THE RELATIVE IMPACT OF EXPOSURE AND COGNITIVE EXTENSIVENESS
IN SESSION-BY-SESSION AND POST-TREATMENT CBT OUTCOMES
FOR YOUTH ANXIETY

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MINA YADEGAR

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APPROVED:  
Brian C. Chu, Ph.D.

Brenna H. Bry, Ph.D.

DEAN:  
Francine Conway, Ph.D.
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Abstract

Although Cognitive Behavioral Therapy (CBT) represents the gold standard treatment for pediatric anxiety disorders, research has indicated opportunities to further advance its effectiveness and efficacy. Studies identifying the core active CBT ingredients and moderators of treatment outcome can facilitate such advancement. The current study utilized an observational coding measure to evaluate the relative strength of therapist Exposure and Cognitive Extensiveness associated with session-by-session and post-treatment outcomes. Participants (aged 8-17) were 73 youth with a principal anxiety disorder diagnosis who completed a manual-based CBT protocol (Coping Cat; Kendall & Hedtke, 2006). Video recordings of two exposure sessions per participant was observed and coded for Exposure and Cognitive Extensiveness. Anxiety symptoms were rated by clinicians at pre- and post-treatment, as well as by children and parents prior to each session and at post-treatment. Session-by-session multiple regression analysis indicated a trend for Exposure Extensiveness to be associated with increased child and parent-reported symptom severity in the first half of exposure sessions (Sessions 9-12), and statistically significant child-reported improvement in the second half of exposure sessions (Session 13-16). Although Cognitive Extensiveness was not associated with outcomes when analyzing the sample as a whole, age and frequency of negative automatic thoughts significantly moderated the relationship between average Cognitive Extensiveness and post-treatment clinician-rated outcomes.
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The Relative Impact of Exposure and Cognitive Extensiveness 

in Session-By-Session and Post-Treatment CBT Outcomes for Youth Anxiety

Anxiety disorders affect 6-18% of youth and cause significant impairment in school, family, and social functioning (Woodward & Fergusson, 2001). Although Cognitive Behavioral Therapy (CBT) is an empirically supported treatment for anxiety disorders, it is only considered “probably efficacious” (Silverman, Pina & Viswesvaran, 2008) with a vast range of diagnostic recovery rates that range from 25 to 79%. This wide range, along with a considerable non-response rate, suggests that there is still room for improvement. Identifying the critical components of CBT can augment the efficacy of youth anxiety disorder treatments.

CBT combines cognitive and behavioral exposure strategies to target youth anxiety. Exposure therapy is rooted in the negative reinforcement cycle of avoiding anxiety-provoking situations to temporarily decrease anxiety, thereby maintaining avoidance and anxiety in the long-term. Exposure to feared situations breaks the negative reinforcement cycle, and promotes habituation of anxiety (Foa & Kozak, 1986) and distress tolerance (Craske et al., 2008) over time. Exposures are graduated according to each patient’s fear hierarchy so that easier exposures are targeted first, which are followed by increasingly difficult exposures. In order to promote generalizability outside of the session and increase success, the patient practices the exposures on a daily basis in between sessions.

The cognitive component of CBT focuses on identifying, challenging, and modifying anxious thoughts, and then creating more rational coping thoughts. Current protocols utilize the cognitive component to support and complement exposure therapy, as coping statements and other cognitive strategies (e.g., probability estimation) are utilized to prepare, push through, and debrief after exposures (Salkovskis, 1996). In this way, discussions prior to exposures challenge
patients’ assumptions to reduce their anxiety (Salkovskis, 1996). According to cognitive theorists, these cognitive tools teach patients to manage anxiety, without which patients may be more resistant to complete exposures (Butler, Cullington, Munby, Amies, & Gelder, 1984).

The mechanism of exposure therapy for anxiety disorders, proposed by Rachman's (1980) Emotional Processing Theory and later expanded by Foa and Kozak (1986), is that exposing oneself to one’s fears activates the fear structure, which then leads to habituation and fear reduction. A fear structure is a series of cognitive representations about a feared object, situation, or physiological reaction, which are stored in one's memory and are activated when faced with one's fears (Foa & Kozak 1986; Lang, 1971). The activation of the fear structure by exposure allows the integration of corrective information that is incompatible with the fear structure (stimulus-response dissociation; Foa & Kozak, 1986). This results in a modified structure that replaces (Foa & Kozak, 1986) or competes with (Foa & McNally, 1996) the original fear structure. Thus, corrective learning or habituation takes place based on classical conditioning, in which the original excitatory association (CS [conditioned stimulus] – US [unconditioned stimulus]) is undermined and erased (or "un-learned"). Learning, which in Foa and Kozak’s (1986) terms is equivalent to structural change, occurs as exposures promote lasting fear reduction.

The corrective information is learned by both within-session habituation (WSH, or short-term habituation) and between-session habituation (BSH, or long-term habituation) of the fear response. WSH measures the decline in anxiety levels during one exposure, whereas BSH measures the decline in anxiety levels from the first to the last exposure. WSH disconfirms an individual’s belief that anxiety is persistent, indicating that anxiety decreases without engaging in avoidance behaviors. BSH, through repeated exposures, weakens links between the feared
situation and associated threat representations, thus replacing elements of the preexisting fear structure and disconfirming beliefs about the feared situation. Through repeated exposures, anxiety regarding a feared situation decreases as the individual learns that the feared consequences of facing an anxiety-provoking situation do not come true. Successful learning, or extinction, is measured by the decrease in fear in both WSH and BSH, and that BSH must be preceded by WSH (Foa & Kozak, 1986). Many treatment manuals for exposure therapy associate this decline in fear with treatment success, and emphasize the importance of continuing the exposure until anxiety decreases.

However, recent research has argued against the primary mechanism of habituation as a predictor of treatment success. Instead of waiting for anxiety levels to decline, Craske and Barlow (2008) recommend continuing an exposure until the patient learns that what the patient is worried about is not likely to come true, or that he or she is able to tolerate the anxiety. In comparison, tolerating anxiety lessens distress in future anxiety provoking situations. Thus, instead of maintaining an exposure until the decline of anxiety levels, Craske and colleagues (2008) recommend to continue exposures at a sustained excitation level of high anxiety, until inhibitory learning takes place, or until anxiety is tolerated. This practice is in line with the inhibitory learning approach, which proposes that the previously learned CS-US association in fear conditioning remains while a new non-threatening CS-US association is learned (Bouton, 2000; Craske et al., 2008). Similar to how contingency awareness is correlated to conditioned responding, extinction occurs through the absence of the expected adverse event (the US), so that patients learn that the fear (CS) is not dangerous (Craske et al. 2008). In this way, new learning inhibits the original excitatory association (CS-US) so that the CS no longer signals the US,
thereby inhibiting the fear response (Hofmann, 2007). According to Craske et al. (2008), inhibitory learning is acquired during an exposure independent of the reduction of anxiety.

While behavioral theorists emphasize the role of avoidance to temporarily decrease anxiety (the function of the behavior), cognitive theorists argue that anxiety is stemmed within a patients’ distorted cognitions about the dangerousness of anxiety-provoking situations (content of the thought; Beck, Emery, & Greenberg, 2005; Clark, 1999). These cognitions can be evaluated into three different levels: automatic thoughts (brief and spontaneous thoughts that arise during a situation), intermediate beliefs (generalized attitudes, assumptions, and rules that control automatic thoughts), and schemas (global, generalized, and enduring beliefs that influence both intermediate and automatic thoughts). Schemas are maintained and elaborated upon overtime by situations that trigger content which are parallel to automatic thoughts. Instead of the behavioral negative reinforcement cycle of the avoidance of anxiety, cognitive theorists utilize a feedback loop that is centered on and originated from cognitive appraisals (Beck & Clark, 1997; Beck et al., 2005). These negative appraisals of danger trigger anxiety, which is further increased by (and interferes with) behavioral performance (e.g., escape). When an anxious individual perceives threat, the relevant cognitive schema is activated and used to evaluate the situation.

Within the cognitive model there are three stages of processing threat stimuli: orienting, primal, and secondary elaborative reappraisal (Beck & Clark, 1997; Beck et al., 2005; Clark & Beck, 2010). Once triggered by an activating event, the threatening stimulus is involuntarily recognized within the orienting mode of the model. To increase the chance of survival, the process rapidly and efficiently evaluates potentially threatening stimuli. For anxious individuals, the orienting mode is biased toward detecting negative stimuli, thus creating a tendency to be
more focused on negative stimuli rather than neutral or positive stimuli. Once a negative stimulus is recognized, the primal mode (a constellation of interconnected schemas) is activated, as the focus is centered on increasing safety and decreasing potential danger. Consequently, the following primal responses are triggered: fight or flight, behavioral mobilization and inhibition (escape and avoidance), fear, hypervigilance towards threat cues, and primal thinking (constriction of thoughts centered around the potential threat stimuli). These thoughts are skewed to be hypersensitive to threats that are overestimated in severity of outcome and probability of occurrence, leading to catastrophizing automatic thoughts. As the individual engages in secondary elaborative reappraisal, information processing is more controlled, effortful, and semantic-centered. Other schemas of the self and the world are activated as the individual considers the current context and one’s coping resources. Within this stage, constructive reappraisal and a resultant decrease in anxiety is possible as the individual is better able to rationally evaluate the probability and severity of the threat.

Based on Beck’s cognitive model, anxiety treatment focuses on strengthening the elaborative reappraisal process through cognitive restructuring and weakening the primal threat mode. This involves monitoring, challenging, and adjusting the individual’s thoughts to be more realistic and rational. These modified thoughts and initial assumptions are tested in exposures (referred to by cognitive therapists as “behavioral experiments”) that allow the opportunity to directly disconfirm the patients’ fears. Therefore the effectiveness of exposures relies on changes in an individual’s dysfunctional fear schemas. However, compared to behavioral therapy, exposures in cognitive therapy are not guided by the mechanism of habituation (Foa & Kozak, 1986) or distress tolerance (Craske et al., 2008), but rather as an experiment to test and collect data about a patient’s assumptions about the dangerousness of anxiety-provoking situations.
Therefore, adjusting maladaptive cognitions is viewed as the primary mechanism of change for cognitive therapy. In addition, while behaviorists focus on practicing exposures outside of the session to promote generalizability, cognitive therapists do not consider the repetition of exposures to be effective on their own (Clark, 1999).

Hofmann (2008) goes as far to say that it is “impossible to conduct successful exposure therapy without changing these cognitive processes” (p. 6). Hofmann draws on studies whereby a decrease in CS-US expectancy is correlated with decreases in CR during extinction; these results indicate that extinction is driven by changes in expectancies and beliefs. In exposure therapy, patients behaviorally challenge their expectancies (or worries) and are asked to re-evaluate harm expectancy (or CS-US expectancy) through exposures. Thus, Hofmann theorizes that treatment outcomes in exposure therapy are mediated by change in perceived harm expectancy.

This cognitive component may be incorporated into the primary process of inhibitory learning or habituation as a secondary mechanism. Similar to Hofmann’s hypothesis, through inhibitory learning patients are able to learn that the CS no longer signals the US. Thus inhibitory learning produces cognitive change by learning that the feared situation is not dangerous (i.e. a “false alarm”), or does not signal the US. For example, a child with separation anxiety learns through gradual exposure of separating from the parent that her feared outcome (harm to self or to the parent) will likely not occur. The child may also learn that the anxiety and worry are temporary as the anxiety dissipates overtime with repeated exposures through habituation.

Research likewise supports cognitive changes to mediate CBT treatment gains for adult (Smits, Powers, Cho, & Telch, 2004; Smits, Rosenfield, McDonald, & Telch, 2006) and youth anxiety disorders (Chu & Harrison, 2007; Hogendoorn et al., 2014; Kendall & Treadwell, 2007;
Lau, Chan, Li & Au, 2010; Muris, Mayer, den Adel, Roos, van Wamelen, 2009; Weersing & Weisz, 2002). For example, in a study of adult panic disorder, Smits et al. (2004) found that CBT was associated with reduction in Fear of Fear (anxiety about bodily sensations). Smits et al. (2006) studied the temporal relations between cognitive mediators and fear for socially anxious adults who received CBT with and without video feedback. The results indicated a reciprocal relationship whereby decreases in probability bias (overestimating the probability of harm) predicted decreases in fear, which in turn predicted decreases in probability bias. However, results do not indicate a uniform conclusion. Smits et al. (2006) also found that cost bias (exaggerating negative consequences) was a consequence of fear reduction, indicating that it may not mediate the effects of treatment outcome. Thus, not all cognitive variables served as critical mediators.

Hogendoorn et al. (2014) likewise found variations in the cognitive mediators of CBT for child anxiety disorders. While change in negative thoughts was not associated in symptom relief, increase in positive thoughts preceded a decrease in child-reported anxiety symptoms. Similarly, positive cognitive restructuring was followed by a decrease in parent-reported anxiety symptoms. However, as these studies utilize combined CBT approaches, it is difficult to pinpoint whether the cognitive or exposure component primarily contributed to the mediating relationship.

**Exposure as the Centerpiece of CBT**

While the majority of anxiety treatment protocols combine cognitive and exposure components, the general consensus is that exposure is the central tenet of CBT for anxiety disorders (Arch & Craske, 2009). Within the adult anxiety literature, a handful of meta-analyses have consolidated the outcomes of CBT anxiety studies. An initial meta-analysis of CBT and exposure treatment for social phobia (SP) indicated equivalent efficacy between treatment
outcomes of both modalities, although a greater number of exposure sessions produced enhanced outcomes for social phobia participants (Feske & Chambless, 1995). In a comprehensive review of a decade of meta-analytic studies of psychotherapy for anxiety disorders, Deacon and Abramowitz (2004), similarly found exposure treatment to be as effective as CBT for SP as well as Obsessive Compulsive Disorder (OCD), but not for Panic Disorder (PD), Generalized Anxiety Disorder (GAD), and Posttraumatic Stress Disorder (PTSD). However, due to the limited number of controlled outcome studies of pure cognitive treatments, effectiveness of a treatment relying solely on cognitive strategies could not be determined. Furthermore, while meta-analyses provides a broad and consolidated perspective, randomized control studies with dismantling designs further shed light on the active components of CBT.

The majority of randomized control studies that dismantle and discretely examine the efficacy of exposure and cognitive components of CBT for anxiety disorders have produced equivalent results between exposure, cognitive, and combined CBT treatments. Borkovec, Newman, Pincus, and Lytle (2002) analyzed the components of a complete CBT package for GAD by comparing 14 sessions of cognitive therapy, self-control desensitization (progressive relaxation therapy and imaginal exposure), and CBT (a combination of the first two components) treatments. At post-treatment and 2-year follow-up all treatment groups were found to be equally effective. Similar results have also been indicated within PTSD treatment studies. Tarrier et al. (1999) found no post-treatment or 6-month follow-up outcome differences between the effectiveness of imaginal exposure and cognitive therapy, with the exception of nine imaginal exposure participants experiencing an increase in symptoms at post-treatment, compared to three cognitive therapy participants, creating a significant group difference. However, this group difference dissipated at the 6-month follow-up. Imaginal exposure versus cognitive therapy for
PTSD was also compared in Lovell, Marks, Noshirvani, Trasher, and Livanou’s (2001) PTSD study, in addition to a comprehensive CBT condition that combined these components. At post-treatment, Lovell and colleagues (2001) also found no significant outcome differences between the treatment conditions.

These findings have also extended to social phobia, whereby randomized dismantling studies (Emmelkamp, Mersch, Vissia, & van der Helm, 1985; Mattick, Peters, & Clarke, 1989) indicated equivalency between groups that received in vivo exposure and cognitive treatment. While Hope, Heimberg, and Brunch (1995) also found comparable outcomes between CBT and exposure treatments, the results also suggested that subjects within the exposure treatment demonstrated some better outcomes (e.g., larger univariate effect size, greater improvement across all four social phobia measures, and a nonsignificant but notable finding in double the number of treatment responders compared to the CBT group). Although participants within the exposure treatment failed to indicate significant improvement in their subjective anxiety ratings during an individualized exposure of a role play at post-treatment compared to the CBT participants, this difference disappeared at the 6-month follow-up assessment. Hope and colleagues (1995) conclude that exposures in and of itself are likely to counter maladaptive thoughts without directly targeting the cognitions. These dismantling studies suggest that exposures are a more critical component of the effectiveness of CBT for anxiety disorders, as compared to the cognitive component.

Exposure and relapse prevention (ERP) is considered the gold standard treatment for Obsessive Compulsive Disorder (OCD; National Institute for Health and Clinical Excellence, 2005). ERP encompasses gradually facing obsessions (exposure) without engaging in the compulsions (response prevention). Olatunji et al. (2013) compared 20 sessions of ERP to
cognitive therapy for adult OCD sufferers. While the ERP condition consisted of solely of imaginal and in vivo exposures, the cognitive therapy involved psychoeducation, elicitation, and modification of intrusive thoughts followed by behavioral experiments. Although both treatments provided significant symptom relief, compared to cognitive therapy, the slope of change in OCD severity over the course of treatment and follow ups was significantly greater for ERP. At the 52-week follow-up, ERP participants also had significantly lower OCD severity scores than cognitive therapy participants. The authors suggest that although the two treatments overlap in encouraging behavioral change (through exposures and behavioral experiments), the greater amount of session time dedicated to behavior change in ERP may have produced greater gains.

Compared to the adult anxiety field, research on the active change ingredients of CBT treatment for anxious children is sparse. However similarly, the general knowledge is that exposure is the main component of CBT. In a randomized clinical trial (Kendall et al., 1997) that studied the efficacy of 16 weeks of CBT (consisting of 8 weeks of psychoeducation, relaxation, and cognitive restructuring followed by 8 weeks of exposures) to an 8-week waitlist group, mid-treatment assessments revealed no significant differences between the two groups. However, at post-treatment, the CBT group had significantly enhanced outcomes as opposed to the waitlist group, suggesting that the majority of the improvement occurred during the later exposure-focused phase of treatment. It is still unclear whether the exposure portion alone can be just as effective independently, or if the inclusion of the first half of treatment and the foundation of cognitive skills are necessary to induce such change. Furthermore, within the latter phase of treatment, exposures are viewed as “experiments” to collect “data” on evidence regarding participants’ expected worries (Kendall et al., 2005) and as an opportunity to challenge
dysfunctional cognitions (Kendall, Choudhury, Chung, & Robin, 2002). Cognitive strategies are also utilized before and after each exposure to promote the generalization of coping in other anxiety-provoking situations (Kendall et al., 2005). This blend of cognitive and exposure strategies during the latter phase of treatment makes it difficult to draw conclusions regarding the active ingredients of the treatment.

In a comprehensive review of evidence-based treatments, Chorpita, Daleiden and Weisz (2005) aggregated the common clinical elements of effective anxiety disorder treatments. In their analysis, exposure was identified as the sole “universal” component and core ingredient of child anxiety interventions, especially for specific phobias. Following this finding, Chorpita (2007) developed a modular therapy approach for anxiety disorders that highlights the importance of exposures as the “centerpiece” of the protocol and one of the four core procedures. The other core procedures (psychoeducation, fear hierarchy development) prepare the child and reviews (education of maintaining skills) the exposure component. Supplemental modules (e.g., rewards, social skills, cognitive restructuring) that follow a treatment planning flowchart may also be included in order to primarily support exposures, and also to individualize treatment according to each patient’s case conceptualization. For example, if negative misperceptions of threat or hopeless thoughts interfere with competing exposures, cognitive restructuring techniques are used to re-evaluate such thoughts prior to and in preparation for future exposures. However, cognitive restructuring is not one of the core procedures, and is thus only utilized as needed. This modular approach has indicated initial efficacy with anxious children (Chorpita, Taylor, Francis, Moffit, & Austin, 2004); the transdiagnostic modular design for anxiety, depression, and conduct problems (MATCH) has demonstrated greater outcomes than standard CBT manuals and usual care treatment (Chorpita et al., 2013; Weisz et al., 2012).
The effectiveness of exposure therapy is supported across the anxiety disorders in various formats. For example, the Social Effectiveness Therapy for Children (SET-C; Beidel, Turner, & Morris, 1998), the highest ranking intervention specifically for Social Phobia (Silverman et al., 2008), with gains maintained five years post-treatment (Beidel, Turner, & Young, 2006), is comprised of purely behavioral strategies, psychoeducation, social skills training, in vivo exposures, and peer generalization (practice sessions of the skills learned). Although it is difficult to decipher if exposure is the active ingredient within the other components of SET-C, exposures to anxiety provoking social situations are naturally embedded within social skills training and peer generalization practices, which indicate the importance of exposure therapy. Another behaviorally-based treatment for child anxiety, as well as mood disorders, is the Group Behavioral Activation Therapy (GBAT) Program, a transdiagnostic exposure-based behavioral activation protocol. GBAT showed positive results in an initial pilot study where three of four GBAT completers reached clinical remission of their principal diagnosis (Chu, Colognori, Weissman, & Bannon, 2009) and in a small randomized control trial where GBAT youth had greater post-treatment remission rates than waitlisted youth (Chu et al., 2013). Exposure therapy effectiveness has been shown even when consolidated into one three-hour session for specific phobia with gains maintained 6 months (Öst, Svensson, Hellström, & Lindwall, 2001) and one year (Ollendick et al., 2009) after treatment termination. Encouraging children to attend to the exposure (Chu et al., 2015) and anxiety-eliciting comments by therapists during exposure has also been associated with greater gains in OCD treatments (Chu et al., 2015).

Do cognitive interventions enhance exposure therapy?

Despite the above evidence indicating equivalent efficacy, and possible superiority, of exposure treatment to the more complex combined CBT treatment, some studies have suggested
that outcomes are enhanced with the addition of cognitive restructuring. For example, in a group CBT versus exposure treatment outcome study for adult social phobia, Hofmann (2004) found equivalent outcomes at post-treatment for both groups, which were similarly mediated by estimated social cost. At 6-month follow up, however, the CBT participants continued to improve in self-reported social anxiety compared to the exposure participants, indicating continued gains with the addition of cognitive therapy techniques. The exposure treatment was limited, however, to public speaking exposures, which may not have produced as generalized effect as the CBT treatment, which targeted a broader range of fears. In another group treatment efficacy study, Butler et al. (1984) found superior outcomes from an Exposure and Anxiety Management treatment (EX/AM; exposure, relaxation, distraction, and rational self-talk) vs. an Exposure and nonspecific control treatment (taking equivalent amount of time as anxiety management) for social phobia at post-treatment and 6-month follow-up. While these results suggest incorporating cognitive restructuring with exposure therapy to enhance outcomes, the medley of various techniques in addition to rational self-talk in the EX/AM condition makes it difficult to pinpoint the active treatment components.

Mattick and Peters (1988) improved on this methodological limitation by comparing group exposure therapy to a group CBT therapy that included only cognitive restructuring and in vivo exposure components for social phobia, both of which passed treatment integrity checks. The combined CBT treatment yielded significantly greater improvement at post-treatment based on clinician-rated functioning, self-rated avoidance, performance on the Behavioral Approach Test (BAT; an individualized exposure), than did the exposure treatment, but there were no significant differences in subjective fear on the BAT. Although Mattick et al. (1989) were unable to exactly replicate these findings in a study utilizing the same treatment protocols, therapists,
and outcome measures, with the addition of a cognitive-only treatment group, the authors reported that the CBT and cognitive therapy group outperformed the exposure only group.

In a more recent social phobia study comparing cognitive therapy and exposure and applied relaxation (EXP + AR), Clark et al. (2006) reported that 86% of cognitive therapy participants no longer met diagnostic criteria for social phobia at the end of treatment, compared to 45% of EXP + AR participants. Likewise cognitive therapy was significantly superior to EXP + AR on all outcome measures. Differences in treatment outcome persisted after one year of treatment termination. Overall, as research has not been able to replicate consistent findings, further research is needed to clarify the most important components of CBT for anxiety disorders.

There has been mixed and limited literature regarding the contribution of cognitive strategies when added to exposure therapy for youth anxiety treatment. Silverman et al. (1999) randomized phobic children to a control condition or one of two exposure-based conditions: contingency management (CM; positive reinforcement, shaping, extinction, and contingency contracting) or cognitive self-control (SC; cognitive restructuring, self-evaluation, and self-reward). Although all conditions indicated substantial and sustained improvement across the self-report measures, at post-treatment differences between the groups of children meeting diagnostic criteria were evident. Eighty-eight percent of SC children recovered compared to 55% and 56% in the CM and control condition respectively. These results suggest that the addition of cognitive strategies (together with self-evaluation and self-reward) enhance the effectiveness of exposure therapy.
The Current Study

This study utilized an observational coding measure to examine the relative strength of exposure and cognitive interventions in facilitating proximal and distal change in CBT for youth anxiety. Extensiveness of therapists’ use of cognitive and exposure strategies were rated by independent raters viewing weekly outpatient therapy sessions of anxious youth (ages 8-16) receiving the “Coping Cat” program (Kendall & Hедтke, 2006), a 16-session CBT program. Observational coding provides objective, highly specific, and accurate measures of therapy components adhered to in session, thus representing the gold standard in integrity research (Hogue, Liddle, & Rowe, 1996; McLeod, Southam-Gerow, & Weisz 2009; Mowbray, Holter, Teague, & Bybee, 2003). In addition research has indicated for higher adherence to be related to improved CBT treatment outcome (DeRubeis & Feeley, 1990). Despite these benefits, only a handful of studies (Benito, Conelea, Garcia, & Freeman, 2012; Chu et al., 2015; Hедтke, Kendall, & Tiwari, 2009; Morgan et al., 2013) have used observational coding to evaluate specific treatment components in relation to outcome in anxious youth. The current study draws on the strengths and further improves the methodology of these studies by analyzing both proximal (session-by-session) and distal (pre- to post-treatment) outcomes. The measure of precise session-by-session changes decreases the impact of confounding factors so that meaningful associations can be uncovered. At the same time, pre- to post-treatment changes allows us to examine if these session-by-session improvements can be maintained overtime.

Using multiple linear regressions, this study examined if therapists’ use of exposure strategies is a critical factor in effectiveness, above and beyond cognitive strategies. This hypothesis was parsed using both session-by-session and post-treatment outcomes as dependent variables. The first hypothesis predicted that therapists’ more extensive use of exposure
strategies within an exposure session, controlling for cognitive strategies, would be significantly related to greater improvements in the following week’s pre-session child and parent-report of anxiety than the prior session. Second, we predicted that these gains would be maintained, as higher average use of exposure strategies across exposure sessions would be significantly related to improved post-treatment outcomes, above and beyond the contribution of cognitive strategies. If these hypotheses are supported, therapists can be trained in simpler (exposure-heavy) and time-effective treatments, which can be more easily disseminated. The analytic approach also permitted an assessment of the inverse hypothesis: whether the extensiveness with which cognitive strategies are applied associated with short- and long-term gains above and beyond Exposure Extensiveness.

The third and fourth hypotheses considers if there are important factors that moderate the effectiveness of various interventions. It is hypothesized that cognitive interventions might be particularly important for youth who present with high frequencies of negative thinking. Previous research in the depression literature (Brent et al., 1998) suggests that youth with multiple adverse predictors, including high degrees of cognitive distortion and hopelessness, performed better after receiving CBT than systemic-behavioral family therapy or nondirective supportive therapy. In addition, across all three treatment conditions, higher levels of cognitive distortion and hopelessness predicted continued depressive episodes. Similarly, high degrees of cognitive distortion predicted poorer outcomes for panic disorder participants receiving cognitive therapy, than those with lower degrees (Meuret, Rosenfield, Seidel, Bhaskara, & Hofmann, 2010). These results suggest that greater negative thinking may require greater degrees of cognitive intervention to be effective. Thus, we hypothesize for frequency of negative automatic thoughts to moderate the relation between Cognitive Extensiveness and session-by-session and
post-treatment outcomes, such that youth with higher frequency of negative thoughts would
demonstrate a stronger relation between cognitive interventions and improvement than youth
with lower frequency of negative thoughts.

In addition to the frequency of negative thoughts, preliminary research suggests for age to
also moderate the relationship between cognitive strategies and outcome. Studies in particular
have reflected on increased cognitive and emotional maturation as children develop, and noted
for younger children’s difficulty in simultaneously experiencing opposite emotions (Harter,
1986), possibly as the ability to integrate different emotions is acquired later in childhood
between the age of 10 and 12 (Caroll & Steward, 1984). This challenge in integrating multiple
emotions, may likewise make it problematic for younger children to report both a negative
thought and then generate a positive or coping thought. As youth increase in age, cognitive
abilities develop (Alfano, Beidel, & Turner, 2002), and some cognitive errors (e.g.,
catastrophizing and personalizing) become more strongly associated with anxiety symptoms in
adolescents, compared to younger children (Weems, Berman, Silverman, & Saavedra, 2001).
This may in part be due to worries becoming more internal and stable traits across development
(Alfano et al., 2002; Graziano, DeGiovanni, & Garcia, 1979), and also increased awareness of
social evaluation during adolescence (Vasey, 1993). Literature in the effectiveness of cognitive
interventions across age is sparse, however in a study of youth aged 8-18, Brown, O’Keeffe,
Sanders, and Baker (1986) found that use of positive self-talk, during imaginal stressful
situations, significantly increased in age. Given these findings, we hypothesize that due to
growth in cognitive development over time, the association between Cognitive Extensiveness
and anxiety symptoms increase as participants’ age increases. Despite the need to identify
treatment moderators, research analyzing age and frequency of negative cognitions as a
moderator of CBT outcome for youth anxiety disorders is lacking. Identifying such moderators of treatment outcome may help clarify who may benefit the most from CBT, thereby improving the efficiency and effectiveness of anxiety treatments.

Methods

Participants

Participants were 73 youth (age 8-16, $M = 10.78$, $SD = 2.09$) who were originally part of an open effectiveness trial at a university specialty research clinic that consisted of 16-20 weeks of CBT for anxiety disorders. The sample of participants was 54.8% female, 75.3% White, 8.2% African American, 5.5% Asian American, 1.4% Latino, 8.2% multiethnicity, and 2.8% identified as another ethnicity. These participants met Diagnostic and Statistical Manual of Mental Disorders (4th ed.; DSM-IV-TR; American Psychological Association, 2000) criteria for a principal anxiety disorder, with the exception of 2 School Refusal participants. 36 youth (49.3%) met criteria for a principal diagnosis of Generalized Anxiety Disorder (GAD), 14 (19.2%) for Social Anxiety Disorder (SOC), 9 (12.3%) for Separation Anxiety Disorder (SAD), 7 (9.6%) for Specific Phobia, 5 (6.8%) for Panic Disorder (PD), and 2 (2.7%) for School Refusal. Youth with a primary diagnosis of PTSD or a non-anxiety disorder, or who have received any diagnosis of intellectual disability, autism spectrum disorder, Schizophrenia, and/or bipolar disorder were excluded from this study. History of suicide attempt within three months prior to the pre-treatment assessment, or with suicidal ideation or intent severe enough to require hospitalization were also excluded and referred to appropriate services.

Measures

*Coping Cat Adherence and Extensiveness Checklist and Coding Manual (CCAE).* The CCAE Checklist is a 12-item treatment adherence checklist adapted from Southam-Gerow et al.,
Each item reflects a specific therapeutic intervention from the Coping Cat. An additional item was added to incorporate treatment planning. The CCAE Coding Manual is an observational coding scheme that is used alongside the CCAE Checklist. The CCAE Coding Manual provides guidelines for objective coders to rate the therapist’s adherence and extensiveness of each of the interventions on a Likert-type, 0 (not at all) to 5 (highly extensive/major component of session), scale. Adherence is defined as the presence of an intervention; a rating of 2 signifies that the intervention was adherent. Extensiveness is defined as the degree, frequency, or intensity the therapist applies the intervention without taking into account the therapist’s competence or quality of executing the intervention. For each intervention, observational descriptions and examples of ratings are included to represent threshold markers. Coded interventions are not designed to be mutually exclusive to one another, as some therapist behaviors can be double coded in order to independently analyze their respective functions. This measure has shown adequate reliability (i.e., kappa=0.82) in a previous study (Southam-Gerow et al., 2010).

For the purpose of the current study, we focused on two of the rated interventions, Exposure and Cognitive (Identification and/or Modification of Anxious Self-Talk) strategies of the Coping Cat. Exposure strategies encompass active in vivo and imaginal exposures completed in session, including setting up and debriefing the exposure, as well as active role plays. An Exposure Extensiveness rating of a 2 (indicating adherence) may introduce and set-up an exposure; this rating is increased to a 3 if a “standard” exposure is executed and completed, although brief; a rating of a 5 defines exposures as the central feature of the session. Cognitive strategies include psychoeducation on thoughts and its relationship to emotions, using thought bubbles, identifying thinking traps, challenging anxious self-talk, and generating coping
thoughts. A Cognitive Extensiveness rating of a 2 (indicating adherence) may elicit anxious thoughts and some coping thoughts; this rating is increased to a 3 in a more discrete lesson about thoughts (e.g., identifying and weighing evidence for worry thoughts), their triggers, and their impact on mood; a rating of a 5 is reserved for sessions whereby cognitive techniques are clearly the major task.

Previously Collected Data (at weekly sessions and pre- and post-treatment)

Anxiety Disorders Interview Schedule for Children-Child/Parent versions (ADIS-IV; Silverman & Albano, 1996). The ADIS-IV was administered at pre and post-treatment to assess for principal and comorbid diagnoses. The ADIS-IV is a semi-structured interview with independent parents and youth interviews that have demonstrated good interviewer reliability (e.g., $\kappa = .98$, parent interview; $\kappa = .93$, child interview; Silverman & Nelles, 1988), test-retest reliability ($r = .76$; Silverman & Eisen, 1992), and sensitivity to treatment effects (e.g., Kendall et al., 1997). The anxiety disorders section of the ADIS-C/P for DSM-IV has demonstrated strong concurrent validity (Wood, Piacentini, Bergman, McCracken, & Barrios, 2002). Clinician Severity Ratings (CSR), ranging from 0 (no impairment) to 8 (disabling impairment), are determined for each diagnosis meeting pre-interference criteria. The ADIS-IV CSR residualized change score of the youth’s primary diagnosis from pre- to post-treatment will be used as an index of treatment outcome.

State-Trait Anxiety Inventory for Children – Trait – Child/Parent Versions (STAIC-T/STAIC-T-P; Spielberger, 1973; Strauss, 1987). The STAIC-T is a 20-item scale of youth-reported state (temporal and situational) and trait (enduring and stable) anxiety. Items measure frequency of anxiety symptoms (e.g., “I get a funny feeling in my stomach”) on a 3-point Likert scale (1 = almost never, 2 = sometimes, and 3 = often), yielding a total range of 20 to 60. The STAIC has
been shown to have high internal consistency, high stability for trait anxiety, and adequate validity (Spielberger, 1973). A modified parent-version of the STAIC (STAIC-T-P; Strauss, 1987) has been used as a complementary parent-rating of child’s trait and state anxiety (range = 26-78). Parents and children completed their respective STAIC ratings prior to each therapy session, and at pre- and post-treatment assessments. In the current sample, internal consistencies of Child ($\alpha = .94$) and Parent ($\alpha = .93$) STAIC were excellent.

**Child Automatic Thoughts Scale** (CATS; Schniering & Rapee, 2002). The CATS is a 40-item child-report measure designed to assess the frequency of negative self-statements (e.g., “I’m going to look silly”) in youth. Response options include a 5-point Likert scale from 0 (“not at all”) to 4 (“all the time”). The CATS was developed and validated on a wide age range of youth (7 – 16 years old) and CATS total scores discriminated between non-clinical youth and those with clinical anxiety, depression, and behavior disorders. Confirmatory factor analyses supported four distinct but strongly correlated factors relating to automatic thoughts on physical threat, social threat, personal failure and hostility. The internal consistency of the total score and subscales was high ($\alpha > .85$) and test–retest reliability at 1 and 3 months was acceptable ($r = .91$). For the purpose of the current study, the CATS total score completed at pre-treatment was used; the internal consistency was excellent ($\alpha = .97$).

**Procedure**

*Original Open Trial Procedure*

Treatment seeking youth were referred to the clinic by mental health professionals, school personnel, and parents for anxiety disorder treatment. Following an initial phone screen, those who described symptoms of anxiety were invited for an intake interview. As part of an intake battery, participants were administered the ADIS-IV, STAIC-T-C/P, and CATS as well as
additional self-report study questionnaires not included in the current study. All participants consented/assented to all procedures, including having all sessions videotaped. All procedures were approved by the university institutional review board.

Following the initial interview, eligible youth participated in a 16-20 week CBT treatment for anxiety (Coping Cat; Kendall & Hedtke, 2006). The first half of treatment focused on psychoeducation, cognitive restructuring, and relaxation strategies, while the latter half continued to practice these skills during exposure exercises. Youth and their parents completed a comprehensive assessment prior to and concluding treatment. The Coping Cat has been shown to produce reliable change in several clinical trials (Kendall, 1994; Kendall et al., 1997; Kendall, Hudson, Gosch, Flannery-Schroeder, & Suveg, 2008) and is distinguished as “probably efficacious” (Ollendick & King, 1998) and “empirically supported” (Albano & Kendall, 2002). Youth and parent participants completed symptom assessments prior to each session and at pre- and post-treatment.

Therapists were doctoral clinical psychology students who received initial training on the Coping Cat through a graduate level course and ongoing supervision by a licensed clinical psychologist with expertise in CBT for anxiety. The same group of doctoral students also interviewed youth and parents; therapists never completed the post-treatment evaluation of their own patients. All interviewers were trained to reliability on the ADIS-IV (Cohen’s $k > .80$ for all diagnoses) and received supervision for each completed assessment. All therapy sessions and interviews were videorecorded.

**Sampling Procedure for Coding**

Two exposure sessions from each participant were selected for coding. In order to sample sessions throughout the exposure phase of therapy, the exposure sessions of each case were split
in half, with sessions 9-12 comprising the first half (Segment A) and sessions 13-16 comprising the second half (Segment B). Of the videorecorded therapy sessions in each phase, one session was randomly selected for coding, creating two sessions to be coded for each case. This sampling procedure allowed for each phase to be equally represented.

Observational Coding Procedures

Reliability Training. Seven coders (one licensed psychologist, five clinical psychology doctoral students, and one post-baccalaureate) served as coders. All but one coder had experience in treating anxious youth using the Coping Cat manual. All coders received three months of didactic training on the Coping Cat protocol (Kendall & Hedtke, 2006) by a licensed psychologist with expertise in the protocol, followed by three months of reliability training on the CCAE. Coders were blind to participants’ data other than the data gathered from watching sessions, and to the hypotheses of the study (with the exception of the licensed psychologist and first author). Coders were asked to be “objective observers” collecting evidence from watching videorecorded sessions to inform their ratings. After an initial orientation of the CCAE, coders independently completed practice codes and then discussed the sessions to form consensus. To establish reliability, coders rated 5 sets (each consisting of 3 therapy sessions) until they reached an agreement level of intraclass correlation coefficients (ICC) ≥ .6 on all 12 interventions against gold-standard ratings. Two-way mixed, single measure pre-study ICC reliabilities for the relevant variables for this study were excellent: Exposure (.87) and Cognitive (.70).

Coding. Coders received lists of cases and video-recorded exposure sessions to observe and code. For the purpose of internal reliability, each coder rated two randomly selected exposure sessions for each assigned case. Coders were instructed to watch the session, in entirety, independently and to refer to the CCAE to determine ratings. In addition, coders also
met biweekly to discuss a different observed universal session to prevent drift and ensure that excellent inter-rater reliability was maintained. Ratings from universal sessions were used to compute study inter-rater reliability after completion of coding.

**Results**

**Reliability of Exposure and Cognitive Ratings**

To assess for inter-rater reliability and prevent drift, coders rated 17 universally coded sessions, following reliability training and throughout the duration of the study. Two-way mixed, single measure ICC reliabilities were excellent (Exposure = .98, Cognitive = .86), indicating reliability remained strong throughout the study.

**Descriptive Statistics**

*Extensiveness Descriptive Statistics*

For each of the 73 participants, one randomly selected session was coded for Segment A (Sessions 9-12) and Segment B (Sessions 13-16), creating a total of 146 sessions coded. No extensiveness ratings were missing. The full range of extensiveness (0-5) was observed for both exposure and cognitive ratings in Segment B (Sessions 13-16). This full range was also observed in Segment A (Sessions 9-12) for exposure ratings, but not for cognitive ratings (Min = 0, Max = 4). Mean Exposure Extensiveness was 3.32 ($SD = 1.86$) in Segment A and 2.96 ($SD = 1.89$) in Segment B, indicating that on average therapists were adherent to exposure strategies and in the middle range of the extensiveness scale, which was reasonably distributed across a normal curve. Mean Cognitive Extensiveness was 2.23 ($SD = 1.01$) in Segment A and 1.93 ($SD = 1.28$) in Segment B, suggesting that on average some cognitive strategies were used during the exposure phase of the treatment, although not as extensive as that of exposure strategies.

*Predictors’ Descriptive Statistics*
Missing outcome data included 19.18% of CATS scores, 4.24% of Parent STAIC scores, 2.97% of Child STAIC scores, and 5.48% of ADIS C/P CSR post-treatment scores. Little’s MCAR test was not significant ($\chi^2 = 206.61, df = 225, p = .81$), suggesting that data was missing at random. Missing data were imputed by an expectation maximization algorithm. No ADIS CSR pre-treatment scores were missing. Descriptive statistics for Child and Parent STAIC, CAT, as well as ADIS CSR pre- and post-treatment scores, are presented in Tables 1 and 2, respectively. The ADIS CSR pre-treatment score indicates that on average, the participants in this study have moderate to moderately severe interference ratings ($M = 5.86, SD = .89$). At post-treatment, interference ratings were on average in the subthreshold range ($M = 2.36, SD = 2.29$), however, with a wider spread of scores than at pre-treatment.

Several multiple regression analyses were calculated in order to examine the unique contribution of Exposure and Cognitive Extensiveness associated with session-by-session and pre- to post-treatment outcomes. Data was screened for violation of assumptions of linearity, homogeneity of variance, multicollinearity, normality, and independence. The data did not violate any of these assumptions.

**Hypothesis 1: Predicting Session-by-Session Outcomes**

Multiple hierarchical regressions were conducted to analyze whether Exposure and Cognitive Extensiveness predicted change in Child and Parent STAIC scores between two consecutive sessions (Session x-1 and Session x). As part of the regression, session-by-session STAIC residualized change scores were calculated, by entering STAIC at Session x as the DV and STAIC at Session x-1 (the coded session) as a covariate in Block 1. The use of residualized change scores accounts for individual differences of Session x scores. Cognitive Extensiveness
at Session x-1 was entered as a predictor in Block 1, and Exposure Extensiveness at Session x-1 was entered as a second predictor in Block 2.

In Segment A, there was a trend for Exposure Extensiveness to predict an increase in the following session’s Child ($b = 0.40, t = 1.57, p = .12$) and Parent ($b = 0.48, t = 1.62, p = .11$) STAIC score, after controlling for Cognitive Extensiveness (see Table 3). A positive $b$ reflects a positive association, where an increase in Exposure Extensiveness is associated with an increase in the severity of STAIC scores. This nonsignificant trend of Exposure Extensiveness predicting subsequent Child ($b = 0.40, t = 1.59, p = .12$) and Parent ($b = 0.48, t = 1.63, p = .11$) STAIC scores was not strengthened, even after removing Cognitive Extensiveness from the model.

In Segment B, Exposure Extensiveness did not predict change in Parent STAIC score, with ($b = 0.01, t = 0.03, p = .98$) or without ($b = -0.05, t = -0.17, p = .86$) controlling for Cognitive Extensiveness. There was a trend for greater Exposure Extensiveness to predict a decrease (and thereby improvement) in the subsequent session’s Child STAIC score ($b = -0.36, t = -1.71, p = .09$). Similarly, this trend was still nonsignificant after taking Cognitive Extensiveness out of the model ($b = -0.33, t = -1.6, p = .11$). Casewise diagnosis revealed an outlier in a Child STAIC Session x score with a standardized residual of 4.46. This outlier was also previously a missing data point which was imputed. Analysis was re-run without the outlier and indicated that greater Exposure Extensiveness, after controlling for Cognitive Extensiveness, significantly predicted decline in the following session’s Child STAIC score ($b = -0.42, t = -2.37, p = .02$). This indicates that for every 1 point increase in Exposure Extensiveness, while controlling for Cognitive Extensiveness, Child STAIC scores decrease by 0.42 points. Cognitive extensiveness was not associated with changes in the following session’s Child or Parent STAIC scores in either segment.
Hypothesis 2: Predicting Post-Treatment Outcomes

The second hypothesis predicted that higher average Exposure Extensiveness across exposure sessions would be significantly related to improved post-treatment outcomes, above and beyond the contribution of average Cognitive Extensiveness. The mean ratings across two exposure sessions (one in Segment A and one in Segment B) was calculated to represent average Exposure and Cognitive Extensiveness. Residualized change scores of pre- to post-treatment ADIS CSR, and Session 9 (the first exposure session) to Post-treatment STAIC scores, were calculated in separate hierarchical multiple regressions. Mean Cognitive Extensiveness was entered as a predictor in Block 1; mean Exposure Extensiveness was entered as a second predictor in Block 2.

Mean Exposure Extensiveness did not predict Child STAIC ($b = -0.50, t = -1.09, p = .28$), Parent STAIC ($b = 0.10, t = 0.22, p = .82$), or ADIS CSR ($b = 0.03, t = 0.18, p = .86$) post-treatment scores after controlling for mean Cognitive Extensiveness (see Table 4). Mean Exposure Extensiveness also did not predict change in Child STAIC ($b = -0.49, t = -1.08, p = .29$), Parent STAIC ($b = 0.11, t = 0.24, p = .81$), or ADIS CSR ($b = 0.03, t = 0.18, p = .86$) post-treatment scores, even without controlling for mean Cognitive Extensiveness.

Hypothesis 3: Predicting Frequency of Negative Thoughts to Moderate Cognitive Extensiveness and Outcomes

We predicted that those with higher frequency of negative automatic thoughts (measured by the CATS total score) will benefit from cognitive strategies during exposure sessions, as indicated by both session-by-session (STAIC), pre-exposure to post-treatment (STAIC) and pre-to post-treatment (ADIS CSR) outcomes. These outcomes were regressed onto their respective moderator interaction term (CATS * Session x-1 Cognitive extensiveness, CATS * Mean...
Cognitive Extensiveness), after controlling for individual main effects (CATS and Cognitive Extensiveness) as well as prior session’s STAIC, Session 9 STAIC, or pre-treatment ADIS CSR scores. This allowed us to assess whether frequency of negative automatic thoughts moderate the contribution of Cognitive Extensiveness on residualized change scores.

The moderator CATS * Session x-1 Cognitive Extensiveness did not predict session-by-session Parent or Child STAIC outcome in Segment A or Segment B (see Table 5). Similarly, the moderator CATS * Mean Cognitive Extensiveness did not predict Child or Parent post-treatment STAIC (see Table 6). However, the interaction between CATS and Mean Cognitive Extensiveness did significantly predict post-treatment ADIS CSR ratings ($b = 0.02$, $t = 2.35$, $p = .02$). To examine the direction of this moderator, an interaction plot with ADIS CSR standardized residualized change scores entered as the DV, Mean Cognitive Extensiveness as the IV, and CATS score as the Moderator was created by PROCESS macro (Hayes, 2013). Lower ADIS CSR standardized residualized change scores indicated greater improvement from pre- to post-treatment. This interaction plot (Figure 1) illustrated that for participants with high CATS scores (measured by one SD above the mean), ADIS CSR post-treatment outcomes improved as Mean Cognitive Extensiveness decreased. For participants with low CATS scores (measured by one SD below the mean), ADIS CSR post-treatment outcomes improved as Mean Cognitive Extensiveness increased. These results are contrary to our third hypothesis.

**Hypothesis 4: Predicting Age to Moderate Cognitive Extensiveness and Outcomes**

An additional moderator, Age * Cognitive Extensiveness, was also analyzed. We predicted for the association of Cognitive Extensiveness and outcomes to increase in strength as the age of participants’ increases. Session-by-session and post-treatment outcomes were regressed onto their respective moderator interactions terms (Age * Session x-1 Cognitive
extensiveness, Age * Mean Cognitive Extensiveness), while controlling for Age, Session x-1 or Mean Cognitive Extensiveness, and the prior session’s STAIC, Session 9 STAIC, and pre-treatment ADIS CSR scores.

The moderator Age * Mean Cognitive Extensiveness was significantly associated with post-treatment ADIS CSR ratings ($b = 0.34, t = 2.34, p = .02$; Table 8). An interaction plot (Figure 2) showed that for younger participants (1 SD below the mean), ADIS CSR post-treatment scores improved as Mean Cognitive Extensiveness increased. In comparison, for the higher age group (1 SD above the mean), lower Mean Cognitive Extensiveness ratings predicted greater ADIS CSR post-treatment scores. While this moderator is significant, the direction of the moderation is contrary to our hypothesis.

There was a nonsignificant trend for this age moderator to be associated with post-treatment Parent STAIC scores ($b = 0.63, t = 1.65, p = .11$). The age moderator, however, did not predict session-by-session Parent or Child STAIC outcomes in either of the segments (see Table 7), or post-treatment Child STAIC scores.

**Discussion**

This study examined the relative strength of Exposure and Cognitive Extensiveness in relation to session-by-session and post treatment outcomes. Although not statistically significant, contrary to our hypothesis, Exposure Extensiveness tended to be associated with increased session-by-session severity of parent and child-report measures, in the first half of the exposure phase (Segment A). Conversely, in the second half of the exposure phase (Segment B), Exposure Extensiveness, after controlling for Cognitive Extensiveness, predicted a statistically significant improvement in the next session according to child-report (after removing an outlier from the data). This improvement was not detected, however, by parent-report.
This possible difference in the impact of Exposure Extensiveness in the first and second half of the exposure phase, suggests that either the introduction of exposures would benefit from a low level of Exposure Extensiveness that increases over time, or that targeting exposures higher in an individual’s hierarchy produces more positive outcomes in the following session. The literature supports the latter, as greater intensity of anxiety during an exposure predicts better outcomes (Foa, Riggs, Massie, & Yarczower, 1995; Kozak, Foa, & Steketee, 1988). This is consistent with the suggestion that anxiety levels may “spike” to reflect the activation of the fear structure that is desired (Hayes, Laurenceau, Feldman, Strauss, & Cardaciotti, 2007). Preliminary research supports this theory of the benefit of an anxiety spike in prolonged exposure therapy for PTSD (Nishith, Resick, & Griffin, 2002), whereby increase in anxiety is followed by a decrease across sessions. Using multilevel growth analysis, Chu, Skriner, and Zandberg (2013) indicated that in a sample of anxious youth receiving CBT, anxiety levels were flat during the first half of exposure sessions before decreasing in the second half of exposure sessions. This decline in anxiety levels in later exposure sessions is consistent to results in the current study.

At the same time, flooding and overly high levels of anxiety may obstruct habituation (Foa et al., 1983, Foa & Kozak, 1986), as well as impede opportunities to collaborate on realistic and successful graduated exposures (Kendall et al., 2009). It is possible for the beginning sessions of Segment B to target an appropriate moderate level of anxiety, which later allows for the greater effectiveness of increasingly difficult exposures. Alternatively, it may be unrealistic to expect any improvements in general anxiety outcomes (as measured by the STAIC) to appear before treatment starts to address challenges high on the fear hierarchy. Furthermore, exposures may not initially generate positive outcomes until exposures are repeated across sessions (Foa &
Kozak, 1986). Additional research that analyzes within-session effects and temporal relationships would help clarify which of these possibilities may be at work.

Nonetheless, the majority of our predicted relationships between Exposure Extensiveness and session-by-session outcomes did not bear out. This may be due to a myriad of conceptual and methodological reasons. Conceptually, results from prior studies suggest for therapist adherence of initial sessions to predict treatment outcome. For example, DeRubeis and Feeley (1990) found that adherence to concrete cognitive techniques in Session 2 significantly predicted decreased depression symptoms in Session 12. In comparison, adherence to these same techniques from Session 4-12 did not predict outcome, although it was predicted by prior symptom change. These findings of early adherence predicting later outcome were replicated in another cognitive therapy study for depression (Feeley, DeRubeis, & Gelfand, 1999), and are also consistent with the phenomenon of “rapid response.” In a comprehensive review, Ilardi and Craighead (1994) indicated for 60-70% of total improvements in CBT efficacy studies for depression to occur in the first four weeks of treatment. Similar rapid responses have been reported by Tang and DeRubeis (1999), whereby 51% of total depression symptom decreased with median gains occurring between Session 5 and 6. Hofmann, Schulz, Meuret, Moscovitch, and Suvak (2006) found comparable rates of sudden gains mostly occurring in Session 5. These rapid responses may have contributed to some of the nonsignificant findings in the current study, which focused on later exposure sessions (Session 9-16). However, this rapid response may be more reflective of the impact of beginning treatment than the specific therapeutic components. Contradictory to an initial rapid response, Kendall et al., (1997) found no difference between waitlist and CBT conditions at midtreatment (prior to the onset of exposure sessions), although later significant post-treatment differences were evident between the complete 16 week CBT and
8 week waitlist groups. Future research would benefit from comparing rapid response rates of combined CBT, cognitive, and exposure therapies.

The nonsignificant results of Exposure Extensiveness predicting symptom change in the following session may also be due to methodological limitations of the study design. As STAIC at Session x-1 was a significant predictor of the following session’s STAIC score in all of the regression analyses, the impact of Exposure Extensiveness may have been diminished. In addition, there was a lag time between therapist’s use of cognitive and exposure strategies within a session, and the following session’s parent and child measures. Between these two time points, outcome could have been confounded by amount of between-session exposure compliance or life events (e.g., the first day of school, argument with a parent). Research has suggested that although anxiety decreases across exposures within a session, there is a rebound effect between sessions, whereby observed anxiety levels are consistent at the start of each session (Chu et al., 2015). Future studies may benefit from analyzing data collected in a smaller window of time, so that data from the DV is collected soon after the IV in order to limit the effect of these possible confounding variables. Furthermore, although the results of the current study reflect a temporal relationship, reciprocal relationships between therapeutic strategies and outcome were not examined and would allow for a clearer understanding of causation. A cross-lagged panel analysis might provide a more accurate picture of which points in therapy that exposure and cognitive strategies would be most helpful (preceding, during, or following an exposure).

In addition to a possible rebound effect, although weekly repeated child and parent-reports pinpoint specific temporal relationships with other variables, they may also have been influenced by order effects (e.g. decreased accuracy due to fatigue or boredom from completing the same questionnaire prior to each session). The influence of order effects is further
highlighted in the small Session 9-to post-treatment mean differences, and even smaller session-by-session mean differences, in child and parent reports (see Table 1). Furthermore, the ease of completing questionnaires, also come with costs of statements that reflect prototypical responses and may not accurately reflect an individual’s true symptom or thought (Cameron & Meichenbaum, 1980). The field would benefit from further research evaluating the effectiveness of cognitive and exposure strategies, perhaps utilizing objective measures that can be repeated without bias (e.g., skin conductance, heart rate, blood pressure).

Average Exposure Extensiveness across the exposure phase did not predict treatment outcome for child, parent, or clinician rated measures. It is possible these null findings are related to the relationships we found in the individual segments. For example, Exposure Extensiveness was positively related (trend) to session-by-session symptoms in Segment A, but negatively related to symptoms (greater exposure led to decreased anxiety) in Segment B. When averaged together, these associations may have cancelled out relationships between overall exposure averages and distal post-treatment outcomes. This reinforces the importance of examining multiple segments of exposure therapy. In addition, only two sessions across eight theoretically completed sessions were averaged to represent the overall Exposure Extensiveness in the exposure phase of treatment. This limited representation of two exposures may not have adequately represented the actual degree of exposure strategies implemented. Future research would benefit from a larger representation of coded exposure sessions to predict post-treatment outcomes. As observational coding is time-intensive and burdensome, future studies may utilize therapist-reported adherence as a more practical measure. Preliminary research indicates good observer and therapist-report agreement for Exposure Extensiveness (Durland, Yadegar, & Chu, 2016).
The addition of cognitive strategies did not enhance session-by-session or post-treatment outcomes. This finding is inconsistent with some of the prior adult and child anxiety research (e.g., Clark et al., 2006; Mattick & Peters, 1988; Silverman et al., 1999) that alludes to the addition of cognitive strategies to enhance the effectiveness of exposures. However, the lack of impact Cognitive Extensiveness had on outcomes in our current study, is more consistent with other studies that indicate comparable outcomes between exposure therapy and combined CBT treatments (e.g., Deacon & Abramowitz, 2004; Feske & Chambless, 1995; Hope et al., 1995), as well as treatment outcome studies that are solely behaviorally based (e.g., Beidel et al., 2006; Chu et al., 2009; Chu et al., 2013; Ollendick et al., 2009; Öst et al., 2001). In another Coping Cat study utilizing observational coding, coping behavior (which includes thought challenging and coping self-talk to manage anxiety) during exposure sessions did not predict clinician, child, or parent-rated outcomes (Hedtke et al., 2009). Similar results across measures of observed cognitive strategies, participant’s coping behavior in Hedtke et al. (2009) and therapist’s extensive use of cognitive strategies in the current study, support our hypothesis for the lack of contribution cognitive strategies add to exposure effectiveness.

The lack of impact Cognitive Extensiveness had on outcomes in the current study, as well as prior mixed findings, suggest that this relationship may be impacted by influential moderator variables. The current study investigated two possible moderators: frequency of negative automatic thoughts and age. Examination of these moderators on clinician-rated post-treatment outcomes was significant, but the direction of the moderators contradicted our hypothesis. Our results suggest for decreased Cognitive Extensiveness to be associated with greater clinician-rated outcomes for children with frequent negative thoughts. In comparison, increased Cognitive Extensiveness predicted an increase in clinician-rated outcomes, for children with low frequency
of negative thoughts. Perhaps greater focus on cognitive strategies for youth, who already endorse and are highly aware of their frequent maladaptive cognitions, further increases avoidance during exposures. Similarly, Chu et al. (2015) found a positive relation between therapist cognitive strategies and observed youth anxiety and escape during exposures for pediatric OCD. While avoidance decreases anxiety temporarily, it reinforces anxiety in the long-term (Foa & Kozak, 1986), as indicated in post-treatment outcomes. However, perhaps children with infrequent thoughts, who may avoid thinking about anxiety-provoking situations, may benefit from cognitive strategies that encourage conscious effort towards their fears.

Another significant finding was that age moderated the association between Cognitive Extensiveness and clinician-rated outcomes. Contrary to our hypothesized direction of this moderator, increased Cognitive Extensiveness was associated with greater post-treatment outcomes, for younger children; however, decreased Cognitive Extensiveness was associated with greater post-treatment improvement for older children. Perhaps this unexpected direction of the age moderator was impacted by study therapists using developmentally appropriate language and strategies (e.g., thought bubbles, cognitive cartoons) that builds on the cognitive skills developed in the pre-exposure sessions to ease young children to approach their fears. Treatment strategies can be adapted to increase their applicability and understanding in young children while still remaining adherent to the protocol (Kendall, Gosch, Furr, & Sood, E., 2008). In a CBT study for childhood Social Phobia, Spence, Donovan, and Brechman-Toussaint (2000) found that young children (age 7-9) struggled to understand the concept of challenging and testing the evidence for negative thoughts. As such, this concept was reduced and replaced with a self-instructional approach that focuses on positive self-talk (e.g., “I can do this”). Utilizing a
content focus on approaching feared situations, instead of modifying negative cognitions, may also decrease avoidance and thereby be associated with symptom improvement.

Despite prior research indicating the continued development of cognitive abilities, and limitations of such abilities in young children, in the current study, younger children benefitted from integrating cognitive techniques during exposure sessions. Researchers who suggest cognitive maturation across development (Alfano et al., 2002; Weems et al., 2001), may wonder if age and frequency of negative thoughts may be positively related, and thereby explain the similar unexpected direction in their respective moderator analysis. Further research is thereby needed to better understand the relationship these two moderators and their independent impacts on the relationship between cognitive strategies and treatment outcome.

Although results were significant for moderators to impact relationships between Cognitive Extensiveness and clinician rated outcomes, these results were not replicated in session-by-session and post-treatment child and parent measures. This may be due to the possible influence of order effects in repeated child and parent measures, as described above. The CATS moderator adds an additional limitation as the CATS was only administered at intake, leaving an extended period of time before exposures began. During this period of time, the coping skills (cognitive restructuring, relaxation, problem-solving) participants learned prior to the first exposure session may have already made an impact on their negative thoughts. Future research interested in negative thoughts moderating the relationship between cognitive strategies and exposure outcome, may benefit from administering the CATS prior to the first exposure session. Alternatively, degree of maladaptive thoughts can also be measured by ratings of observed negative self-statements prior to exposures.
Limitations

A number of limitations should be considered and have been mentioned including: the use of only two sessions to compose mean ratings of Exposure and Cognitive Extensiveness, mixed results based on outcome variables, order effects, and lag time between DV, IV, and moderator variables. Our results are also limited to the analyzed variables. In addition to therapist’s extensiveness to exposure and cognitive strategies, therapist’s competence, child’s comprehension of cognitive and exposure strategies, child’s use of internal coping statements, involvement in session, and therapeutic alliance may be confounding factors. Hedtke et al. (2009) also suggested for safety seeking behavior during exposures to be significantly associated with poorer treatment outcome. Safety seeking behaviors impedes the effectiveness of exposures, as they represent a form of avoidance and prevents complete activation of the fear structure, and thereby also prevents corrective or inhibitory learning.

As all of the participants completed the first eight sessions of the Coping Cat, and thereby were already exposed to cognitive strategies, the design of the current study does not allow us to analyze the effectiveness of exposures alone. During the exposure phase of the Coping Cat, all participants received both exposure and cognitive strategies. The absence of a control condition allows for outcomes to be influenced by external factors (e.g., time) that are not specific to the experimental condition. Future research may draw further on these findings by creating randomized dismantling studies with control conditions to compare exposure and cognitive strategies in child anxiety, while moderating for age and frequency of negative thoughts.

Conclusions

In spite of these limitations, the current study identifies novel findings that, with further research, may enhance the effectiveness and efficacy of treatment for youth anxiety. To our
knowledge, this is the first study to examine the relative strength of observed Exposure and Cognitive Extensiveness in proximal and distal CBT outcomes for child anxiety. This study supports the use of observational coding to uncover meaningful relations between therapist extensiveness and outcomes. Our findings suggest for increased Exposure Extensiveness to be associated with improvements in the latter segment of the exposure sessions. In comparison such an increase may deter improvement in the first segment. Although the reason for this contradiction is currently unclear, clinicians should be mindful of the implications for exposure to have varying impact on outcome depending on the time point of treatment or level of anxiety within an individual’s fear hierarchy. In addition, while cognitive strategies did not enhance Cognitive Extensiveness for our sample as a whole, their addition may be helpful for younger children or youth with a low level frequency of negative thoughts. Simplifying treatments to their active core ingredients (e.g., exposures) can enhance the feasibility, efficiency, and scalability of evidence-based treatments, allowing them to be more easily disseminated in a range of settings. Simultaneously, tailoring treatments based on individual differences (e.g., age) through supplemental strategies (e.g., cognitive strategies) may increase their effectiveness for a wider range of patients.
References


Table 1

*Means and Standard Deviations of Child and Parent STAIC scores*

<table>
<thead>
<tr>
<th></th>
<th>Child</th>
<th>Parent</th>
</tr>
</thead>
<tbody>
<tr>
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<tr>
<td>Session 9</td>
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</tr>
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</tr>
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<td>Parent</td>
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<td>Segment B</td>
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<td></td>
</tr>
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<td></td>
<td>Child</td>
<td>Parent</td>
</tr>
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<td>9.13</td>
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<td>Session x</td>
<td>28.87</td>
<td>9.21</td>
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</table>

*Note.* Segment A = randomly coded session from Sessions 9-12; Segment B = randomly coded session from Sessions 13-16; STAIC = State-Trait Anxiety Inventory for Children
Table 2

*Means and Standard Deviations of CATS and ADIS CSR*

<table>
<thead>
<tr>
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<th>SD</th>
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</thead>
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<td>Pre-Treatment</td>
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</tr>
<tr>
<td>Post-Treatment</td>
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<td>2.29</td>
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</table>

*Note.* CATS = Child Automatic Thoughts Scale; ADIS CSR = Anxiety Disorders Interview Schedule for Children - Clinician Severity Ratings.
Table 3

*Multiple Hierarchical Regression Predicting Session-by-Session Outcome in STAIC with Exposure and Cognitive Extensiveness*

<table>
<thead>
<tr>
<th></th>
<th>Child STAIC x</th>
<th></th>
<th>Parent STAIC x</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Segment A</td>
<td>Segment B</td>
<td>Segment A</td>
<td>Segment B</td>
</tr>
<tr>
<td></td>
<td><strong>B</strong></td>
<td><strong>SE</strong></td>
<td><strong>β</strong></td>
<td><strong>p</strong></td>
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<tr>
<td>Block 1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>STAIC x-1</td>
<td>0.92</td>
<td>0.05</td>
<td>0.91</td>
<td>.00*</td>
</tr>
<tr>
<td>Cog</td>
<td>0.08</td>
<td>0.47</td>
<td>0.01</td>
<td>.87</td>
</tr>
<tr>
<td>Block 2</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>STAIC x-1</td>
<td>0.91</td>
<td>0.05</td>
<td>0.90</td>
<td>.00*</td>
</tr>
<tr>
<td>Cog</td>
<td>0.05</td>
<td>0.46</td>
<td>0.01</td>
<td>.91</td>
</tr>
<tr>
<td>Exp</td>
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<td>0.25</td>
<td>0.08</td>
<td>-.12</td>
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<tr>
<td></td>
<td><strong>b</strong></td>
<td><strong>SE</strong></td>
<td><strong>β</strong></td>
<td><strong>p</strong></td>
</tr>
<tr>
<td>Block 1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>STAIC x-1</td>
<td>0.74</td>
<td>0.06</td>
<td>0.83</td>
<td>.00*</td>
</tr>
<tr>
<td>Cog</td>
<td>0.01</td>
<td>0.55</td>
<td>0.00</td>
<td>.99</td>
</tr>
<tr>
<td>Block 2</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>STAIC x-1</td>
<td>0.74</td>
<td>0.06</td>
<td>0.83</td>
<td>.00*</td>
</tr>
<tr>
<td>Cog</td>
<td>-0.03</td>
<td>0.55</td>
<td>0.00</td>
<td>.96</td>
</tr>
<tr>
<td>Exp</td>
<td>0.48</td>
<td>0.30</td>
<td>0.11</td>
<td>.11</td>
</tr>
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</table>

*Note.* STAIC = State-Trait Anxiety Inventory for Children; x-1 = Session x-1 (coded session); x = Session x; Cog = Session x-1 Cognitive Extensiveness; Exp = Session x-1 Exposure Extensiveness; Segment A = randomly coded session from Sessions 9-12; Segment B = randomly coded session from Sessions 13-16; b = Unstandardized regression weight; SE = Unstandardized Standard Error; β = Standardized regression weight. p ≤ .12; * p ≤ .05.
Table 4

*Multiple Hierarchical Regression Predicting Post-Treatment Outcome in STAIC and ADIS CSR scores with Mean Exposure and Cognitive Extensiveness Across Exposure Sessions*

<table>
<thead>
<tr>
<th></th>
<th>Post ADIS CSR</th>
<th>Child Post STAIC</th>
<th>Parent Post STAIC</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
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<td>SE</td>
<td>B</td>
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<tr>
<td>Mean Ext</td>
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<tr>
<td>Block 1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PreTx</td>
<td>0.74</td>
<td>0.31</td>
<td>0.29</td>
</tr>
<tr>
<td>Mean Cog</td>
<td>0.07</td>
<td>0.29</td>
<td>0.03</td>
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<tr>
<td>Block 2</td>
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</tr>
<tr>
<td>PreTx</td>
<td>0.73</td>
<td>0.32</td>
<td>0.28</td>
</tr>
<tr>
<td>Mean Cog</td>
<td>0.07</td>
<td>0.29</td>
<td>0.03</td>
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<tr>
<td>Mean Exp</td>
<td>0.03</td>
<td>0.18</td>
<td>0.02</td>
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</table>

*Note.* ADIS CSR = Anxiety Disorders Interview Schedule for Children - Clinician Severity Ratings; STAIC = State-Trait Anxiety Inventory for Children; Post = Post-treatment; PreTx = pretreatment ADIS CSR score or pre-exposure STAIC score; Mean Cog = Mean Cognitive Extensiveness; Mean Exp = Mean Exposure Extensiveness; $b =$ Unstandardized regression weight; $SE =$ Unstandardized Standard Error; $β =$ Standardized regression weight. *$p ≤ .05.$
Table 5

Multiple Hierarchical Regression Predicting Session-by-Session Outcome in STAIC with CATS * Cognitive Extensiveness Moderator

<table>
<thead>
<tr>
<th>Segment A</th>
<th>Child STAIC x</th>
<th>Parent STAIC x</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$b$</td>
<td>$SE$</td>
</tr>
<tr>
<td>STAIC x-1</td>
<td>0.87</td>
<td>0.06</td>
</tr>
<tr>
<td>CATS</td>
<td>0.00</td>
<td>0.04</td>
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<tr>
<td>Cog</td>
<td>-0.53</td>
<td>0.83</td>
</tr>
<tr>
<td>CATS * Cog</td>
<td>0.01</td>
<td>0.02</td>
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<table>
<thead>
<tr>
<th>Segment B</th>
<th>Child STAIC x</th>
<th>Parent STAIC x</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$b$</td>
<td>$SE$</td>
</tr>
<tr>
<td>STAIC x-1</td>
<td>0.95</td>
<td>0.05</td>
</tr>
<tr>
<td>CATS</td>
<td>-0.02</td>
<td>0.02</td>
</tr>
<tr>
<td>Cog</td>
<td>-0.04</td>
<td>0.52</td>
</tr>
<tr>
<td>CATS * Cog</td>
<td>0.00</td>
<td>0.01</td>
</tr>
</tbody>
</table>

Note. STAIC = State-Trait Anxiety Inventory for Children; x-1 = Session x-1 (coded session); x = Session x; CATS = Child Automatic Thoughts Scale; Cog = Session x-1 Cognitive Extensiveness; $b$ = Unstandardized regression weight; $SE$ = Unstandardized Standard Error; $\beta$ = Standardized regression weight.  
* $p \leq .05$. 

IMPACT OF COGNITIVE TECHNIQUES ON EXPOSURE
Table 6

Multiple Hierarchical Regression Predicting Post-Treatment Outcome in STAIC and ADIS CSR with CATS * Mean Cognitive Extensiveness Moderator

<table>
<thead>
<tr>
<th></th>
<th>Post ADIS CSR</th>
<th>Child Post STAIC</th>
<th>Parent Post STAIC</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>b</td>
<td>SE</td>
<td>β</td>
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<tr>
<td>PreTx</td>
<td>0.84</td>
<td>0.35</td>
<td>0.33</td>
</tr>
<tr>
<td>CATS</td>
<td>-0.05</td>
<td>0.02</td>
<td>-0.66</td>
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<tr>
<td>Mean Cog</td>
<td>-0.81</td>
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<td>-0.34</td>
</tr>
<tr>
<td>CATS*Mean Cog</td>
<td>.02</td>
<td>.01</td>
<td>.82</td>
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Note. ADIS CSR = Anxiety Disorders Interview Schedule - Clinical Severity Ratings; STAIC = State-Trait Anxiety Inventory for Children; Post = Post-treatment; PreTx = pre-treatment ADIS CSR score or pre-exposure STAIC score; CATS = Child Automatic Thoughts Scale; Mean Cog = Mean Cognitive Extensiveness; \(b\) = Unstandardized regression weight; \(SE\) = Unstandardized Standard Error; \(\beta\) = Standardized regression weight.

\(+p \leq .10; *p \leq .05.\)
Table 7

*Multiple Hierarchical Regression Predicting Session-by-Session Outcome in STAIC with Age * Cognitive Extensiveness Moderator*

<table>
<thead>
<tr>
<th></th>
<th>Child</th>
<th></th>
<th>Parent</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>b</td>
<td>SE</td>
<td>β</td>
<td>P</td>
</tr>
<tr>
<td>STAIC x-1</td>
<td>0.92</td>
<td>0.05</td>
<td>0.90</td>
<td>.00*</td>
</tr>
<tr>
<td>Age</td>
<td>0.40</td>
<td>0.61</td>
<td>0.09</td>
<td>.52</td>
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<td>Cog</td>
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<td>2.55</td>
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<td>.81</td>
</tr>
<tr>
<td>Age * Cog</td>
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Segment B

<table>
<thead>
<tr>
<th></th>
<th>Child</th>
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<th>Parent</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>b</td>
<td>SE</td>
<td>β</td>
<td>P</td>
</tr>
<tr>
<td>STAIC x-1</td>
<td>0.93</td>
<td>0.05</td>
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<td>.37</td>
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<tr>
<td>Age * Cog</td>
<td>0.15</td>
<td>0.16</td>
<td>0.26</td>
<td>.36</td>
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</table>

Note. STAIC = State-Trait Anxiety Inventory for Children; Cog = Session x-1 Cognitive Extensiveness; b = Unstandardized regression weight; SE = Unstandardized Standard Error; β = Standardized regression weight.  
* p ≤ .05.
Table 8

Multiple Hierarchical Regression Predicting Post-Treatment Outcome in STAIC and ADIS CSR with Age * Mean Cognitive Extensiveness Moderator

<table>
<thead>
<tr>
<th></th>
<th>Post ADIS CSR</th>
<th>Child Post STAIC</th>
<th>Parent Post STAIC</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
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<td>SE</td>
<td>β</td>
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<tr>
<td>PreTx</td>
<td>0.78</td>
<td>0.30</td>
<td>0.30</td>
</tr>
<tr>
<td>Age</td>
<td>-0.57</td>
<td>-0.35</td>
<td>-0.52</td>
</tr>
<tr>
<td>Mean Cog</td>
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<td>1.9</td>
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</table>

Note. ADIS CSR = Anxiety Disorders Interview Schedule - Clinical Severity Ratings; STAIC = State-Trait Anxiety Inventory for Children; Post = Post-treatment; PreTx = pretreatment ADIS CSR score or pre-exposure STAIC score; Mean Cog = Mean Cognitive Extensiveness; b = Unstandardized regression weight; SE = Unstandardized Standard Error; β = Standardized regression weight.

+ p ≤ .10; * p ≤ .05.
Figure 1. Relationship between Cognitive Extensiveness and ADIS CSR as a Function of Negative Automatic Thoughts

Note. ADIS CSR = Anxiety Disorders Interview Schedule - Clinician Severity Ratings; Pre-to Post- Treatment Change = Pre- to post- treatment standardized residualized change score; CATS = Child Automatic Thoughts Scale; Cog = Cognitive Extensiveness; Low = 1 standard deviation below average; High = 1 standard deviation above average.
Figure 2. Relationship between Cognitive Extensiveness and ADIS CSR as a Function of Age

Note. ADIS CSR = Anxiety Disorders Interview Schedule - Clinician Severity Ratings; Pre-to Post-Treatment Change = Pre- to post- treatment standardized residualized change score; Cog = Cognitive Extensiveness; Low = 1 standard deviation below average; High = 1 standard deviation above average.