Physical, Mental, and Social Catastrophic Cognitions as Prognostic Factors in Cognitive–Behavioral and Pharmacological Treatments for Panic Disorder

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The authors explored the prognostic value of 3 different types of catastrophic cognitions in the treatment of panic disorder with and without mild-to-moderate agoraphobia using a sample of 143 participants who received either cognitive–behavioral therapy (CBT) or imipramine in a randomized controlled trial. Stronger fears of social catastrophes both prior to and following treatment with CBT or imipramine were associated with a poorer outcome. In contrast, cognitions involving physical or mental catastrophes were unrelated to outcome, regardless of whether these thoughts were measured prior to or following treatment. These findings are consistent with the notion that although the intensity of physical catastrophe cognitions may best discriminate between panic disorder and other anxiety disorders, it is the intensity of social catastrophe cognitions that is most closely tied to success in treating this disorder.

Keywords: cognitions, catastrophic cognitions, panic disorder, panic disorder treatment, cognitive–behavioral therapy, imipramine, prognostic factors
with other anxiety disorders, whereas fears of social and behav-
ioral catastrophes do not (e.g., Bouvard et al., 1998; Chambless &
Gracely, 1989). Although their value in discriminating between
different diagnoses may be clear, little is known about the use of
these different types of catastrophic thoughts in predicting panic
disorder treatment outcome. One study, however, suggests that
fears of social–behavioral catastrophes may be the most important
type of catastrophic cognition in predicting the course of panic
disorder in the absence of treatment. Arrindell (1993) reported that
thoughts involving social and behavioral catastrophes predicted
agoraphobic avoidance measured 3 months later, whereas the
intensity of physical catastrophe cognitions did not emerge as a
significant predictor.

Unfortunately, to our knowledge only two studies have at-
ttempted to directly examine the role of catastrophic cognitions in
predicting panic disorder treatment outcome, and they have pro-
duced inconsistent findings. Chambless and Gracely (1988) failed
to find a significant association between pretreatment levels of
either physical or social–behavioral cognitions and behavioral
treatment outcome (though they noted trends indicating that more
severe physical catastrophe cognitions were associated with poorer
posttreatment outcome). In contrast, Keijsers, Hoogduin, and
Schapp (1994) reported that participants who endorsed stronger
catastrophic cognitions in general prior to exposure-based treat-
ment tended to report poorer outcomes. Although Keijsers et al.
used a measure that included items assessing physical, mental, and
social catastrophe cognitions, they did not distinguish between
these different types of catastrophic thoughts in their analysis of
the data.

Thus, there is a substantial gap in the literature regarding the
relative importance of different types of catastrophic cognitions
measured prior to the start of treatment in predicting outcome for
individuals with panic disorder undergoing treatment. Although
catastrophic cognitions are often seen as central to panic disorder
and are expected to change during treatment, it is far from certain
that the strength of these cognitions at pretreatment predicts out-
come in either of the two most prominent empirically supported
treatments for panic disorder: cognitive–behavioral therapy (CBT)
and pharmacotherapy. The major aim of this study is to determine
whether pretreatment physical, mental, and social catastrophe cog-
nitions predict short-term and long-term outcome in both of these
treatments as might be expected on the basis of several existing
theoretical conceptualizations of panic disorder (e.g., Barlow,
2002; Beck et al., 1985; Clark, 1986; Goldstein & Chambless,
1978; Salkovskis, 1988; van den Hout & Griez, 1982). However,
if these cognitions are secondary to other variables determining
success in CBT and pharmacotherapy, then catastrophic cognitions
might not be expected to have any relation to panic treatment
outcome. It is also possible that pretreatment levels of catastrophic
cognitions may be more closely associated with treatment outcome
in CBT that includes a cognitive restructuring component, as
compared with pharmacotherapy, which does not. Finally, in terms
of clinical implications, if certain types of catastrophic cognitions
emerge as more significant prognostic indicators than others, then
it could suggest that treatment protocols should be altered so as to
direct more attention to addressing particular types of catastrophic
fears.

Beyond these questions related to pretreatment levels of cata-
strophic cognitions, it remains unclear to what extent posttreatment
levels of different types of catastrophic cognitions are related to
panic treatment outcome measured at the end of treatment and at
a long-term follow-up. Clark et al. (1994) partially addressed this
issue in a study comparing cognitive therapy, applied relaxation,
and imipramine. Clark et al. (1999) also addressed this issue in a
second study comparing cognitive therapy and brief cognitive
therapy with a wait-list control condition. In both studies, the
authors found a significant association between posttreatment
scores on a schema questionnaire designed to measure the misin-
terpretation of body sensations and long-term outcome, collapsing
across all treatment conditions. However, they did not find an
association between long-term outcome and posttreatment scores
on a questionnaire designed to measure specific catastrophic cog-
nitions experienced by participants when feeling anxious. These
two studies, though, did not distinguish between different types of
specific catastrophic cognitions, so the question remains open as to
whether particular types of catastrophic cognitions (physical, men-
tal, or social) might be more predictive of long-term treatment
outcome.

It could be hypothesized that physical and mental catastrophe
cognitions would have the closest connection to treatment outcome
because they represent fears of such extreme personal disasters
(e.g., death or going crazy). However, perhaps fears of these types
of catastrophes are more easily dispelled as compared with social
catastrophe cognitions that may seem more trivial in nature (e.g.,
making a fool of oneself). It also remains to be determined whether
there is any interaction between type of catastrophic cognition and
type of treatment received in predicting long-term outcome. In the
current study, we seek to address these remaining questions.

Method

Design

The Multicenter Comparative Treatment Study of Panic Disorder
(MCCTSPD) was a randomized clinical trial conducted by Barlow, Gorman,
Shear, and Woods (2000) to compare the effectiveness of CBT,
imipramine plus medical management, and their combination, in the treat-
ment of participants diagnosed with panic disorder with and without
mild-to-moderate agoraphobia. Four nationally known anxiety disorder
treatment sites participated in the study. Randomization to treatment con-
dition was stratified by treatment site as well as the presence of current
major depression as defined by DSM-III-R (American Psychiatric Asso-
ciation, 1987) criteria.

Participants initially entered a pretreatment assessment phase that in-
cluded a 2-week drug washout period. They then entered an acute treatment
phase that consisted of 11 sessions within a 12-week period for all condi-
tions. CBT sessions lasted approximately 50 min, whereas medical man-
agement sessions were approximately 30 min in duration. Participants were
reassessed after this initial 3-month acute treatment period to determine
treatment responder status. Classification as a responder was defined as a
40% reduction from baseline levels of panic disorder symptom severity. If
they completed acute treatment, then CBT participants automatically en-
tered a maintenance treatment phase that consisted of six CBT monthly
sessions. In contrast, imipramine participants needed to complete acute
treatment and be rated as a treatment responder to enter a maintenance
phase that consisted of six medical management monthly sessions. Partici-
pants in the pharmacotherapy condition continued to receive imipramine
during this 6-month maintenance phase. At the end of the maintenance
phase, participants were again reassessed and treatment was discontinued.
For participants in the imipramine condition, discontinuation of treatment
was accomplished by tapering during a 1- to 2-week period, according to
an established protocol. Follow-up data were collected from all available participants 6 months after treatment discontinuation (15 months after treatment was initiated). It should be noted, however, that the majority of participants who took part in the follow-up assessment were individuals who had completed the maintenance treatment phase. A more detailed description of this study’s design, therapists, and treatment conditions has been provided elsewhere (Barlow et al., 2000).

Participants

A total of 497 participants passed an initial screening that confirmed a principal diagnosis of panic disorder with or without mild-to-moderate agoraphobia. These participants entered a pretreatment phase that included a drug washout for participants taking antianxiety or antidepressant medication. Participants in all treatment conditions were, however, permitted up to 10 doses of benzodiazepine medication in the 2 weeks prior to treatment and up to 20 doses during baseline and acute treatment combined. Two weeks prior to the first treatment session, diagnosis was reconfirmed with the Anxiety Disorders Interview Schedule—Revised (DiNardo & Barlow, 1988). Mild-to-moderate agoraphobia was operationally defined as a score of 18 or lower on the Anxiety Disorders Interview Schedule—Revised avoidance scale. Participants who scored higher than 18 (severe agoraphobia) were excluded from the study. Inclusion in the study also required at least one full or limited panic attack in the 2 weeks prior to the first treatment session. Participants with comorbid unipolar depression were included in the study (unless actively suicidal). Aside from severe agoraphobia, other exclusion criteria included (a) the presence of a psychotic disorder, (b) the presence of bipolar disorder, (c) having a significant medical illness or a substance abuse problem, (d) having prior nonresponse to similar treatments, (e) participation in a concurrent competing treatment, and (f) having a pending disability claim.

A total of 312 participants were randomized to five different treatment conditions (CBT alone, imipramine alone, placebo alone, CBT plus imipramine, and CBT plus placebo). In the current study, we only used data from participants assigned to the CBT (n = 71) or imipramine plus medical management (n = 72) conditions, which created a total sample size of 143 participants for the current analyses. Although the original MCCCTSPD sought to examine potential benefits in combining CBT and pharmacotherapy for panic, we were focused in the current analyses on determining the association between catastrophic cognitions and outcome among participants receiving one of these well-established psychosocial or pharmacological treatments. Furthermore, by examining data from participants in these pure treatment conditions, we aimed to identify potential interaction effects between particular types of cognitions and specific treatments methods. Given these goals for the current study, the combined treatment groups were eliminated from the current analyses for the sake of clarity. Excluding these combined groups clearly had no impact on the results of the analyses based on the two pure treatment groups. The placebo group might also have been included, but because of the uneven treatment cell allocation of participants used to maximize trial efficiency in the original study, as well as missing data on cognitive measures used in the current analyses, the n for this group was too small. The final sample comprised 55 men and 88 women, with a mean age of 36.7 years (SD = 10.3). Of these 143 participants, 135 provided information on their pretreatment catastrophic cognitions. Of these 135 participants, 96 completed acute treatment, whereas 33 of these acute treatment completers provided data at the follow-up assessment. These sample sizes differ from those listed in earlier descriptions of the MCCCTSPD sample (Barlow et al., 2000) because not all study participants provided data on their catastrophic cognitions.

Treatment Conditions

CBT used in the current study was based on the “panic control treatment” developed by Barlow, Craske, Cerny, and Klosko (1989). This treatment combines interoceptive exposure, cognitive restructuring, and breathing retraining and was described in a manual that included detailed therapist instructions for each session (Barlow & Craske, 1994). The imipramine treatment was administered in a double-blind, fixed flexible-dose design, according to a manual developed for the study. Patients in this treatment condition attended 30-min medical management appointments that were used to monitor adverse side effects, assess mental and physical condition, and maximize treatment compliance. Initial doses of imipramine were 10 mg per day. The dosage was increased every other day by 10 mg until 50 mg per day was reached. The dosage was then increased more rapidly, with the goal of reaching 100 mg per day by the end of Week 3 and 200 mg per day by Week 5. These dosage goals were pursued even if the patient became symptom-free earlier, unless adverse side effects became intolerable. If the patient was not symptom free at 200 mg per day, then the dosage could be increased up to 300 mg per day by Week 5. Blood levels of imipramine were assessed at Weeks 6 and 12 of acute treatment.

Measures

Panic Disorder Severity Scale (PDSS). The average item score on the PDSS (Shear et al., 1997) was used as the primary continuous outcome measure. The PDSS is a seven-item, clinician-rated scale of panic disorder severity modeled after the Yale–Brown Obsessive Compulsive Scale (Goodman et al., 1989). The PDSS was administered with a scripted interview in which the clinician rated the severity of seven features of panic disorder on a scale ranging from 0 (none) to 4 (extreme). The features that were rated included panic frequency, panic distress, anticipatory anxiety, agoraphobic fear–avoidance, interoceptive fear–avoidance, work impairment–distress, and social impairment–distress. It is important to note that these seven features rated on the PDSS did not include any catastrophic cognitions. Because catastrophic cognitions were used in the current study to predict panic disorder severity and treatment outcome as measured by the PDSS, it was essential that this measure of severity not overlap with the measure of catastrophic cognitions.

The PDSS has been shown to have excellent interrater reliability, moderate internal consistency, and very good validity and sensitivity to change. Internal consistency reliability for the PDSS based on pretreatment data from the participants in the treatment conditions relevant to the current study would be considered moderate (α = .67). However, prior research on the psychometric properties of the PDSS has noted that the internal consistency of this scale is reduced when used with a sample with a restricted range on panic-related variables, such as the limited agoraphobia sample used in the current study (Shear et al., 1997).

PDSS data were collected through an independent evaluator assessment at baseline and were also collected following the acute, maintenance, and follow-up phases. The evaluators were blind as to treatment condition throughout the study. In addition to these independent evaluator assessments, PDSS data were recorded by clinicians at the end of each treatment session. These clinician ratings served as a weekly assessment of treatment outcome that could be carried forward for participants who dropped out of the study early and were included along with independent evaluator data in intent-to-treat (ITT) analyses. These ITT analyses therefore included early termination PDSS data obtained in session by clinicians, as well as data obtained by independent evaluators who assessed participants who completed the phase of the study in question. Analyses for the current study used assessment data obtained at baseline, following acute treatment, and at the 6-month follow-up. Postmaintenance treatment data were not used in the current analyses because of the treatment condition differences in criteria necessary for entering this phase of treatment.

Thoughts Rating Form (TRF). The TRF is an 18-item, self-report questionnaire developed for use in the MCCCTSPD to assess the extent to which participants experienced particular catastrophic cognitions commonly associated with panic disorder. Respondents rated how much they believe each of 18 different cognitions when experiencing somatic symp-
toms of anxious arousal for no apparent reason. Belief in these cognitions was rated on a scale ranging from 0 (not at all) to 8 (extremely). TRF data were not collected during each treatment session but were collected as part of the independent evaluator assessments that took place at baseline and following each treatment phase.

In keeping with other measures of catastrophic cognitions, the TRF’s 18 items were designed to measure fears of (a) physical catastrophes, (b) mental catastrophes, and (c) social catastrophes. The TRF is a transparent measure with clear face validity. However, to ensure that the TRF was actually measuring three underlying factors, we conducted an exploratory factor analysis using principal axis factoring with oblique rotation. This analysis was based on data from the 273 participants from all five treatment conditions who completed the TRF prior to the start of treatment. Factor extraction procedures indicated three factors with eigenvalues greater than one. A three-factor solution was also supported by an examination of change in slope on the scree test of eigenvalues plotted against factors. This three-factor solution accounted for 60.5% of the common variance.

Using .40 as the cutoff point for loading, each item loaded on one of the three factors but not on the other two. There was one exception. The item “I will lose control of my bodily functions” did not load above .30 on any of the three factors and was eliminated. Table 1 provides the wording of the remaining 17 items that were divided into three factors labeled as physical, mental, and social catastrophe cognitions. Internal consistency reliabilities measured with Cronbach’s (1951) alpha were excellent for all three factors in this model (physical catastrophes α = .86, mental catastrophes α = .83, social catastrophes α = .88). Correlations between factors ranged from .18 (physical with mental) to .44 (social with mental). On the basis of these results, in the current study we used this three-factor system for categorizing catastrophic cognitions, which distinguished between fears of physical catastrophes (e.g., “I will have a heart attack”), mental catastrophes (e.g., “I will go crazy”), and social catastrophes (e.g., “People will laugh at me”).

Continuous variables were used to represent the intensity of the three different types of catastrophic cognitions both prior to and immediately following acute treatment. These variables were created by simply averaging scores on all of the items in a given factor. Traditional test–retest reliability data were not available for the TRF. However, reliability was examined with pretreatment and postacute treatment data from the 30 individuals in the current study that were considered treatment nonresponders at the end of the acute treatment phase. Although this method involved a 3-month interval between administrations, test–retest reliability statistics were respectable for physical, mental, and social catastrophe average item scores, with intraclass correlation coefficients of .81, .80, and .68, respectively.

**Results**

In the current study, we were exclusively focused on determining whether catastrophic cognitions were predictive of short-term and long-term treatment outcome in both CBT and imipramine treatments for panic and whether there were any Treatment Condition × Cognition Intensity interaction effects in predicting outcome. The main effects of treatment condition on short-term and long-term outcome in panic disorder were reported elsewhere (Barlow et al., 2000).

### Pretreatment and Postacute Treatment Levels of Catastrophic Cognitions

Table 2 displays pretreatment and postacute treatment descriptive statistics for all three types of catastrophic cognitions among CBT and imipramine participants who completed acute treatment. Table 2 shows that when collapsing across treatment conditions, the pretreatment means and standard deviations for physical, mental, and social cognitions were quite similar. Furthermore, there were no treatment condition differences in the levels of any of the three types of cognitions at pretreatment or postacute treatment. Finally, Table 2 shows that all three types of catastrophic cognitions were significantly reduced during the course of acute treatment in both the CBT and imipramine treatment conditions.

### Predicting Postacute Treatment Outcome Based on the Intensity of Pretreatment Catastrophic Cognitions

Following the procedure described by Cohen and Cohen (1983), a hierarchical multiple regression methodology was used to determine whether the pretreatment intensity of the three different types of cognitions was predictive of postacute treatment panic disorder severity after controlling for pretreatment panic disorder severity levels. The dependent variable in these analyses was panic disorder severity as measured by the PDSS at the postacute treatment assessment. The first independent variable, entered as Step 1 in the hierarchical regressions, was pretreatment panic disorder severity as measured by the PDSS. The second step involved simultaneously entering (a) a dummy coded dichotomous variable that represented treatment condition (0 = CBT, 1 = Imipramine) and (b) three centered continuous variables that represented the pretreatment intensity of fears of physical, mental, and social catastrophes. The third and final step in the regression analyses was to simultaneously enter three interaction terms created from the centered and dummy coded variables entered at Step 2. These interaction term variables represented the following: (a) Treatment Condition × Physical Cognition Intensity, (b) Treatment × Mental Cognition Intensity, and (c) Treatment × Social Cognition Intensity.

Table 3 shows the results of two separate regression analyses that were conducted with the procedure described above. The first analysis was based on participants who completed acute

### Table 1

**Factor Structure of the Thoughts Rating Form**

<table>
<thead>
<tr>
<th>Factor</th>
<th>Item</th>
</tr>
</thead>
<tbody>
<tr>
<td>I. Physical catastrophes</td>
<td>I will die.</td>
</tr>
<tr>
<td></td>
<td>I will pass out.</td>
</tr>
<tr>
<td></td>
<td>I will have a stroke.</td>
</tr>
<tr>
<td></td>
<td>I will have a heart attack.</td>
</tr>
<tr>
<td></td>
<td>I will suffocate.</td>
</tr>
<tr>
<td></td>
<td>I will choke.</td>
</tr>
<tr>
<td>II. Mental catastrophes</td>
<td>I will go crazy.</td>
</tr>
<tr>
<td></td>
<td>I will become hysterical.</td>
</tr>
<tr>
<td></td>
<td>I will uncontrollably try to escape.</td>
</tr>
<tr>
<td></td>
<td>I will think irrationally.</td>
</tr>
<tr>
<td>III. Social catastrophes</td>
<td>People will think I’m weird.</td>
</tr>
<tr>
<td></td>
<td>People will laugh at me.</td>
</tr>
<tr>
<td></td>
<td>People will feel sympathy for me.</td>
</tr>
<tr>
<td></td>
<td>People will think I’m crazy.</td>
</tr>
<tr>
<td></td>
<td>People will think I’m irresponsible and/or incompetent.</td>
</tr>
<tr>
<td></td>
<td>I will be unable to function competently.</td>
</tr>
<tr>
<td></td>
<td>People will stare at me.</td>
</tr>
</tbody>
</table>
treatment, whereas the second was based on an ITT methodology. This more conservative ITT approach involved carrying forward early termination data for participants who dropped out of the study prior to completing the acute treatment phase. The results of these two analyses were virtually identical. Table 3 shows that, in both approaches to the data, the pretreatment intensity of fears of physical and mental catastrophes was not a significant predictor of postacute treatment panic disorder severity after controlling for pretreatment panic disorder severity. In contrast, the pretreatment intensity of fears of social catastrophes was a significant predictor of postacute treatment panic disorder severity, even after controlling for pretreatment panic levels in both the treatment completers and ITT analyses. The positive beta values indicate that regardless of pretreatment panic disorder severity, participants with more severe pretreatment fears of social catastrophes reported more severe panic symptoms after the acute phase of treatment.

Table 3 also shows that none of the Treatment Condition × Catastrophic Cognition interaction terms entered as Step 3 of the regressions approached statistical significance. These results indicate that the nature of the connection (or lack thereof) between all three types of catastrophic cognitions and postacute treatment outcome was the same for participants in both the CBT and imipramine conditions. In other words, the pretreatment intensity of participants’ thoughts regarding physical and mental catastrophes was unrelated to short-term treatment outcome in both the CBT and pharmacotherapy conditions. However, higher pretreatment levels of social catastrophe cognitions were associated with a poorer response to both CBT and imipramine. This again was true regardless of whether these analyses were conducted with an ITT methodology or were restricted to data from acute treatment completers.

**Predicting Treatment Outcome at Follow-Up Based on the Intensity of Catastrophic Cognitions at Pretreatment**

Additional hierarchical regression analyses were conducted to determine whether the intensity of pretreatment catastrophic cognitions was predictive of long-term treatment outcome as measured at the 6-month follow-up assessment. The dependent variable in these analyses was panic disorder severity as measured by the PDSS at the 6-month follow-up. The independent variables were again entered in a hierarchical fashion and were identical to the postacute treatment analyses already described.

Table 3 shows the results of two separate regression analyses that predicted panic disorder severity at follow-up: one based on data from participants who completed acute treatment and a second based on an ITT methodology. These two different approaches to the data again produced very similar results. In both analyses, variables that represented pretreatment physical and mental catastrophe cognitions were not significant predictors of panic disorder severity at follow-up after controlling for pretreatment panic levels. However, the pretreatment intensity of fears of social catastrophes was a significant predictor of long-term treatment outcome, even after controlling for pretreatment panic levels in both the acute completers and ITT analyses. Again, none of the Treatment Condition × Catastrophic Cognition interaction terms entered as Step 3 of the regressions approached statistical significance.

**Predicting Postacute Treatment Outcome Based on the Intensity of Catastrophic Cognitions at the End of Acute Treatment**

Thus far, analyses have been presented in which the pretreatment levels of catastrophic cognitions were used to predict short-term and long-term treatment outcome. Additional analyses were conducted to determine whether the intensity of these cognitions measured at the end of acute treatment would be predictive of short-term and long-term response to CBT and imipramine. It was not possible to use an ITT methodology in these analyses because catastrophic cognitions were only measured at the formal assessment points at the end of each phase of the study (see Table 2 for postacute treatment means and standard deviations for physical, mental, and social cognitions). Therefore, the analyses with post-
Table 3

Summary of Hierarchical Regression Analyses for Pretreatment Variables Predicting Postacute Treatment and Follow-Up Panic Severity Based on Both Acute Treatment Completers and an Intent-to-Treat (ITT) Methodology

<table>
<thead>
<tr>
<th>Variable</th>
<th>Predicting postacute panic</th>
<th></th>
<th>Predicting follow-up panic</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>B</td>
<td>SE B</td>
<td>β</td>
<td>B</td>
</tr>
<tr>
<td>Step 1: Pretreatment panic disorder severity</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pretreatment panic severity</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acute completers</td>
<td>0.45</td>
<td>0.13</td>
<td>0.34***</td>
<td>0.40</td>
</tr>
<tr>
<td>ITT</td>
<td>0.46</td>
<td>0.11</td>
<td>0.35***</td>
<td>0.67</td>
</tr>
<tr>
<td>Physical cognitions</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acute completers</td>
<td>-0.02</td>
<td>0.03</td>
<td>-0.06</td>
<td>0.02</td>
</tr>
<tr>
<td>ITT</td>
<td>-0.01</td>
<td>0.03</td>
<td>-0.02</td>
<td>0.03</td>
</tr>
<tr>
<td>Mental cognitions</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acute completers</td>
<td>0.01</td>
<td>0.04</td>
<td>0.02</td>
<td>0.02</td>
</tr>
<tr>
<td>ITT</td>
<td>0.00</td>
<td>0.03</td>
<td>-0.01</td>
<td>-0.05</td>
</tr>
<tr>
<td>Social cognitions</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acute completers</td>
<td>0.11</td>
<td>0.05</td>
<td>0.30*</td>
<td>0.16</td>
</tr>
<tr>
<td>ITT</td>
<td>0.08</td>
<td>0.04</td>
<td>0.21*</td>
<td>0.14</td>
</tr>
<tr>
<td>Step 3: Pretreatment Catastrophic Cognitions × Treatment Condition interaction terms</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Physical × Treatment</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acute completers</td>
<td>0.00</td>
<td>0.06</td>
<td>0.01</td>
<td>-0.06</td>
</tr>
<tr>
<td>ITT</td>
<td>0.04</td>
<td>0.06</td>
<td>0.09</td>
<td>0.02</td>
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<tr>
<td>Mental × Treatment</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acute completers</td>
<td>0.04</td>
<td>0.08</td>
<td>0.08</td>
<td>-0.11</td>
</tr>
<tr>
<td>ITT</td>
<td>-0.02</td>
<td>0.07</td>
<td>-0.04</td>
<td>-0.15</td>
</tr>
<tr>
<td>Social × Treatment</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acute completers</td>
<td>-0.10</td>
<td>0.09</td>
<td>-0.21</td>
<td>-0.04</td>
</tr>
<tr>
<td>ITT</td>
<td>-0.06</td>
<td>0.08</td>
<td>-0.13</td>
<td>0.02</td>
</tr>
</tbody>
</table>

Note. n = 96, R² = .24, p ≤ .01 for acute completers analysis predicting postacute panic; n = 135, R² = .18, p ≤ .001 for ITT analysis predicting postacute panic; n = 53, R² = .43, p ≤ .001 for acute completers analysis predicting follow-up panic. N = 123, R² = .24, p ≤ .001 for ITT analysis predicting follow-up panic.

*p ≤ .05. **p ≤ .01. ***p ≤ .001.

Acute treatment cognitions as predictors were based on participants who completed the acute treatment phase.

The first analysis of this type involved a hierarchical multiple regression in which variables representing the postacute treatment intensity of fears of physical, mental, and social catastrophes were tested as predictors of postacute treatment panic disorder severity after controlling for pretreatment panic levels. This analysis was designed to answer the following question: “Is ending treatment with lower levels of particular types of catastrophic cognitions associated with a better response to cognitive–behavioral and drug treatment?” This method was deemed superior to simply correlating cognition intensity levels and panic severity with both variables measured at posttreatment because, in essence, it allows one to examine how cognitions at the end of treatment are related to change in panic severity from pretreatment to posttreatment. It would also have been possible to examine cognitive change scores as predictors of change in panic severity. However, the use of change scores has been shown to be statistically problematic in outcome studies in general (e.g., Mintz, Luborsky, & Christoph, 1979) and specifically within the panic literature (e.g., Chambless & Gracely, 1988).

This hierarchical regression was identical to the analyses already described except that cognitive variables were measured at the end of acute treatment rather than at pretreatment. Variables were again entered into the regression in three steps: Step 1—pretreatment panic disorder severity; Step 2—treatment condition and postacute treatment fears of physical, mental, and social catastrophes, and Step 3—variables that represented the interaction of treatment and the three posttreatment cognition variables.

Table 4 displays the results of this hierarchical multiple regression and shows that a significant main effect was found for postacute treatment social catastrophe cognitions. The positive coefficient for this cognitive variable indicates that higher levels of fears of social catastrophes at the end of acute treatment were associated with higher levels of panic disorder severity following treatment, even after controlling for pretreatment panic disorder severity. In contrast, the variables that represented the postacute treatment intensity of fears of physical and mental catastrophes were not associated with acute treatment outcome. Neither of the Treatment Condition × Catastrophic Cognition intensity interaction terms were statistically significant.
Table 4
Summary of Hierarchical Regression Analysis for Postacute Treatment Catastrophic Cognitions Predicting Postacute Treatment Panic Disorder Severity Based on Acute Treatment Completers

<table>
<thead>
<tr>
<th>Variable</th>
<th>B</th>
<th>SE B</th>
<th>β</th>
</tr>
</thead>
<tbody>
<tr>
<td>Step 1: Pretreatment panic disorder severity</td>
<td>0.48</td>
<td>0.13</td>
<td>0.38***</td>
</tr>
<tr>
<td>Pretreatment panic severity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Step 2: Postacute treatment catastrophic cognitions</td>
<td>0.01</td>
<td>0.04</td>
<td>0.03</td>
</tr>
<tr>
<td>Physical cognitions</td>
<td>0.08</td>
<td>0.05</td>
<td>0.23</td>
</tr>
<tr>
<td>Mental cognitions</td>
<td>0.13</td>
<td>0.04</td>
<td>0.34**</td>
</tr>
<tr>
<td>Social cognitions</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Step 3: Postacute Catastrophic Cognitions × Treatment Condition interaction terms</td>
<td>-0.03</td>
<td>0.08</td>
<td>-0.06</td>
</tr>
<tr>
<td>Physical × Treatment</td>
<td>-0.01</td>
<td>0.10</td>
<td>-0.01</td>
</tr>
<tr>
<td>Mental × Treatment</td>
<td>-0.04</td>
<td>0.10</td>
<td>-0.07</td>
</tr>
</tbody>
</table>

Note. n = 88, R² = .44, p ≤ .001.
** p ≤ .01. *** p ≤ .001.

Predicting Panic Disorder Severity at Follow-Up Based on the Intensity of Catastrophic Cognitions at the End of Acute Treatment

An analysis was conducted to examine the value of postacute treatment catastrophic cognition variables in predicting long-term treatment outcome. This analysis used a 3-step hierarchical regression that was identical to the postacute treatment analysis described above, except that (a) the dependent variable in this case was panic disorder severity at follow-up and (b) postacute treatment panic disorder severity was controlled for in Step 1 of the regression. In the current study, this methodology allowed us to examine whether cognitive variables at the end of acute treatment were associated with relapse of panic symptoms during the 6-month period following the end of treatment.

Table 5 shows the results of this analysis. Participants who completed acute treatment with more severe panic symptoms continued to report more severe symptoms at the follow-up assessment, as would be expected. In keeping with the results of analyses already described, postacute treatment fears of physical and mental catastrophes were not significant predictors of long-term outcome. However, in contrast to the current study’s other findings, postacute treatment fears of social catastrophes also failed to emerge as a significant predictor of long-term outcome. Finally, Table 5 shows that none of the Treatment Condition × Catastrophic Cognition intensity interaction terms were statistically significant.

Discussion

In the current study, thoughts involving physical and mental catastrophes did not emerge as significant predictors of CBT or imipramine treatment outcome. In contrast, individuals in both these treatment conditions who reported more intense fears of social catastrophes prior to treatment tended to report more substantial panic symptoms at the end of 12 weeks of treatment. Participants who reported stronger pretreatment social catastrophe thoughts also tended to report higher panic symptoms at a follow-up assessment conducted 6 months after CBT or drug treatment discontinuation. Furthermore, individuals who continued to report higher levels of social catastrophe cognitions after the first 12 weeks of treatment were also likely to report a poorer response to both types of treatment at that time point. These findings are generally consistent with prior research conducted by Keijsers et al. (1994) who found that stronger pretreatment catastrophic cognitions in general are associated with a poorer response to behavioral treatment. However, the results of the current study partially contradict earlier work by Chambless and Gracely (1988) who found that pretreatment social and behavioral catastrophe cognition intensity was not significantly related to behavioral treatment outcome. However, for their predictor variable, Chambless and Gracely used a factor that included items that would be considered mental catastrophe cognitions along with items that would be considered social catastrophe cognitions. This methodological difference might account for the discrepancy.

In contrast to the association between pretreatment social catastrophe cognitions and treatment outcome, posttreatment social catastrophe cognitions were not predictive of panic disorder severity at follow-up after controlling for postacute treatment panic severity. This is consistent with two earlier studies by Clark et al. (1994, 1999), which showed that posttreatment catastrophic cognitions did not predict long-term outcome (though a schema measure of body sensation misinterpretation did). These earlier studies, however, did not distinguish between different types of cognitions but instead used a single summary score that represented the strength of catastrophic cognitions in general. Because we evaluated the predictive role of the three different types of catastrophic cognitions separately in the current study, it was somewhat surprising that posttreatment social cognitions did not emerge as a significant predictor of long-term outcome, as social cognitions were shown to be a significant predictor at pretreatment. The failure of posttreatment social cognitions to emerge as a significant predictor of panic disorder severity at follow-up is almost certainly because of the very strong correlation.
between postacute treatment panic levels and follow-up assessment panic levels in the current study. Postacute treatment panic disorder severity accounted for more than 55% of the variance in panic disorder severity measured at follow-up. Given that this large proportion of variance was parcelled out prior to testing the predictive value of the postacute treatment cognitive variables, it is easy to understand why social catastrophe cognitions did not emerge as a significant predictor.

Prior research (e.g., Bouvard et al., 1998; Chambless & Gracely, 1989) has indicated that thoughts of physical catastrophes are the only type of cognitions capable of discriminating panic disorder from other anxiety disorders. This implies that physical catastrophe cognitions are more central to the development of panic disorder as distinguished from other anxiety disorders. Nevertheless, the results of the current study suggest that it is social catastrophe cognitions that are most closely tied to success in overcoming panic disorder. There are a number of possible explanations for this combination of results.

Perhaps the most obvious explanation would be to assume that physical and mental catastrophe cognitions were uniformly reduced among all participants regardless of outcome, whereas only those individuals who showed the strongest response to treatment were able to achieve significant reductions in social catastrophe cognitions. However, the data do not support this explanation. The different types of catastrophic thoughts were equally reduced during acute treatment (Hicks, 2002) and the pretreatment and postacute treatment descriptive statistics were quite similar for all three types of cognitions (see Table 2). Nevertheless, only the level of social cognitions at the end of treatment was related to postacute treatment outcome. Taken together, these findings may imply that even at equally low levels, thoughts regarding social catastrophes are more likely to interfere with successful panic treatment as compared with physical and mental catastrophe cognitions. Research by Arrindell (1993) showed that only social–behavioral catastrophe cognitions are predictive of future agoraphobic avoidance in the absence of treatment. On the basis of this finding, it seems reasonable to suppose that lingering fears of social embarrassment could prevent individuals receiving CBT or pharmacotherapy from engaging in the naturalistic exposure to previously avoided situations that may be an important element of success in either treatment.

It may simply be more difficult to believe one is relatively safe from embarrassing oneself socially than from going crazy or experiencing a heart attack or other physical catastrophes. This is partly because there is often less objective evidence one can marshal regarding one’s safety in the social arena as compared with the mental or physical health arenas. A patient can be somewhat reassured by a mental health professional that they are not in fact losing their mind, or they can be informed by a physician that they have not actually experienced a cardiac emergency. Judging social embarrassment is a comparatively subjective task. An individual experiencing a panic attack is likely to grossly exaggerate the extent to which those around them notice their anxious symptoms. Despite this often distorted viewpoint, an individual experiencing a panic attack in public may actually attract some amount of social attention. As a result, many of these individuals will experience intense shame, as well as dread of this occurring again in the future. Although this social attention does not inevitably represent a catastrophe, there is an element of reality in this type of fear that is entirely absent in the vast majority of physical and mental catastrophe fears.

The findings of the current study imply that further reduction of fears of social catastrophes beyond what is already being achieved may be an important target in panic disorder treatments. This assumes, of course, that catastrophic cognitions have more of an influence on panic attacks than panic attacks have on catastrophic cognitions, an assumption with intuitive clinical appeal but in need of further empirical verification. If this assumption proves to be true, then it seems clear that CBT providers should increase their emphasis on effecting more substantial reductions in social catastrophe cognitions in the context of exposure exercises. Furthermore, given that fears of social catastrophes were also closely linked to poorer outcome in the imipramine treatment condition, the current study’s findings imply that patients receiving pharmacological treatments should also be further encouraged to reenter social situations they had been avoiding prior to seeking treatment.

Some limitations of the current research should be noted. Perhaps most importantly, the panic disorder sample used in the current study was limited to individuals with mild-to-moderate agoraphobia. It is an empirical question whether the findings from the current study, which so strongly suggest that social catastrophe cognitions are of much greater prognostic value than physical or mental catastrophe cognitions, are applicable to panic disorder samples with more severe agoraphobia. It may be the case that among individuals with severe agoraphobia, these other types of cognitions are more significant. Another limitation is that, because of their slimmer side effects profile (as opposed to superior efficacy; Barlow et al., 2000), selective serotonin reuptake inhibitors (SSRIs)—rather than tricyclic antidepressants—are currently considered the first-line medication for the treatment of panic. Although there is little reason to think that the prognostic roles of social versus physical and mental catastrophe cognitions would vary depending on which type of antidepressants are used, this also is an empirical question awaiting further research. It should also be noted that because catastrophic cognition data were not the focus of the original study used for the current analyses, the rate of missing data for cognitive variables was relatively high (though randomly distributed), and analyses were limited to participants who provided these data. Finally, the follow-up assessment in the current study was predominantly based on individuals who had completed and responded to treatment. The follow-up should, therefore, be viewed as primarily an attempt to determine whether treatment responders experienced a relapse in symptoms or continued to report that their symptoms remained reduced. Future studies may be more successful in collecting follow-up data from all participants who initially entered treatment. Such data would be most informative about the long-term clinical outcome for individuals who drop out of treatment early.

Despite these limitations, the findings presented here represent an important step forward in our understanding of the role of catastrophic cognitions in predicting response to cognitive–behavioral and pharmacological treatments for panic disorder.

References