May, 2004

The longitudinal relationship of personality traits and disorders

Megan B. Warner, *Texas A & M University - College Station*
Leslie C. Morey, *Texas A & M University - College Station*
John F. Finch, *Texas A & M University - College Station*
John G. Gunderson
Andrew E. Skodol, et al.

Journal of Abnormal Psychology
2004, Vol. 113, No. 2, 217–227

0021-843X/04/$12.00 DOI: 10.1037/0021-843X.113.2.217

The Longitudinal Relationship of Personality Traits and Disorders

Megan B. Warner, Leslie C. Morey, and John F. Finch
Texas A&M University

John G. Gunderson
McLean Hospital and Harvard Medical School

Andrew E. Skodol
New York State Psychiatric Institute and Columbia University

Charles A. Sanislow
Yale University School of Medicine

M. Tracie Shea
Brown University

Thomas H. McGlashan and Carlos M. Grilo
Yale University School of Medicine

Personality disorders are presumed to be stable because of underlying stable and maladaptive personality traits, but while previous research has demonstrated a link between personality traits and personality disorders cross-sectionally, personality disorders and personality traits have not been linked longitudinally. This study explores the extent to which relevant personality traits are stable in individuals diagnosed with 4 personality disorders (schizotypal, borderline, avoidant, and obsessive–compulsive personality disorders) and examines the assumption that these personality disorders are stable by virtue of stable personality traits. This assumption was tested via the estimation of a series of latent longitudinal models that evaluated whether changes in relevant personality traits lead to subsequent changes in personality disorders. In addition to offering large consistency estimates for personality traits and personality disorders, the results demonstrate significant cross-lagged relationships between trait change and later disorder change for 3 of the 4 personality disorders studied.

With the publication of *DSM–III* (American Psychiatric Association, 1980) a separate and distinct axis was created to distinguish personality disorder (PD) from mental state disorders. These disorders were presumed to reflect difficulties associated with personality traits that were inflexible or maladaptive, and the personality disorders were thought to be stable over time relative to Axis I disorders by virtue of the assumed stability of personality (see Roberts & DelVecchio, 2000, for a review of research addressing this assumption). Accompanying this assumption was the belief that to produce lasting change in PD, personality change must occur. Livesley (1999) points out the paradox for clinicians, noting that "personality disorder is defined in terms of enduring traits. . . yet we seek to treat personality disorder, and treatment implies change" (p. 26).

Although PDs have conventionally been thought of as stable and unchanging, observed stability estimates have in fact varied widely (Ferro, Klein, Schwartz, Kasch, & Leader, 1998; Grilo, McGlashan, & Oldham, 1998; McDavid & Pilkonis, 1996; Perry, 1993). In his review of the longitudinal course of PDs, Perry (1993) reported that at an average follow-up of 8.7 years, 57% of individuals diagnosed with borderline personality disorder (BPD) retained the diagnoses. In a model based on previous studies, Perry suggested that 3.7% of cases remit each year, resulting in 52% retaining the diagnosis at 10 years, and 33% at 15 years. In a review of the PD stability literature, McDavid and Pilkonis (1996) found that, irrespective of length of time and method of assessment, the stability of 11 studies looking at specific PD diagnoses ranged from 25% to 78%. Follow-ups longer than 5 years showed 50% stability whereas those less than 5 years had a mean of 62% stability. McDavid and Pilkonis (1996) note a variety of explanations for these findings, which included methodological problems (such as diagnostic reliability) as well as potential limitations in the construct validity of PD concepts. However, to this point few studies have been directed toward determining whether some portion of these observed changes is attributable to changes in the underlying personality traits.

The trait model that has received the most theoretical and research attention as applied to PD has been the five-factor model (FFM; e.g., Costa & Widiger, 2002). The five higher order traits of the FFM (Neuroticism, Extraversion, Agreeableness, Conscientiousness, and Openness) are thought to be fairly stable after the age of 30 (McCrae & Costa, 1990), cross-sectional research suggests that the traits do show statistically significant changes with age, with declines in Neuroticism, Extraversion, and Openness, and increases in Agreeableness and Conscientiousness; although correlations with age tend to be small (Costa & McCrae, 1994). Previous longitudinal research, however, did not find consistent effects of age on personality stability, and found that over a 6-year interval, the stability estimates for the five domains are large

Megan B. Warner, Leslie C. Morey, and John F. Finch, Texas A&M University; John G. Gunderson, McLean Hospital, Belmont, MA, and Harvard Medical School; Andrew E. Skodol, New York State Psychiatric Institute, and Columbia University; Charles A. Sanislow, Thomas H. McGlashan, and Carlos M. Grilo, Yale University School of Medicine; M. Tracie Shea, Department of Psychiatry, Brown University.

Correspondence concerning this article should be addressed to Leslie C. Morey, Department of Psychology, 230 Psychology Building, 4235 Texas A&M University, College Station, TX 77843-4235. E-mail: lcm@psyc.tamu.edu

(Costa & McCrae, 1988). This stability may itself be age related. In a recent meta-analysis of this literature, Roberts and DelVecchio (2000) demonstrated that individuals' rank order consistency increases with age, with consistency estimates peaking during middle age. Costa and McCrae (1994) argued that stability estimates are even higher when retest reliability is accounted for, and concluded that "behaviors, attitudes, skills, interests, roles, and relationships change over time, but in ways that are consistent with the individual's underlying personality." (p. 35)

Cross-sectional research has demonstrated clear associations between the traits of the FFM and PDs, leading some to speculate that PDs may best be understood as extreme, maladaptive levels of these five normal personality traits (e.g., Trull & McCrae, 1994). Costa and Widiger (1994) suggest that "the FFM is the most adequate and comprehensive taxonomy for describing personality and for understanding problems associated with personalities or personality disorders" (p. 3), and the *DSM–IV* (American Psychiatric Association, 1994) PD categories have been described in terms of the FFM and its lower order facets (Costa & Widiger, 2002). Although limitations of the FFM as applied to PDs have been noted (e.g., Clark, 1993; Morey et al., 2002; Coolidge et al., 1994), there are clear cross-sectional relationships between certain of these traits and the PD constructs. However, there is little evidence of any longitudinal relationship among these disorders, evidence of which could clarify the nature of causal linkages between these traits and disorders.

The limited evidence in this area is consistent with a pattern of temporal relatedness of these traits and disorders. In a study of the stability of BPD, Trull et al. (1997) looked at assessments of borderline PD features, general personality traits, and affect in a nonclinical sample. They found that stability estimates for BPD were comparable to stability estimates for Neuroticism, Agreeableness, and Conscientiousness. After 2 years, mean scores of BPD decreased, as did the base rate of borderline diagnoses in the sample. Neuroticism and negative affectivity also decreased, whereas Agreeableness and Conscientiousness increased over the 2-year period. Unfortunately, these authors did not link the change in the traits to the change in the diagnosis of BPD. Along similar lines, Ferro et al. (1998) compared the stability of the PD diagnoses with personality stability coefficients derived from the Eysenck Personality Questionnaire; they noted that Extraversion and Psychoticism tended to show higher stability than dimensional scores from their PD measure, but that Neuroticism was comparable to many of the stability estimates for the different PD diagnoses. This finding suggests that personality traits may be generally more stable than PD features. These authors again did not link the changes in the disorder to the changes in personality traits, however, so it is unclear whether or not changes in traits corresponds to change in disorder.

The present study seeks to further explore the link between personality traits and disorders by relating patterns of change in these two domains. The present study explicitly links data on both personality traits and symptoms to examine the assumption that changes in PD are brought about, at least in part, through changes in personality. Data from the Collaborative Longitudinal Study of Personality Disorders (CLPS), a multisite longitudinal study, are used to test this assumption in four target personality disorders: BPD, schizotypal (STPD), avoidant (AVPD), and obsessive–compulsive personality disorders (OCPD). This sample of patients, most of whom were in treatment at entry to the study, provides a unique opportunity to determine whether changes in personality that appeared to occur during this interval had subsequent effects on PD status.

Method

Participants

Participants were 376 patients selected from a larger sample of 668 recruited for a longitudinal study of PD (Gunderson et al., 2000) from four different study sites in the Northeast United States. Initial recruitment for the sample was targeted for four of the *DSM–IV* (American Psychiatric Association, 1994) PDs: avoidant, borderline, obsessive–compulsive, and schizotypal. Exclusion criteria for the study included a history of schizophrenia-spectrum psychosis, organic mental disorder, acute substance intoxication or withdrawal, or mental retardation that would invalidate assessments; patients could manifest other Axis I diagnoses and did so at rates typical of PD samples (McGlashan et al., 2000). For this report, the original sample of 668 individuals was reduced to 376 to assure complete data for all participants at the baseline, 1-year, and 2-year time points. Some loss was accounted for by sample attrition; by the end of 2 years of data collection, 63 individuals were no longer participating in the study because of a variety of factors, including inability to be found for continued assessment, withdrawal from the study, or they were deceased. A number of the remaining 605 participants could not be included because of missing or incomplete data at one of the observational time points; for most participants, exclusion was because they did not fully complete or did not return their self-report materials at all three time points. A chi-square test examined the distribution of individuals for cell assignment in order to make sure that there were no systematic differences between those participants who were excluded from the analyses, and the final sample of 376. The results suggest that the most notable difference in the samples was cell distribution. There were more individuals with AVPD in the incomplete data sample, and this sample had fewer individuals with STPD, BPD, and OCPD $\chi^2(4, N = 668) = 10.47$, $p < .05$. Additionally, *t* tests were completed to determine whether there were mean differences in domain scores between the complete and incomplete data samples at baseline. The differences in the personality domains indicated that the incomplete data sample had lower scores on Neuroticism ($M = 116.18$, $SD = 23.40$) than the complete data sample ($M = 123.10$, $SD = 24.73$), $t(605) = 3.49$, $p < .01$, and that they had higher scores on Extraversion ($M = 95.64$, $SD = 22.26$) than the complete data sample ($M = 91.70$, $SD = 22.62$), $t(589) = -2.11$, $p < .05$. Thus, the effects of attrition and incomplete responding resulted in the study sample being slightly more ill than the original sample and having a higher concentration of Axis II pathology.

Measures and Procedure

Baseline assessment. All participants signed written informed consent, following a full explanation of study procedures. A shortened version of the Personality Diagnostic Questionnaire (PDQ–IV; Hyler et al., 1990; Hyler, 1994) consisting of items for the four targeted PDs was used to screen for potential participants. Participants screening positive for one or more of the PDs were referred for further diagnostic assessment with the Structured Clinical Interview for *DSM–IV* Axis I disorders (SCID–I; First et al., 1996) and the Diagnostic Interview for *DSM–IV* Personality Disorders (DIPD–IV; Zanarini, Frankenburg, Sickel, & Yong, 1996). Participants were interviewed in person by experienced interviewers with master's or doctoral degrees in a mental health field. Interviewers received extensive training and continued reliability monitoring in the administration of the major diagnostic measures (Axis I and II; Gunderson et al., 2000). The DIPD–IV is a semistructured interview for assessing each of the 10 *DSM–IV* Axis II disorders. It is designed for use by interviewers trained to make clinical judgments. One or more questions are asked for each of the criteria, which are then rated on a 3-point scale (0 = *not present*, 1 =

present but of uncertain clinical significance, 2 = *definitely present*). The time frame covered is the prior 2 years, but traits must be reported to be characteristic of the person for most of his or her adult life to be counted toward a diagnosis. The median kappa for interrater reliability was .68 (range = .58–1.00), and the median test–retest kappa was .69 (range = .39–1.00). For the four study PDs, the interrater and test–retest kappas were .68 and .69 for BPD, .68 and .64 for AVPD, and .71 and .74 for OCPD, respectively. The test–retest kappa for STPD was .64; there was an insufficient sample size in the interrater reliability sample to calculate the kappa for STPD, but diagnostic agreement was 100% (Zanarini et al., 2000).

The NEO-Personality Inventory—Revised (NEO-PI–R; Costa & McCrae, 1992) was administered at baseline as part of a comprehensive battery of instruments. The NEO–PI–R is a self-report questionnaire that generally requires 30–40 minutes to complete. It is designed to provide a comprehensive assessment of the FFM of personality. In addition, the NEO–PI–R measures six facet scales that define each of the five domains (see Table 1 for a list of the domains and facets). The 240 items are answered on a 5-point Likert scale. In this sample, internal consistency reliabilities for the five domain scales in the baseline assessment ranged from .87 to .92 (*Mdn* = .89); for the facet scales, they ranged from .58 to .85 (*Mdn* = .75).

Follow-up evaluations. Participants were interviewed at 6 months, 1 year, and 2 years following the baseline assessment. Changes in personality traits were determined via a readministration of the NEO–PI–R at the 1-year and 2-year follow-up. The course of each of the four study personality disorders was assessed using a modified version of the DIPD–IV. To assess the longitudinal course of the study PDs, the DIPD–IV was modified to record the presence of each criterion for the four PDs for each month of the follow-up interval. Follow-up interviews were not blind and were conducted by the same interviewer whenever possible. Interviewers asked the standard DIPD–IV probes for presence of each criterion; if present at all during the interval, the participant was then queried about any change over the interval, to determine whether or when the criterion was absent. Ratings (0, 1, or 2) were then made for each month of the interval for each criterion. To estimate the reliability of retrospective reporting by month on the DIPD–Follow Along Version (FAV), an additional reliability study was conducted. At the 12-month interview, interviewers assessed and rated Month 6, in addition to Months 7–12. Hence Month 6 was rated twice, first at the 6-month interview, then again 6 months later at the 12-month interview. Based on 453 cases with overlap data, the kappas for diagnostic agreement at the 2 time points were .78 (STPD), .70 (BPD), .73 (AVPD), and .68 (OCPD).

Analyses

To examine the overtime relationship between personality traits and PD criteria, a series of longitudinal latent variable models were estimated using the AMOS structural equation modeling software package (Arbuckle,

Table 1
Expert Ratings of Prototypical Personality Traits for Selected Personality Disorders

Trait/facet	Schizotypal	Borderline	Avoidant	Obsessive–Compulsive
Neuroticism				
N1 Anxiousness	+	+	+	+
N2 Angry hostility		+		
N3 Depressiveness		+		
N4 Self-consciousness	+		+	
N5 Impulsiveness		+	−	−
N6 Vulnerability		+	+	
Extraversion				
E1 Warmth	−			
E2 Gregariousness	−		−	
E3 Assertiveness			−	
E4 Activity				
E5 Excitement seeking			−	−
E6 Positive emotions	−		−	
Openness				
O1 Fantasy				
O2 Aesthetics				
O3 Feelings		+		−
O4 Actions		+	−	−
O5 Ideas	+			−
O6 Values				−
Agreeableness				
A1 Trust				
A2 Straightforwardness				
A3 Altruism				
A4 Compliance				
A5 Modesty			+	
A6 Tendermindedness				
Conscientiousness				
C1 Competence				+
C2 Order	−			+
C3 Dutifulness				+
C4 Achievement striving				+
C5 Self-discipline				+
C6 Deliberation		−		+

Note. From "Using the Five-Factor Model to Represent the *DSM–IV* Personality Disorders: An Expert Consensus Approach," by D.R. Lynam and T.A. Widiger, 2001, *Journal of Abnormal Psychology, 110*, p. 404. Copyright 2001 by the American Psychological Association. Adapted with permission of the authors.

1995). The latent personality constructs theoretically underlying the four PDs were represented by Lynam and Widiger's (2001) recent FFM formulation of the PDs, which used expert consensus to determine the FFM traits most prototypic of certain PDs. Lynam and Widiger (2001) asked experts in PD to use the 30 facets of the FFM to rate prototypic cases of each PD, and concluded that these experts showed interrater agreement among themselves, and also found that these ratings corresponded with previously hypothesized relationships (Widiger, Trull, Clarkin, Sanderson, & Costa, 1994). Table 1 lists the consensus results of the most prototypic traits, as well as the hypothesized direction of the relationships.

Before testing the hypothesized structural models of lagged relationships among traits and disorders, the measurement models for each personality trait construct specified by Lynam and Widiger (2001) were tested using confirmatory factor analysis (CFA). In these models, the errors of measurement in a given manifest variable were expected to be similar at each time point, so corresponding error terms were allowed to correlate across time points. In addition, because they were expected to have identical relationships with the latent constructs across time, the loadings for corresponding manifest variables were fixed as equal across the three time intervals (i.e., the loading of the first indicator of STPD at Time 1 was set equal to the loading of the same indicator of STPD at Time 2, etc.). The fit indices for the initial measurement models were then examined to evaluate how well each model fit the data. Revisions to the initial measurement models were made by removing manifest variables that shared less than 10% common variance with their target factor at baseline (a standardized factor loading of less than .30). The Openness facet, *actions*, for example, was one facet hypothesized as salient for several disorders by Lynam and Widiger (2001), but in our analyses it was removed for the BPD, AVPD, and OCPD models to improve the fit of these measurement models.

The four personality disorders were represented as latent variables underlying the criterion scores obtained from the DIPD–IV, which assesses the presence and clinical significance of individual *DSM–IV* PD criteria. Items specific to the *DSM–IV* criteria for the PD model under study were used to represent the PD construct. Because each DIPD–IV item is on a 0–2 point scale, the DIPD–IV indicators were grouped into parcels of approximately three DIPD–IV items to achieve indicators that more closely approximated a normal distribution (Little et al., 2001). Each PD construct was thus based on 3 DIPD–IV "parcels" at each time point.

Figure 1 presents the general longitudinal model linking the traits and disorders estimated from these measurement models. The stability of the trait constructs at each time point is represented by the horizontal paths labeled T_{AB} and T_{BC}. Similarly, D_{AB} and D_{BC} represent the stability coefficients linking the PD measures at consecutive points in time. These parameters are of interest because they represent autoregressive effects and

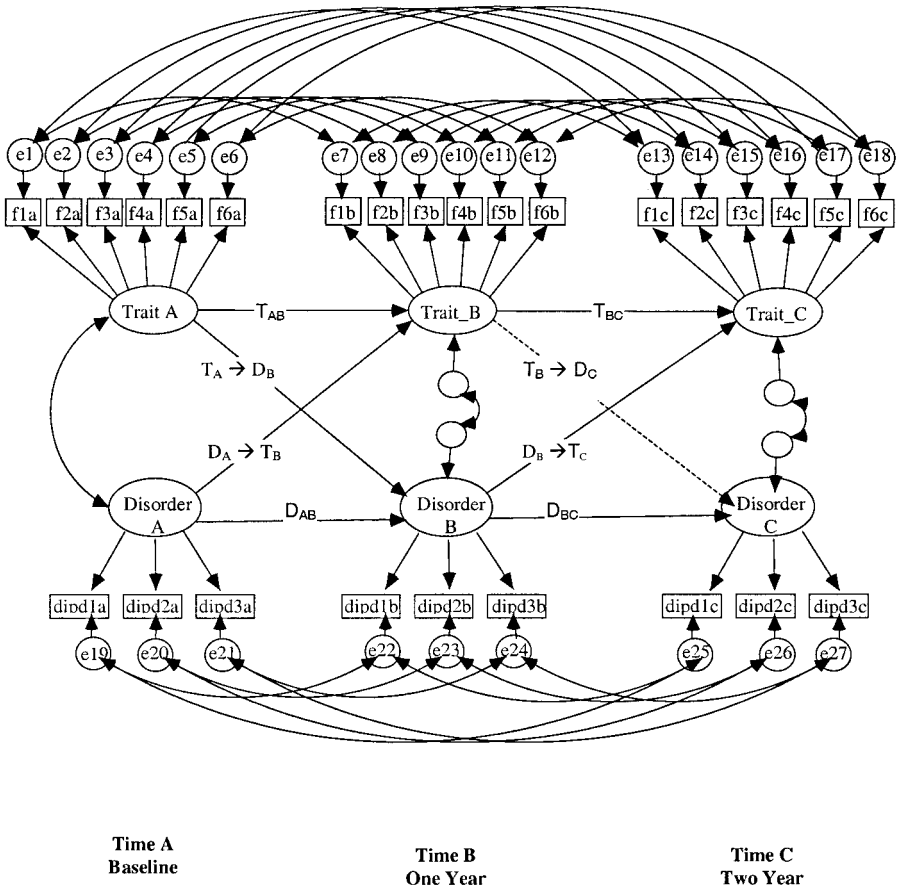

Figure 1. Hypothesized latent longitudinal model of personality traits and personality disorder. Large ovals represent latent constructs, and rectangles represent manifest variables. Small circles reflect residual variables (disturbances). The bidirectional arrows represent correlations among constructs (e.g., the double-headed arrows linking the errors of measurement in the manifest variables indicate autocorrelation of these error terms across time points). F1—F6 represent hypothesized facets measured at each time point. Diagonal arrows represent the lagged effects, and the dotted arrow represents the hypothesized lagged effect of personality change on disorder change.

provide information about the extent to which the latent personality trait and disorder constructs reflect enduring individual differences as measured over these intervals. Estimation of these coefficients controls for the stability of the latent trait and latent PD constructs. Therefore, any significant cross-lagged associations represent incremental effects that are present above and beyond those attributable to the stability of personality traits and PD.

The cross-lagged effects are represented by the diagonal arrows in Figure 1. There are important differences in the interpretation of each lag. The lags from the first panel of the design (the period from baseline to Year 1) relate the baseline levels of each construct to change in the other construct at the next time point. For example, the $D_A \rightarrow T_B$ path in Figure 1 represents the influence of baseline PD on personality change at Year 1, while the $T_A \rightarrow D_B$ reflects the prediction of PD changes at Year 1 from baseline trait configurations. These first panel lags are primarily informative in investigating traits and disorders as static predictors of subsequent change; for example, determining whether traits serve as a static diathesis that helps to predict subsequent changes in PD status. However, these first panel lags do not answer the central question of dynamic relatedness, for example, whether trait change subsequently influences disorder change. To address this issue, there must be data on changes in both traits and disorder, and this information can be found by examining the lagged paths from the second to the third observation point. The lagged association labeled $T_B \rightarrow D_C$ estimates the predictive relationship between personality change at Year 1 to PD change at Year 2; $D_B \rightarrow T_C$ estimates the influence of PD change at Year 1 onto personality trait change at Year 2. Because each lag of the model provides different information, neither the autoregressive effects nor the corresponding cross-lagged effects were constrained to be equivalent, a constraint sometimes placed upon models assumed to have reached some form of equilibrium (Dwyer, 1983). Instead, the significance of each lag in each PD model was tested separately. In these models, the manifest variable loadings for specific indicators of the latent variables were fixed to be equal across successive time points for both trait and disorder indicators. The synchronous correlations linking the disturbances for the trait and the disorder constructs at each time point were also specified to be equal across time points. The primary paths of interest for this study (i.e., the lagged effects linking trait and disorder, as well as the stability estimates of the trait and disorder constructs) were freed to be estimated.

Results

Separate hypothesized structural models were tested for each PD following the general model presented in Figure 1. The results for each of the four disorder models are described below.

STPD

To assure that the measurement model was providing a good representation of the underlying trait structure of STPD, two facets accounting for less than 10% of the latent trait variable were removed from all three time points. These were the Openness facet, *ideas* (O5), and the Conscientiousness facet, *order* (C2). At baseline, *ideas* had a standardized regression weight of -.19, and *order* was -.05. Model fit was assessed with three widely used measures, the normed fit index (NFI; Bentler & Bonett, 1980), the comparative fit index (CFI; Bentler, 1990), and the root-mean-squared error of approximation (RMSEA; Steiger, 1990). For the former two measures, values of approximately .90 or greater reflect an adequate fit, whereas Browne and Cudeck (1993) suggested that values of the RMSEA of .05 or less indicate a close fit, values between .05 and .08 indicate adequate fit, and values of greater than .10 suggest room for improvement. The fit of the final schizotypal structural model was acceptable (NFI = .91, CFI = .94, RMSEA = .06). The standardized parameter estimates reflecting the stability for the latent trait variable across time were significant and quite large ($\beta = .76$ and $\beta = .83$, both ps $< .01$) as were the stability estimates for STPD ($\beta = .90$ and $\beta = .81$, both ps $< .01$).

As can be seen in Figure 2, there were no significant cross-lagged effects of STPD on schizotypal personality traits, nor was the initial trait value significantly related to the second measurement of STPD. However, there was a significant lagged effect from the latent personality trait variable at Time 2 to STPD at Time 3 ($\beta = .16$, $p < .01$). This suggests a significant relationship between personality change and later disorder change that is predictive above and beyond the rather large stability effects of both personality traits and STPD over time.

BPD

As described earlier, the original borderline personality trait measurement model was refined by removing those facets that accounted for less than 10% of the variance. This resulted in removing the two Openness facets in the model, *feelings* (O3) and *actions* (O4), which were $\lambda = .10$ and $\lambda = -.20$ at baseline, respectively. The final borderline structural model appeared to be a good representation of the data (NFI = .91, CFI = .95, RMSEA = .06). The stability estimates were significant for the latent personality trait variables over time ($\beta = .70$, $p < .01$ and $\beta = .77$, $p < .01$) as were the stability estimates for PD over time ($\beta = .77$, $p < .01$ and $\beta = .70$, $p < .01$).

As can be seen in Figure 3, there was a significant cross-lagged effect from BPD at baseline to borderline traits at Year 1 ($\beta = .15$, $p < .01$). This suggests a relationship between disorder and change in personality traits but does not account for change in disorder. More informative in that regard was the significant cross-lagged effect ($T_B \rightarrow D_C$) from the measure of personality at Year 1 to PD at the Year 2 time point ($\beta = .14$, $p < .01$). Because both of the latent variable measurements account for earlier measures of personality and disorder, and the cross-lagged effects have predictive power over and above the large stability estimates, the significance of this cross-lagged effect suggests that personality trait change has a relationship with later BPD change.

AVPD

As was done with earlier models, the trait measurement model for AVPD was refined, resulting in the exclusion of the Extraversion facet of *excitement seeking*, and the Openness facet of *actions* ($\lambda = -.17$ and $\lambda = -.29$ at baseline, respectively). The fit of the resulting AVPD structural model was acceptable as indicated by the various fit indices (NFI = .89, CFI = .94, RMSEA = .05). The standardized parameter estimates reflecting the stability of the latent trait variable over time were both significant ($\beta = .74$, $p < .01$ and $\beta = .77$, $p < .01$) as were the parameter estimates which represent the stability of AVPD ($\beta = .73$, $p < .01$ and $\beta = .60$, $p < .01$).

As can be seen in Figure 4, there was a significant cross-lagged relationship from avoidant trait change to AVPD change ($\beta = .27$, $p < .01$), suggesting that as traits change there is a later effect on AVPD. Notably, the causal effect in the opposite direction was not significant—there was no relationship between AVPD change at Year 1 to avoidant traits at the Year 2 time point, indicating that

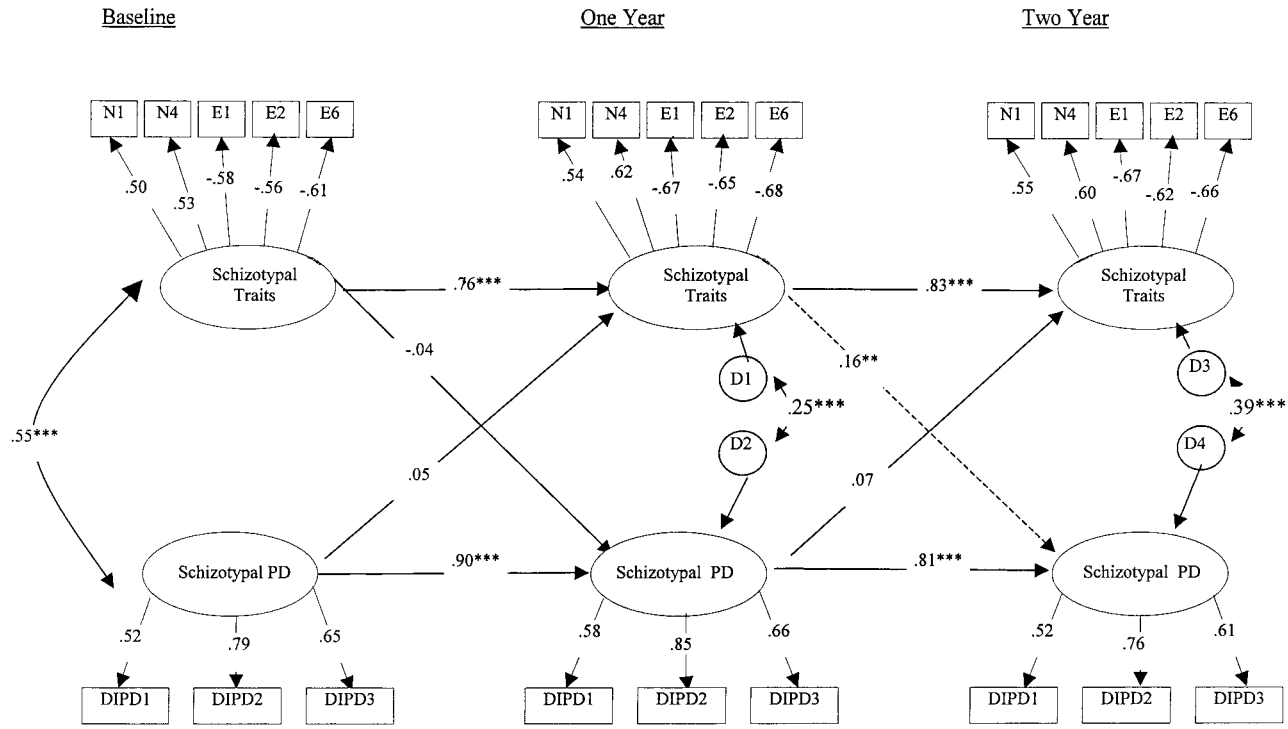

Figure 2. Final structural model for schizotypal personality disorder. The dashed line represents the lagged effect of personality trait change on personality disorder change. Parameter estimates are standardized. All factor loadings were significant at $p < .001$; stability and lagged effects: $*p < .05$; $**p < .01$; $***p < .001$. N represents facets of Neuroticism; E represents facets of Extraversion; D represents the disturbances; DIPD represents the Diagnostic Interview for *DSM–IV* Personality Disorders.

change in AVPD does not have a predictive relationship with change in traits.

OCPD

The original measurement model for the latent personality trait variables hypothesized to underlie OCPD yielded a marginal fit (NFI = .85, CFI = .90, RMSEA = .07). Those facets with standardized estimates less than .30 were removed from the model, resulting in the removal of the Extraversion facet *excitement seeking* (E5; $\lambda = .04$ at baseline), the Openness facets *feelings* (O3; $\lambda = -.12$ at baseline), *actions* (O4; $\lambda = .10$), *ideas* (O5; $\lambda = -.23$), and *values* (O6; $\lambda = -.07$).

With these revisions, the resulting structural model for OCPD fit the data more adequately (NFI = .90, CFI = .94, RMSEA = .05). As can be seen in Figure 5, both the personality trait stability estimates ($\beta = .84$ and $\beta = .86$, $p < .01$) and the obsessive compulsive PD estimates were significant ($\beta = .76$ and $\beta = .80$, $p < .01$).

The parameter estimates linking OCPD to later personality traits was examined, but neither the first nor the second lag from disorder to trait was significant. Similarly, initial measurement of personality traits did not have a significant relationship with later measurements of disorder, nor did personality change at Year 1 impact OCPD at the Year 2 time point. These results suggest that there were no predictive effects of personality traits on PD, or vice versa, that were significant above and beyond the large stability effects of the personality traits and disorder.

Discussion

This study brings to light several important findings. First, and most central to this article, is that for three of the four PDs examined, change in personality traits appears to have a significant predictive relationship for subsequent change in PDs, providing supporting evidence that, as many have speculated, the two are theoretically linked. Second, the study provides further empirical support that the latent variables underlying both personality traits and personality disorders reflect enduring properties that demonstrate considerable ordinal stability in individuals over a 2-year period. Finally, the results of the measurement model component of each structural equation model can help refine hypotheses recently made by Lynam and Widiger (2001) on those personality traits predicted to underlie specific disorders.

STPD, BPD, and AVPD all demonstrated statistically significant lagged effects from the second to the third panel of the structural models relating FFM traits and *DSM–IV* disorder. As noted earlier, the lagged effects from the second to the third panel provide an estimate of the relationship between the changes in each variable because both the latent trait and latent PD variables accounted for participant status at earlier time points. Interpreting these lagged effects suggests that, as is reflected in the *DSM–IV* definition of PDs, there is an important conceptual link between personality traits and PDs. The results indicate that there is a specific temporal relationship between traits and disorder whereby changes in the personality traits hypothesized to underlie PDs lead to subsequent changes in the disorder. This relationship does not

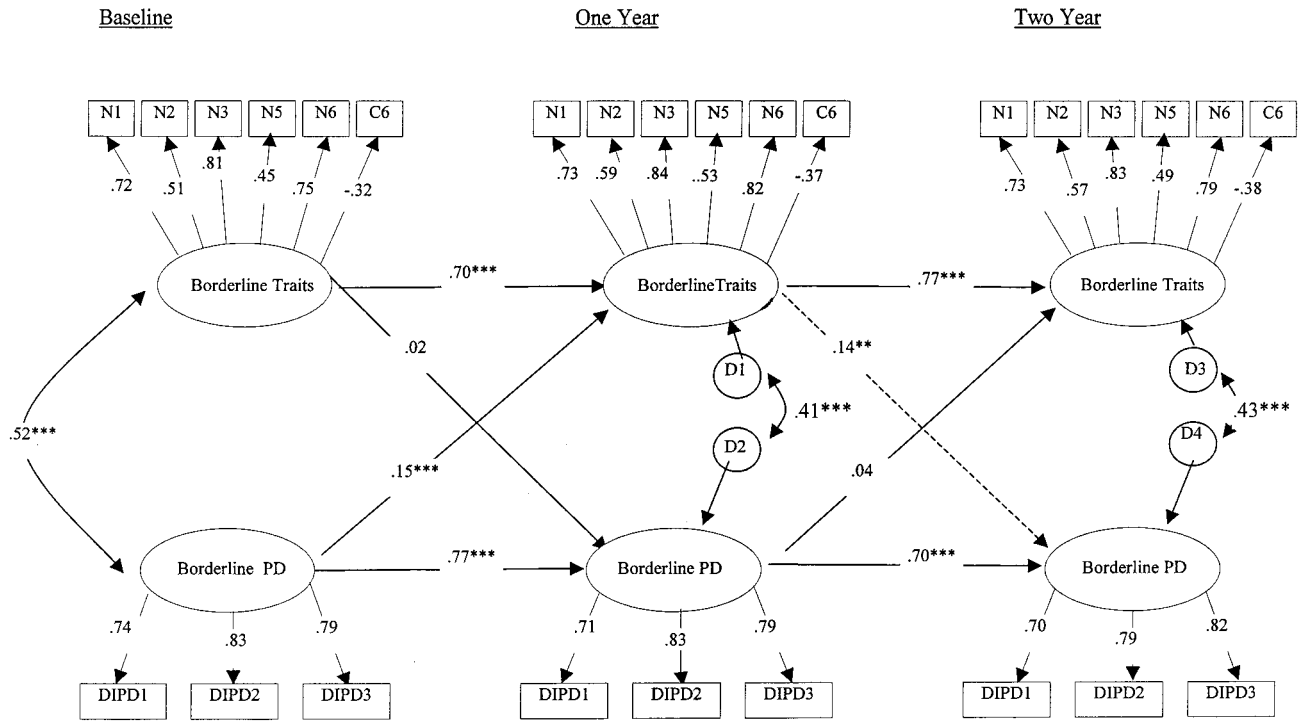

Figure 3. Final structural model for borderline personality disorder. The dashed line represents the lagged effect of personality trait change on personality disorder change. Parameter estimates are standardized. All factor loadings were significant at $p < .001$; stability and lagged effects: $*p < .05$; $**p < .01$; $***p < .001$. N represents facets of Neuroticism; C represents facets of Conscientiousness; D represents the disturbances; DIPD represents the Diagnostic Interview for *DSM–IV* Personality Disorders.

seem to hold in the opposite direction, which supports the contention that PDs stem from particular constellations of traits. This finding also supports the commonly held belief of clinicians that modifying personality traits is an important component of treating PD (e.g., Paris, 1998; Tickle, Heatherton, & Wittenberg, 2001). Though the relationships between selected personality traits and PDs have been demonstrated several times in the past, the limitations of cross-sectional data have precluded the ability to more fully understand the link between traits and disorder.

The sole exception to this finding, OCPD, was selected for inclusion in this study specifically because previous factor-analytic studies using *DSM* and non-*DSM* criteria have demonstrated that OCPD does not group well with other PDs (e.g., Kass et al., 1985; Livesley, Jackson, & Schroeder, 1992; Livesley, Jang, & Vernon, 1998; Morey, 1986; Tyrer & Alexander, 1979). In the present study, OCPD also appeared to be different than the other three disorders for which structural models were tested. Contrary to the other three PDs studied, OCPD did not show any significant lagged effects between trait change and disorder change, or the reverse. In addition, unlike the other three PDs, the cross-sectional disturbances for OCPD failed to achieve significance, suggesting that there was also little cross-sectional association between the traits and disorder after the stability and lagged effects were accounted for. Possible explanations for this latter finding are discussed in more detail below.

In contrast to the effects of trait change on disorder change, the effects of PD change did not predict personality change. None of the PDs studied demonstrated significant cross-lagged effects from the PD measure at the second observation period (representative of disorder change) to the latent personality construct at the third observation period (which represented personality change). This result, paired with the previously discussed findings that demonstrate that trait change results in disorder change, suggests that the relationship between personality traits and PD is not simply a reciprocal one, but instead seems to show specific directionality. That is, trait changes are required to alter PD, but PD changes in a group of individuals do not necessarily lead to a change in traits. These results coincide with the clinical belief that lasting personality change may require more than targeting specific behaviors or symptoms.

The analyses of lagged effects in the first panel of the study provide information about the impact of baseline personality traits on disorder change, and baseline disorder on personality change. Results indicate that the baseline measures of personality traits did not predict the 1-year measure of PD above and beyond the influence of the baseline measures of PD. That is, the baseline trait profile does not appear to have much impact on PD change independent of its concurrent association with PD.

Interestingly, BPD demonstrated a predictive relationship between the baseline disorder measure to changes in the latent trait variable. It is possible that these results reflect affective state effects on personality assessment. Individuals with BPD are characterized by affective lability (American Psychiatric Association, 1994), in contrast to STPD and OCPD, which do not tend to show marked affective variability. Indeed, a criterion for STPD is "inappropriate or constricted affect" (American Psychiatric Associa-

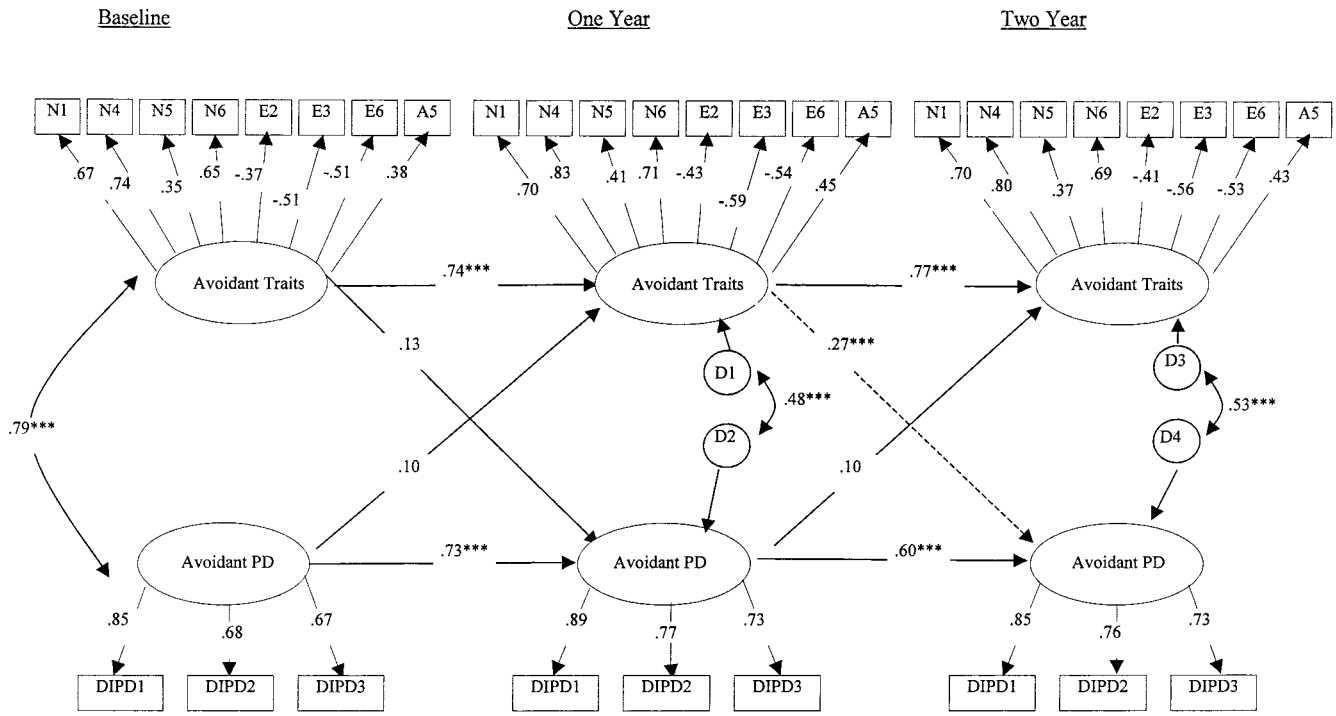

Figure 4. Final structural model for avoidant personality disorder. The dashed line represents the lagged effect of personality trait change on personality disorder change. Parameter estimates are standardized. All factor loadings were significant at $p < .001$; stability and lagged effects: $*p < .05$; $**p < .01$; $***p < .001$. N represents facets of Neuroticism; E represents facets of Extraversion; A represents facets of Agreeableness; D represents the disturbances; DIPD represents the Diagnostic Interview for *DSM–IV* Personality Disorders.

tion, 1994, p. 645) and the *DSM–IV* states that individuals with OCPD "may be very uncomfortable in the presence of others who are emotionally expressive" and that they "may be intolerant of affective behavior in others." Because emotional state does seem to show some impact on later trait measurement (Hirschfeld et al., 1983; Zimmerman, 1994) as well as on measures of PDs (Grilo et al., 1998; Zimmerman, 1994), it is possible that the finding that BPD predicts later personality trait change is due to affective state impact upon the measurement of these traits. As an example, consider an individual with BPD who presents at baseline in prominent affective distress, perhaps related to recent relationship crises. This individual is likely to show elevations on certain personality traits, most likely facets of Neuroticism such as Anxiety and Angry Hostility. One year later, the emotional state of this individual may be experienced very differently (e.g., perhaps his or her mood is euphoric as a result of a new relationship), and earlier elevations of Neuroticism facets may have drastically decreased.

An examination of the correlational results about the stability of PDs provide additional evidence that PDs appear to reflect enduring individual differences over a 2-year interval, even though noteworthy changes in the categorical diagnosis may be observed and in fact were observed in these patients (Shea et al., 2002; Grilo et al., in press). This finding is congruent with the *DSM–IV* (American Psychiatric Association, 1994) description that PDs reflect enduring phenomena, although it is important to note there may also be considerable fluctuation in the severity of these problems over time. In addition, the findings support the widely asserted stance of personality researchers that personality traits are stable (Costa & McCrae, 1988; McCrae & Costa, 1990) in that this study found that the personality trait constellations hypothesized to underlie relevant PDs endured over time. Thus, although the study provides additional empirical evidence in support of widely held beliefs about the enduring nature of both personality traits and personality disorders, it is unique in that it links the two findings. Many have speculated that it is the stability of personality traits which underlie the stability of PD (Costa & Widiger, 1994), and the finding that changes in personality traits lead to later PD changes appears to support personality trait stability as a mechanism for PD stability.

Results of the current study also shed light on hypotheses made by Lynam and Widiger (2001) in which, using the consensus of experts, they articulate which of the facets of the FFM that were thought to best represent specific PDs. The confirmatory factor analyses of the initial and revised measurement models for each PD studied provides a test of these hypotheses on a representative group of patients. In general, the results of these analyses confirmed significant hypothesized relationships between FFM trait facets and specific personality disorders, consistent with our previous results (Morey et al., 2002) that found a majority of hypothesized relationships were supported in terms of distinguishing these four specific personality disorders, both from a major depression group (70.8% of hypothesized relationships confirmed) and from other personality disorders (62.5% confirmed). With respect to the facets which were predicted to relate to specific PDs, the majority of the hypothesized model elements fared well. Schizotypal, borderline, and avoidant each had only two facets

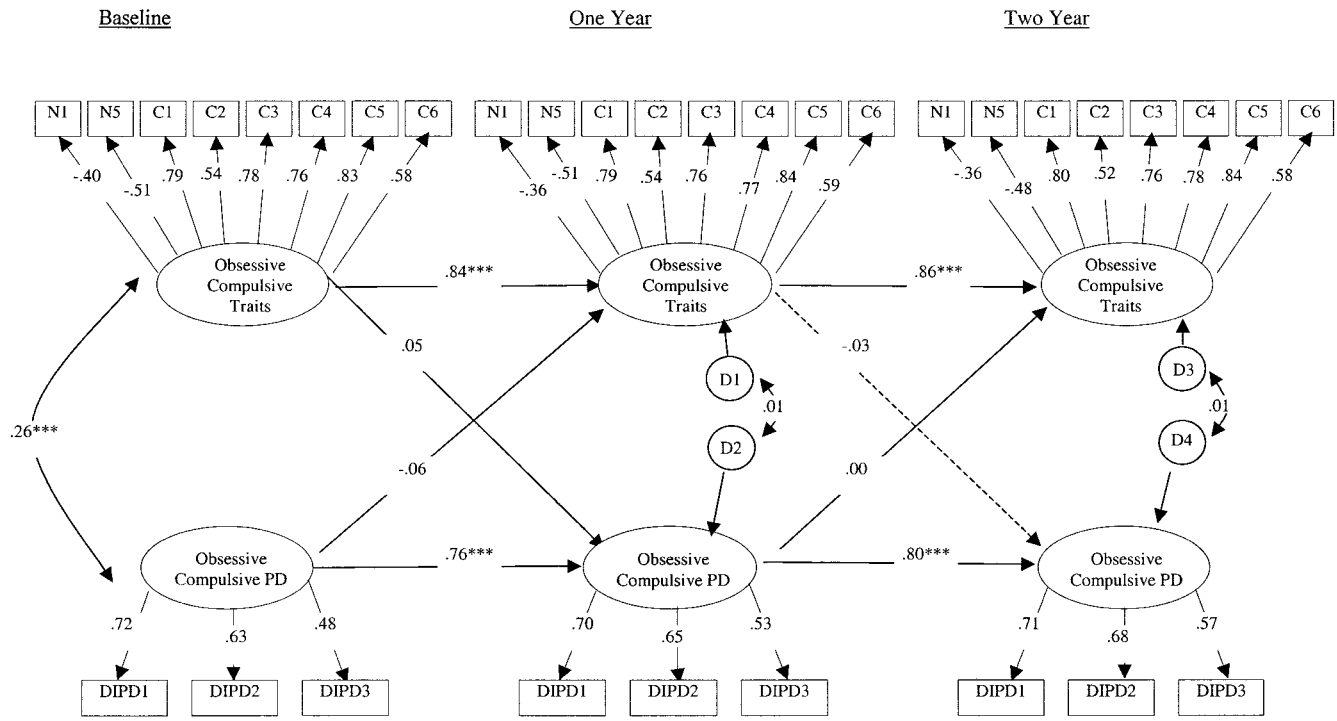

Figure 5. Final structural model for obsessive–compulsive personality disorder. The dashed line represents the lagged effect of personality trait change on personality disorder change. Parameter estimates are standardized. All factor loadings were significant at $p < .001$; stability and lagged effects: $*p < .05$; $**p < .01$; $***p < .001$. N represents facets of Neuroticism; C represents facets of Conscientiousness; D represents the disturbances; DIPD represents the Diagnostic Interview for *DSM–IV* Personality Disorders.

from the initial hypothesis made by the consensus of experts that failed to meet the .30 criterion for the factor loadings. In contrast, the model proposed for OCPD fared the worst of the four disorders. The fit of the initial measurement model for OCPD was marginal, and five of the hypothesized traits, primarily facets of Openness, were removed to improve the fit. As stated earlier, the Openness facets did not seem to adequately capture the disorder. In fact, the facets which were removed from all four PDs were primarily facets from the Openness domain; of the 11 total facets removed, 8 of those were Openness facets. Previous research has indicated that Openness does not seem to have a strong relationship to personality pathology (Schroeder, Wormworth, & Livesley, 2002). The results support the idea that Openness has limited utility in terms of a predictive relationship for these four PDs.

Interestingly, Lynam and Widiger (2001) examined the convergence of the experts' hypothesized relationships with those made previously by Widiger et al. (1994), and of the four examined in our study, the convergence was considerably poorer for OCPD relative to the other three disorders. It is possible that the domain and facets of the FFM are of limited utility in capturing the traits underlying OCPD. For example, the domain of conscientiousness has been hypothesized to be central in describing individuals with OCPD (Widiger et al., 1994), but a previous examination of these patients has found that though individuals with OCPD tend to be more conscientious than individuals with other PDs, they are actually less conscientious than the normative sample which was used for the development of the NEO (Morey et al., 2002). Haigler and Widiger (2001) noted that this result may be a relative emphasis on adaptive rather than maladaptive variants of conscientiousness in the NEO-PI items, and found that experimental manipulation of these items to target maladaptive variants resulted in scores that were indeed related to OCPD. As this study used the standard NEO-PI-R items, it is not clear whether the lack of a finding of a cross-lagged relationship between trait change and subsequent change in OCPD indicates similar measurement issues, or whether current understanding of the latent traits underlying OCPD needs to be reconceptualized.

The current study had some limitations that need to be addressed in future research. The study was limited to only four PDs. It would be beneficial to examine other PDs, but the selection of the four that were included was strategic in that each PD cluster was represented, suggesting that there should be some generalization of the results to other PDs in the same cluster. A second limitation pertains to the length of the intervals between the assessments, which makes it difficult to know how quickly the change in traits impacted the change in disorder. There was a lapse of one year between each time point. If more frequent measurements of the disorders and traits were available, it might demonstrate that the effects of trait change on disorder change occurred in more rapid intervals of time. A final limitation involves the restriction of the sample to individuals selected from clinical settings; the study of individuals in the community in future research could provide more information about changes that occur in the direction of increasing trait extremity/more pathology (rather than the lessening of extremity/pathology that was typical of this treatment sample). Furthermore, the extension of the hypothesized models into

nonclinical samples could clarify the extent to which these models involve disorder-specific implications for subsequent change, or whether they represent a more general pattern of trait changes that tend to be shared across different disorders (Morey et al., 2002) and that have similar implications for change across all disorders.

The limitations of the study are balanced by several strengths. In addition to the methodological strengths such as large sample size and a multimethod approach, the study is the first which has used a longitudinal design to link the hypothesized personality traits that underlie PDs with actual diagnostic criteria in a sample of individuals with PDs. It is also the first to use a panel design to examine lagged associations between latent constructs over time. Kessler and Greenberg (1981), and others, have noted that cross-sectional models are misspecified as true causal models because they ignore the effects that variables exert on them over time. When longitudinal designs that control for prior levels on the outcome variable are used, the case for the causal importance of a predictor often diminishes—at times to nonsignificant levels. By examining associations between traits and disorders while explicitly modeling autoregressive effects, the present investigation represents a comparatively strong test of the hypothesized causal influence of personality on subsequent PD. By comparing the lagged associations of traits and subsequent disorder against the relationship of disorder with subsequent trait levels, the present investigation also provides a clearer picture of the directionality of the relationship between personality and PD.

The study provides an important starting point for several different directions for future research. The determination that personality trait change leads to later PD change is an important piece of the puzzle in terms of understanding the relationship between traits and disorder. These results suggest that personality traits are not merely a static diathesis for PD, as individuals did change on these traits and that change proved to be predictive of later changes in PD features. The results also suggest that personality traits and disorders are not synonymous. In other words, the disorders appear to be more than simple manifestations of extreme positioning on traits, as the patterns of change observed were not reciprocal and not restricted to covariance that was entirely cross-sectional. In contrast to these two views, it appears that personality traits may represent causally proximal mechanisms that influence the expression and variability of PD over time. If so, a greater understanding of the process of personality trait change becomes an important consideration for theory and treatment. For example, is it the case that certain types of treatment impact on traits more than others? Is there an interactive effect between personality traits and type of treatment on treatment efficacy? There is also a need for a better understanding of the impact that extratreatment events, such as critical life events, can have on personality traits. These may include such major life events as marriage, divorce, job change, loss of a parent, or the birth of a child. These events may play a significant role in changes of personality and, subsequently, changes in PD. However, it should also be understood that personality traits might be one of many such influences on the expression of PD, and that changes in disorder may also come about independent of personality change. For example, individuals may move to an environment in which certain traits are less problematic, or they may learn to manage their expression of these traits in ways that are less maladaptive.

This study demonstrates that, in individuals diagnosed with PDs, the traits hypothesized to underlie PD and the diagnoses of PDs reflect enduring individual differences, as has been assumed. However, when changes in personality traits do occur, this appears to lead to subsequent changes in PD. These changes were demonstrated in three of the four PDs examined, and did not simply reflect a reciprocal relationship between trait and disorder. Though it has been speculated by some clinicians that treating traits should be a main focus of psychological treatment, others have encouraged clinicians to be realistic in their belief in the possibility of changing traits. Costa and McCrae (1986) wrote the following:

> Behaviors do change, and some learning techniques are effective in modifying behavior. Attitudes and expectations change, and information and counseling can be useful in making them more realistic. Moods come and go, and catharsis or support may temporarily relieve feelings of distress. But basic personality is to a remarkable degree fixed, and therapies aimed at altering fundamental dispositions face a difficult task. (p. 420)

To some degree, the present results can serve to counter the pessimism expressed about the feasibility and implications of personality trait change. The results should lend some hope to clinicians and researchers alike that both personality traits and PDs, though stable, may be somewhat malleable, and that when altered can have a subsequent beneficial impact on PD.

References

American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: Author.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Arbuckle, J. (1995). *AMOS user's guide*. Chicago: SmallWaters.

Bentler, P. M. (1990). Comparative fit indexes in structural models. *Psychological Bulletin, 107,* 238–246.

Bentler, P. M., & Bonett, D. G. (1980). Significance tests and goodness of fit in the analysis of covariance structures. *Psychological Bulletin, 88,* 588–606.

Browne, M. W., & Cudeck, R. (1993). Alternative ways of assessing model fit. In K. A. Bollen & J. S. Long (Eds.). *Testing structural equation models* (pp. 136–162). Newbury Park, CA: Sage.

Clark, L. A. (1993). *Manual for the Schedule for Nonadaptive and Adaptive Personality (SNAP).* Minneapolis: University of Minnesota Press.

Coolidge, F. L., Becker, L. A., DiRito, D. C., Durham, R. L., Kinlaw, M. M., & Philbrick, P. B. (1994). On the relationship of the five-factor personality model to personality disorders: Four reservations. *Psychological Reports, 75,* 11–21.

Costa, P.T., & McCrae, R.R. (1986). Personality stability and its implications for clinical psychology. *Clinical Psychology Review, 6,* 407–423.

Costa, P. T., & McCrae, R. R. (1988). Personality in adulthood: A 6-year longitudinal study of self-reports and spouse ratings on the NEO Personality Inventory. *Journal of Personality and Social Psychology, 54,* 853–863.

Costa, P. T., & McCrae, R. R. (1992). *Professional manual: Revised NEO Personality Inventory (NEO-PI-R) and NEO Five-Factor Inventory (NEO–FFI).* Odessa, FL: Psychological Assessment Resources.

Costa, P. T., & McCrae, R. R. (1994). Set like plaster? Evidence for the stability of adult personality. In T. F. Heatherton & J. L. Weinberger (Eds.), *Can personality change?* (pp. 21–40). Washington, DC: American Psychological Association.

Costa, P. T., & Widiger, T. A. (1994). Introduction: Personality disorders and the five-factor model of personality. In P. T. Costa & T. A. Widiger (Eds.), *Personality disorders and the five-factor model of personality* (pp. 1–10). Washington, DC: American Psychological Association.

Costa, P. T., & Widiger, T. A. (2002). *Personality disorders and the*

five-factor model of personality (2nd ed.). Washington, DC: American Psychological Association.

Dwyer, J. H. (1983). *Statistical models for the social and behavioral sciences.* New York: Oxford University Press.

Ferro, T., Klein, D. N., Schwartz, J. E., Kasch, K. L., & Leader, J. B. (1998). 30-month stability of personality disorder diagnoses in depressed outpatients. *American Journal of Psychiatry, 155,* 653–659.

Grilo, C. M., McGlashan, T. H., & Oldham, J. M. (1998). Course and stability of personality disorders. *Journal of Practicing Psychiatry and Behavioral Health, 4,* 61–75.

Grilo, C. M., Shea, M. T., Sanislow, C. A., Skodol, A. E., Gunderson, J. G., Stout, R. L., et al. (in press). Two-year stability and change in schizotypal, borderline, avoidant, and obsessive–compulsive personality disorders. *Journal of Consulting and Clinical Psychology*.

Gunderson, J.G., Shea, T., Skodol, A. E., McGlashan, T. H., Morey, L. C., Stout, R. L., et al. (2000). The Collaborative Longitudinal Personality Disorders Study: Development, aims, design, and sample characteristics. *Journal of Personality Disorders, 14,* 300–315.

Haigler, E. D., & Widiger, T. A. (2001). Experimental manipulation of NEO-PI-R items. *Journal of Personality Assessment, 77,* 339–358.

Hirschfeld, R. M., Klerman, G. L., Clayton, P. J., Keller, M. B., McDonald-Scott, P., & Larkin, B.H. (1983). Assessing personality: Effects of the depressive state on trait measurement. *American Journal of Psychiatry, 140,* 695–699.

Hyler, S. E. (1994). *The PDQ–4 Personality Questionnaire*. New York: New York State Psychiatric Institute.

Hyler, S. E., Skodol, A. E., Kellman, D., Oldham, J. M., & Rosnick, L. (1990). Validity of the Personality Diagnostic Questionnaire: Comparison with two structured interviews. *American Journal of Psychiatry, 147,* 1043–1048.

Kass, F., Skodol, A. E., Charles, E., Spitzer, R. L., & Williams, J. B. (1985). Scaled ratings of *DSM–III* personality disorders. *American Journal of Psychiatry, 142,* 627–630.

Kessler, R. C., & Greenberg, D. F. (1981). *Linear panel analysis: Models of quantitative change*. San Diego, CA: Academic Press.

Little, T. D., Cunningham, W. A., Shahar, G., & Widaman, K. F. (2001). To parcel or not to parcel: Exploring the question, weighing the merits. *Structural Equation Modeling, 9,* 151–173.

Livesley, W. J. (1999). The implications of recent research on the etiology and stability of personality and personality disorder. In Derksen, J., Cesare, M., & Groen, H. (Eds.), *Treatment of personality disorders* (pp. 25–37). New York: Kluwer Academic/Plenum Publishers.

Livesley, W. J., Jackson, D. N., & Schroeder, M. L. (1992). Factorial structure of traits delineating PDs in clinical and general population samples. *Journal of Abnormal Psychology, 101,* 432–440.

Livesley, W. J., Jang, K. L., & Vernon, P. A. (1998). Phenotypic and genetic structure of traits delineating personality disorder. *Archives of General Psychiatry, 55,* 672–675.

Lynam, D. R., & Widiger, T. A. (2001). Using the five-factor model to represent the *DSM–IV* personality disorders: An expert consensus approach. *Journal of Abnormal Psychology, 110,* 401–412.

McCrae, R. R., & Costa, P. T. (1990). *Personality in adulthood.* New York: Guilford Press.

McDavid, J. D., & Pilkonis, P. A. (1996). The stability of personality disorder diagnoses. *Journal of Personality Disorders, 10,* 1–15.

McGlashan, T. H., Grilo, C. M., Skodol. A. E., Gunderson, J. G., Shea, M. T., Morey, L.C., et al. (2000). The Collaborative Longitudinal Personality Disorders Study: Baseline Axis I/II and II/II diagnostic co-occurrence. *Acta Psychiatrica Scandinavica, 102,* 256-264.

Morey, L. C. (1986). A comparison of three personality disorder assessment approaches. *Journal of Psychopathology and Behavioral Assessment, 8,* 25–30.

Morey, L. C., Gunderson, J. G., Quigley, B. D., Shea, M. T., Skodol, A. E., McGlashan, T. H., et al. (2002). The representation of borderline, avoidant, obsessive–compulsive, and schizotypal personality disorders by the five-factor model. *Journal of Personality Disorders, 16,* 215–234.

Paris, J. (1998). *Working with traits: Psychotherapy of personality disorders.* Northvale, NJ: Jason Aronson.

Perry, J. C. (1993, Spring). Longitudinal studies of personality disorders. *Journal of Personality Disorders* (Suppl. 1), 63–85.

Roberts, B. W., & DelVecchio, W. F. (2000). The rank-order consistency of personality traits from childhood to old age: A quantitative review of longitudinal studies. *Psychological Bulletin, 126,* 3–25.

Schroeder, M. L., Wormworth, J. A., & Livesley, W. J. (2002). Dimensions of personality disorder and the five-factor model of personality. In P. T. Costa & T. A. Widiger (Eds.), *Personality disorders and the five-factor model of personality* (2nd ed., pp. 149–160). Washington, DC: American Psychological Association.

Shea, M. T., Stout, R. L., Gunderson, J. G., Morey, L. C., Grilo, C. M., McGlashan, T., et al. (2002). Short-term diagnostic stability of schizotypal, borderline, avoidant, and obsessive–compulsive personality disorders. *American Journal of Psychiatry, 159,* 2036–2041.

Steiger, J. H. (1990). Structural model evaluation and modification: An interval estimation approach. *Multivariate Behavioral Research, 25,* 173–180.

Tickle, J. J., Heatherton, T. F., & Wittenberg, L. G. (2001). Can personality change? In W. J. Livesley (Ed.). *Handbook of personality disorders: Theory, research, and treatment.*(pp. 242–258). New York: Guilford Press.

Trull, T. J., & McCrae, R. R. (1994). A five-factor perspective on personality disorder research. In P. T. Costa & T. A. Widiger (Eds.), *Personality disorders and the five-factor model of personality* (pp. 59–71). Washington, DC: American Psychological Association.

Trull, T. J., Useda, D., Conforti, K., & Doan, B. (1997). Borderline personality disorder features in non-clinical young adults: 2. Two-year outcome. *Journal of Abnormal Psychology, 106,* 307–314.

Tyrer, P., & Alexander, J. (1979). Classification of personality disorder. *British Journal of Psychiatry, 135,* 163–167.

Widiger, T. A., Trull, T. J., Clarkin, J. F., Sanderson, C., & Costa, P. T. (1994). A description of the *DSM–III–R* and *DSM–IV* personality disorders with the five-factor model of personality. In P. T. Costa & T. A. Widiger (Eds.), *Personality disorders and the five-factor model of personality* (pp. 41–56). Washington, DC: American Psychological Association.

Zanarini, M. C., Frankenburg, F. R., Sickel, A. E., & Yong, L. (1996). *The Diagnostic Interview for DSM–IV Personality Disorders (DIPD–IV).* Belmont, MA: McLean Hospital.

Zanarini, M. C., Skodol, A. E., Bender, D., Dolan, R., Sanislow, C., Schaefer, E., et al. (2000). The Collaborative Longitudinal Personality Disorders Study: Reliability of Axis I and II diagnoses. *Journal of Personality Disorders, 14,* 291-299.

Zimmerman, M. (1994). Diagnosing personality disorders: A review of issues and research methods. *Archives of General Psychiatry, 51,* 225–245.

Received December 20, 2002
Revision received October 1, 2003
Accepted October 16, 2003 ■