Journal of Psychiatric Research 45 (2011) 469–474

Contents lists available at ScienceDirect

Journal of Psychiatric Research

journal homepage: www.elsevier.com/locate/psychires

The effects of comorbid personality disorders on cognitive behavioral treatment for panic disorder☆

Michael J. Telch [a,*], Jan H. Kamphuis [b], Norman B. Schmidt [c]

[a] Laboratory for the Study of Anxiety Disorders, Department of Psychology, 1 University Avenue, A8000, University of Texas, Austin, TX 78712, USA
[b] Department of Clinical Psychology, University of Amsterdam, Amsterdam, The Netherlands
[c] Department of Psychology, Florida State University, Tallahassee, FL, USA

ARTICLE INFO

Article history:
Received 6 January 2010
Received in revised form
11 August 2010
Accepted 18 August 2010

Keywords:
Panic disorder
Cognitive-behavioral treatment
Personality disorders
Predictors of treatment outcome

ABSTRACT

The present study investigated the influence of personality pathology assessed both dimensionally and categorically on acute clinical response to group cognitive-behavioral treatment in a large sample of panic disorder patients ($N = 173$) meeting DSMIII-R criteria for panic disorder with or without agoraphobia. Nearly one-third of the sample met for one or more personality disorders, with the majority meeting for a Cluster C diagnosis. Patients with one or more comorbid personality disorders displayed higher baseline and higher post treatment scores across multiple indices of panic disorder severity compared to those without personality disorders. After controlling for panic disorder severity at baseline, the presence of both Cluster C and Cluster A Pers-Ds predicted a poorer outcome, whereas when assessed dimensionally, only Cluster C symptoms predicted a poorer treatment response. However, the influence of personality pathology was modest relative to that of baseline panic disorder severity.

1. Introduction

Panic disorder is frequently complicated by the presence of both psychiatric comorbidity (Brown et al., 1995) and non-psychiatric medical comorbidity (Schmidt et al., 1996). In terms of psychiatric comorbidity, as many as 70% of patients with panic disorder present with co-occurring psychiatric diagnoses (Reich and Troughton, 1988). The high rate of comorbidity in panic disorder has naturally led to evaluation of the effects of co-occurring conditions on treatment outcome.

Cognitive-behavioral treatment (CBT) has established efficacy in the treatment of panic disorder (Barlow et al., 2000; Clark et al., 1994; Gould et al., 1995; Hofmann, 2008). However, following CBT, many patients continue to display residual symptoms requiring some to seek out additional treatment (Brown and Barlow, 1995). Consequently, identifying factors that predict a poor response to CBT is an important research goal for optimizing the clinical management of panic disorder (Wolfe and Maser, 1994).

Personality disorder comorbidity is frequently cited as a factor implicated in poor treatment response to both pharmacotherapy (Slaap and den Boer, 2001) and psychosocial treatments (Milrod et al., 2007; Reich and Green, 1991; Reich and Vasile, 1993). Although not studied systematically, personality dysfunction may negatively affect treatment outcome through its potential influence on other moderators of treatment outcome such as patient drop-out (Grilo, Money, Barlow, Goddard, Gorman, Hofmann et al, 1998), compliance with treatment regimens (Schmidt and Woolaway-Bickel, 2000), the therapeutic alliance, or motivation for treatment (Persons et al., 1988).

The presence of a comorbid personality disorder as measured by either structured interview (i.e., SCID II) or questionnaire has been shown to predict treatment non-response (Marchesi et al., 2006; Noyes et al., 1990; Reich, 1988) or relapse upon medication discontinuation (Green and Curtis, 1988). Despite the claim that patients displaying comorbid Axis II pathology respond less favorably to cognitive-behavioral treatment (Mennin and Heimberg, 2000), evidence from controlled prospective studies is inconclusive. This is due to the small number of prospective studies and the significant methodological limitations of the existing studies i.e., small sample size, use of questionnaires to assess personality dysfunction, and failure to control for baseline severity of Axis I pathology (Dreessen and Arntz, 1998; Shear et al., 1994).

Of the prospective studies investigating the linkage between Pers-D comorbidity and treatment response in panic disorder, three published studies have investigated the effects of Pers-D pathology (as assessed by structured clinical interview) on panic patients'

☆ This research was funded by National Institute of Mental Health Grant MH74-600-203.

* Corresponding author. Tel.: +512 560 4100; fax: +512 471 6572.
E-mail address: telch@austin.utexas.edu (M.J. Telch).

0022-3956/$ – see front matter © 2011 Published by Elsevier Ltd.
doi:10.1016/j.jpsychires.2010.08.008

response to cognitive-behavioral therapy (Black et al., 1994; Dreessen et al., 1994; Kampman et al., 2008). Several additional studies have examined response to naturalistic treatment e.g. (Mellman et al., 1992; Noyes et al., 1990), which may have involved some cognitive-behavioral treatment.

In a sample of 31 panic patients undergoing individual cognitive-behavioral treatment, Dreessen et al. (1994) found some evidence to suggest that patients with a comorbid personality disorder, based on the SCID II, were more likely to show greater psychopathology at baseline. Neither the presence of a personality disorder nor the number of SCID II personality traits predicted treatment response. However, the negative findings may have been due to low statistical power given the small sample size.

Black and colleagues (Black et al., 1994) examined the influence of personality pathology on the short-term (i.e., 3 weeks) treatment response in 66 panic disorder patients receiving cognitive therapy, fluvoxamine, or placebo. The presence of a comorbid personality disorder based on the SIDP structured interview did not predict recovery as defined by the absence of panic attacks and a Clinical Global Improvement score of 'very much' or 'much' improved. However, higher scores on a self-report personality disorder questionnaire were associated with a less favorable outcome (Black et al., 1994). Unfortunately, due to the small sample size, separate analyses were not reported for the patients receiving cognitive therapy. Kampman and colleagues examined whether cluster C personality disorders predicted treatment response in a sample of 161 panic disorder patients treated with 15 sessions of CBT. Although initial panic severity predicted posttreatment severity, the presence of one or more cluster C personality disorders did not affect treatment outcome (Kampman et al., 2008). The principal limitation of this study is its exclusive focus on cluster C personality disorders.

Based on the meager evidence to date, no firm conclusions can be drawn concerning the effects of Pers-D comorbidity on panic disorder patients' clinical response to cognitive-behavioral treatment. The primary aim of this study was to examine several indicators of Pers-D comorbidity and their relationship to treatment outcome among a large sample of panic disorder patients receiving cognitive-behavioral treatment. Several design features were included to address the limitations of previous studies. These included: (a) inclusion of a larger sample, (b) followed recommended guidelines for the assessment of panic disorder (Shear and Maser, 1994) (c) followed the evidence-based recommendations for the assessment of personality disorders (Widiger and Samuel, 2005); (d) utilized both categorical and dimensional analyses of Pers-Ds; and (e) controlled for pretreatment levels of panic disorder severity.

2. Methods

2.1. Subjects

The sample consisted of 173 panic disorder patients (128 women and 45 men) who had completed an eight-week group-administered cognitive-behavioral treatment as part of their participation in several different clinical trials. Participants were recruited through local media channels and letters to physicians and mental health workers in the Austin, TX area. Further details of the subject recruitment and screening are provided elsewhere (Telch et al., 1993b; Telch et al., 1995a). All participants met the following entry criteria: (a) principal Axis I diagnosis of panic disorder with or without agoraphobia; (b) at least one panic attack during the past 30 days; (c) age 18–65; (d) no recent change in psychotropic medications; and (e) negative for current psychosis, bipolar disorder and substance abuse disorder. Panic disorder diagnoses were derived from the Structured Clinical Interview for DSMIII-R. Mean age of the sample was 35.2 years and mean duration of illness was 9.1 years. The ethnic breakdown of the sample was as follows: Caucasian (81%), African-American (8%), Hispanic (6%), Asian (1%), and other 4%. Over half of the subjects were married (56%), 27% were never married, and 18% were divorced or separated.

2.2. Measures

2.2.1. Assessment of panic disorder

Three primary outcome measures tapping each of the three major symptom facets consisting of panic attacks, anticipatory anxiety, and panic-related avoidance (agoraphobia) were selected for this study. They included: (a) Sheehan Patient-Rated Anxiety scale (Kick et al., 1994); (b) Agoraphobia scale of the Marks and Mathews Fear Questionnaire (Marks and Mathews, 1979); and (c) panic attack frequency as assessed through a prospective self-monitoring approach modified from that used in the Upjohn Cross National Study (Ballenger et al., 1988). Selection of patient rating scales was based on several considerations including their psychometric qualities, availability of normative information, and recommendations of a 1994 consensus panel on the assessment of panic disorder (Shear and Maser, 1994).

To assess clinical response, a continuous composite clinical response index was computed based on standardized scores from the SPRAS, FQ-Ago, and panic-diary measures. In addition to this continuous composite index, we constructed a dichotomous classification of clinically significant change (yes or no). Specifically, patients were classified as achieving clinically meaningful if their scores on all three of the primary outcome measures moved into the normal range i.e., SPRAS < 30, FQ-Ago < 12, and when no panic attacks were reported in the previous week.

2.2.2. Assessment of personality disorders

The SCID II was administered to assess for the presence of personality disorders. The SCID II is a widely used semi-structured diagnostic interview with good inter-rater reliability (Zimmerman, 1994) Advanced clinical psychology graduate students who had received specialized training in SCID administration conducted the interviews. Adequate levels of inter-rater reliability were found for a randomly selected subset of the present sample. These data are reported elsewhere (Telch et al., 1995b).

2.3. Treatment

All patients received treatment previously described by Telch et al. (1993a,1995a). This multi-component group CBT treatment consists of four major treatment components: (a) education and corrective information concerning the nature, causes, and maintenance of anxiety and panic, (b) cognitive therapy techniques aimed at helping the patient identify, examine, and challenge faulty beliefs of danger and harm associated with panic, anxiety, and phobic avoidance; (c) training in methods of slow diaphragmatic breathing to help patients eliminate hyperventilation symptoms and reduce physiological arousal; (d) interoceptive exposure exercises designed to reduce patients' fear of somatic sensations through repeated exposure to various activities (e.g., running in place) that intentionally induce feared bodily sensations (e.g., heart racing), and (e) self-directed exposure to patients' feared situations designed to reduce agoraphobic avoidance. Treatment sessions were led by an experienced doctoral level clinician (MJT or NBS) and co-led by one of several advanced doctoral student clinicians. Treatment consisted of 12 two-hours highly structured sessions conducted over an eight-week period. Sessions were conducted twice weekly for the first four weeks and then once each week for

the remaining four weeks. Patients were required to tape record each session and were encouraged to listen to the tape between sessions. Skill-based home practice was assigned each session and patients' completed home practice monitoring forms to track their adherence to home skill building exercises.

2.4. Statistical analyses

Our analyses were guided by the a priori working hypothesis that both *level* of personality dysfunction (i.e., severity) and *type* of personality disorder may negatively influence treatment outcome. Descriptive analyses were used to describe the sample. Next, ANOVAs were conducted to compare patients with- and without personality disorders on the three principal panic outcome facets, at both baseline and post treatment. Comparison groups included patients who met criteria for (a) one or more Pers-Ds, (b) any Cluster A Pers-D; (c) any Cluster B (Pers-D); (d) any Cluster C Pers-D; and (e) two or more Pers-Ds. To examine the relation between PD cluster membership and overall treatment outcome, multiple regression analyses were conducted. Independent variables included a composite score for baseline panic disorder severity and alternatively (a) presence or absence of a comorbid personality disorder or (b) presence or absence of diagnoses in each of the three PD clusters. A standardized composite of panic disorder severity was used in all analyses. It was computed as follows. First, the panic-frequency data were log-transformed to generate a less skewed distribution. Next, Z-scores were calculated for each of the three primary outcome measures (i.e., SPRAS, FQ-Ago, and panic-frequency). These Z-scores were then summed to derive the standardized composite outcome score.

3. Results

3.1. Prevalence and distribution of personality disorders

The distribution of personality disorders broken down by cluster is presented in Table 1. Approximately one third of the sample (31.2%) met full diagnostic criteria for one or more Per-Ds at intake. Most prevalent were Cluster C diagnoses (24.3%), followed by Cluster B (8.7%), and Cluster A (6.9%). Twenty panic disorder patients (11.6%) met criteria for more than one personality disorder.

3.2. Association between comorbid personality pathology and panic disorder at pretreatment

Data on panic disorder symptoms at pretreatment as a function of the presence or absence of personality disorders are presented in Table 2. Patients presenting with one or more personality disorders scored significantly higher on the composite measure of panic disorder severity relative to those without a personality disorder, $F(1, 171) = 14.75$, $p < .001$, $\eta^2 = .079$. The correlation between the panic disorder severity composite score at pretreatment and the total Pers-D score from the SCID II was .36 ($p < .001$).

Table 1
Breakdown of personality disorder status.

Personality Disorder status	N	%
No personality disorder	119	68.8
Any personality disorder	54	31.2
Two or more personality disorders	20	11.6
Any Cluster A	12	6.9
Paranoid	9	5.2
Schizoid	0	0.0
Schizotypal	3	1.7
Any Cluster B	15	8.7
Antisocial	2	1.2
Borderline	10	5.8
Histrionic	3	1.7
Narcissistic	3	1.7
Any Cluster C	42	24.3
Avoidant	37	21.4
Dependent	12	6.9
Obsessive Compulsive	10	5.8

3.3. Influence of personality disorders on treatment outcome after controlling for panic disorder severity at intake

Because patients displaying Pers-Ds displayed significantly higher panic disorder severity at baseline, we examined whether Pers-Ds influence patients' response to CBT after controlling for panic disorder severity at pretreatment.

3.3.1. Categorical analyses of personality disorders

Separate multiple regression analyses were constructed to assess the influence of each of the categorical Pers-D indices after controlling for baseline severity of panic disorder. For each analysis, panic disorder severity at baseline was entered in Step 1 and the Pers-D variable of interest was entered in Step 2. Results of these analyses are presented in Table 3 and revealed that the presence of any Pers-D [$F(1,170) = 4.34$, $B = .14$, $p < .05$, R^2 change $= .018$], Cluster A Pers-D [$F(1,170) = 7.94$, $B = .182$, $p < .01$, R^2 change $= .032$], and Cluster C [$F(1,170) = 5.28$, $B = .152$, $p < .03$, R^2 change $= .022$] each predicted a less favorable response to CBT at the posttreatment assessment. Next, the relative contribution of these categorical Pers-D indices were evaluated in a multivariate model controlling for panic disorder severity at baseline. Results of this analysis indicated that the presence of a Cluster A diagnosis uniquely predicted posttreatment outcome after controlling for the other predictors in the model [$t = 2.35$, $B = .168$, $p < .03$. None of the other predictors in the model were significant.

3.3.2. Dimensional analyses of Pers-Ds predicting treatment outcome

A series of four regression analyses were performed using the SCID II total score and each of the three Pers-D cluster scores. The approach for these analyses were identical to those reported above for the categorical Pers-D indices. Of these analyses, only Cluster C scores predicted treatment outcome after controlling for panic disorder severity at baseline [$F(1,170) = 6.19$, $B = .170$, $p < .02$, R^2 change $= .025$].

3.3.3. Influence of comorbid personality pathology on clinically significant change

Finally, we examined the influence of personality disorder comorbidity on patients' likelihood of attaining clinically significant change as determined by the criteria outlined by (Jacobson and Truax, 1991). Data describing the percentage of patients achieving clinically significant change by their pretreatment personality disorder status are presented in Fig. 1. Logistic regression models (both unadjusted and adjusted for pretreatment panic disorder severity) were constructed to evaluate the relationship between the four Pers-D variables and the more conservative categorical outcome of achieving clinically significant change.

In the unadjusted models, a significantly lower odds of achieving clinically significant improvement was observed for patients presenting with one or more Pers-Ds [Any Pers-D: RO = .347, 95% CI = .179 to .674], Cluster A Pers-D [RO = .060, 95% CI = .008 to.476], and Cluster C Pers-D [RO = .368, 95% CI = .180 to.753] were each associated with lower odds of achieving clinically significant change relative to those without Pers-Ds. After adjusting for pretreatment panic disorder severity, only the presence of a Cluster A Pers-D predicted a significantly lower odds of achieving

Table 2
Means and standard deviations of anxiety, agoraphobic avoidance, and panic frequency at baseline and posttreatment among patients with and without comorbid personality disorders.

Outcome measure	Two or more					
	No Pers-D (N = 119)	Any Pers-D (N = 54)	Pers-Ds (N = 20)	Cluster A (N = 12)	Cluster B (N = 15)	Cluster C (N = 42)
Pretreatment Anxiety						
M	54.60	73.13**	77.50***	87.00***	73.47**	74.24***
SD	25.63	27.00	22.11	20.05	26.70	26.11
Agoraphobic avoidance						
M	12.43	18.04**	17.35*	11.00	19.33*	18.50**
SD	9.25	11.64	10.97	8.28	12.61	11.54
Panic Attacks						
M	2.86	3.39	5.50*	7.83**	2.33	3.64
SD	5.23	5.36	7.94	9.50	2.19	5.95
Posttreatment Anxiety						
M	18.07	27.41**	27.65**	41.25***	24.27	26.68***
SD	17.67	19.13	19.96	21.25	17.74	18.87
Agoraphobic avoidance						
M	4.55	6.81*	7.55**	4.83	7.27	7.43**
SD	5.14	6.23	6.76	4.32	8.21	5.94
Panic Attacks						
M	0.31	0.87**	1.55**	2.17***	0.67	1.02**
SD	0.67	1.90	2.91	3.27	1.63	2.11

NOTE: Pers-D = Personality Disorder; Anxiety was assessed with the Sheehan Patient-Rated Anxiety Scale (SPRAS); Agoraphobia was assessed with the agoraphobia subscale of the Marks & Mathews Fear Questionnaire (Fq-Ago); Panic Attacks during the past week were assessed using a prospective panic diary method. *p < .05; **p < .01; ***p < .001, for each patient category when compared to the No PD patient group.

significant improvement [RO = .073, 95% CI = .008 to .653], relative to those without a personality disorder. The proportion of variance in treatment outcome explained by each of the personality disorder status indices after controlling for pretreatment panic disorder severity is presented in Fig. 2.

4. Discussion

The influence of comorbid personality pathology on panic disorder patients' clinical response to cognitive-behavior therapy was examined. Consistent with previous treatment studies (Reich and Troughton, 1988) and prospective naturalistic follow-up studies of panic disorder patients (Massion et al., 2002), approximately one-third of panic disorder patients presented with a comorbid personality disorder. Of these, most patients displayed personality disorders in Cluster C with the modal diagnosis being avoidant personality disorder. The similarity in the distribution of personality disorders observed in our sample relative to those reported in other treatment studies as the panic disorder sample followed in the Harvard-Brown naturalistic follow-up study (Massion et al., 2002) provides some evidence that the level of personality disorder comorbidity in our sample is representative of panic disorder patients seeking treatment.

A significant relationship was observed between the presence of personality disorders and baseline severity of panic disorder. The nature of this relationship was quite consistent across panic disorder indices – namely, patients displaying a comorbid personality disorder scored significantly higher at baseline on most indices of panic disorder severity including panic frequency, panic-related apprehension, and panic-related avoidance. This finding is consistent with those reported by Dreessen & Arntz (Dreessen et al., 1994) and is consistent with findings from the TDCRP suggesting a positive relationship between personality disorder comorbidity and baseline severity of major depression (Ablon and Jones, 1999).

What do our findings reveal about the influence of comorbid personality pathology and clinical response to CBT? The answer depends in large part on whether one controls for pretreatment levels of panic disorder severity, which accounted for 27% of the

Table 3
Results of Linear Regression Analyses Examining Categorical and Dimensional Personality Disorder Variables as Predictors of Post-Treatment Outcome After Controlling for Baseline Panic Disorder Severity (N = 173).

Variable	*B*	*SE*	*b*
Dimensional Analyses			
Baseline Panic Disorder Severity Index	.47	.07	.43***
Total Pers-D Sxs	.02	.01	.09
Cluster A Sx Score	.06	.12	.03
Cluster B Sx Score	−.03	.12	−.02
Cluster C Sx Score	.29	.12	.17*
Categorical Analyses			
Baseline Panic Disorder Severity Index			
Any Pers-D (Yes vs. No)	.53	.25	.14*
Cluster A (Yes vs. No)	1.26	.45	.18**
Cluster B (Yes vs. No)	.05	.41	.01
Cluster C (Yes vs. No)	.63	.27	.15*
Multiple Pers-D's (Yes vs. No)	.63	.36	.11

Note: *p < .05; ** p < .001. Sxs = Symptoms; Pers-D = Personality Disorder.

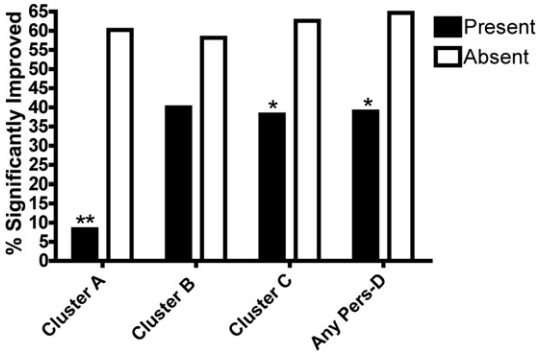

Fig. 1. Percent of patients displaying clinically significant improvement as a function of personality disorder status without controlling for baseline panic disorder severity.

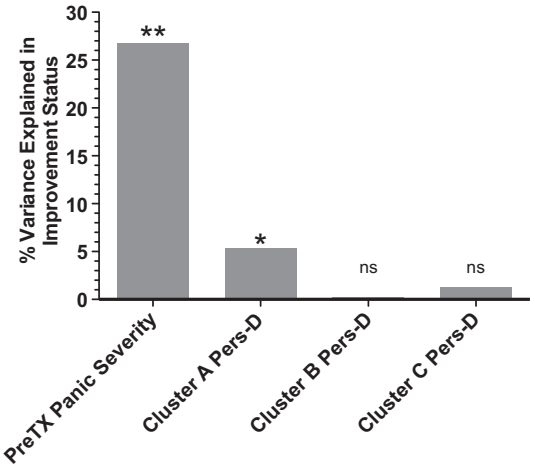

Fig. 2. Proportion of variance in clinically significant change explained by pretreatment panic disorder severity, and the presence of Cluster A, B, and C personality disorders.

explained variance in clinically significant change at posttreatment. Without controlling for pretreatment panic severity, patients presenting with one or more personality disorders showed greater posttreatment symptoms on the continuous panic outcome measures and were significantly less likely (39% vs. 65%) to achieve clinically meaningful change at posttreatment. From these findings, one might conclude as have some (see Mennin and Heimberg, 2000) that the presence of comorbid personality disturbance significantly diminishes panic patients' therapeutic response to CBT. However, after controlling for pretreatment panic disorder severity, our findings suggest that the presence of personality disturbance – whether assessed via dimensional or categorical indices - confers a very modest - but statistically significant deleterious effect on treatment outcome.

Our findings also suggest that conclusions regarding the influence of personality pathology on treatment outcome in panic disorder depends on whether personality pathology is assessed dimensionally or categorically. The most notable example of this was observed with respect to the influence of Cluster A personality pathology. When assessed categorically, the presence of a Cluster A diagnosis was the most influential Pers-D predictor of treatment outcome, whereas when assessed dimensionally, Cluster A symptoms were not associated with treatment outcome. However, it should be noted that only 7% of our sample met for a Cluster A personality disorder diagnosis. Of those, most (9 of 12) were diagnosed with paranoid personality disorder. These findings provide tentative support that panic patients presenting with paranoid personality features respond more poorly to cognitive-behavioral group treatment. We can only speculate as to what mediates this effect. One possibility is that patients presenting with paranoid features have difficulty with a group treatment format. Alternatively, it is possible that change in maladaptive beliefs – one of the targets of CBT – is more difficult to achieve among patients with highly entrenched paranoid beliefs.

Implications for clinical practice deserve mention. Although the data support the widely held view that patients presenting with personality comorbidity are more difficult to treat, they also suggest that it is the severity of panic disorder symptoms, not the presence of personality dysfunction that most significantly accounts for the poorer outcome. Consequently, our findings offer little support for the view that panic disorder patients presenting with personality disorder pathology are not good candidates for CBT, since the symptom change data suggest that patients with personality disturbance start out with more severe illness but improve markedly and almost as much as those without personality dysfunction. Further research is needed to determine whether these patients could attain levels of posttreatment functioning equal to panic patients w/o personality dysfunction, by increasing either the duration and/or intensity of CBT sessions.

Limitations of the study should be considered in drawing inferences about personality disorder comorbidity and panic disorder. First, it should be noted that the present findings are restricted to predicting patients' acute response to CBT. One cannot rule out the possibility that personality dysfunction may prove more influential in predicting relapse or other indices of long-term functioning. Second, patients' personality pathology was not reassessed following cognitive-behavioral treatment. Although the CBT group treatment did not target personality dysfunction directly, it is possible that improvement in panic disorder symptoms may have led to reductions on measures of personality dysfunction. Findings from the Multi-Center Panic Disorder Study revealed that both imipramine and CBT led to significant reductions in personality disturbance as measured by questionnaire (Hofmann et al., 1998). Finally, it should be noted that our findings are limited to patients receiving CBT. It is possible that personality dysfunction will be more influential in predicting clinical response to alternative psychotherapeutic modalities such as psychodynamic psychotherapy (Milrod et al., 2007).

Contributors

Dr. Telch was the major contributor to the design of the original trials in which these analyses are based. He also took the lead in writing the bulk of the manuscript, and contributed to the planning of the data analysis.

Dr. Kamphuis contributed to the design and execution of the analyses used in this manuscript, and was a major contributor in the writing of the results section of the manuscript.

Dr. Schmidt contributed to the coordination of the original trials for which these analyses are based. He also contributed to the selection of the personality disorder indices and worked with Dr. Kamphuis in conducting the data analyses.

Role of Funding Source

Funding for this study was provided by National Institute of Mental Health Grant MH74-600-203; the NIMH had no further role in study design; in the collection, analysis, and interpretation of data; in the writing of the report; and in the decision to submit the paper for publication.

Conflict of interest

There are no conflicts of interests for any of the authors of this manuscript.

Acknowledgements

We thank Drs Patrick Harrington and LaNae Jaimez who provided valuable assistance in the clinical assessment and group CBT sessions.

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