# Comorbid ADHD: Implications for Cognitive-Behavioral Therapy of Youth with a Specific Phobia

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ABSTRACT

*Objective*: Although findings have been mixed, accumulating evidence suggests that co-occurring attention-deficit/hyperactivity disorder (ADHD) diagnoses and symptoms negatively predict cognitive-behavioral therapy (CBT) outcomes for anxious youth. The current study extends past research by examining the association of not only ADHD but also other features of ADHD with treatment outcomes of youth who received an intensive CBT for a specific phobia. *Method*: 135 youth (ages 6-15; 52.2% female; 88.2% white) were randomized to either an individual or parent-augmented intensive CBT targeting a specific phobia. Latent growth curve models were used to explore the association of ADHD symptoms, effortful control, sluggish cognitive tempo, maternal depression and the two treatment conditions (i.e., individual versus parent-augmented) with pre-treatment severity of the specific phobia and the trajectory of change in the severity of the specific phobia from pre-treatment to the 6-month follow up after the intervention. *Results*: As expected, higher levels of ADHD symptoms were associated with lower levels of effortful control and increased maternal depression at pre-treatment. Contrary to expectations, ADHD symptoms and its associated difficulties were not significantly associated with treatment outcomes. *Conclusion*: Overall, the findings lend support to the generalizability of intensive CBT for a specific phobia to youth with comorbid ADHD and associated difficulties. Implications and limitations of the study are discussed.

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# I. Introduction

Several evidence-based treatments have been identified for a variety of disorders in children and adolescents (Ollendick & King, 2012; Pelham & Fabiano, 2008). However, a significant number of youth do not respond to these treatments (Ollendick & Sander, 2012). Given high rates of comorbidity (Angold, Costello, & Erkanli, 1999; Costello, Mustillo, Erkanli, Keeler, & Angold, 2003, Merikangas et al., 2010), it is possible that some youth do not respond because of co-occurring disorders that limit the effectiveness of these interventions. Indeed, accumulating evidence suggests that co-occurring attention-deficit/hyperactivity (ADHD) negatively predicts outcomes in children and adolescents treated for a variety of anxiety disorders (Halldorsdottir & Ollendick, 2013); however, it is poorly understood at present *how* ADHD impedes successful treatment outcomes in anxious youth. In an attempt to address this gap, the current study expands on the literature by exploring various facets of ADHD that may shed light on how the disorder may hinder optimal treatment gains in children and adolescents with a specific phobia, a common anxiety disorder, but one that has not yet been examined in terms of comorbidity with ADHD. Toward that end, various difficulties associated with ADHD in addition to the diagnosis itself will be examined as predictors and moderators of treatment outcome. Predictors inform what baseline characteristics or factors facilitate or hinder successful treatment outcomes across treatment conditions, whereas moderators indicate which specific treatment works best for whom (Kraemer, Wilson, Fairburn, & Agras, 2002).

## Attention-Deficit/Hyperactivity Disorder (ADHD)

ADHD is among the most common psychiatric disorders in youth (Merikangas et al., 2010). There are three different subtypes of ADHD: ADHD predominately inattentive presentation (ADHD-I), ADHD predominately hyperactive/impulsive presentation (ADHD-H),

and ADHD combined presentation (ADHD-C, APA, 2000). ADHD-I is characterized by developmentally inappropriate inattention, whereas ADHD-H is characterized by excessive hyperactivity/impulsivity. Children with ADHD-C present with a combination of the inattentive and hyperactive/impulsive symptoms seen in the other two subtypes. Lifetime prevalence rates of ADHD subtypes range from 8.5-19% (Kessler, Berglund, Demler, Jin, & Walters, 2005; Merikangas et al., 2010). In addition to the core symptoms, children with ADHD are known to have deficits with executive functioning, emotion regulation, and social skills (Barkley, 1997; Hinshaw, 2003; Nigg, 2006).

Treatments building on operant strategies (i.e., behavior modification techniques) have been identified as evidence based treatments for ADHD (Pelham & Fabiano, 2008). These interventions involve teaching caregivers and/or teachers to manage the child's behavior systematically through attending to and reinforcing appropriate behaviors and ignoring or punishing inappropriate behaviors (MTA Cooperative Group, 1999). Behavioral interventions have been shown to lead to significant reductions in core ADHD symptoms (MTA Cooperative Group, 1999); although the treatment gains are frequently transitory and not maintained over time (Molina et al., 2009). In regard to barriers to long-term outcomes, parental psychopathology, including depression, has been found to interfere with achieving an optimal treatment response (Chronis, Chacko, Fabiano, Wymbs, & Pelham, 2004).

## Comorbid ADHD and Anxiety

Rates of comorbid ADHD and anxiety disorders (AD) are greater than would be expected by chance (Angold et al., 1999), ranging from 11-25% (Biederman, Newcorn, & Sprich, 1991; Jensen et al., 2001, Kendall et al., 2010; Larson, Russ, Kahn, & Halfon, 2011). Children with dual diagnoses of ADHD and AD evince greater impairment than youth with ADHD or AD

alone (Bowen, Chavira, Bailey, Stein, & Stein, 2008; Jensen et al., 2001). Specifically, when compared to youth with either ADHD or AD alone, youth with comorbid ADHD and AD present with greater symptom severity, more attentional problems, more school fears, and decreased social competence (Bowen et al., 2008). Furthermore, youth with comorbid ADHD and AD are more likely to display symptoms of sluggish cognitive tempo (i.e., daydreaming, being slow to respond, and easily confused) and poor response inhibition (i.e., inhibiting an automatic response for a more task appropriate response) when compared with youth with ADHD alone (Lee, Burns, Snell, & McBurnett, 2013; Pliszka, 1989; 1992; Schatz & Rostain, 2006; Skirbekk, Hansen, Oerbeck, & Kristensen, 2011). Consequently, identifying and, if needed, devising effective treatments for this population is of clinical importance (Jarrett & Ollendick, 2008).

Comorbidity Treatment Outcome Studies

Cognitive-behavioral treatments (CBTs) have been shown to be effective in treating youth with various ADs. CBT traditionally includes components of psychoeducation, teaching the child to identify and challenge dysfunctional cognitions, and exposure to the anxiety-provoking stimuli along with graduated exposure inside and outside the therapy session. Using CBT principles, the One-Session Treatment (OST; Öst, 1989; 1997) – the focus of the present study - has been shown to be effective in treating youth with SPs (Ollendick et al., 2009). The intervention involves psychoeducaton, intensive graduated *in vivo* exposures, cognitive challenges, participant modeling and reinforced practice. During the intervention, change is thought to be facilitated through habituation, the elicitation and challenging of dysfunctional cognitions, skill acquisition, and reduction in behavioral avoidance (Davis & Ollendick, 2005).

Although EBTs have been identified for ADHD and ADs, treating youth with comorbid ADHD and anxiety may pose difficulties in need of further consideration. As noted, evidence is

beginning to accumulate that anxious youth with co-occurring ADHD diagnoses and symptoms, when examined independent of other disruptive behavior disorders, respond less well to CBT treatments (Garcia et al., 2010; Halldorsdottir et al., 2013; Storch et al., 2008). Still, some studies have found comparable outcomes between anxious youth with and without ADHD (Manassis et al., 2002; Southam-Gerow, Kendall, & Weersing, 2001).

Among these studies, Storch et al. (2008) examined the influence of comorbid ADHD on treatment outcomes in 96 children (ages 7 to 19; 45% females) with a primary diagnosis of obsessive-compulsive disorder (OCD) after receiving family-based CBT with exposure and response prevention (ERP). Approximately 26% of the participants had comorbid ADHD ($n$ = 24). Response rates were significantly lower and remission rates were marginally lower ($p$ = .09) in participants with comorbid ADHD than those without this comorbid disorder. In explaining their findings, Storch and colleagues indicated that youth with comorbid ADHD may have been unable to attend long enough to habituate to anxiety-provoking stimuli and engage in deliberate cognitive restructuring during the sessions (Storch et al., 2008). They also postulated that the comorbid children may have had difficulties with planning and implementing exposures or other therapeutic tasks outside of the treatment session. To accommodate for these difficulties, the authors suggested teaching parents specific behavior modification techniques prior to pursuing CBT for OCD and implementing these adjunctive procedures throughout treatment.

Similar findings were shown in the multisite Pediatric OCD Treatment Study (POTS, 2004). In the POTS trial, treatment outcomes were evaluated for 112 youth (ages 7 to 17; 50% females) with a primary diagnosis of OCD who were randomly assigned to receive individual CBT, medication, the combination of the individual CBT and medication regimen, or a pill placebo. Approximately 15% of the sample ($n$ = 17) met diagnostic criteria for ADHD (Garcia et

al., 2010). Across treatment conditions, children with high levels of ADHD symptoms and associated behavioral problems had lower remission rates than their non-ADHD counterparts. In their concluding remarks, commensurate with Storch et al. (2008), the authors noted that additional treatment components may be needed for treating OCD in youth with comorbid ADHD.

In a recent re-analysis of data from the Child/Adolescent Multimodal Study (CAMS; ages 7-17; 76% Caucasian; 52% female), a co-occurring ADHD diagnosis ($n$ = 12) was found to predict poor immediate treatment outcomes in youth who received individual CBT for generalized anxiety disorder, separation anxiety disorder or social anxiety disorder (Halldorsdottir et al., 2013). In addition, at both the diagnostic and dimensional level, presence of attentional and/or overactivity difficulties was negatively associated with successful acute remission rates and maintenance of treatment gains at 6-month follow up. Based on these findings, the authors echoed the need to augment treatment of anxiety in comorbid youth to accommodate for difficulties associated with ADHD.

These findings notwithstanding, two other studies failed to find negative outcomes associated with ADHD in the treatment of anxious youth and reported comparable outcomes for comorbid and noncomorbid youth after receiving CBT for anxiety (Manassis et al., 2002; Southam-Gerow et al., 2001). Southam-Gerow and colleagues (2001) explored the influence of ADHD symptoms on immediate and long-term treatment outcomes of 135 children (ages 7 to 15; 39% females) with generalized anxiety disorder, separation anxiety disorder or social anxiety disorder who received individual CBT. Approximately 14% of the participants had a comorbid diagnosis of ADHD ($n$ =19). No differences were found in treatment response between the comorbid and noncomorbid youth. However, when discussing their findings, the authors'

advised caution in interpreting the results and in generalizing the findings to other clinical settings due to the relatively low levels of ADHD symptoms in the children and adolescents in their anxiety specialty clinic.

In the second study, Manassis et al. (2002) compared individual CBT and group CBT in a sample of 78 children (ages 8 to 12; 46% females) with generalized anxiety disorder, separation anxiety disorder, social anxiety disorder, specific phobia, or panic disorder. Six children met diagnostic criteria for comorbid ADHD (8% of the sample). In this study, symptoms of ADHD-H only were examined as a predictor across the treatment conditions (i.e., individual- and group-based CBT) after no difference in outcomes was found between the conditions (Manassis et al., 2002). No differences were observed between the children with high or low levels of hyperactivity in regard to treatment gains across conditions. In response to these findings, the authors indicated that greater parental involvement in both treatment conditions and the small group sizes may have helped to redirect the hyperactive children, thereby eliminating potential treatment barriers for youth with comorbid ADHD in the group-based CBT condition (Manassis et al., 2002).

ADHD and CBT Outcomes

Although somewhat mixed, these findings raise the question of what it is about ADHD that interferes with successful CBT outcomes for pediatric anxiety. At present the current literature has been limited to examining ADHD diagnosis and/or symptoms as a predictor of outcomes (Halldorsdottir & Ollendick, 2013). However, the current study expands on the current literature by exploring various facets of ADHD that may shed light on how co-occurring difficulties with inattention and overactivity impede successful treatment outcomes in anxious youth after receiving CBT, in addition to analyses at the symptomatic level. Based on previous

findings and prominent ADHD theories, the associations of CBT outcomes and the following aspects of ADHD were explored: ADHD core symptoms, effortful control, and sluggish cognitive tempo symptoms. In addition, maternal depression and parental involvement in treatment and their contributions to treatment outcomes were also examined.

*ADHD Symptoms.* As stated previously, effective treatments for anxiety build on cognitive and behavioral principles (Ollendick & King, 2012). Consistent with the observation by Storch et al. (2008), there is evidence to suggest that comorbid ADHD may interfere with the implementation of these principles, such as habituation which is thought to be necessary for successful CBT outcomes to occur in anxious youth. Habituation refers to a decrease in anxiety level as a result of *sustained* contact with the anxiety-provoking stimulus during exposures (Davis & Ollendick, 2005; Grayson, Foa, & Steketee, 1986; Tiwari, Kendall, Hoff, Harrison, & Fizur, 2013; Tryon, 2005). Given its dependence on uninterrupted anxious arousal, inattention and/or overactivity may interfere with habituation. Indeed, studies have found that distractibility, a hallmark symptom of ADHD, decreases the effectiveness of exposure treatments (Benito, Conelea, Garcia, & Freeman, 2012; Grayson, Foa, & Steketee, 1982; Grayson, Foa, & Steketee, 1986). In terms of the importance of exposures for CBT, there is a general consensus in the field that exposures are among the most effective 'ingredients' in CBT (Bouchard, Mandlowitz, Coles & Franklin, 2004; Hedtke, Kendall, & Tiwari, 2009). Accordingly, any disruption in the underlying process related to exposures may pose a barrier to successful treatment outcomes.

Furthermore, as detailed above, CBT traditionally includes components of psychoeducation, teaching the child to identify and challenge dysfunctional cognitions and use effective problem-solving skills. In essence, it is a skills-building approach aimed to change maladaptive thoughts and learning patterns. In sessions, the introduction of skills, initial practice of exposures and

problem-solving occurs. Throughout therapy, the effectiveness of these components relies heavily on the child's ability to stay involved and engaged. These types of cognitive interventions have largely been found to be ineffective in treating the core symptoms of ADHD and its associated difficulties (e.g., poor academic performance; Abikoff, 1991; Pelham, Wheeler, & Chronis, 1998).

Taken together, there is reason to believe that ADHD on a symptomatic level may interfere with the mechanism of CBT and children's ability to engage fully in the cognitive and behavioral aspects of the intervention. However, this may not be the whole story. If the disorder interfered solely on a symptomatic level, these behaviors would be observable in CBT sessions and they would be linked to poor treatment outcomes. To the contrary, Edmunds and Kendall (2013) found no in-session differences in ADHD-like behaviors between comorbid and noncomorbid youth. Moreover, in-session behaviors typical of youth with ADHD were not specifically associated with treatment outcomes. Accordingly, there is a call for examining other potentially interfering factors associated with ADHD and their relation to CBT outcomes.

*Effortful Control.* Theoretically proposed unobservable processes, rather than overt behaviors, may also pose difficulties for children and adolescents with ADHD seeking treatment for anxiety. Although the development of psychopathology is an intricate multifactorial process, certain temperament influences have been widely recognized to increase the risk of psychopathology and may be involved in its maintenance (e.g., Muris & Ollendick, 2005; Nigg, 2006). Temperament has been defined as inherent differences in reactivity and self-regulation (Martel & Nigg, 2006; Rothbart, 2004). Within temperament, three broad traits have been identified: negative emotionality, extraversion/surgency and effortful control (Rothbart, Ahadi, Hershey, & Fisher, 2001). In the current study, an emphasis was placed on effortful control as it

has been proposed as a core feature of the development of ADHD (Nigg, Goldsmith, & Sachek, 2004) and because of its clear potential to impede CBT outcomes. Also, effortful control has been conceptually related to neuropsychological executive functioning (Nigg, 2006; Rothbart & Bates, 1998) and emotion regulation (Martel, 2009), central features in other prominent theories of ADHD (Barkley, 1997; Brown, 2009). Effortful control refers to purposeful regulation of emotional states and ensuing behavior (Rothbart, 2004). Specifically, it involves the ability to deliberately focus and shift attention as well as the ability to suppress a dominant response in order to perform a subdominant response (i.e., response inhibition; Martel & Nigg, 2006; Rothbart, 2004). Despite the vast literature on temperament influences, few studies have examined how they impact treatment outcomes. However, similar to how overt symptoms may interfere with habituation, it is easy to see how deficits in these abilities may also be disruptive to CBT sessions. Accordingly, low levels of effortful control may be driving the difficulties experienced by children and adolescents with ADHD when receiving CBT. In other words, effortful control may mediate the negative association of ADHD symptoms and treatment outcomes. This possibility was explored in the current study.

*Sluggish Cognitive Tempo.* Alternatively, the heterogeneous presentation of ADHD may in part explain why some studies have found ADHD to be a barrier to successful outcomes in youth with anxiety, whereas other studies have not. For instance, as previously noted, a significant number of children with ADHD and anxiety displays symptoms of sluggish cognitive tempo (SCT; Becker & Langberg, 2012; Skirbekk et al., 2011). SCT has been defined by a range of behavioral symptoms, such as daydreaming, being slow to respond and process information, easily confused, having difficulties staying alert/awake in minimally stimulating situations, and lacking initiative to complete work (Barkley, 2013; McBurnett, Pfiffner, & Frick, 2001). SCT

symptoms have been found to negatively affect academic and social functioning above and beyond that of the ADHD core symptoms (Becker & Langberg, 2012). Given similarities between CBT sessions and school settings (new skills are taught and practiced), these symptoms might be hypothesized to affect treatment outcomes of anxious youth with co-occurring ADHD. Despite emerging research on SCT in recent years, no study has examined its relation to treatment outcomes, either for ADHD or anxiety to the author's knowledge. Accordingly, the examination of SCT and treatment outcomes was exploratory in this study.

*Maternal Depression*. Parental factors may also play a role in determining treatment outcomes in youth with co-occurring ADHD. As stated above, parental psychopathology, in particular depression, has been linked to poor treatment outcomes, both in youth receiving treatment for ADHD and anxiety (Chronis, Chacko, Fabiano, Wymbs, & Pelham, 2004; Southam-Gerow et al., 2001). Furthermore, studies have shown that parents of youth with ADHD have an increased risk of having Major Depressive Disorder (Chronis et al., 2004). In explaining poor CBT response in children of mothers with elevated levels of depressive symptoms, researchers have speculated that maternal depression may affect motivation for creating exposure activities and/or inadvertently reinforcing nonavoidant behaviors (Hops, 1992; Southam-Gerow et al., 2001). Additionally, there is evidence indicating decreased parental encouragement of applying new strategies among youth with ADHD (Costin, Vance, Barnett, O'Shea, & Luk, 2002). Accordingly, differences in parental psychopathology between youth with and without comorbid ADHD were compared and the predictive effects of parental psychopathology on treatment outcomes were examined.

*Parental involvement in Treatment.* On a more positive note, there could be factors that may help mitigate the unfavorable outcomes in anxious youth with comorbid ADHD that also

need to be explored. As suggested in previous literature, augmenting CBT for anxious youth with comorbid ADHD may be conducive of good outcomes. As such, increasing parental involvement in treatment may be a step in the right direction. Parental involvement is generally encouraged when treating anxiety in children and adolescents and thought to be associated with good outcomes (Bouchard et al., 2004; Kendall, Robin, Hedtke, & Suveg, 2005). Moreover, EBTs for ADHD rely mainly on parental participation in therapy. Accordingly, there is reason to believe that youth with co-occurring ADHD may have better outcomes when receiving parent-augmented CBT in which the parent is taught to conduct exposure and operant techniques are incorporated compared to individual-based CBT. As noted above, Storch et al. (2008) examined the differential treatment outcomes of anxious youth with and without co-occurring ADHD after receiving family-based CBT, whereas the other studies used individual-based or group-based CBT. Thus, it appears that youth with co-occurring ADHD may have poorer outcomes compared to their non-ADHD peers regardless of parental involvement. However, at present, no study has compared treatment outcomes in comorbid youth after receiving parent-augmented versus individual-based CBT. In the current study, differential treatment outcomes of comorbid youth receiving individual-based or parent-augmented CBT were explored.

## Current Study

To summarize, the relationship between various aspects associated with ADHD and outcomes in youth receiving CBT for a specific phobia was explored. Specifically, the current study first examined the predictive and moderating effect of ADHD symptoms on treatment outcomes of youth receiving CBT for a specific phobia. Additionally, the study expanded upon past work by also exploring various features of ADHD that may hinder successful outcomes in anxious youth as predictors and moderators (in separate analyses) of treatment outcomes. That is,

in independent analyses, how effortful control, SCT symptoms, and maternal depression in combination with ADHD symptoms predict treatment outcomes in youth receiving CBT for a specific phobia was examined. Given literature suggestive that an ADHD diagnosis may pose a barrier to successful treatment outcomes (Storch et al., 2008; Halldorsdottir et al., 2013), ADHD diagnosis was also examined as a predictor and moderator of treatment outcomes in exploratory nature due to the small sample size ($n$ = 28). The study is therefore important in determining for whom and under what conditions the one-session treatment for SPs is effective and may shed light on how ADHD may interfere with optimal treatment response in comorbid youth receiving CBT for anxiety.

## Specific Aims

The aims of the proposed study are as follows:

*Aim 1.* Examine the predictive effect of ADHD (at the dimensional and diagnostic level) on CBT outcomes in children and adolescents seeking treatment for a specific phobia.

*Aim 2.* Explore whether effortful control mediated the relationship between ADHD and CBT outcomes.

*Aim 3.* Investigate the relationship between SCT and CBT outcomes

*Aim 4.* Inspect differences in parental psychopathology between anxious youth with and without ADHD and the relationship between parental psychopathology and treatment outcomes.

*Aim 5.* In independent analyses, examine whether ADHD (on a dimensional and diagnostic level) moderate treatment outcomes between youth receiving individual-based or parent-augmented CBT for a specific phobia. Two forms of OST will be contrasted: individual OST and a parent-augmented OST.

## Hypotheses

Based on the previous literature, the following hypotheses are offered:

*Hypothesis 1.* ADHD, on a dimensional and diagnostic level (in separate analyses) will negatively predict successful treatment outcomes at post-, 1-month and 6-month follow-ups across treatment conditions.

*Hypothesis 2.* Participants with higher levels of ADHD symptoms will present with lower levels of effortful control than participants with lower levels of ADHD symptoms.

*Hypothesis 3.* Effortful control will mediate the relationship between ADHD and poor treatment outcomes, independent of treatment condition.

*Hypothesis 4.* The predictive effect of SCT symptoms, in combination with ADHD symptoms, to treatment outcomes will be explored. Given the lack of prior research, no directional hypothesis is offered.

*Hypothesis 5.* Greater levels of maternal depression will be observed in parents of youth with elevated levels of ADHD symptoms compared to parents of those with lower levels of ADHD symptoms.

*Hypothesis 6.* Greater levels of maternal depression, in combination with ADHD symptoms, will predict poor treatment outcomes across treatment conditions.

*Hypothesis 7.* Treatment response will be moderated by the presence of ADHD (on a dimensional and diagnostic level). Specifically, participants with ADHD will experience greater reduction in the targeted specific phobia after receiving parent-augmented CBT than their counterparts in the individual-based condition at all time points.

## II. Method

### Participants

Participants were 135 children and adolescents (ages 6-15; 52.2% females; 88.2% white) diagnosed with a specific phobia who participated in an NIMH-sponsored randomized controlled trial examining the efficacy of two cognitive-behavioral interventions for specific phobias (Ollendick, 2007). These participants largely came from 2-parent households (78.5%), most mothers (64.4%) and fathers (51.9%) graduated from a 4-year college/university, with an average household income of $106,889.64 (*SD* = $80515.16). Of these individuals, 28 (ages 7-13; 34.5% female; 89.7% white) met full diagnostic criteria for ADHD. Within the comorbid ADHD group, ADHD-C was the most common subtype of ADHD (*n* = 18, 64.3%) and ADHD was most commonly the secondary diagnosis (*n* = 13, 46.4%) (see Table 1 for further description of comorbid ADHD group). Prior to treatment, 11 participants were on medication for ADHD symptoms. Of those, nine met diagnostic criteria for ADHD.

### Interventions

Participants were randomized to either the individual-based one-session treatment or the parent-augmented one-session treatment (Ollendick, 2007; Öst & Ollendick, 2001). Both treatment conditions involved a functional analysis (FA), a 3-hour therapy session, and 4 weekly phone checks following treatment. The differences in the two treatment conditions, parental involvement, are described below.

*Individual-based CBT* (Öst & Ollendick, 2001). In this condition, the child alone received treatment with minimal parental involvement. During the FA, the parent received brief feedback on the assessment findings. Thereafter, information to inform relevant exposures was obtained from the child and the rationale and structure of treatment was explained. Approximately a week

after the FA, the family returned for the treatment session. As noted earlier, in this condition, the child alone received treatment. The objective of the three-hour session was to gradually expose the child to the phobic stimuli and to assist the child in the cognitive challenge of thoughts and beliefs associated with the feared stimulus. Following treatment, the parents were contacted once a week for four consecutive weeks to determine how the child was doing.

*Parent-augmented CBT* (Ollendick, 2007). Two clinicians were assigned to each family; one clinician worked with the child and the other with the parent. As noted above, the family attended a FA in which the child and parent each met with a clinician in separate rooms. Information relevant for devising appropriate exposures was obtained from both parent(s) and the child. Additionally, the parent(s) received psychoeducation on anxiety. The rationale for treatment was explained to the family at the end of the FA. In the treatment phase, similar to the individual-based condition, the child worked with a clinician in gradual exposures to the phobic stimuli. The parent and parent clinician observed the treatment behind a one-way mirror. Furthermore, in addition to observing the child clinician conduct exposures, the parent received coaching in strategies to reinforce appropriate approach behavior and decrease avoidance and distress associated with the phobic object. Parents were also assisted in designing exposures for the child after treatment and utilizing a reward system to motivate exposures. In the last part of the session, the parent joined the child clinician and the child and practiced the skills they had observed. Afterwards, the parent clinician contacted the parent once a week for four weeks to encourage the family to continue with ongoing exposures and to help problem solve any difficulties with continued exposures outside of session.

Measures

*Anxiety Disorders Interview Schedule for DSM–IV: Child and Parent Versions (ADIS–C/P*; Silverman & Albano, 1996*).* The ADIS–C/P is a semi-structured diagnostic interview in which clinicians assign a clinician severity rating (CSR) on a 9-point scale (0–8, with a rating ≥ 4 suggesting a clinical level of interference) for each endorsed disorder. Test-retest kappa coefficients for the ADIS have been found to range from 0.65-1.00, depending on the disorder (Silverman, Saavedra, & Pina, 2001). The ADIS has been recommended for diagnosing anxiety in youth with ADHD since other measures (e.g., DISC) may tend to over-report anxiety disorders (March et al., 2000). Furthermore, the reliability and validity of the ADIS for the diagnosis of ADHD has been established (Jarrett, Wolff, & Ollendick, 2007). In the current study, diagnoses of specific phobia and ADHD were derived from the ADIS. Reliability for the top three parent (primary diagnosis kappa coefficient = .93, secondary diagnosis kappa coefficient = .92, and tertiary diagnosis kappa coefficient = .73) and child diagnoses (primary diagnosis kappa coefficient = .80, secondary diagnosis kappa coefficient = .89, and tertiary diagnosis kappa coefficient = .90) diagnoses were acceptable. Changes in CSR of the specific phobia following treatment served as the primary outcome measure in this study.

*Brief Symptom Inventory* (BSI; Derogatis, 1975). The BSI is composed of 53 items designed to assess psychological functioning in adults. It is a self-report in which individuals are asked to rate their functioning on a 5-point scale (1= *not at all* to 5 = *extremely*). The measure includes the following subscales: somatization, obsessive-compulsive, interpersonal sensitivity, depression, anxiety, hostility, phobic anxiety, psychoticism, and paranoid ideation and a global severity index. The BSI has been found to have adequate reliability and validity (Boulet & Boss, 1991). Maternal report on the depression subscale of the BSI was used to assess maternal

depression in the current study. The internal consistency for the maternal depression scale was good (Cronbach's α = .790).

*Child Behavioral Checklist/6-18* (CBCL, Achenbach & Rescorla, 2001). The CBCL is a well-validated multidimensional parent-report measure of the child's behavioral and emotional functioning. Parents rate how true each item is for their child on a 3-point scale (0 = *not true*, 1 = *somewhat or sometimes true*, and 2 = *very true or often true*). The measure has two broad band scales, Internalizing and Externalizing behaviors. The measure includes the following narrow band scales: anxious/depressed symptoms, withdrawn/depressed symptoms, somatic complaints, attention problems, delinquent behavior, social problems, thought problems and aggressive behaviors. Although less studied at present, the CBCL also has the following clinical scales: affective problems, anxiety problems, somatic problems, attention deficit/hyperactivity problems, oppositional defiant problems, conduct problems, sluggish cognitive tempo, obsessive-compulsive problems, and post-traumatic stress problems. Test-retest reliability over a 1-week interval has found to be .95 for the problem scales (Achenbach, 1991).

For the purpose of this study, the *t*-scores on the ADHD and sluggish cognitive tempo problems subscales were used. The ADHD problems scale was used to assess ADHD symptoms (7 items; *fails to finish things he/she starts, can't concentrate, can't sit still, impulsive or acts without thinking, can't pay attention for long, talks too much*, and *unusually loud*). Symptoms of sluggish cognitive tempo were measured with the sluggish cognitive tempo subscale (4 items; *confused or seems to be in a fog, daydreams or gets lost in his/her thoughts, stares blankly*, and *underactive, slow moving or lacks energy*). The scale has been found to be reliable and distinct from measures of ADHD (Garner, Marceaux, Mrug, Patterson, & Hodgens, 2010; Wahlstedt & Bohlin, 2010). Internal consistency for the ADHD problems was acceptable (Cronbach's α =

.878), whereas the internal consistency for the SCT problems (Cronbach's α = .640) was low in the current study. Given that the ADHD and SCT problems subscales are less known in the literature and the SCT problems subscale had relatively low internal consistency, in independent analyses, the attention problems subscale, which is essentially a composite of the aforementioned scales (10 items: *acts young, fails to finish things he/she starts, can't concentrate, can't sit still, confused or seems to be in a fog, daydreams or gets lost in his/her thoughts, impulsive or acts without thinking, poor schoolwork, can't pay attention for long,* and *stares blankly*), was also examined. Internal consistency for the attention problems scale was adequate (Cronbach's α = .872).

*Early Adolescent Temperament Questionnaire, Revised* (EATQ-R; Ellis & Rothbart, 2001). The EATQ-R is a parent-report measure designed to assess subcomponents of temperament related to self-regulation, emotionality, and reactivity. The scale is composed of 62 items rated on a 5-point scale (1 = *almost always untrue of your child* to 5 = *almost always true of your child*). The scale contains 12 scales: activation control, affiliation, attention, fear, frustration, high intensity pleasure, inhibitory control, perceptual sensitivity, pleasure sensitivity, shyness, aggression and depressed mood. The EATQ-R has been found to be a reliable measure of temperament (Ellis & Rothbart, 2001). The composite score of the activation control, attention and inhibitory control served as a measure of effortful control in the present study (Ellis & Rothbart, 2001). Internal consistency of the effortful control scale (Cronbach's α = .903) was good in the current study.

Procedure

Interested parents completed a brief phone interview to assess eligibility and appropriateness for study inclusion. After the phone screen procedure, eligible families came to

the Child Study Center for a psychological assessment to determine final eligibility and symptom severity. Upon arrival, the study was described and verbal and written consent/assent was obtained. During the assessment session, one clinician worked with the parent while another worked with the child. After the assessment, eligibility and diagnoses were determined during a consensus process. If appropriate for the study, the family was randomized to a treatment condition (individual or parent-augmented CBT). Thereafter, the family came in for the FA and treatment (described above). After the intervention, depending on the treatment condition, the child or parent clinician conducted weekly phone check-ins with the parents about ongoing exposures. Finally, the families returned for assessments at one-week, 1-month and 6-months following treatment. Overall, 135 children and their parent(s) met inclusion and exclusion criteria and participated in the randomized trial (see Figure 1).

Data Analysis

Chi-square and *t*-tests were conducted to compare pre-treatment characteristics between the two treatment groups and participants who completed follow up assessment versus those who did not.

Latent growth curve analyses, which are a special case of structural equation modeling, were performed to examine each proposed model (see Figures 2-7). Such analyses have been found to provide good estimates of treatment effects (Bentler, 1991). In the proposed study, latent growth curve models are favored over more traditional statistical methods (e.g., repeated measures ANOVA) for four reasons: a) the current study examines the relationship between latent variables (i.e., nonobservable constructs); b) this type of analysis is recommended when evaluation designs contain some sources of variance (Lipsey & Cordray, 2000; Muthén & Curran, 1997). For instance, there was variation in targeted specific phobia (i.e., animal, natural

environment) in the current study; c) this type of analysis has been found to be more effective in detecting group differences in small sample sizes than many traditional statistical methods (for review, see Muthén & Curran, 1997) and d) a better capacity to accommodate for missing data points (for review, see Kline, 2011). With respect to missing data, maximum likelihood estimation for incomplete data sets, available through structural equation model computer programs, has been found to outperform more traditional statistical methods in dealing with missing data. Attrition in longitudinal studies is common and to be expected (for attrition in the current study, see Table 2). Accordingly, an approach that deals well with missing data was advantageous. Although structural equation modeling is generally a large-sample technique, relatively small sample sizes are sufficient when continuous outcome variables are normally distributed and their association with one another is linear (Kline, 2011), as were those in the present study. With a sample of 135 participants, the present study has the power to detect a medium effect size at $\beta$=.80 (Muthén & Curran, 1997).

Analyses were conducted in two steps. First, latent growth analyses were performed to examine the nature of individual-level changes in treatment outcomes. Secondly, analyses were conducted to assess the degree to which these individual-level changes relate to the proposed predictors. In the current study, the intercept corresponds to pre-treatment level of the child's specific phobia, as measured by the CSR, and the slope corresponds to the rate of change in the target specific phobia (decrease or increase in CSR) over the period of the study (see Figures 2-6). The intercept, which represents the pre-treatment level of the child's specific phobia, is a latent factor that is constant for individuals across time. The intercept factor represents information about the mean ($\mu_I$) and variance ($\sigma^2_I$) across the individual intercepts of each participant's growth curve. The factor loadings for the intercept have been fixed to a value of one

(represented as $\lambda_{1I}$ to $\lambda_{4I}$). The slope factor denotes information about the mean ($\mu_S$) and variance ($\sigma^2_S$) of the collection of individual slopes of the growth curves. Loadings on the slope are fixed to constants that represent the times of measurement, starting with 0 for the first measurement prior to treatment ($\lambda_{1S}$), 1 for the post-treatment ($\lambda_{2S}$), and the loadings for 1-month and 6-month follow up were freely estimated ($\lambda_{3S}$ and $\lambda_{4S}$). As indicated by a double-headed arrow, the two latent factors are allowed to covary. This covariance (estimated as $\sigma_{SI}$) represents the degree to which pre-treatment levels of the child's specific phobia predict rates of linear change in regard to the child's specific phobia through the study (see Figure 2 for the unconditional model).

In the current study, in five latent growth models, the degree to which 1) ADHD symptoms/diagnosis, 2) effortful control, 3) sluggish cognitive tempo, 4) maternal depression, and 5) parental involvement, respectively, influenced individual slopes associated with changes over the course of 6-months in a child's specific phobia after receiving either individual-based or parent-augmented CBT were examined (see Figures 3-7). Specifically, the relationship between ADHD symptoms and treatment outcomes across the different time points was explored. As noted above, it was hypothesized that lower levels of ADHD symptoms would yield greater change in the slope (i.e., CSR of the targeted specific phobia over time) compared to participants with higher levels of ADHD symptoms (represented as $\gamma_{SP1}$ in Figure 3). The second model illustrates how the negative relationship between ADHD symptoms and treatment outcomes is mediated by effortful control. This is represented by the indirect effect of effortful control through ADHD symptoms ($\gamma_{indSP2}$) on the slope in Figure 4. The third model exemplifies the correlation between ADHD and SCT symptoms ($\sigma_{P1P3}$) and how higher levels of these symptoms negatively predict change in the slope (represented as $\gamma_{SP1}$ and $\gamma_{SP3}$ in Figure 5). Figure 6 shows higher levels of maternal depression were correlated with higher levels of ADHD symptoms

($\sigma_{P1P4}$) and that both greater levels of maternal depression and child ADHD symptoms negatively predict change in the slope ($\gamma_{SP1}$ and $\gamma_{SP4}$, respectively). Lastly, Figure 7 illustrates how the differential treatment outcomes of youth with elevated levels of ADHD symptoms/diagnosis based on treatment condition was explored. As noted earlier, the negative association of ADHD with the slope is expected to be greater in participants in the individual-based CBT condition than those in the parent-augmented CBT condition (represented with the interaction term $\gamma_{SP1*P5}$ in Figure 7).

Maximization likelihood method of estimation was used to fit the models and three well-established fit indices were used to assess the model fit (Hu & Bentler, 1998). Specifically, the model chi-square, non-normed fit indices (NNFI; Bentler & Bonett, 1980) and root-mean-square error of approximation (RMSEA; Steiger, 1990). The NNFI ranges from 0 to 1 with values approaching 1 indicating a good fit for the data. The cutoff of .90 or above is considered a good fit. For the RMSEA, a value equivalent to or smaller than .08 is considered indicative of a good fit. The computer software Mplus 7 was used to fit the models.

## III. Results

### Attrition

Table 2 illustrates attrition/retention and comparison of demographic variables at each follow up appointment. All 135 participants completed the treatment. Of those, 120 (88.2%) came in for the post-treatment assessment, 107 (78.7%) at 1-month follow up and 76 (55.9%) completed the 6-month follow up assessment. No differences were noted in gender, age, race, locality, type or severity of the targeted specific phobia between participants who completed follow up and those who did not at the various time points. Retention of participants was comparable in the individual and parent-augmented intervention across time points. Similarly,

differences in ADHD symptoms, SCT symptoms, effortful control, and maternal depression between completers and noncompleters were nonsignificant. Attrition rates did not differ significantly across participants with and without an ADHD diagnosis at posttreatment (p = .20), 1-month follow up (*p* = .25) or 6-month follow up (*p* = .34, see Table 2).

Pretreatment Differences

Differences between participants with and without an ADHD diagnosis and low versus elevated ADHD symptoms were compared on key demographic factors with *t*-test and chi-square statistics. As expected, youth with a co-occurring ADHD diagnosis had significantly higher levels of ADHD symptoms (see Table 3). There was also a significant gender difference between participants with a diagnosis of ADHD. Specifically, there was a higher ratio of males to females with comorbid ADHD than in the noncomorbid group. However, when broken down by associated ADHD symptoms, no other differences in terms of demographics were observed (see Table 4). Similarly, within the participants with comorbid ADHD, no differences were noted in treatment outcomes between participants on medication for ADHD (*n* = 11) versus those who were not on such medication (*n* = 19) (Table 5).

Latent Growth Curve Models

Table 6 displays the means, standard deviations, skewness, kurtosis and intercorrelations of all study variables. Weak to high intercorrelations were found in the severity of the specific phobia across the time points, ranging from .120 to .677, with the correlations higher among the time points following treatment. Additionally, as expected, significant positive correlations among ADHD symptoms and SCT symptoms (r = .533) and ADHD symptom and levels of maternal depressions (r = .188) were observed. Similarly, a significant negative correlation was found among ADHD symptoms and levels of effortful control (r = -.737). All variables had

univariate normal distribution, although the *t*-scores of ADHD problems, SCT symptoms and maternal depression were somewhat positively skewed. However, multivariate normality was not met (multivariate skew = 21.06, $p = .04$; multivariate kurtosis = 99.80, $p = .11$) (Mardia, Kent, & Bibby, 1979). As previously noted, the expectation maximization algorithm was used to accommodate for missing data, which has been found to be robust to minor non-normality in small samples.

*Unconditional Model.* Data preparation revealed that neither a linear or quadratic slope fit the model well. Accordingly, as recommended by Kline (2011), the model was run with the first two loadings fixed to 0 and 1 and the last two time points freely estimated. Based on this analysis, a non-linear model with the slope factor loadings fixed at 0, 1, 1.324 and 1.472 was found to fit the data well. The slope factor loadings indicate that there was a substantial drop in the CSR of the specific phobia from pretreatment (CSR = 6.47) to posttreatment (CSR = 4.10). Although continued decrease in CSR across time was observed, the decrease plateaued, indicating that treatment gains were maintained (CSR = 3.33 and CSR = 2.98 at the 1- and 6-month follow up, see Figure 8). Based on the gender discrepancies noted above, all the following analyses were examined with and without gender as a covariate. However, given that the models yielded comparable findings, the unstandardized results from the models without gender as a covariate are presented below. Figure 2 illustrates the unconditional model ($\chi^2$ (5, 135) = 5.563, $p = .351$, TLI = .993, RMSEA = .029; see unconditional model in Table 7). In the model, participants had an average specific phobia CSR of 6.471 ($p < .001$) prior to treatment. The mean slope was significant and negative ($\mu_S$= -2.375, $p <.001$), indicating a decrease in the severity of the specific phobia after treatment. A summary of all the unstandardized and standardized results

from all the latent growth models with and without gender as a covariate is presented in Tables 7 to 9.

*ADHD Symptoms.* Figure 3 illustrates the growth curve model depicting the study's first hypothesis regarding the relationship between ADHD and treatment outcomes. To significantly increase model fit, the measurement errors of the post-treatment and 1-month follow were allowed to correlate ($\chi^2$ (6, 131) = 7.155, $p$ = .307, TLI = .978, RMSEA = .039; see Model 1 in Table 7). ADHD symptoms did not predict pre-treatment phobia severity ($\gamma_{IP1}$ = .011, $p$ = .405) or changes in phobia severity across time ($\gamma_{SP1}$ = -.027, $p$ = .096).

*Effortful Control and ADHD Symptoms.* Mediation testing was conducted based on Baron and Kenny's (1986) four step process in establishing mediation. As previously noted, ADHD symptoms did not significantly predict treatment outcomes. This finding indicates that there was no effect that may be mediated. Accordingly, no further testing of mediation occurred. However, the indirect effect of effortful control on treatment outcomes was explored. Figure 4 shows the growth curve model illustrating the relationship between effortful control, ADHD symptoms and treatment outcomes ($\chi^2$ (11, 129) = 19.649, $p$ = .050, TLI = .939, RMSEA = .078; see Model 2 in Tables 7 to 9). ADHD symptoms negatively predicted effortful control symptoms ($\gamma_{P1P2}$ = -.216, $p$ < .001). In other words, higher levels of ADHD symptoms were associated with lower levels of effortful control, as expected. However, the indirect effect of ADHD on pre-treatment levels of phobia severity ($\gamma_{indIP1}$ = .011, $p$ =.277) and trajectory of change after treatment ($\gamma_{indSP1}$ = .004, $p$ = .773) were not significant.

*SCT and ADHD Symptoms.* The relationship between SCT and ADHD symptoms with treatment outcomes is presented in Figure 5 ($\chi^2$ (9, 135) = 12.114, $p$ = .207, TLI = .948, RMSEA = .050; see Model 3 in Tables 7 to 9). The covariance between SCT and ADHD symptoms was

significant ($\sigma_{P1P3}$ = 24.941, $p < .001$). However, neither ADHD nor SCT symptoms predicted pre-treatment phobia severity ($\gamma_{IP1}$ = .015, $p$ = .328 and $\gamma_{IP3}$ = -.005, $p$ = .754, respectively) or change in phobia severity over time ($\gamma_{SP1}$ = -.039, $p$ = .073 and $\gamma_{SP3}$ = .029, $p$ = .211, respectively). Similarly, the composite score of ADHD and SCT symptoms, as measured by the attention problems of the CBCL ($\chi^2$ (6, 130) = 7.922, $p$ = .243, TLI = .974, RMSEA = .041), was not significantly associated with pre-treatment phobia severity ($\gamma_{IP1}$ = .012, $p$ = .317) or change in phobia severity over time ($\gamma_{SP1}$ = -.014, $p$ = .370; see Model 3[b] in Tables 7 to 9).

*Maternal Depression and ADHD Symptoms.* Figure 6 demonstrates the relationship between maternal depression, ADHD symptoms and treatment outcomes ($\chi^2$ (8, 135) = 11.659, $p$ = .167, TLI = .932, RMSEA = .058; see Model 4 in Tables 7 to 9). Measurement errors of phobia severity at 1-month and 6-months following treatment were allowed to covary to increase the fit of the model. The covariance between ADHD symptoms and maternal depression was significant ($\sigma_{P1P4}$ = 9.775, $p$ = .028). ADHD symptoms and maternal depression did not predict pre-treatment severity of the specific phobia ($\gamma_{IP1}$ = .012, $p$ = .361 and $\gamma_{IP4}$ = -.002, $p$ = .909) nor change in the targeted phobia after treatment ($\gamma_{SP1}$ =-.033, $p$ = .076 and $\gamma_{SP4}$ = .022, $p$ = .235).

*Treatment Condition and ADHD Symptoms.* Treatment condition was dummy coded (0 = individual-based CBT, 1 = parent-augmented CBT) and ADHD symptoms were grand-mean centered. Figure 7 illustrates the association between ADHD, treatment conditions and treatment outcomes ($\chi^2$ (11, 129) = 7.918, $p$ = .721, TLI = 1.061, RMSEA = .000; see Model 5 in Tables 7 to 9). Treatment condition was not significantly associated with pre-treatment specific phobia severity ($\gamma_{IP5(\text{Treatment condition})}$ = .282, $p$ = .128), indicating that randomization was successful. The interaction between ADHD symptoms and treatment condition was not significantly associated with changes over time in specific phobias after receiving treatment ($\gamma_{SP1*P5}$ = .020, $p$ = .512). In

other words, participants with ADHD had comparable treatment outcomes independent of receiving individual or parent-augmented treatment.

*ADHD diagnosis.* As noted above, in exploratory analyses, an ADHD diagnosis was examined as a predictor ($\chi^2$ (7, 135) = 8.626, $p$ = .281, TLI = .974, RMSEA = .041) of treatment outcomes (Model 6 in Tables 7 to 9). An ADHD diagnosis was not significantly associated with pre-treatment phobia severity ($\gamma_{IP1}$ = -.202, $p$ = .351) or change in phobia severity over time ($\gamma_{SP1}$ = -.057, $p$ = .853). Also in an exploratory fashion, ADHD diagnosis was examined as a moderator ($\chi^2$ (11, 135) = 7.590, $p$ = .750, TLI = 1.063, RMSEA = .000) of treatment outcomes (Model 7 in Tables 7 to 9). The interaction between ADHD diagnosis and treatment condition was not significantly associated with changes over time in specific phobias after receiving treatment ($\gamma_{SP1*P5}$ = .385, $p$ = .509). The findings suggest that participants with a comorbid ADHD diagnosis had comparable treatment outcomes independent of treatment condition (i.e., individual-based or parent-augmented treatment).

## IV. Discussion

Findings on whether ADHD is associated with CBT outcomes have been mixed. Some evidence has been found that ADHD negatively predict CBT outcomes. However, what conditions and what mechanism associated with ADHD may be associated with CBT outcomes are poorly understood at this time. Toward this end, the relationship between various facets of ADHD and CBT outcomes were explored in the current study. More precisely, the predictive effects of overt ADHD (dimensionally and diagnostically), symptoms of sluggish cognitive tempo, levels of effortful control, symptoms of maternal depression and treatment modality with treatment outcomes of youth who received an intensive CBT for a specific phobia were examined.

Given pretreatment differences in gender based on the presence of ADHD diagnosis, all analyses were conducted with and without gender as a covariate. Comparable findings were observed independent of gender included as a covariate. To summarize the findings, contrary to our first hypothesis, ADHD symptoms were not predictive of the severity of the specific phobia prior to treatment or the trajectory of change in the severity of the specific phobia up to 6-months following the treatment. As previously noted, the findings in terms of whether ADHD is associated with treatment outcomes in youth have been mixed (Halldorsdottir & Ollendick, 2013). Within the literature, the findings have been mixed especially when the presence of ADHD symptoms are based on parental report (Halldorsdottir et al., 2013; Manassis et al., 2002; Southam-Gerow et al., 2001). However, other studies have found that an ADHD diagnosis derived from a structured interview negatively predicts treatment outcomes in youth (Halldorsdottir et al., 2013; Storch et al., 2008). Accordingly, exploratory analyses were conducted examining the predictive effect of an ADHD diagnosis on treatment outcomes. Also contrary to our hypothesis, an ADHD diagnosis did not predict treatment outcomes. In other words, ADHD symptoms and an ADHD diagnosis were not significantly associated with treatment outcomes in this treatment-seeking sample of 135 youth with SPs. This finding suggests that the one-session treatment is a robust treatment for specific phobia. Several aspects of the treatment may have facilitated the successful treatment outcomes in youth with comorbid ADHD and perhaps some of these elements are shared with other studies in which ADHD was not found to be associated with treatment outcomes (Manassis et al., 2002; Southam-Gerow et al., 2001). Specifically, the intensive CBT used in this study (i.e., OST) entails many components consistent with recommendations on how to accommodate for the deficits in youth with ADHD (Halldorsdottir & Ollendick, 2013; Hudson, Krain, & Kendall, 2001; Pelham &

Fabiano, 2008). For instance, the interventions were highly interactive with psychoeducation and exposures presented in a multimodal fashion (i.e., auditory, visual, and tactile presentation), which may prevent fatigue or boredom. Throughout treatment an emphasis was placed on positive reinforcement and in-session practice. Participants were provided with specific short-term single step commands/goals (e.g., 'Let us move one step closer to the dog') and specifically praised for accomplishing these goals (e.g., 'Look how brave you are taking one step closer to the dog!'). The intervention was also structured in that children were provided with regular breaks every hour. During the break, children in both conditions were encouraged to share their success in treatment with their parents, thereby often eliciting more positive reinforcement from caregivers.

There may also be other aspects of treating a specific phobia that may be conducive of successful treatment outcomes in comorbid youth. For instance, in comparison to the treatment of other anxiety disorders, the circumscribed nature of a specific phobia and the OST's focus on this one anxiety (i.e., the phobic stimuli) may be advantageous for treating youth with comorbid ADHD. Treatment for other anxiety disorders tends to have a broader focus. Specifically, there may be multiple anxiety-provoking areas (e.g., public speaking and speaking to adults) that need to be addressed. On a similar note, exposures for a specific phobia can be more tangible than exposures for other anxiety disorders. That is, the top of the hierarchy for a child with a specific phobia may involve an *in-vivo* exposure of petting a dog or staying in a dark room for an extended period of time. Alternatively, a child with generalized anxiety disorder or separation anxiety disorder may be fearful of dying or a loved one dying. Therefore, the exposure may need to rely more heavily on *imaginal* exposures or cognitive challenging (e.g., discussion of thought-action fusion or probabilities; Kendall & Hedtke, 2006). Also related to the circumscribed nature

of specific phobias, the OST only requires the parents to attend one therapy session in addition to the functional analysis, thereby eliminating concerns of treatment dropout, which some studies have found to be higher among comorbid youth (Edmunds & Kendall, 2013; Rapee, 2003). Future research is necessary to explore these possible explanations.

In line with the proposed hypotheses, lower levels of effortful control were significantly associated with higher levels of ADHD symptoms. However, no support was found for the hypothesis that effortful control mediated the relationship between ADHD symptoms and change in treatment outcomes over time. This finding was surprising as it was expected that high levels of effortful control would be needed to successfully engage in an exposure-based therapy. To elaborate, a child seemingly needs to be able to regulate his/her emotional state and ensuing behaviors in order to actively engage in an activity when there is a strong tendency to avoid it. With such an interesting finding, it is unfortunate that research on temperament and treatment outcomes is limited. To the author's knowledge only one study has examined the predictive effect of effortful control on CBT outcomes in youth (Festen et al., 2013). However, similar to the current findings, Festen and her colleagues found that effortful control did not predict CBT outcomes in anxious youth. As such, it remains unclear how children with low levels of effortful control accommodate for their difficulties and evidence successful treatment outcomes comparable to youth with higher levels of effortful control.

Similarly, SCT symptoms were not significantly associated with the severity of the specific phobia prior to treatment nor did SCT symptoms predict change over time in the severity of the specific phobia after treatment. Given low internal consistency of the SCT subscale, the attention problems subscale (a composite of ADHD and SCT symptoms) was also examined as a predictor of change in the specific phobia over time. Similar to the SCT findings, the composite

score was not significantly associated with treatment outcomes. Although also initially surprising, there are several possible explanations for this finding. Along with having low internal consistency of the SCT measure, our measure did not include all the symptoms/deficits typically found in youth with SCT. More precisely, our measure did not include SCT symptoms pertaining to slow processing speed of information, difficulties staying alert/awake in minimally stimulating situations, increased withdrawal and lack initiative and/or transient efforts to complete work (Barkley, 2013). It is possible that these symptoms may have greater influence on treatment outcomes than the symptoms included in our measure. Alternatively, the sample size may not have been sufficient to capture whether SCT symptoms interfere with CBT outcomes. Approximately 40% of youth with ADHD have accompanying SCT (Barkley, 2013). In the current study, only a small portion of participants ($n$ = 15) presented with elevated levels of SCT symptoms ($t$-score $\geq$ 65). Furthermore, recent studies have found that SCT symptoms become more impairing with age and are more associated with depressive disorders than anxiety disorders (Barkley, 2012; Barkley, 2013). Therefore, further research of the association of SCT with CBT outcomes is needed with a measure that more fully captures the construct in a larger and more diverse sample.

Consistent with previous literature and our hypothesis, maternal depressive symptoms were significantly associated with ADHD symptoms. Specifically, mothers' of youth with elevated levels of ADHD symptoms were more likely to endorse higher levels of depressive symptoms. However, maternal depressive symptoms in combination with ADHD symptoms were not significantly associated with the severity of the specific phobia prior to treatment or change over time in the targeted specific phobia. This was also a surprising finding as maternal depression has been implicated with poor treatment outcomes in youth receiving psychosocial

treatment for either ADHD (Chronis et al., 2004) or anxiety (Southam-Gerow et al., 2001). It should be noted that in the current study there was relatively limited variability in maternal depressive symptoms in the mothers of the children treated at our clinic. The majority of the mothers presented with average to mild depressive symptoms ($n$ = 117 or 92.9% < $t$-score 60). Indeed, in different clinical settings where mothers experience greater levels of maternal depression treatment outcomes may not have been as positive.

Lastly, the interaction between ADHD (dimensionally and diagnostically) and treatment outcomes was not significantly related to the trajectory of change in the specific phobia after the two treatments. In other words, comparable trajectories were found in the individual-based and parent-augmented treatment independent of ADHD. This was an also unexpected finding given that evidence-based treatments for ADHD rely on parental involvement. Therefore, we expected the parent-augmented treatment to yield better outcomes for comorbid youth as parents were coached in conducting exposures outside of treatment and encouraged to increase positive reinforcement through a reward system similar to the operant strategies found to be effective in managing ADHD (Pelham & Fabiano, 2008). However, across the two treatment conditions, parents were called once a week for four consecutive weeks by a clinician for a brief check in on how the child was doing. In the individual-based condition, this phone call may have served as a reminder to the child and parent to continue to overcome his/her fear leading to continued out-of-session exposure and/or reinforcement of treatment gains. Accordingly, there may have been greater parental involvement in both treatment conditions than typically seen in individual-based therapy. On the other hand, within the anxiety literature, findings on increasing parental involvement in CBT for childhood anxiety have been inconsistent with the majority of the studies not finding additional gains despite research indicating that parent factors contribute to

the development and maintenance of childhood psychopathology (for review see Breinholst, Esbjorn, Reinholdt-Dunne, & Stallard, 2012). In their recent review, Breinholst and her colleagues offered several reasons for why this may be the case. Of relevance to the current study, the specific parental factors for comorbid youth (e.g., elevated symptoms of maternal depression) were not specifically targeted in either treatment condition. Although not identified as a barrier to treatment outcomes, incorporating treatment components for psychopathology in parents may increase response rates to treatment above and beyond that of individual treatment.

*Limitations*. Findings in the current study must be interpreted in light of study limitation. In addition to the aforementioned limitations, the majority of the sample was Caucasian and from largely middle-class families. Secondly, all the comorbid youth were diagnosed with ADHD-C or ADHD-I, with the exception of one participant. Therefore, no exploration of the differential influence of the various subtypes of ADHD on treatment outcomes for anxiety could be conducted. Future research is needed to examine whether the distinct subtypes of ADHD, in particular ADHD-H, differentially influence treatment outcomes. Third, maternal report of ADHD symptoms or a clinician rating based on parent and child report were used as a predictors and moderators of treatment outcomes in the current study. Therefore, the study is missing confirmation of ADHD symptoms in two settings (i.e., at home and at school) which is part of the diagnostic criteria. Fourth, the present study was conducted in a research university-based clinic in which the family only needed to attend one treatment session and therapists received careful training and supervision by peer clinicians and a licensed clinical psychologist.. Clinicians were also trained in flexible administration of the protocol while adhering to the fidelity of the treatment. As such, accommodations were made for youth with co-occurring difficulties associated with ADHD. Accordingly, caution is advised in generalizing the findings

to community settings wherein higher levels of attrition are found and behavioral comorbidity are more prevalent in anxious youth (Jarrett & Ollendick, 2008; Southam-Gerow et al., 2003; Tannock, 2009). Additionally, caution in interpreting the findings is also advised due the lack of control for medication status during analyses. ADHD would presumably not interfere with treatment gains when symptoms are being effectively controlled by medication. However, descriptive analyses suggested that there were no differences in treatment outcomes between comorbid youth on ($n$ = 9) versus not on medication ($n$ = 19) (see Table 5). Furthermore, multivariate normality was not met, which may increase Type I errors (West, Finch, & Curran, 1995). However, it should be noted that maximum likelihood estimation has been found to perform better than asymptotically distribution free techniques under violations of normality in terms of chi square goodness of fit test and the parameter estimates in small samples ($n$ < 200) (Chou & Bentler, 1995). Lastly, other facets associated with ADHD were not considered as predictors in the current study. Consequently, future studies should examine the predictive effect of other aspects of ADHD, such as other executive functioning deficits (e.g., working memory deficits) and between-session assignment compliance, on treatment outcomes as well. Addressing these limitations in future studies will strengthen conclusions regarding the predictive and moderating effect of ADHD on anxiety treatment outcomes. These limitations notwithstanding, the current study supports that the one-session treatment (regardless of treatment modality) for SPs is generally effective for anxious youth with a wide range of difficulties. If replicated, particularly in community-based service clinics, this finding will be important for implementing and disseminating evidence-based treatments for anxiety and ADHD as anxiety and ADHD frequently co-occur.

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## VI. Tables

Table 1. Breakdown of pre-treatment characteristics of comorbid ADHD group.

| | *n(%)* |
|---|---|
| ADHD-C | 18(64.3) |
| ADHD-I | 9(32.1) |
| ADHD-H | 1(3.6) |
| Primary diagnosis | 8(28.6) |
| Secondary diagnosis | 13(46.4) |
| Tertiary diagnosis | 7(25.0) |

*Note*. ADHD-C = attention-deficit/hyperactivity disorder, combined type, ADHD-H = attention-deficit/hyperactivity disorder, predominantly hyperactive-impulsive type, ADHD-I = attention-deficit/hyperactivity disorder, predominantly inattentive type, *n* = sample size.

Table 2. Comparison of completers at each time point on key demographic variables.

| | Pretreatment ($n$ = 135) | Posttreatment ($n$ = 120) | 1-month fu ($n$ = 107) | 6-month fu ($n$ = 76) |
|---|---|---|---|---|
| Categorical Variable | *n(%)* | *n(%)* | *n(%)* | *n(%)* |
| Gender | | | | |
| *Female* | 70(51.9) | 62(51.7) | 60(56.1) | 40(52.6) |
| *Male* | 65(48.1) | 58(48.3) | 47(43.9) | 36(47.4) |
| Race | | | | |
| *White* | 119(88.1) | 105(87.5) | 96(89.7) | 69(90.8) |
| *Non-white* | 16(11.9) | 15(12.5) | 11(10.3) | 7(9.2) |
| Town | | | | |
| *Local* | 97(71.9) | 89(74.2) | 83(77.6) | 60(78.9) |
| *Out of town* | 38(28.1) | 31(25.8) | 24(22.4) | 16(21.1) |
| Type of phobia | | | | |
| *Animal* | 54(40.0) | 48(40.0) | 42(39.3) | 28(36.8) |
| *Other* | 81(60.0) | 72(60.0) | 65(60.7) | 48(63.2) |
| Treatment condition | | | | |
| *Individual CBT* | 48(35.6) | 45(37.5) | 42(39.3) | 31(40.8) |
| *Family-based CBT* | 87(64.4) | 75(62.5) | 65(60.7) | 45(59.2) |
| ADHD diagnosis | | | | |
| *Comorbid* | 28(20.7) | 23(19.2) | 20(18.7) | 18(23.7) |
| *Noncomorbid* | 107(79.2) | 97(80.8) | 87(81.3) | 58(76.3) |
| Continuous Variable | *M(SD)* | *M(SD)* | *M(SD)* | *M(SD)* |
| Age | 8.94(1.82) | 9.00(1.85) | 9.08(1.92) | 8.96(1.64) |
| Pretreatment SP CSR | 6.47(1.03) | 6.46(1.00) | 6.44(1.03) | 6.28(1.03) |
| ADHD sx | 55.81(7.06) | 55.79(7.09) | 56.12(7.28) | 56.01(7.15) |
| SCT sx | 54.48(6.49) | 54.58(6.69) | 54.68(6.90) | 54.41(6.67) |
| Effortful control | 9.65(2.06) | 9.70(2.09) | 9.57(2.16) | 9.69(2.25) |
| Maternal depressive sx | 46.75(6.94) | 46.81(7.08) | 47.02(7.17) | 46.26(7.04) |

*Note*. ADHD = attention-deficit/hyperactivity disorder, CBT = cognitive-behavioral therapy, CSR = clinician severity rating, fu = follow up, SP = specific phobia, sx = symptoms.

Table 3. Pre-treatment descriptives for participants with and without a co-occurring ADHD diagnosis.

| | Comorbid (*n* = 28) *n(%)/M(SD)* | Not Comorbid (*n* = 107) *n(%)/M(SD)* | $\chi^2/t$ | *p* |
|---|---|---|---|---|
| Gender | | | | |
| *Female* | 9(32.1) | 61(57.0) | 4.64 | **.031** |
| *Male* | 19(67.9) | 46(43.0) | | |
| Ethnicity | | | | |
| *White* | 25(89.3) | 94(87.9) | .07 | .789 |
| *Non-white* | 3(10.7) | 13(12.1) | | |
| Age | 8.91(1.52) | 9.00(1.90) | .28 | .78 |
| ADHD symptoms | 63.28(7.22) | 53.89(5.65) | 7.25 | **<.001** |
| Specific Phobia CSR | 6.32(1.06) | 6.51(1.03) | .94 | .35 |
| Phobia Type | | | | |
| *Animal* | 9(32.1) | 45(42.1) | 1.16 | .282 |
| *Other* | 19(67.9) | 62(57.9) | | |
| Treatment Condition | | | | |
| *Individual CBT* | 9(32.1) | 39(36.4) | .04 | .845 |
| *Parent-augmented CBT* | 19(67.9) | 68(63.6) | | |

*Note*. ADHD = attention-deficit/hyperactivity disorder, CBT = cognitive behavioral therapy, CSR = clinician severity rating.

Table 4. Comparison of pre-treatment descriptives broken down by participants with and without elevated ADHD symptoms.

| | Average ADHD sx (*n* = 108) *n(%)/M(SD)* | Elevated ADHD sx (*n* = 23) *n(%)/M(SD)* | $\chi^2/t$ | *p* |
|---|---|---|---|---|
| Gender | | | | |
| *Female* | 59(54.6) | 11(47.7) | .35 | .553 |
| *Male* | 49(45.4) | 12(52.2) | | |
| Ethnicity | | | | |
| *White* | 95(88.0) | 22(95.7) | 1.18 | .278 |
| *Non-white* | 13(12.0) | 1(4.3) | | |
| Age | 8.84(1.79) | 9.39(1.97) | 1.30 | .198 |
| Comorbid ADHD dx | | | | |
| *Comorbid* | 11(10.2) | 15(65.2) | 36.1 | **<.001** |
| *Noncomorbid* | 97(89.8) | 8(34.8) | | |
| Specific Phobia CSR | 6.50(1.05) | 6.48(.90) | .09 | .926 |
| Phobia Type | | | | |
| *Animal* | 46(42.6) | 7(30.4) | 1.16 | .281 |
| *Other* | 62(57.4) | 16(69.6) | | |
| Treatment Condition | | | | |
| *Individual CBT* | 37(34.3) | 11(47.8) | 1.50 | .220 |
| *Parent-augmented CBT* | 71(65.7) | 12(52.2) | | |

*Note*. ADHD = attention-deficit/hyperactivity disorder, CBT = cognitive behavioral therapy, CSR = clinician severity rating, sx = symptoms.

Table 5. Comparison of treatment outcomes across time within participants with comorbid ADHD broken down by medication status.

| | | Pretreatment | Posttreatment | 1-month fu | 6-month fu |
|---|---|---|---|---|---|
| No medication (*n* = 19) | *M*(*SD*) | 6.26(1.10) | 4.07(2.02) | 3.00(1.92) | 2.00(2.18) |
| On medication (*n* = 9) | *M*(*SD*) | 6.44(1.01) | 4.33(1.87) | 2.57(1.90) | 2.50(2.08) |

*Note*. fu = follow up, *M* = mean, *n* = sample size, *SD* = standard deviation.

Table 6. Means, standard deviation, skewness, kurtosis and correlations of study variables

| Variable | *M* | *SD* | Skewness | Kurtosis | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1. ADHD sx | 55.78 | 6.96 | 1.23 | .68 | 1 | | | | | | | | | | |
| 2. EC | 9.65 | 2.05 | -.34 | -.55 | -.737* | 1 | | | | | | | | | |
| 3. SCT sx | 54.58 | 6.46 | 1.37 | .80 | .533* | -.540* | 1 | | | | | | | | |
| 4. Attention px | 56.31 | 7.24 | 1.35 | 1.37 | .858* | -.755* | .763* | 1 | | | | | | | |
| 5. DEP | 46.67 | 6.91 | 1.14 | .11 | .188* | -.093 | .208* | .178* | 1 | | | | | | |
| 6. Tx | .64 | .48 | -.61 | -1.65 | -.110 | .023 | -.093 | -.118 | -.150 | 1 | | | | | |
| 7. ADHD dx | .21 | .41 | 1.46 | .13 | .526* | -.462* | .356* | .512* | .048 | .017 | 1 | | | | |
| 8. Pre CSR | 6.47 | 1.03 | -.52 | -.06 | .063 | -.114 | -.005 | .075 | .025 | .105 | -.081 | 1 | | | |
| 9. Post CSR | 4.14 | 1.74 | -.06 | -.79 | .062 | -.216* | .082 | .130 | .045 | .075 | .012 | .314* | 1 | | |
| 10. 1-month fu | 3.30 | 1.84 | .24 | -.89 | -.112 | -.059 | .029 | .002 | .136 | .044 | -.122 | .214* | .677* | 1 | |
| 11. 6-month fu | 2.83 | 2.03 | .48 | -.65 | -.172 | -.026 | -.008 | -.134 | -.048 | .113 | -.104 | .120* | .483* | .544* | 1 |

*Note.* ADHD = attention-deficit/hyperactivity disorder, CSR = clinician severity rating of the targeted specific phobia, DEP = maternal depression, EC = effortful control, fu = follow up, *M* = mean, px = problems, SCT = sluggish cognitive tempo, *SD* = standard deviation, sx = symptoms, Tx = treatment condition.

*$p < .05$.

Table 7. Unstandardized results of the latent growth analyses for all models.

| Parameter/ fit measure | Unconditional Model | Model 1 | Model 2 | Model 3 | Model 3[b] | Model 4 | Model 5 | Model 6 | Model 7 |
|---|---|---|---|---|---|---|---|---|---|
| $\mu_I$ | 6.471* | 5.892* | 6.980* | 5.902* | 5.798* | 5.857* | 6.320* | 6.514* | 6.369* |
| $\sigma_I^2$ | .973* | .983* | .728 | .841 | .956 | 1.002* | .083 | 1.021* | 1.223* |
| $\mu_S$ | -2.375* | -.904 | -2.221* | -1.758 | -1.641 | -1.590 | -2.428* | -2.366* | -2.359* |
| $\sigma_S^2$ | 1.465* | .932* | 1.117* | 1.312* | 1.010* | 1.549* | 1.840* | 1.500* | 1.339* |
| $\sigma_{IS}$ | -.415 | -.356 | -.154 | -.290 | -.354 | -.436 | -.336 | -.463 | -.649* |
| $\gamma_{IP1(ADHD)}$ | - | .011 | - | .015 | .012 | .012 | .013 | -.202 | -.219 |
| $\gamma_{SP1(ADHD)}$ | - | -.027 | - | -.039 | -.014 | -.033 | -.039 | -.057 | -.369 |
| $\gamma_{P1P2(EC)}$ | - | - | -.216* | - | - | - | - | - | - |
| $\gamma_{IP2(EC)}$ | - | - | -.049 | - | - | - | - | - | - |
| $\gamma_{SP2(EC)}$ | - | - | -.017 | - | - | - | - | - | - |
| $\gamma_{indIP1(ADHD)}$ | - | - | .011 | - | - | - | - | - | - |
| $\gamma_{indSP1(ADHD)}$ | - | - | .004 | - | - | - | - | - | - |
| $\gamma_{IP3(SCT)}$ | - | - | - | -.005 | - | - | - | - | - |
| $\gamma_{SP3(SCT)}$ | - | - | - | .029 | - | - | - | - | - |
| $\sigma_{P1P3}$ | - | - | - | 24.941* | - | - | - | - | - |
| $\gamma_{IP4(DEP)}$ | - | - | - | - | - | -.002 | - | - | - |
| $\gamma_{SP4(DEP)}$ | - | - | - | - | - | .020 | - | - | - |
| $\sigma_{P1P4}$ | - | - | - | - | - | 9.775* | - | - | - |
| $\gamma_{IP5(Tx)}$ | - | - | - | - | - | - | .282 | - | .228 |
| $\gamma_{SP5(Tx)}$ | - | - | - | - | - | - | -1.127 | - | -.042 |
| $\gamma_{SP1*P5(ADHD*Tx)}$ | - | - | - | - | - | - | .020 | - | .385 |
| $\chi 2$ | 5.563 | 7.155 | 19.649 | 12.114 | 7.922 | 11.659 | 7.918 | 8.626 | 7.590 |
| df | 5 | 6 | 11 | 9 | 6 | 8 | 11 | 7 | 11 |
| RMSEA | .029 | .039 | .078 | .050 | .050 | .058 | .000 | .041 | .000 |
| TLI | .993 | .978 | .939 | .948 | .974 | .932 | 1.061 | .974 | 1.063 |

*Note.* ADHD = attention-deficit/hyperactivity disorder symptoms/diagnosis, DEP = maternal depression, df = degrees of freedom, EC = effortful control, RMSEA = root mean square error of approximation, SCT = sluggish cognitive tempo symptoms, TLI = Tucker Lewis Index, Tx = treatment condition.

*$p < .05$.

Table 8. Standardized results of the latent growth analyses for all models.

| Parameter/ fit measure | Unconditional Model | Model 1 | Model 2 | Model 3 | Model 3[b] | Model 4 | Model 5 | Model 6 | Model 7 |
|---|---|---|---|---|---|---|---|---|---|
| $\mu_I$ | 6.561* | 5.924* | 6.124* | 6.401* | 5.905* | 5.829* | 6.456* | 6.426* | 5.714* |
| $\sigma_I^2$ | 1.00 | .994* | .986* | .990* | .991* | .993* | .975* | .993* | .984* |
| $\mu_S$ | -1.963* | -.918 | -2.100* | -1.504 | -1.624 | -1.252 | -2.493* | -1.932* | -2.032* |
| $\sigma_S^2$ | 1.00 | .962* | .999* | .960* | .989* | .961* | .956* | 1.000* | .994* |
| $\sigma_{IS}$ | -.348 | -.372 | -.171 | -.276 | -.360 | -.350 | -.366 | -.374 | -.507* |
| $\gamma_{IP1(ADHD)}$ | - | .077 | - | .117 | .093 | .086 | .095 | -.199 | -.081 |
| $\gamma_{SP1(ADHD)}$ | - | -.195 | - | .235 | -.360 | -.183 | -.283 | -.047 | -.130 |
| $\gamma_{P1P2(EC)}$ | - | - | -.* | - | - | - | - | - | - |
| $\gamma_{IP2(EC)}$ | - | - | -.117 | - | - | - | - | - | - |
| $\gamma_{SP2(EC)}$ | - | - | -.032 | - | - | - | - | - | - |
| $\gamma_{indIP1(ADHD)}$ | - | - | .087 | - | - | - | - | - | - |
| $\gamma_{indSP1(ADHD)}$ | - | - | .024 | - | - | - | - | - | - |
| $\gamma_{IP3(SCT)}$ | - | - | - | -.037 | - | - | - | - | - |
| $\gamma_{SP3(SCT)}$ | - | - | - | .160 | - | - | - | - | - |
| $\sigma_{P1P3}$ | - | - | - | .541* | - | - | - | - | - |
| $\gamma_{IP4(DEP)}$ | - | - | - | - | - | -.011 | - | - | - |
| $\gamma_{SP4(DEP)}$ | - | - | - | - | - | .122 | - | - | - |
| $\sigma_{P1P4}$ | - | - | - | - | - | .201* | - | - | - |
| $\gamma_{IP5(Tx)}$ | - | - | - | - | - | - | .139 | - | .098 |
| $\gamma_{SP5(Tx)}$ | - | - | - | - | - | - | -.559 | - | -.017 |
| $\gamma_{SP1*P5(ADHD*Tx)}$ | - | - | - | - | - | - | .571 | - | .115 |
| $\chi 2$ | 5.563 | 7.155 | 19.649 | 12.114 | 7.922 | 11.659 | 7.918 | 8.626 | 7.590 |
| df | 5 | 6 | 11 | 9 | 6 | 8 | 11 | 7 | 11 |
| RMSEA | .029 | .039 | .078 | .050 | .050 | .058 | .000 | .041 | .000 |
| TLI | .993 | .978 | .939 | .948 | .964 | .932 | 1.061 | .974 | 1.063 |

*Note.* ADHD = attention-deficit/hyperactivity disorder symptoms/diagnosis, DEP = maternal depression, df = degrees of freedom, EC = effortful control, RMSEA = root mean square error of approximation, SCT = sluggish cognitive tempo symptoms, TLI = Tucker Lewis Index, Tx = treatment condition.

*$p < .05$.

Table 9. Unstandardized results of the latent growth analyses for all models with gender as covariate.

| Parameter/ fit measure | Unconditional Model | Model 1 | Model 2 | Model 3 | Model 3[b] | Model 4 | Model 5 | Model 6 | Model 7 |
|---|---|---|---|---|---|---|---|---|---|
| $\mu_I$ | 6.471* | 5.957* | 7.015* | 5.894* | 5.879* | 5.842* | 6.394* | 6.586* | 6.444* |
| $\sigma_I^2$ | .973 | .964 | .705 | .847 | .929 | 1.019* | .909 | 1.008* | 1.208* |
| $\mu_S$ | -2.375* | -1.510 | -2.595* | -2.420 | -2.362* | -2.460 | -2.827* | -2.901* | -2.877* |
| $\sigma_S^2$ | 1.465 | .905* | 1.080* | 1.308* | .962* | 1.563* | .873 | 1.471* | 1.299* |
| $\sigma_{IS}$ | -.415 | -.344 | -.140 | -.302 | -.336 | -.458 | -.318 | -.454 | -.638 |
| $\gamma_{IP1(ADHD)}$ | - | .011 | - | .015 | .012 | .012 | .013 | -.212 | -.230 |
| $\gamma_{SP1(ADHD)}$ | - | -.032 | - | -.035 | -.011 | -.029 | -.037 | .021 | -.365 |
| $\gamma_{P1P2(EC)}$ | - | - | -.216* | - | - | - | - | - | - |
| $\gamma_{IP2(EC)}$ | - | - | -.048 | - | - | - | - | - | - |
| $\gamma_{SP2(EC)}$ | - | - | -.034 | - | - | - | - | - | - |
| $\gamma_{indIP1(ADHD)}$ | - | - | .010 | - | - | - | - | - | - |
| $\gamma_{indSP1(ADHD)}$ | - | - | .007 | - | - | - | - | - | - |
| $\gamma_{IP3(SCT)}$ | - | - | - | -.005 | - | - | - | - | - |
| $\gamma_{SP3(SCT)}$ | - | - | - | .028 | - | - | - | - | - |
| $\sigma_{P1P3}$ | - | - | - | 24.938* | - | - | - | - | - |
| $\gamma_{IP4(DEP)}$ | - | - | - | - | - | -.002 | - | - | - |
| $\gamma_{SP4(DEP)}$ | - | - | - | - | - | .024 | - | - | - |
| $\sigma_{P1P4}$ | - | - | - | - | - | 9.786* | - | - | - |
| $\gamma_{IP5(Tx)}$ | - | - | - | - | - | - | .285 | - | .231 |
| $\gamma_{SP5(Tx)}$ | - | - | - | - | - | - | -1.267 | - | -.093 |
| $\gamma_{SP1*P5(ADHD*Tx)}$ | - | - | - | - | - | - | .02 | - | .505 |
| $\chi 2$ | 5.563 | 12.171 | 26.002 | 21.114 | 13.373 | 20.989 | 13.262 | 14.052 | 12.624 |
| df | 5 | 8 | 14 | 13 | 8 | 12 | 13 | 9 | 13 |
| RMSEA | .029 | .064 | .082 | .068 | .072 | .074 | .013 | .064 | .000 |
| TLI | .993 | .918 | .913 | .882 | .898 | .863 | .995 | .917 | 1.007 |

*Note.* ADHD = attention-deficit/hyperactivity disorder symptoms/diagnosis, DEP = maternal depression, df = degrees of freedom, EC = effortful control, RMSEA = root mean square error of approximation, SCT = sluggish cognitive tempo symptoms, TLI = Tucker Lewis Index, Tx = treatment condition.

*$p < .05$.

Table 10. Standardized results of the latent growth analyses for all models with gender as covariate.

| Parameter/ fit measure | Unconditional Model | Model 1 | Model 2 | Model 3 | Model 3[b] | Model 4 | Model 5 | Model 6 | Model 7 |
|---|---|---|---|---|---|---|---|---|---|
| $\mu_I$ | 6.561* | 6.050* | 6.293* | 6.370* | 6.070* | 5.767* | 6.616* | 6.537* | 5.814* |
| $\sigma_I^2$ | 1 | .994* | .986* | .990* | .991* | .993* | .973* | .993* | .984* |
| $\mu_S$ | -1.963* | -1.544 | -2.459* | -2.066 | -2.358* | -1.917 | -2.930* | -2.369* | -2.490* |
| $\sigma_S^2$ | 1 | .946* | .971* | .953* | .959* | .949* | .937* | .981* | .973* |
| $\sigma_{IS}$ | -.348 | -.368 | -.160 | -.286 | -.355 | -.363* | -.357 | -.373 | -.509* |
| $\gamma_{IP1(ADHD)}$ | - | .075 | - | .116 | .092 | .085 | .093 | -.086 | -.085 |
| $\gamma_{SP1(ADHD)}$ | - | -.170 | - | -.208 | -.080 | -.157 | -.269 | .007 | -.129 |
| $\gamma_{P1P2(EC)}$ | - | - | -.747* | - | - | - | - | - | - |
| $\gamma_{IP2(EC)}$ | - | - | -.116 | - | - | - | - | - | - |
| $\gamma_{SP2(EC)}$ | - | - | -.066 | - | - | - | - | - | - |
| $\gamma_{indIP1(ADHD)}$ | - | - | .087 | - | - | - | - | - | - |
| $\gamma_{indSP1(ADHD)}$ | - | - | .049 | - | - | - | - | - | - |
| $\gamma_{IP3(SCT)}$ | - | - | - | -.037 | - | - | - | - | - |
| $\gamma_{SP3(SCT)}$ | - | - | - | .120 | - | - | - | - | - |
| $\sigma_{P1P3}$ | - | - | - | .541* | - | - | - | - | - |
| $\gamma_{IP4(DEP)}$ | - | - | - | - | - | -.011 | - | - | - |
| $\gamma_{SP4(DEP)}$ | - | - | - | - | - | .130 | - | - | - |
| $\sigma_{P1P4}$ | - | - | - | - | - | .201* | - | - | - |
| $\gamma_{IP5(Tx)}$ | - | - | - | - | - | - | .143 | - | .100 |
| $\gamma_{SP5(Tx)}$ | - | - | - | - | - | - | -.635 | - | -.039 |
| $\gamma_{SP1*P5(ADHD*Tx)}$ | - | - | - | - | - | - | .636 | - | .152 |
| $\chi 2$ | 5.563 | 12.171 | 26.002 | 21.114 | 13.373 | 20.989 | 13.262 | 14.052 | 12.624 |
| df | 5 | 8 | 14 | 13 | 8 | 12 | 13 | 9 | 13 |
| RMSEA | .029 | .064 | .082 | .068 | .072 | .074 | .013 | .064 | .000 |
| TLI | .993 | .918 | .913 | .882 | .898 | .863 | .995 | .917 | 1.007 |

*Note.* ADHD = attention-deficit/hyperactivity disorder symptoms/diagnosis, DEP = maternal depression, df = degrees of freedom, EC = effortful control, RMSEA = root mean square error of approximation, SCT = sluggish cognitive tempo symptoms, TLI = Tucker Lewis Index, Tx = treatment condition.

*$p < .05$.

## VII. Figures

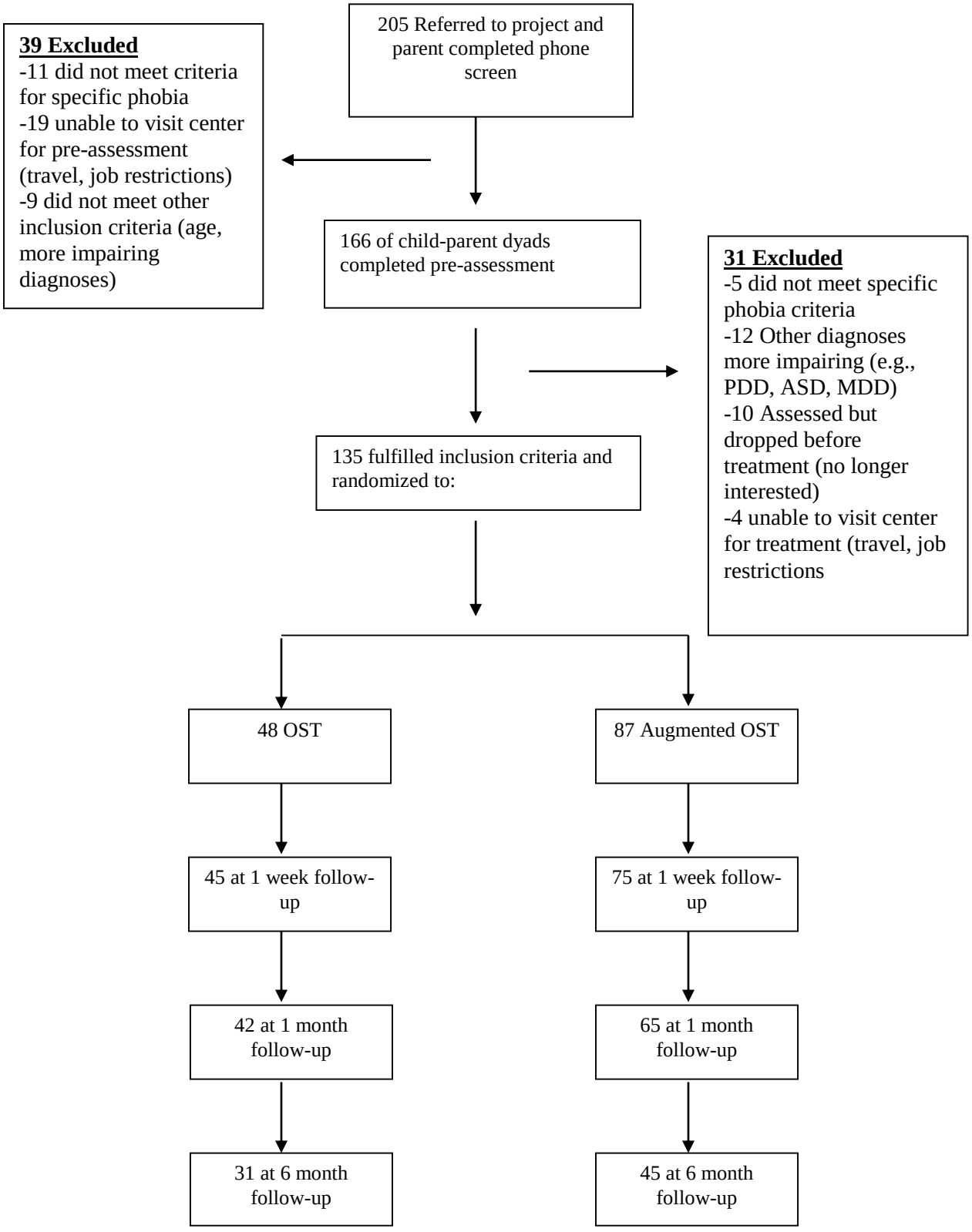


*Figure 1*. Flow chart of all participants through the study.

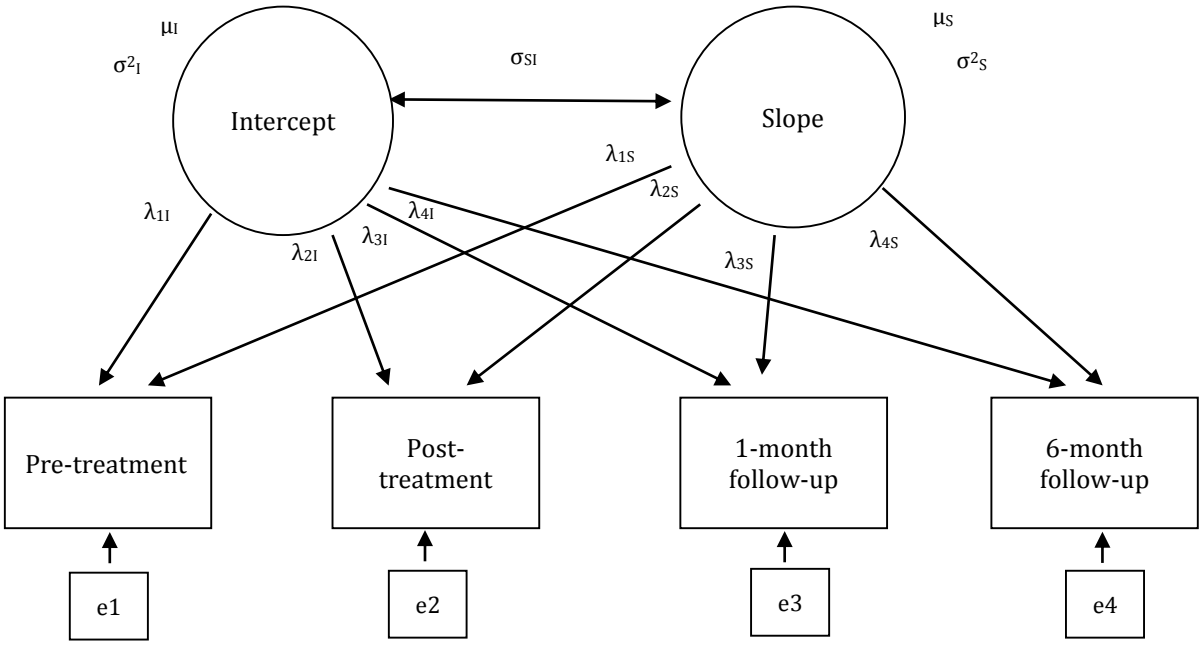


*Figure 2.* Unconditional growth curve model of treatment outcomes at posttreatment and at the 1-month and 6-month follow ups for participants across treatment conditions.

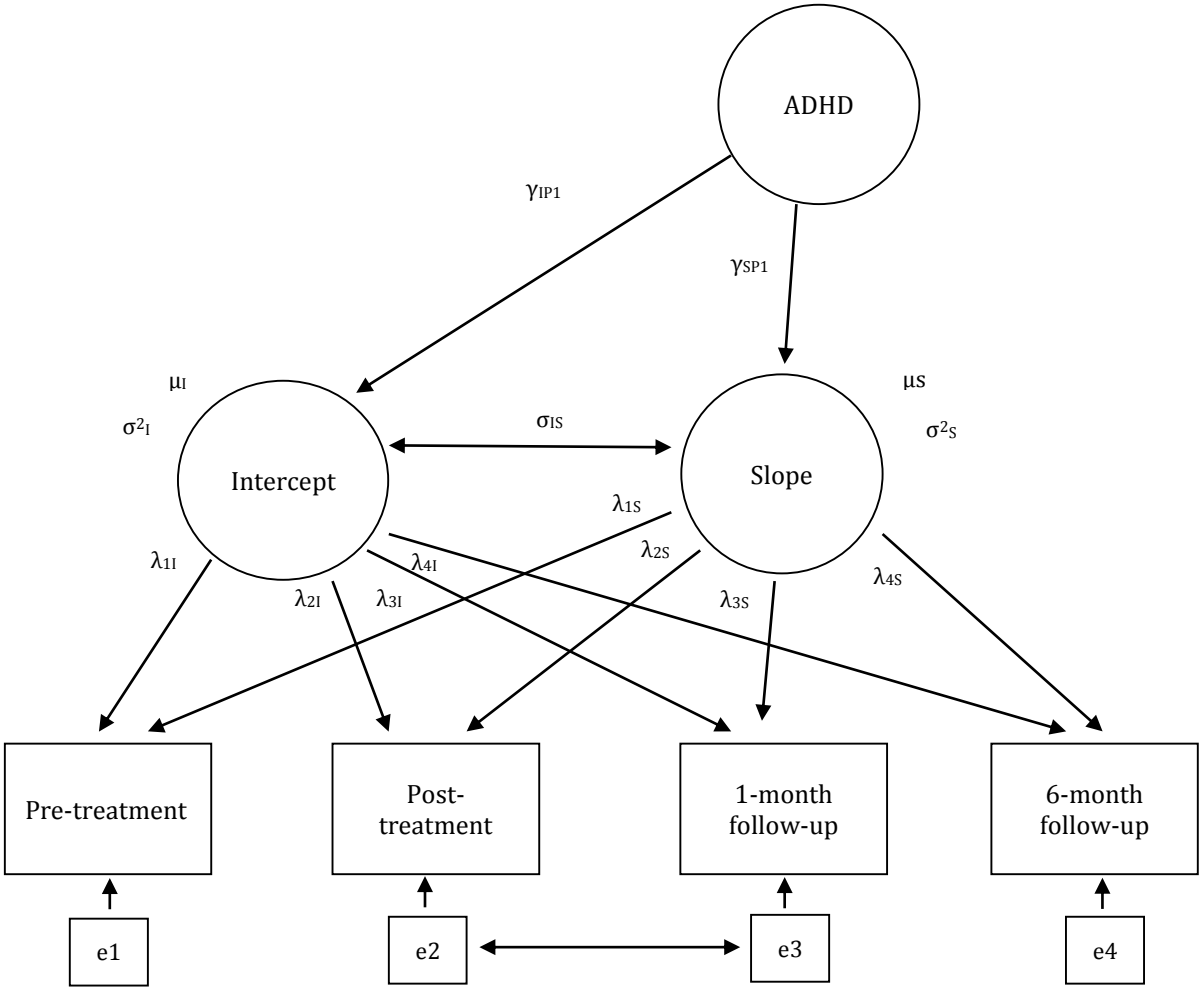


*Figure 3*. Growth curve model with ADHD as a predictor of treatment outcomes at posttreatment and at the 1-month and 6-month follow ups for participants across treatment conditions.

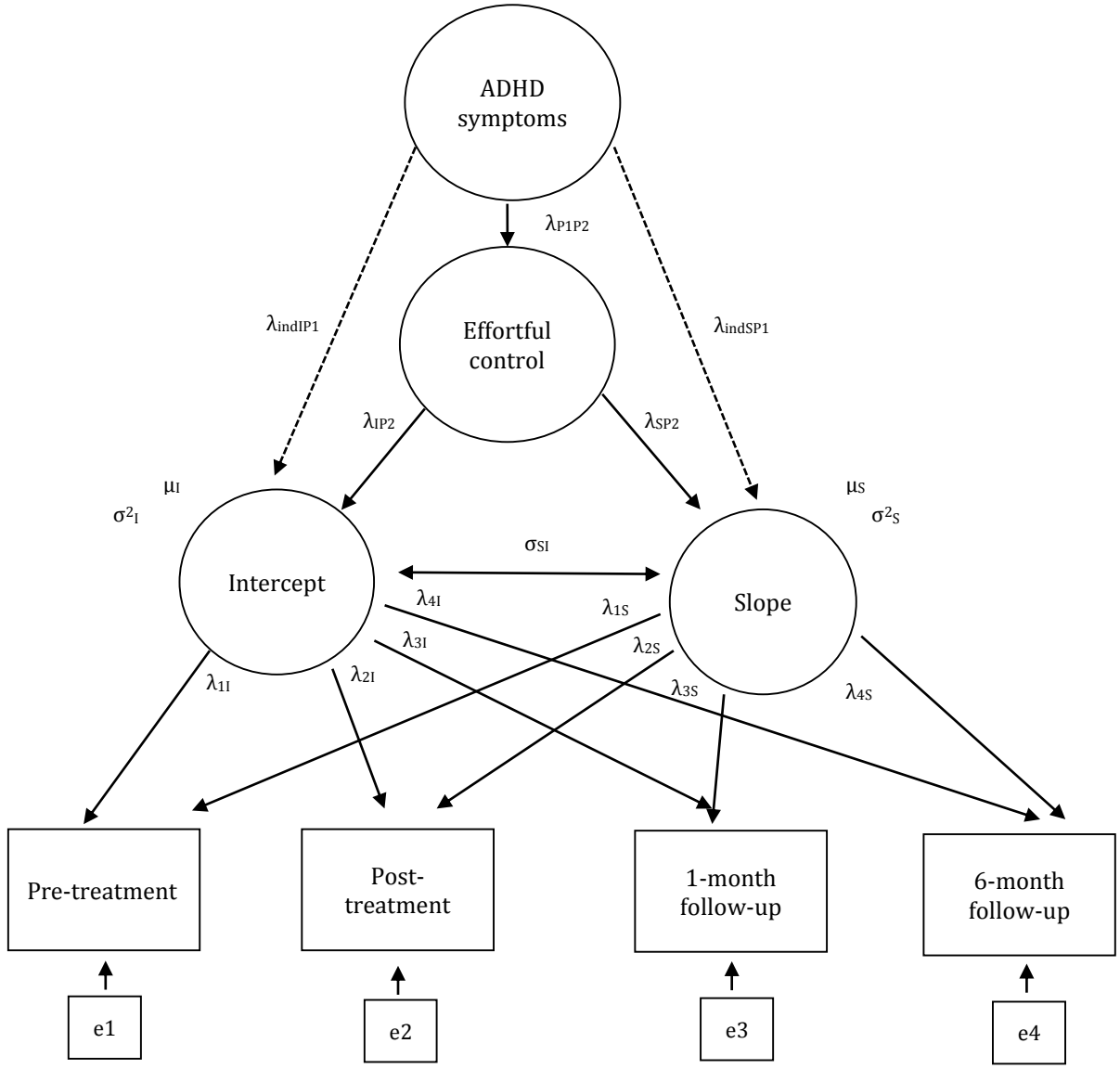


*Figure 4*. Growth curve model with effortful control as a mediator of ADHD symptoms and treatment outcomes at posttreatment and 1-month and 6-month follow ups.

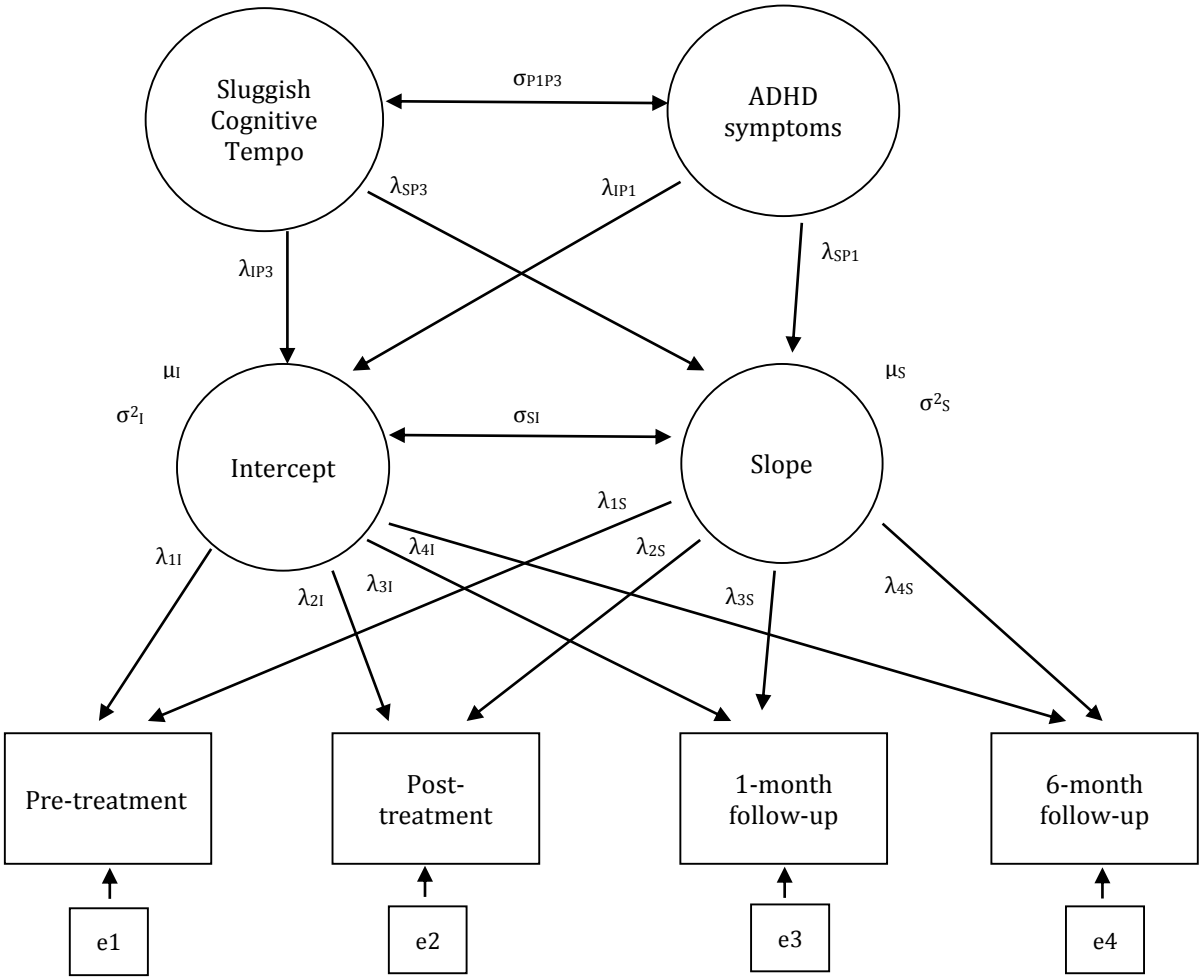


*Figure 5*. Growth curve model with symptoms of sluggish cognitive tempo and ADHD symptoms as predictors of treatment outcomes at posttreatment and at the 1-month and 6-month follow ups.

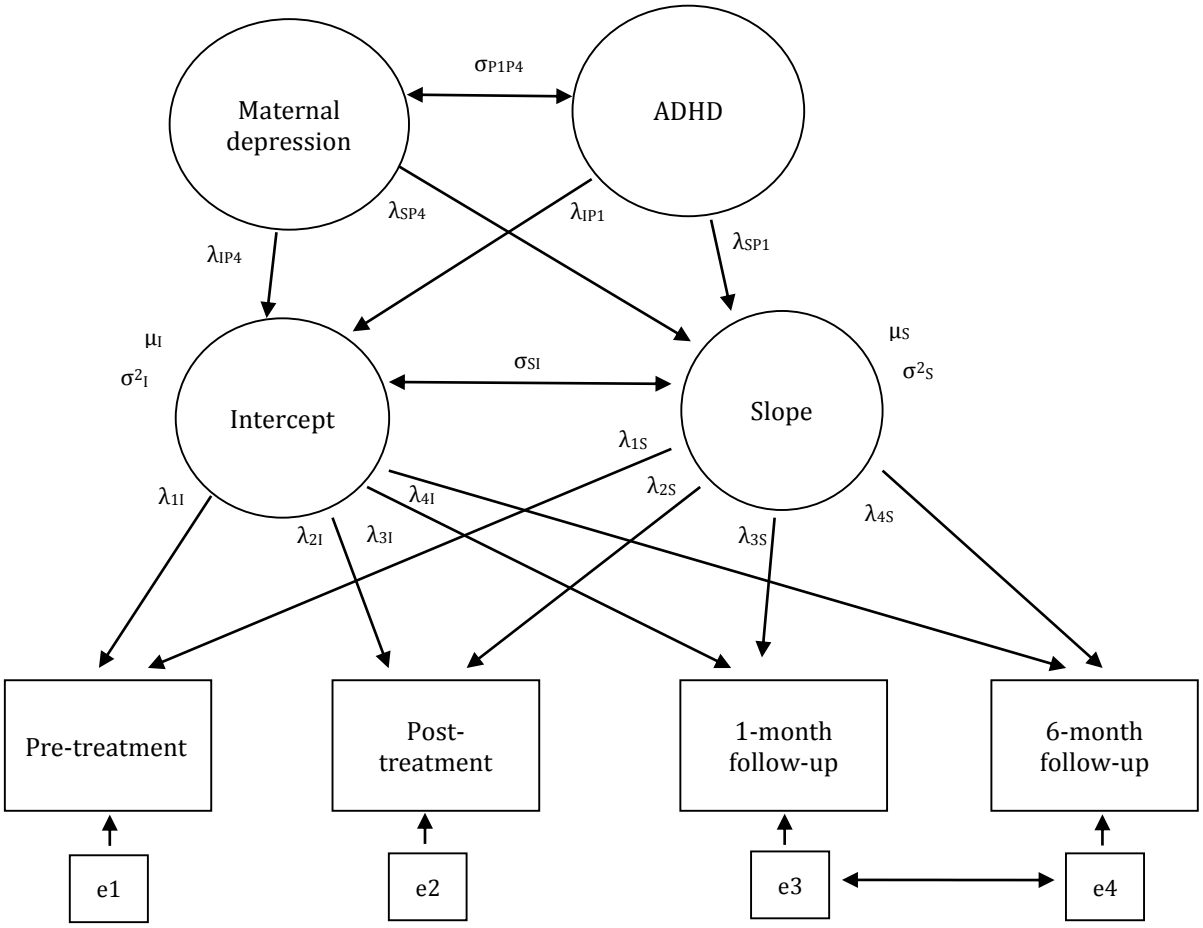


*Figure 6*. Growth curve model with maternal depression and ADHD symptoms as a predictor of treatment outcomes at posttreatment and 1-month and 6-month follow ups.

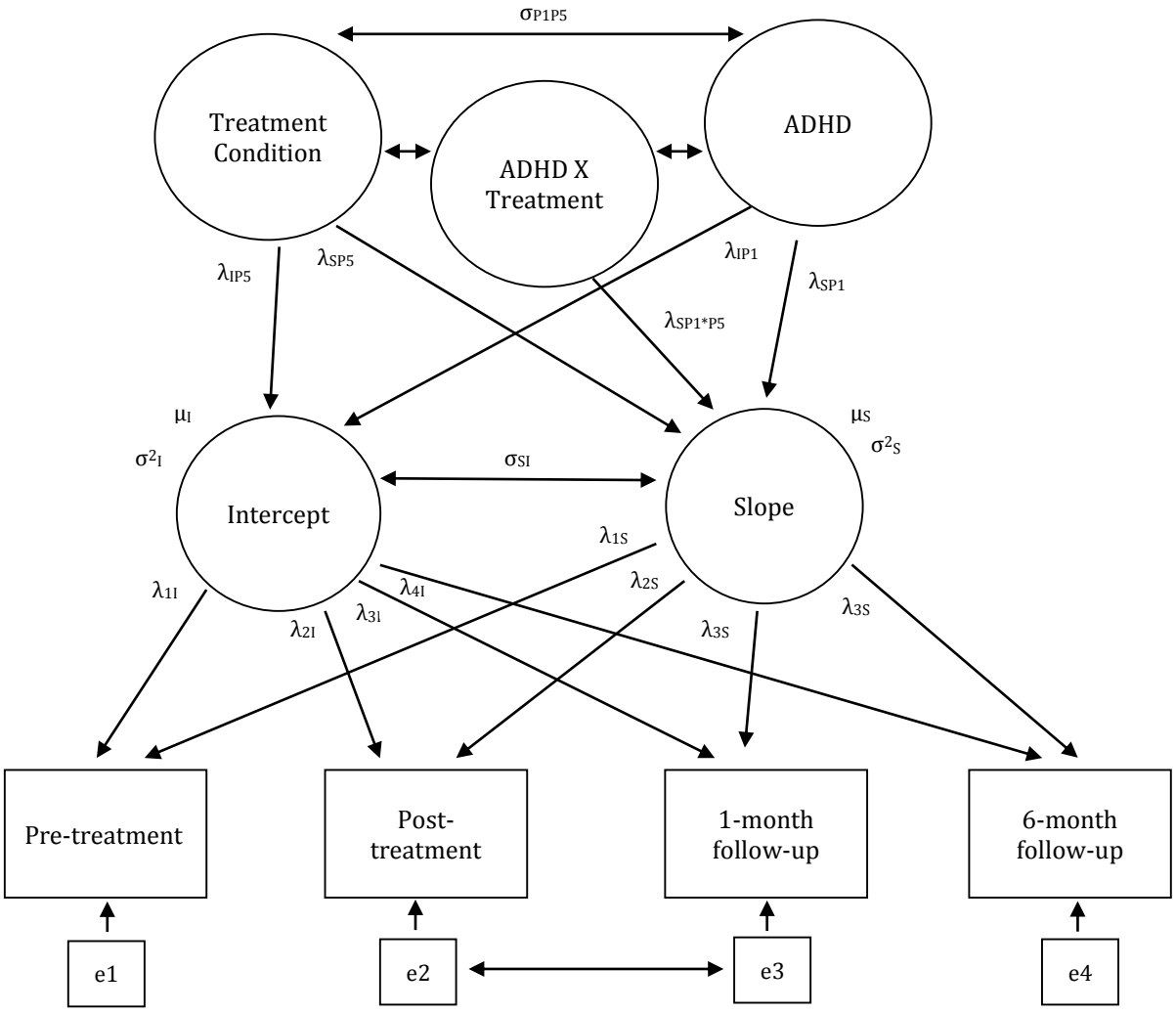


*Figure 7*. Growth curve model with ADHD as a moderator of treatment outcomes at posttreatment and at the 1-month and 6-month follow ups.

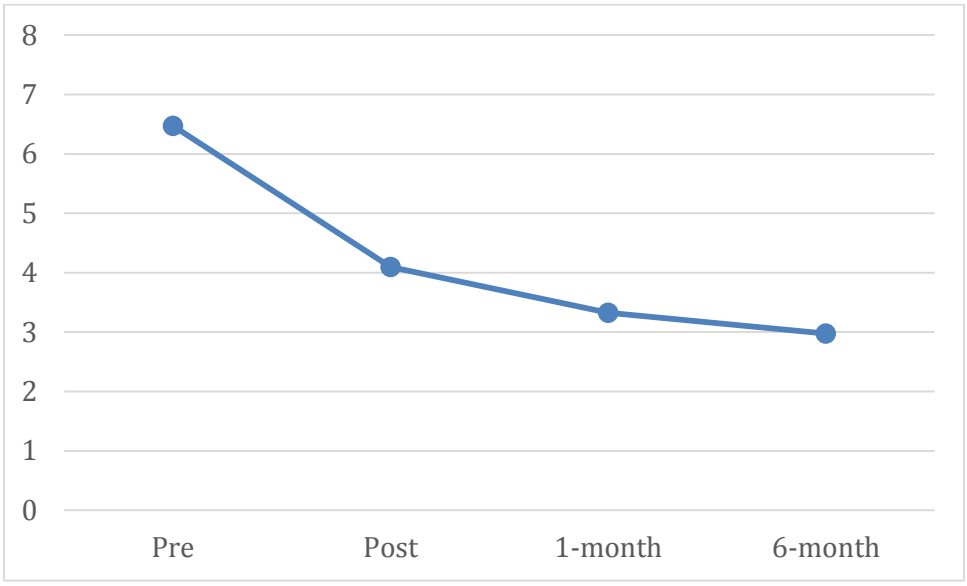


*Figure 8*. Clinician severity rating means for the treated specific phobia across time derived from the unconditional model.