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SLEEP, EXECUTIVE CONTROL, AND PSYCHOPATHOLOGY IN CHILDREN:
A LONGITUDINAL STUDY AND AN EXAMINATION OF BRIEF SLEEP
TREATMENT

by

Katherine M. Kidwell

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SLEEP, EXECUTIVE CONTROL, AND PSYCHOPATHOLOGY IN CHILDREN:
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University of Nebraska, 2016

Adviser: Timothy D. Nelson

Researchers have acknowledged that poor sleep is not merely a symptom of psychopathology but also a contributing factor to the development of psychopathology in children (Walker & Harvey, 2010). However, more research is needed to explicate the associations among sleep, executive control (EC), and psychopathology. Specifically, there are few studies using longitudinal designs and limited research on how treating sleep can improve mental health symptoms. This dissertation provides a conceptual framework for the associations among sleep, EC, and psychopathology. The conceptual framework is bolstered by two studies. Study 1 is an examination of early sleep problems and preschool EC as predictors of risk for attention-deficit/hyperactivity disorder (ADHD) using an extended longitudinal design within a community sample. Study 2 is a pilot of a brief sleep treatment for children presenting to an outpatient clinic for emotional and behavioral disorders.

Study 1 consisted of a cohort sequential, longitudinal design ($N = 271$). At study entry, parents answered questions about sleep when children were three years old. Then, children completed nine behavioral EC tasks in the laboratory at 4.5 years. In the spring of 4th grade, teachers completed measures of ADHD symptoms. The results of a latent

moderated structural equation (LMS) model demonstrated that early sleep problems and EC deficits predicted later ADHD symptoms. EC moderated the relationship between sleep problems and ADHD symptoms, such that children with both sleep problems and poor EC were particularly vulnerable to experiencing ADHD symptoms.

Study 2 was an evaluation of a treatment protocol involving one to three sessions of sleep treatment followed by behavioral treatment of mental health symptoms. Parents of children ages six to 11 years ($N = 13$) completed measures of psychopathology symptoms and EC at baseline and following sleep treatment. Additionally, children wore actigraphs to objectively measure sleep, and parents completed weekly assessments of children's symptoms. The results indicated that there was a rapid effect of sleep treatment such that psychopathology and EC improved from baseline. A full understanding of the associations among sleep, EC, and psychopathology is critical for improving existing theories and developing more effective interventions.

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CHAPTER 1: INTRODUCTION

Children with psychopathology, including both externalizing problems (Chervin, Dillon, Archbold, & Ruzicka, 2003; Cortese, Faraone, Konofal, & Lecendreux, 2009) and internalizing problems (Alfano, Ginsburg, & Kingery, 2007; Chorney, Detweiler, Morris, & Kuhn, 2008), often have poor sleep. Although the National Sleep Foundation recommends that children ages five to 12 years old obtain 10 to 11 hours of sleep each night, research with a clinical sample found that on average children slept only 7.6 hours (Van Dyk, Thompson, & Nelson, 2016), resulting in about a three hour sleep deficit every night. Sleep deficits are problematic, because poor sleep can exacerbate mental health symptoms and degrade cognitive functioning (see Alfano & Gamble, 2009 and Beebe, 2011 for reviews). More research is necessary to explicate the associations among sleep, EC, and mental health. Moreover, there is a lack of research on how treating sleep can improve mental health symptoms. Developing and implementing a brief sleep treatment at the beginning of behavioral treatment would be expected to improve current interventions, because sleep treatment would target underlying cognitive and regulatory deficits.

To develop the framework for the current projects, several relevant literatures will be reviewed. First, the association between externalizing disorders and sleep disorders will be reviewed with an exploration of potential brain-based mechanisms underlying the relationship. Second, the literature on internalizing disorders and sleep will be reviewed with an examination of potential brain-based mechanisms. These literature reviews will demonstrate that although existing research has found associations among sleep, EC, and mental health, there is a lack of research using longitudinal designs and examining the

three constructs in the same study. The reviews will lay the foundation for Study 1, which is a longitudinal study examining early sleep problems and preschool EC as predictors of risk for ADHD. Following the chapters covering Study 1, Study 2 (a pilot treatment study) will be introduced by reviewing literature on the treatment of externalizing disorders, treatment of internalizing disorders, and the brief literature on the use of sleep treatment for mental health problems. These reviews will demonstrate that sleep appears to be an important contributor to various forms of child psychopathology and is an appropriate, yet overlooked, target for treatment.

Externalizing Disorders and Sleep

A significant body of research links externalizing behavior problems and poor sleep in children. The majority of this research has focused on ADHD, a prevalent neurodevelopmental disorder characterized by inattention and impulsivity (Barkley, 1997). ADHD is estimated to occur in about 7-8% of children and adolescents (Thomas, Sanders, Doust, Beller, & Glasziou, 2015). About 80% of children with ADHD are estimated to have a comorbid psychological disorder (Pliszka, 2000), and about half have comorbid oppositional defiant disorder (ODD; Kutcher et al., 2004). Longitudinal studies have found that ADHD leads to ODD and conduct disorder (CD), but the reverse relationship is not supported (Thapar et al., 2006). Children with ADHD also are more likely to experience various sleep disorders.

Children with ADHD often have comorbid sleep problems. Research consistently finds that parents report high levels of sleep problems in children with ADHD (see Owens, 2005 and Owens, 2008 for a review). For instance, Sung and colleagues estimated that 73% of children with ADHD have sleep problems (Sung, Hiscock,

Sciberras, & Efron, 2008). Furthermore, a meta-analysis of six studies revealed that non-medicated children with ADHD had more parent-reported sleep problems than controls (Cortese, Faraone, Konofal, & Lecendreux, 2009). The most commonly reported sleep problems were poor sleep quality, short sleep duration, bedtime resistance, and daytime sleepiness (Cortese et al., 2009). Additionally, parents indicated that their children sleepwalked, had night terrors, had confusional arousals, snored, and were more physically active while sleeping (Silvestri et al., 2009).

Objective measurement also indicates that children with ADHD have difficulties with sleep. Cortese and colleagues' (2009) meta-analysis included 13 articles with objective measurement. The objective measures indicated children with ADHD had problems including fragmented sleep, poor sleep efficiency, daytime sleepiness, and sleep-disordered breathing (Cortese et al., 2009). The two most common types of objective measurement that are used in sleep studies are actigraphy and polysomnography (PSG). Actigraphy uses a small device typically worn on the wrist to assess movement and light to determine sleep/wake patterns. PSG assesses multiple physiological aspects of sleep including brain activity, eye movements, muscle activity, and heart rhythm. Studies using actigraphy have tended to find higher motor activity during sleep and greater daily variability in sleep and wake times (Gruber & Sadeh, 2004; Gruber, Sadeh, & Raviv, 2000; Konofal, Lecendreux, Bouvard, & Mouren-Simeoni, 2001).

Empirical research using PSG to assess the sleep of children with ADHD has produced mixed findings (Gruber et al., 2009; Kirov et al., 2004). Several studies have found that children with ADHD have increased slow wave activity in the delta frequency

range (Ringli et al., 2013; Silvestri et al., 2009). Specifically, high-density electroencephalography (EEG) revealed that slow wave activity occurred over the bilateral central and frontal regions of the brain (Pisarenco, Caporro, Prosperetti, Manconi, 2014). Some studies have found shorter sleep durations, decreases in rapid eye movement (REM) sleep, and more sleep cycles in children with ADHD compared to control groups (Gruber et al., 2009; O'Brien et al., 2003; Owens, Mehlenbeck, Lee, & King, 2008). For example, Gruber and colleagues (2009) conducted PSG with 15 children with ADHD and 23 controls. The children (ages seven to 11) were instructed to not consume any stimulant medication or caffeine during the course of the study. Children with ADHD had shorter REM sleep, a lower percentage of REM sleep, and shorter sleep duration (Gruber et al., 2009). In contrast to studies finding shorter REM sleep, Kirov and colleagues (2004) found an increased duration of REM sleep and number of sleep cycles in preadolescent boys with ADHD. They theorized that increased sleep cycles reflected an altered regulation of the sleep cycle leading to a faster transition to REM sleep. Forced REM initiation suggests sleep may be another self-regulation impairment in children with ADHD.

Because of the inconsistent findings using PSG, Sadeh and colleagues (2006) conducted a meta-analysis on PSG sleep parameters in children with ADHD. Overall, sleep architecture was not very different between children with ADHD and controls (Sadeh, Pergamin, & Bar-Haim, 2006). However, they found a moderating effect of age on ADHD and sleep such that studies with younger children tended to find shorter sleep than controls, whereas older children had longer sleep than controls. The authors explained that the studies were so inconsistent in their methodologies and samples that

comparing across studies with enough power to find effects was challenging (Sadeh et al., 2006). The one significant main effect was that children with ADHD were more likely to have restless leg syndrome or periodic limb movement disorder (Sadeh et al., 2006). These findings indicate that children with ADHD have heterogeneous sleep problems.

Finally, children with ADHD often present with increased daytime sleepiness when measured objectively using the Multiple Sleep Latency Test (MSLT). Children with ADHD were more likely to fall asleep in the daytime than peers without ADHD. They also had shorter sleep-onset latency (Golan, Shahar, Ravid, & Pillar, 2004; Lecendreux, Konofal, & Mouren-Siméoni, 2000). Owens (2008) has postulated that MSLT results suggest that ADHD is associated with hypoarousal, and thus, hyperactivity serves to offset daytime sleepiness. Daytime sleepiness can be considered a measure of impairment as it reflects sleep deficits (Anderson, Storfer-Isser, Taylor, Rosen, & Redline, 2009). Thus, children with ADHD display clinical signs of sleepiness reflecting their sleep debt.

Empirical literature provides evidence that sleep problems lead to EC difficulties in both clinical and community samples. EC is conceptualized as a set of neurocognitive processes including attentional flexibility, inhibitory control, and working memory (Carlson, 2005; Garon, Bryson, & Smith, 2008). The research literature has used many terms to describe these neurocognitive processes including *executive function*, *effortful control*, and *self-regulation*. The term *EC* is often used in the field of neuropsychology and when studies include individually administered laboratory tasks, as is the case in Study 1 (Espy, Sheffield, Wiebe, Clark, & Moehr, 2011). As EC deficits are implicated

in the etiology of ADHD (Barkley, 1997; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005), sleep problems may lead to symptoms of ADHD by impacting underlying EC. For example, in a longitudinal study of 916 twins, parents reported on their children's sleep and youth completed laboratory tasks of EC at multiple time points throughout childhood and adolescence (Friedman, Corley, Hewitt, & Wright, 2009). When analyzing the data, the researchers created a latent trait of EC abilities to examine the impact of sleep on overall EC. The results indicated that change in sleep over time was related to EC in adolescence (Friedman et al., 2009). Specifically, when sleep problems improved, EC improved (Friedman et al., 2009). Furthermore, in terms of attentional flexibility, daytime sleepiness has been associated with worse flexible shifting when measured by a neurocognitive task (Anderson et al., 2009), indicating that sleep impairment may mimic symptoms of ADHD.

Although findings have been mixed, experimental studies that have manipulated sleep have generally found that sleep restriction led to deficits in EC (Gruber et al., 2011) by impacting the prefrontal cortex (Sadeh, 2007; Yoo, Gujar, Hu, Jolesz, & Walker, 2007). Experimental studies that have examined attention have tended to find that youth had worse attention following sleep restriction (Beebe et al., 2008; Beebe, 2011; Fallone, Acebo, Seifer, & Carskadon, 2005; Gruber et al., 2011). Research on inhibition/impulsivity has found disparate results. For example, Gruber and colleagues (2012a) presented evidence that children who underwent experimental sleep extension had better inhibition. In contrast, other experimental studies have not demonstrated a difference in inhibition and hyperactivity after sleep manipulation (Fallone, Acebo, Arnedt, Seifer, & Carskadon, 2001; Sadeh, Gruber, & Raviv, 2003). A meta-analysis of

experimental sleep restriction/extension studies with children found that hyperactivity/impulsivity was not significantly affected by sleep loss (Lundahl, Kidwell, Van Dyk, & Nelson, 2015). However, disparate inhibition results can be explained by distinguishing between two different types of inhibition. Response inhibition involves inhibiting a behavioral response and interference suppression involves inhibiting attention to distractions (Nelson, Nelson, Kidwell, James, & Espy, 2015). Therefore, response inhibition may not be impacted by sleep problems, as poor sleep may lead to hypoactivity that delays responses, giving impulsive children time to inhibit unwanted automatic responses (Fallone et al., 2005). Interference suppression is more likely to be impacted by sleep loss, because it involves the more complex, higher-order task of directing and maintaining attention (Nelson et al., 2015).

Theoretically, the results from experimental studies suggest that sleep deficits may have differential effects on cognition (Friedman et al., 2009; Nelson et al., 2015). The consequences of poor sleep are most apparent on tasks with a substantial cognitive load, particularly for those assessing abilities centered in the prefrontal cortex (Sadeh, 2007). Therefore, general intelligence should not theoretically be impacted by sleep problems. This theory has been supported by research showing that sleep deficits impact EC, but not general IQ (Bernier, Beauchamp, Bouvette-Turcot, Carlson, & Carrier, 2013; Nelson et al., 2015). Taken as a whole, these findings suggest that even when examined in healthy samples, sleep problems have been associated with increases in ADHD-like symptoms (Gruber et al., 2011).

The other line of research that has examined sleep problems predicting ADHD-like impairments in EC involves primary sleep disorders including sleep-disordered

breathing, restless leg syndrome, periodic limb movement disorder, and circadian rhythm disorders. Sleep-disordered breathing includes obstructive sleep apnea, gasping, and paradoxical chest and abdominal wall movements (see Owens, 2008 for a review). Sleep-disordered breathing has been associated with inattention and hyperactivity symptoms measured cross-sectionally (Chervin et al., 2002) and an increased risk for developing hyperactivity over time (Chervin, Ruzicka, Archbold, & Dillon, 2005). Owens and colleagues (2008) reviewed the medical records of 235 children aged three to 18-years-old undergoing PSG for symptoms of sleep-disordered breathing. They found that 23% of the sample had a reported diagnosis of ADHD (Owens et al., 2008). Neurobehavioral deficits from sleep-disordered breathing have been theoretically tied to repeated episodes of hypoxia leading to inflammation (Bourke et al., 2011). Additionally, frequent night awakenings due to sleep-disordered breathing could lead to daytime sleepiness and impact EC. The theories are supported by studies indicating that children who have received treatment for sleep-disordered breathing have reductions in ADHD symptoms. For example, 50% of children initially meeting criteria for ADHD did not after adenotonsillectomy (Chervin et al., 2006). In addition to sleep-disordered breathing, researchers have investigated the impact of periodic limb movement disorder and restless leg syndrome (Cortese et al., 2012; Gagliano et al., 2011). Konofal et al. (2007) found that in a sample of five to eight-year-olds, ADHD symptoms were worse in children who had comorbid restless leg syndrome. Other research has found that restless leg syndrome correlates with inattention, impulsivity, hyperactivity, and oppositional behaviors (Silvestri et al., 2007). To demonstrate the comorbidity of ADHD and restless leg syndrome, 2% of healthy eight to 17-year-olds met the diagnostic criteria for restless

leg syndrome (Picchietti et al., 2007), but 44% of children with ADHD had restless leg syndrome, and 26% of restless leg syndrome patients had symptoms of ADHD (Owens et al., 2008).

Because ADHD symptoms and sleep problems frequently co-occur, researchers have investigated whether sleep impairment is a symptom or cause of ADHD (i.e., “the sleep and ADHD conundrum;” Owens, 2005). However, ADHD and sleep problems may share a common pathophysiology that could explain how problems in one system can impact the other. Exploration of physiological systems and processes underlying both sleep and ADHD can help explicate the mechanisms driving problems with both sleep and EC.

Research on the association between sleep and other externalizing problems, such as ODD and CD, is much more limited than for ADHD; however, theory and findings from sleep restriction studies suggest a possible connection. Theoretically, sleep loss increases activation in emotional centers (e.g., amygdala), decreases activation in areas responsible for inhibition [e.g., prefrontal cortex (PFC)], and reduces connectivity between the two (Dahl & Harvey, 2007; Gujar, McDonald, Nishida, & Walker, 2011). This process possibly results in a failure of “top-down” control of emotion and behavior, leading to emotionally-dysregulated externalizing behavior. Dahl and Harvey (2007) proposed that sleep loss can lead to difficulties monitoring emotional state. They argue that the lack of self-monitoring of emotions and behavior may lead to aggression (Dahl & Harvey, 2007). Moreover, an empirical study with a sample of adolescents with substance use problems demonstrated that aggressive and oppositional behavior improved as sleep problems improved (Haynes et al., 2006). Similarly, a study

conducted by Sadeh, Gruber, and Raviv (2002) found that sleep problems in school-age children were associated with behavior problems as measured by the total behavior problems scale and the delinquent subscale of the Child Behavior Checklist (CBCL). Overall, sleep deprivation promotes irritability, a decline in self-monitoring, and aggressive thoughts that are symptomatic of externalizing disorders (Dahl & Harvey, 2007).

Prefrontal cortex. Several biological explanations for the mechanisms linking sleep problems and externalizing problems are possible, including various brain-based mechanisms. The first explanation focuses on the PFC. The main brain structure most often implicated in both sleep and in ADHD is the PFC (Beebe & Gozal, 2002; Cassoff, Weibe, & Gruber, 2012; Owens, 2008). The PFC houses EC and regulates sleep and wakefulness (Walters, Silvestri, Zucconi, Chandrashekariah, & Konofal, 2008; Vriend et al., 2013). Moreover, Beebe and Gozal (2002) implicated disrupted cellular homeostasis in the association between sleep restriction and EC in the PFC. Essentially, sleep disruption can lead to deficits in the PFC which leads to EC deficits that underlie ADHD and ODD.

Dopaminergic and noradrenergic systems. A host of research has highlighted the role of PFC catecholamines (e.g., dopamine, norepinephrine) in ADHD and sleep problems (Cassoff et al., 2012; Harvey, Murray, Chandler, & Soehner, 2011; Gruber, 2009; Owens, 2008; Sagvolden, Johansen, Aase, & Russell, 2005). This research provides evidence that ADHD and sleep problems share a common pathophysiology (Gruber, 2009). Deviations in typical neurotransmitter actions in the noradrenergic and dopaminergic systems have been connected to both ADHD and sleep problems (Gruber

2009; Gruber et al., 2006; Owens, 2005). In terms of the noradrenergic system, deviations in typical norepinephrine in the locus ceruleus have been related to both ADHD and sleep problems (Faraone, Bonvicini, & Scassellati, 2014; Owens et al., 2013; Yang et al., 2013). Norepinephrine and the locus coeruleus are related to the circadian rhythm and with attention (Owens et al., 2013). In terms of the dopaminergic system, dopamine in the PFC has been shown to impact attention (Arnsten & Pliszka, 2011), while dopamine in the substantia nigra pars compacta and ventra tegmental area maintains wakefulness (Cassoff et al., 2012; Harvey et al., 2011). Essentially, dopamine is critical for maintaining the sleep-wake cycle (Lima et al., 2008). For example, in an animal study, researchers injected dopamine agonists into the brains of rats and found that rats with higher dopamine levels were more awake and alert (Isaac & Berridge, 2003). In humans, sleep restriction has been shown to decrease dopamine levels and lead to impaired EC (Cassoff et al., 2012). Some researchers have investigated how neurotransmitters regulate sleep in children with ADHD and have found that amphetamines, a class of medication used to treat ADHD, relies on the dopamine transporter gene to promote wakefulness (Harvey et al., 2011; Wisor et al., 2001).

Some researchers have investigated the genetic influence of catechol-*O*-methyltransferase (COMT). COMT is an enzyme that breaks down catecholamines such as dopamine, epinephrine, and norepinephrine (Gruber, 2009). Although COMT is found all over the body, COMT plays the biggest role in the neurons of the PFC. In humans, there is a COMT gene that influences the COMT enzyme and has two functional polymorphisms, Valine and Methionine (Gruber et al., 2006). Valine (Val) is a high activity allele that degrades dopamine four times faster than Methionine (Met). Because

Met breaks down dopamine slower, there is more dopamine in the postsynaptic neuron. Having more dopamine in the prefrontal postsynaptic neurons can influence EC which is theoretically the important piece for ADHD (Barkley, 1997; Gruber et al., 2006). In a pioneering study on COMT polymorphism, Gruber and colleagues (2006) found that children with ADHD who had a Val allele had worse sleep continuity than children with the Met allele. Overall, children with ADHD who had a Val-Val or Val-Met genotype had more risk for sleep impairment than those with the low-activity Met-Met genotype. In line with research on COMT, researchers have proposed the tonic-phasic dopamine hypothesis (Bilder, Volavka, Lachman, & Grace, 2004; Gruber, 2009). In this theory, the COMT Met allele increases baseline levels of dopamine (tonic) and decreases the release of large, brief pulses of dopamine (phasic) in subcortical regions. The Met allele also increases D1 transmission cortically (Bilder et al., 2004). Bilder and colleagues (2004) propose that increased tonic dopamine and reduced phasic dopamine in subcortical areas increases the stability of neural networks underlying EC, but makes the neural network less flexible. In contrast, the Val allele increases phasic dopamine and decreases subcortical tonic dopamine (Bilder et al., 2004). These patterns of dopamine release also play a role in REM sleep and could contribute to REM sleep disorders (Gruber et al., 2006).

Default mode network. The default mode network refers to the network of brain areas that are active when a person is *not* engaged in a task and is instead introspective or daydreaming (Castellanos & Proal, 2012). During goal-oriented tasks, the default mode network is deactivated. Researchers have proposed that ADHD is a default mode network disorder (Sonuga-Barke & Castellanos, 2007), such that children will have

lapses in attention if the default mode network is not suppressed (Castellanos et al., 2005). Some research has shown that children with ADHD have decreased default mode network suppression (Fassbender et al., 2009). Moreover, methylphenidate, a stimulant medication for ADHD, normalized the functioning of the default network suppression in the ventromedial PFC and posterior cingulate cortex (PCC) in youth with ADHD (Liddle et al., 2011). Children who were not medicated only suppressed the default network under high incentive conditions during a go/no-go task, while those on stimulant medications suppressed the default network under low and high incentive conditions (Liddle et al., 2011). In participants without ADHD, those who were sleep deprived for 24 hours showed an increase in default mode network variability and inattention similar to those with ADHD. Finally, the default mode network had decreased connectivity after sleep deprivation (De Havas, Parimal, Soon, & Chee, 2012; Kelly, Uddin, Biswal, Castellanos, & Milham, 2008).

Circadian system. The sleep/wake process is regulated by two processes, the circadian cycle (Process C) and sleep pressure (Process S). The suprachiasmatic nuclei (SCN) controls the circadian cycle (Harvey et al., 2011). Because the SCN does not maintain an internal clock that is exactly as long as external clocks, the SCN relies on external cues to maintain synchrony. One set of external cues comes from social information such as meal times and physical activity. A second external cue is light information, which travels from the photoreceptor ganglion cells in the retina via melanopsin. Melanopsin moves along the retinohypothalamic tract, which leads to the SCN. The SCN also is connected to the pineal gland, which secretes melatonin based on light information (Harvey et al., 2011). Melatonin is critical for the initiation of sleep and

reaches its highest level in the middle of the night (Harvey et al., 2011). Adversely, Process S is influenced by the history of sleep and wake periods, such that sleep pressure increases during time spent awake and decreases during sleep (Harvey et al., 2011). Sleep pressure builds over about 16 hours of being awake until the individual falls asleep. Process C and Process S maintain wakefulness during the day and sleep at night. In children with ADHD and externalizing problems, there is evidence that their circadian process is delayed.

The circadian system has been implicated in sleep problems of children with ADHD. A circadian phase delay shifts sleep and waking times. In particular, researchers have identified a delay in circadian phase characterized by melatonin that is released later in non-medicated youth with ADHD compared with controls (van der Heijden, Smits, Someren, & Boudewijn Gunning, 2005). When treated with exogenous melatonin, sleep problems improved in youth with ADHD, providing further evidence of a circadian phase delay (van Geijlswijk, Korzilius, & Smits, 2010). Circadian phase delay interferes with children being able to fall asleep and wake up at socially appropriate times. In a circadian model of sleep problems, children demonstrate habitual bedtime resistance, because they are sent to bed before they are tired. Children with circadian phase delay may have increased daytime sleepiness and bedtime resistance imitating the appearance of youth with ADHD and ODD (Gruber & Sheshko, 2008). Moreover, children with circadian phase delay may have ADHD-like symptoms caused by sleep deficits.

Gruber and colleagues (2012b) hypothesized that children with ADHD may have circadian phase delays that are creating sleep-onset difficulties and bedtime resistance. They assessed the sleep of 26 children with ADHD and 49 controls seven to 11 years old

using home PSG. The participants were told not to take stimulant medications or caffeine during the study. The researchers found that circadian phase delay was connected with bedtime problems including bedtime refusal and longer sleep latency in all children. However, children with ADHD were more likely to have a circadian phase delay and to have sleep problems than control children (Gruber et al., 2012b). This study provides evidence that circadian phase delay can lead to bedtime resistance and longer sleep-onset latency that are commonly seen in children with ADHD. These sleep problems lead to a sleep deficit which impairs EC-tasks, particularly attention shifting.

In line with the hypothesis that circadian phase delay leads to ADHD symptoms, researchers have revealed connections between ADHD and genes controlling the circadian system [Per1 and brain-derived neurotropic factor (BDNF)] (Lasky-Su et al., 2008). For example, some research has found increased BDNF in the dorsal hippocampus during REM sleep (Tononi & Cirelli, 2006) indicating that increased levels of BDNF enhanced sleep (Suntsova, Stewart, Gong, Szymusiak, & McGinty, 2003). Moreover, BDNF polymorphisms were associated with ADHD (Li et al., 2014; Liu et al., 2014). In general, it appears that BDNF may be another common physiological component underlying both ADHD and sleep impairment.

Internalizing Disorders and Sleep

In addition to associations with ADHD and externalizing disorders, sleep problems are also common among youth with internalizing problems (i.e., anxiety and depression). Anxiety disorders are the most common childhood mental health disorders, occurring in 20% of children (Costello, Egger, & Angold, 2005). Anxious children often experience a range of sleep problems including nightmares, nighttime fears, and

avoidance of sleeping away from home. In a study using PSG, children with anxiety had increased awakenings, increased sleep latencies, and decreased slow wave sleep (Forbes et al., 2008). Alfano and colleagues (2007) examined the frequency of sleep problems across anxiety disorders in children receiving medication treatment. Overall, they found that 88% of children with anxiety had a parent-reported sleep problem such as insomnia or nightmares. After medication treatment for anxiety symptoms, children also had improved sleep. These results indicate that sleep and anxiety symptoms are highly correlated, and improvements in one problem area can improve the other. Despite evidence that there are reciprocal interactions between sleep and internalizing disorders, we have found no research investigating how treating sleep with behavioral methods may improve internalizing symptoms.

Primary sleep problems may vary slightly across anxiety disorders. Children with generalized anxiety disorder have excessive worries and often present to treatment with insomnia (Alfano et al., 2007; Bagley, Kelly, Buckhalt, & El-Sheikh, 2015). Children with panic disorder have recurring panic attacks and may have fears about sleeping alone (Garland, 1995). Children with social anxiety fear evaluation in social situations and may have short sleep in anticipation of anxiety-provoking situations (Chorney et al., 2008). Children with separation anxiety disorder demonstrate excessive anxiety when separating from attachment figures and have high levels of bedtime resistance when parents encourage sleeping independently (Eisen & Schaefer, 2005).

In addition to anxiety, depression is a common internalizing disorder. Depression is estimated to occur in 5% of school-aged children and 10-20% of adolescents in community samples (Avenevoli, Knight, Kessler, & Merikangas, 2008). Structured

clinical interviews conducted with parents and children indicate that about 73% percent of children with depression have a sleep problem such as insomnia (Liu et al., 2007). In a review article, Riemann, Berger, and Voderholzer (2001) demonstrated that sleep and depression have a bidirectional relationship, with evidence of early sleep problems predicting later depression. Despite mixed findings across objective studies, overall, sleep problems in children with depression include difficulties in sleep continuity, REM latency, and REM density (for a review, see Riemann et al., 2001). In a study of 94 children ages eight to 16 years who were diagnosed with depression, anxiety, or no clinical diagnosis, youth completed Ecological Momentary Assessment (EMA) to assess daily affect and wore actigraphs to measure sleep (Cousins et al., 2011). Youth with a clinical diagnosis presented with a bidirectional association between objectively measured sleep and affect. Children with depression or anxiety who had longer sleep latencies had more negative mood the next day. Alternatively, longer sleep duration and shorter sleep latency were predictive of more positive affect the next day (Cousins et al., 2011). These findings indicate that improving sleep quality and duration can improve mood for children with internalizing disorders.

Anxiety and depression are highly comorbid in children and are often measured under the unifying construct of internalizing disorders (Brady & Kendall, 1992). A literature review examining the association between sleep, anxiety, and depression revealed significant co-occurring sleep problems, anxiety, and depression in children (Chorney et al., 2008). Specifically, Mayes and colleagues found that children diagnosed with anxiety/depression had more daytime sleepiness and slept more than children with externalizing disorders or healthy controls (Mayes, Calhoun, Bixler, & Vgontzas, 2009).

Another study by Johnson, Chilcoat, and Breslau (2000) investigated the correlation between sleep problems and anxiety/depression cross-sectionally at ages six and 11 in 823 children, using the CBCL sleep scale and the anxiety/depression scale. Children with sleep problems had increased anxiety/depression symptoms at six and 11 years old. When examining the depression subscale, the relationship between depression and sleep problems was stronger in 11 year olds than in six year olds, reflecting the developmental differences in depression. Objective measures of sleep have also found that children with internalizing disorders have high rates of sleep problems. A study investigating the predictive factors of excessive daytime sleepiness included 508 children who underwent PSG and had parent-report of anxiety and depressive symptoms (Calhoun et al., 2011). Calhoun and colleagues (2011) found that anxiety/depressive symptoms and objectively measured sleep problems predicted excessive daytime sleepiness. Both parent-report and objective measures indicate associations between internalizing disorders and sleep problems.

Longitudinal research suggests the importance of early intervention for sleep problems and internalizing disorders, as patterns tend to persist across individuals. Gregory and O'Connor (2002) examined how sleep problems predicted anxiety/depression from preschool to mid-adolescence in a prospective longitudinal study. This adoption study included 360 age- and sex-matched participants who had been adopted or raised by their biological parents. Parents completed the CBCL at multiple time points, and the sleep scale from the CBCL was used in the analyses. Sleep problems at four years significantly predicted anxiety/depression at 15 years. However, in this study early anxiety/depression at four years did not predict sleep problems at 15 years,

which makes sense given the low prevalence rate of preschool internalizing problems in this sample (Gregory & O'Conner, 2002). Extending this association further, Gregory and colleagues (2005) examined how children's sleep problems at five, seven, and nine years predicted their anxiety and depression symptoms at 21 and 26 years. Gregory et al. (2005) included 943 children in New Zealand who had participated in a national birth cohort study. Forty-six percent of children with a persistent sleep problem at nine years old had anxiety in the clinical range in adulthood. However, sleep problems at nine years old did not predict later depression. The authors explained that the results were puzzling and require replication given the high comorbidity between anxiety and depression. The results provide some evidence of the brain processes underlying the anxiety-sleep connection, as the authors suggest that poor regulation may be the common factor in both sleep problems and anxiety disorders.

Prefrontal cortex. Biological explanations for the mechanism linking sleep problems and internalizing problems include various brain-based mechanisms. Although there is limited research with children, adult research indicates that sleep can modulate affective brain responses (Gujar et al., 2011). The study included 36 non-clinical young adults who rated the intensity of emotions in different photographs. The participants who were randomized to the experimental group underwent PSG during a ninety minute daytime nap and those in the control group did not take a nap. The participants who had less sleep had decreased positive mood. Moreover, the participants who did not nap had increased sensitivity to fear and anger expressions. The authors proposed that sleep may modulate the prefrontal regulatory control of emotion processing. Specifically, the authors noted that the medial and ventral portions of the PFC are likely important brain

areas in the relationship between sleep and anxiety sensitivity (Gujar et al., 2011). The medial and ventral PFC regulate the subcortical limbic and basal ganglia areas that have been associated with internalizing disorders (Drevets, 2000), and the medial PFC plays a role in producing slow wave sleep (Peyrache, Battaglia, & Destexhe, 2011). The findings of the study provide further evidence of underlying brain areas that may be associated with both sleep and anxiety.

One study that examined how poor sleep can predict impaired emotional processing in children tested 94 healthy children at three time points from 10 years to 13 years (Soffer-Dudek, Sadeh, Dahl, & Rosenblat-Stein, 2011). The emotional processing task employed the identification of emotional expressions. A neutral task was included for comparison. Sleep was assessed with actigraphy. Children with more night awakenings and decreased sleep efficiency had worse emotional processing over time. The results demonstrate that sleep is critical for emotional processing but not for processing neutral information. The authors argue that early sleep intervention could improve the emotional functioning of youth (Soffer-Dudek et al., 2011).

Other research on the medial-prefrontal cortex (MPFC) has examined the interaction of the MPFC and the amygdala, the structure in the brain that processes emotional information (Yoo et al., 2007). The MPFC inhibits the amygdala through top-down control to regulate emotional responses. Functional magnetic resonance imaging (fMRI) research reveals that experimental sleep restriction leads to weaker connections between the MPFC and the amygdala resulting in poor regulation of emotions. EC abilities such as inhibition are housed in the PFC. When the PFC is unable to inhibit strong emotional reactions due to sleep problems, emotional dysregulation ensues.

Hypothalamic pituitary adrenal (HPA) axis. Sleep problems and internalizing disorders are linked to HPA axis dysregulation. First, research has found that the HPA axis is related to both depression (Goodyer et al., 1996) and anxiety (Dietrich et al., 2013). Second, research has found that sleep is related to the HPA axis. One study examined neuroendocrine alterations in 282 eight-year-olds by measuring cortisol and α -amylase in response to a stressful task. Sleep was assessed via actigraphy. Shorter sleep efficiencies resulted in higher levels of α -amylase and cortisol in response to the stressful task demonstrating the relationship between sleep problems and the HPA axis (Raikkonen et al., 2010). Finally, research has examined the interactions of sleep and internalizing disorders with the HPA axis. For instance, one study found that children with anxiety often have higher levels of cortisol before they fall asleep than children with depression or matched control children (Forbes et al., 2006). This series of studies demonstrates that the HPA axis is another physiological system that underlies both the regulation of sleep and anxiety/depression. Essentially, sleep problems appear to increase emotional reactions through the increased release of stress hormones. When EC deficits in the PFC are combined with increased stress hormones, children experience both increased emotionality and decreased emotional regulation, exacerbating internalizing disorders.

CHAPTER 2: STUDY 1 INTRODUCTION

Study 1 examined early sleep problems and preschool EC as predictors of risk for ADHD using an extended longitudinal design with a community sample. Because ADHD symptoms and sleep problems frequently co-occur, researchers have investigated whether sleep impairment is a symptom or a cause of ADHD (Owens, 2005). The

question of whether sleep is a symptom or a cause of ADHD fits within a larger trend of examining sleep as a predictor of psychopathology more broadly. Recent research has begun to acknowledge that sleep is not merely a symptom of psychopathology but also a predictor of the development of psychopathology during childhood (Alfano & Gamble, 2009; Harvey, 2001; Roberts, Roberts, & Duong, 2008; Walker & Harvey, 2010). For instance, Roberts and colleagues (2008) found that insomnia in youth predicted internalizing and externalizing disorders and interpersonal problems one year later. Research demonstrating that sleep problems may predict ADHD symptoms provides support for the theory that sleep problems are a risk factor for ADHD symptoms (Cassoff et al., 2012; Gregory, Eley, O'Connor, & Plomin, 2004), but much more research is needed to replicate and extend current findings [see argument in Gregory and Sadeh's (2012) clinical review].

Moreover, ADHD and sleep problems may share a common pathophysiology that could explain how problems in one system can impact another (see Chapter 1 for a thorough review). To briefly reiterate, while the relationship between sleep and ADHD may be bidirectional, it is very likely that they are comorbid disorders linked via common physiological impairments in the central nervous system (for a review see Cassoff et al., 2012). Sleep problems and ADHD symptoms share many of the same biological risk factors including cellular impairments in the PFC (Beebe & Gozal, 2002), dopaminergic system differences (Harvey et al., 2011), low iron levels (Cortese et al., 2012), and circadian phase delay (van der Heijden et al., 2005). In summary, sleep and ADHD are likely to co-occur because they share the same pathogenesis that includes the biological mechanisms driving problems with both sleep and EC.

In addition to the likely co-occurrence of sleep problems and ADHD, EC is a construct strongly linked to ADHD. Adequate EC allows for problem-solving, self-regulation, and planning, precisely the areas that are often difficult for children with ADHD. Thus, researchers have theorized that difficulties with EC contribute to the self-regulation difficulties inherent to ADHD (Barkley, 1997; Barkley, 2012). As EC deficits are implicated in the etiology of ADHD (Barkley, 1997; Willcutt et al., 2005), those with poor EC in preschool may be especially likely to have ADHD symptoms in elementary school.

Researchers have found support for the theory that EC deficits contribute to ADHD symptoms (Brocki, Eninger, Thorell, Bohlin, 2010; Espy et al., 2011; Rabinovitz, O'Neill, Rajendran, Halperin, 2016). For example, a meta-analysis conducted by Willcutt and colleagues (2005) found that youth with ADHD had impairments on all examined EC tasks. They argued that the moderate effect sizes indicated that EC deficits were not a sufficient explanation for all cases of ADHD, but rather EC deficits were one contributing factor in a complex constellation of causes. Halperin and Schulz (2006) have proposed a slightly different theory of the role of EC in ADHD. They argue that ADHD is caused, at least in part, by subcortical dysfunction. As EC develops, it exerts top-down influence on subcortical regions which allows for improvement in ADHD symptoms. Fitting with Halperin and Schulz's (2006) theory, children experiencing EC deficits would experience increased ADHD symptoms due to the weaker top-down influence of EC on subcortical regions. However, there have been few studies examining EC and ADHD symptoms in typically developing samples, and longitudinal studies of the association between behaviorally-measured EC and ADHD are rare. Research with a

community sample would allow for questions of generalizability to be addressed.

Building on research demonstrating that sleep and EC are independently associated with ADHD, some studies have investigated the relationships among sleep, EC, and ADHD. For instance, Schneider, Lam, and Mahone (2016) examined the associations among sleep, EC, and ADHD in a sample of children with and without ADHD ages four to seven at a single developmental time point. They found that sleep problems were associated with ratings of ADHD symptoms, but sleep problems were not associated with performance on EC tasks. Further, children with ADHD demonstrated worse performance on EC tasks. Additionally, Sadeh and colleagues (2002) examined objectively measured sleep, EC (neurobehavioral functioning using computerized measures), and behavior problems in a community sample of school aged children. Children with more fragmented sleep experienced problems with both EC and behavior problems. Although the interaction of sleep and EC has not yet been examined, it is possible that when children experience problems with both sleep and EC, they may have worse ADHD symptomatology.

CHAPTER 3: PRIMARY PURPOSE AND RESEARCH HYPOTHESES

Study Rationale

The current study seeks to address gaps in the literature on sleep, EC, and ADHD. This study examines the longitudinal associations among sleep problems at three years of age, EC deficits at 4.5 years, and ADHD symptoms in 4th grade. Although research has begun to identify sleep problems as a precursor to mental health problems, more research is necessary. In particular, extended longitudinal designs are needed to demonstrate that early sleep problems predict ADHD symptoms later. To maintain the temporal sequence

of the theory, the current study assessed sleep, EC, and ADHD symptoms at separate time points. Further, much of the past research has relied on parent-report of EC. This study included behavioral tasks designed to assess EC in order to build on past parent-report research. Although Barkley's (1997) theory that EC deficits underlie ADHD has empirical support (Willcutt et al., 2005), the current study provides a rigorous testing of the theory that those with poor EC will be more likely to demonstrate ADHD symptoms later. Finally, research has not yet examined the interaction of sleep and EC in predicting ADHD symptoms. Sleep and EC are both independently predictive of ADHD, but they could also interact with each other. Because sleep and EC are both theoretically related to ADHD, when both are present, children may be especially vulnerable to the occurrence of ADHD symptoms.

Hypotheses

The current study had three hypotheses. The first hypothesis was that preschool sleep problems would predict later ADHD symptoms in 4th grade. This hypothesis was based on previous findings of significant associations between early sleep problems and ADHD symptoms, but extends previous research into later elementary school using a longitudinal design. Second, it was expected that preschool EC deficits would predict symptoms of ADHD later in development. This hypothesis was based on research and theory demonstrating that EC deficits often underlie ADHD symptoms. The third hypothesis was that EC would moderate the association between sleep and ADHD symptoms, such that those with both sleep problems and EC deficits would experience more ADHD symptoms than those with high EC. This hypothesis was based on findings

that sleep and EC problems independently predict ADHD symptoms and may exacerbate symptoms when both are present.

CHAPTER 4: METHOD

Participants

Participants were 271 children recruited through flyer distribution at two Midwestern study sites for a longitudinal study spanning preschool to elementary school. Children with diagnosed developmental, behavioral (including ADHD), or language disorders at study entry were excluded. To be eligible for the study, the primary language spoken at home had to be English. In terms of race and ethnicity, 73.43% of children reported as European American, 7.38% as Hispanic/Latino, 5.17% as African American, and 14.02% as multiracial. Participant recruitment was stratified by both sex (49.4% female) and sociodemographic risk, with 46.5% of the sample considered at-risk based on qualification for Medicaid/CHIP or by falling below the federal poverty guidelines. Descriptive statistics for sample demographics are presented in Table 1.

Table 1

Descriptive Statistics on Sample Demographics and Observed Variables for Study 1

Variable	Mean	SD	Min	Max
Age in 4 th Grade	10.1	.36	9.3	11.3
Conners-3 Inattention	50.7	11.5	41	89
Conners-3 Hyperactivity	52.7	13.8	41	90
Gender	<i>N</i>	<i>%</i>		
<i>Male</i>	137	50.6%		
<i>Female</i>	134	49.4%		
Ethnicity	<i>N</i>	<i>%</i>		
<i>European American</i>	199	73.4%		
<i>African American</i>	14	5.2%		
<i>Hispanic</i>	20	7.4%		
<i>Multiracial</i>	38	14.0%		
SES – At-Risk	<i>N</i>	<i>%</i>		
<i>Yes</i>	126	46.5%		

No 145 53.5%

Procedures

The larger longitudinal study consisted of a cohort sequential design spanning multiple time points beginning in preschool. For the current study, parents completed sleep questions at study entry when children were three years old. Behavioral EC tasks were completed in the laboratory at 4.5 years for all participants. Both parent and child visited the university laboratory to participate, with a technician administering the battery of EC tasks to the child. In the spring of 4th grade, teachers completed measures of ADHD symptoms. Teachers were mailed questionnaire packets along with a prepaid return envelope. All study procedures were approved by the Institutional Review Board at the University of Nebraska-Lincoln.

Measures

Child sleep. Sleep problems were assessed using the sleep problem questions from the Child Behavior Checklist (CBCL), a widely used caregiver-completed measure of children's emotional and behavioral functioning (Achenbach & Rescorla, 2001; Ebesutani et al., 2010). The sleep subscale is a commonly used measure of sleep issues in childhood (Gregory et al., 2011; Gregory & O'Connor, 2002), consisting of seven items measured on a three-point Likert scale (*Never/Not True, Sometimes/Somewhat True, and Often/Very True*) gauging common sleep problems (e.g., difficulty sleeping, short/long typical sleep duration). Recent research has demonstrated that the CBCL sleep problems composite score is highly correlated with well-validated measures of sleep (e.g., Children's Sleep Habits Questionnaire; Owens, Spirito, & McGuinn, 2000) and

clinical diagnosis of sleep problems (Becker, Ramsey, & Byars, 2015). Internal consistency for the sleep problem questions in the current sample was acceptable ($\alpha = .70$).

Executive control. The EC battery consisted of nine tasks reflecting the three major aspects of EC (i.e., inhibitory control, working memory, and flexible shifting). Three tasks in the battery measured working memory: *Delayed Alternation*, *Nine Boxes*, and *Nebraska Barnyard*. Four tasks gauged inhibitory control by requiring the children to inhibit a prepotent response: *Big-Little Stroop*, *Go/No-Go*, *Shape School (Inhibit condition)*, and *modified Snack Delay*. Two tasks measured flexible shifting: *Shape School (Switching Condition)* and *Trails (Switching Condition)*. See Table 2 for a brief description of each EC task.

Table 2

Description of Executive Control Tasks

EC Task	Brief Description	Source
<i>Working Memory</i>		
Delayed Alternation	Child searches for reward by choosing one of two locations on a board. The location of the reward alternates, so the child must remember the last location following a delay.	Espy et al. (1999); Goldman et al. (1971)
Nine Boxes	Child searches among nine boxes for a reward. Boxes are different colors and shapes, and the child must remember previously searched boxes to be able to find the reward in the fewest number of trials.	Adapted from Diamond et al. (1997)
Nebraska Barnyard	Child is shown locations of animal figures on a 3x3 field of boxes and then must remember the locations in order to indicate locations of animal sequences after pictures are removed.	Adapted from Hughes et al. Noisy Book task (1998)
<i>Inhibitory Control</i>		
Big-Little Stroop	Child is presented with stimuli made of smaller objects embedded within a larger objects. Child is asked to name the smaller object, which requires suppression of the larger object name.	Adapted from Kochanska et al. (2000)

Go/No-Go	Child is presented a series of fish or shark stimuli on the screen. Child is asked to press a button when a fish is shown but inhibit pressing the button when a shark is shown.	Adapted from Simpson & Riggs (2006)
Shape School – Inhibit	Child is presented with cartoon stimuli with either a happy or sad faces and is asked to name the color of the stimulus when the face is happy and to inhibit naming the color when the face is sad.	Espy (1997); Espy et al. (2006)
Modified Snack Delay	Child is seated in close proximity to a candy reward and is asked to remain still until a bell rings.	Adapted from Konchanska et al. (1996); Korkman et al. (1998)
<i>Flexible Shifting</i>		
Shape School - Switching	The child is presented with cartoon stimuli of different colors and shapes that are either wearing a hat or not. Child is asked to say the color of the stimulus when not wearing a hat and say the shape when wearing a hat.	Espy (1997); Espy et al. (2006)
Trails - Switching	Child is asked to use a stamp to mark stimuli on a page, alternating between dog and bone stimuli.	Adapted from Espy & Cwik (2004)

Child ADHD symptoms. Child ADHD symptoms were assessed using the Conners 3rd Edition Teacher Ratings Scale (Conners 3-T; Conners, Pitkanen, & Rzepa, 2011), a teacher-report measure of hyperactive-impulsive and inattentive symptoms in the classroom. Teacher-report of ADHD symptoms is highly important, because symptoms are typically more apparent in structured settings such as school classrooms (DuPaul & Stoner, 2014). The Conners-3 is a validated measure that includes updated *t*-scores based on a geographically representative normalization sample (Kollins, Epstein, & Conners, 2014). Each subscale is comprised of four-point Likert scale items, with high scores corresponding to DSM-IV diagnostic criteria for ADHD subtypes. The internal

consistency for both the hyperactivity and inattention scales were excellent (hyperactivity $\alpha = .94$; inattention $\alpha = .95$).

CHAPTER 5: ANALYTIC RATIONALE

Latent moderator analyses were used to determine if EC was a moderator of the relationship between sleep problems and ADHD symptoms. The seven sleep items were used to make a latent sleep factor, and the nine EC task scores were standardized before making the single latent EC variable. Previous research using the nine EC tasks found that preschool EC was best modeled using a single factor based on fit and parsimony (Nelson, James, Chevalier, Clark, & Espy, 2016). The fit of the single factor structure was confirmed with the current sample. Both inattention and hyperactivity were included as observed outcomes in the same model.

The two-step procedure for latent moderator analyses recommended by Maslowsky and colleagues (2015) was implemented in Mplus version 6.12 (Muthén & Muthén, 2010) using maximum likelihood. First, the measurement model that estimated the relationships between the latent predictors and observed variables was evaluated to ensure acceptable fit statistics. Second, the latent moderator (latent sleep problems X latent EC) was added to the structural equation model. Because the latent moderated structural equation (LMS) model does not provide fit statistics, a likelihood ratio test was used to compare the LMS model with the measurement model (Maslowsky, Jager, Hemken, 2015). Maximum likelihood estimation handles missing data well and allows all cases to be included in the analyses regardless of missing data. In the present study, the average percentage of missing data was 6.21%.

CHAPTER 6: RESULTS

Preliminary Analyses

Correlations were calculated to determine if there were any gender, ethnicity/race, or SES differences on the outcome variables. Gender significantly correlated with inattention and hyperactivity symptoms, but neither ethnicity/race nor SES were significantly correlated with either ADHD scale ($p > .05$). Thus, only gender was included in the subsequent models for parsimony. *A priori* power analyses were conducted to ensure that there was enough power to find significant effects, with results indicating that a sample size of 271 provided ample power to find a medium effect.

Structural Equation Model

The measurement model demonstrated excellent fit, RMSEA = .03, CFI = .96, TLI = .95, SRMR = .05. All item loadings contributing to the latent sleep problems and EC constructs were significant. The likelihood ratio test comparing the latent moderator model to the original measurement model indicated that the model with the latent moderator was the better model, $\chi^2(1) = 234.46, p < .001$. See Figure 1 for a depiction of the latent moderator model results.

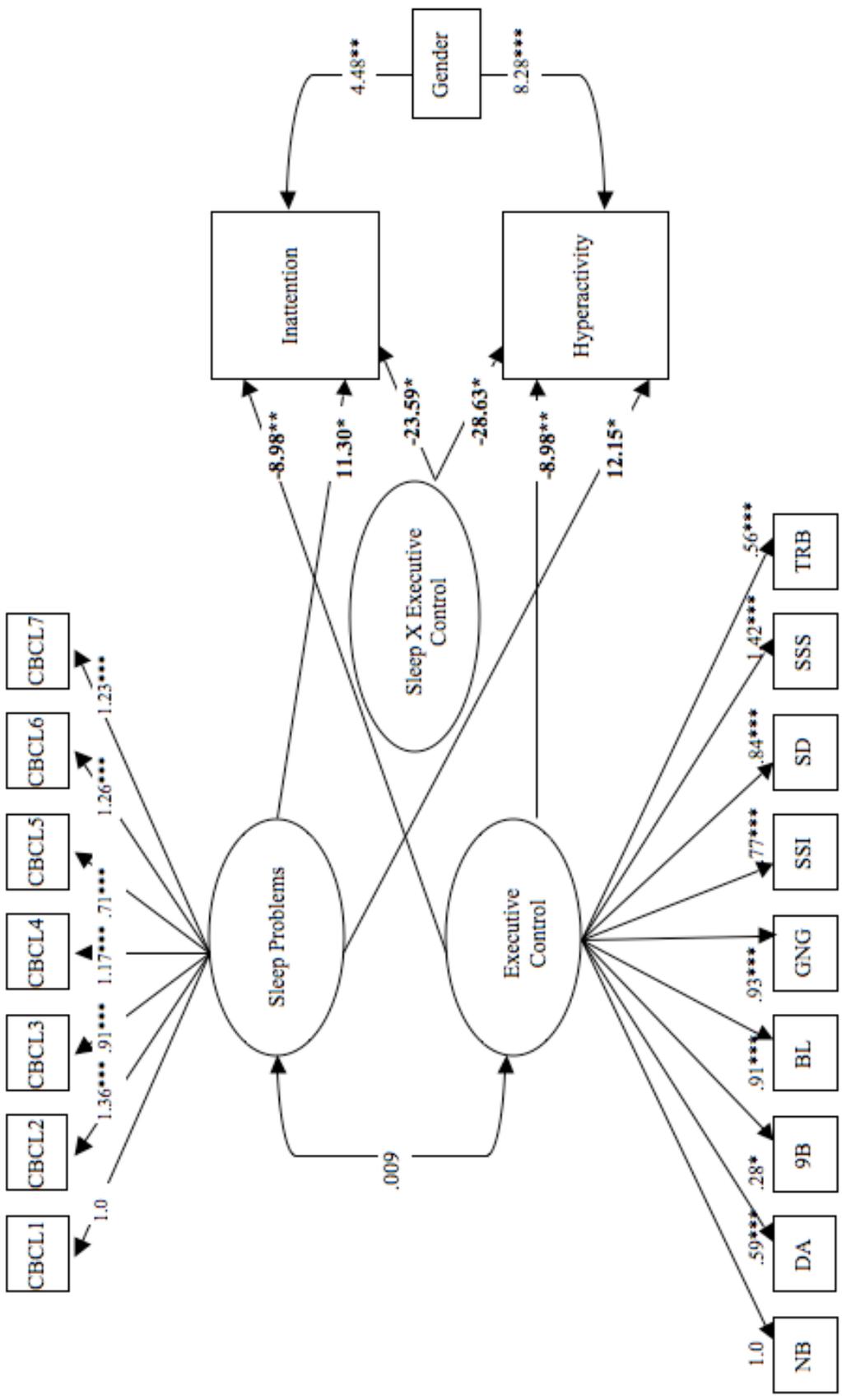


Figure 1. Results of Latent Moderated Structural Equation Model (LMS)

Notes. All results are unstandardized, because standardized results have not been developed for LMS models. * $p < .05$, ** $p < .01$, *** $p < .001$; NB = Noisy Book; DA = Delayed Alternation; 9B = 9 Boxes; BL = Big Little Stroop; GNG = Go-No-Go; SSI = Shape School Inhibit; SD = Snack Delay; SSS = Shape School Switch; TRB = Trail Making Test

Inattention. In the latent moderator model controlling for gender, sleep problems significantly predicted more inattention symptoms, $b = 11.30$, $SE = 4.34$, $t = 2.60$, $p = .009$. EC also significantly predicted inattention symptoms, such that children with worse EC had more inattention symptoms, $b = -9.95$, $SE = 2.34$, $t = -4.25$, $p < .001$. The latent sleep problems X EC moderator variable significantly predicted greater inattention, such that those with both sleep problems and EC deficits experienced more inattention symptoms than those with high EC, $b = -23.59$, $SE = 11.34$, $t = -2.08$, $p = .037$.

Hyperactivity. In the latent moderator model controlling for gender, sleep problems significantly predicted more hyperactivity symptoms, $b = 12.15$, $SE = 4.93$, $t = 2.47$, $p = .014$. EC also significantly predicted hyperactivity symptoms, such that children with worse EC had more hyperactivity symptoms, $b = -8.98$, $SE = 2.63$, $t = -3.41$, $p = .001$. The latent sleep problems X EC moderator variable significantly predicted greater hyperactivity, such that those with both sleep problems and EC deficits experience more hyperactivity symptoms than those with high EC, $b = -28.63$, $SE = 12.62$, $t = -2.27$, $p = .023$.

CHAPTER 7: STUDY 1 DISCUSSION

The current study examined early sleep and EC problems as predictors of ADHD symptoms in 4th grade. Consistent with the first hypothesis that preschool sleep problems would predict later ADHD symptoms, results showed that sleep problems at age three significantly predicted greater ADHD symptomatology in 4th grade. In support of the second hypothesis that early EC deficits would predict elementary ADHD symptoms, EC deficits at age 4.5 years predicted ADHD symptoms in 4th grade. Finally, the results indicated that EC deficits moderated the relationship between sleep problems and ADHD symptoms.

The finding that sleep problems at age three significantly predicted higher levels of inattention and hyperactivity in 4th grade builds on previous research demonstrating that sleep problems may predate symptoms of ADHD (Chervin et al., 2005; Gregory et al., 2004). By using an extended longitudinal design with children who had not yet been diagnosed with ADHD at study entry, this study was able to examine sleep problems at an early age predicting later ADHD symptoms in a non-clinical population. This study provides support for the theory that sleep problems are a risk factor for ADHD symptoms.

Results also indicated that EC deficits predicted greater ADHD symptoms, an outcome that maps onto findings in the literature of associations between EC and ADHD (Espy et al., 2011; Sadeh et al., 2002; Willcutt et al., 2005) and provides support for Barkley's (1997) theory of EC deficits underlying ADHD. This study also builds on previous studies by examining the constructs in a longitudinal design starting early in development. Moreover, the results support the theory that over time, EC becomes

increasingly important in processes related to self-regulation and behavior management (Halperin & Schulz, 2006). Thus, children with poor EC have worse behavioral control, which may exacerbate ADHD symptomatology (Rabinovitz et al., 2016).

This study also found that EC was a moderator of the relationship between sleep problems and ADHD symptoms, with findings showing that those children with both sleep problems and EC deficits experienced more ADHD symptoms than those with high EC. In other words, children with low EC may be particularly vulnerable to the effects of sleep loss. These findings are in line with results from Schneider et al. (2016), who found associations between sleep and ADHD symptoms, and that children with ADHD demonstrated worse performance on EC tasks. However, Schneider and colleagues' (2016) study was limited by a focus on associations at a single developmental time point and by a lack of mediation or moderation analyses. The current study extends the work of Schneider et al. (2016) by examining associations among sleep, EC, and ADHD at three time points in a longitudinal design and by examining EC as a moderator of the sleep-ADHD relationship.

Implications

The findings that early sleep and EC deficits predicted inattention and hyperactivity symptoms later in childhood have important clinical implications. Healthcare providers are encouraged to provide early sleep and EC interventions. First, for young children presenting with sleep problems, practitioners can provide behavioral sleep treatments such as techniques for managing bedtime resistance and education on sleep hygiene. Research has begun to demonstrate that treating sleep problems as part of routine clinical care reduces ADHD symptoms (Hiscock et al., 2015; Keshavarzi et al.,

2014). Second, healthcare providers are encouraged to assess for EC difficulties starting in preschool. For young children who demonstrate poor EC, practitioners are encouraged to recommend interventions that may improve EC. Research has demonstrated that EC is modifiable (Halperin et al., 2013; Rueda, Rothbart, McCandliss, Saccomanno, & Posner, 2005), and studies have found support for computerized interventions (Klingberg et al., 2005; Mackey, Hill, Stone, & Bunge, 2011), school-based curriculums (Diamond, Barnett, Thomas, & Munro, 2007; Raver et al., 2011; Riggs, Greenberg, Kusché, & Pentz, 2006), tae kwon do (Lakes & Hoyt, 2004), and yoga / mindfulness programs (Gould, Dariotis, Mendelson, & Greenberg, 2012; Razza, Bergen-Cico, & Raymond, 2015).

In the context of future research, these findings lay the groundwork for research on the efficacy of sleep treatments for children with ADHD by demonstrating that early sleep problems predict ADHD symptoms. Because a typically-developing sample was recruited (children with diagnosed emotional and behavioral disorders were excluded at the initial time point), this study demonstrates that the associations among sleep, EC, and ADHD symptoms are generalizable to a community sample. Sleep and EC appear to underlie the development of ADHD and together contribute to the complex constellation of predictive factors of ADHD.

Limitations and Future Directions

The current findings should be considered in the context of the study's limitations. First, the parent-report sleep measure at age three and the teacher-report ADHD measure completed during 4th grade are limited by the parent/teacher-report format. Although the Conners-3 is a reliable, valid, and widely-used measure, school

observations of ADHD symptoms may have provided richer data. Further, this study used the parent-report CBCL sleep questions to make a latent sleep scale. Although not an ideal measure of sleep duration and quality, items from the CBCL have been shown to be useful predictors of objectively assessed child sleep problems (Gregory et al., 2011). Recent research has demonstrated that the CBCL sleep problems composite score is highly correlated with well-validated measures of sleep (e.g., Children's Sleep Habits Questionnaire; Owens, Spirito, & McGuinn, 2000) and clinical diagnosis of sleep problems (Becker, Ramsey, & Byars, 2015). Although actigraphy may provide a more accurate measurement of sleep duration, the CBCL is able to identify sleep problems and symptoms (Lewandowski, Toliver-Sokol, & Palermo, 2011). Future research is needed to replicate the current findings using a combination of subjective and objective measurement of sleep. Second, ADHD symptoms were assessed in a community sample with a teacher-report rating scale rather than conducting a thorough assessment of whether children had diagnosable, clinical ADHD. Future researchers may choose to examine the interaction of sleep and EC in subsamples of youth diagnosed with ADHD to build on the current study that examined the constructs in a community sample. Third, the current study was unable to examine the reverse association that early ADHD symptoms would predict later sleep problems. Future researchers are encouraged to examine the constructs at multiple time point and to control for ADHD symptoms in early development to add rigor.

In practice, it is difficult to distinguish between inattentiveness that derives from sleepiness and inattentiveness that is symptomatic of ADHD (Owens, 2005). The results of the current study indicate that when examined longitudinally, sleep problems at three

years old predict greater teacher-rated inattention and hyperactivity symptoms in 4th grade. It could be that sleep problems are a risk factor for inattention and hyperactivity years later, or that concurrent sleep problems result in ADHD-like symptoms, or that early sleep problems are a prodromal sign of ADHD, or that sleep loss results in impaired functioning in the prefrontal cortex leading to ADHD symptoms. More research is needed on sleep, EC, and ADHD to elucidate the complex relationships at work.

Despite the limitations of the current study, there are many strengths of the research design. First, the extended longitudinal design allows for more confidence in the findings that sleep problems at three years and EC problems at 4.5 years precede symptoms of ADHD. To examine the temporal sequence of the developmental trajectory, sleep, EC, and ADHD symptoms were assessed at different time points. Second, because sleep and EC change across development, children were assessed within two weeks of turning three and 4.5 years old to control for age. Third, teachers reported on symptoms of ADHD, as inattention and hyperactivity are typically more apparent in structured settings such as school classrooms (DuPaul & Stoner, 2014). Finally, rather than using parent-rated EC, nine developmentally appropriate behavioral tasks were used to assess the various components of EC.

Conclusions

In conclusion, this study found that very early sleep problems at three years old and EC deficits in preschool predicted ADHD symptoms in elementary school. EC moderated the relationship between early sleep problems and ADHD symptoms, such that children with both sleep problems and poor EC were particularly susceptible to

experiencing ADHD symptoms. It is recommended that healthcare providers assess and treat early sleep problems and EC problems.

CHAPTER 8: STUDY 2 INTRODUCTION

Study 2 was a pilot of a brief sleep treatment for children presenting to an outpatient clinic for emotional and behavioral disorders. To develop the framework for Study 2, first, the effectiveness of current behavioral and medical treatments for both externalizing and internalizing disorders will be reviewed. Second, the very brief literature on treating sleep within child psychopathology will be reviewed. These literature reviews will demonstrate that sleep appears to be an important contributor to various forms of child psychopathology and is an appropriate, yet overlooked, target for treatment.

Treatment of Externalizing Problems

Current evidence-based treatments for ADHD and externalizing problems such as ODD, CD, noncompliance, and aggression fall into the following two categories: behavioral and medication. Behavioral treatments include parent training, educational interventions, and summer treatment programs. Behavioral parent training uses strategies such as contingency management and differential attention to decrease disruptive behavior at home and reduce parent-child conflict (for an example see Barkley, 2014). Educational interventions focus on reducing disruptive classroom behavior and improving academic performance. Summer treatment programs typically combine parent training, medication, support groups, and individual counseling in an intensive program (for an example see Fabiano, Schatz, & Pelham, 2014). Medications that are commonly used to treat ADHD but are also prescribed for other externalizing disorders include

stimulants, noradrenergic medications, tricyclic antidepressants, and hypertension medications. Stimulant medications are the most commonly used medications. They can be classified as short-, medium-, or long-acting. The two most prescribed formulas are methylphenidate (e.g., Ritalin™, Concerta™, Daytrana™) and amphetamine (e.g., Adderall™, Dexedrine™, Vyvanse™). Stimulants are effective in increasing sustained attention and improving inhibition (Charach, Ickowicz, & Schachar, 2004). However, stimulants only work as long as they are in the children's bodies and do not "cure" ADHD (Barkley, 2014).

A review of the largest randomized control trial for ADHD treatment allows for examination of effectiveness of behavioral treatments, stimulant medications, and combined treatment. The Multimodal Treatment Study of Children with ADHD (MTA Study) was a multi-site clinical trial comparing the effectiveness of medication, behavioral treatment, and combined treatment (MTA Cooperative Group, 1999). Children randomized to the medication management group received stimulant medication every day. In the behavioral treatment group, parents attended 35 sessions of parent training, clinicians visited the school up to ten times, and children attended an eight week summer treatment program. In the combined treatment group, children took medication and received the behavioral treatment. A typical community treatment condition was also included as a control group. The typical community treatment group included 66% of children who received medication. All children showed improvements over the 14 months of treatment. In treating ADHD symptoms and common co-occurring problems (noncompliance, ineffective parenting, and poor social skills), combined treatment performed best, then medication, behavioral treatment, and routine community treatment

in that order (Conners et al., 2001; Swanson et al., 2001). For two subgroups of children who had anxiety or had parents on public assistance, behavioral treatment was just as effective as medication (Jensen et al., 2001). At the two year follow-up, combined treatment continued to outperform the other treatments (MTA Cooperative Group, 2004). At the 36 month follow-up, the differential effects of treatment had disappeared (Jensen et al., 2007). One caveat to the follow-up findings is that some children who received only behavioral treatment began taking stimulant medications during the 36 months after the initial study ended, and some children discontinued stimulants.

The MTA study also provided a unique opportunity to examine normalization rates with different treatments. The MTA Study defined the normalization rate as the percentage of children at the end of treatment who had an overall symptom-severity level less than one when parent- and teacher-rating scales were averaged. Essentially, the normalization rate refers to the percentage of children who would no longer be diagnosed with ADHD after treatment. The MTA Study had a normalization rate of 34% for behavioral treatment and 56% for stimulants (while the stimulant is active in the child's body), which is surprisingly low for frontline treatments. The typical community treatment had a normalization rate of 25%. Perhaps, treatments could have higher normalization rates by targeting the brain-based impairments that underlie the disorder.

While the MTA study demonstrated that combined behavioral and stimulant treatment is effective, the American Academy of Pediatrics (AAP) have included recommendations for broadening the scope of assessment and treatment beyond the targets in the MTA study. In recognition of the high comorbidity of ADHD and sleep difficulties, the AAP included recommendations for the direct assessment of sleep

problems in their guideline for the treatment of ADHD (AAP, 2011). Given the relatively high rate of sleep problems in children with ADHD, clinicians and physicians are likely to encounter comorbid sleep impairments during the course of treating ADHD. Although there are many effective behavioral treatments for pediatric insomnia (Meltzer & Mindell, 2014), there have been no medications approved for pediatric insomnia in the US. However, behavioral sleep treatments are not implemented for children with ADHD, and instead, physicians commonly prescribe sedatives to treat insomnia (Owens, Rosen, & Mindell, 2003).

Treatment of Internalizing Disorders

Treatments for anxiety often address distorted information processing, physiological reactions, and excessive avoidance (Kendall, Furr, & Podell, 2010). Evidence-based treatments for anxiety include behavioral treatment, cognitive-behavioral therapy (CBT), family therapy, and medications. The evidence-based behavioral treatment for anxiety is exposure and response prevention. There are multiple ways of conducting exposures, but the same principles of extinguishing the response to a feared stimuli apply. The child is exposed to the anxiety-provoking situation or object and he/she remains in the setting despite the fear. Eventually, the fear response is extinguished (Chorpita & Southam-Gerow, 2006). In behavioral treatment and CBT, children may also learn problem solving and coping skills such as relaxation and deep breathing before exposures. Normalization rates for a popular behavioral anxiety treatment lasting 16 sessions (Coping Cat) range from 50% to 73% across randomized clinical trials (Flannery-Schroeder & Kendall, 2000; Kendall, 1994; Kendall et al., 1997). Selective serotonin

reuptake inhibitors (SSRIs) may be prescribed for anxiety, but there have been very few effectiveness trials for SSRIs in children with anxiety (Reinblatt & Riddle, 2007).

Treatments for child depression include behavior therapy, cognitive therapy, CBT, and medication. Behavior therapy focuses on increasing behaviors that lead to positive reinforcement and decreasing punishing aspects of the environment (Birmaher & Brent, 2007). Children may also learn coping skills in behavior therapy (Stark, Streusand, Krumholz, & Patel, 2010). Cognitive therapy focuses on altering depressogenic beliefs and thought patterns and may be difficult for young children (Stark et al., 2010). For children in the United States, SSRIs are often used to treat depression (only fluoxetine is approved by the Food and Drug Administration for use in children). However, SSRIs may have unwanted side effects in children (Goodyer et al., 2007). For youth with MDD, a large randomized control trial found that a combination of CBT and fluoxetine led to a faster decline in depression symptoms and outperformed medication alone at follow-up (Kratohvil et al., 2006). Moreover, the combination of CBT with medication had the highest normalization/remission rate (37%) compared to the other conditions (23% with Fluoxetine and 16% with CBT; Kennard et al., 2006), indicating that combined treatment is currently the most effective option for youth with depression.

Conceptual Framework

Current treatments for externalizing and internalizing disorders have relatively low normalization rates, indicating that more effective treatments need to be developed and tested. The rationale for how treating sleep would improve emotional and behavioral functioning is based on the conceptualization of externalizing disorders reflecting EC impairment and internalizing disorders reflecting impairments in brain-based emotional

regulation. The proposed mechanism of change for both types of disorders is through improved regulation and EC. Given the complex reciprocal relationships between sleep and mental health symptoms, improving sleep may be one way to address brain-based impairments in regulation. Sleep is a modifiable behavior that can have a critical impact on attention, aspects of inhibition, anxiety, depression, and oppositional behaviors. Study 2 is grounded in theory and research that suggests that change in one regulatory system (i.e., sleep) can impact another (i.e., emotional reactivity, impulsivity, inattention, noncompliance) by impacting the PFC, amygdala, and related brain systems.

Sleep Treatment Studies

Based on information on the underlying physiological correlates of psychopathology and sleep impairments, more insightful treatments can be offered that include a focus on sleep improvement. However, very little research has been conducted in this area. Keshavarzi and colleagues (2014) designed a study to treat sleep problems to improve functioning in children with ADHD. The study included 40 eight to 13-year-olds in Iran who had no comorbid disorders (i.e., oppositional defiant disorder, conduct disorder, anxiety, nor depression). All children in the study took stimulant medication throughout treatment. Parents in the sleep-treatment condition received 12 weeks of sleep hygiene education in a group format. Sleep treatment significantly improved children's sleep. More importantly, sleep treatment improved children's physical well-being, psychological well-being, family relationships, social support, school environment, and social acceptance. The study did not include measures of children's core ADHD symptoms such as inattention and hyperactivity. The study also did not use objective measures of sleep such as actigraphy (Keshavarzi et al., 2014). Despite the

limitations of the study, the findings indicate that a lengthy sleep treatment can improve sleep and overall functioning of children with ADHD.

A pilot study conducted by Haynes and colleagues (2006) used a six-session behavioral sleep treatment with adolescents with substance use issues. After substance use treatment, the adolescents received sleep treatment. Adolescents had reduced aggressive thoughts and behaviors after the sleep treatment. Similarly to Keshavarzi (2014), this study did not use objective measurement of sleep and tested a long form of behavioral sleep treatment. It is expected that even a short-form of behavioral sleep treatment would be effective and would be easier to implement in routine clinical care. This study also investigated the relationship between aggression and sleep in adolescents, whereas the proposed study seeks to demonstrate the association between psychopathology and sleep in a younger, middle childhood sample.

Hiscock and colleagues (2015) expanded on an initial pilot investigation of brief sleep treatment for children with ADHD and sleep problems (Sciberras, Fulton, Efron, Oberklaid, & Hiscock, 2011) to examine the sleep treatment in a sample of 244 children (113 children actually receiving sleep treatment) ages five to 12 years with comorbid ADHD and sleep problems. Psychologists or a pediatrician provided information about sleep cycles, sleep hygiene, and behavior management techniques for sleep during two in-person sessions. At follow-up assessments conducted three months and six months after the sessions, children had fewer sleep problems, reduced ADHD symptoms, and improved working memory. Objective measurement of sleep with actigraphy with a subset of the sample indicated that children slept longer after the intervention. Hiscock et al.'s (2015) study only included children who had both sleep problems and ADHD. The

current study (Study 2) expands on the work of Hiscock and colleagues (2015) by providing sleep intervention for children with a variety of presenting mental health problems. The current study also assessed the *rapid* improvement of symptoms following sleep treatment, while Hiscock and colleagues (2015) examined improvements at three- and six-month follow-ups.

Additionally, our research group has conducted an initial study investigating the effectiveness of a brief sleep intervention for noncompliance (Nelson, Van Dyk, McGinnis, Nguyen, & Long, 2016). The study included 54 children ages two to 16 who received outpatient behavioral treatment for noncompliance. Findings indicated that a brief sleep treatment significantly improved parent-report of sleep. More importantly, sleep treatment significantly improved compliance. However, the study did not include objective or validated measurements. Instead, parents verbally rated key problems on a one to seven scale of problem severity each week at the beginning of treatment. Despite the lack of validated measures, Nelson and colleagues (2016) found a pre-post effect size several times larger than those published in the literature for behavioral treatments and medications ($d = 3.25$). Sleep treatment improved noncompliance in the first few weeks of treatment, and the authors noted the importance of rapid improvement in symptoms to prevent drop-out from treatment. Moreover, compared to the typical length of treatment in the literature, children needed fewer sessions of behavioral treatment after the sleep intervention.

Research examining the effectiveness of incorporating sleep treatment into child emotional/behavioral treatment is scant, and the existing studies, while promising, have notable limitations. In particular, the little research that has been published has rarely

included objective measurement of sleep, measured child emotional/behavioral symptoms, assessed EC, or included children with internalizing disorders. Additionally, testing a brief sleep treatment at the beginning of behavioral treatment is rare.

CHAPTER 9: PRIMARY PURPOSE AND RESEARCH HYPOTHESES

In light of the minimal research examining sleep interventions in child mental health treatment and the notable limitations of the few existing studies, the current study aims to test the effectiveness of a brief sleep treatment at the beginning of treatment for emotional/behavioral disorders. Study 2 addresses gaps in the literature through the implementation of a brief behavioral treatment of sleep problems in children presenting for treatment of externalizing and internalizing disorders. This study uses objective measure of sleep (i.e., actigraphy) and validated measures of behavior, emotion, and EC at multiple time points to evaluate therapy outcomes.

There were four main aims of the present study. First, this study aimed to examine the effect of the sleep treatment. We hypothesized that objectively measured sleep duration and efficiency would improve, and that emotional/behavioral symptoms and EC would show an initial decline after sleep treatment. We based this hypothesis on theory and experimental research linking sleep with emotional/behavioral and cognitive functioning. Second, this study aimed to examine the total change in emotional/behavioral symptoms after combined sleep and behavioral treatment. We expected that children's symptoms would continue to decline after combined treatment and show a significant pre-post difference. This hypothesis was based on studies indicating the effectiveness of current behavioral treatments for internalizing and externalizing disorders. The third aim was to compare the total change in symptoms

after combined treatment (sleep and behavioral treatment) to the effects in the literature for behavioral treatments alone. We hypothesized that the pre-post effect size would be larger than effect sizes currently found in the literature for behavioral treatments (e.g., $d = .63$ for ADHD; Fabiano et al., 2009). This hypothesis was based on preliminary results from Nelson et al. (2016) demonstrating that combined brief sleep treatment and parent training on children's noncompliance had a very large effect size ($d = 3.25$), much larger than typical results in behavioral treatment. Moreover, we expected that children receiving combined sleep and behavioral treatment would have higher normalization rates than rates in the literature for current behavioral treatments. The fourth aim of the study was to examine changes in EC as a potential mechanism of treatment effects for sleep interventions and child psychopathology symptoms. We hypothesized that change in EC would mediate the relationship between treatment and symptom reduction, such that sleep would improve EC and greater EC would improve mental health symptoms. This hypothesis was based upon a series of research studies demonstrating that sleep impacts EC and EC influences emotional reactivity and behavioral functioning.

In summary, current behavioral treatments may be improved upon to be more effective for more children. Sleep is a promising yet untapped target with the potential to improve treatment outcomes for a wide range of child psychopathology. A sleep module, if found to be effective, would fit well within the modular approaches that are becoming popular and well-supported. There has been a relatively recent shift in the field of pediatric psychology and in practice to a modular approach to treatment (Weisz et al., 2012). Modular approaches tend to outperform traditional evidence-based treatments and

may be easier for clinicians to use in practice (Weisz et al., 2012). Although there are treatment modules for behavioral treatment of anxiety, depression, and ADHD, there has yet to be a module for sleep problems. This study tested the effectiveness of a brief sleep module in a behavioral health clinic.

CHAPTER 10: METHOD

Procedures

Study 2 is an evaluation of sleep treatment that was administered by psychologists at the Boys Town South Florida Behavioral Health Clinic. The treatment involved one to three sessions of sleep treatment followed by behavioral treatment of mental health symptoms (e.g., noncompliance, anxiety, inattention, impulsivity). See Appendix A for a sample treatment guide. At the beginning of the first session, the therapist administered the CBCL and the Behavior Rating Inventory of Executive Function (BRIEF) for children ages six to 11 years in the clinic to establish a baseline of mental health symptoms and EC. The first session was only an intake with no intervention. If the child met the inclusion and exclusion criteria during the initial session (described below), the clinician explained the study and obtained consent from the parent and written assent from the child. Parents provided authorization for the Boys Town clinicians to release the baseline measures, medication information, and dates of treatment for the research study. The parent then completed one brief page of demographic information and an additional measure of mental health functioning, the Brief Problem Monitor (BPM). To monitor weekly change, participants completed the BPM each week of treatment.

After completion of the baseline measures, parents and youth were given detailed instruction on how to wear the actigraph. Youth were asked to wear the actigraph all day

and night for one week to measure baseline sleep. During this week, the parents completed simple sleep diaries of when the child went to bed and woke up. The participant returned the actigraph at their second therapy session.

Next, the participants received one to three weeks of behavioral sleep treatment by a Boys Town clinician employed at the Behavioral Health Clinic. The sleep treatment was informed by expert recommendations for treating sleep problems (Hamilton, 2009; Kuhn & Elliot, 2003). The treatment included psychoeducation on bedtime routines, sleep hygiene, and behavioral strategies (e.g., contingency management) in response to problematic behaviors around bedtime. The sleep protocol also included a session to extinguish nighttime fears and worries if the child presented with anxiety that interfered with sleep. The protocol was developed with the intention of being a brief treatment that could be used in the context of psychotherapy treatment for externalizing/internalizing disorders. The protocol was developed by J. Christopher McGinnis, PhD while at the McGinnis Psychology Group in Fort Meyers, Florida.

After sleep treatment, the caregivers completed the same measures of mental health and EC as at baseline (CBCL and BRIEF) to determine the effectiveness of the sleep treatment. Youth were asked to wear an actigraph wristwatch all day and night for one week to measure change in sleep after treatment. The participants then received standard behavioral treatment for the presenting problems (e.g., exposure and response prevention for anxiety; behavioral activation for depression, etc.). Children attended an average of 8.69 therapy sessions (range 1 - 17). At the end of treatment, the participants completed the CBCL and the BRIEF a final time. Participants were compensated with \$10 for initial measures, \$10 for post-sleep measures, \$15 for final measures, \$1/day for

adherence to actigraph protocol, and \$1 bonus for complete measures all paid out at study completion to the parent/legal guardian (total of \$50). All study procedures were approved by the Institutional Review Boards at the University of Nebraska-Lincoln and Boys Town National Research Institute.

Participants

Participants included 13 youth between the ages of six and 11 ($M = 8.08$ years, $SD = 1.32$) with emotional and/or behavioral problems and their parent or legal guardian. Parents and children who presented to the Boys Town South Florida Behavioral Health Clinic for behavioral health services were recruited to participate. To be eligible to participate in the study, parents and children had to be able to speak and understand English and children had to be between the ages of six and 11. Exclusion criteria included the following: (1) Children with a previously diagnosed cognitive/developmental disability (e.g., severe language delay) and (2) Children with autism or a pervasive developmental disorder. These exclusion criteria were necessary as we were interested in the cognitive mediators of the effect of sleep treatment on emotional/behavioral problems. The conditions in the exclusion criteria would present as confounds to investigating the cognitive mediators of the effect of sleep treatment on emotional/behavioral disorders.

See Table 3 for demographic information. In terms of gender, 46.2% were female. The participants were in grade school with 16.7% in first grade, 33.3% in second grade, 25% in fourth grade, and 8.3% in fifth grade. The sample was ethnically diverse with 61.5% of children reported as European American, 30.8% as Hispanic/Latino, and 7.7% as African American. Mothers reported their highest level of education as the

following: 15.4% high school or equivalent, 7.7% vocational or technical school, 46.2% some college, 7.7%, bachelor's degree, 7.7% master's degree, 7.7% doctoral degree, and 7.7% professional degree. Fathers' highest level of education was the following: 30.8% high school or equivalent, 7.7% vocational or technical school, 15.4% some college, 15.4%, bachelor's degree, 15.4% master's degree, 7.7% other (e.g., pilot), and 7.7% professional degree. See Table 4 for prescription medication information. Families were instructed to hold medications constant unless instructed by the primary care physician/psychiatrist to change medication regimen.

Table 3

Descriptive statistics on sample demographics for Study 2

Demographic Variable	N	%	Mean	SD	Min	Max
Age			8.08	1.32	6	11
Gender						
<i>Male</i>	7	53.8%				
<i>Female</i>	6	46.2%				
Ethnicity						
<i>European American</i>	8	61.5%				
<i>African American</i>	1	7.7%				
<i>Hispanic</i>	4	40.8%				
Mother's Education						
<i>High School or Equivalent</i>	2	15.4%				
<i>Vocational/Tech School</i>	1	7.7%				
<i>Some College</i>	6	46.2%				
<i>Bachelor's Degree</i>	1	7.7%				
<i>Master's Degree</i>	1	7.7%				
<i>Doctoral Degree</i>	1	7.7%				
<i>Professional Degree</i>	1	7.7%				
Father's Education						
<i>High School or Equivalent</i>	4	30.8%				
<i>Vocational/Tech School</i>	1	7.7%				
<i>Some College</i>	2	15.4%				
<i>Bachelor's Degree</i>	2	15.4%				
<i>Master's Degree</i>	2	15.4%				
<i>Professional Degree</i>	1	7.7%				

Other 1 7.7%

Table 4

Prescription medication information for participants in Study 2

Baseline Medications	<i>N</i> Taking Medication	Post-Sleep Treatment Medications	<i>N</i> Taking Medication
Adderall XR	1	Adderall XR	0
Concerta	1	Concerta	2
Vyvanse	3	Vyvanse	4
Methylphenidate	1	Methylphenidate	1
Quillivant XR	1	Quillivant XR	1
Tenex	1	Tenex	0
Clonidine	1	Clonidine	1
Strattera	0	Strattera	1
Melatonin	2	Melatonin	2
Risperdal	1	Risperdal	1
Abilify	1	Abilify	1
Cogentin	1	Cogentin	0
No Medications	5	No Medications	4

Notes. Six children had no changes in their medications across treatment. One child switched from one medication to a similar medication. One child who had no prescriptions at baseline started taking medication during Study 2. One child changed medications about once a month.

Twenty participants agreed to participate in the study. Thirteen received sleep treatment and completed baseline and post-sleep rating scales (65%). Only two families completed the final BRIEF and CBCL rating scales at the third time point (15.4%), but eight children had BPMs for the last session they attended following the initiation of behavioral treatment of the presenting problem (61.5%). Ten children wore the

actigraphs at both time points (76.9%). One child wore the actigraph at baseline but did not return it after sleep-treatment. A second child refused to wear the actigraph, and a third child lost the actigraph at school. Of the children not included in the analyses (35%), one case closed due to the discovery of a brain tumor, three participants dropped out of therapy before receiving sleep treatment, two received sleep treatment but did not return for additional sessions, and one withdrew consent early.

Measures

Sleep measurement. *Actigraphy.* Objective measurement of child sleep duration and quality was measured using actigraph wristwatches (Actigraph Corporation), which were worn continuously for one week from the first therapy session to the second (baseline sleep) and for one week after sleep treatment is complete. The parents completed simple sleep diaries of when the child went to bed and woke up to set the parameters of the actigraphy algorithms (100% completion rate). The actigraphs were waterproof and, therefore, were able to be worn continuously, minimizing the chance for lost data. Research supports that actigraphs provide valid and reliable measurement of child sleep (Dayyat, Spruyt, Molfese, & Gozal, 2011; Werner et al., 2014). Actigraphy was used, because it is an objective measure of children's sleep that is more accurate than parent-report alone (Werner et al., 2014). Actigraphy is ideal, because, unlike PSG, it non-intrusively captures regular sleep patterns in the natural environment. It also allows for extended measurement of sleep, unlike PSG which is typically implemented during only one or two nights. Sadeh (2015) recommends that actigraphy be used in intervention studies due to its non-intrusive ability to collect quality data across time.

Child behavioral/emotional symptoms. *The Child Behavior Checklist.* The CBCL is a 113-item checklist that measures the emotional and behavioral problems of children aged six through 18 (Achenbach & Rescorla, 2001). The CBCL is a well-established and comprehensive measure of child symptoms. Parents reported the problems that their children have by responding to items using a 3-point response scale including Never/Not True, Sometimes/Somewhat True, and Often/Very True. Raw scores on the CBCL were then converted to *t*-scores for standardization purposes. The CBCL has demonstrated excellent internal reliability (coefficients ranging from .76 to .92; Achenbach & Rescorla, 2001). Parents completed the CBCL at three time points (baseline, post-sleep treatment, and post-behavioral treatment).

The CBCL composite scores that were included in analyses are Total Problems, Externalizing Behavior Problems, and Internalizing Problems. The empirically based syndrome scales that were included in the analyses are Social Problems, Attention Problems, Rule-Breaking Behavior, Aggressive Behavior, Anxious/Depressed, and Withdrawn/Depressed. The DSM-oriented scales that were included in analyses are Affective Problems, Anxiety Problems, ADHD, ODD, and Conduct Problems.

Brief Problem Monitor. Each session the parents were asked to complete the BPM (Achenbach, McConaughy, Ivanova, & Rescorla, 2011). The BPM is a brief version of the CBCL and an ideal evidence-based tool for weekly assessment. The BPM takes about 2 minutes to complete, minimizing participant burden. The BPM provides standardized scores for Internalizing, Externalizing, Attention Problems, and Total Problems, corresponding to major scales on the CBCL. This measure provides a convenient, valid, and broad assessment of weekly mental health symptoms via parent

report. A recent study examining the validity and reliability of the BPM demonstrated that the BPM subscales have high correlations with the corresponding CBCL subscales (Piper, Gray, Raber, & Birkett, 2015). The BPM scales were sensitive and correctly identified children's symptoms that coincided with previous diagnoses (Piper et al., 2015). The authors argue that their findings provide strong support for the validity and reliability of the BPM (Piper et al., 2015)

EC. *Behavior Rating Inventory of Executive Function.* The BRIEF (Gioia, Isquith, Guy, & Kenworthy, 2000) assesses EC abilities, which we hypothesized would be an important mechanism for how sleep treatment is correlated with mental health symptoms. The BRIEF is an 86-item parent-report measure that uses a 3-point likert-scale including Never, Sometimes, and Often as response options. The BRIEF is normed for children ages five to 18 years old. The BRIEF has demonstrated excellent internal reliability (Cronbach alpha coefficients ranging from .80 to .98; Gioia et al., 2000). Test-retest reliability correlations across clinical scales was .81 in a previous study (Gioia et al., 2000). Parents completed the BRIEF at three time points (baseline, post-sleep treatment, and post-behavioral treatment).

The BRIEF yields the following eight subscales which combine to form the Global Executive Composite: Inhibit, Shift, Emotional Control, Initiate, Working Memory, Plan/Organize, Organization of Materials, and Monitor. Each of the scales was included in analyses examining immediate change in EC and total change in EC across treatment. The BRIEF Global Executive Composite was included in analyses as the primary mediator. The subscale scores were included as additional mediators to examine the different aspects of EC separately. For one child, only the Shift, Initiate, and

Organization of Materials scales were computed due to missing item responses on the second page of the BRIEF at baseline.

CHAPTER 11: ANALYTIC RATIONALE

Preliminary Analysis Plan

First, an *a priori* power analysis was conducted. Second, descriptive statistics of all study variables were calculated. Correlations were calculated between demographic variables and treatment outcomes (14 CBCL scores, four BPM scores, and the composite BRIEF scores across each time point). Next, demographic variables that were statistically significantly correlated with treatment outcomes were included in subsequent models as control variables.

Analysis Plan for Primary Research Questions

Aim 1: Immediate effect of sleep treatment. To determine the immediate effect of sleep treatment for children presenting for treatment across internalizing and externalizing disorders, a series of repeated measures ANOVAs were conducted in SPSS version 23 to compare the mean change in sleep and externalizing / internalizing symptoms from baseline to immediately after sleep treatment.

Immediate change in sleep. Children's actigraphy scores from baseline to post-sleep treatment were analyzed with repeated measures ANOVAs to determine if sleep treatment objectively improved children's sleep duration and sleep efficiency.

Immediate change in total psychopathology symptoms. A repeated measures ANOVA was conducted using the CBCL Total Problems scale at baseline and post-sleep treatment. For analyses using the BPM, the dependent variable was Total Problems.

Immediate change in externalizing problems. The following CBCL scales were entered into repeated measures ANOVAs from baseline to post-sleep treatment: Externalizing Problems, Attention Problems, ADHD, ODD, Conduct Problems, Aggressive Behavior, Rule-Breaking Behavior, and Social Problems. For analyses using the BPM, the dependent variables was Externalizing Problems and Attention Problems.

Immediate change in internalizing problems. The following CBCL scales were entered into repeated measures ANOVAs from baseline to post-sleep treatment: Internalizing Problems, Anxious/Depressed, Withdrawn/Depressed, Affective Problems, and Anxiety Problems. For analyses using the BPM, the dependent variable was Internalizing Problems.

Immediate change in executive control. The following BRIEF scales were entered into repeated measures ANOVAs from baseline to post-sleep treatment: Inhibit, Shift, Emotional Control, Initiate, Working Memory, Plan/Organize, Organization of Materials, Monitor, and Global Executive Composite.

Aim 2: Total effect of all treatment. To examine the total change in emotional/behavioral symptoms after combined sleep and behavioral treatment, a series of repeated measures ANOVA analyses were conducted in SPSS version 23 to compare the mean change in psychopathology symptoms from baseline to the end of all treatment (combined sleep and behavioral treatment). Only two parents completed the final BRIEF and CBCL measures at the end of treatment. However, eight children had BPMs for the last session that they had attended. Thus, the change in BPM ratings was examined to compare baseline ratings to the end of all treatment ratings. Multilevel

modeling (MLM) was conducted in SAS version 9.3 to examine the change in psychopathology across treatment. The BPM scores across therapy sessions were entered into the longitudinal models for all 13 participants.

Total change in psychopathology symptoms. The BPM Total Problems score from baseline and from the final therapy session were entered into a repeated measures ANOVA. Multilevel modeling was conducted in SAS version 9.3 to examine the change in BPM Total Problems scores across treatment. Because children attended different numbers of therapy sessions, maximum likelihood estimation was used as it handles missing data well (Enders, 2010). Time was entered as the predictor of BPM Total Scores to examine trajectories of change. An unstructured covariance matrix was used.

Total change in externalizing problems. The BPM Externalizing Problems and Attention Problems were entered into repeated measures ANOVAs from baseline to the end of *all* treatment. Gender was included as a covariate in the analyses with Attention Problems as the dependent variable, because gender was significantly correlated with Attention Problems at post-sleep and the end of all treatment. Multilevel modeling was conducted in SAS version 9.3 to examine the change in BPM Externalizing Problem and Attention Problem scores across treatment. Time was entered as the predictor to examine trajectories of change, maximum likelihood estimation was used, and an unstructured covariance matrix was specified.

Total change in internalizing problems. The BPM Internalizing Problems was entered into a repeated measures ANOVA from baseline to the end of *all* treatment. Multilevel modeling was conducted in SAS version 9.3 to examine the change in BPM

Internalizing Problem scores across treatment. Time was entered as the predictor to examine trajectories of change, maximum likelihood estimation was used, and an unstructured covariance matrix was specified.

Analysis Plan for Secondary Research Questions

Aim 3: Comparison of combined treatment to effects in the literature. Aim 3 was to compare the total change in symptoms after combined treatment (sleep and behavioral treatment) to the effects in the literature for behavioral treatments alone.

Comparison of effect sizes. The effect sizes for pre-post treatment change on the CBCL and BPM Total Problems scales were calculated. Cohen's d (i.e., the mean difference divided by the pooled variance) was calculated as the effect size. The effect sizes for the current study were compared to $d = .63$ for behavioral treatment ADHD (Fabiano et al., 2009) and $d = .34$ for behavioral or CBT treatment of depression (Weisz, McCarty, & Valeri, 2006).

Comparison of normalization rates. First, the normalization rates of the CBCL Total Problems scale pre-post sleep treatment and the BPM Total Problems scale pre-post sleep treatment and pre-post all treatment were calculated by examining the number of participants who had BPM scores less than 65 after treatment. Second, the normalization rates in the current study were compared to those published for behavioral treatments (e.g., MTA study; Coping Cat; TADS).

Aim 4: Executive control mediating sleep-psychopathology. To examine changes in EC as a potential mechanism of treatment effects for sleep intervention and child psychopathology symptoms, two mediation analyses were conducted using bootstrapping and difference scores across treatment. The sleep duration difference score

from baseline to post-sleep treatment was entered as the independent variable. The BRIEF Global Executive Composite difference score was entered as the mediator. CBCL and BPM difference scores were entered in separate models as the dependent variable. Direct and indirect paths were calculated to determine mediation.

CHAPTER 12: RESULTS

Preliminary Analyses

A priori power analysis. Based on preliminary results from Nelson et al. (2016) examining combined brief sleep treatment and parent training on children's noncompliance, a pre-post treatment effect size of $d = 3.25$ was converted to an effect size of $r = .85$ to be used in the power analysis. Based on an effect size of $r = .85$, only 11 participants would be necessary to have 80% power to detect large effects. With a more conservative estimate of anticipated effect sizes, the inclusion of 20 participants provides 80% power to detect an $r = .55$.

Descriptive statistics and correlations. Descriptive statistics of sleep variables are portrayed in Table 5, and descriptive statistics for emotional/behavioral and EC variables are presented in Table 6. Correlations were calculated between demographic variables and treatment outcomes (14 CBCL scores, four BPM scores, and the composite BRIEF scores across each time point). Gender significantly correlated with the CBCL's Attention Problems scale at baseline ($r = .60, p = .031$) and post-sleep treatment ($r = .64, p = .026$). Gender also correlated with BPM's Attention Problems scale at the second session ($r = .66, p = .013$) and third session ($r = .70, p = .011$) indicating that parents rated girls as experiencing more attention problems. Because gender was statistically

significantly correlated with the BPM Attention Problems, gender was included in subsequent Attention Problem models as a control variable.

Table 5

Descriptive statistics for baseline and post-sleep treatment sleep variables

Baseline	<i>N</i>	Mean	Median	<i>SD</i>	Min	Max
Efficiency (%)	11	80.16	79.11	8.84	62.26	91.76
Total Sleep Time (min/hours)	11	466.71 / 7.78	465.43 / 7.76	64.76 / 1.08	348.00 / 5.80	570.71 / 9.51
# of Awakenings	11	21.85	21.86	5.29	12.57	29.29
Post-Sleep Treatment	<i>N</i>	Mean	Median	<i>SD</i>	Min	Max
Efficiency (%)	10	77.89	77.74	5.10	70.84	85.78
Total Sleep Time (min/hours)	10	475.33 / 7.92	468.16 / 7.80	80.72 / 1.35	358.63 / 5.98	599.29 / 9.99
# of Awakenings	10	23.03	22.52	3.82	15.00	29.25

Notes. The National Sleep Foundation recommends that children ages 6-11 sleep 10-11 hours each night for optimal health. Sleep efficiency refers to the quality of sleep; above 85% is ideal.

Table 6

Descriptive statistics for baseline, post-sleep treatment, and post-combined treatment emotional/behavioral and executive control variables

Baseline	Mean	Median	SD	Min	Max
CBCL Internalizing Problems	57.69	58.00	11.49	41	74
CBCL Externalizing Problems	63.69	65.00	10.21	44	73
CBCL Total Problems Scale Score	64.69	68.00	8.52	49	74
CBCL DSM Affective	61.85	60.00	7.93	50	75
CBCL DSM Anxiety	61.31	65.00	9.53	50	73
CBCL DSM ADHD	64.85	66.00	8.34	53	77
CBCL DSM ODD	65.92	70.00	9.19	51	77
CBCL DSM CD	63.46	63.00	9.02	50	77
CBCL Social Problems	60.38	57.00	8.37	50	77
CBCL Attention Problems	66.46	68.00	8.93	53	87
CBCL Rule-Breaking Behavior	62.08	63.00	8.62	50	74
CBCL Aggressive Behavior	64.69	65.00	8.87	50	76
CBCL Anxious/Depressed	59.46	57.00	8.72	50	76
CBCL Withdrawn/Depressed	57.23	56.00	6.61	50	70
BRIEF Inhibit	66.75	64.50	10.93	46	89
BRIEF Shift	65.15	64.00	13.98	40	84
BRIEF Emotional Control	61.08	62.00	13.32	35	80
BRIEF Initiate	66.15	69.00	10.51	39	78
BRIEF Working Memory	70.00	69.50	11.13	48	86
BRIEF Plan/Organize	68.42	69.50	11.63	46	87
BRIEF Organization of Materials	59.15	59.00	9.33	43	73
BRIEF Monitor	70.42	70.50	7.88	56	84
BRIEF Global Executive Composite	70.00	69.00	11.13	46	86
BPM Internalizing	62.46	63.00	8.54	50	75
BPM Attention	67.00	70.00	6.95	52	75
BPM Externalizing	65.69	68.00	6.12	51	73
BPM Total Problems	68.31	68.00	4.97	56	77

Post-Sleep Treatment	Mean	Median	SD	Min	Max
CBCL Internalizing Problems	57.42	60.50	11.11	41	72
CBCL Externalizing Problems	63.50	63.00	7.13	49	74
CBCL Total Problems Scale Score	63.17	64.00	7.58	51	74
CBCL DSM Affective	61.42	64.00	8.46	50	73
CBCL DSM Anxiety	59.25	57.00	9.27	50	75
CBCL DSM ADHD	62.58	60.00	9.03	51	78
CBCL DSM ODD	62.42	62.50	7.75	52	80
CBCL DSM CD	63.58	64.00	5.79	52	72
CBCL Social Problems	57.42	57.00	5.55	51	69
CBCL Attention Problems	63.67	59.00	10.73	52	90
CBCL Rule-Breaking Behavior	64.67	65.00	5.33	55	73
CBCL Aggressive Behavior	62.17	62.00	7.94	50	75
CBCL Anxious/Depressed	57.33	52.00	9.03	50	72
CBCL Withdrawn/Depressed	60.00	61.00	6.88	50	67
BRIEF Inhibit	63.58	64.50	9.13	41	75
BRIEF Shift	61.50	58.00	13.17	43	83
BRIEF Emotional Control	57.25	62.00	12.32	38	72
BRIEF Initiate	61.50	63.00	11.44	41	75
BRIEF Working Memory	64.00	64.00	9.20	52	86
BRIEF Plan/Organize	62.67	64.50	8.96	49	77
BRIEF Organization of Materials	57.50	56.50	6.87	45	66
BRIEF Monitor	61.83	61.00	12.06	47	91
BRIEF Global Executive Composite	64.08	65.50	10.72	47	81
BPM Internalizing	56.15	51.00	8.17	50	69
BPM Attention	61.92	60.00	7.09	52	73
BPM Externalizing	58.85	60.00	6.80	51	70
BPM Total Problems	61.00	61.00	6.82	52	72
Post-Combined Treatment	Mean	Median	SD	Min	Max
BPM Internalizing	55.88	51.00	7.72	50	70
BPM Attention	62.38	60.00	6.12	55	73
BPM Externalizing	62.00	62.50	5.50	53	70
BPM Total Problems	62.50	61.00	6.82	52	72

Notes. CBCL = Child Behavior Checklist; DSM = Diagnostic and Statistical Manual; ADHD = Attention-Deficit/Hyperactivity; ODD = Oppositional Defiant Disorder; CD = Conduct Disorder; BRIEF = Behavior Rating Inventory of Executive Function; BPM = Brief Problems Monitor

Primary Research Questions

Aim 1: Immediate effect of sleep treatment. The first hypothesis was that emotional/behavioral symptoms, EC, and sleep problems would show an initial decline after sleep treatment.

Immediate change in sleep. In contrast to the hypothesis, children's actigraphy scores from baseline to post-sleep treatment did not significantly change. There was not a significant change in mean sleep duration in minutes from baseline ($M = 458.80$, $SD = 62.44$) to end of sleep treatment ($M = 475.33$, $SD = 80.72$), $F(1, 9) = .53$, $p = .487$, $Mse = 2594.43$. There was also not a significant change in mean sleep efficiency from baseline ($M = 79.14\%$, $SD = 8.61\%$) to end of sleep treatment ($M = 77.89\%$, $SD = 5.11$), $F(1, 9) = .27$, $p = .615$, $Mse = 28.34$.

Immediate change in total psychopathology symptoms. In contrast to the research hypothesis, there was not a significant change in the mean ratings on the CBCL Total Problems scale from baseline ($M = 66.00$, $SD = 7.41$) to end of sleep treatment ($M = 63.17$, $SD = 7.58$), $F(1, 11) = 2.48$, $p = .144$, $Mse = 19.44$.

In support of the research hypothesis, there was a significant change in the mean ratings on the BPM Total Problems scale from baseline ($M = 68.31$, $SD = 4.97$) to end of

sleep treatment ($M = 61.00$, $SD = 6.82$), $F(1, 12) = 15.82$, $p = .002$, $Mse = 21.95$. When gender was entered as a covariate, there continued to be a significant change in the mean ratings on the BPM Total Problems scale from baseline to end of sleep treatment, $F(1, 11) = 11.77$, $p = .006$, $Mse = 22.58$.

Immediate change in externalizing problems. In contrast to the research hypothesis, there was not a significant change in the mean ratings on the CBCL Externalizing Problems scale from baseline ($M = 65.08$, $SD = 9.29$) to end of sleep treatment ($M = 63.50$, $SD = 7.13$), $F(1, 11) = .55$, $p = .47$, $Mse = 27.34$. There was also not a significant change in the mean ratings on the CBCL Attention Problems scale from baseline ($M = 65.92$, $SD = 9.10$) to end of sleep treatment ($M = 63.67$, $SD = 10.73$) with gender included as a covariate, $F(1, 10) = 2.30$, $p = .161$, $Mse = 19.45$. Moreover, there was not a significant change in the mean ratings on the CBCL ADHD scale from baseline ($M = 65.00$, $SD = 8.69$) to end of sleep treatment ($M = 62.58$, $SD = 9.03$), $F(1, 11) = 1.55$, $p = .240$, $Mse = 22.68$.

In contrast to the research hypothesis, there was not a significant change in the mean ratings on the CBCL ODD scale from baseline ($M = 67.17$, $SD = 8.38$) to end of sleep treatment ($M = 62.42$, $SD = 7.75$), $F(1, 11) = 3.06$, $p = .108$, $Mse = 44.28$; nor was there a change in the mean ratings on the CBCL Conduct Problems scale from baseline ($M = 64.42$, $SD = 8.70$) to end of sleep treatment ($M = 63.58$, $SD = 5.79$), $F(1, 11) = .31$, $p = .589$, $Mse = 13.44$. There was not a significant change in the mean ratings on the CBCL Aggressive Behavior scale from baseline ($M = 65.92$, $SD = 8.04$) to end of sleep treatment ($M = 62.17$, $SD = 7.94$), $F(1, 11) = 2.85$, $p = .120$, $Mse = 29.65$; nor was there a change in the mean ratings on the CBCL Rule-Breaking Behavior scale from baseline

($M = 62.92$, $SD = 8.43$) to end of sleep treatment ($M = 64.67$, $SD = 5.33$), $F(1, 11) = 1.10$, $p = .317$, $Mse = 16.74$.

Furthermore, there was not a significant change in the mean ratings on the CBCL Social Problems scale from baseline ($M = 61.25$, $SD = 8.11$) to end of sleep treatment ($M = 57.42$, $SD = 5.55$), although the change approached significance, $F(1, 11) = 4.03$, $p = .070$, $Mse = 21.89$.

Supporting the research hypothesis, there was a significant change in the mean ratings on the BPM Externalizing Problems scale from baseline ($M = 65.69$, $SD = 6.12$) to end of sleep treatment ($M = 58.85$, $SD = 6.80$), $F(1, 12) = 12.97$, $p = .004$, $Mse = 23.49$. There was also a significant change in the mean ratings on the BPM Attention Problems scale from baseline ($M = 67.00$, $SD = 6.95$) to end of sleep treatment ($M = 61.92$, $SD = 7.09$) with gender included as a covariate, $F(1, 11) = 8.87$, $p = .013$, $Mse = 14.90$.

Immediate change in internalizing problems. In contrast to the research hypothesis, there was not a significant change in the mean ratings on the CBCL Internalizing Problems scale from baseline ($M = 58.92$, $SD = 11.07$) to end of sleep treatment ($M = 57.42$, $SD = 11.12$), $F(1, 11) = 1.11$, $p = .314$, $Mse = 12.14$. There was also not a significant change in the mean ratings on the CBCL Anxious/Depressed scale from baseline ($M = 60.17$, $SD = 8.72$) to end of sleep treatment ($M = 57.33$, $SD = 9.93$), $F(1, 11) = 1.74$, $p = .214$, $Mse = 27.71$. Further, there was not a significant change in the mean ratings on the CBCL Withdrawn/Depressed scale from baseline ($M = 57.83$, $SD = 6.52$) to end of sleep treatment ($M = 60.00$, $SD = 6.88$), $F(1, 11) = 1.15$, $p = .307$, $Mse = 24.53$. Finally, there was not a significant change in the mean ratings on the CBCL

Affective Problems scale from baseline ($M = 62.83$, $SD = 7.40$) to end of sleep treatment ($M = 61.42$, $SD = 8.46$), $F(1, 11) = 1.37$, $p = .266$, $Mse = 8.77$.

In support of the research hypothesis, there was a significant change in the mean ratings on the BPM Internalizing Problems scale from baseline ($M = 62.46$, $SD = 8.54$) to end of sleep treatment ($M = 56.15$, $SD = 8.17$), $F(1, 12) = 8.83$, $p = .012$, $Mse = 29.28$. Additionally, although not significant, there was a trend toward a significant change in the mean ratings on the CBCL Anxiety Problems scale from baseline ($M = 62.25$, $SD = 9.30$) to end of sleep treatment ($M = 59.25$, $SD = 9.27$), $F(1, 11) = 4.75$, $p = .052$, $Mse = 11.36$.

Immediate change in executive control. In support of the research hypothesis, there was a significant difference on the Global Executive Composite from baseline ($M = 70.64$, $SD = 11.44$) to post-sleep ($M = 63.36$, $SD = 10.94$), $F(1, 10) = 6.06$, $p = .034$, $Mse = 48.01$. Also in support of the hypothesis, there was a significant difference on the Emotional Control scale from baseline ($M = 62.00$, $SD = 13.57$) to post-sleep ($M = 56.45$, $SD = 12.60$), $F(1, 10) = 8.08$, $p = .017$, $Mse = 20.94$. There was also a significant difference from baseline ($M = 70.73$, $SD = 8.19$) to post-sleep ($M = 62.00$, $SD = 12.63$) on the Monitor scale, $F(1, 10) = 7.84$, $p = .019$, $Mse = 53.41$.

There was a difference that trended toward significance from baseline ($M = 69.27$, $SD = 11.80$) to post-sleep ($M = 61.82$, $SD = 8.88$) on the Plan/Organize scale, $F(1, 10) = 4.83$, $p = .053$, $Mse = 63.24$. There was also difference that trended toward significance from baseline ($M = 69.64$, $SD = 11.60$) to post-sleep ($M = 63.45$, $SD = 9.44$) on the Working Memory scale, $F(1, 10) = 3.75$, $p = .081$, $Mse = 55.98$.

Contrary to the research hypothesis, there was no difference between the mean ratings on the other BRIEF scales from baseline to post-sleep treatment. There was no difference from baseline ($M = 67.36$, $SD = 11.25$) to post-sleep ($M = 63.91$, $SD = 9.50$) on the Inhibit scale, $F(1, 10) = 1.33$, $p = .275$, $Mse = 49.24$; from baseline ($M = 66.17$, $SD = 14.10$) to post-sleep ($M = 61.50$, $SD = 13.17$) on the Shift scale, $F(1, 11) = .96$, $p = .348$, $Mse = 136.21$; from baseline ($M = 66.50$, $SD = 10.90$) to post-sleep ($M = 61.50$, $SD = 11.44$) on the Initiate scale, $F(1, 11) = 2.62$, $p = .134$, $Mse = 57.27$; nor from baseline ($M = 59.08$, $SD = 9.74$) to post-sleep ($M = 57.50$, $SD = 6.87$) on the Organization of Materials scale, $F(1, 11) = .27$, $p = .614$, $Mse = 55.77$.

Aim 2: Total effect of all treatment. The second hypothesis was that children's symptoms would continue to decline after combined treatment and show a significant pre-post difference.

Total change in psychopathology symptoms. When gender was included as a covariate, the change in the mean ratings on the BPM Total Problems scale trended in the hypothesized direction from baseline ($M = 69.00$, $SD = 3.96$) to end of all treatment ($M = 62.25$, $SD = 6.63$), $F(1, 7) = 5.57$, $p = .056$, $Mse = 39.66$. When gender was not included as a covariate, the change in the mean ratings on the BPM Total Problems scale trended in the hypothesized direction from baseline ($M = 69.00$, $SD = 3.96$) to end of all treatment ($M = 62.25$, $SD = 6.63$), $F(1, 7) = 4.48$, $p = .072$, $Mse = 40.68$. The observed power in the analysis was 44.7%.

Multilevel models were estimated in SAS version 9.3 with time as the predictor of BPM Total Problems to examine trajectories of change. Refer to Figure 2 for the model equation including a fixed slope of time. Model A examined the BPM Total Problems

across each therapy session. Because children attended an average of eight therapy sessions, the BPM was examined across eight sessions. The significant effect of time indicated that for every session a child attended, Total Problems decreased by .62, $b = -.62$, $SE = .19$, $t = -3.30$, $p = .002$. Model B included the BPM Total Problems at the three main time points (baseline, post-sleep treatment, post-combined sleep and behavioral treatment). The significant effect of time indicated that following sleep treatment and at the end of treatment, Total Problems decreased by 3.56 at each time point, $b = -3.56$, $SE = 1.25$, $t = -2.84$, $p = .009$. See Figures 3 and 4 for spaghetti plots of each participant's BPM Total Problems across treatment.

$$\begin{aligned}
 y_{ti} &= \pi_{0i} + \pi_{1i} \text{TIME} + e_{ti} && \text{Level 1} \\
 \pi_{0i} &= \beta_{00} + r_{0i} && \text{Level 2} \\
 \pi_{1i} &= \beta_{10}
 \end{aligned}$$

Figure 2. The MLM equation including a fixed slope for time to examine change in BPM scores across treatment

Notes. i indexes individual and t indexes time; Level 1 is the within person variation and Level 2 is the between-person variation; y_{ti} is the BPM Total Problems score at measurement t for person i ; β_{00} is the fixed intercept or the sample grand mean of the BPM Total Problems score; β_{10} is the fixed linear time slope; r_{0i} is the individual intercept deviation or person-specific deviation; e_{ti} is the residual error or the time specific deviation

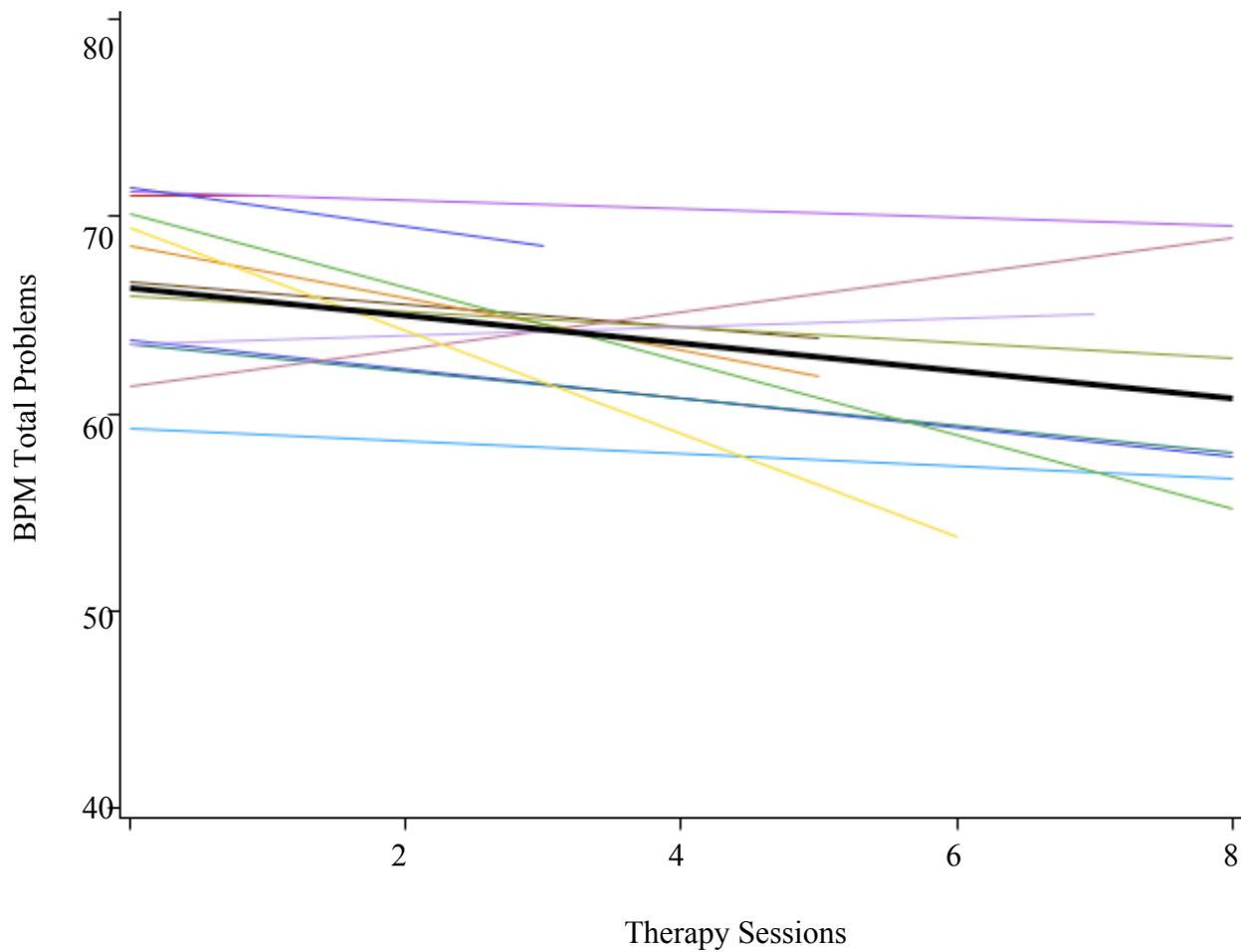


Figure 3. Spaghetti plot for each participant plotting the BPM Total Problems across treatment

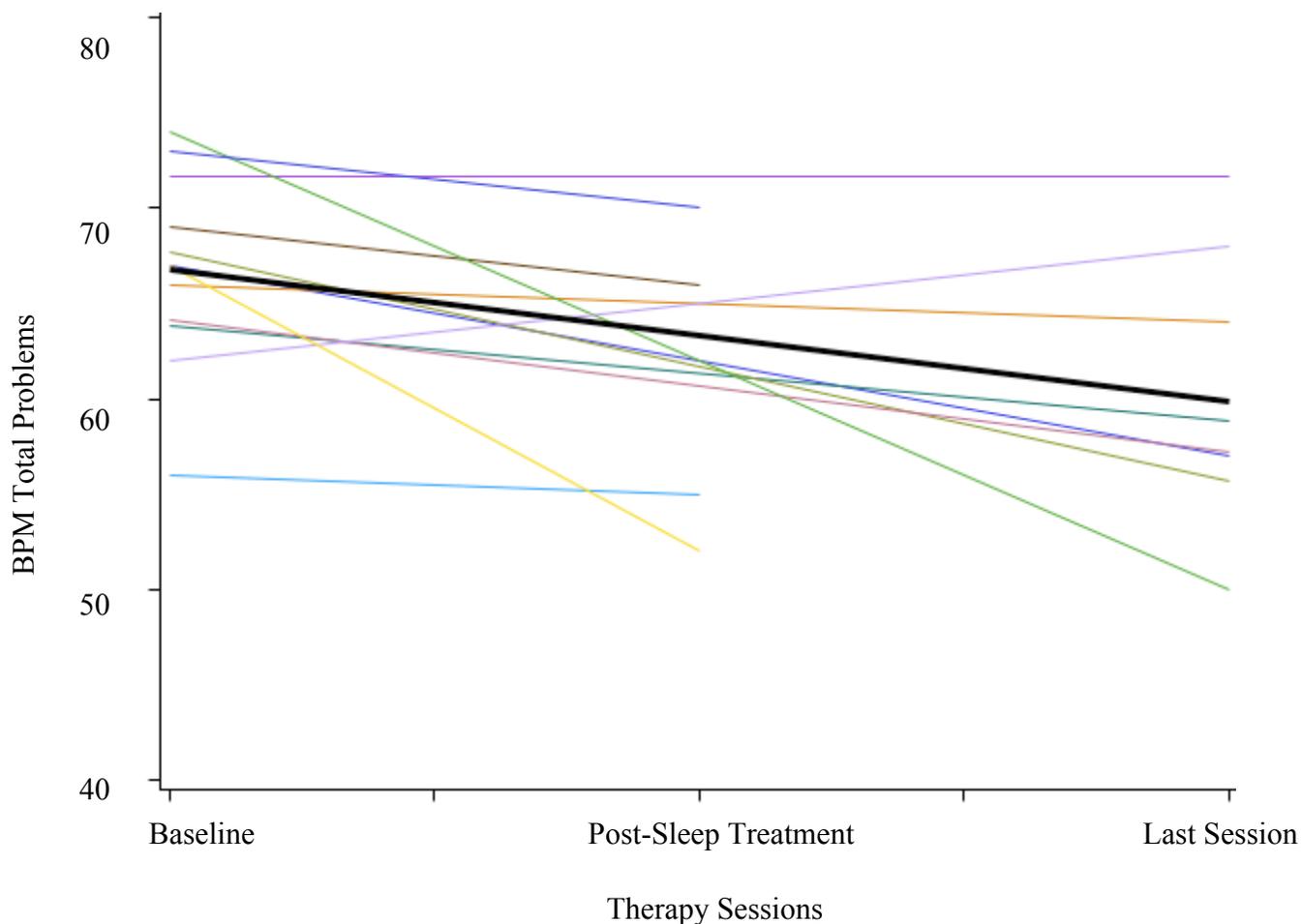


Figure 4. Spaghetti plot for each participant plotting the BPM Total Problems across the three main time points

Total change in externalizing problems. Contrary to the research hypothesis, there was no difference between the mean ratings on the BPM Externalizing Problems scale from baseline ($M = 65.88$, $SD = 7.22$) to post-sleep treatment ($M = 62.00$, $SD = 5.50$), $F(1, 7) = 1.00$, $p = .351$, $Mse = 60.06$. When gender was included as a covariate,

there was no difference between the mean ratings on the BPM Attention Problems scale from baseline ($M = 67.50$, $SD = 6.12$) to post-sleep treatment ($M = 62.38$, $SD = 6.12$), $F(1, 7) = 2.34$, $p = .177$, $Mse = 35.90$.

Multilevel models were estimated in SAS version 9.3 with time as the predictor of BPM Externalizing Problems and Attention Problems to examine growth trajectories. Model A examined the BPM Externalizing Problems across each therapy session. Because children attended an average of eight therapy sessions, the BPM was examined across eight sessions. There was a trend toward a significant effect of time, such that for every session a child attended, Externalizing Problems decreased by .40, $b = -.40$, $SE = .21$, $t = -1.92$, $p = .059$. Model B included the BPM Externalizing Problems at the three main time points (baseline, post-sleep treatment, post-combined sleep and behavioral treatment). There was not a significant fixed effect of time for Externalizing Problems, $b = -.64$, $SE = 1.65$, $t = -1.75$, $p = .093$.

Model C examined the BPM Attention Problems across each therapy session. The significant effect of time indicated that for every session a child attended, Attention Problems decreased by .51, $b = -.51$, $SE = .16$, $t = -3.19$, $p = .002$. Model D included the BPM Attention Problems at the three main time points (baseline, post-sleep treatment, post-combined sleep and behavioral treatment). The significant effect of time indicated that following sleep treatment and at the end of treatment, Attention Problems decreased by 2.84 at each time point, $b = -2.84$, $SE = 1.05$, $t = -2.71$, $p = .013$.

Total change in internalizing problems. The change in the mean ratings on the BPM Internalizing Problems scale trended in the hypothesized direction from baseline

($M = 62.38$, $SD = 7.25$) to end of all treatment ($M = 55.88$, $SD = 7.72$), $F(1, 7) = 4.46$, $p = .072$, $Mse = 37.86$.

Multilevel models were estimated in SAS version 9.3 with time as the predictor of BPM Internalizing Problems to examine growth trajectories. Model A examined the BPM Internalizing Problems across each therapy session. The significant effect of time indicated that for every session a child attended, Internalizing Problems decreased by .45, $b = -.45$, $SE = .20$, $t = -2.32$, $p = .023$. Model B included the BPM Internalizing Problems at the three main time points (baseline, post-sleep treatment, post-combined sleep and behavioral treatment). The significant effect of time indicated that following sleep treatment and at the end of treatment, Internalizing Problems decreased by 3.29 at each time point, $b = -3.29$, $SE = 1.27$, $t = -2.59$, $p = .017$.

Secondary Research Questions

Aim 3: Comparison of combined treatment to effects in the literature. The third hypothesis was that the pre-post effect size would be larger than effect sizes currently found in the literature for behavioral treatments (e.g., $d = .63$ for ADHD; Fabiano et al., 2009). Moreover, we expected that children receiving combined sleep and behavioral treatment would have higher normalization rates than rates in the literature for current behavioral treatments.

Comparison of effect sizes. First, Cohen's d was calculated as the effect size of the CBCL Total Problems scale pre-post sleep treatment ($d = .45$). Second, Cohen's d was calculated as the effect size of the BPM Total Problems scale pre-post sleep treatment. In a model with gender as a covariate, Cohen's d for the analysis from

baseline to post-sleep treatment was $d = .99$. In a model without gender as a covariate, Cohen's d for the analysis from baseline to post-sleep treatment was $d = 1.10$.

Third, Cohen's d was calculated as the effect size of the BPM Total Problems scale pre-post all treatment. In a model with gender as a covariate, Cohen's d for the analysis from baseline to the end of all treatment was $d = .83$. In a model without gender as a covariate, Cohen's d for the analysis from baseline to the end of all treatment was $d = .75$.

For a comparison, an effect size of $d = .63$ was found for behavioral treatment ADHD (Fabiano et al., 2009) and $d = .34$ for behavioral or CBT treatment of depression (Weisz et al., 2006). Figure 5 presents a visual comparison of the effect sizes of the examined studies.

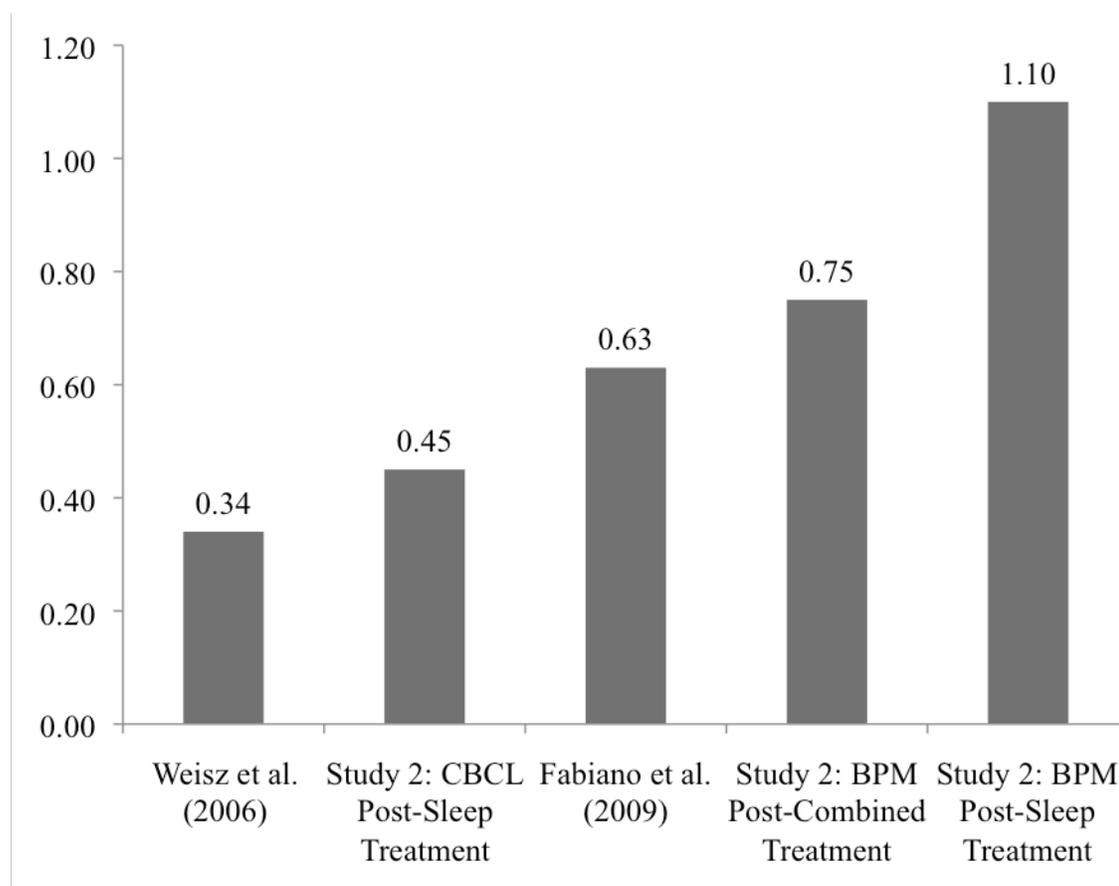


Figure 5. Comparison of Cohen's *d* effect sizes

Notes. The effect size in Weisz et al. (2006) is for CBT for depression. The effect size in Fabiano et al. (2009) is for behavioral treatment of ADHD. BPM = Brief Problems Monitor; CBCL = Child Behavior Checklist

Comparison of normalization rates. The normalization rate of the CBCL Total Problems scale following sleep treatment was 50.0% (out of $N = 12$). The normalization rate of the BPM Total Problems scale following sleep treatment was 69.2% (out of $N = 13$). The normalization rate of the BPM Total Problems scale at the end of combined sleep and behavioral treatment was 62.5% (out of $N = 8$). Flannery-Schroeder & Kendall (2000) reported a 73% normalization rate following individual treatment of

anxiety using the Coping Cat protocol and a 50% normalization rate following group therapy. Jensen et al. (2001) reported a 34% normalization rate in the MTA study for behavioral treatment of ADHD and a 68% normalization rate for combined medication and behavioral treatment. Kennard et al. (2006) reported a 16% normalization rate following CBT for depression in the TADS study. See Table 7 and Figure 6 for a comparison of normalization rates.

Table 7

Comparison of normalization rates

Current Study: Study 2	Normalization Rate	Published RCTs	Normalization Rate	Difference in Normalization Rates
BPM Post-Sleep Treatment (N = 13)	69.2%	Coping Cat: Individual	73.0%	-5.8%
		Coping Cat: Group	50.0%	19.2%
		MTA: Behav	34.0%	35.2%
		MTA: Comb	68.0%	1.2%
		TADS	16.0%	53.2%
BPM Post-Combined Treatment (N = 8)	62.5%	Coping Cat: Individual	73.0%	-10.5%
		Coping Cat: Group	50.0%	12.5%
		MTA: Behav	34.0%	28.5%
		MTA: Comb	68.0%	-5.5%
		TADS	16.0%	46.5%
CBCL Post-Sleep Treatment (N = 12)	50.0%	Coping Cat: Individual	73.0%	-23.0%
		Coping Cat: Group	50.0%	0%
		MTA: Behav	34.0%	16.0%
		MTA: Comb	68.0%	-18%
		TADS	16.0%	34%

Notes. BPM = Brief Problems Monitor; CBCL = Child Behavior Checklist; MTA:

Behav = Multimodal Treatment Study of ADHD: Behavioral Treatment; MTA: Comb =

Multimodal Treatment Study of ADHD: Combined Medication and Behavioral Treatment; TADS = Treatment of Adolescents with Depression Study

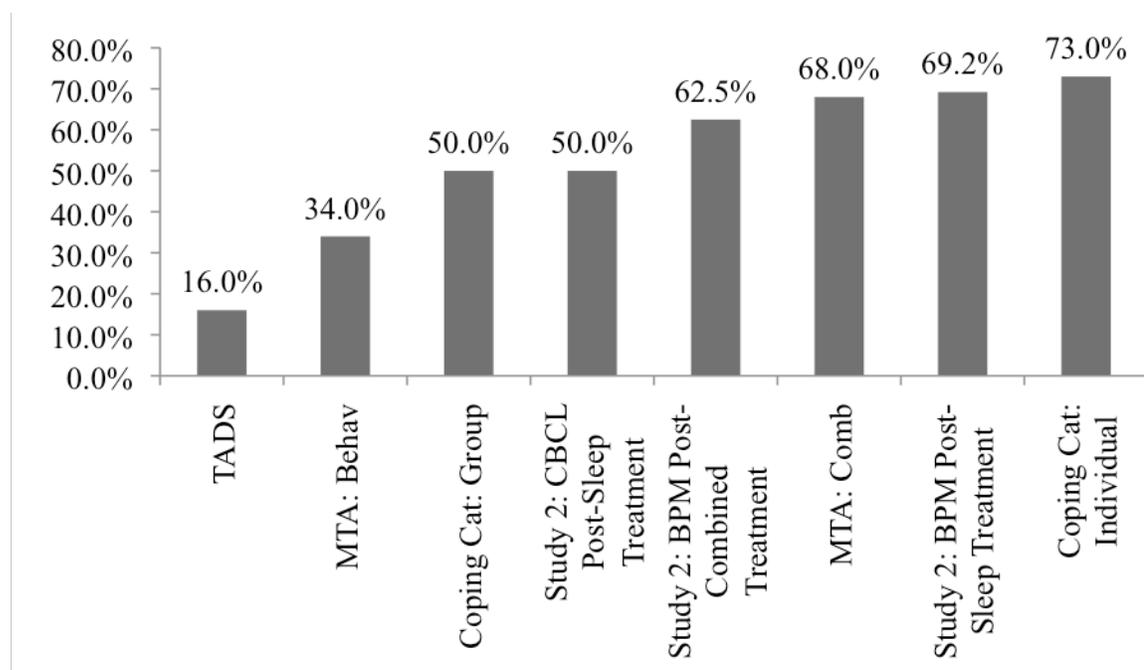


Figure 6. Comparison of normalization rates

Notes. BPM = Brief Problems Monitor; CBCL = Child Behavior Checklist; MTA: Behav = Multimodal Treatment Study of ADHD: Behavioral Treatment; MTA: Comb = Multimodal Treatment Study of ADHD: Combined Medication and Behavioral Treatment; TADS = Treatment of Adolescents with Depression Study

Aim 4: Executive control mediating sleep-psychopathology. The fourth hypothesis was that change in EC would mediate the relationship between treatment and symptom reduction, such that sleep would improve EC and greater EC would improve mental health symptoms.

CBCL. Results for the mediator analyses are presented in Figure 7. The first path established change in sleep duration (independent variable) was associated with change in EC (mediator), $b = -.13$, $SE = .04$, $t = -2.99$, $p = .024$. Second, the direct effect of EC (mediator) on CBCL Total Problems (dependent variable) was estimated. EC was associated with CBCL Total Problems, $b = .66$, $SE = .18$, $t = 3.58$, $p = .016$. Third, the path estimating the total effect of the change in sleep duration (IV) on CBCL Total Problems (DV) was estimated. Sleep duration did not significantly predict Total Problems, $b = -.03$, $SE = .03$, $t = -1.00$, $p = .354$. Fourth, the direct effect of sleep duration on CBCL Total Problems through change in EC (mediator) was estimated. With the inclusion of the mediator, the coefficient for sleep duration was not significant, $b = .05$, $SE = .03$, $t = 1.67$, $p = .155$. The sleep duration coefficient switched from being negative to positive when the mediator was included. The overall model including the mediator was significant, $F(2, 5) = 7.90$, adjusted $R^2 = .66$, $p = .028$.

Although the pathways were not significant, nonparametric bootstrapping analyses were repeated 5000 times to test the model of EC as a mediator of the relationship between sleep duration and CBCL Total Problems scores. In these analyses, mediation is significant if the 95% Bias Corrected confidence intervals for the indirect effect do not include 0 (Hayes, 2009; Preacher & Hayes, 2008). Using bootstrapping procedures, EC was found to mediate the relationship between sleep duration and Total Problems (Indirect Effects 95% CI = $-.17$ to $-.01$), such that children with longer sleep duration were more likely to have improved EC, and due to improved EC, had lower CBCL Total Problems scores.

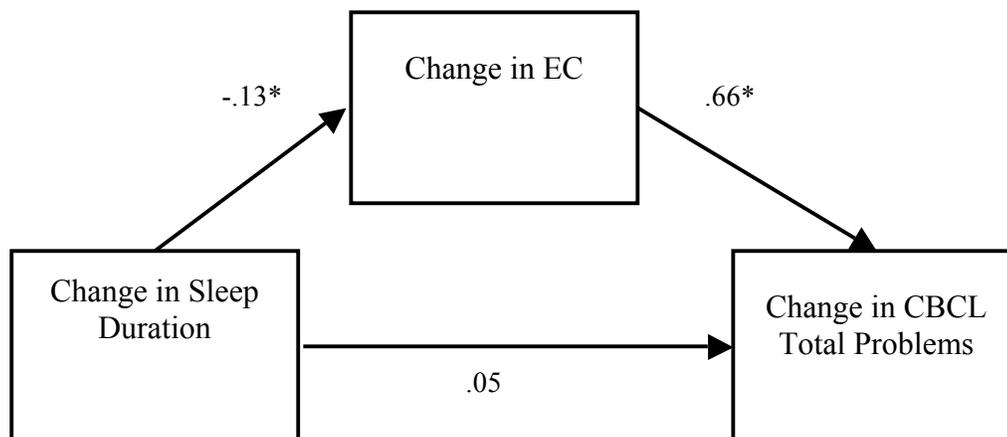


Figure 7. Unstandardized mediation results for CBCL

Notes. The direct path from sleep duration to CBCL Total Problems was not significant before the mediator was added, $b = -.03$, $p = .354$. $*p < .05$

BPM. Results for the mediator analyses are presented in Figure 8. The first path established change in sleep duration (independent variable) was associated with change in EC (mediator), $b = -.13$, $SE = .04$, $t = -2.99$, $p = .024$. Second, the direct effect of EC (mediator) on BPM Total Problems (dependent variable) was estimated. EC was not significantly associated with BPM Total Problems, $b = .75$, $SE = .41$, $t = 1.85$, $p = .124$. Third, the path estimating the total effect of the change in sleep duration (IV) on BPM Total Problems (DV) was estimated. Sleep duration did not significantly predict Total Problems, $b = .03$, $SE = .05$, $t = .55$, $p = .602$. Fourth, the direct effect of sleep duration on BPM Total Problems through change in EC (mediator) was estimated. With the inclusion of the mediator, the coefficient for sleep duration was not significant, $b = .12$,

$SE = .07, t = 1.84, p = .125$. The overall model including the mediator was not significant, $F(2, 5) = 1.92$, adjusted $R^2 = .21, p = .241$.

Nonparametric bootstrapping analyses were repeated 5000 times to test the model of EC as a mediator of the relationship between sleep duration and BPM Total Problems scores. In these analyses, mediation is significant if the 95% Bias Corrected confidence intervals for the indirect effect do not include 0 (Hayes, 2009; Preacher & Hayes, 2008). Using bootstrapping procedures, EC was not found to mediate the relationship between sleep duration and Total Problems (Indirect Effects 95% CI = $-.33$ to $.03$).

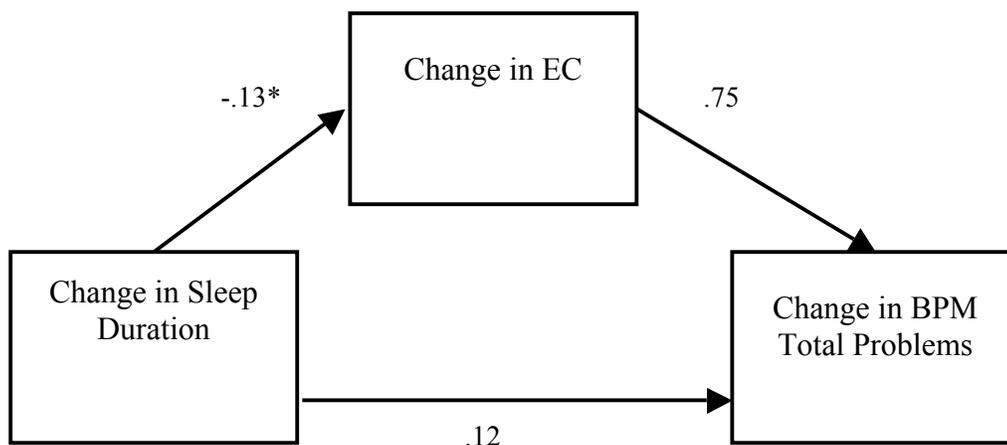


Figure 8. Unstandardized mediation results for BPM

Notes. The direct path from sleep duration to BPM Total Problems was not significant before the mediator was added, $b = .03, p = .602$. $*p < .05$

CHAPTER 13: STUDY 2 DISCUSSION

Study 2 was a preliminary pilot sleep intervention for children presenting to an outpatient clinic. There were four main hypotheses of the present study. First, the findings partially supported the hypothesis that sleep problems, emotional/behavioral symptoms, and EC would show an initial decline after sleep treatment. Second, the findings supported the hypothesis that children's symptoms would continue to decline after combined treatment and show a significant pre-post difference. Third, this results provided partial support for the hypothesis that Study 2 would have larger effect sizes and normalization rates compared to previous studies that did not include a sleep treatment component. Fourth, this study found partial support for the hypothesis that EC was a mediator of the association between objectively assessed sleep improvement and psychopathology outcomes.

Overview of Results

Aim 1: Immediate effect of sleep treatment. The first hypothesis was that emotional/behavioral symptoms, EC, and sleep problems would show an initial decline after sleep treatment.

Immediate change in sleep. Objectively measured sleep did not significantly change from the beginning of the intervention to after sleep treatment. There are several reasons why this may be the case. First, the analyses with actigraphy only had 10 children, resulting in low power. Second, the two actigraphy periods were separated by only one to two weeks. There may not have been enough time to see clinically significant or statistically significant changes in sleep duration, sleep efficiency, or number of awakenings. Perhaps, children had fewer sleep *problems*, such as bedtime

resistance or oppositional behaviors at bedtime, that actigraphs were unable to measure. Third, the results invite the question of whether the dose of the sleep treatment was strong enough to result in a clinical difference. Although objectively measured sleep did not improve, children who received sleep treatment before any other behavioral treatment demonstrated emotional/behavioral symptom reduction and EC improvement. It appears that the “dose” of the brief sleep treatment was likely strong enough, because change was apparent in other symptoms. Fourth, it is also possible that baseline sleep was not a *true* baseline, because children wore actigraphs after the first session. Although no formal intervention occurred in that first session, there still could have been an effect of beginning treatment, which made the baseline better than expected. Fifth, the current study did not include an assessment of intervention fidelity. Deviations from the protocol/session content may be another reason why the expected effects on sleep were not significant. Assessment of intervention fidelity would be an important consideration in a larger trial of the intervention. Sixth, the level of participation and engagement in treatment may have been variable and perhaps only a subset of the participants were compliant with the sleep recommendations. However, it is difficult to know who adhered to the sleep recommendations and who did not, so adherence to treatment is unable to be examined statistically.

The results indicating that objectively measured sleep did not improve diverge from findings by Hiscock et al. (2015). Hiscock and colleagues (2015) examined change in sleep as measured by actigraphy in a subset of the sample who received sleep treatment. It could be that the subset of participants who wore the actigraphs were more dedicated to the treatment as demonstrated by those who were willing to participate

further in the research study. Thus, perhaps those who wore the actigraph in Hiscock et al. (2015) were more adherent to the sleep treatment recommendations than those who did not wear the actigraph. Alternatively, sleep treatment might improve objectively measured sleep, but Study 2 was underpowered to detect small changes.

Immediate change in psychopathology symptoms. Emotional/behavioral symptoms as measured by the BPM reduced across treatment. Children had improved scores on the Total Problems, Externalizing Problems, Attention Problems, and Internalizing Problems. However, emotional/behavioral symptoms did not significantly improve when measured by the CBCL. Only one of the CBCL scales, Anxiety Problems, demonstrated a trend toward significant change from baseline to end of sleep treatment ($p = .052$). These results are consistent with research finding an association between sleep problems and psychopathology (Mayes et al., 2009; Sung et al., 2008), and extend the research on the basic association between sleep problems and psychopathology by examining how sleep *treatment* connects with psychopathology.

Study 2 also builds on the limited literature on sleep treatment in several important ways. First, this study uses a *brief* sleep treatment which is thought to be more practical for therapists to administer than a 12-week treatment protocol (as in Keshavarzi et al., 2014). Second, this study is a first examination of sleep treatment with children with internalizing disorders and comorbid conditions. Previous research has focused only on ADHD and has tended to exclude those with comorbid mental health conditions like ODD (Hiscock et al., 2015; Keshavarzi et al., 2014). Third, this study builds on sleep treatment research that had not included validated measures (i.e., Nelson et al., 2016) by using objective measurement of sleep and validated measures of behavior, emotion, and

EC at multiple time points to evaluate therapy outcomes. The first published study of this brief sleep intervention (Nelson et al., 2016) had significant limitations in terms of measurement issues. By using validated and objective measurement, Study 2 was more than an initial feasibility study, and was rather a very preliminary pilot intervention. Fourth, this study examined the immediate change in psychopathology symptoms following sleep treatment, whereas most studies have examined change at the end of all treatment provided (Hiscock et al., 2015; Keshavarzi et al., 2014).

There are several possible reasons for why the BPM presented changes in symptoms while the CBCL did not. First, although the pre- and post- CBCL and BPM were completed during the same sessions, parents apparently responded differently to the questions. The CBCL was administered once at baseline and once at post-sleep treatment, while the BPM was administered each session. Perhaps, parents responded in a different way to the 113 questions of the CBCL in comparison to the 19 questions of the BPM. Although they are possibilities, it is unclear how the administration frequency and length of the instrument would affect the responses to the questions. Second, the CBCL had 12 participants in the analyses, and the BPM had 13 people. Perhaps, the one extra person shifted the averages of the results. Third, the CBCL was designed to examine change over six months, and the BPM was designed to examine weekly change. Maybe the CBCL was not sensitive enough to examine rapid change over only one to three weeks, whereas the BPM was designed for that purpose. Fourth, the results may have been diminished by having such broad diagnostic eligibility criteria (e.g., any presenting internalizing or externalizing problem). Because some of the participants would not have clinical scores on some scales, there would not be a chance to improve.

Immediate change in executive control. Some of the BRIEF scales improved following sleep treatment and some did not (e.g., Inhibit, Shift, Initiate, Organization of Materials did not improve; Plan/Organize and Working Memory approached significance). The overall EC scale, the Global Executive Composite, improved after sleep treatment. In terms of subscales, Emotional Control and Monitoring also improved following treatment. What could explain the difference in which scales improved and which did not? First, it could be that sleep treatment truly is not associated with improvement in certain EC abilities. Second, it could be that the BRIEF subscales vary in their ability to measure certain abilities. Some abilities may be better measured by behavioral tasks than through parent-report. Thus, the BRIEF subscales may not be the most valid measure of EC abilities. Third, many of the analyses contained only 11 participants (some contained 12 participants), and thus, the sample was not large enough to detect small effects. It is promising that the overall composite EC scale and the Emotional Control scale significantly improved following sleep treatment, even with a smaller sample size. This result is consistent with research finding an association between sleep and EC (Fallone et al., 2005), and indicates that EC may be an important mechanism for how sleep problems are correlated with psychopathology.

Aim 2: Total effect of all treatment. The second hypothesis was that children's symptoms would continue to decline after combined treatment and show a significant pre-post difference.

Total change in psychopathology symptoms. This set of analyses examined change in psychopathology symptoms after combined sleep treatment and behavioral treatment. Because children had to have at least one session of behavioral treatment in

addition to the sleep treatment, only eight children were included in the ANOVA analyses. There were not significant differences from baseline to post-treatment on overall psychopathology, externalizing problems, or internalizing problems, although the analyses for the Internalizing Problems and Total Problems scales approached significance. However, when change was examined in a longitudinal growth curve MLM, all 13 participants' data were included and plotted over time. Multilevel analyses indicated that the Total Problems, Attention Problems, and Internalizing Problems scales significantly improved across time.

MLM is a sensitive method for modeling change over time, because maximum likelihood estimation allows all participants to be included in the analyses which increases power to detect effects. MLM is also an appropriate analytic technique for the current data, because parents completed BPMs at each therapy session resulting in an average of eight measurement time points. The MLM findings are novel, because Study 2 represents the first examination of the associations between combined sleep and behavioral treatments and psychopathology outcomes.

Aim 3: Comparison of combined treatment to effects in the literature. The third hypothesis was that the pre-post effect size would be larger than effect sizes currently found in the literature for behavioral treatments. Moreover, it was expected that children receiving combined sleep and behavioral treatment would have higher normalization rates than rates in the literature for current behavioral treatments.

Comparison of effect sizes. The effect sizes (Cohen's d) for Study 2 ranged from .45 to 1.10 which are medium to large effect sizes. Analyses examining change in BPM scores resulted in larger effect sizes than analyses that included the CBCL. For a

comparison, an effect size of .63 was found for behavioral treatment ADHD (Fabiano et al., 2009) and .34 for behavioral or CBT treatment of depression (Weisz et al., 2006). To visually inspect differences across studies, refer to Figure 5. It is worth noting that the comparison studies (like MTA) were over many more sessions than the current study.

Comparison of normalization rates. The normalization rates in Study 2 ranged from 50% to 69.2% depending on the time frame selected (either after sleep treatment or at the end of all treatment) and the measure used to determine whether symptoms were in the clinical range. The highest normalization rate of the selected published studies was 73% following individual treatment of anxiety using the Coping Cat protocol (Flannery-Schroeder & Kendall, 2000). The lowest normalization rate of the selected published studies was 16% following CBT for depression in the TADS study (Kennard et al., 2006). Many of the children in the current study presented with symptoms of ADHD. When compared to behavioral treatments of ADHD, the sleep treatment resulted in comparable normalization rates. For instance, Jensen et al. (2001) reported a 34% normalization rate in the MTA study for behavioral treatment of ADHD and a 68% normalization rate for combined medication and behavioral treatment. Some of the children in Study 2 were on stimulant medications for ADHD, and thus, the more appropriate comparison may be to compare the 69.2% normalization rate of Study 2 following sleep treatment with the 68% normalization rate for the MTA combined medication and behavioral treatment. Importantly, the comparison studies contained many more therapy sessions than Study 2. The easiest way to visualize the comparison of normalization rates is to examine Figure 6. Overall, the normalization rates of the current study either outperformed or were comparable to published studies.

Aim 4: Executive control mediating sleep-psychopathology. The fourth hypothesis was that change in EC would mediate the relationship between treatment and symptom reduction, such that sleep would improve EC and greater EC would improve mental health symptoms. The results demonstrated that changes in objectively measured sleep duration predicted changes in EC following sleep treatment. Yet, change in objectively measured sleep duration did not predict change in psychopathology. Thus, mediation was not initially evident when examining the significant/non-significant pathways. However, bootstrapping is recommended for small sample sizes in which the models could be underpowered. When bootstrapping methods were used, EC acted as a mediator between sleep duration and psychopathology as assessed via the CBCL, but not the BPM. The results partially supported the hypothesis that sleep improvements would lead to improved EC which would be associated with improved psychopathology. Research examining EC as a mediator is extremely rare, and thus, this study presents a novel examination of EC mediating the sleep treatment – psychopathology connection.

Research Implications

The current study included validated measures, extending the work of Nelson and colleagues (2016). When validated measures were used instead of one to seven ranking scales about problem areas, the effect sizes were smaller, thus, slightly tempering conclusions about the effectiveness of a brief sleep intervention. Specifically, Nelson and colleagues (2016) reported an effect size of 3.25 with non-validated measures, and the current study had an effect size of .45 with the CBCL and 1.10 with the BPM. Future researchers are encouraged to continue using validated measures of sleep, psychopathology, and EC to provide a stringent test of sleep treatment. Furthermore, the

BPM was more sensitive to change than the CBCL as evidenced by the significant weekly changes in BPM ratings and the non-significant change in ratings on the CBCL. Researchers may want to consider including the BPM in future treatment studies to examine weekly change over time.

This study was the first to examine treatment of sleep and internalizing symptoms. The results of this first investigation indicate that researchers need to examine sleep treatment and internalizing disorders further. Researchers may want to examine subsamples of youth with specific internalizing disorder diagnoses to investigate if there are differential associations between sleep treatment and various internalizing disorders (e.g., for example, maybe sleep treatment works better for those with only depression or anxiety than combined depression and anxiety).

Clinical Implications

The results of objectively measured sleep quantity and quality indicated that the current sample of children presenting for treatment for emotional/behavioral disorders had shorter sleep duration and worse sleep quality than what is recommended by the National Sleep Foundation. Although the recommendation is that children should sleep 10 to 11 hours each night, the children in the current study slept an average of 7.78 hours each night before receiving sleep treatment ($SD = 1.08$ hours, range = 5.80 – 9.51 hours) which results in a two to three hour sleep deficit every night. None of the children had average sleep duration in the recommended range. The results are consistent with research with a clinical sample that found children slept only 7.6 hours on average (Van Dyk et al., 2016). The average sleep efficiency in the current sample was below the recommended 85% as well ($M = 80.16\%$, $SD = 8.84$, range = 62.26% - 91.76%). Note

that the children in the sample were not presenting to the clinic because of sleep problems, but rather sleep problems were a comorbid condition to the primary presenting problems of noncompliance, anxiety, depression, disruptive behaviors, hyperactivity, or inattention. These results indicate that children presenting with emotional/behavioral disorders are likely to have sleep problems. Clinicians are encouraged to assess and treat sleep problems as part of routine clinical care.

This study found partial support for the effectiveness of a brief sleep module delivered universally at the beginning of treatment for emotional/behavioral disorders. The sleep treatment protocol provides an example of how to implement a brief treatment module in therapy. Clinicians and researchers in the field of pediatric psychology have been developing many treatment modules (Weisz et al., 2012), likely because pediatric psychologists often deliver treatments quickly in a hospital setting and value being able to choose a two-session module rather than a 12-week manualized protocol. Modular approaches tend to outperform traditional evidence-based treatments and may be easier for clinicians to use in practice (Weisz et al., 2012). Study 2 adds a brief sleep treatment to the existing behavioral modules.

Limitations and Future Directions

The findings from the sleep treatment pilot (Study 2) must be interpreted with caution given the low number of participants ($N = 13$). Findings with small samples are not as reliable or stable as those with larger samples. More research is needed with a larger sample to fully investigate the effectiveness of a brief sleep intervention at the start of therapy. Moreover, possibly due to the small sample size, the objective actigraph sleep data did not change from baseline to post-sleep treatment. Thus, the current study

is unable to support the hypothesis that sleep treatment improves sleep quantity and quality which, in turn, improves outcomes. Future research with larger samples may be better able to address this concern.

Although the sleep treatment was effective in reducing psychopathology symptoms as measured by the BPM and improving EC, there was high attrition. Attrition is common in clinical practice, but study attrition may have affected the results. For instance, the change in EC from start of treatment to the end of all treatment was unable to be examined, because only two children completed the third EC measure. Although, EC changed from baseline to post-sleep treatment, the change from baseline to post-combined sleep and behavioral treatments was not analyzed. Overall, the treatment appears to be effective in reducing psychopathology symptoms for the children who attended therapy, with the caveat that there was a high drop out rate. Future researchers with large grant funding may want to provide greater compensation following each session attended and/or provide transportation to therapy to mitigate drop out risk.

In the current investigation, participants were not randomized to provider or treatment type. Thus, the study does not provide information on causality, but instead, demonstrates an association between sleep treatment and outcomes. Future examinations of sleep treatment would benefit from random assignment of clients to treatment conditions and a waitlist control condition. Future researchers could randomly assign participants to providers to strengthen the design of the study. A multi-site examination of the treatment would also be beneficial for examining both generalizability and increasing the number of participants.

Despite the limitations, there were many strengths of the current study. First, the study provided an initial examination of sleep treatment for internalizing disorders, adding a novel investigation to the field. Second, the study expanded on previous research to examine sleep treatment at the onset of treatment, demonstrating that sleep treatment was effective in reducing symptoms, even before behavioral treatment for the presenting problem was implemented. Third, youth with comorbid conditions were included in the study which provided a realistic, generalizable test of the effectiveness of sleep treatment for youth who commonly present for treatment. Fourth, this study included objective measurement of sleep and validated measures of child emotional/behavioral symptoms and EC. Fifth, examination of EC in relation to the sleep-psychopathology association is rare. Study 2 provided a novel examination of EC as a mediator between sleep improvement and psychopathology improvement.

Conclusions

Study 2 was a preliminary pilot study of an innovative brief sleep treatment module for both internalizing and externalizing disorders. Sleep treatment was associated with improvements in psychopathology symptoms and overall EC. Therapists are encouraged to implement sleep treatment into their work with children. Researchers are encouraged to conduct large-scale, randomized trials of sleep treatment for children.

CHAPTER 14: GENERAL DISCUSSION

The two studies in this dissertation answer questions about the associations among sleep, EC, and psychopathology. The findings generally support the hypothesis that sleep problems are correlated with psychopathology and that sleep treatment can

reduce symptoms of psychopathology. These two studies also raise new questions for future researchers to consider.

Does psychopathology reflect brain dysfunction? The current dissertation has cited research supporting the proposition that psychopathology and sleep problems reflect underlying dysfunctions in the brain. The findings of Study 1 that poor EC was associated with ADHD symptoms and the findings of Study 2 that sleep treatment led to changes in both EC and psychopathology provide some support for the conceptualization that psychopathology, sleep problems, and poor EC may be due to shared pathophysiology. Alternatively, psychopathology (e.g., ADHD) may not be indicative of a disease state or brain dysfunction status. Whether one is able to maintain a repertoire of good sleep habits corresponds with measures of EC (Nelson, Kidwell, Hankey, Nelson, & Espy, in press), and may be more important for sleep than whether someone is diagnosed with ADHD. Moreover, poor EC may be more a function of sleep quality and quantity than a reflection of dysfunctional brain processes in those with ADHD. In Study 2, sleep treatment was correlated with improvements in the overall BRIEF composite and many of the subscales, providing some support that sleep improvement leads to improved EC. However, the results of the current study are not able to address the underlying physiological mechanisms of how change in sleep can improve both mental health and EC. Future studies that investigate cellular, dopaminergic, and brain processes are necessary to fully understand the processes involved.

Do sleep problems predict poor EC? Mixed findings exist on whether sleep problems predict EC problems. There are several examples of studies finding an association between sleep and EC. Bernier and colleagues (2013) investigated the

relationship between children's sleep at 12 and 18 months old and their EC at 18 and 26 months. The researchers found that children who had a higher proportion of their sleep occurring at nighttime performed better on EC tasks later. Adequate sleep appeared to be particularly important for impulse control (Bernier et al., 2013). In experimental designs in which the amount of sleep has been manipulated, school aged children who slept less than usual each night had more impulsive behaviors and more attention problems than when they had adequate sleep (Fallone et al., 2005; Gruber et al., 2011; Sadeh et al., 2003). For example, Sadeh and colleagues (2003) tested 77 children between the ages of nine years old and twelve years old. Children whose sleep was experimentally extended by one-hour had significantly better memory and attention compared to the children who lost an hour of sleep. As some research demonstrates, sleep problems can lead to impairment in EC when children receive less sleep than is optimal for their developmental level. Conversely, some research has not found an association between sleep and EC. For instance, Schneider et al. (2016) examined the associations among sleep, EC, and ADHD. They found that sleep problems were associated with ratings of ADHD symptoms, but sleep problems were not associated with performance on EC tasks. Moreover, Nelson and colleagues (2015) found that sleep problems in preschool predicted working memory and interference suppression inhibition, but not flexible shifting or response inhibition. The study indicates that sleep problems may interfere with certain EC tasks but not others.

In Study 1, latent EC did not act as a mediator between sleep and ADHD symptoms, but rather as a moderator. When sleep problems and EC problems were both present, children experienced many more problems with attention and impulsivity. In

Study 2, it was expected that sleep improvements would lead to improved EC which would be associated with improved psychopathology. The results demonstrated that changes in objectively measured sleep duration predicted changes in EC following sleep treatment. When bootstrapping methods were used, EC acted as a mediator between sleep duration and psychopathology as assessed via the CBCL, but not the BPM. Why was a mediator model proposed for Study 2 when Study 1 focused more on moderation? Interventions are rarely as precise and theoretical as theories of causality. Although there are mixed findings on whether sleep problems predict EC problems, many studies have found an association between sleep and EC (Anderson et al., 2009; Fallone et al., 2005; Friedman et al., 2009; Gruber et al., 2011), and between EC and psychopathology (Mogg et al., 2015; Snyder, 2013), that conceptually, pathways from sleep improvement to EC improvement to psychopathology improvement were supported by research.

A meta-analysis is a way of summarizing the available literature in a rigorous and statistical way to answer questions when individual studies vary in their findings. When more mediation research has been conducted in this area, a meta-analysis may be necessary to examine the conditions under which EC is a mediator of the sleep-psychopathology relationship. Currently, there may be enough research to warrant a meta-analysis examining the association between sleep and EC. Lundahl and colleagues (2015) conducted a meta-analysis examining experimental sleep restriction on attention and hyperactivity symptoms. They found that sleep-restricted youth had worse attention, but hyperactivity symptoms did not change. However, another meta-analysis may be needed examining a range of EC abilities and various sleep problems (rather than experimental sleep-restriction or extension). The statistics underlying meta-analyses do

not currently support investigations of latent variables, but perhaps, studies may have found disparate results when individual EC tasks were used in analyses (as in Study 2) as opposed to a latent, composite EC variable (as in Study 1). Further, the way EC was assessed may explain some of the difference in findings (behavioral tasks in Study 1 and parent-report in Study 2). The literature has resulted in mixed findings and the true association between sleep and EC is unclear.

What are the clinical implications of sleep problems? Study 1 demonstrated that sleep problems are associated with increased ADHD symptoms, and Study 2 tested a sleep treatment module. There are many reasons why an effective sleep treatment module is necessary in clinical practice.

Sleep problems that have a behavioral or psychological etiology affect between 20% to 40% of children and adolescents (Blunden, 2012). The percentage of children impacted by sleep problems is particularly troubling given research has shown that childhood sleep problems predict cognitive abilities, behavior problems, emotional lability, neurological functioning, physical health problems, risk of injury, and academic success (for reviews see Beebe, 2011; Blunden, 2012; Buckhalt, Wolfson, & El-Sheikh, 2009). Multiple childhood sleep disorders exist including snoring/sleep apnea, restless legs syndrome, parasomnias (e.g., sleep terrors and sleepwalking), and insomnia (Hamilton, 2009; Kuhn & Elliott, 2003). It is estimated that between 20 to 30 percent of all children will have difficulties with insomnia (Morgenthaler et al., 2006), such as bedtime resistance, difficulties staying asleep, and non-restorative sleep.

Behavioral insomnia of childhood (BIC) is classified into two types. BIC, limit-setting type is characterized by parents who have a difficult time effectively putting their

child to bed. BIC, sleep onset association type occurs when children depend on specific cues to fall asleep at bedtime and to return to sleep when they awaken during the night (Blunden, 2012). The etiology of BIC involves many factors including biological, circadian, neurodevelopmental, environmental, and behavioral components, which fits within a socioecological model of development (Bronfenbrenner, 1986). One environmental factor that interacts with children's circadian rhythms is media use before bed. Children with a television in their bedroom watched more TV and were more likely to have a sleep problem. Each additional hour of evening media use was associated with a significant increase in sleep problems. Violent content and evening media use predicted increased sleep problems (Garrison, Liekweg, & Christakis, 2011). Given the correlation between media use and poor sleep, treatments are needed that address media use and sleep hygiene. Furthermore, parental practices may lead to the development of insomnia (Morgenthaler et al., 2006). In terms of parental practices, research has linked maternal psychological problems to their settling strategies at infancy, 12 months, and 18 months and their children's later sleep problems (Sheridan et al., 2013). Maternal involvement in infant settling was greater in mothers with depression and anxiety, and predicted less optimal sleep at five years old, which in turn, was related to child adjustment. Brief sleep treatments delivered early in development can address parenting practices, as well as the many other predictors of sleep problems.

Parents are typically the ones to report that their children have problems with sleep. Often parents find their children's bedtime resistance and frequent night awakenings problematic. For example, when a mother holds her child's hand each night to help him fall asleep, the child learns an association between falling asleep and his

mother's presence. When he awakens at night, he is unable to return to sleep unless his mother holds his hand again. As one can imagine, parents often become distressed that their child frequently awakens them at night when seeking assistance in returning to sleep (Morgenthaler et al., 2006).

The problem of insomnia extends beyond the distress parents experience due to their children's attempts to resist bedtime and frequent awakenings. Research also has demonstrated a link between short sleep duration and academic problems. Study 1 demonstrated that children with sleep problems early in development had increased teacher-report symptoms of inattention and hyperactivity in elementary school. Furthermore, delayed sleep schedules and early school start times are associated with daytime sleepiness, dozing in class, attention difficulties, and lower grades (Buckhalt et al., 2009). For children with no known clinical disorders, research has established a strong connection between the effects of daytime sleepiness and poorer academic performance as defined by teacher ratings, grades, individual and group achievement tests, and lower scores on specialized tests of neurocognitive functioning and intelligence tests (Buckhalt et al., 2009). The combination of sleep-induced attention impairments and daytime sleepiness likely worsens academic performance (Buckhalt et al., 2009).

Research has also found that the inadequate sleep was related to emotional lability and affective problems in children. Gruber and colleagues (2012a) used an experimental design to compare children who were sleep restricted to children who had extended sleep. Sleep restricted children had significantly worse emotional lability than children who had extended sleep. In addition to experiencing emotional lability, research also suggests that children who have short sleep also have less positive affect than children who had

adequate sleep (Dagys et al., 2012). Youth who experimentally received less sleep than usual, rated themselves as being more tense, anxious, angry, confused, and irritable (Baum et al., 2013). Other research has shown that poorer five-year-old sleep was associated with child anxiety, depression, and aggression based on maternal report (Sheridan et al., 2013). Similarly, in a study in which children's sleep was assessed in third grade and their internalizing symptoms were examined in 5th grade, sleep problems were associated with worse anxiety, self-esteem, and depression symptoms both cross-sectionally and longitudinally. African American children and those from lower socioeconomic status homes were at particular risk for the sleep problems-mental health relationship (El-Sheikh, Kelly, Buckhalt, & Hinnant, 2010). Finally, Blunden (2012) summarized literature demonstrating that insomnia was related to increased stress and decreased ability to cope with stressors in school-aged children.

Childhood insomnia is a common condition with multiple causal factors. Insomnia presents a problem for both parents and children, although the research indicates the problems for children may be more extensive than the distress caused to parents. Because of the widespread impact of sleep problems on nearly every aspect of children's adjustment, effective treatments (e.g., Study 2's brief sleep module) are needed that address the multifaceted causal factors of sleep problems.

When is the best time to offer interventions? Study 1 found that sleep problems assessed at *three years* were associated with ADHD symptoms in 4th grade. Study 2 offered a brief sleep intervention to children ages *six to 11 years* that was associated with improved psychopathology as measured by the BPM. However, it may be difficult to treat sleep problems in school-aged children. Children with comorbid sleep and

compliance problems may represent some of the toughest cases to treat, because children sometimes aggressively resist bedtime and are emotionally volatile due to sleep loss.

Early childhood (infancy to five years) may be an easier time to start treating sleep problems, because it may be easier for parents to return younger children to bed.

Adolescence represents a mixed case, because adolescents are often easy to motivate to get more sleep by providing psychoeducation and motivational interviewing techniques.

However, adolescents often have shifts in circadian rhythm that make it easier to stay up late, but they must awaken early due to school start times, resulting in short sleep durations. The results of the two studies in this dissertation indicate that sleep problems may be best treated in early childhood. Furthermore, the ideal path might be to prevent sleep problems from occurring and to address sleep problems as soon as they are identified early in development.

How could sleep problems be addressed from the prenatal period to early childhood? A hypothetical prevention and treatment program would focus on prevention, early diagnosis, and a brief intervention module (as described in Appendix A and examined in Study 2). Partnerships with community agencies to implement various aspects of prevention and treatment would be necessary (i.e., pediatricians, hospital staff, psychologists, social workers, teachers, and school personnel).

Prevention. Prevention efforts would begin during pregnancy and would rely on routine mental health screenings by obstetrician-gynecologists (OB/GYNs). Past research has found that maternal involvement in infant settling was greater in mothers with psychological problems and predicted worse sleep when children were five years old (Sheridan et al., 2013). OB/GYNs would provide psychoeducation emphasizing the

importance of minimizing involvement in helping infants settle, and referrals would be offered for mental health intervention. Prenatal classes offered through hospitals would deliver a thorough lesson on pediatric sleep training (Mindell et al., 2006). Prevention efforts soon after birth would involve partnerships with hospital staff to provide early psychoeducation on how to settle infants (Sheridan et al., 2013), how to set bedtime routines in infancy (Mindell et al., 2006), and the importance of allowing toddlers to return to sleep without parent interference during frequent night awakenings (Mindell et al., 2006). Parents who did not receive pediatric sleep training during pregnancy would have access to this information from nurses and other hospital staff after delivery.

Early diagnosis. Because children's sleep problems tend to persist from infancy through childhood (Beebe, 2011), a critical part of the solution is early diagnosis. Ideally, pediatricians would conduct routine screenings at each well-child visit for sleep problems. If sleep problems were identified, they would then offer education and refer the child to a pediatric psychologist for intervention. At the beginning of each school year, school psychologists and/or school social workers could implement mass screenings to assess childhood sleep and identify problems.

Intervention. As tested in Study 2, children who presented to behavioral health clinics would receive the brief sleep module at the onset of therapy. Alternatively, therapists could use the sleep module as needed during the course of therapy. As outlined in Appendix A, the sleep treatment includes sleep hygiene, sleep routines, and behavioral strategies for decreasing night awakenings and bedtime resistance. The intervention is flexible enough to be delivered by a range of healthcare providers in many different settings.

Conclusions

The two studies in this dissertation provide evidence that sleep and psychopathology are correlated, EC and psychopathology are correlated, and EC may be an important mechanism (e.g., a mediator and/or a moderator) of the sleep-psychopathology association. The studies highlight the importance of early intervention for sleep and EC problems, and Study 2 provides a test of a sleep treatment module available for clinicians to implement in therapy. A full understanding of the associations among sleep, EC, and psychopathology is critical for improving existing theories and developing more effective interventions.

CHAPTER 15: REFERENCES

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APPENDIX A: SAMPLE SLEEP TREATMENT PROTOCOL

Session 1

Psychoeducation about Sleep Problems and Emotion/Behavioral Disorders

- Children with ADHD (*substitute with information on the presenting problem*) often have a difficult time falling asleep and staying asleep.
- When children do not sleep well, they tend to have problems paying attention the next day. They may also be more emotionally reactive.
- Parents frequently feel distressed, as well.
- There are strategies that will help your child fall asleep and may help improve his/her ability to pay attention. Over the next few weeks, we will discuss strategies to improve your child's sleep. At first, you can expect for your child to resist change and things may get worse before they get better. Helping your child to improve his sleep will pay off though! [*Address any concerns the parent may have.*]

Proactive Measures

Note: As you explain each proactive measure, ask the parent if this is a problem for the child. Brainstorm how the parent can implement each recommendation that is not already established in the family. Explain topics in developmentally appropriate ways to child.

- As a parent, you will need to take proactive measures to promote good sleep.
- Encourage the development of self-quieting skills throughout the day. Your child must learn to be able to spend time alone, as well as handle his own negative emotions.
 - Be “unavailable” to him at times, and do not offer comfort for upset feelings following any natural/logical consequence (except, of course, in cases of physical pain such as a scraped knee or illness). For example, if your child refuses to wear mittens, the logical consequence is that her hands will be cold.
 - Also, catch your child being independent throughout the day and praise it! For example, you could say “Wow! I love how you got the cup of water on your own!”
- Allow your child to take one transition object to bed with him, such as a favorite blanket or stuffed animal. Make sure YOU are not the transition object your child requires!
- Eliminate all sources of caffeine (e.g., chocolate, soda) and restrict late snacks. [*Identify substitutes for soda: i.e., plain water, fruit-infused water, or water with lime or lemon juice*]
- Make your child's bedroom for sleeping only. Remove everything that distracts from sleep. Absolutely no television, videogames, or smart phone. The hamster and the dog must leave at bedtime, too. Having a roommate of any kind hurts our chances of success.
- If the house or neighborhood remains rather noisy after lights-out and the noise disrupts your child's sleep, use a white noise generator in his room such as a well-

made fan or an electronic device designed to emit white noise (no rain or seagulls, however). If you are going to use this option, use it consistently.

- There is no reason a child needs a nightlight (one in the hallway or bathroom is okay though) or to sleep anywhere other than his own bed. Remediating these issues cold-turkey should facilitate the onset of sleep, following several predictable evenings of protest. Your child's room should be dark. Close the curtains at night.
- Make sure your child's room is cool enough for sleeping.

Make a Family Schedule

Note: You will need a piece of paper and pen for the parent to write down the routine. Remind the parent to write down the routine during this activity.

- The timing of various activities throughout the day is important for promoting sleep. We are going to establish a routine that will work for your family. It is critical that you follow this routine consistently every day for the child to learn it.
- Your child should go to bed and be awakened everyday at the same times regardless of day of the week or season of the year. The flow of the day's events should be very predictable to your child. What time does your child need to wake up? Write that time down on your paper.
- I know waking your child up at the same time every day can be very difficult. At first, you may want to wake him up the same time each Sunday and school day. Waking your child up on Sunday helps prevent a miserable Monday by ensuring they do not feel "jet lagged" by constantly changing schedules. Waking your child up and putting him to bed at the same time every day may be the most important goal for you to have.
- The bare minimum of sleep that your child needs (Ferber, 1985; Friman, 2005):

○ 2 to 3 years old = 12 hours	○ 9 years old = 10 hours
○ 4 years old = 11.5 hours	○ 10 years old = 10 hours
○ 5 years old = 11 hours	○ 11 years old = 9 hr, 45 min
○ 6 years old = 10 hr, 45 min	○ 12 years old = 9.5 hours
○ 7 years old = 10 hr, 30 min	○ 13 years old = 9 hr, 15 min
○ 8 years old = 10 hr, 15 min	○ 14 years old = 9 hours
- The National Sleep Foundation recommends that your child sleep...

○ 1-3 years old = 12-14 hours a day; by 18 months naptime will decrease to once a day lasting about one to three hours
○ 3-5 years old = 11-13 hours each night; most do not nap after 5 years old
○ 5-12 years old = 10-11 hours each night; no daytime naps
- Bedtime scheduling should reflect these requirements plus the time it takes to fall asleep, perhaps a half-hour. What time does your child need to go to sleep based on the time he needs to be awake?
- Make sure your child is getting vigorous exercise throughout the day but not within a few hours of bedtime. By bedtime, he should be fatigued from the day. When would you be able to plan active time? What kind of activities does your child enjoy?
- Do not allow hot baths or showers within a few hours of bedtime; an increase in core body temperature is associated with problems falling asleep. What time would work for showering?
- An hour or so before bedtime each evening, the whole household should slow down

- and quiet down. Voices are slower and softer, the television is turned off, and interesting toys have been put away. No roughhousing or running around the house is allowed.
- Watching TV close to bedtime has been connected with bedtime resistance, difficulty falling asleep, and anxiety around sleep. What time can you turn off the TV in your house? [Encourage parent to aim for at least an hour before bedtime]

Homework Assignment

- Post the schedule where you and your child can see it (perhaps on the refrigerator?). Follow the schedule, even when your child resists.
- Identify 3 of the proactive measures to focus on this week that are the most problematic for your child. Put a check mark down on a tracking sheet for each day you followed the plan. [Remind parent that their child may put up some resistance. Normalize resistance and bolster parent's resolve to follow through with the plans.]

Session 2

Check In

- How did your child react to the new routine? How did you handle any resistance? [Commend parent for any progress they have made with following a routine. Problem-solve issues.]
- From last week, you agreed to really focus on 3 proactive measures. [Remind parent of the 3 measures they were going to focus on.] How did that go? How many days last week were you able to follow the recommendations?

Teaching Measures

- This week you will focus on teaching your child how to go to bed and stay in bed. We have many strategies that will make bedtime easier. It is important for you to follow these strategies as closely as you possibly can.
- Set two specific times 30 minutes apart (e.g., 8:00 and 8:30). The first one is for the announcement, "Time to Get Ready for Bed" and the other is for "Lights-Out." Getting ready for bed means that ongoing activities stop and teeth brushing, etc., begins. If you have more than one child, you may wish to stagger their bedtimes so you can focus on one child at a time.
- The time interval between the time your child is actually in bed, and Lights-Out, will be bonding time with either Mom or Dad. This quality time should be light and interactive, such as reading a book, recapping the day's events, and so on. This also serves as a built-in incentive for your child to get ready for bed more quickly.
- A minute before Lights-Out, point out the time on the clock, lovingly tuck your child in, provide his favorite stuffed animal and a big kiss, and say, "I love you. Have a great night. I can't wait to see you in the morning." Then LEAVE THE ROOM at exactly Lights-Out time. The door should be shut or opened just a crack to limit noise, light, and access.
- The Bedtime Pass: Before Lights-Out, give your child a poker chip (or something similar) redeemable after Lights-Out for one easily satisfied request such as getting a sip of water or one more goodnight kiss. He calls for you, you enter and obtain the pass, and grant the request all very matter-of-factly. Then back to bed. There is only

- one bedtime pass to be redeemed each night.
- Except for his use of the bedtime pass and bona fide safety concerns, you must ignore all requesting, yelling, protesting, and crying. Do not enter to correct or calm him even if he is keeping everybody awake for hours. You will avoid giving your child attention for bedtime misbehavior, because even corrective attention is rewarding for children. If you give in to your child, he will learn that he only needs to yell and cry for 30 minutes for you to come back. If you don't give in, your child will not yell for your attention, because he knows he won't get it. Bedtime will become easier after a few days of not responding. In the morning, no matter how difficult last night was for everyone, greet him with a hug and praise him for doing it all by himself!
- If he leaves his bedroom after Lights-Out (without or after using the pass), he will encounter only a robot that looks a lot like Mom or Dad but who is programmed to only walk him slowly back to bed, and who does not look at him, talk to him, or lovingly tuck him back in. You, as robot, are programmed to take him back to bed 100 times if you have to without ever slipping out of character.
- Some additional tips: To be successful with this plan, you have to be fully willing to sacrifice your sleep for one to several nights. You may wish to consider starting all of this over a long weekend devoted solely to establishing good sleep habits in your child. If your child seems to be getting pleasure out of the “robotic return” procedure, other treatment components may need to be included; be sure to tell me if he seems to enjoy the “robotic return”. If you don't hear your child leave the room, install a cowbell or similar noise-maker atop his bedroom door to alert you to his great escape.
- [Address and problem-solve all parent concerns. Help parent feel confident about their abilities to carry out this process. Correct any misunderstandings about procedures. If parent is willing, ask them to practice the strategies in your office.]

Homework Assignment

- Continue to follow the daily schedule, even when your child resists.
- Continue to focus on 3 of the proactive measures. You can pick the same or different ones depending on what your child needs help with. Put a check mark down on a tracking sheet for each day you followed the plan.
- Follow the teaching procedures exactly. Put a check mark down on a tracking sheet for each day you followed the steps.

Session 3

Check In

- Last week, I explained the teaching procedures that included giving exact times for “Time to Get Ready for Bed” and “Lights-Out,” spending time with your child before Lights-Out, using the bed-time pass, ignoring all yelling/crying, and robotically returning the child to bed. How did those procedures go in general? What problems did you run into? [Initiate problem-solving with the parent] What went well?
- How is the daily routine working for your child and family? [Commend parent for any progress they have made with following a routine. Problem-solve issues.]
- You've been working on using proactive measures. [Remind parent of the 3 measures they were going to focus on.] How did that go? How many days last week were you able to follow the recommendations?

Managing Childhood Fears

Note: Spend more or less time on this section depending on if the child has fears that interfere with sleep.

- Some children have fears that interfere with sleep. Has your child expressed any fears? First, understand that many childhood fears are typical and expected at certain ages.
 - 1 year olds are typically afraid of strangers, separation from parents
 - 2 year olds: thunder, monsters, burglars
 - 3 year olds: the dark, thunder, being alone, burglars, animals, masks/clowns
 - 4 year olds: the dark, thunder, loud sirens/alarms, animals, parents leaving at night
 - 5 year olds: the dark, injury, falling, dogs
 - 6 year olds: the dark, ghosts, someone under the bed, natural disasters, stern tone of voice
 - 7 to 10 yrs: the dark, ghosts, burglars, injury, medical and dental procedures, death
 - 10 to 18 yrs: being hit by a car, not being able to breathe, fire, injury, death, getting bad grades
- Additionally, children ages 2 to 12 average at least three fears, with up to seven by age 12.
- For age-appropriate fears, let your child know that he is not alone in having that fear and that most kids his age feel the same way right now.
- Do not talk to your child about his fear, do not try to talk him out of it, and do not try to discover why he has it. Such efforts will likely feed into it and make things worse. You may listen to your child's description of and rationale for his fear any time he brings it up, but do not respond – just listen. Toward the end of your listening session, simply say with a loving and reassuring smile, “Everyone is afraid of something when they're little.”
- Do not let your child's fear result in his avoidance of daily life. Fear of the dark should not result in use of a nightlight in his bedroom. Fear of school (i.e., school avoidance) should not result in tardiness or absence. When fear results in avoidance of what is feared, the fear grows stronger and life is increasingly not lived to the fullest. Do not accommodate or compensate for his fears (e.g., giving him “monster-repellent spray” or keeping him home from school), for if you do, you will not be part of the solution.
- Reassure your child – only at times when he is not complaining of his fear – that you take your job very seriously as the one responsible for his safety and welfare. Expect and demand his trust in you. In this context, when he complains of his fear he essentially is saying that he holds little trust in you to keep him safe and healthy.
- For bona fide fears (i.e., fears that make a lot of sense), you may have to take some action to remove the reason for the fear, and/or empower your child to advocate for himself in this regard. For example, if your child was the victim of a bully at school yesterday, you may have to contact the school to demand and ensure a safe and orderly learning environment for your child. A neighbor's unleashed pit bull is likely

something you would fear if you had to walk to school every day; you may have to contact your neighbor, or the police, if necessary.

- For fears that are not common for your child's age, or for age-appropriate fears that are causing undue and prolonged stress on family functioning, additional steps may be required. Such steps we can discuss if necessary will likely involve techniques employing systematic desensitization, increasing your child's perception of predictability and control over the feared situation, and/or altering the social and/or physical environment as indicated.
- If you suffer from fear and anxiety, seek help for yourself in order to minimize or eliminate any cues or accommodations you may inadvertently be giving your child that may be maintaining or worsening his fear and anxiety. Do you think that your fears could be interfering? I can give you a referral if you need it.

Review

- We have covered a lot of information in the last few weeks. Let's spend some time reviewing the information.
- [Review proactive measures and check on status of each one. Look for places where the parent could improve. Problem-solve barriers to implementation.]
 - Self-quieting skills? Child spending time alone? Child handling negative emotions? Parent praising independence?
 - One transition object to bed?
 - Eliminating all sources of caffeine? Restricting late snacks?
 - Is the child's bedroom for sleeping only? Have you removed everything that distracts from sleep? Absolutely no television, videogames, or smart phone? Any pets at bedtime?
 - Consistently using noisemaker at bedtime if needed to block out noisy neighborhood?
 - Removing the nightlight from the room?
 - Child only sleeping in his own bed?
- [Review teaching measures and check on status of each one. Look for places where the parent could improve. Problem-solve barriers to implementation.]
 - Have you set two specific times 30 minutes apart (e.g., 8:00 and 8:30)? The first one is for the announcement, "Time to Get Ready for Bed" and the other is for Lights-Out.
 - Spending time with child when child is in bed before Lights-Out?
 - Leaving the room right at Lights-Out? Saying "I love you. Have a great night. I can't wait to see you in the morning"? Shutting door to block out light?
 - Using the Bedtime Pass only once and very matter-of-factly?
 - Ignoring all requests, yelling, protests, and crying? Praising child in morning for doing it all by himself?
 - Robotically returning child to his room?

Homework Assignment

- Continue to follow the daily schedule, even when your child resists.

- Continue to focus on 3 of the proactive measures. You can pick the same or different ones depending on what your child needs help with. Put a check mark down on a tracking sheet for each day you followed the plan.
- Follow the teaching procedures exactly. Put a check mark down on a tracking sheet for each day you followed the plan.

Appendix A References

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Appendix A Goal Tracking Sheet

	Mon	Tue	Wed	Thur	Fri	Sat	Sun
Proactive Sleep Goal:							
Proactive Sleep Goal:							
Proactive Sleep Goal:							
Proactive Sleep Goal:							
Teaching Measures followed almost exactly?							

Directions: In the proactive sleep goal box, write down your goal for the week (e.g., eliminate all caffeine from child's diet). Place a check mark for each day that you met your goal. Place a check mark for each day you followed the teaching measures very closely (e.g., announce time to get ready for bed, bond with child, announce lights out, leave room at the exact time, etc.).