



Childhood Anxiety Disorders: Developmental Risk Factors and Predictors of Treatment Response

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Childhood Anxiety Disorders:
Developmental Risk Factors and Predictors of Treatment Response

A dissertation presented

by

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to

The Department of Psychology

in partial fulfillment of the requirements

for the degree of

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in the subject of

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Childhood Anxiety Disorders:

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Abstract

Cognitive Behavioral Therapy (CBT) is the evidence-based treatment of choice for childhood anxiety disorders. Its blend of cognitive and behavioral coping strategies for anxiety management has more empirical support than any other intervention approach. Yet even after receiving CBT, more than 40% of anxious children still meet criteria for their anxiety disorder. Research is needed to identify factors associated with treatment response and non-response, and ultimately to inform treatment improvement. Three studies, reflecting this broad objective, focus on factors that may relate to child treatment response—environmental, biological, and cognitive factors suggested by theoretical models of anxiety and potentially relevant to treatment effectiveness research. **Study 1** examined whether parental anxiety is a negative predictor, and child perception of control a positive predictor, of treatment outcome in CBT within a randomized controlled trial for childhood anxiety disorders. We found that parental anxiety and child perception of control were not associated with treatment outcomes in the CBT or Usual Care treatment conditions with the exception of child perceived social control. In addition, parental anxiety levels did not change from pre- to post-treatment but child perceived control increased from pre- to post-treatment in response to both CBT and Usual Care. **Study 2** examined biological stress response in the context of exposure, the treatment component widely regarded as the heart of CBT for anxiety. Analyses of salivary analytes focused on activation of biological systems implicated in the social stress response (i.e., the hypothalamic-pituitary-adrenal axis and the autonomic nervous system) in children with Social Anxiety Disorder and

age-matched non-anxious controls, and we found that socially anxious children do not exhibit abnormally elevated biological reactivity. The study also tested whether heightened physiological arousal facilitates habituation and fear extinction, and we found that children who experienced greater biological activation over the course of a graduated exposure intervention appeared to benefit most. Finally, we found that subjective reports of heightened anxiety did not correspond to objective levels of biological arousal, suggesting that social anxiety may be associated with excessive self-monitoring and hypersensitivity to normative physiological response to stress and anxiety rather than biological dysregulation. **Study 3** examined whether socially anxious children exhibit social skills deficits and/or negative cognitive appraisal biases in a social-evaluative speech task. We found that socially anxious children did not exhibit negative cognitive appraisal biases, but they did exhibit specific social skills deficits in comparison to age-matched non-anxious peers. Taken together, these studies shed light on child and parent characteristics that are positively and negatively associated with youth anxiety treatment outcome from a biopsychosocial perspective.

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Introduction

Anxiety disorders are among the most common psychological disorders in children and adolescents, with lifetime prevalence rates of 15% to 20% (Beesdo, Knappe, & Pine, 2009; Costello, Egger, & Angold, 2005) and particularly early age of onset (Kessler et al., 2005). Manifestations of anxiety symptoms include emotional and physiological arousal, negatively distorted cognitive appraisals, and behavioral avoidance of feared stimuli. For example, a child with test-taking anxiety may be afraid of failing a test, and worry that the bad grade will lead to his teacher thinking he is stupid and his parents being disappointed in him, and to a ruined academic future. As the child prepares for school in the morning, his anxiety may increase along with physiological arousal (e.g., racing heartbeat) in response to the perceived threat. Feeling overwhelmed, the child may refuse to go to school. Following this behavioral avoidance, the child's anxious arousal declines, reinforcing the avoidance and thus helping to sustain the anxious response in the long run.

Anxiety disorders are associated with academic failure, low self-esteem, poor social relationships, and—in adolescence and beyond—suicidal ideation, suicide attempts, and comorbid depression and substance use disorders (Kendall & Ollendick, 2004; Muroff & Ross, 2011; Wood & McLeod, 2008). One of the most important advances in the field of child anxiety disorders has been the establishment of effective treatment in the form of Cognitive Behavioral Therapy (CBT) (Silverman, Pina, & Viswesvaran, 2008). CBT is a short-term intervention focused on anxiety management skills training and coping strategies (e.g., cognitive restructuring, problem-solving, exposures) (Kendall, Robin, Hedtke, & Suveg; 2005). CBT has more extensive empirical support than any other treatment for child and adolescent anxiety (Silverman et al., 2008; Weisz, Ng, Rutt, Lau, & Masland, 2013).

Despite the advances in child anxiety treatment, and the success of CBT in particular, up to 50% of anxious children still exhibit significant symptoms after treatment (Ginsburg & Schlossberg, 2002), and more than 40% fail to recover from their anxiety disorder (Cartwright-Hatton, Roberts, Chitsabesan, Fothergill, & Harrington, 2004; Kendall, Settapani, & Cummings, 2012). Given the pervasiveness of and severe functional impairments associated with anxiety disorders, research has targeted its risk and maintenance factors, with the ultimate goal of making prevention and treatment efforts more effective (Marques, Pereira, Barros, & Muris, 2013). As no single mechanism accounts for the development and maintenance of anxiety disorders, research has explored a complex set of risk factors implicated in theoretical models of anxiety disorders, including biological factors (e.g., physiological stress reactivity and dysregulation), cognitive aspects (e.g., information-processing biases), and environmental influences (e.g., parenting and family environment) in order to provide a more complete picture of anxiety in childhood (Rapee et al., 2009). Furthermore, expanding our knowledge of factors associated with treatment response may help us identify individuals who are more likely to benefit from CBT, identify others who may need alternative approaches, and inform efforts to tailor and personalize treatment for specific clients (Kazdin & Kendall, 1998; March & Curry, 1998).

My dissertation studies are designed to provide distinct and complementary perspectives from core domains implicated in theoretical models of anxiety disorders (including environmental, biological, and cognitive factors), with the ultimate goal of informing treatment effectiveness research. **Study 1** tests whether parental anxiety is a negative predictor, and whether child perception of control (over emotional experiences and environmental stressors) is a positive predictor, of treatment outcome in a randomized controlled trial (RCT) comparing the

effectiveness of CBT to Usual Care (UC) for anxiety disorders in community mental health clinics. Therapists in the UC condition conducted treatment using the methods they typically would in everyday practice for clinically anxious children. The study examines associations between parental anxiety and child perception of control, on the one hand, and treatment outcome, on the other, in the context of CBT (compared to UC). Client diagnoses included Generalized Anxiety Disorder, Separation Anxiety Disorder, Social Anxiety Disorder, and Specific Phobia, consistent with the multi-anxiety disorder samples of many child RCTs (e.g., Norton & Price, 2007; Rapee, Schniering, & Hudson, 2009; Silverman et al., 2008). In addition to furthering knowledge of conditions under which CBT may be more effective (e.g., whether the client characteristic of perceived control matters), study findings could also suggest ways to improve upon standard CBT (e.g., whether it might be helpful to include parental anxiety management as a treatment component).

Including multiple anxiety disorders within child anxiety RCTs is appropriate for a number of reasons—for example, the high level of comorbidity among anxiety disorders in childhood (see e.g., Benjamin, Costello, & Warren, 1990; Kashani & Orvaschel, 1988; Last, Hersen, Kazdin, & Francis, 1987)—but the multi-disorder focus may have limited our opportunity to understand treatment effects for specific disorders (Davis III, May, & Whiting, 2011). Although there are some shared clinical characteristics among anxiety disorders such as excessive fearfulness, unwanted physiological arousal, and behavioral avoidance of feared stimuli (Beesdo et al., 2009), there are also distinct differences between specific anxiety disorders, such as the target of the individuals' fears, level of distress and impairment, emotion regulation (e.g., Turk, Heimberg, Luterek, Mennin, & Fresco, 2005), and cognitive problems

(e.g., Holaway, Heimberg, & Coles, 2006). Thus, a narrower diagnostic focus is adopted for Studies 2 and 3.

Studies 2 and 3 focus specifically on Social Anxiety Disorder (SAD), one of the most prevalent anxiety disorders among children, with an estimated lifetime prevalence rate of 12.1% (Ruscio et al., 2008). Epidemiological studies have found SAD to be the third-most prevalent psychological disorder after depression and alcohol abuse (Hidalgo et al., 2001; Schneier, 2006); it is associated with decreased quality of life, academic and occupational impairment, family dysfunction, and social withdrawal and isolation (Baldwin & Buis, 2004). SAD in particular may have distinctive cognitive and biological features that warrant separate research attention. For example, there is content specificity of cognitive appraisal biases in SAD individuals such that they are triggered by situations that involve social evaluation. SAD individuals are more likely to hold negative expectations for social interactions, make negative interpretations of ambiguous social cues, and overestimate the probability and cost associated with negative social interactions (Vassilopoulos & Banerjee, 2012; Wilson & Rapee, 2005). Additionally, physiological hyperarousal symptoms have been implicated as a key factor contributing to disorder maintenance in cognitive models of SAD (e.g., Clark & Wells, 1995; Rapee & Heimberg, 1997), and the hypothalamic-pituitary-adrenal (HPA) axis and the autonomic nervous system (ANS) have been studied extensively within the context of social-evaluative stress (Kramer et al., 2012).

Study 2 examines biological dysregulation in response to an acute psychological stressor in children with SAD. Children (ages 8-14) with SAD and age-matched non-anxious controls participated in a lab visit involving a public speaking exposure intervention. Exposure is commonly accepted as an integral component of CBT for anxiety disorders, in which anxious

individuals are systematically exposed to feared stimuli in order to facilitate habituation and fear extinction (Arch & Craske, 2009). A meta-analysis conducted by Deacon and Abramowitz (2004) suggests that exposure during CBT may constitute the primary active ingredient in the treatment of SAD. We collected saliva samples at standardized timepoints throughout the exposure intervention in order to assess biological response to the exposure. Analyses of salivary analytes focused on activation of the major biological systems implicated in the social stress response (i.e., the HPA axis and the ANS) in children with SAD as compared to age-matched non-anxious controls. In addition, the study examined whether individual differences in biological response to exposures in SAD children are related to intervention outcome.

Finally, **Study 3** explores theoretical models that have implicated both social skills deficits and negative interpretation biases in children with SAD (Spence, Donovan, & Brechman-Toussaint, 1999; Rapee & Heimberg, 1997). A subsample of SAD children and age-matched non-anxious controls from **Study 2** evaluated their own speech performance. Speech performance was also rated separately by the child's parent, and by external observers who were blind to study group. We examined whether SAD children (as compared to age-matched non-anxious controls) exhibit social skills deficits or negative self-evaluation biases regarding their own social skills in the public speaking task. We also examined whether parents of SAD children exhibit negative evaluation biases in regard to their child's social skills. Assessing the accuracy of SAD children's and their parents' perceptions of performance could have significant implications for the treatment of childhood SAD (e.g., whether social skills training or cognitive restructuring is more appropriate).

Together, these three studies investigate factors associated with anxiety in children that may relate to their responses to treatment. The findings may suggest new directions for research on CBT and other treatments for anxious children.

Study 1

Anxiety disorders are among the most prevalent psychological disorders in children and adolescents, and they have a particularly early age of onset (Kessler et al., 2005). Anxiety disorders are associated with serious distress and functional impairments, including comorbid depression and substance use disorders, suicidal ideation, and suicide attempts (Kendall & Ollendick, 2004). Cognitive Behavioral Therapy (CBT) has been shown to be the most effective Evidence-Based Treatment (EBT) for children with anxiety disorders (Silverman, Pina, & Viswesvaran, 2008). CBT is delivered primarily as an individual and child-focused treatment. Although research has supported the success of CBT, a substantial percentage of anxious children remain symptomatic after treatment. In fact, a systematic review showed that the overall remission rate (i.e., diagnosis-free at posttreatment) of child anxiety disorders after CBT was only 56.5% (Cartwright-Hatton, Roberts, Chitsabesan, Fothergill, & Harrington, 2004). Thus, it is important to identify parent and child characteristics associated with treatment response and nonresponse to standard CBT, as this will inform intervention research and potential treatment adaptations for nonresponders (Southam-Gerow, Kendall, & Weersing, 2001). This study examined parental anxiety and child perception of control as predictors of treatment response for child anxiety disorders.

Extensive research has shown that parents' own anxiety disorders increase the risk for anxiety disorders in their children (Beidel & Turner, 1997; Biederman et al., 2006; Ginsburg, Siqueland, Masia-Warner, & Hedtke, 2004). Studies show that 80% of anxious youths have a parent with an anxiety disorder (Silverman, Cerny, & Nelles, 1988). However, research on the effects of parental anxiety on child anxiety treatment outcome is limited, and previous findings are mixed. There is some evidence in support of parental anxiety as a negative predictor of child

anxiety treatment outcome. For example, Cobham, Dadds, and Spence (1998) found that after a program of individual child-focused CBT for anxiety disorders, only 39% of children with an anxious parent exhibited significant symptom improvement at posttreatment, compared to 82% of children without an anxious parent. Similarly, Gar and Hudson (2009) found parental anxiety to be a negative predictor of treatment outcome in a group CBT program for child anxiety disorders; only 28% of children with an anxious parent showed significant treatment gains compared to 58% of children without an anxious parent. However, the evidence is inconclusive as other CBT studies have found no relationship between parental anxiety and treatment outcome for child anxiety disorders (Berman, Weems, Silverman, & Kurtines, 2000; Crawford & Manassis, 2001; Southam-Gerow et al., 2001).

Parental anxiety has also been linked to maladaptive parenting practices such as overprotection, overinvolvement, and controlling behaviors, which may lead to a reduced sense of self-efficacy and perception of control in children (Hudson & Rapee, 2001; Siqueland, Kendall, & Steinberg, 1996). Lack of perceived control over stressful events and emotional experiences has been supported as a general psychological vulnerability risk factor for anxiety disorders (Barlow, 2004). Research has shown that diminished perception of control predicts greater severity of SAD (e.g., Hofmann, 2005), Generalized Anxiety Disorder (e.g., Cannon & Weems, 2010), and trait anxiety (e.g., Brown et al., 2004). A lack of perceived control may develop due to early childhood experiences such as a controlling family environment; this may lead the child to believe that the world is a dangerous place over which s/he has no control and ultimately result in the child's avoidance of real or perceived threat in the environment (Chorpita & Barlow, 1998; Hudson & Rapee, 2001). Although there is limited research examining perceived control during CBT, Muris, Mayer, den Adel, Roos, and van Wamelen (2009) found

that perception of control significantly increased in anxious children who successfully underwent CBT. Similar results were found in clinically anxious adults who underwent CBT (Gallagher et al., 2013).

Parental anxiety may have a particularly strong *negative* impact on CBT, a structured skills-based and goal-oriented intervention in which children are encouraged to actively think about their worries and fears, and approach and engage with feared stimuli. CBT, as compared to other talk therapies, may differentially and directly trigger parents' anxiety as their children are systematically challenged to engage with fearful and anxiety-provoking thoughts, experience negative emotions and somatic symptoms of fear and anxious arousal, and face feared situations (Barlow, 2004). Anxious parents may be fearful of and resistant to their child's engagement in CBT due to their own negative beliefs about the catastrophic consequences of anxiety, beliefs about their child's inability to cope with stress, or concerns that challenging their child to confront feared stimuli could damage the parent-child relationship (Ginsburg et al., 2004). Parental anxiety may also lead to treatment-interfering behaviors such as reinforcement of child avoidance (Burstein & Ginsburg, 2010). On the other hand, child perception of control may have a particularly strong *positive* impact on CBT in comparison to other treatment modalities. If a subset of anxious children perceive situations to be within their control, this can naturally be leveraged in a treatment modality that emphasizes building problem-solving skills to effectively engage with fearful and anxiety-provoking thoughts, stimuli, and situations (Muris et al., 2009).

Southam-Gerow et al. (2010) conducted a randomized controlled trial (RCT) comparing the effectiveness of CBT to Usual Care (UC) in the treatment of child anxiety disorders. Therapists in the CBT condition were trained to use Coping Cat (Kendall, Kane, Howard, & Siqueland, 1990), an evidence-based treatment program for child anxiety disorders. Therapists

in the UC group delivered the treatment approaches they regularly used and considered to be effective. UC drew from an eclectic range of therapeutic approaches, and was heavily psychodynamic. Southam-Gerow and colleagues (2010) found that children in both the CBT and UC conditions improved, and there were no significant differences between CBT and UC in regard to treatment outcome, duration, or cost. These results are fairly consistent with two meta-analyses of RCTs comparing child EBTs (many involving CBT) to UC that found only a modest mean effect size favoring EBTs over UC for a range of clinical problems (Weisz, Jensen-Doss, & Hawley, 2006); these findings were recently replicated in a second meta-analysis with a larger sample of studies (Weisz et al., 2013). Additionally, there were no significant group differences between EBT and UC outcome for many of the studies in these meta-analyses. This suggests the value of research investigating whether there are conditions under which EBTs such as CBT may be able to outperform UC.

We used data collected from the Southam-Gerow et al. (2010) RCT to determine the effects of parental anxiety and child perception of control on child anxiety treatment outcome in the CBT vs. UC groups. To our knowledge, no previous studies have assessed this relationship in UC alone or in any comparison of CBT with UC.

Study aim/research questions. The aim of the current study was to test whether specific theoretically significant child and parental factors are associated with treatment response. The study addressed the following research questions: (1) Is parental anxiety a negative predictor of child anxiety disorders treatment outcome? (2) Does parental anxiety have a greater negative association with child treatment outcome in the CBT group as compared to the UC group? (3) Is child perception of control a positive predictor of child anxiety disorders treatment outcome? (4) Does child perception of control have a greater positive association with child treatment outcome

in the CBT group as compared to the UC group? (5) Are parent and child expectations for treatment outcome related to parental anxiety and child perception of control?

Hypotheses

(1) We hypothesized that parental anxiety (assessed at pretreatment) would have a greater negative association with treatment outcome in the CBT group than the UC group. (2) We hypothesized that child perception of control (assessed at pretreatment) would have a greater positive association with treatment outcome in the CBT group than the UC group. We posited these hypotheses as a child's sense of perceived control should be associated with behavior consistent with the therapeutic goal of overcoming anxiety symptoms, whereas parental anxiety may model and reinforce a child's fear and avoidance; such child and parent characteristics are especially salient in CBT which necessitates active engagement with fearful thoughts, stimuli, and situations. The remaining research questions posed were exploratory.

Method

Sample

Participants were 48 children (27 girls, 21 boys) ages 8 to 15 ($M = 10.85$, $SD = 2.09$) enrolled in a RCT comparing the effectiveness of individual CBT to UC for anxiety disorders, with 24 children in the CBT group and 24 in the UC group. Inclusion criteria consisted of a primary anxiety disorder diagnosis of Generalized Anxiety Disorder, Separation Anxiety Disorder, Social Anxiety Disorder, or Specific Phobia. Comorbidity was prevalent, with participants meeting criteria for an average of 3.2 diagnoses at pretreatment (see diagnostic assessment procedures below). Exclusion criteria included diagnoses of pervasive developmental disorder, psychotic disorder, or mental retardation. 31.3% of the children self-

identified as Caucasian, 12.5% were African American, 27.1% were Hispanic/Latino, 10.4% were multi-ethnic, and 18.7% did not provide ethnicity information.

Measures

Assessments were administered prior to the start of therapy (pretreatment) and at the end of therapy (posttreatment), and the assessment battery consisted of various diagnostic and symptom measures described below.

Diagnostic Interview Schedule for Children Version 4.0 (DISC 4.0). The DISC 4.0 (Rubio-Stipec et al., 1996; Shaffer, Fisher, Lucas, Dulcan, & Schwab-Stone, 2000) is a commonly used structured diagnostic interview that has demonstrated good reliability and validity (Schwab-Stone et al., 1996; Shaffer, Fisher, Dulcan, & Davies; 1996). Study interviewers were trained on standardized DISC 4.0 administration, and were blind to study condition (CBT vs. UC). Parent and child interviews were administered separately, and responses were combined to generate DSM-IV diagnoses and symptom counts.

State-Trait Anxiety Inventory for Children-Trait Version (STAIC-T). The STAIC-T (Spielberger, 1973) is a commonly used 20-item child self-report scale that measures general trait anxiety and has demonstrated strong reliability and validity.

State-Trait Anxiety Inventory for Children-Trait Version (STAIC-P-T). The STAIC-P-T (Spielberger, 1973) is the parent-report version of the scale assessing the child's trait anxiety and has also demonstrated strong reliability and validity.

Child Behavior Checklist (CBCL). The CBCL (Achenbach, 1991) is a 118-item parent-report questionnaire supported by extensive psychometric data and is designed to assess child behavioral and emotional functioning.

Mood and Anxiety Symptom Questionnaire (MASQ). The MASQ (Watson, Clark, Weber, & Assenheimer, 1995a; Watson et al., 1995b) is a 90-item self-report questionnaire shown to have good psychometric properties (Reidy and Keogh, 1997; Keogh and Reidy, 2000). The MASQ is used to assess negative affect, positive affect, and somatic symptoms associated with anxiety and depression (Wardenaar et al., 2010). The MASQ was administered to parents. The MASQ Anxious Arousal scale, MASQ General Distress Anxious Symptoms scale, and a composite of the MASQ Anxious Arousal scale and MASQ General Distress Anxious Symptoms scale were used as the criterion measures of parental anxiety for this study.

Perceived Control Scale for Children (PCSC). The PCSC (Weisz et al., 1998; Weisz, Southam-Gerow, & McCarty, 2001) is a 24-item child self-report questionnaire used to assess perception of control over outcomes in academic, social, and behavioral contexts. The PCSC has demonstrated good reliability and validity (Weisz et al., 2001).

Parent-Reported Anxiety Factor and Child-Reported Anxiety Factor. An exploratory factor analysis was conducted by Southam-Gerow and colleagues (2010) to identify latent factors underlying the child- and caregiver-report symptom measures of child anxiety (Weisz, et al., 2009). Two factors were identified using maximum-likelihood estimation and scree test with an oblique Promax rotation (Hendrickson, & White, 1964). Factor 1, the Parent-Reported Anxiety Factor, represented all parent-report measures of child anxiety: the STAIC-T-P, DISC-P anxiety symptoms, and the CBCL Anxious-Depressed Narrowband Scale. Factor 2, the Child-Reported Anxiety Factor, represented all child self-report measures of anxiety: the STAIC-T and DISC-C anxiety symptoms. Southam-Gerow and colleagues (2010) computed a standardized pretreatment factor score and posttreatment factor score for each client (Kline, 2004). The

parent-reported anxiety factor and child-reported anxiety factor served as the treatment outcome measures of the current study, and separate sets of primary analyses were conducted for each.

Expectations of Therapy Outcome Scale (ETOS). Children and parents filled out the ETOS (Bonner & Everett, 1982, 1986) on their expectations for treatment at pretreatment (Sample item: “How helpful do you expect that therapy will be?” 1= not at all helpful, 9 = very helpful). ETOS scores have been found to significantly relate to information youths and parents receive pre-treatment and therapist expectations pre-treatment (Bonner & Everett, 1982, 1986).

See *Table 1* for study measurement model.

Table 1. Study measurement model.

Pre-Treatment	Post Treatment
CHILD	
DISC 4.0-C	DISC 4.0
STAIC-T	STAIC-T
PCSC	
ETOS-C	
PARENT	
DISC 4.0-P	DISC 4.0
STAIC-P-T	STAIC-P-T
CBCL	CBCL
MASQ	
ETOS-P	

Note. DISC 4.0 is the Diagnostic Interview Schedule for Children Version 4.0. STAIC-T is the State-Trait Anxiety Inventory for Children-Trait Version. PCSC is the Perceived Control Scale for Children. ETOS is the Expectations of Therapy Outcome Scale. CBCL is the Child Behavior Checklist. MASQ is the Mood and Anxiety Symptom Questionnaire.

Procedures

Recruitment. Participants were recruited for the study during routine client intake procedures in six community mental health clinics. Children were invited to participate if (a) they met diagnostic criteria for at least one anxiety disorder (i.e., Generalized Anxiety Disorder,

Separation Anxiety Disorder, Social Anxiety Disorder, Specific Phobia), and (b) anxiety was the treatment priority for the family.

Randomization of Children and Therapists. Child and therapist participants were assigned to the UC or CBT condition via block randomization (Friedman, Furberg, & DeMets, 1998; for further procedural details, see Southam-Gerow et al., 2010).

CBT Treatment Procedures. Therapists in the CBT condition were trained to use Coping Cat (Kendall, Kane, Howard, & Siqueland, 1990). The Coping Cat manualized treatment consists of 16 to 20 sessions and is centered around a FEAR acronym: Feeling frightened (identifying feelings of fear and anxiety and co-occurring somatic symptoms), Expecting bad things to happen (identifying automatic negative thoughts and cognitive restructuring), Actions that can help (planning and practicing imaginal and in-vivo exposures), and Rating and Reward (self-evaluation of how one is coping with feelings of anxiety, and rewards for progress made in treatment) (Kendall & Hedtke, 2006). For example, an imaginal exposure for a perfectionistic child with Generalized Anxiety Disorder who worries about getting a bad grade may consist of the child imagining and writing a detailed story about failing an upcoming exam (Kendall et al., 2005). An in-vivo exposure task, on the other hand, is one in which the child faces a feared stimulus in person. For example, an in-vivo exposure for a child with SAD may consist of a conversation with a child she just met or placing a food order at a counter service restaurant.

Usual Care Treatment Procedures. Therapists in the UC group delivered the treatment approaches they regularly used and considered to be effective. UC therapists used a range of treatment procedures, including Psychodynamic, Family, Client-Centered approaches, and to a limited extent general CBT (Southam-Gerow et al., 2010).

Results

Pre- to Post-Treatment Changes in Parental Anxiety

CBT. Paired sample *t*-tests showed there were no significant changes in parent-report of their own anxiety from pre-treatment to post-treatment for participants who received CBT on the MASQ General Distress Anxious Symptoms scale ($t(18) = .83, p = .41$), MASQ Anxious Arousal scale ($t(18) = -.44, p = .66$), and MASQ Anxiety Composite Score ($t(18) = .21, p = .84$).

See Figure 1.

UC. Paired sample *t*-tests showed there were no significant changes in parent-report of their own anxiety from pre-treatment to post-treatment for participants who received treatment as usual on the MASQ General Distress Anxious Symptoms scale ($t(18) = -.023, p = .98$), MASQ Anxious Arousal scale ($t(18) = 1.02, p = .32$), and MASQ Anxiety Composite Score ($t(18) = .57, p = .57$). *See Figure 1.*

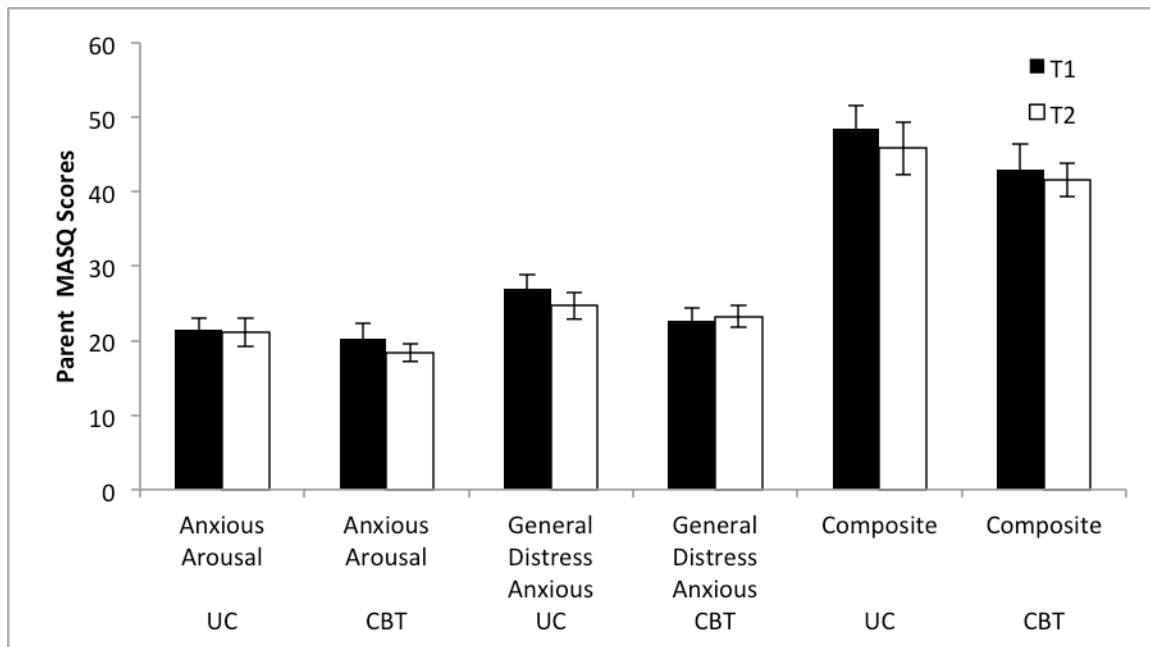


Figure 1. Parent mean scores on the MASQ Anxious Arousal scale, General Distress Anxious Symptoms scale, and Anxiety Composite pre- and post-treatment in both treatment conditions, CBT and UC.

Research Question #1: Is parental anxiety a negative predictor of child anxiety disorders treatment outcome?

Child-Reported Anxiety Factor. A multiple regression analysis showed that pretreatment parent-report of their own anxiety symptoms on the MASQ Anxiety Composite Score at T1 did not significantly predict improvements in child anxiety post-treatment scores (as represented by T2 child-reported anxiety factor) ($\beta = .069, t(30) = .30, p = .77, ns$). Similarly, the MASQ General Distress Anxious Symptoms scale at T1 did not significantly predict improvements in child anxiety post-treatment scores (as represented by T2 child-reported anxiety factor) ($\beta = .054, t(30) = .22, p = .82, ns$), nor did the MASQ Anxious Arousal scale ($\beta = .077, t(30) = .34, p = .73, ns$).

Parent-Reported Anxiety Factor. A multiple regression analysis showed that parent-report of their own anxiety symptoms on the MASQ Anxiety Composite Score at T1 did not significantly predict improvements in child anxiety post-treatment scores (as represented by T2 parent-reported anxiety factor) ($\beta = .25, t(30) = 1.15, p = .26, ns$). Similarly, the MASQ General Distress Anxious Symptoms scale at T1 did not significantly predict improvements in child anxiety post-treatment scores (as represented by T2 parent-reported anxiety factor) ($\beta = .35, t(30) = 1.57, p = .13, ns$), nor did the MASQ Anxious Arousal scale ($\beta = .14, t(30) = .66, p = .51, ns$).

Research Question #2: Does parental anxiety have a greater negative association with child treatment outcome in the CBT group as compared to the UC group?

Child-Reported Anxiety Factor. The aforementioned regression analyses also showed that there were no significant interaction effects of treatment condition x T1 MASQ Parent Anxiety Composite scores in predicting improvements on the T2 child-reported anxiety factor ($\beta = .062,$

$t(30) = .27, p = .79, ns$). Similarly, there were no significant interaction effects of treatment condition x T1 MASQ General Distress Anxious Symptoms scale or treatment condition x T1 MASQ Anxious Arousal scale on the T2 child-reported anxiety factor.

Parent-Reported Anxiety Factor. The aforementioned regression analyses also showed that there were no significant interaction effects of treatment condition x T1 MASQ Anxiety Composite Scores on the T2 parent-reported anxiety factor ($\beta = -.04, t(30) = -.19, p = .85, ns$). Similarly, there were no significant interaction effects of treatment condition x T1 MASQ General Distress Anxious Symptoms scale or treatment condition x T1 MASQ Anxious Arousal scale on the T2 parent-reported anxiety factor.

Pre- to Post-Treatment Changes in Child Perceived Control

CBT. Paired sample *t*-tests showed there was a significant increase in child perceived control from pre-treatment to post-treatment for participants who received CBT on the PCSC Total Score ($t(14) = -2.14, p = .05$), and the PCSC Social Subscale ($t(14) = -3.35, p = .005$). However, there were no significant changes in PCSC Academic Subscale ($t(14) = -1.71, p = .11$) and PCSC Behavioral Subscale ($t(14) = -.34, p = .74$) from pre-treatment to post-treatment. *See Figure 2.*

UC. Paired sample *t*-tests showed there was a significant increase in child perceived control from pre-treatment to post-treatment for participants who received UC on the PCSC Total Score ($t(13) = -2.35, p = .035$). There was a marginal increase in the PCSC Social Subscale ($t(13) = -1.79, p = .096$). However, there were no significant changes in PCSC Academic Subscale ($t(13) = -1.36, p = .20$) and PCSC Behavioral Subscale ($t(13) = -.88, p = .40$) from pre-treatment to post-treatment. *See Figure 2.*

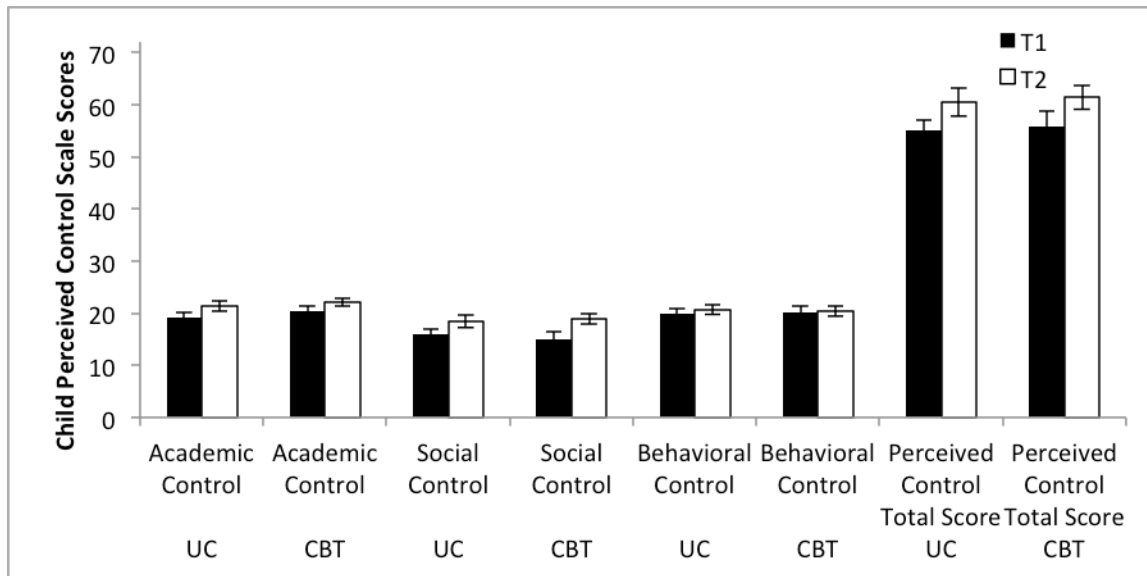


Figure 2. Child mean scores of perceived control on the Child Perceived Control Scale Total score, and on the academic, social, and behavioral subscales pre- and post-treatment in both treatment conditions, CBT and UC.

Research Question #3: Is child perception of control a positive predictor of child anxiety disorders treatment outcome?

Child-Reported Anxiety Factor. A multiple regression analysis showed that child perceived control on the PCSC Total Score at T1 did not significantly predict improvements in child anxiety post-treatment scores (as represented by T2 child-reported anxiety factor) ($\beta = -.28$, $t(24) = -.92$, $p = .37$). Similarly, the PCSC Academic Subscale at T1 did not significantly predict improvements in child anxiety post-treatment scores (as represented by T2 child-reported anxiety factor) ($\beta = -.038$, $t(24) = -.17$, $p = .87$), nor did the PCSC Behavioral Subscale ($\beta = -.026$, $t(24) = -.098$, $p = .92$) or the PCSC Social Subscale ($\beta = -.38$, $t(24) = -1.14$, $p = .16$).

Parent-Reported Anxiety Factor. A multiple regression analysis showed that child perceived control on the PCSC Total Score at T1 did not significantly predict improvements in child anxiety post-treatment scores (as represented by T2 parent-reported anxiety factor) ($\beta = .11$, $t(24) = .44$, $p = .66$). Similarly, the PCSC Academic Subscale at T1 did not significantly

predict improvements in child anxiety post-treatment scores (as represented by T2 parent-reported anxiety factor) ($\beta = -.082, t(24) = -.41, p = .68$), nor did the PCSC Behavioral Subscale ($\beta = -.065, t(24) = -.29, p = .78$).

A multiple regression analysis showed a marginal trend of child perceived control on the PCSC Social Subscale at T1 negatively predicting improvements in child anxiety post-treatment scores (as represented by T2 parent-reported anxiety factor) ($\beta = .38, t(24) = 1.74, p = .095$); higher pre-treatment perceived control in social contexts predicted worse treatment outcomes.

Research Question #4: Does child perception of control have a greater positive association with child treatment outcome in the CBT group as compared to the UC group?

Child-Reported Anxiety Factor. The aforementioned regression analyses also showed that there were no significant interaction effects of treatment condition x T1 child perceived control total score in predicting improvements on the T2 child-reported anxiety factor ($\beta = -.023, t(24) = -.086, p = .93, ns$). Similarly, there were no significant interaction effects of treatment condition x T1 PCSC Academic Subscale, treatment condition x T1 PCSC Behavioral Subscale, or treatment condition x T1 PCSC Social Subscale on the T2 child-reported anxiety factor.

Parent-Reported Anxiety Factor. The aforementioned regression analyses also showed that there were no significant interaction effects of treatment condition x T1 child perceived control total score in predicting improvements on the T2 parent-reported anxiety factor ($\beta = .13, t(24) = .54, p = .59, ns$). Similarly, there were no significant interaction effects of treatment condition x T1 PCSC Academic Subscale, treatment condition x T1 PCSC Behavioral Subscale, or treatment condition x T1 PCSC Social Subscale on the T2 parent-reported anxiety factor.

Research Question #5: Are parent and child expectations for treatment outcome related to parental anxiety and child perception of control?

There was a marginal positive correlation between MASQ Anxiety Composite Score and parental expectations for therapy outcome at pre-treatment, such that higher parental expectations for therapy outcome were associated with higher parental anxiety at pre-treatment, $r = .26, p = .084$. There was no significant correlation between the MASQ Anxiety Composite Score and child expectations for therapy outcome at pre-treatment, $r = .071, p = .64, ns$.

There was no significant correlation between child perceived control on the PCSC Total Score and parental expectations for therapy outcome at pre-treatment, $r = .067, p = .69$. There was no significant correlation between child perceived control on the PCSC Total Score and child expectations for therapy outcome at pre-treatment, $r = -.053, p = .76$.

Discussion

Anxiety disorders are the most common psychological disorders among children and adolescents, and are associated with severe functional impairment and a myriad of adverse mental health problems including comorbid mood disorders and substance use disorders (Higa-McMillan, Francis, Rith-Najarian, & Chorpita, 2015). The high prevalence rates and significant distress and impairment associated with anxiety disorders emphasize a need to focus on effective interventions. As the EBT outcome literature for youth anxiety disorders continues to expand, an important current and future direction of research is to examine parent and child characteristics that are associated with treatment outcomes. Our study focused on parental anxiety and child perception of control as candidate predictors of treatment outcome in CBT and UC.

Although research has shown that parents' anxiety disorders increase the risk for anxiety disorders in their children, research on the effects of parental anxiety on child anxiety treatment outcome is limited. We found that parental anxiety did not change over the course of treatment for youth anxiety in the CBT or UC treatment conditions. We also found that parental anxiety at

pre-treatment was not associated with youth anxiety treatment outcomes in CBT or UC.

Additionally, treatment condition did not have a differential effect on the relationship between parental anxiety and treatment outcome. Previous CBT studies showed mixed findings with some support for parental anxiety as a negative predictor of treatment outcome (e.g., Gar & Hudson, 2009) while others showed no relationship (e.g., Berman et al., 2000; Southam-Gerow et al., 2001). Our findings were consistent with the body of literature that found no relationship between parental anxiety and youth anxiety disorder treatment outcome over the course of CBT.

In a meta-analysis conducted in 2005, Barmish and Kendall reported that treatments with parent-involvement showed larger effect sizes than child-alone treatments. However, there have only been 9 CBT outcome trials with anxious youths that included parents as co-clients in treatment (i.e., at least four sessions with parent-involvement). Due to the limited number of parent-involvement outcome trials and variability across studies in parent treatment content (e.g., discouraging parent reinforcement of child's anxious behavior, teaching parents to model appropriate behaviors, reducing family conflict), Barmish and Kendall (2005) suggest that "the clearest and safest conclusion is that additional comparative research is needed and that the acceptance of either approach as superior is not yet justified" (p. 579). Our findings suggest that although parental anxiety is implicated in the etiology of childhood anxiety disorders, it may not play a very prominent role in the effects of child treatment and may not serve as a barrier to successful youth anxiety disorders treatment. In fact, higher parental anxiety at pre-treatment was associated with greater expectations for therapy.

Previous research has also shown that lower perceived control predicts higher severity of SAD and other anxiety symptoms (Hofmann, 2005; Cannon & Weems, 2010). Some evidence suggests that low perceived control may contribute to anxiety, as children's perception that they

lack control over life circumstances (Weems & Silverman, 2006) leads to behavioral avoidance of real or perceived environmental threat (Chorpita & Barlow, 1998; Hudson & Rapee, 2001). We found that child perceived control, and particularly in social contexts, increased in response to CBT. This is consistent with a previous study that found that perceived control increased in anxious children who received CBT (Gallagher et al., 2013). However, the improvements in child perceived control were not limited to CBT and were also found for UC. Thus, fostering increased child perceived control may not be specific to CBT but may instead be a byproduct of a course of treatment for youth anxiety across multiple treatment approaches.

Most domains of child perceived control at pre-treatment were not associated with youth anxiety treatment outcomes in CBT or UC. Additionally, treatment condition did not have a differential effect on the relationship between child perceived control and treatment outcome. Unexpectedly, lower pre-treatment perceived social control predicted better treatment outcomes in CBT and UC. One possible interpretation is that a low level of initial perceived control may motivate children to work for change through therapy, whereas children who perceive higher levels of control are less motivated to change. In a partially related perspective, Weems & Silverman (2006) suggest that internal locus of control (i.e., attributing life circumstances to personal skills and characteristics) may not be a universally adaptive cognitive style, and that an external locus of control (i.e., attributing life circumstances to external factors outside of one's control) may at times represent an accurate assessment of environmental circumstances. They posit that this accurate assessment may then lead to adaptive responses such as practicing acceptance, or actively seeking a different environment.

There were several limitations of the current study. Firstly, we did not examine parenting behaviors that are often implicated in the etiology of youth anxiety, such as parental overcontrol

(Adam, Gunnar, & Tanaka 2004), negativity/criticism (Hudson & Rapee, 2001), and lack of warmth (Whaley, Pinto, & Sigman, 1999) and such behaviors' association with parental anxiety. Secondly, we used a child self-report measure of perceived control but did not ask parents to evaluate their children's perceived control; parents might have provided a distinct perspective. Thirdly, we did not assess for parent psychopathology or anxiety disorder diagnosis and therefore do not know whether and which parents in the study exhibited clinical levels of anxiety. Finally, the small sample size limits our ability to detect effects.

We recommend the following directions for future research. First, in addition to the identification of treatment predictors that are specific to CBT, it may be useful to expand our search to child and parent characteristics that are associated with a successful treatment intervention regardless of treatment approach. Secondly, it may be helpful to identify treatment processes that are effective in UC and capitalize on them in EBTs given the comparable treatment benefits of both approaches. Third, parent-child behavioral observation tasks may help shed light on parental anxiety and child perception of control processes in the context of treatment, in ways that are not subject to the limitations of self- and collateral-report measures.

Study 2

Social Anxiety Disorder (SAD) is one of the most prevalent psychological disorders among children, with an estimated lifetime prevalence rate of 12.1% (Ruscio et al., 2008). SAD is characterized by an intense fear of embarrassment, humiliation, and negative evaluation, and avoidance of social situations in which these might occur (e.g., public speaking; DSM-IV-TR¹; American Psychiatric Association, 2000). In children, SAD is associated with social isolation, academic impairment and truancy, and the development of comorbid depression and substance abuse (Beidel, Turner, & Morris, 1999). The disorder typically follows a chronic course if left untreated, seriously undermining children's social and emotional development (Kessler et al., 2005). This highlights the importance of effective early intervention.

Cognitive Behavioral Therapy (CBT) has been established as the most effective evidence-based treatment for children with anxiety disorders (Silverman, Pina, & Viswesvaran, 2008), and is delivered primarily as an individual child-focused treatment. An essential component of CBT for anxiety is exposure, in which a therapist coaches the client to engage with a feared stimulus in a systematic way and tolerate the anxious arousal until it diminishes via habituation (Kendall, Robin, Hedtke, & Suveg, 2005). According to Foa and Kozak's (1986) emotional processing theory, anxious arousal while confronting the feared stimulus is required for habituation and fear extinction to occur. Engaging with the feared stimulus leads to the formation of competing nonfearful memories associated with the feared stimulus (i.e., counterconditioning of fear response). A meta-analysis suggests that exposure during CBT may serve as the main active ingredient in the treatment of SAD (Deacon & Abramowitz, 2004). For

¹ The American Psychiatric Association has recently published a fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders*, but some time will be required for standardized diagnostic interviews to be updated to match the DSM-V. As the diagnostic interview included in the current study (the ADIS—see below) is based on the DSM-IV-TR, so is the measurement model and conceptual framework for the study. Fortunately, the diagnostic criteria for SAD have not changed significantly with the introduction of the DSM-V.

SAD, an exposure task often used in research involves delivering a speech to an audience. Successful exposures that lead to a reduction in fear towards one type of social situation (e.g., public speaking) tend to generalize to other related fears (Kendall et al., 2005).

Although research has supported the efficacy of CBT and exposures, the treatment is not effective for all anxious children. In fact, up to 50% of anxious children still exhibit significant symptoms after treatment (Ginsburg & Schlossberg, 2002). Thus, an important challenge for research is to determine which kinds of children benefit from exposures and which kinds of children need an alternate approach. This fits a major theme of the National Institute of Mental Health (NIMH) Strategic Plan: supporting research to identify predictors of treatment response, to inform effective matching of clients to the specific treatment approaches most likely to be effective for them (NIMH, 2008). Consistent with this theme, we examined biological response to exposure therapy as a predictor of treatment response for children with SAD.

Biological Stress Response

Physiological hyperarousal symptoms play a prominent role in cognitive models of SAD (e.g., Clark & Wells, 1995; Rapee & Heimberg, 1997), and there are two major physiological systems implicated in human response to social stress: the hypothalamic-pituitary-adrenal (HPA) axis and the autonomic nervous system (ANS). Acute and chronic emotional and physical stress activates the HPA axis: the hypothalamus increases production of corticotropin-releasing hormone, which triggers the pituitary release of adrenocorticotropin hormone, and in turn generates increased cortisol production in the adrenal cortex (Axelrod & Reisine, 1984). Cortisol released from the adrenal cortex binds to glucocorticoid receptor-dense brain regions including the hippocampus, amygdala, and frontal lobe, which influences fear learning (Bentz, Michael, de Quervain, & Wilhelm, 2010). The hypothalamus acts as a command center that

communicates with the rest of the body through the ANS. The ANS helps the body self-regulate and maintain homeostasis, and controls involuntary body functions such as respiration, blood pressure, and heart rate (Mendes, 2009).

Although theoretical models have implicated HPA axis dysregulation in the onset and maintenance of SAD, only a few studies have examined this process in samples diagnosed with SAD (de Kloet, Joels, & Holsboer, 2005). In one of only two studies to assess HPA axis reactivity in children with SAD, van West, Claes, Sulon, and Deboutte (2008) found significantly increased cortisol levels in children with SAD compared to non-anxious controls in response to a public speaking exposure. Kramer et al. (2012), however, found that children with SAD experienced similar increases in cortisol levels compared to non-anxious controls in response to social-evaluative tasks involving mental arithmetic and telling a story in front of an audience.

In a study conducted by Furlan, DeMartinis, Schweizer, Rickels, and Lucki (2001) comparing SAD adults to adult non-anxious controls for a public speaking exposure task, non-anxious controls ($n = 14$) experienced a 50% net increase (from baseline) in salivary cortisol levels in response to the exposure. SAD individuals showed dichotomous results, with a *cortisol increase* subgroup averaging a 92% increase in cortisol ($n = 7$), and a *cortisol decrease* subgroup averaging a 32% decline ($n = 11$). These findings suggest that there may be marked individual differences among individuals with SAD in their biological response to the exposure component of CBT. Moreover, there is preliminary evidence that these individual differences may relate to whether exposure works as intended in treatment—i.e., that cortisol may facilitate the consolidation of extinction learning during exposure therapy. de Quervain et al. (2011) found that administering cortisol to adults with Specific Phobia preceding exposure sessions facilitated

the extinction of phobic fear. If HPA axis activation during exposure facilitates habituation and fear extinction, then research assessing HPA axis reactivity during exposure may help guide prediction of treatment response, and ultimately efforts to personalize treatment.

More recently, studies have discovered that salivary alpha-amylase (sAA) serves as a noninvasive and accurate biological marker of ANS activity in the “next generation of biobehavioral research” (Granger, Kivlighan, El-Sheikh, Gordis, & Stroud, 2007). The ANS is involved in the “fight or flight” response to acute emotional and physical stress, and studies have shown significant sAA increases in response to the Trier Social Stress Test (TSST; Kirschbaum, Pirke, & Hellhammer, 1993), a standardized laboratory social stressor task involving giving a speech and performing mental arithmetic in front of an audience (Nater et al., 2005). Few sAA studies have been conducted with clinical populations, although ANS dysregulation is potentially implicated in anxiety disorders. Individuals with Panic Disorder (Coupland, Wilson, Potokar, Bell, & Nutt, 2003) and Obsessive-Compulsive Disorder (Kawano et al., 2013) have been found to exhibit heightened ANS activity. However, in the only study to assess ANS reactivity in children with SAD, Kramer et al. (2012) found that children with SAD experienced similar increases in sAA levels compared to non-anxious controls in response to social-evaluative tasks involving finishing a story and mental arithmetic. More research with this promising new analyte is warranted.

Cognitive models of SAD (e.g., Clark & Wells, 1995; Rapee & Heimberg, 1997) have proposed that when individuals with SAD experience somatic sensations of anxious arousal (e.g., blushing, sweating, racing heart), they become even more anxious because others may notice their anxiety. However, it is unclear whether individuals with SAD experience abnormally elevated levels of physiological arousal or if this impression is an effect of self-monitoring and

catastrophic interpretations of somatic symptoms (Kramer et al., 2012). Further research is needed to clarify the relationship between physiological reactivity and the subjective experience of anxiety in children with SAD.

Study aim/research questions. The aim of the current study was to examine HPA axis and ANS reactivity in children with SAD compared to non-anxious controls in response to psychosocial stress. In addition, individual differences in cortisol and sAA response during a single-session exposure was examined in relation to intervention outcome. The study addressed the following research questions: (1) Do children with SAD show a greater mean increase in cortisol and sAA in response to a public speaking exposure than non-anxious controls? (2) Among children with SAD, do those who exhibit a greater physiological response (higher levels of cortisol and sAA) to an acute social stressor benefit more from exposure? This research question was of special interest and based on findings by de Quervain et al. (2011) that suggest higher levels of physiological arousal may predict greater benefit from exposure therapy. However, as the previous study involved treatment for adults with a different anxiety disorder, there was insufficient empirical basis to justify a study hypothesis. (3) Does biological stress response correspond to subjective reports of anxiety? To our knowledge, no previous studies have examined the effects of HPA axis and ANS activation on treatment outcome among children with SAD. The current study may expand our understanding of the biological mechanisms underlying effective exposure therapy, and ultimately contribute to personalizing treatment through identification of those children most likely to benefit from exposure.

Method

Sample

68 children (22 boys, 46 girls) participated in the study, along with their parents. Child age ranged from 8-14 ($M = 10.96$ years, $SD = 2.04$). 36 were children with a SAD diagnosis, and 32 were age-matched healthy control children. The majority of participants identified as Caucasian (54.4%), 14.7% percent identified as Asian, 7.4% identified as Black/African American, and 14.7% identified as multiracial. Participants were recruited through community organizations (e.g., afterschool programs), local schools, hospitals, and online advertisements (e.g., Craigslist, parenting listservs). Exclusion criteria for child participants in the SAD group included current comorbid non-anxiety psychological disorder(s), and current psychotropic medication use.

Procedure

The study session consisted of a pre-stressor baseline period (75 minutes), public speaking exposure (20 minutes), and a post-stressor recovery period (40 minutes). Researchers strictly adhered to the timing of tasks as they were designed to capture the rise and fall of cortisol and sAA.

Baseline. During the baseline period, an advanced clinical psychology doctoral student administered the SAD module of the Anxiety Disorders Interview Schedule for DSM-IV: Child and Parent Versions (ADIS-IV-C/P; Silverman and Albano, 1996) separately to the child and parent. The parent and child also separately completed a set of baseline questionnaires about the child's psychological symptoms (see below).

Public speaking exposure. van West, Claes, Sulon, & Deboutte (2008) found that the most commonly endorsed feared situations of children with SAD, as reported on the ADIS-IV, were giving a report or reading aloud in front of the class, public performances, and speaking to new or unfamiliar people, all of which were elements of this study's public speaking exposure.

The study employed a child-adapted version of the Trier Social Stress Test (TSST; Kirschbaum et al., 1993), shortened in duration and with child-relevant speech topics. First, a clinical psychology graduate student experimenter briefly described the rationale for exposures to the parent and child. Then, the child and parent were given five minutes to prepare a 5-minute speech on one of five topics (family, favorite vacation, hobbies, school, or sports). Next, the child gave a 5-minute practice speech to one of three “committee members” they were told would judge their speech. Finally, the child gave a 5-minute speech in front of the full committee of three. The committee consisted of undergraduate lab assistants who maintained neutral affect and took notes on the child’s performance during the speech. If the child stopped talking before time was up (i.e., a 10-second pause), committee members gave the child standard prompts including “you still have some time left, please continue,” and “try to tell us something more about (topic).” At the end of the child’s speech, the committee left the room to deliberate and then returned to give the child positive feedback on speech performance, following the van West et al. (2008) study procedure. The positive feedback demarcated the end of the stressor so that the recovery period could begin.

Post-stressor recovery. During the post-stressor recovery period, the parent and child were given eight minutes to discuss the child’s cognitive and emotional experience during the speech. This was followed by an 8-minute cognitive-restructuring discussion between the child and graduate student, in which they examined the evidence for and against the child’s catastrophic thoughts and negative expectations for speech performance (e.g., freezing up and not being able to talk). Finally, the lab visit ended with a 20-minute resting period during which the parent and child watched a calming Planet Earth documentary in order to wind down from the stressor.

One month follow-up. For the SAD group only, follow-up questionnaires were administered to parents and children via a phone interview conducted one month after the lab visit to assess child anxiety symptoms post-study visit.

See Figure 3 for study procedures flow chart.

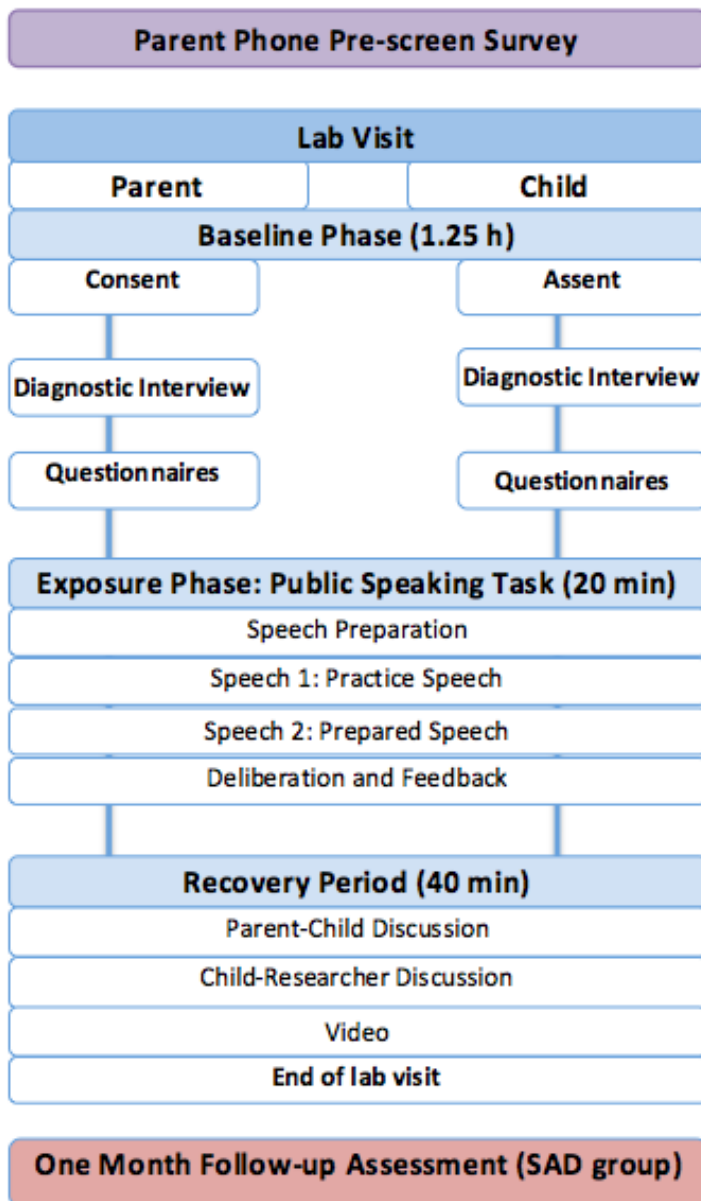


Figure 3. Study procedures flow chart. Lab visit procedures were consistent for the SAD and non-anxious control groups. One month follow-up phone calls were only conducted with the SAD group.

Assessment Measures

Anxiety Disorders Interview Schedule for DSM-IV: Child and Parent Versions (ADIS-IV-C/P). The ADIS-IV-C/P (Silverman & Albano, 1996) is a semi-structured diagnostic interview designed to assess anxiety and related disorders. The interview was separately administered to the parent and child, and has shown good to excellent inter-rater reliability and test-retest reliability (Silverman, Saavedra, & Pina, 2001). The SAD module assesses fear of 22 social situations, and a diagnosis is made taking into account both symptom count and impairment level.

Child Measures

Saliva assays. Child stress reactivity and regulation in response to an acute stressor was measured via changes in levels of salivary analytes (i.e., cortisol, sAA). Saliva samples were collected at seven timepoints, following the optimal “pre-pre-[task]-post-post-post-post” design for assessing changes in salivary analytes in response to an acute stressor (see Granger et al., 2012). Two saliva samples were collected pre-stressor (i.e., after study consent, immediately pre-stressor), one after speech preparation, and four were collected post-stressor (i.e., immediately post-stressor, and then 5-, 20-, and 40-minutes post-stressor). All study sessions were scheduled for the late afternoon, when HPA-axis activity is most stable (Kirschbaum & Hellhammer, 1994). Participants were asked not to eat or drink for at least one hour preceding the lab visit because food in saliva affects analyte levels. Saliva samples were collected from participants by asking them to hold oral swabs that absorb saliva in their mouths for two minutes at a time. The samples were then stored in Salivette collection devices in a -20°C freezer until they were sent to the Salimetrics Laboratory for analysis.

Subjective Units of Distress Scale (SUDS). SUDS ratings are commonly used to measure anxiety levels during exposures. SUDS ratings were administered to the child at 11 timepoints to measure change in anxiety levels throughout the study session. The scale ranges from “0-not at all anxious” to “8-very, very much anxious,” and a “feelings thermometer” was displayed as a visual aid for the ratings scale (Kendall et al., 2005). See Figure 4 for study timeline of saliva samples and SUDS administration.

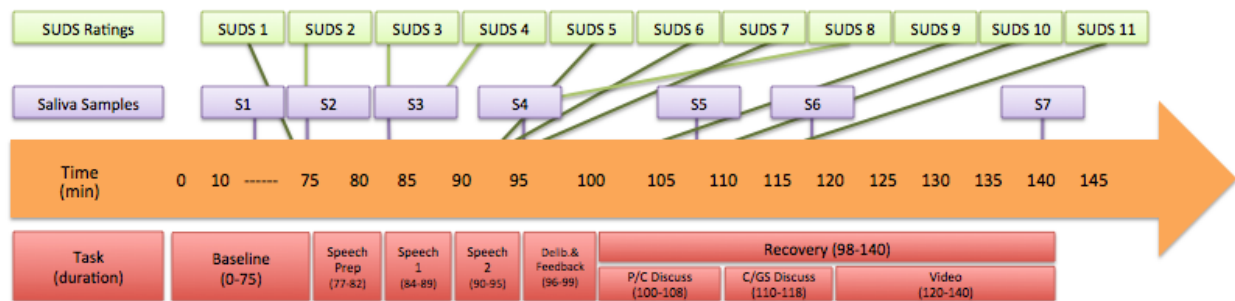


Figure 4. Study timeline for saliva samples and SUDS administration.

Spielberger State-Trait Anxiety Scale for Children (STAI-C). Child participants completed the STAI-C (Spielberger, 1973), a questionnaire that has demonstrated strong reliability and validity and is commonly used to measure anxiety in children. It consists of a state-version anxiety scale in which children rate how they are feeling at the time of questionnaire completion, and a trait-version anxiety scale in which children rate how they generally feel. The trait-version anxiety scale was administered along with a battery of child self-report questionnaires during the baseline period and the state-version was administered pre-stressor (immediately after the public speaking task was introduced) and-post-stressor (immediately after the speech in front of the full committee) in order to assess whether trait and state anxiety would be reflected in biological responses.

Revised Child Anxiety and Depression Scale (RCADS). The RCADS (Chorpita, Yim, Moffitt, Umemoto & Francis, 2000) is a 47-item measure that assesses child self-report of

anxiety and depression symptoms, including a Social Anxiety subscale. The RCADS has demonstrated good reliability and validity (Chorpita, Moffitt, & Gray, 2005). This measure was administered at baseline and one month follow-up.

Social Worries Questionnaire-Child Version (SWQ-C). The SWQ-C (Spence, 1995) is a 13-item measure that assesses child self-report of social anxiety symptoms, and was administered at baseline and one month follow-up. The SWQ-C has been used to screen for social anxiety symptoms and assess change in social anxiety symptoms in RCTs and pediatric care settings although its psychometric properties have not been extensively assessed (e.g., Bailey, Chavira, Stein, & Stein, 2006; Sofronoff, Attwood, & Hinton, 2005).

Parent Measures

Demographics questionnaire. Parent participants completed a demographics questionnaire at baseline providing background information about their marital status, ethnicity, level of education, employment status, and household income.

Tanner stages of pubertal development. Tanner stages of pubertal development (score range: 1-5) is a common and well-validated parent-report tool for measuring a child's current stage of pubertal development (Marshall & Tanner, 1969; 1970), and was administered at baseline. It consists of gender-appropriate depictions of secondary sex characteristics representing five stages of pubertal development for boys and girls. As a child's pubertal status affects hormone levels in saliva, it was important to control for the effects of pubertal stage in all data analyses involving salivary analytes.

Revised Child Anxiety and Depression Scale-Parent Version (RCADS-P). The RCADS-P (Ebesutani, Bernstein, Nakamura, Chorpita, & Weisz, 2010) is a 47-item measure that assesses parent report of child symptoms of anxiety and depression, including a Social Anxiety subscale.

The RCADS-P has demonstrated good reliability and validity (Ebesutani, 2011). This measure was administered at baseline and one month follow-up.

Social Worries Questionnaire-Parent Version (SWQ-P). The SWQ-P (Spence, 1995) is a 10-item measure that assesses parent report of child social anxiety symptoms, and was administered at baseline and one month follow-up. The SWQ-P has been used to screen for child social anxiety symptoms and assess change in child social anxiety symptoms in RCTs although its psychometric properties have not been extensively assessed (e.g., Bailey, Chavira, Stein, & Stein, 2006; Sofronoff, Attwood, & Hinton, 2005).

See *Table 2* for study measurement model.

Table 2. Study measurement model.

Baseline	Pre-TSST	Post-TSST	One Month Follow-Up
CHILD			
ADIS-IV-C			RCADS
STAIC-T	STAIC-S	STAIC-S	SWQ-C
RCADS			
SWQ-C			
PARENT			
ADIS-IV-P			RCADS-P
RCADS-P			SWQ-P
SWQ-P			
Family Demographics Questionnaire			
Tanner Stages of Pubertal Development			

Note. ADIS-IV is the Anxiety Disorders Interview Schedule for DSM-IV. STAIC-T is the State-Trait Anxiety Inventory for Children-Trait Version. RCADS is the Revised Child Anxiety and Depression Scale. SWQ is the Social Worries Questionnaire. SASC-R is the Social Anxiety Scale for Children-Revised. STAIC-S is the State-Trait Anxiety Inventory for Children-State Version, and was administered to the child pre- and post-Trier Social Stress Test (i.e., the child public speaking task).

Statistical Analyses

All statistical analyses were conducted using IBM SPSS Statistics 22.0. If there were three or less missing data points for a given participant’s trajectory of either sAA or salivary

cortisol over the course of the experiment, the missing data points were imputed using linear transformation. Of the 68 child participants, there was only one saliva data point missing for one participant due to an insufficient quantity of saliva provided during sampling, accounting for 0.2% of the total number of samples.

Before all statistical analyses were conducted, both salivary cortisol and sAA data were tested for normality and skewness using the Kolmogorov-Smirnov test. The results showed significant deviations from a normal distribution for both salivary cortisol and sAA. Therefore, all data points were natural log transformed (Engert et al., 2013).

Differences in sAA and salivary cortisol reactivity in response to the modified TSST between children with SAD and healthy controls were assessed with repeated-measures ANCOVAs, controlling for pubertal stage and age. Additionally, individual reactivity was calculated using Pruessner et al.'s (2003) area under the curve with respect to ground (AUC_G). This AUC_G computation allows us to quantify, using a single metric, changes in hormone concentration over the course of the entire exposure using every saliva timepoint. Higher AUC_G scores equate to greater hormone reactivity over the course of the exposure. In order to examine the recovery slopes for each participant, AUC_G was separately calculated for saliva sample timepoints 5-7 over the course of the recovery period only.

In addition to group categorization based on presence or absence of anxiety disorder diagnosis, we examined biological responder vs. non-responder subgroups (as found in Furlan et al., 2001, for SAD). Independent of anxiety disorder diagnosis, each participant was categorized as a cortisol responder or nonresponder based on criteria established by Petrowski et al. (2013) which required an increase of 2.5 nmol/L of salivary cortisol concentration above the individual's baseline in response to the TSST. The researchers established a contingent

secondary criterion for categorizing salivary cortisol responders as an increase in concentration of 10% above the baseline value (Granger et al., 2012). Baseline salivary cortisol level was calculated for each participant by selecting the minimum concentration from the baseline phase of the study (Allwood et al., 2011); the first saliva sample collected tended to be higher due to anxiety from being in a novel lab setting, but this was not always the case. Similarly the peak salivary cortisol value was calculated for each participant by selecting the maximum value of saliva samples four through six during the graduated public speaking exposure. The criterion for categorizing participants as sAA responders and non-responders consisted of an increase of 10% from the baseline phase to the peak concentration during the exposure phase of experimentation (Granger et al., 2006). In order to maintain consistency with salivary cortisol responder calculations, baseline and peak sAA measurements were calculated by selecting the minimum concentration from the first three saliva samples and the maximum concentration from saliva samples four through six, respectively.

To address physiological response as a predictor of intervention outcome, multiple regression analyses were conducted to examine whether children with SAD who exhibit a greater physiological response (i.e., higher levels of salivary cortisol and/or alpha amylase concentration, and therefore AUC_G) are more likely to benefit from exposure. AUC_G for salivary cortisol and alpha amylase were included as predictors in the same regression model, controlling for pubertal status and baseline RCADS symptoms at pre-exposure. Parent- and child-reported RCADS at one month follow-up were used to assess symptom improvement. The same set of multiple regression models was also tested using the recovery slopes of sAA and salivary cortisol (i.e., AUC_G for saliva sample timepoints 5-7 over the course of the recovery period). All statistical analyses reported in this paper in which we controlled for pubertal stage were also

tested with age as a covariate, with consistent results. We chose to report on analyses with pubertal stage as a covariate instead of age due to normative changes in HPA axis activity during puberty (Gunnar, Wewerka, Frenn, Long, & Griggs, 2009).

In order to determine whether biological stress response correlates with subjective reports of anxiety, simple bivariate correlations were calculated between salivary cortisol/sAA levels and child-reported STAI-state scores pre- and post-exposure for each corresponding saliva sample administration timepoint. Similarly, bivariate correlations were calculated between each saliva sample administration timepoint and their corresponding SUDS ratings. Significant positive correlations would suggest that subjective ratings of anxiety do correspond to levels of physiological arousal. While STAI-S and SUDS ratings represent real-time ratings of subjective distress, autonomic and HPA-axis reactivity experience a time lag from initial introduction of an anxiety-provoking stimuli until an increased analyte concentration is measurable in saliva. For sAA, peak concentration increase is exhibited approximately five minutes after initial exposure to anxiety-provoking stimuli, and for salivary cortisol approximately 15-20 minutes after initial exposure. *Figure 5* provides an example of hormone concentration spikes in response to the psychosocial stressor based on the aforementioned lag times in analyte production.

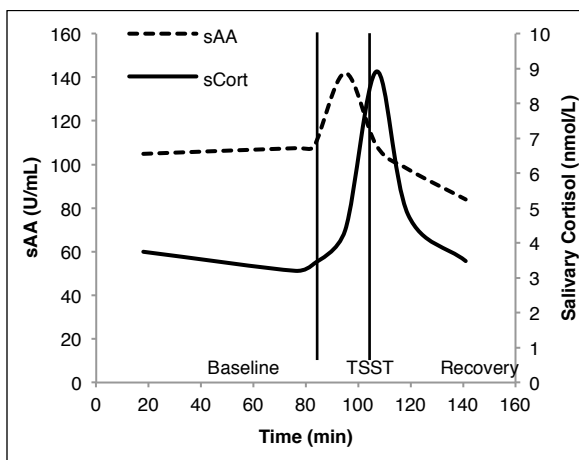


Figure 5. Illustrative example data meant to simulate the time lag between stimulus exposure and subsequent hormone concentration increase.

Additionally, one-way ANOVAs were conducted comparing the SAD group with healthy control group on anxiety as reported on the STAIC-T, the RCADS-C Total Anxiety Subscale, and on the STAIC-S immediately pre- and post-exposure. The preceding set of analyses was also conducted comparing salivary cortisol responders and non-responder groups regardless of diagnosis.

Chi-Square tests were used to measure categorical differences. For all analyses, the significance level was set at $\alpha = 0.05$ (two-tailed). It should also be noted that due to a few missing data points for a subset of the symptom-report questionnaires that were missing at random (i.e., parent accidentally skipped an item) and missing parent-reported pubertal stage for a few of the participants, statistical analyses reported have varying degrees of freedom.

Results

Autonomic and Hypothalamic-Pituitary Adrenal Responses to Psychosocial Stressor

Salivary cortisol. An ANCOVA revealed that there were no significant differences in baseline salivary cortisol concentration between children with SAD and healthy controls controlling for pubertal status ($F(1,66) = 0.031, ns$). For salivary cortisol reactivity, a repeated measures ANOVA revealed a significant main effect of time over the course of exposure when controlling for pubertal stage; the concentration of salivary cortisol increased in response to the stressor and subsequently fell during the recovery phase, $F(6,59) = 8.14, p < .001$, but there was no significant main effect of group ($F(1,59) = 1.51, ns$) nor was there an interaction of time x group, ($F(6, 59) = 1.24, ns$) when controlling for pubertal stage. Paired-sample *t*-tests revealed that salivary cortisol concentration was significantly higher during the exposure phase than both the baseline phase, $t(67) = -2.98, p = .004$, and recovery phase of the experiment, $t(67) = 6.49, p < .001$. An ANCOVA also showed no significant differences in recovery slopes (i.e., recovery

period AUC_G) between children with SAD and healthy controls controlling for pubertal stage ($F(1,66) = 1.95, ns$).

sAA. Similar to salivary cortisol concentration at baseline, an ANCOVA revealed that there were no significant differences in baseline sAA concentration between children with SAD and healthy controls ($F(1,66) = 0.16, ns$). For sAA reactivity, a repeated-measures ANOVA revealed that there was no significant main effect of time over the course of the exposure when controlling for pubertal stage ($F(1,59) = .65, ns$). This is unsurprising due to the restricted timeframe in which the action for sAA occurs; a paired sample *t*-test (as reported below) would better capture this relatively fast-acting analyte's rise and fall over the course of 5-10 minutes in response to an acute stressor. Similarly to salivary cortisol, there was also no significant main effect of group ($F(1,59) = .22, ns$), nor was there an interaction of time x group ($F(6,59) = .50, ns$) when controlling for pubertal stage. Paired-sample *t*-tests revealed that sAA concentration was significantly higher during the exposure phase than both the baseline phase ($t(67) = -3.74, p < .001$), and recovery phase of the experiment, $t(67) = 5.08, p < .001$. An ANCOVA also showed no significant differences in recovery slopes (i.e., recovery period AUC_G) between children with SAD and healthy controls controlling for pubertal stage ($F(1,66) = .67, ns$).

Figures 6 and 7 depict the similar levels of HPA axis and autonomic reactivity of children with SAD and healthy controls over the course of the experiment.

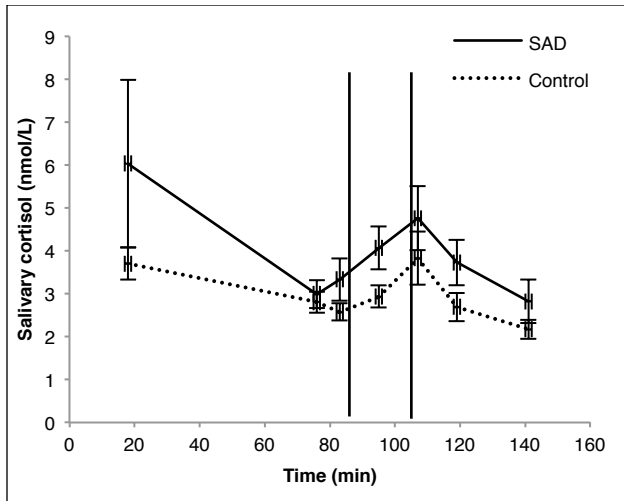


Figure 6. Means of salivary cortisol response to an adapted TSST for children with Social Anxiety Disorder and a healthy control group.

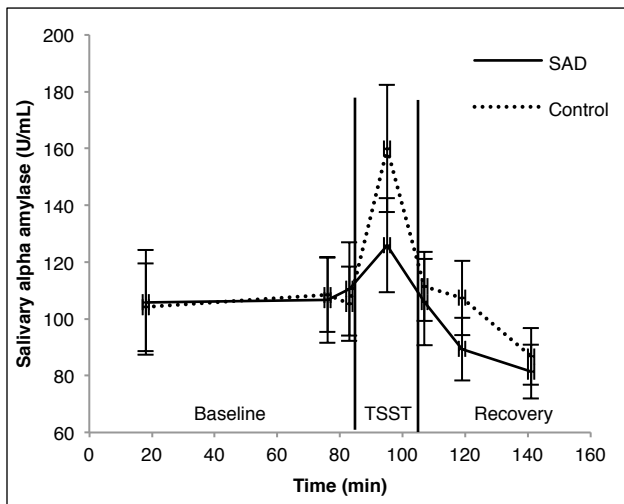


Figure 7. Means of salivary alpha amylase response to an adapted TSST for children with Social Anxiety Disorder and a healthy control group.

Although there were no significant group differences in salivary cortisol response when comparing the socially anxious and healthy control groups, chi-square analyses revealed that the percentage of SAD children categorized as salivary cortisol responders was significantly higher than the percentage of healthy control children categorized as salivary cortisol responders ($\chi^2(1, N=68) = 4.17, p < .05$). Within the SAD group, 15 of 36 participants (41.7%) were categorized as salivary cortisol responders, and in the control group, 6 out of 32 participants

(18.8%) were categorized as responders. *Figure 8* shows the pronounced difference in biological activation in salivary cortisol responders compared to biologically blunted nonresponders within the SAD group. *Figure 9* shows salivary cortisol response of cortisol responders and non-responders for both the SAD and healthy control groups. Interestingly, patterns of cortisol responder and non-responder profiles are indistinguishable between the SAD and healthy control groups.

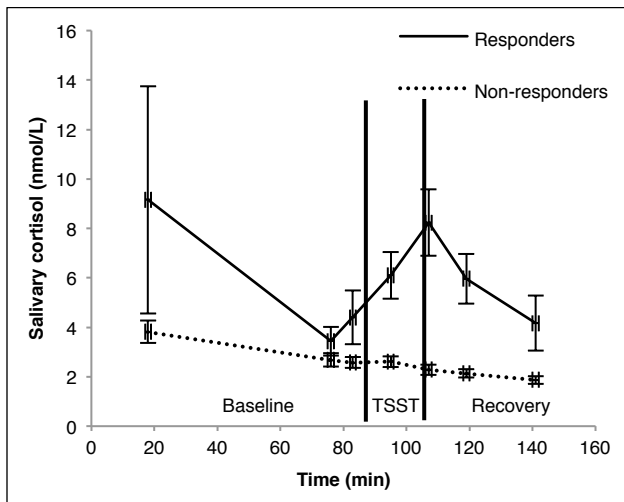


Figure 8. Means of salivary cortisol responders and non-responders to an adapted TSST for children with Social Anxiety Disorder.

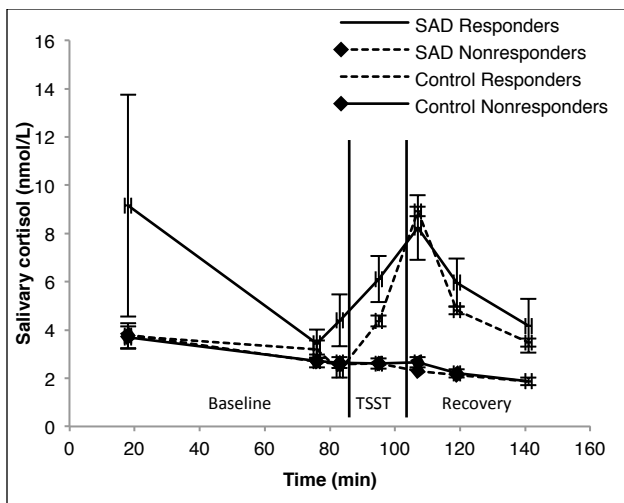


Figure 9. Salivary cortisol means of salivary cortisol responders and non-responders in response to an adapted TSST for children with Social Anxiety Disorder and a healthy control group.

Using the 10% increase in sAA concentration from baseline to peak criterion to categorize sAA response to exposure, there were no significant differences in sAA response status between experimental groups ($\chi^2(1, N=68) = .90, ns$). Within the SAD group, 34 of 36 (94%) participants were categorized as responders, and within the control group, 30 of 32 (94%) of participants were categorized as responders. Our results are consistent with previous sAA studies that have found that the majority of participants are categorized as responders using the established criterion (Granger et al., 2006). Due to the limited utility of responder categorization for sAA, analyses using this categorization were foregone. This suggests that we may need better methods of delineating responder-cutoffs that are appropriate for sAA given that this is still a newer analyte that is less well-researched than salivary cortisol.

Biological Reactivity as a Predictor of Symptom Improvement

Separation Anxiety

A multiple regression analysis showed that sAA ($\beta = -.33, t(28) = -3.29, p = .003$) and salivary cortisol ($\beta = -.26, t(28) = -2.56, p = .016$) reactivity (measured by AUC_G) positively and significantly predicted parent-reported symptom improvement at one month follow-up on the RCADS-P Separation Anxiety Scale, controlling for baseline symptoms and pubertal stage. Child salivary cortisol and alpha amylase also explained a significant proportion of variance in parent-reported child separation anxiety scores, $R^2 = .73, F(4,32) = 19.16, p < .001$. However, a second multiple regression analysis showed that neither sAA ($\beta = -.049, t(28) = -.40, ns$) nor salivary cortisol ($\beta = -.049, t(28) = -.40, ns$) reactivity (measured by AUC_G) significantly predicted child-reported symptom improvement at one month follow-up on the RCADS-C Separation Anxiety Scale, controlling for baseline symptoms and pubertal stage.

General Anxiety

A multiple regression analysis showed that sAA ($\beta = -.33, t(28) = -1.79, p = .084$) and salivary cortisol ($\beta = -.21, t(28) = -1.85, p = .075$) reactivity (measured by AUC_G) trended towards significance in positively predicting parent-reported symptom improvement at one month follow-up on the RCADS-P General Anxiety Scale, controlling for baseline symptoms and pubertal stage. However, a second multiple regression analysis showed that neither sAA ($\beta = .056, t(28) = .053, ns$) nor salivary cortisol ($\beta = -.077, t(28) = -.73, ns$) reactivity (measured by AUC_G) significantly predicted child-reported symptom improvement at one month follow-up on the RCADS-C General Anxiety Scale controlling for baseline symptoms and pubertal stage.

Depression

A multiple regression analysis showed that salivary cortisol ($\beta = -.24, t(28) = -2.36, p = .026$) reactivity (measured by AUC_G) positively and significantly predicted parent-reported symptom improvement at one month follow-up on the RCADS-P Depression Scale, controlling for baseline symptoms and pubertal stage, but alpha amylase reactivity did not ($\beta = -.05, t(28) = .53, ns$). Child salivary cortisol reactivity also explained a significant proportion of variance in parent-reported depression scores, $R^2 = .73, F(4,32) = 18.08, p < .001$. However, a second multiple regression analysis showed that neither sAA ($\beta = -.030, t(28) = -.26, ns$) nor salivary cortisol ($\beta = -.061, t(28) = -.52, ns$) reactivity (measured by AUC_G) significantly predicted child-reported symptom improvement at one month follow-up on the RCADS-C Depression Scale controlling for baseline symptoms and pubertal stage.

Social Phobia

A multiple regression analysis showed that neither sAA ($\beta = -.01, t(28) = -.06, ns$) nor salivary cortisol ($\beta = .22, t(28) = 1.66, ns$) reactivity (measured by AUC_G) significantly predicted parent-reported symptom improvement at one month follow-up on the RCADS-P Social Phobia

Scale controlling for baseline symptoms and pubertal stage. A second multiple regression analysis showed that sAA ($\beta = -.20$, $t(28) = -1.69$, $p = .10$) reactivity (measured by AUC_G) trended towards significance in positively predicting child-reported symptom improvement at one month follow-up on the RCADS-C Social Phobia Scale, controlling for baseline symptoms and pubertal stage, but salivary cortisol reactivity did not ($\beta = -.039$, $t(28) = -.33$, *ns*).

A multiple regression analysis also showed that neither sAA ($\beta = -.041$, $t(28) = -.43$, *ns*) nor salivary cortisol ($\beta = -.084$, $t(28) = -.87$, *ns*) reactivity (measured by AUC_G) significantly predicted parent-reported symptom improvement at one month follow-up on the SWQ-P controlling for baseline symptoms and pubertal stage. Similarly, an additional multiple regression analysis showed that neither sAA ($\beta = .030$, $t(28) = .18$, *ns*) nor salivary cortisol ($\beta = .098$, $t(28) = .59$, *ns*) reactivity (measured by AUC_G) significantly predicted child-reported symptom improvement at one month follow-up on the SWQ-C controlling for baseline symptoms and pubertal stage.

Total Anxiety

A multiple regression analysis showed that sAA ($\beta = -.22$, $t(28) = -1.89$, $p = .069$) reactivity (measured by AUC_G) trended towards significance in positively predicting parent-reported symptom improvement at one month follow-up on the RCADS-P Total Anxiety Scale, controlling for baseline symptoms and pubertal stage, but salivary cortisol reactivity did not ($\beta = -.14$, $t(28) = -1.20$, *ns*). A second multiple regression analysis also showed that neither sAA ($\beta = -.098$, $t(28) = -.64$, *ns*) nor salivary cortisol ($\beta = -.088$, $t(28) = -.56$, *ns*) reactivity (measured by AUC_G) significantly predicted child-reported symptom improvement at one month follow-up on the RCADS-C Total Anxiety Scale controlling for baseline symptoms and pubertal stage.

Biological Recovery Period Slopes as a Predictor of Symptom Improvement

We also examined recovery slopes as measured by recovery period AUC_G as a predictor of intervention outcome. A multiple regression analysis showed that sAA ($\beta = -.32, t(28) = -3.29, p = .004$) and salivary cortisol ($\beta = -.23, t(28) = -2.49, p = .033$) recovery slopes positively and significantly predicted parent-reported symptom improvement at one month follow-up on the RCADS-P Separation Anxiety Scale, controlling for baseline symptoms and pubertal stage. Child salivary cortisol and sAA also explained a significant proportion of variance in parent-reported separation anxiety scores, $R^2 = .73, F(4,32) = 16.82, p < .001$. A second multiple regression analysis showed that salivary cortisol ($\beta = -.23, t(28) = -2.32, p = .028$) recovery slopes positively and significantly predicted parent-reported symptom improvement at one month follow-up on the RCADS-P Depression Scale, controlling for baseline symptoms and pubertal stage, but sAA reactivity did not ($\beta = -.05, t(28) = .53, ns$). Child salivary cortisol reactivity also explained a significant proportion of variance in parent-reported depression scores, $R^2 = .73, F(4,32) = 18.23, p < .001$. These were consistent with the aforementioned AUC_G findings over the course of the exposure. None of the other multiple regressions revealed sAA or salivary cortisol recovery slopes to significantly predict symptom improvement.

Correspondence between Physiological and Subjective Stress Responses

The self-reported STAI-S administered pre-exposure did not correlate with its corresponding salivary alpha amylase timepoint measurement ($r = .11, p = .37, ns$) or its corresponding salivary cortisol timepoint measurement ($r = .19, p = .13, ns$) taking into account aforementioned lag times in analyte production. Similarly, the STAIC-S administered post-exposure did not correlate with its corresponding salivary alpha amylase timepoint measurement ($r = .09, p = .45, ns$) or salivary cortisol timepoint measurement ($r = .01, p = .95, ns$) taking into account aforementioned lag times in analyte production.

SUDS and salivary cortisol. During the baseline phase, salivary cortisol concentration at saliva sample timepoint 1 did not correlate with its corresponding SUDS rating ($r = -.068, p = .58, ns$), nor did salivary cortisol concentration at saliva sample timepoint 2 ($r = -.071, p = .56$). During the exposure phase, salivary cortisol concentration at saliva sample timepoint 3 ($r = .010, p = .94$) did not correlate with its corresponding SUDS rating, nor did salivary cortisol concentration at saliva sample timepoints 4 ($r = .045, p = .72$) or 5 ($r = .012, p = .92$). However, during the recovery phase, *lower* salivary cortisol concentration at saliva sample timepoint 6 significantly correlated with *higher* corresponding SUDS ratings ($r = -.26, p = .035$), and lower salivary cortisol concentration at saliva sample timepoint 7 trended towards significance in correlating with higher corresponding SUDS ratings ($r = -.21, p = .088$).

SUDS and sAA. During the baseline phase, sAA concentration at saliva sample timepoint 1 did not correlate with its corresponding SUDS rating ($r = -.084, p = .50$), nor did sAA concentration at saliva sample timepoint 2 ($r = .0040, p = .98$). During the exposure phase, sAA concentration at saliva sample timepoint 3 did not correlate with its corresponding SUDS rating ($r = -.044, p = .72$), nor did sAA concentration at saliva sample timepoints 4 ($r = -.011, p = .92$) or 5 ($r = -.014, p = .91$). During the recovery phase, sAA concentration at saliva sample timepoint 6 did not correlate with its corresponding SUDS rating ($r = -.0040, p = .98$), nor did sAA concentration at saliva sample timepoint 7 ($r = .097, p = .43$).

One-way ANOVAs showed that children with SAD demonstrate significantly higher trait anxiety than healthy controls on the STAIC-T ($F(1,66) = 13.93, p < .001$) and RCADS-C Total Anxiety Subscale ($F(1,66) = 20.10, p < .001$). One-way ANOVAs also showed that SAD children experienced significantly higher state anxiety pre-exposure ($F(1,66) = 5.65, p < .05$), and trended towards significance post-exposure on the STAIC-S ($F(1,66) = 3.75, p = .057$),

despite no significant differences between the social anxiety and healthy control groups in salivary cortisol or alpha amylase reactivity.

However, when individuals were grouped based on responder and non-responder status, one-way ANOVAs showed that salivary cortisol responders demonstrated significantly higher trait anxiety than non-responders on the RCADS-C Total Anxiety Subscale ($F(1,66) = 5.55, p < .05$), and trended towards significance on the STAIC-T ($F(1,66) = 3.75, p = .057$). Additional one-way ANOVAs showed that there were no significant differences between salivary cortisol responders and non-responders on the STAIC-S pre- ($F(1,66) = 3.00, ns$) or post-exposure ($F(1,66) = .24, ns$).

Discussion

Although physiological hyperarousal and biological dysregulation have been implicated in theoretical models of SAD, little is known about the neuroendocrine and nervous system response of children with SAD to acute psychosocial stressors (e.g., Clark & Wells, 1995; Rapee & Heimberg, 1997). The purpose of the current study was to examine biological dysregulation in children with SAD in response to psychosocial stress. In addition, individual differences in biological response profiles of cortisol and sAA were evaluated as predictors of intervention outcome.

Children with SAD and healthy control children exhibited similar baseline levels of sAA and salivary cortisol. Similarly, the psychosocial stressor produced a similar level of sAA and cortisol reactivity in youths with SAD as compared to children in the healthy control group. Our findings for sAA are consistent with findings from Kramer and colleagues' 2012 study, the only previous study to have assessed ANS reactivity in children with SAD. As sAA and salivary cortisol are well-established indicators of stress and anxious arousal, this suggests that the

anxiety experienced by children with SAD may be more cognitive than biological. However, a significantly greater number of children in the SAD group were categorized as cortisol responders (41.7%) as compared to the healthy control children (18.8%). This suggests that although children with SAD do not experience *abnormally* high elevations in physiological arousal overall, SAD youths are more likely to experience significantly elevated cortisol in response to an acute psychosocial stressor in comparison to their psychopathology-free peers. Healthy control children, on the other hand, are more likely to experience a physiologically blunted response to a psychosocial stressor. Similarly, Furlan and colleagues (2001) have also found a dichotomization of cortisol response patterns in socially phobic adults and age-matched healthy controls.

SAD youths exhibited individual differences in their biological response to an exposure intervention. However, there were mixed findings in whether such individual differences serve as a reliable predictor of intervention outcome. Heightened cortisol reactivity over the course of an exposure intervention predicted symptom improvement on parent-reported separation anxiety, generalized anxiety, and depression. In addition, heightened alpha amylase reactivity predicted symptom improvement on parent-reported separation anxiety, generalized anxiety, and total anxiety. Heightened alpha amylase reactivity also predicted symptom improvement on child-reported social anxiety. This suggests that exposures targeting social anxiety symptoms may generalize to other unrelated fears. Our findings lend some promising preliminary evidence that greater physiological response to exposure may facilitate habituation and fear extinction. As this is the first study to examine HPA axis and ANS reactivity as predictors of exposure intervention outcome for youths with SAD, further research is necessary to determine whether physiological arousal during exposure enhances the therapeutic effect of exposure.

Our study also helps to shed light on the relationship between physiological stress response and the subjective experience of anxious arousal in children with SAD. Unsurprisingly, we found that children with SAD exhibited significantly greater trait anxiety and anticipatory anxiety/state anxiety immediately preceding an acute stressor task. Interestingly, this was despite the fact that we found no significant group differences in biological stress reactivity experienced by socially anxious youths and age-matched healthy control children. We also found that self-reported anxiety ratings over the course of the exposure did not correlate with corresponding salivary cortisol and sAA timepoints, suggesting that an individual's subjective experience of anxiety may not correspond to objective measures of biological arousal. These findings support a theoretical model of SAD in which increased state anxiety may be attributed to excessive self-monitoring and cognitive biases such as catastrophic interpretations of a normative physiological response to anxiety and stress rather than an abnormally elevated physiological response that results in increased anxiety. This can inform the cognitive component of targeted anxiety interventions in that physiological symptoms of anxious arousal which everyone experiences to a similar degree can be normalized; clinically anxious individuals do not in fact experience an abnormally elevated and dysregulated level of biological reactivity in comparison to their psychopathology-free peers.

In conclusion, the current study provides information on the biological stress response of children with SAD and in relation to the subjective experience of anxious arousal. Results of the current study may expand our understanding of the biological mechanisms underlying effective exposure therapy, and ultimately contribute to personalizing treatment through identification of those children most likely to benefit from exposure. Although exposure therapy is the gold standard form of intervention for anxiety disorders, it involves intensive and repeated

interactions with feared stimuli and has been shown to be ineffective for a significant number of individuals. Thus, it is important to identify patient characteristics and not solely based on self-report that are associated with non-response to exposure in order to prevent unnecessary stress, ineffective treatment, and potential iatrogenic effects. To the best of our knowledge, this is the first report on HPA axis and ANS biological stress response as a predictor of intervention outcome for youths with SAD. Although the current study findings are mixed, they show preliminary evidence that individual differences in biological reactivity may shed light on who may benefit most from exposure interventions.

There are some methodological limitations to consider for the current study. Firstly, biological response was only explored in the context of a single-session intervention for SAD. Although one-session interventions have been demonstrated to be effective for social anxiety and specific phobia (e.g., Hindo & Gonzalez-Prendes, 2011, Ollendick, Ost, Costa & Cederlund, 2009), a more typical course of CBT involves 16 weekly 60-minute sessions (Kendall, Hudson, Gosch, Flannery-Schroeder, & Suveg, 2008). Secondly, the graduated exposure task was standardized in order to present consistent experimental stimuli to all patients whereas fear hierarchies are typically developed in collaboration with the patient and individualized for patients' specific needs (Kendall et al., 2005). Third, biological response measures were limited to salivary biomarkers and such analytes exhibit a lag time in relation to real-time stress response. Finally, the statistical power to detect an effect are limited due to small sample sizes of children with SAD and healthy control children.

The current study has important implications for future research. First, it would be interesting to examine whether biological stress response can change and be regulated with intervention. Secondly, it may be useful to explore other objective measures of physiological

symptoms of anxious arousal in conjunction with salivary analytes such as plasma cortisol, heart rate, and skin conductance, as they have an immediate response (or relatively shorter time lag) after exposure to anxiety-provoking stimuli. Third, we recommend that biological activation in response to exposure to other fearful stimuli be evaluated in the context of single-session and longer term episodes of care over a full course of CBT. Fourth, continued research on sAA reactivity in clinically anxious populations is warranted as it is a relatively newer analyte. Finally, further research should be conducted with salivary biomarkers to determine whether they may function as a viable assessment tool for studying the exposure process in anxiety treatment.

Study 3

Social Anxiety Disorder (SAD) is one of the most prevalent psychological disorders among children, with an estimated lifetime prevalence rate of 12.1% (Ruscio et al., 2008). SAD is characterized by an intense fear of embarrassment, humiliation, and negative evaluation, and avoidance of social situations in which these might occur (e.g., public speaking; DSM-IV-TR; American Psychiatric Association, 2000). Unlike many other anxiety and mood disorders, SAD onsets at a relatively early age, with diagnosis as early as age 8 and a mean onset of age 15.5 years (Kashdan & Herbert, 2001). In children, SAD is associated with social isolation, academic impairment and truancy, and the development of comorbid depression and substance abuse (Beidel, Turner, & Morris, 1999). The disorder seriously undermines children's social and emotional development, and typically follows a chronic course if left untreated (Kessler, Chiu, Demler, & Walters, 2005). This can further lead to low quality of life into adulthood, including comorbid depression and alcoholism, and functional impairment in work, education, and relationships (Weeks, Heimberg, Rodebaugh, & Norton, 2008).

The current study is designed to examine social skills deficits and biases in self-evaluation in children with SAD. Two bodies of theory and research suggest a connection between childhood SAD and such deficits and biases. First, psychologists have theorized that social skills deficits serve as an underlying causal and maintaining factor of SAD (e.g., Spence, Donovan, & Brechman-Toussaint, 1999). Specifically, it was hypothesized that poor social skills lead to ineffective and discouraging interpersonal interactions which then cause significant anxiety and distress (Inderbitzen-Nolan, Anderson, & Johnson, 2007). Assessment of social skills in the initial stages of social anxiety is important not only in contributing to research on SAD in childhood, but also to the understanding of deficits that may be exhibited in adults with

long-term SAD (Cartwright-Hatton, Tschernitz, & Gomersall, 2005). Second, cognitive theorists and researchers have proposed that individuals with SAD devote attentional resources to negative threat cues and exhibit a hypercritical cognitive style in evaluating their own performance in social interactions (Rapee, & Heimberg, 1997). This can lead to negative expectations for and subsequent avoidance of future social interactions, which further decreases opportunities to develop social skills (Wallace & Alden, 1997). In this way, social anxiety may be maintained and further exacerbated.

Unsurprisingly, previous studies have found that children high in social anxiety (e.g., Schmitz, Kramer, & Tuschen-Caffier, 2011), and SAD children (e.g., Schmitz, Kramer, Blechert, & Tuschen-Caffier, 2010), judge themselves more negatively than non-anxious control children on social-evaluative tasks such as giving a speech or telling a story in front of an audience. However, it is unclear whether these significant group differences are indicative of SAD children's underlying social skills deficits or of negative cognitive biases. In fact, few studies have assessed objective social skills deficits in children with SAD, and the findings are mixed. Some direct observation studies have found that external observers rate SAD children's performance worse than age-matched non-anxious controls on social interaction tasks (including conversational role plays and reading aloud) (Beidel et al., 1999; Norton & Hope, 2001), whereas Tuschen-Caffier, Kuhl, & Bender (2011) found no group differences. Tuschen-Caffier et al. (2011) found that SAD children performed no worse than non-anxious controls on social interaction tasks when rated by external observers. However, the SAD children's subjective ratings of their performance were significantly lower than those of non-anxious controls. Cartwright-Hatton, Tschernitz, and Gomersall (2005) found similar results with a sample of

children high in social anxiety rating their own performance significantly lower than external observers on a conversational role play with a stranger.

Thus, it remains unknown whether socially anxious individuals are accurate in their self-appraisal of social skills deficits or whether they may just experience negative cognitive biases and hold inaccurate and maladaptive beliefs regarding their social abilities. On the other hand, social psychology researchers have consistently found that the general population exhibits a positive illusory bias in which individuals exhibit a tendency toward overly positive self-serving biases (Alicke & Govorun, 2005). Perhaps SAD children lack such unrealistic and overly positive illusory biases that researchers have argued promote well-being and effective coping (Taylor & Brown, 1988). In the current study, we examined whether SAD children exhibit negative cognitive biases and whether non-anxious control participants exhibit cognitive biases in the opposite direction.

From a cognitive-behavioral perspective, social experiences should affect self-evaluation. Specifically, it has been theorized that positive social interactions increase confidence and a sense of self-efficacy in SAD individuals, whereas negative interactions decrease them (e.g., Beck & Emery, 1985; Hammerlie & Montgomery, 1982). However, it may be the case that SAD individuals actually experience a fear of both positive and negative social experiences (Weeks et al., 2008). Gilboa-Schechtman, Franklin, and Foa (2000) found that adults with SAD underestimated the probability of positive social interactions and exaggerated their cost. Researchers (Alden & Wallace, 1995; Wallace & Alden, 1997) found that adults with SAD who participated in a conversational role play (designed to go well) with a friendly confederate and who received positive feedback on the role play subsequently rated their own performance in that social interaction as positive. However, those SAD participants predicted that they would

experience even greater anxiety in a follow-up interaction with that same partner. Unlike non-anxious control participants, SAD individuals were concerned about falling short of the partner's heightened expectations. Thus, it is possible that SAD individuals may not experience positive social feedback as reassuring and enjoyable (Alden, Taylor, Mellings, & Laposa, 2008). To our knowledge, previous studies have not assessed whether SAD children would experience increased anxiety in response to positive social feedback.

Similarly, few studies have examined whether parents of SAD children have negative interpretation biases regarding their children's social skills. Mothers of children with an anxiety disorder have been found to exhibit lower expectations of their children's ability to cope in stressful situations and an increased likelihood of encouraging behavioral avoidance compared to mothers of non-anxious controls (Kortlander, Kendall, & Panichelli-Mindel, 1997; Micco & Ehrenreich, 2008). Parents of SAD children have also rated their children as less skilled on social skills questionnaires as compared to parents of non-anxious control children (Ginsburg, La Greca, & Silverman, 1998; Spence et al., 1999), but it is unclear whether these are accurate appraisals. To our knowledge, no previous studies have assessed whether parents of SAD children exhibit negative interpretation biases regarding their children's social abilities.

Examining whether SAD children's and their parents' perceptions of performance are accurate or negatively biased could have significant treatment implications. If SAD children exhibit skills deficits, and the children are accurate in their self-assessments of social skills, then social skills training would seem appropriate. In fact, therapy designed to make their self-assessments more positive rather than addressing underlying skills deficits could serve as a real disservice to these children. On the other hand, if SAD children's self-perceptions and parents'

perceptions are in fact negative cognitive distortions, a cognitively focused intervention approach might be appropriate.

Taken together, previous studies highlight the importance of examining social skills deficits in SAD children, along with biases in social-evaluative appraisals in SAD children and their parents, in order to further understanding of the nature of SAD and generate hypotheses about strategies for effective treatment.

Study aim/research questions. The aim of the current study was to examine the accuracy of SAD children's and their parent's evaluations of performance in social interactions. The study addressed the following research questions: (1) Do children with SAD perform worse than age-matched non-anxious controls on a public speaking task? (2) Do SAD children rate themselves worse than non-anxious control children on a public speaking task? Additionally, are SAD children more likely than non-anxious control children to rate themselves worse than objective observers do on a public speaking task? (3) Do SAD children's self-ratings of performance increase in response to positive external feedback? (4) Does SAD children's level of anxiety decrease after receiving positive external feedback? (5) Are parents of SAD children more likely than parents of non-anxious control children to rate their children worse than objective observers do on a public speaking task?

Hypotheses

(1) We hypothesized that SAD children would rate themselves worse than non-anxious control children on a public speaking task based on findings from previous studies (Cartwright-Hatton et al., 2005; Tuschen-Caffier et al., 2011). (2) We hypothesized that SAD children's self-ratings of performance on a public speaking task would increase in response to positive external feedback. This hypothesis was based on previous studies have shown that SAD adults who receive positive

feedback regarding their social performance have subsequently self-rated their performance in a more positive direction (Alden & Wallace, 1995; Wallace & Alden, 1997). The remaining research questions posed were exploratory.

Method

Sample²

68 parent-child dyads participated in the study. 34 of the child participants had a SAD diagnosis, and 34 were non-anxious healthy controls. Exclusion criteria for the SAD group included current comorbid non-anxiety psychological disorder, and current psychotropic medication use. The age range of children was 8 – 14 years ($M = 11.08$, $SD = 1.98$), 22 were male and 46 were female. 54.4% self-identified as Caucasian, 7.4% were African American, 4.4% were Hispanic/Latino, 13.2% were Asian, 14.7% were multiethnic, 1.5% self-identified as “other”, and 4.4% did not provide ethnicity information. Participants were recruited through community organizations (e.g., afterschool programs), local schools, hospitals, and online advertisements (e.g., Craigslist, parenting listservs).

Procedure

During the lab visit, an advanced clinical psychology doctoral student administered the SAD module of the Anxiety Disorders Interview Schedule for DSM-IV: Child and Parent Versions (ADIS-IV-C/P; Silverman and Albano, 1996) to the child and parent separately. The parent completed a set of baseline questionnaires, which includes parenting questionnaires, measures of their own anxiety, and their child’s psychological symptoms. The child also completed a set of baseline questionnaires, which included self-report measures of anxiety and psychological symptoms. Next, the child engaged in a public speaking task. We used a child-

² Study 3 used a subsample of Study 2 because the Perception of Performance Questionnaires (see below) were added to the study measures after data collection was already underway.

adapted version of the Trier Social Stress Test (TSST; Kirschbaum, Pirke, & Hellhammer, 1993), shortened in duration and with child-relevant speech topics. The child and parent were given five minutes to prepare a 5-minute speech on one of five topics (family, favorite vacation, hobbies, school, or sports). Next, the child gave a 5-minute practice speech to one of three committee members they were told would be judging their speech. Finally, the child gave a 5-minute speech in front of the full committee of three. The committee consisted of undergraduate lab assistants, who maintained neutral affect and took notes on the child's performance during the speech. If the participant stopped talking before time was up (i.e., a 10-second pause), committee members gave the child standard prompts such as "you still have some time left, please continue," and "try to tell us something more about (topic)". The speeches were videotaped for observational coding purposes. After the child's speech, the committee left the room to deliberate and returned to give the child positive feedback on speech performance.

Measures

Anxiety Disorders Interview Schedule for DSM-IV: Child and Parent Versions (ADIS-IV-C/P). The ADIS-IV-C/P (Silverman & Albano, 1996) is a semi-structured diagnostic interview designed to assess anxiety and related disorders; it has shown good to excellent inter-rater reliability and test-retest reliability (Silverman, Saavedra, & Pina, 2001).

Perception of Performance Questionnaire-Child³. The child's brief speech evaluation questionnaire consists of two items rated on an 11-point Likert scale. Item #1: "How do you think you did on the speech?" 0 = really badly, 10 = really good. Item #2: "How much do you think the committee liked your speech?" 0 = not at all, 10 = very much. The child completed the

³ Only 24 of the children in the SAD group, and 17 of the healthy control children had Perception of Performance Questionnaire-Child data as this measure was added to study procedures after data collection was underway.

questionnaire on two occasions, once immediately after the speech and again after receiving feedback from the committee.

Perception of Performance Questionnaire-Parent⁴. Immediately after the speech, the parent completed a brief speech evaluation questionnaire consisting of two items rated on an 11-point Likert scale. Item #1: “How do you think your child performed on the speech?” 0 = very badly, 10 = very well. Item #2: “How much do you think the committee liked your child’s speech?” 0 = not at all, 10 = very much.

Subjective Units of Distress Scale (SUDS). SUDS ratings are commonly used to measure anxiety levels during exposures. SUDS ratings were administered at 11 timepoints (including post-speech and post-committee feedback) to measure change in anxiety levels throughout the study session. The scale ranges from “0-not at all anxious” to “8-very, very much anxious” and a “feelings thermometer” was displayed as a visual aid for the ratings scale (Kendall, Robin, Hedtke, & Suveg, 2005).

Observational Coding. A main coder rated all videotaped speeches, and a reliability coder rated half of the videotaped speeches. Coders were blind to psychopathology status (i.e., whether the child was socially anxious or in the healthy control group). Evaluations on global speech performance were rated on the Perception of Performance Questionnaire and specific social skills were rated on a modified version of the Social Performance Rating Scale (SPRS; Fydrich, Chambless, Perry, Buergener, & Beazley, 1998). See Appendix A for modified version of the SPRS used for the current study.

⁴ Only 24 of the parents of children in the SAD group, and 17 of the parents of healthy control children had Perception of Performance Questionnaire-Parent data as this measure was added to study procedures after data collection was underway.

Perception of Performance Questionnaire-External Observer.⁵ External observers blind to participants' study group viewed the videotaped speeches and completed a brief speech evaluation questionnaire consisting of two items rated on an 11-point Likert scale. Item #1: "How do you think the child performed on the speech?" 0 = very badly, 10 = very well. Item #2: "Item #2: "How much do you think the committee liked the child's speech?" 0 = not at all, 10 = very much.

Social Performance Rating Scale (SPRS). The SPRS is an observational coding system on specific social skills designed for ratings of conversations between two people. The SPRS has shown excellent interrater reliability for assessing both individuals with social phobia and healthy controls (Fydrich et al., 1998). For the purposes of our study, we retained the original skills categories of eye contact, vocal quality (e.g., vocal inflections/nuances, appropriate volume), discomfort, and speech flow (e.g., speech content) and made slight modifications to coding manual descriptions so that the content was more appropriate for a child speech.

See *Table 3* for study measurement model.

Table 3. Study measurement model.

Baseline	Post-Speech	Post-Feedback
CHILD		
ADIS-IV-Child	PoP-Child SUDS	PoP-Child SUDS
PARENT		
ADIS-IV-Parent	PoP-Parent	
EXTERNAL OBSERVER		
	PoP-External Observer	

Note. ADIS-IV is the Anxiety Disorders Interview Schedule for DSM-IV. PoP is the Perception of Performance Questionnaire. SUDS are Subjective Units of Distress Scale ratings.

⁵ We did not have the committee members rate the Perception of Performance-External Observer because they were not blind to children's psychopathology (SAD or healthy control) group status.

Results

Interrater Reliability

Interrater reliability was calculated via intra-class correlations (using methodology described in Hallgren, 2012) between 2 coders on a random sample of 35 videos. Intra-class correlations showed good to excellent interrater reliability (Cicchetti, 1994), and ranged from 0.669 (discomfort) to 0.829 (speech flow) on speech microcodes. Global ratings of speech performance showed ICCs of 0.835 for how well the child did on the speech, and 0.872 for how well the committee liked the speech, respectively.

Speech Performance of Children with SAD vs. Healthy Controls

Perception of Performance Questionnaire. A one-way ANOVA showed no significant group differences on external observer ratings of children with SAD compared to healthy control children on global ratings of child speech performance, $F(1,66) = 0.84, p = .36$. Similarly, a one-way ANOVA showed no significant group differences between children with SAD and healthy control children in self-ratings of speech performance as compared to external observer ratings of speech performance, $F(1,39) = 0.00, p = .995$. See Figure 10.

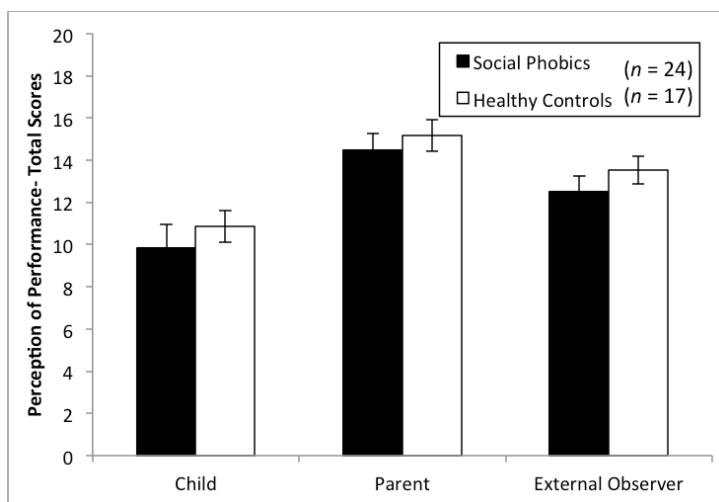


Figure 10. Mean ratings of child speech performance on the Perception of Performance-Total Scores as evaluated by the child, parent, and an external observer.

Social Performance Rating Scale. One-way ANOVAs showed no significant group differences on external observer ratings of eye contact ($F(1,66) = 0.79, p = .38$) or speech flow/content ($F(1,66) = 0.73, p = .40$) in children with SAD compared to healthy control children during their speeches. One-way ANOVAs showed a significant group difference on external observer ratings of physical comfort level ($F(1,66) = 5.15, p = .027$) and a marginal group difference on vocal quality ($F(1,66) = 3.58, p = .063$) in children with SAD compared to healthy control children during their speeches. Healthy control children were given higher ratings than children with SAD in physical comfort level and vocal quality. *See Figure 11.*

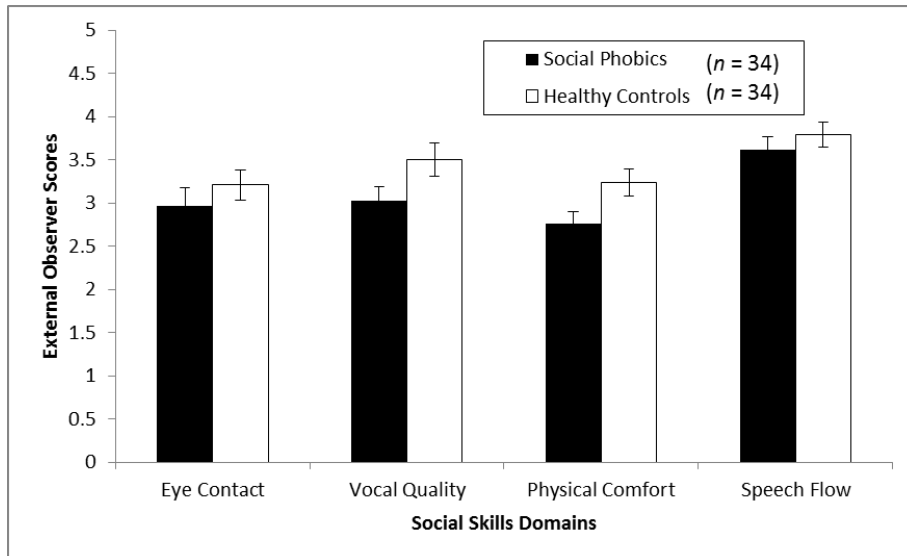


Figure 11. External observer mean ratings of child speech performance for the social skills domains of eye contact, vocal quality, physical comfort, and speech flow/content for children with Social Anxiety Disorder compared to healthy controls.

Age Effects

A multiple regression analysis showed that a diagnosis of SAD was not a significant predictor of global self-ratings of speech performance on the Perception of Performance Questionnaire-Child ($\beta = 0.10, t(37) = 0.75, p = .46$) nor was child's age ($\beta = -0.088, t(37) = -0.40, p = .69$). However, there was a significant group (SAD vs. healthy control group) x age

interaction effect; group x age was a significant predictor of child self-ratings of speech performance ($\beta = -0.49, t(37) = -2.25, p = .031$). Increasing age predicted lower self-ratings of speech performance in the SAD group only, but there was no such age effect for healthy control children. See Figure 12 for group x age interaction effect.

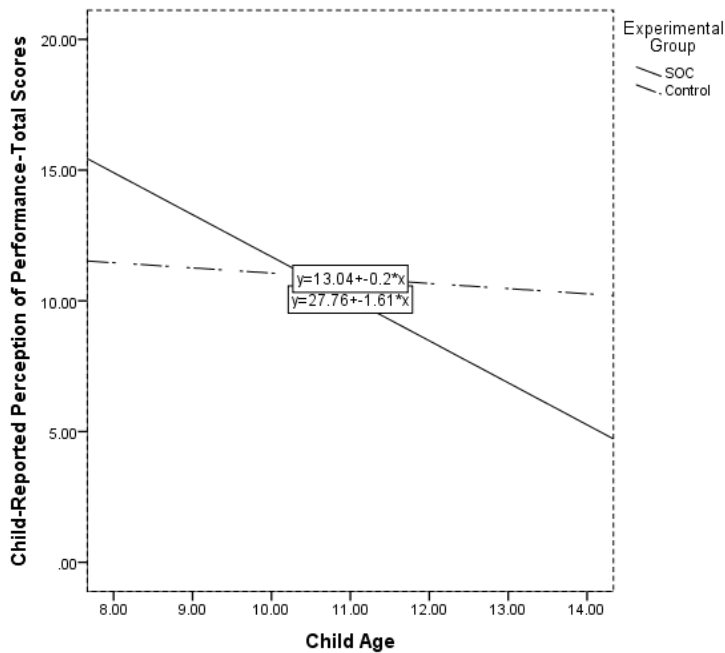


Figure 12. Interaction effect of child age x group (children with SAD vs. healthy controls) on self-evaluations of speech performance on the Perception of Performance-Total Scores.

A multiple regression analysis showed that a diagnosis of SAD was not a significant predictor of self-observer discrepancy scores of global ratings of speech performance on the Perception of Performance-Total Scores ($\beta = -0.008, t(37) = -0.05, p = .96$) nor was child's age ($\beta = -0.13, t(37) = -0.55, p = .59$). However, the group (SAD vs. healthy control group) x age interaction was trending towards significance, $\beta = -0.41, t(37) = -1.75, p = .09$. Increasing age was associated with lower self-ratings of speech performance in the SAD group in comparison to ratings made by an external observer, but there was no such age effect for healthy control children.

Children's Response to Positive Feedback

Among children with SAD, a paired sample *t*-test showed a significant increase in global self-ratings of speech performance after receiving positive committee feedback on their speech (as compared to global self-ratings of speech performance immediately prior to committee feedback), $t(23) = -5.40, p < .001$. In addition, a paired sample *t*-test showed a significant decrease in SAD children's self-ratings of anxiety after receiving positive feedback from the committee (as compared to self-ratings of anxiety immediately prior to committee feedback), $t(33) = 7.43, p < .001$. Similarly, among healthy control children, a paired sample *t*-test showed there was a significant increase in global self-ratings of speech performance after receiving positive committee feedback on their speech (as compared to global self-ratings of speech performance immediately prior to committee feedback), $t(16) = -9.21, p < .001$. In addition, a paired sample *t*-test showed a significant decrease in healthy control children's self-ratings of anxiety after receiving positive feedback from the committee (as compared to self-ratings of anxiety immediately prior to committee feedback), $t(33) = 6.37, p < .001$.

Parent Evaluations of Child Performance

A one-way ANOVA showed no significant group differences between parents of children with SAD and parents of healthy control children in global ratings of their children's speech performance on the Perception of Performance Questionnaire-Parent, $F(1,39) = 0.377, p = .543$. Similarly, a one-way ANOVA showed no significant group differences in parent ratings of their children's speeches as compared to that of an external observer's ratings regardless of child psychopathology status, $F(1,39) = 0.076, p = .784$.

Discussion

Psychologists have theorized that social skills deficits serve as an underlying causal and maintaining factor of SAD (Spence et al., 1999). Alternatively, cognitive theorists and researchers have proposed a theoretical model in which socially anxious individuals exhibit negative cognitive biases and a hypercritical cognitive style in evaluating their own performance in social situations which serve as an underlying causal and maintaining factor of SAD (Rapee & Heimberg, 1997). An important question for psychotherapy for children with SAD is whether the disorder primarily involves a skills deficit or a cognitive distortion involving underestimates of skill. Research that sheds light on this question has implications for appropriate intervention such as whether the main focus of psychotherapy should be on skills-building or cognitive restructuring. Answering this question could help us improve existing therapeutic interventions for socially anxious youths, as although CBT is the gold standard method of treatment for anxious youths, up to 50% still exhibit symptoms after treatment (Ginsburg & Schlossberg, 2002). Additionally, effective early intervention is especially important given the serious long-term consequences of SAD if left untreated, including decreased quality of life and pervasive impairments in academic, work, and social domains.

Our findings suggest that a comprehensive theoretical model for youth SAD that implicates both specific social skills deficits and cognitive biases may be the most accurate. Overall, socially anxious youths seem to display comparable levels of social skills to their age-matched non-anxious peers on global ratings of speech performance by external observers, with some exceptions that pertain to specific skills deficits in vocal quality and physical comfort level. Interestingly, the quality of speech content of socially anxious youths was rated at a similar level to that of healthy controls. This suggests that socially anxious youths may not need help with

what to say but rather *how* to say it. Appropriate targets for skills-based interventions are incorporating vocal inflections into speech and working on body language in order to minimize signs of physical discomfort. Promisingly, our findings showed that socially anxious children rated themselves more favorably on social performance after receiving positive external feedback and reported decreased anxiety in response to the positive feedback. This suggests that negative cognitive schemas may be malleable in psychotherapy with fear-conditioning reversal and that positive feedback can be internalized and experienced as reassuring and enjoyable.

Overall, we found that socially anxious youths were not unrealistically critical of their own speech performance and their perceptions of how well their speech was received by an external committee. Children with SAD were generally able to accurately evaluate their own performance and skills on a social-evaluative task involving public speaking. Parents of children with SAD were similarly accurate in their appraisals of their child's speech performance which suggests that they are not hypercritical of their child's social skills. However, we did find age effects such that older children/young adolescents who were socially anxious were most self-critical of their own performance, and significantly more so than their age-equivalent non-anxious peers. Adolescence is commonly considered a critical developmental stage for identity formation and social skills development, and demarcated by preoccupations with peer acceptance and body image (Petersen & Leffert, 1995). This suggests that adolescence as experienced by socially anxious youths may warrant special research attention due the vulnerability of this developmental period to cognitive distortions and pessimism about one's performance in social situations. Previous research has shown that in youths with SAD, adolescents are distinct from younger children in that they present with higher rates of suicidal ideation (Francis, Last, & Strauss, 1992) and excessive self-focused attention in social situations (Albano et al., 1995).

Clark and Wells's (1995) cognitive model of SAD posits that socially anxious individuals exhibit excessive self-focused attention, and that self-focused attention increases access to negative thoughts and feelings, leading individuals to generate and internalize a negatively distorted impression of themselves. Thus, the cognitive restructuring component of CBT may be especially relevant for anxious adolescents and less so for younger children.

Several methodological limitations of the current study are to be noted. Firstly, although the focus of our experimental task on public speaking was appropriate, because this is the most commonly endorsed feared situation among children with SAD (van West, Claes, Sulon, & Deboutte, 2008), our focus on that task did limit our ability to generalize findings to other types of social scenarios. Secondly, we only obtained global ratings of the children's and parents' perception of speech performance whereas ratings on specific deficits may provide more nuanced information on perceived strengths and weaknesses. Third, we did not evaluate older adolescents (>14 years of age) in our study, and older youths may experience even more pronounced age effects of self-criticism and negative cognitive biases. We did not anticipate that there would be an age effect with cognitive biases differing from childhood to early adolescence. Finally, small sample size may limit statistical power to detect an effect.

Further research on social skills deficits and cognitive distortions in children and adolescents with SAD is warranted. Firstly, youths with SAD have been studied much less than their adult counterparts. It is important to examine unique differences in SAD symptomatology across the lifespan, including changing presentations from early childhood into adolescence due to increased social-evaluative concerns associated with typical development. Theoretical models for SAD may need to be modified based on developmental stage. Secondly, CBT manualized protocols may be most beneficial when targeted to specific age groups based on utility of

cognitive vs. behavioral principles. Given accuracies in self-appraisals of social performance, exposure may be explored as a beneficial standalone treatment in younger children. Given a pronounced increase in self-criticism and negative cognitive biases in adolescence, cognitive restructuring may be an especially important treatment focus. Third, it may be important to evaluate social skills deficits and negative cognitive biases in a range of commonly endorsed fear situations such as talking to new people, talking to adults, and starting or joining in on a conversation (examples from van West et al., 2008) in order to explore whether there are situation-specific deficits that may arise. Finally, research should continue to incorporate the use of independent evaluators of children's social competence in order to tease apart the role of objective skills deficits vs. subjective interpretation biases in the onset and maintenance of SAD. Our findings also suggest that a finer grained level of analysis is warranted. Future research should focus on strategies for precise assessment of the nature of deficits for specific individuals in order to permit better personalizing of interventions.

General Discussion

Anxiety disorders are among the most common psychological disorders in children and adolescents. They are associated with impaired social and emotional development, and typically follow a chronic course if left untreated. Cognitive Behavioral Therapy (CBT) has more extensive empirical support than any other treatment for child and adolescent anxiety. CBT is a short-term intervention focused on anxiety management skills training, equipping the child with a “toolkit” of coping “tools” such as problem solving, relaxation, cognitive restructuring, and exposures. However, as many as half of anxious children still exhibit significant symptoms after treatment (Ginsburg & Schlossberg, 2002), and over 40% fail to recover from their anxiety disorder (Kendall et al., 2012).

As no single mechanism can explain the onset and maintenance of anxiety disorders, research has explored an intricate set of risk factors implicated in theoretical models of anxiety disorders in order to provide a more complete picture of anxiety in childhood and adolescence (Rapee et al., 2009). Research is needed to identify factors associated with treatment response, and ultimately to inform treatment improvement. It’s especially important to understand the distinctive developmental needs of children and adolescents in treatment. CBT interventions are much more effective for anxious adults than anxious children, and perhaps this isn’t too surprising because we know a lot more about anxiety disorders in adults, and child treatment manuals are also oftentimes directly adapted from adult manuals. In thinking about the unique developmental needs of children, there’s what’s going on with the child and arguably, as importantly, what’s going on with the parent, who controls access to treatment, can be considered a co-client in therapy, and may be the child’s support after treatment ends.

My dissertation focused on risk factors implicated in theoretical models of anxiety disorders, including (1) biological factors (i.e., physiological stress reactivity and dysregulation), (2) cognitive functioning (i.e., information processing biases), and (3) environmental influences (i.e., parenting and family environment). I used a multi-method, multi-informant approach (including parent- and child-report measures, behavioral observations of videotaped lab tasks, and salivary biomarkers) to examine factors that influence the response of anxious youths and their parents to treatment interventions.

Study 1 used data from a youth anxiety disorders randomized controlled trial comparing CBT to Usual Care (UC) conducted by Southam-Gerow and colleagues (2010) to determine the impact of parental anxiety and child perceived control on treatment outcomes. Southam-Gerow and colleagues (2010) found that child anxiety symptoms improved in both treatment conditions, and there were no significant differences between CBT and UC for treatment outcome, length of treatment, or cost. To my knowledge, this is the first study to examine parental anxiety and child perceived control as predictors of treatment outcome in UC, and in CBT compared to UC. We found that parental anxiety and child perceived control generally were not associated with treatment outcomes in the UC or CBT treatment condition. Additionally, parental anxiety levels did not decrease over the course of treatment for UC or CBT. The substantial reduction in symptoms and diagnoses in both CBT and UC, combined with the finding that parental anxiety did not predict negative treatment outcomes, suggests that parents' anxiety may not significantly interfere with their children's treatment, even though evidence indicates that it plays a strong role in childhood anxiety disorders etiology (Rapee et al., 2009). Notably, parental anxiety was associated with higher expectations for therapy at pretreatment. Child perception of control, on the other hand, improved from pre- to post-treatment in both CBT and UC. This suggests that

child perception of control may increase over the course of treatment regardless of therapeutic orientation and approach. In addition to evaluating CBT-specific treatment moderators, it may be beneficial to expand our search to child and parent characteristics that are related to a successful treatment intervention regardless of theoretical orientation or treatment modality.

Study 2 examined physiological symptoms of arousal implicated in cognitive models of Social Anxiety Disorder (SAD). To my knowledge, previous research has not assessed the role of HPA axis and ANS activation in treatment outcome for socially anxious youths. We evaluated biological reactivity in SAD children and age-matched non-anxious children in the context of a single-session graduated exposure intervention. We found that children with SAD and age-matched non-anxious children showed similar levels of salivary cortisol and sAA at baseline, and children with SAD did not experience *abnormally* elevated levels of biological arousal overall in response to a psychosocial stressor task. However, in response to the stressor task, SAD children were more likely than healthy control children to meet criteria for classification as salivary cortisol responders (41.7% versus 8.8%), and healthy control children were more likely to experience physiological blunting. With regard to biological arousal as a predictor of treatment effect, there were mixed findings with preliminary evidence for heightened cortisol and sAA reactivity over the course of a graduated exposure intervention predicting anxiety symptom improvement; this of course would be consistent with the notion that arousal during exposure enhances the benefits of the exposure. Our study also helped to clarify the relationship between objective stress reactivity and the subjective experience of anxiety in children with SAD. We found that children with SAD show significantly higher trait anxiety and anticipatory anxiety preceding a psychosocial stressor. However, self-reports of state anxiety did not correlate with biological stress response over the course of a graduated exposure. Our

findings support a theoretical model of SAD in which clinical levels of anxiety may be due to excessive self-monitoring and overly negative interpretations of normative biological response to stress rather than abnormally elevated biological arousal.

Study 3 focused on assessing social skills deficits and negative cognitive biases that have been implicated in theoretical models of SAD. We had SAD children and age-matched non-anxious controls evaluate their own social performance on a public speaking task, along with their parents and independent observers blind to group. Our findings suggest that a comprehensive theoretical model for youth SAD that incorporates both specific social skills deficits and negative interpretation biases may be the most appropriate. Children with SAD exhibited similar levels of social skills as non-anxious peers overall, including independent evaluations on speech content. However, children with SAD did exhibit deficits in the specific domains of vocal quality and physical comfort level. This suggests that socially anxious youths may not need help with *what* to say but rather *how* to say it. Children with SAD also generally had the ability to accurately rate their own speech performance, as did their parents. However, increasing age in the SAD group was associated with lower self-ratings of speech performance. Adolescence, as experienced by socially anxious youths may be an especially vulnerable period for cognitive distortions and hypercriticism over one's self-perceptions of social performance. Thus, behavioral interventions alone may be appropriate for younger children and the cognitive restructuring component of CBT may be especially salient for adolescents.

The purpose of my dissertation studies is to further our understanding of the distinctive developmental needs of youths in treatment, reflecting their biological and cognitive development, and the influences of parenting. Each study is intended to provide a distinct and complementary perspective from core domains implicated in theoretical models of anxiety

disorders, with the ultimate goal of informing treatment effectiveness research. Expanding our knowledge of risk and protective factors associated with treatment response may help us identify youths who are more likely to benefit from specific treatments, identify others who may need alternative approaches, and inform efforts to personalize treatment for individual youths.

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Appendix

Appendix A. Behavioral anchors for the Social Performance Rating Scale (SPRS)
(as adapted from the SPRS; Fydrich et al, 1998)

A.1. GAZE

(1) Very Poor: Participant completely avoids looking at the audience or stares continually and intently at audience member directly in front of him/her in an uncomfortable manner (i.e., no eye contact or stares down audience member). Gaze pattern is very disruptive to performance.

(2) Poor: Participant frequently avoids looking at the audience (or stares intently at audience member directly in front of him/her) for majority of time. Gaze pattern is disruptive to performance.

(3) Fair: Participant avoids eye contact or tends to look too much (staring intently) at specific audience member. Gaze pattern is mildly disruptive to performance.

(4) Good: Participant shifts focus during pauses, but occasionally avoids eye contact or stares at audience members.

(5) Very Good: Participant keeps eye contact during the speech, does not stare; shifts focus during pauses.

A.2. VOCAL QUALITY

(1) Very Poor: (a) Participant speaks in a flat, monotonous voice; or (b) speaks at a low volume or mumbles; or (c) speaks overly loudly, or has intrusive tone (harsh or unpleasant voice quality).

(2) Poor: (a) Participant demonstrates minimal vocal inflections/nuances, enthusiasm, or interest in verbal expression; or (b) volume somewhat low and speech somewhat unclear; or (c) speaks a little bit too loudly, or tone is somewhat intrusive, unpleasant, or sarcastic.

(3) Fair: (a) Participant shows some inflections/nuances in verbal expression but at most times sounds unenthusiastic or uninterested; and (b) speaks in appropriate volume; has clear voice quality; and (c) does not have an intrusive or sarcastic tone.

(4) Good: (a) Participant shows moderate inflections/nuances and but inconsistent enthusiasm or interest. Could also be too 'over the top' (seems fake or forced); and (b) speaks in appropriate volume; has clear voice quality and (c) does not have an intrusive, unpleasant, or sarcastic tone.

(5) Very Good: Participant is emphatic and enthusiastic in verbal expression; and (b) speaks in appropriate volume; has clear voice quality and (c) does not have an intrusive, unpleasant, or sarcastic tone.

A.3. DISCOMFORT

(1) Very High: Complete rigidity of arms, legs or whole body. Constant leg movements or fidgeting with hands, hair or clothing. Extremely stiff face or constant facial tics. Frequent nervous throat clearing, swallowing, or stuttering. Frequent inappropriate giggling or laughing. Look of extreme discomfort and desire to flee situation.

(2) High: Rigidity or fidgeting for majority of time. Difficulty staying still is somewhat disruptive to conversation. Stiff face or frequent facial tics. Some nervous throat clearing or swallowing. Some inappropriate giggling or laughing. Participant shows signs of discomfort by frequently looking around.

(3) Moderate: No rigidity. Slight movement of legs, fidgeting, throat clearing, or swallowing. Participant shows only brief periods of discomfort.

(4) Low: No rigidity, nervous throat clearing, or swallowing. Minimal fidgeting that is not disruptive to performance. No notable signs of discomfort. At times may appear relaxed and at ease (smiling or gesturing).

(5) Very Low: Relaxed body posture and natural body movement. Participant laughs and smiles at appropriate times. S/he shows effective gesturing (to be distinguished from fidgeting). Participant does not appear at all uncomfortable and is at ease in situation.

A.4. SPEECH FLOW

(1) Very Poor: Participant frequently trails off, making few attempts to continue the speech. Participant does not respond appropriately to audience's prompts, (does not acknowledge prompts and/or does not react to prompts). Even when prompted by the audience, participant cannot maintain the speech.

(2) Poor: Participant tries to initiate and continue the speech but is only successful about half the time. The speech does not flow smoothly – participant trails off, participant does not follow up information on topics in a fluid manner or provide relevant examples. Participant sometimes repeats the same factual information during the speech (repeats himself/herself). Participant occasionally responds appropriately to audience's prompts, (does not acknowledge prompts and/or does not react to prompts).

(3) Fair: For the most part, the participant is able to continue the speech with little to no help/prompts from the audience, although the speech is still somewhat awkward and stalls at times, with participant occasionally trailing off. Participant provides little follow up information on topics or provide relevant examples. Participant responds appropriately to audience's prompts.

(4) Good: Participant is able to maintain the speech with no help/prompting from the audience. The speech flows smoothly with few awkward pauses. Participant rarely trails off. The participant readily shares information and examples. Shows interest in engaging the audience, and follows up appropriately on participant's own remarks. No obvious deficits.

(5) Very Good: Participant easily maintains the speech with minimal pauses and smooth transitions, often following up on previous information provided by making appropriate follow-up remarks and offering additional information on a related topic. Participant introduces new topics fluidly and speaks fluently.