A Meta-Analytic Review of Adult Cognitive-Behavioral Treatment Outcome Across the Anxiety Disorders

Peter J. Norton, PhD, and Esther C. Price, MA

Abstract: The efficacy of cognitive behavioral treatments (CBT) for anxiety in adults has been supported by multiple meta-analyses. However, most have focused on only 1 diagnosis, thereby disallowing diagnostic comparisons. This study examined the efficacy of CBT across the anxiety disorders. One hundred eight trials of CBT for an anxiety disorder met study criteria. Cognitive therapy and exposure therapy alone, in combination, or combined with relaxation training, were efficacious across the anxiety disorders, with no differential efficacy for any treatment components for any specific diagnoses. However, when comparing across diagnoses, outcomes for generalized anxiety disorder and posttraumatic stress disorder were superior to those for social anxiety disorder, but no other differences emerged. CBT effects were superior to those for no-treatment and expectancy control treatments, although tentative evidence suggested equal effects of CBT when compared with relaxation-only treatments.

Key Words: Meta-analysis, anxiety disorder, treatment outcome, CBT.

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According to a number of sources (Barlow and Lehman, 1996; Chambless and Gillis, 1993; Craske, 1999), encouraging efficacy data have been obtained across the range of cognitive-behavioral treatment (CBT) protocols for anxiety disorders. Several meta-analyses of anxiety disorder treatment outcome studies support these assertions, showing strong treatment effects of CBT for panic disorder and agoraphobia (Bakker et al., 1998; Clum et al., 1993; Gould et al., 1995; van Balkom et al., 1997), social anxiety disorder (Fedoroff and Taylor, 2001; Feske and Chambless, 1995; Taylor, 1996), obsessive-compulsive disorder (OCD; Abramowitz, 1997; Kobak et al., 1998; van Balkom et al., 1994), generalized anxiety disorder (GAD; Borkovec and Ruscio, 2001; Chambless and Gillis, 1993; Gould et al., 1997), and posttraumatic stress disorder (PTSD; Bradley et al., 2005; Sherman, 1998; Van Etten and Taylor, 1998). (For a detailed review of anxiety disorder meta-analyses, see Deacon and Abramowitz, 2004.)

For the treatment of panic disorder with or without agoraphobia, Gould et al. (1995), Clum et al. (1993), and van Balkom et al. (1997) reported that CBT produced significant posttreatment improvement compared with respective control groups. Treatment effect maintenance data (minimum 6-month follow-up) are also supportive, with Gould et al. and Bakker et al. describing stable maintenance of treatment effects following CBT. Meta-analyses of social anxiety disorder (Fedoroff and Taylor, 2001; Feske and Chambless, 1995; Taylor, 1996) also support the immediate and long-term efficacy of behavioral and CBTs, particularly cognitive therapy (CT) plus exposure.

Abramowitz (1997) reported a meta-analytic review of OCD psychotherapy outcome studies. Results indicated that exposure with response prevention (E/RP) was significantly more effective than relaxation (RLX) training; however, no significant differences were noted between E/RP and CT, nor between E/RP and either exposure-only or response prevention only. Kobak et al. (1998) and van Balkom et al. (1994) provided strong support for the effectiveness of E/RP for OCD. Borkovec and Ruscio (2001), Chambless and Gillis (1993), and Gould et al. (1997) all provided analyses of clinical trials for GAD. These studies showed strong post-treatment and follow-up effects for CBT when compared with various control conditions. Gould et al. (1997) also reported the superiority of CBT over RLX and biofeedback. Gould et al. (1997) also compared psychological and pharmacological treatment effects on GAD and observed a slight advantage of CBT over pharmacological approaches in the short-term.

Meta-analyses of PTSD treatments have been provided by Van Etten and Taylor (1998), Sherman (1998), and Bradley et al., (2005). Van Etten and Taylor suggested that behavior therapy and eye movement desensitization and reprocessing therapy (EMDR) were equally efficacious treatments, with a possible advantage favoring behavior therapy. EMDR and behavior therapy both showed evidence for maintenance of treatment gains. Similarly, Bradley et al. (2005) reported substantial improvement for individuals receiving CBT or EMDR, and noted that such improvement was significantly superior to that observed following waitlist or supportive control participants. Bradley et al., however, noted no superiority of CBT or EMDR over each other. Finally, Sherman (1998) found similar efficacy support. The majority of the studies employed a cognitive-behavioral or behav-

Department of Psychology, University of Houston, Houston, Texas.
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Send reprint requests to Peter J. Norton, PhD, Department of Psychology 126 Heyne Building, University of Houston, Houston, TX 77204-5022.

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ioral approach, such as flooding, systematic desensitization, and CT, or EMDR. A significant effect was noted posttreatment, as well as at follow-up, which ranged from 3 months to 2 years.

Implications From Anxiety Treatment Meta-Analyses

Most, albeit not all (Gould et al., 1995; Kobak et al., 1998), of the aforementioned meta-analyses have noted that cognitive and behavioral treatments show positive therapeutic effectiveness that is superior, or at least equivalent, to other treatment modalities. However, the varied inclusion and exclusion criteria, methods of estimating effect sizes, and different meta-analytic strategies used, do not allow for direct comparison of effect size estimates *across* meta-analyses. In partial response to this, Westen and Morrison (2001) conducted a meta-analytic review of the efficacy of empirically supported treatments for major depressive disorder (MDD), panic disorder, and GAD. Although concerns have been raised regarding their methodology and conclusions, such as the inclusion criteria and the measures from which effect sizes were drawn (Aikins et al., 2001), Westen and Morrison reported very high pre- to posttreatment average effect sizes for each diagnostic group. Although the pre- to posttreatment effects sizes for MDD and GAD appeared larger than that for panic disorder, these estimates were not statistically compared. Interestingly, despite panic disorder having a lower effect size, percent responder rates were much higher for panic disorder than for MDD or GAD. Panic disorder was associated with sustained response to treatment whereas MDD and GAD were not.

Westen and Morrison's (2001) work aside, the general lack of comparative outcome analyses between diagnoses has not quelled beliefs about the responsiveness of certain disorders, particularly GAD and PTSD, to CBT (Brown et al., 1994; Norton et al., 2000). Further, questions have been raised as to whether specific cognitive or behavioral techniques may be differentially indicated for specific disorders (Deacon and Abramowitz, 2004). GAD, for example, had traditionally been treated using RLX and cognitive techniques, while exposure to worry (Borkovec and Costello, 1993; Craske et al., 1992) or intolerance of uncertainty (Ladouceur et al., 2000) have been applied more recently to treatment programs. Conversely, treatments for OCD have been almost exclusively exposure based, with cognitive models and treatments only recently beginning to receive recognition (van Oppen et al., 1995). The value of adding RLX training to CBTs for anxiety disorders has been contested (De Ruiter et al., 1989), with some suggestions that it may be contraindicated for panic disorder (Schmidt et al., 2000). Apart from a few well-conducted comparative outcome studies, however, little evidence exists to suggest whether cognitive and exposure therapy (EXP), alone or in combination, and with or without RLX training, are differentially indicated among the anxiety disorders.

Exploration of the relative efficacy of cognitive and behavioral techniques may not only help identify the optimal treatment approaches for each diagnosis, but may also provide suggestions about alternate nosological structures for anxiety disorders. Indeed, similar response to similar treatments has been a strong argument underlying calls for the consideration of clustering anxiety and other emotional disorders as a single pathology (Barlow et al., 2004; Erickson, 2003; Norton, 2006; Norton and Hope, 2005). However, these arguments have been based on assumptions of comparable response, as opposed to empirical demonstrations. The purpose of this study was therefore to examine the efficacy of EXP, CT, or their combination across the anxiety disorders using meta-analytic methods. Two primary questions were examined to explore current gaps of knowledge: (1) Is the efficacy of CBT, broadly defined as CT, exposure, or their combination, comparable across the anxiety disorders? and (2) Are specific treatment components (e.g., exposure, cognitive restructuring, etc.) or combinations of components particularly indicated or contraindicated for specific anxiety disorder diagnoses?

METHODS

Inclusion and Exclusion Criteria

Studies included for the meta-analysis were randomized clinical trials (RCTs) for adults with any anxiety disorder, excepting specific phobia. We could not ensure complete coverage of all specific phobia treatment studies because of the variety of terminology used, such as snake phobia, ophidiophobia, fear of snakes, or snake anxiety. Dissertations that were not published were not included.

To ensure the quality of the studies and the treatments delivered, all potential studies were required to meet several specific criteria: (1) published in a peer-reviewed journal, (2) completely random assignment of clients to conditions, (3) use of at least 1 control or comparison condition, (4) diagnoses strictly based on DSM-III-R (American Psychiatric Association, 1987) or DSM-IV (American Psychiatric Association, 1994; 2000) criteria, (5) attempts made to maintain or assess experimental blinds and treatment fidelity, and (6) use of valid outcome measures with means and standard deviations for each of the conditions. Given the variability in the quality and appropriateness of outcome measures for different diagnoses, effect sizes were computed only from established measures for each disorder. Established measures were identified as those described in extended format by Antony et al., (2001), and effects from these measures were included only if the measure was appropriate for the diagnostic group being treated. Antony et al. (2001) provided a careful review of measures of anxiety and each anxiety disorder that (a) held a sound psychometric evidence-base, (b) were in relatively regular use for clinical or research purposes, and (c) held some degree of clinical utility. Several measures were identified as assessing a more general anxiety construct that was applicable across diagnoses. In addition, several measures were identified as assessing features or constructs considered to be specific to a diagnosis. Effect sizes were computed from both the general anxiety measures, as well as from the disorder-specific measures only if they were appropriate for the diagnostic groups being treated. For example, effect sizes for a panic disorder treatment study would be generated from general anxiety measures (e.g., Beck Anxiety Inventory) and

panic-specific measures (e.g., Anxiety Sensitivity Index), but not from unrelated disorder-specific measures if any (e.g., Penn State Worry Questionnaire).

All studies were required to have at least 1 treatment condition that could be broadly defined as CT, EXP, or some combination of CT, EXP, and RLX. Studies including RLX training in conjunction with EXP or CT were also included, although RLX-only trials were not included as they were not considered CBT. Studies were included only if they explicitly stated that DSM-III-R or DSM-IV criteria were used for diagnosis. Studies using DSM-III (American Psychiatric Association, 1980) and earlier criteria were excluded because of the many changes in anxiety disorder diagnostic criteria from DSM-III to DSM-III-R. To avoid any studies being missed owing to a lag in the study appearing in electronic databases, only studies published through the end of 2005 were included. Five studies were excluded on the basis of these criteria: 1 only presented results graphically, another presented only data pooled across conditions, 2 did not present any indices of variability, and 1 pooled data across multiple measures. Efforts were made to contact the authors of these studies, but either (1) more amenable data were not available from the authors or (2) we were unable to contact any of the authors.

Potentially eligible studies were identified from previous meta-analyses of specific anxiety disorder treatments, searches through recent review papers and chapters, and searches of multiple electronic reference databases using a liberal set of search terms {PsycINFO and Medline search parameters: [(anxiety) or (panic) or (phobia) or (stress disorder) or (obsessive) or (compulsive)] and [(cognitive) or (behavioral) or (CBT) or (exposure)] and [(treatment) or (therapy)[and (random*)}. The authors reviewed all potential studies to ensure that they met inclusion and exclusion criteria. Within each diagnostic group, a list of all included studies was sent to an established researcher of that diagnosis to review for possible missed outcome studies. Missed studies, if any, were then obtained, reviewed, and coded for inclusion in the meta-analysis.

All studies were reviewed and coded by the lead author, while roughly 30% of the studies were also coded by the second author to ensure reliability in coding. Although infrequent, discrepancies were discussed until a consensus was achieved. Studies were coded for study characteristics (e.g., research design), sample characteristics (e.g., demographics, diagnosis, medication status), treatment characteristics (e.g., specific treatment components used, treatment intensity and duration), and treatment effects.

Coding of treatment components followed a rigorous examination of the description of the treatment in each study and/or any ancillary cited treatment manuals. RLX was defined as any active technique specifically designed to immediately reduce physiological arousal, typically progressive or passive muscle RLX, biofeedback, or breathing retraining. CT was defined as any verbal or behavioral strategy designed to directly identify and challenge erroneous or maladaptive beliefs or assumptions regarding the client's fears. Typical strategies included Socratic questioning, cognitive or behavioral experiments, or other techniques such as pie charting. Finally, behavior therapy was defined as behavioral techniques used to promote habituation or extinction or fear responses. As many treatment components, such as behavioral experiments and in vivo exposure, are similar in form but differ in intended function, caution was used in coding treatment components. In general, we relied on authors' description of the intent of each treatment component; for example, whether the component was used primarily for cognitive change or habituation. Most of the studies reviewed were very clear in their explanations of which components were used, and why. In the few cases where the intent of a treatment component was in question, we either contacted the study authors for clarification or used ancillary information such as the number of times an exposure/behavioral experiment was repeated (single experiment to highlight discrepancy between beliefs and actual events versus repeated exposure to promote habituation) to form a best guess. The authors consulted on coding the few ambiguous treatments. Finally, the first author re-reviewed all studies and completed coding forms to ensure consistency in the coding of treatment components.

Effect Size Calculation and Statistical Procedures

Cohen *d* (1988) standardized mean change effect sizes and variance weights were computed for each reported treatment or control condition, using bias corrections provided by Morris (2000). Where available, intent-to-treat data were used over completer data.

$$g = \frac{(Mean_{pre} - Mean_{post})}{SD_{pre}}$$

As effect size estimates tend to be upwardly biased with small samples (Hedges, 1981), the effect size estimates were corrected for this bias using Hedges' (1981) adjustment:

$$d = \left(1 - \frac{3}{4(n-1) - 1}\right)g$$

The variance of each effect size was estimated using the following formula, where δ was estimated using published estimates of the test-retest correlation for each measure:

$$\sigma_d^2 = \frac{2(1-\rho)}{n} + \frac{\delta^2}{2(n-1)}$$

Weighted effect sizes were computed from pre- to posttreatment, and posttreatment to follow-up, as opposed to posttreatment and follow-up comparisons with control groups. This approach was selected for 2 reasons. First, many studies, such as comparative outcome trials, did not use any no-treatment control conditions. Rather, a sizable proportion of the studies reviewed used some form of placebo or alternate treatment as a comparison, thereby attenuating the magnitude of their effect sizes. Conversely, only utilizing studies with no-treatment controls would have required excluding the

many studies without such controls. Second, while recognizing that pre to post effect sizes are influenced by both technique-specific and nonspecific treatment effects, the purpose of this meta-analysis was to examine the effects of these treatment packages *as delivered*, not to tease apart the relative contributions of nonspecific and technique-specific treatment elements. Further, given that different studies used different standards for control group (i.e., no treatment, pill or attention placebo, supportive therapy, etc.) subsequent analyses would need to be restricted to each subset, such as examinations of treatment versus wait-list control condition effect sizes followed by examinations of treatment versus supportive control condition effect sizes (Bradley et al., 2005). This would undoubtedly impact the statistical power of the analyses, and increase the frequency of unobserved treatment component by diagnosis cells.

One concern when conducting meta-analyses is that the average effect size may be artificially inflated due to the "file drawer" problem (Rosenthal, 1979). This problem is based on the proposition that studies obtaining null results are less likely to be published, while those with large effects are more likely to be published. The file drawer problem can be statistically estimated, however, as the number of studies with zero effect sizes that would be required to reduce the average effect to a "trivial" value. Although definitions of a trivial value vary (0.20 in Orwin, 1983; 0.05 in Van Etten and Taylor, 1998), we selected a trivial effect size to match the average effect size of the no-treatment conditions in the studies used for this meta-analysis (pre to post $d = 0.25$).

All effect size estimates were screened for outlying values, with outliers being defined as values 1.5 times the interquartile range beyond the upper and lower quartiles (Hoaglin et al., 1983). One CBT study showed extremely high outlying values (Biswas et al., 1995) across multiple measures and multiple distinct conditions. This study was removed from the dataset. Two control conditions (defined below) also showed outlying values ($d = 3.77$ and 4.60 in Bond et al., 2002 and Borkovec and Costello, 1993, respectively). Interestingly, participants in this condition received both nonspecific psychotherapy and pill placebo, which may suggest an additive expectancy effect. This condition was removed from subsequent analyses.

File-drawer analyses suggested that the observed CBT effects would not be likely to have been artificially inflated because of publication biases. The Orwin (1983) formula for determining the failsafe *n* was used:

$$k_0 = k_1\left(\frac{ES_k}{ES_c} - 1\right)$$

Result suggested that over 915 additional CBT conditions showing null effects would be required to reduce the observed CBT effects to the predetermined trivial level.

Following removal of outliers, a total of 108 treatment studies, described in 119 published articles, and accounting for 291 distinct conditions, were identified as meeting inclusion and exclusion criteria (Appendix). These conditions had an average sample size of 24.84 ($SD = 16.41$), while each treatment study had an average sample size of 72.92 ($SD = 72.06$). Treatment conditions were coded as CT-only ($n = 26$), EXP only ($n = 54$), CT+EXP ($n = 39$), CT+RLX ($n = 5$), EXP+RLX ($n = 11$), or CT+EXP+RLX ($n = 58$). For the comparison conditions 45 were coded as no-treatment or delayed treatment, 14 as RLX-only, 15 as medication, 20 as expectancy control (e.g., attentional or pill placebo, supportive therapy), 17 combined medication and CBT, and 6 psychotherapies not classifiable as CT, EXP, CBT, RLX, or attention placebo. Of the 119 outcome studies, 34 were for the treatment of panic disorder with or without agoraphobia (92 conditions), 17 for social anxiety disorder (51 conditions), 14 for OCD (38 conditions), 17 for GAD (45 conditions), and 23 for PTSD (58 conditions). Individual treatment (58.1%) was more commonly used than group (16.2%) or other or mixed format treatments (2.7%), although a surprising number (23.0%) failed to report treatment format. The average age of the participants was 37.98 ($SD = 6.93$) and 64.79% were women. Six conditions (Marks et al., 2004; Norton and Hope, 2005; Schneider et al., 2005) were identified as "transdiagnostic," in that 2 or more diagnostic groups were included in the treatment. Treatment effects were potent ($d_{\text{prepost}} = 2.62$, $d_{\text{postfollow-up}} = 0.37$); however, because of their heterogeneous nature and small number, these studies were not included in subsequent analyses.

The effect sizes were next tested for homogeneity using the Hedges and Olkin (1985) formula.

$$Q = \sum w_i(ES_i - ES)^2$$

Results indicated that the effect sizes of pre- to posttreatment change, $Q\ (291) = 50.01$, $p > 0.05$, as well as the effect sizes of posttreatment to follow-up change, $Q\ (148) = 0.70$, $p > 0.05$, each estimate from the same populations of effect sizes. Data were examined to ensure no violations of normality, and were analyzed using a weighted least squares model.

In addition to computing effect sizes for all CBT treatments, average effect sizes were computed for those conditions that involved *no treatment* (e.g., no treatment, waitlist control, etc.; $d = 0.250$), *expectancy control* (e.g., pill placebo, nonspecific treatment, attentional placebo, etc.; $d = 1.04$), and RLX-only ($d = 1.60$). Expectancy control conditions were specifically defined and coded as those conditions that were designed to account for the elements common to that treatment modality, but do not have any active pharmacologic (e.g., pill placebo) or cognitive-behavioral components (supportive listening, attentional placebo, etc.). It must be noted that these effect size estimates were not derived from *all* no-treatment, expectancy control, and RLX conditions for anxiety, but rather only those from anxiety RCTs that also included an EXP, CT, or CBT condition. Furthermore, these estimates were obtained from a small number of conditions; therefore, these values will be used as a cautious "best guess" estimate of no-treatment, expectancy control, and RLX effects.

RESULTS

Treatment Effects

Table 1 presents mean weighted effect sizes for each diagnostic group by treatment type for those treatments using CT, EXP, or any combination of CT, EXP, and RLX. For pre- to post treatment, effect sizes were obtained from 169 separate treatment conditions, whereas 109 posttreatment to follow-up effect sizes were obtained. When examining pre- to posttreatment change no main effect of treatment component [$F(5,146) = 0.77$, $p = 0.57$] or interaction of treatment component by diagnosis were observed [$F(13,146) = 1.03$, $p = 0.43$]. However, a marginally significant main effect of diagnosis was noted [$F(4,146) = 2.39$, $p = 0.05$]. Simple effects analysis of the main effect indicated that higher weighted effect sizes were obtained from treatments for GAD and PTSD, than from treatments for social anxiety disorder. No other diagnostic groups differed significantly. Maintenance of gains from post to follow-up did not differ by diagnosis [$F(4,87) = 0.39$, $p = 0.81$] or treatment component [$F(5,87) = 0.53$, $p = 0.75$], and no interaction was observed [$F(12,87) = 0.57$, $p = 0.87$; Table 1].

CBT Versus Control Conditions

We next sought to compare the average weighted effects of CBT and its components to the effect size estimates obtained from the various control conditions. However, these analyses should be interpreted with caution, as the best guess effect size estimates are not derived from the entire population of no treatment and expectancy control conditions; rather, they represent only those included in trials that also included at least 1 condition of CT and/or EXP. Therefore, the control condition effect size estimates may be biased or otherwise not be representative of the true effect.

For pre- to posttreatment, weighted effect sizes for CBT were significantly greater than the weighted no-treatment effects sizes [$F(1,203) = 74.39$, $p < 0.001$], and this main effect did not vary by diagnosis [$F(4,203) = 0.63$, $p = 0.64$]. In addition, pre- to posttreatment weighted effect sizes for CBT were significantly greater than the weighted expectancy control effects sizes [$F(1,179) = 8.69$, $p < 0.01$], and this main effect did not vary by diagnosis [$F(4,179) = 1.66$, $p = 0.16$]. Finally, when comparing CBT to RLX-only, no main effect of treatment type was observed [$F(1,172) = 0.03$, $p = 0.87$], although the interaction of treatment type by diagnosis [$F(3,172) = 2.49$, $p = 0.06$] was nearly significant. This nonsignificant interaction suggested that CBT tended to be associated with higher weighted effects sizes for OCD than did RLX, although RLX-only was observed in only 1 treatment condition for OCD. In addition, RLX-only was not observed in any included trial for social phobia, and was observed only once in trials for PTSD. Indeed, while the nonsignificant main effect suggests no benefit of CBT over

TABLE 1. Average Weighted Effect Size by Diagnosis and Treatment Components

	Primary Diagnosis Treated					
	PD/A	**SAD**	**OCD**	**GAD**	**PTSD**	**Overall**
CT						
Pre–Post	1.37 (6)	1.03 (3)	1.16 (7)	2.06 (6)	2.23 (3)	1.67
Post–FU	0.08 (5)	0.21 (3)	0.08 (3)	0.29 (5)	0.54 (3)	0.22
EXP						
Pre–Post	0.97 (10)	1.53 (9)	1.39 (14)		2.03 (9)	1.48
Post–FU	0.29 (5)	0.23 (5)	0.11 (5)		0.23 (7)	0.20
RLX + CT						
Pre–Post	0.72 (1)			2.08 (2)		1.14
Post–FU	0.40 (1)					0.40
RLX + EXP						
Pre–Post	2.11 (2)		1.68 (1)	1.72 (2)	2.04 (5)	1.98
Post–FU	−0.05 (1)		0.04 (1)	0.11 (2)	0.27 (4)	0.19
CT + EXP						
Pre–Post	1.97 (11)	1.16 (19)	2.03 (3)	2.02 (3)	1.74 (3)	1.56
Post–FU	0.23 (5)	0.19 (15)	0.12 (2)	0.17 (1)	−0.10 (2)	0.15
RLX + CT + EXP						
Pre–Post	1.52 (28)			1.54 (9)	1.63 (13)	1.55
Post–FU	0.07 (17)			0.19 (7)	0.20 (10)	0.12
Any						
Pre–Post	1.53[a,b]	1.27[b]	1.50[a,b]	1.80[a]	1.86[a]	1.58
Post–FU	0.12	0.20	0.10	0.21	0.22	0.17

Reported average effect sizes derived only from established measures identified in Antony et al. (2001) for each anxiety diagnosis and are weighted by sample size.

Means with different superscripts (e.g., [a,b]) differ significantly ($p < .05$).

Values in parentheses represent number of conditions contributing to the mean.

CT indicates cognitive restructuring techniques; EXP, exposure techniques; RLX, relaxation training.

RLX, it must be interpreted with caution because of the limited number of RLX-only conditions ($n = 12$). No main effects or interactions were observed in comparison to any control condition for posttreatment to follow-up weighted effect sizes.

Predictors of Treatment Effects

Treatment effects were next included in a series of WLS simple regressions to explore whether any treatment (number, frequency, and duration of sessions, group versus individual), sample (average age), or study variables (publication year) significantly predicted CBT treatment outcome. Somewhat surprisingly, no variables were significantly related to pre- to posttreatment outcome [$R^2 = 0.056$, $F = 1.45$, $p = 0.19$] or post to follow-up change [$R^2 = 0.055$, $F = 1.33$, $p = 0.24$].

DISCUSSION

Consistent with the results of the aforementioned diagnosis-specific meta-analyses (Abramowitz, 1997; Bakker et al., 1998; Borkovec and Ruscio, 2001; Bradley et al., 2005; Chambless and Gillis, 1993; Fedoroff and Taylor, 2001; Feske and Chambless, 1995; Gould et al., 1995, 1997; Kobak et al., 1998; Sherman, 1998; Taylor, 1996; van Balkom et al., 1994, 1997; Van Etten and Taylor, 1998; Westen and Morrison, 2001), the current meta-analysis provided evidence supporting the efficacy of CBT techniques with the anxiety disorders. More specifically, treatments using CBT techniques showed significantly larger treatment effect sizes than no-treatment or placebo across all of the anxiety disorders. Moreover, CBT treatment effects were larger with GAD and PTSD than for social anxiety disorder, although strong treatment effects were observed across all diagnoses.

Several somewhat intriguing sets of results were observed. First, no differences were observed in treatment effect sizes across the various combinations of CBT treatment components. Furthermore, no interaction of treatment component and diagnosis was observed, suggesting that the lack of differences in effect size across treatment components held consistent across diagnoses. This point must be considered in light of the fact that several treatment components, and combinations thereof, were not used for certain diagnoses in any of the randomized controlled trials included in this study. Most notably, CT+RLX, EXP+RLX, and CT+EXP+RLX were not included in any of the trials for social anxiety disorder or OCD. CT+RLX was also not used for any of the PTSD trials, while EXP-only was not used for any of the GAD trials. Therefore, the comparability of treatment effects for these combinations with these diagnoses should not be assumed. Future well-conducted trials should examine the efficacy of these under-studied treatment component combinations for these diagnoses, as important treatment advances have resulted from the incorporation of novel CBT elements for diagnoses traditionally treated using other CBT components (van Oppen et al., 1995; Salkovskis, 1996).

Second, treatment effects for CBT did not differ from the estimate of RLX-alone effects, with the possible exception that CBT effects were stronger that the effects of the one included RLX-only condition for OCD. This may provide support for comprehensive RLX training as an alternative treatment option. In fact, the estimated effect size for RLX only may *underestimate* the true effect, as RLX training was almost always employed in the reviewed studies as a control or alternative treatment. Given the well-demonstrated allegiance effects in treatment outcome research (Luborsky et al., 1999), differences may exist between studies examining RLX only as a primary treatment versus those including RLX only as a control or comparison condition. However, these results should be interpreted with caution because an exhaustive search for studies exploring the efficacy of RLX only in the treatment of anxiety disorders was not conducted. The studies included herein were only those that contained a component of CBT such as CT or EXP. Moreover, not all diagnoses were adequately represented in the RLX-only condition. Even so, these data do suggest a re-examination of the comparative efficacy of comprehensive RLX programs and CBT for the treatment of anxiety disorders.

Finally, and somewhat surprisingly, results suggested that across CBT components, weighted effect sizes were significantly larger for treatment of PTSD and GAD, than for social anxiety disorder. The superior efficacy for PTSD and GAD was unexpected in that it has been inferred that treatment efficacy for these disorders has lagged behind that of the other anxiety disorders (Brown et al., 1994; Norton et al., 2000) while CBT approaches for social anxiety disorder are typically considered to be well established. It may be that the large effect size for GAD may not be reflective of response rates to CBT, as Westen and Morrison (2001) reported higher effect sizes for GAD treatment than for panic disorder, but much lower response rates. It is unknown whether a similar phenomenon is occurring for PTSD. Still, we must consider that the ratio of symptom reduction and treatment response may be incongruous across the anxiety disorders. Similarly, it must also be considered that measures specific to different disorders, such as GAD and PTSD, may be systemically more sensitive to treatment effects than those for other disorders. Indeed, measures for GAD and PTSD may be, on average, more sensitive to change, show less error variance, or oversample symptoms or features that are particularly amenable to change. This possibility seems less likely, however, as effect sizes were derived from both disorder-specific outcome measures as well as common established measures such as the Beck Anxiety Inventory or Hamilton Anxiety Rating Scale. Unfortunately, no single measure was used with enough frequency across diagnoses and treatments to allow for re-analysis of the studies using a common outcome measure.

One of the largest concerns when conducting meta-analytic research lies in variance in the quality of included research studies. While we selected studies that met our inclusion criteria (RCT, DSM-III-R or newer diagnostic criteria, adult-only, etc.), and many of these inclusion criteria are Delphi committee indicators of high quality studies (Verhagen et al., 1998), there still remains some variability in the quality of studies used and the quality of the study reporting. More specifically, there was considerable variability in reporting of important variables such as attrition rates, ethnicity

of participants, or treatment duration and frequency. Additionally, the strict adherence to our inclusion and exclusion criteria may have resulted in an over-sampling of trials that reported significant and large treatment effects. Consequently, there is a possibility that the effect sizes may be exaggerated, although file-drawer analyses indicate that a substantial number of unpublished studies would be needed to nullify these results.

Other limitations of this study are tied to the stringent inclusion and exclusion criteria determined a priori. Most notably, the decision to include only studies that explicitly stated that patients met DSM-III-R or IV diagnostic criteria resulted in a number of published and well-conceived studies to be excluded. This was particularly evident in outcome studies for PTSD where numerous studies had less-stringent diagnostic inclusion criteria, [e.g., "PTSD or posttraumatic stress symptoms coupled with a clear Criterion A trauma link" (Krakow et al., 2000, p. 592)]. Similarly, the inclusion of only DSM-III-R and DSM-IV resulted in the exclusion of many otherwise appropriate studies that used DSM-III diagnostic criteria. However, given the extent of changes in some diagnostic criteria, particularly GAD, from DSM-III to III-R, this exclusion criterion was deemed necessary to ensure effect sizes could reasonably be combined within diagnoses. Lastly, it must be reiterated that the effect size estimates for no-treatment, expectancy control treatments, and RLX-only treatments are only estimates based on the effect sizes from control conditions of the studies included in this meta-analysis. Obtaining all treatment studies employing one of these conditions, while extraordinarily time-consuming, would provide more reliable effect size estimates.

The results of this study may also provide some interesting suggestions about alternate nosologies for anxiety. Although not statistically different, with the exception of the significant difference from social anxiety disorder, both GAD and PTSD showed somewhat larger weighted effect sizes than the other diagnostic groups. Westen and Morrison (2001) found comparable results that suggested response to treatment differs between panic disorder and GAD. Combined with our results, the data may suggest a differentiation between anxiety disorders wherein GAD, and possibly PTSD, may represent a different pathology from panic, social anxiety disorder, and OCD. Indeed, this hypothesis is congruent with emerging nosological models (Watson, 2005) and some of the preliminary transdiagnostic anxiety treatment data, suggesting different treatment response for individuals with GAD than for those with panic disorder, social anxiety disorder, and/or OCD (Barlow et al., 2004; Schmidt, 2003; Smith and Schmidt, 2005). While response to treatment alone is not the best basis for a psychological nosology, it does provide an interesting framework for continued investigation.

Overall, it appears that the various components of CBT, whether individually or in different combinations, tend to yield strong effect sizes from pre- to posttest across all anxiety disorders. However, none of the components of CBT evidenced longer-term treatment gains from post to follow-up, suggesting that patients do not continue to improve dramatically after treatment. However, the absence of a negative effect size suggests that, overall, treatment effects tend to be sustained after treatment cessation. It may be tentatively concluded that any CBT component is likely to be at least effective, irrespective of diagnosis, and these effects are likely to be sustained after treatment cessation. However, given that several combinations of treatment components were not evaluated for some diagnostic conditions, treatment components that have demonstrated efficacy for each diagnosis, such as the combination of CT+EXP, would appear to be the treatment of choice for the anxiety disorders.

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APPENDIX: STUDIES USED IN THE META-ANALYSIS

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