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Moderators and Non-Specific Predictors of Treatment Outcome for Anxiety Disorders: A Comparison of Cognitive Behavioral Therapy to Acceptance and Commitment Therapy

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Objective: Understanding for whom, and under what conditions, treatments exert their greatest effects is essential for developing personalized medicine. Research investigating moderators of outcome among evidence-based treatments for anxiety disorders is lacking. The current study examined several theory-driven and atheoretical putative moderators of outcome in cognitive behavioral therapy (CBT) and acceptance and commitment therapy (ACT). **Method:** Eighty-seven patients with a *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text rev.; *DSM-IV-TR*; American Psychiatric Association, 2000) anxiety disorder completed 12 sessions of ACT or CBT and were assessed with a self-report measure of anxiety at baseline, post-treatment, and 6- and 12-month follow-up assessments. **Results:** CBT outperformed ACT among those at moderate levels of baseline anxiety sensitivity, and among those with no comorbid mood disorder. ACT outperformed CBT among those with comorbid mood disorders. Higher baseline neuroticism was associated with poorer outcome across treatment conditions. Neither moderation nor general prediction was observed for baseline anxiety disorder comorbidity, race/ethnicity, gender, age, or baseline severity of the principal anxiety disorder. When including all randomized participants who completed the pre-treatment assessment ($N = 121$), a similar pattern was observed. **Conclusions:** Prescriptive recommendations for clinical practice and directions for future research are discussed.

Keywords: treatment moderators, cognitive behavioral therapy, acceptance and commitment therapy

The National Institutes of Health (NIH) has called for a focus on personalized medicine. Creating guidelines for the selection of treatments likely to yield the greatest efficacy based on an individual's baseline characteristics should have a significant impact on improving the effectiveness of mental health treatment. In order to achieve this overarching goal, researchers must uncover pre-treatment variables (e.g., baseline demographics, clinical characteristics) that have a predictive relation with outcome measures. Two distinct approaches can be taken: (a) understanding which types of individuals will respond best to treatment, regardless of the nature of the treatment (*non-specific predictors*); and (b) un-

derstanding which treatment works best for a particular individual (*moderators*) (see Driessen, Cuijpers, Hollon, & Dekker, 2010; Fournier et al., 2009). General, non-specific predictors of outcome across treatment groups provide prognostic information by clarifying what types of patients will respond more or less favorably to treatment in general. Treatment moderators provide prescriptive information about optimal treatment selection. *Moderators*, as opposed to *predictors*, are more useful in identifying subgroups of patients who will respond differentially to one treatment over another, thereby increasing the utility of the findings in making treatment decisions (Hollon & Najavits, 1988; Simon & Perlis, 2010). Thus, although there are benefits to identifying baseline predictors of overall treatment success (i.e., see Kraemer, Wilson, Fairburn, & Agras, 2002), identifying treatment moderators (who will do best in which treatment) may have more important clinical implications.

Researchers have attempted to address personalized medicine in the context of the treatment of depression (Simon & Perlis, 2010), but much less has been done in the context of anxiety treatment. Although treatment efficacy for anxiety disorders is often good (e.g., Bergström et al., 2009; Westra, Arkowitz, & Dozois, 2009), a significant number of individuals drop out of treatment, need additional treatment, do not significantly improve, or show a return of symptoms at follow-up assessments (e.g., Hofmann, Schulz,

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Meuret, Moscovitch, & Suvak, 2006; van Apeldoorn et al., 2008). Given the high prevalence (Kessler, Chiu, Demler, Merikangas, & Walters, 2005) and substantial cost (Greenberg et al., 1999) of anxiety disorders, more work is needed to match patients to the appropriate treatments in order to improve overall efficacy.

Extant literature has focused primarily on general predictors of cognitive behavioral therapy (CBT) outcomes. CBT for anxiety disorders appears to work similarly across gender, age, and socioeconomic status (e.g., Piacentini, Bergman, Jacobs, McCracken, & Kretchman, 2002; Schuurmans et al., 2009; Watanabe et al., 2010). With respect to clinical variables, however, the outcomes are mixed. For example, the extent to which baseline severity of a disorder impacts outcome differs across studies (Kampman, Keijsers, Hoogduin, & Hendriks, 2008; Meuret, Rosenfield, Seidel, Bhaskara, & Hofmann, 2010; Watanabe et al., 2010). Also, several studies have observed that comorbid depression does not predict anxiety symptom outcomes following CBT (e.g., Kampman et al., 2008; Rief, Trenkamp, Auer, & Fichter, 2000; Schuurmans et al., 2009; van Balkom et al., 2008), whereas others have found that it predicts worse outcomes (Chambless, Beck, Gracely, & Grisham, 2000; Chambless, Tran, & Glass, 1997; Steketee, Chambless, & Tran, 2001; Watanabe et al., 2010).

For the most part, other (non-mood disorder) psychiatric comorbidity appears to have little to no influence on CBT outcomes for anxiety disorders (Kampman et al., 2008; Mennin, Heimberg, & Jack, 2000; Ollendick, Ost, Ruetterskiold, & Costa, 2010; Schadé et al., 2007; Turner, Beidel, & Dancu, 1996). There are some exceptions (Steketee et al., 2001), such as the finding that certain additional anxiety disorders are associated with greater improvement in the targeted anxiety disorder (e.g., Brown, Antony, & Barlow, 1995). Finally, both poor health and high baseline neuroticism have been associated with a poorer prognosis from CBT for those with late-life anxiety (Schuurmans et al., 2009).

To our knowledge, only one study has compared the effect of putative moderators on two distinct psychological treatments for anxiety disorders (Meuret et al., 2010). In that study of treatment for panic disorder, lower perceived control at baseline was associated with poorer outcomes from a brief (4-week) treatment aimed at changing respiration as opposed to brief cognitive therapy, whereas higher levels of cognitive misappraisal of anxiety symptoms (i.e., anxiety sensitivity) at baseline were related to poorer outcomes in cognitive therapy as opposed to treatment aimed at changing respiration. No study has compared moderators between standard (i.e., 12-week) CBT and an established alternative treatment.

The goal of the current study was to evaluate potential moderators of a traditional full package of CBT compared to acceptance and commitment therapy (ACT) for anxiety disorders. Whereas CBT uses logical empiricism and exposure to feared stimuli in order to replace misappraisals with more evidence-based thinking, and aims to replace avoidance with approach behavior (see Craske, 2011), ACT uses cognitive defusion and acceptance to increase willingness to experience anxiety, and to engage in behavioral actions toward life values (Hayes, Strosahl, & Wilson, 1999). There is growing interest in ACT as an alternative approach, and initial data indicate that ACT is an effective treatment for anxiety disorders (e.g., Arch, Eifert, Plumb, Rose, & Craske, 2012). Although CBT and ACT share some common elements (Arch & Craske, 2008), they are derived from different theoretical models

and involve different treatment strategies; thus, differential moderation may be expected.

Because CBT focuses on challenging cognitive misappraisals through cognitive and behavioral strategies, and because cognitive misappraisals of anxiety symptoms have been shown to mediate CBT outcomes (Meuret, Rosenfield, Hofmann, Suvak, & Roth, 2009; Smits, Rosenfield, McDonald, & Telch, 2006), baseline level of cognitive misappraisals could be one moderator of treatment outcome, and may be more influential in CBT compared to ACT. However, the direction of that association is unclear. On the one hand, patients high in cognitive misappraisals may improve more with CBT than patients low in cognitive misappraisals, since the CBT focus upon cognitive restructuring would match the dysregulation underlying their anxiety; conversely, those low in baseline anxiety sensitivity may show poorer CBT outcomes because the treatment focus does not match the underlying dysregulation. On the other hand, those high in anxiety sensitivity at baseline may improve less in CBT because strongly held misappraisals may be resistant to change through direct attempts at challenging and replacing them with more evidence based thinking. In support of the latter hypothesis, Meuret et al. (2010) found that higher levels of cognitive misappraisal of anxiety symptoms predicted poorer outcomes from cognitive therapy for panic disorder than lower levels. Additionally, both of these possibilities may be true: that a non-linear relation exists between baseline anxiety sensitivity and treatment outcome in CBT, with high and low levels of anxiety sensitivity associated with poorer outcome than moderate levels. There are no studies to our knowledge that have examined anxiety sensitivity, or any other clinical variable, as a non-linear predictor or moderator of treatment outcome. Exploring nonlinear associations between anxiety sensitivity and treatment outcome may improve our ability to uncover moderators and more precisely match patients with appropriate treatments. Based on the limited previous research, we speculated that higher baseline anxiety sensitivity would be associated with poorer outcome. However, we also considered the possibility that the association may be non-linear.

A potential moderator of ACT may be experiential avoidance. Because ACT focuses on increasing willingness to experience and accept negative emotion, and because willingness has been shown to mediate ACT outcomes (Arch, Wolitzky-Taylor, Eifert, & Craske, 2012), those high in experiential avoidance may show more improvement in an ACT approach. In support, Zettle (2003) found that higher baseline levels of experiential avoidance positively predicted outcomes in a small sample of students with mathematics anxiety who were treated with ACT. Thus, it was hypothesized that higher experiential avoidance would be associated with more favorable outcomes in ACT. As with anxiety sensitivity, we explored the possibility that the association between baseline experiential avoidance and treatment outcome was non-linear in order to uncover potential moderating effects that may not be observed when looking only at linear associations. Given the lack of research exploring experiential avoidance as a predictor of outcome in CBT, no specific hypotheses were made.

Also, we hypothesized that ACT would outperform CBT among those with mood disorder comorbidity because ACT taps into constructs presumed to be shared across anxiety and mood disorders, whereas CBT is more disorder-specific in content (although its effects extend to comorbidity; e.g., Craske et al., 2007). Finally,

given prior findings regarding predictors, we hypothesized that the presence of an additional anxiety disorder would not impact outcome across groups, whereas neuroticism and baseline severity of the principal disorder would be associated with poorer outcomes. No other specific hypotheses were made.

To test our hypotheses, we used a sample of patients with mixed principal anxiety disorders. Such an approach is in line with current directions toward a transdiagnostic approach (e.g., Allen, McHugh, & Barlow, 2008; Barlow, Allen, & Choate, 2004), given the number of elements common across the anxiety disorders (Craske et al., 2009). Moreover, identification of treatment moderators across a variety of anxiety disorders may be of greater clinical utility in high demand real-world practice settings than moderators for each anxiety disorder.

Method

Participants

Participants were eligible for the study if they (a) met *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text rev.; *DSM-IV-TR*; American Psychiatric Association, 2000) diagnostic criteria for one or more anxiety disorders with a Clinician Severity Rating (CSR) ≥ 4 on the Anxiety Disorders Interview Schedule (ADIS-IV; Brown, Di Nardo, & Barlow, 1994; see the Measures section below; also see Craske, DeCola, Sachs, & Pontillo, 2003; Craske et al., 2007); (b) were between 18 and 60 years of age; (c) were either medication-free or stabilized on psychotropic medication; (d) were not undergoing other psychotherapy; and (e) were English-speaking. Exclusion criteria included the following: (a) history of psychiatric hospitalization in the past 5 years; (b) serious medical conditions or pregnancy; (c) active suicidal ideation and/or severe depression; (d) history of a psychotic disorder, bipolar disorder, mental retardation, or organic brain damage; and (e) substance abuse or dependence within the last 6 months. ADIS-IV principal anxiety disorder diagnoses show good inter-rater reliability, with kappas ranging from .67 to .86 (mean $\kappa = .78$; Brown, Di Nardo, Lehman, & Campbell, 2001).¹ A total of 147 participants were randomized to treatment. Sixteen of these participants did not complete any treatment sessions, and 44 did not complete all 12 sessions, leaving 87 participants who completed all 12 treatment sessions ($N = 49$ in CBT, $N = 38$ in ACT). Although study staff randomized participants before the baseline assessment, participants were blind to treatment condition assignment. All participants were told they would be undergoing a behavioral treatment but the specific type of treatment was not disclosed. Therapists did not interact with participants until the first treatment session. The Consolidated Standards of Reporting Trials (CONSORT) diagram shows the flow of participants through the study.

The completer sample was 47.1% female, with a mean age of 37.93 years ($SD = 11.79$). The sample was predominantly Caucasian/White (66.7%), with 14.9% Hispanic/Latino, 5.7% Black/African American, 9.2% Asian/Pacific Islander, 1.1% Native American/Alaskan Native, and 2.3% other race. The most common principal diagnosis was panic disorder with agoraphobia (PD/A; 36.8%), followed by generalized anxiety disorder (GAD; 20.7%), social anxiety disorder (SAD; 19.5%), obsessive compulsive disorder (OCD; 13.8%), specific phobia (SP; 3.4%), panic disorder

without agoraphobia (PD; 3.4%), and post-traumatic stress disorder (PTSD; 2.3%). The intent-to-treat (ITT) sample consisted of those who completed a pre-treatment assessment ($N = 121$). Similar to the completers sample, this ITT, or “completed baseline only” sample was 57.1% female and was predominantly Caucasian/White (66.7%), with 15.4% Hispanic/Latino, 6.0% Black/African American, 8.5% Asian/Pacific Islander, 0.9% Native American/Alaskan Native, and 2.6% other race. The most common principal diagnosis was PD/A (38.7%), followed by GAD (19.3%), SAD (19.3%), OCD (10.9%), SP (5.0%), PD (4.2%), and PTSD (2.5%).

Measures

Outcome measure.

Mood and Anxiety Symptom Questionnaire (MASQ; Watson & Clark, 1991). Because this study examined treatment moderators across a heterogeneous anxiety disorders sample, a measure with good psychometric properties that taps into general anxiety was chosen as the primary outcome measure. The MASQ is a 90-item measure based on the tripartite model of anxiety and depression. The measure asks participants to rate the degree to which they experienced a number of anxiety and mood symptoms in the past week, using a 6-point scale. The MASQ General Anxiety (MASQ-GA) subscale, which taps into general experiences of anxiety that cause distress, is comprised of 11 items—such as “Felt nervous,” “Felt nauseous,” and “Felt tense and high strung”—and was selected as the outcome measure. The MASQ-GA subscale shows good convergent and construct validity (Watson et al., 1995). Cronbach’s α for this subscale in the current sample was .81 at the baseline assessment, .86 for the post-treatment assessment, .78 at the 6-month follow-up, and .85 at the 12-month follow-up. Although the MASQ has not been used widely in clinical outcome research, it was chosen because it is sensitive to change after treatment (see the Results section) and is highly relevant across anxiety disorders. In the current sample, the baseline MASQ-GA score was greater than 1 *SD* above the mean for the normal adult population ($M = 20.65$, $SD = 7.1$; Watson et al., 1995) for all anxiety disorders except for specific phobia ($N = 6$), in which the baseline MASQ-GA score was within 1 *SD* of the norm.

Potential moderators.

Baseline demographic characteristics. Gender, age (coded as a continuous variable), and race/ethnicity (coded dichotomously as White vs. non-White status to increase statistical power) were analyzed.

Principal anxiety disorder diagnosis. Principal anxiety disorder diagnosis was determined using data from the ADIS-IV. This was the diagnosis deemed to be the most severe and causing the most distress and impairment, and was the focus of treatment in CBT.

Comorbidity. Diagnostic comorbidity data were collected during the administration of the ADIS-IV. The presence of the following current comorbid diagnoses was coded (yes/no): (1) a comorbid *DSM-IV-TR* anxiety disorder rated with a CSR ≥ 4 (i.e., one or more additional anxiety disorder diagnoses not including the principal diagnosis), and (2) one or more *DSM-IV-TR* mood disorders rated with a CSR ≥ 4 . Because more severe psychopa-

¹ For more information about the reliability of the ADIS-IV administration in this study, see Arch, Eifert, et al. (2012).

thology was an exclusion criterion (e.g., psychotic disorder, substance dependence) and personality disorders were not assessed, no other comorbidity was examined.

Baseline severity. Baseline severity of the principal diagnosis was determined by the CSR on the ADIS-IV (Brown et al., 1994). This semi-structured diagnostic interview was used to make *DSM-IV-TR* diagnoses of anxiety disorders, mood disorders, somatoform disorders, and substance disorders, and to screen for psychotic disorders. For each disorder, clinical ratings of severity of distress and disablement were made by group consensus on a 0–8 scale (CSR scale: 0 = *none*, 8 = *extreme*).

Anxiety Sensitivity Index (ASI; Peterson & Reiss, 1992; Reiss, Peterson, Gursky, & McNally, 1986). The ASI assesses beliefs that anxiety-related bodily sensations (e.g., rapid heartbeat, dizziness) are harmful, with sensations categorized as having either negative social, physical, or mental consequences. ASI total scores are elevated across all of the anxiety disorders (except specific phobia; see Taylor, Koch, & McNally, 1992). The ASI has good internal consistency ($\alpha = .82-.91$) and stable test-retest reliability over a 3-year period ($r = .71$; Maller & Reiss, 1992). Change in ASI has been shown to predict CBT outcomes for panic disorder (Smits, Powers, Cho, & Telch, 2004). Current study α at baseline was .84.

Acceptance and Action Questionnaire-16 (AAQ; Bond & Bunce, 2000). This 16-item measure is based on the validated nine-item AAQ and is hypothesized to be more sensitive to clinical change than the nine-item version (Hayes et al., 2004). The Willingness/Acceptance subscale, which taps into willingness to experience and accept negative emotion (as opposed to experiential avoidance), asks participants to rate statements such as “I am not afraid of my feelings” and “It’s OK to feel depressed and anxious.” Higher scores indicated lower experiential avoidance (i.e., greater willingness/acceptance of emotional experience). In this sample, α for the Willingness/Acceptance subscale at baseline was .65.

NEO Personality Inventory—Revised (NEO-PI-R; Costa & McCrae, 1992). The NEO-PI-R is a 60-item measure based on the 5-factor model of personality (Costa & McCrae, 1992), focused on identifying the degree to which an individual possesses the neurotic personality trait. The NEO-PI-R shows high internal consistency (α from .85 to .95) and long-term stability (Costa, Herbst, McCrae, & Seigler, 2000; Costa & McCrae, 1992; Rolland, Parker, & Stumpf, 1998). Current study α at baseline was .94.

Procedure

Experimental design. Participants completed baseline eligibility and diagnostic assessments, in addition to a questionnaire battery. Eligible participants were randomly assigned to either CBT or ACT. Following the 3-month treatment phase of the study (see below), participants were administered a post-treatment assessment consisting of a diagnostic interview and questionnaire battery. Participants completed similar assessments at the 6-month since baseline follow-up period (i.e., 3 months post-treatment) and 12-month since baseline follow-up period (i.e., 9 months post-treatment).

Treatment procedures common to both conditions. Participants received 12 weekly, 1-hr individual CBT or ACT therapy sessions by doctoral student therapists. Treatment protocols were standardized through the use of detailed treatment manuals.² The principal authors of the treatment manuals and/or advanced ther-

apists for CBT and ACT led weekly hour-long group supervision for study therapists. See Arch, Eifert, et al. (2012) for additional details of therapist training, randomization, and treatment.

Cognitive behavioral therapy (CBT). CBT followed Craske and Barlow’s (2005) protocol that included cognitive and behavioral treatment strategies of self-monitoring (introduced in Session 1), psychoeducation (introduced in Session 1), breathing retraining (emphasized in Sessions 2 and 3), cognitive therapy techniques (i.e., cognitive restructuring and behavioral experiments; emphasized in Session 2–4), and exposure (in vivo, interoceptive, and imaginal, as indicated), which was introduced in Session 5 and was emphasized throughout the remainder of treatment. The manual included a brief problem assessment in Session 1 in which the focus of treatment (typically the principal anxiety diagnosis) was established. The manual also included a branching mechanism that allowed tailoring of cognitive restructuring and exposure for each anxiety disorder. Exposure in CBT focused on hypothesis testing and anxiety reduction in the long term. Session 12 focused on relapse prevention including planning additional hypothesis testing exercises and exposures as needed.

Acceptance and commitment therapy (ACT). ACT was conducted following Eifert and Forsyth’s (2005) manual. In the current study, exercises were individualized to accommodate the clients’ principal anxiety disorder (see also Eifert et al., 2009). Session 1 focused on psychoeducation and an orientation to treatment. Session 2 emphasized creative hopelessness (i.e., exploring whether the client’s efforts to control anxiety “worked” and experiencing how these efforts led to the diminishment or elimination of valued life activities). Sessions 3–5 emphasized mindfulness, acceptance, and cognitive defusion. Acceptance was explored as an alternative to controlling anxiety via experiential exercises and practicing acceptance- and mindfulness-based meditations in session and at home. Sessions 6–11 continued to hone acceptance, mindfulness, and cognitive defusion skills and also emphasized values clarification with the goal of increasing clients’ willingness to pursue valued life activities. Personal values were explored via experiential exercises and participants were encouraged to behave in ways that reflected their values rather than spend time managing anxiety. In vivo and interoceptive exposures were included during Sessions 6–11 to match the amount of exposure in CBT but were framed as opportunities to practice engaging in valued activities while experiencing anxiety. In the final session (Session 12), clients discussed how to skillfully manage obstacles while pursuing a meaningful and workable life.

Statistical Analysis

We obtained up to 4 assessments of the outcome variable (MASQ-GA) over time: baseline, post-treatment, 6-month follow-up (FU), and 12-month FU. No assessments were obtained during the treatment. Such an assessment schedule does not lend itself to accurate modeling using typical growth curves (e.g., linear, quadratic, or exponential curves), since we expect a dramatic change from baseline to post-treatment, with some unknown pattern of change during FU (probably involving some worsening

² Contact Michelle G. Craske for a copy of the CBT treatment manual; the ACT manual is published (Eifert & Forsyth, 2005).

of symptoms). In such situations, recent research suggests the use a repeated measures analysis of variance (ANOVA) type model (with enhancements to allow for missing data and complex covariance structures), in which the means at each assessment are allowed to vary without fitting a specific growth curve (Liu, Rovine, & Molenaar, 2012). Such models more accurately reflect the true change that occurs and are less likely to result in false conclusions (Liu et al., 2012). Since our main objectives involved how potential moderators/predictors affect results at each post-treatment assessment, and did not involve the shape of growth curve during FU, we followed the recommendations of Liu et al. (2012) and treated the repeated assessments of MASQ-GA as levels of a repeated measures independent variable: TIME. Further, in pre/post designs, it is recommended that the pre-treatment assessment be used as a covariate rather than as a level of the repeated measures independent variable (IV), because using it as a covariate more fully equates groups on baseline levels of the outcome, minimizes the variance in the outcomes (thus increasing power), and is not subject to potential problems with “regression to the mean” (Tabachnick & Fidell, 2007). Therefore, we chose a mixed effects, repeated measures analysis of covariance (ANCOVA)-like design, implemented using the “mixed effects models” in PASW Version 19.0. Baseline MASQ-GA was included as a covariate, and the three post-treatment assessments (post, 6-month FU, and 12-month FU) were considered three levels of the repeated measures independent variable (TIME). This mixed effects routine allows the inclusion of all participants who have data on the baseline assessment and at least one post-treatment assessment. Further, using mixed effects models (referred to as MRMs [mixed effects regression models]) allows us to model the covariance matrix of the repeated measures more flexibly than standard repeated measures ANOVA, which assumes compound symmetry. We modeled the covariance matrix as autoregressive, with heterogeneous variances at the three assessment time points (this covariance matrix was the matrix with the fewest parameters whose “deviance” which was not significantly different from the “unstructured” covariance matrix; Heck et al., 2009).

Between subjects variables consisted of treatment condition (CBT or ACT), baseline level of MASQ-GA (as a covariate), and our potential predictors/moderators of outcome (for simplicity, we use the term “moderator” rather than moderator/predictor, except when the analysis demonstrates that a variable is a predictor). A separate MRM analysis was performed for each potential moderator.

Since a moderator might interact with treatment group or TIME, both of these interactions, and the triple interaction between the moderator, treatment group, and TIME were included in each analysis. Further, because few studies have examined these moderators in a study with two distinct treatments, and because the associations between psychological variables are often non-linear, we also explored whether the predictive or moderating relations were non-linear. Thus, quadratic terms for the moderator and its interaction with group and TIME (i.e., moderator^2 , $\text{Group} \times \text{moderator}^2$, $\text{TIME} \times \text{moderator}^2$, and $\text{Group} \times \text{Time} \times \text{moderator}^2$) were also included in the model. To summarize the variables entered on each level, all models included MASQ-GA as the outcome measure and TIME as a repeated measures IV (post, 6-month FU, and 12-month FU). All models also included the following between-subjects variables: MASQ-GA at baseline, the moderator, Group (CBT vs. ACT), and $\text{Group} \times \text{moderator}$. Mod-

els with continuous moderators (e.g., ASI) further included moderator² (e.g., ASI²), and $\text{moderator}^2 \times \text{Group}$. All models included the interactions between each between-subjects variable and TIME. See Table 1 for an overview of the analyses conducted.

Effect sizes for between subjects effects are reported as the proportion of the between subject variance (the “Level 2” variance) that is accounted for by the effect (Singer & Willett, 2003). The effect size for TIME is the proportion of the “Level 1” variance accounted for TIME. We will refer to these proportions as P², since they can be interpreted as roughly similar to R² for the error variance in the Level 1 or Level 2 equation in which the effect is included.

When we report a significant interaction, we do not report lower level components of the interaction (e.g., we do not report main effects when there is a significant interaction because, by definition, the effect of each variable depends on the level of the other variables in the interaction). Instead, we examine the effect of each variable in the interaction at relevant levels of the other variables in the interaction. Therefore, when a $\text{Group} \times \text{moderator}$ interaction was observed (i.e., moderating effect), the interaction was investigated from two perspectives: (1) the effect of the moderator within each Group, and (2) the effect of Group at different levels of the moderator. These follow-up tests were important for illustrating the specific nature of the interaction. To ascertain the effect of the moderator within each Group, simple slopes were computed within each Group by dummy coding, in turn, one treatment group as 0, and the other as 1 (e.g., CBT = 0, ACT = 1; Aiken & West, 1991). To ascertain the effect of Group at different levels of the moderator, simple slopes for Groups (i.e., differences between groups) were computed at different levels of the moderator. If the moderator was categorical, this was accomplished by dummy coding different levels of the moderator as 0 and examining whether a Group effect emerged at each level (Aiken & West, 1991). If the moderator was continuous, the moderator was “centered” alternately at “low” and “high” levels, thus allowing the calculation of the effect of Group at low (i.e., 1 SD below the mean) and high (i.e., 1 SD above the mean) levels of the moderator (thus deriving the effect of treatment for those low in ASI [for example], and the effect of treatment for those high in ASI). This procedure for determining simple effects at different levels of the moderator (or for the different treatment groups) includes *all* participants in the data analysis, and produces *model based estimates* of the relation between Group and the outcome for individuals at the “centered” level of the moderator. Thus, if one centers ASI at 1 SD below the mean, the analysis yields the *model based estimate* for the Group effect for those 1 SD below the mean. This approach to decomposing interactions is preferred to a more typical alternative approach to examining the effect of Group for patients low in ASI, in which one would only select those with low levels of ASI (1 SD below the mean or lower) and examine the Group effect within this small subsample. This latter “subsample” approach often yields more unreliable and unreplicable results because it is often based on small samples.

An interaction of a moderator with TIME indicates that the effect of the moderator was different at the different time points. Therefore, in such situations, we investigated the effect of the moderator on outcome at each time point. Thus, decisions to look at effects at each time point were driven by a significant interaction over TIME. Again, these simple effects tests at each time point

Table 1
List of Moderator Analyses Performed

Predictor/Moderator	Variables included in the model	Predictor/Moderator	Variables included in the model
1. Anxiety Sensitivity Index (ASI)	Mood and Anxiety Symptom Questionnaire–General Anxiety subscale (MASQ-GA) at baseline Group ASI ASI × Group ASI ² *ASI ² × Group TIME × Group TIME × ASI TIME × ASI × Group TIME × ASI ² TIME × ASI ² × Group	8. Gender	MASQ-GA at baseline Gender Gender × Group TIME × Group TIME × Gender TIME × Gender × Group
2. Acceptance and Action Questionnaire–16 Willingness subscale (AAQ)	MASQ-GA at baseline AAQ AAQ × Group AAQ ² AAQ ² × Group TIME × Group TIME × AAQ TIME × AAQ × Group TIME × AAQ ² *TIME × AAQ ² × Group	9. Race/ethnicity (Race)	MASQ-GA at baseline Race Race × Group TIME × Group TIME × Race TIME × Race × Group
3. Neuroticism (N)	MASQ-GA at baseline *N N × Group N ² N ² × Group TIME × Group TIME × N TIME × N × Group TIME × N ² TIME × N ² × Group	10. Principal anxiety disorder diagnosis (Principal)	MASQ-GA at baseline Principal Principal × Group TIME × Group TIME × Principal TIME × Principal × Group
4. Mood disorder comorbidity (Mood)	MASQ-GA at baseline Mood Mood × Group TIME × Group TIME × Mood *TIME × Mood × Group		
5. Anxiety disorder comorbidity (Anx)	MASQ-GA at baseline Anx Anx × Group TIME × Group TIME × Anx TIME × Anx × Group		
6. Baseline severity (Clinician Severity Rating [CSR])	MASQ-GA at baseline CSR CSR × Group TIME × Group TIME × CSR TIME × CSR × Group		
7. Age	MASQ-GA at baseline Age Age × Group Age ² Age ² × Group TIME × Group TIME × Age TIME × Age × Group TIME × Age ² TIME × Age ² × Group		

Note. Each row of this table includes all variables and their interactions included in a single analysis conducted to test the overall moderating effect. Follow-up tests were only conducted when a significant moderating effect was observed.

* Statistically significant moderating effects.

were performed using the entire sample and the full MRM model, but time was alternatively dummy coded such that the reference group was either post, or the 6-month FU, or the 12-month FU (see Tabachnick & Fidell, 2007). Thus, this reference group dummy coding produced model based estimations of the effect at each time point.

Although it is impossible to know if our results would be different if we were able to include data from those who dropped out of treatment before being administered the MASQ, we can use multiple imputation to impute the missing data for these dropouts and determine whether our results would change if imputed data for these treatment dropouts were added to the models. Multiple imputation is a multistep procedure that “imputes” the missing data in the model and produces multiple complete data sets that can then be analyzed, from which the results can be “pooled” (e.g., Allison, 2002; Rubin, 1987). We used the “fully conditional specification” and included all available relevant variables in the imputation: all the IVs (including all moderators), dependent variables (DVs), demographic variables, and the interactions used in every moderator analysis, plus available auxiliary variables. We performed 20 imputations and “pooled” the results according to the method recommended by Schafer (1997). *Multiple* imputations are required to prevent underestimation of the variance of the imputed variables (Rubin, 1987). The data set with the imputation that included dropouts was used to run an identical set of moderator analyses as a secondary approach to analyzing these data.

Results

Table 2 reports descriptive data for all potential moderators in the completer sample and ITT samples. Table 3 reports the de-

Table 2
Means (Standard Deviations) for Continuous Moderators at Baseline and Percentages (Ns) for Categorical Moderators at Baseline Between Treatment Groups

Variable	Completers		ITT	
	CBT	ACT	CBT	ACT
Age (SD)	37.86 (11.10)	37.84 (12.90)	38.12 (11.26)	37.65 (12.22)
% female (N)	51 (22)	51 (18)	60 (39)	26 (50)
% Caucasian (N)	63 (27)	71 (25)	66 (41)	69 (36)
Primary diagnosis CSR (SD)	5.53 (1.03)	5.80 (0.87)	5.63 (0.97)	5.70 (0.89)
% anxiety disorder comorbidity (N)	28 (12)	26 (9)	28 (18)	28 (15)
% mood disorder comorbidity (N)	16 (7)	17 (6)	22 (14)	23 (12)
ASI (SD)	26.27 (10.51)	32.32 (11.54)	27.76 (11.74)	31.98 (11.19)
NEO-PI-R (SD)	189.66 (33.10)	197.78 (33.87)	191.69 (34.58)	194.85 (35.24)
AAQ Willingness subscale (SD)	21.47 (6.17)	21.73 (6.32)	21.49 (6.79)	22.73 (6.50)

Note. Ns do not always add up to the total number in each group because of some missing data for individual variables. ITT = intent-to-treat; CBT = cognitive behavioral therapy; ACT = acceptance and commitment therapy; CSR = Clinician Severity Rating; ASI = Anxiety Sensitivity Index; NEO-PI-R = NEO Personality Inventory—Revised; AAQ = Acceptance and Action Questionnaire–16.

scriptive data for the dependent variable (i.e., MASQ-GA) in the completer and ITT samples.

Treatment Credibility, Therapist Competence, and Treatment Integrity

Participants completed treatment credibility ratings after Session 2 (see Arch, Eifert, et al., 2012, for details). Treatment credibility scores differed significantly by Group, $F(1, 76) = 9.08, p < .01, \eta_p^2 = .11$, with higher scores in CBT than ACT. There were no differences between CBT and ACT on therapist competence ratings ($p = .28$).

As expected, treatment integrity ratings indicated that cognitive therapy (CT) adherence scores were higher for CBT than ACT, $F(1, 87) = 316.88, p < .001, \eta_p^2 = .76$. Also as expected, ACT adherence scores were higher for ACT than CBT, $F(1, 87) = 813.58, p < .001, \eta_p^2 = .90$. On the behavioral adherence scale, which included items such as exposure and behavioral modeling, CBT scored significantly higher than ACT, $F(1, 87) = 22.77, p < .001, \eta_p^2 = .21$. The results show that therapists exhibited strong adherence to their assigned treatment. See Arch, Eifert, et al. (2012) for details about these measures and results.

Differences Between Treatment Completers and Treatment Dropouts

To examine whether dropouts and completers differed on the baseline measures of the variables included in the analyses, a multivariate analysis of variance (MANOVA) was run with baseline levels of both MASQ-GA and the potential moderators as dependent variables, and dropout status as the independent variable. There were no significant differences between dropouts and completers on the six continuous variables ($p = .68$). Chi-square tests were conducted to examine whether differences existed between dropouts and completers on the categorical variables. Dropouts differed from completers on one of these five variables, with more dropouts having a mood disorder diagnosis at baseline compared to completers, $\chi^2(1, N = 121) = 4.04, p < .05$.

Correlations Between Baseline MASQ-GA (DV) and Moderators (IVs)

Baseline MASQ-GA scores were significantly correlated with AAQ-Willingness ($r = -.25, p < .05$), Neuroticism scores ($r = .43, p < .001$), ASI ($r = .28, p < .05$), and age ($r = -.23, p < .05$).

Table 3
Means (Standard Deviations) Across Groups and Across Assessment Periods on the MASQ-GA

MASQ-GA assessment	Completers		ITT	
	CBT	ACT	CBT	ACT
Pre-treatment	26.95 (7.32)	28.93 (8.17)	27.18 (7.74)	28.24 (7.92)
Post-treatment	22.41 (8.22)	23.06 (7.58)	20.97 (12.07)	19.52 (11.64)
6-month follow-up	21.73 (5.75)	22.50 (6.91)	20.34 (10.23)	20.74 (10.19)
12-month follow-up	21.80 (7.20)	25.09 (6.89)	22.41 (13.60)	22.49 (11.41)

Note. Intent-to-treat (ITT) data at post-treatment, 6-month follow-up, and 12-month follow-up are based on the 20 imputation samples. MASQ-GA = Mood and Anxiety Symptom Questionnaire–General Anxiety subscale; CBT = cognitive behavioral therapy; ACT = acceptance and commitment therapy.

None of the other moderators were significantly associated with baseline MASQ-GA scores (all $ps > .26$).

Outcome Analyses

To verify the efficacy of our treatments, we examined whether MASQ-GA scores generally declined over time, and whether this decline varied by treatment. For this analysis only, the TIME variable included the baseline measure of MASQ-GA, and thus comprised four levels of MASQ (pre, post, 6-month FU, and 12-month FU). Group and the Group \times TIME interaction were also added to the TIME variable in the MRM model. Results indicated a significant effect only for TIME, $F(1, 84) = 12.04, p < .001, P^2 = 17.2\%$. Multiple comparison tests using the Sidak correction showed that baseline MASQ-GA scores were significantly higher than those at post-treatment ($p < .001$), 6-month FU ($p < .001$), and 12-month FU ($p < .005$; see Table 3). The post, 6-month FU, and 12-month FU scores did not differ from one another (Sidak corrected $ps > .99$).

Below, we present the results of our analyses of each potential moderator as both a general, non-specific predictor of outcome across treatment conditions (and TIME), and as a differential predictor (i.e., moderator) of outcome dependent upon treatment condition and/or TIME.

Moderator Analyses

Anxiety sensitivity. The quadratic ASI term interacted with Group to moderate outcome on the MASQ-GA, $F(1, 44) = 8.29, p < .01, P^2 = 13.7\%$.³ As displayed in Figure 1, simple effects tests showed a significant quadratic ASI effect for those in CBT, $F(1, 45) = 8.28, p < .01$, such that ASI scores near the mean were associated with lower MASQ-GA scores, but high and low ASI scores were associated with higher MASQ-GA scores. In ACT, neither the linear nor quadratic ASI effects were significant ($ps > .33$). Examining group differences as a function of ASI scores, no Group effects were observed among those with ASI scores 1 SD above the mean ($p = .73$) or 1 SD below the mean ($p = .72$) (see Figure 2). However, there was a significant difference between CBT and ACT for those with ASI scores at the mean, $F(1, 45) = 4.33, p < .05$, with CBT outperforming ACT. No other significant effects that were not subcomponents of this interaction were observed in this analysis.

Experiential avoidance. There was a significant $AAQ^2 \times$ Group \times Time interaction, $F(2, 54) = 3.20, p < .05, P^2 = 3.7\%$. This triple interaction can best be understood by examining the $AAQ^2 \times$ Group interaction at each time point. This was accomplished by alternately coding Time as 0, 1, 2 (to obtain predictors of MASQ-GA at post-treatment), $-1, 0, 1$ (to obtain predictors of MASQ-GA at 6-month FU), and $-2, -1, 0$ (for 12-month FU). Hence, this approach included all data in all the analyses, while still calculating the $AAQ^2 \times$ Group interaction effects at each time point separately. AAQ did not significantly interact with Group to predict outcome at either post or at the 6-month FU ($ps > .47$), nor did AAQ itself (not interacting with Group) predict outcome at either of these time points ($ps > .15$). However, there was a significant $AAQ^2 \times$ Group interaction for MASQ-GA at the 12-month FU, $F(1, 35) = 4.61, p < .05$. As seen in Figure 3, in CBT there was a tendency for MASQ-GA scores to increase as

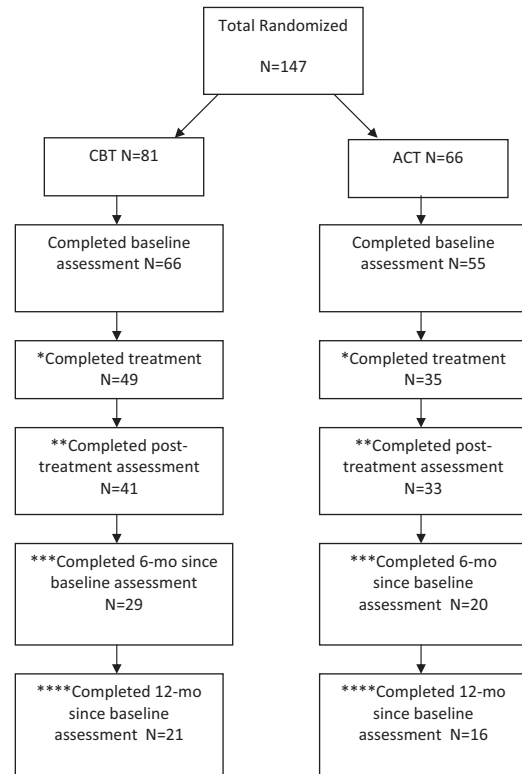


Figure 1. Consolidated Standards of Reporting Trials (CONSORT) diagram. Because some participants failed to complete a baseline Mood and Anxiety Symptom Questionnaire–General Anxiety subscale (MASQ-GA) but completed other portions of the study, and because we attempted to gather follow-up data from participants even if they missed previous assessment time-points, further details are provided: A single asterisk indicates that six participants in cognitive behavioral therapy (CBT) and three participants in acceptance and commitment therapy (ACT) completed treatment but failed to complete a baseline MASQ-GA questionnaire during the baseline assessment. Thus, 43 participants in CBT and 35 participants in ACT completed the baseline MASQ-GA and completed treatment. Two asterisks indicate that 35 participants in CBT and 32 participants in ACT completed the baseline MASQ-GA, all sessions of treatment, and the post-treatment MASQ-GA. Three asterisks indicate that 26 participants in CBT and 18 participants in ACT completed the baseline MASQ-GA, treatment, the post-treatment assessment, and the 6-month follow-up. Four asterisks indicate that 20 participants in CBT and 15 participants in ACT completed all four assessments and treatment.

AAQ increased, but this increase leveled off for AAQ scores greater than 0.5 SD above the mean. In the ACT condition, on the other hand, outcomes were best for those near the mean in AAQ, and worse for those either high or low in AAQ. Although these relations between AAQ and outcome within the two treatment conditions were in the opposite directions (thus resulting in the significant Group \times AAQ^2 interaction), neither tendency in either

³ Denominator degrees of freedom in our analyses are based on Satterthwaite's (1946) approximation, which has been shown to be more accurate than Wald tests in small samples (Hox, 2010). Thus, degrees of freedom reported vary substantially between analyses.

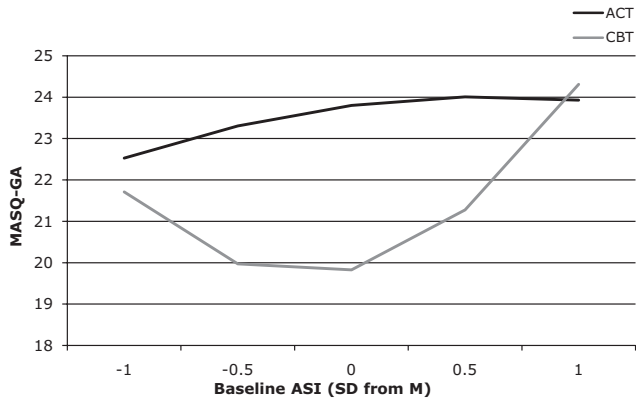


Figure 2. Baseline Anxiety Sensitivity Index (ASI) as a moderator of the Mood and Anxiety Symptom Questionnaire–General Anxiety subscale (MASQ-GA). ACT = acceptance and commitment therapy; CBT = cognitive behavioral therapy.

condition reached statistical significance ($p < .08$ for CBT and $p = .20$ for ACT). Similarly, testing the differences between the two treatment conditions at low (1 SD below the mean), medium (at the mean), and high AAQ (1 SD above the mean) revealed no significant differences between the groups ($ps > .38$; see Figure 2). No other significant effects that were not subcomponents of this interaction were observed.

Mood disorder comorbidity. A significant mood disorder \times Group \times TIME interaction was observed, $F(2, 50) = 7.12, p < .005, P^2 = 5.6\%$ (see Figure 4). Examining the mood disorder \times Group interaction at each assessment point, we found that, at post and at the 12-month FU, the mood disorder \times Group interaction was significant, $F(1, 61) = 7.68, p < .01$, and $F(1, 34) = 15.37, p < .001$, respectively. In terms of within-group effects, for CBT, those with a mood disorder at baseline had higher MASQ-GA scores at post and at the 12-month FU than those without a mood disorder, $F(1, 61) = 5.93, p < .05$, and $F(1, 34) = 12.95, p = .001$, respectively. In ACT, on the other hand, although not statistically

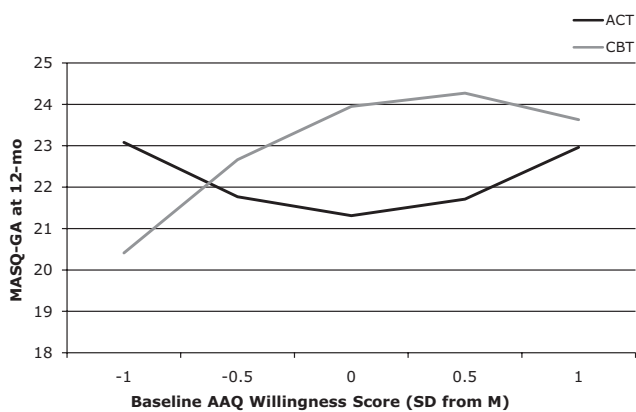


Figure 3. Baseline Acceptance and Action Questionnaire–16 (AAQ) as a moderator of the Mood and Anxiety Symptom Questionnaire–General Anxiety subscale (MASQ-GA). ACT = acceptance and commitment therapy; CBT = cognitive behavioral therapy.

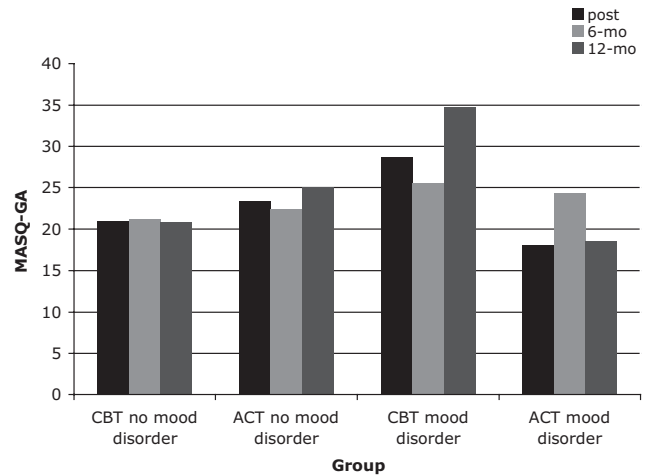


Figure 4. Mood disorder status as a moderator of the Mood and Anxiety Symptom Questionnaire–General Anxiety subscale (MASQ-GA). CBT = cognitive behavioral therapy; ACT = acceptance and commitment therapy.

significant, those with a mood disorder *tended* to have lower MASQ-GA scores at post and at 12-month FU than those without a mood disorder ($ps > .07$).

Looking at the interactions another way (i.e., between-group effects), at post and 12-month FU, patients with a mood disorder fared better in ACT than CBT, $F(1, 34) = 6.06, p < .05$, and $F(1, 34) = 11.37, p < .01$, respectively. In contrast, those without a mood disorder tended to do better in CBT than ACT, but CBT only outperformed ACT at the 12-month FU, $F(1, 34) = 4.37, p < .05$ ($p = .21$ at post-treatment). Thus, ACT significantly outperformed CBT among those with mood disorders at both time points and CBT significantly outperformed ACT among those with no mood disorders at the 12-month FU. At the 6-month FU, these same patterns of means were displayed; however, the mood disorder \times Group interaction was not significant ($p = .60$). Accordingly, the simple effects tests were not conducted at 6-month FU. Note that pretreatment level of MASQ-GA was included as a covariate in these analyses (as it was in all the analyses); therefore, these mood disorder effects were over and above any effects due to initial MASQ scores. No other effects that were not subcomponents of this interaction were observed.

Neuroticism. Neuroticism did not interact with treatment group or TIME to effect MASQ-GA ($ps \geq .08$), but it was a significant predictor of outcome, $F(1, 56) = 4.39, p < .05, P^2 = 5.4\%$. Higher baseline levels of neuroticism was predictive of higher levels of anxiety ($b = 1.74$) across groups and across assessment time points.

Anxiety disorder comorbidity, baseline severity, demographics, and principal anxiety disorder diagnosis. Anxiety disorder comorbidity was not significantly related to MASQ-GA, either as a predictor ($ps > .39$) or as a moderator ($ps > .50$). Likewise, baseline severity (i.e., baseline CSR of principal diagnosis) did not moderate outcome ($ps > .42$) nor did it predict outcome ($ps > .27$), even when baseline levels of MASQ-GA were not used as a covariate in the analysis ($ps > .11$). Principal anxiety disorder diagnosis also did not moderate outcome ($ps \geq$

.08) nor did it predict outcome ($ps > .60$).⁴ Finally, gender, age, and race/ethnicity did not significantly moderate outcomes on the MASQ-GA, neither as a predictor of outcome in general across groups ($ps > .07$) nor as a moderator ($ps > .08$).

Multiple Imputation for Analysis of the Full Randomized Sample

We used all variables from all our analyses (IVs, DVs, demographics, interactions, plus auxiliary variables) for imputing the data. We generated 20 complete imputed data sets (multiple imputation uses “different draws” from random variables to generate the different data sets). We then repeated all of the moderator/predictor analyses on 20 data sets and pooled the results (Schafer, 1997). Each of our above reported moderator/predictor findings was replicated in the pooled results except for the group \times AAQ² \times TIME interaction, which was not found to be significant. Thus, the overall, pooled results from the imputed full samples reinforced those from our “completer” analyses (except the AAQ² finding) and showed that (1) for ASI, there was a significant Group \times ASI² interaction ($p < .05$); (2) for comorbid mood disorder, there was a significant Group \times comorbid mood disorder \times TIME interaction ($p < .05$); (3) for neuroticism, there was a significant neuroticism main effect ($p < .05$); and (4) none of the other potential moderators showed any significant moderator or predictor effects.

Discussion

The present study examined putative moderators of treatment outcome for those receiving CBT and ACT in an effort to provide prescriptive information to aid in matching patients to specific psychological treatments for heterogeneous anxiety disorders. Both CBT and ACT were efficacious, and effects were comparable to previous clinical trials of CBT for anxiety disorders (Loerinc, Meuret, Twohig, Rosenfield, & Craske, 2012; Norton & Price, 2007). With two equally effective choices, it is especially important to identify for whom each treatment may be the *most* efficacious. In addition to atheoretical putative moderators, pre-treatment clinical characteristics associated with the theories underlying these treatments were examined, adding to the understudied body of research exploring whether these variables moderate treatment outcome. Findings identified only one baseline variable that generally predicted outcomes across both treatment groups, whereas several were identified as moderators, thus providing information to guide treatment matching. The results were largely consistent between completer and intent-to-treat samples, suggesting that the majority of the findings are robust regardless of treatment completion.

This study identified several psychological characteristics at baseline that moderated treatment outcome. Consistent with hypotheses, baseline level of anxiety sensitivity had a greater impact on treatment outcomes for CBT than for ACT. Whereas anxiety sensitivity at baseline did not impact anxiety symptoms for those in ACT, low and high levels of baseline anxiety sensitivity were associated with the least favorable outcomes in CBT, whereas moderately elevated levels were associated with the greatest improvement. These findings point to the unique relation between anxiety sensitivity and treatment outcomes in CBT. Because

change in anxiety sensitivity mediates change in symptoms from CBT, at least for panic disorder (Meuret et al., 2009; Smits et al., 2004), it is reasonable that baseline levels of this variable would be important in predicting response to CBT.

In particular, low levels of baseline anxiety sensitivity were associated with unfavorable outcomes from CBT. It may be that treatments targeting misappraisals are less relevant or meaningful to participants with low levels of misappraisal at the start of treatment. Indeed, those 1 *SD* below the mean of the current sample had ASI scores that were similar to the normal population (Donnell & McNally, 1989), suggesting that anxiety sensitivity may not be functionally important to their principal disorders. Higher levels of baseline anxiety sensitivity were also associated with unfavorable outcomes from CBT, which is consistent with findings from a brief 4-week cognitive therapy reported previously (Meuret et al., 2010). In this sample, ASI scores 1 *SD* above the mean were higher than the mean for a “clinical panicker” (Donnell & McNally, 1989). Conceivably, highly rigid beliefs about anxiety symptoms may be more difficult to address or modify through cognitive and behavioral strategies, resulting in less improvement. Also, strongly held beliefs that anxiety is dangerous may result in lower treatment engagement due to fears about completing anxiety-activating exercises in session and for homework (i.e., exposure). Alternatively, patients who begin treatment with high levels of anxiety sensitivity may ascend their fear hierarchies more slowly, resulting in less improvement by the twelfth session. Taken together, these findings indicate that those with baseline anxiety sensitivity that is low (i.e., comparable to a normal population), or high, may not improve as much in CBT as those with moderate anxiety sensitivity. Importantly, CBT outperformed ACT among those whose baseline anxiety sensitivity fell around the mean on the ASI. This suggests that those with moderate anxiety sensitivity within a clinical anxiety disorder population may be better suited for CBT than ACT.

Baseline experiential avoidance moderated treatment outcome, but inconsistent with hypotheses, experiential avoidance did not have a greater impact in ACT compared to CBT. Baseline experiential avoidance appeared to impact both CBT and ACT by the 12-month follow-up, suggesting that this pre-treatment variable may be useful in identifying trajectories of outcome in each treatment group over the long-term. Long-term improvement in CBT was the greatest among those with high experiential avoidance at baseline, whereas moderate levels of experiential avoidance were optimal for outcome among those in ACT. The current findings are somewhat discrepant from Zettle (2003) who found higher baseline experiential avoidance to be associated with more favorable outcome in ACT. Given that our simple effects tests did not attain statistical significance, and that these findings did not hold in the more conservative intent-to-treat analyses, these findings should be interpreted with caution and need further replication. Still, it appears that those with high baseline experiential avoidance may be best suited for CBT and those with moderate baseline experiential avoidance may be best suited for ACT. It is interesting how this latter finding parallels the findings for ASI in

⁴ Note that for anxiety disorder diagnosis, there were only three participants diagnosed with specific phobia and only two participants with PTSD, so they were not included in this analysis due to their small number.

CBT, in that both CBT and ACT appear to be most effective for patients with *moderately* high baseline levels of the characteristic targeted most by the treatment.

In the current study, high baseline levels of experiential avoidance were associated with a poorer long-term, but not short-term, prognosis for ACT than CBT. Perhaps high levels of experiential avoidance were initially mitigated by the focus on acceptance during ACT, thereby leading to anxiety symptom reduction, but resurged after therapy ended, resulting in a return of symptoms in the long-term. Future research is needed explore whether baseline experiential avoidance relates to continued acceptance, mindfulness, and pursuit of life goals after ACT is discontinued. High baseline experiential avoidance was associated with better long-term treatment response in CBT than moderate or high baseline levels. Possibly, this subgroup was more motivated to continue practicing CBT skills after CBT in an attempt to keep negative affect to a minimum, leading to greater improvement over time (e.g., Schmidt & Woolaway-Bickel, 2000). On the other hand, one might also argue that participants high in baseline experiential avoidance are more reluctant to engage in exposure therapy. However, breathing retraining and cognitive restructuring may have provided patients high in experiential avoidance with increased self-efficacy to undergo exposure during treatment; and successful learning experiences during treatment may have increased motivation for self-directed exposure during follow-up. Moreover, although effective exposure increases anxiety in the short-term, reductions in anxiety over the course of CBT may have encouraged continued exposure. Also, it is possible that those high in baseline experiential avoidance who were treated with CBT had greater increases in willingness during treatment compared to those in ACT, leading to more engagement in self-directed exposure during the follow-up period.

Consistent with our hypothesis, ACT showed a significant advantage over CBT for those with baseline comorbid mood disorders across multiple time points. Past research on the role of comorbid mood disorders and CBT outcomes has been mixed. Inconsistencies across studies may be due to different exclusionary criteria (e.g., exclusion of suicidality in some studies and not others). Results from the current study indicated that ACT may be a better choice than CBT for patients with comorbid mood disorders. In comparison to ACT, which addresses negative affect in general, CBT for anxiety disorders more narrowly addresses anxiety and fear. Thus, symptoms of depression may impede progress in CBT more than in ACT. In contrast, CBT showed a significant advantage over ACT for those without baseline comorbid mood disorders at the 12-month follow-up. Reasons for this finding are not clear. Perhaps those without comorbid mood disorders are able to take better advantage of 12 sessions focused strictly on their anxiety disorder, as opposed to the more diffuse approach in ACT. This finding suggests a helpful prescriptive picture: those with comorbid mood disorders should be treated with ACT whereas those without comorbid mood disorders may benefit more in the long-term from CBT.

Consistent with hypothesis and prior research (Schuurmans et al., 2009), higher baseline levels of neuroticism were associated with poorer outcome across both groups. This finding provides a prognostic indication that those who enter treatment higher in neuroticism may be at risk for lower treatment response. Clinicians

may be advised to assess neuroticism at baseline to identify those individuals who may be in need of additional treatment.

Consistent with previous research, age (e.g., Watanabe et al., 2010), gender (e.g., Piacentini et al., 2002), and race/ethnicity did not moderate or predict outcome. Thus, there was no evidence that ACT or CBT were more effective for certain genders, races/ethnicities, or ages. Also, current results add to the body of work suggesting that pre-treatment severity of the principal diagnosis neither moderates nor predicts outcome (e.g., Watanabe et al., 2010), suggesting that patients with severe anxiety psychopathology can improve to the same degree as those with less severe symptoms. Although no specific hypotheses were made about comorbid anxiety disorders, no moderating relation was observed. We suspected that covarying baseline MASQ-GA controlled for the additional anxiety symptom severity that may be indicated by additional anxiety disorders. However, removing the baseline MASQ-GA covariate did not change this finding. Thus, there was no evidence from these data that the presence of additional, clinically significant anxiety disorders impacts treatment outcome generally or differentially between CBT and ACT. On the other hand, the relatively small sample size may have resulted in Type II error. Although our power analysis suggested we had sufficient power for the analyses, future studies with larger samples should be conducted to examine whether these null findings are replicated.

Conclusions

The current study is the first to directly compare putative moderators for CBT and ACT for the treatment of anxiety disorders. In addition, the current study illustrated the importance of evaluating nonlinear relations between moderating variables and treatment outcome. Indeed, two of our main findings uncovered important moderators that had a quadratic association with treatment outcome. These findings, which can directly aid in treatment matching, would not have been observed with an approach that simply relied on the assumption that psychological constructs such as anxiety sensitivity and willingness to experience emotion at baseline have a linear relation with outcome.

Taken together, several preliminary prescriptive recommendations can be made from this study, should the results be replicated and observed across multiple indices of change and consistently over time. First, those with baseline anxiety sensitivity in the moderate range (relative to patients with anxiety disorders), those with higher baseline experiential avoidance, and/or those without mood disorder comorbidity may improve more in CBT compared to ACT. On the other hand, those who have mood disorder comorbidity, and/or moderate baseline experiential avoidance, may be better suited for ACT.

The prescriptive recommendations drawn from the findings are tempered by limitations of the current study. The primary limitation of the study was the relatively small sample size which precluded examination of disorder-specific moderators. Although the inclusion of all anxiety disorders is a strength of the study and in line with current movement in the field toward a transdiagnostic approach to treatment of anxiety disorders (see Barlow et al., 2004), it does present a limitation regarding power to detect disorder-specific moderating effects. Future research with larger samples may benefit from examining baseline levels of disorder-specific measures of appraisal in specific subgroups (e.g., measures that assess social concerns for social anxiety disorder or

beliefs about worry for generalized anxiety disorder), as well as examining moderators of outcome on disorder-specific measures. Indeed, the choice of outcome measures inherently has an impact on findings, a common limitation of treatment outcome research. The outcome measure and theory-driven predictor measures in this study were selected because they were relevant across the anxiety disorders. However, the ASI and MASQ-GA may be less relevant for specific phobia. Thus, it is possible findings may have differed for those with specific phobia. Finally, this study explored several theoretical and atheoretical predictors, which is a reasonable step for the first study of its kind. However, despite using a statistical approach that reduced the number of tests needed to examine the effects of these predictors across a number of assessment periods, several tests were still conducted, leaving open the possibility of Type I error. Again, future research should examine whether these findings are replicated.

Personalized medicine can only be developed when researchers and clinicians have information to guide treatment selection based on pre-treatment characteristics. This study provides preliminary evidence that there may be a number of pre-treatment variables exerting different patterns of influence on outcomes in CBT compared to ACT.

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