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Development of Personality and the Remission and Onset of Personality Pathology

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Abstract

The current study uses the Longitudinal Study of Personality Disorders dataset (Lenzenweger, 1999) to examine the development of personality traits in the context of the remission and onset of personality disorder (PD) symptoms. Despite high levels of stability, past research that has examined the development of basic personality traits has also found a mean trend towards increased maturity, and that individuals vary in their trajectories of trait development. Research on PD change has shown a similar pattern. We employ individual growth curve modeling to examine the relationship between personality trait development and PD symptom course. We found that the two are indeed related, and that remission in PD symptoms is associated with patterns of trait development associated with more rapid maturity. In contrast, deviating from the mean of trait development either through no change (i.e., stagnation) or change in the opposite direction (i.e., regression) was associated with developing PD symptoms over the course of the study.

Keywords

Personality traits; personality disorder; personality development; growth curve modeling

Research finds personality traits to be highly stable using a number of samples, measures, and analytic approaches (Roberts, Walton, & Viechtbauer, 2006; Costa, Herbst, McCrae, & Siegler, 2005). However, research also demonstrates that basic personality traits, although highly stable, are not fixed, and in fact mean levels change significantly through the life span following normative developmental trends (Roberts et al., 2006). Moreover, mean level changes appear to capture only part of the story, and there is rich interindividual heterogeneity in the personality trajectories individuals chart through the life course (Vaidya, Gray, Haig, Mroczek, & Watson, 2008). Interindividual differences in change immediately raise the question: What are the determinants of those differences? Although the determinants are undoubtedly numerous, one possible source of this deviation is abnormality in the personality system itself. The current research investigates the effect of

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remission and onset of personality disorder (PD) symptoms on interindividual variability in personality trait trajectories in early adulthood.

During the transition from late adolescence to early adulthood individuals are struggling with a number of important normative developmental tasks (Arnett, 2000). It is during this time that individual differentiation would be expected to occur vis-à-vis those who are managing this transition successfully, and those who are struggling to achieve mature functioning. Development of personality traits in early adulthood is characterized by decreasing Neuroticism, and increasing Agreeableness, Conscientiousness, and Openness. Extraversion has been less consistent, with some investigators finding increases, and others finding stability in mean scores over time. This pattern of trait development has been characterized as a trend towards personality maturity (Roberts et al., 2006). Individuals who are more emotionally stable, agreeable, conscientious, and open to new experiences are better suited to engage society and get ahead while getting along (Hogan, 1996). However, despite high rank order stability coefficients, researchers find individual differences in rates of change (Robins et al., 2001). In the early years of young adulthood (i.e., 18–21) stability coefficients are at their most modest (Roberts & DelVecchio, 2000), and there is significant heterogeneity in developmental trajectories (Vaidya et al., 2008). The majority of individuals follow the trend towards increases in personality maturity (albeit at varying rates) while there are those who depart markedly from the norm by either failing to mature or changing in the opposite direction. Yet, relatively little is known about the processes of change in personality and it is essential for developmental theories of personality that the determinants of this variability in personality growth are identified.

Psychopathology may account for interindividual variability in personality development. Contemporary theoretical models directly link personality with psychopathology (Depue & Lenzenweger, 2005; Pincus, Lukowitsky, & Wright, 2010; Widiger & Smith, 2008), and cross-sectional research finds links between personality and psychopathology of most types (Clark & Watson, 1991; Krueger et al., 2002) and PDs in particular (Samuel & Widiger, 2008). Empirically, the personality trait profile associated with increased maturity is the opposite of the trait profile associated with PD generally—High Neuroticism, Low Conscientiousness, Low Agreeableness (Morey et al., 2002). Indeed, lack of development in the personality system is the cornerstone of PD in much of the clinical literature (e.g., Clarkin, Lenzenweger, Yeomans, Levy, & Kernberg, 2007; Freud, 1909/1961; McWilliams, 2011). Given the strong conceptual and cross-sectional empirical links, PD and its processes are intuitive potential predictors of the variability in personality development. However, for the two to be developmentally linked, PD would need to demonstrate similar patterns of instability as basic personality traits.

The results of a number of large-scale longitudinal studies that have assessed the stability of PD diagnoses and symptom criteria indicate that PD symptoms are, in fact, notably unstable and plastic. In both clinical (Shea et al., 2002; Zanarini et al., 2003) and community samples (Johnson et al., 2000) results show that there are significant declines in the number of individuals meeting diagnostic threshold, and accordingly significant declines in symptoms when treated as dimensions. Further results indicate that the mean decline masks significant interindividual heterogeneity in symptom change over time (Lenzenweger, Johnson, & Willett, 2004). These patterns of change in PD mirror those in personality trait development, with the average trend towards increased effectiveness in functioning but with significant individual differences in growth trajectories. Thus, the pattern of instability and individual variability in growth in PD suggests that it has the potential to be dynamically related to growth in basic personality traits.

Past research supports the notion that personality traits assessed cross-sectionally are associated with PD trajectories (Lenzenweger & Willet, 2007; Wright, Pincus, & Lenzenweger, 2010). Other research (Warner et al., 2004) suggests that personality is predictive of subsequent PD symptoms. However, by adopting a cross-lagged modeling approach Warner et al. could not examine development trajectories in either system. Hopwood and colleagues (2009) found that individuals diagnosed with borderline personality disorder (BPD) demonstrate more rapid declines in Neuroticism and increases in Openness compared to other PD diagnoses. Nevertheless, as the authors point out, this was found in a clinical sample for which the primary inclusionary criterion is the diagnosis of a serious mental disorder and within which the strong mean trend is a decline in symptoms. Therefore, what has primarily been captured is the increase in health of the sample. Additionally, these were patients seeking or who had recently sought treatment, which makes firm conclusions as to what is driving this change more difficult (i.e., general personality development, treatment response, regression to the mean).

Taken together, past research indicates that personality traits and PD both exhibit mean change in early adulthood, and there is significant variability in the rates and directions of this change. Although it is intuitively appealing to conclude that because personality and PD show similar patterns of mean growth (in opposite directions) across samples the change be related to each other, within individuals this is not necessarily the case, and no prior study has directly examined the relationship between Big Five trait growth trajectories and PD symptom change within the same. Thus it remains an untested assumption that growth in personality traits and PD symptom change are developmentally linked. Testing this assumption has important implications because most contemporary theories directly link the normative and non-normative expressions of personality, and empirically demonstrating a longitudinal link during a pivotal developmental epoch (i.e., early adulthood) would add an important piece of scientific evidence to theoretical assertions.

The Current Study

The purpose of the present study is to examine the role of PD in basic personality trait development. We use the Longitudinal Study of Personality Disorders (Lenzenweger, 1999) sample, which is a naturalistic, prospective study of PD and personality. The LSPD includes a broad range of individuals that both *did* and *did not* possess PD symptoms at the outset of the study thereby allowing for the investigation of those who remit *and* develop PD criteria over time. The limited prior research that has examined the effect of PD status on trait development has looked at PD symptoms diagnosed at one time point and individual trait trajectories going away from that initial assessment time point. This analytic strategy only elucidates half of the story—the effect of PD remission on trait growth. Furthermore, it potentially capitalizes on sampling strategies and the law of initial values in change over time. However, individual growth curve (IGC) modeling allows for a flexible shift in focus between the start and finish of a study in the prediction of the trajectories one takes, thereby illuminating both patterns of personality trait growth—those associated with symptom remission *and* symptom onset. Accounting for initial and final PD status within an IGC framework in a sample that is balanced with respect to PD sampling will allow for an empirical examination of the key theoretical assertion that personality and PD are developmentally linked (see e.g., Lenzenweger & Clarkin, 2005).

We measure personality traits using the Revised Interpersonal Adjective Scales – Big Five (IASR-B5; Trapnell & Wiggins, 1990), a measure based on the interpersonal traits of Dominance and Affiliation with additional scales for Neuroticism, Conscientiousness, and Openness. Dominance and Affiliation can be understood as rotational variants of Extraversion and Agreeableness (Pincus, 2002). Analytically, we will first establish the mean and individual change in the dimensions measured by the IASR-B5 using IGC models.

Subsequently, the main analyses of this study relate PD symptoms to personality trait trajectories as a function of PD remission and onset in early adulthood. We hypothesize that the remission and onset of PD symptoms over time should be associated with patterns of trait development that move toward and away from the normative maturational trend.

Method

Participants

Extensive detail concerning the initial participant selection procedure and sampling is given elsewhere (see e.g., Lenzenweger, 1999). Of the initial 258 participants, 250 (53% Female) completed all three assessment waves and are included in these analyses. The mean age of the participants at entry into the study was 18.88 years (*SD* = 0.51). Participants were subsequently assessed at their second and fourth years of college.

Procedure

At each time point participants completed self-report measures of personality and clinical assessments were conducted by experienced Ph.D. or M.S.W. clinicians. The mean time between entry into the study (T1) and Wave 2 (T2) and Wave 3 (T3) was 0.95 years (*SD* = 0.14) and 2.82 years (*SD* = 0.23), respectively. All participants were assessed on the same schedule, although the time between assessments varied from case to case. Participants were sampled to ensure an adequate coverage of PD and non-PD by selecting approximately half of the included individuals based on a possible positive PD diagnosis using a self-report screener of PD symptoms, and the other half with no full diagnosis and fewer than 10 individual criteria based on the same screener. At T1, 11% of the participants qualified for a full Axis II diagnosis of some sort. As of T3, 16% of the sample had received probable or definite diagnoses of PD.

Measures

Revised Interpersonal Adjective Scales – Big Five (IASR-B5)—The IASR-B5 (Trapnell & Wiggins, 1990) consists of adjective based 124-items (e.g., dominant, coldhearted) on an 8-point scale assessing the dimensions of Dominance, Affiliation, Conscientiousness, Neuroticism, and Openness. Coefficient alphas for all scales at all waves ranged from .88 to .96.

International Personality Disorder Examination (IPDE)—The IPDE (Loranger, 1999) interrater reliability (based on intraclass correlation coefficients) was excellent at all three waves, ranging between .84 and .92 for all PD dimensions. The PD dimensional scores were used for this study. For each symptom an individual receives a score of 0 (Absent or Normal), 1 (Exaggerated or accentuated), or 2 (Criterion or Pathological). These values are summed to create a count of disorder related features. We use the total cluster scores for each of the well-known *DSM*-based Cluster A, Cluster B, and Cluster C PD domains (American Psychiatric Association, 2000). 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 count for borderline, antisocial, histrionic, and narcissistic PD features (i.e., the erratic/impulsive PD cluster). Cluster C total scores reflected the total count for avoidant, obsessive–compulsive, and dependent PD features (i.e., the anxious/avoidant PD cluster). The resulting distributions are skewed as is commonly the case for psychiatric criteria. Descriptive statistics are presented. T1 Cluster A Features: M = 2.4, Mdn = 1.0, SD = 3.8; Cluster B: M = 4.8, Mdn = 2.0, SD = 6.9; Cluster C: M = 2.9, Mdn = 1.0, SD = 4.0. T3 Cluster A Features: M = 1.2, Mdn = 0.0, SD = 2.8; Cluster B: M = 2.8, Mdn = 1.0, SD = 3.9; Cluster C: M = 1.8, Mdn = 1.0, SD = 3.0.

Statistical Analyses

IGC models were fitted within a multilevel modeling framework using HLM-6 (Raudenbush, Bryk, Cheong, Congdon, & Du Toit, 2004) and employing full maximum likelihood estimation. This approach was supported due to the lack of missing data and a post hoc analysis of the residuals that indicated they conformed to a normal distribution. First, we estimated unconditional models to establish the average intercepts and rate of change per year (i.e., fixed effects) and test for individual variability (i.e., random effects) in intercepts and slopes. Second, we estimated conditional models in which we examined systematic interindividual differences in intercept and slope as a function of between-participant predictors (e.g., age, sex, PD). Improvement in model fit going from the unconditional to the conditional models was assessed by comparison of −2LL (i.e., deviance) statistics. Fixed-effects and variance components were tested for statistical significance using the provided *t* (2-tailed) and χ^2 statistics.

Results

All basic personality dimensions at each wave were first standardized on the original IASR-B5 sample to provide a common metric. Therefore, although the results presented here are not standardized, they are in standard deviation units. The results of the unconditional IGC models are presented in Table 1. These models capture the average elevation and net change over time with the fixed effects, and model the heterogeneity in individual growth using random effects. The fixed effects for the intercepts suggest that the sample is more affiliative, open, and less neurotic than the original IASR-B5 sample, reflecting the inclusion of the screened non-PD group in the LSPD. The fixed and random effects of the slopes are of central interest here, and capture the average rate of change in the personality dimensions per year and variability around that mean. Significant mean increases were found for Affiliation, Conscientiousness, and Openness, with Neuroticism significantly decreasing over time. Dominance did not have significant mean change. These results are consistent with similarly aged samples (Robins et al., 2001; Vaidya et al., 2008). Significant individual variability was found for rates of change in each dimension.

In the conditional models, we included as predictors age of entry to the study, sex (males = 1, females = 0), and the three DSM PD Clusters (each in separate models). However, the set of predictors was not identical for the intercept and the slope parameters. Only T1 PD features were included as predictors as the intercept was fixed at T1, whereas for the slope both T1 and T3 PD criteria were included. By including both T1 and T3 PD features in predicting rate of change, they serve as covariates of each other and make for specific interpretations of the coefficients. In this context, the coefficients for the PD features at a given time point in predicting slope is the average value per PD feature, when the other time point's PD feature value is fixed. The T1 PD feature count represents the rate of change of a given personality trait each year per PD feature controlling for an individual's value at T3. This can be used to estimate the effect for an individual if they end the study having remitted in PD features. In contrast, the T3 PD coefficient represents the rate of change in a given personality trait each year per PD feature controlling for an individual's value at T1. This can be used to estimate the effect if an individual were to develop PD features after having started with none. Accordingly, these estimates can be understood as the personality trajectories for those whose symptoms remit (T1 PD) and for those who develop personality pathology (T3 PD). This analytic approach statistically reigns in the strong mean trends (i.e., declines) in PD harnessing the relationship between personality and PD as it emerges over time.

The results are organized in three tables (2, 3, and 4) associated with each of the broad PD Clusters. Due to space limitations, we focus on the results associated with the slopes in these

models. However, we note that the patterns of associations between the intercepts and PD T1 across models were highly consistent with past meta-analytic results (Samuel & Widiger, 2008). Additionally, across clusters males reported lower levels of Affiliation and Neuroticism than females. Older individuals reported lower Neuroticism.

The results for Cluster A PD features are catalogued in Table 2. Only the rate of change in Dominance was associated with these PD features, such that T3 PD features were associated with declines in Dominance over the course of the study. Thus, individuals who develop Cluster A PD features break the normative pattern of unchanging Dominance and demonstrate a decline.

The results for Cluster B PD features are presented in Table 3. Cluster B PD features significantly predicted personality trait development in a number of ways. First, T1 PD is related to steeper rates of decline in Neuroticism. Those individuals whose PD features remitted decreased more rapidly in Neuroticism. In contrast, T3 PD features were *positively* associated with the slopes for Neuroticism and *negatively* associated with the Conscientiousness slopes. Those individuals who developed Cluster B PD features demonstrate more shallow declines in Neuroticism and more shallow increases in Conscientiousness relative to the normative rates of change. Males demonstrated a more modest increase in Conscientiousness over time.

Table 4 summarizes the effect of Cluster C PD features. PD T1 was associated with increases in Dominance and with steeper declines in Neuroticism. In contrast, PD T3 was related to decline in Dominance and Affiliation, and was positively associated with slope in Neuroticism. Taking normative trends into account, individuals who develop Cluster C PD features demonstrate decreases in Dominance, more modest increases in Affiliation, and less rapid decreases in Neuroticism relative to the average trait development trajectory.

Discussion

A wealth of prior research demonstrates that personality traits show a normative trend towards increased maturity in early adulthood while PD decreases on average over the same time span. Additionally, both show individual differences in the rate and direction of this change. The majority of past work relating personality to PD has occurred at the cross-sectional level, yet longitudinally, the interface between the two remains poorly understood. Without direct empirical examinations, discussions of the longitudinal relationships remain speculative (cf. Clark, 2009). The current research examined Big Five trait change in early adulthood, and the effect of PD remission and onset on individual variability in that change. Baseline model results were similar to previous studies, showing mean increases in Affiliation, Conscientiousness, and Openness, a decrease in Neuroticism, but no change in Dominance, but with significant individual variability. The PD fixed effects for the elevation, which can be understood as the static cross-sectional PD trait profile, showed patterns that are highly consistent with the PDs in each of these clusters respectively, lending confidence to the remaining results (Samuel & Widiger, 2008). Further, personality trait change was significantly associated with the remission and onset of PD criteria. Using a novel analytic approach, we used the PD symptoms assessed at the outset and the end of the study as covariates in these models. This isolates the effects of those who decline and those who increase in PD over time allowing for the study of trait trajectories associated with both the remission and onset of PD symptoms. Broadly, we found support for the hypothesized relationship such that as PD symptoms decline personality shows more rapid change in the direction of maturation, and in contrast, stagnates or regresses as PD develops.

More specifically, there appears to be some differentiation in the patterns of trait development associated with each cluster. Cluster A features exhibited the most modest pattern of association with trait development. This is a bit surprising given the relatively strong associations with higher Neuroticism, lower Affiliation and Dominance (Samuel & Widiger, 2008). Although the reason is unclear, it may be that the Cluster A PDs do not solely reflect the functioning of personality systems. It is possible that paranoid and schizotypal PDs represent variants of schizophrenia and thus the maintenance processes are less related to personality traits (cf. Lenzenweger, 2010). The Cluster B predicted change is highly consistent with previous results using clinical samples with BPD (Hopwood et al., 2009). The result of more rapid declines in Neuroticism over time as PD declines was replicated here. Moreover, more modest declines in Neuroticism and more shallow increases in Conscientiousness were associated with the development of Cluster B PD features. This is understandable given the strong associations between Cluster B PD's and disconstraint. Depending on the amount of PD features that develop over time, the net growth for an individual could range from one of no growth in these traits (i.e., stagnation as others develop) to increases in Neuroticism and decreases in Conscientiousness (i.e., regression from developmental norms). The fact that Cluster B features were unrelated to the development of the interpersonal trait dimensions may seem surprising. However, some of the prominent PDs captured in this Cluster (i.e., BPD) have shown inconsistent interpersonal profiles (see Wright et al., 2010 for a review), and other PDs have contrasting associations with affiliation (e.g., Narcissistic PD vs. Histrionic PD). Finally, Cluster C PD feature remission was associated with increases in Dominance and more rapid decline in Neuroticism, but onset was associated with declines in Dominance, Affiliation, and increases in Neuroticism. Thus, those who develop Cluster C PD features over time end of college being less affiliative, more submissive and neurotic. These findings mesh well with past results that have shown strong associations with Neuroticism and Submissiveness in the Cluster C PDs.

It is important to recognize that although these techniques treat personality traits and their rate of change as the dependent variable, they are ultimately correlationally based and therefore serve more to illuminate the links in the trajectories as opposed to establishing predictive primacy. Therefore, these results have implications for personality science and the full spectrum of personality functioning, from normal to abnormal. Although *pre*diction based models (i.e., predicting developmental trajectories going forward) may be maximally helpful for clinical scientists, *post*diction based models (i.e., examining the trajectories leading up to an outcome) are equally or more valuable from a developmental science perspective. Whether the relationship is predictive or postdictive does not detract from the fact that these results empirically demonstrate for the first time that the development of personality and its pathology are intimately linked. What is more, the postdictive nature of T3 PD features in the LSPD serving as predictors of trait trajectories should not be confused with studies that use retrospective reporting which is associated with increased bias and error. Because these traits and PD features were assessed concurrently across time in a prospective study, we can be more confident in these results. The modeling approach here distinguishes between the trait trajectories associated with remission and onset of symptoms, although additional latent modeling techniques such as linked latent-difference scores or parallel process growth models provide interesting alternative approaches (see McArdle, 2009).

Compared to the large amount of descriptive research on personality trait development, comparatively little research has examined the determinants of long term developmental change. Undoubtedly a large portion of this development is associated with biological maturation and normative socialization processes (Roberts et al., 2008). However, the fact remains that there is significant heterogeneity in the paths individuals forge as they develop

(Robins et al., 2001). Interindividual variability in change offers an exciting avenue for researchers interested in individual differences. Emerging research points to the contribution of genetics and environment at a very broad level (Hopwood et al., 2011). Still, both basic personality development models and developmental psychopathology models must account for more specific processes of change that can produce deviation from the mean trend. The results of this study attest to the importance of considering abnormal personality features and processes when studying these differences. With 9%–16% of individuals in the population meeting criteria for PD of some type (Lenzenweger, 2008), the effect of these processes are likely to be non-trivial in any study that uses a broad and representative sample. The inverse is also true; models of PD can no longer ignore the clear link with basic personality processes. These results strengthen the assertions made by clinical personality researchers who have called for a greater recognition of the role of basic personality processes (e.g., maturation) in our diagnostic nosology (Depue & Lenzenweger, 2005; Pincus & Hopwood, in press).

Our goal was not to develop specific models of PD and personality change, but instead to empirically demonstrate that normal and the abnormal aspects of personality functioning are indeed developmentally related. Nevertheless, clear conclusions can be drawn from these results. It is not just the case that as PD remits personality matures, but rather PD is reliably associated with the pattern and direction of trait development. What has also been affirmed is the view that maturation in the personality system is associated with increased functioning and health (Clarkin et al., 2007; McWilliams, 2011). Undoubtedly, contained within the months, weeks, and days that make up the years are innumerable interactions and life experiences that serve to cumulatively push and pull an individual's trajectory one way or another, inching them towards maturity or edging them towards significant problems in functioning.

Several caveats must also be considered. First, our sample was more homogeneous in age, educational achievement, and social class than the U.S. population. Perhaps the most effective way to assess the generalizability of findings is to evaluate whether prior LSPD findings have been replicated, and they have. 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 community (Johnson et al., 2000) samples. Thus, although the sample is more compressed in terms of demographic characteristics, the findings mesh with those obtained in other longitudinal PD research. Second, we note that the results obtained here are highly consistent with prior research charting the developmental trajectories of basic personality traits in young adults. 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, subjects who happen to be selected for academic achievement are not immune to psychopathology. In fact, some forms of PD might actually be enriched in such a sample (e.g., obsessive–compulsive PD, narcissistic PD). We note that 16% (or 1 in every 6) of the LSPD sample subjects was diagnosed with a formal PD by the by Wave 3 using the highly conservative IPDE. Many other subjects met intermediate levels of PD criteria (e.g., 2 or 3 criteria) indicating the presence of some degree PD disturbance of clinical intensity. Also, 45.2% of the LSPD subjects had a lifetime (or current) Axis I disorder by the end of college, and these data are consistent with the rates in the U.S. population (see Kessler, Chiu, Demler, & Walters, 2005).

Fourth, we are mindful that there are undoubtedly many predictors of rates of development in personality. Some may be a consequence of important time-varying processes of a broad (e.g., other temperament factors) or more specific (e.g., romantic relationships, developing

friendships) nature. The results of this study are at too coarse of a level of analysis to speak directly to person-environment transaction theories (Caspi & Roberts, 2001). At this broad level, our results provide the first empirical evidence that growth in PD and personality traits are dynamically related, demonstrate that the conceptual and analytic frameworks are indeed viable, and allow for future research testing more focused hypotheses.

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Table 1

Unconditional Growth Models for the Five Personality Dimensions

	Elevation (Intercept) of Individual Trajectory			Rate of Change (Slope) of Individual Trajectory			Variance Components								
	Fixed Effect	***p***	**ES r**	**Fixed Effect**	***p***	**ES *r***	**Residual**	***p***	**Intercept**	***p***	**Slope**	***p***	**Covariance**	***p***	**−2LL**
DOM	−0.05	0.47	0.05	0.00	0.99	0.00	0.15	0.00	1.24	0.00	0.04	0.00	−0.09	0.00	1643
LOV	**0.64**	**0.00**	**0.46**	**0.04**	**0.02**	**0.15**	0.23	0.00	1.37	0.00	0.03	0.00	−0.05	0.04	1859
CONS	0.11	0.11	0.10	**0.04**	**0.02**	**0.15**	0.16	0.00	1.11	0.00	0.03	0.00	−0.04	0.01	1645
NEUR	**−0.95**	**0.00**	**0.62**	**−0.10**	**0.00**	**0.26**	0.34	0.00	1.20	0.00	0.05	0.00	−0.05	0.12	2034
OPEN	**0.28**	**0.00**	**0.25**	**0.06**	**0.00**	**0.21**	0.19	0.00	1.06	0.00	0.04	0.00	−0.05	0.02	1734

Note. N = 250. DOM = Dominance; LOV = Affiliation; CON = Conscientiousness; NEUR = Neuroticism; OPEN = Openness; −2LL = −2 log likelihood, also known as the deviance, an index of fit. Tabled values represent the final estimates of the fixed effects with robust standard errors. The fixed effects and variance component parameters were tested to determine if they differ from zero. ES *r*, effect size *r*, .10 = small effect, .24 = medium effect, .37 = large effect (Rosenthal & Rosnow, 1991, p. 446). For all models −2LL statistics are based on six estimated parameters. Model estimation was done using full maximum likelihood with the HLM-6 program. Significant fixed effects values ($p < .05$) bolded.

Table 2

Conditional Growth Models with Cluster A Features as Predictors of Interindividual Differences in Change

	Dominance			Affiliation			Conscientiousness			Neuroticism			Openness		
	Coeff.	***p***	**ES *r***	**Coeff.**	***p***	**ES *r***	**Coeff.**	***p***	**ES *r***	**Coeff.**	***p***	**ES *r***	**Coeff.**	***p***	**ES *r***
Fixed Effects															
Elevation															
Intercept	−0.52	0.84	0.01	2.51	0.31	0.06	1.41	0.64	0.03	4.60	0.11	0.10	2.16	0.36	0.06
Age	0.03	0.83	0.01	−0.07	0.62	0.03	−0.07	0.67	0.03	**−0.30**	**0.05**	0.13	−0.10	0.44	0.05
Sex	0.23	0.13	0.10	**−0.79**	**0.00**	**0.34**	0.16	0.27	0.07	**−0.33**	**0.02**	**0.15**	−0.26	0.08	0.11
PD T1	**−0.08**	**0.00**	**0.22**	**−0.11**	**0.00**	**0.28**	−0.04	0.06	0.12	**0.09**	**0.00**	**0.25**	0.03	0.09	0.11
Rate of Change															
Intercept	−0.23	0.72	0.02	−0.63	0.28	0.07	−0.27	0.66	0.03	0.85	0.33	0.06	−0.50	0.45	0.05
Age	0.01	0.72	0.02	0.04	0.24	0.07	0.02	0.55	0.04	−0.05	0.27	0.07	0.03	0.39	0.05
Sex	0.01	0.88	0.01	−0.02	0.51	0.04	−0.05	0.13	0.10	0.01	0.75	0.02	0.05	0.15	0.09
PD T1	0.01	0.14	0.09	0.00	0.75	0.02	−0.01	0.07	0.12	0.00	0.69	0.03	−0.01	0.21	0.08
PD T3	**−0.02**	**0.05**	**0.13**	−0.01	0.37	0.06	−0.01	0.08	0.11	0.01	0.37	0.06	−0.01	0.24	0.08
Random Effects															
Intercept	1.15	0.00	--	0.99	0.00	--	1.09	0.00	--	1.05	0.00	--	1.03	0.00	--
Slope	0.03	0.00	--	0.03	0.00	--	0.02	0.00	--	0.04	0.00	--	0.03	0.00	--
Covariance	−0.09	0.00	--	−0.06	0.01	--	−0.05	0.02	--	−0.05	0.07	--	−0.05	0.05	--
Residual	0.15	0.00	--	0.23	0.00	--	0.16	0.00	--	0.34	0.00	--	0.19	0.00	--
−2LL	1613	--	--	1770	--	--	1618	--	--	1996	--	--	1723	--	--
$\Delta\chi^2$	−30	0.00	--	−89	0.00	--	−27	0.00	--	−40	0.00	--	−11	0.14	--

Note. N = 250. PD T1 = Personality disorder features at first assessment; PD T3 = Personality disorder features at final assessment. Sex is coded as 1 = males, 0 = females. Tabled values represent the final estimates of the fixed effects with robust standard errors. −2LL = −2 log likelihood, also known as the deviance, an index of fit. ES *r*, effect size *r*, .10 = small effect, .24 = medium effect, .37 = large effect (Rosenthal & Rosnow, 1991, p. 446). For all models −2LL statistics are based on 13 estimated parameters. $\Delta\chi^2$ = Chi-square test for improvement of model fit compared to unconditional model (*df*=7). Model estimation was done using full maximum likelihood with the HLM-6 program. Significant fixed effects values ($p < .05$) bolded.

Table 3

Conditional Growth Models with Cluster B Features as Predictors of Interindividual Differences in Change

	Dominance			Affiliation			Conscientiousness			Neuroticism			Openness		
	Coeff.	***p***	**ES *r***	**Coeff.**	***p***	**ES *r***	**Coeff.**	***p***	**ES *r***	**Coeff.**	***p***	**ES *r***	**Coeff.**	***p***	**ES *r***
Fixed Effects															
Elevation															
Intercept	−0.17	0.95	0.00	1.70	0.47	0.05	0.88	0.77	0.02	**5.92**	**0.04**	**0.13**	2.49	0.29	0.07
Age	0.00	0.98	0.00	−0.03	0.84	0.01	−0.04	0.81	0.02	**−0.37**	**0.01**	**0.16**	−0.12	0.36	0.06
Sex	0.05	0.75	0.02	**−0.85**	**0.00**	**0.33**	0.18	0.23	0.08	**−0.37**	**0.01**	**0.17**	−0.26	0.07	0.12
PD T1	**0.03**	**0.01**	**0.18**	**−0.04**	**0.01**	**0.16**	**−0.03**	**0.00**	**0.19**	**0.07**	**0.00**	**0.40**	0.02	0.06	0.12
Rate of Change															
Intercept	−0.39	0.56	0.04	−0.68	0.25	0.80	−0.30	0.62	0.03	0.69	0.43	0.05	−0.64	0.34	0.06
Age	0.02	0.55	0.04	0.04	0.20	0.09	0.02	0.53	0.04	−0.04	0.36	0.06	0.04	0.32	0.06
Sex	0.00	0.98	0.00	−0.01	0.72	0.02	**−0.06**	**0.05**	**0.12**	0.02	0.61	0.03	0.03	0.40	0.05
PD T1	0.00	0.46	0.05	0.00	0.70	0.00	0.00	0.32	0.06	**−0.01**	**0.00**	**0.19**	0.00	0.49	0.04
PD T3	0.00	0.74	0.02	−0.01	0.12	0.02	**−0.01**	**0.01**	**0.18**	**0.02**	**0.00**	**0.19**	0.01	0.07	0.12
Random Effects															
Intercept	1.19	0.00	--	1.09	0.00	--	1.07	0.00	--	0.93	0.00	--	1.03	0.00	--
Slope	0.04	0.00	--	0.03	0.00	--	0.02	0.00	--	0.04	0.00	--	0.04	0.00	--
Covariance	−0.09	0.00	--	−0.07	0.01	--	−0.05	0.01	--	−0.04	0.17	--	−0.05	0.02	--
Residual	0.15	0.00	--	0.23	0.00	--	0.16	0.00	--	0.34	0.00	--	0.19	0.00	--
−2LL	1631	--	--	1786	--	--	1621	--	--	1966	--	--	1722	--	--
$\Delta\chi^2$	−12	0.10	--	−12	0.10	--	−24	0.00	--	−68	0.00	--	−12	0.10	--

Note. N = 250. PD T1 = Personality disorder features at first assessment; PD T3 = Personality disorder features at final assessment. Sex is coded as 1 = males, 0 = females. Tabled values represent the final estimates of the fixed effects with robust standard errors. −2LL = −2 log likelihood, also known as the deviance, an index of fit. ES *r*, effect size *r*, .10 = small effect, .24 = medium effect, .37 = large effect (Rosenthal & Rosnow, 1991, p. 446). For all models −2LL statistics are based on 13 estimated parameters. $\Delta\chi^2$ = Chi-square test for improvement of model fit compared to unconditional model (*df*=7). Model estimation was done using full maximum likelihood with the HLM-6 program. Significant fixed effects values ($p < .05$) bolded.

Table 4

Conditional Growth Models with Cluster C Features as Predictors of Interindividual Differences in Change

	Dominance			Affiliation			Conscientiousness			Neuroticism			Openness		
	Coeff.	***p***	**ES *r***	**Coeff.**	***p***	**ES *r***	**Coeff.**	***p***	**ES *r***	**Coeff.**	***p***	**ES *r***	**Coeff.**	***p***	**ES *r***
Fixed Effects															
Elevation															
Intercept	−0.50	0.84	0.01	2.47	0.31	0.06	1.46	0.62	0.03	4.39	0.13	0.10	2.23	0.35	0.06
Age	0.03	0.83	0.01	−0.06	0.62	0.03	−0.07	0.66	0.03	**−0.30**	**0.05**	**0.12**	−0.10	0.45	0.05
Sex	0.16	0.27	0.07	**−0.89**	**0.00**	**0.36**	0.14	0.32	0.06	**−0.30**	**0.02**	**0.14**	−0.22	0.12	0.10
PD T1	**−0.06**	**0.00**	**0.19**	**−0.07**	**0.00**	**0.24**	**−0.04**	**0.01**	**0.16**	**0.14**	**0.00**	**0.35**	0.00	0.83	0.01
Rate of Change															
Intercept	−0.38	0.56	0.04	−0.66	0.25	0.07	−0.36	0.55	0.04	0.98	0.22	0.08	−0.60	0.37	0.06
Age	0.02	0.55	0.04	0.04	0.20	0.08	0.02	0.46	0.05	−0.06	0.17	0.09	0.03	0.33	0.06
Sex	−0.01	0.87	0.01	−0.03	0.46	0.05	**−0.07**	**0.03**	**0.13**	0.03	0.50	0.04	0.03	0.34	0.06
PD T1	**0.01**	**0.04**	**0.13**	0.00	0.28	0.07	0.00	0.55	0.04	**−0.02**	**0.00**	**0.20**	0.00	0.40	0.05
PD T3	**−0.02**	**0.02**	**0.15**	**−0.02**	**0.00**	**0.19**	−0.01	0.25	0.07	**0.03**	**0.00**	**0.22**	−0.01	0.37	0.06
Random Effects															
Intercept	1.17	0.00	--	1.07	0.00	--	1.08	0.00	--	0.87	0.00	--	1.04	0.00	--
Slope	0.03	0.00	--	0.03	0.00	--	0.02	0.00	--	0.04	0.00	--	0.04	0.00	--
Covariance	−0.10	0.00	--	−0.06	0.02	--	−0.05	0.02	--	−0.04	0.18	--	−0.04	0.04	--
Residual	0.15	0.00	--	0.24	0.00	--	0.16	0.00	--	0.34	0.00	--	0.19	0.00	--
−2LL	1616	--	--	1783	--	--	1623	--	--	1946	--	--	1728	--	--
$\Delta\chi^2$	−27	0.00	--	−76	0.00	--	−22	0.00	--	−90	0.00	--	−8	0.33	--

Note. N = 250. PD T1 = Personality disorder features at first assessment; PD T3 = Personality disorder features at final assessment. Sex is coded as 1 = males, 0 = females. Tabled values represent the final estimates of the fixed effects with robust standard errors. −2LL = −2 log likelihood, also known as the deviance, an index of fit. ES *r*, effect size *r*, .10 = small effect, .24 = medium effect, .37 = large effect (Rosenthal & Rosnow, 1991, p. 446). For all models −2LL statistics are based on 13 estimated parameters. $\Delta\chi^2$ = Chi-square test for improvement of model fit compared to unconditional model (*df*=7). Model estimation was done using full maximum likelihood with the HLM-6 program. Significant fixed effects values ($p < .05$) bolded.