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An Open Trial of a Transdiagnostic Cognitive-Behavioral Group Therapy for Anxiety Disorder

Peter J. Norton
University of Houston

Transdiagnostic models of anxiety, and cognitive-behavioral treatments based on these models, have been gaining increased attention in recent years. Preliminary efficacy studies generally suggest strong treatment effects, although few of these studies have examined to what extent treatment effects are similar across clients with different anxiety disorders. The purpose of the current study was to examine the efficacy of a 12-week transdiagnostic group cognitive-behavioral therapy for anxiety disorders and compare outcome across diagnoses. Mixed-effect regression modeling of data from 52 participants with anxiety disorders (predominantly panic disorder and social phobia) participating in an open outcome trial indicated that participants tended to improve over treatment, with no differential outcome for any primary or comorbid disorders. The results of this study add to the growing evidence base for transdiagnostic anxiety treatment models and provide preliminary support for the assumption that individuals with different anxiety diagnoses can be treated equally within the same treatment protocol.

Recent years have seen a resurgence of research examining common elements across, and within, diagnostic groups. Research and treatment of eating disorders, for example, appear to have benefited greatly from such *transdiagnostic* conceptualizations (Fairburn, Cooper, & Shafran, 2003), and transdiagnostic conceptualizations that extend beyond broad *DSM* groupings (e.g., negative affect syndromes; Barlow, Allen, & Choate, 2004) are also being proposed. Transdiagnostic models of anxiety disorders have also begun to emerge (Barlow, 2000; Barlow et al., 2004; Norton, 2006) and generally hold that the common elements across the anxiety disorders outweigh the differences. These models draw from the genetic and comorbidity literatures which suggest an extremely high level of overlap between disorders, as well as from the cognitive-behavioral and pharmacological treatment literature suggesting similar response to highly similar medications or treatment elements. From these models, comparable treatments (Erickson, 2003; Norton, Hayes, & Hope, 2004; Norton & Hope, 2005; Lumpkin, Silverman, Weems, Markham, & Kurtines, 2002) have been developed, which incorporate individuals with different anxiety disorders under the same treatment protocol. Despite this, published outcome data on treatments based on these models have been limited (for a review, see Norton, in press).

Erickson (2003) reported the results of an uncontrolled trial of a transdiagnostic cognitive-behavioral therapy (CBT) program for 70 individuals with anxiety disorders. His results suggested significant decreases in self-reported anxiety and depression among clients completing the 11-week treatment. Further 6-month follow-up data from 16 participants suggested maintenance of treatment gains. No analyses of outcome by diagnosis were conducted due to power limitations. Lumpkin et al. (2002) reported similar treatment effects following a 12-week transdiagnostic treatment with anxious youths. Multiple baseline results suggested notable reductions on measures of anxiety occurring during treatment, but no change during the baseline periods. As well, treatment gains were maintained at 6

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Address correspondence to Peter J. Norton, Ph.D., Department of Psychology, 126 Hyne Bldg., University of Houston, Houston, TX, 77204-5022; e-mail: pnorton@uh.edu.

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and 12 months. Again, no analyses by diagnosis were conducted due to the limited sample size.

Norton and Hope (2005) published the first randomized controlled trial of a 12-week transdiagnostic group treatment and found that, compared to waitlist controls, clients receiving treatment improved significantly. Roughly 67% of those receiving treatment, as compared to none of the waitlist controls, showed a reduction in diagnostic severity to subclinical levels, and significant improvement was also noted on several indices of anxiety. Unfortunately, again, the limited sample size of this study ($n = 23$) precluded analyses of outcome by diagnosis. However, in a reanalysis of the treatment data, Norton et al. (2004) also noted significant decreases in depressive symptoms and the severity of depressive disorders among those receiving treatment, despite the fact that depression was not targeted during treatment.

In a series of conference proceedings, several groups have also reported unpublished data from outcome trials (Laposa, Janeck, Erickson, & Tallman, 2003; Larkin, Waller, & Combs–Lane, 2003; Schmidt, 2003; Schmidt & Smith, 2005). For example, Larkin et al. (2003) presented preliminary data from an outcome trial of participants diagnosed with generalized anxiety disorder (GAD), social anxiety disorder, and panic disorder/agoraphobia. Data from 25 treatment completers suggested consistent reductions on self-report measures of anxiety, clinician-rated global assessment of functioning, and self-monitored anxiety and depression. Unfortunately, no control group was utilized for comparison purposes. Similarly, in his unpublished presentations, Schmidt (2003; Schmidt & Smith 2005) reported that participants with panic disorder, social anxiety disorder, and GAD showed considerable improvement compared to controls in his transdiagnostic anxiety treatment protocol. Treatment effects for panic disorder and social anxiety disorder were larger than those for GAD, although clients with GAD still showed good response. Laposa et al. (2003) evaluated the efficacy of their treatment protocol in a large multi-site randomized controlled trial by comparison to waitlist controls. Initial reports suggest that, compared to controls, participants receiving treatment evidenced a significantly larger decrease in Beck Anxiety Inventory scores from pre- to posttreatment than did waitlist controls.

Finally, Barlow, Allen, and Choate (2003) reported preliminary evidence from two initial groups, describing treatment effect sizes similar to those typically seen in diagnosis-specific treatments. Following a revision to their treatment protocol, Allen, Ehrenreich, and Barlow (2005) individually treated six clients with different anxiety and depressive disorders and noted that five of the six clients showed decreases in the severity of their primary diagnoses to subclinical levels. Data from self-report questionnaires generally supported these findings.

Overall, the published and unpublished data reported thus far converge on the conclusion that participants undertaking transdiagnostic treatment programs for anxiety disorders show significant improvement and that such change is greater than that experienced by control participants not receiving treatment. What is less clear, however, is the relative efficacy of these treatments for individuals with different anxiety disorder diagnoses. As noted above, no published trials have compared outcomes by diagnosis. The purpose of the current study was therefore to further add to the growing efficacy evidence base underlying transdiagnostic treatments for anxiety disorders using mixed-effects regression modeling analyses and to compare treatment efficacy across primary and comorbid disorders. Additionally, this study extended previous works by modeling anxiety across each session as opposed to only at pre- and posttreatment periods. It was hypothesized that participants would show a significant reduction in anxiety over the course of treatment and that treatment effects would not differ significantly by diagnosis, whether primary or comorbid.

Method

PARTICIPANTS

Participants were 52 individuals presenting for services at the University of Houston Anxiety Disorder Clinic. They were recruited for participation via advertisements and articles in local and neighborhood newspapers, referrals from health and mental health professions, and public service media announcements. The following criteria were established for inclusion in the study: (a) age 18 or older, (b) principal *DSM-IV* diagnosis of any anxiety disorder, (c) adequate proficiency in English, (d) no evidence of dementia or other neurocognitive conditions that would impair ability to provide informed consent or participate in treatment, and (e) absence of serious suicidality, substance abuse, or other conditions that would require immediate intervention.

The sample of treatment initiators consisted of 22 men and 29 women (1 unreported), and was somewhat racially diverse (51.9% Caucasian, 15.4% Hispanic/Latino[a], 5.8% African American, 3.8% Asian American, 3.8% other or mixed, and 19.2% unreported). The sample ranged in age from 19 to 71 years old, with a mean of 33.13 ($SD = 12.02$). Most were single (51.9%) or married (30.8%) and were fairly well educated (38.5% some undergraduate, 25.0% bachelor's degree or equivalent, 3.8% some professional/graduate school, 13.5% graduate/professional degree).

Participants were assigned to treatment groups based on order of presentation to the clinic, such that when the first six to eight participants had completed pretreatment assessments, they were assigned to begin group sessions together. In a small number of cases, scheduling issues required a participant to wait until a subsequent group began. No efforts were made to influence the composition of the group by diagnosis or other characteristic. In all, clients from 10 groups participated in the current study.

MEASURES

All participants received a structured diagnostic assessment at intake, the Anxiety Disorders Interview Schedule for *DSM-IV* (Brown, Di Nardo, & Barlow, 1994) and Clinician Severity Ratings for each diagnosis, and completed one self-report measure, the State-Trait Anxiety Inventory–State version (Spielberger, 1983) immediately prior to the beginning of each session.

Anxiety Disorders Interview Schedule for DSM-IV. The Anxiety Disorders Interview Schedule for DSM-IV (ADIS-IV; Brown et al., 1994) is a semistructured diagnostic interview designed to assess the presence, nature, and severity of *DSM-IV* anxiety, mood, and somatoform disorders, as well as previous mental health history. The interview also contains a brief screen for psychotic symptoms and alcohol or substance abuse. All ADIS-IV interviewers, advanced doctoral students, were trained to reliability standards by observing an an experienced interviewer and thereafter conducting at least three interviews under observation. A reliable match involved matching the experienced interviewer on diagnoses and matching the Clinician Severity Rating (see below) within 1 point for the primary diagnosis. A recent large-scale analysis of the ADIS-IV offers strong support for the reliability of diagnoses using the ADIS-IV (Brown, Di Nardo, Lehman, & Campbell, 2001).

Clinician Severity Ratings. Clinician Severity Ratings (CSRs), a component of the ADIS-IV, are subjective ratings applied by diagnosticians to quantify the degree of severity for each disorder diagnoses with the ADIS-IV. CSR range from 0 (*not at all severe*) to 8 (*extremely severe/distressing*). A CSR of 4 (*moderate impairment*) is generally considered the cutoff for a disorder of clinical significance (e.g., Heimberg et al., 1990).

State-Trait Anxiety Inventory–State version. The state form of the State-Trait Anxiety Inventory (STAI; Spielberger, Gorsuch, Luschene, Vagg, & Jacobs, 1993) is a 20-item measure designed to assess state anxiety. STAI items are scored on 1 (*not at all*) to 4 (*very much so*) scales of how much each statement indicates how the participant feels at that moment, with a total score ranging from 20 to 80. The psychometric properties of the STAI-S are strong across multiple populations (Spielberger et al., 1993), with anxiety disorder sample means ranging from 44 to 61 (see Antony, Orsillo, & Roemer, 2001), and the measure has demonstrated sensitivity to treatment effects (e.g., Fisher & Durham, 1999). At the initial time-point (Session 1), the STAI was highly internally consistent in this sample ($\alpha = .95$). The STAI was administered immediately prior to each treatment session.

PROCEDURE

Assessment and treatment were conducted at the University of Houston Anxiety Disorder Clinic. All methods and procedures were reviewed by the Institutional Review Board of the University of Houston. All potential participants underwent a brief telephone screen to provide initial evidence of suitability for the study. Potential participants who appeared to be eligible for participation were scheduled for the structured diagnostic evaluation. Following the evaluation, eligible participants were enrolled in a cognitive behavioral transdiagnostic group for anxiety. Informed consent was obtained from all participants.

Treatment protocol and therapists. Treatment consisted of 12 weekly 2-hour sessions following a manualized treatment protocol (Norton & Hope, 2002; Norton & Price, 2005). This protocol deemphasizes diagnostic labels and focuses instead on challenging and confronting feared stimuli regardless of their specific nature. Indeed, clients are encouraged to conceptualize their own network of fears, and those of the others in the group, as "an excessive or irrational fear of [blank]" rather than as, for example, "panic disorder with comorbid OCD."

Over the first nine sessions of treatment, three core ingredients of CBT were utilized: psychoeducation and self-monitoring, cognitive restructuring, and exposure to feared stimuli. Although the composition of the groups differed from diagnosis-specific CBT and typically adopted a more individualized case formulation stance, the mechanisms of action are thought to be similar to those of diagnosis-specific CBT protocols. Psychoeducation focuses on the nature of anxiety and anxiety disorders and the components of treatment and their purpose. During the first session, the concept of a fear-avoidance hierarchy is discussed, and each client develops a hierarchy with assistance from the therapists. Cognitive restructuring emphasizes identifying fear-related automatic thoughts and challenging evidence of catastrophic thinking and overestimating probabilities of negative outcomes. Exposure, which is conducted in vivo or through role-played, imaginal, or interoceptive methods, depending on client needs and the nature of the feared stimuli, is conducted in session and assigned as part of weekly

homework exercises. During the final sessions, the focus shifts from the presenting fear to the underlying perceptions of uncontrollability, unpredictability, and threat. This phase of treatment utilizes cognitive techniques to identify and challenge core beliefs regarding threat, negativity, and personal control over events. Although similar to the cognitive restructuring in the first phase of treatment, the emphasis is not on the immediate and most salient fears but rather the application of cognitive restructuring skills to general distress-producing aspects of daily life.

Therapists in this trial were doctoral-level graduate students under the supervision of the study author. All therapists were trained in the treatment protocol through video observation of previous groups and were then paired with senior graduate student co-therapists who had previously delivered the treatment. The study author directly observed all sessions for supervision purposes and to ensure treatment fidelity. The study author did not conduct any ADIS interviews or treatment sessions.

Results

PRELIMINARY ANALYSES

Of the sample of treatment initiators, 25 received a primary diagnosis of social anxiety disorder, 22 received a primary diagnosis of panic disorder with or without agoraphobia, 2 received primary diagnoses of GAD and obsessive-compulsive disorder (OCD), and 1 received a primary diagnosis of specific phobia. Over half (55.8%) of the sample were given one or more additional diagnoses, based on lower CSR scores, including GAD ($n = 13$), major depressive disorder, dysthymia, or other depressive mood disorder ($n = 11$), social anxiety disorder ($n = 6$), specific phobia ($n = 5$), panic disorder with or without agoraphobia ($n = 4$), substance abuse ($n = 2$), and ADHD ($n = 1$). Ignoring the hierarchy of principal versus comorbid diagnoses, 48.1% of the sample had clinically significant panic disorder/agoraphobia, 61.5% social anxiety disorder, 30.8% GAD, 11.5% a specific phobia, and 3.8% OCD.

Clients attended an average of 7.10 sessions ($SD = 3.31$), with a median of 8.00 and the modal number of sessions attended being 10. Number of sessions attended was unrelated to diagnosis, $F(4, 47) = 1.68$, $p = .171$. Further, no differences in CSRs, $F(4, 47) = 0.91$, $p = .464$, were observed across the primary diagnoses. Due to the limited representation of primary OCD, GAD, and specific phobia in the current sample, participants with primary diagnoses of panic disorder and social anxiety disorder were compared. Again, no difference in the number of sessions attended was found, $F(1, 45) = 0.15$, $p = .700$, nor were differences in CSR, $F(1, 45) = 0.04$, $p = .700$.

MIXED-EFFECTS REGRESSION MODELING OF CHANGE

To fully utilize the entire sample of treatment initiators, session-by-session STAI measures were examined using mixed-effect regression modeling (MRM). MRM can be conceptualized as an extension of linear regression, but with the incorporation of individual-level effects in addition to group-level effects. In essence, individual regression lines are modeled for each participant, such that their severity and change can be expressed as a combination of individual intercept and slope parameters, thereby providing estimates of both the intercept and slope of the sample as well as estimates of the average deviations of individual participants from these intercepts and slopes. Missing data are ignored, as the individual regression lines are fitted to the available longitudinal data, assuming at least two time points are available[1] (for an accessible introduction, see Hedeker, 2004). All participants attending at least two sessions were included in the sample.

Using a restricted maximum likelihood (REML) estimator,[2] the data were fitted to a random intercepts and slopes model with session-by-session STAI scores serving as a time variant regressor and primary diagnosis as a time invariant factor Table 1. First, STAI scores were modeled with only time as a predictor to establish the extent to which anxiety scores changed over the course of treatment. Results indicated that the intercept of the STAI scores (i.e., prior to Session 1) was within the clinical range, maximum likelihood estimate ($MLE = 48.01$, Wald $z = 27.89$, $p < .001$, and significantly decreasing STAI scores were observed throughout treatment, $MLE = -1.19$, Wald $z = -5.32$, $p < .001$, although a significant amount of variability was observed around both the average intercept, $MLE = 121.48$, $se = 31.37$, and slope, $MLE = 1.25$, $se = 0.49$. Furthermore, a negative slope-by-intercept correlation suggested that greater initial severity was associated with a more negative slope, $MLE = -6.21$, $se = 3.25$, $r = -.50$. Put another way, clients who were initially more severe, as measured by the STAI, showed greater improvement during treatment than did those with lower initial severity.

Given the significant average improvement and the significant variability around the intercept and slope,

[1] Primary analyses were re-run with participants attending a minimum of 3, 4, 5, and 6 sessions. Conclusions were not altered in any case.

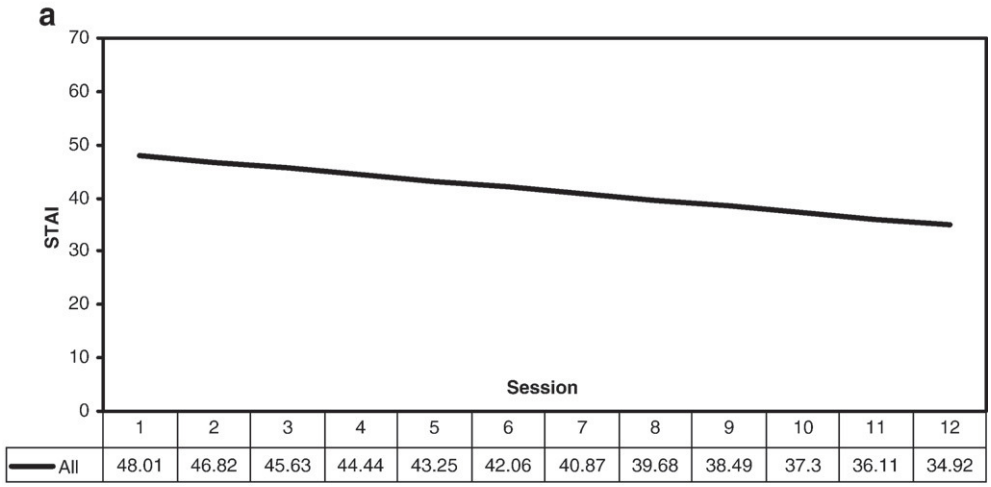
[2] Analyses were also run using the maximum likelihood (ML) estimator and no differences in results were noted.

Table 1
Fixed effect and covariance parameter estimates for each model

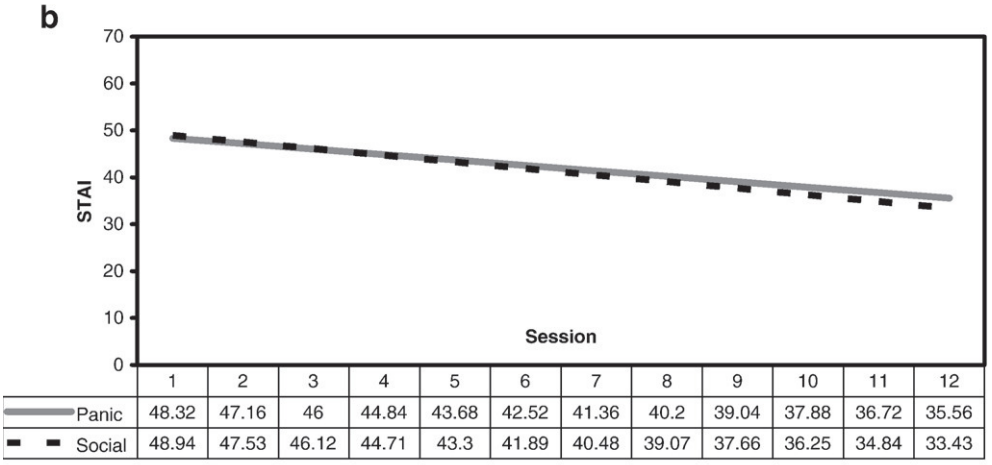
Fixed Effect Comparison	Variable	REML Estimate	*se*	*z*	*p*
Slopes as outcomes only	Intercept	48.01	1.72	27.89	<.001
	Session Slope	−1.19	0.22	−5.32	<.001
	$\sigma^2_{\upsilon_0}$	121.48	31.37		
	$\sigma_{\upsilon_0\upsilon_1}$	−6.21	3.25		
	$\sigma^2_{\upsilon_1}$	1.25	0.49		
	σ^2	55.21	4.76	Log *L*=−1345.5	
Panic disorder and social phobia	Intercept	48.32	1.85	26.17	<.001
	Session Slope	−1.16	0.23	−4.99	<.001
	Dx1 (Social vs. Panic)	0.62	1.83	.34	.737
	Dx1×Slope	−0.25	0.24	−1.06	.296
	$\sigma^2_{\upsilon_0}$	128.43	34.74		
	$\sigma_{\upsilon_0\upsilon_1}$	−6.34	3.53		
	$\sigma^2_{\upsilon_1}$	1.28	0.50		
	σ^2	52.46	4.70	Log *L*=−1228.5	
Presence/absence of clinically significant panic, social phobia, GAD, OCD, or specific phobia	Intercept	49.28	4.33	11.38	<.001
	Session Slope	−1.54	0.67	−2.29	.026
	PDA	−3.71	3.64	−1.02	.311
	Social	−3.86	3.65	−1.06	.293
	GAD	14.51	3.04	4.78	<.001
	OCD	−2.49	7.21	−0.35	.730
	SpPh	−11.74	4.55	−2.58	.014
	PDA×Session	0.55	0.61	0.90	.373
	Social×Session	0.27	0.57	0.48	.634
	GAD×Session	−0.21	0.54	−0.39	.696
	OCD×Session	1.13	1.36	0.83	.411
	SpPh×Session	−0.25	0.66	0.38	.704
	$\sigma^2_{\upsilon_0}$	76.22	23.31		
	$\sigma_{\upsilon_0\upsilon_1}$	−5.69	3.10		
	$\sigma^2_{\upsilon_1}$	1.30	0.54		
	σ^2	53.79	4.71	Log *L*=−1262.4	
Severity of clinically significant panic, social phobia, GAD OCD, or specific phobia	Intercept	44.03	3.76	11.71	<.001
	Session Slope	−1.22	0.53	−2.31	.024
	PDA	0.19	0.59	0.32	.750
	Social	0.10	0.56	0.19	.853
	GAD	2.63	0.65	4.07	<.001
	OCD	0.55	1.03	0.54	.592
	SpPh	−1.51	0.98	−1.54	.130
	PDA×Session	0.06	0.09	0.68	.502
	Social×Session	−0.04	0.08	−0.43	.666
	GAD×Session	−0.04	0.10	−0.42	.680
	OCD×Session	0.12	0.19	0.62	.538
	SpPh×Session	0.05	0.13	0.42	.675
	$\sigma^2_{\upsilon_0}$	91.68	26.04		
	$\sigma_{\upsilon_0\upsilon_1}$	−6.08	3.14		
	$\sigma^2_{\upsilon_1}$	1.34	0.54		
	σ^2	54.13	4.66	Log *L*=−1336.2	

diagnostic data were entered into the model. Due to the small sample sizes for participants with primary diagnoses of GAD, OCD, and specific phobia, analyses were run several times to ensure consistency. First, analyses were restricted only to individuals with principal diagnoses of panic disorder and social anxiety disorder to examine the extent to which improvement was differentially associated with diagnosis. Results again suggested a significant average slope, *MLE* = −1.16, Wald *z* = −4.99, *p* = .001, and that slope and intercept were negatively correlated, *MLE* = −6.34, *se* = 3.53, *r* = −.49. No significant diagnosis intercept effect was observed, *MLE* = 0.62, Wald *z* = 0.34, *p* = .737, nor was a significant Diagnosis × Time interaction, *MLE* = −0.25, Wald *z* = −1.06, *p* = .296, suggesting the participants with principal diagnoses of panic disorder and social anxiety disorder did not differ from each other in initial severity or average improvement (see Figure 1).

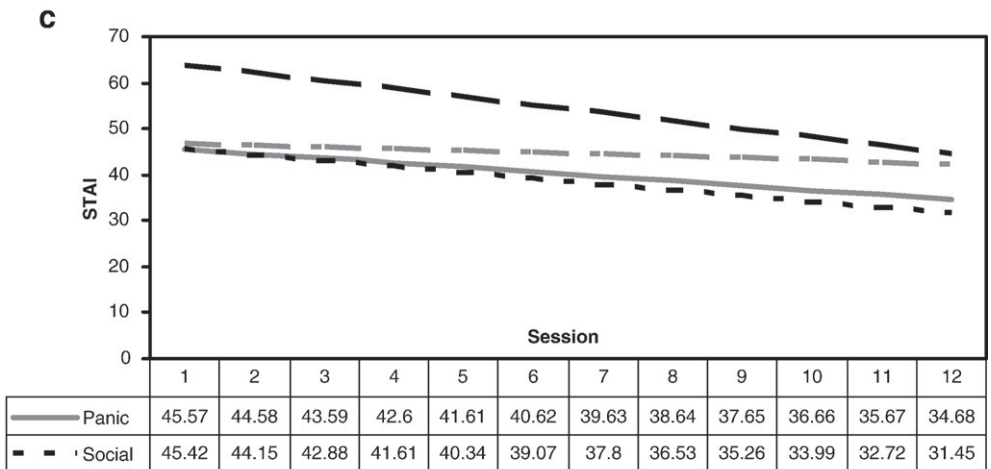
One problem associated with comparisons between individuals with different primary diagnoses is that they ignore additional comorbid disorders. Put another way, principal diagnosis analyses presuppose that, for example, someone with a principal



	1	2	3	4	5	6	7	8	9	10	11	12
All	48.01	46.82	45.63	44.44	43.25	42.06	40.87	39.68	38.49	37.3	36.11	34.92



	1	2	3	4	5	6	7	8	9	10	11	12
Panic	48.32	47.16	46	44.84	43.68	42.52	41.36	40.2	39.04	37.88	36.72	35.56
Social	48.94	47.53	46.12	44.71	43.3	41.89	40.48	39.07	37.66	36.25	34.84	33.43



	1	2	3	4	5	6	7	8	9	10	11	12
Panic	45.57	44.58	43.59	42.6	41.61	40.62	39.63	38.64	37.65	36.66	35.67	34.68
Social	45.42	44.15	42.88	41.61	40.34	39.07	37.8	36.53	35.26	33.99	32.72	31.45
GAD	63.79	62.04	60.29	58.54	56.79	55.04	53.29	51.54	49.79	48.04	46.29	44.54
OCD	46.79	46.38	45.97	45.56	45.15	44.74	44.33	43.92	43.51	43.1	42.69	42.28
SpPh	37.54	35.75	33.96	32.17	30.38	28.59	26.8	25.01	23.22	21.43	19.64	17.85

FIGURE 1 Plots of mean STAI change over sessions for (a) the entire sample, (b) primary diagnoses of panic disorder or social phobia only, and (c) the presence/absence of a diagnosis of panic disorder, social phobia, GAD, OCD, or specific phobia. Values in each table represent MRM estimated means for each session.

diagnosis of panic disorder and comorbid social anxiety disorder is categorically different from someone with a principal diagnosis of social anxiety disorder and comorbid panic disorder. Therefore, the data were reconfigured to examine all diagnoses of clinically significant severity (i.e., CSR $\geq$ 4).

Dichotomous (yes/no) variables for panic, social, OCD, GAD, and specific phobia were created along with interaction terms of each diagnosis by session. MRM revealed again a significant session effect, $MLE = -1.54$, Wald $z = -2.29$, $p = .026$, suggesting a significant average improvement over sessions, and a negative Slope × Intercept correlation, $MLE = -5.69$, $se = 3.10$, $r = -0.57$. Main effects for GAD, $MLE = 14.51$, Wald $z = 4.78$, $p < .001$, and specific phobia, $MLE = -11.74$, Wald $z = 2.58$, $p = .014$, were also observed, suggesting that individuals with any diagnosis of GAD (principal or comorbid) had a significantly higher intercept, or initial severity, whereas individuals with any specific phobia diagnosis (principal or comorbid) had a significantly lower intercept, or lower severity, than did the average participant. No other main effects were observed. Similarly, despite the two main effects, no interactions of any diagnosis by session were observed, indicating that the presence of any principal or comorbid anxiety diagnosis was not associated with differential treatment slopes.

As a final test of the possible impact of diagnosis on outcome, the previous presence/absence data were reconfigured to reflect the diagnostic severity of each principal or comorbid diagnosis, with the absence of a diagnosis being coded as "0," and interaction terms were again computed by session. As with the previous analyses, a significant session effect, $MLE = -1.22$, Wald $z = -2.31$, $p = .024$, was observed. A main effect of GAD severity, $MLE = 2.63$, Wald $z = 4.07$, $p < .001$, indicated that the severity of GAD diagnoses was associated with an intercept that was higher than the grand mean. No other main effects were observed. Again, no interactions of any diagnosis by session were observed, indicating that the presence of any diagnosis was not associated with differential treatment slopes. As before, a negative Slope × Intercept correlation was observed, $MLE = -6.08$, $se = 3.14$, $r = -.55$.

Discussion

The current study had two primary goals. The first goal was to further evaluate the efficacy of a transdiagnostic treatment for anxiety beyond previously published trials. Second, this study sought to examine the data for possible differential outcomes as a function of diagnosis. Using MRM procedures, the data showed a significant negative slope across sessions, suggesting that participants experienced a significant decline in STAI scores over time during treatment. On average, participants showed a 1.19-point decline in STAI scores per session, or an average decrease of more than 14 points over the course of the 12-week treatment. Prior to the first session, participants showed an average score of roughly 48, a score within the range of reported norms for anxiety disorder samples, whereas the posttreatment score of 34 is similar to scores reported by nonclinical samples of adults (see Antony et al., 2001). Computation of a Cohen's *d* effect size indicated an average effect of 1.06, an effect size very similar to the average effect ($d = 1.14$) previously obtained by Norton and Hope (2005).

To explore the possible effects of diagnosis on outcomes, the data were reanalyzed in a variety of ways using MRM given that OCD, GAD, and specific phobia were poorly represented as principal diagnoses. First, only participants with panic disorder and social phobia were compared. Next, distinctions between principal and comorbid diagnoses were removed, and the effects of any panic, social phobia, GAD, OCD, or specific phobia diagnosis were examined. In no case did any Diagnosis × Session interaction approach significance, suggesting that differential improvement during treatment for participants with different anxiety disorders did not occur.

In examining model fit using -2 Log Likelihood criteria, the model restricted to only panickers and social phobics showed the best fit to the data. In examining models that included all of the data, however, the model including dichotomous presence/absence of any diagnosis fit the data most closely. In this model, a significant negative overall slope indicated that, on average, STAI scores decreased over sessions. No significant Diagnosis × Session interactions were observed, suggesting that the presence or absence of any diagnosis was not associated with any differential outcome. Diagnoses of GAD were associated with higher-than-average initial STAI scores, whereas diagnoses of specific phobia were associated with lower initial STAI scores. Explanations for these effects are less clear, but several hypotheses seem tenable. First, it may well be that in this sample individuals with GAD were indeed more severe than were those without GAD, whereas those with a specific phobia were initially less severe than those without a specific phobia. However, it may also be that the STAI is particularly sensitive to symptoms associated more diffuse *anxiety*-dominant syndromes such as GAD and less sensitive to syndromes more associated with predominantly *fear*-based responses such as specific phobias.

In addition to the main effects, the covariance parameters suggested a significant correlation between intercepts and slopes, wherein participants with higher initial STAI scores tended to show more negative slopes (i.e., greater improvement) during treatment. Several explanations can be offered for the consistently robust Slope × Intercept correlation. First, it simply could be that those who were more anxious improved more than those who were

initially less anxious. Alternatively, floor effects on the STAI might have limited the amount of measurable improvement for those at a lower starting point on the measure. Finally, it is plausible that when anxiety levels decrease to an ideal range, client efforts shift to maintenance of gains whereas those clients who remain at higher levels of anxiety continue to work toward greater anxiety reduction.

Despite these encouraging findings, several limitations must be considered in evaluating the current study. First, the data were obtained during an open uncontrolled treatment trial without any follow-up assessments. Consequently, causal attributions regarding the effect of the treatment protocol cannot be directly assumed and statements of the persistence of any changes cannot be offered. However, given the positive treatment effects described by Norton and Hope (2005) and Schmidt (2003; Schmidt & Smith 2005) in comparison to no-treatment controls, it seems unlikely that the effects are simply a function of unrelated improvement over time. Still, modeling of longitudinal data from an outcomes trial employing adequate controls and blinds is necessary before firm conclusions can be offered. Furthermore, a trial comparing transdiagnostic and diagnosis-specific CBT is necessary to explore the relative efficacy of this treatment format, and subsequent studies should endeavor to collect follow-up data to examine the persistence of treatment effects.

Second, given the limited representation of participants with principal diagnoses of GAD, OCD, or specific phobia, firm conclusions about the efficacy of the treatment for individuals with these principal diagnoses cannot be made. However, in exploring *all* diagnoses of clinical severity, no differential improvement slopes were found, lending some support for the conclusion that similar improvement occurs across diagnoses during a transdiagnostic anxiety treatment. Still, given the small sample sizes, the analyses by diagnosis, even those comparing panic disorder and social phobia, may have been underpowered to detect small but possibly clinically significant differences.

Third, the use of only one outcome measure was less than optimal, as cross-validation of treatment effects using multiple and multimodal measures is ideal (Kazdin, 1992). However, the participant burden of collecting multiple measures from multiple sources prior to each session is extremely impractical in a clinical setting. Perhaps future trials could make use of simple session-by-session clinician ratings of severity to corroborate the data obtained through self-report.

Finally, the general lack of formal pre- and posttreatment assessment data is an additional limitation of the current study. Indeed, complete data across all time points would enhance confidence in the results of any outcome trial. Unfortunately, complete data are rarely, if ever, obtained. More realistically, missing data due to session absences, treatment discontinuation, and skipped assessment appointments are common. Several approaches to treating missing data have been utilized in previous studies. Completers-only analyses are commonly reported, but may yield inaccurate estimates of treatment effects, as those who discontinue (e.g., due to lack of response, anxiety reduction goals being met prior to the trial's end, or for any other reason) are omitted from the analyses. Intent-to-treat analyses carrying forward the last available data may produce similarly biased estimates of the treatment effects, particularly in trials using only pre- and posttreatment assessment data. These analyses assume that discontinuers did not change in any direction over their partial course of treatment, an assumption that is not consistent with many analyses of treatment discontinuers (e.g., Krishnamurthy, Khare, Klenck, and Norton, in preparation). Furthermore, data-carried-forward approaches may underestimate standard error terms by assuming no random or error variability over time. Maximum likelihood linear modeling methods, such as those used in the current study, or multiple data imputation analyses, are widely seen as being much more appropriate for examining clinical trials with missing data (Houck et al., 2004).

Overall, this study yielded two sets of results that held consistent across the multiple sets of analyses. First, across all analyses, a significant negative slope effect was observed indicating that, regardless of the other variables in the equations, anxiety scores decreased significantly over the course of treatment. This finding generally replicates the efficacy data from the previous randomized controlled trial of this treatment protocol (Norton & Hope, 2005), as well as the results of published trials of other transdiagnostic anxiety treatments (e.g., Erickson, 2003; Lumpkin et al., 2002). Second, despite coding and analyzing diagnostic data in a number of ways, in no case were any diagnostic variables associated with slopes of change during treatment, lending support to the hypothesis that transdiagnostic treatments for anxiety disorders are equally efficacious across diagnoses.

References

Allen, L. B., Ehrenreich, J. T., & Barlow, D. H. (2005). A unified treatment for emotional disorders: Applications with adults and adolescents. *Japanese Journal of Behavior Therapy*, *31*, 3–31.

Antony, M. M., Orsillo, S. M., & Roemer, L. (2001). *Practitioner's guide to empirically based measures of anxiety*. New York: Plenum.

Barlow, D. H. (2000). Unraveling the mysteries of anxiety and its disorders from the perspective of emotion theory. *American Psychologist*, *55*, 1247–1263.

Barlow, D. H., Allen, L. B., & Choate, M. L. (2003, November). A unified treatment protocol for the emotional disorders. In P. J. Norton (Chair), *Integrative treatment approaches across anxiety and related disorders*. Symposium presented at the annual meeting of the Anxiety Disorders Association of America, Toronto, ON.

Barlow, D. H., Allen, L. B., & Choate, M. L. (2004). Toward a unified treatment for emotional disorders. *Behavior Therapy*, *35*, 205–230.

Brown, T. A., Di Nardo, P. A., & Barlow, D. H. (1994). *Anxiety disorders interview schedule for DSM-IV (Adult Version)*. Albany, NY: Graywind.

Brown, T. A., Di Nardo, P. A., Lehman, C. L., & Campbell, L. A. (2001). Reliability of *DSM-IV* anxiety and mood disorders: Implications for the classification of emotional disorders. *Journal of Abnormal Psychology*, *110*, 49–58.

Erickson, D. H. (2003). Group cognitive behavioural therapy for heterogeneous anxiety disorders. *Cognitive Behaviour Therapy*, *32*, 179–186.

Fairburn, C. G., Cooper, Z., & Shafran, R. (2003). Cognitive behaviour therapy for eating disorders: A "transdiagnostic" theory and treatment. *Behaviour Research and Therapy*, *41*, 509–528.

Fisher, P. L., & Durham, R. C. (1999). Recovery rates in generalized anxiety disorder following psychological therapy: An analysis of clinically significant change in the STAI-T across outcome studies since 1990. *Psychological Medicine*, *29*, 1425–1434.

Hedeker, D. (2004). An introduction to growth modeling. In D. Kaplan (Chair), *The Sage handbook of quantitative methodology for the social sciences* (pp. 215–234). Thousand Oaks, CA: Sage.

Heimberg, R. G., Dodge, C. S., Hope, D. A., Kennedy, C. R., Zollo, L. J., & Becker, R. E. (1990). Cognitive behavioral group treatment for social phobia: Comparison with a credible placebo. *Cognitive Therapy and Research*, *14*, 1–23.

Houck, P., Mazumdar, S., Koru-Sengul, T., Tang, G., Mulsant, B., Pollock, B., & Reynolds III, C. (2004). Estimating treatment effects from longitudinal clinical trial data with missing values: Comparative analyses using different methods. *Psychiatry Research*, *129*, 209–215.

Kazdin, A. E. (1992). *Research design in clinical psychology* (2nd ed.). Boston: Allyn and Bacon.

Laposa, J., Janeck, A., Erickson, D., & Tallman, K. (2003). *Group CBT for heterogeneous anxiety disorders: Preliminary evidence for effectiveness*. Presentation to the 31st annual convention of the British Association for Behavioural and Cognitive Psychotherapies, York, England.

Larkin, K. T., Waller, S., & Combs-Lane, A. (2003, March). Anxiety management group therapy for multiple anxiety disorder diagnoses. In P. J. Norton (Chair), *Integrative treatment approaches across anxiety and related disorders*. Symposium presented at the annual meeting of the Anxiety Disorders Association of America, Toronto, ON.

Lumpkin, P. W., Silverman, W. K., Weems, C. F., Markham, M. R., & Kurtines, W. M. (2002). Treating a heterogeneous set of anxiety disorders in youth with group cognitive behavioral therapy: A partially nonconcurrent multiple-baseline evaluation. *Behavior Therapy*, *33*, 163–177.

Norton, P. J. (2006). Toward a clinically oriented model of anxiety disorders. *Cognitive Behaviour Therapy*, *35*, 88–105.

Norton, P. J. (in press). Integrated psychological treatment of multiple anxiety disorders. In M.M. Antony & M.B. Stein (Eds.), *Handbook of anxiety and anxiety disorders*. New York: Oxford.

Norton, P. J., Hayes, S. A., & Hope, D. A. (2004). Effects of a transdiagnostic group treatment for anxiety on secondary depressive disorders. *Depression and Anxiety*, *20*, 198–202.

Norton, P. J., & Hope, D. A. (2002). *Anxiety treatment program: Therapist manual*. Unpublished treatment manual.

Norton, P. J., & Hope, D. A. (2005). Preliminary evaluation of a broad-spectrum cognitive-behavioral group therapy for anxiety. *Journal of Behavior Therapy and Experimental Psychiatry*, *36*, 79–97.

Norton, P. J., & Price, E. P. (2005). *Anxiety treatment program: Client manual*. Unpublished treatment manual.

Schmidt, N. B. (2003, November). Unified CBT for anxiety: Preliminary findings from False Safety Behavior Elimination Therapy (F-SET). In D. F. Tolin (Chair), *Increasing the cost-effectiveness and user-friendliness of cognitive behavior therapy for anxiety disorders*. Symposium presented at the 37th annual convention of the Association for Advancement of Behavior Therapy, Boston.

Schmidt, N. B., & Smith, J. D. (2005, March). Unified CBT for anxiety: Preliminary findings from False Safety Behavior Elimination Therapy (F-SET). In P. J. Norton (Chair), *Transdiagnostic issues across anxiety and mood disorders*. Symposium presented at the annual meeting of the Anxiety Disorders Association of America, Seattle.

Spielberger, C. D. (1983). *Manual for the State-Trait Anxiety Inventory (STAI)*. Palo Alto: Consulting Psychologists Press.

Spielberger, C. D., Gorsuch, R. L., Luschene, R. E., Vagg, P. R., & Jacobs, G. A. (1993). *State-Trait Anxiety Inventory for adults*. Palo Alto: Mind Garden.

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