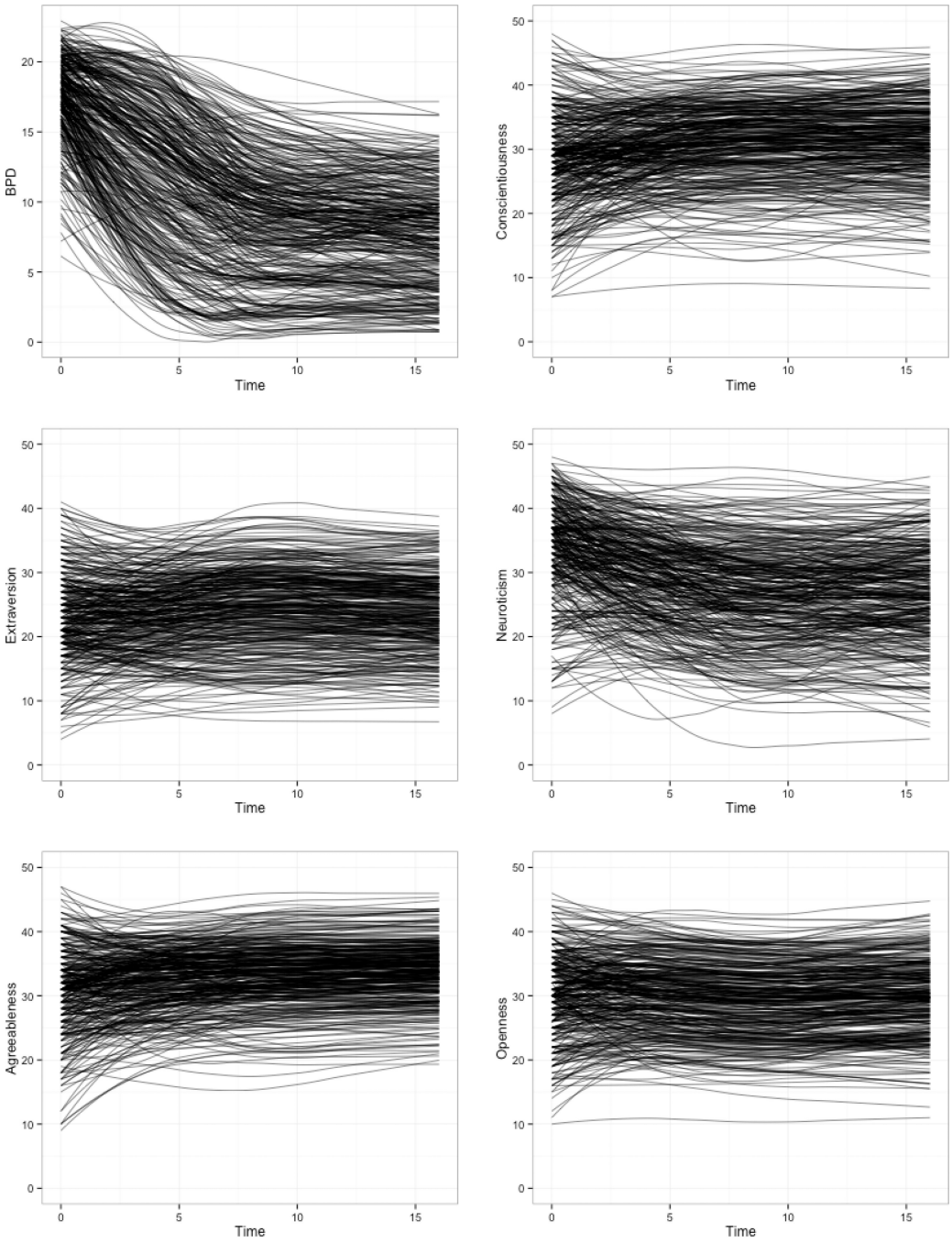


Figure 3.

Plot of individual estimated growth trajectories by study variable.

Note. Individual trajectories reflect Loess smoothed curves based on model estimated values. BPD Scores are on a different scale than NEO-FFI trait scores. Data were collected every 2 years.

Table 1

Parameter Estimates and Fit Indices for Parallel Process Growth Models of Normative Traits and Borderline Personality Disorder.

	Extraversion		Agreeableness		Conscientiousness		Neuroticism		Openness	
	Estimate	**95% CI**	**Estimate**	**95% CI**	**Estimate**	**95% CI**	**Estimate**	**95% CI**	**Estimate**	**95% CI**
Fixed Effects[†]										
BPD I	17.29***	[15.30, 19.29]	17.29***	[15.29, 19.29]	17.26***	[15.26, 19.26]	17.21***	[15.21, 19.22]	17.26***	[15.27, 19.26]
BPD S1	−2.12***	[−3.26, −0.99]	−2.02***	[−3.15, −0.88]	−2.11***	[−3.25, −.98]	−2.18***	[−3.32, −1.04]	−2.13***	[−3.27, −0.99]
BPD Q	0.10	[−0.03, 0.23]	0.09	[−0.04, 0.22]	0.10	[−0.03, 0.23]	0.10	[−0.03, 0.23]	0.10	[−0.03, 0.23]
BPD S2	−0.11	[−0.43, 0.22]	−0.12	[−0.44, 0.20]	−0.13	[−0.45, 0.19]	−0.11	[−0.43, 0.21]	−0.13	[−0.45, 0.20]
Trait I	31.42***	[27.67, 35.17]	27.40***	[23.76, 31.04]	26.01***	[21.81, 30.21]	24.38***	[20.17, 28.60]	30.88***	[27.26, 34.50]
Trait S1	−1.02	[−2.49, 0.45]	0.83	[−0.55, 2.20]	−0.32	[−1.96, 1.32]	−1.85	[−3.90, 0.21]	0.02	[−1.26, 1.30]
Trait Q	0.13	[−0.05, 0.31]	−0.06	[−0.22, 0.11]	0.16	[−0.03, 0.35]	0.14	[−0.11, 0.38]	−0.02	[−0.17, 0.13]
Trait S2	−0.72**	[−1.18, −0.25]	−0.07	[−0.45, 0.31]	−0.28	[−0.75, 0.20]	0.46	[−0.16, 1.09]	0.20	[−0.17, 0.57]
Factor Correlations										
BPD I - BPD S1	.05	[−.31, .41]	.06	[−.31, .43]	.05	[−.31, . 41]	.09	[−.26, .44]	.10	[−.30, .49]
BPD I - BPD Q	−.17	[−.51, .18]	−.18	[−.53, .17]	−.17	[−.51, .17]	−.20	[−.53, .13]	−.21	[−.57, .16]
BPD S1 - BPD Q	−.95***	[−.97, −.94]	−.95***	[−.97, −.93]	−.95***	[−.97, −.94]	−.96***	[−.97, −.94]	−.96***	[−.97, −.94]
BPD I - BPD S2	−.01	[−.24, .23]	.01	[−.23, .26]	.00	[−.23, .24]	−.01	[−.25, .23]	−.01	[−.25, .23]
BPD S1 - BPD S2	−.19	[−.45, .06]	−.19	[−.46, .07]	−.19	[−.44, .07]	−.18	[−.44, .07]	−.19	[−.45, .07]
BPD S2 - BPD Q	.04	[−.22, .30]	.04	[−.23, .31]	.04	[−.23, .30]	.03	[−.23, .30]	.04	[−.23, .31]
Trait I - Trait S1	−.46***	[−.63, −.29]	−.55***	[−.66, −.43]	−.55***	[−.68, −.41]	−.46***	[−.59, −.33]	−.40***	[−.53, −.27]
Trait I - Trait Q	.31**	[.08, .54]	.42***	[.27, .56]	.41***	[.22, .61]	.31***	[.16, .47]	.30**	[.12, .48]
Trait S1 - Trait Q	−.88**	[−.94, −.81]	−.95***	[−.97, −.93]	−.93***	[−.96, −.90]	−.94***	[−.96, −.91]	−.95***	[−.98, −.92]
Trait I - Trait S2	.00	[−.17, .18]	−.12	[−.32, .09]	.02	[−.15, .18]	−.10	[−.26, .05]	−.12	[−.28, .03]
Trait S1 - Trait S2	−.16	[−.48, .15]	−.10	[−.38, .19]	−.03	[−.28, .22]	.10	[−.10, .30]	.11	[−.11, .33]
Trait S2 - Trait Q	−.05	[−.43, .34]	.03	[−.31, .37]	−.11	[−.41, .20]	−.21	[−.43, .00]	−.21	[−.47, .06]
Trait I - BPD I	**−.24****	**[−.37, −.10]**	**−.33*****	**[−.47, −.18]**	**−.15***	**[−.29, −.02]**	**.62*****	**[.48, .77]**	**.07**	**[−.07, .21]**
Trait I - BPD S1	−.11	[−.26, .03]	−.21**	[−.36, −.07]	−.14	[−.28, .01]	.04	[−.11, .19]	−.10	[−.25, .05]
Trait I - BPD Q	.11	[−.05, .26]	.23**	[.08, .38]	.12	[−.03, .27]	−.09	[−.24, .06]	.11	[−.04, .26]

	Extraversion		Agreeableness		Conscientiousness		Neuroticism		Openness	
	Estimate	**95% CI**	**Estimate**	**95% CI**	**Estimate**	**95% CI**	**Estimate**	**95% CI**	**Estimate**	**95% CI**
Trait I - BPD S2	.05	[−.13, .22]	.02	[−.16, .20]	.01	[−.16, .19]	−.12	[−.30, .06]	−.16	[−.33, .01]
Trait S1 - BPD I	.04	[−.20, .28]	.14	[−.05, .33]	.00	[−.21, .21]	−.18	[−.38, .01]	−.15	[−.36, .06]
Trait S1 - BPD S1	**−.17**	**[−.42, .08]**	**−.26***	**[−.46, −.06]**	**−.33****	**[−.54, − .12]**	**.62*****	**[.45, .78]**	**.07**	**[−.15, .29]**
Trait S1 - BPD Q	.18	[−.09, .44]	.26*	[.06, .05]	.36**	[.14, .57]	−.60***	[−.77, −.44]	−.06	[−.29, .17]
Trait S1 - BPD S2	.07	[−.21, .36]	−.12	[−.36, .11]	−.03	[−.28, .23]	.08	[−.15, .31]	.07	[−.17, .32]
Trait Q - BPD I	.03	[−.27, .33]	−.11	[−.33, .11]	.02	[−.25, .29]	.09	[−.13, .31]	.13	[−.13, .39]
Trait Q - BPD S1	.15	[−.17, .47]	.28*	[.05, .51]	.37*	[.10, .64]	−.59***	[−.78, −.40]	.01	[−.27, .28]
Trait Q - BPD Q	**−.22**	**[−.56, .11]**	**−.31***	**[−.55, −.07]**	**−.45****	**[−.73, −.18]**	**.65*****	**[.47, .84]**	**−.04**	**[−.32, .25]**
Trait Q - BPD S2	.03	[−.33, .39]	.17	[−.10, .44]	.16	[−.16, .48]	−.20	[−.46, .06]	−.04	[−.35, .27]
Trait S2 - BPD I	.01	[−.22, .25]	−.08	[−.35, .19]	.01	[−.22, .23]	−.04	[−.25, .17]	−.03	[−.25, .19]
Trait S2 - BPD S1	.00	[−.24, .24]	.22	[−.06, .50]	.07	[−.16, .30]	−.10	[−.31, .12]	−.04	[−.27, .18]
Trait S2 - BPD Q	.11	[−.14, .36]	−.12	[−.41, .18]	.02	[−.22, .26]	−.01	[−.23, .21]	.11	[−.12, .34]
Trait S2 - BPD S2	**−.34***	**[−.62, −.06]**	**−.58****	**[−.91, −.25]**	**−.45****	**[−.72, .18]**	**.72*****	**[.54, .91]**	**−.18**	**[−.47, .10]**
Model Fit Statistics										
df	302		302		302		302		302	
χ^2	534.18		637.84		575.40		588.51		628.69	
RMSEA	.05	[.04, .05]	.06	[.05, .06]	.05	[.04, .06]	.05	[.05, .06]	.06	[.05, .06]
RMSEA *p*	*p* = .84		*p* = .07		*p* = .49		*p* = .37		*p* = .10	
CFI	.96		.95		.96		.96		.95	
TLI	.95		.93		.94		.94		.94	

Note. I = Intercept; S1 = Linear slope for piece 1 of growth; Q = Quadratic slope for piece 1 of growth, S2 = Linear slope for piece 2 of growth. Piece 1 = Year 0 thru year 8, piece 2 = year 8 thru year 16. BPD = Borderline Personality Disorder. *df* = Degrees of Freedom, RMSEA = Root Mean Square Error of Approximation, CI = Confidence Interval, CFI = Comparative Fit Index, TLI = Tucker-Lewis Index, SRMR = Standardized Root Mean Square Residual. Covariates of growth factors included sex and age, and treatment since last measurement was included as a time-varying covariate.

Bolded coefficients reflect parameters of primary interest. All factor variances significant at $p < .001$.

†Fixed effects presented in unstandardized form, factor covariances presented as standardized (i.e., correlations).

*$p < .05$,

**$p < .01$,

***$p < .001$.