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Exploring Personality Diagnosis Stability Following Acute Psychotherapy for Chronic Posttraumatic Stress Disorder

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Abstract

Background—Axis I comorbidity complicates diagnosing Axis II personality disorders (PDs). PDs might influence Axis I outcome. No research has examined psychotherapy effects on PDs of treating Axis I comorbidity. Secondary analysis of a randomized controlled trial examined PD diagnostic stability after brief psychotherapy of chronic posttraumatic stress disorder (PTSD).

Methods—Patients with chronic PTSD were randomly assigned to 14 weeks of Prolonged Exposure, Interpersonal Psychotherapy, or Relaxation Therapy. Assessments included SCID-P and SCID-II at baseline, week 14, and for treatment responders (30% Clinician-Administered PTSD Scale improvement, defined *a priori*) at week 26 follow-up. We hypothesized patients whose PTSD improved would retain fewer baseline PD diagnoses posttreatment, particularly with personality traits PTSD mimics: e.g., paranoid and avoidant.

Results—Forty-seven (47%) of 99 SCID-II patients evaluated at baseline received a SCID-II diagnosis: paranoid (28%), obsessive-compulsive (27%), and avoidant (23%) PDs were most prevalent. Among 78 patients who repeated SCID-II evaluations posttreatment, 45% (N=35) had baseline PD diagnoses, which 43% (N=15/35) lost at week 14. Three (7%) patients without baseline PDs acquired diagnoses at week 14; ten others shifted diagnoses. Treatment modality and PTSD response were unrelated to PD improvement. Of treatment responders re-evaluated at follow-up (N=44), 56% with any baseline Axis II diagnosis had none at week 26.

Discussion—This first evaluation of Axis I psychotherapy effects on personality disorder stability found that acutely treating a chronic state decreased apparent trait – across most PDs observed. These exploratory findings suggest personality diagnoses may have limited prognostic meaning in treating chronic PTSD.

Keywords

personality disorder; PTSD; IPT; CBT; assessment/diagnosis

Researchers and clinicians have long recognized the difficulty in diagnosing personality disorders (PDs) in the setting of an Axis I disorder (e.g., 1). This report describes the effect

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on comorbid DSM-IV Axis II PD status of short-term psychotherapies targeting an Axis I disorder, posttraumatic stress disorder (PTSD).

Psychiatric nosology has historically distinguished between episodic illness (“state”) and underlying, putatively stable personality or temperament (“trait”). This dichotomy has proved simplistic, however. The phrase “state versus trait” describes a longstanding confusion in psychiatric comorbidity. In 1980, DSM-III codified these categories as Axis I disorders and Axis II personality disorders. They frequently co-occur (2,3), and seeming distinctions between Axis I “state” episodes and persisting Axis II “trait” PDs quickly blur. The passive, anxious, socially-withdrawn “state” of major depressive disorder, for example, overlaps Axis II cluster C “trait” diagnoses such as dependent or avoidant PD (4). Acutely ill patients often act and experience their lives as if following chronically established patterns, conflating attributes of an episode with their premorbid personality.

Axis I and II disorders have complex interactions. Personality disorders, developed by late adolescence and characterized by dysfunctional behavioral patterns, may produce life and relationship problems that trigger depressive or anxiety episodes in vulnerable individuals (5). The impulsivity of borderline PD might increase risk for traumatic life events, increasing likelihood of developing comorbid PTSD (6); conversely, childhood abuse might influence borderline PD. Repeated or prolonged mood or anxiety disorder episodes may “scar” personality, phenocopying Axis II diagnoses (7). Axis I and Axis II disorders might share heritability (8); or individuals might develop etiologically independent Axis I and II disorders (9).

The two diagnostic categories blur further when Axis I disorders persist chronically: passive, avoidant patients with dysthymic or social anxiety disorders frequently meet Cluster C PD criteria (10). Clinicians and researchers often struggle to determine whether a patient’s Axis I disorder is exacerbating a PD, or *vice versa*; or whether an apparent PD is an epiphenomenon of chronic Axis I disorder. This difference matters clinically, affecting clinical stance and treatment selection (11,12): which disorder should clinicians treat first? Although DSM-5 (13) recently discarded the multiaxial system, the diagnostic problem remains.

Chronic PTSD exemplifies this dilemma. PTSD has high Axis I and II comorbidity. Zanarini et al., e.g., found 58% comorbidity of DSM-III-R PTSD among 290 patients with borderline PD (14). Conversely, among 115 male combat veterans with DSM-IV PTSD and comorbid major depression, Dunn and colleagues found 45% prevalence of PDs, particularly paranoid (17%), obsessive-compulsive (17%), avoidant (12%), and borderline (9%)(15). A national survey (N=34,653) found 30% of individuals with borderline PD also met PTSD criteria; 24% with PTSD met borderline criteria (16).

Many patients presenting for treatment with chronic PTSD have apparent PDs: they mistrust their environments and the people therein (paranoid); withdraw socially (avoidant); have anger outbursts (borderline); etc. Do these apparent traits reflect underlying, stable, pre-trauma personality pathology? Or simply the distressing state of chronic PTSD? Do PDs

place individuals in harm's way, increasing the risk of developing PTSD? Are illness and PD independent, hence truly "comorbid"? All these scenarios doubtless occur.

Compounding this confusion, individuals often lack insight into their personalities. Moreover, longitudinal studies of personality-disordered individuals indicate that diagnostic stability, once considered to define PDs, may fluctuate considerably (17). In the state-of-the-art, naturalistic Collaborative Longitudinal Personality Disorders Study (CLPS), only 44% of individuals retained Axis II diagnoses during the first study year: 56% retained avoidant, 42% obsessive-compulsive, 41% borderline, and 34% schizotypal PD (18).

One approach to disentangling this conundrum is to treat comorbid Axis I disorders to see whether Axis II diagnoses evanesce. In conducting a randomized controlled 14-week trial of three brief psychotherapies (Prolonged Exposure, Relaxation Therapy, and Interpersonal Psychotherapy) for chronic PTSD (19), we assessed patients' PD status before and after treatment. We diagnostically re-assessed a subset of PTSD treatment responders at 26 weeks.

To our knowledge, no prior research has reassessed PDs following acute psychotherapeutic treatment of an Axis I disorder. Literature searches of PubMed, Scopus, and Google Scholar using key words "trauma," "traumatic stress," "treatment," "personality disorder," and "personality stability" as well as Axis I disorders ("depression," "mood disorder," "bulimia," "substance use," etc.) uncovered few Axis I treatment studies even assessing Axis II diagnoses, and none specific to PTSD.

A few trials have examined acute PD changes after treating Axis I disorders. Fava and colleagues (20) described treating 384 outpatients with major depression in an eight week trial of fluoxetine. Sixty-four percent met baseline criteria for at least one PD on the self-report Personality Diagnostic Questionnaire-Revised scale (21). After eight weeks, rates of avoidant, dependent, passive-aggressive, paranoid and narcissistic PDs significantly decreased (20). Lopez-Castroman and colleagues similarly treated 82 patients with major depression for eight weeks with antidepressant pharmacotherapy (22). Of the 66% who had a baseline PD by SCID-II, 20.4% (N=11) had a SCID-II change. Mulder et al. treated 149 patients with major depression for 18 months with fluoxetine, nortriptyline, or their combination (23), then repeated the SCID-II. They found a dropoff in PDs at 18 months, although also some new PD diagnoses. Patients with better antidepressant responses showed a trend for losing more personality disorder symptoms, but lesser responders lost, on average, nearly three personality disorder symptoms. The authors concluded that "Personality disorders are neither particularly stable nor treatment resistant." (23, page 219). Lasting 18 months, this trial cannot be considered brief.

Vergara-Moragues et al. reassessed PDs using the self-report Millon Multiaxial Inventory III (24) among 127 patients with cocaine dependence before and after completing three months in a therapeutic community (25). Somewhat confusingly, they found a decrease in personality dimensions, but a slight increase in PD diagnoses over time.

No research we could locate addressed change in PDs following brief psychotherapy targeting an Axis I disorder. Nor could personality experts we polled, including CLPS

Principal Investigators, identify such studies. Articles we located generally focused on PD effects on Axis I treatment adherence and outcome, not on PD changes. For example, a study comparing dialectical behavioral therapy (DBT; $n=9$) to DBT integrated with prolonged exposure ($n=17$) for patients with comorbid borderline PD and PTSD reported greater improvements among treatment completers in the latter therapy (26). Although clinicians often fear that Axis II comorbidity may worsen treatment outcome, Milrod et al. found that presence of Cluster C PDs at baseline predicted better outcome in psychodynamic psychotherapy for panic disorder ($N=49$, 24 with a PD) (27). Neither study reported PD diagnostic changes.

Patients who meet criteria for one PD often meet criteria for several (9,28). Presence of a comorbid baseline PD diagnosis raises important clinical questions: How likely is this diagnosis to persist after brief Axis I treatment? What prognostic value does the comorbid PD impart? This exploratory study addressed both questions. We hypothesized: 1) PD diagnostic rates would decrease post treatment (week 14) and remain lower for responders at 26 weeks; 2) baseline PD would not predict PTSD treatment outcome. We further explored 3) whether loss of PD diagnosis depended upon *a priori* defined PTSD treatment response, and 4) whether PDs whose symptom criteria overlapped with PTSD symptoms (paranoid, avoidant) were more likely to resolve with effective PTSD treatment than non-overlapping PDs. Beyond categorical personality disorders, we explored effects on the three Axis II PD “clusters” and overall (across SCID-II) PD symptom severity.

We did not hypothesize differences between the three psychotherapies in PD change, lacking statistical power to test these. Nor did we hypothesize that PD diagnoses would worsen PTSD treatment outcome, another area of comorbidity controversy (2).

Methods

Principal study outcomes are published elsewhere (19,29). Briefly, patients recruited by advertisement were carefully screened by research psychiatrists and master’s ($n=1$) and doctoral ($n=3$) level trained independent evaluators using the Clinician-Administered PTSD Scale (CAPS, 30) for PTSD severity, the Structured Clinical Interview for DSM-IV, Patient Version (SCID-P; 31) and SCID-II (32–35) to establish current and lifetime Axis I and II diagnoses. The same evaluators assessed both Axis I and Axis II disorders. The SCID-II, a widely used, semi-structured PD interview, has shown good test/retest reliability (33–35). Patients were 18–65 years old, had primary DSM-IV chronic PTSD and CAPS score ≥ 50 (at least moderate PTSD severity [36]). Exclusion criteria comprised psychotic disorders, bipolar disorder, unstable medical condition, current alcohol or other substance dependence, active suicidal ideation; antisocial, borderline, schizotypal, or schizoid PD; prior non-response to ≥ 8 weeks of study therapy; and current psychotherapy or psychopharmacotherapy.

Patients enrolled in the study were randomly assigned to 10 90-minute sessions of Prolonged Exposure ($N=38$), 14 50-minute sessions of Interpersonal Psychotherapy ($N=40$), or 9 90-minute and one 30 minute session of Relaxation Therapy ($N=32$). All three treatments yielded large pre/post effect sizes: $d=1.32$ – 1.88 (19).

Blinded to treatment condition, independent evaluators readministered SCID-II assessments immediately post treatment (week 14). Treatment responders were invited to enter a 12 week, no-treatment follow-up, after which SCID-II was repeated (week 26). Although 14 and even 26 weeks seem too brief intervals for overall “personality change,” and study treatments did not target PDs, Axis I symptomatic improvement might explain a decrease in apparent PDs.

Initial SCID-II interviews asked patients to describe their characteristic functioning (“about the kind of person you are, that is, how you generally have felt or behaved” [32]). Evaluators ascertained early age onset criteria, limiting the risk of baseline false positive PD diagnoses. SCID-II readministration at weeks 14 and 26 could not meaningfully ask about personality over prior years but used the same probe. Evaluators asked: “Has this [trait] changed in any direction?” – asking patients to consider time since treatment began (at week 14), or since treatment ended (at week 26). Evaluators spent extra time re-rating traits patients reported to have changed, avoiding influencing them or pushing for improvement. The same evaluators generally re-evaluated patients across time points.

Multiple (N=2 or 3) raters assessed 19 videotaped SCID-II interviews to evaluate reliability. Across the 10 individual PDs, percent agreement between raters ranged from 85% to 100%. Kappa coefficient (κ) for agreement ranged from 0.2 to 1. We caution that within the interviews with multiple ratings used for computing κ , many PD diagnoses had very low prevalence, rendering the κ coefficient unstable and unreliable for characterizing rater agreement. Despite these limited formal SCID-II reliability data, the raters met in weekly supervision to discuss reliability over five years; their reliabilities on the CAPS (primary outcome measure; Shrout-Fleiss interclass reliability coefficient = 0.93) and Hamilton Depression Rating Scale (Shrout-Fleiss = 0.89) were excellent (19).

This study conducted secondary analysis of data from the primary study, which was designed to compare outcomes of three psychotherapies for chronic PTSD (19). Of 110 randomized patients with chronic PTSD, 99 (90%) had usable baseline SCID-II data and 78 underwent SCID-II reassessment at week 14. We defined PTSD response *a priori* as 30% decrement from baseline CAPS score, and remission as final CAPS ≤ 20 (37). Patients missing outcome data post treatment were considered non-responders.

Data Analysis

To evaluate generalizability of the inferences, we compared (i) the subject sample with baseline PD assessment against those without baseline PD assessment, and (ii) the subject sample with assessments at both baseline and week 14 against those with only baseline PD assessment. Comparison (i) evaluated patients’ baseline clinical and demographic characteristics; comparison (ii) added baseline PD diagnosis and symptoms at week 14.

We evaluated persistence of any PD by classifying patients with PD assessments at both weeks 0 and 14 with respect to presence/absence of the diagnosis at weeks 0 and 14. We further estimated proportions of patients with each baseline PD diagnosis who lost this diagnosis by week 14, testing whether this proportion exceeded 0 using tests for comparison of proportions.

The prognostic value of a baseline comorbid PD was assessed by modeling PTSD outcome as a function of treatment and baseline PD diagnosis (main effects only) and evaluating significance of the coefficient for baseline PD diagnosis. To address whether losing a PD diagnosis related to PTSD symptom improvement, analyses focused on patients with a given PD diagnosis at baseline; losing the PD diagnosis by week 14 was modeled as a function of PTSD outcome and treatment, with a significant effect of PTSD outcome indicating a relationship. These questions were investigated defining PTSD outcome by (i) response (30% decrease in baseline CAPS at week 14) and (ii) remission (post-treatment CAPS <20).

To evaluate whether PDs with criteria overlapping PTSD symptoms (paranoid, avoidant) associated differently with PTSD outcome than other PDs, we created a category “avoidant and/or paranoid” including patients having 1 of these diagnoses. All PD analyses were conducted on each of the 10 Axis II diagnoses, the three Axis II clusters, on presence/absence of any PD diagnosis, and on the variable “avoidant and/or paranoid,” totaling 10+3+1+1=15 PD measures. Recognizing the small sample size and exploratory nature of these analyses, to avoid omitting potentially important effects we judged statistical significance at $\alpha=0.05$. Raw p -values are reported without correction for multiple testing.

Results

In this highly traumatized, long-suffering sample, a mean 40.3 (s.d.=11.3) years old and 71% female, only 17% of patients were married or cohabitating; only 37% held full-time employment (11% were students). Ninety-four percent reported interpersonal traumas, generally more disturbing than impersonal traumas (38,39). Most (55%) reported chronic traumata (mean duration since primary trauma= 14.7 [14.6] years), including sexual (36%) and physical (63%) abuse. Twenty-four percent reported childhood or adolescent trauma. Three-quarters of patients (73%) reported prior psychotherapy, and 46% pharmacotherapy, for PTSD. No demographic factor was associated with presence of PD diagnosis at baseline.

Of the 99 patients with baseline SCID-II data, 47 (47%) met criteria for 1 of the ten standard PDs at baseline: paranoid (28%), obsessive-compulsive (27%), and avoidant (23%) PD were most prevalent; 15% had narcissistic, 3% dependent, 0% histrionic PDs. Despite these being exclusion criteria, 5 patients with borderline, 4 with schizoid, 4 with antisocial, and 3 with schizotypal PDs inadvertently slipped into enrollment. No differences in distribution of PD diagnoses emerged across the three treatment groups.

Personality disorder stability

After 14 weeks of psychotherapy, 78 of the 99 study patients received PD reassessment. (The remainder declined, or data were incomplete.) Of 35 patients with week 14 Axis II data who had met diagnostic criteria for 1 baseline PD, 15 (43%) no longer met any Axis II diagnosis (Table 1). Proportions of patients who lost baseline PD diagnoses were high and statistically greater than 0 across PD diagnoses (Table 1). Personality disorder diagnostic improvement did not reflect dropping just below threshold: patients no longer meeting PD criteria decreased scores for their lost diagnosis by a mean 3.6 (s.d.=1.7) points, achieving a final mean score of 1.5 (s.d.=1.2) traits.

Prognostic value

No evidence suggested any association between baseline PD presence and PTSD response or remission (all p -values >0.3): PTSD response rate among patients without baseline PD diagnosis was 64%, versus 62% among those with any baseline Axis II diagnosis.

PTSD response status had no effect on losing a specific PD diagnosis or decreasing SCID-II symptom severity total. However, among patients with Axis II reassessment ($n=78$), at week 14 none of 26 PTSD remitters had avoidant and/or paranoid diagnosis, whereas 15 (29%) of 52 non-remitters had avoidant and/or paranoid diagnoses ($p=0.006$). Similarly, focusing only on patients with baseline avoidant and/or paranoid PD, PTSD remitters ($n=6$) all lost their avoidant and/or paranoid diagnosis, versus only 7 (35%) non-remitters ($n=20$) ($p=0.015$). (Overall, 33% of the $N=78$ sample remitted [19].)

The $N=99$ patients with baseline SCID-II assessment did not differ significantly from the 11 not SCID-II assessed, nor did the $N=78$ patients with repeat SCID-II assessments differ meaningfully from the 21 without reassessment, on clinical or demographic variables.

Curiously, three (7%) of 43 patients who had lacked any baseline PD diagnosis developed one at week 14: two developed paranoid PD, one obsessive-compulsive (Table 1). At baseline, one new paranoid PD cases had had three paranoid traits, hence only needed one to reach threshold; the other had met two of four criteria; both were PTSD non-responders. An additional ten subjects who had had other baseline PDs developed new ones: three developed paranoid, three obsessive-compulsive, one apiece borderline and narcissistic, and two PD not otherwise specified. Six of the ten were within two criteria of threshold at baseline. Five of these diagnoses occurred among PTSD non-responders. Patients gaining PD diagnoses had mean increase of 2.3 ($sd=1.5$) points, starting at mean 1.7 ($s.d.=1.5$) and ending with an average score of 4 ($s.d.=1.87$).

At week 26, the 44 treatment responders who entered the follow-up phase showed a similar if further heightened pattern of improvement (Table 2). Of 18 subjects who had 1 baseline PD, 10 (56%) had no PD diagnosis at week 26. Again, a few subjects who had lacked baseline PD diagnoses acquired them at week 26.

Discussion

This is the first study to examine effects of acute psychotherapeutic treatment of an Axis I disorder on Axis II personality disorders. Many PDs disappeared with brief treatment of chronic PTSD. We had not expected so many PD diagnoses to fade, even with successful PTSD treatment. These exploratory findings open new vistas of research on the interaction of personality and other psychiatric disorders.

Findings supported several of our hypotheses: 1) personality disorder diagnostic rates decreased after fourteen weeks of PTSD treatment, and remained lower for responders at 26 weeks. 2) Baseline personality disorder did not predict PTSD treatment outcome. Perhaps due to limited sample size, the range of PDs excluded by study criteria, and the broad level of PD improvement, we did not find that 3) loss of PD diagnosis depended upon PTSD

treatment response. 4) Specific PDs whose symptom criteria overlapped PTSD symptoms (paranoid, avoidant) were more likely to resolve with PTSD remission (though not response); no such relationship appeared for other PDs, clusters, or severity.

The temporal decrease in PD diagnoses echoes early CLPS findings (18). Not only did personality disorders diminish with PTSD treatment, but baseline PDs did not worsen PTSD treatment prognosis. These results suggest – within the study’s restricted range of PDs – that whereas many clinicians may consider PDs worrisome prognostic factors, such fear might be baseless. Acutely treating Axis I may clarify the need to treat PDs. There may be a halo effect of generalized improvement: treating the target disorder may generally lower psychopathology. Resolving a given symptom might affect several disorders due to the polythetic construction of DSM diagnoses. Some apparent PDs may be epiphenomena of chronic PTSD, may fade with acute treatment, and may not carry negative prognostic value. Our debriefing study patients posttreatment echoed these findings in suggesting considerable changes in outlook and functioning after symptomatic PTSD improvement.

Our study could not address all comorbidity issues. Questions about the etiological interaction of PTSD and borderline PD extend back decades to Herman’s clinical observations (40). As borderline PD was a study exclusion criterion (albeit a few, milder cases slipped in), we could not address that question.

One might speculate about differential effects of the three psychotherapies on personality disorders. A seven month adaptation of IPT showed promise for patients with borderline personality disorder in a small open trial (41), and maintenance IPT for recurrently depressed patients was associated with regression of Cluster C diagnoses over a two year course (42). Might IPT, which focuses on interpersonal interactions, be more likely to treat personality disorder traits than somatically-focused Relaxation Therapy? The treatment duration was too short to expect true personality change, and the study sample size too small to test this interesting question.

The emergence of new personality diagnoses following brief treatment surprised us, although researchers have reported this phenomenon (23). Some readers might interpret this finding as rater artifact and criticize the paucity of interrater reliability data. Our study focused on the primary PTSD outcomes; in retrospect, we could have prioritized more extensive testing of SCID-II interrater reliability ratings. Even had we done so, the limited PD range and sample size would have precluded testing interrater reliability as studies with that primary purpose have. First et al., studying SCID-II psychometrics in 284 subjects, still produced kappas ranging from 0.24 to 0.74 (34). We recognize our kappa statistics as a study limitation but feel they inadequately reflect the clinical integrity of our experienced, carefully supervised evaluators.

Alternatively, that new PDs arose might suggest that independent evaluators made truly objective assessments rather than tracking patients’ Axis II course across rating sessions. Most patients with *de novo* PDs had scored within two criteria of PD threshold at baseline, and thus needed only mild fluctuation to attain the diagnosis. This finding, and the regular evaluator review meetings and supervision, offer some reassurance about the results.

Zimmerman, in an excellent review (9), described methodological factors potentially contributory to “the (so-called) diagnostic instability problem” of PDs (9, p. 448). He enumerates as explanations: 1) methodological error, due to limited rater test-retest reliability (43), heightened baseline distress subsiding over time (regression to the mean) (44,45), or repeated assessment effects; 2) therapeutic improvement; 3) spontaneous improvement; and 4) nosological artifact (9). We concur that measurement limitations might account for some diagnostic decline over time – Zimmerman suggests potentially 20–30% on the SCID-II – but probably do not account for the overall magnitude of rapid change. Therapeutic improvement here would take on new meaning: brief Axis I treatment “improving” Axis II disorders. More likely, symptom overlap of chronic PTSD with PDs and the burden of Axis I psychopathology often created the appearance of PDs, which then improved with PTSD treatment.

Several study limitations include a relatively small sample of patients with PD diagnoses, a brief follow-up interval, and limited evaluator interrater reliability testing. Missing data further diminished the available sample. Study exclusions of some PDs, alcohol/substance dependence, and suicidality may have limited the range of PDs and resulted in milder PD presentations more prone to evanescence. The selection of a sample with chronic PTSD may also have influenced PD presentations. These factors limit the generalizability of our results, which we consider preliminary and requiring replication. Other PTSD therapies might conceivably yield different personality disorder findings. Nonetheless, the findings break new ground in suggesting that seeming PD diagnoses may shift with acute psychotherapy of chronic PTSD, and that baseline PD presence need not lead clinicians to anticipate poorer treatment prognosis. Our research team had long regretted not having re-assessed PDs post-treatment in dysthymic disorder trials. Researchers conducting Axis I clinical trials might follow our example by repeating personality disorder assessments post treatment.

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Table 1

Personality Disorder Diagnoses Pre/Post Treatment (Weeks 0, 14) (N=78)[/]

Personality Disorder Diagnosis	Axis II Dx at baseline				% Lost Baseline Axis II Dx
	No		Yes		
	Axis II Dx at week 14		Axis II Dx at week 14		
	No	Yes	No	Yes	
Avoidant	63	0	10	5	67%
Paranoid	54	5	10	9	53%
Obsessive Compulsive	56	3	12	7	63%
Schizoid	75	0	3	0	100%
Antisocial	77	0	1	0	100%
Borderline	74	1	2	1	67%
Narcissistic	68	1	5	4	56%
Dependent	76	0	2	0	100%
NOS	68	3	4	3	57%
Any Axis II	40	3	15	20	43%

Note: Columns do not sum because some patients had multiple Axis II diagnoses. NOS= diagnosis not otherwise specified. No patients met schizotypal or histrionic personality disorder criteria.

[/] All p-values for testing whether the proportion of those who lost a baseline Axis II diagnosis is greater than 0 are <0.001.

Table 2

PTSD Treatment Responders: Personality Disorder Diagnoses at Baseline, Week 26 Follow-up (N=44)¹

Personality Disorder Diagnosis	Dx at baseline				% Lost Baseline Axis II Dx
	No		Yes		
	Dx at W 26		Dx at W 26		
	No	Yes	No	Yes	
Avoidant	37	0	6	1	86%
Paranoid	35	1	4	4	50%
Obsessive Compulsive	32	1	10	1	91%
Schizoid	43	0	1	0	100%
Borderline	42	0	2	0	100%
Narcissistic	39	0	5	0	100%
Histrionic	43	1	0	0	100%
Dependent	42	0	2	0	100%
NOS	39	2	3	0	100%
Any Axis II	25	1	10	8	56%

Note: Columns do not sum because some patients had multiple Axis II diagnoses. NOS= diagnosis not otherwise specified. No patients met schizotypal or histrionic personality disorder criteria.

¹ All p-values for testing whether the proportion of those who lost a baseline Axis II diagnosis is greater than 0 are <0.001.