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Predicting Individual Change in Personality Disorder Features by Simultaneous Individual Change in Personality Dimensions Linked to Neurobehavioral Systems: The Longitudinal Study of Personality Disorders

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Personality disorders (PDs), long thought to be immutable over time, show considerable evidence of individual change and malleability in modern prospective longitudinal studies. The factors responsible for the evident individual change in PDs over time, however, remain essentially unknown. A neurobehavioral model that posits negative emotion (NEM), nonaffective constraint (CON), communal positive emotion (PEM-C), and agentic positive emotion (PEM-A) as important systems underlying PD provides a theoretical basis for investigating predictors of change in PD features over time. Thus, in this study, the authors investigated how individual change in NEM, CON, PEM-C, and PEM-A over time predicted individual change in PD features over time, using longitudinal data on PD assessed by the International Personality Disorders Examination (A. W. Loranger, 1999), as well as data on normal personality features gathered within a 4-year prospective multiwave longitudinal study ($N = 250$). The authors used the method of latent growth modeling to conduct their analyses. Lower initial levels of PEM-C predicted initial levels of the growth trajectories for those with elevated Cluster A PD features. Elevated NEM, lower CON, and elevated PEM-A initial levels were found to characterize the initial levels of growth trajectories for those with increased Cluster B PD features. Interestingly, subjects with higher initial levels of PEM-A revealed a more rapid rate of change (declining) in Cluster B PD features over time. Elevated NEM and decreased PEM-C initial levels were found to characterize the growth trajectories for subjects with increased Cluster C PD features. The substantive meaning of these results is discussed, and the methodological advantages offered by this statistical approach are also highlighted.

Keywords: personality disorder, personality, negative emotion, nonaffective constraint, longitudinal individual growth trajectories

It has long been assumed that personality disorder (PD) features remain stable over time, within the individual. Indeed, for years, the prevailing diagnostic nomenclature (*Diagnostic and Statistical Manual of Mental Disorders* [*DSM*]; American Psychiatric Association [APA], 1980, 1987, 1994) embraced this assumption in asserting that PDs, which are relatively prevalent disorders affecting about 10% of the population (Lenzenweger, Lane, Loranger, & Kessler, in press; Lenzenweger, Loranger, Korfine, & Neff, 1997), are "enduring patterns" of individual behavior that are "inflexible" and "stable over time" (APA, 1994, pp. 629–639). However, an emerging corpus of data from modern prospective longitudinal studies has revealed that there is considerable individual change in personality pathology over time and, in addition, that this change differs across individuals whether PDs are conceptualized as dimensions or diagnostic entities (Johnson et al., 2000; Lenzenweger, 1999; Lenzenweger, Johnson, & Willett, 2004; Shea et al., 2002; Zanarini, Frankenburg, Hennen, & Silk, 2003). Lenzenweger (1999), reporting from the Longitudinal Study of Personality Disorders (LSPD), found nontrivial declines in mean levels across PD dimensions over a 4-year study period. However, although these mean level declines were statistically significant, the analytic method (repeated measures analysis of variance) by which they were tested and estimated represented a first pass at the LSPD data and was deemed subsequently to be insufficiently sensitive to individual growth and change in PD. More recently, individual growth curve analysis of the LSPD data (Lenzenweger, Johnson, & Willett, 2004) revealed a decline in the total number of PD features over the study period at the rate of 1.4 features per year (a medium effect size, using criteria established by Rosenthal & Rosnow, 1991). Moreover, statistically significant average annual declines in PD features were observed on measures of all three *DSM* PD clusters: The annual average declines in PD cluster feature counts were 0.35 for Cluster A, 0.65 for Cluster B, and 0.40 for Cluster C. At the individual level, these investigators observed statistically significant rates of decline on 9 of the 11 PDs; antisocial PD showed a statistically significant increase over time, whereas there was little change in paranoid PD (Lenzenweger, Johnson, & Willett, 2004). A particularly remarkable feature evi-

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dent in the LSPD data on PD was the degree of heterogeneity among the individual growth curves across subjects on each of the various PDs. It is important to note that this interindividual heterogeneity suggested that change was not uniform across all individuals within each specific PD feature (i.e., despite an overall mean trend that may be a decline, some subjects showed declines, whereas others showed increases or no change), and it was suggested that this interindividual variability in change should be explored to identify its determinants. Additional evidence for change (generally declines) in PD features over time has been reported subsequently. For example, also using a nonclinical community sample, the Children in the Community study directed by Cohen (Cohen, Crawford, Johnson, & Kasen, 2005) reported 28% to 48% reductions in PD symptoms (continuous dimensional count format) over time across the various individual PDs (Johnson et al., 2000). Additionally, Johnson et al. reported substantial and significant declines in the Cluster A, Cluster B, and Cluster C composite PD feature counts as well as in the total PD feature count index for the Children in the Community data.

Two studies that studied specific subsets of PDs, or a single PD, in clinical populations also reported substantial declines in the level of PD features over time. In a study that focused on four of the *DSM* PDs (schizotypal, borderline, avoidant, and obsessive–compulsive PDs), the Collaborative Longitudinal Study of Personality Disorders, Shea et al. (2002) found that 66% of their patients dropped below diagnostic thresholds after 1 year. Moreover, treating the PD feature counts for the four studied PDs as continuous variables, Shea et al. used repeated measures analysis of variance to demonstrate highly statistically significant and substantial mean level declines for all four PDs assessed over a 2-year time span. Also, in the McLean Study of Adult Development, which focuses solely on borderline PD, Zanarini and colleagues (2003) found that nearly 75% of a sample of individuals diagnosed initially with borderline PD (implying relatively high levels of borderline PD features when considered dimensionally) no longer met the diagnostic threshold for the disorder at a 6-year assessment. Although data on mean level changes were not reported for the borderline PD symptom dimension, it is clear that substantial borderline PD symptom declines must have occurred in order that nearly three quarters of the sample was considered remitted at 6-year follow-up.

Clearly, the overall picture that emerges from these four major prospective, multiwave longitudinal studies is one of change in levels of PD over time. On average, PD features tend to decline over time, whether one considers community-based subjects (LSPD, Children in the Community) or clinic/hospital subjects (Collaborative Longitudinal Study of Personality Disorders, McLean Study of Adult Development). This finding, which is important to take note of, flies in the face of nearly 100 years of clinical impressions and teaching in both psychiatry and clinical psychology. In short, it has been assumed heretofore that PD features are set firmly in place early on in life and are relatively fixed, in most cases, and such inflexibility and resistance to change has always been described as a defining aspect of this class of psychopathology. However, the empirical picture that has emerged over the past several years from the consideration of both trends in group means and individual growth trajectories does not support this assumption of enduring stability.

At the same time, it is important to emphasize that there are other aspects of stability beyond the stability of group means over time, such as rank-order stability or the stability of individual differences over time. In this context, it is worth noting that relatively high levels of rank-order stability from wave to wave were reported for PD features for three of the four available longitudinal studies (Johnson et al., 2000; Lenzenweger, 1999; Shea et al., 2002). This suggests that interindividual differences in PD features tended to maintain themselves over time. Despite this, the declines in mean levels of the PD symptom dimensions observed across these same three studies (and implied by the Zanarini et al., 2003, study) reveal a certain plasticity to PD that has been previously unknown. This plasticity and change has been most clearly and vividly demonstrated in a study of individual growth trajectories in PDs (e.g., Lenzenweger, Johnson, & Willett, 2004) that illustrated the use of a statistical approach to longitudinal data analysis that transcends more traditional group-based analyses of means and level stability. Clearly, there is evidence of considerable change in PDs over time, and so the task confronting longitudinal research on PDs is to explore the nature of this change process. In short, what must now be determined is the mechanism that is driving these changes, and the interindividual differences among them, in PDs over time.

One could consider any number of potential predictors of change in PD symptomatology over time, however one immediate research vector presents itself, namely personality and those processes subserving personality. Thus, one possible mechanism that might account for the change could be those underlying personality processes that themselves show plasticity over time. Indeed, recent models linking underlying neurobehavioral processes, personality, and PD suggest a potentially fruitful avenue for exploration as such models have specified clear links between personality systems and individual PDs (Depue & Lenzenweger, 2001, 2005, 2006; cf. Cloninger, Svrakic, & Przybeck, 1993). In recent theoretical work, we (Depue & Lenzenweger, 2001, 2005, 2006) proposed a neurobehavioral model of PDs that emphasizes interactive neurobiologically mediated systems that impact both normal personality and PD.[1] This model links well-established underlying

[1] We are mindful of other approaches to describing the relationship between personality disorders and either normal personality features (e.g., five-factor model; Saulsman & Page, 2004) or Axis I psychopathological conditions (Siever & Davis, 1991). However, this is not the forum for extended discussion of the relative merits of alternative substantive viewpoints. We note, briefly, that the Depue–Lenzenweger model (DLM) (Depue & Lenzenweger, 2001, 2005, 2006) of neurobehavioral systems differs from the popular five-factor model (FFM) of personality trait dimensions in critical ways. The DLM model separates agentic and affiliative components of extraversion, whereas the FFM combines them; the DLM separates anxiety and fear into distinct systems, whereas the FFM combines them; and the DLM emphasizes a nonaffective constraint system, whereas such a system is not represented in the FFM (conscientiousness is not nonaffective constraint). The DLM emerged from a consideration of the neurobiological and theoretical literature on motivation, affect, and behavior, whereas the FFM emerged from factor analytic studies of language-based/lexical trait descriptors. The DLM also differs substantially from the Cloninger model (Cloninger et al., 1993) in that the primary temperament components of the Cloninger model represent heterogeneous blends of underlying personality dimensions (see Waller, Lilienfeld, Tellegen, & Lykken, 1991), whereas the DLM model emphasizes relatively homogenous dimensions corresponding to underlying neurobehavioral systems. Interested readers are referred to Depue and Lenzenweger (2001, 2005, 2006) for additional details.

neurobehavioral systems with higher order personality trait processes and specifies interactions among the underlying neurobehavioral systems that will manifest ultimately, given certain configurations, in PD (Depue & Lenzenweger, 2001, 2005, 2006). In short, Depue and Lenzenweger (2005) argued that "the higher-order traits of personality, which are general and few, most likely reflect the activity of the few, general neurobehavioral systems" (p. 396), and this position was based on an extensive review of the relevant animal and human neurobiological literature. The primary neurobehavioral systems identified in this model are (a) positive incentive motivation (a reward-based behavioral approach system), (b) affiliative reward (for establishment and maintenance of social closeness/bonds), (c) anxiety (for assessment of the risk of danger), (d) fear (for escape from unconditioned aversive stimuli), and (e) neural constraint (a tonic inhibitory influence on behavioral responding). These systems are manifested respectively in the following personality traits: (a) agentic extraversion (agentic positive emotion; PEM-A), (b) affiliation (social closeness or communal positive emotion; PEM-C), (c) negative emotion (NEM), (d) fear (often referred to as *harm avoidance*), and (e) nonaffective constraint (CON). According to Depue and Lenzenweger (2005), the positive incentive motivation system is mediated largely by dopamine; the affiliative reward system is mediated by a complex interaction of vasopressin, oxytocin, and the endogenous opiates; the anxiety system is mediated in part by corticotrophin-releasing hormone and tonic norepinephrine activity; the fear system is mediated by phasic norepinephrine activity; and the neural constraint system is mediated largely by serotonin (5-HT). An important assumption of the model is that individual differences in the underlying neurobehavioral systems will be reflected in individual differences in the higher order personality traits deriving from these underlying systems. To be clear, the model does maintain that neurobiological substrates underpin and, by implication, shape what is known as personality (or temperament). Moreover, and this is important to note, the underlying neurobehavioral processes can configure interactivity to create the substrate basis for PD. Thus, in short, Depue and Lenzenweger (2005) have argued that PD or personality disturbance is best viewed as an emergent phenomenon reflective of these interacting neurobehavioral processes that are represented phenotypically by the personality dimensions associated with the constructs NEM, CON, PEM-A, and PEM-C.[2]

The Depue and Lenzenweger (2001, 2005, 2006) model provides a substantive basis for exploring the relationship between personality and PD; however, one must consider the nature of personality from a longitudinal perspective if one is to examine change in PD as a function of change in personality over time. Is normal personality stable over time? Does it make sense to posit that change can occur in normal personality systems? Although there is some evidence for personality constructs, such as those described in the Depue–Lenzenweger model, to maintain rank-order stability over time (see Roberts & DelVecchio, 2000, for an excellent review), the overall picture that has emerged from contemporary reviews of the stability of normal personality is one that suggests change (or plasticity) over time, particularly in alterations of mean levels of personality trait dimensions (see Roberts, Walton, & Viechtbauer, 2006, for an extended review). In fact, the patterns of change in mean levels of well-known personality dimensions were extensive (in four of six trait categories) and substantial according the Roberts et al. (2006) meta-analysis. The Roberts et al. (2006) review is particularly relevant to questions regarding change in normal personality for the subject sample under consideration in this study, namely persons between the ages of 18 and 22. Roberts et al. (2006) found that the normal personality constructs of social vitality (an extraversion facet), social dominance (an extraversion facet), emotional stability, and openness to experience all revealed statistically significant change effects for the population parameters estimated. These constructs, derived from a multitude of personality measures, bear some resemblance to aspects of the core constructs in the Depue–Lenzenweger model described previously. Furthermore, considering studies that specifically examined normal personality constructs that are highly similar to those we have discussed, the picture that emerges is also one of change over time. Indeed, four studies that explicitly examined NEM, CON, and the facets of PEM-A (achievement, social potency) and PEM-C (well-being, social closeness) showed evidence of change over time in these constructs, particularly during the early adulthood years (Blonigen, Carlson, Hicks, Krueger, & Iacono, in press; Donnellan, Conger, & Burzette, 2007; McGue, Bacon, & Lykken, 1993; Roberts, Caspi, & Moffitt, 2001; see also Roberts et al., 2006). It is plausible, therefore, to assume that individual change in the NEM, CON, PEM-A, and PEM-C systems occurs over time, as indexed by psychometric measures of these personality traits, and that these changes may be related to individual change in PD features over time. This is the broad research question that we address in the current article.

A necessary and logical step in addressing this question is to devise an analytic approach that simultaneously captures, in a fine-grained manner, individual growth in measures of the NEM, CON, PEM-A, and PEM-C over time and relates those changes to individual change in PD features over time. In this study, we used longitudinal data on PD symptomatology and on selected neurobehavioral systems indicator variables to test our hypotheses about how individual change in NEM, CON, PEM-A, and PEM-C impacts individual change in PD features over time. As our hypotheses concerned potential relationships among individual change over time in both our outcome and our predictors, in our analyses we had to simultaneously model change over time in PDs and in the interactive neurobiologically mediated systems—NEM, CON, PEM-A, and PEM-C—that we believe impact that change in PD. We were able to do this by applying latent growth modeling, a recently developed analytic approach that has mapped the methods of individual growth modeling onto the general covariance structure model (Willett & Bub, 2006; Willett & Keiley, 2000).

Our application of latent growth modeling is relatively new in psychopathology research and builds directly on more traditional methods of analyzing longitudinal data. It is an extension of earlier work on the analysis of individual growth, in which multilevel modeling was used to investigate systematic interindividual differences in growth by examining the relationships between time-invariant predictors, such as a subject's gender, and an individual's

[2] Depue and Lenzenweger (2001, 2005, 2006) discussed the roles played by fear and aggression in the development of some PDs, however the model focuses principally on agentic extraversion, affiliation, negative emotion (anxiety), and nonaffective constraint.

change in an outcome, like PDs, over time. Under the standard individual growth modeling approach, individual change over time in PD outcome is described as a function of time in a "Level 1" growth model. With limited numbers of waves of longitudinal data available for analysis, this latter model often describes only linear growth over time and contains an intercept parameter to represent the individual's initial status and a slope parameter to represent his or her linear rate of change. These individual growth parameters then stand in place of the change in subsequent interindividual analyses. Thus, a Level 2 model can then be specified in which the Level 1 parameters (i.e., individual initial status and rate of change) are related to the hypothesized time-invariant predictors of change, like subject gender. Both models are fit simultaneously to longitudinal data, usually by maximum likelihood methods. By testing and interpreting the Level 2 parameter estimates, the investigator can detect and assess systematic interindividual differences in change that are present, perhaps concluding that the initial status and rate of change in PD differ for men and women and, consequently, that the personality growth trajectory differs by gender in the population (Singer and Willett, 2003, provides an extensive description of the approach). Our current application of latent growth modeling is a direct extension of these earlier methods and differs only in that our approach permits the concurrent specification of multiple Level 1 growth models to describe change in both the predictors and the outcome simultaneously, rather than requiring the predictors to be time invariant. Consequently, at Level 2 we can examine potential relationships among the multiple kinds of change.

Other analytic approaches have been suggested and successfully implemented in longitudinal data like ours. One approach introduces the hypothesized time-varying predictors—such as measures of personality dimensions—directly into the Level 1 individual growth model itself, along with the time predictor. Although this is a feasible way of handing time-varying covariates analytically, it addresses a different research question from ours, in which the relationship between the time-varying levels of outcome and predictor is examined concurrently over occasions (thereby asking "Is PD symptomatology related to measures of personality, controlling for time?"), rather than the relationship that we have examined here between concurrent changes in both outcome and predictor. A second approach uses path analysis to investigate lagged pairwise relationships between the levels of the time-varying predictors on one occasion and the level of the outcome on a subsequent occasion, over multiple waves of data (this is a path-analytic extension of the now mostly unused and largely discredited cross-lagged correlation approach). Again, although this kind of analysis can succeed in detecting the presence of multiple simultaneous pairwise relationships and examining their persistence over time, it cannot address a research question about change over time or about hypothesized links between simultaneous changes in both outcome and predictors, as does the method we implemented.

In the present study, therefore, we sought to determine, using prospective multiwave data from the LSPD (Lenzenweger, 1999, 2006; Lenzenweger, Johnson, & Willett, 2004; Lenzenweger et al., 1997), whether individual change in PEM-A, PEM-C, NEM, and CON was related to individual change in PD features over time. For the purposes of this study, we defined our outcome as the total number of Cluster A, Cluster B, or Cluster C PD features. The cluster taxonomy is the well-known and easily appreciated clustering of PDs proposed in the *DSM* system. To address our research questions, we modeled individual change simultaneously in PEM-A, PEM-C, NEM, CON, and the PD cluster features using latent growth modeling and examined the prediction of individual change in the latter outcome variable by the individual changes in PEM-A, PEM-C, NEM, and CON.

Method

Longitudinal Study of PD Data Set

The LSPD (Lenzenweger, 1999, 2006; Lenzenweger, Johnson, & Willett, 2004), begun in 1991 as the first longitudinal study of PDs sponsored by the National Institute of Mental Health, has a prospective multiwave longitudinal design with subjects evaluated at three points in time, corresponding to their 1st, 2nd, and 4th years in college. Interview assessments for PD (i.e., Axis II) and Axis I disorders are conducted at each of the three assessment waves by PhD or advanced MSW clinicians with extensive Axis II diagnostic experience (Lenzenweger et al., 1997). Finally, as the LSPD is a naturalistic prospective study, subjects are free to seek psychological treatment of their own accord. Extensive detail regarding the LSPD, including reliability assessments and other technical matters, is given in prior publications, and the interested reader is referred to those articles (Lenzenweger, 1999, 2006; Lenzenweger et al., 1997).

Subjects

The 258 subjects in the LSPD were drawn initially from a population consisting of 2,000 first-year undergraduate students (Lenzenweger, 1999; Lenzenweger, Johnson, & Willett, 2004; Lenzenweger et al., 1997). The subjects were recruited from both the private and public (State University of New York) colleges at Cornell University in Ithaca, New York, which helped to ensure diversity across sociodemographic background variables. All subjects gave voluntary written informed consent and received an honorarium of $50 at each wave of data collection. Extensive detail concerning the initial subject selection procedure and sampling is given elsewhere (Lenzenweger, 1999; Lenzenweger et al., 1997) and is not repeated here. Of the initial 258 subjects, 250 completed all three assessment waves and are included in our current analysis. We summarize their selected characteristics in Table 1.

Initial Screening Measure: International Personality Disorder Examination (IPDE) DSM-III-R Screen (IPDE-S)

The IPDE-S is a 250-item self-administered true/false PD screening inventory developed by Armand W. Loranger. The diagnostic efficiency and psychometric properties of the IPDE-S, in a two-stage screen application, are generally excellent and have been described previously (Lenzenweger et al., 1997). The grouping variable for the study subjects described here was based on this measure and was thus retained in the analyses we report; specific scores from the IPDE-S were not used in the analyses reported here.

Table 1
Descriptive Statistics for Selected Demographic Characteristics in the Longitudinal Study of Personality Disorders Sample (N = 250)

Variable	Father (%)	Mother (%)	Participant (%)
Parental education			
1–8 years	1.6	0.8	
9–11 years	2.4	2.8	
12 years	8.4	15.2	
13–15 years	16.0	20.4	
16+ years	68.8	58.8	
Not available	2.8	2.0	
Parental occupation			
Laborer/service	2.0	2.4	
Operatives (machine)	2.8	1.2	
Craftsman/foreman	3.2	1.6	
Clerical/sales	4.0	16.8	
Management/official	26.8	12.8	
Professional/technical	52.4	42.0	
Not available or homemaker	8.8	23.2	
Race			
African American			3.6
Latin/Hispanic			4.8
Caucasian/Anglo			72.0
Asian or Pacific Islander			17.2
Native American			0.8
Other			1.6

Outcome Measure: IPDE

The IPDE is the well-known semistructured interview procedure that assesses both *DSM* and *International Classification of Diseases-10* PD features (Loranger, 1999; Loranger et al., 1994; Loranger, Sartorius, & Janca, 1996) and was used in the International Pilot Study of Personality Disorders sponsored by the World Health Organization and the Alcohol, Drug Abuse, and Mental Health Administration (Loranger et al., 1994, 1996). The *DSM-III-R* criteria were assessed in this study as these were the criteria in effect at the time the LSPD was undertaken. We note that the *DSM-III-R* and the later *DSM-IV* criteria bear considerable resemblance to one another, and the fundamental PD constructs are the same in both nomenclatures. Clinically experienced interviewers received intensive training in IPDE administration and scoring by Dr. Armand W. Loranger and were supervised throughout the project by author Mark F. Lenzenweger, who was blind to the subjects' identity, putative PD status, and all prior assessment information. The interrater reliability for IPDE assessments (based on intraclass correlation coefficients) was excellent at all three waves, ranging between .84 and .92 for all PD dimensions. The interviewers (a) were blind to the putative PD group status of the subjects, (b) were blind to all prior LSPD PD assessment data, and (c) never assessed the same subject more than once.

For the purposes of this study, we defined three separate outcome variables to describe subjects' PD—these were the total cluster scores for each of the well-known *DSM*-based Cluster A, Cluster B, and Cluster C PD domains (APA, 1980, 1987, 1994). Cluster A total scores reflected the total dimensional count for paranoid, schizoid, and schizotypal PD features (i.e., the odd/eccentric PD cluster). Cluster B total scores reflected the total dimensional count for borderline, antisocial, histrionic, and narcissistic PD features (i.e., the erratic/impulsive PD cluster). Cluster C total scores reflected the total dimensional count for avoidant, obsessive–compulsive, dependent, and passive-aggressive PD features (i.e., the anxious/avoidant PD cluster). However, the sample distributions of the Cluster A, B, and C PD feature counts were all heavily positively skewed. This comes as no surprise, as positive skewing is common for psychopathology variables that are formed from counts, particularly when studied in nonclinical community populations. Hence, we followed standard statistical practices for counted variables, taking a square-root transformation of the raw count as the outcome in our latent growth modeling analyses. This reduces the skewness in the raw counts of PD features, linearizes individual growth in PD features over time, and renders the distribution of the residuals in our analysis more hospitable to the required normal theory assumptions. However, we detransformed our findings, reversing this transformation, before plotting and interpreting the findings presented later in this article.

Axis I Diagnostic Measure: Structured Clinical Interview for DSM-III-R: Non-Patient version (SCID-NP; Spitzer, Williams, Gibbon, & First, 1990)

This is a semistructured *DSM-III-R* Axis I clinical interview for use with nonpatients. The clinical interviewers trained on the use of the SCID-NP by using the videotape-based training system provided with the SCID-NP. All interviewers achieved high reliability with criterial cases (all intraclass correlation coefficients $>$ .80 for symptom assessments). The SCID interview was done first, followed by the IPDE as is customary practice for thorough Axis II assessments. The Axis I diagnostic information generated by the SCID is not a primary focus of this report, although we do note the results of some sensitivity analyses that incorporated data from the SCID later in this report.

Predictors

Time. Time was the important Level 1 predictor in our analyses, and it was measured in years from a subject's entry into the study. All subjects contributed three waves of data, being assessed repeatedly on the same schedule, although the time between assessments varied somewhat from case to case due to the practical constraints in the data collection. The average age of subjects at each assessment was 18.78 years ($SD = 0.51$) at the first wave, 19.83 years ($SD = 0.54$) at the second wave, and 21.70 years ($SD = 0.56$) at the third wave. In order to apply the latent growth methodology, we had to impose a common assessment schedule across all subjects, setting the times of measurement to their between-person averages at each wave. From entry into the study, then, the Level 1 predictor time took on values of 0, 0.95 years and 2.82 years at the first, second, and third waves, respectively. Sensitivity analyses—in which we systematically modified the assessment times to reflect extremes in the variation of the assessment schedule over subjects within wave—left our findings unaffected.

Neurobehavioral indicator dimensions. The NEM, CON, PEM-A, and PEM-C dimensions were hypothesized to be reflective of the four primary neurobehavioral systems that Depue and

Lenzenweger (2001, 2005) posited as critical to PD development and maintenance. The PEM-A, PEM-C, NEM, and CON dimensions described by Depue and Lenzenweger (2001, 2005) correspond phenotypically with those identified by Tellegen (1982, 1985; Watson & Tellegen, 1985); however, Depue and Lenzenweger relied heavily on the neurobiological animal and human literature in deriving the neurobehavioral systems thought to underlie these dimensions (see Footnote 1). Although Tellegen's measure for these dimensions was not included in the LSPD at the time of initial data collection, the LSPD did include another personality measure, the original NEO-PI (Costa & McCrae, 1985). Reasonable representations of the PEM-A, PEM-C, NEM, and CON dimensions can be extracted from the NEO-PI dimensions using algorithms derived from the factor analytic work of Church (1994) comparing the NEO-PI and Tellegen's constructs (see Appendix A for technical detail regarding our estimation of the PEM-A, PEM-C, NEM, and CON trait scores). The NEO-PI is the original version of the well-known, Likert-style self-report measure of normal personality traits. The NEO-PI has generally excellent psychometric properties as an assessment instrument, with dimensions showing high internal consistency and reliability over time (Costa & McCrae, 1985). Finally, we note that scores on the four personality trait dimensions were relatively normally distributed at each assessment point and were therefore not transformed prior to analysis.

Covariates That Capture Important Features of the Study Design

Although none of their effects were a focus of the findings reported here, we also included three covariates to control for intrinsic features of the study's sampling and design in all of our analyses. These were the subject's (a) age at entry into the study, (b) assignment to putative PD risk group prior to study onset, and (c) gender. Age at entry was measured in years and was included to account for marginal heterogeneity in the ages of subjects at the beginning of the study. The group variable described subjects' initial assignment (established prior to IPDE data collection) to either a possible personality disorder (PPD) or no personality disorder (NPD) group according to the IPDE-S (Lenzenweger, 1999; Lenzenweger et al., 1997). As detailed in Lenzenweger at al. (1997), PPD subjects met the diagnostic threshold on the IPDE-S for at least one specific *DSM-III-R* PD, whereas NPD subjects (a) did not meet the *DSM-III-R* threshold for diagnosis and (b) had fewer than 10 PD features across all disorders on the screener (see Lenzenweger, 2006, for greater detail). Five PPD and three NPD subjects did not complete all three waves of data collection; two of these noncompleting subjects died during the course of the study, one in each subject group. Control predictor *male* identified the subject as either male or female—the sample contained 121 men (47%) and 137 women (53%). We also included a two-way interaction of the group and gender predictors as a covariate in our analyses.

Statistical Analysis

In order to evaluate the simultaneous impact of individual change in PEM-A, PEM-C, NEM, and CON on individual change in the Cluster A, Cluster B, or Cluster C PD features over time, we used latent growth modeling (Willett & Bub, in press; Willett & Keiley, 2000). Under this approach, we used the Y and X measurement submodels available within the general covariance structure model to specify individual growth models simultaneously for our time-varying outcome and for all four of our time-varying predictors. With only three waves of data on both outcome and predictors for each subject, we were limited in the complexity of our growth specification. Consequently, after preliminary exploratory analysis and the subsequent square-root transformation of the outcome (as described above), we specified linear trajectories to represent change over time in each of the measures. As a result, individual changes in the outcome and each of the predictors are implicitly represented, in each case, by an initial status and a rate of change parameter for each subject. Then, in the structural component of the general covariance structure model, we specified Level 2 relationships among these individual growth parameters in order to articulate our Level 2 hypotheses about the prediction of change in PD features over time by changes in NEM, CON, PEM-A, and PEM-C. We repeated these analyses separately for the three subscores that we used to describe the details of subjects' PD features, namely Cluster A, Cluster B, and Cluster C.

Figure 1 presents a simplified path diagram that displays, for a generic PD features outcome (either Cluster A, Cluster B, or Cluster C), the several hypothesized measurement models and structural model required for conducting our covariance structure analyses. As noted above, the Y and X measurement models on the right and left sides of the path model, respectively, establish Level 1 change trajectories in the outcome and the predictors, and the structural model that occupies the center of the path diagram defines hypothesized Level 2 relationships among the individual growth parameters. Although we included several additional control variables (as described above) in all of our statistical models as predictors of change in PD features, we omitted them from the path diagram in Figure 1 to avoid clutter. We fit the complete path model to our longitudinal data using the LISREL software (Version 8.7; Jöreskog & Sörbom, 2004), separately for each of the three PD outcomes (Cluster A, Cluster B, and Cluster C).[3]

As can be seen in Figure 1, the three available waves of longitudinal data permitted us to specify individual growth models in which predictors NEM, CON, PEM-A, and PEM-C were simultaneously hypothesized to be linear functions of time. This implied the presence, in the path model, of four latent constructs representing the initial levels (or *elevations*, π_1^{NEM}, π_1^{CON},π_1^{PEMA},π_1^{PEMC}) and slopes (or *rates of change*, π_2^{NEM}, π_2^{CON}, π_2^{PEMA}, π_2^{PEMC}) of the growth trajectories for these neurobehavioral indicators. Growth in the square root of total PD features, within each of the A, B, and C clusters, was similarly specified in the latent growth model, yielding another pair of latent constructs that represented the initial level (π_1^{PD}) and rate of change (π_2^{PD}) of the PD features change trajectory in each of the clusters. The latent growth parameters representing change over time in PEM-A, PEM-C, NEM, and CON were then hypothesized to predict the latent growth parameters representing

[3] We also fit full and reduced models without the control predictors (i.e., age at entry, group, gender, and the Group × Gender two-way interaction term).

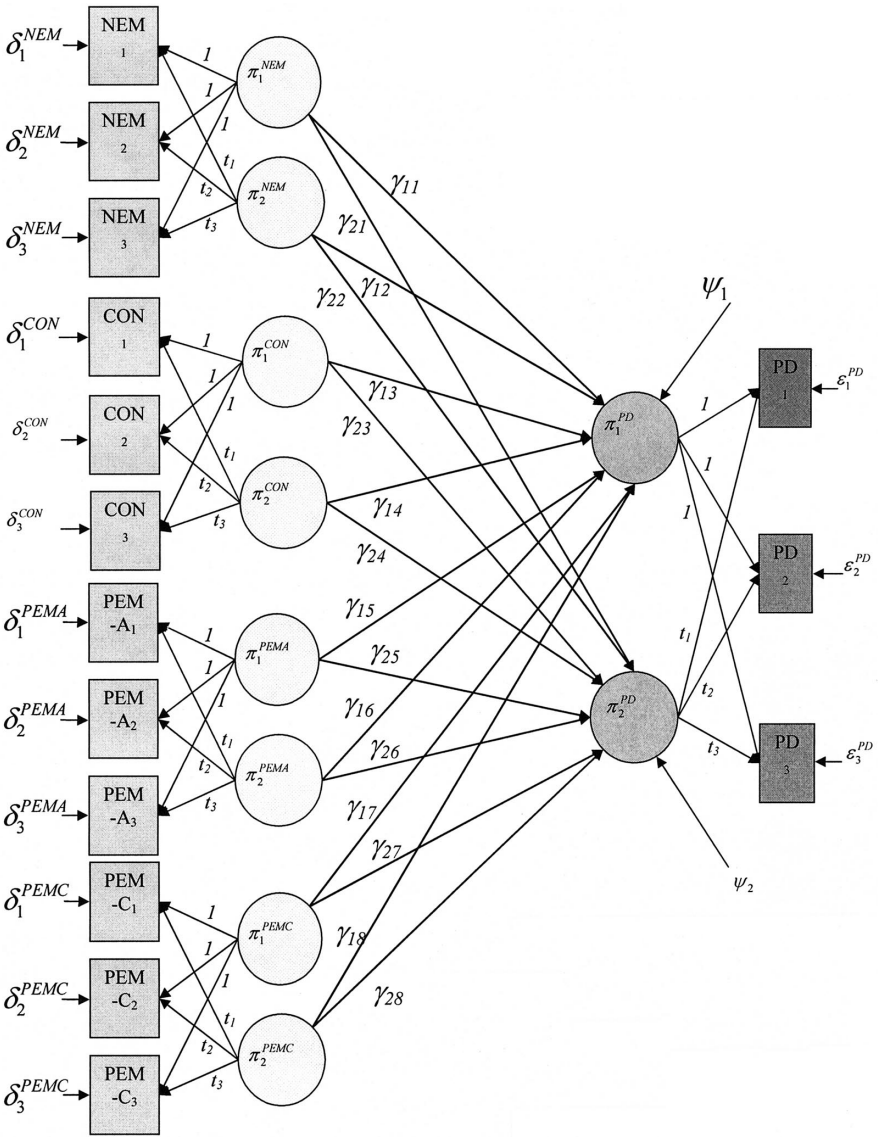

Figure 1. Hypothesized path model depicting relationships among initial status (elevation) and rate of change in four personality indicators of neurobehavioral systems and initial status (elevation) and rate of change in personality disorder (PD) symptomatology over time (control predictors omitted for clarity). NEM = negative emotion; CON = nonaffective constraint; PEM-A = positive emotion–agentic; PEM-C = positive emotion–communal.

change in total PD features in the structural part of the path model, providing 16 structural regression parameters, the γs, that addressed our research questions.

In an initial round of statistical analysis, we fit the full path model specified in Figure 1 (and also including all covariates) for each of our three outcomes (Cluster A PD features, Cluster B PD features, and Cluster C PD features). Then we tested a set of sequential nested hypotheses to obtain a parsimonious final fitted model in which several of the originally hypothesized paths were omitted because they were not required (see Singer & Willett, 2003, for a description of this approach). We provide parameter estimates and associated inferential statistics for both the full and the reduced models later in the article. (A full taxonomy of fitted models is available from Mark F. Lenzenweger on request.) All hypothesized latent growth models were fit assuming that the Level 1 measurement errors over time within each growth process, because supplementary analyses in which the errors were permitted to be heteroscedastic, confirmed that this was reasonable. Finally, we also conducted sensitivity analysis in which we fit models that included two additional predictors of initial level and rate of change in the three PD dimensions, namely the presence or absence of any form of Axis I disorder before or during the study period as well as the presence or absence of any form of psychological/psychiatric treatment before or during the study period, as predictors.

Results

Clinical Characteristics of the Sample

The lifetime *DSM-III-R* Axis I diagnoses for the study subjects have been reported previously (Lenzenweger, 1999; Lenzenweger, Johnson, & Willett, 2004). In overview, 81 (62.8%) of the PPD subjects received a definite or probable Axis I diagnosis compared with 32 (26.4%) NPD subjects ($\Pi^2 = 33.30$, $df = 1$, $p < .00001$). Also, 41 (31.8%) PPD subjects versus 21 (17.4%) NPD subjects reported a prior history of treatment by Wave 3 ($\Pi^2 = 6.97$, $df = 1$, $p < .008$). Finally, at Wave 1, 11% of the subjects qualified for an Axis II diagnosis of some sort. The raw rates of diagnosed PDs in the LSPD sample as of Wave 1 were as follows: paranoid = 1.2%, schizoid = 1.2%, schizotypal = 1.6%, antisocial = 0.8%, borderline = 1.6%, histrionic = 3.5%, narcissistic = 3.1%, obsessive-compulsive = 1.6%, passive-aggressive = 0.8%, avoidant = 1.2%, dependent = 0.8%, and not otherwise specified = 4.3%. Moreover, as of the Wave 3 assessment (i.e., 4 years), 16% of the sample had received a probable or definite diagnosis for at least one Axis II PD (or PD not otherwise specified). Thus, by Wave 3, approximately 1 in every 6 subjects in the sample met formal *DSM-III-R* thresholds for PD diagnoses based on the highly conservative IPDE (Loranger, 1999). When converted to weighted population prevalence rates (see Lenzenweger, 2006), the rates of diagnosed PD accorded well with estimates for the general population of the United States (Lenzenweger et al., in press). Descriptive data for the personality dimension correlations across time are given in Appendix B, Table B1, and group mean (*SD*) data for the personality and PD features are given in Appendix C.

Latent Growth Analyses

Table 2 reports our full and reduced fitted latent growth models for each of the three outcomes (Cluster A, Cluster B, and Cluster C PD features).[4] In the table, we report parameter estimates, approximate *p* values, and selected goodness-of-fit statistics (goodness-of-fit χ^2, Akaike's information criterion, root-mean-square residual, and standardized root-mean-square residual [SRMSR]) for all fitted models. We also report a pseudo-R^2 statistic for the prediction of each PD growth parameter in each model. This latter statistic is akin to the adjusted R^2 statistic, more familiar in regular regression analyses, and estimates the proportion of variance in the relevant PD growth parameter explained simultaneously by all predictors in the particular model. The overall trend for PD growth across all three clusters over the study period was toward a lessening of PD features with the passage of time. We discuss the results for each outcome, separately.

In our final parsimonious (i.e., reduced) model for change in the Cluster A PD features, we found that only the initial status of the PEM-C individual growth trajectory predicted the initial status of the Cluster A PD growth trajectories. No other statistically significant relationships were evident, particularly no relationships among the several rates of change for the personality and Cluster A PD variables. None of the control variables predicted either initial status or rate of change in the Cluster A PD features. Both the full and reduced models provided reasonably good fit to the data (e.g., for both, SRMR $< .08$). The values of the pseudo-R^2 statistics for the Cluster A full model were 46% (initial status) and 33.6% (rate of change); for the reduced model they were 40.2% (initial status) and 13.6% (rate of change). The parameter estimate associated with the relationship between the initial status of the PEM-C trajectory and the Cluster A PD trajectory was negative, suggesting that subjects whose PEM-C growth trajectories began at higher elevations on study entry tended to have Cluster A PD trajectories that began at lower elevations. We illustrate this pattern in Panel A of Figure 2, in which we display fitted change trajectories in Cluster A PD features for prototypical subjects who were either high (+1 *SD*) or low (–1 *SD*) in the elevation of their PEM-C trajectories on entry into the study (we also plot a fitted trajectory for a prototypical participant of average initial status on PEM-C for comparison).[5]

In the final parsimonious model for change in the Cluster B PD features, we found that the initial status of the NEM, PEM-A, and CON growth trajectories predicted the initial status of the Cluster B growth trajectories. Both the full and reduced models provided reasonably good fit to the data (e.g., in both cases, SRMR $< .08$). The values of the pseudo-R^2 statistics for the Cluster B full model were 64.0% (initial status) and 52.5% (rate of change); for the reduced model they were 55.8% (initial status) and 37.8% (rate of change). Subjects whose NEM and PEM-A growth trajectories began at higher elevations on study entry and whose CON growth trajectories began at lower elevations on study entry tended to have Cluster B PD trajectories that began at higher elevations on entry into the study. It is interesting to note that we also found that the initial status of PEM-A growth trajectories was inversely related to the rate of change in Cluster B growth over time. This means that subjects who entered the study at higher initial levels of PEM-A displayed more rapid rates of decline in Cluster B PD features over time. We illustrate this pattern in Panel B of Figure 2, in which we display fitted change trajectories in Cluster B PD features for prototypical subjects who were either high (+1 *SD*) NEM, low (–1 *SD*) CON, and low (–1 *SD*) PEM-A; or low (–1 *SD*) NEM, high (+1 *SD*) CON, and high (+1 *SD*) PEM-A in the elevation of their respective NEM, CON, and PEM-A trajectories on entry into the study (we also plot a trajectory for a prototypical subject of average initial status for NEM, CON, and PEM-A for comparison). Furthermore, there were also interesting statistically significant relationships between the control predictors and growth in the Cluster B PD outcome. Participants who were classified as PPD subjects on entry tended to have more rapid rates of change in Cluster B PD growth over time, and subjects who were older at study entry displayed more rapid rates of change.

In the final parsimonious model for change in the Cluster C PD features, we found that the initial status of the NEM and PEM-C growth trajectories predicted the initial status of the Cluster C

[4] The full and reduced models without the control predictors led to virtually identical results to those obtained with models that included them. We report the results for the full and reduced models with the control variables here to illustrate that these captured critical features of the study design and to be consistent with prior analyses of the LSPD. Results for the full and reduced models without the control predictors may be requested from Mark F. Lenzenweger.

[5] The fitted prototypical trajectories for Cluster A, B, and C PD features depicted in Figure 2 are based on the respective fitted reduced models with the control predictors set to their sample averages.

Table 2
Predicting Individual Growth in Personality Disorder Features (Initial Status and Linear Rate of Change) by Individual Growth in Neurobehavioral Systems Indicators

	Growth in Cluster A personality disorder features				Growth in Cluster B personality disorder features				Growth in Cluster C personality disorder features			
	Full model		Reduced model		Full model		Reduced model		Full model		Reduced model	
Variable	Initial status	Rate of change	Initial status	Rate of change	Initial status	Rate of change	Initial status	Rate of change	Initial status	Rate of change	Initial status	Rate of change
	Parameter estimates and approximate p values											
Intercept	2.648	0.016	2.881	0.575	−8.941**	2.148†	−9.133***	2.079*	0.439	−0.629	−0.210	−0.257
NEM												
Initial status	0.009	0.001	—	—	0.037**	−0.002	0.037***	—	0.028***	0.001	0.030***	—
Rate of change	−0.020	0.030	—	—	−0.161	0.048	—	—	0.003	0.011	—	—
CON												
Initial status	−0.014	0.0005	—	—	−0.096***	0.011	−0.065***	—	−0.018	0.003	—	—
Rate of change	−0.100	0.053	—	—	−0.464	0.034	—	—	−0.044	0.003	—	—
PEM-A												
Initial status	−0.008	0.002	—	—	0.161***	−0.025*	0.123***	−0.017**	−0.024	0.018†	—	0.006
Rate of change	−0.098	0.017	—	—	0.273	0.045	—	—	−0.153	0.160	—	—
PEM-C												
Initial status	−0.043***	0.004	−0.042***	—	−0.003	−0.002	—	—	−0.017†	−0.008*	−0.022***	0.003
Rate of change	−0.058	−0.004	—	—	−0.327	0.025	—	—	−0.015	−0.067	—	—
Gender	−0.031	0.003	−0.059	−0.034	0.102	0.024	0.223	0.031	0.083	0.021	0.043	0.038
Group	−0.001	0.020	0.183	−0.039	−0.051	−0.048	0.263	−0.149*	0.347†	−0.088	0.304†	−0.061
Group × Gender	0.338	−0.051	0.366	−0.073	0.853*	−0.133	0.611*	−0.152	0.057	−0.150	0.113	−0.223*
Age at Entry	0.052	−0.025	0.037	−0.034	0.338*	−0.082	0.360*	−0.092†	0.061	0.040	0.076	0.018
	Goodness-of-fit statistics											
χ^2 (df)	162.04 (85)		190.33 (100)		154.86 (85)		180.95 (97)		279.03 (85)		303.47 (97)	
Pseudo R^2 (%)	46.0	33.6	40.2	13.6	64.0	52.5	55.8	37.8	52.7	70.7	52.3	47.5
AIC	481.37		603.80		447.29		624.64		512.32		878.75	
RMSR	2.22		3.20		2.63		3.04		3.38		3.54	
SRMSR	.034		.058		.037		.048		.055		.066	

Note. Table contains parameter estimates and approximate p values from the structural portion of selected full and reduced (final) fitted latent growth models. NEM = negative emotion; CON = nonaffective constraint; PEM-A = positive emotion-agentic; PEM-C = positive emotion-communal; AIC = Akaike's information criterion; RMSR = root-mean-square residual; SRMSR = standardized root-mean-square residual.
† $p < .10$. * $p < .05$. ** $p < .01$. *** $p < .001$ (two-tailed).

growth trajectories. Both the full and reduced models provided reasonably good fit to the data (e.g., SRMR $<$ .08 in both cases). The values of the pseudo-R^2 statistics for the full model were 52.7% (initial status) and 70.7% (rate of change); for the reduced model they were 52.3% (initial status) and 47.5% (rate of change). Subjects whose NEM growth trajectories growth began at higher elevations on study entry and whose PEM-C growth trajectories began at lower elevations on study entry tended to have Cluster C PD trajectories that began at higher elevations on entry into the study. We illustrate this pattern in Panel C of Figure 2, in which we display fitted change trajectories in Cluster C PD features for prototypical subjects who were either high (+1 *SD*) NEM and low (–1 *SD*) PEM-C, or low (–1 *SD*) NEM and high (+1 *SD*) PEM-C in the elevation of their respective NEM and PEM-C trajectories on entry into the study (we also plot a trajectory for a prototypical participant of average initial status for NEM and PEM-C for comparison). Again, there were also interesting statistically significant relationships between the control predictors and growth in the Cluster B PD outcome. Participants who were classified as PPD subjects tended to have higher initial status for their Cluster C feature growth trajectories. In addition, the two-way Group × Gender interaction predicted rate of change in the Cluster C growth trajectories such that being a male PPD subject was associated with a more rapid rate of decline in Cluster C PD features over time.

Sensitivity Analyses: Impact of Treatment and/or Axis I

In addition to our primary analyses described above, we conducted sensitivity analyses to evaluate the impact of the presence or absence of any Axis I disorder and/or the presence or absence of any form of psychiatric/psychological treatment in the subjects either before or during the study period on the growth observed for the PD feature dimensions. We believed that this was a reasonable set of analyses to undertake given the possibility that comorbid Axis I pathology might alter growth trajectories, as might the impact of any form of treatment. We found that the presence of any form of Axis I disorder either before or during the study period was unrelated to change for Cluster A, B, or C PD features over time, and that the presence of any form of psychiatric/psychological treatment was also unrelated to change for Cluster A, B, or C PD features over time.

Discussion

The emerging corpus of data regarding the natural history of PD symptomatology over time suggests that this form of psychopathology is considerably more flexible and plastic than previously thought (Johnson et al., 2000; Lenzenweger, 1999; Lenzenweger, Johnson, & Willett, 2004; Shea et al., 2002; Zanarini et al., 2003). However, to our knowledge, no prior empirical investigation has sought to identify predictors of change in PD psychopathology within a longitudinal framework. In the present study, we utilized multiwave data from the LSPD to address this issue. In our exploration, we focused on four major neurobehavioral systems predictors, namely NEM, CON, PEM-C, and PEM-A, on the basis of their hypothesized substantive importance in models of PD (Depue & Lenzenweger, 2001, 2005) as well as of normal personality (Tellegen, 1982, 1985).

Using latent growth modeling, we simultaneously modeled individual change over time in the NEM, CON, PEM-C, and PEM-A systems and investigated whether growth in these systems was predictive of changes observed in Cluster A, B, and C PD features over time. In short, we modeled and explored the effects of change in personality on change in PD features over time. Our primary findings suggest that initial levels of personality dimensions on entry into the study, hypothesized to be reflective of underlying neurobehavioral systems, predict individual variation in the initial levels of the growth trajectories for subjects with elevated Cluster A, B, or C PD features. This general pattern of results is consistent with the model proposed by Depue and Lenzenweger (2001, 2005, 2006) that ascribes importance to the level and configuration of elevations of neurobehavioral systems in the determination of PD. Specifically, we found that the initial level of PEM-C was related inversely to higher initial levels of growth trajectories for those subjects with elevated Cluster A PD features. This finding accords well with the diminished affiliative tendencies seen in persons with schizoid, schizotypal, and/or paranoid PD features. Such individuals do not seek out social connection and affiliation with others. Disengagement from affiliation may suggest a more focal pathology in these individuals, and one might consider probing those neurobehavioral processes and genetically influenced structures (e.g., *u* opiate receptors) known to underpin affiliation (see Depue & Lenzenweger, 2005, 2006). High initial levels of the growth trajectories for those subjects with increased Cluster B PD features were associated with increased initial levels of NEM and PEM-A and decreased initial levels of CON. This configuration of predictors also accords well with what is known about Cluster B psychopathology and theoretical conjectures in the Depue–Lenzenweger model. Individuals who present with increased amounts of borderline, antisocial, histrionic, and narcissistic PD features are frequently characterized by decreased amounts of behavioral constraint (with ample evidence of impulsive and dyscontrolled behaviors); increased levels of anxiety, depression, and hostility; and increased levels of PEM-A reflective of an outwardly directed "externalizing" approach to the world in which rewards are sought actively (cf. Kendler, Prescott, Myers, & Neale, 2003; Krueger, Markon, Patrick, & Iacono, 2005). The diminished constraint construct is of particular interest for Cluster B PDs as it reflects underlying neural constraint, which is a system that modulates stimulus elicitation of motor behavior, both positive and negative emotions, and cognition (e.g., Lenzenweger, Clarkin, Fertuck, & Kernberg, 2004). High initial levels of the growth trajectories for those subjects with increased Cluster C PD features were associated with increased initial levels of NEM (reflecting anxiety and other negative affective features) and decreased initial levels of PEM-C, which reflects, again, diminished social engagement and affiliation. This characterization accords well with what is known about the dependent, obsessive–compulsive, and other Cluster C PDs, which represent anxiety-riddled and socially frightened individuals.

A principal goal in undertaking this study was to determine if individual change in the personality systems underpinning PD was related to observed individual change in PD over time. Generally, our results provide only a provisional answer to this issue. We found that those subjects who presented with initially higher levels of PEM-A revealed accelerated declines in Cluster B PD features over time. This may reflect a benefit of being engaged with the

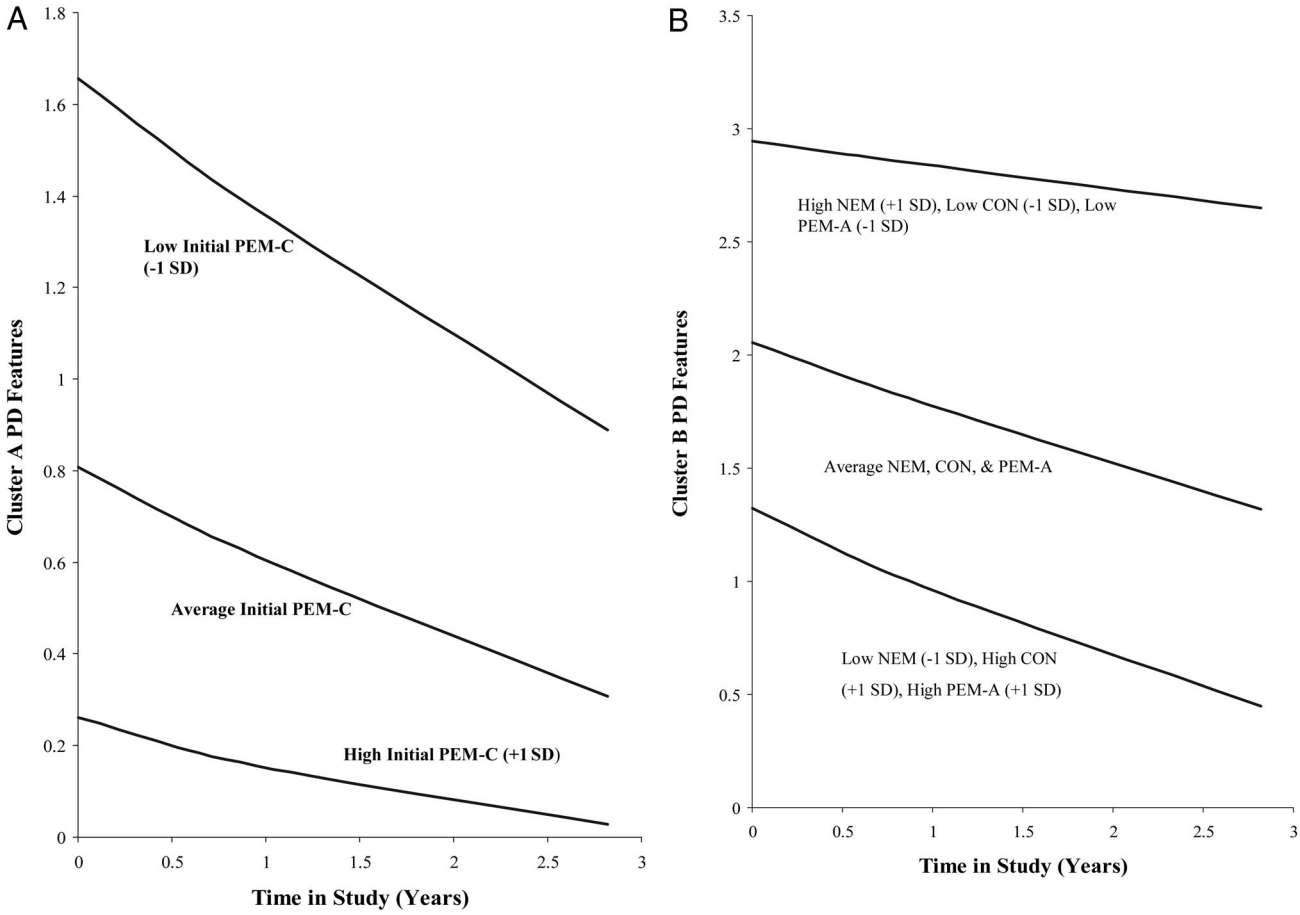

Figure 2. Fitted personality disorder (PD) trajectories for prototypical individuals with selected initial levels on personality variables. A: Fitted growth trajectories in Cluster A PD features as a function of deviation on PEM-C initial levels. B: Fitted growth trajectories in Cluster B PD features as a function of deviation on initial levels of NEM, CON, and PEM-A. C: Fitted growth trajectories in Cluster C PD features as a function of deviation on initial levels of NEM and PEM-C. The fitted values displayed are estimated from the respective parsimonious (i.e., reduced) fitted models with the values of all control predictors (age at entry, group, and sex) set to their sample averages. PEM-C = positive emotion–communal; NEM = negative emotion; CON = nonaffective constraint; PEM-A = positive emotion–agentic.

world in terms of reward and incentive motivation. Such continued engagement with the world may represent a form of resilience or the buffering role of PEM-A. To the extent that the PEM-A system is reflective of dopamine activity, dopaminergic-mediated engagement with external rewards, many of which are social in nature, may serve to moderate or offset Cluster B PD features more quickly over time. However, a precise mechanism for this pattern of results cannot be specified from these analyses of correlational data. We did not detect other statistically significant predictors of the rate of change in the either the Cluster A or Cluster C PD dimensions.

It is especially salient that the rate of change parameters in the four personality systems we investigated did not predict the rate of change in the PD clusters across the board. This may be an issue of reduced statistical power. It may reflect one of the known analytic challenges in seeking to relate change to change in growth modeling due to the difficulty of estimating rates of changes precisely with a limited number of waves of longitudinal data (Singer & Willett, 2003). Alternatively, it could be that our results indicate a genuine absence of relationships between rates of change in the personality variables and the rates of change in the PD features. At the moment, we cannot distinguish between these explanations for this set of results. It is certainly plausible that our analyses, limited to three waves of longitudinal data, did not provide sufficient statistical power to reject null hypotheses about the impact of change in personality on change in PD. If this is the case, then it will certainly be alleviated in the future with the planned collection of additional waves of data on the LSPD subjects, which will yield increased power and precision for all of the analyses reported here. The subjects in the LSPD were initially assessed across the 4-year time span during their college years but are now approaching their mid-30s. With the passage of such a substantial period of time and the developmental possibilities contained therein, it is certainly plausible that the incorporation of additional waves of data into these analyses will shed more light on whether change in personality predicts change in PD. It is also

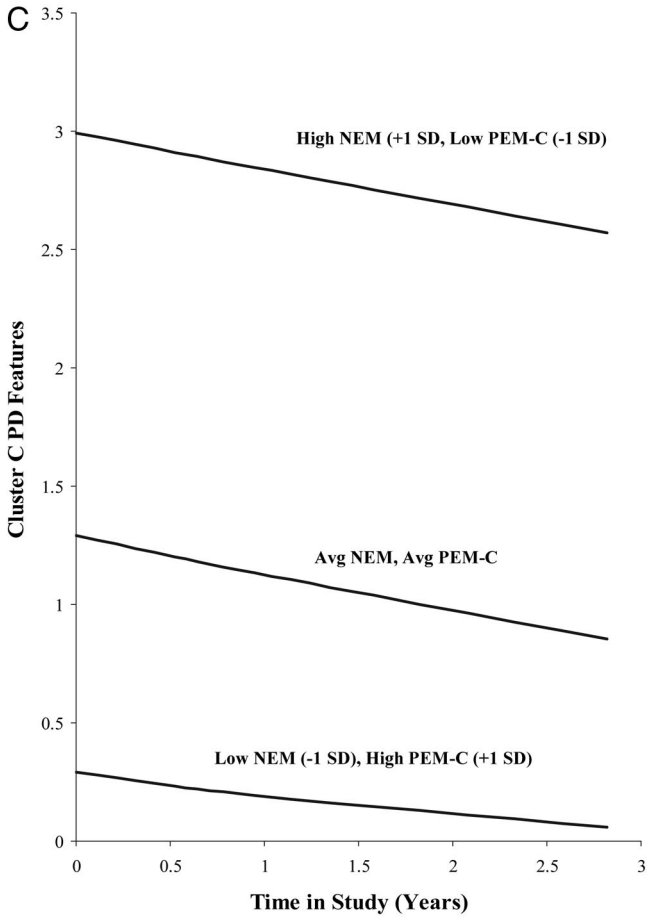

Figure 2. (continued)

important to note, in this context, that the addition of assessment waves will serve to further increase the reliability of the estimates themselves (Singer & Willett, 2003).

We did observe some interesting findings for the background/control variables that we included in our analyses. For example, it was male PPD subjects who were slightly older at entry whose Cluster B PD growth trajectories tended to begin at higher elevations. However, a more rapid rate of change (i.e., decline) for Cluster B PD features was also associated with being a PPD subject. Finally, being a male PPD subject was associated with a more rapid decline for Cluster C PD features. More importantly, however, the various relationships that we detected between the personality system growth parameters and the PD growth parameters were largely unaffected by either the inclusion or exclusion of these background/control predictors, which suggests that the background/control variables do not stand in for the personality constructs in any meaningful manner. Moreover, our sensitivity analyses—which added predictors describing Axis I disorders and prior history of treatment—suggested that neither of these conditions was related to change in any of the three PD cluster dimensions.

Do our results match findings from other multiwave longitudinal studies of PD? Although there are three other major longitudinal studies of PD currently underway using nonclinical (Johnson et al., 2000) and clinical (Shea et al., 2002; Zanarini et al., 2003) samples, data are not currently available from these projects that bear directly on the central question of whether change in personality systems impacts change in PD over time. Clearly, to fully address this question and to compare results with those reported here, one needs to focus on data that have been collected with methodological safeguards in place, such as blinded PD assessments and no treatment/time confounds. One broadly relevant study examined the extent to which levels (status) in personality traits (five-factor model) corresponded to levels (status) of PD symptom features (for four different Axis II disorder dimensions) across time using a cross-lagged correlational design (Warner et al., 2004). Mindful of the limitations of such a statistical approach (see Rogosa, 1980) as well as the fact that all subjects in their study were in treatment for PDs, Warner et al. reported that levels of some normal personality constructs were predictive of later levels of PD features for the four PDs examined. Their analysis, however, is not directly comparable to ours as the statistical approach of Warner et al. did not parse out the effects of level of personality traits from the effects attributable to the rate of change in personality traits in the prediction of level and rate of change parameters for PDs over time. In this context, we also note that although numerous cross-sectional studies have explored relationships between various personality dimensions and the PDs (see Saulsman & Page, 2004, for an excellent review), such cross-sectional studies are mute with respect to the question of impact of change in personality systems on change in PD over time. Thus, just because high levels of neuroticism, for example, have been found to be correlated with high levels of PD in cross-sectional analyses, it does not follow that change in neuroticism would impact change in PD over time. Our study allowed us to address this question directly.

On a broader methodological note of interest to psychopathologists, the latent growth modeling approach that we utilized in the present study offers powerful advantages in the study of the predictors of change or growth in psychopathology research. This is particularly true with respect to systems that involve multiple component parts or processes that could be simultaneously affecting (or correlating with) a given outcome. For example, as it is likely that PD features reflect the phenotypic manifestations of multiple dynamic interactive neurobehavioral systems at work and each of these interactive systems themselves likely shows change and growth across the life course, then an understanding of this process requires an appropriate analytic strategy that must be able to handle simultaneous growth in several systems, as well as take various background variables into account (e.g., gender, age). Our latent growth modeling approach achieves this goal as it unifies the powerful growth curve methodology (Rogosa, Brandt, & Zimowski, 1982; Rogosa & Willett, 1985) that has proven so useful in the investigation of change in psychological and behavioral phenomena over time with the flexibility of covariance structure analysis. In this way, we have been able to move freely through an exploration of the impact of multiple important variables, some changing (NEM, CON, PEM-C, PEM-A) and others not (gender, group status, age at entry), in predicting the growth trajectory in PD. We also note that the present approach to the study of growth on growth represents a compelling and superior statistical alternative to the analysis of cross-lagged correlations, which has been a common, but problematic (Rogosa, 1980), way of examining re-

lations between two variables that are changing simultaneously over time.

The nature of the LSPD subject selection may ultimately prove especially helpful in dissecting the issues of change with which we have been concerned. In this regard, we highlight the scientific utility of a sample of subjects drawn initially from a nonclinical sample for prospective longitudinal study, particularly where some subjects are at increased risk for a PD and others are not, which has been described in detail (Lenzenweger, 2006). In brief, the utility derives from avoidance of (a) sampling artifacts that characterize subjects recruited through clinics (Berkson's bias); (b) treatment effects that necessarily impact growth trajectories for clinically identified patients (a Treatment × Time confound); and (c) study restricted only to extreme cases (i.e., diagnosed PD cases), wherein change over time is likely to be in the direction of symptom decline. By including subjects at Wave 1 who were at low risk for a PD in the LSPD, the study is therefore well positioned to examine the development of a PD across the life course. It is important from a developmental psychopathology perspective to study the development of PD features in previously unaffected individuals, and, indeed, we have actually seen such development of PD in the low-risk (i.e., NPD) LSPD subjects over time. Such a developmental opportunity would have been lost had we restricted the LSPD at the outset to the longitudinal study of only diagnosed PD cases.

Several caveats must also be considered with these data. First, our present sample (a) was clearly more homogenous in age, educational achievement, and social class than the U.S. population at large and (b) consisted of young adults, features that may have differentially affected the study results. Perhaps the most effective way to assess the generalizability of findings from the LSPD is to evaluate whether prior core LSPD findings have been replicated, and they have. For example, the LSPD-based estimate for PD prevalence in the community (11%; Lenzenweger et al., 1997) has now been broadly replicated several times in U.S. nonclinical community samples (Crawford et al., 2005; Samuels et al., 2002) and the U.S. general population (Lenzenweger et al., in press), as well as in Norwegian (Torgersen, Kringlen, & Cramer, 2001) and United Kingdom (Coid, Yang, Tyrer, Roberts, & Ullrich, 2006) samples. Furthermore, the patterns of change in mean levels of PD features over time initially reported for the LSPD sample (Lenzenweger, 1999) were subsequently replicated in both clinical (Shea et al., 2002; Zanarini et al., 2003) and nonclinical community (Johnson et al., 2000) samples. Thus, although the present sample is somewhat more compressed in terms of demographic background characteristics, this has not led to findings at odds with those obtained in other epidemiological or longitudinal PD research.

Second, we also note that adjustment to university life across the college years may have played some role in the changes we observed, however we also note that the IPDE specifically requires one to respond after reflecting on 5 years of behavior (not just the current time or recent past; Loranger, 1999), and it is relatively robust in the face of mental state alterations (Loranger et al., 1991). Third, given that the LSPD subjects were selected from a population of first-year university students, the sample may have been somewhat censored for individuals affected by some of the most severe PDs. However, one must be cautious in ascribing undue levels of mental health to subjects who happen to be selected for academic achievement, as such selection does not confer immunity to psychopathology. In fact, some forms of PD might actually be enriched in such a sample (e.g., obsessive–compulsive PD, narcissistic PD, schizoid PD). To this end, we note that 16% (or 1 in every 6) of the LSPD sample subjects was diagnosed with a formal Axis II disorder by the end of the study period (i.e., by Wave 3) using the highly conservative IPDE. Many other subjects met intermediate levels of PD criteria (e.g., 2 or 3 criteria) that fell short of *DSM* diagnostic threshold counts but indicated the presence of some degree PD disturbance of clinical intensity nonetheless according to the IPDE. Such intermediate levels of PD pathology are frequently seen even in clinical samples (e.g., Loranger et al., 1991). We note also that 45.2% of the LSPD subjects had a lifetime (or current) Axis I disorder by the end of college, and these data are broadly consistent with the ubiquitous nature of Axis I disorders in the U.S. population (see Kessler, Chiu, Demler, & Walters, 2005; Kessler et al., 1994).

Fourth, we are mindful that there may be other predictors of change in PD. Some may be a consequence of important time-varying processes (e.g., other temperament factors), whereas others may not (e.g., presence of severe childhood sexual abuse). Thus, there may well be other factors that would be predictive of rate of change in PDs over time. We hope to establish such hypotheses and probe the LSPD database more deeply in the coming years to explore such possibilities as well as collect additional data relevant to this possibility upon upcoming Wave 4 and Wave 5 assessments. On a related note, it may also be the case that some of the *DSM* PDs may not reflect solely the functioning of personality systems, specifically the Cluster A PDs. It may be the case that paranoid and schizotypal PDs represent variants of schizophrenia and thus are less related to personality (cf. Lenzenweger, 1998). Finally, we emphasize that our research design is observational, not experimental, and so, although specified the hypothesized direction of relations in the growth model, we cannot address questions of causation. The Depue–Lenzenweger model does hypothesize that underlying neurobehavioral, brain-based systems are responsible ultimately for personality and PD, however our data analyses cannot test these conjectures.

It has long been assumed that PDs are in place relatively early in the life course (i.e., late adolescence, early adulthood; see *DSM,* APA, 1980, 1987, 1994) and that they stay in place for the most part across the lifespan, as if carved in granite. In our prior analyses of the LSPD data (Lenzenweger, 1999; Lenzenweger, Johnson, & Willett, 2004), we reported evidence of flexibility in most PD features over time, a fact that challenged this long-held assumption regarding the trait-like quality of PDs over time. In short, those prior reports documented that an appreciable amount of change was occurring in the PDs on average over time. However, those reports did not specify what was driving, or what was correlated with, such change. The present article substantially extends those initial observations by using an approach that sought to identify the variables (constructs) that predict individual change in PD features over time, thus moving from a merely descriptive to a more explanatory, model-testing research posture. Although William James (1890/1950, p. 121) claimed "by the age thirty, the character has set like plaster, and will never soften again," researchers now know this is true only for some aspects of normal personality (Roberts et al., 2006; Srivastava, John, Gosling, & Potter, 2003). Of importance is the fact that experts now know,

insofar as one might want to suggest PD is a form of character or personality, that PD is not set like plaster at an early age with little prospect for change as assumed by the official psychiatric nomenclature and common clinical lore (Johnson et al., 2000; Lenzenweger, 1999; Lenzenweger, Johnson, & Willett, 2004; Shea et al., 2002; Zanarini et al., 2003). Our research findings provide some initial coordinates for future explorations of those aspects of both personality and PD where the plaster seems not to harden.

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Appendix A

Compositing the Depue–Lenzenweger (2001, 2005, 2006) Personality Dimensions Linked to Neurobehavioral Systems From the NEO-PI

We employed the published factor solution of Church (1994, Table 4) as the source of weights for creating optimal composite measures from the NEO-PI data, which we then used as indicators of the neurobehavioral systems dimensions in our analyses. Church used a principal-axis factor extraction method coupled with a varimax rotation in his analysis of the NEO-PI and Tellegen Multidimensional Personality Questionnaire inventories ($N = 575$). The precise weights that we used to construct our measures are given here, illustrating that we capitalized only on salient loadings (.35 or higher) in the construction of our NEM, CON, PEM-A, and PEM-C factor scores. Moreover, based on substantive considerations, we required that each NEO-PI facet load on only one of our constructs (NEM, CON, PEM-A, PEM-C). For instance, NEO-PI Agreeableness was assigned to PEM-C composite, and NEO-PI Conscientiousness was assigned to CON. Our composite indicators of the NEM, CON, PEM-A, and PEM-C dimensions, therefore, reflected the application of Church's weights in the Longitudinal Study of Personality Disorders data set.

Negative Emotion (NEM) = (.62 × anxiety) + (.58 × hostility) + (.66 × depression) + (.58 × self-consciousness) + (.35 × impulsivity) + (.63 × vulnerability).

Constraint (CON) = (–.40 × excitement seeking) + (–.37 × actions) + (.49 × conscientiousness).

Positive Emotion–Agentic (PEM-A) = (.65 × assertiveness) + (.50 × activity).

Positive Emotion–Communal (PEM-C) = (.68 × warmth) + (.57 × gregariousness) + (.63 × positive emotions) + (.35 × feelings) + (.41 × agreeableness).

Appendix B

In our sample, the median values of sample Pearson bivariate correlations among the NEM, CON, PEM-A, and PEM-C composites across the three waves of LSPD data were as follows:

Table B1
Sample Intercorrelations of the Personality Dimensions

Measure	NEM	CON	PEM-A	PEM-C
NEM	—	−.32	−.25	−.32
CON		—	.22	.10
PEM-A			—	.38
PEM-C				—

Note. NEM = negative emotion; CON = nonaffective constraint; PEM-A = positive emotion-agentic; PEM-C = positive emotion-agentic.

(*Appendixes continue*)

Appendix C

Table C1
Descriptive Statistics for Personality and Personality Disorder Features, Longitudinal Study of Personality Disorders (N = 250)

	No personality disorder		Possible personality disorder	
Variable/Wave	*M*	*SD*	*M*	*SD*
	Personality dimensions			
PEM-A 1	20.01	3.91	20.04	4.78
PEM-A 2	19.87	3.99	19.93	4.53
PEM-A 3	20.25	4.07	20.11	4.65
PEM-C 1	71.14	8.80	64.59	10.49
PEM-C 2	71.59	7.41	65.30	10.58
PEM-C 3	71.31	8.98	65.84	10.92
NEM 1	35.29	8.86	51.11	14.39
NEM 2	33.05	8.80	47.47	14.22
NEM 3	33.21	10.33	44.66	13.29
CON 1	10.84	4.57	8.84	5.48
CON 2	11.22	4.62	8.80	5.83
CON 3	11.23	4.87	9.06	5.17
	Personality disorder features			
Cluster A 1	1.29	2.68	3.36	4.32
Cluster A 2	0.79	2.55	1.84	3.48
Cluster A 3	0.65	1.79	1.73	3.35
Cluster B 1	1.83	2.96	7.59	8.28
Cluster B 2	1.55	2.84	4.51	6.50
Cluster B 3	1.69	2.38	3.74	4.78
Cluster C 1	1.78	2.47	5.77	5.82
Cluster C 2	0.89	2.37	2.56	3.76
Cluster C 3	1.52	2.88	3.21	4.77

Note. Wave refers to assessment wave as indicated by 1, 2, or 3. PEM-A = positive emotion-agentic; PEM-C = positive emotion-communal; NEM = negative emotion; CON = nonaffective constraint; Clusters A, B, and C = *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed., revised) Cluster A, B, and C personality disorder features, respectively.

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