Behaviour Research and Therapy 50 (2012) 469–478

Contents lists available at SciVerse ScienceDirect

Behaviour Research and Therapy

journal homepage: www.elsevier.com/locate/brat

Longitudinal treatment mediation of traditional cognitive behavioral therapy and acceptance and commitment therapy for anxiety disorders

Joanna J. Arch [a,*], Kate B. Wolitzky-Taylor [b], Georg H. Eifert [c], Michelle G. Craske [b]

[a] University of Colorado Boulder, Department of Psychology and Neuroscience, 345 UCB Muenzinger, Boulder, CO 80309-0345, USA
[b] University of California, Los Angeles, Department of Psychology, 405 Hilgard Avenue, Los Angeles, CA 90095, USA
[c] Chapman University, Department of Psychology, One University Drive, Orange, CA 92866, USA

ARTICLE INFO

Article history:
Received 12 August 2011
Received in revised form
20 April 2012
Accepted 23 April 2012

Keywords:
Cognitive behavioral therapy (CBT)
Acceptance and commitment therapy (ACT)
Anxiety
Mediation
Cognitive defusion
Anxiety sensitivity

ABSTRACT

Objective: To assess the relationship between session-by-session putative mediators and treatment outcomes in traditional cognitive behavioral therapy (CBT) and acceptance and commitment therapy (ACT) for mixed anxiety disorders.
Method: Session-by-session changes in anxiety sensitivity and cognitive defusion were assessed in 67 adult outpatients randomized to CBT ($n = 35$) or ACT ($n = 32$) for a DSM-IV anxiety disorder.
Results: Multilevel mediation analyses revealed significant changes in the proposed mediators during both treatments ($p < .001$, $d = .90–1.93$), with ACT showing borderline greater improvements than CBT in cognitive defusion ($p = .05$, $d = .82$). Anxiety sensitivity and cognitive defusion both significantly mediated post-treatment worry; cognitive defusion more strongly predicted worry reductions in CBT than in ACT. In addition, cognitive defusion significantly mediated quality of life, behavioral avoidance, and (secondary) depression outcomes across both CBT and ACT ($p < .05$, R^2 change $= .06–.13$), whereas anxiety sensitivity did not significantly mediate other outcomes.
Conclusions: Cognitive defusion represents an important source of therapeutic change across both CBT and ACT. The data offered little evidence for substantially distinct treatment-related mediation pathways.

Determining if and how psychotherapies work is a central task of clinical science. Thus far, the field has succeeded greatly at demonstrating that specific therapies are effective for specific disorders, but we are still working to demonstrate "how." Addressing how therapies work requires identifying the specific variables that drive change within specific treatments, or treatment mediators.

Anxiety disorders, the most common class of psychiatric disorders (Kessler, Berglund, Demler, Jin, & Walters, 2005), are characterized by fear, anxiety, and behavioral avoidance. Over the past few decades, cognitive behavioral therapy (CBT) has become the most empirically supported psychotherapy for anxiety disorders (see Craske, 2010). Meta-analyses confirm the efficacy of CBT for the treatment of anxiety disorders relative to wait-list, expectancy and attention control conditions (Butler, Chapman, Forman, & Beck, 2006; Hofmann & Smits, 2008) and psychodynamic therapy (Tolin, 2010). However, relatively few studies rigorously examine mediators of CBT for anxiety disorders. Understanding CBT-specific vs. treatment-common processes requires comparing CBT mediators to those of another active psychotherapy (see Arch & Craske, 2008; Kraemer, Wilson, Fairburn, & Agras, 2002). Yet, relatively few studies compare CBT for anxiety disorders to fully active psychotherapy-based treatments (Tolin, 2010). Of those that do, very few compare treatment mediation between the two approaches.

Acceptance and commitment therapy (ACT; Hayes, Strosahl, & Wilson, 1999, 2012), an acceptance-based behavioral therapy, has been applied specifically to the treatment of anxiety disorders (e.g., Arch et al., in press; Eifert & Forsyth, 2005; Eifert et al., 2009). With roots in the behavioral and experiential therapy traditions, ACT cultivates mindfulness, acceptance and cognitive defusion with the aim of decreasing avoidance of internal discomfort, increasing psychological flexibility, and above all, promoting behavior change consistent with personal values (Hayes et al., 1999). Case studies, multiple-baseline studies, and an initial randomized study (Twohig et al., 2010) provide nascent evidence that ACT is an effective treatment for anxiety disorders, including obsessive compulsive disorder (Twohig, Hayes, & Masuda, 2006; Twohig et al., 2010), social anxiety disorder (Dalrymple & Herbert, 2007), panic disorder (Eifert et al., 2009), and posttraumatic stress disorder (Orsillo &

* Corresponding author. Tel.: +1 303 492 4634; fax: +1 303 492 2967.
E-mail addresses: Joanna.Arch@Colorado.edu (J.J. Arch), ktaylor@psych.ucla.edu (K.B. Wolitzky-Taylor), geifert@chapman.edu (G.H. Eifert), Craske@psych.ucla.edu (M.G. Craske).

0005-7967/$ – see front matter © 2012 Elsevier Ltd. All rights reserved.
doi:10.1016/j.brat.2012.04.007

Batten, 2005). In addition, an acceptance-based behavioral therapy for generalized anxiety disorder has shown effectiveness relative to a wait-list control condition (Roemer, Orsillo, & Salters-Pedneault, 2008). We recently completed a randomized trial comparing ACT and CBT for the treatment of a mixed anxiety disorder sample, showing similar effectiveness of ACT compared to traditional CBT (Arch et al., in press). Thus, initial evidence suggests that ACT is effective for the treatment of anxiety disorders.

Within the context of anxiety disorder treatment, the question remains of how traditional CBT (herein abbreviated as CBT) and ACT work and whether they work for the same reasons. The cognitive model for CBT identifies reductions in anxiety-related thoughts and beliefs as the central treatment process, leading to subsequent fear or symptom reduction (see Craske, 2010). Although the evidence has been mixed, several CBT studies report that changes in cognitions predict or mediate symptom improvements. For example, Hofmann (2004) found that pre- to post-treatment reductions in negative social cost ratings (patient ratings of "how bad would it be" if a feared social event occurred) predicted symptom outcomes in CBT for social phobia, though such changes also mediated outcomes in a comparison behavioral therapy condition. Smits, Rosenfeld, McDonald, and Telch (2006) established that session-by-session estimates for probability of a feared social outcome predicted subsequent fear reduction in social phobia. Further, Hofmann et al. (2007) demonstrated that changes in panic-related cognitions across pre-post-follow up predicted panic disorder symptom improvement in conditions that included CBT (CBT-only, CBT plus medication) but not in a medication-only condition. Meuret, Rosenfield, Hofmann, Seidel, and Bhaskara (2010) demonstrated changes in negative beliefs about panic and anxiety-related sensations were bidirectionally related to panic symptom severity in cognitive therapy but not in capnometry-assisted respiratory training for panic disorder. Elsewhere, pre- to post-treatment reductions in fear of and negative beliefs about panic and anxiety-related sensations accounted for significant variance in panic disorder symptom change in CBT (Smits, Powers, Cho, & Telch, 2004). These studies provide initial evidence for a significant relationship between cognitive and symptom changes in CBT for panic disorder and social phobia, and suggest that this relationship is sometimes specific to CBT. However, with two notable exceptions (Meuret et al., 2010; Smits et al., 2006), these studies did not assess changes in the mediator variable during treatment, and thus did not establish the temporal precedence of the mediator required for full mediation testing. Furthermore, few compared mediation of CBT to that of another active psychotherapy.

Whereas the cognitive model of CBT posits that change in thoughts and beliefs about anxiety predicts symptom reductions, ACT posits that "buying into" or being fused with anxiety-related thoughts and beliefs is a core cause of disordered anxiety (Eifert & Forsyth, 2005; Forsyth, Eifert, & Barrios, 2006). Consequently, ACT aims to reduce fusion with anxiety-related thoughts through "cognitive defusion" or flexibly distancing from the literal meaning of cognition so that cognition no longer rigidly dictates behavior. Acceptance is employed for approaching rather than avoiding anxiety-related thoughts, feelings, and physical sensations. A small, multiple-baseline study of ACT for social phobia (Dalrymple & Herbert, 2007) established that pre- to mid-treatment changes on a measure of cognitive defusion, acceptance, and valued action predicted subsequent symptom improvement. A randomized trial comparing ACT to relaxation training for obsessive compulsive disorder (Twohig et al., 2010) found greater pre- to post-treatment increases on a measure of cognitive defusion, acceptance, and value-guided action in ACT than in relaxation training, although the differences were no longer significant at 3 month follow up. Two small studies in samples of psychotic inpatients (e.g., Gaudiano & Herbert, 2006a,b; see Hayes, Luoma, Bond, Masuda, & Lillis, 2006) and depressed outpatients (Zettle & Hayes, 1986), support the notion that changes in the believability of distressing hallucinations or cognitions (a measure of cognitive defusion) predict outcomes in ACT.

Collectively, these studies identify cognitive defusion, acceptance, and value-guided action as likely mediators of ACT for anxiety disorders. Furthermore, evidence (Twohig et al., 2010) suggests that changes in these variables may be specific to ACT at post-treatment, at least compared to relaxation training. The evidence to date is only preliminary, however, and the methodological quality varies widely. For example, only one study (Dalrymple & Herbert, 2007) evaluated the temporal precedence of the mediator and none compared ACT mediators to those of CBT.[1]

In summary, few previous studies fulfill the highest standards for tests of treatment mediation. Full testing of treatment mediation requires that the mediation measure be administered *during* treatment, and preferably at multiple time points during treatment, to establish the temporal precedence of the mediator relative to outcome measures (Kraemer et al., 2002; MacKinnon, Fairchild, & Fritz, 2007). Also, the mediation measure should be treatment-specific and psychometrically valid. Methodologies such as multilevel modeling provide greater statistical power and permit fine-grained analyses such as the time sequencing of effects (Kenny, Kashy, & Bolger, 1998). Several exemplary studies in the anxiety disorders literature fulfill these standards (e.g., Meuret, Rosenfield, Hofmann, Suvak, & Roth, 2009; Moscovitch, Hofmann, Suvak, & In-Albon, 2005) but address different mediation questions than does the present study. Our aim is to apply these rigorous standards to investigating treatment mediation of CBT and ACT for anxiety disorders.

Our discussion has focused more on cognitive mediation (broadly defined) of CBT and ACT for two reasons. First, extant studies of mediation in CBT and ACT have focused on cognitive mediation, namely, reductions of anxiety-related beliefs in CBT (thought content) and reductions of cognitive fusion or believability in ACT (thought context). Second, we believe that cognitive mediators offered greater potential to distinguish CBT and ACT than behavioral mediators. In that CBT and ACT are both behaviorally-based therapies, they share the goal of reducing behavioral avoidance. ACT focuses on engaging in valued behaviors that have been avoided due to life narrowing that results from attempts to control symptoms, whereas CBT focuses on engaging in behaviors that have been avoided due to their association with fear and anxiety. In practice, however, these approaches may result in similar reductions in behavioral avoidance, which would likely drive change in both treatments (see Arch & Craske, 2008). For these two reasons, we focused the present investigation on assessing cognitive mediation of CBT and ACT, defined in the broad sense of mediators that reflect either the context (how we relate to) or content of cognition.

Our study investigated two central questions: 1) Do CBT and ACT affect the theorized mediators for each treatment, showing greater reductions in beliefs about the harmful effects of anxiety (i.e. anxiety sensitivity) in CBT and greater increases in cognitive defusion in ACT? 2) Do changes in anxiety sensitivity and cognitive defusion mediate treatment outcomes? Specifically, do treatment-specific processes mediate outcomes within the specified treatment only (anxiety sensitivity mediates CBT but not ACT outcomes, whereas cognitive defusion mediates ACT but not CBT outcomes), *or, alternatively,* do treatment-specific processes mediate outcomes across *both* treatments (anxiety sensitivity and cognitive defusion mediate outcomes across both CBT and ACT)?

[1] For effectiveness studies of ACT vs. CBT in undiagnosed patient samples, see Forman, Herbert, Moitra, Yeomans, & Geller, 2007; Lappalainen et al., 2007.

Methods

Participants

Sixty-seven adult outpatients (Ps) meeting criteria for one or more DSM-IV anxiety disorders were included in the mediation analyses, including Ps randomized to CBT ($n = 35$) and ACT ($n = 32$). Ps were included if they completed at least 6 sessions of psychotherapy (half of treatment) and at least one session-by-session mediation measure during treatment (see Measures) in order to provide a sufficient foundation for investigating therapy process. Seven Ps (10%) who completed 6 or more sessions did not finish treatment; they were included in analyses of mediator change during treatment but not in post-treatment outcome analyses. Thus, the *n*'s for post-treatment outcome analyses are lower than for change in mediators during treatment.[2]

Ps were recruited from the greater West Los Angeles area; 51% were female, 63% were Caucasian, 13% were Latino/Hispanic-American, 12% were Asian-American, 6% were African-American, 1% were Native American, and 4% were biracial. The mean age was 37 years (11.8 SD, range 19–60) with 15 years of education (2.1 SD, range 9–21) and 22% were married, 55% were single, 10% were cohabiting, and 12% were separated or divorced. CBT and ACT Ps did not differ significantly on any demographic variables, all $ps > .3$. Clinical diagnoses were ascertained by highly trained doctoral students and professional research assistants using the Anxiety Disorders Interview Schedule-IV-Revised (ADIS-IV-R) (DiNardo, Brown, & Barlow, 1994). Principal diagnoses included 36% (24/67) with panic disorder with or without agoraphobia, 24% (16/67) with social anxiety disorder, 19% (13/67) with generalized anxiety disorder, 13% (9/67) with obsessive compulsive disorder, 4% with specific phobia (3/67) and 3% with posttraumatic stress disorder (2/67). Of these, 11% (7/66)[3] additionally met criteria for major depression. CBT and ACT Ps did not differ significantly on principal diagnoses. Although CBT (45.7%, 16/35) had more Ps with principal PD/A than ACT (25.0%, 8/32), this difference did not reach significance, $\chi^2(1) = 3.12$, $p = .08$. Diagnostic assessments and treatment took place at the Anxiety Disorders Research Center at the University of California Los Angeles (UCLA), Department of Psychology.

Exclusion criteria were active suicidal ideation, very severe depression (ADIS-IV-R clinical severity rating >6, see Outcome Measures), substance abuse or dependence within the last 6 months, psychiatric hospitalization within the last five years, a history of bipolar disorder, psychosis, mental retardation, or organic brain damage. If taking psychotropic medication, Ps were required to be stabilized: one month for benzodiazepines, or three months for SSRIs/SNRIs/heterocyclics. Please refer to Arch et al. (in press) for additional study criteria, assessment, and assessment training procedures.

Measures

Session-by-session measures (purported mediators)

The Anxiety Sensitivity Index (ASI) and a brief version of the Believability of Anxious Feelings and Thoughts questionnaire (BAFT) were administered as measures of therapy process at the beginning of alternating therapy sessions (Sessions 2, 4, 6, 8, 10). Clients completed these questionnaires in the clinic waiting room before each treatment session, placed them in a large envelope, and turned them in to the clinic staff (i.e. not to the study therapists). The ASI was conceptualized as a CBT process measure whereas the BAFT was conceptualized as an ACT process measure.

Anxiety Sensitivity Index (ASI). The *Anxiety Sensitivity Index* (Peterson & Reiss, 1992; Reiss, Peterson, Gursky, & McNally, 1986) assesses fear of anxiety-related symptoms (e.g., rapid heart beat) based on the belief that such sensations have negative social, physical, or mental consequences. In that fear and negative beliefs about experiencing fear and anxiety are common features of anxiety disorders (Craske, Rauch, et al., 2009), the ASI captures a core component of these disorders. ASI total scores are elevated across all anxiety disorders (except specific phobia, see Taylor, Koch, & McNally, 1992) relative to nonclinical samples, and particularly panic disorder and posttraumatic stress disorder (Olatunji & Wolitzky-Taylor, 2009). Psychometrically, the ASI has good internal consistency ($\alpha = .82$–$.91$) and stable test–retest reliability over a three-year period ($r = .71$) (Maller & Reiss, 1992). ASI assessed at pre- and post-treatment has been shown to mediate CBT for panic disorder outcomes (Smits et al., 2004). Current study α's ranged from .87 to .90 depending on the assessment point.

Believability of Anxious Feelings and Thoughts (BAFT). The original *Believability of Anxious Feelings and Thoughts* questionnaire was a 30-item measure designed to assess cognitive defusion processes within ACT for anxiety disorders (see Eifert et al., 2009). More extensive psychometric analyses (BAFT; Herzberg et al., in press) resulted in a version with 16 items. Higher scores indicate lower levels of cognitive defusion (or higher levels of cognitive fusion). Previous ACT studies (e.g., Bach & Hayes, 2002; Gaudiano & Herbert, 2006b; Twohig et al., 2006) have developed similar measures on an as-needed basis to assess treatment-related change in cognitive defusion, a central hypothesized ACT process (see Hayes et al., 2006). In contrast to the non-psychometrically validated cognitive defusion measures used in past research, the BAFT is well-validated with high internal consistency in both healthy ($\alpha = .90$) and highly anxious ($\alpha = .90$) samples, and possesses good divergent, convergent, and predictive validity in a treatment-seeking high anxiety sample (Herzberg et al., in press). We administered the entire BAFT at pre- and post-treatment, and a briefer 7-item version as a session-by-session measure to reduce participant burden. We selected these 7 items from the original BAFT based on their strong face validity. Six of these 7 items are included in the revised BAFT (items 1, 4, 8, 14, 15, 16) and demonstrate an overall BAFT factor loading of $M = .65$ (range .61–.70). Current study α's ranged from .79 to .86[4] across assessment points.

Treatments

Following the baseline eligibility and diagnostic assessments, Ps were randomly assigned to CBT or ACT. Ps received twelve weekly, 1-h individual CBT or ACT therapy sessions based on detailed treatment manuals delivered by doctoral student therapists.[5] The principal authors of the treatment manuals for CBT and ACT led

[2] Participants who dropped out of treatment after Session 6 (2 by the PI, 5 on their own) were invited to complete the post-treatment assessments but generally declined due to time constraints (assessments included a 2–3 h laboratory assessment in addition to an extensive clinical interview and questionnaires).

[3] Mood disorder data was missing for 1 participant.

[4] Comparing these 7 BAFT items to the 6 included in the revised BAFT shows that Cronbach α's at each time point ranged from .75 to .85 ($M = .81$) for the 6 item version and .79 to .86 ($M = .82$) for the 7-item version, i.e. α's were identical or increased when the 7th item is added. Therefore, it seemed statistically sound in the current sample to include the 7th item despite its exclusion from the revised BAFT.

[5] See author MGC for a copy of the CBT treatment manual; the ACT manual is published (Eifert & Forsyth, 2005).

weekly hour-long group supervision for study therapists.[6] See Arch et al. (in press), for additional details of therapist training, randomization, and treatment.

Traditional cognitive behavioral therapy (CBT). Traditional CBT for anxiety disorders followed a protocol (Craske, 2005) successfully employed in previous studies (Craske, Rose, et al., 2009; Craske et al., 2011) which included general treatment components shared across the anxiety disorders along with a 'branching' mechanism that listed specific cognitive restructuring strategies and exposure methods for each anxiety disorder. CBT began in Session 1 with problem assessment, self-monitoring, and psychoeducation. Cognitive restructuring (logical empiricism, evidence-based analysis and hypothesis testing), self-monitoring, and breathing retraining were introduced in Sessions 2–4. Exposure, including interoceptive, in-vivo, and imaginal exposure, was introduced in Session 5 and emphasized strongly in sessions 6–11. Cognitive restructuring and exposure were tailored to the client's principal diagnosis. Clients were taught to answer a series of questions immediately following each exposure: 1) Did the feared catastrophe (e.g., collapsing, losing control) come true?; 2) Did I survive being distressed or afraid?; and 3) Did the fear subside with repetition? Through this framing, exposure in CBT focused on hypothesis testing and anxiety reduction over time. Session 12 focused on relapse prevention including planning additional hypothesis testing exercises and exposures as-needed.

Acceptance and commitment therapy (ACT). ACT for anxiety disorders followed a manual (Eifert & Forsyth, 2005; Eifert et al., 2009); Session 1 focused on psychoeducation and an orientation to treatment. Session 2 emphasized "creative hopelessness", or exploring whether the client's efforts to manage and control anxiety had been successful and experiencing the costs of these efforts (e.g., diminishment or elimination of valued life activities). Sessions 3–5 emphasized mindfulness, acceptance, and cognitive defusion, or the process of distancing oneself from the literal meaning of anxiety-related thoughts, obsessions, and self-talk. Acceptance was explored as an alternative to controlling anxiety via experiential exercises (such as Chinese finger traps, see Eifert & Forsyth, 2005) 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 engage in valued life activities on a daily basis. Personal values were explored via experiential exercises and Ps were encouraged to behave in ways that reflected their values rather than spend time managing anxiety. In-vivo and interoceptive exposures were employed during Sessions 6–11 to match the CBT condition on amount of exposure, and were framed as opportunities to practice acceptance and defusion skills and engage in valued activities while experiencing anxiety. In the final session (Session 12), clients discussed how to skillfully manage obstacles while pursuing a meaningful and value-guided life.

Outcome measures

We assessed whether changes in ASI and BAFT during treatment mediated outcomes at post-treatment. Given that CBT focused explicitly on symptom reduction whereas ACT focused on broader goals of valued living, we investigated both anxiety disorder specific (e.g., symptom reduction related) and nonspecific or broader outcomes across both treatments. Clinical severity ratings for the principal anxiety disorder diagnosis, clinically relevant worry, and anxiety-related behavioral avoidance comprised the anxiety disorder specific outcomes. Worry and behavioral avoidance were selected because they represent central shared features of anxiety disorders (Craske, Rauch, et al., 2009). Quality of life and depression symptoms[7] comprised the nonspecific, broader outcomes. The questionnaires assessing the outcome indices are described briefly. At pre-treatment, CBT and ACT did not differ on any outcomes, $ps \geq .10$.

Anxiety disorder specific outcomes

Diagnostic interview assessment. The information extracted from the ADIS-IV-R included 'clinical severity ratings' (CSR; distress and disability) made by group consensus on a 0 to 8 scale (0 = none, 8 = extreme) for each disorder. Baseline ratings of "4" or higher indicated a clinically significant degree of distress or disability; ratings of 4 or higher for at least one anxiety disorder served as the cutoff for study eligibility (see Craske, DeCola, Sachs, & Pontillo, 2003; Craske et al., 2007). Averaging across the various principal anxiety disorders, the CSR pre-treatment mean was 5.7 (SD = .93, range 4–8) and post-treatment mean was 3.0 (SD = 2.2, range = 0–7). Pre to post reductions in principal anxiety disorder CSR ratings did not differ by treatment group (see Arch et al., in press, for more detailed analyses).

To assess inter-rater reliability, ADIS-IV-R assessments were audiotaped and 15% of Ps in the parent study ($n = 22$) were selected at random for blind ratings by a second rater. One-way intraclass correlation coefficients for diagnostic status (clinically significant distress and disability [CSR of 4+] vs. subclinical [CSR of 1–3] vs. none [CSR of 0]) were social anxiety disorder and obsessive compulsive disorder ICC = 1.00 (100% agreement), panic disorder ICC = .91, generalized anxiety disorder ICC = .85, and specific phobia ICC = .75. Dimensional CSRs across all principal diagnoses evidenced an ICC of .65[8] (Arch et al., in press).

The *Penn State Worry Questionnaire* (PSWQ; Meyer, Miller, Metzger, & Borkovec, 1990) is a 16-item measure of trait worry which focuses on the intensity, excessiveness, and uncontrollability of clinically relevant worry (Molina & Borkovec, 1994). The PSWQ demonstrates good internal consistency (α of .86 to .93 across clinical and college samples) and test–retest reliability (r = .74–.93 across 2–10 week periods). Although particularly elevated in GAD, the PSWQ shows elevations across all anxiety disorders relative to non-anxious controls (Brown, Antony, & Barlow, 1992). The current sample demonstrated αs of .89 (pre-treatment) and .93 (post-treatment).

The *Fear Questionnaire* (FQ; Marks & Mathews, 1979) is a questionnaire that assesses fear, anxiety, and behavioral avoidance among different anxiety disorders. The Main Target Phobia Scale, an avoidance rating for each P's "main phobia", was used as the behavioral avoidance outcome.

Broader outcomes

The *Quality of Life Inventory* (Frisch, 1994) is a 32-item questionnaire assessing life satisfaction across 16 broad life domains.

[6] For the second half of the study, ACT supervision was led by a highly ACT-experienced doctoral candidate working with Dr. Steven Hayes at the University of Nevada at Reno.

[7] We also assessed experiential avoidance/acceptance with the 16-item Acceptance and Action Questionnaire (AAQ; Bond & Bunce, 2000; Hayes et al., 2004) and considered using it as a broader treatment outcome. At post-treatment, however, the AAQ and full BAFT correlated at r = .71, indicating a high degree of overlap between these measures. Due to this overlap, we did not use the AAQ as an outcome measure in the mediator analyses. For analyses of AAQ outcomes, see Arch et al. (in press).

[8] The ICC across all principal diagnoses was lower than for individual diagnoses because the individual diagnoses included no symptoms (e.g., none) and were rated on a simpler 3-point scale (relative to the 0–8 CSR scale), which promoted a higher level of agreement.

Quality of life is calculated by multiplying an importance rating by satisfaction rating for each area and summing them to produce an overall quality of life score. Psychometric properties include good test–retest reliability (at 2-week intervals $r = .73$), as well as good convergent, discriminant, and treatment validity (Frisch et al., 2005). The αs in the current sample were .83 (pre-treatment) and .85 (post-treatment).

The *General Depression scale, Mood and Anxiety Symptom Questionnaire* (Watson & Clark, 1991) is a 12 item measure of depression symptoms that demonstrates good divergent ($r = .02–.09$) and convergent validity ($r = .67–.76$) with other depression scales and strong incremental validity across student, adult, and patient samples (Watson, Clark, et al., 1995; Watson, Weber, et al., 1995). Current αs were .88 (pre-treatment) and .94 (post-treatment).

Results

Analyses were performed in HLM 6.0 (Raudenbush, Bryk, Cheong, & Congdon, 2004) and SPSS 16.0. Analyses included Ps with post-treatment data as well as at least one session-by-session data point. Thus, Ps with missing data were included in the analyses as long as they met these criteria.[9] ASI and BAFT data points refer to alternating-session scores on the ASI and BAFT (7-item version) from baseline through Session 10 (6 total data points). We computed effect sizes (d) based on the magnitude effect size formula for HLM analyses presented by Feingold (2009) based on earlier work by Raudenbush and Liu (2001). Statistical significance was defined as $p < .05$.

We assessed whether the presence or amount of missing data predicted any of the post-treatment outcomes that were linked to the mediators (covarying outcome measures at pre-treatment). On average, 32.4% of data was missing at each session-by-session time point; the majority of Ps (83.6%) had 2 or fewer sessions of missing data. Importantly, the *presence* of missing data did not predict any of the post-treatment outcomes (all $ps > .13$). The *amount* of missing data for each participant also did not predict behavioral avoidance, depression, or quality of life outcomes ($ps > .11$), though approached significance for worry outcomes ($p = .06$). In summary, there were no statistically significant associations between missing data and the predicted outcomes.

Decline in ASI and BAFT across assessment periods during treatment

A series of 2-level hierarchical linear growth curve models (HLMs) were conducted with ASI or BAFT during treatment as the outcome variable, Time as the level-1 predictor, and Group (CBT vs. ACT) as the level-2 predictor. In the first model, baseline ASI scores (i.e. intercept) were borderline significantly higher in ACT, $\beta = 33.59$, than in CBT, $\beta = 28.48$, between group $\beta = 5.12$, $t(65) = 1.96$, $p = .053$, $d = .47$. ASI significantly declined across time in CBT, $\beta = -2.16$, $t(65) = -5.46$, $p < .001$, $d = -1.00$[10], and in ACT, $\beta = -3.70$, $t(65) = 7.34$, $p < .001$, $d = -1.71$ with those in ACT showing steeper ASI decline slopes than CBT, $\beta = -1.55$, $t(65) = -2.80$, $p = .007$, $d = -.71$ (see Fig. 1a).

In the second model, baseline BAFT scores did not differ between CBT, $\beta = 18.75$, and ACT, $\beta = 18.45$, between group $\beta = .30$, $t(65) = .33$, $p = .74$, $d = .08$. BAFT significantly declined across time[11] in CBT, $\beta = -1.36$, $t(65) = -6.39$, $p < .001$, $d = -1.85$, and in ACT, $\beta = -1.96$, $t(65) = -9.27$, $p < .001$, $d = -2.67$, with ACT showing nearly significantly steeper BAFT decline slopes than CBT, $\beta = -.60$, $t(65) = -1.99$, $p = .050$, $d = .82$ (see Fig. 1b).

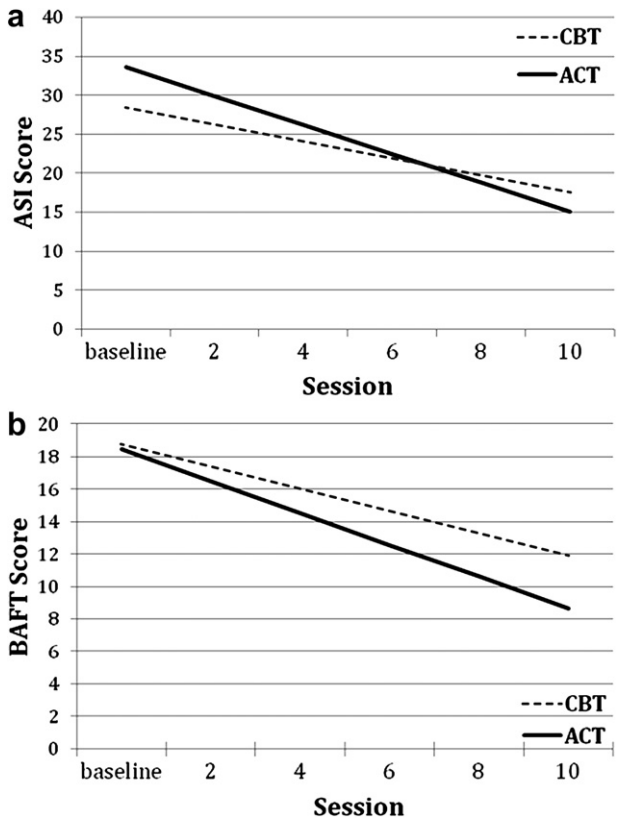

Fig. 1. a. Change in anxiety sensitivity during treatment. b. Change in cognitive fusion during treatment.

BAFT and ASI as mediators of treatment outcome

Generalized least squares estimates analysis was performed within HLM to obtain *y*-intercept (i.e. baseline score on ASI or BAFT) and decline slope (i.e. change over time on ASI or BAFT during treatment) parameter estimates for each participant. These slope and intercept parameters were then entered as predictors into a series of linear regression analyses (described below). We used the MacArthur guidelines as outlined by Kraemer et al. (2002) to test for mediation. In this approach, a variable can be considered a mediator of treatment outcome if: (a) it is measured during treatment; (b) rate of change during treatment is correlated with treatment Group; and (c) it either has a direct relation with the outcome variable or interacts with the treatment Group in its relation to the outcome.

For all analyses including BAFT and ASI slopes as putative mediators, criterion (a) was clearly met, as these variables were measured during treatment. Criterion (b) was also met since change in BAFT and ASI during treatment correlated with Group (see Results above; albeit only a borderline significant [$p = .050$] group effect for the BAFT). Therefore, for each mediation analysis below, we focus on whether criterion (c) was met. If criterion (c) is

[9] Ps who were missing all mediator and post-treatment data, i.e. those who did not begin treatment or who were treated prior to the implementation of the mediator measures, were logically excluded because it did not make sense to examine treatment mediators for Ps who lacked treatment data.

[10] Effect sizes characterize change *during* treatment, that is, from baseline to Session 10 (of 12 Sessions), and thus exclude post-treatment data. Assuming a constant linear change rate, they are approximately 20% smaller than if post-treatment data were included.

[11] Please note that declines in BAFT indicate *increases* in cognitive defusion (i.e. decreases in cognitive fusion).

met for the ASI analyses below, the requirements for treatment mediation were met fully; if criterion (c) is met for the BAFT analyses, the requirements for treatment mediation were met largely but our conclusions are more cautious given the partial fulfillment of criterion (b).

Regression analyses in SPSS were conducted for the *y*-intercept with the putative mediating variable (i.e. estimated baseline score on BAFT or ASI) in the first block to adjust for baseline scores on these measures, Group and BAFT or ASI slope (i.e. main effect terms) in the second block, and their interaction term (BAFT or ASI slope × Group) in the third block. That is, the Group × Time interaction effect was tested on the final block of the regression equation. Significant Group × Time interaction effects indicated mediated moderation, that is, the mediation of treatment outcome that was moderated by treatment group. If the Group × Time interaction term was non-significant, the analysis was re-run without it to examine the main effect of the mediator. Significant main effects indicated that mediation took place but was not moderated by treatment group (e.g., no moderated mediation). The dependent variable was the outcome variable at post-treatment (see Outcome Measures). Slopes represented change in the putative mediators *during* treatment (post-treatment scores were excluded from slope estimates) and therefore, temporally preceded the post-treatment outcomes that served as the dependent variable. By measuring the putative mediators *prior* to the post-treatment outcomes, we have established their temporal precedence. Separate analyses were conducted for BAFT and ASI mediation. Note that the reason we did not conduct the entire analysis in HLM is because our outcome variables were single data points (e.g., the post-treatment outcomes) whereas in HLM, outcome variables must be time varying or longitudinal. See Table 1 for significant regression results, which are summarized below.

Anxiety disorder specific outcomes

Does change on the BAFT and/or ASI mediate clinical severity ratings for the principal diagnosis? Within the model described above, BAFT slope × Group interaction did not significantly predict principal diagnosis CSR at post-treatment ($p = .72$). A simplified model removing the interaction term was conducted. Still there was no significant main effect of BAFT slope on CSR at post-treatment ($p = .21$). Similarly, in the final model described above, the ASI × Group interaction did not significantly predict principal diagnosis CSR at post-treatment ($p = .21$); nor did a simplified model removing the interaction term, ASI slope ($p = .47$). Thus, neither putative mediator was found to mediate the primary outcome measure of clinical severity.

Does change on the BAFT and/or ASI mediate change in worry symptoms? A significant interaction of BAFT slope × Group was observed in predicting post-treatment PSWQ, $\beta = -8.98$, $t(39) = -2.25$, $p < .05$, Adjusted $R^2 = .44$, $\Delta R^2 = .07$. Thus, criterion (c) was met and BAFT served as a mediator of change in worry outcomes. The nature of this interaction was observed by plotting estimated regression lines, which showed that overall, BAFT decline was associated with lower PSWQ scores, and that those in CBT with steeper BAFT decline slopes had lower PSWQ scores than those in ACT with similarly steep decline slopes (see Fig. 2).

An identical model was run with ASI variables. The ASI slope × Group interaction was not significant ($p = .89$). A simplified model removing the interaction term was conducted to examine main effects. ASI slope significantly predicted PSWQ scores at post-treatment, $\beta = 11.73$, $t(39) = 2.95$, $p < .01$, Adjusted $R^2 = .34$, $\Delta R^2 = .15$, such that steeper ASI decline slopes over treatment were associated with lower PSWQ scores at post-treatment across both groups. This result fulfilled the criterion (c) requirement

Table 1
Linear regression statistics for mediation analyses.

	B	SE	*t*	*p*
BAFT as a mediator of PSWQ outcome				
Model 1 (interaction)				
Pre-treatment PSWQ	.30	.16	1.87	<.01
BAFT intercept	3.50	1.99	1.75	.09
BAFT slope	15.26	3.79	4.03	<.001
Group	−8.11	3.90	−2.08	<.05
BAFT slope × Group	−8.99	4.00	−2.24	.03
ASI as a mediator of PSWQ outcome				
Model 1 (interaction)				
Pre-treatment PSWQ	.47	.16	2.88	<.01
ASI intercept	2.64	2.47	1.07	.29
ASI slope	12.01	4.54	2.64	.01
Group	−6.20	4.40	−1.41	.17
ASI slope × Group	−.41	3.04	−.13	.89
Model 2 (main effects)				
Pre-treatment PSWQ	.47	.16	2.92	<.01
ASI intercept	2.67	2.42	1.10	.28
ASI slope	11.73	3.98	2.95	<.01
Group	−6.11	4.28	−1.43	.16
BAFT as a mediator of FQ1 outcome				
Model 1 (interaction)				
Pre-treatment FQ1	−.001	.003	−.28	.78
BAFT intercept	1.01	.59	1.71	.10
BAFT slope	.87	.84	1.04	.31
Group	−1.14	1.00	−1.13	.27
BAFT slope × Group	1.27	1.01	1.25	.22
Model 2 (main effects)				
Pre-treatment FQ1	−.001	.003	−.44	.66
BAFT intercept	.99	.60	1.66	.11
BAFT slope	1.46	.70	2.10	.04
Group	−1.08	1.01	−1.06	.30
ASI as a mediator of FQ1 outcome				
Model 1 (interaction)				
Pre-treatment FQ1	−.001	.003	−.36	.73
ASI intercept	−.15	.57	−.27	.79
ASI slope	1.25	1.14	1.10	.28
Group	−1.09	1.04	−1.05	.30
ASI slope × Group	.73	.87	.83	.41
Model 2 (main effects)				
Pre-treatment FQ1	−.001	.003	−.45	.66
ASI intercept	−.14	.57	−.25	.81
ASI slope	1.73	.99	1.75	.09
Group	−1.09	1.03	−1.05	.30
BAFT as a mediator of QOLI outcome				
Model 1 (interaction)				
Pre-treatment QOLI	.66	.10	6.30	<.001
BAFT intercept	−12.73	3.34	−3.81	.001
BAFT slope	−10.87	5.86	−1.86	.07
Group	3.82	5.75	.66	.51
BAFT slope × Group	−2.54	6.78	−.38	.71
Model 2 (main effects)				
Pre-treatment QOLI	.65	.10	6.44	<.001
BAFT intercept	−13.00	3.23	−4.03	<.001
BAFT slope	−12.51	3.84	−3.26	<.01
Group	4.04	5.65	.71	.48
BAFT as a mediator of MASQ general depression outcome				
Model 1 (interaction)				
Pre-treatment MASQ	.85	.67	5.79	<.001
BAFT intercept	3.14	.26	1.74	.09
BAFT slope	3.26	.24	1.18	.25
Group	−2.42	−.09	−.75	.46
BAFT slope × Group	2.00	.11	.63	.53
Model 2 (main effects)				
Pre-treatment MASQ	.83	.14	5.79	<.001
BAFT intercept	3.14	1.79	1.75	<.001
BAFT slope	4.43	2.04	2.17	<.05
Group	−2.65	3.19	−.83	.41

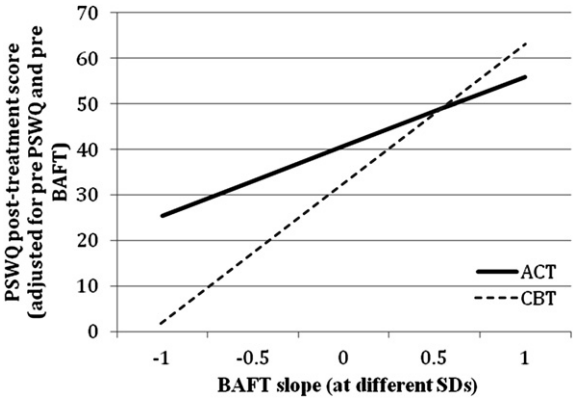

Fig. 2. Cognitive defusion × group interaction in predicting post-treatment worry.

for mediation and ASI was considered a mediator of worry outcomes.

Does change in BAFT and/or ASI mediate changes in behavioral avoidance? There was no significant BAFT slope × Group interaction on FQ1 ($p = .22$). The simplified model revealed a significant main effect of BAFT slope on FQ1, $\beta = 1.46$, $t\,(37) = 2.10$, $p < .05$, Adjusted $R^2 = .03$, $\Delta R^2 = .13$,[12] such that steeper BAFT decline slopes were associated with lower FQ1 at post-treatment. This result fulfilled the criterion (c) requirement for mediation and BAFT was considered a mediator of behavioral avoidance outcomes. In contrast, there was neither an ASI slope × Group interaction on FQ1 ($p = .41$) nor a significant main effect of ASI slope on FQ1 ($p = .09$). However, the main effect of ASI slope on FQ1 was in the same direction as that of the BAFT slope, albeit not statistically significant, Adjusted $R^2 = .02$, $\Delta R^2 = .09$.

Broader outcomes

Does change on the BAFT and/or ASI mediate change in quality of life? The BAFT slope × Group interaction did not significantly predict quality of life at post-treatment ($p = .71$). After removing the interaction term, there was a significant main effect of BAFT slope, $\beta = -12.52$, $t\,(41) = 3.56$, $p < .01$, Adjusted $R^2 = .61$, $\Delta R^2 = .10$, such that steeper BAFT declines were associated with increases in quality of life. This result fulfilled the criterion (c) requirement for mediation and BAFT was considered a mediator of quality of life outcomes. The ASI × Group interaction did not significantly predict quality of life ($p = .42$). In the simplified model, ASI slope did not significantly predict quality of life at post-treatment ($p = .07$) but the effect was in the same direction as that of the BAFT slope, Adjusted $R^2 = .48$, $\Delta R^2 = .05$.

Does change on the ASI and/or BAFT mediate Depression outcomes? The BAFT slope × Group interaction did not significantly predict MASQ General Depression ($p = .53$). In the simplified model, the BAFT slope significantly predicted depression scores at post-treatment, $\beta = 4.43$, $t\,(42) = 2.17$, $p < .05$, $\Delta R^2 = .06$, such that steeper BAFT decline slopes were associated with lower depression at post-treatment. This result fulfilled the criterion (c) requirement for mediation and BAFT was considered a mediator of depression outcome.

The models including ASI variables yielded no significant interaction or main effect terms for MASQ General Depression scores ($ps > .43$). Thus, ASI did not mediate depression outcomes.

Discussion

Our analyses addressed two central research questions regarding CBT or ACT for the treatment of a heterogeneous anxiety disorder sample. First, we addressed whether CBT and ACT affected the purported mediators for each treatment (anxiety sensitivity in CBT, cognitive defusion in ACT). Both mediators evidenced large effect size improvements during both treatments. ACT resulted in greater improvements than CBT, of medium to large effect size, for both anxiety sensitivity and cognitive defusion. Group differences in cognitive defusion were not fully significant ($p = .05$), but were large in magnitude. The finding therefore supported our hypothesis that cognitive defusion would decline significantly and to a greater extent in ACT than CBT. Furthermore, the lack of group differences in cognitive defusion at pre-treatment clarifies that the observed effects were due to treatment. Our hypothesis that anxiety sensitivity would decline significantly and to a greater extent in CBT than ACT was not supported, however, since anxiety sensitivity declined more in ACT. At pre-treatment, ACT showed marginally higher anxiety sensitivity than CBT, suggesting that ACT's steeper improvement in anxiety sensitivity during treatment was due in part to greater room for improvement. Our second question addressed whether session-by-session changes in anxiety sensitivity and cognitive defusion mediated treatment outcomes and whether this differed by group. We categorized outcomes as anxiety disorder specific vs. broader treatment outcomes. Anxiety disorder specific outcomes included the clinical severity rating of the principal diagnosis, clinical worry, and anxiety disorder-related behavioral avoidance. Broader outcomes included quality of life and depression.

For anxiety disorder specific outcomes, neither session-by-session change in cognitive defusion nor anxiety sensitivity predicted the clinical severity ratings of the principal diagnosis at post-treatment. Their high variability at post-treatment may account for the non-significant findings on this outcome. Alternatively, processes other than the specified mediators, such as treatment credibility, homework adherence, or therapist adherence,[13] may account for changes in clinical severity. On the other hand, session-by-session change in anxiety sensitivity and cognitive defusion both mediated clinical worry outcomes. For anxiety sensitivity, steeper declines in anxiety sensitivity during treatment predicted lower worry at post-treatment across both treatment groups, with a large effect size. For cognitive defusion, a significant interaction of medium effect size demonstrated that increases in cognitive defusion during treatment predicted diminished worry at post-treatment to a greater extent in CBT than ACT. This finding was not due to steeper overall cognitive defusion increases in CBT. In fact, as noted above, the opposite is true: ACT evidenced steeper increases in cognitive defusion than CBT. This unexpected finding suggests that the ACT-specific mediator (cognitive defusion) played a more central role in predicting worry reduction in the non-ACT treatment (CBT). We return to this point below. Finally, increases in cognitive defusion during treatment significantly mediated reductions in anxiety-related behavioral avoidance at post-treatment across both treatments, by a medium to large effect size. Anxiety sensitivity did not significantly mediate behavioral avoidance outcomes, though outcomes were in the predicted direction with a smaller effect size than for cognitive defusion.

[12] Although the *Adjusted* R^2 is smaller than the ΔR^2, the *Total Unadjusted* $R^2 = .13$, which is equivalent or larger than the ΔR^2 for both ASI and BAFT models.

[13] Per Arch et al. (in press), general therapist competency ratings (i.e. general therapy skills, non-condition specific) were not related to post-treatment clinical severity ratings. Other potential predictors of clinical severity ratings will be tested and reported elsewhere.

For the broader outcomes, session-by-session change in cognitive defusion significantly mediated both quality of life and depression outcomes, whereas anxiety sensitivity was not a statistically significant mediator. For quality of life, increases in cognitive defusion mediated higher post-treatment quality of life across both treatments with a medium to large effect size. Although not statistically significant, anxiety sensitivity evidenced a borderline significant mediator effect across treatments of medium effect size. The finding that changing the relationship with anxiety-related thoughts (via cognitive defusion) was a more robust mediator of quality of life increases than changing the content of anxiety and fear-related beliefs (via anxiety sensitivity) is consistent with the ACT model. According to the ACT model of psychopathology (Hayes et al., 2006, 2012), increasing cognitive defusion results in greater psychological flexibility, or the capacity to respond flexibly according to all available options and move forward in valued life directions rather than react rigidly to the content of (anxiety-related) thoughts and other unhelpful conceptions. Psychological flexibility creates space to enact personally valued actions (rather than anxiety-driven actions), which should lead to higher quality of life. Further, by decreasing the narrow, rigid sense that life is dictated by the need to respond to anxiety-related cognitions, cognitive defusion should decrease despondency, avolition, and associated depressive symptoms. In support of this notion, increases in cognitive defusion predicted lower depression at post-treatment across both treatment groups, with a medium effect size. Cognitive defusion in ACT parallels the construct of decentering in Mindfulness Based Cognitive Therapy, in which thoughts and feelings are viewed from a detached perspective as 'mental events' that are not necessarily aspects of the self or accurate reflections of reality (Segal, Williams, & Teasdale, 2002). It is notable that even in an anxiety disorder sample, defusion/decentering mediated improvements in depression.

In summary, cognitive defusion – the capacity to not be rigidly governed by cognitive content – appears to be an important mediator of both CBT and ACT for anxiety disorders. Cognitive defusion significantly mediated all post-treatment outcomes except one (4/5 outcomes) whereas anxiety sensitivity significantly mediated only one outcome, although two others evidenced borderline significant effects. Across both ACT and CBT, therefore, cognitive defusion served as a stronger mediator of post-treatment outcomes than anxiety sensitivity. Further, in CBT, cognitive defusion more strongly mediated worry outcomes than in ACT, suggesting that even within the CBT model that does not explicitly discuss cognitive defusion, this process occurs, and underlies change to the same or greater extent than in ACT. Several CBT strategies implicitly promote cognitive defusion, including self-monitoring and cognitive restructuring, both of which aim to cultivate a 'scientific observer perspective' and therapeutic distance from anxiety-related cognitions. In ACT, cognitive defusion-based exercises more directly target this process (see Arch & Craske, 2008). Not surprisingly, given its more direct targeting in ACT, cognitive defusion increased more during ACT than CBT. Despite the different approaches to increasing cognitive defusion in each CBT and ACT, improvement on this variable represents a key therapeutic process across both therapies.

The overall findings do not provide evidence that CBT and ACT for anxiety disorders differ substantially in terms of mediation pathways, at least with regard to anxiety sensitivity and cognitive defusion. We have previously theorized that CBT and ACT for anxiety disorders share some common underlying therapeutic processes or mediators (Arch & Craske, 2008). To our knowledge, this is the first empirical test of this hypothesis.

Several study limitations should be noted. First, our outcome measures were limited to independent diagnostic evaluations and self-report questionnaires and our mediators were limited to self-report questionnaires only. Because questionnaires rely on often-limited conscious awareness of mental processes, future studies would benefit from integrating behavioral, psychophysiological, and brain-based measurements of mediators and outcomes. Second, we could not test more than one hypothesized mediator of each treatment model due to limited patient time (patients had to complete mediators immediately prior to alternating therapy sessions, and often ran somewhat late for sessions). Future studies should aim to assess a variety of different potential mediators, including thought frequency, controllability, and/or content for CBT, using both implicit and explicit paradigms, and self-as-context (e.g., self-defusion), experiential avoidance, acceptance, behavioral commitments, or values clarification for ACT. Third, ACT and CBT were matched on the number of sessions devoted to behavioral exposure, which likely minimized group differences in mediation pathways. The original ACT manual (Hayes et al., 1999) did not emphasize traditional exposure methods. Fourth, for the ACT mediator we used a brief version of a little-known measure (BAFT) relative to the widely used Anxiety Sensitivity Index for CBT. Well-validated measures of ACT processes, however, were still under development when we began this study. The BAFT represents one of the best validated among them and is the only validated ACT process measure for disordered anxiety. In addition, although anxiety sensitivity is elevated among all of the anxiety disorders assessed in this study (see Taylor et al., 1992), it is higher in panic disorder and posttraumatic stress disorder (Olatunji & Wolitzky-Taylor, 2009) and therefore may represent a more robust mediator within those disorders. It is challenging, however, to find a psychometrically sound CBT-relevant mediator with strictly equal relevance across all of the anxiety disorders. As such, anxiety sensitivity may be among the best candidates. Fifth, our use of beginning therapists who were relatively inexperienced in CBT and new to ACT may have reduced the overall effectiveness of both treatments. Sixth, our mixed anxiety disorder sample limits the conclusions that can be drawn regarding mediation for a specific anxiety disorder. Future studies will need to assess whether our findings hold for individual anxiety disorders. On the other hand, given the overlapping features of anxiety disorders (Craske, Rauch, et al., 2009; Watson, 2005) a strength of this approach was that it facilitated an examination of mediation across a broader nosologic category. Seventh, although we established temporal precedence for our mediators, they were measured through session 10 and a session 10 mediator is likely to be correlated with the outcomes assessed (several weeks) after the final session (session 12). It can be challenging, therefore, to narrow precisely when one variable influenced another. Nonetheless, this study represents a preliminary step in identifying treatment mediators and progresses beyond most previous studies with regard to temporal precedence. Finally, this study was underpowered to detect between group differences that were small in magnitude. Future studies should aim to replicate the present findings in larger samples.

In summary, this study, to our knowledge, represents the first empirical investigation of treatment mediation in CBT vs. ACT for anxiety disorders. Cognitive defusion emerged as a robust mediator of outcomes within both CBT and ACT; anxiety sensitivity mediated outcomes to a less significant degree. Findings are consistent with our previous assertion that treatment mediation is partially shared across both CBT and ACT for anxiety disorders (Arch & Craske, 2008). Further, findings suggest that cognitive defusion contributes considerably to the success of behaviorally-based anxiety disorder treatments.

Acknowledgments

First author J.J.A. was assisted in the preparation of this manuscript by startup funds from the University of Colorado Boulder.

References

Arch, J. J., & Craske, M. G. (2008). Acceptance and commitment therapy and cognitive behavioral therapy for anxiety disorders: different treatments, similar mechanisms? *Clinical Psychology: Science and Practice, 15*(4), 263–279.

Arch, J. J., Eifert, G. H., Davies, C., Plumb, J. C., Rose, R. D., & Craske, M. G. Randomized clinical trial of cognitive behavioral therapy versus acceptance and commitment therapy for the treatment of mixed anxiety disorders. *Journal of Consulting and Clinical Psychology*, in press.

Bach, P., & Hayes, S. C. (2002). The use of acceptance and commitment therapy to prevent the rehospitalization of psychotic patients: a randomized controlled trial. *Journal of Consulting and Clinical Psychology, 70*(5), 1129–1139.

Bond, F. W., & Bunce, D. (2000). Mediators of change in emotion-focused and problem-focused worksite stress management interventions. *Journal of Occupational Health Psychology, 5*(1), 156–163.

Brown, T. A., Antony, M. M., & Barlow, D. H. (1992). Psychometric properties of the Penn State Worry Questionnaire in a clinical anxiety disorders sample. *Behaviour Research and Therapy, 30*(1), 33–37.

Butler, A. C., Chapman, J. E., Forman, E. M., & Beck, A. T. (2006). The empirical status of cognitive-behavioral therapy: a review of meta-analyses. *Clinical Psychology Review, 26*, 17–31.

Craske, M. G. (2005). *Cognitive-behavioral treatment of anxiety disorders*. Unpublished manuscript.

Craske, M. G. (2010). *Cognitive-behavioral therapy*. Washington, D.C.: American Psychological Association.

Craske, M. G., DeCola, J. P., Sachs, A. D., & Pontillo, D. C. (2003). Panic control treatment for agoraphobia. *Journal of Anxiety Disorders, 17*(3), 321–333.

Craske, M. G., Farchione, T. J., Allen, L. B., Barrios, V., Stoyanova, M., & Rose, R. (2007). Cognitive behavioral therapy for panic disorder and comorbidity: more of the same or less of more? *Behaviour Research and Therapy, 45*(6), 1095–1109.

Craske, M. G., Rauch, S. L., Ursano, R., Prenoveau, J., Pine, D. S., & Zinbarg, R. E. (2009). What is an anxiety disorder? *Depression and Anxiety, 26*, 1066–1085.

Craske, M. G., Rose, R. D., Lang, A., Welch, S. S., Campbell-Sills, L., Sullivan, G., et al. (2009). Computer-assisted delivery of cognitive behavioral therapy for anxiety disorders in primary-care settings. *Depression and Anxiety, 26*(3), 235–242.

Craske, M. G., Stein, M. B., Sullivan, G., Sherbourne, C., Bystritsky, A., Rose, R. D., et al. (2011). Disorder-specific impact of coordinated anxiety learning and management treatment for anxiety disorders in primary care. *Archives of General Psychiatry, 68*(4), 378–387. doi:10.1001/archgenpsychiatry.2011.25.

Dalrymple, K., & Herbert, J. D. (2007). Acceptance and commitment therapy for generalized social anxiety disorder: a pilot study. *Behavior Modification, 31*(5), 543–568.

DiNardo, P. A., Brown, T. A., & Barlow, D. H. (1994). *Anxiety Disorders Interview Schedule for DSM-IV (ADIS-IV)*. Albany, NY: Graywind Publications.

Eifert, G. H., & Forsyth, J. P. (2005). *Acceptance and commitment therapy for anxiety disorders: A practitioner's treatment guide to using mindfulness, acceptance, and values-based behavior change strategies*. New York, NY: Guilford Press.

Eifert, G. H., Forsyth, J. P., Arch, J. J., Keller, M., Langer, D., & Espejo, N. (2009). Acceptance and commitment therapy for anxiety disorders: case studies using a unified treatment protocol. *Cognitive and Behavioral Practice, 16*, 368–385.

Feingold, A. (2009). Effect sizes for growth-modeling analysis for controlled clinical trials in the same metric as for classical analysis. *Psychological Methods, 14*(1), 43–53.

Forman, E. M., Herbert, J. D., Moitra, E., Yeomans, P. D., & Geller, P. A. (2007). A randomized controlled effectiveness trial of acceptance and commitment therapy and cognitive therapy for anxiety and depression. *Behavior Modification, 31*(6), 772–799.

Forsyth, J. P., Eifert, G. H., & Barrios, V. (2006). Fear conditioning in an emotion regulation context: a fresh perspective on the origins of anxiety disorders. In M. G. Craske, D. Hermans, & D. Vansteenwegen (Eds.), *From basic processes to clinical implications*. Washington, D.C.: American Psychological Association.

Frisch, M. B. (1994). *Quality of Life Inventory (QOLI)*. Minneapolis: National Computer Systems.

Frisch, M. B., Clark, M. P., Rouse, S. V., Rudd, M. D., Paweleck, J., & Greenstone, A. (2005). Predictive and treatment validity of life satisfaction and the Quality of Life Inventory. *Assessment, 12*, 66–78.

Gaudiano, B. A., & Herbert, J. D. (2006a). Acute treatment of inpatients with psychotic symptoms using acceptance and commitment therapy: Pilot results [Peer Reviewed]. *Behaviour Research and Therapy, 44*(3), 415–437. doi:10.1016/j.brat.2005.02.007.

Gaudiano, B. A., & Herbert, J. D. (2006b). Believability of hallucinations as a potential mediator of their frequency and associated distress in psychotic inpatients [Peer Reviewed]. *Behavioural and Cognitive Psychotherapy, 34*(4), 497–502. doi:10.1017/s1352465806003080.

Hayes, S. C., Luoma, J. B., Bond, F. W., Masuda, A., & Lillis, J. (2006). Acceptance and commitment therapy: model, processes and outcomes. *Behaviour Research and Therapy, 44*(1), 1–25.

Hayes, S. C., Strosahl, K. D., & Wilson, K. G. (1999). *Acceptance and commitment therapy: An experiential approach to behavior change*. New York, NY: Guilford Press.

Hayes, S. C., Strosahl, K. D., & Wilson, K. G. (2012). *Acceptance and commitment therapy: The process and practice of mindful change* (2nd ed.). New York, NY: Guilford Press.

Hayes, S. C., Strosahl, K., Wilson, K. G., Bissett, R. T., Pistorello, J., Toarmino, D., et al. (2004). Measuring experiential avoidance: a preliminary test of a working model. *Psychological Record, 54*(4), 553–578.

Herzberg, K. N., Sheppard, S. C., Forsyth, J. P., Eifert, G. H., Crede, M., & Earleywine, M. The believability of anxious feelings and thoughts questionnaire (BAFT): psychometric properties in a healthy undergraduate and high-anxious community sample. *Psychological Assessment*, in press.

Hofmann, S. G. (2004). Cognitive mediation of treatment change in social phobia. *Journal of Consulting and Clinical Psychology, 72*(3), 392–399.

Hofmann, S. G., & Smits, J. A. J. (2008). Cognitive-behavioral therapy for adult anxiety disorders: a meta-analysis of randomized placebo-controlled trials. *Journal of Clinical Psychiatry, 69*(4), 621–632.

Hofmann, S. G., Suvak, M. K., Barlow, D. H., Shear, M. K., Meuret, A. E., Rosenfeld, D., et al. (2007). Preliminary evidence for cognitive mediation during cognitive-behavioral therapy of panic disorder. *Journal of Consulting and Clinical Psychology, 75*(3), 374–379.

Kenny, D. A., Kashy, D. A., & Bolger, N. (1998). Data analysis in social psychology. In (4th ed.).. In Gilbert, D. T., Fiske, S. T., & Lindzey, G. (Eds.), *The handbook of social psychology, vols. 1 and 2* (pp. 233–265) New York, NY, US: McGraw-Hill, x, 1085.

Kessler, R. C., Berglund, P., Demler, O., Jin, R., & Walters, E. E. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the national comorbidity survey replication. *Archives of General Psychiatry, 62*(6), 593–602.

Kraemer, H. C., Wilson, T., Fairburn, C. G., & Agras, W. S. (2002). Mediators and moderators of treatment effects in randomized clinical trials. *Archives of General Psychiatry, 59*(10), 877–883.

Lappalainen, R., Lehtonen, T., Skarp, E., Taubert, E., Ojanen, M., & Hayes, S. C. (2007). The impact of CBT and ACT models using psychology trainee therapists: a preliminary controlled effectiveness trial. *Behavior Modification, 31*(4), 488–511.

MacKinnon, D. P., Fairchild, A. J., & Fritz, M. S. (2007). Mediation analysis. *Annual Review of Psychology, 58*, 593–614.

Maller, R. G., & Reiss, S. (1992). Anxiety sensitivity and panic attacks: a longitudinal analysis. *Journal of Anxiety Disorders, 6*, 241–247.

Marks, I. M., & Mathews, A. M. (1979). Brief standard self-rating for phobic patients. *Behaviour Research and Therapy, 17*, 263–267.

Meuret, A. E., Rosenfield, D., Hofmann, S. G., Seidel, A., & Bhaskara, L. (2010). Respiratory and cognitive mediators of treatment change in panic disorder: evidence for intervention specificity. *Journal of Consulting and Clinical Psychology, 78*(5), 691–704.

Meuret, A. E., Rosenfield, D., Hofmann, S. G., Suvak, M. K., & Roth, W. T. (2009). Changes in respiration mediate changes in fear of bodily sensations in panic disorder. *Journal of Psychiatric Research, 43*, 634–641.

Meyer, T. J., Miller, M. L., Metzger, R. L., & Borkovec, T. D. (1990). Development and validation of the Penn State Worry Questionnaire. *Behaviour Research and Therapy, 28*, 487–495.

Molina, S., & Borkovec, T. D. (Eds.). (1994). *The Penn State Worry Questionnaire: Psychometric properties and associated characteristics*. Oxford, England: John Wiley & Sons.

Moscovitch, D. A., Hofmann, S. G., Suvak, M. K., & In-Albon, T. (2005). Mediation of changes in anxiety and depression during treatment of social phobia. *Journal of Consulting and Clinical Psychology, 73*(5), 945–952.

Olatunji, B. O., & Wolitzky-Taylor, K. B. (2009). Anxiety sensitivity and the anxiety disorders: a meta-analytic review and synthesis [Peer Reviewed]. *Psychological Bulletin, 135*(6), 974–999. doi:10.1037/a0017428.

Orsillo, S. M., & Batten, S. V. (2005). Acceptance and commitment therapy in the treatment of posttraumatic stress disorder. *Behavior Modification, 29*(1), 95–129.

Peterson, R. A., & Reiss, S. (1992). *Anxiety sensitivity index manual* (2nd ed.). Worthington, OH: International Diagnostic Systems.

Raudenbush, S. W., Bryk, A. S., Cheong, Y. F., & Congdon, R. T. (2004). *HLM 6: Hierarchical linear and nonlinear modeling*. Lincolnwood, IL: Scientific Software International.

Raudenbush, S. W., & Liu, X.-F. (2001). Effects of study duration, frequency of observation, and sample size on power in studies of group differences in polynomial change. *Psychological Methods, 6*(4), 387–401.

Reiss, S., Peterson, R., Gursky, D., & McNally, R. (1986). Anxiety sensitivity, anxiety frequency, and the prediction of fearfulness. *Behavioural Research and Therapy, 24*, 1–8.

Roemer, L., Orsillo, S. M., & Salters-Pedneault, K. (2008). Efficacy of an acceptance-based behavior therapy for generalized anxiety disorder: evaluation in a randomized controlled trial. *Journal of Consulting and Clinical Psychology, 76*(7), 1083–1089.

Segal, Z. V., Williams, J. M., & Teasdale, J. D. (2002). *Mindfulness-based cognitive therapy for depression: A new approach to preventing relapse*. New York, NY: Guilford Press.

Smits, J. A. J., Powers, M. B., Cho, Y., & Telch, M. J. (2004). Mechanism of change in cognitive-behavioral treatment of panic disorder: evidence for the fear or fear mediational hypothesis. *Journal of Consulting and Clinical Psychology, 72*(4), 646–652.

Smits, J. A. J., Rosenfeld, D., McDonald, R., & Telch, M. J. (2006). Cognitive mechanisms of social anxiety reduction: an examination of specificity and temporality. *Journal of Consulting and Clinical Psychology, 74*(6), 1203–1212.

Taylor, S., Koch, W. J., & McNally, R. (1992). How does anxiety sensitivity vary across the anxiety disorders? *Journal of Anxiety Disorders, 6*, 249–259.

Tolin, D. F. (2010). Is cognitive behavioral therapy more effective than other therapies?: a meta-analytic review. *Clinical Psychology Review, 30*(6), 710–720.

Twohig, M. P., Hayes, S. C., & Masuda, A. (2006). Increasing willingness to experience obsessions: acceptance and commitment therapy as a treatment for obsessive-compulsive disorder. *Behavior Therapy, 37*(1), 3–13.

Twohig, M. P., Hayes, S. C., Plumb, J. C., Pruitt, L. D., Collins, A. B., Hazlett-Stevens, H., et al. (2010). A randomized clinical trial of acceptance and commitment therapy vs. progressive relaxation training for obsessive compulsive disorder. *Journal of Consulting and Clinical Psychology, 78*(5), 705–716.

Watson, D. (2005). Rethinking the mood and anxiety disorders: a quantitative hierarchical model for DSM-V. *Journal of Abnormal Psychology, 114*(4), 522–536.

Watson, D., & Clark, L. A. (1991). *The mood and anxiety symptom questionnaire*. Unpublished Manuscript. University of Iowa, Department of Psychology, Iowa City.

Watson, D., Clark, D. A., Weber, K., Assenheimer, J. S., Strauss, M. E., & McCormick, R. A. (1995). Testing a tripartite model: II. Exploring the symptom structure of anxiety and depression in student, adult, and patient samples. *Journal of Abnormal Psychology, 104*, 15–25.

Watson, D., Weber, K., Assenheimer, J. S., Clark, L. A., Strauss, M. E., & McCormick, R. A. (1995). Testing a tripartite model. I. Evaluating the convergent and discriminant validity of anxiety and depression symptom. *Journal of Abnormal Psychology, 104*, 3–14.

Zettle, R. D., & Hayes, S. C. (1986). Dysfunctional control by client verbal behavior: the context of reason giving. *The Analysis of Verbal Behavior, 4*, 30–38.