



Published in final edited form as:

*Personal Disord.* 2013 July ; 4(3): 230–238. doi:10.1037/a0027773.

## A Parallel Process Growth Model of Avoidant Personality Disorder Symptoms and Personality Traits

**Aidan G. C. Wright,**

Western Psychiatric Institute and Clinic, University of Pittsburgh Medical School, The Pennsylvania State University

**Aaron L. Pincus,** and

The Pennsylvania State University

**Mark F. Lenzenweger**

The State University of New York at Binghamton and Weill Cornell Medical College

### Abstract

**Background**—Avoidant personality disorder (AVPD), like other personality disorders, has historically been construed as a highly stable disorder. However, results from a number of longitudinal studies have found that the symptoms of AVPD demonstrate marked change over time. Little is known about which other psychological systems are related to this change. Although cross-sectional research suggests a strong relationship between AVPD and personality traits, no work has examined the relationship of their change trajectories. The current study sought to establish the longitudinal relationship between AVPD and basic personality traits using parallel process growth curve modeling.

**Methods**—Parallel process growth curve modeling was applied to the trajectories of AVPD and basic personality traits from the Longitudinal Study of Personality Disorders (Lenzenweger, 2006), a naturalistic, prospective, multiwave, longitudinal study of personality disorder, temperament, and normal personality. The focus of these analyses is on the relationship between the rates of change in both AVPD symptoms and basic personality traits.

**Results**—AVPD symptom trajectories demonstrated significant negative relationships with the trajectories of interpersonal dominance and affiliation, and a significant positive relationship to rates of change in neuroticism.

**Conclusions**—These results provide some of the first compelling evidence that trajectories of change in PD symptoms and personality traits are linked. These results have important implications for the ways in which temporal stability is conceptualized in AVPD specifically, and PD in general.

### Keywords

Avoidant Personality Disorder; Personality Traits; Latent Growth Curve Analysis; Parallel Process Latent Growth Curve Analysis

---

The core pathology of avoidant personality disorder (AVPD) centers on a keen sensitivity to interpersonal rejection, exquisite fears of humiliation and judgment, and accordingly,

avoidance of social and interpersonal situations, especially when it involves new people or new situations (American Psychiatric Association, 2000). The symptoms of AVPD, like all personality disorders (PDs), have historically been construed as highly stable. However, recent empirical findings from prospective longitudinal studies suggest that symptoms of PDs generally, and AVPD specifically, demonstrate instability and plasticity over time (Grilo et al., 2004; Johnson et al., 2000; Lenzenweger, 1999; Shea et al., 2002; Zanarini, Frankenburg, Hennen, & Silk, 2003). These results immediately raise questions about which other aspects of psychological functioning are associated with an individual's AVPD symptom trajectory. Identifying other psychological systems that change in tandem with AVPD would provide strong evidence for systems that play a causal or maintenance role in the disorder (cf., Lenzenweger & Willett, 2007 for a discussion relating personality change to PD change). We propose that basic personality traits demonstrate just this pattern, and we test whether individual trajectories in AVPD and personality traits are dynamically linked over time.

A recent literature review found that, of the currently recognized PD diagnoses, AVPD was among the most prevalent and impairing across clinical and community samples, leading to the recommendation that it be retained as a diagnostic construct in DSM-5 (Skodol et al., 2011). However, of the PDs to be retained in DSM-5, AVPD has received relatively less empirical attention, which argues for more research that will elucidate the nature and mechanisms of the disorder. One proposal suggests that the diagnostic features of AVPD can be understood as rigid and maladaptive manifestations of basic personality dimensions (Alden, Lapsa, Taylor, & Ryder, 2002). Indeed, cross-sectionally, AVPD is associated with low trait Dominance and Affiliation (Wiggins & Pincus, 1989) and socially-avoidant interpersonal problems (Pincus & Wiggins, 1990). Additionally, two recent meta-analyses of the associations between PDs and the Five-Factor Model (FFM) of personality traits have found that AVPD shows a strong positive relationship with neuroticism, or the tendency to experience negative emotions (e.g., anxiety, anger, depression, guilt), and a strong negative relationship with extraversion,<sup>1</sup> or the tendency to be outgoing, gregarious, and experience positive emotions (Saulsman & Page, 2004; Samuel & Widiger, 2008). Importantly, these are among the strongest effect sizes for any of the PDs. Nevertheless, the wealth of cross-sectional associations cannot speak to the longitudinal relationship between basic personality traits and AVPD, about which very little is known.

Prospective longitudinal studies in both clinical and non-clinical samples reveal that there are significant average declines in AVPD symptoms when considered as dimensions of pathology (Lenzenweger, 1999), and significant declines in the number of individuals meeting diagnostic threshold in AVPD (50% after two years; Grilo et al., 2004). However, not all individual's decline in the number of AVPD criteria they meet over time, and a subset remain stable, while others increase over time. This is illustrated in Figure 1, which plots the estimated individual AVPD trajectories of the Longitudinal Study of Personality Disorders (LSPD, Lenzenweger, 1999; 2006) participants, the sample analyzed in this study. The empirical literature on stability and change in normal range personality traits and the processes they represent has developed separately from that of PD, but striking similarities have emerged. Once thought to be entirely stable (James, 1890), it is now understood that an individual's personality traits are indeed highly stable, but not fixed (Roberts, Walton, & Viechtbauer, 2006). Rates of mean change in broad personality traits are modest but

---

<sup>1</sup>Past research has shown that the FFM traits of Extraversion and Agreeableness can be located within the interpersonal circumplex as rotations of the primary dimensions of Dominance and Affiliation (McCrae & Costa, 1989; Pincus, 2002). For the purposes of the current study, it is important to note that low Extraversion is a blend of low Dominance *and* low Affiliation. Thus, variables with a negative relationship with Extraversion would in turn be expected to have a negative relationship with *both* Dominance and Affiliation.

significant, and this normative change masks significant interindividual heterogeneity in individual trajectories around the population's mean rate of change (Mroczek & Spiro, 2003; Vaidya Gray, Haig, Mroczek, & Watson, 2008; Wright, Pincus, & Lenzenweger, 2011).

Thus, the finding that both AVPD and basic personality traits are not as stable as once thought suggests that the observed cross-sectional relationship between the two may extend to a developmental relationship across time. To date, this question remains mostly unexplored and therefore the developmental relationship between these systems remains unknown.<sup>2</sup> If the rate of change in AVPD and personality were shown to be significantly related, it would represent an important advance in the science of personality and its pathology, suggesting that these constructs are developmentally linked. This would provide strong evidence in favor of a unified science of personality and psychopathology, allowing for more confident assertions that normal personality traits and PD comprise manifestations of the same psychological system (Depue & Lenzenweger, 2005; Pincus & Hopwood, in press; Widiger & Trull, 2007).

## The Current Study

The current investigation sought to evaluate whether the longitudinal trajectories of AVPD and personality traits were linked using data from the LSPD. The LSPD is ideally suited to study this question as it is a naturalistic study and designed to include individuals who were at a putative risk for PD pathology at the outset and also individuals who exhibited no significant pathology, but might develop symptoms over the course of the study (see Lenzenweger, 2006). Our analytic approach was designed to measure multivariate change (i.e., simultaneous change in multiple dimensions or systems) through the use of parallel process growth curve modeling (sometimes known as associative or multivariate growth curve modeling, Bollen & Curran, 2006). In latent growth curve models (LGM), an individual's scores at each time point are modeled as a function of latent growth factors that are estimated from the observed scores. With three time points, linear rates of change can be estimated with one latent factor for the intercept, or level of the curve at the measurement time of the intercept, and another latent growth factor is estimated for the slope, or rate of change. Parallel process LGMs are so called because they chart trajectories of change or growth processes in two or more variables in parallel. By allowing for associations among the growth factors of each parallel set of variables, it is possible to examine whether the intercept and growth in one is related to the intercept and growth in the others. This offers a very powerful analytic approach for the study of stability, change, and development across time in multiple psychological variables.

This analytic framework allowed us to test whether changes in AVPD symptoms and personality traits are related to each other. Our analyses focused on AVPD symptomatology, the interpersonal traits of Dominance and Affiliation, and the remaining FFM traits of Conscientiousness, Neuroticism, and Openness. We hypothesized that the same traits that demonstrate significant cross-sectional relationships (i.e., Dominance, Affiliation, and Neuroticism) with AVPD symptoms would demonstrate the same dynamic relationships over time. This is the first study to directly examine the conjoint change in personality traits and AVPD symptoms using parallel process growth curve models.

---

<sup>2</sup>The lone study to examine AVPD and personality traits over time found that personality traits predicted subsequent AVPD symptoms one year later (Warner et al., 2004). Although informative, this work did not test whether the rates of change in each system directly corresponded.

## Method

### Participants

The 258 LSPD participants were drawn from a population of 2,000 first-year undergraduates. Detail concerning the initial participant selection procedure and sampling is given elsewhere (Lenzenweger, 2006; Lenzenweger et al., 1997). The 258 participants were balanced on gender (53% Female). The mean age of the participants at study entry was 18.88 years ( $SD = 0.51$ ). Participants were assessed at their first, second, and fourth years of college at Cornell University (Ithaca, NY). Cornell University consists of both private colleges as well as statutory units of the State University of New York, which provides for a more diverse student population as contrasted with many purely private universities and/or colleges. All participants gave voluntary written informed consent and received an honorarium of \$50.00 at each wave. These data were collected and analyzed with the full approval of the Institutional Review Boards at Cornell University and the Pennsylvania State University respectively. Of the initial 258 participants, 250 completed all three assessment waves and are included in these analyses. Six left the study, and two died in automobile accidents.

### Procedure

**Structure of the LSPD Dataset**—As noted above, the LSPD has a prospective, multiwave, longitudinal design with participants evaluated at three points in time (i.e., first, second, and fourth years in college). At each time point, participants completed self-report measures of personality and clinical assessments were conducted by experienced Ph.D. or advanced M.S.W. clinicians. The LSPD selected approximately half of the included individuals based on putative positive PD status as assessed by a self-report measure of PD symptoms and the other half based on their current lack of pathology (see Lenzenweger et al., 1997). This method was employed to ensure a balance among those with possible current PD pathology, and those who might develop it over time. At Wave 1, 11% of the participants qualified for an Axis II diagnosis of some sort based on clinical interviews. The raw rates of diagnosed PDs in the LSPD sample at 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%.

### Measures

**International Personality Disorder Examination (IPDE)**—The IPDE (Loranger, 1988, 1999) was used as the PD measure in this study. The IPDE has excellent psychometric properties, and it has been shown to be robust to mental state (anxiety, depression) changes. The DSM-III-R criteria were assessed in this study because these were the criteria in effect at the time the LSPD was undertaken. We note the DSM-III-R and DSM-IV criteria bear considerable resemblance to one another and the fundamental PD constructs are the same in both nomenclatures. The interrater reliability for IPDE assessments (based on intraclass correlation coefficients) was excellent, 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. The IPDE yields categorical PD diagnoses and PD dimensional scores. The IPDE AVPD dimensional scores were used for this study as they are the metrics that will capture changes in the most sensitive manner.

**Revised Interpersonal Adjective Scales – Big Five (IASR-B5)**—The IASR-B5 (Trapnell & Wiggins, 1990) contains 124 trait descriptive adjectives rated on a 0 to 8 scale that provides scores for the personality trait dimensions of dominance, affiliation,

conscientiousness, neuroticism, and openness. Participants completed the IASR-B5 at each assessment wave of the LSPD. Coefficient alphas for all scales at all waves ranged from .82 to .96.

## Data Analysis

We estimated parallel process LGMs in Mplus 6.11 (Muthén & Muthén, 2010). Figure 2 provides a conceptual diagram of the estimated models with the paths labeled for ease of communication. The loadings for the waves of measurement on the slope factor were fixed to 0.0 for Wave 1, thereby setting the start of the study as the intercept, followed by the mean assessment time between waves for the remaining two loadings (i.e., .95 for Wave 2, 2.82 for Wave 3). Of most interest in this study is the covariance between the slope factors (i.e., growth rates), indicating the degree to which change in AVPD symptoms and personality traits are associated with each other (Path B in Figure 2). The estimated relationship between intercepts (Path A) captures the cross-sectional association between traits and AVPD at the start of the study. The estimated growth models included a series of important covariates. First, slope factors within each system were regressed on the corresponding intercepts in order to control for any spurious association in the relationship between the growth factors (e.g., regression to the mean; Paths C and D). Second, the effect of sex, age-of-entry to the study, and sampling group are included as covariates of the trajectories of change by regressing the latent growth factors on each. Age of entry was included to account for potentially important individual heterogeneity in development at the outset of the study because standard LGMs treat all individuals as starting at the same time. Sex was included as it demonstrates a significant relationship to basic personality traits (Costa et al., 2001). Sampling group is included as a covariate to account for the LSPD's selection strategy. Finally, we tested whether initial status on personality or AVPD had an effect on the rate of change in the other after controlling for the above mentioned covariates (Paths E and F). The AVPD symptom counts were modeled using a Poisson distribution. Symptom counts of psychiatric disorders rarely follow a normal distribution (which is assumed in standard SEM), and significant violations of this assumption can lead to problems in estimation and severe misspecification of model parameters if a more appropriate distribution is not employed (e.g., the prediction of negative values; Atkins & Gallop, 2007; Cameron & Trivedi, 1998).

We estimated five parallel process LGMs, one for each personality dimension of the IASR-B5. Individual models were required because attempts to estimate a model with growth in all five personality dimensions and AVPD symptoms modeled simultaneously led to problems with specification (i.e., negative residual variances), which is not uncommon in very large structural models (Kline, 2011). When growth is modeled using a Poisson distribution, traditional SEM fit indices (e.g., chi-square, root mean square error of approximation [RMSEA], comparative fit index [CFI], etc.) are not available. Therefore the log-likelihood and indices of relative fit, the Akaike (AIC) and Bayesian information criteria (BIC) are provided. A careful evaluation of the resulting parameter estimates indicates that all variances were positive and resulting parameter estimates were plausible (e.g., no Heywood cases). Finally, Table 4.1 provides the raw parameter coefficients and standard errors. The parameters associated with the AVPD symptoms are on the logit scale, making direct interpretation difficult. When exponentiated these values provide the estimated counts, but leave parameters associated with the growth factors (e.g., covariance of personality variables vs. covariance of PD counts) on vastly different scales. Therefore, to aid with interpretability, the estimated effect size on the  $r$  metric is also provided (where  $r = \sqrt{(\chi^2)/(df)}$  see Rosenthal & Rosnow, 1991).

## Results

Past work with the LSPD sample has shown that on the average, AVPD symptoms decrease over the course of the study, but prior work did not find statistically significant heterogeneity in individual trajectories (Lenzenweger et al., 2004) despite the compelling plots of individual trajectories (see Figure 1 for a representation of the heterogeneity in intercept and slope using individually estimated OLS regression lines). Significant heterogeneity in individual rates of change (i.e., slopes) in AVPD symptoms over time is an important precondition for the parallel process models. However, the previously reported results were estimated using a standard growth model whose distributional assumptions may not have been adequate for the estimation of count distributed variables leading to potential misspecifications in model parameters. Therefore, as an initial step, we estimated an unconditional Poisson growth model in AVPD to more accurately capture the distribution of the AVPD feature counts. Results for the means of the growth parameters (intercept  $ES\ r = .39$ ,  $p < .001$ ; slope  $ES\ r = -.19$ ,  $p = .002$ ) were highly consistent with the previously reported model leading to the same interpretation of the mean rate of change (i.e., modest decline over the course of the study). Importantly, significant variability was found in the intercept and slope parameters ( $p$ 's  $< .001$ ) indicating that there is individual heterogeneity in starting values and trajectories of change when estimated using a Poisson based LGM. Using the BIC to compare the fit of models without (BIC = 1889.66) and with (BIC = 1379.72) randomly varying slopes confirms that the estimation of these extra parameters is warranted. Also in this sample, we previously found that on average, Affiliation ( $ES\ r = .15$ ,  $p = .02$ ), Conscientiousness ( $ES\ r = .15$ ,  $p = .02$ ), and Openness ( $ES\ r = .21$ ,  $p < .01$ ) increased over the course of the study waves, whereas Dominance ( $ES\ r = .00$ ,  $p = .99$ ) showed no mean change, and Neuroticism ( $ES\ r = -.26$ ,  $p < .01$ ) decreased significantly (Wright et al., 2011). Results also indicated that there was significant individual variability around mean personality dimension trajectories (all  $p$ 's  $< .01$ ). We have briefly summarized the previously published findings' effect sizes to provide a context for this study's results.

The resulting parameter values of the parallel process growth models are presented in Table 1. Age, as a covariate, was never a significant predictor of the intercepts or slopes in either personality or AVPD. Male sex was associated with lower Affiliation and Neuroticism intercepts, but sex was otherwise not predictive of intercepts or slopes. Those individuals selected for the study based on a positive putative PD status demonstrated higher AVPD and Neuroticism intercepts, lower Affiliation and Conscientiousness intercepts, and steeper declines in Neuroticism, but otherwise sampling group was unassociated with study parameters. AVPD intercept was significantly associated with lower Dominance, Affiliation, and Openness, and higher Neuroticism intercepts (A Paths). The rate of change in AVPD was significantly negatively related to the rates of change in Dominance and Affiliation, and positively to Neuroticism (B Paths). The rate of change in AVPD was never related to initial AVPD status (D Paths), whereas initial personality status was significantly negatively related to slope for all traits with the exception of Affiliation (C Paths). AVPD intercept was unrelated to personality trait trajectories (E Paths). Finally, only initial status on Openness positively predicted rate of change in AVPD symptoms (F Paths).

## Discussion

In this study we tested whether the rates of change in AVPD symptoms and five normal personality trait dimensions were related over the course of approximately 3 years. The results revealed that individual trajectories in AVPD symptoms were indeed associated with rates of change in personality traits. This is the first study to demonstrate such a dynamic relationship for any PD and provides an important contribution to the science of personality

and the psychopathology of PD by demonstrating personality traits and PD symptoms are developmentally linked.

Specifically, we found that at the outset of the study, an individual's level of AVPD symptoms was related to higher neuroticism (i.e., the A Paths in Figure 2), but lower dominance, affiliation, and openness, replicating the results of many prior studies (see Alden et al., 2002; Saulsman & Page, 2004; Samuel & Widiger, 2008 for reviews). Replication of these well-known cross-sectional associations in the LSPD provides confidence in the novel results associated with the growth factor relationships, the primary target of this study. In terms of the relationship between the growth factors, we found that the rate of change in AVPD symptoms was associated with the rate of change in Dominance, Affiliation, and Neuroticism. Notably, these relationships are in the same directions as was found in the intercepts. Individuals' AVPD symptoms decrease as they become more dominant (i.e., more self-assured, assertive, and less submissive), more affiliative (i.e., more cooperative, engaging, less aloof), and less neurotic (i.e., less inclined to experience negative emotions and anxiety). The reverse is also true, such that as trait dominance and affiliation decline and neuroticism increases, AVPD symptoms increase.

An individual's initial status on AVPD was not predictive of the rates of change in personality traits, nor was an individual's initial status on personality traits predictive of AVPD change, with the exception of openness. Lower in openness at the outset of the study predicted more rapid declines in AVPD symptoms. This relationship may best be understood in the context of the mean trend of decline in AVPD symptoms that has been reported earlier (Lenzenweger, 1999), and is likely reflective of the law of initial values, whereby those who are more extreme in an initial value exhibit more change. However, this was the only trait for which this occurred, was not predicted, and therefore we suggest it be interpreted cautiously.

By looking longitudinally within a LGM framework, this study adopts a more person-centered approach in the study of the relationship between personality and its pathology. This is because the variability in intercept and slope across individuals is modeled using growth factors, and not simply reduced to a mean as might be the case in an ANOVA. These longitudinal data capture the very important dynamic interplay between an individual's personality and AVPD symptoms. Although each person experiences changes in personality and PD symptoms, the rates and patterns of change vary across individuals. Past work has examined the heterogeneity in AVPD and personality growth separately, but this is the first evidence that they are dynamically linked in the paths they take. The longitudinal relationships observed here are consistent with hypotheses based on the diagnostic features of AVPD, the aspects of personality measured by these traits, and prior cross-sectional work. Other work using the LSPD employed similar models to investigate the longitudinal relationship between neurobehavioral markers and Cluster C symptoms (Lenzenweger & Willett, 2007) and between temperament and schizoid PD features (Lenzenweger & Willett, 2009). However, no longitudinal relationship was found among the normal range scales and the PD symptoms in those studies. In the case of the Cluster C PD feature change examined by Lenzenweger and Willett (2007), it is possible that they were at too coarse a level of analysis (i.e., including dependent and obsessive-compulsive PD symptomatology) to capture this change that is more specific to AVPD. Schizoid PD possibly represents a variant or early marker of schizophrenia and thus the maintenance process is likely less related to personality traits (Lenzenweger, 2010; Lenzenweger & Willett, 2009).

From a psychopathology perspective, these results offer compelling evidence that normal personality and personality pathology should be understood as expressions of the same system, at least as it pertains to AVPD. This implication is not minor, as the modern

empirical literatures in each are often blind to the other's advances. A number of theorists have called for greater synthesis in conceptualizing basic personality and personality disorder, with theoretical proposals spanning the neurobiological (Depue & Lenzenweger, 2001, 2005), temperament (Clark, 2007), psychometric (Widiger & Trull, 2007), and interpersonal (Pincus & Hopwood, in press) domains. Each of these theoretical perspectives, although distinct in some respects, is also quite similar in others (Widiger & Simonsen, 2005). The findings reported here bolster this argument, and provide novel evidence for the notion that personality and its disorder belong within a coherent and comprehensive model of normal and abnormal functioning (Pincus, 2011; Wright, 2011).

## Limitations

Several caveats must be considered with these data and analyses. First, despite the impressive ability of these models to capture association between changes over time in both systems, they do not determine the direction of causality among them. It remains to be determined whether personality changes drive symptom changes, or vice versa. Undoubtedly, contained within the days, weeks, and months 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. Additionally, 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).

In terms of the data, the present sample was clearly more homogenous in age, educational achievement, and social class than the U.S. population at large. 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 nonclinical community samples and the U.S. general population (e.g., NCS-R, Lenzenweger, Lane, Loranger, & Kessler, 2007; see Lenzenweger, 2008 for a review). 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 more compressed in terms of demographic background characteristics, the findings accord with those obtained in other epidemiological and longitudinal PD research. Second, given that the LSPD subjects were selected from a population of first-year university students, the sample may have been 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. 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 thresholds. An additional strength of these results is that they are based on clinical interviews, and self-reported personality traits, and therefore the method of assessment is not confounded.

Third, we are mindful that there are undoubtedly many predictors of rates of development in personality and change in PD symptoms. Some change may be driven by important time-varying processes of a broad (e.g., other temperament factors) or more specific nature (e.g., romantic relationships, developing friendships). Finally, we emphasize that these analyses measure personality traits broadly as opposed to more specifically. Future analyses might go beyond the personality trait domains to look at the more specific facet/octant level of analysis. Past research would suggest that this might be a fruitful avenue for future investigation (Samuel & Widiger, 2008).



## Conclusion

In this study we demonstrated that the rates of change in AVPD symptoms and personality traits are directly related to each other during a critical developmental period for the diagnosis of PD. This is the first demonstration of this link and is important in the context of enduring efforts to unify the study of basic personality/temperament and PD. The multidimensional growth in personality and PD across the span of 3 years argues strongly for the close conceptual relationship between basic traits and the problematic expression of functioning that is captured by DSM defined PD criteria. The findings reported here confirm that the cross sectional relationships between traits and PD criteria are further observed in the developmental trajectories in each. The developmental patterns and pathways of PDs remain poorly understood and require systematic investigation. The current study is an initial effort in this regard and provides a model for future studies that seek to further illuminate the covariates, predictors, and patterns of PD development. Anticipated future data collection for the LSPD (i.e., Wave IV), when the study subjects will be approaching age 40, will allow for additional examination of the relations we discovered for AVPD in the current LSPD database.

## Acknowledgments

This research was supported by grants (MH45448, Lenzenweger; F31MH087053, Wright) from the National Institute of Mental Health, Washington, DC. The views and opinions contained within are solely those of the authors and do not reflect the official position of the funding source.

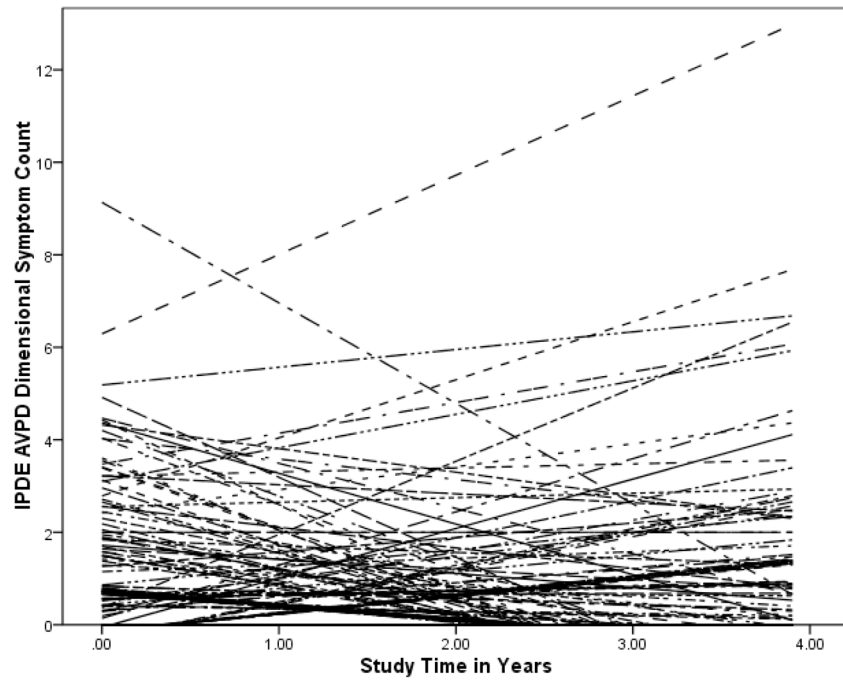
This manuscript reports on work that represents a portion of the first author's dissertation. We thank Jerry S. Wiggins for providing consultation on the initial use of the Revised Interpersonal Adjectives Scales – Big Five. We are grateful to Lauren Korfine for project coordination in the early phase of the study, and for comments on an earlier draft by Michael N. Hallquist.

## References

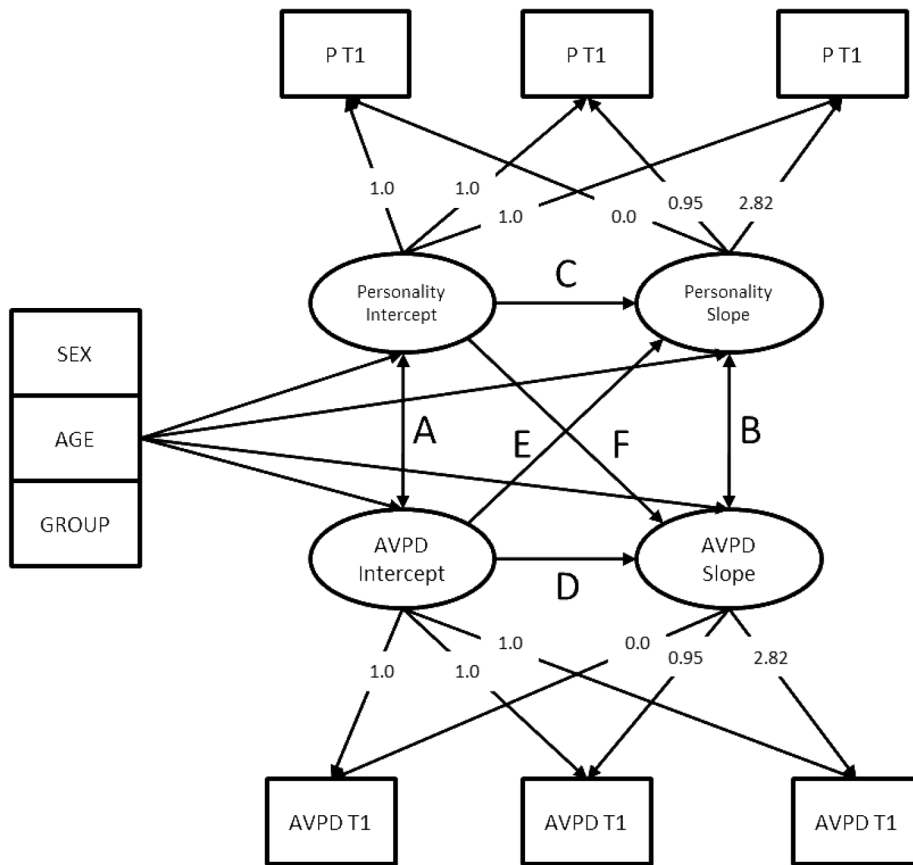
- Alden LE, Laposa JM, Taylor CT, Ryder AG. Avoidant personality disorder: Current status and future directions. *Journal of Personality Disorders*. 2002; 16:1–29. [PubMed: 11881158]
- American Psychiatric Association. *Diagnostic and statistical manual of mental disorders*. 4. Author; Washington, DC: 2000. Text Revision
- Atkins DC, Gallo RJ. Rethinking how family researchers model infrequent outcomes: A tutorial on count regression and zero-inflated models. *Journal of Family Psychology*. 2007; 21:726–735. [PubMed: 18179344]
- Bollen, KA.; Curran, PJ. *Latent curve models: A structural equation perspective*. John Wiley & Sons; Hoboken: 2006.
- Cameron, AC.; Trivedi, PK. *Regression analysis of count data*. Cambridge: Cambridge University Press; 1998.
- Caspi A, Roberts BW. Target article: Personality development across the life course: The argument for change and continuity. *Psychological Inquiry*. 2001; 12:49–66.
- Clark LA. Assessment and diagnosis of personality disorder: Perennial issues and an emerging reconceptualization. *Annual Review of Psychology*. 2007; 58:227–257.
- Costa PT, Terracciano A, McCrae RR. Gender differences in personality traits across cultures: Robust and surprising findings. *Journal of Personality and Social Psychology*. 2001; 81:322–331. [PubMed: 11519935]
- Depue, RA.; Lenzenweger, MF. A neurobehavioral dimensional model of personality disturbance. In: Lenzenweger, MF.; Clarkin, JF., editors. *Major theories of personality disorder*. 2. Guilford Press; New York, NY: 2005. p. 391-453.
- Grilo CM, Shea TM, Sanislow CA, Skodol AE, Gunderson JG, McGlashan TH. Two-year stability and change of schizotypal, borderline, avoidant, and obsessive-compulsive personality disorders. *Journal of Consulting and Clinical Psychology*. 2004; 72:767–775. [PubMed: 15482035]

- James W. The principles of psychology. 1890
- Johnson JG, Cohen P, Kasen S, Skodol AE, Hamagami F, Brook JS. Age-related change in personality disorder trait levels between early adolescence and adulthood: A community-based longitudinal investigation. *Acta Psychiatrica Scandinavica*. 2000; 102:265–275. [PubMed: 11089726]
- Kline, R. Principles and practices of structural equation modeling. 3. Guilford; New York: 2011.
- Lenzenweger MF. Stability and change in personality disorder features: The longitudinal study of personality disorders. *Archives of General Psychiatry*. 1999; 56:1009–1015. [PubMed: 10565501]
- Lenzenweger MF. The longitudinal study of personality disorders: History, design considerations, and initial findings. *Journal of Personality Disorders*. 2006; 20:645–670. [PubMed: 17192142]
- Lenzenweger MF. Epidemiology of personality disorders. *Psychiatric Clinics of North America*. 2008; 31:395–403. [PubMed: 18638642]
- Lenzenweger, MF. Schizotypy and schizophrenia: The view from experimental psychopathology. Guilford Press; New York: 2010.
- Lenzenweger MF, Johnson MD, Willett JB. Individual growth curve analysis illuminates stability and change in personality disorder features: The longitudinal study of personality disorders. *Archives of General Psychiatry*. 2004; 61:1015–1024. [PubMed: 15466675]
- Lenzenweger MF, Loranger AW, Korfine L, Neff C. Detecting personality disorders in a nonclinical population: Application of a 2-stage for case identification. *Archives of General Psychiatry*. 1997; 54:345–351. [PubMed: 9107151]
- Lenzenweger MF, Willett JB. Predicting individual change in personality disorder features by simultaneous individual change in personality dimensions linked to neurobehavioral systems: The longitudinal study of personality disorders. *Journal of Abnormal Psychology*. 2007; 116:684–700. [PubMed: 18020716]
- Lenzenweger MF, Willett JB. Does change in temperament predict change in schizoid personality disorder? A methodological framework and illustration from the Longitudinal Study of Personality Disorders. *Development and Psychopathology*. 2009; 21:1211–1231. [PubMed: 19825265]
- Loranger, A. The Personality Disorder Examination (PDE) manual. DV Communications; Yonkers: 1988.
- Loranger, AW. *International Personality Disorder Examination: DSM-IV and ICD-10 Interviews*. Psychological Assessment Resources Inc; Odessa: 1999.
- McCrae RR, Costa PT. The structure of interpersonal traits: Wiggins's circumplex and the five-factor model. *Journal of Personality and Social Psychology*. 1989; 56(4):586–595. [PubMed: 2709308]
- Mroczek DK, Spiro A. Modeling intraindividual change in personality traits: Findings from the normative aging study. *The Journals of Gerontology: Series B: Psychological Sciences and Social Sciences*. 2003; 58B:P153–P165.
- Muthén, LK.; Muthén, BO. Mplus User's Guide. Sixth Edition. Muthén & Muthén; Los Angeles: 1998–2010.
- Pincus, AL. Constellations of dependency within the Five-Factor Model of personality. In: Costa, PT., Jr; Widiger, TA., editors. *Personality Disorders and the Five-Factor Model of Personality*. 2. Washington, DC: American Psychological Association; 2002. p. 203-214.
- Pincus AL. Some comments on nomology, diagnostic process, and narcissistic personality disorder in the DSM-5 proposal for personality and personality disorders. *Personality Disorders: Theory, Research, and Treatment*. 2011; 2:41–53.
- Pincus, AL.; Hopwood, CJ. A contemporary interpersonal model of personality pathology and personality disorder. In: Widiger, TA., editor. *The Oxford handbook of personality disorders*. Oxford University Press; New York: in press
- Pincus AL, Wiggins JS. Interpersonal problems and conceptions of personality disorders. *Journal of Personality Disorders*. 1990; 4:342–352.
- Roberts BW, Walton KE, Viechtbauer W. Patterns of mean-level change in personality traits across the life course: A meta-analysis of longitudinal studies. *Psychological Bulletin*. 2006; 132:1–25. [PubMed: 16435954]
- Samuel DB, Widiger TA. A meta-analytic review of the relationships between the five-factor model and DSM-IV-TR personality disorders: A facet level analysis. *Clinical Psychology Review*. 2008; 28:1326–1342. [PubMed: 18708274]

- Saulsman LM, Page AC. The five-factor model and personality disorder empirical literature: A meta-analytic review. *Clinical Psychology Review*. 2004; 23:1055–1085. [PubMed: 14729423]
- Shea MT, Stout R, Gunderson J, Morey LC, Grilo CM, McGlashan T, ... & Keller MB. Short-term diagnostic stability of schizotypal, borderline, avoidant, and obsessive-compulsive personality disorders. *The American Journal of Psychiatry*. 2002; 159:2036–2041. [PubMed: 12450953]
- Skodol AE, Bender DS, Morey LC, Clark LA, Oldham JM, Siever LJ. Personality disorder types proposed for the DSM-5. *Journal of Personality Disorders*. 2011; 25:136–169. [PubMed: 21466247]
- Trapnell PD, Wiggins JS. Extension of the interpersonal adjective scales to include the big five dimensions of personality. *Journal of Personality and Social Psychology*. 1990; 59:781–790.
- Vaidya JG, Gray EK, Haig JR, Mroczek DK, Watson D. Differential stability and individual growth trajectories of big five and affective traits during young adulthood. *Journal of Personality*. 2008; 76:267–304. [PubMed: 18331279]
- Warner MB, Morey LC, Finch JF, Gunderson JG, Skodol AE, Sanislow CA, ... & Grilo CM. The longitudinal relationship of personality traits and disorders. *Journal of Abnormal Psychology*. 2004; 113:217–227. [PubMed: 15122942]
- Widiger TA, Simonsen E. Introduction to part two of the special section on the research agenda for the development of a dimensional classification of personality disorder. *Journal of Personality Disorders*. 2005; 19:211–211.
- Widiger TA, Trull TJ. Plate tectonics in the classification of personality disorder: Shifting to a dimensional model. *American Psychologist*. 2007; 62:71–83. [PubMed: 17324033]
- Wiggins JS, Pincus AL. Conceptions of personality disorders and dimensions of personality. *Psychological Assessment: A Journal of Consulting and Clinical Psychology*. 1989; 1:305–316.
- Wright AGC. Qualitative and quantitative distinctions in personality disorder. *Journal of Personality Assessment*. 2011; 93:370–379. [PubMed: 22804676]
- Wright AGC, Pincus AL, Lenzenweger MF. Development of personality and the remission and onset of personality pathology. *Journal of Personality and Social Psychology*. 2011; 101:1351–1358. [PubMed: 21967009]
- Zanarini MC, Frankenburg FR, Hennen J, Silk KR. The longitudinal course of borderline psychopathology: 6-year prospective follow-up of the phenomenology of borderline personality disorder. *The American Journal of Psychiatry*. 2003; 160:274–283. [PubMed: 12562573]



**Figure 1.**  
Plot of Individual Ordinary Least Squares Growth Trajectories  
*Note.* IPDE = International Personality Disorder Examination; AVPD = Avoidant Personality Disorder



**Figure 2.** Conceptual Diagram of Parallel Process Growth Model.  
*Note.* AVPD = Avoidant Personality Disorder Symptoms; P = Personality Trait Score; T1–T3 = Study Wave 1–3; Single headed arrows denote regression paths, double headed arrows denote covariances. Path A = Covariance between personality and AVPD intercepts; Path B = Covariance between personality and AVPD slopes; Path C = Personality slope regressed on personality intercept; Path D = AVPD slope regressed on AVPD intercept; Path E = Personality slope regressed on AVPD intercept; Path F = AVPD slope regressed on personality intercept.

**Table 1**

Parameter estimates and indices of fit for the five estimated parallel process growth models.

	Dominance			Affiliation			Conscientiousness			Neuroticism			Openness		
	Coef.	S.E.	ES r	Coef.	S.E.	ES r	Coef.	S.E.	ES r	Coef.	S.E.	ES r	Coef.	S.E.	ES r
<i>Model Intercepts</i>															
AVPD Intercept	-3.514	4.136	0.05	-3.947	4.251	0.06	-3.816	4.350	0.06	-5.330	4.330	0.08	-3.377	4.244	0.05
AVPD Slope	0.142	2.853	0.00	0.185	2.674	0.00	0.242	2.825	0.01	1.381	2.704	0.03	-0.485	2.753	0.01
Personality Intercept	-0.617	2.686	0.01	3.635	2.157	0.11	2.076	2.986	0.04	2.828	2.549	0.07	2.246	2.348	0.06
Personality Slope	-0.502	0.552	0.06	-0.450	0.554	0.05	-0.315	0.552	0.04	1.544	0.887	0.11	-0.509	0.614	0.05
<i>Growth Factor Associations</i>															
Path A	-0.831***	0.153	0.33	-0.344**	0.110	0.19	-0.173	0.136	0.08	0.648***	0.129	0.30	-0.366***	0.138	0.17
Path B	-0.034*	0.016	0.13	-0.041*	0.019	0.13	-0.017	0.015	0.07	0.061**	0.022	0.18	-0.015	0.016	0.06
Path C	-0.086**	0.026	0.20	-0.044	0.037	0.07	-0.055**	0.020	0.17	-0.097*	0.045	0.13	-0.080***	0.020	0.25
Path D	0.043	0.100	0.03	0.031	0.078	0.03	0.043	0.080	0.03	0.048	0.088	0.03	0.064	0.072	0.06
Path E	-0.015	0.021	0.04	0.019	0.019	0.06	-0.026 <sup>†</sup>	0.014	0.12	0.031	0.027	0.07	-0.003	0.015	0.01
Path F	0.023	0.070	0.02	0.022	0.078	0.02	0.001	0.061	0.00	-0.093	0.075	0.08	0.176*	0.069	0.16
<i>Regression on Covariates</i>															
<i>AVPD Intercept</i>															
Sex	0.136	0.250	0.03	0.149	0.257	0.04	0.195	0.259	0.05	0.202	0.257	0.05	0.169	0.263	0.04
Age	0.077	0.220	0.02	0.096	0.225	0.03	0.089	0.231	0.02	0.169	0.229	0.05	0.064	0.225	0.02
Group	1.330***	0.263	0.31	1.388***	0.274	0.31	1.365***	0.272	0.30	1.323***	0.271	0.30	1.384***	0.272	0.31
<i>AVPD Slope</i>															
Sex	-0.210	0.135	0.10	-0.156	0.153	0.06	-0.201	0.135	0.09	-0.213	0.141	0.10	-0.150	0.136	0.07
Age	-0.014	0.152	0.01	-0.020	0.141	0.01	-0.021	0.150	0.01	-0.088	0.144	0.04	0.018	0.147	0.01
Group	-0.265	0.197	0.08	-0.236	0.182	0.08	-0.242	0.178	0.09	-0.117	0.174	0.04	-0.303	0.159	0.12
<i>Personality Intercept</i>															
Sex	0.133	0.151	0.06	-0.883***	0.142	0.37	0.143	0.141	0.06	-0.316*	0.135	0.15	-0.234	0.140	0.10
Age	0.029	0.143	0.01	-0.115	0.115	0.06	-0.095	0.159	0.04	-0.222	0.136	0.10	-0.102	0.125	0.05
Group	-0.080	0.143	0.04	-0.803***	0.134	0.36	-0.464**	0.137	0.21	1.149***	0.129	0.49	0.152	0.135	0.07
<i>Personality Slope</i>															

	Dominance			Affiliation			Conscientiousness			Neuroticism			Openness		
	Coef.	S.E.	ES r	Coef.	S.E.	ES r	Coef.	S.E.	ES r	Coef.	S.E.	ES r	Coef.	S.E.	ES r
Sex	0.008	0.029	0.02	-0.063	0.051	0.08	-0.058 <sup>†</sup>	0.031	0.12	-0.005	0.045	0.01	0.018	0.034	0.03
Age	0.024	0.031	0.05	-0.032	0.029	0.07	0.019	0.029	0.04	-0.089 <sup>†</sup>	0.047	0.12	0.030	0.032	0.06
Group	0.036	0.042	0.05	-0.058	0.039	0.09	-0.014	0.035	0.03	-0.026	0.059	0.03	0.031	0.038	0.05
<b>Model Fit</b>															
LL	-1452.42			-1540.79			-1468.65			-1613.92			-1517.40		
AIC	2962.85			3139.58			2995.30			3285.83			3090.80		
BIC	3064.97			3241.71			3097.42			3296.02			3189.40		

Note. N = 250. AVPD = Avoidant Personality Disorder; LL = Log Likelihood; AIC = Akaike Information Criterion; BIC = Bayesian Information Criterion. Coef. = Raw Estimated Coefficient; S.E. = Standard Error; ES r = Effect Size r.

<sup>†</sup>  $p < .10$ ;  
 \*  $p < .05$ ;  
 \*\*  $p < .01$ ;  
 \*\*\*  $p < .001$ .