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A Randomized Controlled Pilot Trial of the Unified Protocol for the Treatment of Emotional Disorders in Children (UP-C)

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A RANDOMIZED CONTROLLED PILOT TRIAL OF THE UNIFIED PROTOCOL
FOR THE TREATMENT OF EMOTIONAL DISORDERS IN CHILDREN (UP-C)

By

Sarah Michelle Kennedy

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Although cognitive-behavioral therapy (CBT) is a well-established treatment for anxiety

and depressive disorders in children (Chorpita et al., 2011), a significant proportion of

children do not respond to existing CBT protocols, and many protocols do not adequately

address high rates of comorbidity among emotional disorders. Transdiagnostic

approaches may help to improve treatment response in children, particularly for clinical

or sub-clinical comorbid conditions not adequately targeted by disorder-specific CBT,

and to prevent the later development of commonly occurring comorbid conditions. The

Unified Protocol for the Treatment of Emotional Disorders in Children (UP-C;

Ehrenreich-May et al., in press) is a transdiagnostic treatment protocol for children ages

6-13 that is organized around a set of core principles addressing emotion reactivity and

regulation deficits common across emotional disorders. Results of an open trial in 22

children supported the initial efficacy and feasibility of the UP-C (Bilek & Ehrenreich-

May, 2012) but were limited due to the lack of a comparison condition. This study

examined additional efficacy data for the UP-C, utilizing a RCT comparing UP-C to an

active, anxiety-specific intervention condition (Lyneham, Abbott, Wignall, & Rapee,

2003). Participants were 47 children with a primary anxiety or depressive disorder

diagnosis (55.30% female; *M* age = 9.31) evaluated at baseline (Pre-Tx), mid-treatment (Mid-Tx), post-treatment (Post-Tx), and six months after treatment completion (FU) using multi-informant assessments. Condition-related differences in diagnostic outcomes were evaluated using Pearson's chi-square test, while condition-related differences in dimensional outcomes were evaluated using latent growth curve models (LGMs) with treatment condition specified as a dummy-coded covariate. As hypothesized, no condition-related differences were found with respect to diagnostic outcomes, including remission of principal diagnosis and all emotional disorder-diagnoses at Post-Tx or FU. We also did not find significant differences in the slope of child- or parent-rated anxiety symptoms, nor in the mean levels of child- or parent-rated anxiety symptoms at Post-Tx or FU. Results provide preliminary evidence that the UP-C is at least as efficacious in treating anxiety disorders and anxiety symptoms as well-supported anxiety-specific treatment protocols. However, UP-C was superior to the active treatment control on a number of variables including treatment response status at FU, the shape of change in child-rated depression symptoms, rate of decrease in parent-rated sadness and worry dysregulation, and rate of increase in cognitive reappraisal. Additionally, UP-C participants demonstrated lower levels of parent-rated depression symptoms at Post-Tx, lower levels of parent-rated sadness dysregulation at Post-Tx and FU, and higher levels of child-rated cognitive reappraisal at Post-Tx and FU. Results provide initial support for the efficacy of the UP-C, which may produce greater gains in emotion reactivity and regulation variables compared to standard domain-specific CBT protocols without sacrificing gains in the area of anxiety.

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Chapter 1: Introduction

By the time they reach adulthood, a significant proportion of individuals have experienced an emotional disorder. Anxiety, one of the most common disorders, impacts up to 20% of youth (Beesdo, Knappe, & Pine, 2009), while approximately one in four youth experience a major depressive episode and 4-7% of youth a unipolar depressive disorder by age 18 (Costello, Erkanli, & Angold, 2006; Lewinsohn, Rohde, & Seeley, 1998). Emotional disorders more often occur together than alone, and comorbidity, particularly among anxiety and depressive disorders, is associated with higher overall severity and functional impairment (Masi, Favilla, Mucci, & Millepiedi, 2000), more peer problems (Klitzing et al., 2014), greater likelihood of disorder recurrence (Moffitt et al., 2007), and higher frequency of mental health service utilization (Essau, 2008; Moffitt et al., 2007). Despite the deleterious impact of comorbidity upon children's disorder trajectories and overall functioning, cognitive behavioral therapy (CBT) for emotional disorders has traditionally overlooked the way in which comorbidity complicates the clinical picture for youth, as the majority of previous efficacy trials have targeted a single disorder or class of disorders. Although approximately 50-60% of children treated with anxiety-specific CBT experience a remission of their principal diagnosis at post-treatment (Kendall, Hudson, Gosch, Flannery-Schroeder, & Suveg, 2008; Walkup et al., 2008), such protocols have often excluded youth with comorbid conditions, do not adequately address high rates of concurrent or sequential comorbidity among emotional disorders, and may leave youth at risk for disorder recurrence or emergence of other disorders.

Transdiagnostic or unified approaches to the treatment of emotional disorders in youth may help to improve treatment response rates, particularly for clinical or sub-

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clinical comorbid conditions not adequately targeted by disorder-specific CBT, and to prevent the later development of commonly occurring comorbid conditions in youth. Unified approaches are those that apply a common set of core principles to the treatment of multiple disorders, rather than tailoring treatment components to specific diagnoses (McEvoy, Nathan, & Norton, 2009). The Unified Protocols for Treatment of Emotional Disorders (UP; Barlow et al., 2011), as well as downward extensions of the UP for adolescents (UP-A; Ehrenreich-May et al., in press) and children (UP-C; Ehrenreich-May et al., in press), take a transdiagnostic approach to the treatment of anxiety, depressive, stress-related, and OC-spectrum disorders (hereafter referred to as "emotional disorders") by focusing on core processes common across these disorders identified by research in the areas of emotion science, affective neuroscience, clinical trials, and basic risk and vulnerability factors for emotional disorders. Briefly, the premise of the UP models is that emotional disorders are rooted in a temperamental predisposition to experience high levels of negative emotion (i.e., neuroticism) and high emotional reactivity, resulting in problematic and often ineffective attempts to regulate emotional experiences (Barlow, Sauer-Zavala, Carl, Bullis, & Ellard, 2014). The UP affects temperamental processes implicated in emotional disorders by targeting problematic attempts at regulation that increase the frequency and intensity of negative emotions (Barlow et al., 2014).

Randomized controlled trials (RCTs) of the UP and UP-A have supported the efficacy of these protocols in treating emotional disorders in both adults and adolescents, respectively (Ehrenreich-May et al., under review; Farchione et al., 2012; Queen, Barlow, & Ehrenreich-May, 2014). Although results from an initial open trial of the UP-C in 22 children appear promising, with 78% of participants no longer meeting criteria for any

anxiety disorder at post-treatment (Bilek & Ehrenreich-May, 2012), conclusions about the efficacy of the UP-C have thus far been limited due to the lack of a comparison condition. The current study examines additional efficacy data for the UP-C in a primarily anxious sample with some depression comorbidity, utilizing a RCT design comparing UP-C to an active, anxiety-specific treatment condition (Lyneham, Abbott, Wignall, & Rapee, 2003). Participants were evaluated just prior to beginning treatment (Pre-Tx), at the treatment mid-point (Mid-Tx), directly after completing treatment (Post-Tx), and six months after completing treatment (FU). Analyses used a multi-informant approach, evaluating three separate types of outcome measures: 1) condition-related differences in clinician-rated diagnostic remission and treatment response; 2) condition-related differences in parent- and child-rated anxiety and depression symptoms; and 3) condition-related differences in parent- and child-rated emotion reactivity and regulation variables theorized to be treatment mediators (e.g., emotion dysregulation, cognitive reappraisal, expressive suppression, positive affect, negative affect). The following sections will provide a rationale for the use of transdiagnostic treatments in youth by discussing the limitations of current evidence-based treatment approaches, providing an overview of research-based core processes underlying emotional disorders, and reviewing the evidence base for transdiagnostic treatments in adults and youth.

Evidence-Based Treatments for Emotional Disorders in Youth

As a result of nearly three decades of effort into the development, testing, and dissemination of evidence-based treatments (EBTs) for children with anxiety disorders, various efficacious treatment protocols now exist. Cognitive-behavioral and exposure-based therapies are currently considered Level 1 or well-established treatments for

anxiety in youth, yielding a mean effect size of .85 when delivered in individual or group formats across multiple settings (Chorpita et al., 2011). Although depressive disorders are much less common than anxiety disorders in youth under 13, CBT is also considered a Level 1 or well-established treatment for depression in children over eight, with a mean effect size of .87 across clinic and community settings (Chorpita et al., 2011). Further, gains have been shown to be maintained or to even slightly improve up to six months post-treatment for youth receiving CBT for anxiety (e.g., Piacentini et al., 2014) and for depression (TADS Team, 2007).

Despite the positive strides made by the EBT movement, it is important to note that up to 40% of youth receiving CBT for anxiety are not considered to be treatment responders (e.g., Walkup et al., 2008), and one large-scale RCT of CBT for adolescent depression found only a 43% 12-week response rate, which was not significantly different from pill placebo and lower than fluoxetine monotherapy (TADS Team, 2004). Clearly, there is room for improvement in post-treatment outcomes for emotional disorders. Beyond immediate outcomes, existing long-term studies of current EBTs suggest that relapse, as well as the emergence of new mental health concerns, are common in previously treated youth. Ginsburg and colleagues (2014) found that relapse occurred in nearly 50% of acute responders within six years of treatment for an anxiety disorder during childhood, while Benjamin, Harrison, Settipani, Brodman, and Kendall (2013) found that both successfully and unsuccessfully treated anxious youth remained at significantly increased risk for GAD and substance dependence compared to healthy controls an average of 16 years later. Although additional data on the long-term efficacy

of EBTs is needed, initial data suggests that many immediate responders experience later relapse, and youth treated for anxiety remain at increased risk for the onset of depressive disorders and other related conditions later in development.

Interpreting the results of EBTs for youth anxiety and depressive disorders is further complicated by the fact that many efficacy studies have traditionally excluded participants with commonly occurring comorbid conditions (e.g., participants with depressive disorders in efficacy trials for anxiety; Walkup et al., 2008). This omission is unfortunate, given that the majority of youth with an anxiety or depressive disorder have one or more comorbid disorders. Approximately two-thirds of clinic-referred youth with a primary anxiety diagnosis receive at least one additional diagnosis, with other anxiety disorders being the most common comorbid conditions (Angold, Costello, Erkanli, 1999). Rates of comorbidity between anxiety and depressive disorders have also been observed to be as high as 75% in some clinical samples (Weersing, Gonzalez, Campo, & Lucas, 2008) and appear to be particularly elevated in youth with primary depression (Axelson & Birmaher, 2001; Garber & Weersing, 2010; Ollendick et al., 2008). Several studies have shown that depression comorbidity predicts poorer outcomes in youth receiving EBTs for anxiety disorders (e.g., Berman, Weems, Silverman, & Kurtines, 2000), and anxiety comorbidity has been shown to predict poorer outcomes in youth receiving EBTs for depressive disorders (Curry et al., 2006; Young, Mufson, & Davies, 2006).

Flexible treatment formats that allow clinicians to address multiple disorders within the same protocol thus have the potential to improve immediate outcomes for both principal and comorbid disorders. Further, transdiagnostic protocols, which target higher-order temperamental vulnerabilities for multiple emotional disorders (e.g., neuroticism,

extraversion) and generalize the delivery of evidence-based skills across a range of emotions, may help to prevent more distal negative outcomes later in development. It is now well-documented that anxiety disorders often temporally precede the onset of depressive disorders, with the onset of anxiety disorders typically occurring in early to middle childhood and depressive disorders most commonly beginning in adolescence (Chavira, Stein, Bailey, & Stein, 2004; Mathew, Petit, Lewinsohn, Seeley, & Roberts, 2011). Early treatment of anxiety using a transdiagnostic framework may thus help to prevent the development of later depression, although empirical evidence in support of such a preventative hypotheses is still forthcoming.

Core Processes Underlying Emotional Disorders

Given that comorbidity is so often the rule rather than the exception, targeting shared vulnerability and maintenance factors that cut across emotional disorders may be one way of improving both the short-term and long-term efficacy of current treatments. Although high comorbidity between anxiety and depressive disorders may be in part accounted for by symptom overlap among disorders, correlations between anxiety and depressive symptoms remain in the moderate to strong range after overlapping items on self-report measures are excluded, suggesting that comorbidity is not merely the result of shared symptoms (Stark & Laurent, 2001). Alternatively, it has been asserted that comorbidity may result from overlapping risk or vulnerability factors, or that impairment arising from one disorder may place youth at risk for developing another, related disorder (Seligman & Ollendick, 1998). Research in the areas of genetics, structural modeling, affective neuroscience, and longitudinal risk studies have provided support for both of

these latter models of comorbidity by elucidating shared core processes that may help account for comorbidity among anxiety disorders and among anxiety and depressive disorders.

Genetic vulnerability. Classical twin studies, which use structural modeling approaches to decompose genetic variance in a particular trait into genetic, shared environmental, and unique environmental components, have consistently found that genetic factors account for a significant proportion of the variance in anxiety and depression. Internalizing problems appear to be moderately to highly heritable, with estimates ranging from approximately 30 to 75% (e.g., Boomsma, Van Beijsterveldt, & Hudziak, 2005; Eley et al., 2003; Hansell et al., 2012; Hudziak et al., 2000) and varying based on differences in child age, measurement, and study methodology. Genetic overlap has also been identified among anxiety-related syndromes (Eley et al., 2003), and among depression and anxiety (Middeldorp, Cath, Van Dyck, & Boomsma, 2005), suggesting that there may be a shared genetic basis for emotional disorders. This shared genetic basis may be conferred through endophenotypes, or heritable intermediate traits between genes and symptoms that are stable over time and are associated with disease (Gottesman & Gould, 2003). Neuroticism, or the temperamental propensity to experience frequent and intense negative emotions in response to stress (Barlow, Ellard, Sauer-Zavala, Bullis, & Carl (2014), is one potential endophenotype that is between 40 and 60% heritable (e.g., Bouchard & Loehlin, 2001; Kendler, Prescott, Myers, & Neale, 2003), is strongly associated with symptoms of anxiety and depression, and shares a large proportion of genetic variance with internalizing and somatic symptoms (Hansell et al., 2012; Boomsma et al., 2005; Middledorp et al., 2005).

Other lines of research have supported the hypothesis of Barlow and colleagues that a "general neurotic syndrome" functions as a shared vulnerability factor for a range of emotional disorders (Brown & Barlow, 2009; Brown, Chorpita, & Barlow, 1998). Clark and Watson's (1991) tripartite model, which has now been extensively evaluated in both youth and adults, proposed that propensity to experience negative affect (NA) is a shared process that accounts for symptom overlap and comorbidity among anxiety and depressive disorders, while decreased positive affect (PA) differentiates depressive from anxiety disorders, and increased physiological hyperarousal (PH) differentiates anxiety from depressive disorders. Several studies have supported this three-factor model of emotional disorders in youth (e.g., Chorpita, Plummer, & Moffitt, 2000; Laurent, Catanzaro, & Joiner, 2004), although it has been suggested that social anxiety disorder may be better characterized by both low NA and low PA, much like depressive disorders (see Anderson & Hope, 2008, for review). Findings from affective neuroscience have supported the hypothesis that individuals with both anxiety and depressive disorders have a propensity to experience increased NA and high emotional reactivity to NA relative to individuals without an emotional disorder. Individuals with both anxiety and depression, as well as individuals high in neuroticism, appear to experience hyperactivity in limbic areas associated with the experience of negative emotion, as well as hypoactivity in cortical structures associated with inhibitory control of emotions (see Wilamowska et al., 2010 for review).

Parenting. Heritable vulnerabilities such as neuroticism may thus set the stage for the development of emotional disorders in some youth. These heritable vulnerabilities interact with other emotional, cognitive, and behavioral vulnerabilities, some of which

are shared across emotional disorders and some of which are unique to a single disorder. This model is consistent with Barlow's (2000, 2002) triple vulnerability model of emotional disorders, whereby biological vulnerabilities (e.g., neuroticism), general psychological vulnerabilities resulting from early life experiences, and disorder-specific psychological vulnerabilities interact to contribute to the development and maintenance of emotional disorders. Some vulnerabilities, such as exposure to certain parenting styles, are present early in a child's life and may interact with genetic risk factors or their phenotypic expression to set the stage for later emotional disorders. Several parenting behaviors have been broadly associated with the development of emotional disorders in youth. Rejection/criticism and low parental warmth have been associated with the development of both anxiety and depressive disorders (Drake & Ginsburg, 2012), while parental overcontrol or overprotection, which may reinforce child avoidance and prevent the development of healthy self-efficacy, has been broadly linked with anxiety disorders in youth (Ginsburg, Siqueland, Masia-Warner, & Hedtke, 2005; Drake & Ginsburg, 2012).

Cognitive and behavioral emotion regulation strategies. In addition to heritable vulnerabilities and early parenting experiences, both anxiety and depressive disorders have been associated with over-utilization of cognitive and behavioral emotion regulation strategies that exacerbate negative mood states (e.g., expressive suppression, repetitive negative thinking, avoidance), and under-utilization of strategies that effectively repair negative mood (e.g., cognitive reappraisal). Cognitive factors, such as styles of repetitive negative thinking (RNT) like rumination or worry, have been linked to anxiety and depressive disorders in both adults and youth. While worry has been

traditionally conceptualized as a future-oriented cognitive process most characteristic of anxiety, and rumination a past-oriented process most characteristic of depression, this distinction has more recently been collapsed into a broad vulnerability for emotional disorders termed RNT, partly as a result of the finding that rumination is associated with both anxiety and depression. McLaughlin and Nolen-Hoeksema (2011), for example, found that rumination mediated the longitudinal relationship between both anxiety and later depressive symptoms and depressive symptoms and later anxiety. Drost and colleagues (2014) obtained similar results in a community sample of adults, in which rumination, in addition to worry, mediated the longitudinal relationship between fear disorders and distress disorders in adults. Similarly, Hankin (2008) found that rumination moderated the relationship between anxious arousal and depressive symptoms, whereby anxious arousal predicted fluctuations in depressive symptoms in youth who exhibited a ruminative response style. Conversely, youth with emotional disorders may use purportedly adaptive strategies, such as cognitive reappraisal, infrequently and/or ineffectively (Gross & Thompson, 2007). Anxious children appear to use less spontaneous and cued reappraisal (Carthy, Horesh, Apter, Edge, & Gross, 2010), and reappraisal use has been negatively associated with psychopathology (Aldao, Nolen-Hoeksema, & Schweizer, 2010). Together, these results suggest that certain cognitive response styles may up-regulate negative mood and, when used consistently or perhaps in absence of more effective regulation strategies, may contribute to the development and maintenance of emotional disorders.

The use of avoidant strategies to regulate emotions has also been broadly linked to the development and maintenance of emotional disorders in youth. Avoidant strategies

and overreliance upon safety behaviors, although immediately reinforcing, may maintain or even increase negative affect in the longer term, prevent habituation to negative stimuli or disconfirmation of cognitive errors about stimuli, and may limit access to positively reinforcing activities or social opportunities (Harvey, Watkins, Mansell, & Shafran, 2004; Campbell-Sills & Barlow, 2007). These long-term negative consequences of avoidant emotion regulation strategies may contribute to the development and maintenance of emotional disorders. For example, expressive suppression has been associated with heightened symptoms of both anxiety and depression in youth (Gullone & Taffe, 2012; Aldao et al., 2010) and with increased sympathetic activation, increased negative affect, and decreased positive affect in adults (Gross, 1998; Gross & John, 2003). Similarly, the use of behavioral avoidance, as well the use of more subtle safety behaviors to reduce the intensity of an emotional situation, is thought to be a transdiagnostic risk and maintenance factor for both anxiety and depressive disorders. Use of behavioral avoidance has been positively associated with symptoms of both anxiety and depression in youth (e.g., Ottenbreit, Dobson, & Quigley, 2014; Thomas, Daruwala, Goepel, & De Los Reyes, 2012; Whiteside et al., 2013), while several studies have found evidence to suggest that behavioral avoidance may mediate the relationship between anxiety and depressive disorders in adolescence and later negative outcomes in adulthood (e.g., Allen, Chango, Szwedo, & Schad, 2014; Jacobson & Newman, 2014).

Interpersonal factors. Finally, social deficits or interpersonal conflict may be additional broad vulnerability and maintenance factors for emotional disorders, and may also help to explain the longitudinal association between anxiety and later depressive disorders. Youth with anxiety disorders, particularly social anxiety disorder, have been

observed to have deficits in social skills and social competence as rated by teachers (Greco & Morris, 2005) and independent observers (Alfano, Beidel, & Turner, 2006; Beidel et al., 2007). These deficits may confer vulnerability for depression by resulting in peer rejection, low peer acceptance, peer victimization, and loneliness, all of which have been associated with both symptoms of anxiety and depression (see Epkins & Heckler, 2011, for review). Several studies have indeed linked the longitudinal association between anxiety and later depression to interpersonal difficulties. Starr, Hammen, Connolly, and Brennan (2013), for example, found that anxiety disorders in adolescence were associated with broad interpersonal impairment, including interpersonal oversensitivity, low sociability, and chronic social stress, and these interpersonal factors partially mediated the association between anxiety and later depression in adulthood. Early treatment of anxiety disorders in youth alone may improve interpersonal difficulties and help prevent the development of depressive disorders, but infusion of social skills and interpersonal training into current EBTs for anxiety disorders may be particularly important for youth with underlying interpersonal deficits above and beyond those directly resulting from their anxiety disorder.

Transdiagnostic Treatment Movement: Targeting Core Processes

Transdiagnostic treatment approaches, which apply a common set of core principles to the treatment of more than one disorder, address some of the limitations of single disorder or syndrome EBTs by targeting shared vulnerability and maintenance factors underlying multiple disorders. These shared factors include many of those outlined in the preceding section, such as heritable vulnerabilities (e.g., neuroticism), ineffective use of cognitive and behavioral strategies to regulate emotions, and

interpersonal conflict and deficits. The Unified Protocols for the Treatment of Emotional Disorders are transdiagnostic treatments for adults, children, and adolescents guided by the principle that emotional disorders have a shared pathophysiology and are expressed as a common set of core features. The UP model is thus potentially easier to disseminate than single target EBTs because the flexible treatment strategies employed in the UP, UP-A, and UP-C allow clinicians to address diverse problems parsimoniously within a single protocol. EBTs for emotional disorders, although now numerous, do not reach the majority of youth in need or services (Riemer, Rosof-Williams, & Bickman, 2005) and do not appear to perform as well in real-world clinical contexts when disseminated in effectiveness studies (Weisz, Jensen-Doss, & Hawley, 2006; Weisz, Ugueto, Cheron, & Herren, 2013). Transdiagnostic protocols may help bridge the gap between research and practice because they are flexible enough to address varying client characteristics and comorbidities in a single package via a focus on risk and maintaining factors across emotional disorders, limiting the burden associated with training clinicians in multiple EBTs. Indeed, initial effectiveness data shows that a modular treatment approach allowing for flexible implementation of evidence-based techniques for a range of emotional and behavioral disorders resulted in faster improvement and fewer post-treatment diagnoses than either usual care or standard single-diagnosis manual treatment (Weisz et al., 2012). Transdiagnostic treatments may be an even more parsimonious alternative to modular treatments because the same set of therapeutic techniques, guided by a single unified theory, can be used to simultaneously target multiple problem areas within a single protocol.

Children may respond particularly well to a transdiagnostic treatment approach because there is high heterotypic continuity of emotional disorders across childhood and adolescence, resulting in frequently shifting symptom and diagnostic profiles over time. The Unified Protocol for the Treatment of Emotional Disorders in Children (UP-C) is a downward extension of the UP and UP-A that delivers the core components of the UP models in an interactive, child-friendly group format incorporating caregivers. Similar to both the UP and UP-A, the UP-C applies a singular set of evidence-based treatment strategies to a range of emotional experiences, including anxiety, sadness, and anger. Treatment is delivered in interactive, child-friendly format with significant caregiver involvement. A detective theme serves as the organizing principle, and treatment is divided into five primary sections around the CLUES skills ("Consider How I Feel;" "Look at My Thoughts;" "Use Detective Thinking;" "Experience My Fears and Feelings;" "Stay Healthy and Happy"). Table 1 specifies the skills delivered in each of these five sections of treatment, as well as hypothesized risk and maintaining factors for emotional disorders that are targeted by each skill. Although no formal tests of mediation have been conducted for the UP, UP-C and its sister protocols are hypothesized to lead to symptom and diagnostic improvement by decreasing emotional reactivity, improving emotion regulation capabilities, and reducing avoidance and withdrawal behaviors. Indeed, several recent examinations of associations between treatment-related changes in hypothesized mediators and emotional disorder symptoms in the adult UP have shown that change in adaptive and maladaptive emotion regulation strategies, negative affect,

fear of negative emotions, and anxiety sensitivity were significantly related to changes in symptom measures (Conklin et al., 2016; Farchione et al., 2012; Sauer-Zavala et al., 2012).

Transdiagnostic treatments are a relatively recent development in EBT research, but support for their efficacy in adults, adolescents, and children with a range of emotional disorders is growing. Several reviews of transdiagnostic treatments for adults have found that participants receiving transdiagnostic CBT experience significantly greater symptom and diagnostic improvement than waitlist controls or participants receiving treatment as usual (McEvoy, Nathan, & Norton, 2009; Reinholt & Krogh, 2014). Additionally, transdiagnostic treatments are associated with improvements in severity of comorbid diagnoses and symptoms, and diagnostic comorbidity does not appear to moderate outcome (McEvoy et al., 2009). Findings from a recent waitlist-controlled RCT of the UP in adults were consistent with these results. Significant reductions in principal diagnosis symptom severity and symptom severity of comorbid diagnoses were observed, and these changes were large in magnitude and significantly greater than those observed in the WL condition (Farchione et al., 2012). The majority of changes were maintained six months post-treatment (Farchione et al., 2012), and all participants who met responder status at the six-month follow-up retained their status a year later (Bullis, Fortune, Farchione, & Barlow, 2014). However, as of yet there are still relatively few RCTs comparing transdiagnostic treatments to waitlist or active conditions, and there is only one RCT comparing a transdiagnostic treatment to disorder-specific CBT in adults (Norton & Barrera, 2012).

Evidence for the efficacy of transdiagnostic treatments in adolescents and children is even more limited, but a recently completed waitlist-controlled RCT of the UP-A provided support for the extension of the UP models to youth. Participants in the UP-A condition experienced significantly greater improvement than those in the WL condition on all outcomes, with a greater effect for clinician-rated measures than for self- or parent-rated measures, and participants continued to improve on most measures up to six months post-treatment (Ehrenreich-May et al., under review). Further, in a recent open trial of the UP-C in 22 children, a large effect was observed for reduction in principal anxiety disorder severity from pre- to post-treatment (Cohen's d = 1.38), and 78% of participants no longer met criteria for any anxiety disorder at post-treatment (Bilek & Ehrenreich-May, 2012). A significant change in parent-reported child depressive symptoms was also observed (Cohen's d = .54), supporting the efficacy of the UP-C in targeting symptoms of both anxiety and depressive disorders (Bilek & Ehrenreich-May, 2012). The next step in testing the efficacy of the UP-C is a RCT design, and given that no known transdiagnostic treatments for youth have been compared to an active treatment condition, this type of comparison would make a substantial contribution to the current literature.

Further, examining treatment-related changes in theorized mediators is an essential next step for understanding why transdiagnostic treatments work. Few studies to date have examined whether single-domain CBT, let alone transdiagnostic CBT, significantly impacts emotion reactivity and regulation processes that may be risk and maintaining factors across emotional disorders. With regard to single-domain CBT, Suveg, Sood, Comer, and Kendall (2009) examined whether 37 children who received

CBT for anxiety experienced treatment-related changes in several emotion regulation processes. They found that, at post-treatment, children exhibited significantly greater emotional awareness, significantly less inhibition of worry and sadness, and significantly less dysregulated expression of worry and sadness. In the transdiagnostic literature, several waitlist-controlled trials have examined the impact of transdiagnostic treatment on hypothesized mechanisms of change. In a pilot RCT of a transdiagnostic group behavioral activation and exposure therapy (GBAT) in a sample of youth ages 12-14, Chu and colleagues (2016) examined the impact of treatment on a hypothesized cognitive (i.e., negative automatic thoughts) and behavioral mechanism of change (i.e., behavioral avoidance). Chu and colleagues found a marginally significant treatment effect for both negative automatic thoughts and behavioral avoidance, but only negative automatic thoughts continued to decline through a four-month follow-up period. Farchione and colleagues (2012) examined the impact of the adult UP on positive and negative affect and found that, compared to WL, the UP had a large effect on increase in positive affect at post-treatment and a medium effect on decrease in negative affect at post-treatment; changes were maintained during a follow-up period. Continued examination of treatment-related changes in theorized mediators is a crucial step in identifying potential mechanisms of change, which have been long been understudied in the child and adolescent CBT literature (Weersing & Weisz; 2002).

Current Study

The current study advances the transdiagnostic treatment literature by examining additional support for the UP-C using a RCT design. Given that only one study in the adult literature and no studies in the child and adolescent literature have compared

transdiagnostic treatments with disorder-specific protocols, we chose an anxiety-specific group treatment protocol (Cool Kids [CK]; Lyneham et al., 2003) as the comparison condition for our RCT. Condition-related differences will be examined for three types of outcome measures, using a multi-information approach: 1) condition-related differences in clinician-rated diagnostic remission and response; 2) condition-related differences in parent- and child-rated anxiety and depression symptoms; and 3) condition-related differences in parent- and child-rated emotion reactivity and regulation variables thought to reflect shared risk and maintaining factors for emotional disorders (e.g., emotion dysregulation, cognitive reappraisal, expressive suppression, positive affect, negative affect). It should be noted that, with regard to diagnostic severity/improvement and parent and child-rated anxiety symptoms, condition-related differences were not anticipated due to strong existing research support for the CK protocol (e.g., Hudson et al., 2009). However, a lack of condition-related differences on diagnostic outcomes and anxiety measures would indicate that UP-C can be used to parsimoniously target multiple problem areas without sacrificing gains in the area of anxiety. Finally, given that there have been so few examinations of mediation within the youth CBT treatment literature, let alone within the transdiagnostic treatment literature, we planned to examine changes in emotion reactivity and regulation as potential mediators of the UP-C. Specific aims and hypotheses for this project are as follows:

Specific Aim 1: Examine condition-related differences in remission of principal diagnosis, remission of all emotional disorder diagnoses, and treatment responder status.

Specific Aim 1, Hypothesis 1: UP-C and CK conditions will not differ in the proportion of children free of their primary diagnosis at either the Post-Tx or FU time points.

Specific Aim 1, Hypothesis 2: UP-C and CK will not differ in the proportion of children free of all emotional disorder diagnoses at Post-Tx and FU time points.

Specific Aim 1, Hypothesis 3: UP-C and CK will not differ in the proportion of children achieving treatment responder status at either Post-Tx or FU.

Specific Aim 2: Examine the effect of treatment condition on the slope of parent- and child-rated anxiety symptoms from Pre-Tx to FU, as well as on mean levels of parent- and child-rated anxiety at Post-Tx and FU.

Specific Aim 2, Hypothesis 1: The slope of parent- and child-rated anxiety symptoms will not differ between conditions.

Specific Aim 2, Hypothesis 2: Mean levels of parent- and child-rated anxiety symptoms will not differ between conditions at Post-Tx or FU.

Specific Aim 3: Examine the effect of treatment condition on the slope of parent- and child-rated depression symptoms from Pre-Tx to FU, as well as on mean levels of parent- and child-rated depression symptoms at Post-Tx and FU.

Specific Aim 3, Hypothesis 1: The slope of both parent- and child-rated depression symptoms will differ between treatment conditions, such that UP-C participants will experience a faster rate of decrease than CK participants.

Specific Aim 3, Hypothesis 2: UP-C participants will have lower levels of both parent- and child-rated depression symptoms at Post-Tx and FU.

Specific Aim 4: Examine the effect of treatment condition (UP-C vs. CK) on the slope of emotion reactivity and regulation variables theorized to be risk and maintaining factors across emotional disorders (i.e., parent- and child-rated emotion dysregulation, child-rated cognitive reappraisal, child-rated expressive suppression, child-rated positive affect, child-rated negative affect), as well as on mean levels of these variables at Post-Tx and FU.

Specific Aim 4, Hypothesis 1: The slope of parent- and child-rated anger and sadness dysregulation will differ between conditions, such that UP-C participants will experience a faster rate of decrease than CK participants. The slope of parent- and child-rated worry dysregulation will not differ between conditions. Additionally, UP-C participants will have lower levels of anger and sadness dysregulation, but not worry dysregulation, at Post-Tx and FU.

Specific Aim 4, Hypothesis 2: The slope of child-rated cognitive reappraisal will not differ between conditions, nor will levels of child-rated cognitive reappraisal at Post-Tx and FU, as both interventions target this emotion regulation process.

Specific Aim 4, Hypothesis 3: The slope of child-rated expressive suppression will differ between conditions, such that UP-C participants will experience a faster rate of decrease than CK participants. Additionally, UP-C participants will have lower levels of expressive suppression at Post-Tx and FU.

Specific Aim 4, Hypothesis 4: The slope of child-rated positive affect will differ between conditions, such that UP-C participants will experience a faster rate of increase in positive affect than CK participants. Additionally, UP-C participants will have higher levels of positive affect than CK participants at Post-Tx and FU.

Specific Aim 4, Hypothesis 5: The slope of child-rated negative affect will not differ between conditions, nor will levels of child-rated negative affect at Post-Tx and FU, as both interventions target this higher order factor.

Exploratory Specific Aim 5: Examine whether changes in emotion reactivity and regulation variables (i.e., emotion dysregulation, cognitive reappraisal, expressive suppression, positive affect, negative affect) mediate treatment outcomes for UP-C. This aim will only be explored if significant changes are found in these variables between Pre-Tx and Mid-Tx. Mediational aims will be tested by examining the indirect effects of intervention condition on Post-Tx symptom variables through Mid-Tx emotion dysregulation, cognitive reappraisal, expressive suppression, positive affect, and negative affect. Given the lack of meditational analyses in the child anxiety treatment literature, no a priori hypotheses are advanced.

Chapter 2: Method

Study Design

Approval was obtained from the University of Miami's Institutional Review board prior to beginning this investigation. Participants were recruited using a variety of methods, including flyers and list-serve announcements. Additionally, many children were referred to the clinic by school personnel, pediatricians, other health care professionals, and through word of mouth. Parents who contacted the clinic were asked to complete a brief phone screen assessing presenting concerns, prior treatment and psychiatric history, and the presence of exclusion criteria. Children whose parents reported a primary anxiety or mood-related concern were scheduled for an initial diagnostic interview with a trained Clinical Psychology doctoral student, Ph.D.-level clinical psychologist, or advanced post-baccalaureate research assistant. If the primary concern did not appear to be anxiety or mood-related, appropriate community referrals were provided to the parent.

All participants provided informed consent and assent to participate in an initial diagnostic assessment to determine study eligibility. During this assessment, the child and parent were interviewed separately to determine DSM-IV-TR diagnoses and to assess for inclusion and exclusion criteria. Primary inclusion criteria for participation were a principal diagnosis of an anxiety and/or depressive disorder, as determined by the Anxiety Disorders Interview Schedule for the DSM-IV, Child and Parent Reports (ADIS-IV-C/P; Silverman & Albano, 1996). Criteria for exclusion included: 1) inability of at least one parent or guardian to attend all assessment and treatment sessions; 2) inability of the child or a primary caregiver to speak, read, and understand English well enough to

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complete study procedures; 3) a DSM-IV-TR diagnosis of schizophrenia, bipolar I or II disorder, pervasive developmental disorder, or mental retardation; 4) severe current suicidal/homicidal ideation; and/or 5) having previously received CBT. Participants were provided with diagnostic feedback following the initial assessment. Participants meeting inclusion criteria were then given the opportunity to provide informed consent and assent for treatment, while participants not meeting inclusion criteria received treatment through another study or were referred out.

Participants were block randomized (in blocks of four) to complete either the UP-C ($n = 24$) or CK ($n = 23$) treatment protocol. Participants waiting to begin the group treatment program were contacted every two weeks prior to the start of the study to monitor for deterioration. Additionally, participants who were assessed more than one month prior to the start of treatment completed an abbreviated diagnostic interview (the "Mini" ADIS-IV-C/P [Silverman & Albano, 1996]) in the two weeks before treatment to confirm eligibility, and data from this second assessment was used in place of data from the initial assessment as the Pre-Tx time point. All RCT participants completed an assessment at Mid-Tx (week 7), at Post-Tx, and at FU.

Participants

Participant flow through treatment and follow-up is presented in the consort diagram in Figure 1. A total of 47 participants consented to treatment and were randomized to either the UP-C or CK condition, and all 47 presented for treatment and received at least one session of intervention. Data from the sample of 47 participants was used in all analyses, unless specified otherwise. Of these 47 participants, 55.31% were female ($n = 26$), and participants ranged in age from 6 to 12 years old ($M = 9.31$). The

majority of participants identified themselves as White Hispanic (n = 27, 57.4%), followed by White Non-Hispanic (n = 17, 36.2%), Black or African-American (n = 1, 2.10%), Asian or Pacific Islander (n = 1, 2.10%), and Other (n = 1, 2.10%). Mean family income was $98,830 (range = $20,000 - $250,000; median = $90,000). During the Pre-Tx assessment, one participant reported taking a psychotropic medication (SSRI), and two participants reported taking a stimulant medication for management of attention deficit/hyperactivity disorder (ADHD; i.e., Concerta). All participants were on a stable dose of their medication for at least one month prior to their Pre-Tx assessment, and no medication changes were reported at any time during the trial.

The most common principal diagnoses for the sample were generalized anxiety disorder (n = 22, 46.81%) and social anxiety disorder (n = 10, 21.28%). Comorbidity was high in this sample; the majority of children (69.4%) received more than one clinical diagnosis, with 2.10 being the mean number of diagnoses assigned at a clinical level (range = 1 to 4). With respect to depressive disorder diagnoses, 8.49% of children (n = 4; 3 in UP-C, 1 in CK) received a diagnosis at a clinical level at the Pre-Tx assessment, while 6.40% of children (n = 3; 3 in CK, 0 in UP-C) received a subclinical diagnosis of a depressive disorder. Despite the low percentage of children with a clinical or subclinical depressive disorder, it should be noted that 27.70% of children reported experiencing elevated depression symptoms (i.e., CDI Total >12) on the *Children's Depression Inventory* (CDI; Kovacs, 1992), while 36.20% of parents reported their children to be experiencing elevated depression symptoms on the CDI. No condition-related differences were observed in the number of children above the cutoff on either child report (χ^2[1] =

.05, p = .82) or parent report of depression symptoms ($\chi^2[1]$ = .01, p = .94). Table 2 displays principal clinical diagnoses and comorbid clinical diagnoses for the sample as a whole.

Treatment

  UP-C condition. Treatment consisted of 15, 90-minute group treatment sessions incorporating children and one or more caregivers. UP-C is comprised of five core treatment principles which, together, are designed to target difficulties in emotion reactivity and regulation across emotional disorders: 1) Increasing emotional awareness; 2) Increasing cognitive flexibility and linking thoughts to sensations; 3) Challenging maladaptive threat appraisals and negative thinking using antecedent cognitive reappraisal; 4) Preventing emotional avoidance by developing and practicing present-focused awareness; and 5) Identifying and modifying maladaptive action tendencies through exposure and behavioral activation. Treatment is organized into five primary sections around these core principles, using the acronym "CLUES" to reinforce UP-C's detective theme ("Consider How I Feel;" "Look At My Thoughts;" "Use Detective Thinking;" "Experience My Fears and Feelings;" "Stay Healthy and Happy"). Table 1 specifies the skills delivered in each of these five sections of treatment, as well as aspects of emotion reactivity and regulation targeted by each skill. A parent treatment component was held concurrently with the child treatment component to teach parents anxiety management strategies, reinforce skills, and plan for at-home exposures. For a more comprehensive discussion of manual development, see Ehrenreich-May and Bilek (2012).

Cool Kids condition. The Cool Kids (CK) treatment program (Lyneham et al., 2003) is a manual-based group treatment protocol adapted from the Coping Cat (Kendall & Hedtke, 2006). CK delivers CBT-based skills in an anxiety-focused (rather than transdiagnostic) format, including emotion recognition, cognitive restructuring, child management, social skills training, and graduated exposure (Hudson et al., 2009). The CK program has strong research support as a group protocol for the treatment of anxiety disorders in children ages seven and above (Hudson et al., 2009). For the current trial, each treatment session was two hours in length, and clinicians met with parents for a portion of each session (approximately 45 minutes) to discuss management of child anxiety and the parent's role in supporting their child's treatment goals. Clinicians met with parents and children on consecutive weeks for the first five treatment sessions and on alternating weeks for the final five treatment sessions, such that start and completion dates were consistent across the two interventions. Despite the difference between conditions in number of sessions, the amount of time spent in treatment was roughly equivalent across conditions (20 hours for CK and 22.5 hours for UP-C). Table 3 provides a week-by-week and session-by session description of the primary treatment components and the timing of their delivery for both conditions.

Therapists and Treatment Integrity

Therapists for both the UP-C condition and CK condition were primarily beginning pre-doctoral Clinical Psychology graduate students who provided group treatment through a research protocol and/or as part of their clinical practicum. Several more advanced pre-doctoral students also served as group therapists. Prior to the onset of the investigation, one of the UP-C developers received training in the CK condition

directly from the developers in Australia, including meetings with clinical supervisors, shadowing of CK treatment groups, and supervised coding of CK adherence. All therapists received at least one four-hour training in their respective protocols prior to the first group session, led by Dr. Ehrenreich-May and advanced pre-doctoral graduate students who were part of the manual development team for UP-C. All therapists received weekly supervision, during which treatment adherence was closely monitored. Treatment fidelity was established by randomly selecting and rating 20% of treatment sessions from each condition. UP-C and CK videos were coded for adherence by two separate independent raters, and 50% of videos were double-coded to establish inter-rater reliability. All skills in each session were rated dichotomously (0 = skill not covered by clinicians; 1= skill covered by clinicians). Based on ratings provided by the primary coder, therapist adherence for both the UP-C condition (95.80%) and the CK condition (91.00%) were high and did not differ between conditions, $\chi^2(1) = .25$, $p = .63$. There was 100% agreement between raters on UP-C sessions that were double-coded, and there was excellent agreement between raters on CK sessions that were double-coded, $\kappa = .85$, p<.001.

Measures

    ***Anxiety Disorders Interview Schedule for the DSM-IV-Child and Parent Reports* (ADIS-IV-C/P; Silverman & Albano, 1996)**. The ADIS-IV-C/P is a semi-structured interview that facilitates the diagnosis of DSM-IV anxiety, mood, and externalizing disorders in children ages 6 to 17. Based on parent and child report of symptoms and impairment, each disorder is assigned a clinician severity rating (CSR) ranging from 0 to 8, indexing the severity of the disorder from "none" to "extreme,"

respectively. Disorders assigned a CSR rating of between 0 and 3 are considered subclinical, while a CSR rating of between 4 and 8 indicates the presence of a clinical diagnosis. The principal diagnosis for this investigation was the disorder assigned the highest CSR rating, indicating the highest amount of disorder-associated impairment. The ADIS-IV-C/P has excellent inter-rater reliability (Lyneham, Abbott, & Rapee, 2007), test-retest reliability (Silverman, Saavedra, & Pina, 2001), and concurrent validity (Wood, Piacentini, Bergman, McCracken, & Barrios, 2002). Clinicians conducting evaluations for this investigation participated in a rigorous training procedure. Before conducting the ADIS-IV-C/P independently, clinicians were required to observe three ADIS assessments, conduct one collaborative assessment with a trained interviewer, and reach agreement with an expert rater (Dr. Ehrenreich-May) on all clinical diagnoses and CSR levels (i.e., within ± 1 CSR value) across three separate assessments. To protect against rater drift, all assessments were reviewed in weekly diagnostic meetings, at which the primary supervisor (Dr. Ehrenreich-May) and other expert raters were present. An adolescent sample assessed at the same clinic demonstrated very high inter-rater reliability for principal diagnoses and CSR values ($\kappa = 0.82$, $p < .001$; Ehrenreich-May et al., under review). For this trial, a principal diagnosis that received a CSR of 3 or below was considered to be indicative of diagnostic remission for the principal diagnosis; if all emotional disorder diagnoses received a CSR of 3 or below, remission of all emotional disorder diagnoses was indicated.

Clinician Global Impression-Improvement Scale **(CGI-I; Guy, 1976).** The CGI-I is a global rating of improvement in severity of all emotional disorder diagnoses, with lower scores indicating greater improvement. The CGI-I is rated on a 7-point scale

ranging from 1 (*very much improved*) to 7 (*very much worse*). All improvement is rated relative to Pre-Tx severity. A score of 1 (*very much improved*) or 2 (*much improved*) at Post-Tx or FU indicated meaningful improvement in emotional disorder severity and was considered indicative of treatment response, consistent with other trials (e.g., Walkup et al., 2008).

   ***Screen for Child Anxiety Related Emotional Disorders—Child and Parent Reports* (SCARED; Birmaher et al., 1997).** The SCARED is a 41-item child- and parent-report questionnaire assessing symptoms of anxiety in youth ages 7 to 19. Respondents are asked to rate the frequency with which they (or their child) have experienced each symptom over the past three months using a 3-point Likert type scale (0 = *almost never*; 1 = *sometimes*; 2 = *often*). Items are summed to yield an overall score of anxiety symptoms, and five distinct subscales assess symptoms of panic, social anxiety, school avoidance, separation anxiety, and generalized worry. The SCARED has good internal consistency (Muris, Merckelbach, van Brakel, Mayer, & van Dongen, 1998; Muris et al., 1999) and test-retest reliability for the overall anxiety composite (.81; Muris et al., 1999), as well as rater agreement between parent and child in the moderate range (Birmaher et al., 1997; Birmaher et al., 1999). Convergence has been established with other self- and parent-reported measures of anxiety (Muris et al., 1998), and the measure's factor structure has been replicated in youth from a variety of ethnic/racial

backgrounds (Gonzalez et al., 2012; Skriner & Chu, 2014). Internal consistency for the current sample was excellent for both child (Cronbach's α = .94) and parent reports (Cronbach's α = .91).

Children's Depression Inventory—Child and Parent Reports **(CDI; Kovacs, 1992)**. The CDI is a 27-item self- and parent-report questionnaire assessing depressive symptoms in youth ages 7 to 18. Each item asks the respondent to select one of three options that best describes their (or their child's) feelings, thoughts, and behaviors during the past two weeks. The CDI yields an overall score, with scores over 12 indicating the presence of significant depressive symptoms (Kovacs, 1992). The CDI is one of the most widely used measures of depression in youth, and pooled estimates indicate that the measure has good reliability (Cronbach's α = .86), good sensitivity and specificity, and high diagnostic accuracy (Stockings et al., 2015). Internal consistency for the current sample was excellent for child-reported symptoms (Cronbach's α = .94) and good for parent-reported symptoms (Cronbach's α = .84).

Children's Emotion Management Scales **(CEMS; Zeman, Shipman, & Penza-Clyve, 2001; Zeman, Cassano, Suveg, & Shipman, 2010)**. The CEMS are comprised of the Children's Sadness Management Scale (CSMS; Zeman et al., 2001), the Children's Anger Management Scale (CAMS; Zeman et al., 2001), and the Children's Worry Management Scale (CWMS; Zeman et al., 2010). The CEMS assesses children's methods of regulating or managing their emotions, including anger, sadness, and worry. Three subscales assess separate dimensions of emotion management, including Inhibition (i.e., suppression of emotion), Dysregulated Expression (i.e., non-constructive expression of emotion), and Coping (i.e., adaptive coping with emotion). Children are asked to

indicate the frequency with which they exhibit each emotion management strategy on a 3-point scale (1 = *hardly ever*; 2 = *sometimes*; 3 = *often*). Only the Dysregulated Expression subscales for anger, sadness, and worry were used in the current study. In a community sample of fourth and fifth grade children, the Sadness Dysregulation subscale demonstrated adequate internal consistency (Cronbach's α = .60) and was positively related to other self-report measures of emotion dysregulation and both self- and parent-reported internalizing problems, including both anxiety and depression (Zeman et al., 2001). In a community sample of children ages 6 to 12, the Worry Dysregulation subscale demonstrated adequate internal consistency (Cronbach's α = .72) and was significantly associated with emotional lability, poor emotional awareness, and anxiety/depression (Zeman et al., 2010). Scores on the Worry Dysregulation subscale also discriminated between children with and without an anxiety disorder diagnosis in a clinical sample (Zeman et al., 2010). In the current sample, internal consistency for the child-reported Anger Dysregulation subscale (Cronbach's α = .68) and parent-reported Anger Dysregulation subscale (Cronbach's α = .70) were adequate. Internal consistency for the child-reported Sadness Dysregulation subscale was also adequate (Cronbach's α = .69), while internal consistency for the parent-reported Sadness Dysregulation subscale was somewhat low (Cronbach's α = .58). Internal consistency for the child-reported Worry Dysregulation subscale (Cronbach's α = .64) and parent-reported Worry Dysregulation subscale (Cronbach's α = .67) were adequate.

***Emotion Regulation Questionnaire for Children and Adolescents (*ERQ-CA; Gullone & Taffe, 2012)**. The ERQ-CA is a downward adaptation of the Emotion Regulation Questionnaire (ERQ; Gross & John, 2003) assessing the emotion regulation

strategies of cognitive reappraisal (6 items) and expressive suppression (4 items) in children and adolescents. Participants rate the extent to which they agree with each statement using a 5-point scale (0 = *strongly disagree*; 1 = *disagree*; 2 = *half and half*; 3 = *agree*; 4 = *strongly agree*). In a large community sample of youth ages 10 to 17, internal consistency was good for both the reappraisal subscale (Cronbach's α = .83) and the expressive suppression subscale (Cronbach's α = .75). The expressive suppression subscale was positively correlated with self-reported depressive symptoms and self-reported neuroticism, while the cognitive reappraisal subscale was negatively correlated with self-reported depressive symptoms and neuroticism (Gullone & Taffe, 2012). In the current sample, internal consistency for both the Cognitive Reappraisal subscale (Cronbach's α = .77) and the Expressive Suppression subscale (Cronbach's α = .62) were adequate.

Positive and Negative Affect Scale **(PANAS; Watson, Clark, & Tellegen, 1988)**. The version of the PANAS used in this study included a combination of 10 positive and 10 negative affect items from the original PANAS developed in 1988 by Watson and colleagues, as well as seven additional items. Four of these items (sad, mad, happy, worried, afraid) also appear on the child version of the PANAS (PANAS-C; Laurent et al., 1999), and three additional items were added for the purpose of assessing affective states targeted by the current study protocol (depressed, worried, uneasy). Participants were asked to rate the degree to which they have experienced a variety of positive and affective states within the past week (1 = *very slightly or not at all*; 2 = *a little*; 3 = *moderately*; 4 = *quite a bit*; 5 = *extremely*). The original PANAS has good internal consistency for the PA scale (Cronbach's α = .87) and the NA scale (Cronbach's

α = .87), good test-retest reliability, and demonstrated convergent validity with symptoms of depression and generalized distress (Watson et al., 1988). The psychometric properties of the PANAS have been replicated in adolescent samples (Huebner & Dew, 1995). Internal consistency for the current sample was good for both the Positive Affect (Cronbach's α = .84) and Negative Affect (Cronbach's α = .90) subscales.

Data Analysis Plan

Data screening examined univariate normality; estimates of skewness and kurtosis were within normal limits, defined by Kline (2011) as skewness value less than 3 and kurtosis value less than 8. No significant outliers ($z \geq 3$) were identified on any variable of interest at any time point. Mahalanobis D and Cook's D were computed to screen for multivariate outliers, and results did not indicate the presence of multivariate outliers.

There were several sources of missing data, including missed assessments (see consort diagram in Figure 1) and missing items. All participants had complete data at the Pre-Tx assessment (i.e., baseline assessment for those assessed < one month prior to group start and waitlist assessment for those assessed > one month before group start). The Post-Tx assessment was completed by 85.10% of participants ($n = 40$), and 59.60% of participants ($n = 28$) completed the FU assessment. There were no significant differences between those with and without missing data at Post-Tx and FU with respect to treatment condition, demographic variables (e.g., age, gender, ethnicity, family income), diagnostic variables (e.g., principal diagnosis CSR, number of diagnoses), symptom measures (e.g., child- and parent-rated CDI and SCARED), or other relevant outcome measures (e.g., child- and parent-rated CEMS subscales, child-rated ERQ-CA subscales, child-rated PANAS subscales). Additionally, missing data pattern analysis

using Little's Missing Completely at Random (MCAR) Test (Little, 1998) did not reveal any significant patterns of missing data, $\chi^2(31368) = 1980.99$, p=.98. Given no evidence that missingness was related to any variables of interest, data were assumed to be at least missing at random (MAR). Diagnostic remission and treatment response status were analyzed in two ways, first including only those participants who completed the Post-Tx or FU time points. Diagnostic remission and treatment response status were also analyzed using an intention to treat (ITT) approach, including all children who were randomized to a condition with the last observation carried forward (LOCF) in the case of missing data. For continuous self- and parent-report variables, missing data was imputed using full information maximum likelihood (FIML) estimation procedures in MPlus.

As data were clustered not only by intervention condition but also by group, we first tested for non-independence of scores clustered within groups by examining the intraclass correlation (ICC) and the design effect (DE) for each outcome variable at each time point. The ICC estimates the proportion of total score variability explained by the cluster variable; thus, if the ICC = .10, then scores within the same cluster are 10% more likely to have a similar value compared with two scores selected completely at random in the population (Kline, 2011). The ICC can be used to calculate the design effect, or the increase in sampling error in a complex sample compared with simple random sampling of individual cases with no cluster, using the formula 1 + (mean cluster size – 1)*ICC. The recommended cutoff for design effect is 2.0, meaning that the variance is two times greater in the complex sampling design than in the design with simple random sampling (Kline, 2011). We examined all outcome variables for a design effect over 2.00 when considering whether to cluster individuals within groups. Design effects for relevant

outcome variables ranged from 1.01 to 2.49, with only three variables exhibiting a design effect greater than the recommended cutoff of 2.00. Given that independence of scores within group clusters was supported for the vast majority of variables, we decided not to take clustering by group into account in our analyses.

For categorical, diagnostic outcomes (i.e., remission of principal diagnosis, remission of all emotional disorder diagnoses, CGI-I treatment response), treatment conditions were compared with Pearson's chi-square test. Diagnostic remission of principal and all emotional disorder diagnoses at Post-Tx and FU was classified as the absence of the principal diagnosis and the absence of all emotional disorder diagnoses, respectively (i.e., ADIS CSR<4), while treatment response was classified as a CGI-I score of "1" or "2." Effect sizes for chi-square analyses were calculated as Odds Ratios (ORs).

For dimensional outcomes, latent growth curve models (LGMs) were estimated for each outcome variable in Mplus, Version 7.0 (Muthén & Muthén, 2012). In LGM, observed measurements at each time point are represented as indicators of an intercept latent growth factor and a slope latent growth factor. Unstandardized loadings of each indicator on the intercept latent growth factor are fixed to 1, and loadings on the slope latent growth factor are fixed to constants that correspond to times of measurement for observed variables, which may be evenly or unevenly spaced. One measurement time point (typically but not necessarily the first time point) is selected as the centering point by setting its indicator loading on the slope latent growth factor to 0. The mean of the intercept growth factor is then interpreted as the mean level of the outcome variable at the centering time point, and the centering time point can be varied to describe mean levels

of the outcome variable at different time points (Muthén & Muthén, 2000). In addition to estimating the mean of the intercept growth factor, its variance can also be estimated to provide information about individual variation in levels of the outcome variable at the centered time point. The mean of the slope latent growth factor represents the average amount of change in the outcome variable for a one-unit change in time, adjusted for measurement error, and the variance of the slope factor corresponds to individual variation in slope. Covariance of the intercept and slope growth factors is also estimated to indicate whether the level of the outcome variable at the centering time point predicts subsequent rate of change in the outcome. Unlike in ANOVA, where error variances of outcome variables at adjacent time points are assumed to be equal and independent, error covariance for adjacent variables is assumed in LGM models.

For the current study, we first examined plots of estimated means for each treatment condition at Pre-Tx, Mid-Tx, Post-Tx, and FU to determine whether the slope of both treatment conditions appeared to exhibit the same functional form (e.g., both linear, both quadratic, etc.). If functional forms differed between conditions, separate LGMs were estimated to describe the slope for each condition. If functional forms were equivalent between conditions, we began by testing a linear slope for the entire sample and adding a quadratic slope in a second step as necessary and when supported by visual inspection of means plots. For the linear growth factor, slope factor loadings were fixed to -2, -1, 0, and 2.5 (corresponding to Pre-Tx, Mid-Tx, Post-Tx, and FU time points, respectively) for the majority of outcome variables. Each one unit change in time corresponds to a period of eight weeks. Centering the slope growth factor on Post-Tx allowed us to examine whether intervention condition significantly impacted the intercept

growth factor for each outcome variable at Post-Tx. Multiple indices of model fit were examined to evaluate goodness of model fit to the data, including a non-significant chi-Square ($p>.05$), a comparative fit index (CFI) $>.95$, a root mean square error of approximation (RMSEA) $<.06$, and an SRMR $<.08$ (Kline, 2011). After testing the linear growth factor in the overall sample, a quadratic growth factor was also specified when warranted by squaring the time score loadings on the latent slope factor (e.g., 4, 1, 0, -6.5 for most outcome variables). If the mean of the quadratic latent factor was significant and an examination of the estimated mean slope plots for each time point supported the addition of the quadratic factor, the quadratic factor was retained. Significance tests for parameter estimates were conducted using the two-tailed z-statistic ($z = \pm1.96$, $p<.05$).

Next, the intercept and slope growth factors were regressed on a dummy-coded covariate representing the treatment effect ($0 = $ CK; $1 = $ UP-C). A significant mean intercept was interpreted to mean a between-condition difference in the outcome variable at Post-Tx, while a significant mean slope was interpreted to mean a between-condition difference in the average rate of change in the outcome variables between conditions. A final LGM was specified with the slope factor loading centered on the FU time point to examine between-condition differences in each outcome variable at FU. Figure 2 illustrates the basic linear LGM model tested for all continuous outcome variables, including the dummy-coded covariate representing the effect of treatment on the intercept and slope factors.

Chapter 3: Results

Randomization Success

There were no significant differences between the UP-C and CK conditions at Pre-Tx with respect to demographic variables of interest, including age ($t[45] = -.15$, $p = .88$), sex ($\chi^2[1] = 2.55$, $p = .15$), or annual family income ($t[45] = -.51$, $p = .611$). Additionally, conditions did not differ with respect to diagnostic variables, including severity of the principal diagnosis ($t[45] = .70$, $p = .49$), the number of clinical emotional disorder diagnoses (e.g., DSM-IV-T/R anxiety or mood disorders; $t[45] = -.37$, $p = .71$), the number of subclinical emotional disorder diagnoses ($t[45] = .76$, $p = .45$), or the presence of either a clinical or sub-clinical depressive disorder diagnosis ($\chi^2[2] = 4.08$, $p = .13$). With regard to CDI scores, there were no condition-related differences in number of children above the CDI cutoff of 12 on either child report of depression symptoms ($\chi^2[1] = .05$, $p = .82$) or parent report of depression symptoms ($\chi^2[1] = .01$, $p = .94$). Further, conditions did not differ on any other parent- or child-rated measure (see Table 4).

Attendance and Attrition

Due to the unequal number of possible sessions between conditions (15 for UP-C vs. 10 for CK), binomial logistic regression was conducted using the generalized linear model function in SPSS to examine possible group differences in attendance. Results revealed that there were no between-condition differences in the proportion of number of

sessions attended relative to total sessions, Wald $\chi^2(1) = .03$, p = .86. CK participants attended an average of 86.52% of sessions, while UP-C participants attended an average of 85.42% of sessions.

In total, 40 out of 47 randomized subjects completed the post-treatment assessment. Completion rates for the follow-up time point were somewhat lower, with 28 out of 47 randomized subjects (59.60%) participating in the follow-up assessment. There were no significant between-condition differences in either completion of the Post-Tx assessment ($\chi^2[1] = .22$, p = .70) or completion of the FU assessment ($\chi^2[1] = 2.58$, p = .14).

Aim 1: Posttreatment and Follow-Up Diagnostic Outcomes

Treatment remission. For all clinician-rated outcomes, parenthetical remission/response rates are listed for UP-C first, followed by CK. UP-C and CK participants were equally likely to achieve remission of their principal diagnosis at Post-Tx when both those who completed the Post-Tx assessment (61.9% [$n = 13$] vs. 57.9% [$n = 11$]), $\chi^2[1] = .067$, $p = .80$, $OR = 1.18$) and the ITT sample (54.2% [$n = 13$] vs. 45.8% [$n = 11$], $\chi^2[1] = .19$, $p = .77$, $OR = 1.28$) were examined. UP-C and CK participants were also equally likely to achieve remission of all emotional disorder diagnoses at post-treatment when both those who completed the Post-Tx assessment (57.1% [$n = 12$] vs. 40.00% [$n = 8$], $\chi^2[1] = .90$, $p = .34$, $OR = 1.83$) and the ITT (50.00% [$n = 12$] vs. 40.00% [$n = 8$], $\chi^2[1] = 1.11$, $p = .38$, $OR = 1.88$) were examined.

UP-C and CK participants did not differ in likelihood of achieving remission of their principal diagnosis at FU when both those who completed the FU assessment (70.60% [$n = 12$] vs. 63.60% [$n = 7$], $\chi^2[1] = .15$, $p = .70$, $OR = 1.39$) and the ITT sample

(58.30% [n = 14] vs. 43.50% [n = 10], χ^2[1] = 1.04, p = .31, OR = 1.83) were examined. UP-C and CK participants also did not differ in likelihood of achieving remission of all emotional disorder diagnoses at follow-up when both those who completed the FU assessment (64.70% [n = 11] vs. 54.50% [n = 6], χ^2[1] = .29, OR = 1.52) and the ITT sample (50.00% [n = 12] vs. 34.80% [n = 8], χ^2[1] = 1.11, OR = 1.88) were examined.

Treatment response. There was no difference between UP-C and CK participants in likelihood of achieving treatment responder status at Post-Tx when both those who completed the Post-Tx assessment (71.4% [n = 15] vs. 68.4% [n = 13], χ^2[1] = .043, p = .84, OR = 1.11) and when the ITT sample were examined (62.5% [n = 15] vs. 56.50% [n = 13], χ^2[1] = .17, p = .68, OR = 1.28). However, UP-C participants were more likely than CK participants to achieve treatment responder status at FU when those participants who completed the FU were examined (94.10%, [n = 16] vs. 54.4% [n = 6], χ^2[1] = 6.21, p = .01, OR = 13.33). When FU responder status was examined using the ITT sample, UP-C and CK participants did not differ in their likelihood of achieving responder status (75.0% [n = 18] vs. 52.20% [n = 12], χ^2[1] = 2.65, p = .10, OR = 2.75).

Aim 2: LGMs for Parent- and Child-Rated Anxiety Symptoms (SCARED)

Correlations between all continuous parent- and child-rated study variables, including symptom measures and measures of emotion regulation and reactivity, can be found in Table 5. Results of LGMs for each variable can be found in Table 6.

Parent-rated anxiety symptoms (SCARED). Figure 3 presents estimated mean levels of parent-reported SCARED Total Anxiety scores for the sample as a whole, for the UP-C condition, and for the CK condition. An examination of the plotted means suggested that, for both conditions, there was little change in parent-reported SCARED

scores from Pre-Tx to Mid-Tx, a substantial decrease in scores from Mid-Tx to Post-Tx, and a smaller decrease in scores from Post-Tx to FU. Based on an examination of the plotted means, the UP-C and CK conditions appeared to have the same functional form. The linear LGM for the combined sample was a poor fit for the data, $\chi^2(5) = 19.84$, $p = .001$, RMSEA = .26, CFI = .81, SRMR = 13. Given the poor model fit, as well as evidence of a leveling out of parent-rated anxiety symptoms between Post-Tx and FU, a second linear LGM was estimated in which the first three loadings from the slope to the indicators were set at -2, -1, and 0, and the loading from the FU time point was freely estimated. This final model demonstrated acceptable fit by most indices, $\chi^2(5) = 10.59$, $p = .06$, RMSEA = 1.61, CFI = .93, SRMR = .09. The final loading for the slope factor was estimated at .44, indicating that reductions in parent-reported anxiety leveled out approximately 4 weeks after the Post-Tx time point and showed little continued change. The slope factor mean was significant and negative (M_s= -5.02, SE = .91, $p<.001$), indicating that, on average, parent-rated SCARED scores decreased 5.02 points every eight weeks between Pre-Tx and 4 weeks after the Post-Tx time point.

When the dummy-coded treatment covariate was added to the final model, the effect of the intervention on the slope factor was not significant ($B = -.85$, $SE = 1.55$, $\beta = -.22$, $z = -.54$, $p = .59$), indicating that the intervention was unrelated to the rate of decrease in parent-reported SCARED scores between Pre-Tx and 4 weeks after the Post-Tx time point. The effect of the intervention on the intercept factor with time score loadings centered at Post-Tx was also not significant, indicating that treatment condition was unrelated to parent-rated SCARED scores at Post-Tx ($B = -4.33$, $SE = 3.49$, $\beta = -.42$, $z = -1.26$, $p = .58$). In the final model where the time score was centered at 4 weeks after

Post-Tx (the point when parent-rated anxiety levels out), the effect of the intervention on the intercept factor was also not significant, indicating that treatment condition was unrelated to parent-rated SCARED scores (B = -3.92, SE = 3.60, β = -.20, z = -1.10, p = .27).

Child-rated anxiety symptoms (SCARED). Figure 4 presents estimated mean levels of child-rated SCARED Total Anxiety scores for the sample as a whole, for the UP-C condition, and for the CK condition. An examination of the plotted means provided evidence of a negative, linear slope for both conditions, suggesting that the slopes for UP-C and CK have the same functional form. Fit statistics for overall the linear LGM for the combined sample were adequate for most indices, $\chi^2(5)$ = 7.48, p = .19, RMSEA = .11, CFI = .95, SRMR = .08. The mean of the slope factor was significant and negative (M_s = -3.93, SE = .62, $p<.001$), indicating that, on average, child-reported SCARED Total Anxiety scores decreased by 3.93 points every eight weeks during the treatment and follow-up periods. When the quadratic LGM was tested, the mean of the quadratic slope factor was not significant (M_q= .73, SE = .38, p = .07), confirming that the model specifying a linear slope factor best fit the data and should be retained.

When the dummy-coded treatment covariate was added to the model, the effect of the intervention on the slope factor was not significant (B = -.40, SE = 1.25, β = -,17, z = -.32, p = .75), indicating that treatment condition was unrelated to the rate of decrease in child-reported anxiety symptoms during treatment and follow-up. The effect of the intervention on the intercept factor with time score loadings centered at Post-Tx was also not significant, indicating that treatment condition was unrelated to level of child-rated anxiety at Post-Tx (B = .64, SE = 3.61, β = .06, z = .18, p = .86). In the final model

where the time score was centered at the FU time point, the effect of the intervention on the intercept factor was also not significant, indicating that treatment condition was unrelated to levels of child-rated anxiety at FU (B = -.35, SE = 3.23, β = -.07, z = -.11, p = .92).

Aim 3: LGMs for Parent- and Child-Rated Depression Symptoms (CDI)

Parent-rated depression symptoms (CDI). Figure 5 presents estimated mean levels of parent-rated CDI Total Depression scores for the sample as a whole, for the UP-C condition, and for the CK condition. An examination of the plotted means provided evidence of a negative, linear slope for both conditions, suggesting that the slopes for UP-C and CK have the same functional form. The linear LGM for parent-rated CDI Total Depression was an adequate fit for the data by some indices, $\chi^2(6)$ = 12.50, p = .052, RMSEA = .16, CFI = .84, SRMR = .10. The slope factor mean was significant and negative (M_s= -1.30, SE = .213, p<.001), indicating that, on average, parent-rated depression scores decreased 1.30 points every eight weeks during the treatment and follow-up periods.

Despite the lack of significant between-condition difference in observed sample means for Pre-Tx parent-rated CDI Total Depression, the effect of treatment condition on Pre-Tx parent-rated CDI Total Depression using estimated scores was significant (B = -3.85, SE = 1.75, β = -.77, z = -2.42, p <.05), indicating that scores of UP-C participants were .77 SD lower than scores of CK participants at Pre-Tx. Therefore, we controlled for Pre-Tx differences in parent-rated CDI Total Depression scores when examining the effect of the intervention on slope and intercept. When the dummy-coded treatment covariate was added to the model, the effect of the intervention on the linear slope factor

was not significant ($B = .39$, $SE = .42$, $\beta = .41$, $z = .95$, $p = .34$), indicating that treatment condition did not impact rate of linear change during the treatment or follow-up periods. Controlling for between-condition differences in Pre-Tx scores, the effect of the intervention on the intercept factor with time score loadings centered at Post-Tx was significant, indicating that participants in the UP-C condition had lower levels of parent-reported CDI Total Depression scores at Post-Tx ($B = -2.55$, $SE = 1.18$, $\beta = -.72$, $z = -2.50$, $p < .05$). Specifically, scores of UP-C participants were .72 SD lower than scores of CK Participants at Post-Tx. In the final model where the time score was centered at the FU time point, the effect of the intervention on the intercept factor controlling for between-condition differences in parent-rated depression scores at Pre-Tx was no longer significant, indicating that participants had similar parent-rated depression scores at FU ($B = .23$, $SE = 2.44$, $\beta = .03$, $z = .09$, $p = .93$).

Child-rated depression symptoms (CDI). Figure 6 presents estimated mean levels of child-rated CDI Total Depression scores for the sample as a whole, for the UP-C condition, and for the CK condition. An examination of the plotted means suggested that the slopes for the UP-C and CK conditions differed with respect to their functional form. An examination of the plotted means for the UP-C condition revealed a roughly linear, decreasing slope, while examination of the plotted means for the CK condition revealed decreasing scores from the Pre-Tx through Post-Tx, and then increasing scores from the Post-Tx through FU periods (i.e., a quadratic slope). Given the different functional forms, models were estimated separately for each condition.

For the UP-C condition, a linear LGM demonstrated good model fit by most indices, $\chi^2(6) = 6.70$, $p = .35$, RMSEA = .07, CFI = .97, SRMR = .19. The mean of the

slope factor was significant and negative (M_s = -1.04, SE = .37, p <.01), indicating that, on average, children's CDI scores decreased by 1.04 points every eight weeks during the treatment and follow-up periods in the UP-C condition. When a quadratic LGM was tested, the mean of the quadratic slope factor was not significant (M_q= -.09, SE = .18, p = .59), suggesting that the model specifying a linear slope factor best fit the data and should be retained for the UP-C condition.

For the CK condition, as expected based on the estimated means plot, the linear LGM was a very poor fit for data, $\chi^2(7)$ = 33.71, RMSEA = .41, CFI = .35, SRMR = 1.17. When a quadratic LGM was estimated, the model was a good fit for the data by most indices, $\chi^2(1)$ = 3.00, p = .084, RMSEA = .29, CFI = .95, SRMR = .14. The mean of the linear slope factor was significant and negative (M_s = -1.02, SE = .43, p <.05), indicating that, on average, children's CDI Total Depression scores decreased by 1.02 points every 8 weeks between Pre-Tx and Post-Tx for children in the CK condition. The mean of the quadratic slope factor was significant and positive (M_q = .48, SE = .09, p<.001), reflecting a significant increase in child-reported CDI Total Depression scores between the Post-Tx and FU periods.

Finally, given that the slope from Pre-Tx to Post-Tx appeared roughly linear for both groups, we estimated a linear LGM for the Pre-Tx through FU time points for the combined sample (time scores = -2, -1, 0). The model was an excellent fit for the data, $\chi^2(2)$ = .84, p = .66, RMSEA = .00, CFI = 1.00, SRMR = .04. When the dummy-coded intervention covariate was added to the model, neither the effect of intervention on the slope facor (B = 1.46, SE = .94, β = .37, z = 1.55, p = .12) nor on the intercept factor (B = 1.81, SE = 1.53, β = .54, z = 1.18, p = .24) was significant. Both conditions exhibited

similar rates of decrease in CDI Total Depression scores from Pre-Tx to Post-Tx, and scores did not differ between conditions at Post-Tx.

Aim 4: LGMs for Parent- and Child-Rated Emotion Dysregulation, Cognitive Reappraisal, Expressive Suppression, Positive Affect, and Negative Affect

Parent-rated anger dysregulation (CEMS). An examination of the estimated mean plots revealed a flat slope across conditions, suggesting minimal change in parent-rated CEMS Anger Dysregulation over time. The linear LGM was an adequate fit for the data by some indices, $\chi^2(5) = 9.04$, $p = .11$, RMSEA = .13, CFI = .93, SRMR = .28. The linear slope factor mean was not significant, indicating that there was no significant linear change in parent-rated CEMS Anger Dysregulation during the treatment and follow-up periods ($M_s = -.08$, $SE = .05$, $p = .12$). Given the insignificant findings and the apparently flat slope, treatment condition was not examined as a predictor of slope or intercept.

Parent-rated sadness dysregulation (CEMS). Figure 7 presents estimated mean levels of parent-rated CEMS Sadness Dysregulation scores for the sample as a whole, for the UP-C condition, and for the CK condition. An examination of the estimated mean plots revealed that participants in the CK condition evidenced a flat slope over time, while participants in the UP-C condition evidenced a gradual decrease between Pre-Tx and FU. Despite the apparent difference in rate of change, the slopes for both conditions were judged to exhibit the same functional form. Due to a small and non-significant negative residual variance for the slope factor, the residual variance was fixed to zero. The linear LGM for parent-rated CEMS Sadness Dysregulation for the entire sample was a good fit for the data by most indices, $\chi^2(7) = 8.00$, $p = .33$, RMSEA = .06, CFI = .98, SRMR = .18. The linear slope factor mean was significant and negative ($M_s = -.22$, $SE =$

.05, *p*<.001), indicating that, on average, there was a .22 point decrease in sadness

dysregulation for every eight weeks between the Pre-Tx and FU time points.

When the dummy-coded treatment covariate was added to the final model, the

effect of the intervention on the slope factor was significant (*B* = -.11, *SE* = .11, β = -

2.00, *z* = -9.38, *p* <.001), indicating that participants in the UP-C group experienced a

2.00 SD faster decrease in parent-reported CEMS Sadness Dysregulation than

participants in the CK condition. The effect of the intervention on the intercept factor

with time score loadings centered at post-treatment was also significant, indicating that

participants in the UP-C condition had .69 SD lower levels of parent-reported CEMS

Sadness Dysregulation at post-treatment (*B* = -.94, *SE* = .43, *β* = -.69, *z* = -2.31, *p* <.05).

In the final model where the time score was centered at the FU time point, the effect of

the intervention on the intercept factor was also significant and negative, indicating that

participants in the UP-C condition had .87 SD lower levels of parent-reported sadness

dysregulation at FU (*B* = -1.23, *SE* = .55, *β* = -.87, *z* = -2.53, *p* <.05).

Parent-rated worry dysregulation (CEMS). Figure 8 presents estimated mean

levels of parent-rated CEMS Worry Dysregulation scores for the sample as a whole, for

the UP-C condition, and for the CK condition. An examination of the estimated mean

plots revealed that participants in the CK condition evidenced a relatively flat slope over

time with minimal fluctuations, while participants in the UP-C condition evidenced

minimal change between Pre-Tx and Mid-Tx and gradual change between Mid-Tx and

FU. The slopes for both conditions were initially analyzed together, with both linear and

quadratic models tested based on visual inspection of estimated means. Due to a small

and insignificant negative residual variance for the linear slope factor, the residual

variance was fixed to zero. The linear LGM for parent-rated worry dysregulation for the entire sample was an adequate fit for the data by most indices, $\chi^2(5) = 10.65$, $p = .06$, RMSEA = .16, CFI = .93, SRMR = .18. The linear slope mean factor was significant and negative ($M_s = -.31$, $SE = .08$, $p<.001$), indicating that, on average, there was a .31 point decrease in parent-reported CEMS Worry Dysregulation for every eight weeks between the Pre-Tx and FU time points. The mean for the quadratic slope factor was not significant and was not included in the final model ($M_q = .06$, $SE = .05$, $p = .27$).

When the dummy-coded treatment covariate was added to the final model, the effect of the intervention on the slope factor was significant ($B = -.23$, $SE = .16$, $\beta = -2.00$, $z = -9.27$, $p <.001$), indicating that participants in the UP-C group experienced a 2.00 SD faster decrease in parent-reported CEMS Worry Dysregulation than participants in the CK condition. The effect of the intervention on the intercept factor with time score loadings centered at Post-Tx was not significant, indicating that participants in the UP-C condition did not have lower levels of parent-reported CEMS Worry Dysregulation at Post-Tx ($B = -.03$, $SE = .17$, $\beta = -.06$, $z = -.17$, $p = .87$). In the final model where the time score was centered at the FU time point, the effect of the intervention on the intercept factor was not significant, indicating that participants in the UP-C and CK conditions did not differ with respect to parent-reported CEMS Worry Dysregulation at FU ($B = -.66$, $SE = .74$, $\beta = -.37$, $z = -.91$, $p = .36$).

Child-rated anger dysregulation (CEMS). An examination of the estimated mean levels of child-reported anger dysregulation for the sample as a whole, the UP-C condition, and the CK condition revealed a flat slope across conditions, suggesting minimal change in anger dysregulation over time. The linear LGM for child-reported

CEMS Anger Dysregulation was a poor fit for the data, $\chi^2(5) = 13.62$, $p = .018$, RMSEA = .19, CFI = .68, SRMR = .17. The linear slope factor mean was not significant, indicating that there was no significant linear change in child-reported CEMS Anger Dysregulation during the treatment and follow-up periods ($M_s = .030$, $SE = .10$, $p = .77$). Given the insignificant findings and the apparently flat slope, intervention condition was not examined as a predictor of slope or intercept.

Child-rated sadness dysregulation (CEMS). An examination of the estimated mean plots revealed a flat slope across conditions, suggesting minimal change in child-rated CEMS Sadness Dysregulation over time. The linear LGM was a poor fit for the data by most indices, $\chi^2(6) = 10.24$, $p = .07$, RMSEA = .15, CFI = .72, SRMR = .18. The linear slope factor mean was not significant, indicating that there was no significant linear change in child-reported anger dysregulation during the treatment and follow-up periods ($M_s = -.16$, $SE = .10$, $p = .09$). Given the insignificant findings, and give the apparently flat slope, intervention condition was not examined as a predictor of slope or intercept.

Child-rated worry dysregulation (CEMS). An examination of the estimated mean plots revealed a flat slope across conditions, suggesting minimal change in child-reported CEMS Worry Dysregulation over time. The linear LGM was an excellent fit for the data, $\chi^2(5) = .54$, $p = .99$, RMSEA = .00, CFI = 1.00, SRMR = .05. The linear slope factor mean was not significant, indicating that there was no significant linear change in child-rated CEMS Worry Dysregulation during the treatment and follow-up periods ($M_s = -.06$, $SE = .08$, $p = .45$). Given the insignificant findings and the apparently flat slope, intervention condition was not examined as a predictor of slope or intercept.

Child-rated cognitive reappraisal (ERQ-CA). Figure 9 presents estimated mean levels of child-rated ERQ-CA Cognitive Reappraisal scores for the sample as a whole, for the UP-C condition, and for the CK condition. An examination of the estimated mean plots revealed that participants in the CK condition evidenced a slight increase in ERQ-CA Reappraisal between Pre-Tx and Mid-Tx, followed by a decrease between Mid-Tx and Post-Tx, followed by another slight increase between Post-Tx and FU. Of note, the Pre-Tx mean for CK participants (M = 14.20) was approximately the same as the Post-Tx mean (M = 13.70). In contrast, UP-C participants evidenced little change in ERQ-CA Reappraisal scores from Pre-Tx to Mid-Tx but a steady linear increase in scores between Mid-Tx and FU. The slopes for both conditions were initially analyzed together, with both linear and quadratic models tested based on visual inspection of estimated means. Due to a small and insignificant negative residual variance for the FU time point, the residual variance for FU was fixed to zero. The linear LGM for child-reported ERQ-CA Reappraisal for the entire sample was an excellent fit for the data by most indices, $\chi^2(6)$ = 1.74, p = .94, RMSEA = .00, CFI = 1.00, SRMR = .07. The linear slope factor mean was not significant (M_s = .36, SE = .31, p = .25), indicating that, on average, the combined sample did not evidence changes in ERQ –CA Reappraisal over treatment or follow-up. The mean for the quadratic slope factor was not significant and was not included in the final model (M_q = -.09, SE = .20, p = .64).

When the dummy-coded treatment covariate was added to the final model, the effect of the intervention on the slope factor was significant (B = 1.27, SE = .58, β = 1.01, z = 2.63, p <.01), indicating that participants in the UP-C group experienced a 1.01 SD faster increase in child-reported ERQ-CA Reappraisal scores compared to participants in

the CK condition. The effect of the intervention on the intercept factor with time score loadings centered at Post-Tx was marginally significant, indicating that participants in the UP-C condition had marginally higher (.65 SD) ERQ-CA Reappraisal scores at Post-Tx compared to CK participants (B = .33, SE = .18, β = .65, z = 1.85, p = .06). In the final model where the time score was centered at the FU time point, the effect of the intervention on the intercept factor was significant, indicating that participants in the UP-C condition evidenced .97 SD higher ERQ-CA Reappraisal scores compared to participants in the CK condition at FU (B = 5.83, SE = 2.24, β = .97, z = 3.18, p = .001).

Child-rated expressive suppression (ERQ-CA). Figure 10 presents estimated mean levels of child-rated ERQ Expressive Suppression scores for the sample as a whole, for the UP-C condition, and for the CK condition. An examination of the estimated mean plots revealed a flat slope across conditions, suggesting minimal change in expressive suppression over time. The linear LGM was an adequate fit for the data by most indices, $\chi^2(5)$ = 6.78, p = .24, RMSEA = .09, CFI = .94, SRMR = .17. As expected from the examination of estimated mean plots, the linear slope factor mean was not significant, indicating that there was no significant linear change in child-reported ERQ Expressive Suppression during the treatment and follow-up periods (M_s = .09, SE = .13, p = .49). Given the insignificant findings and apparently flat slopes, treatment condition was not examined as a predictor of slope or intercept.

Child-rated positive affect (PANAS-PA). Figure 11 presents estimated mean levels of child-rated PANAS Positive Affect scores for the sample as a whole, for the UP-C condition, and for the CK condition. An examination of the estimated mean plots revealed a relatively flat slope across conditions, suggesting minimal change in positive

affect over time. The slopes for both conditions were therefore judged to exhibit the same functional form. Due to a small and insignificant negative residual variance for the linear slope factor, the residual variance was fixed to zero, and Mid-Tx and Post-Tx PANAS Positive Affect scores were allowed to covary after examining modification indices. The final linear LGM for child-reported positive affect was an adequate fit for the data by most indices, $\chi^2(6) = 9.00$, $p = .17$, RMSEA = .10, CFI = .93, SRMR = .21. As expected from the examination of estimated mean plots, the linear slope factor mean was not significant, indicating that there was no significant linear change in child-reported PANAS Positive Affect during the treatment and follow-up periods ($M_s = -.52$, $SE = .46$, $p = .25$). Given the insignificant findings and apparently flat slopes, treatment condition was not examined as a predictor of slope or intercept.

Child-rated negative affect (PANAS-N). Figure 12 presents estimated mean levels of child-rated PANAS Negative Affect scores for the sample as a whole, for the UP-C condition, and for the CK condition. An examination of the plotted means provided evidence of a slight increase in PANAS Negative Affect scores in both groups from Pre-Tx to Mid-Tx, followed by a negative, linear slope for both conditions from Mid-Tx to FU, suggesting that the slopes for UP-C and CK have the same functional form. Fit statistics for overall the linear LGM for the combined sample were good for most indices, $\chi^2(5) = 5.79$, $p = .33$, RMSEA = .06, CFI = .98, SRMR = .12. The mean of the slope

factor was significant and negative (M_s = -1.41, SE = .50, p<.01), indicating that, on average, children's PANAS Negative Affect scores decreased by 1.41 points every eight weeks during the treatment and follow-up periods.

When the dummy-coded treatment covariate was added to the model, the effect of the intervention on the slope factor was not significant (B = .054, SE = 1.19, β = .03, z = .05, p = .96), indicating that intervention group did not impact that rate of change in child-rated negative affect during treatment and follow-up. The effect of the intervention on the intercept factor with time score loadings centered at Post-Tx was also non-significant, indicating that treatment condition was unrelated to levels of child-reported PANAS Negative Affect at Post-Tx (B = -2.67, SE = 2.77, β = -.34, z = -.97, p = .33). In the final model where the time score was centered at the FU time point, the effect of the intervention on the intercept factor was also not significant, indicating that treatment condition was unrelated to levels of child-reported PANAS Negative Affect at FU (B = -2.54, SE = 3.69, β = -.48, z = -.68, p = .50).

Exploratory Aim 5: Mediation Analyses for Higher Order Factors

To examine whether mediation analyses for hypothesized higher order factors were justified, we conducted repeated samples t-tests for each condition to test for significant change in hypothesized mediator variables from Pre-Tx to Mid-Tx. Within the CK sample, there were no significant change from Pre-Tx to Mid-Tx on any hypothesized mediator variable (range ts = -.19 to -1.59, range ps = .11 to .85). Similarly, within the UP-C sample, there were no significant changes in hypothesized mediator variables from Pre-Tx to Mid-Tx (range ts = -.32 to 1.45, range ps = .15 to .75). Therefore, mediation analyses were not pursued.

Chapter 4: Discussion

The current study is the first known randomized controlled trial comparing outcomes of a transdiagnostic CBT intervention for youth with emotional disorders to an active treatment condition. We examined the efficacy of the Unified Protocol for the Treatment of Emotional Disorders in Children (UP-C; Ehrenreich-May et al., in press) against an anxiety-specific, group CBT treatment protocol previously shown to be efficacious in treating anxiety disorders (Cool Kids; Hudson et al., 2009). We examined potential condition-related differences in three types of outcomes derived from clinical interviews, parent-report rating scales, and child-report rating scales: 1) diagnostic response and remission rates; 2) parent- and child-rated emotional disorder symptoms, including anxiety and depression symptoms; and 3) parent- and child-rated measures of emotion reactivity and regulation. Condition-related differences in diagnostic response/remission rates and change in anxiety disorder symptoms were not expected, given that our sample was composed of primarily anxious youth and given that we were examining the efficacy of the UP-C against an anxiety-specific protocol with previously demonstrated efficacy.

However, because the UP-C delivers transdiagnostic CBT skills and emphasizes their application to a range of emotional experiences, we expected that UP-C participants would exhibit greater change in depression symptoms over treatment and FU compared to CK participants, as well as greater decreases in dysregulated expression of sadness and anger. In addition to its transdiagnostic delivery of CBT skills commonly found in anxiety-specific treatments, the UP-C also incorporates skills more commonly used to treat other emotional disorder presentations, such as behavior activation, and takes an

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explicitly anti-avoidant stance toward emotional experiences by incorporating present-moment and non-judgmental awareness activities. Therefore, we also expected that UP-C participants would demonstrate greater increases in positive affect and decreases in expressive suppression. Changes in negative affect and cognitive reappraisal were not expected to differ between conditions.

When we examined remission rates for both conditions, including both remission of primary diagnosis and remission of all emotional disorder diagnoses, we did not find any significant differences between conditions in either the sample of participants who completed Post/FU or the ITT sample. Additionally, the remission rate for principal diagnosis in the UP-C condition at Post-Tx (61.90% for assessment completers, 54.20% for ITT) was very similar to that observed in large-scale efficacy trials of CBT for anxiety disorders (e.g., 59.70%; Walkup et al., 2008). For the UP-C condition, the remission rate for all emotional disorder diagnoses (57.10% for assessment completers, 50.00% for ITT) was also similar to rate of remission of all anxiety disorder diagnoses (46.20%) found by Walkup and colleagues (2008). Participants in both conditions appeared to maintain remission status of both the principal anxiety diagnosis and all emotional disorder diagnoses at FU, as evidenced by an equal or even greater percentage of participants achieving diagnostic remission at FU as at Post-Tx across both groups.

When a more global, clinician-rated measure of treatment response across diagnostic categories (CGI-I; Guy et al., 1976) was examined, condition-related differences did emerge. At Post-Tx, there were no significant condition-related differences in the number of participants achieving treatment responder status, as defined by a CGI-I score of "1" or "2." Once again, responses rates were comparable or even

slightly higher (depending on whether the assessment completer or ITT sample was examined) than those observed in previous clinical trials (Walkup et al., 2008). However, 94.10% of UP-C participants who completed the FU assessment achieved responder status, in contrast with 54.40% of CK participants, a difference that was statistically significant. This difference was no longer statistically significant when the ITT sample was considered using the LOCF approach, although this may be due to small sample size given that the percentage of participants achieving responder status was almost 25% greater in the UP-C condition compared to the CK condition (75.00% vs. 52.20%, respectively). Of note, the response rate for the UP-C condition at FU is greater than the 24 week response rate found by Piacentini and colleagues (2014) in a follow-up of the large scale RCT for child anxiety mentioned above, and the response rate for the UP-C assessment completer sample is outside the confidence interval reported by Piacentini and colleagues (CI = 57.08-81.66). Our results indicate that a transdiagnostic intervention like the UP-C may result in continued response to treatment over a longer time period compared to anxiety-specific treatments, perhaps because the transdiagnostic focus of the intervention supports generalization of skills to a diversity of emotions and symptom presentations.

Similar to our results for diagnostic remission, when we examined the slope of parent- and child-rated anxiety symptoms, treatment condition did not predict the rate of change in anxiety symptoms. Anxiety symptoms exhibited a significant, linear decrease over the treatment and follow-up period in both conditions, and the conditions did not differ in the mean level of parent- or child-rated anxiety symptoms at either Post-Tx or FU. These findings, taken together with the lack of condition-related differences in

remission rates of principal diagnosis at Post-Tx and FU, provide preliminary support that the UP-C may be at least as efficacious in targeting anxiety disorder diagnoses as some of the most widely utilized, currently available CBT treatment protocols for anxiety disorders in children.

Regarding parent- and child-rated depressive symptoms, there were no significant condition-related differences in the rate of change in depression symptoms from Pre-Tx to Post-Tx. Across both groups, parent- and child-rated depressive symptoms decreased significantly from Pre-Tx to Post-Tx. These results are consistent with a number of previous trials showing that, even when depressive symptoms are not explicitly targeted by intervention components, youth receiving CBT for an anxiety disorder experience a significant reduction in depressive symptoms over the course of treatment (e.g., Kendall et al., 1997; Manassis et al., 2002; Suveg et al., 2009).

It should be noted, however, that results from the parent- and child-rated LGM differed from one another, and from previous results in the literature, in several important ways. A linear LGM was an adequate fit for the data from all four time points with regard to parent-rated depressive symptoms, indicating that participants in both conditions experienced a significant linear decrease in symptoms over the course of the intervention and follow-up period. Further, the intervention condition significantly predicted Post-Tx depression symptoms, such that UP-C participants experienced significantly lower parent-rated depression symptoms at Post-Tx compared to CK participants; intervention condition no longer predicted mean levels of parent-rated depressive symptoms at FU. For child-rated depressive symptoms, a quadratic LGM best fit the data from the CK condition, and a linear LGM best fit the data from the UP-C condition. The different

functional forms for each condition indicated that, while child-rated depression symptoms decreased significantly from Pre-Tx through FU for UP-C participants, child-rated depressive symptoms decreased from Pre-Tx through Post-Tx for CK participants and then increased from Post-Tx through FU. Results for the CK condition are consistent with some previous trials of anxiety-specific treatments showing that change in depressive symptoms levels out, or even increases, during follow-up periods (e.g., Kendall et al., 1997; Suveg et al., 2009). Our results suggest that a transdiagnostic intervention such as the UP-C may result in more lasting changes in depressive symptoms over time, perhaps as a result of both the incorporation of depression-specific interventions such as behavioral activation and as a result of the presentation of CBT skills within a more general emotion framework.

When we examined change in child- and parent-rated emotion dysregulation during treatment and follow-up, our hypotheses were partially supported. As expected, UP-C participants experienced a faster rate of change in parent-rated sadness dysregulation compared to CK participants, and they also exhibited lower levels of parent-rated sadness dysregulation at both the Post-Tx and FU time points. However, these results were not consistent across reporters, as children in neither condition reported significant changes in sadness dysregulation over time. Similar to the results for sadness dysregulation, but contrary to hypotheses, UP-C participants also experienced a faster rate of change in parent-rated worry dysregulation over treatment and follow-up, although there were no statistically significant condition-related differences at Post-Tx or at FU. As with child-rated sadness dysregulation, children in neither condition reported significant changes in worry dysregulation over time. The lack of significant findings for

any of the child-rated emotion dysregulation scales (including the anger dysregulation scale discussed further below) suggests that parents may be more reliable reporters of dysregulated expression of emotion, a construct that involves observable behavior.

For our final emotion dysregulation variable—anger dysregulation--our hypothesis that parent- and child-rated anger dysregulation would exhibit a faster rate of change in the UP-C condition was not supported. In fact, the slope for anger dysregulation was not significantly different from zero in either condition. In previous trials, many studies of anxiety-specific youth CBT have failed to find a significant treatment effect for either anger dysregulation (e.g., Suveg et al., 2009) or externalizing symptoms/disorders (e.g., Rapee et al., 2013). Despite the fact that the flexibility of the UP-C allows clinicians to apply skills to anger-related targets, and despite UP-C's greater emphasis on parent training and principles of reinforcement as compared to other anxiety-specific protocols, it appears that the UP-C did not outperform the anxiety-specific protocol in the area of anger dysregulation. One possible explanation for these results is that relatively few children in our sample experienced clinically significant difficulties with anger management, as evidenced by the fact that only one child received a clinical diagnosis of oppositional defiant disorder. However, anger dysregulation is not only relevant to childhood externalizing disorders. As recently noted by Cassiello-Robbins and Barlow (2016), disturbance in anger expression and regulation is also common across anxiety and depressive disorders, although the role of anger in the emotional disorders has been long underemphasized. A revised version of the UP-C and UP-A manuals is currently being prepared that incorporates greater emphasis on the application of core skills--including psychoeducation, cognitive reappraisal, present-

moment awareness, and exposure—to frustration and anger-related targets, and that expands the parenting component to include more material on effective behavior management. These revisions appear particularly important, as the current evidence from this trial suggests that UP-C is more effective at addressing sadness regulation and management than it is at addressing anger regulation and management.

Changes in other emotion regulation variables also departed from our original hypotheses in interesting ways, particularly in the case of child-rated expressive suppression and cognitive reappraisal. Expressive suppression, which we expected to decrease across both conditions, did not change significantly over treatment or follow-up in either condition. These results may be due to a floor effect, as the mean level of expressive suppression in our sample across all conditions and time points was rather low (e.g., means ranged from 5.71-7.47, with the highest possible score being 16.00). Additionally, despite strong associations found in previous studies between expressive suppression and psychopathology (e.g., Aldao, Nolen-Hoeksema, & Schweizer, 2010; Gullone & Taffe, 2012), some studies have begun to suggest that the extent to which strategies such as expressive suppression are maladaptive may depend upon contextual factors and upon the combination of strategies used, rather than on the absolute level of any one emotion regulation strategy (e.g., Aldao, 2013). The degree to which suppression is a maladaptive regulation strategy may vary with developmental stage, but the lack of research on age-related changes in the relationship between suppression and psychopathology makes it difficult to evaluate this interpretation. In contrast, levels of cognitive reappraisal, which we expected to increase significantly in both conditions, exhibited no change for participants in the CK condition but exhibited a positive,

significant linear slope over time for UP-C participants. UP-C participants also had significantly higher levels of cognitive reappraisal at both Post-Tx and FU compared to CK participants. This finding was somewhat surprising, given that both UP-C and Cool Kids prominently feature cognitive reappraisal (i.e., detective thinking) as a core treatment component. However, as with other skills delivered in UP-C, cognitive reappraisal is presented in a transdiagnostic framework, such that children are encouraged to apply reappraisal across a variety of emotional experiences. Additionally, the UP-C protocol heavily utilizes interactive activities such as non-emotional games and skits to deliver reappraisal skills, in addition to encouraging children to apply them to their own emotional thoughts. These key differences in the presentation of cognitive reappraisal across the two interventions may have resulted in increased mastery and generalization of cognitive reappraisal in the UP-C condition and may explain the different growth trajectories observed between the two groups.

Finally, in support of our hypotheses, a significant decrease in negative affect was observed across the two groups over treatment and follow-up, and no significant condition-related differences emerged in either the rate of change or in mean levels of negative affect at Post-Tx or FU. However, in contrast to hypotheses, minimal change was observed in positive affect in either condition, and no condition-related differences were observed in the slope for positive affect or mean levels at Post-Tx and FU. These results stand in contrast to results from a waitlist-controlled trial of the adult UP, in which a large treatment effect for positive affect was observed (Farchione et al., 2012). It should be noted that the mean level of positive affect across both conditions in our study was relatively high at Pre-Tx ($M = 35.79$ out of a possible 55.00), indicating that children

reported experiencing at least moderate levels of positive affect on average. It is also possible that mean levels of positive affect remained stable because children in both conditions may have enhanced their emotional awareness and become less defensive about their emotional experiences, resulting in more realistic endorsements of their levels of both positive and negative emotions.

Although we hypothesized that a number of variables might mediate treatment outcome in this study (e.g., emotion dysregulation, expressive suppression, cognitive reappraisal, positive affect, negative affect), mediation analyses were not conducted due to the lack of significant change in these variables from Pre-Tx to Mid-Tx. Our Mid-Tx assessment occurred seven weeks into treatment, coinciding with the delivery of cognitive reappraisal skills in the UP-C and occurring prior to the initiation of interoceptive and situational exposures. Several studies examining trajectories of symptom change and global functioning over the course of CBT for anxiety have revealed that change in symptoms and global severity accelerates following the introduction of cognitive restructuring and exposure skills (e.g., Peris et al., 2015). Therefore, it is possible that we measured our hypothesized mediators too early in treatment, prior to onset or full delivery of skills that have previously been supported as active treatment ingredients. Additionally, although we hypothesized that the skills delivered in the first six sessions of treatment directly target our identified potential mediators, there may be a delay between delivery of a particular skill and change in the domain that skill is hypothesized to target. This latter possibility was supported by Peris and colleagues (2015), who found that the introduction of therapeutic techniques did not alter the trajectory of improvement within the specific domain of anxiety the skill was

intended to address (e.g., the introduction of relaxation training did not result in accelerated change in physiological anxiety; the introduction of exposure did not result in an accelerated rate of change in avoidance, etc.). Despite the methodological challenges in accurately capturing change processes as they occur, studies of the adult UP have supported the idea that emotion reactivity and regulation change over the course of transdiagnostic treatment (Farchione et al., 2012; Sauer-Zavala et al., 2012), and these changes are predictive of changes in symptoms and global impairment. Studies of trajectories of change in emotion reactivity and regulation variables over the course of the UP-C and other transdiagnostic treatments would be helpful for identifying likely windows of change in these variables.

Limitations

This study has several limitations that should be noted, including statistical and other methodological limitations. Concerning statistical limitations, our sample size was small for an RCT and may have limited our power to detect statistically significant group differences, especially given the fact that we expected only small differences on most variables due to our use of an active comparison condition. Despite this limitation, we did find condition-related differences on a number of variables, including most notably depression symptoms, sadness dysregulation, and cognitive reappraisal. Although we had relatively few participants drop out of treatment, and although the majority of participants completed the Post-Tx assessment, we had significant participation drop-off at FU with only about 60% of participants providing data. This amount of missingness may have resulted in biased parameter estimates for the FU time point, even though FIML was used to impute missing data. Future randomized trials of the UP-C and other UP models

should explore additional methods for retaining participants throughout the study period. Finally, both our small sample size and amount of missing data at FU resulted in difficulty conducting multiple group analyses in SEM, whereby separate growth models are fitted to each group and then groups are compared by systematically freeing model parameters and examining resulting change in fit statistics. Such an approach is recommended as the "gold standard" by Muthén and Curran (1997) and would have allowed us to more fully describe the individual growth trajectories in each condition, although our current approach is acceptable and more feasible with smaller sample sizes.

Limitations in our measurements should also be noted. Although we did use ratings from multiple informants in this study as outcome variables (i.e., clinician, parent, and child), the majority of our measures were confined to rating scales. Future studies should undertake a multi-method approach to examining changes in potential higher order factors/mechanisms during treatment, including but not limited to the collection of physiological data, reaction time data, and behavioral observations. Additionally and as noted above, we only measured our hypothesized mediators once during treatment at the seven-week time point, and this decision limited our ability to examine mediation because very little change occurred in our hypothesized mediator variables during the first seven weeks of treatment. Future studies should examine trajectories of change in hypothesized mediators over the course of the UP-C and other transdiagnostic treatments in order to identify more precisely when these variables exhibit change. Beyond measurement limitations, an additional methodological limitation involves possible experimenter bias. The study team responsible for the development of the UP-C provided training and clinical supervision in both the UP-C and CK manuals, and it is therefore

possible that UP-C clinicians may have received superior supervision and training. This may have resulted in greater clinician competency for the UP-C condition (although competency was not rated for this study). It is important to note, however, that the primary supervisor for both groups has considerable expertise in the treatment of anxiety disorders in youth, and the study team for this investigation made efforts to ensure competency in providing training and supervision in the comparison treatment by receiving on-site training from the manual's developers. It would have been ideal had a separate study team with clinical expertise in the CK protocol provided supervision and training to CK clinicians, but our limited resources for this trial made this unfeasible.

Finally, diagnostic homogeneity is also a limitation in our sample, particularly with regard to principal diagnoses of participants. Few participants (8.51%) received a principal or co-principal diagnosis of a depressive disorder at Pre-Tx, somewhat limiting our ability to examine the efficacy of the UP-C for depression relative to an anxiety-specific treatment protocol. Our low percentage of participants is understandable, given that we recruited children between the ages of 6 and 13 for this study, when rates of depressive disorders are still relatively uncommon. Epidemiological studies have established that prevalence rates of depressive disorders do not increase substantially until adolescence, with the most recent studies establishing a 2.7% past year prevalence rate for children ages 8-15 years old and a 7.5% past year prevalence rate among 13-18 year olds (Avenevoli, Swendsen, He, Burstein, & Merikangas, 2015; Merikangas et al., 2010). Although we did actively recruit depressed children for our study, future trials of the UP-C should explore additional methods for increasing the number of depressed children receiving treatment. Finally, although the UP-C targets difficulties with anger

regulation and frustration tolerance, only one child in our sample received a clinical diagnosis of oppositional defiant disorder, making it difficult to draw conclusions about the efficacy of the UP-C in treating children with significant anger dysregulation or frustration intolerance. As mentioned previously, revisions of the UP-C are underway that will permit greater clinician flexibility in applying core skills to anger regulation targets, and we are actively recruiting more children with ODD and frustration intolerance in ongoing naturalistic trials of the UP-C. Future studies with larger sample sizes should also compare the efficacy of the UP-C in children with and without externalizing problems, including not only ODD but also ADHD.

Conclusion

This study built upon previous open trial results supporting the efficacy of the UP-C (Bilek & Ehrenreich-May, 2012) by conducting a RCT comparing the UP-C against an anxiety-specific treatment protocol with previously supported efficacy. Results demonstrated that UP-C participants did not differ from participants in the active control condition with respect to remission of primary diagnosis or all emotional disorder diagnoses at Post-Tx or FU, nor did they differ with respect to rate of change in anxiety symptoms or mean levels of anxiety at Post-Tx or FU. Unexpectedly, UP-C participants were more likely than Cool Kids participants to achieve responder status at FU, suggesting that receiving a transdiagnostic intervention may lead to more lasting benefits in terms of overall emotional disorder-related impairment. Condition-related differences were also observed in both child- and parent-rated depressive symptoms, and the different functional form of the growth curves for child-rated depression among the two conditions suggests that a transdiagnostic intervention like the UP-C may also lead to

more lasting changes in depression symptoms. The UP-C condition also outperformed the active control condition on a variety of other measures of hypothesized risk and maintenance factors across emotional disorders, including rate of change in parent-rated sadness dysregulation and levels of parent-rated sadness dysregulation at Post-Tx and FU, rate of change in parent-rated worry dysregulation, and rate of change in child-rated cognitive reappraisal and mean levels of cognitive reappraisal at Post-Tx and FU.

Our results suggest that the UP-C is an efficacious protocol for treating children with emotional disorders, adding to the growing body of literature supporting the use of transdiagnostic interventions with individual of varying ages and varying diagnostic presentations. Our results also suggest that there may be a rationale for using a transdiagnostic versus an anxiety-specific intervention even in younger children with primary anxiety disorders, as observed gains in symptoms of common comorbid disorders and in emotion regulation may help to prevent the later development of related disorders. The possible preventative effects of transdiagnostic interventions should be tested empirically using longitudinal designs with long-term follow-ups.

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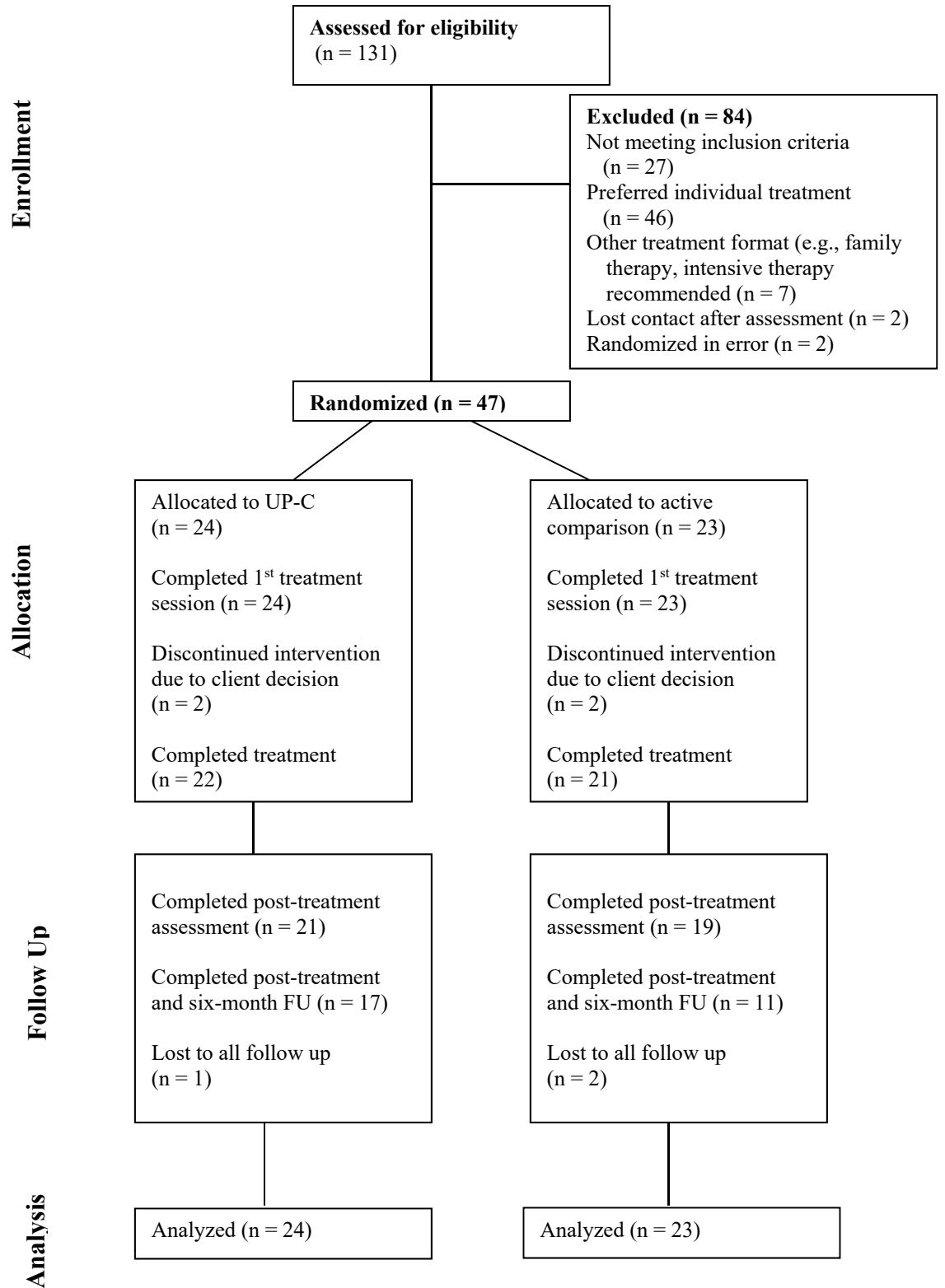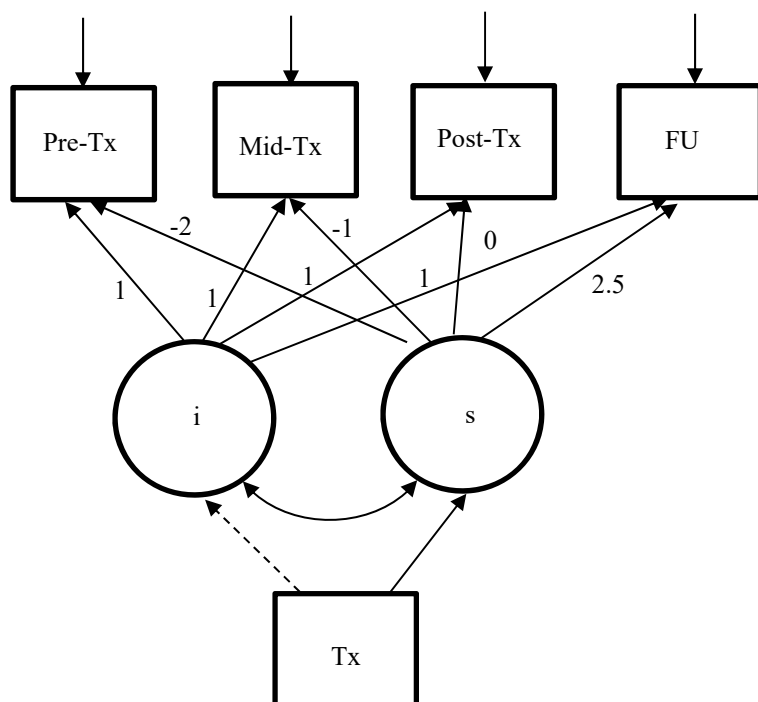
Figures

Figure 1. Consort diagram.

Figure 2. Basic latent growth curve model with treatment condition as a time-invariant covariate.
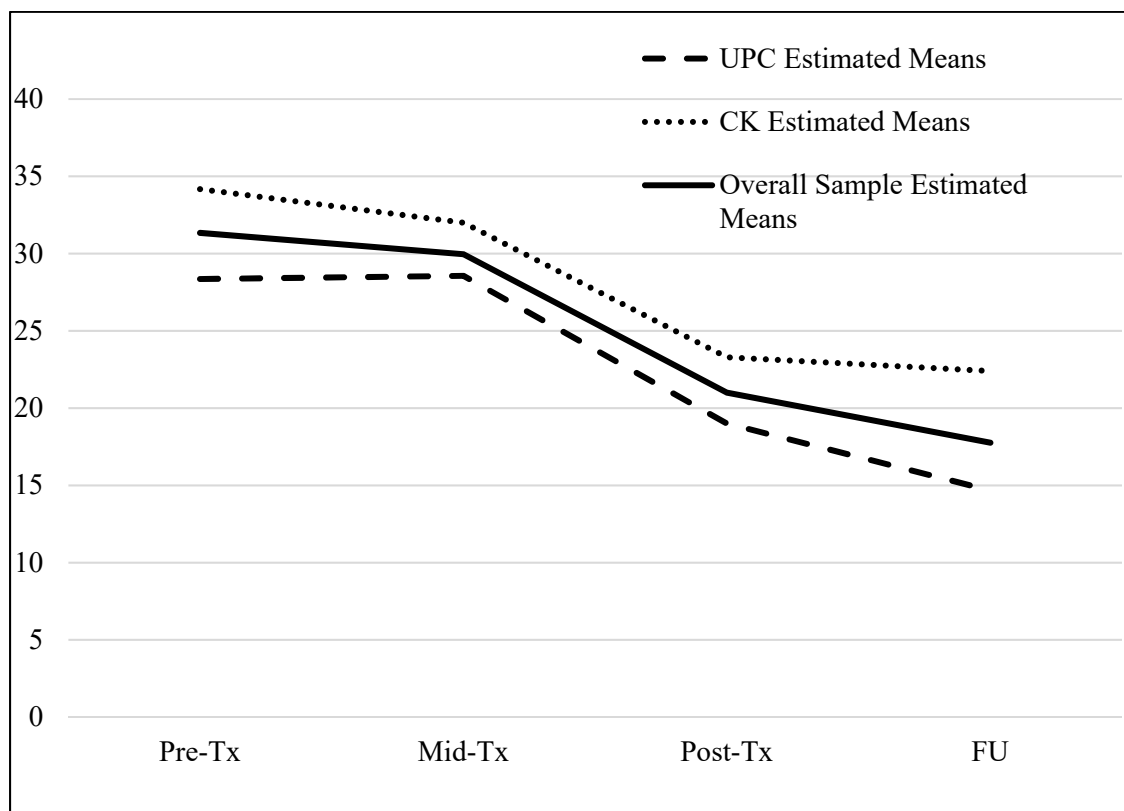
Figure 3. Plot of estimated means of parent-rated SCARED Total Anxiety scores for overall sample, UP-C condition, and Cool Kids condition.
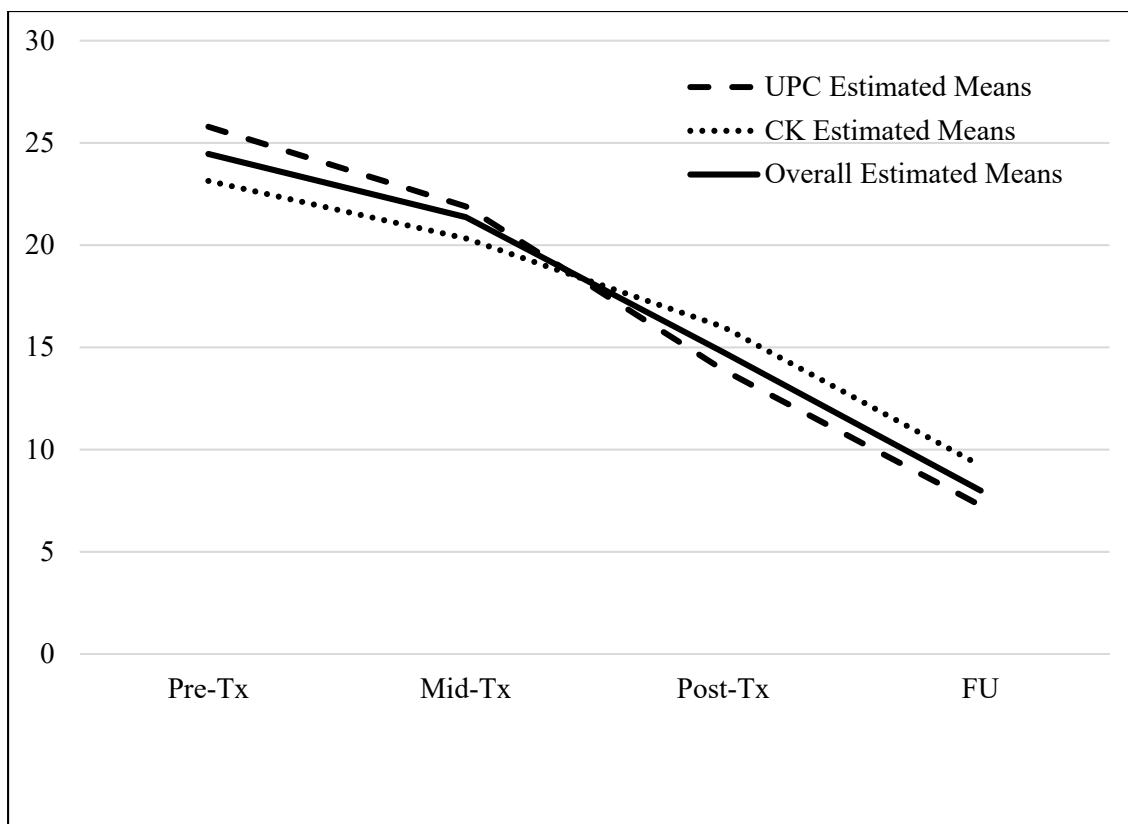
Figure 4. Plot of estimated means of child-rated SCARED Total Anxiety scores for overall sample, UP-C condition, and Cool Kids condition.
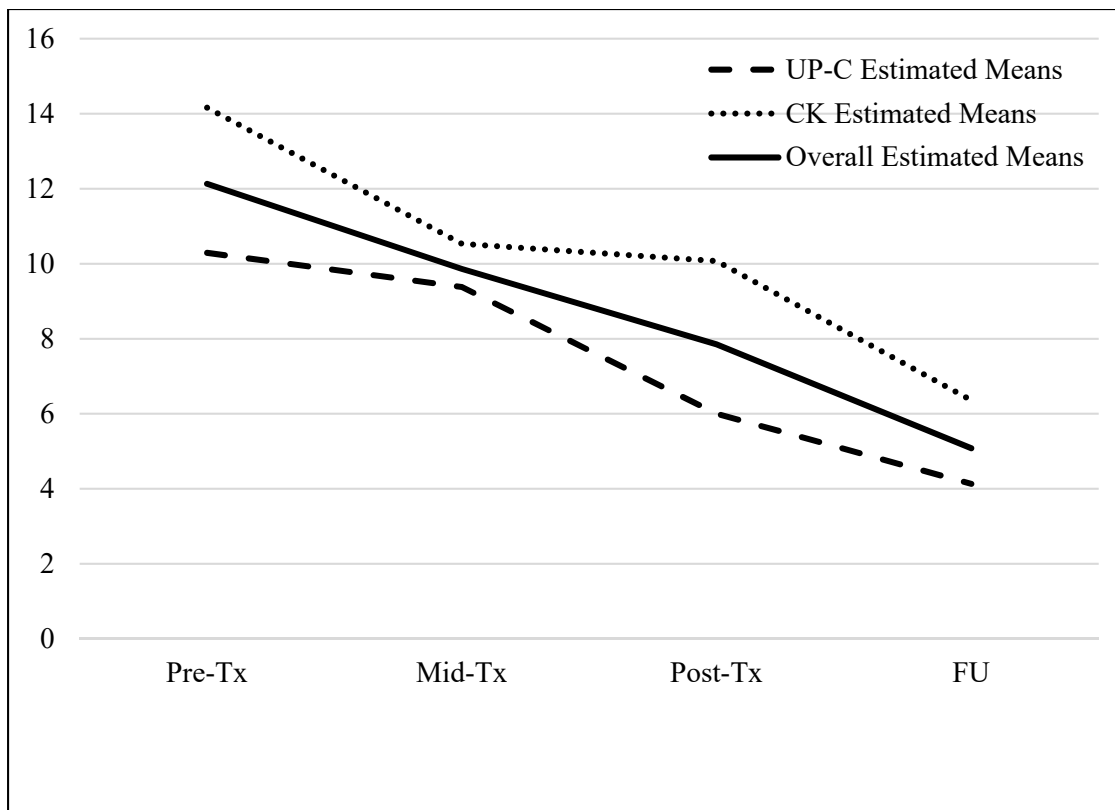
Figure 5. Plot of estimated means of parent-rated CDI Total Depression scores for overall sample, UP-C condition, and Cool Kids condition.

Figure 6. Plot of estimated means of child-rated CDI Total Depression scores for overall sample, UP-C condition, and Cool Kids condition.

Figure 7. Plot of estimated means of parent-rated CEMS Sadness Dysregulation scores for overall sample, UP-C condition, and Cool Kids condition.
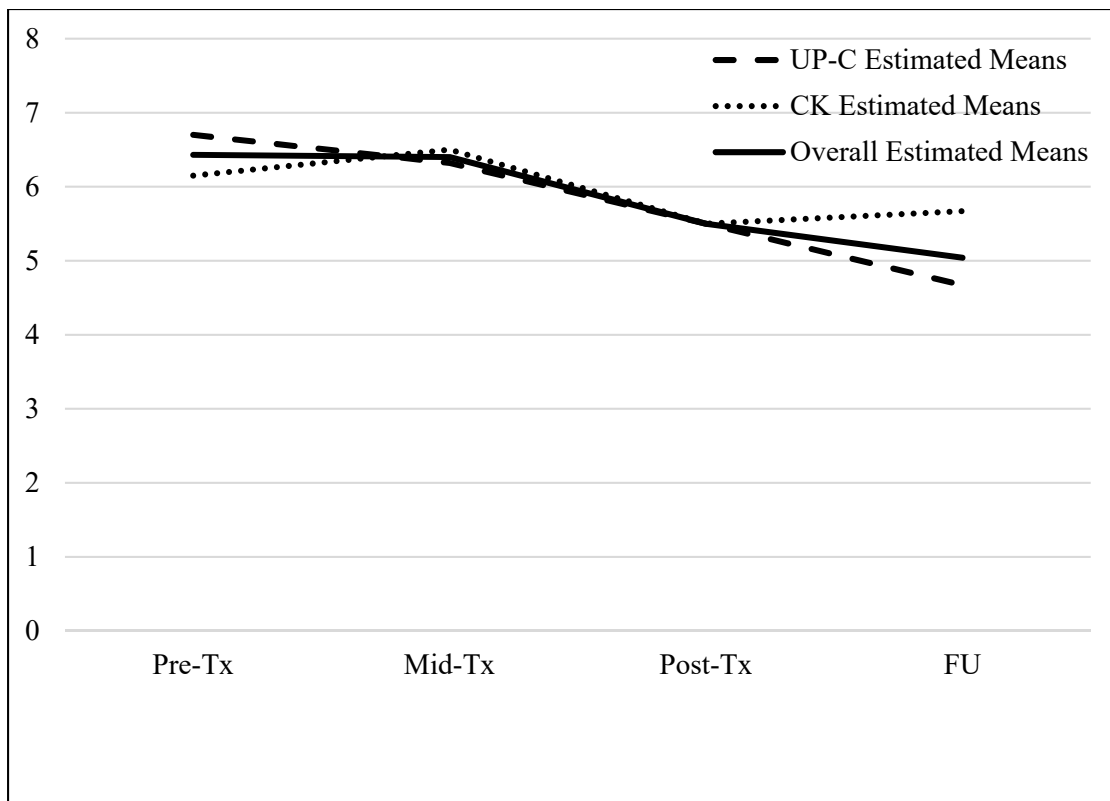
Figure 8. Plot of estimated means of parent-rated CEMS Worry Dysregulation scores for overall sample, UP-C condition, and Cool Kids condition.

Figure 9. Plot of estimated means of child-rated ERQ-CA Reappraisal scores for overall sample, UP-C condition, and Cool Kids condition.
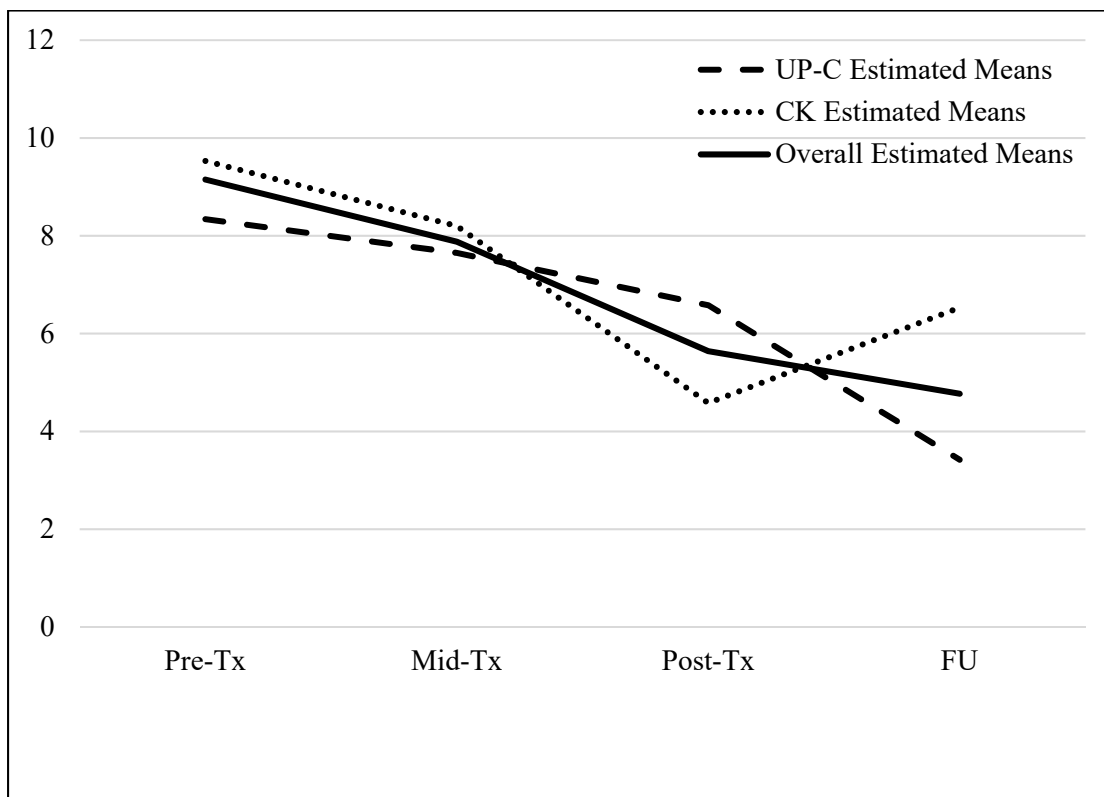
Figure 10. Plot of estimated means of child-rated ERQ-CA Suppression scores for overall sample, UP-C condition, and Cool Kids condition.
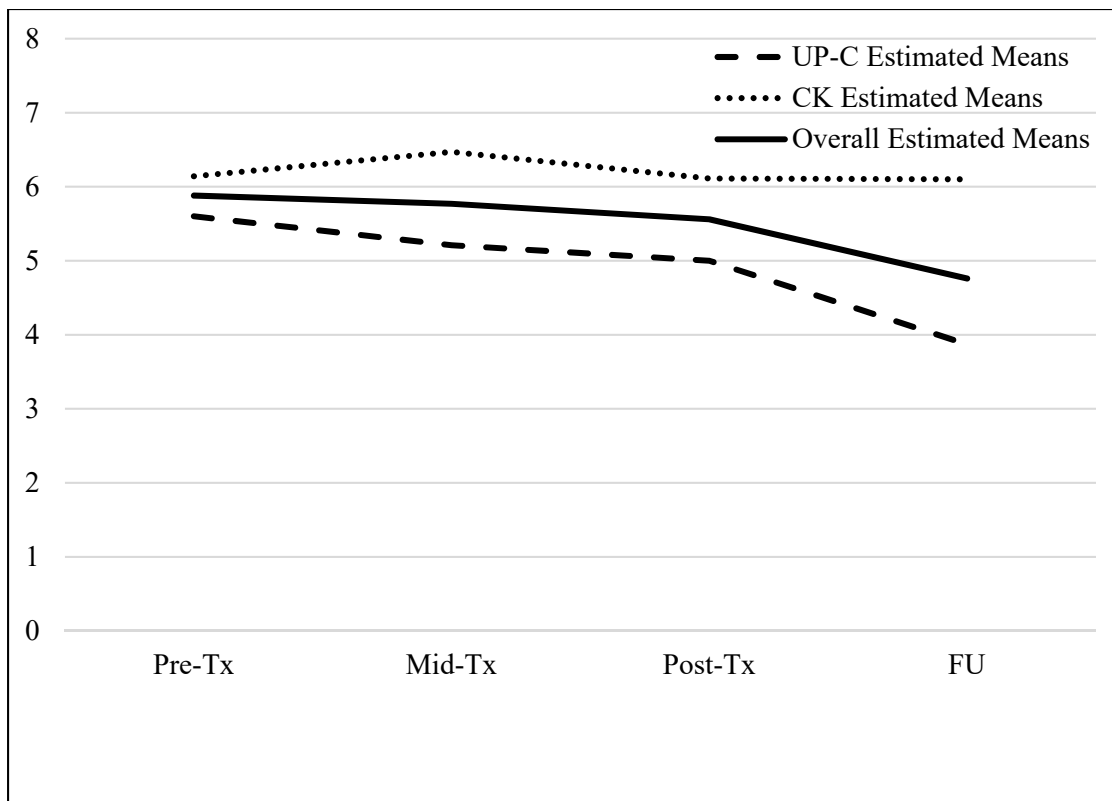
Figure 11. Plot of estimated means of child-rated PANAS Positive Affect scores for overall sample, UP-C condition, and Cool Kids condition.
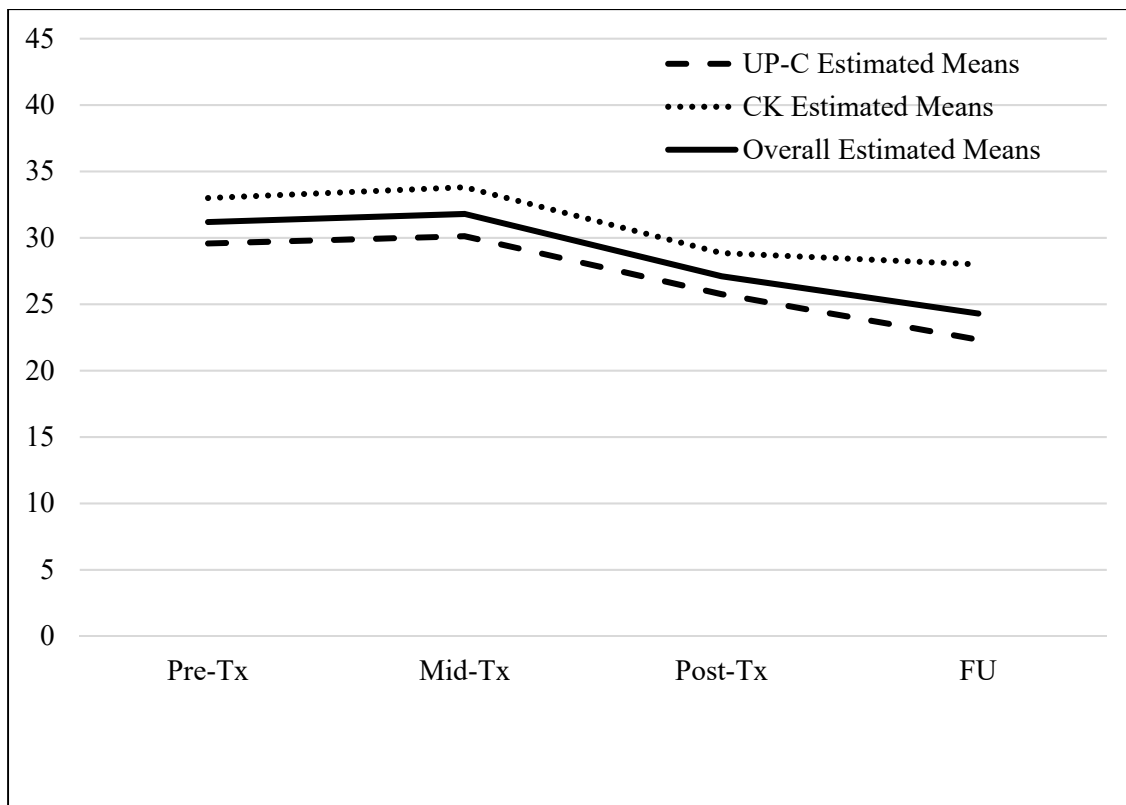
Figure 12. Plot of estimated means of child-rated PANAS Negative Affect scores for overall sample, UP-C condition, and Cool Kids condition.
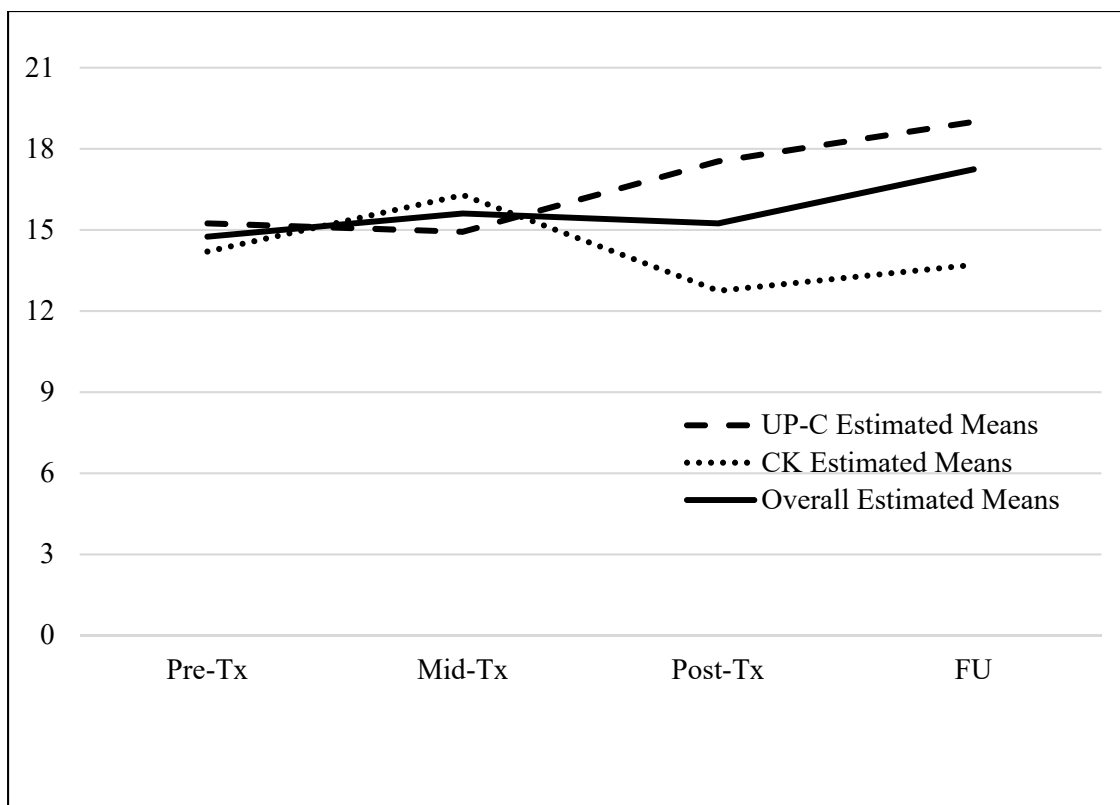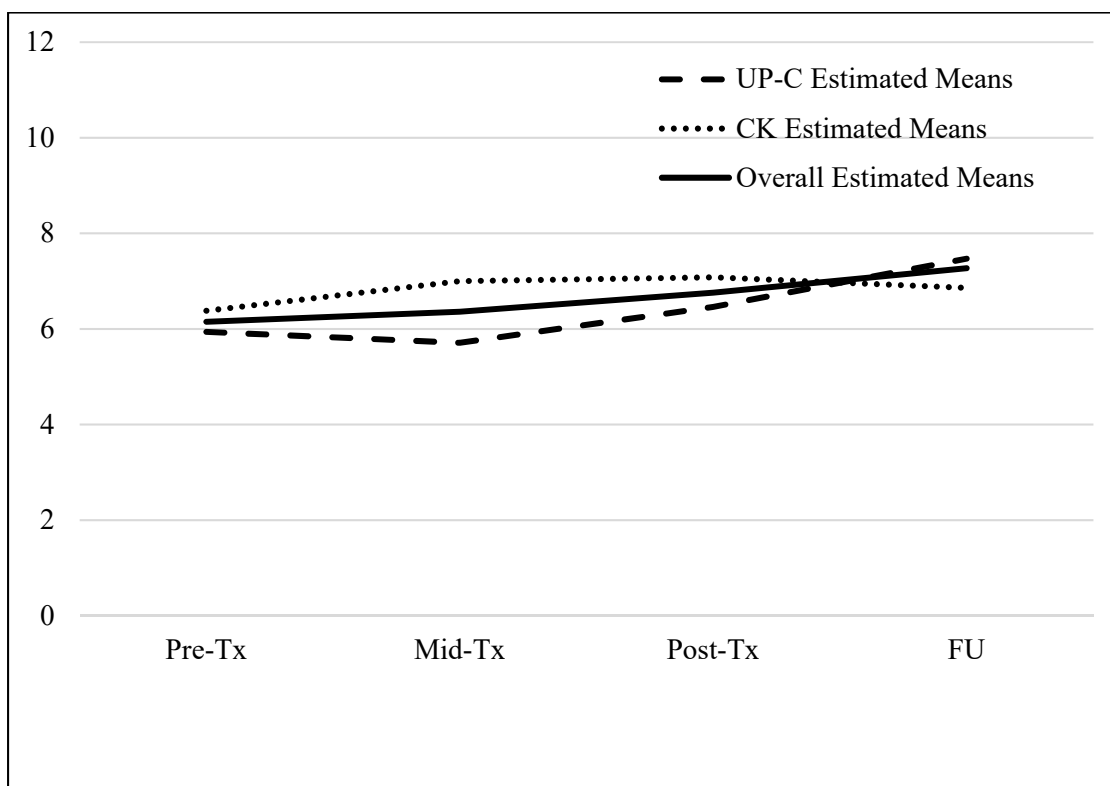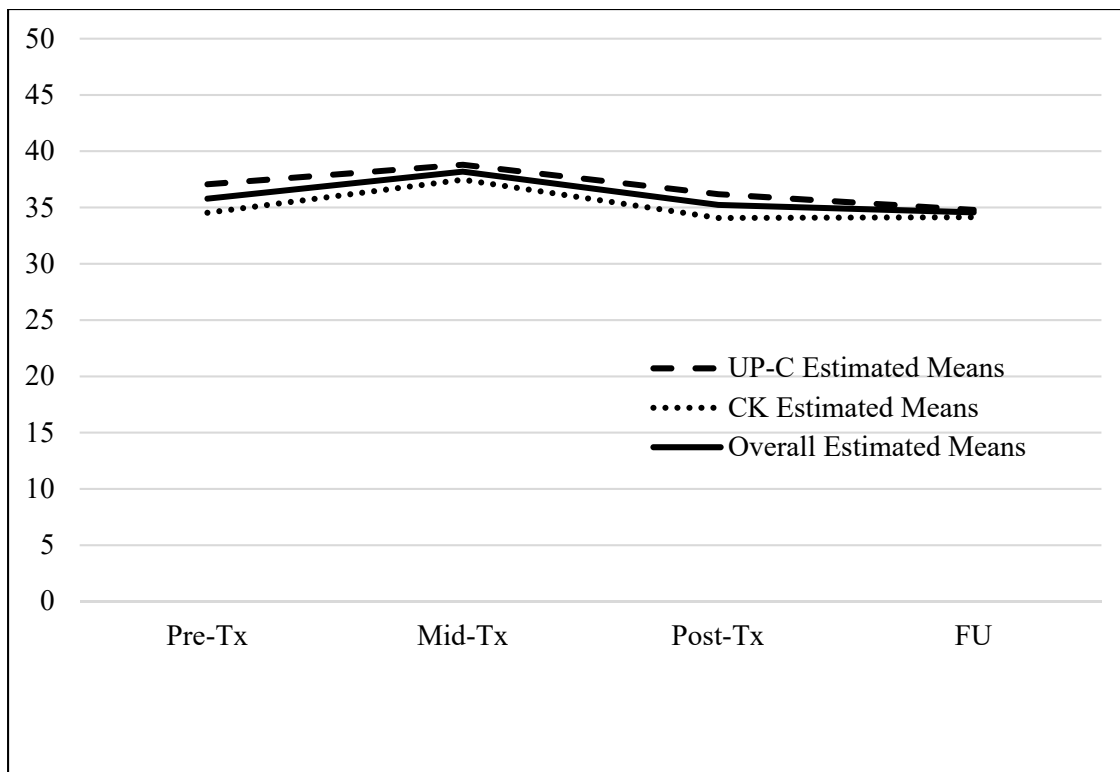
Tables

Table 1
Structure of the UP-C and Common Risk and Maintaining Factors Targeted in Each CLUES Skill Section

Session #	CLUES Skill	Primary Interventions	Risk/Maintenance Factors Targeted by Interventions
Session 1			
Session 2	C Skill (Consider How I Feel)	Emotion Identification Sensational Awareness Behavioral Activation Present-Moment Awareness	Poor Emotional Awareness Emotion Suppression Low Positive Affect High Negative Affect Emotion Dysregulation
Session 3			
Session 4			
Session 5			
Session 6	L Skill (Look At My Thoughts)	Linking Thoughts to Feelings Identifying Cognitive Distortions	Repetitive Negative Thinking Cognitive Inflexibility
Session 7	U Skill (Use Detective Thinking)	Cognitive Reappraisal Problem-Solving	Repetitive Negative Thinking Cognitive Inflexibility
Session 8			
Session 9			
Session 10	E Skill (Experience My Fears and Feelings)	Interoceptive Exposures Situational Exposures	Avoidance High Negative Affect Emotion Dysregulation
Session 11			
Session 12			
Session 13			
Session 14			
Session 15	S Skill (Stay Healthy and Happy)	Relapse Prevention	-------------------------------

Table 2

Principal, Comorbid, and Total Diagnoses for Randomized Participants

	Principal Diagnosis (%)	Comorbid Diagnosis (%)	Total (%)
Generalized Anxiety Disorder (GAD)	22 (46.81%)	11 (18.97%)	33 (70.21%)
Social Anxiety Disorder (SOC)	10 (21.28%)	11 (23.40%)	21 (44.68%)
Separation Anxiety Disorder (SEP)	6 (12.77%)	3 (6.38%)	9 (19.15%)
Anxiety Disorder Not Otherwise Specified (ANX-NOS)	5 (10.64%)	0	5 (10.64%)
Obsessive Compulsive Disorder	3 (6.38%)	3 (6.38%)	6 (12.77%)
Specific Phobia (SP)	1 (2.13%)	6 (12.77%)	7 (14.89%)
Selective Mutism	1 (2.13%)	1 (2.13%)	2 (4.26%)
Panic Disorder with Agoraphobia	1 (2.13%)	0	1 (2.13%)
Major Depressive Disorder	0	1 (2.13%)	1 (2.13%)
Depressive Disorder Not Otherwise Specified	3 (6.38%)	0	3 (6.38%)
Attention Deficit/Hyperactivity Disorder (ADHD)	3 (6.38%)	3 (6.38%)	6 (12.77%)
Oppositional Defiant Disorder (ODD)	0	1 (2.13%)	1 (2.13%)
Tourette's Disorder	1 (2.13%)	0	1 (2.13%)

Note. Principal diagnoses do not total 47 because some children received more than one principal diagnosis.

Table 3
Week-by-Week and Session-by-Session Comparison of Treatment Components Across Conditions

Week	UP-C		Cool Kids	
1	Session 1	Introduction to treatment Emotion identification	Session 1	Introduction to treatment Emotion identification (anxiety) Linking thoughts and feelings
2	Session 2	Emotion identification Three-component model of emotions	Session 2	Self-talk Detective thinking (i.e., reappraisal)
3	Session 3	Sensational awareness	Session 3	Additional detective thinking practice
4	Session 4	Behavioral experiments, including behavioral activation	Session 4	Introduction to graduated exposure
5	Session 5	Present-moment awareness Generalized emotion exposure	Session 5	Individual situational exposure
6	Session 6	Linking thoughts to sensations Thinking traps (i.e., distortions)	---	At-home exposure practice
7	Session 7	Detective thinking (i.e., reappraisal)	Session 6	Revising exposure stepladders Worry-surfing (i.e., mindfulness)
8	Session 8	Problem-solving (including in an interpersonal context)	---	At-home exposure practice
9	Session 9	Introduction to exposure Sensational exposure	Session 7	Problem-solving Social skills & assertiveness
10	Session 10	Group situational exposure	---	At-home exposure practice
11	Session 11	Individual situational exposure	Session 8	Revising stepladders Outsmarting bullies Individual situational exposure
12	Session 12	Individual situational exposure	---	At-home exposure practice
13	Session 13	Individual situational exposure	Session 9	Revising stepladders Individual situational exposures
14	Session 14	Individual situational exposure	---	At-home exposure practice
15	Session 15	Skills review Relapse prevention	Session 10	Skills review Relapse prevention

Table 4
Estimated Means of Primary Study Variables at Pre-Treatment, Mid-Treatment, Post-Treatment, and Follow-Up

Scale	UP-C				Cool Kids			
	Pre-Tx	Mid-Tx	Post-Tx	Fu	Pre-Tx	Mid-Tx	Post-Tx	FU
Clinician Variables								
Principal Dx CSR	5.21	--	3.19	2.82	5.34	--	3.16	3.10
Child Variables								
CDI Total	8.32	7.50	6.72	3.79	10.24	8.46	4.33	6.50
SCARED Total	25.79	21.89	13.90	7.27	23.14	20.33	16.00	9.22
CEMS Anger Dysregulation	3.95	4.10	4.05	5.07	4.86	5.44	4.47	5.29
CEMS Sadness Dysregulation	5.14	5.00	4.47	5.00	5.21	5.00	4.41	4.25
CEMS Worry Dysregulation	4.76	4.62	4.42	4.44	4.78	4.77	4.81	4.88
PANAS Positive Affect	37.06	38.80	36.18	34.79	34.53	37.47	34.07	34.14
PANAS Negative Affect	29.58	30.12	25.76	22.33	33.00	33.81	28.85	28.00
ERQCA (Reappraisal)	15.24	14.93	17.54	19.00	14.20	16.29	12.75	13.71
ERQCA (Suppression)	5.94	5.71	6.46	7.47	6.38	7.00	7.08	6.86
Parent Variables								
CDI Total	10.29	9.38	6.00	4.13	14.16	10.53	10.07	6.36
SCARED Total	28.35	28.56	19.00	14.67	34.17	32.00	23.29	22.40
CEMS Anger Dysregulation	5.24	4.75	4.89	5.00	5.59	5.50	5.74	5.56
CEMS Sadness Dysregulation	5.60	5.21	5.00	3.87	6.14	6.47	6.11	6.10
CEMS Worry Dysregulation	6.70	6.32	5.50	4.67	6.15	6.50	5.50	5.67

Note. Tx = Treatment; Dx = Diagnosis; Emotional Disorder = anxiety, depressive, or oc-spectrum disorder, CDI = *Children's Depression Inventory*, SCARED = *Screen for Childhood Anxiety and Related Disorders*; CEMS = *Children's Emotion Management Scale*; PANAS = *Positive and Negative Affect Scale*; ERQCA = *Emotion Regulation Questionnaire for Children*.

Table 5

Observed Bivariate Correlations Among Study Variables at Pre-Treatment

	dep c	anx c	reapp	supp	PA	NA	ang dys c	sad dys c	wor dys c	dep p	anx p	ang dys c	sad dys c	wor dys c
dep c	1													
anx c	.664**	1												
reapp	.056	.210	1											
supp	.107	-.048	.192	1										
PA	-.149	.118	-.165	-.116	1									
NA	.702**	.785**	-.109	-.039	-.010	1								
ang dys c	.532**	.551**	.066	.038	-.166	.630**	1							
sad dys c	.410*	.706**	.295	.230	.231	.519**	.560**	1						
wor dys c	.412*	.527**	.050	-.116	-.124	.653**	.535**	.574**	1					
dep p	.288	-.150	-.129	-.114	.214	.147	.052	.065	.000	1				
anx p	.322	.286	.066	-.154	.091	.296*	.083	.152	.272	.581**	1			
ang dys p	-.061	-.239	-.081	.248	-.138	.031	.203	-.085	-.112	.183	-.250	1		
sad dys p	.298	.276	-.038	-.054	-.071	.237	.118	.166	.176	.309*	.243	.118	1	
wor dys p	.423*	.520**	.033	-.222	-.267	.405*	.459**	.409*	.405	.264	.295	.459**	.409*	.405*

Note. c = child-rated measure; p = parent-rated measure; dep = CDI Total Depression; anx = SCARED Total Anxiety; reapp = ERQ-CA Reappraisal; supp = ERQ-CA Suppression; PA = Positive Affect; NA = Negative Affect; ang dys = CEMS Anger Dysregulation; sad dys = CEMS Sadness Dysregulation; wor dys = CEMS Worry Dysregulation. *$p<.05$. **$p<.01$.

Table 6
LGM Results for Symptom Outcome Measures and Measures of Higher Order Factors

	Combined Linear Slope Mean	Intervention Effect on Slope	Intervention Effect on Post Intercept	Intervention Effect on FU Intercept
SCARED Parent	-5.02 (.91)***	-.85 (1.55), ns	-4.33 (3.49), ns	-3.92 (3.60), ns
SCARED Child	-3.93 (.62)***	-.40 (1.25), ns	.64 (3.61), ns	-.35 (3.23), ns
CDI Parent	-1.30 (.21), ns	.39 (.42), ns	-2.55)1.18)*	---------
CDI Child	---------	.79 (.92), ns	-.42 (1.16), ns	---------
CEMS A Dys Parent	-.08 (.05), ns	---------	---------	---------
CEMS S Dys Parent	-.22 (.05)***	-.11 (.11)***	-.94 (.43)*	-1.23 (.55)*
CEMS W Dys Parent	-.31 (.08)***	-.23 (.16)***	-.03 (.17), ns	-.66 (.74), ns
CEMS A Dys Child	.03 (.10), ns	---------	---------	---------
CEMS S Dys Child	-.16 (.10), ns	---------	---------	---------
CEMS W Dys Child	-.06 (.08), ns	---------	---------	---------
ERQ Reappraisal	.36 (.31), ns	1.27 (.58)**	.33 (.18), p=.06	5.83 (2.24)***
ERQ Suppression	.09 (.13), ns	---------	---------	---------
PANAS Positive Affect	-.52 (.46), ns	---------	---------	---------
PANAS Negative Affect	-1.41**	.054 (1.19), ns	-2.67 (2.77), ns	-2.54 (3.69), ns

Note. A = Anger; W = Worry; S = Sadness; Dys = Dysregulation.
**p<.05.
**p<.01.
***p<.001.