THE EFFECTS OF COGNITIVE-BEHAVIORAL THERAPY FOR YOUTH ANXIETY ON SLEEP PROBLEMS

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ABSTRACT

Research supports shared neurological, cognitive, and environmental features among youth with sleep-related problems (SRPs) and anxiety. Despite overlap in interventions for SRPs and anxiety, little is known about the secondary benefit on SRPs following anxiety-focused treatment. The present study examined whether SRPs improved following cognitive-behavioral therapy (CBT) for youth with anxiety disorders. It also examined whether variables that may link anxiety and sleep problems (e.g. pre-sleep arousal, family accommodation, sleep hygiene) changed across treatment, and whether said changes predicted SRPs at posttreatment. Youth were diagnosed with anxiety at pretreatment and received weekly CBT that targeted their principal anxiety diagnosis at one of two specialty clinics (N = 69 completers, M_{age} = 10.86, 45% males). Youth completed a sleep diary between pretreatment and session one and again one week prior to posttreatment. All other measures were administered in the first session and at the posttreatment assessment. Results indicated that parent-reported SRPs improved from pre- to post-treatment and that treatment responders yielded greater improvement than nonresponders. Specific areas of bedtime resistance and sleep anxiety showed significant improvement. Youth reported lower rates of SRPs and no pre- to post-treatment changes. Pre-sleep arousal and parental accommodation decreased over treatment but did not predict lower SRPs at posttreatment. However, higher accommodation positively correlated with greater SRPs. Sleep hygiene evidenced no change and did not mediate accommodation and posttreatment SRPs. Clinical implications for the treatment of anxious youth are discussed and suggestions for future research are offered.

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CHAPTER 1

MANUSCRIPT IN JOURNAL ARTICLE FORM

The Effects of Cognitive-Behavioral Therapy for Youth Anxiety on Sleep Problems

Sleep related problems (SRPs) represent a spectrum of sleep issues, including prolonged sleep latency (i.e. difficulty falling asleep), night wakings, bedtime resistance, nighttime anxiety, daytime sleepiness, and parasomnias/nightmares. SRPs are relatively common throughout youth development (Chorney, Detweiler, Morris & Kuhn, 2008) but are particularly prevalent among youth with anxiety disorders (Leahy et al. 2013). Studies show that anxiety and SRPs are positively correlated within samples of anxious youth (Alfano, Beidel, Turner & Lewin, 2006; Alfano, Zakem, Costa, Taylor & Weems, 2009) and that up to 90% of youth with an anxiety disorder endorse a SRP (Alfano, Ginsburg, & Kingery, 2007; Chase & Pincus, 2011). Additionally, research has found that samples of youth with anxiety show significantly higher levels of SRPs compared to controls on parent/self report measures (Alfano et al., 2006; Hansen et al. 2011; Ivanenko, Crabtree, O'Brien, & Gozal, 2006), although differences may exist depending on the specific SRP being investigated (Hudson, Gradisar, Gamble, Schniering & rebelo, 2009). Further evidence comes from studies that use objective measurement of SRPs (e.g. actigraphy, polysomnigraphy; Alfano & Kim, 2011; Alfano, Reynolds, Scott, Dahl & Mellman, 2012; Forbes et al. 2008), although findings are not unanimous (Forbes et al. 2006).

Researchers have posited links between SRPs and anxiety. Conceptually, sleep and anxiety represent opponent processes, such that cognitive and/or physiological

arousal from fear is incompatible with sleep initiation (Dahl, 1996). Youth with anxiety experience difficulties with emotion regulation (Hum, Manassis, & Lewis, 2013) and their ability to self-regulate can be particularly compromised at nighttime (Chorney et al. 2008). Fatigue resulting from SRPs can further exacerbate deficits in emotion regulation (i.e. the ability to inhibit anxiety), which in turn, worsens sleep. Neuroscience research supports this bidirectional relationship. For example, underarousal of the prefrontal cortex has been observed in both elevated anxiety and SRPs (Campbell-Sills, Simmons, Lovero, Rochlin, Paulus & Stein, 2011), and has been associated with increased amygdala activity (Yoo et al. 2007). Similarly, higher levels of cortisol at bedtime for youth with anxiety suggest greater sleep-incompatible arousal (Forbes et al. 2006). The findings are buttressed by parent/self report research showing significant correlations between pre-sleep arousal and anxiety in youth (Alfano et al. 2010). Other behavioral mechanisms have also been suggested, such as parental accommodation of youth anxious symptoms (Storch et al., 2008). Allowance of anxious/avoidance behaviors at bedtime can promote unhealthy sleep habits (i.e. co-sleeping, staying up late, inconsistent bedtime routines; poor sleep hygiene) that result in chronic poor sleep hygiene and SRPs (Peterman, Carper & Kendall, 2014).

Overlap between SRPs and anxiety has sparked interest in transdiagnostic treatment. Cognitive-behavioral therapy (CBT) is the empirically supported treatment (EST) for both anxiety and for SRPs (Hollon & Beck, 2013; Taylor & Roane, 2010). Although important distinctions exist, there are common features such as parent management training, contingency management, and relaxation. Findings support improvement in SRPs following treatment of specific nighttime fears (Gordon et al.

2007; Pincus, Weiner & Friedman, 2012) and when SRPs and anxiety are treated conjointly (Clementi & Alfano, 2013). However, the extent to which CBT for anxiety yields secondary improvement in SRPs remains largely unknown. Within the adult literature, a meta-analysis found moderate effects of CBT for anxiety on SRPs; however, residual SRPs were present at posttreatment (Belleville et al. 2010). Nevertheless, the authors concluded that there are too few studies on the topic to state that SRPs improve following CBT for anxiety. Only two studies have examined the issue in youth. One study found youth treated with CBT for generalized anxiety disorder showed improvement on a one-item marker of general sleep disturbance (Kendall & Pimentel, 2003). In another study, youth treated for OCD experienced improvements in a variety of sleep problems per parent report (Storch et al. 2008). However, these studies are limited by the use of non-validated sleep measures, mono-method/reporter assessment, diagnostically homogeneous samples, and the limited scope of SRPs evaluated. Further, use of secondary data in these studies limits theory driven research to investigate possible factors that mediate the sleep/anxiety relationship.

Although it has yet to be empirically corroborated, secondary effects on SRPs following CBT for anxiety may occur because (a) youth and parents may apply skills from CBT for anxiety to SRPs (e.g. contingency management, relaxation), and/or (b) CBT may resolve underlying anxiety issues that had previously facilitated SRPs (e.g. reduction in pre-sleep arousal, firmer parental limits/decreased accommodation). Unfortunately, youth with comorbid SRPs and anxiety are a particularly at-risk population. Youth with SRPs experience a range of functional impairment, such as problems in academic achievement, attention, impulse control, memory, mood regulation,

and even physical health (Dewald, Meijer, Oort, Kerkhof, & Bögels, 2010; Heiser et al., 2000; Kopasz et al., 2010; Sadeh, 2007; Sadeh, Gruber, & Raviv, 2002). Disturbances from SRPs may further compound the negative outcomes regularly seen in anxious youth (Shanahan, Copeland, Costello & Angold, 2008). Such consequences suggest a need to further our understanding of and enhance our treatments for this population.

The research to date has established clear links between sleep and anxiety in youth, both in theory and in clinical practice. Treatment that targets both problem areas has real clinical utility given interactive effects and impairment when comorbid. Although CBT for anxiety targets unique features of behavioral sleep problems (and vice-versa), there is significant overlap between the treatments in methods of intervention and mechanisms targeted. To date, no trial has investigated the effects of CBT for youth anxiety on SRPs among heterogeneous anxious youth and using validated sleep questionnaires. Such research would help elucidate the relationship between anxiety and SRPs as well as provide practical information to clinicians and families regarding intervention for comorbid problems.

The present study examined (a) whether there are changes to SRPs following CBT for youth anxiety (without specific sleep intervention) and whether treatment response is associated with reduced SRPs at posttreatment, and (b) whether pre-sleep arousal likewise decreases over treatment and whether that change is associated with a reduction of SRPs at posttreatment. In line with treatment effectiveness (rather than efficacy) research, the research approach was a "multiple EST trial" evaluating multiple empirically supported treatments for various anxiety disorders in youth. In addition, we explored change in (a) sleep hygiene (behaviors that promote good sleep, e.g., consistent

wake/bedtimes, calming activities before bedtime, no electronics in bed) and (b) family accommodation pre- to post-treatment as predictors of SRPs, and whether sleep hygiene mediates the relationship between family accommodation and SRPs. It is hypothesized that SRPs will improve from pre- to post-treatment and that favorable treatment response and reduction in pre-sleep arousal will predict better SRP outcome (with pre-sleep arousal also hypothesized to improve from pre- to post-treatment). Regarding exploratory analyses, it is hypothesized that reduced accommodation and improved sleep hygiene will predict SRPs at posttreatment and sleep hygiene will mediate the relationship between family accommodation and SRPs.

Methods

Participants

One-hundred and five youth (and their families) were screened for youth anxiety disorders and met eligibility criteria (intent-to-treat sample). Eligibility criteria included: (a) child was aged 7-17 years at the time of the assessment, (b) child met DSM-IV diagnostic criteria for a *principal* anxiety disorder (Separation Anxiety Disorder (SAD), Generalized Anxiety Disorder (GAD), Social Phobia (SoP), Specific Phobia (SP), Obsessive Compulsive Disorder (OCD), Panic Disorder (PD), Selective Mutism (SM), or Anxiety Disorder- Not Otherwise Specified (AD-NOS)), and (c) families read/spoke English. No exclusions on the basis of non-principal youth comorbidities or presence of medications were made with the purpose of increasing external validity. Of the initial sample, 69 youth and parents completed treatment. The data presented were analyzed using the treatment completer sample to evaluate CBT's effect on sleep. Descriptive information on study participant variables and treatment variables are presented in Tables

1.1-1.3. See figure 1.1 for consort table.

Procedure

Youth with concerns about anxiety were referred for outpatient treatment to either the Temple University Child and Adolescent Anxiety Disorders Clinic (CAADC) or the Boston University Center for Anxiety Related Disorders (CARD) from multiple sources, including school guidance counselors, pediatricians, and parents. Prior to participation, parents provided consent and youth provided assent. Families completed the ADIS C/P at pretreatment to meet eligibility criteria. The ADIS C/P was administered separated to youth and parents. Youth diagnoses were based on the "or rule," in which they received a diagnosis based on criteria from parent *or* youth report. Eligible families completed accommodation measures at the pretreatment assessment. Youth were provided a sleep diary to complete over the following two weeks.

At the beginning of the first treatment session (at least two weeks after the pretreatment assessment), youth and parents completed another set of pretreatment measures. Youth completed measures of sleep hygiene, pre-sleep arousal, and sleep habits; parents completed measures of sleep hygiene and youth sleep habits. The clinician also collected the sleep diary at this time point.

Using a "multiple EST trial," youth received the empirically supported treatment (cognitive-behavioral) for their respective *principal* diagnoses. For example, youth diagnosed with principal SAD, GAD, SoP, SP, or AD-NOS received either the *Coping Cat* (Kendall & Hedtke, 2006a) or *Modular Approach to Therapy for Children with Anxiety, Depression, or Conduct Problems* (MATCH; Chorpita, 2007), youth diagnosed with PD received *Mastery of Anxiety and Panic for Adolescents: Riding the Wave*

(Pincus, Ehrenreich & Mattis, 2008), youth diagnosed with OCD received *Family-Based Treatment for Young Children with OCD* (Freeman & Garcia, 2009), and youth diagnosed with SM were treated with *Treatment for Children with Selective Mutism (*Bergman, 2012). All treatments contained common core features: Psychoeducation, cognitive skills training, and exposure (with response prevention for OCD). Therapists implemented treatment manuals flexibly to address individual differences and comorbidities. Sessions were audio- or video-taped and 10 percent were randomly selected and assessed for treatment adherence by independent coders. Sessions were coded by whether the clinician completed critical components of protocol (e.g. at least 4 exposures conducted, rewards system implemented, cognitive restructuring taught, etc.), rather than a session-by-session adherence, consistent with a "flexibility within fidelity" approach (Kendall & Beidas, 2007). Adherence was very high (97% adherent). Further, among coded sessions, there were no instances of clinicians providing empirically supported behavioral therapy for sleep problems (e.g. sleep hygiene, stimulus control, bedtime fading). All therapists were masters-level clinicians with experience treating youth anxiety but no unique training for sleep problems.

All pretreatment/session one questionnaires were completed for a second time at posttreatment by youth and parents. A one-week version of the sleep diary was completed in the week before the posttreatment assessment. Diagnosticians reevaluated families with the ADIS C/P and CGI-I to determine whether youth were treatment responders. Diagnosticians were blind to initial diagnostic severity, treatment experience, and treatment status. Consistent with the emphasis on *effectiveness*, the timing of the posttreatment assessment was variable, with the following criteria: the child had (a)

reached the recommended number of sessions according to the protocol (e.g. 16 sessions of *Coping Cat*, 12 sessions of *Family OCD Treatment*), *or* (b) received an "adequate dose" of CBT, in which families and/or clinicians terminated due to reported remission of the principal diagnosis or the family needed to end treatment for personal reasons (see Table 1.3). "Adequate dose" was defined as the therapist completing all major CBT skills (e.g. psychoeducation, skills training, contingency management, minimum of four in-session exposures). Several youth continued therapy following the posttreatment assessment but were not tracked further.

Measures

Parent-Report

Children's Sleep Habit Questionnaire (CSHQ; Owens, Spirito, & McGuinn 2000). The CSHQ is a 33-item scale that assesses children's sleep behaviors during the most recent typical week. Parents rate the frequency of sleep behaviors from "rarely" (0-1 times per week), "sometimes" (2-4 times per week), and "usually" (5-7 times per week). Parents also provide information regarding bedtime schedules and estimated nightly sleep. The questionnaire includes a composite scale and eight subscales: sleep duration, bedtime resistance, night wakings, parasomnias, daytime sleepiness, sleep onset delay, sleep disordered breathing, and sleep anxiety. Higher scores indicate greater sleep problems. Composite scores above a 41 signify clinical sleep problems. The composite scale of the CSHQ has demonstrated adequate internal consistency in community (α = .68-83) and clinical samples (α = .78), and acceptable restest reliability (.62-.79; Gregory et al., 2008; Owens, et al, 2000). Internal consistencies (alphas) for subscales in a community sample ranged from .36 (parasomnias) to .70 (bedtime resistance), and .56

(parasomnias) to .93 (sleep-disordered breathing) in a clinical sample (Hart, Palermo & Rosen, 2005; Owens et al., 2000). The CSHQ has been validated on children ages 4 to 10 and can discern clinical from non-clinical sleep problems (sensitivity = .80, specificity = .72; Owens et al., 2000). The measure has also been used in prior studies to detect sleep problems in youth ages 11 to 18 (Goldman, Richdale, Clemons, & Malow, 2012; Hansen, Skirbekk, Oerbeck, Richter, & Kristensen, 2011). Cronbach's alpha in the current sample was, $\alpha = .82$. The current study examined the total scale and subscales, with the exception of the sleep disordered breathing and parasomnia subscales.

The Family Accommodation Checklist and Interference Scale (FACLIS; Thompson-Hollands, Kerns, Pincus & Comer, 2014). The FACLIS is a 20-item modified version of the family accommodation scale (FAS; Calvocoressi et al., 1995). The FACLIS assesses parental accommodation of youth's anxious behaviors (e.g. avoidance) over the past two weeks with yes/no questions as well as interference ratings of 0 to 8 (0 = no interference, 8 = extreme interference). Language on the FACLIS has been adapted to reflect anxiety in general, rather than OCD-specific symptoms. Greater scores indicate more accommodation, modifications in daily living, and participation in the youth's anxious behaviors. The FACLIS consists of three subscales: Accommodation Scope (i.e., sum of all accommodation items endorsed), Mean Accommodation Interference (i.e., average of interference ratings for all endorsed items for mean burden associated with accommodation), and Total Accommodation Interference (i.e., interference ratings for all endorsed items tallied for total burden associated with accommodation). The original FAS has demonstrated good internal constancy ($\alpha = .80$-.90) as well as strong convergent and adequate divergent validity (Calvocoressi et al.,

1995; Flessner et al., 2011). The FACLIS scales have demonstrated internal consistency (α = .70-.86) and convergent validity the FASA (*r* = .23-.58; Thompson-Hollands et al., 2014). Cronbach's alpha in the current sample was α = .82.

The Family Accommodation Scale-Anxiety (FASA; Lebowitz et al., 2012). Like the FACLIS, the FASA is another accommodation questionnaire that has been modified from the family accommodation scale (FAS; Calvocoressi et al., 1995). The measure contains 9 questions assessing the frequency of accommodation behaviors using a 5-point Likert scale from "never" to "daily." Higher scores indicate greater family accommodation of youth anxiety. Language on the FASA has been adapted to reflect anxiety in general, rather than OCD-specific symptoms. The FASA has displayed internal consistency of α = .90. It also demonstrated convergent and divergent validity, correlating with measures of anxious (*r* = .45) but not depressive symptoms (*r* = .17; Lebowitz et al., 2012). Cronbach's alpha in the current sample was α = .84.

Children's Sleep Hygiene Scale (CSHS; Harsh, Easley & LeBourgeois, 2002). The CSHS is a 22-item parent-report measure about youth's sleep habits (ages 2 to 12). Sleep hygiene items are rated on a 6-point scale (1 = never, 6 = always), with higher scores indicating better sleep hygiene. The CSHS demonstrated adequate validity and reliability (α = .76; Harsh et al., 2002; Henderson, Barry, Bader, & Jordan, 2011; Meltzer, Biggs, Reynolds, Avis, Crabtree & Bevans, 2012). A subsample of parents of children (under 12) completed this measure (*n* = 47). Cronbach's alpha in the current sample was α = .76.

Child/Adolescent-Report

Sleep Self Report (SSR; Owens, Maxim, Nobile, McGuinn & Msall, 2000). The SSR is a 26-item scale that assesses children's sleep behaviors during the most recent typical week. Youth rate the frequency of sleep behaviors from "rarely" (0-1 times per week), "sometimes" (2-4 times per week), and "usually" (5-7 times per week). The SSR includes a composite scale and three subscales: Bedtime, sleep behavior, and daytime sleepiness (Pirinen et al., 2010). Higher scores indicate greater sleep problems. The composite scale has demonstrated adequate reliability in a community sample ($\alpha = .71$; Gregory et al., 2008). The scale was designed for children ages 7 to 12 (Gregory & Eley, 2005b; Owens et al., 2000), but minor modifications were made in the present study for adolescent participants. Cronbach's alpha in the current sample was $\alpha = .77$.

Pre-Sleep Arousal Survey for Children (PSAS-C; Nicassio, Mendlowitz, Fussell, & Petras, 1985). The PSAS-C is a 16-item self-report measure that assesses arousal prior to sleep during a recent typical week. The PSAS-C uses the same items from the adult version (Nicassio et al., 1985), but some items have been reworded with age appropriate language (e.g "hands feel wet" in lieu of "perspiration on hands;" Alfano, Pina, Zerr & Villalta, 2010). Items are rated on a 5 point scale (1 = not at all, 5 = extremely), with higher scores indicating greater pre-sleep arousal. The PSAS-C includes a total scale, as well as cognitive and somatic arousal subscales. Questions relating to cognitive arousal include: "Can't shut off my thoughts" and "worry about falling asleep," whereas somatic items include: "Shortness of breath," and "stomach upset." The PSAS-C has demonstrated consistency in a community sample (total scale $\alpha = .85$, subscales $\alpha = .75$; Gregory et al., 2008) and in anxious youth (total scale $\alpha = .90$, subscales $\alpha = .74$-$.85$; Alfano et al., 2010). PSAS scales have demonstrated discriminant and adequate

convergent validity in adult samples (Nicassio, et al., 1985; Jansson- Fröjmark & Norell-Clarke, 2012). Cronbach's alpha in the current sample was $\alpha = .87$.

Sleep Diary. The sleep diary is a self-report measure of sleep patterns, including: bedtime, time sleep was initiated (lights turned off in room), morning wake time, length and number of night awakenings, sleep latency, and morning sleepiness (1 to 5 Likert scale, 1 = "very sleepy," 5 = "very awake"). Variables analyzed were: Total sleep duration (wake time-bedtime), sleep efficiency (time asleep/total time in bed), sleep latency, length of night awakenings, and morning sleepiness. Youth also recorded whether they attended an activity each morning (e.g. school, summer school, religious activity, camp, or work). Thus, days of diaries were tracked by weekdays vs. weekends and school year vs. vacation. Youth were responsible for completing sleep diaries each morning, but were able to request assistance from parents as needed. Participants were also given a choice between a paper-based and computer-based sleep diary. The two versions of the diary were identical. Diary data were only included if the youth completed a minimum of 3 days.

The sleep diary has been shown to be a reliable and valid estimate of sleep behaviors (Buysse, Ancoli-Israel, Edinger, Lichstein & Morin, 2006; Morin & Epsie, 2003). The sleep diary has convergent validity, showing associations with objective sleep measures (e.g. $r = .52$ with actigraphy, Talbot, McGlinchey, Kaplan, Dahl, & Harvey, 2010). Past research has suggested that participants complete sleep diaries over a one to two week period (Alfano & Gamble, 2009).

Adolescent Sleep Hygiene Scale (ASHS; LeBourgeois, Giannotti, Cortesi, Wolfson, & Harsh 2005). The ASHS is a 28-item self-report scale of sleep hygiene

practices during the past month for youth ages 12 to 18 years old. The ASHS was modified from the parent-report CSHS. Sleep hygiene items are rated on a 6-point scale (1 = never, 6 = always), with higher scores indicating better sleep hygiene. The total scale yielded reliability (α = .80), and subscales have demonstrated variable reliabilities (α = .50-.74; LeBourgeois, et al., 2005). A subsample of adolescents completed this measure (*n* = 20). Cronbach's alpha in the current sample was α = .80.

Diagnostician-Report

The Anxiety Disorder Interview Schedule for Children and Parents (ADIS-IV-C/P; Silverman & Albano, 1996) The ADIS-IV-C/P is a semistructured diagnostic interview that assesses child psychopathology, particularly DSM-IV-R anxiety disorders. The ADIS C/P has excellent retest reliability and interrater reliability (Silverman, Saavadra, & Pina, 2001) and is sensitive to changes associated with treatment-produced change (Kendall et al., 1997). The anxiety disorders section has demonstrated concurrent validity (Wood, Piacentini, Bergman, McCracken, & Barrios, 2002). Diagnosticians provide Clinician Severity Ratings (CSRs) using a 9 point scale (i.e., 0-8), with a "0" indicating no impairment, and an "8" signifying severe impairment. Impairment is defined as impacting several domains of the child's life and/or causing significant distress. A CSR ≥ 4 designates a diagnosis.

Clinical Global Impression- Improvement Scale (CGI-I; Guy, 1976). The CGI-I measures improvement in clinical symptoms, with lower scores representing greater improvement (e.g. 1, very much improved; 2, much improved; 3, minimally improved; 4, no change; 5, minimally worse; 6, much worse; 7, very much worse). Youth who

received a CGI-I score of a 1 or 2 were considered treatment responders. Youth with a CGI-I including and above a 3 were classified as non-responders.

***Therapist Treatment Review Form*.** Clinicians completed a brief form at the posttreatment assessment to note the number of treatment sessions, reason for timing of posttreatment assessment, and changes in medications over the course of therapy. Diagnosticians completed a similar form at the pretreatment assessment regarding recent changes to medications and whether participants had a prior sleep study/treatment.

Data Analysis Plan

Preliminary Analyses. Descriptive statistics examined demographic variables and information related to symptomatology and treatment. Independent sample t-tests assessed site differences by age, sex, income, CGI-I, season change (i.e. school year to vacation, pre to post treatment), sleep medication, prior sleep study, pretreatment CSR, and primary outcome variables at pretreatment. Independent sample t-tests also examined differences between the completer (n = 69) and non-completer sample (n = 36) by pretreatment variables (e.g. see above for site differences). Independent sample t-tests also assessed differences on sleep variables at pretreatment by responder status as well as differences on sleep variables by medication status.

Pearson correlations assessed associations between primary outcome measures, including correlations between parent and child report of SRPs. Paired *t*-tests assessed changes in anxiety on the ADIS-IV principal anxiety CSR from pre- to posttreatment. Pearson correlations also examined whether number of treatment sessions is associated with treatment outcome. Finally, paired *t*-tests between the pre- and post-sleep diary examined differences by the ratio of "activity - no activity days/total number of days"

(e.g. did youth cite an activity to get up for in the morning, such as school, camp, work, or a religious service; did the ratio change by time point?).

Prior to analyses, independent-samples t-tests tested whether individuals missing data on any of the variables of interest significantly differed from individuals who were not missing data. Missing data analyses indicated no significant pretreatment differences (all *p*'s > 0.05). We proceeded as data missing at random and used mean imputation and pairwise deletion. Only 1.81% of data was missing. Sphericity was assumed in all repeated measures ANCOVA analyses (> .90).

Aim 1: SRP change from pre- to post-treatment and association with treatment response. A repeated measures ANCOVA assessed whether SRPs (i.e. CSHQ, SSR, sleep diary) reduced from pre- to post-treatment. Interactions with treatment responder status on the CGI-I were examined. Three covariates were entered: (1) age (i.e. to help rule-out sleep changes due to normative developmental sleep-shifts), (2) change in psychotropic medication, and (3) change in season over therapy. Medication status was dummy coded as "change in medication" or "no change in medication." Change in medication was operationalized as change in the type of psychotropic medication (e.g., Zoloft to Cymbalta) or in dosage of medication during or within 6 weeks of starting treatment. Different subclasses of drugs were not broken down and uniquely entered as covariates in the ANCOVA given the small number of participants in each group. Finally, season change was dummy coded (1) pretreatment assessment during school year and posttreatment during vacation, and (2) pretreatment assessment during vacation and posttreatment during school year. Including said covariates addressed concerns that change over treatment may be driven by change in sleep schedules by season. A

Bonferoni correction was applied for CSHQ subscale analyses (e.g. $p = .05/6$ subscales). In cases with measures that use cut-scores, chi squared analyses assessed whether there were differences in SRP improvement by responder status.

Aim 2: Change in pre-sleep arousal over treatment in relation to SRPs at posttreatment. A repeated measures ANCOVA assessed whether pre-sleep arousal differed from pre- to post-treatment and whether treatment responder status predicted arousal at posttreatment, accounting for potential covariates. Change in pre-sleep arousal was calculated by subtracting pretreatment scores from posttreatment scores. Change in arousal was entered into a hierarchical linear regression to predict posttreatment SRPs (e.g. CSHQ total, SSR total, sleep diary variables), accounting for covariates of pretreatment SRPs and other covariates.

Exploratory Aim 1: Change in family accommodation predicting SRPs at posttreatment. Change in family accommodation from pre- to post-treatment was entered into a hierarchical linear regression to predict SRPs at posttreatment, controlling for SRPs at pretreatment and age, season change, and medication change.

Exploratory Aim 2: Sleep hygiene mediating the relationship between family accommodation and SRPs. Paired t-tests assessed whether sleep hygiene significantly changed from pre- to post-treatment. Family accommodation at pretreatment and sleep hygiene measured at session 1 was entered into a multiple regression to predict various SRPs at posttreatment (e.g. CSHQ total, SSR total, sleep diary variables). Analyses were run separately for children and adolescents as different, developmentally sensitive, measures of sleep hygiene were administered.

Results

Preliminary Analyses

Means and standard deviations of outcome variables are presented in Table 1.4. Independent sample t-tests assessed differences by (a) site and (b) completer status (completer [*n* = 69] and non-completer sample [*n* = 36]) among variables of age, sex, income, CGI-I, season change, sleep medication, prior sleep study, pretreatment CSR, and primary outcome variables at pretreatment. There was a significant difference by pretreatment CSR (t=3.35, p=.001), in which average CSR was higher at Temple University (*M* = 5.69, *SD* = .84) compared to Boston University (*M* = 4.85, *SD* = .80). There were no other significant site differences. Because CSR was not directly assessed in the main analyses, site was not included as a control variable. There were no significant differences by completer status.

The average number of days completed on the pre sleep diary was 9.98/14 and 6.17/7 on the post sleep diary. Paired t-tests between the pre- and post-sleep diary examined differences by the ratio of "activity - no activity days/total number of days" (e.g. did youth cite an activity to get up for in the morning, such as school, camp, work, or a religious service; did the ratio change by time point?). There were no significant differences (*t* = -.47, *p* = .64). The type of season change that youth experienced between pre and post-treatment was relatively proportional: School to vacation (*n* =17) and vacation to school (*n* = 15).

Independent sample *t*-tests assessed whether pretreatment sleep means differed by the presence of medication. Youth on medication showed greater SRPs on diary sleep latency (*t* = -3.15, *p* = .03), diary sleep efficiency (t = 2.81, p = .007), CSHQ (*t* = -2.41, *p* = .02), and PSAC (*t* = -2.13, *p* = .04). No differences were present on total time in bed,

sleepiness, nighttime wakings, or SSR. When examined by medication class (e.g. SSRIs, stimulants, or sleep-aides) the findings were not significant; however, these groups may have been too small to detect differences. Another set of independent sample *t*-tests were performed to determine whether responders vs. non-responders differed on any primary sleep measures at pretreatment. No significant differences were found with one exception; responders had better sleep efficiency at pretreatment ($t = -2.56$, $p = .01$). The difference was also present at posttreatment ($t = -2.75$, $p = .008$).

Correlations between main outcome measures are presented in Table 1.5. Parent report of SRPs was positively correlated with child self-report at concurrent time points on the SSR and diary variables. Pre-sleep arousal was not significantly correlated with parent report of SRPs; however, it was positively and significantly correlated with child self-report on the SSR, sleep latency, and sleep efficiency. FACLIS accommodation significantly correlated with a range of SRP metrics. Regression analyses were performed with accommodation predicting pretreatment SRPs, controlling for pretreatment anxiety (i.e. to demonstrate correlation not just due to high-level of anxiety). FACLIS Scope significantly predicted scores on the CSHQ ($\beta = .45$, $t = 3.79$, $p < .001$), SSR ($\beta = .31$, $t = 2.48$, $p = .02$), and sleep diary efficiency ($\beta = .33$, $t = -2.69$, $p = .009$); FACLIS interference predicted CSHQ ($\beta = .43$, $t = 3.62$, $p = .001$) and sleep diary efficiency ($\beta = -.38$, $t = -3.12$, $p = .002$). Of note, there were no significant correlations between pretreatment CSR and any sleep measure at pretreatment.

Anxiety improvement was tested by a paired *t*-test of principal CSR on the ADIS C/P. Participants' CSR was significantly lower at posttreatment ($M_{difference} = 2.25(1.67)$ $t = 11.20$, $p < .001$, $d = 1.50$). On the CGI-I, 47 youth (68.1%) met treatment responder

status. The number of sessions was associated with posttreatment CSR (r = .25, p =.04) and CGI (r = .29, p =.02), such that fewer total sessions were associated with lower (better) CSRs.

Aim 1: To assess whether sleep problems decrease pre- to post-treatment and interact with treatment response

Counter to expectations, relatively few participants presented with elevated SRPs by child report (see Table 1.4). However, the majority of participants had elevated SRPs by parent report. Relative to norms of youth without anxiety (ages 6-11), the total average of CSHQ (see Table 1.4) was higher at both time points (M = 38.80; Owens et al., 2000). Of those classified as "treatment responders," 24 youth were above the CSHQ cut-off (53% of treatment responder subsample), relative to the 16 non-responders above the CSHQ cut-off (80% of the non-responder subsample) at posttreatment. A chi-squared test revealed a non-significant trend of responders as more likely to be below the clinical cut-off at posttreatment (x^2=3.56, p = .059). However, results should be interpreted with caution as the cut-off was established for youth aged 6-11.

There was a significant interaction between treatment response and time: Youth who responded to CBT evidenced a significant decrease in total CSHQ score ($M_{difference}$ = 3.99; F = 8.52, p = .005, η^2=.13; see Table 1.4 and Figure 1.2), with independent samples t-tests yielding significant differences between responders and nonresponders at posttreatment (t = 4.89, p = .009). Of note, CSHQ score at posttreatment for responders hovered just above the clinical cut-off of 41 (M = 41.87). Regarding subscale interactions of treatment response by time, youth who responded to CBT evidenced a significant decrease in the bedtime resistance subscale ($M_{difference}$ = 1.08; F = 8.02, p = .006, η^2=.12),

the sleep anxiety subscale ($M_{difference}$ = .87; F = 11.96, p = .001, η^2=.17), and the sleep duration subscale ($M_{difference}$ = .41; F = 5.49, p = .023, η^2=.09; see figures 1.3-1.4). However, sleep duration was no longer significant following the Bonferroni correction (p = .023 > p = .008). There were no significant interactions for the sleep onset, night wakings, or sleepiness subscales. No main effects of time were observed in any of the CSHQ analyses. When the responder subsample was separately analyzed in a repeated measures ANCOVA, SRPs significantly decreased on the total CSHQ score (F = 5.17, p = .03, η^2=.11), CSHQ bedtime resistance (F = 11.06, p = .002, η^2=.22), and CSHQ sleep anxiety (F = 15.54, p < .001, η^2=.28).

There were no main effects of time or interactions by treatment on any SSR scale or sleep diary variable. Of note, when treatment was analyzed as a continuous variable on the CGI-I, there was a non-significant trend for the SSR bedtime subscale (F = 2.35, p = .065), in which youth who were "very much improved" compared to others had lower scores at posttreatment ($M_{difference}$ = 1.04).

Aim 2: To assess whether change in pre-sleep arousal predicts sleep problems at posttreatment

There was a significant reduction in total pre-sleep arousal (F = 4.51, p = .038, η^2=.07) and cognitive arousal (F = 5.95, p = .02, η^2=.09) from pre- to post-treatment (Figure 1.5). There was no significant change for somatic pre-sleep arousal over treatment. Treatment responder status did not predict pre-sleep arousal at posttreatment. Change in pre sleep arousal (total, cognitive, and somatic) did not predict any main sleep outcome at posttreatment.

Exploratory Aims: Change in family accommodation and hygiene predicting SRPs at posttreatment; hygiene mediation of accommodation and SRPs

Paired t-tests revealed significant change in accommodation on each metric of accommodation: FASA ($M_{difference}$ = 5.13, t = 5.71, p <.001), FACLIS accommodation scope ($M_{difference}$ = 2.21, t = 4.65, p <.001), and FACLIS mean accommodation interference ($M_{difference}$= 12.83, t = 5.93, p <.001). Change in accommodation was entered into a regression to predict SSR total, sleep diary, PSAS-C scales and CSHQ total and previously significant subscales (e.g. sleep anxiety and bedtime resistance). Change in FASA and FACLIS variables did not predict any outcomes (when examined by total sample or responder subsample). There was a non-significant trend in which reduction in FACLIS mean accommodation interference trended toward reduced posttreatment CSHQ sleep anxiety (β = -.22, t = -1.90, p = .063).

There was no significant change in sleep hygiene from pre- to post-treatment on either the CSHS or ASHS (i.e. sleep hygiene). Sleep hygiene did not mediate accommodation and SRPs. Although not included in the initial hypotheses, we assessed whether pretreatment sleep efficiency predicted treatment response given differences by responder/nonresponder status at pretreatment (see preliminary analyses). When entered into a linear regression, sleep efficiency predicted lower CSR on the ADIS (β = -10.19, t = -2.00, p = .05; e.g. greater sleep efficiency predicts lower anxiety at posttreatment) and a trend toward predicting responder status on the CGI-I in logistic regression (β = 12.45, p = .058).[1]

Discussion

[1] The above analyses were run after eliminating an outlier > 2 SDs. When the outlier was included sleep efficiency continued to predict CSR in linear regression (β = -9.85, t = -2.61, p = .01) and CGI-I became significant in logistic regression by treatment responder (β = 13.02, p = .03),

The present findings indicate that parent-report SRPs (not child self-report) decreased after CBT for youth anxiety. Further, youth who respond to CBT, as opposed to nonresponders, showed significant improvement in parent-reported SRPs. The present results are consistent with previously reported improvement to SRPs following CBT for youth anxiety (Kendall & Pimentel, 2003; Storch et al., 2008). In particular, the present findings indicate that sleep anxiety and bedtime resistance evidence change. Thus, sleep issues around bedtime, rather than quantitative sleep variables (e.g. sleep latency, duration), are those sensitive to CBT for youth anxiety.

The findings are consistent with the notion that improvement in general anxiety helps to alleviate nighttime specific anxiety. For example, youth may be less resistant to separate from parents to go to bed. Likewise, general parent management training implemented in treatment may help parents navigate issues related to bedtime limit setting (Adam, Snell & Pendry, 2007; Billows et al., 2009). Importantly, although the present study lacked objective measurement of SRPs, the problem areas that showed change are those that are best measured by parent/self-report.

Despite improvement, elevated parent-reported SRPs remained at posttreatment, with averages among the responder sample hovering at the cut-off on the CSHQ. The results are consistent with adult studies that find modest to moderate improvement in SRPs, yet residual SRPs present at posttreatment (Belleville et al., 2010). Clinically, these data suggest that CBT for anxiety, while successful for anxiety, may not be sufficient on its own to treat SRPs. Although some modest improvement may occur, results suggest that youth with high-level SRPs need additional sleep focused treatment. Future research should investigate the efficacy of anxiety treatment that does or does not

include specific sleep modules (e.g. hygiene training, stimulus control). Additionally, stepped-care research would help determine the optimal order of services for patients with elevated anxiety and SRPs.

Total pre-sleep arousal decreased significantly across treatment, although changes in arousal did not predict SRPs at posttreatment. Consistent with past research (Alfano et al., 2010), cognitive, rather than somatic, arousal was particularly linked in anxious youth. Anxiety treatment may provide youth skills to modulate their anxiety, and accordingly their arousal, at bedtime. Exposure, in addition to relaxation and changes in self-talk, is a pathway to anxiety reduction that may generalize to reduced evening arousal. However, the present results should be interpreted with caution as changes in arousal were not associated with treatment response. Even so, small changes in cognitive arousal over treatment may translate to meaningful impact for youth, even if they are not associated with change in SRPs.

Treatment-produced changes in parent-reported SRPs were found, but changes were not present on child self-reports (Sleep Self Report; Sleep Diary). Although there were significant correlations between parent and child-reported SRPs, youth endorsed relatively fewer sleep problems. The low rate of child-reported SRPs at pretreatment may account for the absence of SRP change over treatment. These results are consistent with anxiety assessment data in which youth report fewer symptoms relative to parents (Safford, Kendall, Flannery-Schroeder, Webb & Sommer, 2005). Additionally, anxious youth underreporting SRPs when contrasted to objective measures has been documented in past research (Forbes et al., 2008). Given these findings, it is important for clinicians to

consider multi-reporter assessment of SRPs in youth with anxiety. In doing so, clinicians can a gain a more comprehensive understanding of youth sleep patterns.

Regarding exploratory analyses, sleep hygiene did not change with treatment and suggests that CBT for youth anxiety does not improve sleep behaviors through generalization of skills. Youth with particularly bad sleep habits may need additional sleep-specific coaching/treatment. Parental accommodation, even after controlling for initial anxiety severity, was positively associated with SRPs. Parents who accommodate inadvertently reinforce anxiety, perhaps contributing to the maintenance of anxiety/arousal at bedtime and/or sleep incompatible behaviors (e.g. co-sleeping, leaving bed for reassurance seeking). Whereas absolute levels of accommodation were related to SRPs, change in accommodation did not predict reduced SRPs at posttreatment. Although these variables may be linked, change in accommodation may not be a direct enough pathway to significantly influence SRPs. Consequently, no mediation was found between sleep hygiene, accommodation, and SRPs.

One unexpected finding was that sleep efficiency at pretreatment predicted both anxiety and treatment response at posttreatment, after controlling for pretreatment anxiety. Both responders and nonresponders to treatment evidenced sleep efficiency that was within the normative range (e.g. > .85) and differences between groups were no greater than .04; nonetheless, small discrepancies in efficiency can represent dramatic differences in sleep. Importantly, sleep efficiency can be said to be a comprehensive metric of self-reported SRPs, taking into account sleep latency, night wakings, and sleep duration. Youth with reduced sleep efficiency struggle with regulation of emotion, attention, and memory (Beebe, Rose & Amin, 2010; Kopasz et al., 2010; Sadeh, 2007)

and their capacity to actively engage and participate in treatment may be limited. Problems in self-regulation due to SRPs may exacerbate and maintain anxiety. Given differences in responder status by sleep efficiency, it is important for anxiety-focused clinicians to assess SRPs and monitor them throughout therapy. Major SRPs identified at onset may represent a barrier to successful treatment.

The study benefited from a number of methodological strengths. The multi-site approach and heterogeneous sample, both in terms of age and diagnosis, contributes to the generalizability of findings. External validity was buttressed by the flexibility of treatment. Additionally, inclusion of participants with non-anxiety comorbidities and on medication mirrored "real-world youth." In contrast with some prior treatment outcome research of youth sleep and anxiety, the present study used validated sleep questionnaires and reports were collected from both parents and children. Treatment adherence was coded and therapists demonstrated very high adherence to CBT protocol. Finally, missing data were minimal across main outcome measures.

Despite study strengths, there are limitations worth noting. First, the study lacked objective sleep measures. Although the subjective measures used have associations with objective measures (Talbot, et al. 2010), polysomnography and actigraphy remain the "gold-standards" of assessment and discrepancy has been shown to exist between subjective and objective measures in anxious youth samples (Alfano & Kim, 2011; Forbes et al., 2008). The use of objective measurement may have yielded findings in quantitative variables (e.g. sleep latency, night wakings) that contrasted youth's self-report of SRPs. Second, due to the longitudinal design, youth were assessed over different seasons (e.g. pretreatment during school and posttreatment during vacation, or

vice-versa). Youth's sleep habits commonly change by season when morning obligations shift. That said, an approximately equal number of youth experienced each type of seasonal shift and seasonal shift was accounted for in all analyses. Even during vacation season, many youth had early morning obligations, which mitigated differences in sleep behaviors during the school year (e.g. camp, work). Further analyses revealed no difference in the proportion of "morning obligation days" to "non-morning obligation days" from pre- to post-treatment. Third, given the heterogeneity of the sample yet need for developmentally sensitive measures, sleep hygiene had to be analyzed separately to reflect the two developmentally-tailored sleep hygiene measures. As a consequence, sleep hygiene may have been underpowered to detect effects, particularly for the adolescents. However, these analyses were exploratory and merit further investigation. The absence of a control condition could be a limitation but this issue was partially addressed by (a) having a two-week baseline of sleep problems by diary report, and (b) finding an interaction with treatment and reduced SRPs at post, indicating that variables in addition to time are related to change in symptoms. Finally, the sample was demographically restricted, with an overrepresentation of upper-middle class Caucasian families.

Whereas the findings support the need for interventions to ameliorate anxiety and to address SRPs, a joint protocol that actively targets both needs may be the next step in research. Although learned skills may overlap in SRP and anxiety treatment and can theoretically cross topics, there may be a limit in the youth's ability to generalize the skills. Families may need therapists to explicitly guide them in applying skills across the shared range of issues. Additionally, a more comprehensive intervention can include elements that are unique to each problem area. Future research will be needed to better

understand the mechanisms that underlie youth anxiety and sleep problems as well as to develop transdiagnostic treatment.

End Notes

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Table 1.1

Descriptive Information for Study Participants

Variable	*n*	%
Child age in years	*M* = 10.86	*SD* = 2.99
Gender		
Males	31	44.9
Race		
Caucasian	58	84.1
Hispanic	1	1.0
Asian-American	3	2.9
African-American	1	1.0
Other	6	5.7
Income		
$0-19,999	1	1.4
$20,000-39,999	6	8.7
$40,000-59,999	8	11.6
$60,000-79,999	5	7.2
Over $80,000	44	63.8
Not reported	5	7.2
Site		
Temple University	56	81.2
Boston University	13	18.8

Table 1.2

Pretreatment Diagnostic Information for Study Participants

Variable	*n*	%
Principal Anxiety Diagnosis		
GAD	33	47.8
Social Phobia	14	20.3
Specific Phobia	9	13.0
SAD	8	11.6
Selective Mutism	2	2.9
OCD	1	1.5
Panic Disorder	1	1.5
AD-NOS	1	1.5
Prior Sleep Study/Treatment		
Yes	6	9.0
No	60	87.0
Missing	3	4.0
Medication		
Sleep Aid	6	8.7
SS(N)RI/Antidepressant	9	13.0
Stimulant	6	8.7
Total	21	30.4
Non-Anxiety Comorbidity		
ADHD	8	11.6
ODD	3	4.3
MDD/Dysthymia	6	8.7

Note. ADHD = Attention Deficit/Hyperactivity Disorder, AD-NOS = Anxiety Disorder-Not Otherwise Specified, GAD = Generalized Anxiety Disorder, MDD = Major Depressive Disorder, OCD = Obsessive Compulsive Disorder ODD = Oppositional Defiant Disorder, SAD = Separation Anxiety Disorder

Table 1.3

Treatment and Posttreatment Diagnostic Information for Participants

Variable	*n*	%
Therapy Protocol		
Coping Cat	57	82.6
MATCH	8	11.6
CBT for SM	2	2.9
Riding the Wave	1	1.4
CBT for OCD	1	1.4
Number of Sessions		
10-11	4	5.8
12-13	6	8.7
14-15	10	14.5
16	49	80.0
Why Therapy Ended		
Remission	25	36.2
Family discontinued	4	5.8
Reached session 16	40	58.0

Note. CBT = Cognitive Behavioral Therapy, MATCH = Modular Approach to Therapy for Children with Anxiety, Depression, Trauma, or Conduct Problems, OCD = Obsessive Compulsive Disorder, SM = Selective Mutism.

Table 1.4. Means and Standard Deviations of Primary Outcome Measures

	Pretreatment			Posttreatment		
	Responder	Nonresponder	Total	Responder	Nonresponder	Total
CSHQ						
Total	45.75(7.91)	45.10(7.75)	45.62(7.71)	41.87(6.90)*	46.76(7.83)	43.38(7.34)
Resist.	8.42(2.00)	8.00(1.86)	8.30(1.95)	7.43(1.66)*	8.27(1.96)	7.71(1.79)
Anx.	5.63(1.45)	5.11(1.41)	5.48(1.46)	4.78(1.20)*	5.50(1.34)	5.01(1.29)
Dur.	4.02(1.09)	3.74(.81)	3.94(1.01)	3.60(.69)	4.07(1.10)	3.38(.86)
Onset	1.30(.70)	1.32(.59)	1.31(.66)	1.31(.67)	1.36(.73)	1.32(.68)
Wak.	3.61(.83)	3.61(.79)	3.61(.81)	3.59(.87)	3.70(1.14)	3.63(.96)
Sleep.	11.47(2.24)	11.43(2.30)	11.46(2.24)	10.78(2.10)	11.22(2.89)	10.93(2.37)
SSR						
Total	35.94(6.86)	36.10(7.76)	35.99(7.08)	34.18(6.25)	35.22(6.26)	34.27(6.20)
Bedtime	18.97(4.10)	19.18(4.06)	19.04(4.06)	17.52(3.64)	18.90(3.67)	17.84(3.64)
Beh.	10.32(3.00)	9.98(2.56)	10.21(2.87)	9.72(2.77)	9.48(2.44)	9.59(2.61)
Sleep.	6.74(1.90)	7.18(2.46)	6.89(2.05)	6.97(2.08)	6.88(2.02)	6.89(2.05)
DIARY						
Wak.	5.99(9.50)	6.29(7.09)	6.51(9.39)	3.26(5.47)	4.10(5.70)	3.50(5.44)
Lat.	18.95(12.45)	24.81(27.43)	21.32(18.50)	17.06(11.86)	31.56(36.64)	21.47(22.61)
Sleep.[a]	3.24(.68)	3.24(.79)	3.24(.69)	3.10(.75)	3.09(.65)	3.09(.74)
Eff.	.93(.04)	.89(.08)	.92(.06)	.93(.06)	.90(.05)	.92(.05)
Dur.	605(37)	601(72)	602(52)	624(89)	627(94)	624(88)
PSAS-C						
Total	28.05(9.58)	30.15(10.92)	28.71(9.99)	25.30(7.76)	26.28(7.69)	25.60(7.70)*
Som.	12.96(5.91)	12.90(5.72)	12.94(5.80)	11.77(3.96)	11.66(4.07)	11.74(3.97)
Cog.	15.10(5.29)	17.27(6.92)	15.78(5.88)	13.53(5.39)	14.62(5.60)	13.87(5.44)*

Note. Anx = Sleep Anxiety, Beh. = Sleep Behavior, Cog = Cognitive Subscale, Dur = Sleep Duration, Eff. = Sleep Efficiency, Lat.= Sleep Latency, Onset = Sleep Onset, Resist = Bedtime Resistance, Sleep = Sleepiness, Som. = Somatic Subscale, Wak = Night Wakings. *$p < .05$. [a]Diary variables are in minutes with the exception of "Sleep" and "Eff".

Table 1.5. Correlations of Pretreatment Outcome Measures

	1	2	3	4	5	6	7	8	9
1	x								
2	.43*	x							
3	.46*	.37*	x						
4	-.37*	-.35*	-.83*	x					
5	.20	.43*	.33*	-.26t	x				
6	.24	.56t	.49t	-.36	.88*	x			
7	.66*	.46*	.32t	-.19	.04	-	x		
8	.24	.25t	.01	-.09	.10	-.11	.27	x	
9	.43*	.28t	.34*	-.42*	.28t	.34	.38*	.55*	x

Note. 1 = Child Sleep Habits Questionnaire (parent report), 2 = Sleep Self Report (child report), 3 = Sleep Diary Latency (child report), 4 = Sleep Diary Efficiency (child report), 5 = Pre-sleep Arousal (child report), 6 = Adolescent Sleep Hygiene Scale (child report), 7 = Child Sleep Hygiene Scale (parent report), 8 = Family Accommodation Scale-Anxiety (parent report), 9 = Family Accommodation Checklist and Interference Scale (accommodation scope subscale; parent report). $^{t}p < .05$, $^{*}p < .01$

Table 1.6

Participants over Clinical Cut-offs for Sleep Problems

Variable	*n*	%
CSHQ (≥ 41)		
Pre	47	72.3
Post	40	60.6
Diary Latency (≥ 30 min)		
Pre	14	20.6
Post	11	16.4
Diary Efficiency (≤ .85)		
Pre	5	7.9
Post	5	7.9

Note. CSHQ = Child Sleep Habit Questionnaire.

Figure 1.1 Consort Diagram

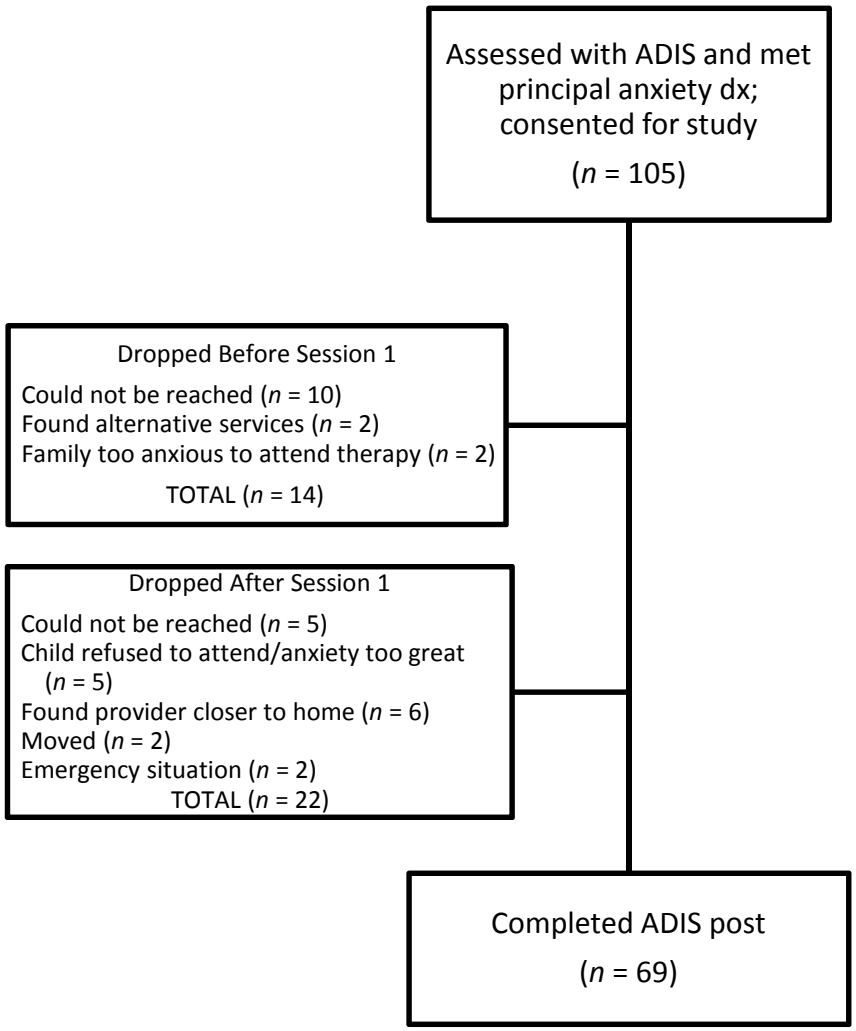
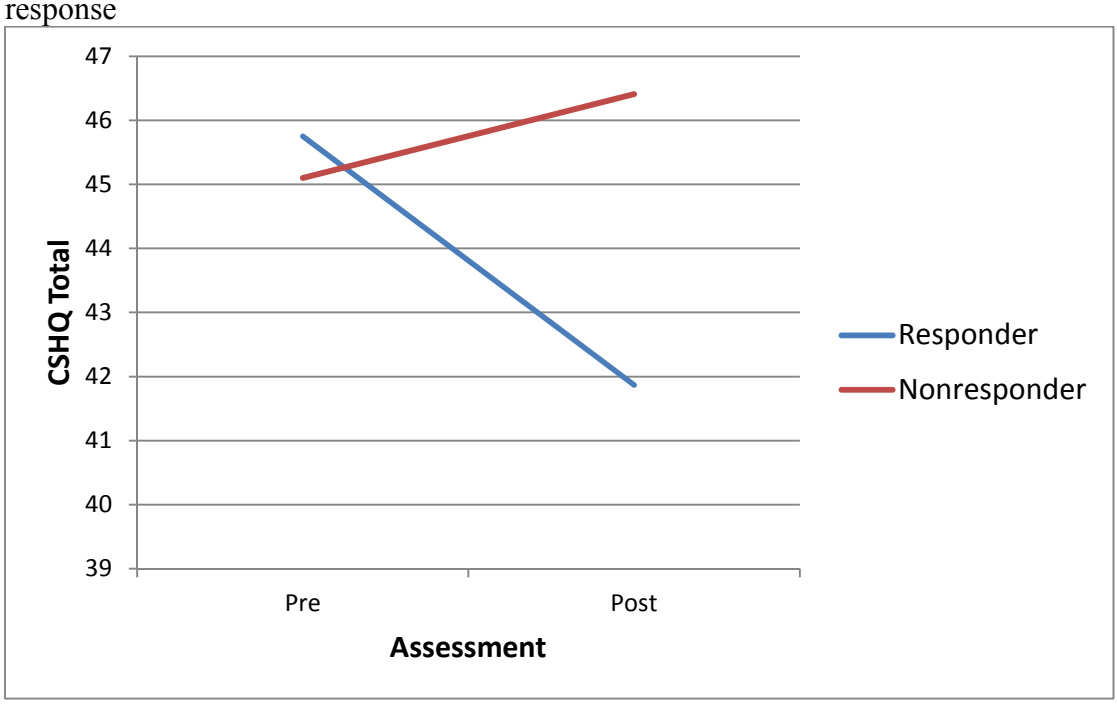
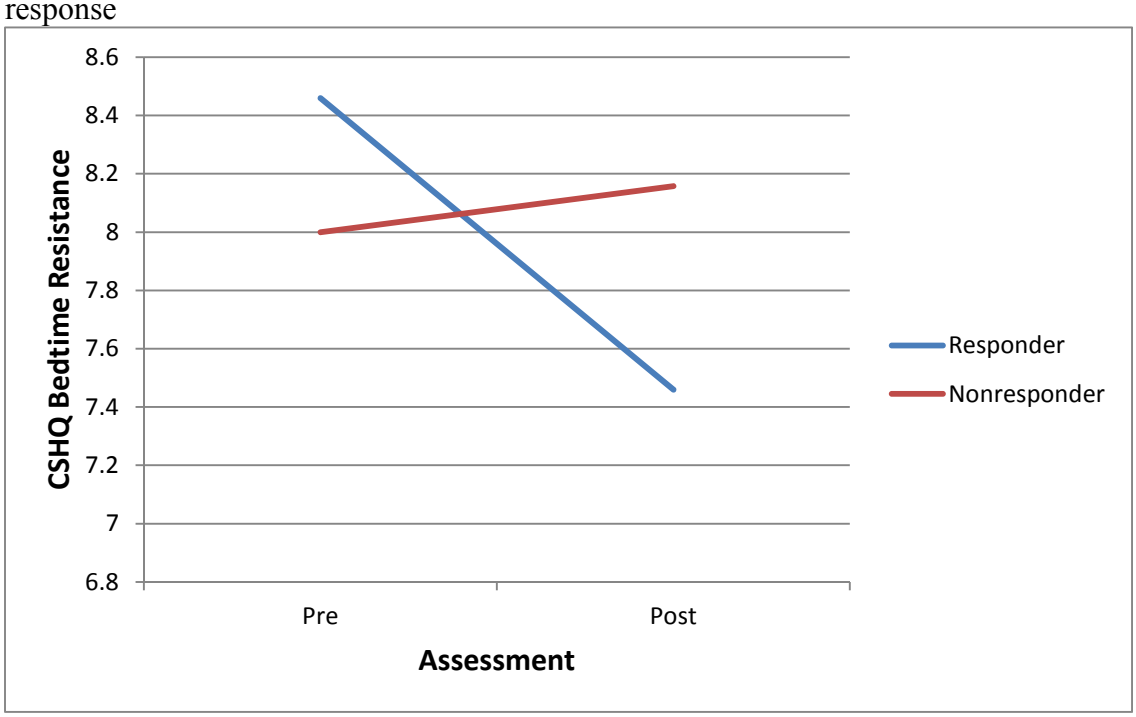


Figure 1.2. Pre- to post- change in parent-reported total sleep problems by treatment response



Note. Higher CSHQ score indicates greater sleep problems.

Figure 1.3. Pre- to post- change in parent-reported bedtime resistance by treatment response



Note. Higher CSHQ score indicates greater bedtime resistance.

Figure 1.4. Pre- to post- change in parent-reported sleep anxiety by treatment response

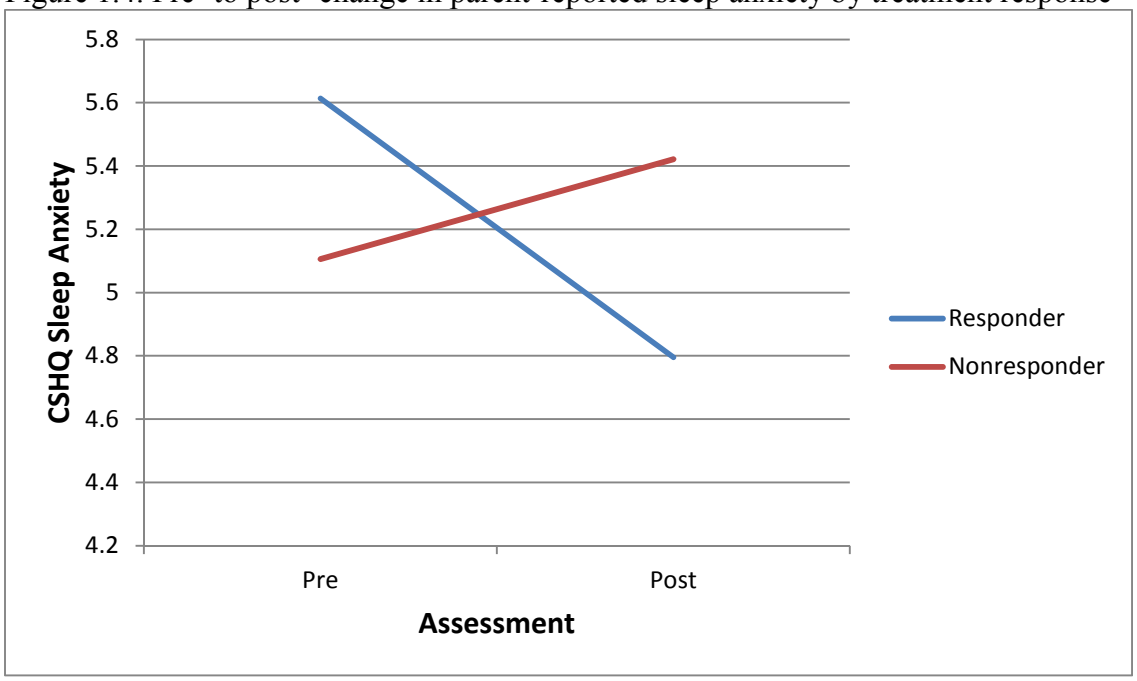
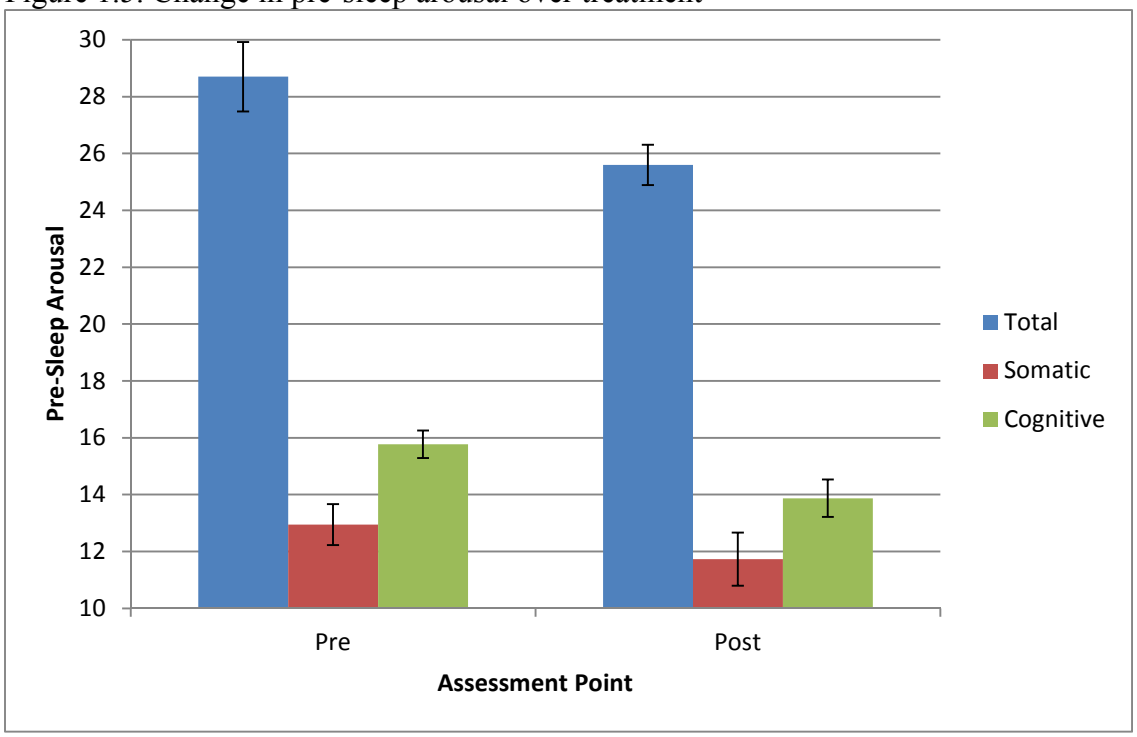


Figure 1.5. Change in pre-sleep arousal over treatment



Note. Higher numbers indicate greater pre-sleep arousal

CHAPTER 2

LITERATURE REVIEW

There is a growing literature regarding the relationship between sleep problems and psychological disorders, particularly anxiety disorders. Among adults, approximately 52% of individuals with sleep problems have a comorbid psychological disorder (Ustun et al., 1996). Sleep problems are a particular concern for youth as nighttime fears, nightmares and involuntary bedtimes are common among this population. Sleep problems are one of the most frequently presented concerns at pediatric clinics, and show high comorbidity rates with internalizing and externalizing disorders (Chorney, Detweiler, Morris & Kuhn, 2008). Furthermore, disturbed sleep has consistently been associated with deficits in executive function, including emotion regulation (Giannotti, Cortesi, Sebastiani & Ottaviano, 2002; Kopasz et al., 2010; Sadeh, Gruber, & Raviv, 2002).

Recent research has emphasized the connection between sleep problems and elevated anxiety in school-aged youth (grades 1-12). Several studies have supported an association between anxiety and sleep problems (Leahy & Gradisar, 2013), yet this area of inquiry remains largely understudied. The dearth of longitudinal, experimental, and more methodologically rigorous research limits conclusions of the current literature. Further, empirical evaluation of underlying mechanisms and shared treatment effects for anxiety and sleep in youth is largely lacking. Such limited understanding of the sleep and anxiety association among youth in surprising given (a) the high comorbidity between the two problems, (b) both are associated with an array of functional impairments, and (c) the field's shift toward a dimensional framework of psychopathology. Research on anxiety

and sleep problems in youth is an exciting and budding area of inquiry with profound implications for clinical case conceptualization, assessment, and treatment.

The present review will provide an overview of the current state of literature regarding anxiety and sleep problems in school-aged youth, and suggest areas for future research. The review begins by outlining general issues, such as sleep problems in non-anxious youth, sleep across development, sleep measurement, and negative functional outcome related to sleep disturbances. Inquiry into these topics will provide context to understand the current state of the anxiety and sleep literature, including methodological shortcomings and areas for further investigation. Second, the review explores associations between anxiety and sleep among youth with anxiety disorders, with an emphasis on studies' collective strengths and limitations. The review transitions to possible shared mechanisms to account for the anxiety and sleep overlap. A brief review of neurological explanations will lead to a discussion of the underlying role of pre-sleep arousal. Other potential shared mechanisms, such as sleep hygiene and parental accommodation, are proposed and reviewed. Review of comorbidity and theoretical mechanisms of sleep and anxiety culminates into a discussion of intervention-focused research and implications for new treatments. Future areas of research are highlighted throughout and are summarized at the end of the article.

Combinations of relevant search terms were entered into PsycINFO and MEDLINE to collect articles for the present review (years 1980-2014). Keywords included "youth, adolescents, children, sleep, insomnia, nightmares, night terrors, parasomnias, sleep hygiene, actigraphy, polysomnography, anxiety, fear, phobia, and panic." Additional articles were found by reviewing the reference sections of relevant

articles. We limited studies to those published in English and in peer reviewed journals. Inclusion criteria for the main review of studies (e.g. SRPs in anxious youth) involved: youth who were diagnosed with a clinical anxiety and/or obsessive-compulsive disorder, measurement of SRPs with objective or subjective report, and non-case study reports. In areas without extant research (e.g. CBT treating concurrent SRPs and anxiety), studies from the adult literature were examined.

Sleep Problems in Youth

Regular disturbances in sleep are classified as "sleep related problems" (SRPs). SRPs are a heterogeneous group of difficulties that interfere with sleep continuity and quality and include: problems initiating or maintaining sleep, bedtime resistance, and parasomnias[2] (Hudson, Gradisar, Gamble, Schniering & Rebelo, 2009). SRPs are relatively common, affecting 25% to 40% of youth (Mindell, Meltzer, Carskadon & Chervin, 2009), and research has shown that up to 90% of youth referred to mental health services have at least one SRP (Alfano, Zakem, Costa, Taylor & Weems, 2009). Indeed, over an 11 year period, there were approximately 18.6 million visits by youth to outpatient settings for SRPs, with the highest rates among school-aged children (Stojanovski, Rasu, Balrishnan & Nahata, 2007). For some youth, SRPs cause substantial distress and impairment and are characterized as sleep disorders by the Diagnostic and Statistical Manual of Mental Disorders—Fifth Edition (DSM-5; American Psychiatric Association, 2013), or the International Classification of Sleep Disorders—Second Edition (ICSD-2; American Academy of Sleep Medicine, 2001). Prevalence rates of sleep disorders range from 3.7% to 20% (Johnson, Roth, Schultz & Breslau, 2006;

[2] Youth parasomnias include nightmares, night terrors, sleep walking, sleep paralysis, and confusional arousals

Meltzer, Johnson, Crosette Ramos & Mindell, 2010; Owens, Spirito, McGuinn & Nobile, 2000), highlighting their ubiquity among youth.

Sleep is of particular importance in youth undergoing maturation. It is recommended that children and adolescents receive 9 to 11 hours of sleep per night (American Academy of Sleep Medicine), yet the average school-aged child spends 8.5 hours asleep, and the average adolescent receives 7.5 hours of nightly sleep (National Sleep Foundation, 2006, 2008. Additionally, 45% of youth have "insufficient sleep" as defined by less than 8 hours of nightly sleep. Daytime sleepiness is frequently reported by youth, particularly adolescents (Dewald, Meijer, Oort, Kerkhof & Bogels, 2010), and teachers commonly report on the "epidemic" of students falling asleep in class (Wolfson & Carskadon, 1996).

Developmental Changes in Sleep

When assessing SRPs it is important to be mindful of normative developmental shifts in sleep. From birth until age 2 a child spends more time asleep than awake (Anders, Sadeh, & Appareddy, 1995), highlighting the importance of sleep to neural maturation during this age. The balance between sleep and wake periods becomes more equivalent as youth reach 5 years of age (Dahl, 1996b). There is also a rise in co-sleeping which peaks at age 4 but is relatively common until age 7 and declines into mid- and late-childhood (Jenni, Fuhrer, Iglowstein, Molinari & Lardo, 2005). Frequency of nighttime awakenings follows a similar pattern. Nocturnal enurersis is relatively common among pre-school and school-aged children and may account for the increase in nighttime awakenings during this period (Meltzer et al., 2010). Additionally, roughly 25% of healthy school-aged children have trouble falling asleep (Stein, Mendelsohn, Obermeyer,

Amromin & Benca, 2001). Longitudinal investigations suggest that sleep onset difficulties and bedtime resistance are often fleeting among healthy children (Jenni et al., 2005).

The transition to adolescence produces major changes to sleep: total sleep time decreases, bedtimes become later, sleep onset latency increases (time to sleep), slow-wave and REM sleep reduces, and variability in weekday/weekend sleep behavior widens. Variations by gender are minor, with girls waking up earlier on weekdays (Sadeh, 2008; Wolfson & Carskadon, 1998). Total sleep time decreases because bedtimes extend yet weekday wake times remain early due to school. Notably, adolescents tend to report greater daytime sleepiness compared to children (Carskadon, 1982; Sadeh, Dahl, Shahar, & Rosenblat-Stein, 2009), even when controlling for total sleep time (Dahl, 1996a). Reductions in slow wave sleep and drastic weekend to weekday shifts in sleep schedules, producing "jet-lag" like symptoms, may contribute to the daytime sleepiness (National Sleep Foundation, 2008)

In sum, despite the increase in several sleep problems in adolescence, others decrease from childhood, such as nighttime awakenings, nightmares and night terrors, bedtime resistance, and nighttime anxiety (Gordon, King, Gullone, Muris & Ollendick 2007; Mindell et al., 2009). Thus, normative development yields a shift rather than an absolute increase in SRPs from childhood to adolescence. Overall, variations in sleep patterns and behaviors between children and adolescents indicate that studies with age-restricted samples may not generalize to all youth. Research examining SRPs must be careful to include measures that are developmentally sensitive and, unless the sample covers the range of ages, not overgeneralize their conclusions across ages.

Assessment of Sleep and SRPs

Researchers have assessed SRPs, and sleep habits in general, with a variety of subjective and objective measures. Polysomnography (PSG) measures sleep objectively, usually in a laboratory, and includes measurement of brain activity (electroencephalography) eye movements (electrooculography), muscle activity (electromyography) and heart rhythm (electrocardiography; Gregory et al., 2011). Although many researchers classify PSG as the gold standard of sleep assessment, it is time-consuming, expensive, and limited (typically measuring two consecutive nights of sleep). Further, assessment using PSG is often laboratory-based, which can lead to irregular sleep due to sleeping in an artificial environment, particularly among youth with anxiety (Forbes et al., 2008). To rectify this latter issue, newer studies take PSG assessments at the youth's home (Gruber, Xi, Frenette, Robert, Vannasinh, & Carrier, 2009). Actigraphy is an alternative to PSG for objective measurement of sleep that is recommended by the American Academy of Sleep Medicine when PSG is unavailable (Morgenthaler, Alessi, & Friedman, 2007). The actigraph is a computerized device worn on the body (usually the wrist) that measures activity level. Tracking of body movement is used to interpret sleep variables, such as total sleep time, sleep latency, and night awakenings. It is advisable that youth wear the actigraph for at least five consecutive nights to achieve a reliable estimate of sleep (Acebo, et al., 1999).

Measurement of sleep through actigraphy tends to agree with PSG, indicating good concurrent validity (Sadeh, 2008). Although actigraphy shows high sensitivity (to detect sleep), it demonstrates lower specificity (to detect wake), particularly after sleep onset (Meltzer, Walsh, Traylor, & Westin, 2012). Moreover, actigraphy cannot assess for

certain sleep problems in youth (e.g. bedtime resistance, parasomnias, sleepiness), so is often used in conjunction with a sleep diary. Compared to PSG, there is evidence that actigraphy underestimates total sleep time and overestimates wake after sleep onset among youth (Spruyt, Gozal, Dayyat, Roman & Molfese, 2011). Technical failures, removal of the actigraph, and other measurement errors, can also misrepresent sleep behaviors.

The gold standard of subjective report of sleep is the 7-day sleep diary, found to be a reliable and valid estimate of sleep behaviors (Buysse, Ancoli-Israel, Edinger, Lichstein, & Morin, 2006; Morin & Epsie, 2003). The measure has good concurrent validity, showing strong associations with objective sleep measures (Goodlin-Jones, Sitnick, Tang, Liu & Anders, 2008; Talbot, McGlinchey, Kaplan, Dahl & Harvey, 2010). Other subjective measures include retrospective questionnaires of SRPs. Among the most widely-used with youth diagnosed with anxiety are the parent-report Children's Sleep Habit Questionnaire (CSHQ; Owens, Spirito, & McGuinn, 2000), and the child-report Sleep Self Report (SSR; Owens, Maxim, Nobile, McGuinn & Msall, 2000; Orgilés, Owens, Espada, Piqueras & Carballo, 2013). The CSHQ can discern clinical from non-diagnosed sleep problems (sensitivity = .80, specificity = .72; Owens et al., 2000a), and both questionnaires have demonstrated adequate reliability (CSHQ internal consistency α = .68-.83; CSHQ retest reliability = .62-.79; SSR internal consistency α = .71 [SSR internal consistency was 0.85 for the Spanish version of the measure]; Owens et al., 2000b; Orgilés et al., 2013; Gregory, Willis, Wiggs, Harvey & the STEPS team, 2008a). The CSHQ has also shown concurrent validity with sleep diaries and actigraphs (total CSHQ with diary sleep onset time, night waking duration, morning wake time, and sleep

duration: r = .21-.65; Goodlin-Jones et al., 2008). Although these scales were designed for youth under age 12, adolescent versions have also been developed (Goldman, Richdale, Clemons, & Malow, 2012; Hansen, Skirbekk, Oerbeck, Richter, & Kristensen, 2011). Alternatively, other measures specifically designed and validated for the assessment of SRPs in adolescents are available, but are less common among investigations of anxious samples (Wolfson et al., 2003).

In sum, parent- and self-report may not be as accurate as PSG or actigraphy in collecting quantitative data about sleep, particularly total sleep time and nighttime awakenings (Acebo, et al., 1999; Goodlin-Jones, et al., 2008; Tikotzky & Sadeh; Wiggs, Montgomery & Stores, 2005). On the other hand, PSG and actigraphy have their own shortcomings. Despite its' limitations, subjective measures do predict to objective measures, particularly in regards to assessment of sleep duration. Additionally, qualitative sleep variables such as daytime sleepiness, bedtime resistance, and parasomnias are better captured by subjective assessment. Overall, a combination of subjective and objective measures is recommended to assess SRPs in youth with and without anxiety, with PSG and the sleep diary being the gold standard objective and subjective measure, respectively.

SRPs and Negative Functional Outcomes

In general, SRPs, particularly sleep deprivation, interfere with the higher cognitive functioning necessary for academic performance, including impulse control/behavioral inhibition (Sadeh et al., 2002), memory (Steenari, Vuontela, Paavonen, Carlson, Fjallberg & Aronen, 2003), and mood regulation (Sadeh, 2007); deficits akin to prefrontal cortex (PFC) abnormalities (Dahl, 1996b). The prefrontal

cortex is particularly sensitive to sleep disruption and can result in limited regulation of executive functions during waking periods, including reduced goal-directed behavior. Similarly, poor sleep subsequently produces daytime sleepiness, which dampens arousal needed for complex cognitive tasks (Dewald et al., 2010). Studies have found attentional deficits in youth with SRPs (Giannotti, et al., 2002; Wolfson et al., 1998), which can resemble symptoms of ADHD (Dahl, 1996a). Sleep problems are also related to physical health outcomes, including risk of physical injury (Boto, et al., 2012), obesity (Hale & Berger, 2011), and compromised immune system functioning (Heiser et al., 2000). Indeed, sleep problems in youth are associated with increased risk-taking behavior, problems in social functioning, and poor school attendance (McLaughlin-Crabtree & Witcher, 2008).

With regard to psychopathology, severe SRPs are strongly associated with emotional, behavioral, and hyperactivity problems (Paavonen et al., 2002), with specific links to youth psychological disorders including ADHD (25% to 50% with comorbid SRPs and higher prevalence compared to healthy controls; (Cortese, Faraone, Konofal, & Lecendreux, 2009) and depression (Chorney et al., 2008). SRPs are also commonly observed in autism spectrum disorder, eating disorders, bipolar disorder, and Tourette's (Dahl & Harvey, 2007). Apropos to the present review, several investigations have found elevated anxiety associated with sleep problems in youth without an anxiety disorder diagnosis, including a number of longitudinal investigations (Gregory, Eley, O'Connor & Plomin, 2004; Gregory & O'Connor, 2002). Research supports that childhood sleep problems prospectively predict elevations in anxiety, including clinical levels of anxiety,

in adolescence and adulthood (OR, .99-1.43; Gregory et al., 2005a; Gregory, Van der Ende, Willis & Verhulst, 2008b).

Anxiety Disorders and SRPs

Anxiety disorders are common among youth with prevalence rates in community samples ranging from 3% to 24% (Cartwright-Hatton, McNicol, & Doubleday, 2006). DSM-5 (American Psychiatric Association, 2013) lists eight anxiety disorders: Generalized Anxiety Disorder (GAD), Social Anxiety Disorder (SoP), Specific Phobia (SP), Panic Disorder (PD), Agoraphobia (AG), Separation Anxiety Disorder (SAD), and Selective Mutism (SM) and Anxiety Not Elsewhere Classified (NEC). Obsessive compulsive disorder (OCD) was formally listed as an anxiety disorder in DSM-IV-R, and has been moved under the heading of Obsessive-Compulsive Disorders in DSM-5. Several of these disorders peak in childhood and adolescence and are unlikely to remit without intervention (Pine, Cohen, Gurley, Brook, & Ma, 1998). Like SRPs, anxiety disorders are associated with impairment in several domains, including academics and interpersonal relationships (Shanahan, Copeland, Costello & Angold, 2008). Early anxiety disorders are also associated with later negative outcomes such as substance abuse and depression (Kendall, Safford, Flannery-Schroeder & Webb, 2004).

Studies have examined the comorbidity of anxiety and SRPs in anxiety disordered youth and have found that 80%-90% of anxious youth have at least one SRP (Chase & Pincus, 2011; Alfano, Ginsburg, & Kingery, 2007). The most commonly reported SRPs are feeling fatigued or tired, insomnia, nightmares, and refusal to sleep alone. Additionally, anxiety is associated with specific SRPs to a moderate degree (*r*'s ranged from .26-.35; Chase et al., 2011) and number of SRPs to a moderate to large degree (*r*'s

ranged from .34-.57; Alfano et al., 2007). Other studies using parent and child self-report have also found small to large associations between anxiety and SRPs among youth diagnosed with anxiety (Alfano & Gamble, 2009a; Alfano, Beidel, Turner & Lewin, 2006; Morrison, McGee & Stanton, 1992; Storch, Murphy, Lack, Geffken, Jacon & Goodman, 2008).

Studies have also compared SRPs between clinically anxious and healthy youth using subjective measures, though these studies have generally had small samples so findings should be interpreted with caution. For example, two studies found greater numbers of SRPs in youth with anxiety disorders compared to healthy youth, with some evidence of greater SRPs in youth with anxiety versus ADHD, and additive effects when anxiety is a comorbid disorder (Hansen et al., 2011; Hansen, Skirbekk, Oerbeck, Wentzel-Larsen & Kristensen, 2013; Ivaenko, McLaughlin-Crabtree, O'Brien & Gozal, 2006). In one study, twice as many anxious children reported taking over 30 minutes to fall asleep (the clinical cutoff; Buysse et al., 2006) compared to healthy controls (Hudson et al., 2009), in addition to evidencing later bedtimes and reduced total sleep time. In another study, anxious adolescents reported greater tiredness, nightmares, and reduced sleep time relative to healthy controls, but more notably, evidenced minimal differences in sleep problems relative to sleep disordered youth (Alfano et al., 2006). This particular finding is striking in that sleep behaviors of anxious youth more closely resemble youth diagnosed with sleep disorders than controls (Alfano et al., 2006). Thus, sleep problems of anxious youth appear to be at a level that causes substantial impairment.

Other studies investigated this topic using objective measures. These studies have tended to find greater SRPs in youth diagnosed with anxiety relative to healthy controls

(Forbes et al., 2008; Alfano et al., 2012). One study using PSG among diagnosed anxious, depressed, and healthy youth (ages 7 to 17; Forbes et al., 2008) found that the anxious group evidenced more nighttime awakenings, less slow-wave sleep, and longer sleep latency compared to the depressed and control groups. Alfano and colleagues (2012) used PSG to compare SRPs in youth (ages 7-11) with GAD (without comorbid depression) to healthy controls. Youth with GAD experienced significantly longer sleep onset latency (54 vs. 29 minutes), reduced latency to REM sleep, longer time to turn on the lights in the morning, and a trend for reduced sleep efficiency. No differences emerged on variables of total sleep time or nighttime awakenings. A similar pilot study using actigraphy with OCD youth did find greater nighttime awakenings and reduced total sleep time, as well as great sleep latency relative to healthy youth (Alfano & Kim, 2011). However, it is important to note that other research has found few differences in PSG measured sleep variables between anxious, depressed, and healthy youth despite documenting higher peri-sleep onset cortisol in anxious youth (Forbes et al., 2006).

Thus, studies using both subjective and objective measures of sleep generally find a relation between anxiety and SRPs, as well as a greater number of SRPs in anxious youth versus healthy controls. However, results differ on the specific sleep-related variables affected by anxiety. Although these studies extend the literature by using objective assessment strategies and controlling for depression, replication with larger samples is needed.

Limitations of Extant Research and Future Directions

Although epidemiological studies consistently support the association between SRPs and anxiety, they share a limitation: many do not use optimal measures to

specifically assess SRPs[3], making cross-study comparisons difficult. Instead they select items from other measures (e.g., the CBCL, MASC [Multidimensional Anxiety Scale for Children; March, 1997], PARS [Pediatric Anxiety Rating Scale; PARS; Research Units on Pediatric Anxiety Study Group, 2002]), and others, to create a "Frankenscale" of SRPs. Additionally, vaguely worded questions, limited scope of SRPs reflected in said measures, and the rescaling of Likert scales hampers the ability to draw conclusions. Even more problematic is the inconsistency across studies in items selected from these non-sleep measures to construct their scales. Perhaps the greatest problem of using sleep measures with unknown psychometric properties occurs when examining correlations with anxiety. Correlating sleep items from anxiety questionnaires with the same anxiety questionnaires inflates the SRP-anxiety association due to shared method variance. Thus, the relationship between anxiety and SRPs may be exaggerated in studies that use shared items for sleep and anxiety. Nonetheless, there is some preliminary support for the CBCL to predict to some variables on validated objective and subjective sleep assessments (Gregory et al., 2011).

Another common shortcoming of the existing literature on sleep and anxiety is that studies often assess SRPs by parent-report only. Parents may not be the most accurate reporters of certain sleep problems, particularly when the sample consists of adolescents. Moreover, research has found that subjective measures of parent- and child-report of SRPs are not highly correlated (Gregory, Riksdijk, Dahl, McGuffin, & Eley, 2006;Owens et al., 2000c; Owens et al., 2009a; Wiggs, Montgomery & Stores, 2005). Unless the youth seeks comfort from the parent during a sleep disturbed episode,

[3] With the exception of the few objective measurement studies above, and a study by Alfano and colleagues that used the CSHQ, finding 85% of youth diagnosed with anxiety having SRPs in the clinical range.

discloses to the parent about sleep problems, or sleeps in the same room as the parent, the parent may be largely ignorant of the youth's sleep behaviors. Parents can provide valuable information (e.g. bedtime resistance, sleep onset), but their report should contribute to, rather than account for all information on youth SRPs.

How do studies address the effects of medications on SRPs? Youth diagnosed with anxiety and SRPs are often prescribed medication (e.g., approximately 25% of youth diagnosed with anxiety (Chase et al., 2011; Owens, Rosen, Mindell & Kirchner, 2010), but there are inconsistencies in the way studies account for psychotropic medications. Whereas some studies exclude participants on medication, others include youth taking a range of medications but do not control for them in the analyses, or only include youth on stable doses of medication, yet other studies make no mention of medication. One study described and analyzed medications of anxious youth in an outpatient clinic, finding no differences in the number of SRPs between those with and without prescriptions (Chase et al., 2011), but these findings require replication given their theoretical impact on sleep-related variables (see below).

Although a review of the efficacy of sleep medications for youth is beyond the present, prescribed and efficacious drugs for anxiety disorders, such as selective serotonin reuptake inhibitors (SSRIs) and benzodiazepines, can have sedating effects and improve SRPs (Alfano et al., 2007). However, some SSRIs (e.g. Fluoxetine) have been linked to nighttime motor restlessness, increase in sleep onset latency, and decrease in sleep efficiency (Owens, 2009b). Benzodiazepines can promote certain SRPs, including daytime sleepiness. It is also not uncommon for youth with anxiety disorders to be on medication for primary sleep problems, such as alpha agonists, short-acting hypnotics,

antihistamines, tricyclics, and melatonin (Owens et al., 2010). To further complicate matters, anxiety disorders' high comorbidity with other psychological problems mean that some youth may be taking other psychotropic medications that may impact sleep. For example, stimulant drugs for ADHD can affect sleep, including increased sleep latency, delayed sleep phase syndrome, reduced sleep efficiency, and reduced total sleep time (Stein, Weiss & Hlavaty, 2012).[4] Thus, analytic strategies and caution are needed when interpreting the relationship between anxiety and SRPs when participants are taking medications. Results from studies may overstate or understate the relationship depending on the types of medications taken by the study's participants. Future studies are encouraged to include large enough sample sizes to control for the effects of medication.

In sum, studies of SRPs and anxiety suffer from inconsistent methodology (e.g., presence of medications, comorbidity, types of SRPs measured; Hansen et al., 2011) as well as use of non-validated questionnaires and reliance on mono-method reporting. Despite the variable methodology and some inconsistencies, anxiety is generally associated with SRPs in clinically diagnosed anxious youth. Anxiety predicts an array of specific SRPs (e.g. insomnia-related SRPs, parasomnias, and bedtime resistance), and clinically anxious youth generally evidence a greater number of SRPs compared to healthy controls and youth with other psychological disorders. More research is needed that measures SRPs with objective measurement, or uses validated subjective measures of SRPs instead of psychological questionnaire-based items. Additionally, it is recommended that future research use *both* parent and child report and control for other factors (e.g., comorbidity).

[4] The type and severity of SRP varies by several factors, including: type of stimulant drug, length of drug trial, when drug was administered, dose, and flexibility of dosing (Stein et al., 2012).

Predictor Variables of SRPs and Anxiety Disorders

Several studies have examined predictors of SRPs and anxiety, including gender, age, principal diagnoses, and ethnicity/SES. With regard to gender, large differences are not to be expected given minimal gender variance in normative sleep patterns, and studies not finding an interaction between gender and SRPs in predicting elevated anxiety in healthy youth (Alfano, Zakem, Costa, Taylor & Weems, 2009b; Gregory et al., 2002; Sadeh et al., 2009). Studies of diagnosed anxious youth been mixed with a few studies finding more SRPs in anxious girls (Alfano, Pina, Zerr & Villalta, 2010; Storch et al., 2008) and several others finding no gender differences (Alfano et al., 2009b; Alfano et al., 2007; Chase et al., 2011; Hansen et al., 2011).

Studies examining age differences in SRPs for anxious youth have yielded inconsistencies as well. As previously mentioned, sleep behaviors fluctuate throughout development, and there is a shift in the types of SRPs from childhood to adolescence. Somewhat consistent with normative trends, research on anxious youth has shown that age predicts certain SRPs, such as greater bedtime resistance (Alfano et al., 2010; Alfano et al., 2007) and nightmares (Alfano et al., 2010; Alfano et al., 2007; Alfano et al., 2006; Storch et al., 2008), as well as nighttime anxiety among younger school-aged children (7 to 12; Alfano et al., 2010). Although Storch and colleagues (2008) found a relative greater frequency of SRPs in adolescents compared to children with OCD, another study found that anxiety did not predict SRPs after controlling for depressive symptoms in adolescents, but did in children (Alfano, Zakem, Costa, Taylor & Weems, 2009). Still other studies found no difference by age for the total number of SRPs, with little variation by subtype of SRP (Alfano et al., 2007; Alfano et al., 2006; Chase et al., 2011). Many

studies include a restricted range of ages or are underpowered, limiting the ability to test for age differences.

Principal diagnoses have been a topic of interest as predictors for anxiety and SRPs. Studies evaluating samples of homogeneous diagnosed anxious youth have found significant associations for SRPs and GAD (Alfano et al., 2012; Kendall & Pimentel, 2003; Masi, et al., 2004; Pina, Silverman, Alfano & Saavedra, 2002) and OCD (Piacentini, Bergman, Keller & McCracken, 2003; Rapoport, et al., 1981; Storch et al., 2008). Unfortunately many of these studies broadly classify sleep problems, and are inconsistent in the SRPs assessed. Other studies with heterogeneous anxious youth have made comparisons between diagnoses. For example, several studies have found that anxious youth with GAD versus without GAD (but other comorbid anxiety) have more SRPs (Alfano et al., 2010; Alfano et al., 2009b; Alfano et al., 2007; Alfano et al., 2006), although one study did not find this (Chase et al., 2011). Three studies (Chase et al., 2011; Alfano et al., 2007) found higher SRPs in anxious youth with SAD versus without SAD, with rates comparable to OCD (Alfano et al., 2010). The significant associations between GAD and SRPs, as well as between SAD and SRPs may be due to the overlap in DSM-5 diagnostic criteria for these anxiety disorders, as both make mention of sleep difficulties. Research on SoP has generally shown high rates of SRPs, but statistically lower frequencies compared to youth with OCD, GAD, and SAD (Alfano et al., 2010; Alfano et al., 2007), though not in all cases (Chase et al., 2011). Research has yet to systematically examine SRPs in youth with Panic Disorder (PD) or Agoraphobia (AG); however, evidence from the adult literature suggests that PD is linked to sleep disruption (Hoge et al., 2011). Some research supports that additional anxiety diagnoses do not

predict greater intensity or frequency of SRPs (Alfano et al., 2009a), yet comorbid ADHD or depression may have an additive effect on SRPs (Alfano, et al., 2009b; Hansen et al., 2011). Despite some discrepancies, all anxiety disorders have been associated with SRPs, although the strength of the relation and type of SRP differs across studies (Chorney et al., 2008).

Few studies have investigated differences by ethnicity or SES. In healthy samples, greater frequency of SRPs have been found in low SES (Meltzer et al., 2010), and ethnic minority youth, particularly African American children (Adam, Snell & Pendry, 2007). Differences may be attributable to greater life stress, cultural differences in sleep practice, and proximity to schools. The one study examining ethnic differences in diagnosed anxious youth found more SRPs across several domains (e.g. total sleep duration, total score on CSHQ) in Latino compared to non-Latino children (Alfano et al., 2010). Substantive conclusions cannot be drawn about SES due to the dearth of studies in this area, making this an important area for future research.

To summarize, the literature on demographic predictors is mixed. Studies on gender mostly indicate little difference in SRPs between demographic subgroups. Although there is some evidence of greater parasomnias among younger children, age does not appear to be a major predictor of the total number of SRPs or insomnia-related SRPs. The addition of more studies with wider-aged samples will allow researchers to test differences by age. With regard to principal diagnosis, although SRPs are related to all anxiety disorders, there is some evidence for a stronger relationship to GAD and SAD. The high comorbidity among the anxiety disorders makes disorder-specific predictions difficult; future studies will require large samples for proper evaluation. Finally, there are

too few studies that have measured SES to draw any firm conclusions. Future research should include more diverse samples, rather than relying on predominately Caucasian upper-to-middle class samples. Additionally, future research would benefit from a better understanding of whether overlap in diagnostic criteria may explain the relationship between primary diagnoses of GAD or SAD and SRPs.

Underlying Mechanisms

An understanding of the mechanisms that underlie the relationship between anxiety and SRP is important not only for etiology, but also for understanding treatment implications. Though these mechanisms are not fully understood, we review several that have received empirical support. Additionally, we propose several mechanisms that have not been empirically explored and may serve as potentially fruitful avenues for future research.

Understanding SRPs and Anxiety from a Neurological Perspective

Conceptual models of sleep and anxiety have been hotly debated. Uhde and colleagues (2009) introduced two theoretical models as a way to conceptualize the relationship between anxiety and SRPs, specifically insomnia. One model proposes that anxiety and insomnia represent different dimensions of a single dynamic neurobiologic diathesis where dysregulation in shared neural regions produces problems in anxiety and sleep; symptoms emerge depending on environmental and genetically-based factors (Uhde et al., 2009). This theory is supported by literature supporting overlapping dysregulation in the PFC and limbic system (discussed later), as well as research showing overlapping genetic factors and neurotransmitter systems (e.g. the serotonin

transporter gene 5HTTLPR is implicated in both anxiety and sleep) (Gregory et al., 2004; Barclay et al., 2011).

Supporting Uhde's (2009) single dynamic neurobiologic diathesis hypothesis, a seminal paper by Dahl (1996b) outlined shared neural pathways for the regulation of anxiety and sleep. As noted, the PFC is both important in the regulation of sleep as well as the regulation of affective, arousal, and attentional systems (i.e. executive functioning). Along these lines, the PFC regulates the emotional areas of the brain (e.g. the limbic system), modulating anxiety. Sleep disruption has been shown to weaken the connection between these areas and the regulatory abilities of the PFC, which in turn, leads to further dysregulation (Gruber, 2014; Payne & Kensinger, 2011). Research in neuroimaging has shown underarousal of the PFC in individuals with anxiety and SRPs (Altena, Van der Werf, Sanz-Argita, Voom, Rombouts, & Kuijer, 2008). Further, research shows that sleep deprivation weakens PFC regulation over these systems, which can be reversed after sufficient sleep (Horne, 1993). For example, Yoo and colleagues (2007) found a 60% increase in amygdala activity and decreased medial-PFC activity in response to an emotional stimulus when subjects were sleep deprived versus rested. Importantly, the medial-PFC appears to be particularly central to the regulation of the amygdala, one region of the brain specifically associated with the fear response (Phelps, Delgado, Nearing & LeDoux, 2004). Likewise, another study found that sleep disruption was associated with increased amygdala and anterior insula activity (excessive anticipatory insula activity is a characteristic of most anxiety disorders (Etkin & Wagner, 2007), particularly among individuals with high trait anxiety (Goldstein, Greer, Saletin, Harvey, Nitschke & Walker, 2013). Such research support a neuropathological model where sleep

disruption is both a symptom of anxiety and a maintaining factor (Goldstein et al., 2013). Additionally, emotion dysregulation, linked with dysfunctional neural pathways between the PFC and limbic system, has shown to mediate SRPs and anxiety in anxiety disordered individuals (Markarian, Pickett, Deveson & Kanona, 2013; Tsypes, Aldao & Mennin, 2013). Problems in emotion regulation contribute to SRPs, which consequently, lead to more emotion dysregulation and anxiety (Schmidt, Harvey & Van der Linden, 2011). Other studies have also found that modest amounts of restricted sleep predict greater anxiety and overall mood dysregulation (Alfano et al., 2009b; Sagaspe et al., 2006).

Further supporting this hypothesis, research findings indicate that sleep deprivation in youth results in less positive affect and a lower positive to negative affect ratio, as well as increased self-reported anxiety (Dagys, McGlinchey, Talbot, Kaplan, Dahl & Harvey, 2012). Interestingly, there are few studies showing increased negative affectivity following sleep disruption (Zohar, Tzischinsky Epstein & Lavie, 2005). An additional study that examined the impact of sleep deprivation on mood in adolescents extended previous research by investigating domains of anxiety (e.g. number of worries, rating of most threatening worry, length of catastrophizing sequence, anxiety ratings, and likelihood of ratings of potential catastrophes (Talbot et al., 2010). Like several past studies, the researchers found that adolescents with sleep disruption experienced less positive affect, a decreased positive to negative affect ratio, more self-reported anxiety following catastrophizing, and reported a higher likelihood that the catastrophic situation would occur. Perhaps most striking is that the experimental manipulation of sleep deprivation had a profound impact on adolescents without an anxiety diagnosis. The consequence of sleep deprivation on worries and catastrophic thinking may be even more

severe among youth diagnosed with anxiety and warrants investigation. Weakened regulation of the PFC may prevent an individual from using higher ordered reasoning skills and accordingly, cause the individual to engage in distorted processing of events. The weakened PFC is related to tiredness and decreased motivation for goal directed tasks which may (1) trigger worry (e.g. worried about the need to accomplish tasks that are not being initiated), and (2) inability to disengage from worry by engaging in other tasks. Thus, poor sleep may result in compromised affect regulation, mood deterioration, cognitive processing, and consequent increased anxiety.

The second theoretical model notes that anxiety and sleep are distinct and due to different neurological processes (Uhde et al., 2009). The comorbidity between the two conditions may be accounted for by either (1) both disorders causing similar symptoms, or (2) both are influenced by an unknown third causative factor (Uhde et al., 2009). Third causative factors may be another type of psychopathology or executive functioning deficit (e.g. depression or attentional problems), or environmental trigger (e.g. poor family functioning, stressful life events). Several studies have accounted for possible third variable psychological factors and still find support for a unique anxiety and SRP relationship (Gregory, Eley, O'Connor, Rijsdijk & Plomin, 2005b; Cartwright & Wood, 1991; Coulombe, Reid, Boyle & Racine, 2010). However, it is possible that these studies did not include the third variable that affects both sleep and anxiety. Additionally, without large-scale longitudinal studies it is difficult to assert whether third factors represent mediators or moderators, linking anxiety and sleep in support of the first model, or whether they predict each condition independently. Overall, contentions of this model are largely speculative and lack considerable research support.

In sum, although specific mechanisms are not fully understood at present, research supports shared biological, cognitive, and environmental risk factors and the notion that the relationship between anxiety and sleep is birdirectional (Dahl et al., 2007; Alfano et al., 2006; Dahl & Lewin, 2002). Whereas some studies of youth found that early SRPs predict subsequent elevated anxiety (Gregory et al., 2005b; Gregory et al., 2004; Gregory et al., 2002), others have found that anxiety disorders commonly precede SRPs, such as insomnia (Johnson et al., 2006), yet directionality of associations remain unclear (Gruber, 2014). A recent review (see, Leahy & Gradisar, 2013) concluded that research supports the role of SRPs as a risk factor for the development of anxiety, but the role of anxiety as a risk factor for the development of SRPs is less conclusive.

To this point the review has presented conceptual and neurological explanations that underlie anxiety and insomnia-type SRPs (e.g. total sleep time, sleep latency, night awakenings). However, SRP is a heterogeneous term and encompasses other problems such as parasomnias and bedtime resistance. How may one conceptually address this group of SRPs? Unfortunately, conceptual models regarding these SRPs are lacking. However, the consequences of many of these SRPs (parasomnias and bedtime resistance) are insomnia-like symptoms. For example, nightmares may reduce total sleep time because they cause nighttime awakenings or increase sleep latency. Night terrors may disrupt essential slow-wave sleep thereby influencing the quality of sleep, and producing daytime fatigue and sleepiness. Bedtime resistance may extend bedtimes, ultimately reducing total sleep time. Additionally, arguing with parents, which often accompanies bedtime resistance, could theoretically increase the child's arousal and increase sleep latency (more discussed in the next section). Thus, parasomnias and bedtime resistance

may predict anxiety because they produce secondary insomnia-like symptoms. In other words, these SRPs may be incorporated into the insomnia-anxiety models.

Pre-Sleep Arousal

Children with anxiety disorders tend to exhibit a bias towards threat, including an increased tendency to interpret ambiguous situations as threatening (Barrett, Rapee, Dadds, & Ryan, 1996; Blossom et al., 2013). Moreover, children with anxiety disorders have been found to have significantly greater levels of peri-sleep-onset cortisol than healthy controls (Forbes et al., 2006). Further, increased arousal at bedtime can interfere with sleep initiation and maintenance. Arousal and sleep represent opponent processes, as sleep requires a lack of responsiveness to the external environment. Research following individuals who experienced a stressful event, such as a trauma, evidence increased nighttime arousal and consequently, disrupted sleep (Harvey, Jones & Schmidt, 2003). Even positive arousal can interfere with sleep, such as the approach of a holiday (Cartwright et al., 1991). Indeed, cortisol has been associated with arousal, and high levels of arousal around bedtime can interfere with sleep initiation (Hatzinger et al., 2012).

Pre-sleep arousal can be classified as either cognitive (worries) or somatic (physiological arousal), with higher cortisol levels reflecting higher somatic arousal. In particular, cortisol is associated with vigilance (a hallmark of anxiety disorders), and may interfere with feelings of safety that are necessary for sleep (Forbes et al., 2008). In other words, if an anxious youth perceives present or future threat, he is likely to experience cognitive and/or somatic arousal that is incompatible with sleep initiation and/or maintenance. Forbes and colleagues (2006) examined differences in cortisol and

objective sleep patterns (EEG) among diagnosed anxious, depressed, and healthy youth. The groups with psychological disorders had higher peri-sleep onset cortisol, yet *children* with anxiety disorders experienced the highest levels of cortisol during two hours before sleep onset. Of note, depressed *adolescents* had higher cortisol levels compared to anxious or healthy adolescents. A subsequent study using PSG did not find higher levels of cortisol in children with GAD compared to controls, but found youth with GAD (compared to controls) showed significantly increased sleep onset latency and reduced latency to REM sleep (Alfano et al., 2012). Crucial deep sleep may be particularly sensitive to increased nighttime vigilance (Tikotzky & Sadeh, 2010). Supporting this contention, a few studies have found that pre-sleep cognitive arousal is a more robust predictor of SRPs in anxious and healthy youth, compared to somatic arousal (Alfano et al., 2010; Gregory et al., 2008a). Unfortunately, these studies are limited by cross-sectional designs and use of retrospective subjective measures.

Cognitive arousal may manifest as nighttime worry and other negative thoughts within the context of anxiety disorders. Pre-sleep worry, negative thoughts, and planning have shown to differentiate insomniacs from normal sleepers (Gellis & Lichstein, 2009). Worrying and negative thoughts can arouse the limbic system, which may already be poorly regulated due to the impact of chronic sleep deprivation on the PFC, creating a vicious cycle. Likewise, SRPs are associated with negative attributional style, anxiety sensitivity, cognitive errors and control beliefs (Alfano, 2009b; Gregory & Eley, 2005c). Anxious youth may not have the coping skills to regulate their cognitive-based arousal, which may account for SRPs. If children commonly experiences nighttime worries, then they may form negative associations regarding their bedroom and bedtime and develop

interoceptive cues, which may subsequently trigger arousal at nighttime. Additionally, certain negative cognitions are likely to present in the evening. In GAD, youth may worry about a burglar breaking into the house, or events relating to school the next morning (e.g. being on time, grades). Likewise, socially anxious youth may ruminate about the next day's social events. Common specific phobias are also present in the evening, such as fear of imaginary creatures and the dark. Youth with separation anxiety may be distressed because of bedtime separation from parents. As mentioned previously, panic attacks commonly occur in the evening and serve as a source of anxiety for panic disordered youth. Finally, some youth with OCD may have specific nighttime routines or compulsive rituals. Unlike other times of the day, anxious youth may not have external stimuli to distract them from their anxiety or the opportunity to seek reassurance (e.g. television or parents). However, if anxious youth do seek these external stimuli to self-soothe, then this is likely to interfere with sleep.

In sum, arousal around bedtime, particularly the cognitive subtype, can interfere with sleep initiation and maintenance. Further, arousal at bedtime is characteristic of anxiety disorders. Neurophysiological and cognitive theories conceptualize how arousal influences SRPs in youth with anxiety disorders. These theories are supported by a handful of studies that have found higher levels of pre-sleep cortisol and self-reported arousal before bedtime in youth with diagnosed anxiety and SRPs. Furthermore, youth diagnosed with specific anxiety disorders may experience arousal due to disordered-specific triggers. More experimental and longitudinal research is needed that examines specific cognitive factors that promote arousal (e.g. control beliefs, negative attributional style) and whether arousal differs by disorder (e.g. more somatic arousal for PD, more

cognitive arousal for GAD). Finally, studies investigating pre-sleep cortisol levels in anxious youth require replication with larger samples.

Sleep Hygiene

Youth who rely on external means to cope with or avoid nightly anxiety (arousal) may have poor sleep hygiene, a variable that may underlie the anxiety and SRPs relationship. Sleep hygiene refers to behaviors and environmental factors that influence adequate sleep initiation, maintenance, and quality. Examples of good sleep hygiene include regular bed and wake times, predictable pre-sleep activities, lack of stimulating or emotionally-laden activities before bedtime (e.g. television, exercise, family arguments), no consumption of high-energy substances in the evening (e.g. tobacco, sugar, caffeine), comfortable sleep environment (low noise/light and comfortable room temperature), avoidance of napping during the day, and the use of the bed for only sleep-related activities. Thus, sleep hygiene can be influenced by both the youth and their family members.

Research has documented a negative relationship between sleep hygiene and SRPs (LeBourgeois, Giannotti, Cortesi & Wolfson, 2005; Mindell et al., 2009). For example, one study found that sleep hygiene was associated with less sleep time and longer sleep onset latency, and it accounted for 15% of the variance in daytime sleepiness (Billows, Gradisar, Dohnt, Johnson, McCappin & Hudson, 2009). Poor sleep hygiene, in the form of digital media use at night (e.g. videogames, computer, or television) predicts later bedtime, reduced total sleep time, greater daytime sleepiness, and even nightmares (Eggermont & Van den Bulck, 2006; Higuchi, Motohashi, Liu, & Maeda, 2005). Theories of media use resulting in SRPs involve exposure to stimulating light, the content

of the media (e.g. scary movies increasing arousal), and the time media use takes away from sleeping (Adam et al., 2007). With regard to those with an anxiety disorder, youth may rely on media to avoid going to bed or as self-distraction from worries. Research shows that anxious individuals watch more television and rely on it as a way to cope with stress (de Wit, van Straten, Lamers, Cuijpers & Penninx, 2011).

Studies have not yet examined sleep hygiene among youth diagnosed with anxiety. Aside from media use, other aspects of sleep hygiene may be poor in this population and account for SRPs. For example, poor sleep hygiene and diagnosed anxiety is associated with family disorganization (Gregory et al., 2005b), with sleep hygiene mediating the relation between family disorganization and sleep problems (Billows et al., 2009). Family disorganization may contribute to poor parental monitoring of the child's nighttime routines, inconsistencies in bedtime, and suboptimal light and noise levels. Likewise, anxious youth may use their bedrooms for non-sleep activities in line with poor sleep hygiene (i.e. poor "stimulus control"). A youth with agoraphobia, socially anxiety, or separation anxiety may spend more time at home (specifically in their bedrooms) because of avoidance dealing with disorder-specific stressors in the outside-home environment. It is possible that anxious youth, who may experience emotional dysregulation broadly, are engaging in pre-bedtime behaviors or activities that are highly emotionally-laden and incongruent with good sleep hygiene.

Overall, research confirms an association between sleep hygiene and SRPs. Sleep hygiene may be particularly poor in youth diagnosed with anxiety, but this has not yet been investigated. Research is needed to determine if sleep hygiene is of particular importance for anxious youth, and if it predicts to the anxiety/SRP relationship. If so,

research can further explore whether unique aspects of sleep hygiene demonstrate differential relationships with anxiety.

Parental Accommodation

Another theoretical (environmental) mechanism that may be responsible for SRPs in anxious youth is parental accommodation. Parental accommodation refers to parents (family members) permitting avoidance behavior, modifying family routines because of youth anxiety, or facilitating other anxious thoughts or behaviors, such as compulsions and reassurance seeking (Lebowitz, Panza, Su & Bloch, 2012). Research of families of OCD youth indicates that parental accommodation may be an important maintaining factor of anxiety (Merlo, Lehmkuhl, Geffken & Storch, 2009). Research on family accommodation is burgeoning with a focus moving away from OCD-specific youth to anxiety disorders more broadly. A common example of sleep-related accommodation is allowing the child to co-sleep with the parent, a common behavior in families with anxious youth (Storch et al., 2008). Unfortunately, co-sleeping can reinforce nighttime worries, facilitate distorted thinking (Tikotzky et al., 2010), and is associated with greater overall sleep disruption, bedtime resistance, night awakenings, sleep anxiety, and reduced sleep duration (Jenni et al., 2005). However, co-sleeping may not be as detrimental to sleep in youth from non-Caucasian families (Mindell, Sadeh, Kohyama & How, 2010).

Accommodating parents may "give in" to their child's anxiety at the expense of rule-setting, which does not make for favorable sleep, as consistency in bedtime is important and rule-setting is part of interventions for bedtime refusal (Tikotzky et al., 2010; Cousins et al., 2011). Specifically, setting rules about sleep has been associated with greater time in bed and improved quality of sleep (Adam et al., 2007). Research

shows that a significant difference between diagnosed anxious and non-anxious youth is bedtime (anxious youth go to bed 30 minutes later; Hudson et al., 2009). Accommodation permits youth to engage in time-consuming behavior without restriction, which may extend into late evening hours (e.g. OCD rituals or GAD-related tasks that are aimed to be completed "perfectly," such as homework). Accommodation supports avoidance behaviors that are incompatible with good sleep habits and hygiene, such as use of media in late hours and reassurance seeking. Thus, accommodation reinforces anxiety at bedtime and also prevents the development of self-regulatory skills (Dahl, 1996b). Although accommodation's role for SRPs in anxious youth has not been systematically evaluated, researchers suggest that it may be important to some sleep behaviors (Storch et al., 2008). Like sleep hygiene, accommodation within the context of anxiety and SRPs is an uncharted area of research. Longitudinal studies are needed to assess family accommodation and SRPs in youth diagnosed with anxiety over time.

To recap, there appears to be shared neurological and cognitive features that account for dysregulation of both sleep and affect, including anxiety. The relationship between SRPs and anxiety is bidirectional, with sleep disruptions exacerbating mood, and anxiety disrupting sleep. Arousal, particularly cognitive arousal, appears to be the central to understanding the relationship between SRPs and anxiety. However, unexplored avenues of research, such as sleep hygiene and parental accommodation may help explain why greater SRPs are observed in youth diagnosed with anxiety. These factors may be sensitive to treatment for youth anxiety and accordingly, result in improved sleep. We have included a proposed model to conceptualize the SRP and anxiety relationship in youth (Figure 1).

CBT for Anxiety and SRPs and Secondary Effects

CBT for youth anxiety has been described as "well established," and it is the first-line psychological treatment for youth anxiety (Hollon & Beck, 2013). Although there is considerable evidence supporting the efficacy of CBT for anxiety disorders, studies on CBT for SRPs among school-aged youth are sparse and there is a great need for more RCTs. Nonetheless, preliminary evidence suggests that CBT has a beneficial effect on SRPs (Paine & Gradisar, 2011; Pincus, Weiner & Friedman, 2012; Taylor & Roane, 2010). Additionally, despite the dearth of evidence for the efficacy of CBT for SRPs, as well as the absence of practice parameters for childhood insomnia from the American Association of Sleep Medicine, researchers and practitioners advocate for CBT interventions to treat SRPs (Meltzer & Mindell, 2006; Rabian & Bottjer, 2008).

CBT for anxiety and CBT for SRPs target shared underlying factors believed to maintain psychopathology. It is believed that avoidance and safety behaviors (e.g. sleeping with parent), as well as distorted cognitive processing are shared among anxiety- and sleep-disordered individuals (Harvey, Watkins, Mansell & Shafran, 2004). Accordingly, SRP and anxiety interventions have common treatment components, including: psychoeducation, parent management training, contingency management, relaxation, and cognitive restructuring. Of course these techniques are individualized depending on the presenting problem. Additionally, each treatment has unique features as well, such as exposures in CBT for anxiety, and bedtime fading and sleep hygiene/stimulus control in CBT for SRPs. In both types of CBT, sleep interventions for very young children are more parental-based and take a behavioral approach. Given the overlap in CBT strategies as well as shared cognitive and behavioral features of SRPs and

anxiety, it is reasonable to consider that an intervention for one presenting problem may have secondary benefits for the other.

CBT for SRPs: Secondary Effects on Anxiety

Research has investigated how CBT interventions for sleep problems impact anxiety in adults. A meta-analysis examined 216 trials of CBT for insomnia (CBT-I) and found that 50 trials (23%) reported quantitative data on comorbid anxiety (Belleville, Cousineau, Levrier, & St-Pierre-Delorme, 2011). The overall effect size of CBT-I on anxiety outcomes was moderate (d = .41) despite residual anxiety symptoms, with no significant differences by type of anxiety, comorbidity, or study design. Unfortunately, most studies examined anxiety with a variety of self-report questionnaires and the majority of studies evaluated did not include participants diagnosed with an anxiety disorder, limiting generalizability to anxiety-disordered youth. Thus, the effects on CBT-I on anxiety is inconclusive.

Studies for CBT-I on anxiety in youth are rare, likely due to the general absence of RCTs for CBT for SRPs. Paine and Gradisar (2011) compared 6 sessions of CBT-I to a waitlist in school-aged youth. The treatment, included elements of CBT for youth anxiety (e.g. cognitive restructuring, exposure targeting separation from parents, relaxation) and produced improvement in self-reported anxiety, specific separation- and OCD-related anxiety (d = .85-1.01), but not fear of the dark. The authors speculated that acquired skills targeting sleep generalized to the domain of anxiety through "the mechanism of reduced reliance on avoidant coping strategies." However, CBT-I included specific anxiety-reduction components not found in some CBT-I protocols, potentially

overestimating its effects on anxiety symptoms. On the other hand, the study supports the utility of transdiagnostic treatments that can target both anxiety and sleep problems.

CBT for Anxiety: Secondary Effects on SRPs

A meta-analysis addressed the question of improvement in SRPs after CBT for anxiety among adults (Belleville, Cousineau, Levrier, St. Piette-Delorme & Marchand, 2010). Of the 1205 trials reviewed, only 19 (1.6%) included quantitative data about sleep, with several using non-validated sleep measures. These 19 studies yielded an overall moderate effect size for CBT on SRPs (d = .53), with some evidence of residual SRPs. Effects did not differ by type of sleep variable examined or by principal anxiety disorder treated. A few additional studies of adults have emerged since the meta-analysis that have also documented improved sleep following CBT for anxiety (e.g. PTSD, GAD; Belleville, Guay & Marchand, 2011; Bush et al., 2012). Results of adult studies should be interpreted with caution as many include specific techniques that target sleep problems, possibly overstating secondary effects. Taken together, preliminary research suggests that CBT for anxiety appears to ameliorate some global sleep difficulties in adults, but residual SRPs remain and too few studies exist to make definitive conclusions.

Although informative, research with adults does not necessarily translate to school-aged youth. The majority of the extant studies on CBT for youth anxiety on SRPs have centered on nighttime fears. These studies find reductions in both SRPs and anxiety after CBT for nighttime fears (an anxiety-, rather than sleep-focused treatment). For example, Graziano and Mooney (1980) randomized 33 school-aged youth to CBT for anxiety or a waitlist. The treatment condition included relaxation, guided imagery, coping self-talk, and contingency management. Although this early study did not implement

validated measures of sleep and anxiety, there was a significant improvement in parent-reported nighttime fear, time in bed, sleep latency, and bedtime resistance (Graziano & Mooney, 1980). Gains for those in the CBT group maintained at a 2.5 year follow-up (Graziano & Mooney, 1982). Most research on this topic has been in the form of case studies or multiple-baseline studies (Friedman & Ollendick, 1989; McMenamy & Katz, 1989; Ollendick, Hagopian & Huntzinger, 1991), and has not implemented a uniform CBT protocol across studies. A review by Gordon and colleagues (2007) surveyed 29 studies of CBT and non-CBT treatments for nighttime fears. The authors found that 11 studies were RCTs, and of these only 4 may be (loosely) classified as a CBT (used inconsistent components of CBT). Additionally, several studies had substantial methodological limitations, such as small sample size, use of mono-method and mono-reporter data, and absence of treatment adherence checks. Of note, since the publication of Gordon and colleagues (2007) review, an additional RCT was published, finding greater maintenance of treatment gains (e.g. reduction of nighttime fears, bedtime resistance) at a 6 week follow-up for CBT over a home monitoring and reinforcement condition (Pincus et al., 2012).

Although studies on nighttime fears provide initial knowledge about the relationship and treatment of comorbid sleep and anxiety, our ability to generalize the findings to all forms of anxiety is restricted. First, the studies discussed above focus on a narrow form of anxiety that occurs within the context of a sleep episode. It can be debated whether nighttime fears fit more under the umbrella of anxiety or SRPs. Second, components of CBT (e.g. relaxation, contingency management, and exposures) are implemented around bedtime in many of these studies. However, most youth with anxiety

do not simply have nighttime fears and the strategies used in CBT for anxiety are needed throughout the day. Accordingly, these studies do not fully address the question whether CBT for anxiety disorders has an impact on SRPs.

Aside from studies treating specific nighttime fears, only two studies have investigated the impact of CBT for anxiety on SRPs. Kendall and Pimentel (Kendall & Pimentel, 2003) found that following CBT for GAD, youth experienced a decrease in somatic symptoms, including reduced "sleep disturbance." However, the study did not specifically examine SRPs and used a one-item measure of sleep disturbance. Another study examined SRPs after CBT treatment for youth OCD (Storch et al., 2008). Of the 41 treatment completers, a total reduction in parent-reported SRPs was observed, as well as specific reductions in sleep variables including, nightmares, being overtired, sleeping less than others, sleeping more than others, having trouble sleeping, sleeping next to someone (ϕ = .44-.68). The authors hypothesized that change in mood and arousal as well as reductions in parental accommodation may account for improved sleep.

Overall, research on the secondary effects of CBT for SRPs on anxiety is in its infancy. Preliminary studies of CBT-I in adults support secondary improvement on anxiety, yet research in youth is largely absent. This is likely due to the general lack of studies evaluating CBT for SRPs in childhood. As future RCTs continue to assess the efficacy of CBT for SRPs, researchers should assess changes to secondary anxiety symptoms. To date, there are no studies that systematically evaluate the impact of CBT for anxiety on SRPs in a sample of youth with diverse anxiety disorders. The existing studies are restricted by a combination of poor measures to assess sleep, samples of diagnostically homogeneous youth, and exclusive focus on nighttime anxiety.

Given the high comorbidity between anxiety and SRPs, shared neurological, cognitive, and social risk factors, and similar mechanisms targeted in CBT for each class of disorder, it is possible that CBT for youth anxiety could reduce SRPs (or vice-versa when treating SRPs would show secondary gains for anxiety). For example, by treating anxiety, youth may experience less nighttime arousal and accordingly, improved sleep. Self-regulatory skills introduced for principal anxiety or SRPs (e.g. cognitive restructuring), may be adapted by the youth to ameliorate the secondary problem. Likewise, parent management training may reduce accommodation of either poor sleep habits or anxious avoidance behavior (depending on whether treatment is for anxiety or SRPs) which in turn, could yield auxiliary benefit for a secondary anxiety or sleep problem. Knowledge of the effects of CBT for anxiety on SRPs (and vice-versa) has implications for both treatment recommendations of mental health professionals, and families of youth with comorbid anxiety and sleep problems who are considering psychological intervention. For example, if CBT for youth anxiety does significantly impact SRPs, then primary anxious youth with comorbid sleep problems may not require a separate sleep-focused intervention. Research into this area will give insight into the generalizability of CBT and foster development of efficient treatment.

Summary

Anxiety and sleep problems commonly present together and share many cognitive, social, and neurological features. Research has been limited by methodological shortcomings, including mono-informant reporting of SRPs, inconsistent and poor measurement of SRPs, and variability in how psychotropic medications are addressed in analyses. The relationship between sleep and anxiety is bidirectional, and research shows

that problems in one domain can exacerbate the other. Arousal, specifically in the cognitive form, appears to be an important mechanism linking sleep and anxiety. Preliminary evidence also suggests that among anxious youth, poor sleep hygiene and parental accommodation may promote SRPs. Research evaluating the efficacy of CBT on SRPs in youth is sparse, and research investigating the impact of CBT for anxiety on SRPs and the impact of CBT for SRPs on anxiety is lacking. Despite a burgeoning interest, sleep and anxiety in youth continues to be understudied.

Based on our review of anxiety and SRPs in youth, the following recommendations are made for future research on this topic:

1. Use of better measurement strategies to assess SRPs and anxiety, such as multi-informant reporting, multi-method assessments (i.e., using both subjective and objective measures to assess SRPs), use of validated measures (e.g. less reliance on items from psychological questionnaires; developmentally sensitive measures), experimental and longitudinal designs, and inclusion of healthy control groups.
2. The use of psychotropic medication should be considered in data analytic plans. All future research should review inclusionary criteria related to medication use. Similar considerations should be made for handling comorbidity in samples.
3. More studies are needed to examine the moderating effect of SES, as SES has been related to variable sleep among non-anxious populations.
4. Additional studies should investigate pre-sleep arousal, particularly the cognitive type of arousal, as this has been found to be a mechanism linking

SRPs and anxiety that could be targeted with transdiagnostic treatments for SRPs and anxiety.

5. Family accommodation and sleep hygiene should be evaluated as mechanisms for understanding the reciprocal relationship between anxiety and SRPs.
6. Studies are needed to examine the secondary benefit of CBT on anxiety and SRPs. Research can further examine specific treatment mechanisms that predict auxiliary benefit.
7. Development of a standardized transdiagnostic treatment of anxiety and SRPs, with an RCT to compare to individual CBT for SRP and/or anxiety treatments.

Such research may improve the quality of care provided to youth with anxiety and SRPs and be a fruitful area of research for investigators, assuring a better night's sleep for all parties involved.

CHAPTER 3

RESULTS

Power Analysis.

Given the novelty of the proposed study, there are few past studies that are indicative of potential effect sizes for anxiety-focused CBT on SRPs. The only study to examine this issue followed treatment-seeking youth with OCD (Storch et al., 2008) and found medium-to-large effect sizes for pre- to post-treatment differences for a variety SRPs (ϕ = .47-.68). In general, studies support medium to large effects for the association between anxiety and SRPs in clinically anxious youth (Alfano et al., 2007, r = .34-.54; Alfano et al., 2009, r = .33-.46), with medium-to-large effects distinguishing SRPs in anxious versus non-anxious youth (ϕ = .34-.67, Hensen et al., 2011; Alfano & Kim, 2011, η^2 = .07-.65). Two cross-sectional studies have observed the effects sizes of pre-sleep arousal and SRPs. Gregory and colleagues (2008) found medium-to-large effects for cognitive arousal and total SRPs (child report SRPs: r = .48, parent report SRPs: r = .33), and small-to-medium and medium-to-large effects for somatic arousal and total SRPs (child report SRPs: r = .44, parent report SRPs: r = .28). Alfano and colleagues (2010) found no significant associations between pre-sleep somatic arousal and parent-reported SRPs, but did find medium effects for cognitive pre-sleep arousal and several parent-reported SRPs (r = .29-.32).

A completer sample size of 69 is adequately powered to detect a medium-to-large effect size (f^2 = .20) for a repeated measures ANCOVA, examining one interaction variables and controlling for three covariates. Power analyses indicate that 69 participants

is greater than the number of participants required to power bivariate correlations and hierarchical regressions (aim 2; medium-to-large effects).

Assumptions

Regarding assumptions, sphericity was assumed in all repeated measures ancova analyses (> .90). A missing data analysis was conducted. Data was considered missing if >10% of items were incomplete on main outcome measures. Missing data on the sleep diary was considered to be < 3 days completed. A total of 1.81% of data was missing. A series of independent t-tests revealed that data were missing at random. Given the low level of data missing at random, cases were excluded pairwise and mean imputation was employed. For a timeline of measures, see figure 1.

Figure 1. Timeline of measures

Pre Treatment Assessment	**Two weeks before session 1 therapy**	**Session 1**	**Week before final assessment**	**Final assessment**
Parent: FASA FACLIS **Diagnostician** ADIS	**Family:** Sleep Diary	**Child:** PSAS-C SSR ASHS* **Parent:** CSHQ CSHS* **Therapist:** Med form	**Family:** Sleep diary	**Child:** PSAS-C SSR ASHS* **Parent:** CSHQ CSHS* FASA FACLIS **Diagnostician** ADIS CGI

Note. ADIS = Anxiety Disorder Interview Schedule, ASHS = Adolescent Sleep Hygiene Scale, CGI = Clinical Global Impressions Scale, CSHS= Child Sleep Hygiene Scale, CSHQ = Child Sleep Habit Questionnaire, FASA = Family Accommodation Scale-Anxiety, FACLIS- Family Accommodation Checklist and Interference Scale, PSAS-C= Pre-sleep Arousal Scale for Children, SSR = Sleep Self Report

Main Findings

Descriptive information on study participant variables and treatment variables are presented in tables 3.1-3.3. See figure 3.2 for consort table. Means and standard deviations of outcome variables are presented in table 3.4. Independent sample t-tests assessed differences by site and the completer (n = 69) and non-completer sample (n = 36) site differences by age, sex, income, CGI-I, season change, sleep medication, prior sleep study, pretreatment CSR, and primary outcome variables at pretreatment. There was a significant difference by pretreatment CSR (t=3.35, p=.001), in which average CSR was higher at Temple University (M = 5.69, SD = .84) compared to Boston University (M = 4.85, SD = .80). There were no other significant site differences. Because CSR was not directly assessed in the main analyses, site was not included as a control variable. There were no significant differences by completer status.

Table 3.1

Descriptive Information for Study Participants

Variable	*n*	%
Child age in years	*M* = 10.86	*SD* = 2.99
Gender		
Males	31	44.9
Race		
Caucasian	58	84.1
Hispanic	1	1.0
Asian-American	3	2.9
African-American	1	1.0
Other	6	5.7
Income		
$0-19,999	1	1.4
$20,000-39,999	6	8.7
$40,000-59,999	8	11.6
$60,000-79,999	5	7.2
Over $80,000	44	63.8
Not reported	5	7.2
Site		
Temple University	56	81.2
Boston University	13	18.8

Table 3.2

Pretreatment Diagnostic Information for Study Participants

Variable	*n*	%
Principal Anxiety Diagnosis		
GAD	33	47.8
Social Phobia	14	20.3
Specific Phobia	9	13.0
SAD	8	11.6
Selective Mutism	2	2.9
OCD	1	1.5
Panic Disorder	1	1.5
AD-NOS	1	1.5
Prior Sleep Study/Treatment		
Yes	6	9.0
No	60	87.0
Missing	3	4.0
Medication		
Sleep Aid	6	8.7
SS(N)RI/Antidepressant	9	13.0
Stimulant	6	8.7
Total	21	30.4
Non-Anxiety Comorbidity		
ADHD	8	11.6
ODD	3	4.3
MDD/Dysthymia	6	8.7

Note. ADHD = Attention Deficit/Hyperactivity Disorder, AD-NOS = Anxiety Disorder-Not Otherwise Specified, GAD = Generalized Anxiety Disorder, MDD = Major Depressive DisorderOCD = Obsessive Compulsive Disorder ODD = Oppositional Defiant Disorder, SAD = Separation Anxiety Disorder

Table 3.3

Treatment and Posttreatment Diagnostic Information for Participants

Variable	*n*	%
Therapy Protocol		
Coping Cat	57	82.6
MATCH	8	11.6
CBT for SM	2	2.9
Riding the Wave	1	1.4
CBT for OCD	1	1.4
Number of Sessions		
10-11	4	5.8
12-13	6	8.7
14-15	10	14.5
16	49	80.0
Why Therapy Ended		
Remission	25	36.2
Family discontinued	4	5.8
Reached session 16	40	58.0

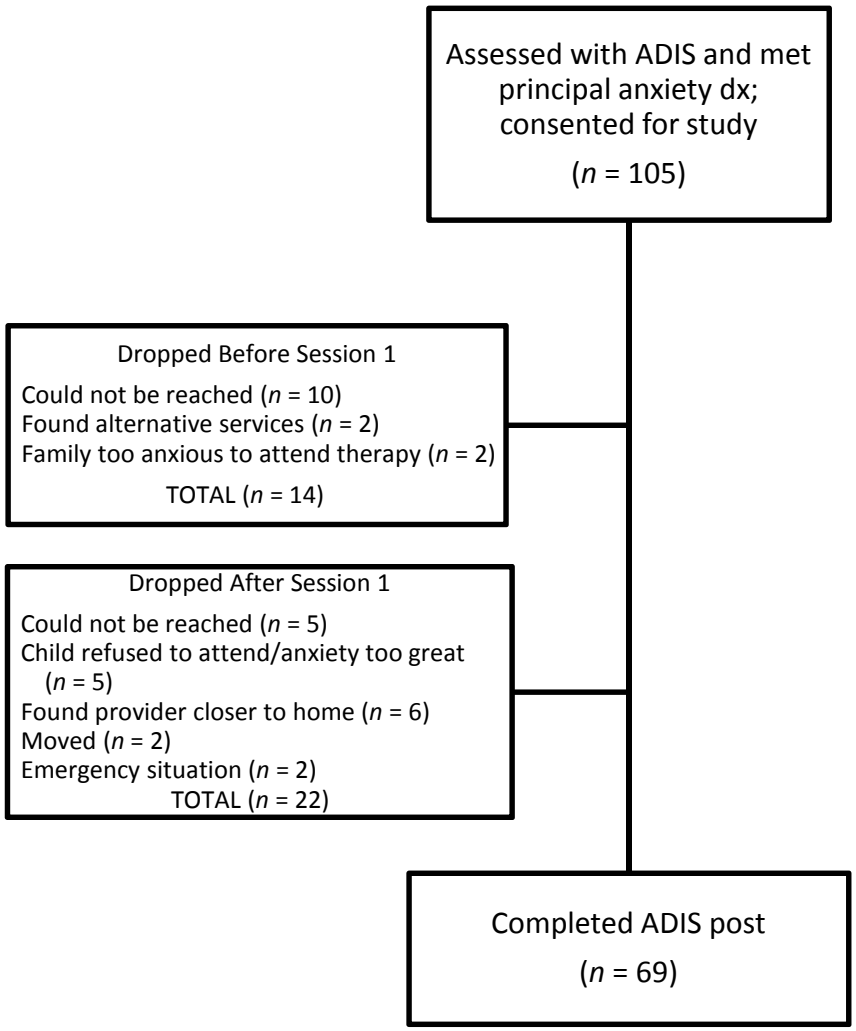
Note. CBT = Cognitive Behavioral Therapy, MATCH = Modular Approach to Therapy for Children with Anxiety, Depression, Trauma, or Conduct Problems, OCD = Obsessive Compulsive Disorder, SM = Selective Mutism,

Table 3.4. Means and Standard Deviations of Primary Outcome Measures

	Pretreatment			Posttreatment		
	Responder	Nonresponder	Total	Responder	Nonresponder	Total
CSHQ						
Total	45.75(7.91)	45.10(7.75)	45.62(7.71)	41.87(6.90)*	46.76(7.83)	43.38(7.34)
Resist.	8.42(2.00)	8.00(1.86)	8.30(1.95)	7.43(1.66)*	8.27(1.96)	7.71(1.79)
Anx.	5.63(1.45)	5.11(1.41)	5.48(1.46)	4.78(1.20)*	5.50(1.34)	5.01(1.29)
Dur.	4.02(1.09)	3.74(.81)	3.94(1.01)	3.60(.69)	4.07(1.10)	3.38(.86)
Onset	1.30(.70)	1.32(.59)	1.31(.66)	1.31(.67)	1.36(.73)	1.32(.68)
Wak.	3.61(.83)	3.61(.79)	3.61(.81)	3.59(.87)	3.70(1.14)	3.63(.96)
Sleep.	11.47(2.24)	11.43(2.30)	11.46(2.24)	10.78(2.10)	11.22(2.89)	10.93(2.37)
SSR						
Total	35.94(6.86)	36.10(7.76)	35.99(7.08)	34.18(6.25)	35.22(6.26)	34.27(6.20)
Bedtime	18.97(4.10)	19.18(4.06)	19.04(4.06)	17.52(3.64)	18.90(3.67)	17.84(3.64)
Beh.	10.32(3.00)	9.98(2.56)	10.21(2.87)	9.72(2.77)	9.48(2.44)	9.59(2.61)
Sleep.	6.74(1.90)	7.18(2.46)	6.89(2.05)	6.97(2.08)	6.88(2.02)	6.89(2.05)
DIARY						
Wak.	5.99(9.50)	6.29(7.09)	6.51(9.39)	3.26(5.47)	4.10(5.70)	3.50(5.44)
Lat.	18.95(12.45)	24.81(27.43)	21.32(18.50)	17.06(11.86)	31.56(36.64)	21.47(22.61)
Sleep.[a]	3.24(.68)	3.24(.79)	3.24(.69)	3.10(.75)	3.09(.65)	3.09(.74)
Eff.	.93(.04)	.89(.08)	.92(.06)	.93(.06)	.90(.05)	.92(.05)
Dur.	605(37)	601(72)	602(52)	624(89)	627(94)	624(88)
PSAS-C						
Total	28.05(9.58)	30.15(10.92)	28.71(9.99)	25.30(7.76)	26.28(7.69)	25.60(7.70)*
Som.	12.96(5.91)	12.90(5.72)	12.94(5.80)	11.77(3.96)	11.66(4.07)	11.74(3.97)
Cog.	15.10(5.29)	17.27(6.92)	15.78(5.88)	13.53(5.39)	14.62(5.60)	13.87(5.44)*

Note. Anx = Sleep Anxiety, Beh. = Sleep Behavior, Cog = Cognitive Subscale, Dur = Sleep Duration, Eff. = Sleep Efficiency, Lat.= Sleep Latency, Onset = Sleep Onset, Resist = Bedtime Resistance, Sleep = Sleepiness, Som. = Somatic Subscale, Wak = Night Wakings. *$p < .05$. [a]Diary variables are in minutes with the exception of "Sleep." and "Eff."

Figure 3.2. Consort Diagram



The average number of days completed on the pre sleep diary was 9.98/14 and 6.17/7 on the post sleep diary. Paired t-tests between the pre- and post-sleep diary examined differences by the ratio of "activity - no activity days/total number of days" (e.g. did youth cite an activity to get up for in the morning, such as school, camp, work, or a religious service; did the ratio change by time point?). There were no significant differences (t = -.47, p = .64). The type of season change that youth experienced between pre and post-treatment were relatively proportional: School to vacation (n =17) and

vacation to school (n = 15; see table 3.3). Taken together, the results suggest a minimal impact by change of season as a confound to the main outcomes.

Independent sample t-tests also assessed whether pretreatment sleep means differed by the presence of medication. Youth on medication showed greater SRPs on diary sleep latency (t = -3.15, p = .03), diary sleep efficiency (t = 2.81, p = .007), CSHQ (t = -2.41, p = .02), and PSAC (t = -2.13, p = .04). No differences were present on total time in bed, sleepiness, nighttime wakings, or SSR. When examined with subsamples by medication class (e.g. SSRIs, stimulants, or sleep-aides) effects were no longer significant; however, groups were likely too small to detect differences. Another set of independent sample-tests were performed to determine whether responders vs. non-responders differed on any primary sleep measures at pretreatment. No differences were found with one exception, responders had better sleep efficiency at pretreatment (t= -2.56, p = .01). The difference was also present at posttreatment (t = -2.75, p = .008).

Correlations between main outcome measures are presented in table 3.5. Parent report of SRPs was positively correlated with child self-report at concurrent time points on the SSR and diary variables. Pre-sleep arousal was not correlated with parent report of SRPs; however, it was positively correlated with child self-report on the SSR, sleep latency, and sleep efficiency. FACLIS accommodation correlated with a range of SRP metrics. Additional regressions were performed with accommodation predicting pretreatment SRPs, controlling for pretreatment anxiety (i.e. to demonstrate correlation not just due to high-level of anxiety). FACLIS Scope predicted scores on the CSHQ (β = .45, t = 3.79, p < .001), SSR (β = .31, t = 2.48, p = .02), and sleep diary efficiency (β = .33, t = -2.69, p = .009); FACLIS interference predicted CSHQ (β = .43, t = 3.62, p =

.001) and sleep diary efficiency (β = -.38, t = -3.12, p = .002). Of note, there were no significant correlations between pretreatment CSR and any sleep measure at pretreatment.

Table 3.5. Correlations of Pretreatment Outcome Measures

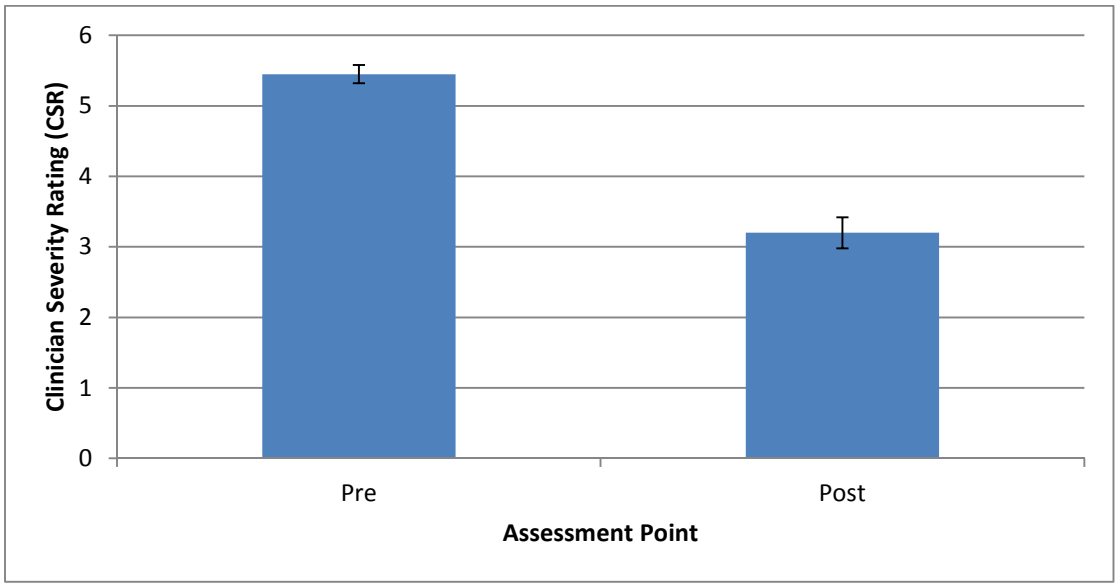
	1	2	3	4	5	6	7	8	9
1	x								
2	.43*	x							
3	.46*	.37*	x						
4	-.37*	-.35*	-.83*	x					
5	.20	.43*	.33*	-.26t	x				
6	.24	.56t	.49t	-.36	.88*	x			
7	.66*	.46*	.32t	-.19	.04	-	x		
8	.24	.25t	.01	-.09	.10	-.11	.27	x	
9	.43*	.28t	.34*	-.42*	.28t	.34	.38*	.55*	x

Note. 1 = Child Sleep Habits Questionnaire (parent report), 2 = Sleep Self Report (child report), 3 = Sleep Diary Latency (child report), 4 = Sleep Diary Efficiency (child report), 5 = Pre-sleep Arousal (child report), 6 = Adolescent Sleep Hygiene Scale (child report), 7 = Child Sleep Hygiene Scale (parent report), 8 = Family Accommodation Scale-Anxiety (parent report), 9 = Family Accommodation Checklist and Interference Scale (accommodation scope subscale; parent report). $^{t}p < .05$, $^{*}p < .01$

Anxiety improvement was assessed by a paired t-test of principal CSR on the ADIS C/P. CSR was significantly lower at posttreatment ($M_{difference}$ = 2.25(1.67) t = 11.20, $p < .001$, d = 1.50; see figure 3.3). On the CGI-I, 47 youth (68.1%) met treatment responder status. The number of sessions was associated with posttreatment CSR (r =

.25, p =.04) and CGI (r = .29, p =.02), such that fewer total sessions were associated with lower (better) CSRs.

Figure 3.3 Change in clinician severity rating for principal anxiety diagnosis over treatment



Note. Higher CSR indicates greater anxiety.

Aim 1: To assess whether sleep problems decrease pre- to post-treatment and interact with treatment response

Counter to expectations, relatively few participants presented with elevated SRPs by child report (see table 3.4, 3.6). However, the majority of participants had elevated SRPs by parent report. Relative to norms of youth without anxiety (ages 6-11), the total average of CSHQ (see table 3.4) was higher at both time points (M = 38.80; Owens et al., 2000). Of those classified as "treatment responders," 24 youth were above the CSHQ cut-off (53% of treatment responder subsample), relative to the 16 non-responders above the CSHQ cut-off (80% of the non-responder subsample) at posttreatment. A chi-squared test revealed a non-significant trend of responders as more likely to be below the clinical cut-off at posttreatment (x^2=3.56, p = .059). However, results should be interpreted with caution as the cut-off was established for youth aged 6-11.

Table 3.6

Participants over Clinical Cut-offs for Sleep Problems

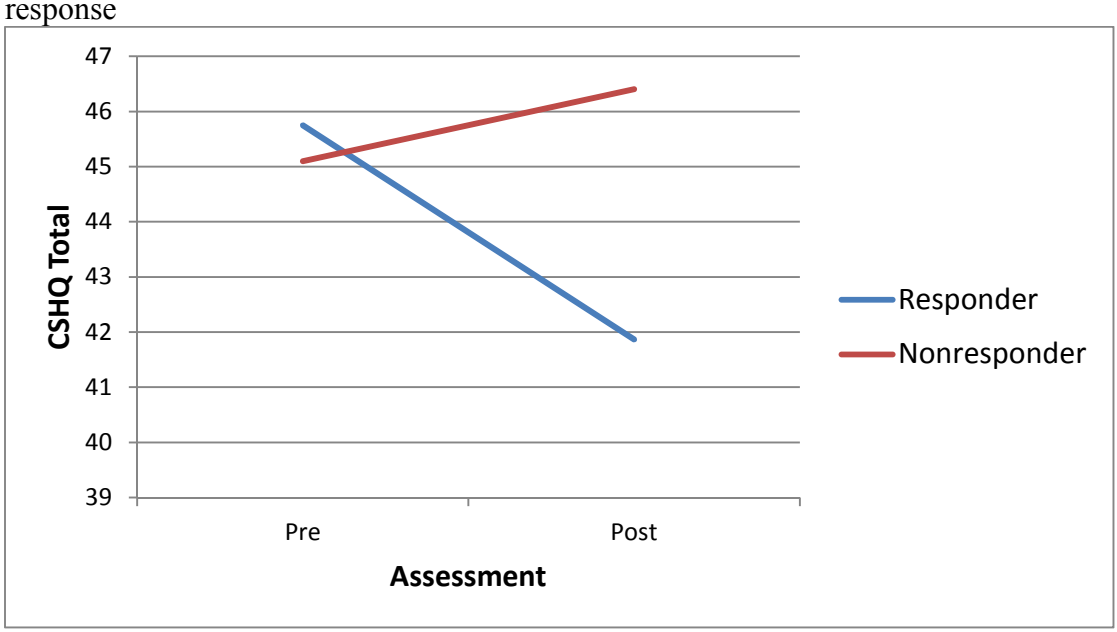
Variable	*n*	%
CSHQ (≥ 41)		
Pre	47	72.3
Post	40	60.6
Diary Latency (≥ 30 min)		
Pre	14	20.6
Post	11	16.4
Diary Efficiency (≤ .85)		
Pre	5	7.9
Post	5	7.9

Note. CSHQ = Child Sleep Habit Questionnaire.

There was an interaction between treatment response and time: Youth who responded to CBT evidenced a significant decrease in total CSHQ score ($M_{difference}$ = 3.99; F = 8.52, p = .005, η^2=.13; see table 3.4 and figure 3.4), with independent samples *t*-tests yielding significant differences between responders and nonresponders at posttreatment (t = 4.89, p = .009). Of note, CSHQ score at posttreatment for responders hovered just above the clinical cut-off of 41 (M = 41.87). Regarding subscale interactions of treatment response by time, youth who responded to CBT evidenced a significant decrease in the bedtime resistance subscale ($M_{difference}$ = 1.08; F = 8.02, p = .006, η^2=.12), the sleep anxiety subscale ($M_{difference}$ = .87; F = 11.96, p = .001, η^2=.17), and the sleep duration subscale ($M_{difference}$ = .41; F = 5.49, p = .023, η^2=.09; see figures 3.5-3.6). However, sleep duration was no longer significant following the Bonferroni correction (p = .023 > p = .008). There were no significant interactions for the sleep onset, night wakings, or sleepiness subscales. No main effects of time were observed in any of the CSHQ analyses. When the responder subsample was separately analyzed in a repeated measures ANCOVA, SRPs significantly decreased on the total CSHQ score (F = 5.17, p

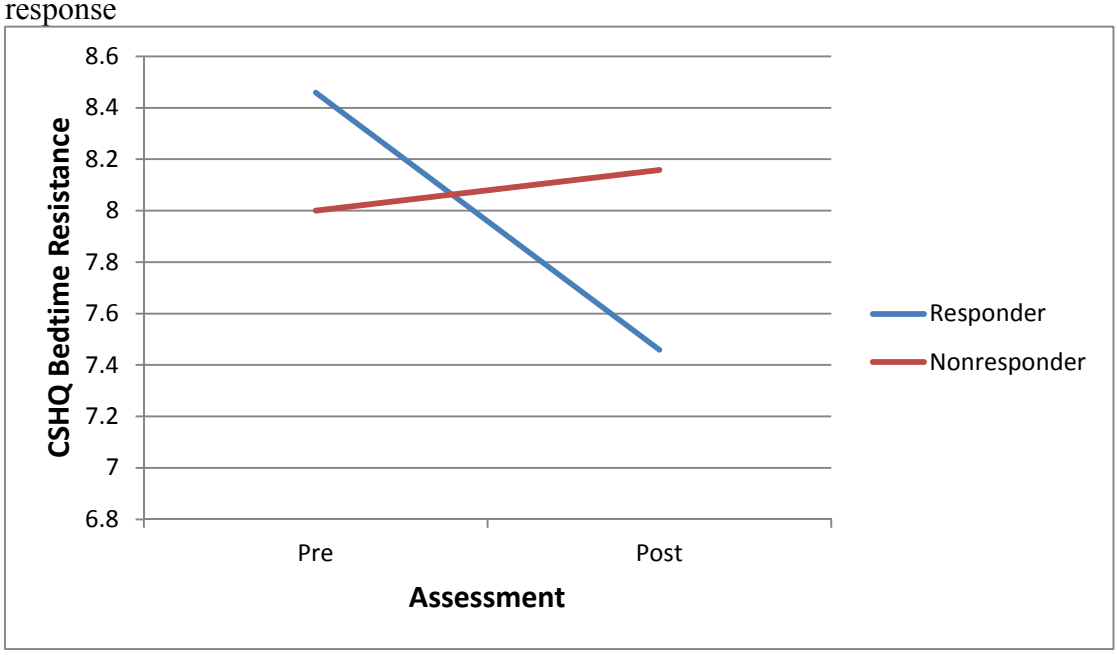
= .03, η^2=.11), CSHQ bedtime resistance (F = 11.06, p = .002, η^2=.22), and CSHQ sleep anxiety (F = 15.54, p < .001, η^2=.28).

Figure 3.4. Pre- to post- change in parent-reported total sleep problems by treatment response



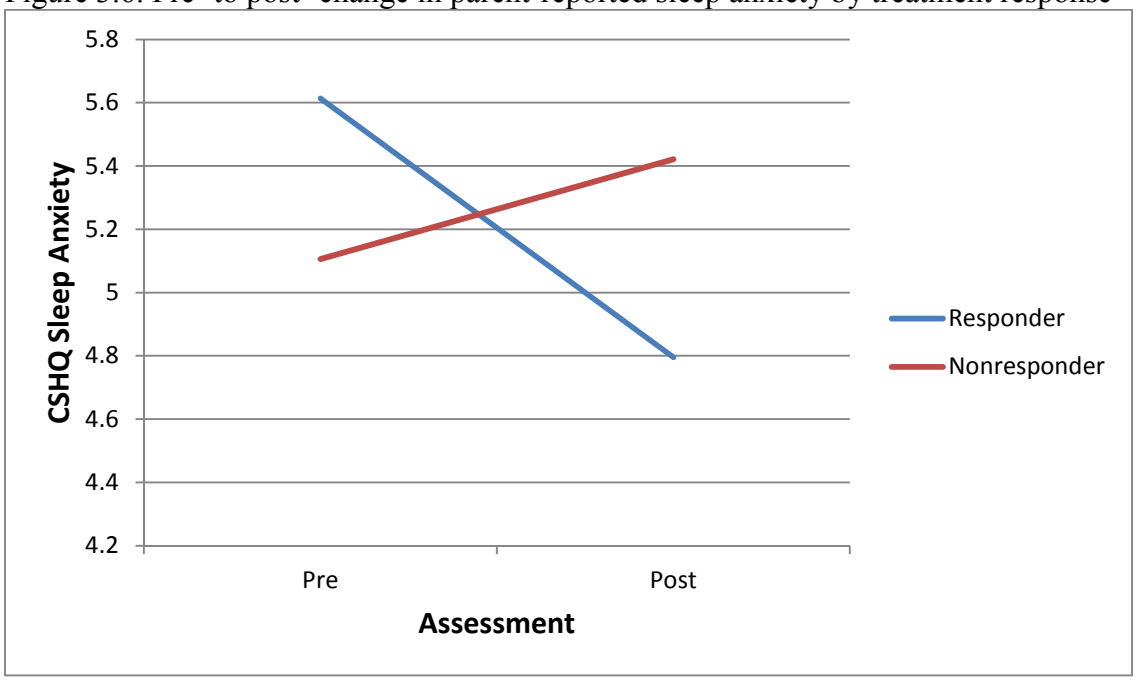
Note. Higher CSHQ score indicates greater sleep problems.

Figure 3.5. Pre- to post- change in parent-reported bedtime resistance by treatment response



Note. Higher CSHQ score indicates greater bedtime resistance.

Figure 3.6. Pre- to post- change in parent-reported sleep anxiety by treatment response



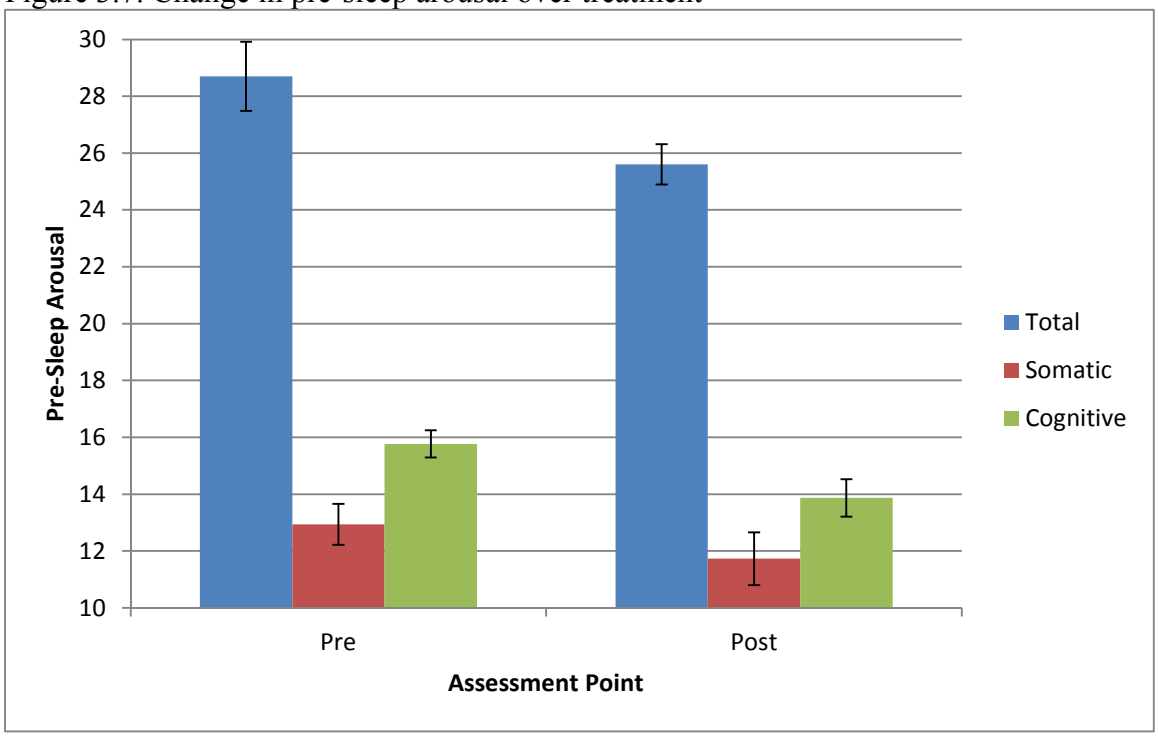
Note. Higher CSHQ score indicates greater sleep anxiety

There were no main effects of time or interactions by treatment on any SSR scale or sleep diary variable. Of note, when treatment was analyzed as a continuous variable on the CGI-I, there was a non-significant trend for the SSR bedtime subscale ($F = 2.35$, $p = .065$), in which youth who were "very much improved" compared to others had lower scores at posttreatment ($M_{difference} = 1.04$).

Aim 2: To assess change in pre-sleep arousal predicts sleep problems at posttreatment

There was a significant reduction in total pre-sleep arousal ($F = 4.51$, $p = .038$, $\eta^2 = .07$) and cognitive arousal ($F = 5.95$, $p = .02$, $\eta^2 = .09$) from pre- to post-treatment (figure 3.7). There was no change for somatic pre-sleep arousal. However, treatment responder status did not predict pre-sleep arousal at posttreatment. Change in pre sleep arousal (total, cognitive, and somatic) did not predict any main sleep outcome at posttreatment.

Figure 3.7. Change in pre-sleep arousal over treatment

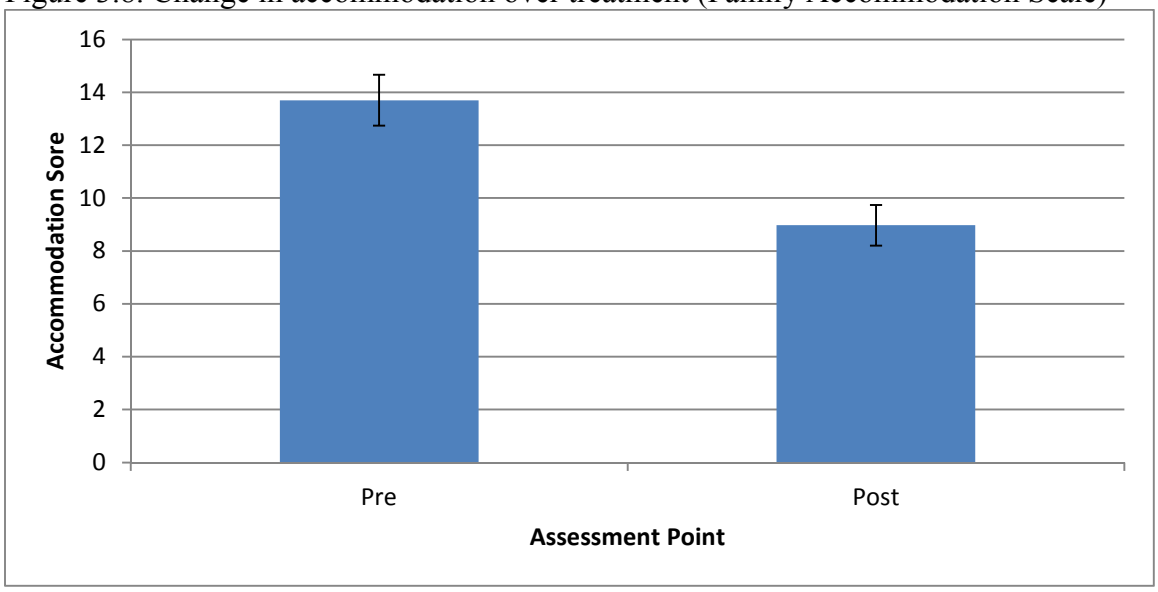


Note. Higher score indicates greater pre-sleep arousal

Exploratory Aims: Change in family accommodation and hygiene predicting SRPs at posttreatment; hygiene mediation accommodation and SRPs

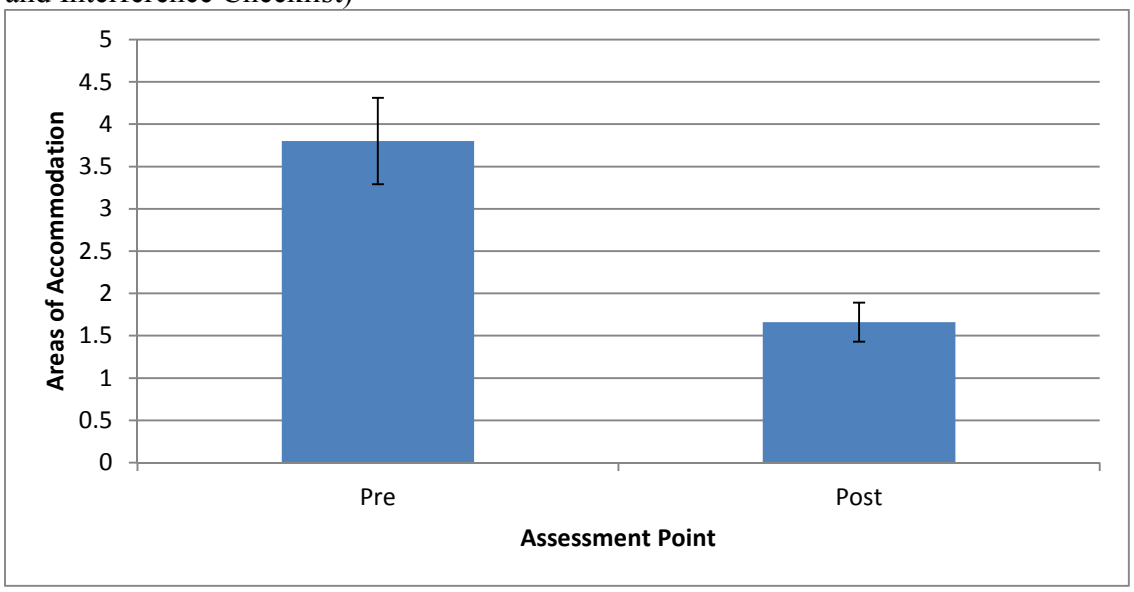
Paired t-tests revealed change in accommodation on each metric of accommodation: FASA ($M_{difference}$ = 5.13, t = 5.71, p <.001), FACLIS accommodation scope ($M_{difference}$ = 2.21, t = 4.65, p <.001), and FACLIS mean accommodation interference ($M_{difference}$= 12.83, t = 5.93, p <.001; see figures 3.8-3.10). Change in accommodation was entered into a regression to predict SSR total, sleep diary, PSAS-C scales and CSHQ total and previously significant subscales (e.g. sleep anxiety and bedtime resistance). Change in FASA and FACLIS variables did not predict any outcomes (when examined by total sample or responder subsample). There was a non-significant trend in which reduction in FACLIS mean accommodation interference trended toward reduced posttreatment CSHQ sleep anxiety (β = -.22, t = -1.90, p = .063).

Figure 3.8. Change in accommodation over treatment (Family Accommodation Scale)



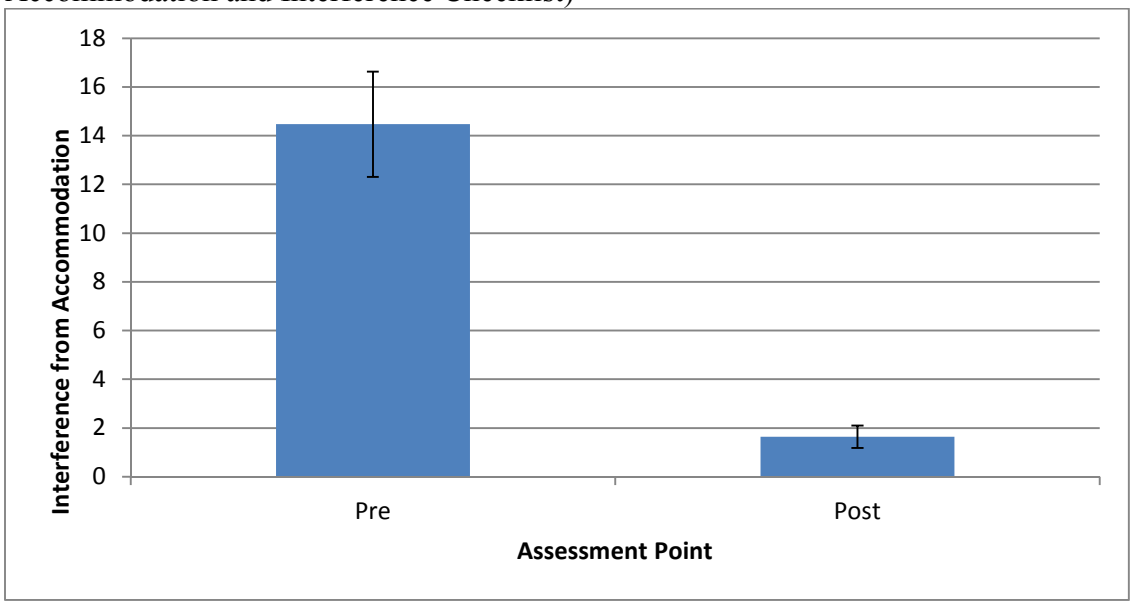
Note. Higher scores indicate greater accommodation

Figure 3.9. Change in areas of accommodation over treatment (Family Accommodation and Interference Checklist)



Note. Higher scores indicate more areas in which parents accommodate youth anxiety

Figure 3.10. Change in accommodation-related interference over treatment (Family Accommodation and Interference Checklist)

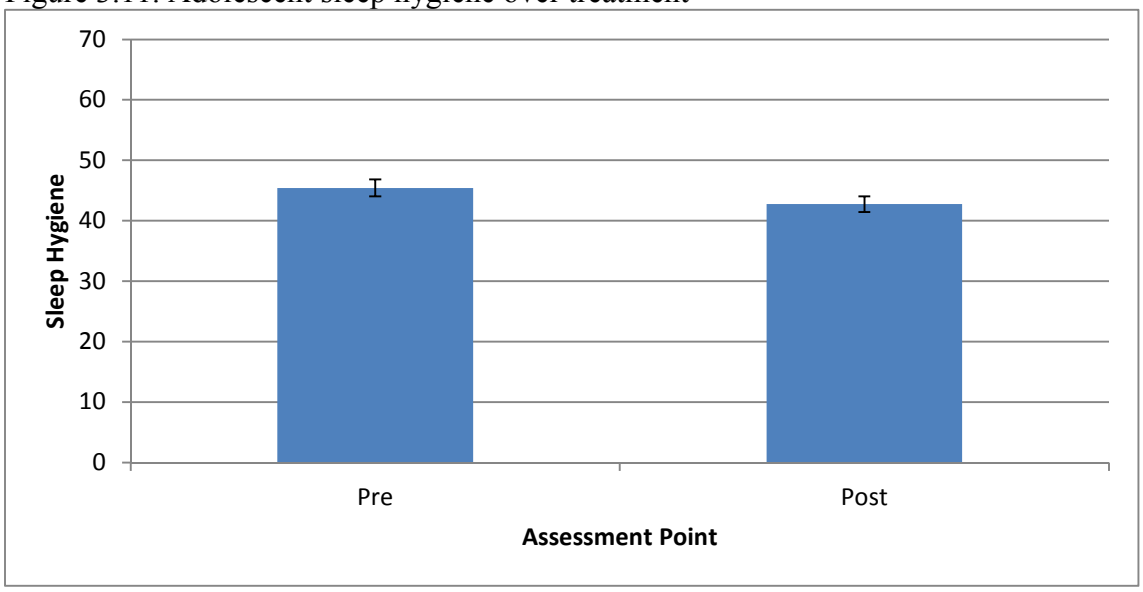


Note. Higher scores indicate greater accommodation-related interference

There was no significant change to sleep hygiene from pre- to post-treatment on either the CSHS or ASHS (i.e. sleep hygiene; figures 3.11-3.12). Sleep hygiene did not mediate accommodation and SRPs. Although not included in the initial hypotheses, we assessed whether pretreatment sleep efficiency predicted treatment response given differences by responder/nonresponder status at pretreatment (see preliminary analyses). When entered into a linear regression, sleep efficiency predicted lower CSR on the ADIS (β = -10.19, t = -2.00, p = .05; e.g. greater sleep efficiency predicts lower anxiety at posttreatment) and a trend toward predicting responder status on the CGI-I in logistic regression (β = 12.45, p = .058).[5]

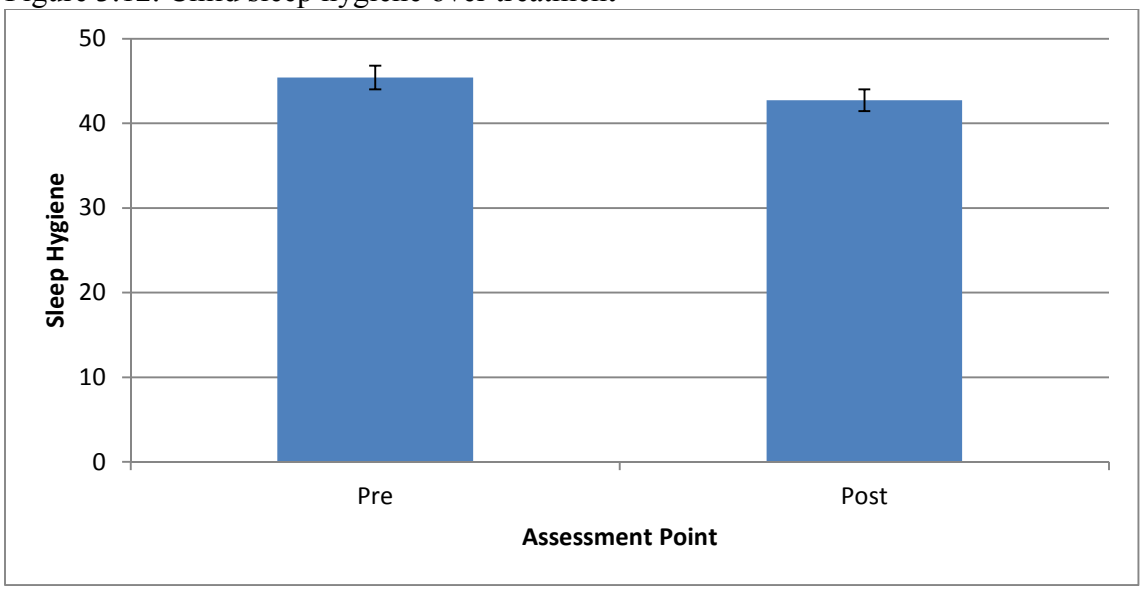
[5] The above analyses were run after an eliminating an outlier > 2 SDs. When the outlier was included sleep efficiency predicted CSR in linear regression (β = -9.85, t = -2.61, p = .01) and CGI-I became significant in logistic regression by treatment responder (β = 13.02, p = .03),

Figure 3.11. Adolescent sleep hygiene over treatment



Note. Higher scores indicate better sleep hygiene

Figure 3.12. Child sleep hygiene over treatment



Note. Higher scores indicate better sleep hygiene

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APPENDIX

SAMPLE TREATMENT ADHERENCE FORMS

The Coping Cat: Adherence Checklist
[Flexible session application when all sessions are viewed entirely]

_____ **1. Establish rapport with family**
Requirement: At least some time (10-15 minutes) from the initial (or 1st 3rd) sessions to play a game with child OR talk about "fun" issues unrelated to anxiety

_____ **2. Psychoeducation**
Requirement: Youth is taught about both (1) Physiological reactions to anxiety, (2) distinguishing between different emotional states (e.g. anxiety vs. sadness or feeling sick)

_____ **3. Create hierarchy of situations**
Requirement: List of anxious situations from least to most anxiety provoking written on paper, revisited at least once later in therapy

_____ **4. Use feelings thermometer or SUDS rating**
Requirement: Construct/present a scale to reflect intensity of anxiety with youth. Use to build hierarchy and check-in with during the exposure tasks

_____ **5. Skills Training**
Requirement: Therapist reviews relaxation and/or problem-solving with the youth

_____ **6. Cognitive Restructuring**
Requirement: Therapist collaborates with the youth to challenge thoughts and develop coping thoughts

_____ **7. Reward system constructed**
Requirement: Therapist works with parent(s) to set up a point/sticker system to earn rewards for homework compliance and exposure tasks. Therapist uses reward (e.g. prizes, praise) in session for exposure tasks.

_____ **8. In-session exposures**
Requirement: Therapy includes at least 4 in-session exposure tasks.

_____ **9. Homework**
Requirement: Therapist assigns parents and/or child homework at least 80% of the time. At least 4 assignments include an exposure task.

Family Based Treatment for Young Children with OCD: Adherence Checklist
[Flexible session application when all sessions are viewed entirely]

_____ **1. Establish rapport with family**
Requirement: At least some time (10-15 minutes) from the initial (or 1st 3rd) sessions to play a game with child OR talk about "fun" issues unrelated to anxiety

_____ **2. Present psychoeducation to family**
Requirement: Discussion of at least one of the following: Development/course of OCD, neurobiology of OCD, rationale of obsessions and compulsions, rationale of exposures (habituation)

_____ **3. Create hierarchy of symptoms**
Requirement: List of anxious situations from least to most anxiety provoking written on paper, revisited at least once later in therapy

_____ **4. Parent training**
Requirement: Elaborated discussion of at least two of the following principles with parents: Differential attention, praise/encouragement, modeling, scaffolding, positive reinforcement

_____ **5. Use feelings thermometer or SUDS rating**
Requirement: Construct/present a scale to reflect intensity of anxiety with youth. Use to build hierarchy and check-in with during the exposure process

_____ **6. Youth taught to "bossing back" OCD**
Requirement: Therapist instructs youth to name their OCD and develop phrases to "talk back" to it when experiencing the urge to compulse

_____ **7. Reward system constructed**
Requirement: Therapist works with parent(s) to set up a point/sticker system to earn rewards for homework compliance and exposure tasks. Therapist uses reward (e.g. prizes, praise) in session for exposure tasks.

_____ **8. In-session exposures**
Requirement: At least 4 in-session exposures conducted. Therapist encourages the youth not to engage in compulsive ritual.

_____ **9. Homework**
Requirement: Therapist assigns parents and/or child homework at least 80% of the time. At least 4 assignments include an exposure.