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GROUP COGNITIVE-BEHAVIORAL TREATMENT OF PANIC DISORDER

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Summary—The present study examined the efficacy of an 8-wk, cognitive-behavioral group treatment for panic disorder. Patients meeting DSM-III-R criteria for panic disorder with or without agoraphobia were randomly assigned to treatment ($N = 34$) or delayed treatment control ($N = 33$). The treatment consisted of: (a) education and corrective information; (b) cognitive therapy; (c) training in diaphragmatic breathing; and (d) interoceptive exposure. At posttreatment, 85% of treated *S*s were panic free, compared to 30% of controls. Treated *S*s also showed clinically significant improvement on indices of anxiety, agoraphobia, depression and fear of fear. Recovery, as estimated conservatively by the attainment of normal levels of functioning on each of the major clinical dimensions of the disorder (i.e. panic, anxiety and avoidance), was achieved in 64% of the treated *S*s and 9% of the controls. At the 6 month follow-up, 63% of the treated patients met criteria for recovery. These findings mirror those from recently-completed trials of individually-administered cognitive-behavioral treatment, and suggest that CBT is a viable alternative to pharmacotherapy in the treatment of panic disorder.

INTRODUCTION

Epidemiologic data indicate that anxiety disorders are the most prevalent of all mental disorders in the U.S. (Karno, Hough, Burnam, Escobar, Timbers, Santana & Boyd, 1987). Panic and its pathological expression have taken on a central role in the conceptualization and classification of the anxiety disorders. A panic attack consists of a sudden onset of intense apprehension, fear or discomfort. The diagnosis of panic disorder is reserved for persons who experience at least four attacks in a 4-wk period or at least one attack followed by a period of at least 30 days of persistent apprehension concerning panic recurrence (American Psychiatric Association, 1987). Panic disorder afflicts approx. 2% of the population (Weissman, 1988) and is one of the leading causes of people seeking out medical and mental health services (Boyd, 1986).

The consequences of panic disorder can be devastating. Many panic sufferers develop agoraphobia, a disorder involving severe anticipatory anxiety and marked avoidance of situations or activities previously associated with a panic attack (Tearnan, Telch & Keefe, 1984; Thyer & Himle, 1985). Other sequelae include alcohol and tranquilizer abuse, depression, lowered self-esteem, marital problems and suicide (Markowitz, Weissman, Ouellete, Lish & Klerman, 1989; Weissman, Klerman, Markowitz & Ouellete, 1989).

Over the past decade significant advances have been made in biological treatments of panic disorder and agoraphobia. Several classes of medications, including the tricyclic antidepressants (e.g. imipramine), MAO inhibitors (e.g. phenelzine) and high-potency benzodiazepines (e.g. alprazolam), have demonstrated panic-blocking effects in over a dozen double-blind placebo controlled trials (Telch, 1988; Telch, Tearnan & Taylor, 1983). Although pharmacological treatments have proved helpful for many panic sufferers, there are problems associated with their use: fear or unwillingness to take medications, troublesome side effects, high attrition rates and relapse upon withdrawal of medication (Telch *et al.*, 1983).

To date, encouraging patients to repeatedly confront fear-provoking situations (i.e. *in vivo* exposure) has been the most consistently effective and well studied psychological treatment for agoraphobia with panic (Jacobson, Wilson & Tupper, 1988). This treatment approach targets the phobia component of the panic syndrome without addressing the underlying panic disorder.

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Although 60–70% of agoraphobics completing exposure-based treatment derive some clinical benefit that endures for 4 or more years (Marks, 1971; Munby & Johnston, 1980), panic attacks are not eliminated in the majority of patients receiving exposure-based therapies (Michelson, Mavissakalian & Marchione, 1985). It has been suggested that failure to eliminate panic accounts for why 30–40% of those who complete exposure treatment derive no significant benefit and why those who do benefit continue to display some level of dysfunction (Janssen & Ost, 1982).

Advances in psychological treatments which target panic directly have recently emerged. This new genre of cognitive-behavioral treatment focuses on correcting the patient's emotional hypersensitivity to certain somatic cues (e.g. lightheadedness) and the misinterpretation of these cues as signaling impending physical, mental or social catastrophe. Treatment typically includes: (a) education about the nature and physiology of anxiety and panic; (b) cognitive restructuring of patients' faulty threat appraisals (e.g. "I will have a heart attack", "I will lose control of my senses"); (c) breathing exercises to control hyperventilation and to help patients cope with stress and anxiety; and (d) interoceptive exposure—purposeful and repeated induction of feared somatic cues (e.g. having the patient repeatedly spin in a chair to reduce her fear of dizziness). Efficacy data from several uncontrolled trials (Barlow, Cohen, Waddell, Vermilyea, Klosko, Blanchard & DiNardo, 1984; Beck, 1988; Clark, Salkovskis & Chalkley, 1985; Gitlin, Martin, Shear, Francis, Ball & Josephson, 1985; Michelson, Marchione, Greenwald, Glanz, Testa & Marchione, 1990; Salkovskis, Jones & Clark, 1986) and a few controlled trials (Barlow, Craske, Cerney & Klosko, 1989; Clark, Gelder, Salkovskis, Hackman, Middleton & Anastasiades, 1990; Klosko, Barlow, Tassinari & Cerny, 1990) indicate that approx. 80–90% of treated patients are panic free by the end of treatment. However, none of the published reports examine the extent to which treatment restores patients to normative levels of functioning (i.e. clinical significance). We present data from the first controlled trial to investigate the efficacy of a *group-administered* cognitive-behavioral treatment for panic disorder. The clinical significance of treatment gains was examined for panic attacks, anxiety, panic-related avoidance, fear of fear and depression.

METHOD

Subject recruitment, screening and selection

Patients ($N = 67$) meeting DSM-III-R criteria for panic disorder took part in the study. Subjects were recruited through local media channels and letters to physicians and mental health workers in the Austin, TX area. Subject demographics are presented in Table 1. A two-step screening procedure was employed. Individuals contacting our laboratory underwent an initial 15 min structured phone interview conducted by a trained doctoral student in clinical psychology. Respondents reporting panic attacks within the past month and who expressed interest in participating were scheduled for a comprehensive clinical screening evaluation at our laboratory. During this second phase of screening, patients were carefully diagnosed using the Structured Clinical Interview for DSM-III-R—Nonpatient Version (SCID; Spitzer, Williams & Gibbon, 1987). This widely used diagnostic interview has demonstrated adequate inter-rater reliability for anxiety disorder samples. SCID interviews were conducted by advanced graduate students in clinical psychology who had received extensive training in SCID administration and scoring. Each case was reviewed by the first author during weekly staff meetings.

Only those patients meeting the following entry criteria were invited to participate: (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. When another Axis I disorder was present (e.g. major depression), the determination of principal diagnosis was based on the disorder that currently resulted in the most distress and impairment for the patient.

Experimental design

Subjects were matched on panic severity as determined by the SCID interview and randomly assigned to the experimental treatment or a delayed-treatment control. A comprehensive assessment

Table 1. Patient demographics

Variable	Treatment condition: Panic inoculation ($N = 34$)	Delayed treatment ($N = 33$)	Total ($N = 67$)
Age (yr)			
Mean	36.9	32.1	34.6
SD	11.7	8.0	10.3
Sex (% female)	76.5	69.7	73.1
Ethnicity (%)			
White	91.2	84.4	87.9
Hispanic	5.9	12.5	9.1
Black	2.9	3.1	3.0
Marital status (%)			
Never married	32.3	36.4	34.3
Married	41.2	48.4	44.8
Divorced/separated	20.5	15.2	17.9
Education (%)			
Less than high school	0	3.2	1.6
High school	9.1	12.9	10.9
Part college	42.4	45.2	43.8
College graduate or beyond	48.5	38.7	43.7
Employment status (%)			
Employed	67.6	66.7	67.2
Unemployed	5.9	9.1	7.5
Student	14.7	21.2	17.8
Housewife	11.8	3.0	7.5
Chronicity (yr)			
Mean	8.0	9.3	8.7
SD	8.3	8.7	8.5
Treatments for panic (%)			
Psychosocial TX (lifetime)	58.8	84.4	71.2
Psychiatric hospitalizations (lifetime)	2.9	15.1	9.0
Prescribed psychotropics (lifetime)	85.3	87.9	87.9
Prescribed psychotropics at intake	55.9	66.7	61.2
None	44.1	33.3	38.8
Benzodiazepines	41.2	39.4	40.3
Antidepressants	5.9	6.1	6.0
Benzo. & antidep.	5.9	9.1	7.4
Other	2.9	12.1	7.5

battery tapping the major clinical dimensions of the disorder (i.e. panic attacks, anxiety, phobic avoidance, depression and fear of fear) was administered at baseline (week 0), posttreatment (week 9) and 6 month follow-up (week 35).

Treatment procedures

Panic-inoculation training. The experimental treatment was derived from the Panic Control Treatment developed at SUNY Albany (Craske & Barlow, 1990). This multicomponent treatment consists of four major components: (a) education and corrective information concerning the nature, etiology and maintenance of panic; (b) cognitive therapy techniques aimed at helping the patient identify, monitor, and alter faulty appraisals of threat that contribute to panic occurrence; (c) training in methods of slow diaphragmatic breathing as a way of reducing or eliminating physical symptoms that often trigger panic attacks; (d) interoceptive exposure exercises designed to reduce patients' fear of somatic sensations through repeated exposure to feared bodily sensations. The protocol was modified to allow for group administration. Moreover, the informational and interoceptive components were expanded in an attempt to increase the potency of treatment. Specific additions included: (a) informational modules on heart disease, fainting and seizures; and (b) repeated self-directed ingestion of 200–400 mg of caffeine.

Treatment was delivered in small groups ranging from 4–6 patients per group. Subjects received 12 90-min treatment sessions over an 8-wk period. Sessions 1–8 were conducted twice weekly; Sessions 9–12 were spaced a week apart. All sessions were conducted by one primary therapist (MJT, JL or NBS) and a graduate student assistant.

Delayed-treatment control. Subjects assigned to the delayed-treatment were evaluated at weeks 0 and 9 but did not receive treatment during the first 8 weeks. Following the posttreatment assessment at week 9, these *S*s were offered the experimental treatment.

Assessment battery

Assessment of the effects of treatment on the panic/agoraphobia syndrome requires reliable and valid measures that tap the major clinical features of the disorder (i.e. panic attacks, anxiety, phobic avoidance, dysphoric mood and fear of fear). Thus, a set of primary outcome measures sampling different clinical features of the syndrome (i.e. panic attacks, anxiety, avoidance, depression and fear of fear) were collected at each of the three assessment periods (i.e. baseline, posttreatment and follow-up). Because one goal was to determine the extent to which the treatment moved patients to normal levels of functioning, the availability of normative data for a nondisordered population was an important consideration in the final selection of assessment instruments. The major outcome measures included the: (a) Sheehan Patient-Rated Anxiety Scale (SPRAS); (b) Agoraphobia scale of the Marks and Mathews Fear Questionnaire (FQ-Ago); (c) Beck Depression Inventory (BDI); and (d) Anxiety Sensitivity Index (ASI).

Assessment of panic attacks. Panic attacks were assessed using a prospective self-monitoring approach. Recent evidence suggests that prospective daily self-monitoring of panic may be less subject to overreporting bias than retrospective recall methods (Margraf, Taylor, Ehlers, Roth & Agras, 1987). Subjects were provided daily panic diary forms modeled after those used in the UpJohn Multi-Center Panic Study (Ballenger, Burrows, DuPont, Lesser, Noyes, Pecknold, Rifkin & Swinson, 1988). For each episode of panic, *S*s were instructed to record the: (a) date; (b) time; (c) duration; (d) severity; (e) symptoms experienced; (f) setting parameters (e.g. place, activities, accompanied); and (g) type of attack (situational or spontaneous). Instructions stressed the importance of recording panic episodes immediately. In order to reduce the likelihood that *S*s would mislabel anxiety episodes as panic attacks, *S*s were provided a clear definition of panic that stressed the *sudden* onset of symptoms. At each assessment, panic diaries were inspected. Panic attacks were classified as either *situational* (attack occurred in the presence of an identifiable fear-provoking cue) or *spontaneous* (attack occurred in the absence of any identifiable fear-provoking cue). Reported panics with fewer than three symptoms (i.e. limited symptom attacks) were excluded in the panic attack count.

Sheehan Patient-Rated Anxiety Scale (SPRAS). The SPRAS (Sheehan, 1983) is a 35-item self-report scale for assessing the intensity of anxiety symptoms. Each of 35 symptoms (e.g. shaking or trembling) is rated on a 5-point scale ranging from 0 (not at all distressing) to 4 (extremely distressing). Consistent with its use in the UpJohn Cross National Panic Study (Ballenger *et al.*, 1988), we modified instructions so that symptom ratings were based on a 1 wk time frame as opposed to the original 6 month time frame.

Fear Questionnaire (FQ-Ago). The Fear Questionnaire (Marks & Mathews, 1979) was used to assess *S*s' level of phobic avoidance. The FQ consists of 15 items representing three separate phobia types (agoraphobia, blood and injury phobia and social phobia). For each item, the *S* rates the degree of avoidance to the specific object or situation. The five-item agoraphobia subscale (FQ-agoraphobia) has demonstrated adequate psychometric properties and is the most widely used measure for indexing level of agoraphobia in treatment outcome research (Jacobsen & Truax, 1991).

Anxiety Sensitivity Index (ASI). The ASI is a widely used 16-item questionnaire that measures the fear of anxiety (Reiss, Peterson, Gursky & McNally, 1986). Each item expresses a concern about a possible aversive consequence of symptoms associated with anxiety (e.g. "When I notice that my heart is beating rapidly, I worry that I might have a heart attack"). Items are rated on a 5-point Likert scale, with total scores ranging from 0 to 64. The psychometric properties of the ASI have been favorable (Telch, Shermis & Lucas, 1989).

Beck Depression Inventory (BDI). The BDI is a widely used and extensively researched self-report instrument for assessing depression in both psychiatric and normal populations. Substantial data support its reliability and validity (Beck & Steer, 1987; Lambert, Hatch, Kingston & Edwards, 1986).

Assessment of treatment integrity

A valid test of treatment efficacy requires that the treatment be delivered in the intended manner. To help protect the integrity of the treatment, therapists and their assistants followed a procedural outline for each therapy session. In addition, all treatment sessions were videotaped and randomly selected segments were rated for consistency with the written treatment protocol.

Overview of analyses

Baseline differences between treated patients and delayed treatment controls on demographic and clinical variables were examined using independent t-tests for continuous variables and χ-square tests for categorical variables. Between-group differences on each of the major clinical outcome measures at posttreatment were examined using one-way ANCOVAs, with treatment group (Panic Inoculation vs Delayed Treatment Control) as the grouping factor and baseline level as the covariate. For each major outcome measure, within-group changes from pretreatment to posttreatment were analyzed separately for each treatment group using dependent t-tests. Within-group changes from pretreatment to follow-up were also examined for the panic inoculation group.

To investigate the clinical significance of the treatment gains independent of their statistical significance, analyses were conducted comparing the proportion of patients in each of the two groups who attained normal levels of functioning on each of the major clinical indices. Finally, the two groups were compared on a composite index of recovery defined as the proportion of patients attaining normal levels of functioning on all three major facets of the disorder (i.e. panic attacks, anxiety and panic-related avoidance).

RESULTS

Differences at baseline

Subjects in the two groups did not differ significantly on any of the demographic variables at intake with the exception that a greater percentage of delayed treatment controls had received psychosocial treatment for panic (see Table 1). Means and standard deviations on the major clinical measures for treated and untreated controls at each of the three assessments are presented in Table 2. As seen in Table 2, the two groups did not differ significantly at baseline on any of the major clinical measures.

However, at posttreatment, a consistent pattern of findings emerged. Compared to untreated *S*s, those receiving the group panic inoculation treatment displayed marked improvement on all major indices of treatment outcome. This difference was evident for all measures in the between-groups analyses, in which the treated *S*s scored significantly less pathological than untreated controls, even after controlling for between-group differences at baseline. Moreover, the within-group analyses revealed that treated subjects evinced highly significant improvement on all measures, whereas untreated *S*s failed to show significant improvement on any measure.

Table 2. Means and SDs for the major outcome measures at pretreatment, posttreatment and follow-up

	Panic inoculation			Delayed TX control	
Measure	Pre ($N = 34$)	Post ($N = 34$)	FU ($N = 30$)	Pre ($N = 33$)	Post ($N = 33$)
Panic total (wk)					
Mean	4.18	0.18	0.46	2.79	2.49
(SD)	(9.47)	(0.46)	(1.33)	(4.93)	(4.45)
Panic (spontaneous)					
Mean	3.27	0.09	0.23	1.00	1.00
(SD)	(9.58)	(0.29)	(0.68)	(1.52)	(1.94)
Panic (situational)					
Mean	0.91	0.09	0.23	1.79	1.49
(SD)	(1.36)	(0.29)	(0.90)	(4.33)	(2.77)
Anxiety (SPRAS)					
Mean	61.12	20.08	22.82	55.79	51.56
(SD)	(21.43)	(15.20)	(19.73)	(29.58)	(28.12)
Agoraphobia (FQ-Ago)					
Mean	12.18	5.06	5.60	15.47	14.85
(SD)	(11.41)	(6.76)	(7.89)	(8.85)	(9.94)
Depression (BDI)					
Mean	16.85	7.68	7.70	15.18	14.24
(SD)	(8.21)	(5.27)	(6.71)	(10.41)	(11.05)
Fear of fear (ASI)					
Mean	33.74	13.94	14.23	34.46	32.03
(SD)	(11.15)	(8.52)	(10.15)	(11.33)	(10.96)

Note: SPRAS = Sheehan Patient Rated Anxiety Scale; FQ-Ago = Agoraphobia subscale of the Fear Questionnaire; BDI = Beck Depression Inventory; ASI = Anxiety Sensitivity Index. Treated vs controls did not differ significantly on any of the measures at pretreatment. All treatment vs control comparisons at posttreatment were significant at the 0.01 level.

Table 3. Percentage of *S*s scoring in the normal range of functioning at pretreatment, posttreatment and 6 month follow-up

		Panic inoculation			Delayed treatment control	
Variable	Criterion for recovery	Pre ($N = 34$)	Post ($N = 34$)	FU ($N = 30$)	Pre ($N = 33$)	Post ($N = 33$)
Panic attacks	Panic attacks = 0	29.4	**85.3**	83.3	27.3	**30.3**
Anxiety	SPRAS < 30	5.9	**73.5**	74.1	24.2	**18.8**
Avoidance	FQ-Ago < 12	61.8	**85.3**	90.0	31.2	**39.4**
Fear of fear	ASI < 27	32.3	**97.1**	86.7	36.4	**33.3**
Depression	BDI < 10	23.5	**64.7**	60.0	33.3	**42.4**
Mean recovery	See Note	30.6	**81.2**	78.8	30.5	**32.8**
Composite recovery	See Note	0.0	**63.6**	63.3	0.0	**9.1**

Note: Mean recovery represents the average recovery rate across the five clinical outcome domains. Composite recovery represents a more conservative index indicating the proportion of *S*s who attain normal functioning on all of the following measures: panic attacks, anxiety and avoidance.

Recovery criteria for the BDI, SPRAS and FQ-Ago were based on well-accepted cutoff scores reported in the literature. The ASI criterion was calculated using the formula of Jacobsen *et al.* (1984). The recovery criterion for panic attacks was conservatively set at zero.

Effects of medication status on treatment outcome

To examine whether treatment outcome was differentially affected by *S*s' medication status at intake, we compared medicated and unmedicated *S*s at each of the three assessments. None of these analyses was significant. Medicated *S*s receiving group panic inoculation treatment did not differ significantly from unmedicated *S*s on any of the clinical measures at baseline, posttreatment or 6 month follow-up.

Clinical significance of treatment gains

We addressed the clinical significance of the treatment findings by examining the proportion of *S*s in each group who attained scores in the normal range on each of the major clinical dimensions of the disorder. These data are presented in Table 3.

Five clinical dimensions or facets were examined: panic attacks, anxiety, avoidance, fear of fear and depression. In addition, two recovery indices were constructed in an attempt to provide upper and lower bounds of recovery. The first index (*Mean recovery*) represents an average across the five clinical outcome domains, in the percentage of *S*s scoring in the normal range of functioning. A more conservative recovery index (*Composite recovery*) was calculated as the proportion of *S*s who attained normal functioning on *all* three of the following measures: panic attacks, anxiety and avoidance.

As seen in Table 3, the mean recovery rate at posttreatment was 81.2% for *S*s receiving group cognitive-behavioral treatment and only 30.5% for untreated controls. Using the more stringent composite criterion, 63.6% of the treated group and 9.1% of those untreated evidenced recovery on all three measures at posttreatment. The recovery estimates at the 6 month follow-up were essentially identical to those at posttreatment (i.e. Mean recovery = 78.8%; Composite recovery = 63.3%) suggesting a general trend for maintenance of improvement.

Relapse

We investigated the extent of relapse among *S*s who evidenced significant improvement at posttreatment. A relapsed case was defined as a *S* who showed statistically reliable improvement* at posttreatment but who was no longer improved at the 6 month follow-up. Two *S*s (6.7% of study completers) met this criterion for relapse at the 6 month follow-up.

DISCUSSION

The present findings demonstrate the efficacy of a group-administered cognitive-behavioral treatment for panic disorder. A complete resolution of panic attacks was observed in over 85%

*Statistically reliable improvement was determined using Jacobson *et al.* (1988) 'reliable change index'. Subjects showing a significant pre to posttreatment reliable change index on all three of the following measures: panic attacks, anxiety and avoidance, were classified as improved. Of those improved, *S*s were classified as having relapsed if they no longer met this same improvement criterion at follow-up.

of the treated cases compared to only 30% for delayed treatment controls. When more stringent criteria were used to classify recovery, between 63 and 80% of the experimentally treated *S*s displayed full recovery. These panic cessation rates compare favorably to those achieved in well-controlled pharmacological trials. For example, in the recently completed UpJohn Cross National Panic Study, 57% of study completers treated with alprazolam vs 50% of those treated with placebo showed a complete resolution of panic attacks (Ballenger, Burrows, DuPont, Lesser, Noyes, Pecknold, Rifkin & Swinson, 1988).

An important dimension of a treatment's utility is the extent to which improvement/recovery is maintained following treatment completion. In contrast to the substantial relapse observed in drug treatment trials (Pecknold, Swinson, Kuch & Lewis, 1988; Telch, 1988), recovery estimates at 6 months posttreatment were essentially identical to those observed at the earlier 8-wk posttreatment assessment. The observed durability of treatment gains is consistent with preliminary findings from several other centers which show lasting improvements for CBT for periods of up to 2 yrs.

Our estimates of recovery deserve comment. Several different methods have been employed to estimate extent of recovery or high endstate functioning in clinical outcome research (Jacobsen & Truax, 1991). These include: (a) global clinical ratings such as the CGI; (b) composite indices arbitrarily set by the investigator; and (c) normative approaches. Although each method has its advantages and disadvantages, we employed a stringent normative approach which conceptualizes recovery as the extent to which *S*s attain normal functional on clinically-relevant dimensions (i.e. panic attacks, anxiety and agoraphobic avoidance). Our findings indicate that requiring *S*s to score in the normal range on multiple indices yields a more conservative estimate of recovery than estimates based solely on panic attack status (i.e. percentage of *S*s who are panic free). However, we believe this normative approach offers a more ecologically-valid index of recovery since it takes into account the multifaceted nature of the panic disorder syndrome. Nevertheless, it should be noted that our recovery estimates are likely to differ from those based on other approaches such as clinical ratings.

Were our findings influenced by *S*s' use of medication during the trial? Approximately 61% of the *S*s at intake were currently taking medications to help control their panic and anxiety. A breakdown by medication type revealed that 40.3% of *S*s were taking only benzodiazepines, 6% were taking only antidepressants, 7.4% were taking benzodiazepines and antidepressants, while 7.5% were taking some other anxiety/panic medication. Patients currently on medications were allowed to enter the trial so long as they: (a) still met full DSM-III-R criteria for panic disorder; (b) had been taking medication for at least 2 months; and (c) agreed to keep their current use of medications stable during the active 8 wk treatment phase.

Subjects assigned to the treatment and control groups did not differ with respect to medication status at intake or posttreatment. If medications were exerting a potent therapeutic influence on patients' status, we would expect to have seen control subjects improve. Such was not the case. However, it is still possible that the experimental group's favorable response was due to a drug by cognitive-behavioral treatment interaction. To examine this possibility, we compared the medicated and unmedicated *S*s in the treated group. The two groups did not differ on any of the measures at baseline, posttreatment or follow-up. Thus, *S*s' medication use did not contribute to the success of this cognitive-behavioral group treatment.

Our study design does not rule out the possibility that nonspecific treatment factors were responsible for *S*s' improvements, but several factors argue against it. First, most of the *S*s had already undergone psychosocial and pharmacologic treatments. Presumably, nonspecific factors were operating in these treatments, yet they failed to produce significant benefits. Second, recent comparative studies of individually-administered CBT have shown it to be superior to supportive psychotherapy (Beck, Sokol, Clark, Berchick & Wright, 1993), imipramine (Clark *et al.*, 1990) or relaxation (Clark *et al.*, 1990). Taken together, these data strongly suggest that this treatment effects change through mechanisms other than nonspecific factors (e.g. demand characteristics).

Were our findings influenced by characteristics of the sample? Several investigators have speculated that CBT's success in treating panic may be due in part to a patient selection artifact. More specifically, it has been suggested that panic patients referred to psychological centers that specialize in CBT may be less severely impaired and thus be more likely to respond to a nonpharmacological treatment compared to patients who are severely impaired. We examined this

hypothesis in several ways. First, we compared our sample to a sample of patients who took part in a recently-completed pharmacological treatment trial conducted in a major medical center (Agras, Telch, Taylor, Roth & Brouillard, 1993). The clinical status of the two samples at baseline were quite comparable on measures of panic frequency, depression and global disability. Next, we tested whether patient demographics or clinical severity at baseline predicted recovery. None of the demographic factors, which included age, education, marital status, ethnicity and employment status were associated with treatment response. Moreover, intake severity of panic, anxiety, agoraphobia and depression failed to predict treatment response. Although additional investigation is needed to rule out the possibility that patient selection factors contribute to the success of CBT, our preliminary analyses failed to support the patient selection artifact hypothesis.

In summary, our results provide additional evidence that panic disorder can be effectively treated in the majority of cases using a cognitive-behavioral treatment focusing on panic control through education, cognitive restructuring, interoceptive exposure and breathing retraining. Moreover, our findings suggest that this treatment can be effective both in the short and long-term using a cost-effective group format.

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