

Associations in the Course of Personality Disorders and Axis I Disorders Over Time

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In this study, the authors examined time-varying associations between schizotypal (STPD), borderline (BPD), avoidant (AVPD), or obsessive–compulsive (OCPD) personality disorders and co-occurring Axis I disorders in 544 adult participants from the Collaborative Longitudinal Personality Disorders Study. The authors tested predictions of specific longitudinal associations derived from a model of crosscutting psychobiological dimensions (L. J. Siever & K. L. Davis, 1991) with participants with the relevant Axis I disorders. The authors assessed participants at baseline and at 6-, 12-, and 24-month follow-up evaluations. BPD showed significant longitudinal associations with major depressive disorder and posttraumatic stress disorder. AVPD was significantly associated with anxiety disorders (specifically social phobia and obsessive–compulsive disorder). Two of the four personality disorders under examination (STPD and OCPD) showed little or no association with Axis I disorders.

Whereas personality disorders and Axis I disorders have repeatedly been found to have high rates of co-occurrence (Dolan-Sewell, Krueger, & Shea, 2001), findings regarding specific associations between pairs of personality disorders and Axis I disorders have been

less consistent. The lack of consistency may be due largely to methodological factors, particularly the frequent use of cross-sectional studies and the variability across studies in sampling methods, base rates of disorders studied, and assessment instruments.

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Oldham and colleagues (1995) examined co-occurrence rates among a range of Axis I and II disorders in a large sample of psychiatric outpatients. That study is also notable for taking base rates into account through the calculation of odds ratios. They reported that personality disorders within Cluster A (“odd-eccentric”) had highly elevated odds of a concurrent psychotic disorder, without increased odds for other Axis I disorders (mood, anxiety, substance use, or eating disorder), which suggests some specificity. However, personality disorders from the other two Axis II clusters also had elevated odds of psychotic disorders (although not as high as for Cluster A).

Borderline personality disorder (BPD), the most frequently studied of the Cluster B (“dramatic–erratic”) personality disorders, has been found to have high rates of co-occurrence with several Axis I disorders. Associations with mood disorders have commonly been reported for BPD, and it has been suggested that BPD may be better conceptualized as a mood disorder (Akiskal, 1994; Akiskal, et al., 1985). However, when the odds of co-occurrence are considered within samples with multiple Axis I and Axis II disorders, the association between BPD and mood disorders appears to be moderate but nonspecific (Dolan-Sewell et al., 2001; Oldham et al., 1995). The lack of specificity applies to bipolar disorder as well as to major depressive disorder (MDD; Dolan-Sewell et al., 2001). BPD has shown a particularly strong association with substance use disorders (Oldham et al., 1995; Skodol, Oldham, & Gallagher, 1999; Tyrer et al., 1997). Elevated rates of anxiety disorders, eating disorders, psychotic disorders, and posttraumatic stress disorder (PTSD) have also been reported for BPD (Dolan-Sewell et al., 2001). The high frequency of childhood abuse and other forms of trauma have led some to consider BPD as a form of chronic and severe PTSD (Herman, 1992).

Personality disorders within Cluster C (“anxious/inhibited”) of Axis II have shown a strong relationship with anxiety disorders (Oldham et al., 1995; Tyrer et al., 1997). However, these disorders have also been associated with elevated odds of a mood, psychotic, or eating disorder (Oldham et al., 1995). Avoidant personality disorder (AVPD) has been associated most frequently with social phobia (Dolan-Sewell et al., 2001) but also with higher rates of obsessive–compulsive disorder (OCD; Skodol et al., 1995) and mood disorders (Oldham et al., 1995). Obsessive–compulsive personality disorder (OCPD) has often been linked with OCD, but findings have been inconsistent (Dolan-Sewell et al., 2001).

Despite the large literature on diagnostic co-occurrence, the meaning of the overlap among Axis I disorders and personality disorders remains unclear. Several conceptual models have outlined the ways in which the apparent comorbidity might be explained (Akiskal, Hirschfeld, & Yerevanian, 1983; Klein, Wonderlich, & Shea, 1993; Lyons, Tyrer, Gunderson, & Tohen, 1997). The simplest explanation is that the disorders are independent in terms of etiology and that apparent high rates of co-occurrence are due to high base rates of each, particularly among individuals who seek treatment. Another conceptualization, frequently referred to as *pathoplasty*, similarly does not assume a shared etiology but emphasizes the influence of one condition on the presentation or course of the other. For example, the presence of alcohol dependence together with diabetes would likely worsen the course of the diabetes. Much of the existing longitudinal research in this area has examined the impact of personality disorders on the treatment outcome or naturalistic course of Axis I disorders, which fits within the framework of a pathoplasty model. Although there is

support for such negative impact of personality disorders (Reich & Vasile, 1993; Shea, Widiger, & Klein, 1992), the findings are not entirely consistent (Mulder, 2002). There are also a large number of studies examining a special form of pathoplasty: the influence of an acute Axis I disorder (usually depression) on the presentation of personality traits or disorders, as expressed through individuals’ self-reports (Widiger, Verheul, & van den Brink, 1999). Depression, in particular, has been reported to negatively distort individuals’ reports of their usual personality, frequently referred to as the *state effect*, in the direction of increased neuroticism, dependency, and introversion, in particular (Widiger, 1993).

Other approaches hypothesize a causal association among disorder pairs, either as risk factors for each other, or as a result of shared etiological factors. Vulnerability models view one disorder or condition as a risk factor, or vulnerability for, another disorder. For example, the features of BPD could result in a vulnerability to develop depression, or alternatively, depression might increase the risk of developing BPD (Gunderson & Phillips, 1991). These models assume that the disorders are distinct but are causally related through conditions of one that increase the risk for the other. Other models suggest that a common disease process underlies what may appear to be distinct disorders. Spectrum models propose that what may appear to be distinct disorders are actually alternate expressions of the same etiological factors. Subclinical models view one disorder as a less severe (subclinical) manifestation of the other. Although these terms are sometimes used interchangeably, the term *subclinical* emphasizes similar but less severe symptoms, whereas the spectrum model may include diverse manifestations of the shared etiological factors.

Another approach relevant to the issue of overlap among disorders is a dimensional view of psychopathology. Whereas dimensional models have focused primarily on personality disorders, Siever and Davis (1991) proposed a model of four psychobiological dimensions that may underlie and crosscut both the Axis I and II disorders. The dimensions are reflected in criteria of both Axis I and II disorders, explaining the frequent co-occurrence of certain disorders via a common pathological process. The four dimensions include impulsivity/aggression (Axis I impulse disorders and borderline and antisocial personality disorders), affective instability (Axis I affective disorders and BPD) cognitive/perceptual (schizophrenia on Axis I and schizotypal personality disorder), and anxiety/inhibition (Axis I anxiety disorders and cluster C “anxious inhibited” personality disorders). Although similar to a spectrum model in the assumption of shared etiological factors, a dimensional model endorses multiple dimensions of psychopathology that may combine in different ways, rather than a single disorder with different expressions or variants (spectrum). Varying combinations of multiple dimensions helps to explain the heterogeneity within disorders as well as the overlap across disorders.

Another often cited explanation of co-occurrence is that the overlap is an artifactual result of overlapping criteria (Widiger & Shea, 1991). Perhaps the clearest example is the association between social phobia and AVPD. The phenomenon of overlapping criteria is essentially consistent with the notion of crosscutting dimensions, or with the subclinical concept, whereby characteristics of each disorder are viewed as manifestations of a common dimension of psychopathology. Either view would suggest that the disorders are not entirely distinct.

No single model is likely to explain the complex associations between personality disorders and Axis I disorders; even among

specific pairs of disorders, the associations may be expressions of more than one model. Further, despite the value of such models in highlighting the various ways that disorders may be related, it is very difficult to distinguish among them empirically (Klein et al., 1993). For example, both the vulnerability and the spectrum models might be characterized by one condition preceding and increasing the risk of the other condition. The conceptual difference is that the vulnerability model assumes two different pathological processes or disorders, whereas the spectrum and dimensional models assume a common pathological process. The expected temporal patterns, or the amount of time between onset or offset of related conditions is typically not specified in these models. However, if two diagnostic conditions are measuring a common dimension of psychopathology, then change in one should be correlated with change in the other. In other words, the changes should occur closely in time. This would not be necessarily true for the vulnerability model, as the condition that represents “vulnerability” might exist without the occurrence of the second condition for any length of time. Similarly, there would be no assumptions about temporal proximity of improvements (or “remissions”) in the conditions. Longitudinal studies, in contrast to cross-sectional studies, provide the advantage of the opportunity to examine associations in the course of disorders over time.

Aside from studies examining the impact of personality disorders on treatment outcome of Axis I disorders, there are only a few existing longitudinal studies designed to examine the association of Axis I disorders and personality disorders. The existing research has shown that personality disorders during adolescence increase risk for Axis I disorders during early adulthood (Johnson et al., 1999) and, similarly, that Axis I disorders during adolescence increase risk for personality disorders in early adulthood (Kasen et al., 2001). However, sample sizes have precluded examination of course associations between specific pairs of Axis I disorders and personality disorders. And although studies with follow-ups several years after initial assessments are valuable in examination of risk, such studies are unable to examine the timing of changes in pairs of disorders. An exception is a recent study that examined the association of depression and BPD symptoms measured in three assessments over five years in 84 outpatients with dysthymic disorder (Klein & Schwartz, 2002). They used structural equation modeling to test different models of associations. Of the various models the authors tested, a model of a common latent factor provided the best fit, suggesting that depressive symptoms and BPD features arise from partially overlapping processes.

Using data from the Collaborative Longitudinal Personality Disorders Study (CLPS; Gunderson et al., 2000), we examined course associations among four personality disorders and Axis I disorders over two years of prospective follow-up. The CLPS included participants with at least one of four *DSM-IV* personality disorders: schizotypal (STPD), BPD, (AVPD, or OCPD. Participants entered the study meeting full criteria for one or more of these four personality disorders and a range of Axis I disorders, which provided the opportunity for us to examine whether pairs of personality disorders and Axis I disorders changed (i.e., improved) together. If the criteria of two disorders are manifestations of one or more shared dimensions, changes in the course of each should occur in close proximity. Our hypotheses were based on the Siever and Davis (1991) dimensions relevant to three of the CLPS personality disorders. For BPD, we predicted course associations with major depression (affective instability dimension), substance abuse

(impulse dimension), and eating disorders (impulse dimension). We also predicted an association between BPD and PTSD (affective instability dimension) because affect regulation difficulties are also characteristic of PTSD, and BPD has been conceptualized as a form of chronic and severe PTSD (Herman, 1992). For the two Cluster C personality disorders, AVPD and OCPD, we predicted course associations with anxiety disorders (anxiety/inhibition dimension).

Method

Participants

Detailed descriptions of the CLPS aims, background, design and methods (Gunderson et al., 2000), and sample characteristics including co-occurring Axis I and II disorders (McGlashan et al., 2000) have been reported separately. Participants were recruited from clinical services affiliated with each of the four recruitment sites participating in the study: Brown University, Department of Psychiatry and Human Behavior; New York State Psychiatric Institute; McLean Hospital; and Yale University, Department of Psychiatry. Additional participants with current or past psychiatric treatment were recruited by postings or advertising. Exclusion criteria were age outside the range of 18 and 45; current or lifetime schizophrenia or schizoaffective disorder; currently active psychosis; confusional states resulting from organic disorders, post-ECT (electroconvulsive therapy) status, or substance intoxication or withdrawal; and estimated IQ of less than 85. The baseline sample included 573 participants with at least one of the four target personality disorders. The CLPS also includes a comparison group of participants with MDD without personality disorder, which is not included in the current report.

The current report is based on 544 personality disorder participants (95% of the baseline personality disorder sample) with follow-up data. Of the participants, 420 (77%) were Caucasian, 62 (11%) were African American, 47 (9%) were Hispanic, and 15 (3%) were other minorities. Women ($n = 348$) comprised 64% of the sample. The mean Hollingshead–Redlich score was 3.07 ($SD = 1.06$). Thirty-seven percent were from the highest two categories (1 and 2), 28% were from Category 3, and 35% were from the lowest categories (4 and 5). The mean age was 32.8 ($SD = 8.1$). Two hundred twenty-three met DIPD-IV criteria for BPD, 312 for AVPD, 94 for STPD, and 251 for OCPD. There were no demographic differences between the current study sample of 544 participants and the 29 participants without follow-up data.

Procedure

Baseline evaluation. All participants signed written informed consent, following a full explanation of study procedures. A shortened version of the Personality Diagnostic Questionnaire (PDQ-IV; Hyler, Skodol, Kellman, Oldham, & Rosnick, 1990) consisting of items for the four targeted personality disorders was used to screen for potential participants. Individuals screening positive for one or more of the personality disorders were referred for further diagnostic assessment with the Structured Clinical Interview for *DSM-IV* Axis I disorders, Patient Edition (SCID-I/P; First, Spitzer, Gibbon, & Williams, 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, or the equivalent clinical experience. Interviewers received extensive training and continued reliability monitoring in the administration of the major diagnostic measures (Axis I and II; Gunderson et al., 2000). DIPD-IV diagnoses were required to have convergent support from either the self-report Schedule for Nonadaptive and Adaptive Personality (SNAP; Clark 1993) or an independent clinician-rated Personality Assessment Form (PAF; Shea, Glass, Pilkonis, Watkins, & Docherty, 1987).

Follow-up evaluations. Participants were interviewed at 6 months, 1 year, and 2 years following the baseline assessment. The course of each of the four study personality disorders over the preceding interval was assessed using a modified version of the DIPD-IV (Follow-Along Version; DIPD-IV-FAV), and the course of all co-occurring Axis I disorders was assessed using the Longitudinal Follow-up Evaluation (LIFE; Keller et al., 1987). The DIPD-IV-FAV interviews were not blind, and, when possible, they were conducted by the same interviewer.

Measures

DIPD-IV. The DIPD-IV is a semi-structured interview for assessing each of the 10 *DSM-IV* personality 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 clinically insignificant*; 2 = *definitely present*). The time frame covered is the prior 2 years. Given the presence of sufficient criteria based on the prior 2 years, the interviewer next asks whether the traits present have been characteristic of the person for most of his or her adult life, to establish the final diagnosis. As described in detail elsewhere (Zanarini et al., 2000), an interrater reliability study was conducted with CLPS interviewers through the use of videotaped interviews generated and rated by interviewers at all sites. A test-retest reliability study was also conducted at each site, with interviews repeated within 1 to 2 weeks by a second interviewer blind to the initial interview. For the four study personality disorders, 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).

DIPD-IV-FAV. To assess the longitudinal course of the study personality disorders, the DIPD-IV was modified to record the presence of each criterion for the four personality disorders for each month of the follow-up interval. Interviewers asked the standard DIPD-IV probes for presence of each criterion; if the criterion was 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. We required a rating of 2 (*definitely present*) for a criterion to be counted as present.

To estimate the reliability of retrospective reporting by month on the DIPD-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. On the basis of 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).

SCID-I. The SCID-I is a semistructured diagnostic interview with established reliability that is used to diagnose 33 Axis I disorders by *DSM-IV* criteria. SCID-I interviews were also included in the interrater and test-retest reliability studies described above. Interrater reliability kappas for Axis I diagnoses for CLPS interviewers ranged from .57 to 1.00, with a median of .76. Test-retest kappas ranged from .35 to .78 with a median of .64 (Zanarini et al., 2000).

LIFE. The LIFE is a semistructured interview rating system with demonstrated reliability for assessing the longitudinal course of mental disorders. On the basis of the information obtained from the interview covering the interval followed, weekly ratings are made through the use of Psychiatric Status Ratings (PSRs) for each Axis I disorder present. Good to excellent reliability has consistently been demonstrated for the LIFE (Warshaw, Dyck, Allsworth, Stout, & Keller, 2001; Warshaw, Keller, & Stout, 1994). A separate reliability study was not conducted for the LIFE with CLPS interviewers. However, CLPS interviewers were trained at the Brown site, by the official LIFE training staff. The training staff was available throughout the study for consultation and questions regarding the LIFE interview and ratings. CLPS uses a 3-point scale for most of the Axis

I disorders, indicating whether the individual meets full criteria (PSR = 3), partial criteria (PSR = 2), or has minimal or no symptoms (PSR = 1) for the given disorder. A 6-point scale is used for the assessment of major depression; the 6 points are collapsed to three categories for the present report (full criteria = PSR 5 or 6; partial criteria = PSR 3 or 4; minimal or no symptoms = PSR 1 or 2).

Data Analyses

Our analyses were based on DIPD-IV positive diagnoses, regardless of co-occurring diagnoses of the other study personality disorders. We judged participants being followed for Axis I disorders to be “remitted” for the purposes of this study if they experienced at least eight successive weeks with minimal or no symptoms. This definition of remission has been used widely in studies of mood and other Axis I disorders (e.g. Keller, Shapiro, Lavori, & Wolfe, 1982; Stout, Dolan, Dyck, Eisen, & Keller, 2001). While it is unclear what period of time should define “remission” for personality disorders, we used a similar definition (minimum of at least 2 months with no more than two criteria) to allow an indicator of change comparable with the Axis I disorders.

We conducted two sets of analyses to examine the associations in course improvement in disorder pairs. Wilcoxon tests for homogeneity of survival curves of time to remission from the personality disorder by a given Axis I disorder status (not present at baseline, present at baseline and remitted during follow-up, present at baseline and not remitted during follow-up) were conducted for personality disorder–Axis I disorder pairs. We required a minimum of 20 participants in each of the three strata for the specific Axis I disorder–personality disorder pair for analyses. These analyses address whether a remission in the Axis I disorder—occurring at any time during the interval—is associated with an increased probability of remission from the personality disorder. Such a finding would suggest the course of the disorders are related in some fashion (ruling out the independence model) but would not provide more support for one versus another of the remaining models.

Without ruling out other models of association, changes occurring closely in time would be most consistent with the model of shared dimensions. For those participants with both disorders in a given pair at baseline, we used proportional hazard regression analysis (Cox, 1972; Allison, 1984) with time-varying covariates to examine whether changes in course were correlated (i.e., whether improvements in both disorders occurred around the same time). Use of time-varying predictors allows the examination of course of one disorder subsequent to a change (in this case improvement) in the status of the time-varying predictor. More specifically, our analyses examined the association of improvement in the Axis I disorder with remission from the personality disorder, and the reverse, improvement in the personality disorder with remission from the Axis I disorder, within a 1-month time frame. We examined the associations in both directions, as the hypotheses predict a temporal association of change but not a particular sequence. In the prediction of personality disorder remission, the time-varying predictor was the Axis I PSR for the last week of the month preceding the time point being analyzed. In the prediction of Axis I remission, the time-varying predictor was the personality disorder status (full criteria; partial criteria [more than 2 but less than full criteria]; minimal criteria [0 to 2]) for the month preceding the time point being analyzed. For each pair of disorders investigated, we included only cases positive at baseline for both. Cases positive for a given disorder at baseline, but remitting in Month 1 of the follow-up (i.e., the first month after baseline) were excluded in analyses examining that disorder as the dependent variable because the value for the predictor variable (preceding month) would be fixed at baseline. Thus, the number of cases in specific pairs of disorders may differ depending on which of the pair is the dependent variable. For AVPD and OCPD, we examined associations with any anxiety disorder as well as specific anxiety disorders. We examined all pairs of the four personality disorders and Axis I disorders that had at least 20 cases.

Hazard (risk) ratios provide an estimate of association between the predictor and dependent variables; the corresponding two-tailed *p* values from the Cox regression analysis test whether the risk ratio (RR) is different from 1.0. A risk ratio that is less than 1 indicates that improvement (lower value) in the predictor variable is associated with a higher likelihood of remission, that is, improvement on the predictor disorder predicts getting better, or remitting, from the disorder that is the dependent variable. In addition to statistical significance, the size of the association can be estimated by the value of the risk ratio: Risk ratios of .50 to .67 and 1.5 to 2.0 are roughly equivalent to medium effect sizes. Risk ratios less than .50 and greater than 2.0 are equivalent to large effect sizes (Schoenfeld, 1983). An alpha level of *p* < .05 was used for statistical significance for all analyses.

Results

A sufficient sample (20 or more) was available to examine associations of one or more of the personality disorders with MDD, any anxiety disorder (and specific anxiety disorders of panic disorder, social phobia, generalized anxiety disorder, OCD, and PTSD), eating disorders, and substance use disorders.

Wilcoxon Tests of Homogeneity of Survival Curves

Remission rates for BPD differed by the status of MDD, $\chi^2(2, N = 223) = 12.4, p = .002$; PTSD, $\chi^2(2, N = 223) = 6.8, p = .031$, (see Figures 1 and 2); and also by the status of any anxiety disorder, $\chi^2(2, N = 223) = 9.0, p = .011$. The latter finding is partially due to the PTSD association, but there was also an unpredicted association with panic disorder status, $\chi^2(2, N = 223) = 6.0, p = .049$. The pattern was similar for each of the associations: Among BPD participants with the respective Axis I

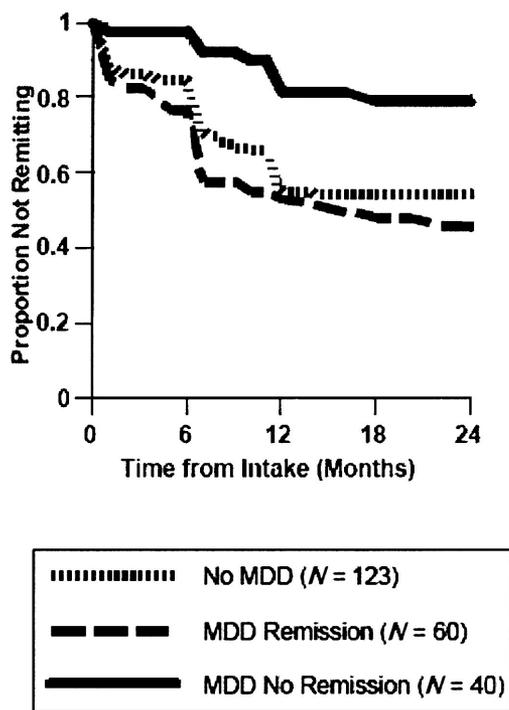


Figure 1. Remission status for borderline personality disorder by major depression status. MDD = major depressive disorder.

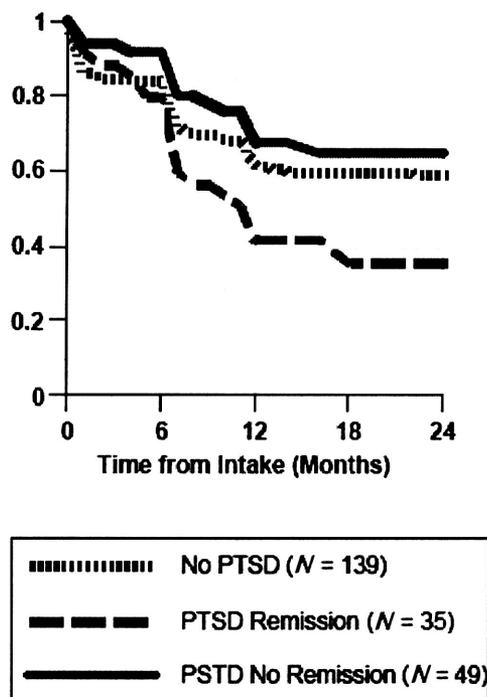


Figure 2. Remission status for borderline personality disorder by post-traumatic stress disorder (PTSD) status.

disorder, those with remission of the Axis I disorder during the follow-up had the highest probability of remitting from BPD, whereas those for whom the Axis I disorder did not remit had the lowest probability of BPD remission. BPD remission did not differ by eating disorder status, or by substance abuse/dependence status.

Remission rates for AVPD differed by the status of any anxiety disorder, $\chi^2(2, N = 312) = 9.9, p = .007$ (see Figure 3). In terms of specific anxiety disorders, differences were found for social phobia, $\chi^2(2, N = 312) = 14.1, p < .001$, and OCD, $\chi^2(2, N = 312) = 8.1, p = .017$. There was also an unpredicted difference for AVPD \times MDD status, $\chi^2(2, N = 312) = 8.2, p = .016$. In all instances, those AVPD participants who did not remit from the Axis I disorder were less likely to remit from AVPD, relative to those remitting from the Axis I disorder. Remission for OCPD did not differ by any anxiety disorder status, $\chi^2(2, N = 251) = 3.2, p = .20$.

Proportional Hazard Regression Analyses

Table 1 summarizes findings from the proportional hazard regression analyses for improvement in Axis I disorders as predictors of personality disorder remission, and Table 2 shows the same for improvement in personality disorders predicting Axis I remission. Risk ratios and their associated *p* values are presented for each combination examined. Two of the four hypothesized associations for BPD were statistically significant (*ps* < .05). BPD and MDD were associated in both directions. Improvement in BPD also predicted remission from PTSD, although the reverse was not true (PTSD improvement did not significantly predict BPD remission). There were no significant associations between BPD and either substance abuse/dependence or eating disorders.

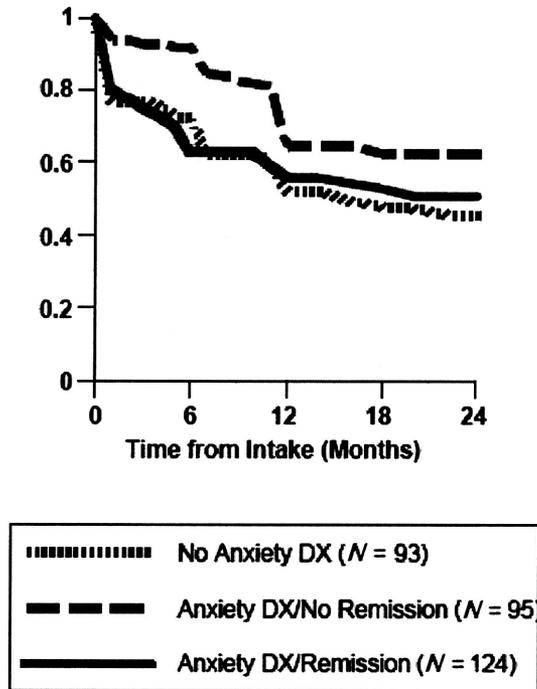


Figure 3. Remission status for avoidant personality disorder by any anxiety disorder (AD) status.

The hypotheses for anxiety disorders were supported for AVPD but not for OCPD. When we examined anxiety disorders as a group, the associations with AVPD were significant in both directions. In terms of specific anxiety disorders, social phobia and AVPD were significantly associated in both directions, as were OCD and AVPD. Additional significant associations that we did not predict included major depression predicting remission from STPD and AVPD improvement predicting remission from major depression.

Given that there is overlap among BPD and AVPD diagnoses, we conducted further analyses to examine the independence of the

associations that each had with MDD. When both AVPD and BPD were treated simultaneously as time-varying predictors of remission for MDD, BPD remained a significant predictor (RR = 0.68, $p = .001$). In contrast, AVPD was no longer significantly predictive of remission from MDD when BPD was present (RR = 0.94, $p = .64$).

Discussion

This is the first study, to our knowledge, to investigate the longitudinal associations between personality disorders and Axis I disorders using a prospective design and continuous measures of course for both. For subgroups of AVPD and BPD participants with the relevant Axis I disorders, the findings provided partial support for the predictions derived from the crosscutting dimensional model (Siever & Davis, 1991). The predicted associations for OCPD were not significant.

BPD course over follow-up showed associations with MDD, consistent with the affective instability dimension proposed to underlie the mood disorders on Axis I and the Cluster B (dramatic-erratic) personality disorders. Failure to remit from MDD was associated with a substantially reduced likelihood of remitting from BPD, and the time-varying associations showed that changes in status for these disorders were closely linked in time. These findings, despite the difference in approach, are consistent with those of Klein & Schwartz (2002), who examined longitudinal associations between BPD and early onset dysthymia. Outpatients with dysthymia were assessed three times over 5 years through the use of semistructured interviews. Using structural equation modeling, the authors tested multiple models of the associations (no association, direct effects in both directions over short periods of time, lagged direct effects in which one condition influenced the other over a longer period, and a fixed common factor underlying both disorders). The fixed common factor model was the best fitting of the models, with an excellent fit to the data. The authors noted that the latent fixed common factor could reflect several possible etiologies, including a shared genetic substrate, a shared temperamental vulnerability such as neuroticism, or environmental events such as early abuse or neglect (Klein & Schwartz, 2002).

Table 1
Improvement in Axis I Disorders as Predictors of Remission From Personality Disorders

Axis I disorder	STPD (N = 94)				BPD (N = 223)				AVPD (N = 312)				OCPD (N = 251)			
	RR	p	n	Remit (N = 32)	RR	p	n	Remit (N = 95)	RR	p	n	Remit (N = 142)	RR	p	n	Remit (N = 142)
MDD	0.69	.04	34	10	0.77	.02	90	30	0.82	.06	114	32	1.03	.79	71	28
Any Anxiety Disorder	1.00	.99	66	15	0.94	.75	137	49	0.66	.01	188	59	0.81	.23	127	45
Panic disorder	0.46	.24	30	4	1.01	.97	62	8	0.51	.28	61	7	1.68	.41	32	5
Social phobia	0.87	.80	20	7	0.79	.49	38	14	0.51	<.01	87	26	1.17	.64	37	14
GAD	—	—	17	4	1.59	.28	35	11	1.23	.44	58	19	0.85	.57	54	15
OCD	0.75	.50	23	7	0.54	.05	28	13	0.41	.01	37	12	1.04	.89	41	16
PTSD	1.10	.88	26	7	0.82	.36	78	32	1.43	.41	63	14	0.71	.33	32	14
Eating disorder	—	—	9	5	0.94	.82	44	17	1.21	.51	54	20	1.29	.40	37	21
Substance abuse/dependence	0.81	.62	24	8	0.77	.43	60	12	0.87	.72	54	9	0.52	.12	25	10

Note. The n column presents the number of participants with both the Axis I and personality disorders. The Remit column presents the number of participants remitting from the personality disorder. Dashes indicate that no analyses were conducted as a result of insufficient sample size. STPD = schizotypal personality disorder; BPD = borderline personality disorder; AVPD = avoidant personality disorder; OCPD = obsessive-compulsive personality disorder; RR = risk ratio; MDD = major depressive disorder; GAD = generalized anxiety disorder; OCD = obsessive-compulsive disorder; PTSD = posttraumatic stress disorder.

Table 2
Improvement in Personality Disorders as Predictors of
Remission From Axis I Disorders

Personality disorder	RR	<i>p</i>	<i>n</i>	Remit
MDD (<i>N</i> = 210; Remit <i>n</i> = 139)				
STPD	0.67	.16	36	18
BPD	0.57	<.01	84	44
AVPD	0.72	.04	105	60
OCPD	0.83	.23	70	47
Any anxiety disorder (<i>N</i> = 358; Remit <i>n</i> = 136)				
STPD	1.02	.96	53	9
BPD	0.68	.09	112	30
AVPD	0.59	<.01	167	45
OCPD	0.80	.21	120	38
Panic disorder (<i>N</i> = 113; Remit <i>n</i> = 62)				
STPD	—	—	7	4
BPD	1.23	.65	24	14
AVPD	—	—	19	12
OCPD	—	—	9	6
Social phobia (<i>N</i> = 116; Remit <i>n</i> = 50)				
STPD	—	—	19	8
BPD	1.17	.68	38	16
AVPD	0.62	.03	87	28
OCPD	1.34	.44	39	13
GAD (<i>N</i> = 120; Remit <i>n</i> = 71)				
STPD	—	—	17	5
BPD	0.78	.42	33	15
AVPD	0.81	.34	53	27
OCPD	0.95	.83	38	9
OCD (<i>N</i> = 80; Remit <i>n</i> = 33)				
STPD	0.91	.89	20	8
BPD	0.78	.47	28	16
AVPD	0.50	.02	38	21
OCPD	0.77	.52	38	20
PTSD (<i>N</i> = 130; Remit <i>n</i> = 48)				
STPD	1.17	.84	28	4
BPD	0.54	.01	84	34
AVPD	0.63	.09	76	25
OCPD	0.66	.18	49	17
Eating disorder (<i>N</i> = 95; Remit <i>n</i> = 48)				
STPD	—	—	9	2
BPD	1.04	.90	40	14
AVPD	0.77	.23	53	21
OCPD	0.98	.96	40	13
Substance abuse/dependence (<i>N</i> = 110; Remit <i>n</i> = 50)				
STPD	—	—	17	1
BPD	1.16	.67	51	19
AVPD	—	—	46	11
OCPD	0.94	.85	29	9

Note. The *n* column presents the number of participants with both the Axis I and personality disorder. The Remit column presents the number of participants remitting from the personality disorder. Dashes indicate that no analyses were conducted as a result of insufficient sample size. Blank cells indicate that stable convergence was not achieved. STPD = schizotypal personality disorder; BPD = borderline personality disorder; AVPD = avoidant personality disorder; OCPD = obsessive-compulsive personality disorder; RR = risk ratio; MDD = major depressive disorder; GAD = generalized anxiety disorder; OCD = obsessive-compulsive disorder; PTSD = posttraumatic stress disorder.

BPD changes were also linked, as predicted, with changes in PTSD. BPD participants with PTSD that remitted over follow-up were more likely to remit from BPD than those with nonremitting PTSD, or than those without PTSD at baseline. Improvement in BPD increased the likelihood of subsequent remission from PTSD in the time-varying analyses, although the time-varying association was not significant in the opposite direction. One possible explanation is that BPD is a more complex disorder, sharing some but not all dimensions with PTSD. Improvement in BPD predicting proximal remission from PTSD could reflect the improvement of one or more shared dimensions (for example, affect dysregulation or brief dissociative episodes). On the other hand, remission from PTSD was associated with increased probability of remission from BPD in the analyses in which we examined associations over the interval as a whole not restricted to the 1-month interval. This discrepancy might suggest that remission from PTSD could over time (i.e., when not restricted to the 1-month interval) lead to improvement in nonshared dimensions of BPD. Alternatively, our methods and data may have been insufficiently sensitive to the associations in both directions, meaning the discrepancy is due to measurement error.

An unpredicted association was found for BPD and panic disorder: BPD participants with unremitting panic disorder were less likely to remit from BPD. Associations between BPD and panic disorder were not found in the time-varying analyses, however, meaning that the changes are not occurring in close proximity. Similarly, a possible reason for the discrepancy may be that conditions facilitating the remission from panic disorder, and/or changes resulting from panic disorder remission, may over a longer period of time effect improvement in BPD, in contrast to a dimension of psychopathology shared by the two disorders.

AVPD showed significant associations with the broad group of anxiety disorders, consistent with the anxiety/inhibition dimension. In terms of specific anxiety disorders, associations were found for social phobia and OCD. AVPD showed unpredicted associations with major depression, but this association disappeared in the time-varying analysis when BPD was controlled. Thus, when both BPD and AVPD were present and both improved, it was the improvement in BPD that had the strongest link with remission from MDD. This finding speaks to the importance of examining the possible influence of co-occurring personality disorder diagnoses when associations between personality disorders and Axis I disorders are found.

OCPD did not show the predicted changes with anxiety disorders, failing to support a crosscutting anxiety/inhibited dimension underlying OCPD. Of note in this regard are findings from studies examining the factor structure of dimensional scores from the 11 *DSM-III* or *DSM-III-R* personality disorders. Although a factor structure consistent with the three Axis II clusters has typically been found, the exception has been for OCPD, which has either not loaded on any factors (Zimmerman & Coryell, 1990) or formed its own factor (Kass et al., 1985). These findings suggest that OCPD may not be well characterized as a disorder of the anxious-inhibited cluster.

Although the findings support some of the hypotheses, it is important to emphasize that the associations found in the time-varying analyses apply only to subsets of the BPD and AVPD participants: those with the co-occurring Axis I diagnosis at baseline. Fewer than half (45%) of the BPD participants had MDD at baseline, and 38% had PTSD. Of the AVPD participants, less than

one third (31%) had social phobia, and only 14% had OCD at baseline. This reflects the heterogeneity and multidimensional nature of the personality disorders, that is, not all individuals meeting criteria for a given personality disorder are characterized by disturbances on all of the dimensions underlying the criteria (Shea, 1992). BPD, in particular, has been shown in several studies to consist of multiple dimensions. Confirmatory factor analyses of BPD criteria from CLPS assessments supported a three-factor model, labeled disturbed relatedness, behavioral dysregulation (impulsivity), and affective dysregulation (Sanislow et al., 2002). Two of these (impulsivity and affective dysregulation) correspond to the Siever and Davis (1991) dimensions. The findings of correlated changes in course for the BPD and AVPD subgroups with the respective Axis I disorders does not mean that the disorders are synonymous, but rather that they may share some dimensions of psychopathology.

Research on biological abnormalities has shown both similarities and differences between BPD and depression. Most biological studies of BPD have not controlled for the presence of major depression, which may contribute to the inconsistent findings (Koenigsberg et al., 1999). It may be that the biological similarities are stronger in those BPD patients with affective dysregulation. A further question to consider in terms of a shared psychobiological dimension of affective dysregulation is the differences in quality of the mood disturbance. Major depression typically involves a more fixed depressed mood, whereas depression in BPD tends to be characterized by more mood reactivity. One hypothesis is that low serotonergic activity and high cholinergic sensitivity may be common to both BPD and depression, but different abnormalities in noradrenergic function, which modulates the extent of engagement with the social environment, may result in the differences in reactivity of mood (Koenigsberg et al., 1999). Though this work is preliminary and far from definitive, it illustrates the more general point, which is that two disorders (BPD and depression, in this case) may share some etiologic mechanisms and differ on others. Furthermore, the shared mechanisms may apply only to a subset of individuals given the multidimensional nature of the personality disorder diagnoses.

The current findings, though consistent with the notion of shared dimensions of psychopathology across Axis I for subgroups of individuals with BPD and AVPD, clearly do not rule out other mechanisms that may contribute to Axis I co-occurrence for these as well as for other personality disorders. Indeed, it is likely that multiple mechanisms contribute to the co-occurrence of the personality and Axis I disorders. The interpersonal difficulties that are the hallmark of personality disorders, for example, put affected individuals at high risk for the kinds of chronic and acute life events (failures and losses) that then increase the risk of the onset and/or maintenance of depressive or anxiety disorders. In the current study, we examined the co-varying associations in improvement across disorders. A vulnerability model would predict that one disorder precedes the other but would not necessarily predict that both disorders would improve together. Thus the current data are not well suited for testing a vulnerability model, which are ideally tested in studies allowing prospective observation of the first onset of disorders. Other methodological approaches are essential to a more definitive understanding of the nature and mechanisms involved in the associations. More intensive study of the timing (sequence) and psychosocial context, including stressors and precipitants, surrounding the onset or re-

currence of the disorders would be informative. Studies of neurobiological correlates would be a valuable strategy to shed light on the extent to which mechanisms may be shared or distinct. For example, time-varying associations of repeated measures of serotonergic activity, with the status of BPD and MDD symptoms over time could be used to test the hypothesis of a shared mechanism for BPD and MDD.

BPD does not show the predicted association with substance use or eating disorders, which are representative of the impulsive aspect of the impulsive/aggressive dimension. This suggests that despite their frequent co-occurrence, changes in the manifestations of these disorders are not closely linked in time. Again, the absence of longitudinal associations does not rule out the possibility of causal associations (for example, vulnerability, complication, or other risk models). Several models have been articulated to explain the high rates of co-occurrence between BPD and substance use disorders (Trull et al., 2000). These include the use of substances to cope with negative affective states in BPD, consistent with a vulnerability or risk model, or that both disorders may be mediated by common constitutional and family environmental factors. Despite likely causal associations in the development of these disorders, the current findings of no associations in course are inconsistent with the notion that both are manifestations of the same currently operative mechanisms.

Another explanation for the current findings is that the associations found are artifacts of a state effect. That is, participants who were in an episode of depression or anxiety disorder at baseline gave a distorted report of their longer term personality as a result of the coloring of their perceptions by their mood state. This would mean that such personality disorder diagnoses were false positives and that the apparent correlation in improvement was really the result of just the depression (or anxiety) and the associated distorted perceptions remitting. However, if this were the case, the personality disorder criteria falsely reported as characteristic when depressed (or anxious), should be denied for the entire assessment interval once the individual is remitted from the Axis I disorder. This would mean, for example, that for participants with major depression that remitted by the 6-month assessment, the personality disorder remission should occur in Month 1 of the follow-up. This did happen in some cases (see Gunderson et al., 2003, for a description and discussion of early remissions), but the improvement in the personality disorder status in relation to the Axis I remission (and vice versa) generally occurred over a period of several months.

A limitation of the current study is the collection of data on four personality disorders, which precludes examinations of other possible personality disorder–Axis I associations. Furthermore, the study exclusion of a history of schizophrenia or schizoaffective disorder precludes examination of personality disorder associations (notably STPD) with Axis I psychotic disorders. Also, the number of cases naturally varied for different pairs of disorders, influencing the statistical power to find associations. For example, the risk ratio of .77 for MDD predicting BPD remission, on the basis of 90 participants, reaches our significance level of .05, whereas the risk ratio of .54 for OCD predicting BPD remission, on the basis of 28 participants, does not. Thus, some relationships may be underestimated. On the other hand, the differences in the size of the pairs is also meaningful, as larger numbers reflect associations among the specific pairs of personality disorders and Axis I disorders.

An additional issue concerns the ability to determine precisely the timing of the symptomatic improvements across the disorders. Interviews covered the prior 6 or 12 months, and despite efforts to determine as precisely as possible the timing of symptoms and criteria changes, the interviewers necessarily relied on participants' memories of these changes. Although our reliability study of retrospective ratings described above suggested good concordance over time, more frequent assessments would provide a better test of the timing of the changes. Additionally, given that the same interviewers assessed the course of the personality disorders and Axis I disorders, it is possible that interviewer bias influenced the results. However, it is unlikely that the interviewers had a specific bias in terms of personality disorder–Axis I relationships; the fact that associations were found for only two of the personality disorders, and for a minority of Axis I disorders, argues against interviewer bias as a major factor in the findings. An advantage of nonblind interviews is increased knowledge of the study participant and thus perhaps the ability to make more valid judgments.

In summary, our findings support some of the hypothesized associations. BPD shows a significant association with MDD and with PTSD. AVPD shows a significant association with anxiety disorders, and specifically with social phobia and OCD. The findings for BPD and AVPD are consistent with the model of crosscutting dimensions (Siever & Davis, 1991) for subsets of individuals with these disorders. Other models, however, may also be applicable. Future reports should examine associated disorders in more depth, including the role of specific personality disorder criteria in the associations found and the investigation of longer time lags. Longer follow-up should also allow for an examination of time-varying associations between the personality disorders and recurrence and new onsets of Axis I disorders. Other studies are needed to address the associations of other personality disorders with Axis I disorders.

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