

ABSTRACT

Title of dissertation: MATERNAL AND PATERNAL
ANXIETY DISORDERS AND EARLY
INTERVENTION FOR BEHAVIORALLY
INHIBITED PRESCHOOL CHILDREN

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Behavioral inhibition (BI) and, subsequent, social withdrawal (SW), are overlapping, but *distinct* concepts, and both are risk factors for the development of later child anxiety. Anxiogenic parenting (i.e. any parenting behavior that promotes anxiety in offspring) and negative peer interactions both contribute to the pathway from child BI/SW to later anxiety (Rubin, Coplan, & Bowker, 2009). Parents' own anxiety is associated with offspring anxiety and may raise the risk for anxiogenic parenting (Murray, Creswell & Cooper, 2009). Thus, through targeting anxiogenic parenting and negative peer relations, early intervention has the potential to alter the trajectory from child BI/SW to later anxiety.

However, several gaps exist in the literature. One, few treatments exist for preschool BI/SW. Two, despite parents' key role during the preschool years and robust evidence showing parent anxiety reduces treatment efficacy for older anxious youth, the role of parent *clinical* anxiety in *preschool* treatment outcomes has not been rigorously assessed. Three, despite meta-analytic data showing fathers' importance in the development of preschool anxiety (Möller, Nikolić, Majdandžić, & Bögels, 2016), few researchers have examined effects of fathers' anxiety on preschool treatment outcomes. Relatedly, the *unique* effects of mothers' and fathers' anxiety on *preschool* treatment outcomes have yet to be tested. Lastly, despite the dual roles of parents and peers in the pathway from child BI/SW to later anxiety, no study has used a true experimental design to test whether treatment for preschool BI/SW that targets both domains can attenuate the effect of parent anxiety on reduced treatment outcomes more than that of interventions that directly target parents only, the most common approach used in existing interventions for preschool BI/SW or anxiety.

The current dissertation study was drawn from a larger NIMH-funded study comparing two early intervention programs for preschool BI/SW. The first program was Cool Little Kids (CLK), a parent-only psychoeducation group, which has been shown to be efficacious in reducing later anxiety among highly inhibited preschoolers (Rapee, Kennedy, Ingram, Edwards, & Sweeney 2005). The second program was the "Turtle Program," which included concurrent parent and child groups, targeting both anxiogenic parenting and child social skills, respectively (Chronis-Tuscano et al., 2015). The first study aim was to examine the separate and *unique* roles of clinician-rated maternal and paternal lifetime anxiety disorders (ADs) as predictors of preschool treatment outcome

across treatment conditions. It was hypothesized that, separately, maternal and paternal lifetime ADs would negatively predict child treatment outcome in both groups. When maternal and paternal lifetime ADs were examined in the same model, it was further hypothesized that paternal lifetime ADs would continue to predict child treatment outcomes.

The second aim was to examine whether the associations between maternal and paternal lifetime ADs and child treatment outcomes differed as a function of treatment condition. It was hypothesized that the in-vivo, intensive nature of the “Turtle Program” might mitigate the negative effects of parents’ anxiety, such as parental avoidance, anxious modeling, and overcontrol, on child treatment outcomes. Further, the child group component of the Turtle Program might improve child social approach behaviors, making it easier for parents to facilitate child social exposures. Thus, it was hypothesized that maternal and paternal lifetime ADs would have weaker associations with child treatment outcome in the “Turtle Program” than in CLK.

Lastly, given that child BI/SW are predictors of *social* anxiety specifically, and social anxiety is heritable (Isomura et al., 2015), the role of maternal and paternal lifetime *social* anxiety disorder (SAD) on child treatment outcomes was also examined as an exploratory aim.

Results indicated that, when examined in the same model, maternal lifetime ADs predicted worse post-treatment child total anxiety, but paternal lifetime ADs predicted *better* post-treatment child total anxiety. This relation did not significantly differ as a function of treatment condition. Regarding the exploratory aim, when examined in the same model, maternal (not paternal) SAD predicted worse post-treatment child total

anxiety. Further, maternal SAD predicted worse post-treatment child total anxiety only in CLK (and not the Turtle Program), suggesting that in-vivo therapist coaching of parents and/or direct child social skills training may have mitigated negative effects of maternal SAD on reduced treatment efficacy. Clinical implications and future directions are discussed.

MATERNAL AND PATERNAL ANXIETY DISORDERS AND TREATMENT OF
PRESCHOOL CHILDREN WITH HIGH BEHAVIORAL INHIBITION

by

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Chapter 1: Introduction

Clinical and Public Health Relevance of Child Anxiety Disorders

Anxiety disorders are common, and they affect approximately 10% of youth (Egger & Angold, 2006). Youth with anxiety disorders often continue to experience anxiety into adolescence and adulthood (Copeland, Shanahan, Costello, & Angold, 2009), and they are at risk for later substance use (Hussong, Jones, Stein, Baucom, & Boeding, 2011) and depression (Beesdo et al., 2007). Even sub-threshold anxiety symptoms prospectively predict adverse psychosocial trajectories (Davies et al., 2016). Further, the public health burden of child anxiety disorders is about 20 times that of non-affected youth (Bodden, Dirksen & Bögels, 2008).

Despite the early onset of childhood anxiety disorders, most studies largely focus on anxiety among school-age children and adolescents, and relatively little research focuses on anxiety during preschool. This is alarming, given that 50% of cases occur before age 6 (Merikangas et al., 2010). Further, in comparison to that of older youth, preschool children experience similar rates of anxiety disorders, anxiety-related impairment, and comorbid disorders (Egger & Angold, 2006; Dougherty et al., 2013). Lastly, an early onset often indicates a more chronic and severe disease course (Ramsawh, Weisberg, Dyck, Stout, & Keller, 2011), *underscoring the significance of early intervention.*

Transactional Nature Among Temperament, Peer Relations, Parenting, and Parent Anxiety in the Development of Child Anxiety

Theoretical and developmental considerations.

Identifying early risk factors for later anxiety is of utmost importance for the purposes of early intervention. One such risk factor is behavioral inhibition (BI), a biologically-based temperamental construct (Fox, Henderson, Marshall, Nichols, & Ghera, 2005), evident in the first few months of life. BI is characterized by fear and withdrawal in the face of novelty (Kagan, Reznick & Snidman, 1988). BI is often operationalized as children's reactions to novel, *non-social* events (e.g. toys, mechanical objects) and *unfamiliar* social events (e.g., novel adult women) in the context of novel settings (e.g., the laboratory) (Garcia-Coll, Kagan & Reznick, 1984). Social withdrawal (SW) is an overlapping, but *distinct* concept (Rubin & Asendorpf, 1993; Rubin, Hastings, Stewart, Henderson & Chen, 1997; Rubin, Burgess & Hastings, 2002; Rubin & Lollis, 1988; Rubin, LeMare & Lollis, 1990; Rubin, Hymel, Mills & Rose-Krasnor, 1991; Rubin & Stewart, 1996). SW is defined as “the consistent (across situations and over time) display of solitary behavior when encountering *familiar* and/or unfamiliar *peers*” (Rubin & Burgess, 2001, p.883). BI/SW are directly relevant to intervention and prevention efforts, as BI in infancy and toddlerhood is a precursor to SW in later childhood, and both BI/SW are risk factors for later social anxiety (Chronis-Tuscano et al., 2018; Clauss & Blackford, 2012; Rubin & Coplan, 2004), specifically when BI/SW are elevated across time (Chronis-Tuscano et al., 2009; Hirshfeld et al., 1992). Further, BI/SW predict later anxiety and mood disorders more broadly (Hirshfeld-Becker et al., 2014; Rubin et al., 2009), as well as social and academic impairments (Rubin et al., 2009).

Child BI/SW and later anxiety: The role of parenting

However, BI/SW do not always lead to negative psychiatric and functional outcomes. Identifying risk factors for later anxiety among children high in BI can further

aid our efforts to intervene earlier in the disease course. One model that discusses such risk factors is Rubin and colleagues' (1991; 2009) developmental-transactional model, which proposes that negative parenting exacerbates pathways from child BI/SW to later negative outcomes (See Figure 1 for a simplified model from Chronis-Tuscano et al., 2018). The model proposes that, during toddlerhood, a transactional process begins, whereby child BI evokes parenting responses that are overcontrolling, intrusive and overly protective. Over time, parents come to perceive their inhibited and withdrawn children as vulnerable (Rubin, Nelson, Hastings & Asendorpf, 1999), so parents inappropriately shield their children from non-threatening, normative tasks by performing the tasks for them. Subsequently, children come to perceive themselves as incapable of independent mastery of social and non-social situations, and this perception maintains child withdrawn behaviors. Overtime, children “miss out” on developmentally necessary opportunities to develop social (and non-social) competencies.

Indeed, ample empirical evidence supports Rubin and colleague's (2009) model (Hastings, Rubin, Smith & Wagner, in press), showing that parenting both predicts the stability of BI/SW and moderates risk for anxiety among children high in BI/SW (Definitional clarity of parenting terms used in this literature is discussed in Appendix A). For example, Williams and colleagues (2009) found that observed toddler BI (in the lab setting) significantly predicted parent-reported internalizing symptoms at age 4 *only* when parents reported permissive parenting. Further, another study found that consistently high childhood inhibition predicted adolescent social anxiety *only* when mothers exhibited observed overcontrol (Lewis-Morrarty et al., 2012). Results underscore

the longitudinal relations between child BI/SW and later anxiety in the context of parent permissiveness and overcontrol.

Importantly, in addition to later anxiety, parenting also longitudinally predicts the *stability* of BI/SW. Hane and colleagues (2008) found that parent-reported observed age 4 reticence significantly predicted observed age 7 SW (in the presence of unfamiliar peers) *only* when mothers exhibited observed high negativity. Further, the relation between age 2 BI (in the presence of an unfamiliar peer) and age 4 social reticence with unfamiliar peers was *only* significant when mothers were highly intrusive and derisive (Rubin, Burgess, & Hastings, 2002). Degnan and colleagues (2008) also found that observed age 4 social reticence predicted observed age 7 social wariness only when mothers showed high observed solicitousness. Lastly, in a study that included both mothers and fathers, Johnson and colleagues (2016) found that parent-reported overprotection predicted the stability of observed child BI (in the presence of novel adults and objects) from age 3 to 6. Overall, because anxiogenic parenting predicts the stability of BI/SW across time, and it is stable BI/SW that predicts later child anxiety (Chronis-Tuscano et al., 2009; Hirshfeld et al., 1992), reducing such parenting behaviors may be a powerful target of early intervention programs.

Child BI/SW and later anxiety: The role of peers

In addition to the role of parenting, Rubin and colleagues (1991; 2009) model also discusses the role of child-peer relationships in the child SW to later anxiety link (see Figure 1). Child SW is associated with less social competence (Bohlin, Hagekull & Andersson, 2005), subsequent peer victimization and rejection (Boivin, Hymel & Bukowski, 1995), and lower quality friendships with similarly-victimized peers (Rubin,

Wojslawowicz, Rose-Krasnor, Booth-LaForce & Burgess, 2006). Lack of social skills and child SW result in peer rejection and victimization because SW is discordant with societally-expected behaviors. According to Rubin and colleagues' (1991; 2009) transactional model, a child-peer transactional cycle also develops, wherein child SW elicits peer victimization and peer rejection, which further exacerbates child SW and increases peer victimization and peer rejection. Withdrawn children become aware of their social difficulties, contributing to negative self-appraisals of their own social skills and relationships, loneliness, and depressive symptoms over time (Boivin et al., 1995; Rubin et al., 1995).

Overall, both negative child-peer *and* parent-child transactional processes exacerbate the pathway from child BI/SW to later anxiety, as well as other negative outcomes, such as depression, negative self-esteem and peer problems by middle childhood and early adolescence (Booth et al., 2012; Oh et al., 2008).

The role of fathers parenting in child development

Notably, most theoretical models and studies of the developmental pathway from child BI/SW to later anxiety do not explicitly discuss fathers' roles (Degnan et al., 2010). Delineating fathers' roles in the pathway from child BI/SW to later anxiety first requires understanding the role of fathers in child development more broadly. Attachment theory (Lamb & Lewis, 2013), social learning theory (Lamb & Lewis, 2013), social capital theory (Coleman, 1988) and essential theory (Pleck, 2007) are all commonly referenced when discussing fathers' roles in child development. Further, *paternal involvement*, defined as paternal engagement, accessibility and responsibility (Lamb, Pleck, Charnov & Levine, 1987; Pleck, 2010), is often used as a complementary construct to the

aforementioned theories. Recently, Cabrera and colleagues (2007, 2014) developed a heuristic model of father-child relationships. They proposed that fathers' parenting is reciprocally and directly influenced by child effects and other family relationships (Cabrera et al., 2014).

While some argue that fathers' parenting is *uniquely* related to child outcomes (Palkovitz, Trask & Adamsons, 2014), others propose that dimensions of mothers' and fathers' parenting (e.g. warmth, sensitivity, harsh punishment) affect children similarly (Fagan, Day, Lamb & Cabrera, 2014). However, one objective difference between mothers and fathers is in their child care time and responsibilities. Riley, Bianchi, and Wang (2012) studied 6,000 married, dual-earning couples, and they found that mothers spent 45-hours per week of total time with children and 15-hours per week on child care duties specifically (compared to 32-hours and 9-hours/week for fathers, respectively). Simply put, children objectively have more weekly exposure to mothers than fathers.

Mothers and fathers also differ in their play styles. Mothers engage in more overall play and play that emphasizes the reactions of others (Cabrera, 2016). Contrarily, play that "stimulates, surprises, and destabilizes the child, encouraging the child to take risks" is characteristic of fathers' interactions with their young children, and such play predicts positive child social competence and peer relations (Paquette, 2004; Bögels & Phares, 2008). Relatedly, Parke and Buriel's (2008) Tripartite Model of Family-Peer Relationships also proposes that fathers and mothers are socialization agents in peer relations. Parke and Buriel (2008) and Cabrera (2016) both propose that fathers have unique effects on children's social development specifically. For example, Isley, O'Neil and Parke (1996) found that fathers' (but not mothers') expressed negativity during

physical play was the strongest predictor of lower teacher-rated peer social acceptance at age 4-6.

Thus, because mothers and fathers differ in their child care time and responsibilities, as well as in their play styles (which is important to child social development; Isley et al., 1996), they each deserve equal attention when studying risks for later anxiety among children high in BI/SW, as each parent may uniquely contribute to early intervention efforts among this at-risk population.

Child BI/SW and later anxiety: Evidence for the role of fathers' parenting.

Among the few studies that have examined the role of paternal parenting in the pathway from child BI/SW to later anxiety, McShane and Hastings (2009) found that, among parents with “cautious, quiet or shy” 2-5-year-old children, observed maternal overprotection and paternal critical control, when examined in the same model, uniquely predicted teacher-reported child internalizing symptoms. However, child anxiety predicted increased overprotection in fathers and, marginally, mothers one year later. Relatedly, mother and father-reported child age 2 social shyness predicted both parents' reports of discouragement of independence at child age 4 (Rubin et al., 1999). However, maternal and paternal discouragement of independence at age 2 did not predict either parents' ratings of age 4 shyness, underscoring the importance of “child effects,” in line with Rubin and colleague's (2009) transactional model (See Figure 1). While Rubin and colleagues (1999) found both parents' discouragement of independence was affected by their prior perceptions of child shyness, they also found both observed BI and mothers' perceptions of child shyness at age 2 predicted mother-rated age 4 child shyness;

however, for fathers, only fathers perceptions of shyness (and *not* observed BI) at age 2 predicted father-rated age 4 shyness.

Lastly, one cross-sectional study with 3- to 4- year-olds found that observed high paternal protection and low paternal support were related to more parent-reported child inhibition and observed social wariness, respectively; these relations were stronger among offspring with physiological risk factors (i.e. less vagal suppression; Hastings et al., 2008). In the same study, observed maternal supportive parenting predicted fewer parent-reported child internalizing problems largely regardless of child physiological risk (i.e. level of vagal suppression). While this study further assessed children 4 years later (Hastings, Kahle & Nuselovici, 2014), only mothers' parenting was examined, highlighting the field's overall lack of attention to fathers' role in the pathway from BI/SW to later anxiety.

Overall, studies that only include mothers are likely limited because a small body of literature with preschoolers shows paternal overprotection and overcontrol also exacerbate the pathway from child BI/SW to later internalizing symptoms. This further elucidates our understanding of factors that put children high in BI/SW at further risk for anxiety, and it highlights a critically understudied area that could, crucially, be a meaningful target of intervention.

Maternal and paternal parenting and the development of preschool anxiety.

Given that the studies of both mothers' and fathers' parenting in the link between child BI/SW and later anxiety are few, the broader research on the relations between maternal and paternal parenting and anxiety in young children is reviewed below.

Among 3- to 5-year-old children, bi-directional positive associations have been found between mother-reported overprotection and later parent-reported child anxiety (Edwards, Rapee, & Kennedy, 2010). In the same study, only unidirectional positive associations existed from father-reported overprotection to later child anxiety, suggesting that paternal parenting was less influenced by “child effects.”. Further, meta-analyses have demonstrated larger effect sizes between parental control and child anxiety in studies that included fathers/both parents compared to those with primarily mothers (van der Bruggen, Stams, & Bögels, 2008). Another meta-analysis of community samples showed that paternal (not maternal) challenging play blunted later preschool anxiety (Möller et al., 2016). Challenging play is defined as play that “encourages [the] child to exhibit risky behavior or to go outside his/her comfort zone” (Möller et al., 2016, p.19). Overall, results suggest increased efforts to include fathers in this area of work could enhance our understanding of risk for later child anxiety disorders broadly.

Particular parenting practices are explicitly anxiogenic, such as the parental modeling of anxiety (see Appendix A). Early observational, mother-only studies found “high” fearful infants modeled their mothers’ anxious behaviors (De Rosnay, Cooper, Tsigaras & Murray, 2006; Gerull & Rapee, 2002). Recent studies that included both mothers and fathers demonstrated that offspring across the age spectrum modeled fathers’ (but not mothers) anxiety, particularly in dangerous situations (Möller, Majdandžić, & Bögels, 2014) or when youth themselves are highly anxious (Bögels, Stevens, & Majdandžić, 2011). Contrarily, a recent study found, among children high (but not low) in BI, *both* mothers and fathers’ anxious modeling at age 2.5 predicted more child age 4.5 avoidance of novel objects and adults (Aktar, Majdandžić, De Vente & Bögels, 2017).

Despite earlier studies of mothers only, results suggest that both fathers' and mothers' anxious modeling significantly predict child anxious behaviors.

Overall, compared to mothers' parenting, paternal parenting has long been neglected from research on child development, broadly, and child BI/SW, specifically. This historical oversight of the role of fathers' parenting practices could have resulted in missed intervention opportunities to further reduce risks for later anxiety among children high in BI/SW.

Maternal and paternal anxiety disorders and parenting.

Given the literature on maternal and paternal anxiogenic parenting and later anxiety among children high in BI, one must identify those parents who are most likely to exhibit anxiogenic parenting behaviors in the context of child BI/SW. Specifically, high child BI and anxiety are associated with parental anxiety and mood disorders, particularly parent SAD (Rosenbaum et al., 1992). Parent anxiety also predicts later offspring anxiety (Murray et al., 2009). Importantly, the concordance of parent and child anxiety has been shown to be strongest in the context of moderate to high (not low) child BI (Wichstrøm, Belsky & Berg-Nielsen, 2013). Regarding the intergenerational transmission of anxiety, Murray and colleagues (2009) theorize that anxious parents transmit risk to offspring through biological/genetic vulnerability (e.g. child BI), child lifestyle/socialization processes, and parental anxiolytic modeling/information transfer. Murray and colleagues' (2009) also propose that child BI predicts overinvolved parenting, which has bi-directional relations with later child anxiety, and parent anxiety *raises the risk* for overinvolved parenting and reduced child encouragement (Figure 2).

Indeed, empirical studies with young children, while few, show that maternal anxiety disorders are linked to anxiogenic behaviors. Compared to that of both non-anxious mothers and mothers with GAD, mothers with SAD exhibited more expressed anxiety toward their 5-year-old offspring during observed social threat (i.e. giving a speech); however, there were no parenting differences between mothers with and without SAD in response to non-social threat (Murray et al., 2012). Thus, maternal behaviors may depend on the congruence between the context (i.e. social versus non-social) and the dimension of maternal fear (i.e. SAD versus GAD). Among 5- to 8-year-old youth, more maternal criticism and less autonomy granting predicted later anxiety among offspring of anxious mothers, but not among offspring of non-anxious mothers (Ginsburg, Grover, & Ialongo, 2005). Similarly, among 4- to 5-year-old youth, compared to that of non-anxious mothers, mothers with SAD verbalized higher threat attribution and lower encouragement. Such parenting then predicted increased offspring anxiety, specifically among children high in observed BI (Murray et al., 2014). Results, again, suggest that reducing anxiogenic parenting behaviors may be an effective intervention target, specifically for families of mothers with anxiety, as children high in BI from such families may be at greater risk for negative outcomes.

Relatedly, fathers' *clinical* anxiety has also been shown to predict anxiogenic parenting (Bögels & Phares, 2008). Among youth 6-12 years old, maternal anxiety disorders cross-sectionally predicted greater reinforcement of child anxious behaviors, whereas paternal anxiety disorders correlated with more overcontrol (Teetsel, Ginsburg, & Drake, 2014). Among toddlers, maternal and paternal anxiety disorders (including SAD) both predicted observed parent anxious modeling (i.e. facial, bodily and verbal

expressions of anxiety; Aktar, Majdandžić, De Vente & Bögels, 2014). Moreover, among 8- to 18-year-old youth, compared to families without anxious fathers, when fathers had clinical anxiety, *both* parents were more rejecting, and fathers were more overcontrolling. However, no parenting differences were found between families of mothers with and without anxiety (Bögels, Bamelis, & van der Bruggen, 2008).

Thus, results suggest intervening with families of fathers with anxiety may not only improve children's anxiety through reducing paternal rejection and control, but it may also have downstream effects on maternal rejection. The aforementioned studies mostly examined children ages 6 and older or during toddlerhood. By continuing to ignore the role of fathers during the preschool years, we risk investing time, efforts and resources in developing early interventions that may, ultimately, miss valuable points of intervention.

**Treatments for Preschool and Young Children with or at Risk for Anxiety
Evidence-based treatment of child/adolescent anxiety disorders.**

Given the extant literature on child BI/SW and later anxiety, both maternal and paternal anxiogenic parenting and peer problems are all compelling targets for intervention among children high in BI/SW. *Cognitive Behavioral Therapy* (CBT) is an efficacious, psychosocial treatment for anxiety disorders in school-aged youth (Weisz et al., 2017). Child CBT programs commonly include psychoeducation, cognitive restructuring, graduated exposure, contingency management, relaxation training, and social skills training (Rapee, Schniering & Hudson, 2009). Parental involvement varies across child anxiety CBT programs, ranging from conceptualizing parents as co-

therapists to treating parents as co-clients with parents' own individual sessions (Breinholst, Esbjorn, Reinholdt-Dunne, & Stallard, 2012).

Parent anxiety and treatment of child/adolescent anxiety disorders.

However, given that parent anxiety raises the risk for overinvolved parenting and reduced encouragement, which is linked to offspring anxiety (Murray et al., 2009), investigators have long postulated that parent anxiety may also thwart the progress of CBT among youth ages 7-17-year-old. Characteristics of parent anxiety, such as parental avoidance, anxious modeling, accommodation of children's anxieties, and parental threat biases, are antithetical to the principles taught in CBT (Cobham, Dadds, & Spence, 1998).

Indeed, among youth ages 7-17, compared to results of studies that used questionnaires to assess parent anxiety, studies that included the use of clinical interviews indicated that parent anxiety disorders predicted worse child anxiety outcomes at post-treatment and follow-up (Hudson et al., 2014; Bodden et al., 2008; Kendall, Hudson, Gosch, Flannery-Schroeder & Suveg, 2008; Cooper, Gallop, Willetts, & Creswell, 2008; see Wood, Piacentini, Southam-Gerow, Chu, & Sigman, 2006 for an exception). One study specifically found that maternal SAD (not GAD) predicted worse child anxiety treatment outcomes among 6- to 15-year-old youth (Cooper et al., 2008). Thus, youth anxiety treatment success may depend upon dimensions of maternal social fear.

Regardless of parent anxiety assessment methods, results suggest paternal and maternal anxiety may have separate, direct effects on child treatment outcome among 7-17-year-old youth. Kendall and colleagues (2008) *separately* examined the effects of clinician-rated maternal and paternal anxiety disorders. Youth ages 7-17 in *family* CBT

improved more on child-reported anxiety symptoms at follow-up than youth in *individual* CBT if fathers (not mothers) experienced anxiety. However, maternal anxiety disorders predicted retaining child anxiety diagnoses at follow-up regardless of treatment condition (Kendall et al., 2008). Further, among 7-17-year-old youth, when examined *separately*, studies have found that higher baseline self-reported paternal (not maternal) anxiety symptoms predicted higher post-treatment parent-and child-rated child anxiety symptoms (Rapee, 2000), slower decrease in parent-and child-rated child anxiety symptoms (van Steensel, Zegers & Bögels, 2017), and lower likelihood of recovery from child anxiety (Liber et al., 2008). Thus, mothers and fathers' anxiety have *separate* effects on child anxiety treatment outcomes. However, none of these studies examined maternal and paternal anxiety variables in the same model. Thus, it is currently unclear whether maternal or paternal anxiety, or both, are *uniquely* linked to reduced treatment effects, which have high implications for intervention efficacy.

Developmental considerations for preschool anxiety treatment.

BI/SW emerge early in development, so preschool is an ideal intervention period. Preschool is also a highly malleable developmental stage, from both a behavioral and neurobiological perspective (Shonkoff et al., 2012). However, special considerations are needed when implementing CBT with preschoolers. CBT for youth ages 7-17 focuses on transferring control from therapists to youth. In contrast, given parents' key role in preschoolers' emotional and physical development, CBT with preschool youth emphasizes transfer of control from therapists to parents (Barmish and Kendall, 2005). Thus, parent characteristics (i.e. anxiogenic parenting and parent anxiety) are especially salient in the success of preschool anxiety treatments. Indeed, researchers have found that

younger children (i.e. <12 years old) benefited more from CBT when parents did not have anxiety, while older children benefited from CBT regardless of parent anxiety status (Bodden et al. 2008; Berman, Weems, Silverman, & Kurtines, 2000), highlighting the importance of parent anxiety and treatment outcome among younger, school-aged children.

However, few researchers have focused on the treatment of preschool anxiety. This may be because CBT relies on cognitive abilities that are beyond the capacity of most preschoolers, such as metacognition, language abilities, theory of mind, higher-order logic, and executive functioning skills (Carpenter, Puliafico, Kurtz, Pincus, & Comer, 2014). Also, compared to CBT for youth ages 7-17, CBT for preschool youth requires higher parent in- and out-of-session involvement, more concrete and age-appropriate language (e.g. “bravery ladder” as opposed to “fear hierarchy”), developmentally-appropriate treatment delivery (e.g. games, cartoons), and more concrete positive and negative reinforcements (e.g. “reward system”) (Carpenter et al., 2014). Further, parents may be less motivated to seek treatment for young children, rationalizing that their children’s difficulties are transitory or are not yet impairing enough. For example, evidence indicates that, with increasing age, counter-normative social behaviors become more noticeable in peer settings, which may explain why SW and peer rejection increases with age (Rubin et al., 2009). Moreover, evidence shows parents are more likely to believe that anxiously withdrawn behaviors among preschoolers are transient, whereas the same behaviors, as exhibited by 7-year-olds, are more likely attributed, by parents, to stable constitutional, dispositional causes (Rubin, Mills, & Krasnor, 1989; Rubin & Mills, 1992; Mills & Rubin, 1993). Thus, these

developmental adaptations are important to consider as the literature on anxiety treatments for young children and preschoolers is reviewed below.

Interventions for young children with clinical anxiety disorders.

Hirshfeld-Becker and colleagues (2010) were the first to adapt a well-established child and adolescent CBT anxiety program (Kendall & Hedtke, 2006a). They tested “Being Brave” for 4-7-year-old children with clinical anxiety using a randomized controlled trial (RCT) design. The first 6 of 20 total sessions were parent-only, and the remaining were parent-child focused. Compared to those in waitlist condition, children in “Being Brave” experienced less clinician-rated anxiety, measured via diagnostic interviews, at post-treatment and one-year follow-up. Interestingly, neither lifetime nor current clinician-rated maternal and paternal anxiety disorders, measured via diagnostic interviews, had effects on post-treatment child anxiety reduction. Perhaps the initial 6-sessions devoted to parents only, as well as the intensive treatment format (20-sessions over 6-months), reduced effects of parents own anxiety symptomatology on child treatment prognosis.

In addition, Parent Child Interaction Therapy (PCIT) has been adapted for child anxiety. PCIT is an evidence-based intervention grounded in attachment, social learning and behavioral theory, originally developed for young children with externalizing behaviors (Eyberg, 1988). Traditional PCIT includes two phases. The first phase, the Child Directed Interaction (CDI), is rooted in attachment theory, and it focuses on building a strong, warm parent-child relationship, which is the foundation for child behavioral change. Parents are taught to refrain from directing their children’s behaviors through using special play therapy skills. The second phase is the Parent Directed

Interaction (PDI), which teaches parents discipline techniques based on operant conditioning principles. The hallmark of PCIT is that parents receive live, in-vivo therapist feedback during interactions with their children. In both phases, parents must meet mastery criteria before moving on to the next phase or ending treatment.

Pincus, Santucci, Ehrenreich and Eyberg (2008) were the first to adapt PCIT for young children with anxiety. Rooted in attachment theory, PCIT is a fitting anxiety intervention, as meta-analytic data show insecure attachment predicts later child anxiety (Colonnesi et al., 2011). Further, because child BI/SW predicts adolescent social anxiety only in the presence of insecure attachment (Lewis-Morrarty et al., 2014), modifying the early parent-child attachment relationship may improve long-term outcomes for children high in BI. Given links between parental overcontrol and intrusiveness and later child anxiety, teaching parents to refrain from directing their children's behaviors during CDI is also clinically fitting. Pincus and colleagues (2008) developed a third phase, the Bravery Directed Interaction (BDI), to target child anxiety symptoms specifically. BDI includes anxiety psychoeducation as well as exposure therapy, the most salient ingredient in anxiety treatments (Weisz et al., 2017). However, Pincus and colleagues' (2008) BDI did not include therapist in-vivo coaching, and BDI was implemented akin to traditional exposure therapy (e.g. parents implemented exposure practices out of session and later reported how exposures went in session with the therapist).

Pincus and colleagues (2008) tested a time-limited version of adapted PCIT for 4- to 8-year-old children with separation anxiety disorder. Compared to those in the waitlist condition, children who received adapted PCIT experienced significant reductions in clinician-rated anxiety severity, measured via diagnostic interviews. Further, Comer and

colleagues (2012) also tested adapted PCIT, known as the CALM program (which included only CDI and BDI), for 3-8-year-old children with any clinical anxiety disorder in a multiple baseline design. Unlike Pincus and colleagues' (2008) treatment, BDI in Comer and colleagues' (2012) study included therapist in-vivo coaching during parent-facilitated exposures. Immediate feedback may be especially salient when parents are implementing exposures, as immediate reinforcement is a stronger predictor of behavioral change than that of delayed feedback (Skinner, 1953). Results showed children experienced reductions in clinician-rated child anxiety disorders (measured via diagnostic interviews). These results are awaiting replication with a RCT design.

While "Being Brave" (Hirshfeld-Becker et al., 2010) and PCIT adaptations (Pincus et al., 2008; Comer et al., 2012) included both parents and children in treatment, most anxiety treatments for young children have included parents-only (Cartwright-Hatton et al., 2011; van der Sluis, van der Bruggen, Brechman-Toussaint, Thissen, & Bögels, 2012; Donovan & March, 2014), and all such studies demonstrated post-treatment reductions in child anxiety symptoms. In the sole child-only program, Barrett and colleagues (2015) tested their universal prevention program, "Fun Friends," with 5-7-year-old children with clinical anxiety in an open trial. Children in "Fun Friends" experienced decreases in clinician-rated child anxiety (obtained via diagnostic interview). Surprisingly, in this study, self-reported maternal and paternal anxiety symptoms also did not separately predict child treatment outcomes. "Fun Friends" was primarily child-only, and parent anxiety may be less relevant when parents are not directly involved in treatment. Further, the questionnaire used to assess parent's own anxiety in this study only assessed symptoms in the past week.

Thus, given the success of combined parent-child (Hirshfeld-Becker et al., 2010; Pincus et al., 2008), parent-only (Cartwright-Hatton et al., 2011) and child-only groups (Barrett et al., 2015), investigators have started to test whether *combined* parent-child treatments would outperform either treatment alone for young children with anxiety (Waters, Ford, Wharton & Cobham, 2009; Monga, Rosenbloom, Tanha, Owens, & Young, 2015). Monga and colleagues (2015) demonstrated, in an RCT, that compared to those in parent-only treatment, 5-7-year-old children in *combined* parent-child treatment experienced more improvements in clinician-rated child functioning and child anxiety at post-treatment and one year later. Results highlight the value of an added child-based group for young children with anxiety. In this study, as self-reported parent anxiety symptoms decreased, parent-reported child anxiety symptoms also decreased. However, mean-levels of parent anxiety symptoms (measured with questionnaires) were in the normative range (Study Mean(SD) = 6.5(5.8), whereas a score of “35” is in the “concerning” range). Thus, it is possible clinician-rated parent *clinical* anxiety may affect young children’s anxiety treatment outcomes differently.

Further, in addition to child anxiety symptom reduction, some of these studies have demonstrated both parent-reported (Barrett et al., 2015) and teacher-reported (van der Sluis et al 2012) reductions in BI at post-treatment and one year later (Barrett et al., 2015). However, in Hirshfeld-Becker and colleagues’ (2010) study, neither parent-reported nor observed BI improved following treatment. Compared to children without BI, children high in observed BI (in the laboratory setting) were less likely to experience anxiety remission following treatment, suggesting BI moderated treatment outcome in “Being Brave” (Hirshfeld-Becker et al., 2010). Thus, available treatments may be less

efficacious for children high in BI.

Overall, current evidence suggests that anxiety psychosocial treatments for young children, particularly those that directly target both parents and children in treatment, are efficacious in reducing child clinical anxiety disorders.

Preventative interventions for preschoolers and young children at risk for anxiety disorders.

Given the adverse impairments associated with anxiety, prevention and early interventions may be equally, if not more, important to develop and evaluate than that of interventions that target children who already have clinical anxiety. Given that one-third of school-age children with clinical anxiety do not benefit from CBT (Silverman & Ollendick, 2005), one way to improve treatment prognosis may be to intervene *earlier* in the disease course. One preventative intervention study with school-age children at risk for anxiety (based on parent anxiety disorders) found that, none of the children in the preventative intervention developed anxiety disorders one year later, compared to 30% of children in the waitlist control condition (Ginsburg, 2009). Thus, prevention and early interventions have the potential to alter the developmental trajectory of at-risk youth.

Given that one-third of children high in BI develop anxiety disorders, several prevention and early intervention programs have been developed for preschoolers and young children *at risk* for anxiety. These programs targeted children based on high BI (Rapee, Kennedy, Ingram, Edwards, & Sweeney, 2005, 2010; Chronis-Tuscano et al., 2015; Coplan, Schneider, Matheson, & Graham, 2010), high BI and parent anxiety (Kennedy, Rapee & Edwards, 2009; Lau, Rapee & Coplan, 2017), parent clinical anxiety

only (Cartwright-Hatton et al., 2018), or high child anxiety/withdrawal symptoms (LaFreniere & Capuano 1997).

LaFreniere and Capuano (1997) developed the first, 20-session, home intervention for parents of anxious/withdrawn 4.5-year-old children ($N=43$). Treatment focused on child development psychoeducation, parent training, and parent social support. Compared to those in the waitlist condition, those in the intervention group demonstrated improvements in teacher-reported child social competence, observed maternal overcontrol, and observed child behavior. Results highlighted that changing parent behaviors alone resulted in improvements in teacher reported and observed child behaviors. However, there were no differences in teacher-rated anxious/withdrawn symptoms at post-treatment, as symptoms decreased in both conditions.

While efficacious, LaFreniere and Capuano's (1997) intervention was quite demanding (e.g. individual format, home-based, 6-months, 20-sessions). Thus, grounded in a sustainability framework, Rapee and colleagues (2005) developed a parent-only, 6-session group preventative intervention for 3-5-year-old children high in BI known as the "Cool Little Kids" (CLK). CLK focused on teaching parents about anxiety psychoeducation, reducing overprotective parenting, implementing exposure therapy, restructuring parent cognitions, and problem-solving for the future (e.g., school transitions). Compared to those in the waitlist condition, children in CLK showed reductions in clinician-rated child anxiety (based on clinical interview) at post-treatment (i.e. 12-months; Rapee et al., 2005), 3 years later (Rapee et al., 2010), and even 11 years later (although this only applied to females) (Rapee, 2013). Surprisingly, change in child BI (defined as a latent variable based on parent-report and observed measures) did not

mediate the relation between treatment condition and child anxiety (Rapee et al., 2005), and, despite improvements in anxiety, there were no changes in parent-reported BI at post-treatment.

However, 50% of youth continued to meet criteria for anxiety one-year following CLK. Thus, Kennedy and colleagues (2009) tested a more targeted program for families of 3-5-year-old children with high BI and parents with current clinical anxiety disorders. Their program included CLK (Rapee et al., 2005) and two additional sessions targeting parents' own anxiety (e.g. parental exposure therapy). At post-treatment, compared to those in the waitlist control condition, families in modified CLK showed decreases in clinician-rated child anxiety, parent-reported child life interference and parent-rated and observed child BI. Surprisingly, when examined *separately*, self-reported current maternal and paternal anxiety symptoms did not change and did not show group by time interaction effects. Though presence of clinician-rated current parent anxiety disorders was a study inclusion criterion, analyses operationalized mothers' and fathers' anxiety using self-reported anxiety symptoms measured with questionnaires, and mean levels of parent anxiety using this questionnaire were in the normal to moderate range. Thus, it is unknown whether lifetime maternal and paternal *clinical* anxiety disorders impact early interventions for preschool BI/SW differently.

Relatedly, a recent study examined a one-time (5-hour), parent-only group intervention for young children ($M_{age} = 5.50$, $SD = 1.99$) of parents with anxiety, who were seen at adult mental health clinics (N=100; Cartwright-Hatton et al., 2018). Compared to young children whose parents received treatment as usual, young children of parents in the intervention group were less likely to develop anxiety (based on

diagnostic interview) at one-year follow-up (i.e. 60% versus 51%). Results are promising with regard to cost and time, and results await replication in a larger, methodologically-rigorous trial.

Most preventative interventions for preschool and young children at risk for anxiety only involve parents. However, as reviewed herein, child BI/SW is also associated with negative social functioning, such as poor social skills, peer rejection/victimization, which are associated with loneliness, anxiety, and depression (Rubin et al., 2009). Thus, to target these child-peer deficits, Coplan and colleagues (2010) developed “Social Skills Facilitated Play (SSFP),” a child-only social skills group for 4-5.5-year-old children high in BI/SW. SSFP uses developmentally-appropriate didactic components that teach children relaxation, emotion identification, and problem solving skills. Children also practice approach behaviors in the peer group context. Compared to those in the waitlist condition, children in SSFP demonstrated improvements in observed child reticent and social behaviors, but there were no group differences in teacher-reported child behaviors. SSFP has also been implemented in Mainland China with socially withdrawn children (Li et al., 2016), and results were favorable.

Prevention programs reviewed thus far either focus on parents-only (Rapee et al., 2005; Kennedy et al., 2009; Cartwright-Hatton et al., 2018; LaFreniere & Capuano, 1997) or on children-only (Coplan et al., 2010; Li et al., 2016). However, given that Rubin’s developmental transactional theory recognizes both child-peer (e.g. rejection) and child-parent (e.g. overcontrol) factors in the child BI/SW to later anxiety pathway (Rubin et al., 2009), combined parent-child treatment that targets both domains may be

most beneficial. Further, the only study of young children with *clinical* anxiety disorders found combined parent-child treatment outperformed parent-only treatment (Monga et al., 2015). Thus, two studies, to date, have examined combined parent-child treatments for those *at risk* for anxiety by virtue of *preschool* BI/SW (Lau et al., 2017; Chronis-Tuscano et al., 2015). Lau and colleagues (2017) compared combined CLK (Rapee et al., 2005) for parents and SSFP (Coplan et al., 2010) for children to CLK only for parents in a *quasi-experimental* design for 3-5.5-year-old children high in BI and mothers with anxious and depressive symptoms (N=72). Compared to those in CLK alone (N = xx), children in combined CLK and SSFP (N = xx) experienced more reductions in clinician (not mother) rated-anxiety severity (Lau et al., 2017). However, due to the *quasi-experimental* design, only tentative inferences regarding the increased efficacy of combined parent-child treatment can be made.

In the second study to examine combined parent-child interventions, Chronis-Tuscano and colleagues (2015) developed the “Turtle Program,” a multi-modal intervention for *preschool* BI/SW. The “Turtle Program” included SSFP (Coplan et al., 2010) and targeted the peer context by teaching preschoolers social skills and emotion regulation. The parent component included a group PCIT program aimed at reducing multiple anxiogenic parenting behaviors (e.g. overcontrol, parent modeling of anxiety, parent-child attachment relationship, parental reinforcement of avoidance). In a pilot RCT (N=40), results demonstrated that, compared to those in the *waitlist* condition, families randomized to receive the “Turtle Program” demonstrated improvements in parent- and teacher-reported child anxiety symptoms, parent-reported BI, and observed parenting behaviors (e.g., xxxx). Notably, compared to those in the waitlist condition,

children in the “Turtle Program” exhibited more (than who?) observed peer initiations in the classroom setting following treatment; this study is among the first to show observed treatment generalizations to school settings (Barstead et al., in press).

Thus, based on the few preventative interventions with preschoolers that exist, the evidence thus far is promising. Most preschool anxiety preventative interventions either target anxiogenic parenting (Rapee et al., 2005, 2010; Kennedy et al., 2009) or child social skills (Coplan et al., 2010; Li et al., 2016). However, thus far, it is only (???) combined parent-child treatment that demonstrates improvements in child anxiety symptoms across contexts (e.g. home and school) based on multi-informant, multimodal assessments (Chronis-Tuscano et al., 2015; Barstead et al., in press).

Gaps in the Literature

Despite our best efforts to develop preschool prevention and early intervention programs, 50% of preschool children continue to meet criteria for anxiety disorders one-year following the most rigorously-tested preschool BI treatment (Rapee et al., 2005). This highlights the need to further develop more targeted, effective interventions. While Rapee and colleagues’ (2005) treatment is parent-only, rooted in the developmental transactional model (Rubin et al., 2009), treatments that reduce *both* peer and parent risk factors may produce more optimal outcomes (Chronis-Tuscano et al., 2015; Lau et al., 2017). However, to date, only one *quasi-experimental* study compared parent-only treatment to parent-child treatment for *preschool BI* (Lau et al., 2017). Further, one other study compared parent-only treatment to combined parent-child treatment, but young children were already diagnosed with clinical anxiety (Monga et al., 2015); thus, no study has tested this comparison of treatments for *preschoolers* at risk for anxiety due to *BI/SW*

using a true *experimental* design.

Further, several gaps exist in the literature. First, the extant literature on interventions and preventions for young children with or at risk for anxiety largely suggests no significant effects of parents anxiety on treatment outcomes (Hirshfeld-Becker et al., 2010; Kennedy et al., 2009; Barrett et al., 2015). However, the current data are limited by lack of information about parents with clinical anxiety (Monga et al., 2015; Kennedy et al., 2009) and primary reliance on self-report questionnaires to measure parent anxiety (Kennedy et al., 2009; Monga et al., 2015; Barrett et al., 2015). Thus, studies that use more methodologically rigorous assessments of parental clinical anxiety are needed.

Second, given the long-standing focus on mothers' anxiety, but recent, increased attention to fathers' anxiety, isolating whether maternal and paternal anxiety are *uniquely* related to child treatment prognosis is imperative. However, current treatment studies with preschool and young children either combine mothers' and fathers' anxiety into one variable (Monga et al., 2015) or examine maternal and paternal anxiety in separate statistical models (Kennedy et al., 2009; Barrett et al., 2015; Hirshfeld-Becker et al., 2010). However, due to both theoretical and statistical issues (e.g. covariation), studies that examine models with mother and father variables together, rather than separately, are needed (Roggman, Bradley, & Raikes, 2013). Such studies could further our understanding of *unique* effects of maternal and paternal anxiety on early intervention outcomes, paving the way for more targeted interventions.

Third, based on theoretical, developmental, and clinical considerations, compared to parent-only treatment, intensive parent-child treatment may be more beneficial for

children with BI/SW and multiple other risk factors, such as parental anxiety, which raises the risk for anxiogenic parenting. Among older youth, parent anxiety is often cited as a risk factor for reduced treatment efficacy. However, few studies have examined whether treatment format attenuates the associations between parent anxiety and reduced child treatment outcomes. Such information aids our understanding of which treatments are appropriate for which families.

The Current Study

To address these gaps in the literature, the current study sought to examine clinician-rated maternal and paternal clinically-significant anxiety disorders as predictors of treatment outcome in a large RCT comparing combined parent-child intervention, the “Turtle Program” (Chronis-Tuscano et al., 2015) to parent-only intervention, CLK (Rapee et al., 2005), for preschool BI/SW among 3.5-5 year old children. The current study examined maternal and paternal anxiety disorders separately and together, in order to determine the *unique* effects of each.

Further, the current study also sought to examine whether maternal and paternal anxiety disorders have different associations with child treatment outcome as a function of treatment format. From a public health perspective, CLK is more resource-efficient, and it has demonstrated efficacious results that have held up 11-years later. However, CLK works well for 50% of children, but the other 50% continue to exhibit clinical anxiety following treatment (Rapee et al., 2005). Thus, the “Turtle Program,” which targets both anxiogenic parenting and child social deficits through in-vivo therapist coaching and social exposures conducted within the peer context, respectively, may be more effective at mitigating the effects of parent anxiety on child treatment outcome.

For this dissertation study, the specific aims and hypotheses were as follows:

Aims and Hypotheses

Aim 1: To examine the separate and *unique* roles of maternal and paternal lifetime anxiety disorders (ADs) as predictors of child treatment outcome across both treatment conditions, the Turtle Program and CLK.

Hypothesis 1a (H1a): Several theoretical models, as well as developmental and clinical studies, highlight the negative effect of maternal anxiety on later child anxiety among inhibited youth (e.g., Murray et al., 2014). Recent research has further demonstrated similar negative effects of fathers' anxiety and later offspring anxiety (Bögels & Phares, 2008), and the negative effects of both mothers' and fathers' anxiety disorders on child anxiety treatment outcome among school-aged youth (e.g. Kendall et al., 2008). **In separate models, it was hypothesized that maternal and paternal lifetime ADs would each separately predict worse child anxiety outcomes regardless of treatment condition (H1a).**

Hypothesis 1b (H1b): Further, given the recent research on the relations among paternal anxiety, parenting, and child anxiety outcomes among youth with and without BI/SW, **it was hypothesized that fathers' lifetime ADs would continue to predict child treatment outcome, even when mothers' lifetime ADs was considered. It was less clear whether mother's lifetime ADs would predict negative outcomes beyond father's lifetime ADs (H1b).** On the one hand, mothers' anxiety, and subsequent anxiogenic parenting, are (?) highly cited as a predictor of offspring anxiety (Ginsburg et al., 2005, 2009). On the other hand, among studies that examined both parents' anxiety, fathers' (not mothers') anxiety predicted reduced anxiety treatment outcome among

school-age children (Rapee et al., 2000; van Steensel et al., 2016; Liber et al., 2008).

Aim 2: To examine whether the associations between maternal and paternal lifetime ADs and child anxiety treatment outcomes differ as a function of treatment conditions.

Hypothesis 2: Fifty percent of preschoolers high in BI continue to exhibit anxiety one-year following the best-available, current treatment, CLK (Rapee et al., 2005). Parent anxiety raises the risk for anxiogenic parenting (Murray et al., 2009). Thus, a more intensive, hands-on treatment, like the “Turtle Program,” where parents receive in-vivo therapist coaching during social exposures with their children and same-age peers, and where children also receive direct instruction and feedback within the peer context, may be more beneficial for families where parents experience anxiety themselves. Other researchers have found that more intensive treatment formats weaken the association between parent anxiety and reduced child treatment outcome (Kendall et al., 2008). **Thus, it was hypothesized that maternal and paternal lifetime ADs would have weaker associations with child treatment outcome in the “Turtle Program” compared to CLK (H2).**

Exploratory Aims:

Exploratory Aim 1: To examine whether lifetime maternal and paternal *social* anxiety disorders (SAD), examined both continuously and dichotomously, predict child anxiety treatment outcome.

Exploratory Hypothesis 1: SAD is the most common disorder among parents of children with BI and anxiety (Rosenbaum et al., 1992), and SAD is also heritable (Isomura et al., 2015). Child BI/SW predicts later social anxiety specifically (Clauss &

Blackford, 2012; Chronis-Tuscano et al., 2009). Further, Cooper et al. (2008) demonstrated that maternal SAD (not GAD) negatively predicted anxiety treatment outcomes among 6- to 15-year-old children. Further, paternal SAD (but not other anxiety disorders) specifically predicted observed fear/avoidance among 2.5-year-old children in an experimental paradigm (Aktar et al., 2014). Both the Turtle Program and CLK also focused on social exposures exclusively. Inherent in all social exposures are interactions with other people. Thus, parent SAD (rather than GAD, for example) may be more relevant to implementing child social (rather than non-social) exposures, given that parents, too, must face these social interactions. **Thus, it was hypothesized that maternal and paternal SAD would each predict worse child anxiety outcomes regardless of treatment condition.**

Exploratory Aim 2: To examine whether the associations between maternal and paternal SAD and child anxiety treatment outcomes differ as a function of treatment condition.

Exploratory Hypothesis 2: Compared to CLK, the “Turtle Program” provides more therapist scaffolding during parent-led child social exposures, due to opportunities for in-vivo coaching in the context of peer, social situations, and it also provides child social skills training with direct peer groups. Given that these additional treatment components may potentially ameliorate characteristics of parent SAD (e.g. avoidance) that are antithetical to CBT, **it was hypothesized that maternal and paternal lifetime SAD would have weaker associations with child treatment outcome in the “Turtle Program” compared to in CLK.**

Chapter 2: Method

This dissertation was drawn from a large ongoing, NIMH-funded R01 comparing two early intervention programs for child BI/SW (Title: Multi-Component Early Intervention for Socially Inhibited Preschool Children; PIs: Andrea Chronis-Tuscano, PhD and Kenneth Rubin, PhD). Research protocols and measures irrelevant to the current dissertation are not described.

Participants

Children, ages 45-64 months, and their custodial caregivers were recruited from local sources (e.g. schools, pediatrician offices, community organizations, newspapers, social media). For the current sample, 125 families were consented, and 111 families were randomized to treatment (CLK: n=55; Turtle Program: n=56). See Figure 1 for the CONSORT diagram.

The median annual household income was $\geq 150,000$ \$. Child demographics were as follows: 48% male, $M_{\text{age}} = 52.8$ months ($SD=5.7$), 6% Hispanic/Latino, 13% Asian, 13% African American, 53% Caucasian, and 21% Other (e.g., biracial).

When only one parent participated in the study, that parent was deemed the primary parent. When two parents participated, the parent who engaged in more child care duties was deemed the primary parent (mothers: 87%; fathers: 13%), and the other parent was deemed the co-parent.

Demographics for mothers were as follows: $M_{\text{age}} = 38.4$ years old ($SD=4.8$), 6% Hispanic/LatinX, 19% Asian, 13% African American, <1% Hawaiian/Pacific Islander, 64% Caucasian, and 3% Other. Further, 95% were biological mothers and 5% were adoptive mothers. Additionally, 24% earned doctoral degrees or equivalent, 42% earned

master's degrees or equivalent, 26% earned bachelor's degrees or equivalent, and 8% completed three years of college or less. Also, 91% were married, 4% were divorced, and 5% were never married.

Demographics for fathers were as follows: $M_{\text{age}} = 40.3$ years old ($SD=5.8$), 7% Hispanic/Latino, 10% Asian, 13% African American, 2% Pacific Islander, 72% White, 3% Other. Further, 96% were biological fathers and 4% were adoptive fathers. Additionally, 26% earned doctoral degrees or equivalent, 35% earned master's degrees or equivalent, 31% earned bachelor's degrees or equivalent, and 8% completed three years of college or less. Regarding marital status, <1% identified as never married, 97% married, <1% separated, and <1% divorced.

Inclusion & Exclusion criteria

For inclusion, children were required to: (1) be between 45-64 months, (2) be attending structured school settings (e.g., daycare, preschool), (3) be in the 85th percentile on the Behavioral Inhibition Questionnaire (BIQ; Bishop, Spence, & MacDonald, 2003), and (4) have custodial parent(s) who consented to participate. Children could meet diagnostic criteria for attention-deficit/ hyperactivity disorder (ADHD), oppositional defiant disorder (ODD) or conduct disorder (CD).

Regarding exclusion criteria, children were required not to: (1) be diagnosed with pervasive developmental disorder (PDD) or mental retardation (MR), (2) score ≥ 15 on the Social Communication Questionnaire (SCQ; Rutter, Bailey, & Lord, 2003; Eaves, Wingert, Ho, & Mickelson, 2006), (3) be in any other anxiety treatment during the treatment phase, and/or (4) meet diagnostic criteria for selective mutism at baseline,

which was assessed with a screening measure first, and then a clinical interview when warranted.

Measures

Screening measures.

The *Behavioral Inhibition Questionnaire* (BIQ; Bishop, Spence & McDonald, 2003) is a 30-item, parent report form that assesses behavior of 3- to 5-year-old children in a range of situations, including peers, adults, separation, performance, novel settings and novel people. The BIQ shows satisfactory test-retest reliability and good internal consistency (Bishop et al., 2003). The BIQ is significantly correlated with measures of observed BI and the parent-report version shows good convergent and divergent validity with similar and distinct traits, respectively (Kim et al., 2011).

The *Social Communication Questionnaire* (SCQ) is a 40-item, parent-report screening form that assesses communication skills and social functioning in children 4 and older who may have autism spectrum disorders (Eaves, Wingert, Ho & Mickelson, 2006; Rutter, 2006). It is based on the Autism Diagnostic Interview-Revised (ADI-R). The SCQ shows strong sensitivity and adequate specificity between children with and without ASD (Allen, Silove, Williams, & Hutchins, 2007). Further, the SCQ shows good internal consistency and construct validity with other autism measures (Ung et al., 2016).

Child anxiety assessment. The Anxiety Disorders Interview Schedule for Children for DSM 5—Parent Version (ADIS-P) is a gold-standard child diagnostic interview. In the current study, it was administered to the primary parent to assess for current child anxiety (separation, specific, social, GAD, selective mutism, panic) and associated disorders (depression, dysthymia, ADHD, ODD, CD) based on DSM-5

criteria. Past versions demonstrate good to excellent test-retest reliability (Silverman & Albano, 1996), inter-rater reliability (Lyneham, Abbott & Rapee, 2007), and concurrent validity (Wood, Piacentini, Bergman, McCracken, & Barrios, 2002). At baseline (hereinafter referred to as “T1”), all disorders were assessed. At post-treatment (hereinafter referred to as “T2”), only child anxiety disorders were assessed, consistent with that of other anxiety intervention studies with young children (Hirshfeld-Becker et al., 2010). Clinicians rated both disorder presence/absence and impairment via the Clinician Severity Rating (CSR) (range: 0-8, with ≥ 4 indicating clinically-significant impairment).

The outcome variables examined were (1) T2 child total anxiety, defined as the sum of all ADIS-P anxiety CSRs assessed at T2 (Ginsburg, Drake, Tein, Teetsel & Riddle, 2015; Teetsel et al., 2014); and (2) T2 child social anxiety CSR. Hereinafter, these two outcome variables are referred to as “T2 child outcome variables.”

Parent anxiety assessment. The Anxiety Disorders Interview Schedule for DSM 5—Adult & Lifetime Version (ADIS-5L) is a semi-structured adult clinical interview. In the current study, it was administered to mothers (n=99) and fathers (n=84) to assess for both current and lifetime adult anxiety (*panic, GAD, social, separation, specific*) and depression based in DSM-5 criteria (Brown & Barlow, 2014). Past versions show good reliability (Di Nardo, Moras, Barlow, Rapee, & Brown, 1993) and inter-rater agreement (Brown, DiNardo, Lehman, & Campbell, 2001). Clinicians rated disorder presence/absence and impairment with the CSR (range: 0-8, with ≥ 4 indicating clinically-significant impairment) for *both* the current and lifetime period. In the current study, maternal and paternal lifetime ADs were defined as total number of current *or* lifetime

clinically-significant anxiety disorders (range: 0-5). In exploratory analyses, maternal and paternal SAD was defined as presence of either current *or* lifetime SAD (dichotomous measure), and the highest CSR for social anxiety for either lifetime *or* current (continuous measure).

Inter-rater agreement was assessed with 10 ADIS-5L and 10 ADIS-P interviews. Agreement was defined as matching on (1) presence/absence of disorder and (2) CSRs within 1 point. For the ADIS-5L, there was 97% agreement. For the ADIS-P, there was 99.9% agreement. There was 100% agreement on SAD for both ADIS-5L and ADIS-P interviews.

Maternal and Paternal Attendance. Examined as a covariate, attendance was defined as the percentage of sessions attended in-person during the scheduled time or, for families who missed the scheduled time, sessions that were made-up through phone calls or outside-session therapist coaching (for the Turtle Program) (mother attendance: CLK 88%, Turtle 91%; father attendance: CLK 34%, Turtle 49%).

Parent Responsibility Scale (PRS), Part 1 and 2. Examined as a covariate, father involvement in caregiving was examined with the PRS-1 and -2, which assesses 14 common child rearing tasks completed by mothers and fathers of preschool-age children (McBride, Schoppe & Rane, 2002). For the PRS-1, primary parents rated each child rearing task on a 5-point scale (i.e. 1= mother always responsible; 3 = both parents responsible; 5= father always responsible). Higher scores indicated higher paternal involvement, with scores \geq “42” indicating “high” paternal involvement (McBride, personal communication, 2017) (range: 14-70; CLK: 30.8 (SD=8.5), Turtle: 33.10 (SD=5.65). On the PRS-2, primary parents indicated the % of time child care duties were

completed by mothers alone (CLK: 54%, Turtle: 53%), both parents together (CLK: 24%, Turtle: 25%), or fathers alone (CLK: 14%, Turtle: 19%), totaling 100%.

Procedures

All interested participants completed brief telephone screens, during which their demographic and contact information, BIQ and SCQ ratings were obtained. ADIS-P interviews were scheduled with families who met inclusion criteria. Families who were excluded were told reasons for ineligibility and were provided referrals when warranted. Prior to treatment initiation, primary parents completed the ADIS-P. All ADIS-P and ADIS-5L interviews were administered by masked, independent evaluators who were unaware of treatment condition. After the ADIS-P was completed, children were block randomized (based on child sex and number of anxiety disorders) to one of two treatment groups.

Treatment Methods

The Turtle Program. The “Turtle Program” was a time-limited program, and it included eight 90-minute concurrent parent and child group sessions, with 6-7 families participating per group (Chronis-Tuscano et al., 2015 and Danko, O’Brien, Rubin & Chronis-Tuscano, in press).

The parent group was an adaptation of PCIT for child anxiety. Child Directed Interaction (CDI) skills (e.g. differential attention, child directed play, and positive social reinforcement) were taught first. Then, the Bravery Directed Interaction (BDI) skills (e.g. exposure therapy, reinforcement, parental differential attention, and antecedent control) were taught. During both CDI and BDI, parents received in-vivo therapist coaching (e.g. therapist coaching in the context of parent-led child peer exposures). Parents observed

therapist coaching of other families, allowing for vicarious learning. Parent Directed Interaction (PDI) skills (e.g. behavior management techniques) were taught last.

The child group included Coplan and colleagues (2010) “Social Skills Facilitated Play” (SSFP) group. SSFP taught children social skills, interpersonal problem-solving, emotion regulation, and relaxation skills through age-appropriate manners (e.g. games and stories). SSFP also included unstructured peer play, which preceded the didactic components, and, at the end of some SSFP sessions, parents observed children practicing the skill of that week (e.g. balloon breathing).

Cool Little Kids. CLK was developed by Rapee and colleagues (2005, 2010). CLK was a parent-only, psychoeducation program with six 120-minute sessions, and 6-7 families per group. CLK content included understanding the etiology of anxiety (e.g. child BI, anxiogenic parenting), implementing exposure therapy, and restructuring cognitions for parent anxiety.

Importantly, Turtle and CLK were balanced for therapist contact time with parents. For both parent groups, families who missed scheduled, in-person sessions were provided opportunities to obtain the material via phone sessions with therapists prior to the next session. Further, for those in the Turtle Program, missed in-vivo therapist coaching sessions were further provided by scheduling visits with the family outside the scheduled group time.

Training and Quality Assurance. All clinical activities were recorded and supervised by licensed clinical psychologists. Clinical psychology post-doctoral fellows and PhD students led all parent groups (i.e. CLK and the parent component of “Turtle Program”). PhD students in the Department of Human Development and Quantitative

Methodology and advanced Bachelors-level students implemented SSFP. Clinical psychology post-doctoral fellows, PhD- and masters-level clinicians administered all clinical interviews. ADIS-P and ADIS-5L training included the following: conducting practice interviews, receiving live supervision during interviews and, subsequently, achieving interrater agreement with supervisors. Prior to leading groups independently, all group leaders received training in PCIT from a PCIT Master Trainer, met mastery criteria in CDI, and observed all sessions of respective groups and supervisions. Ron Rapee, PhD, and Rob Coplan, PhD, the developers of CLK and SSFP, respectively, also provided feedback on a subset of videotapes.

Data Analytic Plan

Missing Data Analyses

All multiple regression analyses were conducted in *Mplus* Version 8, which accounted for missing data using full information maximum likelihood (FIML) procedures for parameter estimates (Dong & Peng, 2013). Missing data rates for the current sample ranged from 12.5-25%. While acceptable cutoffs for missing data rates have not yet been established, one simulation study demonstrated that, when FIML was used, parameters of datasets with 20% missing data did not significantly differ from that of complete datasets (Dong & Peng, 2013). Thus, FIML was an appropriate missing data approach for the current sample.

Multiple Regression Analyses

All study aims were examined using path modeling. All analyses were conducted separately for each T2 child outcome variable, controlling for T1 child functioning. The following variables were examined for group differences and to see if they predicted T2

child outcome variables: child sex, age, race, and T1 externalizing severity; maternal and paternal education, race, attendance, and involvement (i.e. PRS-1 and -2); and family income. These variables were selected based on their theoretical relations with T2 child outcome variables (Doey, Coplan, Kingsbury, 2014; Rapee et al., 2009). Variables that *significantly* predicted T2 child outcome variables were included as covariates in all analyses.

Aim 1: In order to examine the separate and *unique* roles of maternal and paternal lifetime ADs as predictors of child treatment outcome across both treatment conditions, the effects of maternal and paternal lifetime ADs on T2 child outcome variables were examined, both separately and in the same model, along with any significant covariates and T1 child outcome variables. **Aim 2:** In order to examine whether the associations between maternal and paternal lifetime ADs and T2 child outcome variables differed as a function of treatment condition, multiple group analysis, rather than traditional products of factors, with treatment condition (i.e. Turtle Program versus CLK) as the grouping variable, was used to assess for moderation. Multiple group analysis is more flexible, as it allows for unequal variances between groups (Muthen, 2002); however, traditional product terms in multiple regression assume homogeneity of variances (Judd & Kenny, 2010). **Exploratory Aim:** To examine whether maternal and paternal lifetime SAD negatively predict T2 child outcome variables and to examine whether the associations between maternal and paternal SAD and T2 child outcome variables differed as a function of treatment conditions, the Data Analytic Plan listed under Aim 1 and 2, was repeated, but maternal and paternal lifetime ADs were replaced with maternal and paternal SAD (measured continuously and dichotomously).

To examine significant moderation effects, the Wald χ^2 test was used, which examined whether two unstandardized parameters were significantly different from each other, either in the same model (i.e. the effect of maternal and paternal lifetime ADs on T2 child anxiety across the whole sample; Aim 1) or across different groups (i.e. the effect of maternal lifetime ADs on T2 child anxiety between treatment groups; Aim 2). The Wald χ^2 test is asymptotically equivalent to χ^2 difference testing, where one constrains parameters to equality and compares the χ^2 for the constrained model to that of the unconstrained model (where all parameters are estimated freely) (Muthen, 2011). A significant Wald χ^2 test is conceptually equivalent to statistical moderation and indicates two unstandardized parameters significantly differ from each other.

Relative Importance Analyses

As follow-up analyses to multiple regression, the relative contributions of predictor variables to the overall model R^2 were examined through relative importance analysis (Tonidandel and LeBreton, 2011). Relative importance analyses partition the overall model R^2 by the proportion of R^2 contributed by each predictor in the model *without* making assumptions about the statistical or practical significance of each predictor (Tonidandel and LeBreton, 2011).

Relative importance analyses were chosen over Relative Weight Analyses (RWA; Tonidandel and LeBreton, 2015), where the statistical significance of each predictor to the overall model R^2 is tested, for the following reasons. First, study hypotheses related to the statistical significance and unique roles of maternal and paternal lifetime ADs on T2 child outcome variables (as one sample and between treatment conditions). A priori hypotheses did not address the *relative influence* of maternal versus paternal lifetime ADs

on the model R^2 . Second, RWA-Web, the program used to conduct RWA, handled missing raw data through listwise or pairwise deletion, which (1) reduced the current sample size and (2) produced parameters that were discordant from those produced with FIML.

Thus, relative importance analyses were conducted through correlation matrices, produced via FIML in *Mplus*, to generate raw and rescaled relative weight values using R from RWA-Web (Tonidandel and LeBreton, 2015). Raw relative weights (RWA) are the variances in R^2 attributable to each predictor and rescaled relative weights (RS-RW) are the percentages of the overall model R^2 that are attributed to each predictor. By using correlation matrices produced via *Mplus*, RAW-Web produced parameters consistent with parameters generated through FIML.

Rescaled relative weights (expressed as %) were reported for interpretation purposes. The following statistics were also included: unstandardized beta coefficients (b), standardized beta coefficients (β), standard error of the standardized beta coefficients (SE), and p-values.

Chapter 3: Results

Preliminary Analyses

To satisfy requirements for FIML, variables had to be normally distributed and to be, at minimum, missing at random (Dong & Peng, 2013). Thus, Little's Missing Completely at Random (MCAR) test (Little, 1988) in SPSS v. 23 was used to examine patterns of missing data, which indicated data were missing completely at random, $\chi^2(df=80)=99.739, p=.067$. Further, all data met criteria for univariate normality. All skew and kurtosis statistics were in the acceptable range (≤ 3.0), except for paternal lifetime ADs, which was positively skewed. A square root transformation was used on this variable, which led to acceptable skew and kurtosis (Field, 2009). All analyses involving paternal lifetime ADs hereinafter were conducted using this transformed variable. Multicollinearity analyses were conducted, and all variance inflation factor (VIF) values were around 1 (which is in the acceptable range of .1-10; Field, 2009).

First, group differences in demographic and clinical variables were examined. Results indicated CLK had significantly higher percentages of fathers, $\chi^2(4, N=100) = 14.29, p = .006$, and children, $\chi^2(3, N=111) = 10.986, p = .012$, who identified as African American compared to that of the Turtle Program. Further, fathers in the Turtle Program had marginally higher attendance than those in CLK, $t(100) = -1.84, p = .069$. However, these variables did not significantly predict T2 child outcome variables, so they were excluded from further analyses¹.

¹In separate models, linear regression analyses indicated paternal African American race (T2 child total anxiety: $\beta=.028, p=.790$; T2 child *social* anxiety: $\beta=-.041, p=.696$), child African American race (T2 child total anxiety: $\beta=.003, p=.975$; T2 child *social* anxiety: $\beta=-.056, p=.584$) and father's attendance (T2 child total anxiety: $\beta=-.053, p=.602$; T2 child *social* anxiety: $\beta=.067, p=.510$) did not predict either T2 child outcome variable.

Second, demographic and clinical variables were each tested separately in linear regression analyses to examine if any predicted the T2 child outcome variables. Maternal and paternal age both predicted T2 child total anxiety, such that higher maternal age, $\beta=.199, p=.049$, and higher paternal age, $\beta=.218, p=.037$, predicted higher T2 child total anxiety. Paternal age predicted T2 child *social* anxiety, $\beta=.210, p=.045$, such that higher paternal age predicted higher T2 child *social* anxiety. Thus, maternal and paternal age were included as covariates in all subsequent analyses for T2 child total anxiety, and paternal age was included as a covariate in all subsequent analyses for T2 child *social* anxiety.

Of the 111 randomized families, 14 families (i.e. 12.6% of the sample) did not complete T2 ADIS-P interviews (hereinafter referred to as “non-completers”; See Figure 3 for the CONSORT diagram). Of these 14 non-completers, 5 families dropped out after randomization (2 due to time commitment, 3 due to unknown reasons), 3 families dropped out at session 3 or after, 1 family was lost to follow up after randomization, 2 families were lost to follow up at session 2 or after, 1 family was lost to follow up due to unknown reasons, and 2 families completed the treatment and were lost to follow up for their T2 ADIS-P assessments.

Further follow-up analyses indicated non-completers were more likely to be children who were African American, $z=3.70, p<.05$, and less likely to be children who were “Other” race, $z=-2.10, p<.05$, more likely to be families from lower-income households (i.e. \$12.5K/year), $z=2.40, p<.05$, and less likely to be families from high-income households (i.e. \$150K/year), $z=-2.20, p<.05$, more likely to be mothers who have less than bachelor’s degrees, $z=4.10, p<.05$, and less likely to be mothers who have

graduate degrees, $z=-2.60, p < .05$, more likely to be children with higher T1 child total externalizing severity (i.e. $M_{CSR}=1.917(SD=2.10)$ versus $M_{CSR} = .464(SD=1.36)$), and more likely to be mothers who were African American, $z=2.50, p < .05^2$.

Aim 1: To examine the separate and *unique* roles of maternal and paternal lifetime ADs as predictors of child treatment outcome *across* both treatment conditions.

T2 child total anxiety.

In all three separate models examining maternal and paternal lifetime ADs each separately and then together (Hypothesis 1a, 1b), T1 child total anxiety, paternal and maternal age were included as covariates (see Table 5).

When maternal lifetime ADs was examined alone without paternal lifetime ADs in the model (Hypothesis 1a), only maternal lifetime ADs ($b=.593, \beta=.180, SE=.077, p=.019$) and T1 child total anxiety ($b=.480, \beta=.587, SE=.067, p < .001$) significantly predicted T2 child total anxiety, such that higher maternal lifetime ADs and higher T1 child total anxiety both predicted higher T2 child total anxiety (See Model 1, Table 5). Follow-up relative importance analyses indicated T1 child total anxiety and maternal lifetime ADs contributed 76% and 13%, respectively, to the overall model R^2 .

Next, paternal lifetime ADs was examined alone without maternal lifetime ADs (Hypothesis 1a; see Model 2, Table 5). Only T1 child total anxiety significantly predicted T2 child total anxiety ($b=.505, \beta=.619, SE=.063, p < .001$), such that higher T1 child

² These results indicate follow-up analyses conducted after the omnibus tests. The omnibus test results are as follows: child race ($\chi^2(3, N=112)=15.527, p < .001$), family income ($\chi^2(6, N=108) = 20.647, p=.002$), mom's education ($\chi^2(2, N=108) = 18.097, p < .001$), T1 child externalizing severity ($t(11.752)=-1.897, p=.083$) and maternal race ($\chi^2(4, N=109)=8.076, p=.089$). Further, completers and non-completers did not significantly differ by the following clinical and demographic characteristics: treatment group ($\chi^2(1, N=112) = 1.31, p=.253$), child sex ($\chi^2(1, N=112) = 2.473, p=.116$), child age ($t(110)=-1.685, p=.095$), maternal age ($t(106)=-.458, p=.648$), paternal age ($t(5.243) = -1.025, p=.350$), maternal lifetime ADs ($\chi^2(4, N=99) = 1.142, p=.888$), paternal lifetime ADs ($\chi^2(3, N=84) = .683, p=.877$), paternal education ($\chi^2(2, N=98) = 3.572, p=.168$), paternal race ($\chi^2(4, N=100)=1.470, p=.832$) T1 child total anxiety ($t(109)=-.383, p=.703$), and T1 child social anxiety severity ($t(109)=-.304, p=.762$).

total anxiety predicted higher T2 child total anxiety. Follow up relative importance analyses showed T1 child total anxiety contributed 83% to the overall model R^2 .

Finally, when maternal and paternal lifetime ADs were examined together in the same model (Hypothesis 1b), maternal lifetime ADs ($b=.601$, $\beta=.182$, $SE=.075$, $p=.015$), paternal lifetime ADs ($b=-.869$, $\beta=-.157$, $SE=.078$, $p=.045$), paternal age ($b=.111$, $\beta=.240$, $SE=.122$, $p=.039$), and T1 child total anxiety ($b=.463$, $\beta=.567$, $SE=.069$, $p<.001$) significantly predicted T2 child total anxiety, such that higher maternal lifetime ADs, higher paternal age and higher T1 child total anxiety all predicted higher T2 child total anxiety, but higher paternal lifetime ADs predicted *lower* T2 child total anxiety. Further, follow up relative importance analyses indicated T1 child total anxiety, maternal lifetime ADs, paternal lifetime ADs, paternal age contributed 70%, 12%, 5%, and 8%, respectively, to the overall model R^2 . The effects of maternal and paternal lifetime ADs on T2 child total anxiety significantly differed from one another, Wald $\chi^2(1) = 8.52$, $p=.004^3$.

T2 child social anxiety.

In all three separate models examining maternal and paternal lifetime ADs separately and together, controlling for T1 child *social* anxiety and paternal age in all models (see Model 1-3, Table 5), only T1 child *social* anxiety significantly predicted T2 child *social* anxiety, such that higher T1 child *social* anxiety predicted higher T2 child *social* anxiety (all p -values $<.001$). Follow-up relative importance analyses indicated T1 child *social* anxiety contributed 80-86% of the overall model R^2 across the three models.

³ Given the study aim to examine the *unique* roles of maternal and paternal lifetime ADs, follow-up Wald's χ^2 test were conducted to test the differences between unstandardized regression estimates relating to the effects of maternal and paternal lifetime ADs on T2 child total anxiety.

Aim 2: To examine whether the associations between maternal and paternal lifetime ADs and child anxiety treatment outcomes differed as a function of treatment condition.

T2 child total anxiety.

All analyses included maternal and paternal lifetime ADs, maternal and paternal age, and T1 child total anxiety. In CLK, T1 child total anxiety ($b=.396, \beta=.502, SE=.106, p=<.001$) and maternal lifetime ADs ($b=.804, \beta=.278, SE=.111, p=.014$) significantly predicted T2 child total anxiety, such that higher maternal lifetime ADs and higher T1 child total anxiety both predicted higher T2 child total anxiety (Table 6). Follow up relative importance analyses showed T1 child total anxiety and maternal lifetime ADs contributed 60% and 25%, respectively, to the overall model R^2 .

In the Turtle Program, only T1 child total anxiety was a significant predictor of T2 child total anxiety ($b=.552, \beta=.637, SE=.088, p=<.001$), such that higher T1 child total anxiety predicted higher T2 child total anxiety (Table 6). Follow-up relative importance analyses indicated T1 child total anxiety contributed 80% to the overall model R^2 . However, the relation between maternal and paternal lifetime ADs and T2 child total anxiety did not significantly differ by group (maternal lifetime ADs: Wald $\chi^2(1) = .938, p=.333$; paternal lifetime ADs: Wald $\chi^2(1) = .013, p=.909$).

T2 child social anxiety.

All analyses included paternal age, maternal and paternal lifetime ADs, and T1 child social anxiety. In both treatment conditions, only T1 child social anxiety significantly predicted T2 child social anxiety (p -values $\geq .05$ in both groups), such that

higher T1 child *social* anxiety predicted higher T2 child *social* anxiety (Table 6). Follow up relative importance analyses indicated T1 child *social* anxiety contributed 75% and 80% to the overall model R^2 in CLK and Turtle, respectively. Further, the relation between maternal and paternal lifetime ADs and T2 child *social* anxiety did not differ by treatment condition (maternal lifetime ADs: Wald $\chi^2(1) = .001, p = .969$; paternal lifetime ADs: Wald $\chi^2(1) = .302, p = .582$).

Exploratory Aims:

Exploratory Aim 1: To examine whether maternal and paternal SAD (examined both dichotomously and continuously) predicts child treatment outcome

The following statistics were reported using maternal and paternal SAD measured dichotomously. Results remained unchanged when the dichotomous variables were replaced with the continuous measures (i.e., highest paternal and maternal lifetime SAD CSR). Results using the continuous measure of maternal and paternal SAD can be found in Appendix B, Table 2.

T2 child total anxiety.

In models examining maternal and paternal SAD separately, only T1 child total anxiety significantly predicted T2 child total anxiety, such that higher T1 child total anxiety predicted higher T2 child total anxiety (all p -values $\leq .001$; Appendix B, Table 1). Follow up relative importance analyses indicated T1 child total anxiety contributed 80-85% of the overall model R^2 across the two separate models.

When maternal and paternal SAD were examined together, only maternal SAD ($b = 1.071, \beta = .155, SE = .077, p = .044$) and T1 child total anxiety ($b = .480, \beta = .586, SE = .068, p < .001$) significantly predicted T2 child total anxiety, such that the presence of

maternal SAD and higher T1 child total anxiety predicted higher T2 child total anxiety (Appendix B, Table 1). Follow up relative importance analyses indicated T1 child total anxiety and maternal SAD contributed to 76% and 9% to the overall model R^2 . Further, the effects of maternal and paternal SAD on T2 child total anxiety significantly differed from each other, Wald $\chi^2(1) = 4.127, p = .042$.

T2 child *social* anxiety.

In all three models examining maternal and paternal SAD separately and together, controlling for T1 child *social* anxiety and paternal age in all models, only T1 child *social* anxiety significantly predicted T2 child *social* anxiety, such that higher T1 child *social* anxiety predicted higher T2 child *social* anxiety (all p -values $\leq .001$; See Table 5). Follow up relative importance analyses indicated T1 child *social* anxiety contributed to 78-83% of the overall model R^2 across all three models.

Exploratory Aim 2: To examine whether the associations between maternal and paternal SAD and child anxiety treatment outcomes differed as a function of treatment conditions.

T2 child total anxiety.

In CLK, T1 child total anxiety ($b = .388, \beta = .490, SE = .116, p < .001$) and maternal SAD ($b = 2.342, \beta = .332, SE = .114, p = .004$) significantly predicted T2 child total anxiety, such that the presence of maternal SAD and higher T1 child total anxiety both predicted higher T2 child total anxiety (Table 5). Follow up relative importance analyses showed T1 child total anxiety and maternal SAD contributed 56% and 28%, respectively, to the overall model R^2 .

In the Turtle Program, only T1 child total anxiety was a significant predictor of

T2 child total anxiety ($b=.555$, $\beta=.642$, $SE=.088$, $p<.001$), such that higher T1 child total anxiety predicted higher T2 child total anxiety (Appendix B, Table 1). Follow up relative importance analyses indicated T1 child total anxiety contributed 82% to the overall model R^2 .

The relation between maternal SAD and T2 child total anxiety significantly differed between treatment conditions, Wald $\chi^2(1) = 4.610$, $p=.031$. The effect of paternal SAD on T2 child total anxiety did not differ between treatment conditions, Wald $\chi^2(1) = .054$, $p=.816$.

T2 child *social* anxiety.

T1 child *social* anxiety was the only significant predictor of T2 child *social* anxiety in both groups (all p -values $\leq .001$). Follow up relative importance analyses indicated T1 child *social* anxiety contributed 78% and 79% of the overall model R^2 in CLK and Turtle Program, respectively. The relation between maternal and paternal SAD and T2 child social anxiety did not differ by group (maternal SAD: Wald $\chi^2(1) = .481$, $p=.488$; paternal SAD: Wald $\chi^2(1) = .045$, $p=.832$).

Chapter 4: Discussion

The current study was among the first to examine maternal *and* paternal anxiety disorders, separately and together, using gold-standard diagnostic interviews, as predictors of treatment outcome in a large RCT comparing combined parent-child intervention to parent-only intervention for *preschool* BI/SW. The current study was also the first RCT to examine, in preschoolers, whether treatment format (parent-child versus parent-only) attenuated the relations between parent anxiety and reduced child treatment outcome. Results also delineated differences in child treatment outcomes among specific parent anxiety presentations, such as parent SAD versus anxiety disorders broadly.

The most consistent finding in the current study was that baseline child anxiety severity was the strongest predictor of post-treatment child functioning, accounting for nearly 70-80% of the explained model variance. Specifically, more severe baseline child anxiety predicted more severe anxiety at post-treatment, which aligns with findings from anxiety treatments for older youth (Higa-McMillan, Francis, Rith-Najarian & Chorpita, 2016). Notably, over 70% of children in the current study had at least one baseline anxiety diagnosis (predominantly SAD). Thus, perhaps we need to intervene even earlier in the disease *course*, before symptoms become clinically-impairing (Ginsburg, 2009).

Further, another consistent finding was that, maternal and paternal anxiety had more significant effects on T2 child total, rather than *social* anxiety. One reason for this finding is that there was more variability in T2 child total anxiety than that of T2 child *social* anxiety. Further, anxiety disorders are often comorbid with each other, and improvement in one area of anxiety can lead to subsequent improvements in other untargeted areas (Ollendick et al., 2009; Spence, Donovan & Brechman-Toussaint, 2000),

which may also explain why more effects were found for T2 child total, rather than *social*, anxiety. Also, while treatment methods in both conditions specifically focused on *social* exposures, the general theoretical principles underlying and taught in both conditions focused on child anxiety broadly.

Results for Aim 1 and Exploratory Aim 1 remained relatively the same when maternal and paternal lifetime ADs and SAD were examined separately or together (although some effects went from marginal to significant). Broadly, study results conflict with recent advice from parenting scholars, who argued for gender-neutral conceptualizations of parenting (Fagan et al., 2014). Although parenting was not directly measured in the current dissertation, parenting is often the hypothesized mechanism for why children of parents with anxiety experience reduced anxiety treatment outcomes (Murray et al., 2009). Current study results tentatively suggest that conceptualizations of parenting may differ between typical versus clinical populations, as results from the current study highlight the *unique* effects of both maternal and paternal anxiety on early intervention outcomes for preschool BI/SW.

Moreover, the current study found maternal lifetime ADs and SAD predicted worse T2 child total anxiety, conflicting with studies that found no significant effects of parent anxiety on young children's anxiety treatment outcomes (e.g. Hirshfeld-Becker et al., 2010; Kennedy et al., 2009; Barrett et al., 2015). This could be because the current study (1) included parents with clinical ADs, while other studies examined mean-levels of parent anxiety symptoms primarily in the normative range (Monga et al., 2015; Kennedy et al., 2009) and (2) used gold-standard diagnostic interviews, which are more valid and reliable measurement tools (Antony & Rowa, 2005) compared to that of self-

report questionnaires (Monga et al., 2015; Kennedy et al., 2009; Barrett et al., 2015). Notably, Hirshfeld-Becker and colleagues' (2010) study also examined lifetime and current parent anxiety disorders, using diagnostic interviews, but their study found no effects of parent anxiety on young children's anxiety treatment outcomes. However, their treatment was 20-sessions over 6-months, so this very intensive format may have reduced effects of parent anxiety symptomatology on treatment outcome.

The finding that paternal lifetime ADs predicted *better* T2 child total anxiety was unexpected, conflicted with prior evidence (Kendall et al., 2008; Rapee et al., 2000; van Steensel et al., 2016; Liber et al., 2008), and was not better explained by associations with paternal involvement in caregiving or attendance. On one hand, this finding requires replication, as few fathers experienced anxiety in the current study. Moreover, paternal lifetime ADs were unrelated to T1 child total anxiety, which means this finding does not simply mean that children who started off with higher anxiety severity experienced higher symptom improvement. On the other hand, one study with an externalizing population found improved child psychosocial treatment outcome when fathers experienced higher (not lower) psychopathology (van der hofddakker et al., 2014). Regarding these results, van der hofddakker and colleagues (2014) proposed that attending treatment as a couple may improve the marital relationship, which may benefit children's treatment prognosis. The current study unfortunately did not examine marital functioning, but it is certainly an area for future inquiry, as one high-risk study found father emotional support mediated the link between marital quality and child internalizing symptoms (Mahedy et al., 2018).

Further, the finding that paternal SAD had no effect on T2 child outcome conflicts

with theories that postulate the stronger role of fathers' SAD, relative to that of mothers', on children's SAD (Bögels & Perotti, 2010). Of note, the current sample is much younger than that of most studies examining the intergenerational transmission of SAD. Of note, one study found that almost 50% of married *women* with preschool-age children spent 100% of their time in child care activities (Cohen & Bianchi, 1999). Thus, children have more exposure to mothers and, consequently, mothers' social anxiety symptomatology, during the early years. For example, one study found that, among *8-12-year-old children*, fathers' (not mothers') socially anxious behaviors predicted socially anxious behaviors among highly anxious children (Bögels et al., 2011). Thus, fathers' SAD may increase in importance across children's development.

Further, regarding Aim 2 and Exploratory Aim 2, contrary to hypotheses, the relation between maternal and paternal lifetime ADs and T2 child total anxiety did not differ by treatment condition. However, as hypothesized, maternal SAD predicted worse T2 child total anxiety in CLK (but not the Turtle Program), a robust finding that remained regardless of whether maternal SAD was measured dichotomously or continuously. Several explanations exist for why maternal SAD (and not lifetime ADs) predicted worse T2 child total anxiety in CLK (and not Turtle Program). First, maternal SAD was the most common anxiety disorder (CLK: 24%, Turtle: 21%). Mothers with SAD (rather than GAD or specific phobia) may avoid conducting child social exposures due to maternal fears of social evaluation specifically, reducing treatment efficacy in both conditions. So, why was this only true in CLK (and not the Turtle Program)? In the Turtle Program, *in-vivo* coaching allowed for direct therapist observation of parent avoidance and immediate rectification, whereas in CLK, parents conducted child social exposures out of session,

prohibiting immediate therapist feedback. Therefore, the Turtle Program may be more effective at reducing parent social avoidance and/or reinforcement of children's social avoidance, improving outcomes in the context of maternal SAD specifically. Indeed, responsive in-vivo, therapist coaching has been found to partially mediate changes in observed positive parenting between sessions (Barnett, Niec, and Acevedo-Polakovich, 2014), and immediate feedback is a hallmark of effective behavioral therapy (Skinner, 1953). Second, in the Turtle Program, parent skills were performed in front of other parents and therapists. The anxiety performance curve states that moderate levels of anxiety, as opposed to low or high levels, are associated with the most optimal performance outcomes (Hebb, 1955). Adults with SAD use both avoidance and over preparation to manage their anxiety (Morrison and Heimburg, 2013). Thus, mothers with SAD in the Turtle Program may have been more likely to do "in-between-session practices" due to this social performance aspect. In CLK, there was no performance requirement, so mothers with SAD may have used avoidance (of "in-between-session practices") to manage their own social fears. Relatedly, inherent in every social exposure is interaction with another person, so mothers with SAD, too, must face these social situations. Thus, by conducting "in-between-session practices," mothers with SAD may actually be engaging in social exposure therapy for themselves simultaneously. Third, SSFP has been found to increase child social approach (Coplan et al., 2010). Thus, based on the "child effects" literature, SSFP may have led to less preschool BI/SW and, subsequently, socially anxious parents (in the Turtle Program) may have found it easier to implement child social exposures due to less child resistance.

Several unexpected findings emerged from the current study. First, maternal and paternal session attendance were unrelated to T2 child total or *social* anxiety. This finding conflicts with prior studies that have found higher parent treatment attendance predicted better child anxiety treatment outcome (Rapee et al., 2005; Podell & Kendall, 2011). However, in Rapee and colleagues' (2005) study, attendance was only examined as a predictor of child anxiety diagnoses at one-year follow-up, rather than immediately following treatment, as was done in the current study. Parent attendance may, indeed, predict child anxiety prognosis at one-year-follow-up in the current study (for an example of " sleeper effects," see Rapee, 2013). A second unexpected finding in the current study was that fathers' involvement in caregiving was unrelated to child treatment outcome. In the current study, father involvement in caregiving was low (i.e. scores \geq "42" on the PRS-1 indicate high father involvement; PRS-1 Means: CLK = 30 (8.5), Turtle = 33 (5.7)). Thus, there may not have been enough variability in father involvement in caregiving to detect effects. A third unexpected finding was that paternal age contributed more to the overall model R^2 than that of paternal anxiety in some models. Paternal age has long been cited as a risk factor for adverse neurodevelopmental outcomes in offspring (Kong et al., 2012), and one study found paternal, not maternal, age increased risk of obsessive compulsive disorder in offspring (Wu et al., 2012). To our knowledge, parent age is not frequently examined as a predictor of child anxiety treatment outcome, so this finding warrants further replication and empirical investigation.

The current study has several strengths. To our knowledge, this was the first study to examine paternal and maternal anxiety separately and together as predictors of early intervention outcomes for *preschool* BI/SW. This statistical approach allowed us to

isolate *unique* effects of maternal and paternal anxiety, paving the way for more targeted interventions. Furthermore, parent and child anxiety were both assessed using gold-standard diagnostic interviews. About 75% of fathers completed diagnostic interviews, which aligns with that of other methodologically rigorous studies (e.g., Kendall et al., 2008). Also, study operationalization of parent anxiety included distinguishing between clinically and non-clinically significant anxiety levels, whereas past studies (based on an examination of means and standard deviations) examined parent anxiety symptoms mostly in the normative range (Monga et al., 2015; Kennedy et al., 2009; Barrett et al., 2015). In addition, the only other study that examined specific parent anxiety disorder domains also found maternal SAD predicted reduced treatment effectiveness for anxious school-age youth (Cooper et al., 2008). Current results corroborate such findings, and results further highlight the importance of isolating the role of specific dimensions of maternal anxiety. Lastly, robust statistical approaches (i.e. FIML) were used to reduce parameter biases due to missing data. Relatedly, although not commonly used in the clinical sciences, relative importance analyses provided added information above and beyond that provided by correlations, unstandardized or standardized coefficients (Tonidandel and LeBreton, 2011; LeBreton, 2007) regarding the relative influence of maternal and paternal anxiety on T2 child outcome variables.

This dissertation study is not without limitations. One, while both groups were matched for intervention hours with parents, due to the SSFP component of the Turtle Program, children in CLK received no direct intervention. A recent quasi-experimental study found that combined CLK and SSFP outperformed CLK alone (Lau et al., 2017). Thus, based on the current study's experimental design, regarding the finding that

maternal SAD predicted worse child outcomes in CLK (but not in Turtle Program), it was unclear if this was due to different aspects of the parent groups (e.g. in-vivo coaching versus group discussion only) or to the addition of direct child intervention (i.e. SSFP) in Turtle. Two, despite random assignment, a few clinical and demographic differences existed between conditions. Compared to those in Turtle Program, families in CLK had higher rates of children and fathers who identified as African American, marginally lower paternal attendance, and marginally higher T1 child total anxiety. However, compared to those in CLK, fathers in Turtle had significantly more lifetime ADs. Although these variables did not predict T2 child outcome, study results should be considered in light of these group differences. Three, treatment non-completers tended to be ethnic minorities, less educated and lower SES compared to those who completed treatment. While missing data of non-completers was handled using FIML, from a clinical perspective, the demographic characteristics of non-completes suggests this intervention, as delivered in this study, may not be feasible for or acceptable to families from more marginalized backgrounds. Four, both independent and dependent variables were based on clinician ratings that were gathered from parent report. Parents who have invested eight weeks in treatment for their children may have biased reports of their children's post-treatment functioning. Five, the current dissertation focused on parent anxiety specifically, but adults with anxiety often experience co-morbid mood disorders, substance use disorders, and related somatic complaints, and these disorders were not examined in the current dissertation. Six, in the current study, although parenting was the primary focus of both interventions, it was not specifically examined. Parenting is one hypothesized mechanism linking parent anxiety and reduced child anxiety treatment (Murray et al., 2009). Thus, it

was unclear *why* maternal SAD predicted reduced outcomes in CLK (and not the Turtle Program). Lastly, the current study primarily comprised of intact families, with biological mothers and fathers living with their biological children. Results may not generalize to those with different family structures (i.e. single parents, same-sex parents, adoptive parents, or grandparents)

Thus, future studies should include families from diverse backgrounds and use teacher reports and/or assessment of functioning in real-world settings (see Chronis-Tuscano et al., 2018 for a thorough discussion). Future studies should consider implementing the current intervention in school or primary care settings as a means of reaching families from more diverse backgrounds. Moreover, given that the Turtle Program includes modified PCIT, which is rooted in attachment theory, future studies should measure changes in child attachment throughout treatment. Additionally, parents' and children's anxiety may be reciprocally influential during child anxiety treatment, and improvements in child anxiety may also lead to improvements in parent anxiety symptoms (Monga et al., 2015). Thus, measuring change in parent anxiety throughout child anxiety treatment (rather than only at baseline) would clarify directionality of improvement. Relatedly, change in parenting is an important treatment mediator to examine, and effects of fathers' anxiety on children's anxiety can also occur through change in mothers' parenting, or vice versa, as had been found by other studies (Gibler, Kalomiris, Kiel, 2017). Thus, these indirect effects should be examined in future studies. Also, when studying mothers and fathers' parenting simultaneously, measuring marital functioning and co-parenting may also be incrementally beneficial (Parke & Buriel, 2008). In the current study, maternal and paternal ADs were unrelated. However, future

studies may benefit from examining interactive effects between maternal and paternal ADs. Lastly, a study with 1,500 5-18-year-old children found parent anxiety only predicted worse child anxiety at follow-up (and not at post-treatment) (Hudson et al., 2015). Indeed, examining whether maternal and paternal anxiety relate to child outcomes at one-year follow-up is critical to our understanding of maintenance of intervention effects.

In conclusion, early intervention has the potential to change the developmental course of children at risk for anxiety. However, parents' own anxiety has often been cited as a factor that thwarts the progress of child CBT. Our findings suggest that immediate therapist feedback to parents during child social exposures combined with child social skills training may potentially mitigate the negative effect of mothers' SAD on treatment success. Findings have the potential to inform the development of more targeted early interventions for families with multiple risk factors, leading to improved treatment outcomes, and ultimately, setting young children on more adaptive developmental trajectories across the lifespan.

Table 1. Sample Demographic and Clinical Characteristics by Treatment Group

	CLK (n=55)	Turtle (n=56)	Significance test	<i>p</i>
<u>Child Demographics</u>				
Child Gender			$\chi^2(1, N=111) = 1.29$.257
Male	30 (54%)	24 (43%)		
Female	25 (46%)	32 (57%)		
Child Age M (SD)	52.8 (5.7)	52.8 (5.9)	$t(110) = -.115$.909
Child Race			$\chi^2(3, N=111) = 10.986$.012*
White	26 (46%)	33 (59%)		
Asian	4 (7%)	11 (20%)		
Black	11 (21%)**	2 (4%)**		
Other	14 (25%)	10 (18%)		
Child Ethnicity			$\chi^2(1, N=111) = .152$.696
Hispanic/Latino	4 (7%)	3 (5.5%)		
Median Household Income	$\geq 150,000$	$\geq 150,000$	$t(106) = -.254$.800
<u>Baseline Child Anxiety:</u>				
<u>CSR</u>				
Presence of any Anxiety Diagnoses	40 (70%)	39 (71%)		
Total Anxiety (Sum of Anxiety CSRs)	7.9 (3.8)	6.7 (3.0)	$t(109) = 1.76$.080+
Social Anxiety CSR	3.8 (1.2)	3.4 (1.3)	$t(109) = 1.53$.129
Specific Phobia CSR	1.66 (1.67)	1.25 (1.48)	$t(107) = 1.362$.176
Separation Anxiety CSR	1.57 (1.59)	1.07 (1.33)	$t(108) = 1.80$.074+
Generalized Anxiety CSR	.28 (.69)	.29 (.62)	$t(107) = -.021$.983
Selective Mutism CSR	.68 (.94)	.68 (1.05)	$t(107) = .004$.997
Panic CSR	0	0	-	-
Total Externalizing (Sum of ADHD, ODD, CD CSRs)	.68(1.88)	.57 (1.29)	$t(107) = .351$.726
<u>Maternal Demographics</u>				
Age, <i>M (SD)</i>	39.1 (5.3)	37.7 (4.3)	$t(106) = 1.65$.101
Parent education, <i>N (%)</i>			$\chi^2(2, N=108) = 1.04$.596
Less than college	5 (10%)	3 (5.5%)		
4 year college degree	12 (23%)	16 (29%)		
Graduate degree	36 (67%)	36 (66%)		
Race, <i>N (%)</i>			$\chi^2(4, N= 109) = 6.84$.145
White	32 (59%)	38 (69%)		
African-American	11 (20%)	3 (5%)		
Asian	9 (16%)	12 (22%)		
Hawaiian/Pacific Islander	1 (2%)	0 (0%)		

	Other	1 (2%)	2 (4%)		
Maternal Ethnicity					
	Hispanic/Latino	4 (8%)	3 (5.5%)		
Relation to Child					
	Biological mother	51 (96%)	52 (94%)		
	Adoptive mother	2 (4%)	3 (6%)		
% Married		47 (89%)	51 (93%)		
Attendance		88%	91%	$t(104) = -.676$.501
Average % of child rearing tasks conducted by mother alone (PRS-2)		54%	53%		
<u>Baseline Maternal Anxiety</u>		CLK (n=46)	Turtle (n=53)		
Presence of maternal lifetime anxiety disorders		20 (43%)	22 (41%)	$\chi^2(1, N=99) = .039$.843
	0	26 (57%)	31 (59%)		
	1	9 (20%)	19 (36%)		
	2	8 (17%)	2 (4%)		
	3	2 (4%)	1 (2%)		
	4	1 (2%)	0 (0%)		
Maternal Social Anxiety CSR, $M (SD)$		2.04 (2.0)	1.8 (1.7)	$t(100) = .608$.545
Maternal Internalizing Disorders					
	Social Anxiety	11 (24%)	11 (21%)		
	Specific Phobia	9 (20%)	8 (15%)		
	Separation Anxiety	3 (6.5%)	0 (0%)		
	Generalized Anxiety	10 (22%)	5 (9%)		
	Panic	2 (4%)	2 (4%)		
	Depression	15 (32%)	12 (23%)		
<u>Paternal Demographics</u>					
Age, $M (SD)$		41.10 (6.79)	40.18 (5.02)	$t(96) = .768$.444
Parent education, $N (%)$				$\chi^2(2, N=98) = .913$.633
	Less than college	5 (11%)	4 (8%)		
	4 year college degree	16 (34%)	14 (27%)		
	Graduate degree	26 (55%)	33 (65%)		
Race/ethnicity (%)				$\chi^2(4, N=100) = 14.29$.006**
	White	30 (61%)	40 (78%)		
	African-American	13 (26%)**	1 (2%)**		
	Asian	3 (6%)	8 (16%)		

Hawaiian/Pacific Islander	1 (2%)	1 (2%)		
Other	2 (4%)	1 (2%)		
Paternal Ethnicity				
Hispanic/Latino	3 (6%)	3 (7%)		
Relation to Child				
Biological father	46 (95%)	43 (96%)		
Adoptive father	1 (2%)	2 (4%)		
% Married	98%	96%		
Attendance	34%	49%	$t(100) = -1.84$.069+
Average % of child rearing tasks conducted by father alone (PRS-2)	14%	19%		
Average % of child rearing tasks conducted by both parents together (PRS-2)	24%	25%		
Paternal Involvement (PRS-1)	30.84 (8.51)	33.10 (5.65)		
<u>Baseline Paternal Anxiety</u>	CLK (n=39)	Turtle (n=45)		
Presence of Paternal Lifetime Anxiety Disorder	5 (13%)	16 (36%)	$\chi^2(1, N=84) = 5.76$.016*
	0	34 (87%)	29 (64%)	
	1	3 (8%)	12 (27%)	
	2	2 (5%)	2 (4.5%)	
	3	0 (0%)	2 (4.5%)	
Paternal Social Anxiety CSR, <i>M (SD)</i>	1.17 (1.4)	2.04 (1.80)	$t(82)=-2.41$.018*
Paternal Internalizing Disorders				
Social Anxiety	3 (8%)	13 (29%)		
Specific Phobia	0 (0%)	3 (7%)		
Separation Anxiety	0 (0%)	0 (0%)		
Generalized Anxiety	2 (5%)	5 (11%)		
Panic	2 (5%)	1 (2%)		
Depression	2 (5%)	6 (13%)		

Note: CSR = Clinician Severity Rating (Range: 0-8, with 4 indicating clinically significant impairment); ADHD = attention deficit/hyperactivity disorder; ODD = oppositional defiant disorder; CD = conduct disorder; PRS-1 = Parental Responsibility Scale Part 1; PRS-2 = Parental Responsibility Scale Part 2; + = $p < .10$; * = $p < .05$; ** = $p < .01$

*Table 2. Turtle Program Session Content**

Session	Parents Only	Children Only	Parents & Children
1	Psychoeducation: Parents learn about BI/SW and social anxiety; overview and rationale for prevention program.	Introducing yourself and asking someone to play	Child group free-play (child therapists observe)
2	Psychoeducation: CDI skills presented. Parents learn relevance of CDI skills for anxiety and how to apply them to play situations.	Making eye contact Relaxation/Deep breathing Free Play	Child group free-play (child therapists observe); parents observe deep breathing
3	CDI Coach: Parents are coached in CDI skill application in interactions with their children and learn how to apply CDI skills in social situations.	Communicating with friends Free Play	Parent-child coaching from parent therapist; Child group free-play (child therapists coach)
4	Psychoeducation: BDI skills presented. Parents learn to use graduated exposure in social situations, develop a fear hierarchy for their child, and are coached in child relaxation techniques.	Being brave and facing fears Free Play	Child group free-play (child therapists coach)
5	BDI Coach 1: Parents' use of graduated exposure and relaxation is reviewed; feedback is provided; next steps on exposure hierarchy are identified; provision of appropriate rewards for progress is discussed.	Understanding and expressing feelings Free Play	Parent-child dyads engage in bravery challenges while parents are coached by parent therapist
6	BDI Coach 2: Problem-solving around graduated exposure and positive reinforcement provided; additional steps on the hierarchy identified. After preparing child for "show and tell," group views show and tell through one-way mirror.	Coping with rejection Free Play "Show and Tell"	Parent-child dyads engage in "show & tell" bravery challenges while parents are coached by parent therapist (parents observe "show & tell")

			via television monitor)
7	Psychoeducation: PDI skills presented. Parents learn to give effective commands, use time out, and discriminate between anxious and oppositional behaviors.	Negotiating play with friends Free Play “Scavenger hunt”	Child group free-play (child therapists coach)
8	Review and wrap-up	Review “Party” with preschool games	Child group free-play (child therapists coach); Parent-child dyads engage in “party” bravery challenges

*see Chronis-Tuscano et al., 2015 and Danko et al., in press, for more details

*Table 5. CLK Session Content**

S ession	Parents Only
1	Discussion of the nature and development of anxiety
2	Basic principles of parent-management techniques and the role of parent overprotection in the maintenance of anxiety
3-5	Creation and implementation of exposure hierarchies and the application of cognitive restructuring to parents' own worries
6	Application of techniques learned in sessions 1-5 with an emphasis on developmental transitions

See Rapee et al., 2005 for more details.

Table 4. Correlations among child, maternal, and paternal clinical and demographic variables

	T1 child total ANX	T2 child total ANX	T1 child social ANX	T2 child social ANX	Child Age	Child Sex	Child Race - White	Mom Age	Mom EDU - < BA	Mom EDU - BA	Mom EDU - GRAD	Mom Race - White	Dad Age	Dad EDU - < BA	Dad EDU - BA	Dad EDU - GRAD	Dad Race - White	Mom ADs	Dad ADs	Dad ATTEND	Mom ATTEND	Both ATTEND	PRS
1. T1 child total ANX	1	.659**	.595**	.439**	-.080	-.008	-.006	.177	.067	-.047	.006	.004	.181	-.056	-.015	.046	.071	.242*	-.045	-.130	.048	-.029	-.144
2. T2 child total ANX	.659**	1	.393**	.745**	-.106	-.119	-.006	.199*	-.107	-.104	.146	.056	.218*	-.067	-.028	.063	.092	.301**	-.163	-.053	.014	-.009	-.105
3. T1 child social ANX	.595**	.393**	1	.550**	-.060	-.126	.066	.202*	-.176	.118	-.012	.122	.248*	-.068	.131	-.086	.032	.101	-.014	.056	-.036	.028	.054
4. T2 child social ANX	.439**	.745**	.550**	1	-.100	-.037	.017	.146	-.164	-.048	.117	.080	.210*	-.082	-.134	.171	-.009	.097	-.145	.067	-.025	.031	.052
5. Child Age	-.080	-.106	-.060	-.100	1	-.041	-.025	.135	-.021	-.061	.068	-.046	.067	-.050	-.060	.085	.026	-.083	-.078	-.106	-.008	-.044	.149
6. Child Sex	-.008	-.119	-.126	-.037	-.041	1	.016	.021	.068	-.006	-.033	-.037	.009	.046	.008	-.034	.044	-.239*	.231*	.008	-.067	-.025	.045
7. Child Race - White	-.006	-.006	.066	.017	-.025	.016	1	-.068	-.095	.035	.020	.796**	.052	-.154	.253*	-.152	.709**	.050	.217	.026	.013	.104	-.194
8. Mom Age	.177	.199*	.202*	.146	.135	.021	-.068	1	-.169	-.137	.221*	-.067	.733**	.025	-.029	.013	.139	.050	.101	-.001	.028	.055	.041
9. Mom EDU - < BA	.067	-.107	-.176	-.164	-.021	.068	-.095	-.169	1	-.169	-.399**	-.092	-.179	.518**	-.105	-.194	.117	.147	-.058	-.147	-.277**	-.126	-.115
10. Mom EDU - BA	-.047	-.104	.118	-.048	-.061	-.006	.035	-.137	-.169	1	-.836**	.075	-.019	.095	.147	-.193	-.062	.010	.045	.175	-.042	.176	.046
11. Mom EDU - GRAD	.006	.146	-.012	.117	.068	-.033	.020	.221*	-.399**	-.836**	1	-.019	.086	-.264*	-.109	.252*	.016	-.064	-.030	-.092	.177	-.104	-.011
12. Mom Race - White	.004	.056	.122	.080	-.046	-.037	.796**	-.067	-.092	.075	-.019	1	.032	-.047	.330**	-.285**	.552**	.073	.195	.031	-.062	.079	-.108
13. Dad Age	.181	.218*	.248*	.210*	.067	.009	.052	.733**	-.179	-.019	.086	.032	1	.004	.022	-.024	.066	-.014	.173	-.103	.138	-.064	-.081
14. Dad EDU - < BA	-.056	-.067	-.068	-.082	-.050	.046	-.154	.025	.518**	.095	-.264*	-.047	.004	1	-.199	-.376**	-.097	-.012	.086	-.020	-.246*	-.093	.033
15. Dad EDU - BA	-.015	-.028	.131	-.134	-.060	.008	.253*	-.029	-.105	.147	-.109	.330**	.022	-.199	1	-.833**	.076	.028	-.024	-.039	.081	-.033	.016
16. Dad EDU - GRAD	.046	.063	-.086	.171	.085	-.034	-.152	.013	-.194	-.193	.252*	-.285**	-.024	-.376**	-.833**	1	-.017	-.021	-.021	.048	.062	.083	-.036
17. Dad Race - White	.071	.092	.032	-.009	.026	.044	.709**	.139	.117	-.062	.016	.552**	.066	-.097	.076	-.017	1	.148	.253*	.201*	-.015	.277**	.025
18. Mom ADs	.242*	.301**	.101	.097	-.083	-.239*	.050	.050	.147	.010	-.064	.073	-.014	-.012	.028	-.021	.148	1	-.015	.194	-.038	.187	.269*
19. Dad ADs	-.045	-.163	-.014	-.145	-.078	.231*	.217	.101	-.058	.045	-.030	.195	.173	.086	-.024	-.021	.253*	-.015	1	.089	.002	.076	-.110
20. Dad ATTEND	-.130	-.053	.056	.067	-.106	.008	.026	-.001	-.147	.175	-.092	.031	-.103	-.020	-.039	.048	.201*	.194	.089	1	-.034	.849**	.388**
21. Mom ATTEND	.048	.014	-.036	-.025	-.008	-.067	.013	.028	-.277**	-.042	.177	-.062	.138	-.246*	.081	.062	-.015	-.038	.002	-.034	1	.224*	.358**
22. Both ATTEND	-.029	-.009	.028	.031	-.044	-.025	.104	.055	-.126	.176	-.104	.079	-.064	-.093	-.033	.083	.277**	.187	.076	.849**	.224*	1	.258
23. PRS	-.144	-.105	.054	.052	.149	.045	-.194	.041	-.115	.046	-.011	-.108	-.081	.033	.016	-.036	.025	-.269*	-.110	.388**	-.358**	.258	1

Notes. ** = $p < .001$; * = $p < .05$; T1 = baseline; T2 = post-treatment; ANX = anxiety; ADs = anxiety disorders; EDU = highest level of education; <BA = less than college (e.g. high school, associate's degree, some college, etc); BA = Bachelor's degree; GRAD = Graduate degree (e.g. Masters, PhD, MD, JD); ATTEND = attendance (expressed as a %); PRS = Part 1 of the Parental Responsibility Scale (higher scores indicate higher paternal involvement).

Table 5

Maternal and Paternal Lifetime Anxiety Disorders (ADs) and Child Treatment Outcomes

	T2 Child Total Anxiety				T2 Child Social Anxiety			
	<i>b</i> (SE)	β (SE)	RW	RS-RW (%)	<i>b</i> (SE)	β (SE)	RW	RS-RW (%)
<u>Model 1</u>			Model R ² = .482**				Model R ² = .333**	
T1 Child functioning	.480(.065)*	.587(.067)**	.366	76.143	.523(.087)**	.530(.075)**	.289	86.710
Maternal lifetime ADs	.593(.255)*	.180(.077)*	.064	13.314	.089(.123)	.060(.084)	.008	02.507
Paternal Age	.093(.056)+	.200(.122)+	.038	08.027	.024(.020)	.110(.092)	.036	10.783
Maternal Age	-.045(.069)	-.077(.117)	.012	02.514	--	--	--	--
<u>Model 2</u>			Model R ² = .471**				Model R ² = .352**	
T1 Child functioning	.505(.064)**	.619(.063)**	.393	83.016	.521(.085)**	.529(.074)**	.291	82.255
Paternal lifetime ADs	-.853(.448)+	-.154(.081)+	.027	05.731	-.390(.221)+	-.158(.089)+	.022	06.150
Paternal Age	.083(.057)	.177(.122)	.039	08.324	.030(.020)	.139(.093)	.041	11.595
Maternal Age	-.022(.068)	-.037(.116)	.014	02.929	--	--	--	--
<u>Model 3</u>			Model R ² = .504**				Model R ² = .357**	
T1 Child functioning	.463(.064)**	.567(.069)**	.355	70.522	.514(.086)**	.521(.075)**	.285	80.079
Maternal lifetime ADs	.601(.434)*	.182(.075)*	.063	12.679	.085(.121)	.058(.082)	.008	02.267
Paternal lifetime ADs	-.869(.250)*	-.157(.078)*	.027	05.383	-.388(.220)+	-.157(.089)+	.021	06.035
Paternal Age	.111(.056)*	.240(.122)*	.044	08.745	.031(.020)	.145(.093)	.041	11.618

Maternal Age	-0.047(.068)	-0.079(.115)	.013	02.669	--	--	--	--
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Note. N=111; **= $p < .001$, *= $p < .05$, += $p < .10$; R²= overall variance explained; RW = relative weight; RS-RW = Rescaled Relative Weight (expressed as %).

Table 6

Maternal and Paternal Lifetime Anxiety Disorders (ADs) and Child Treatment Outcome as a Function of Treatment Condition

	T2 Child Total Anxiety				T2 Child Social Anxiety			
	<i>b</i> (SE)	β (SE)	RW	RS-RW (%)	<i>b</i> (SE)	β (SE)	RW	RS-RW (%)
<u>CLK</u>			Model R ² =.494**				Model R ² =.393**	
T1 Child functioning	.396(.092)**	.502(.106)**	.301	60.855	.619(.136)**	.563(.106)**	.302	75.508
Maternal lifetime ADs	.804(.326)*	.278(.111)*	.121	24.399	.081(.154)	.062(.117)	.011	02.829
Paternal lifetime ADs	-.946(.859)	-.126(.114)	.022	04.357	-.639(.422)	-.188(.123)	.019	04.666
Paternal Age	.106(.072)	.247(.168)	.041	08.230	.032(.026)	.160(.129)	.068	16.997
Maternal Age	-.026(.091)	-.046(.161)	.011	02.158	--	--	--	--
<u>TURTLE</u>			Model R ² =.515**				Model R ² =.327**	
T1 Child functioning	.552(.093)**	.637(.088)**	.411	79.755	.450(.111)**	.502(.107)**	.262	80.029
Maternal lifetime ADs	.296(.411)	.074(.102)	.012	02.352	.091(.207)	.052(.117)	.005	01.499
Paternal lifetime ADs	-.831(.505)	-.178(.108)	.030	05.782	-.363(.272)	-.176(.131)	.035	01.754
Paternal Age	.094(.092)	.184(.182)	.046	08.878	.029(.031)	.121(.132)	.025	07.717
Maternal Age	-.048(.107)	-.076(.170)	.017	03.233	--	--	--	--

Note. N=111; **= $p < .001$, *= $p < .05$ *, += $p < .10$; R² = overall variance explained; RW = relative weight; RS-RW = Rescaled Relative Weight (expressed as %).

Figure 1. Chronis-Tuscano and colleagues (2018) transactional model adapted from that of Rubin and colleagues (2009).

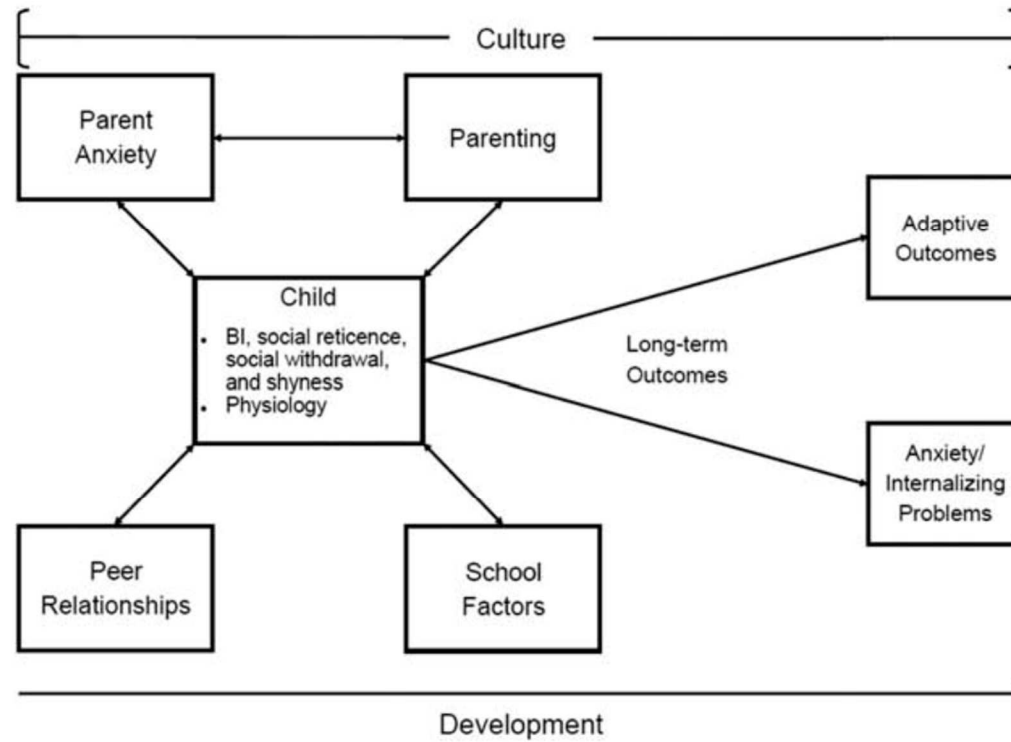


Figure 2. Murray and colleagues (2009) model of the intergenerational transmission of anxiety

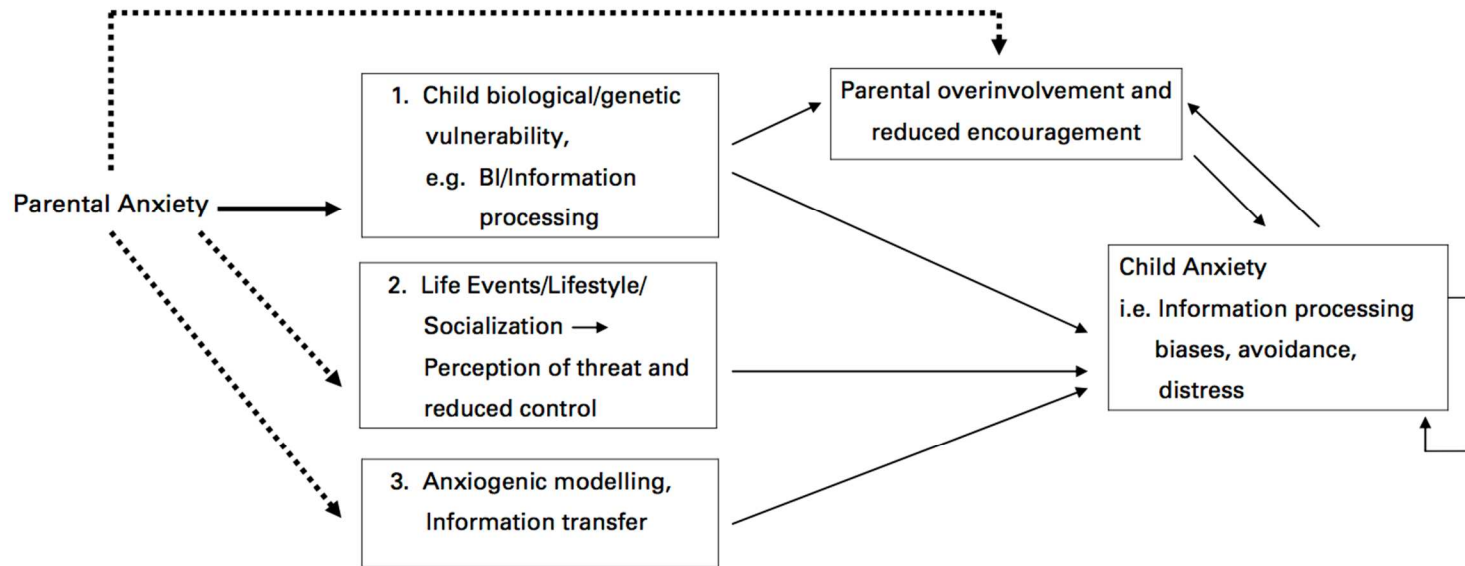
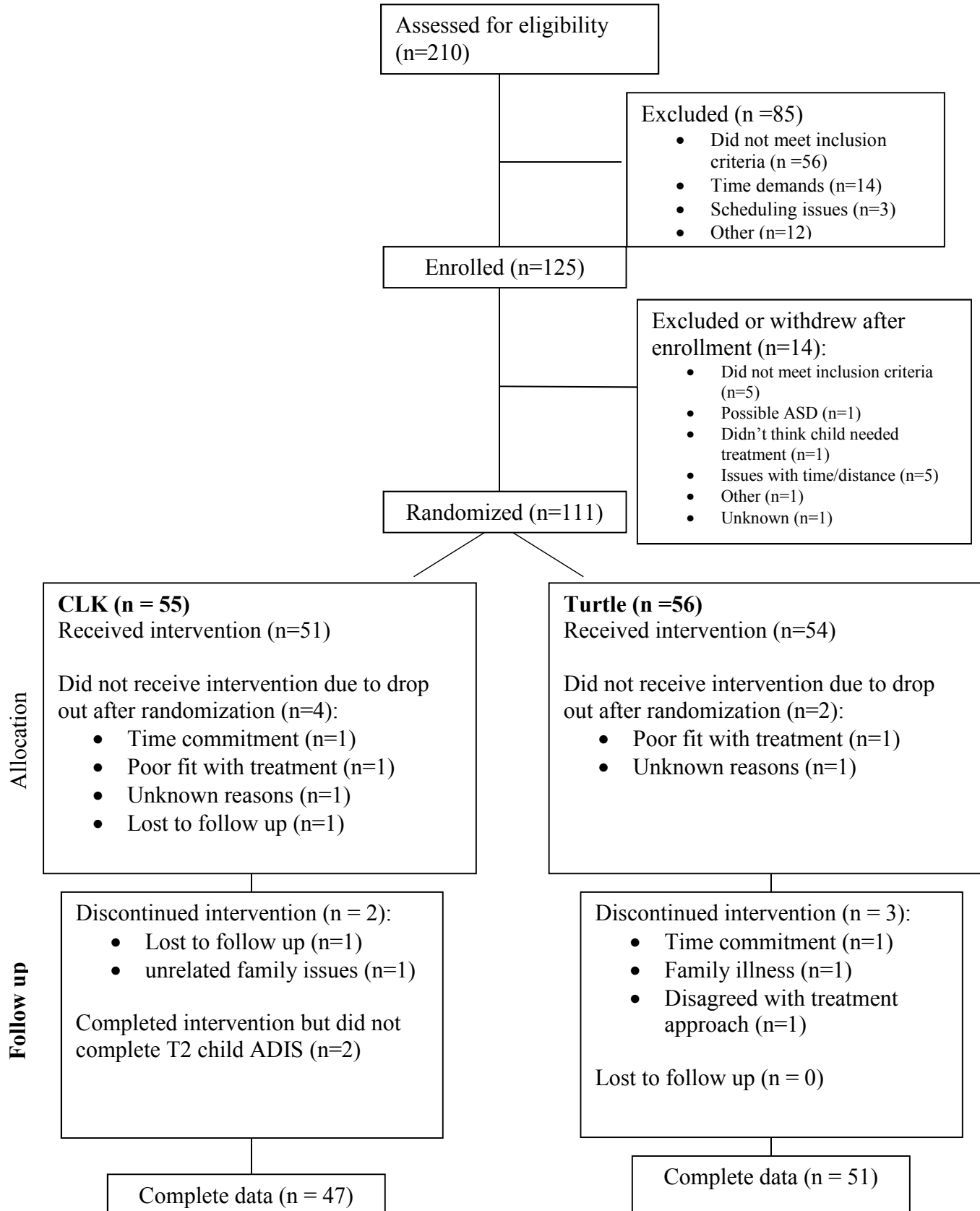


Fig. 1. Pathways to child anxiety. —>, Parental anxiety accounts for the factor;>, Parental anxiety raises the risk of the factor. BI, Behavioural inhibition.

Figure 3. Consort Diagram for Participants



Appendices

Appendix A.

Parenting dimensions: Definitional clarification

Chronis-Tuscano and colleagues (2018) recently called attention to more conceptual clarity regarding the parenting behaviors that have been implicated in the development of anxiety in children. The following is a brief overview.

To start, both parental *overinvolvement* and *overcontrol* have been defined by scholars as parental “interference” or “pressure” to influence “children’s thoughts, feelings and behaviors,” and parenting that “encourages dependence on the parent” (Chorpita & Barlow, 1998; van der Bruggen et al., 2008; McLeod et al., 2007). Similarly, *intrusiveness* has been defined by researchers as parenting behaviors that “take over tasks that children are (or could be) doing independently and impose an immature level of functioning on their children, restricting children’s autonomy” (Wood, 2006). Contrarily, other scholars claim that *overinvolvement* has been inconsistently operationalized, and they believe *overinvolvement* is a broad dimension that describes related subcomponents, such as overcontrol, overprotection, intrusiveness, psychological control (Möller et al., 2016).

Relatedly, *overprotection* has been similarly defined, but some scholars further argue that *overprotection* can further be characterized as “overly sensitive,” “extremely responsive” and difficulty seeing “children in difficulty (Rubin, Stewart & Coplan 1995). Similarly, *oversolicitousness* is defined as “highly affectionate” and “shielding” children “when it is neither appropriate nor sensitive to do so” (Rubin et al., 1997). “Affection” seems to underlie both *overprotection* and *oversolicitousness*. Contrastingly, *autonomy*

granting is characterized by “encouragement of a child's individuality and independence” (Silk, Morris, Tomoe, & Steinberg, 2003), and some believe this to be on the opposite end of the same dimension as *overprotection* (Bögels & van Melick, 2004), while others believe it is a distinct construct (Silk et al., 2003).

In the child anxiety literature, specifically, *parental modeling of anxiety* (often referred to as “parental modeling”) is defined as situations where “children may learn anxiety or avoidance from their parents in a vicarious manner” (Fisak & Grills-Taquechel, 2007). Parental verbal threat transmission, a similar but distinct behavior, refers to situations where “children may become fearful when they hear or read that a stimulus or situation might be dangerous or have another negative connotation” (Muris & Field, 2010).

In the literature, these aforementioned parenting behaviors, and more (e.g. accommodation, rejection, low warmth/support), have all been referred to as “anxiogenic” parenting (for examples, see Kiel & Buss, 2010; Flessner, Murphy, Brennan & Auria, 2017). Thus, to be consistent with the wider field, in this document, the term “anxiogenic parenting” is used as a broad, umbrella term that refers to *any parenting behaviors that has been associated with anxious outcomes in offspring*.

Appendix B

Table 1. Maternal and Paternal Lifetime Social Anxiety Disorder (SAD) and Child Treatment Outcome

	T2 Child Total Anxiety				T2 Child Social Anxiety			
	<i>b</i> (SE)	β (SE)	RW	RS-RW (%)	<i>b</i> (SE)	β (SE)	RW	RS-RW (%)
Model 1			Model $R^2=.475^{**}$				Model $R^2=.334^{**}$	
T1 Child functioning	.496(.064)*	.605(.065)*	.382	80.580	.520(.087)**	.527(.076)**	.280	83.122
	1.018(.537)							
Maternal SAD	+	.147(.077)+	.042	08.937	.163(.263)	.053(.086)	.012	03.651
Paternal Age	.093(.057)	.199(.123)	.038	07.995	.025(.020)	.118(.093)	.045	13.227
Maternal Age	-.045(.069)	-.077(.118)	.012	02.487	--	--	--	--
Model 2			Model $R^2=.454^{**}$				Model $R^2=.343^{**}$	
T1 Child functioning	.508(.065)*	.623(.065)*	.393	85.616	.522(.086)**	.530(.074)**	.286	82.027
	*	*						
Paternal SAD	-.515(.600)	-.072(.083)	.018	03.906	-.395(.287)	-.123(.089)	.017	04.892
Paternal Age	.069(.057)	.149(.122)	.036	07.756	.025(.020)	.118(.092)	.046	13.080
Maternal Age	-.020(.069)	-.034(.117)	.013	02.722	--	--	--	--
Model 3			Model $R^2=.482^{**}$				Model $R^2=.351^{**}$	
T1 Child functioning	.480(.066)*	.586(.068)*	.366	76.058	.511(.087)**	.517(.076)**	.276	78.034
	*	*						
	1.071(.536)							
Maternal SAD	*	.155(.077)*	.044	09.095	.188(.261)	.061(.085)	.013	03.657
Paternal SAD	-.627(.590)	-.087(.082)	.019	04.023	-.412(.287)	-.128(.089)	.018	04.984
Paternal Age	.099(.057)+	.212(.123)+	.040	08.267	.028(.020)	.133(.093)	.047	13.324

Maternal Age	-.045(.069)	-.076(.117)	.012	02.558	--	--	--	--
CLK				Model R ² =.502**			Model R ² =.374**	
T1 Child functioning	.388(.099)*	.490(.116)*	.279	55.669	.578(.140)*	.524(.111)*	.292	78.166
Maternal SAD	2.342(.828)*	.332(.114)*	.143	28.460	.371(.390)	.116(.122)	.025	06.683
Paternal SAD	.450(1.343)	-.042(.125)	.025	05.001	-.384(.620)	-.077(.124)	.007	01.978
Paternal Age	.118(.073)	.275(.170)	.044	08.743	.027(.026)	.134(.131)	.049	13.173
Maternal Age	-.043(.092)	-.076(.163)	.011	02.130	--	--	--	--
TURTLE				Model R ² =.502**			Model R ² =.340**	
T1 Child functioning	.555(.093)*	.642(.088)*	.412	82.054	.459(.110)*	.513(.106)*	.271	79.481
Maternal SAD	.047(.677)	.007(.103)	.003	00.642	.012(.342)	.004(.117)	.003	01.019
Paternal SAD	-.795(.638)	-.136(.109)	.024	04.752	-.533(.330)	-.206(.127)	.038	11.357
Paternal Age	.088(.092)	.174(.182)	.046	09.196	.030(.031)	.128(.130)	.028	8.143
Maternal Age	-.045(.107)	-.071(.170)	.017	03.355	--	--	--	--

Note. $N=111$; ** = $p < .001$, * = $p < .05$, + = $p < .10$; R^2 = overall variance explained; RW = relative weight; RS-RW = Rescaled Relative Weight (expressed as %).

Appendix B

Table 2. Maternal and Paternal Highest Lifetime Social Anxiety Clinician Severity Rating (CSR) and Child Treatment Outcome

	T2 Child Total Anxiety				T2 Child Social Anxiety			
	<i>b</i> (SE)	β (SE)	<i>R</i> <i>W</i>	<i>RS-RW</i> (%)	<i>b</i> (SE)	β (SE)	<i>R</i> <i>W</i>	<i>RS-RW</i> (%)
<u>Model 1</u>			Model <i>R</i> ² =.472**				Model <i>R</i> ² =.331**	
T1 Child functioning	.492(.065)**	.602(.066)**	0.379	80.265	.522(.088)**	.529(.077)**	0.283	85.653
Maternal SAD CSR	.208(.120)+	.137(.078)+	0.0435	09.215	.020(.059)	.029(.087)	0.009	02.584
Paternal Age	.092(.057)	.198(.125)	0.0379	08.027	.025(.020)	.116(.094)	0.039	11.762
Maternal Age	-.043(.070)	-.074(.118)	0.012	02.493	--	--	--	--
<u>Model 2</u>			Model <i>R</i> ² =.455**				Model <i>R</i> ² =.331**	
T1 Child functioning	.518(.064)**	.635(.062)**	0.406	89.129	.531(.086)**	.539(.074)**	0.292	87.050
Paternal SAD CSR	-.141(.136)	-.084(.081)	0.009	01.938	-.051(.067)	-.067(.089)	0.006	01.836
Paternal Age	.062(.056)	.133(.122)	0.029	06.359	.022(.020)	.103(.092)	0.037	11.114
Maternal Age	-.016(.069)	-.028(.118)	0.012	02.574	--	--	--	--

<u>Model 3</u>		Model $R^2=.484^{**}$				Model $R^2=.338^{**}$			
T1 Child functioning	.483(.065)**	.591(.067)**	0.371	76.743	.519(.088)**	.526(.077)**	0.283	83.642	
Maternal SAD CSR	.243(.122)*	.160(.079)*	0.048	09.836	.031(.060)	.045(.088)	0.010	02.908	
Paternal SAD CSR	-.201(.136)	-.120(.081)	0.016	03.233	-.067(.068)	-.089(.091)	0.007	02.106	
Paternal Age	.087(.057)	.189(.124)	0.037	07.690	.025(.020)	.116(.093)	0.038	11.344	
Maternal Age	-.037(.069)	-.063(.118)	0.012	02.498	--	--	--	--	
<u>CLK</u>		Model $R^2=.496^{**}$				Model $R^2=.381^{**}$			
T1 Child functioning	.418(.090)**	.530(.102)**	0.322	64.828	.589(.139)**	.536(.110)**	0.297	77.980	
Maternal SAD CSR	.464(.175)*	.307(.114)*	0.101	20.268	.105(.082)	.153(.120)	0.027	07.186	
Paternal SAD CSR	-.274(.250)	-.136(.125)	0.027	05.532	-	-	0.006	01.456	
Paternal Age	.102(.080)	.242(.192)	0.037	07.540	.026(.027)	.131(.133)	0.051	13.378	
Maternal Age	-.047(.100)	-.083(.178)	0.009	01.832	--	--	--	--	
<u>TURTLE</u>		Model $R^2=.490^{**}$				Model $R^2=.311^*$			
T1 Child functioning	.573(.096)**	.664(.090)**	0.409	83.446	.483(.113)**	.542(.109)**	0.272	87.412	
Maternal SAD CSR	-.014(.168)	-.009(.111)	0.015	03.096	-	-	0.006	02.038	
Paternal SAD CSR	-.141(.163)	-.096(.111)	0.008	01.642	-	-	0.011	03.412	

SAD CSR					.065(.085)	.100(.131)		
Paternal	.076(.091)	.150(.182)						
Age			0.042	08.612	.020(.030)	.087(.129)	0.022	07.138
Maternal	-.049(.107)	-.078(.170)					--	--
Age			0.016	03.204	--	--		

Note. $N=111$; ** = $p < .001$, * = $p < .05$, + = $p < .10$; R^2 = overall variance explained; RW = relative weight; RS-RW = Rescaled Relative Weight (expressed as a %)

Appendix C: Demographics Questionnaire

DEMOGRAPHICS QUESTIONNAIRE

Please fill out this form in its entirety. This information will be used for research purposes only.

1. Date _____

Information about your child

2. Child's Name _____

3. Child's D.O.B. ____/____/____

4. Child's gender 1. Male 2. Female

5. Child's age (in months) _____

6. Child's ethnicity:

1. Hispanic or Latino

2. Not Hispanic or Latino

7. Child's race (please circle all that apply):

1. American Indian or Alaska Native

5. White

2. Asian

6. Other:

3. Black or African American

4. Native Hawaiian or Other Pacific Islander

8. Child's preschool or daycare:

9. Please indicate if this is a preschool or daycare:

10. How many hours/days does your child attend preschool/daycare? _____
hrs _____ days/week

11. When did your child start preschool/daycare? _____
(month/year)

12. Has your child been diagnosed with any psychiatric, emotional or behavioral disorder?

1. Yes 2. No

13. If so, what diagnosis and by whom?

14. Please circle the names of any medications that your child is currently taking

- | | | |
|----------------|----------------|---------------------|
| 1. Adderall | 13. Effexor | 25. Seroquel |
| 2. Adderall XR | 14. Luvox | 26. Mellaril |
| 3. Concerta | 15. Paxil | 27. Zyprexa |
| 4. Cylert | 16. Prozac | 28. Orap |
| 5. Dexadrine | 17. Serzone | 29. Cibalith-S |
| 6. Dextrostat | 18. Sinequan | 30. Depakote |
| 7. Focalin | 19. Tofranil | 31. Eskalith |
| 8. Metadate | 20. Wellbutrin | 32. Lithobid |
| 9. Ritalin | 21. Zoloft | 33. Tegretol |
| 10. Strattera | 22. Clozaril | 34. Other (specify) |

-
- | | |
|---------------|---------------|
| 11. Anafranil | 23. Haldol |
| 12. BuSpar | 24. Risperdal |

15. If your child is taking medication, please specify the dose
_____ and
dose schedule (how often he/she takes the medication)

16. How old was your child I he/she first began medication for any psychiatric disorder?

- 2
3
4
5

17. Which medication did he/she begin taking? _____

18. For what problem did he/she begin taking this medicine?

1. ADHD

2. Depression
3. Bipolar disorder
4. Anxiety disorder or anxiety problems
5. Schizophrenia
6. Post traumatic stress disorder
7. Dysthymia (feeling down or sad most days)
8. Other (specify) _____

19. Is your child currently receiving treatment other than medication from a psychologist, psychiatrist, pediatrician, or other professional for treatment of mental health, emotional, or behavioral problems?

1. Yes
2. No

20. If yes, please circle what type of treatment your child is receiving (you may circle more than 1)

1. Individual therapy
2. Group therapy
3. Family therapy
4. Parent training group
5. Self help for drug or alcohol problems (e.g. Alcoholics Anonymous)
6. Summer program
7. School intervention
8. After-school program
9. Other (specify) _____

21. Please circle what type of professional works with your child (you may circle more than 1)

1. Psychologist
2. Psychiatrist
3. Social worker
4. Physician (MD)
5. Counselor
6. Teacher
7. Other (specify) _____

Information about yourself

22. Your name _____

23. Your age _____

24. Your D.O.B. _____ / _____ / _____

9. Other (specify)

34. What is your occupation?

35. What is your marital status?

1. Never married
2. Married
3. Separated
4. Divorced
5. Widowed

36. Total yearly family income _____

37. Please circle any psychological problems you may experience currently or have experienced in the past

1. Attention problems
2. Hyperactivity problems
3. Depression
4. Mania (extended periods of abnormally elevated mood)
5. Substance use problems
6. Psychosis
7. Post traumatic stress
8. Interpersonal problems
9. Other (specify) _____

38. Are you currently taking medication for a psychological or psychiatric disorder?

1. Yes
2. No

39. If yes, please circle what type of medication you are taking

1. Stimulant ADHD medication [i.e., Concerta, Ritalin (methylphenadate), Adderall (amphetamine)]
2. Non-stimulant ADHD medication [i.e., Strattera (atomoxetine)]
3. Antidepressants or anti-anxiety medication [i.e., Prozac (fluoxetine), Zoloft (sertraline), Wellbutrin (bupropion)]
4. Antipsychotic medications [i.e., Risperdal (risperdone), Clozaril (chlozapine)]

5. Mood stabilizing medications [i.e., Depakote (valproic acid), Lithobid (lithium carbonate)]
6. Other (specify) _____
-

40. Are you currently receiving non-medication treatment (i.e., therapy) for your own psychological problems?

1. Yes
2. No

41. If yes, please circle what type of treatment you are currently receiving (you may circle more than 1)

1. Individual therapy
2. Group therapy
3. Family therapy
4. Self help for drug or alcohol problems (e.g. Alcoholics Anonymous)
5. Other (specify) _____

42. Have you been diagnosed with any chronic health conditions, such as high blood pressure, cardiovascular disease, diabetes, etc.?

1. Yes
2. No

43. If yes, please list the health conditions you have been diagnosed with:

44. Are you currently taking any medications for your chronic health conditions?

1. Yes
2. No

45. If so, please list:

46. Do you live with the above named child? 1. Yes 2. No

Information about your child's co-parent (**IMPORTANT NOTE: Only complete this section if your child's co-parent *has* consented to participate in this study and he is *NOT* completing his own questionnaires).** If you do not need to complete this section please skip to item 88.

47. Co-parent's name _____

48. Co-parent's age _____

49. Co-parent's D.O.B. _____/_____/_____

50. Co-parent's sex: 1. Female 2. Male

51. Co-parent's ethnicity:

1. Hispanic or Latino

2. Not Hispanic or Latino

52. Co-parent's race (please circle all that apply):

1. American Indian or Alaska Native

5. White

2. Asian

6. Other:

3. Black or African American

4. Native Hawaiian or Other Pacific Islander

53. Please choose which of the following best describes the relationship with the above-named child:

1. Biological Mother

2. Biological Father

3. Adoptive Mother

4. Adoptive Father

5. Step-Mother

6. Step-Father

7. Other: _____

54. Co-parent's address

55. Co-parent's phone number (home) _____
(business) _____ (cell) _____

56. What is the best number to reach co-parent? 1. Home 2. Business

3. Cell

57. Co-parent's Email address

58. What is the highest educational degree your child's co-parent has completed?

1. Some high school

2. High school degree or equivalent

3. One year of college

4. Two years of college, or associates degree

5. Three years of college, no Bachelors degree

6. Bachelors degree or equivalent

7. Masters degree or equivalent

8. Doctoral degree or equivalent

9. Other (specify)

59. What is your child's co-parent's occupation?

60. What is your child's co-parent's marital status?

1. Never married
2. Married
3. Separated
4. Divorced
5. Widowed

61. Has your child's co-parent ever been diagnosed with a psychological or psychiatric disorder?

1. Yes
2. No

62. Please circle any psychological problems your child's co-parent may be experiencing currently or has experienced in the past

1. Attention problems
2. Hyperactivity problems
3. Depression
4. Mania (extended periods of abnormally elevated mood)
5. Substance use problems
6. Psychosis
7. Post traumatic stress
8. Interpersonal problems
9. Other (specify) _____

63. Is your child's co-parent currently taking medicine for a psychological or psychiatric disorder?

1. Yes
2. No

64. If yes, please circle what type of medication your child's co-parent is currently taking

1. Stimulant ADHD medication [i.e., Concerta or Ritalin (methylphenadate), Adderall (amphetamine)]
2. Non-stimulant ADHD medication [i.e., Strattera (atomoxetine)]

3. Antidepressants or anti-anxiety medication [i.e., Prozac (fluoxetine), Zoloft (sertraline), Wellbutrin (bupropion)]
 4. Antipsychotic medications [i.e., Risperdal (risperidone), Clozaril (clozapine)]
 5. Mood stabilizing medications [i.e., Depakote (valproic acid), Lithobid (lithium carbonate)]
 6. Other (specify)
-

65. Is your child's co-parent currently receiving non-medication treatment (i.e., therapy) for his own psychological problems?

1. Yes
2. No

66. If yes, please circle what type of treatment your child's co-parent is currently receiving (you may circle more than 1)

1. Individual therapy
2. Group therapy
3. Family therapy
4. Self help for drug or alcohol problems (e.g. Alcoholics Anonymous)
5. Other (specify) _____

67. If your child's co-parent is in treatment, please circle what type of professional he is seeing

1. Psychologist
2. Psychiatrist
3. Social worker
4. Physician (MD)
5. Counselor
6. Teacher
7. Other (specify) _____

68. Does your child's co-parent live with the above named child? 1. Yes
2. No

Appendix D: Screening Measures

BEHAVIOR INHIBITION QUESTIONNAIRE (BIQ)

1	2	3	4	5	6	7
Hardly Ever	Infrequently	Once in a While	Sometimes	Often	Very Often	Almost Always
1. Approaches new situations or activities very hesitantly						
2. Will happily approach a group of unfamiliar children to join in their play #2 RECODE						
3. Is very quiet around new (adult) guests to my home						
4. Is cautious in activities that involve physical challenge (e.g., climbing, jumping from heights)						
5. Settles in quickly when I visit the homes of people I don't know well #5 RECODE						
6. Enjoys being the centre of attention #6 RECODE						
7. Is comfortable asking other children to play #7 RECODE						
8. Is shy when first meeting new children						
9. Happily separates from parent(s) when left in new situations for the first time (e.g., kindergarten, preschool, childcare) #9 RECODE						
10. Is happy to perform in front of others (e.g., singing, dancing) #10 RECODE						
11. Quickly adjusts to new situations (e.g., kindergarten, preschool, childcare) #11 RECODE						
12. Is reluctant to approach a group of unfamiliar children to ask to join in						
13. Is confident in activities that involve physical challenge (e.g., climbing, jumping from heights)						

#13 RECODE
14. Is independent
#14 RECODE
15. Seems comfortable in new situations
#15 RECODE
16. Is very talkative to adult strangers
#16 RECODE
17. Is hesitant to explore new play equipment
18. Gets upset at being left in new situations for the first time (e.g., kindergarten, preschool, childcare)
19. Is very friendly with children he or she has just met
#19 RECODE
20. Tends to watch other children, rather than join in their Games
21. Dislikes being the centre of attention
22. Is clingy when I visit the homes of people I don't know well
23. Happily approaches new situations or activities
#23 RECODE
24. Is outgoing
#24 RECODE
25. Seems nervous or uncomfortable in new situations

26. Happily chats to new (adult) visitors to my home

#26 RECODE

27. Takes many days to adjust to new situations (e.g., kindergarten, preschool, childcare)

28. Is reluctant to perform in front of others (e.g., singing, dancing)

29. Happily explores new play equipment

#29 RECODE

30. Is very quiet with adult strangers

SUM: ____

ADD SCORES FOR ITEMS 1-30. SUM MUST BE 132 OR MORE FOR INCLUSION.

INELIGIBLE IF SUM = 131 OR LESS.

SOCIAL COMMUNICATION QUESTIONNAIRE (SCQ)

1. Is she/he now able to talk using short phrases or sentences? (If “No”, skip to question 8.)	No	Yes
2. Do you have a to and fro “conversation” with her/him that involves taking turns or building on what you have said?	1	Yes No 0
3. Does she/he ever use odd phrases or say the same thing over and over in almost exactly the same way (either phrases that she/he hears other people use or ones that she/he makes up)?		Yes No 1 0
4. Does she/he ever use socially inappropriate questions or statements? For example, does she/he ever regularly ask personal questions or make personal comments at awkward times?		Yes No 1 0
5. Does she/he ever get her/his pronouns mixed up (e.g., saying <i>you</i> or <i>she/he</i> for <i>I</i>)?		Yes No 1 0
6. Does she/he ever use words that she/he seems to have invented or made up her/himself; putting things in odd, indirect ways; or use metaphorical ways of saying things (e.g., saying <i>hot rain</i> for <i>steam</i>)?		Yes No 10
7. Does she/he ever say the same thing over and over in exactly the same way or insist that you say the same thing over and over again?		Yes No 1 0
8. Does she/he ever have things that she/he seems to have to do in a very particular way or order or rituals that she/he insists that you go through?		Yes No 1 0
9. Does her/his facial expression usually seem appropriate to the particular situation, as far as you can tell?		Yes No 0 1
10. Does she/he ever use your hand like a tool or as if it were part of her/his own body (e.g., pointing with your finger or putting your hand on a doorknob to get you to open the door)?		Yes No 1 0
11. Does she/he ever have any interests that preoccupy her/him and might seem odd to other people (e.g., traffic lights, drainpipes, or timetables)?		Yes No 1 0

12. Does she/he ever seem to be more interested in parts of a toy or an object (e.g., spinning the wheels of a car), rather than in using the object as it was intended?	Yes No 1 0
13. Does she/he ever have any special interests that are <i>unusual</i> in their intensity but otherwise appropriate for her/his age and peer group (e.g., trains or dinosaurs)?	Yes No 1 0
14. Does she/he ever seem to be unusually interested in the sight, feel, sound, taste, or smell of things or people?	Yes No 1 0
15. Does she/he ever have any mannerisms or odd ways of moving her/his hands or fingers, such as flapping or moving her/his fingers in front of her/his eyes?	Yes No 1 0
16. Does she/he ever have any complicated movements of her/his whole body, such as spinning or repeatedly bouncing up and down?	Yes No 1 0
17. Does she/he ever injure her/himself deliberately, such as biting her/his arm or banging her/his head?	Yes No 1 0
18. Does she/he ever have any objects (<i>other</i> than a soft toy or comfort blanket) that she/he <i>has</i> to carry around?	Yes No 1 0
19. Does she/he have any particular friends or a best friend?	Yes No 0 1
20. Does she/he ever talk with you just to be friendly (rather than to get something)?	Yes No 0 1
21. Does she/he ever <i>spontaneously</i> copy you (or other people) or what you are doing (such as vacuuming, gardening, or mending things)?	Yes No 0 1
22. Does she/he ever spontaneously point at things around her/him just to show you things (not because she/he wants them)?	Yes No 0 1
23. Does she/he ever use gestures, other than pointing or pulling your hand, to let you know what she/he wants?	Yes No 0 1
24. Does she/he nod her/his head to indicate <i>yes</i> ?	Yes No 0 1

25. Does she/he shake her/his head to indicate <i>no</i> ?	Yes No 0 1
26. Does she/he usually look at you directly in the face when doing things with you or talking with you?	Yes No 0 1
27. Does she/he smile back if someone smiles at her/him?	Yes No 0 1
28. Does she/he ever show you things that interest her/him to engage your attention?	Yes No 0 1
29. Does she/he ever offer to share things other than food with you?	Yes No 0 1
30. Does she/he ever seem to want you to join in her/his enjoyment of something?	Yes No 0 1
31. Does she/he ever try to comfort you if you are sad or hurt?	Yes No 0 1
32. If she/he wants something or wants help, does she/he look at you and use gestures with sounds or words to get your attention?	Yes No 0 1
33. Does she/he show a normal range of facial expressions?	Yes No 0 1
34. Does she/he ever spontaneously join in and try to copy the actions in social games, such as <i>The Mulberry Bush</i> or <i>London Bridge if Falling Down</i> ?	Yes No 0 1
35. Does she/he play any pretend or make-believe games?	Yes No 0 1
36. Does she/he seem interested in other children of approximately the same age whom she/he does not know?	Yes No 0 1
37. Does she/he respond positively when another child approaches her/him?	Yes No 0 1
38. If you come into a room and start talking to her/him without calling her/his name, does she/he usually look up and pay attention to you?	Yes No 0 1

39. Does she/he ever play imaginative games with another child in such a way that you can tell that each child understands what the other is pretending?	Yes No 0 1
40. Does she/he play cooperatively in games that need some form of joining in with a group of other children, such as hide-and-seek or ball games?	Yes No 0 1

Add total from each column:

___ +

___ = ___

IF SCQ SCORE IS ≥ 15 , PARTICIPANT IS **NOT ELIGIBLE.**

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