ACTIGRAPHIC EVALUATION OF SLEEP DISTURBANCE IN YOUNG CHILDREN

by

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Sleep studies have rarely explored individual differences in sleep disruption and associated outcomes at early ages. In two studies, this dissertation addresses both of these limitations using actigraphy, an activity-derived assessment of sleep, to increase understanding of negative impacts of sleep on early development. Study 1 investigated sleep disruption in foster children and sleep-related treatment outcomes of the Multidimensional Treatment Foster Care for Preschoolers (MTFC-P) intervention program. Study 2 explored individual differences in the associations among sleep, children's behavior, and neurohormonal activity. Four groups of participants ages 3- to 7-years-old were included in both studies: 1. Regular foster care (RFC; n=15); 2. MTFC-P intervention (TFC; n=17); 3. Low-income community (LIC; n=18); and 4. Middle-income community (MIC; n=29).

Results of Study 1 indicated greater sleep disruption in foster groups, as evidenced by longer sleep latencies and increased variability of sleep duration, in the TFC group than in community groups. There was also indication of a treatment effect as the TFC group slept longer than RFC and LIC groups and had earlier bedtimes, fell asleep earlier, and spent more time in bed than either community group. LIC children had marginally more active sleep than MIC children, indicating a possible role for socioeconomic status in sleep quality.

In Study 2, correlational and causal modeling approaches were used to investigate associations among sleep disruption, problem behaviors, and diurnal cortisol. Influences of foster care placement, gender, and age were also examined as potential individual difference factors. Results of mixed linear autoregressive models indicated that children were more likely to display inattentive/hyperactive behaviors after shortened sleep durations. Furthermore, at lower sleep durations, differences among care groups and genders emerged as children in foster care and males were at heightened risk for inattentive/hyperactive behavior problems. No associations between sleep and disruptive problem behaviors were found and there were few associations with morning and evening cortisol values.

Results of these studies are discussed in terms of the effectiveness of the MTFC-P program for addressing sleep problems in foster children. Additionally, clinical implications of the heightened likelihood of inattentive/hyperactive behavior problems after disrupted sleep in some children are discussed.

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To my parents who taught me the virtues of a good night sleep.

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CHAPTER I

INTRODUCTION

Project Inception

This dissertation investigating early sleep patterns originated as part of a larger study of foster children. The Early Intervention Foster Care (EIFC) study was a response to the observation that foster children are at elevated risk for a variety of poor outcomes including increased psychopathology, cognitive delays, and stunted growth. Of specific interest was the earliest segment of the foster care population. Preschool-aged foster children appeared to be most vulnerable to negative impacts of foster care placement and had the poorest outcomes of any segment of foster children. Furthermore, children under the age of six represent a large proportion of children in foster care; approximately one third of all children. Many of the intervention resources are disproportionately distributed to adolescents in foster care because the problem behaviors of these children have a greater impact on society as many adolescents in foster care experience school failure, criminal justice involvement, and harm toward themselves or others.

At Oregon Social Learning Center (OSLC), Fisher and colleagues created Multidimensional Foster Care for Preschoolers (MTFC-P) in an effort to address the early needs of preschool-aged children in the foster care system. MTFC-P was designed as an early intervention approach to prevent delays and emotional problems from developing

into the concerning problems seen in older foster children. In the mid 1990's, Fisher and colleagues extended the existing Multidimensional Treatment Foster Care program (Chamberlain, 2003a, 2003b; Chamberlain & Fisher, 2003), originally developed to address chronic deviance in adolescent foster children, to address the needs of preschool-aged foster children. Although the MTFC-P program was designed to help young children meet developmental milestones and facilitate early learning, it retained the core principles of MTFC for adolescents. Specifically, both programs emphasize consistent reinforcement, effective limit setting, and close supervision.

As part of their EIFC randomized efficacy trial of MTFC-P, Fisher's group at OSLC sought to understand how early stressors experienced by foster children may impact their underlying basic neural mechanisms. The OSLC group developed collaborations with field experts on early life stress, such as Gunnar and others, to develop a model of the impacts of stress in young children. From observations of young foster children and internationally adopted children, and information gained from animal models of behavior, they created a conceptual model of impacts of early life stress on underlying processes and from this model began generating questions of plasticity of these underlying systems. Their conceptual model highlighted the importance of high quality caregiving as a protective mechanism against the negative effect of early life stress. Caregivers who are consistent and highly engaging are thought to help regulate the experiences of early stress, thereby protecting their children from developing adverse neurobiological responses to stressful events. Children who do not have a caregiver to help with stress regulation must attempt to manage it on their own. This has been

associated with increases or decreases in cortisol levels, a stress hormone, and abnormal electroencephalographic asymmetry. From this model, the MTFC-P intervention selected the foster care provider as a means for delivering consistent and effective caregiving so that children may develop new, and more adaptive, patterns of stress responding.

As part of the EIFC study, cortisol was collected on regular foster care children, low-income community comparison children, and children in the MTFC-P program to identify any effects placement in the MTFC-P program may have on these basic processes. Early in their study of diurnal patterns of cortisol in foster children and internationally adopted children, Fisher's group noted that the diurnal patterns were different from those children living with their biological parents. The community children showed the expected morning peak with cortisol decreasing throughout the remainder of the day and night. Foster children did not have the morning peak and some showed little change in cortisol across the day. Since they did not have 24 hour samples of cortisol, it was unclear whether the foster children had no cortisol peak, or whether this peak was shifted to occur at a time that they had not collected cortisol data, such as the middle of the day or night. Since there has been a clearly documented relationship between diurnal patterns of cortisol and circadian sleep rhythms, these aberrant cortisol patterns led to questions about whether sleep disruption was driving abnormal cortisol in the children in foster care.

These dissertation studies directly emanated from the early findings of abnormal cortisol in foster children in the EIFC study. To help the group understand how the cortisol patterns and sleep were related in these children, Fisher's group at OSLC

collaborated with Avi Sadeh, a field leader in young children's sleep. Through this collaboration, Sadeh assisted the OSLC team in collecting objective sleep data, via actigraphy, in a subset of children in the EIFC groups. Upon early inspection of the data, the groups did not look especially different on most measures of sleep. However, sleep across all children in the sample looked more disrupted than the sleep information collected by Sadeh on same-aged middle-income children. This discrepancy indicated that income may be an important determinant of children's sleep, and may be even more influential than placement in foster care. A group of upper middle-income children were collected as a comparison group as an effort to separate these potential effects. The dissertation studies investigate sleep across these four groups of children in 1. Regular foster care (RFC); 2. MTFC-P foster care intervention (TFC); 3. Low-income families (LIC); and 4. Upper-middle income families (UMC).

The first study of this dissertation investigates individual differences in vulnerabilities for sleep disruption using actigraphy. Specifically, it investigates sleep differences among foster children, which is an understudied, yet high risk group for sleep disturbance. The second study in this dissertation investigates individual differences in problem behaviors and cortisol following sleep disruptions. Moreover, this second study examines how variability in sleep measures can contribute to dysregulation in children's behaviors and neurohormonal activity. Prior to discussion of these studies, a review of the current sleep literature will be provided as a foundation for the dissertation studies. This review will provide an overview of sleep organization, impacts of sleep restriction, the importance of sleep in childhood, and current sleep assessment approaches. The

dissertation will conclude with a discussion of how these studies may contribute to our understanding of sleep in foster care and general influences of sleep in early childhood. Furthermore, the efficacy of the MTFC-P intervention for sleep disruption in foster care will be discussed.

Review of the Sleep Literature

Sleep Architecture

Sleep is a complex and multi-stage phenomenon which is thought to occur in five stages describing increasingly "deeper" states. Individuals typically cycle through the stages approximately every 60 to 90 minutes throughout the night if sleep is not disturbed, but the time spent in each stage is thought to vary throughout development (Berger, 1969b; Colten & Altevogt, 2006; Dahl, 1996). The sleep stages are further categorized into non-rapid eye-movement (NREM) and rapid eye-movement (REM) sleep. Sleep is typically entered through early stages of NREM sleep (Stages 1 and 2) and progresses into slow wave sleep (SWS), which characterize the deeper NREM stages 3 and 4. REM sleep generally follows Stage 4 sleep and comprises approximately 20 to 25% of total sleep (Colten & Altevogt, 2006). The later stages of sleep (e.g. Stage 3, Stage 4, and REM) are considered the most recuperative stages as disruption of these stages is most related to impairment of cognitive functioning and feeling "sleepy". However, the actual mechanism of recuperation in each of the stages remains unknown.

Polysomnography research indicates that individuals begin sleeping in Stage 1 sleep, which is the lightest stage and most closely resembles a wake state. Stage 1 sleep is

typically maintained for less than seven minutes at sleep onset, but individuals return to this stage periodically throughout the night, and most commonly after REM sleep. Individuals are easily interrupted in this sleep state and many times report that they were not sleeping. Most studies have indicated that Stage 1 sleep provides little to no recuperative value and is simply a transition state from wakefulness to deep slow wave sleep (Wesensten, Balkin, & Belenky, 1999).

Stage 2 sleep is a slightly deeper stage of sleep than is Stage 1 and constitutes approximately half of the total sleep time. Although it is considered a light stage of sleep, there is evidence that Stage 2 sleep provides some recuperative value and that it may provide a minor contribution to the memory consolidation process (Colten & Altevogt, 2006).

Sleep stages 3 and 4, the slow wave sleep stages, are thought to be imperative for restorative sleep and occur primarily in the first third of the night. These are the deepest NREM stages which are characterized by almost a complete loss of consciousness for both external and internal stimuli. Individuals in these stages are difficult to wake and many times are disoriented if sleep in these stages is disrupted (Bonnet, 1985; Dahl, 1996). Stage 3 sleep lasts a few minutes per cycle and Stage 4 sleep lasts approximately 20 to 40 minutes and comprises 10 to 15% of total sleep. The amount of nightly SWS tends to vary in direct proportion to the amount of sleep debt, or wake time, incurred. Laboratory studies have documented positive relationship between sleep debt, or amount of lost sleep, and relative amount of SWS compared with other sleep stages. After a night of disrupted sleep or sleep deprivation, individuals spend a greater percentage of sleep

time in SWS although total sleep does not increase (Bonnet, 1985, 1986; Dahl, 1996; Philip, Stoohs, & Guilleminault, 1994; Wesensten, Balkin, & Belenky, 1999).

REM sleep is commonly called paradoxical sleep because it is comprised of aspects of both light and deep sleep (Berger, 1969a; Colten & Altevogt, 2006; Dahl, 1996). The REM sleep stage is characterized by muscle atonia, or sleep paralysis, and rapid eye movements. Physiological outputs such as sympathetic activity and respiration are many times elevated in comparison with wake states. Unlike SWS, the REM sleep cycles are relatively short at the beginning of sleep and the longest periods occur primarily in the latter half of the night. When awoken from REM sleep, many times, individuals report that they had been dreaming.

Slow wave sleep and REM sleep have been considered the most important sleep stages as they have been strongly implicated in the learning and memory consolidation process. Individuals who are learning new tasks as well as infants and children who are undergoing extraordinary amounts of neurodevelopment and learning sleep a substantially longer percentage of each day than do older children and adults. Children have relatively large amounts of SWS compared to adults, which has been found to peak between the ages of 3 and 6 and steadily decline thereafter (Dahl, 1996). One explanation of this phenomenon is that during SWS, the bulk of learning and neural pathway solidification occurs. This hypothesis has been explicitly tested in animal and adult studies where subjects are taught a new task. SWS and REM sleep increases during sleep periods after learning the task, but returns to baseline after the task is mastered (Maquet, 2001). Another study tested the effects of sleep deprivation on learning by teaching adult

participants a task and either allowing sleep immediately after learning or subsequently depriving sleep for 30 hours and then allowing two nights of recovery sleep (Stickgold, James, & Hobson, 2000). Participants who were allowed to sleep immediately after learning improved performance on subsequent tests and continued to improve over the next week. Those individuals who were deprived of sleep did not show improvement upon retest and did not improve over the following week. The authors concluded that if sleep does not occur within a specific window of time, the learning consolidation process may be permanently interrupted.

Impacts of Sleep Loss

The impacts of sleep loss and sleep deprivation have been studied extensively in adult samples and to a lesser extent using child samples. The results of these studies have uncovered deficits in functioning associated with sleep loss spanning cognitive degradation to poor emotion regulation (Dahl, 1996; Pilcher & Huffcutt, 1996). As a striking example, Pilcher and Huffcutt (1996) reported in their meta-analytical study that sleep-deprived individuals who fell in the 50th percentile for their group on a combination of motor, cognitive and emotional tasks, performed equivalently to individuals scoring in the 9th percentile of a group of non-deprived individuals.

Studies investigating the effects of disrupted sleep have done so by completely depriving participants of sleep for at least 24 hours, partially depriving sleep by significantly reducing typical sleep time, or fragmenting sleep by continuously interrupting sleep throughout the night. Widespread deficits in functioning have been associated with each of these types of sleep disruption. Surprisingly, participants in

fragmentation studies show equivalent or greater impairment than participants in total sleep deprivation conditions (Bonnet, 1985, 1986; Jones & Harrison, 2001; Pilcher & Huffcutt, 1996; Wesensten, Balkin, & Belenky, 1999). This phenomenon has been explained by the observation that sleep fragmentation significantly increases non-recuperative Stage 1 sleep and impairs the ability to sustain slow wave NREM and REM sleep (Bonnet, 1985). Practically speaking, this line of research suggests that individuals who wake up consistently throughout the night may feel less rested and have greater impairment in daytime functioning than individuals experiencing insomnia, or difficulty initiating sleep.

Sleep Deficits and Cognitive Functioning

Regardless of whether sleep is completely deprived or whether sleep is continuously interrupted, or fragmented, the most consistently affected cognitive functions are those that are primarily associated with activation of the prefrontal cortex (PFC), which is implicated in goal-directed behavior and executive functions (Dahl, 1996; Drummond & Brown, 2001). Tasks with low cognitive demand, such as rote procedural and motor ability, appear to remain intact even after extensive periods of sleep deprivation (Pilcher & Huffcutt, 1996; Randazzo, Muehlbach, Schweitzer, & Walsh, 1998).

The majority of studies investigating cognitive impairments associated with sleep have used adult participants and have restricted sleep in a laboratory setting. However, several child and adolescent studies have noted cognitive deficits similar to those seen in adult samples. Two noteworthy adolescent studies found similar cognitive deficits as

adult studies after inducing both complete and partial sleep deprivation (Carskadon, Harvey, & Dement, 1981; Randazzo, Muehlbach, Schweitzer, & Walsh, 1998). Interestingly, the adolescents in both studies only showed decrements in performance on complex cognitive tasks that have been associated with PFC activation. One limitation of both of these studies is that sleep disturbance was artificially induced and was monitored in the lab, thereby limiting extension of the findings to naturalistic sleep disruption.

In one of the few naturalistic actigraphy studies, Sadeh, Gruber, and Raviv (2002) also found that performance differences in young children were limited to complex cognitive tasks. They investigated the relationship between neurobehavioral functioning, measured by a battery of executive functioning tasks, and quality of nightly sleep without a prescribed alteration in schedule in school-age children. Consistent with prior research, they found that performance on only the most cognitively taxing tests in the neurobehavioral battery was associated with fragmented sleep. In a follow-up study, Sadeh and colleagues (2003) experimentally manipulated school-aged children's sleep schedules to either add or subtract an hour to their normal sleep duration. They found that children who extended their sleep one hour performed significantly better on complex neurobiological tasks, while those children who restricted their sleep one hour did not change in performance. Alternatively, on a test of simple reaction time, the sleep restricted children's performance worsened while the sleep enhanced children showed no change in performance. One advantage of both of these studies is that all sleep occurred in the child's home with typical bedtime routines instead of in a laboratory, as in the adolescent studies.

Sleep Deficits and Emotion Regulation

The literature has identified a bidirectional relationship between sleep and emotion. While sleep disruption is a hallmark symptom of many psychological and medical disorders (e.g. depression, post-traumatic stress disorder, and attention deficit hyperactivity disorder), problems with emotion regulation also occur after sleep disruption (Bursztein, Steinberg, & Sadeh, 2006; Dahl, 1996; Sadeh, Hayden, McGuire, Sachs, & Civita, 1994). Sleep has been described as a "barometer to psychological stress" reflecting the close relationship between stress, emotional disturbance, and sleep disruptions (Tikotzky & Sadeh, 2001). Sleep problems have been associated with internalizing-type regulatory problems and have also been linked with a reliance on emotion-focused coping styles (Dollinger, 1986; Fisher & Rinehart, 1990; Sadeh, Keinan, & Daon, 2004). Likewise, sleep disruption in children has been closely linked to the experience of traumatic events, which many times are also associated with affective dysregulation (Hillary & Schare, 1993; Rimsza, Berg, & Locke, 1988; Sadeh, 1996).

Although there are seemingly strong associations between subjectively-reported sleep disturbance and depressive symptoms, studies using objective measures, such as polysomnography (PSG), have been less conclusive. Some studies using PSG have found support for a delay in entering the rapid eye movement (REM) stage of sleep in depressed children rather than impaired duration or quality (Emslie, Rush, Weinberg, Rintelmann, & Roffwarg, 1990), although others have not found any differences (Bertocci et al., 2005). Limited in number, studies using actigraphy have also been somewhat inconclusive and have shown less robust findings than have been reported subjectively.

In a study of children on an inpatient unit, Sadeh and colleagues found negative associations between depressive symptoms and sleep efficiency and some indication of a delay in sleep onset associated with increased hopelessness (Sadeh et al., 1995). However, non-clinical studies of young children have not found any associations between sleep quality and internalizing domains of behaviors, although they did find increases in externalizing behaviors (Aronen, Paavonen, Fjallberg, Soininen, & Torronen, 2000; Lavigne et al., 1999; Sadeh, Gruber, & Raviv, 2002).

Sleep restriction studies have also demonstrated the opposite causal relationship and individuals deprived of sleep commonly report significant decreases in feelings of happiness and increases in negative mood (Carskadon, 2002). Individuals who are sleep deprived exhibit greater irritability and respond with negative emotionality more quickly than if they were fully rested (Dahl, 1996). Mood deterioration is arguably the strongest impact of sleep deprivation. A meta-analysis of experimentally restricted sleep indicated that the effect of mood deterioration was over twice as large as impairment of cognitive or motor functioning (Pilcher & Huffcutt, 1996). Sleep restriction has been primarily studied in adolescents and adults, so it is unclear whether these results may be generalizable to a younger sample.

From the prior literature, it is unclear what mechanisms may account for the associations between negative affect, anxiety, and sleep disturbance. Although sleep restriction studies suggest that internalizing behaviors may increase after a poor night of sleep, other studies suggest that depressed mood and increased anxiety may be the mechanism that drives sleep disturbances. Furthermore, polysomnography studies

indicate that the sleep disruption may be a problem with aberrant sleep architecture and that other sleep characteristics may not be affected.

Importance of Sleep in Early Childhood

One consistent correlate of sleep disruption in young children has been poor school performance and school adjustment (Gozal, 1998; Meijer, Habekothe, & Van Den Wittenboer, 2000). Children who sleep poorly or inconsistently tend to earn lower grades, have more school-based behavioral problems than children who consistently get high quality sleep, and are rated as less socially competent by their teachers.

Early sleep disruption may not only be related to concurrent deficiencies in daily function, it may also be a marker of biological risk for later cognitive, emotional, behavioral, and developmental problems (Dollinger, 1986; Halpern, Maclean, & Baumeister, 1995; McCracken, 2002). For example, adult studies have found that in depressed individuals, sleep abnormalities continued once depressive symptoms subsided, indicating that sleep disruption may be directly related to vulnerability for depression (McCracken, 2002). One important study demonstrated that risk for affective disorders was three times greater in relatives of depressed individuals that showed reduced onset of REM sleep (Giles, Biggs, Rush, & Roffwarg, 1988).

Clinically, sleep problems have been considered a hallmark symptom of ADHD due to the prevalence of parent-reported disturbance (Gruber, Sadeh, & Raviv, 2000). However, mean-levels of objective measures of sleep duration and quality do not consistently verify these reports (Corkum, Tannock, & Moldofsky, 1998; Sadeh, Pergamin, & Bar-Haim, 2006). Instead, night-to-night instability of sleep has been

consistently associated with diagnosis of ADHD and sub-threshold occurrence of symptoms (Gruber & Sadeh, 2004; Gruber, Sadeh, & Raviv, 2000; Owens, 2005).

Symptoms of attention-deficit/hyperactivity disorder (ADHD) are arguably the most studied consequences of sleep disturbance in early childhood. Measures of sleep variability have successfully discriminated between children with and without an ADHD diagnosis (Gruber, Sadeh, & Raviv, 2000). The prominent features of ADHD are problems with attention modulation, poor behavior and emotion regulation, and overactivity. It has also been linked to sleep conceptually as it has been considered a disorder of arousal. Furthermore, neuroanatomical centers that regulate sleep are thought to substantially overlap with pathways regulating attention, implicating that impaired attentional abilities may be directly caused by insufficient sleep.

A large number of studies have investigated inattentive and hyperactive behaviors in relation to sleep-disordered breathing (SDB), which is commonly marked by snoring and open-mouthed breathing. SDB may increase the occurrence of inconsistent and fragmented sleep thereby interfering with children's ability to remain in the deepest and most restorative stages of sleep (i.e., Stages 3 and 4; Kennedy et al., 2004; O'Brien et al., 2003; Weissbluth, Davis, Poncher, & Reiff, 1983). In her review article, Owens (2005) suggests that the mounting evidence linking SDB and inattentive and hyperactive behaviors may indicate that ADHD is a disorder of hypoactivity rather than hyperactivity. If sleep is frequently fragmented, children may experience continual feelings of drowsiness and hyperactivity may be a compensatory mechanism to counteract the urge to sleep (Owens, 2005).

In a study investigating the associations between sleep disruption and conduct disorder, Chervin and colleagues (2003) found striking evidence that sleep fragmentation secondary to parent-reported sleep disordered breathing (SDB) and periodic leg movements during sleep (PLMS) occurred more frequently in young children displaying highly oppositional, aggressive, and fighting behaviors. As the authors point out, the effects were quite large and children displaying symptoms of SDB and PLMS were two to three times more likely to be rated higher on a conduct disorder scale than children without disrupted sleep.

Likewise, it has been acknowledged that sleep as a stable individual trait can also be influenced by environmental variables. Infancy studies have been able to predict developmental status and regulatory abilities in one year old infants from sleep quality variables collected within the first three months of life (Anders, Keener, & Kraemer, 1985; Burnham, Goodlin-Jones, Gaylor, & Anders, 2002). These studies have also found that environmental influences, such as caregiver sensitivity and low family stress, begin to predict developmental outcomes after the first year. In summary, sleep in children is clearly a product of the complex interaction between individual trait-based vulnerabilities and environmental influences.

Assessment of Sleep

Sleep has been measured in a variety of ways in the literature. The gold-standard has been polysomnography (PSG), which is a multi-channel assessment of brain activity with electroencephalography (EEG), muscle tone with electromyography (EMG), eye movement with electrooculography (EOG), and cardiac activity with electrocardiography

(EKG; Halpern, Maclean, & Baumeister, 1995; Sadeh, Hauri, Kripke, & Lavie, 1995). Although PSG provides an immense amount of data, many studies do not elect to use it due to prohibitive cost and requirement that sleep is monitored in the lab. Participants in PSG studies must stay in the sleep laboratory for five days or longer to complete a study. Furthermore, there has been criticism that PSG studies are not naturalistic as participants may not adjust to the unfamiliar surroundings.

The majority of studies utilize sleep diaries for the collection of self-report or parent-report of sleep quality and duration. Typically, participants record sleep and wake times as well as subjective reports of sleep quality in the diaries over consecutive nights. Some strengths of this method are the relatively low cost and unobtrusive nature. Some authors caution against solely relying on this method of assessment due to the high subjectivity and in child studies, the requirement that children must signal their parents in order for them to be able to report sleep difficulties (Sadeh, Lavie, Scher, Tirosh, & Epstein, 1991).

A promising assessment approach that combines ease of home assessment with high reliability is actigraphy (Sadeh & Acebo, 2002). An actigraph is a small watch-like device worn on the wrist or the ankle that can continuously collect data for up to two weeks. Actigraphy measures activity level in 1-min epochs and uses algorithms to assess whether the activity is associated with sleep or wake states. An algorithm converts acceleration of activity into numerical form and from these data, computes variables such as sleep onset, periodic wake episodes, and wake time. Recent studies of sleep algorithms (e.g. Sadeh and Cole-Kripke) validated against PSG have reported correspondence of

measures up to 93% in adults and 89.9% in children (Sadeh, Hauri, Kripke, & Lavie, 1995; Sadeh, Lavie, Scher, Tirosh, & Epstein, 1991; Sadeh, Sharkey, & Carskadon, 1994).

Although actigraphy is a promising means of collecting sleep data, it has been underused in the child sleep literature. Due to its unobtrusive nature and naturalistic quality, in the currently proposed studies, we elected to use actigraphy to assess young children's sleep over the course of five nights.

CHAPTER II

STUDY 1: EARLY SLEEP DISRUPTION IN CHILDREN IN FOSTER CARE

Introduction

The multitude of stressors commonly experienced by children in foster care may place these children in at high risk for sleep disruption. Children in foster care are vulnerable to a host of other regulatory problems that heighten the likelihood for adverse developmental trajectories. Past research has found that cognitive delays, deficits in emotion regulation, and behavioral problems are widespread in these children. In fact, Klee, Kronstadt, and Zlotnick (1997) reported that 80% of young foster children struggle with developmental or emotional delays and numerous other health concerns. Even more striking, they reported that nearly 50% of the children showed delays across multiple areas of functioning. Children in the foster care system are also at higher risk for substance use (Hurlburt et al., 2004), poor academic outcomes (Stock & Fisher, 2006), and disrupted growth (Pears & Fisher, 2005a).

The negative outcomes seen in foster children are very similar to impairments secondary to sleep disruption. Sleep is consistently implicated in the regulation of emotion, cognitive functioning, and behavior. Although the exact function of sleep remains a mystery, researchers have found that sleep plays a key role in daytime alertness and functioning (Sadeh, 2007). Disrupted sleep has been linked with difficulties with

sustained attention (Stores, 1999), working memory (Steenari et al., 2003), and executive control (Dahl, 1996), which has led researchers to address the potential overlap between sleep disruptions and the diagnosis of attention deficit hyperactivity disorder (Blader, Koplewicz, Abikoff, & Foley, 1997; Corkum, Tannock, & Moldofsky, 1998; Gruber & Sadeh, 2004). In both children and adults, disrupted sleep has been related to cognitive impairment, especially in complex tasks, emotional dysregulation, and poor behavioral regulation marked by increased impulsivity, aggression, inattention, and hyperactivity (Buckhalt, El-Sheikh, & Keller, 2007; Dahl, 1996; Owens, 2005; Sadeh, Gruber, & Raviv, 2002; Sadeh et al., 1995). Furthermore, sleep disruptions have been associated with depressive and anxious symptomology (Blader, Koplewicz, Abikoff, & Foley, 1997; Dahl & Harvey, 2007) as well as aggressive and delinquent behavior (Aronen, Paavonen, Fjallberg, Soininen, & Torronen, 2000; Chervin, Dillon, Archbold, & Ruzicka, 2003), although no causal links have been determined.

Among young children, it has been estimated that between 20% to 42% experience sleeping problems (Anders & Eiben, 1997; Kataria, Swanson, & Trevathan, 1987; Mindell, 1993; Paavonen et al., 2000). While some of these early sleep disturbances are mild and transient across development, a large portion of early sleep problems, 41% by some estimates, persist throughout childhood (Blader, Koplewicz, Abikoff, & Foley, 1997; Kataria, Swanson, & Trevathan, 1987; Zuckerman, Stevenson, & Bailey, 1987). Sleep disruptions have been considered especially problematic in childhood due to the enduring nature of the problem, the increased stress experienced by the family, and the negative implications for developing regulatory systems.

Consequently, children with enduring sleep problems are likely more vulnerable to negative psychosocial outcomes.

The current study investigates early sleeping patterns among foster children and several relevant comparison groups. It takes a descriptive approach to understanding potential differences in sleeping behaviors of foster children and both low-income and upper-middle income community children living with their birth families. The community children serve as comparison groups so that the influences of foster care can be separated from potential economic influences. Furthermore, sleeping patterns of foster children participating in the Multidimensional Treatment Foster Care for Preschoolers (MTFC-P) intervention are investigated to better understand whether early intervention may have any influence on sleeping behaviors in young foster children.

Stressful Early Experiences and Sleep in Foster Children

The ability to initiate and maintain sleep is closely related to aspects of stress regulation and the experience of vigilance. Dahl (1996) proposed that individuals who regularly experience high levels of vigilance or who perceive their environments as unsafe are vulnerable to sleep disturbance. That is, both the presence of stress or environmental threat and the absence of safety are associated with sleep disruptions. Sleep and arousal (i.e. heightened vigilance) mark competing and incompatible states that are greatly impacted by perceptions of safety versus threat. Past research suggests that perceptions of safety are essential for humans to engage in sleep (Dahl, 1996; Sadeh, 1996). On a neurobiological level, many of the brain systems that are central to sleep and arousal overlap substantially with the systems that are central to stress responsivity and

vigilance associated with heightened arousal. For example, the hypothalamo-pituitary-adrenal (HPA) axis is closely related to regulation of sleep and arousal cycles as well as to the stress response (Mignot, Taheri, & Nishino, 2002; Van Reeth et al., 2000). As highlighted by Dahl in his 1996 review of the developmental sleep literature, the locus coeruleus (LC) may also be an especially important brain structure involved in these neural processes. The LC has been implicated in the regulation or dysregulation of sleep, vigilance/arousal processes, the stress response, and affect. The LC receives projections from areas of the limbic system, including the amygdala as well as the hypothalamus. It is thought to be instrumental in vigilance and stress responses associated with panic and other anxiety disorders as well as in problems with sleep and arousal associated with sleep disorders (Berridge & Waterhouse, 2003).

For short periods of time, humans are able to resist physiological urges for sleep, which is an adaptive process that allows for addressing potential environmental dangers. However, it is impossible for individuals to resist sleep for long periods of wakefulness, and eventually, the need for sleep prevails, although this sleep may be compromised. Dahl (1996) argues that if sustained vigilance consistently interrupts the balance between sleep and arousal in early development, there may be important ramifications for later development of sleep disorders.

Many of the negative outcomes seen in foster children emerge secondary to the experience of early stressors. Some of the most extreme examples of early life stress, such as severe maltreatment or neglect and losing a primary caregiver, are associated with placement in the foster care system. The majority of foster children have histories of

exposure to multiple stressors prior to entering the foster care system including parental substance abuse, parental imprisonment, and parental mental health problems (Leathers, 2002). In addition to these parenting stressors, most children who enter the foster care system after infancy have experienced some type of maltreatment. Prior research suggests that exposures to acute or chronic traumatic stressors, such as maltreatment experiences, directly causes marked impairment of sleep and interference with sleep architecture in children (Moore, 1989; Rimsza, Berg, & Locke, 1988; Sadeh, 1996; Sadeh, Hayden, McGuire, Sachs, & Civita, 1994; Sadeh et al., 1995). Sleep disruption is the most frequent non-specific consequence of exposure to stress in children due to the associated hypervigilant response (Sadeh, 1996). It has been documented that maltreated children display heightened baseline vigilance as compared with non-maltreated children (De Bellis, 2001; Rieder & Cicchetti, 1989).

Sleep disruption secondary to increased fear and anxiety, may have profound impacts on children's daytime functioning. Disrupted sleep, either shortened duration or impaired sleep quality, may preclude children from engaging in the deepest stages of steep (Stage 4), which is also associated with the greatest amount of restorative benefits (Dahl & Harvey, 2007). However, for some children with histories of maltreatment, it may not be adaptive to enter into the deeper stages of sleep because in these stages, individuals are least responsive to their environments. While foster children may not perceive immediate threat in the foster home, they may continue to perceive their environment as lacking safety, due to uncertainty about their environment and their care provider. To enter into the deepest sleep stages requires that individuals feel secure about

the safety of their surroundings (Dahl & Harvey, 2007), and this may not occur for some children in the foster care system.

Associations between sleep disruptions and child maltreatment have been assessed using parent- and self-report measures as well as objective indicators, such as the activity-derived measure of actigraphy. However, there is some indication that subjective reports of these associations may be inflated by reporter expectations of sleep disruption following stressful experiences (Sadeh, 1996). The current study will add to the growing body of research using actigraphy, a reliable measure of sleep. Results of actigraphy studies have also suggested that sleep disruption is a common outcome of maltreatment. For example, Sadeh and colleagues found that physically abused children on an inpatient unit experienced greater objectively-measured sleep disruption (i.e. extensive nighttime awakening, increased active sleep) than other children (Sadeh et al., 1995). Although these findings have since been replicated (Glod, Teicher, Hartman, & Harakal, 1997), other studies have also reported increased frequency of sleep disturbance following sexual abuse (Goldston, Turnquist, & Knutson, 1989; Rimsza, Berg, & Locke, 1988).

Past research on foster children suggests that maltreatment experiences in these children are associated with negative outcomes, while placement in foster care may increase the risk for problematic long-term consequences. Children in foster care frequently experience ongoing stress related to disruption from their primary attachment figure, decreased feelings of safety while living in a new home, and transitions between foster placements (Field, 1996).

Although it is beyond the scope of the current study to investigate potential causal mechanisms of sleep disruption in foster children, it is possible that these children may be at increased risk for disrupted sleep due to low reliance on their caregivers for assistance during the transition to sleep. There is evidence that foster children can be reluctant to request assistance from their foster caregiver when they are experiencing difficulty regulating stress or emotion (Fish & Chapman, 2004; Schofield & Beek, 2005). This is especially risky behavior in early childhood because at early ages, children rely almost exclusively on caregivers as sources of external regulation of sleep and arousal, emotion regulation, and regulation of stressors (Beltramini & Hertzig, 1983; Thompson, 1994). Across development, typically-developing children become increasingly able to self-soothe by recognizing internal cues that they are tired or are becoming emotionally aroused. In contrast, when caregivers are unavailable or not considered to be acceptable sources of regulation, children may develop poor regulatory skills, and as a result exhibit regulatory problems.

Foster children may resist reliance on foster caregivers for assistance with sleep-related problems. They may neglect to summon their caregiver when feeling fearful prior to sleep or during nighttime awakenings, thereby increasing the likelihood of less restorative sleep due to long latencies prior to sleep onset, increased nighttime awakenings, and increased activity. For young children, it is normative to perceive night as a fearful time when parents are not directly available and they are left alone in the darkness. Many times, parents report that children request "curtain calls" after the lights are out to provide soothing gestures such as additional goodnight kisses and also to

address fears of monsters or nightmares (Beltramini & Hertzig, 1983). Foster children may experience bedtime differently since they may not view foster caregivers as potential sources of regulation of normative fears due to compromised attachment relationships (Dozier, 2005). Furthermore, nighttime and darkness may be directly associated with experiences of abuse, which may further increase anxiety around bedtime (Sadeh, 1996).

Moore (1989) suggests that children who view their caregiver as unresponsive and unavailable to protect them will experience greater vigilance in order to stay "on guard" to protect themselves. One study has empirically tested this hypothesis and further suggested that secure attachment relationships may be important determinants of high quality sleep in children (Benoit, Zeanah, Boucher, & Minde, 1992). The study assessed the attachment style of mothers and found that 100% of the mothers with sleep-disordered children were classified as having an insecure attachment style. This finding may be especially applicable to foster children since many of them do not develop secure attachments with their foster care providers.

Socioeconomic Status and Sleep Disruption

In comparing foster children's sleep with that of community children, it is important to control for SES effects since birth families of foster children are often in the lowest socioeconomic strata. Differences between children in foster care and children living in middle-income homes may be due to economic differences rather than differences attributable to foster care placement. On the other hand, if foster children are compared to only a low-income community group and no differences are found, it is difficult to know whether this is a true lack of difference between foster and community

children. The inclusion of low- and middle-income community comparisons is an optimal design for testing differences between foster children and their community peers. This design allows for understanding effects of foster care beyond what is attributable to economic effects.

The majority of studies in the developmental sleep literature have been conducted on children living in upper middle-income households, and it is unclear whether findings in this group can be generalized to samples of higher risk (Spilsbury et al., 2004). There is evidence to suggest that children in low SES households may also show sleep disruption due to a higher incidence of stressors and greater vulnerability to negative psychosocial and physical health outcomes (Brooks-Gunn & Duncan, 1997; Evans & English, 2002; Lupien, King, Meaney, & McEwen, 2001). Some stressors that have been found to occur in higher frequencies in low SES families that may increase children's experience of stress are harsh parenting and physical punishment, decreased maternal emotional responsiveness, increased frequency of parental psychopathology, and increased marital conflict (Aber, Bennett, Conley, & Li, 1997; Hashima & Amato, 1994).

Measures of SES have also been related to compromised sleep. In a study on adult perceptions of their health, Hunt, McEwen, and McKenna (1985) found that individuals at the lowest socioeconomic levels endorsed three times the sleep disruptions as did those in the highest levels. Similarly, sleep has been implicated in other studies as an important mediator of the well-documented association between SES and diminished psychological and physical health (Moore, Adler, Williams, & Jackson, 2002). In a path analysis, Moore and colleagues found that subjective reports of sleep quality were directly

predicted by income level and in turn, sleep predicted both psychological distress and health after controlling for other potential confounds (e.g., prior health status, ethnicity, and sleep quantity). At the very least, these examples suggest that lower socioeconomic levels are associated with the perception of impaired sleep.

There also evidence that children in low SES homes are vulnerable to poor psychosocial outcomes and impaired sleep. One plausible explanation for this association is that low income rural and urban children experience increased number and intensity of stressors (Evans & English, 2002). Past studies have identified increased family turmoil, low-quality and crowded housing as stressors associated with children living in low income households (Aber, Bennett, Conley, & Li, 1997) and other studies have found these factors to also be related to sleep disruption (Kahn et al., 1989; Rona, Li, Gulliford, & Chinn, 1998; Stein, Mendelsohn, Obermeyer, Amromin, & Benca, 2001). In a survey of school-aged children, Simonds and Parraga (1982) found evidence that SES was inversely related to increased stress and sleep disruption as children in the lowest socioeconomic strata reported the greatest frequency of nighttime awakenings, restless sleeping, snoring, daytime drowsiness, and fearfulness upon going to sleep. Although this study suggests that increased fearfulness or vigilance may be a key factor in explaining higher rates of sleeping problems in low SES homes, the study methodology prohibits any causal explanations. Other groups have found associations between SES and sleep disruption using objective activity-based measures of sleep, which are more accurate and reliable than subjective sleep diaries or retrospective reporting. Although some associations with sleep disruption have been found to decrease in magnitude when using

objective measures, associations with SES remained significant even when controlling for confounding factors (e.g. race) in a study of third graders (El-Sheikh, Buckhalt, Mize, & Acebo, 2006). Overall, studies suggest that income may be associated with factors that interfere with the acquisition of sufficient sleep in children; however, the inconsistencies in the literature indicate that this question deserves further exploration.

Treatment Foster Care and Sleep

Although there is overwhelming evidence that stress inhibits children's ability to obtain adequate sleep, there is also evidence for resilience in children experiencing multiple stressors. Factors that have been related to increased quality, quantity, and regularity of sleep are consistency of sleep schedule, secure attachment with a primary caregiver, responsive and warm caregiving, and a contingent and consistent environment (Bates, Viken, Alexander, Beyers, & Stockton, 2002; Mindell, 1999; Moore, 1989). There is also evidence that placement into reinforcing and consistent foster homes can be a therapeutic mechanism to reduce the occurrence of problem behaviors and dysregulation seen in foster children.

In the current study, we investigate differences in sleep between foster children placed in care as usual as well as children who participated in Multidimensional Treatment Foster Care for Preschoolers (MTFC-P) prevention intervention (Fisher, Ellis, & Chamberlain, 1999). The MTFC-P program primarily targets caregiving behavior as a means for intervention so that the home becomes the treatment setting. Foster care providers receive training to enhance consistent and warm caregiving, thereby increasing the likelihood that foster children experience their environments as reinforcing and safe

(Fisher, Ellis, & Chamberlain, 1999). Many foster children come from chaotic home environments and have experienced inconsistent caregiving. One of the hallmarks of the MTFC-P intervention is to create consistent routines and to pre-teach expectations for upcoming situations so that foster children are able to understand the environmental contingencies and learn that their caregiver is consistently available. The intervention additionally provides support for the foster caregivers to reduce parenting stress, which is a risk factor for sleep disturbance in caregivers and in children. Although the MTFC-P intervention does not specifically address sleep hygiene and sleep schedule behaviors, it targets many areas of risk for disrupted sleep.

Outcomes of MTFC-P intervention trials suggest that the intervention may impact processes related to sleep such as HPA axis processes. There is a close relationship between sleep and HPA axis functioning as functioning of the hypothalamus is instrumental to sleep-arousal processes. Cortisol, a hormonal end product of the HPA axis, is secreted in a diurnal pattern that peaks right before morning rise time and reaches its nadir during sleep. Past research has indicated that foster children are at risk for atypical patterns of cortisol secretion secondary to early stressful experiences. Cortisol in some children may show "blunting" where early morning cortisol levels are low and there is very little change throughout the day. Fisher and colleagues (2007) found that the MTFC-P intervention may impact these key neural regulatory processes. Specifically, they found that throughout the course of the MTFC-P intervention study, cortisol patterns of foster children in the intervention condition began to normalize to look like the community comparison group. On the other hand, cortisol patterns of children in regular

foster care condition flattened more over time and the "blunting" became more pronounced. Since functioning of the HPA axis and sleep are so closely linked, the results of this study may implicate that the MTFC-P intervention also influences children's sleeping behavior. The current study will provide a first step at describing potential differences among treatment and intervention groups, yet it is beyond the scope of this study to investigate the underlying mechanisms of such differences.

The Present Investigation

This study will investigate risk for sleep disruption in young foster children in both regular foster care settings as well as in children participating in the MTFC-P program. Very few studies have investigated the associations between sleep and stress in 3- to 7-year-old children. In this important period of early development, regulatory processes are vulnerable to disruption and enduring regulatory patterns are beginning to form. In all children, there is an exceptionally high rate of sleep disruption during this developmental period.

In addition, the current study will add to the growing body of literature using actigraphy, a home-based, activity-derived measure of sleep. The use of actigraphy is superior to subjective caregiver report of sleep disruption due to increased reliability and accuracy (Sadeh & Acebo, 2002). It may be especially important to use objective measures of sleep in high-risk samples, such as foster care, since caregivers may not be alerted when there is sleep disruption leading to underreporting of sleep problems.

This study used five nights of actigraphy recording to assess sleeping patterns of the following four groups: 1. foster children receiving the MTFC-P prevention

intervention (TFC); 2. foster children receiving care as usual (RFC); 3. low-income community comparison children living with their biological parents with no maltreatment history (LIC), and 4. upper middle-income community comparison children living with their biological parents with no maltreatment history (UMC). Based on past research of effects of early stress, it was expected that the RFC group would show the most disrupted sleep of all the groups across variables of sleep quantity, quality, and schedule. Secondly, the LIC group was expected to show less sleep disruption than the RFC group, but more disruption than the UMC group. If the UMC group showed less sleep disruption than either of the groups, it would suggest that family income may be a stronger determinant of sleep than placement in foster care. To test these specific hypotheses, two sets of contrasts assessed differences between RFC and LIC groups as well as LIC and UMC groups across a number of sleep variables.

Since the treatment group did not receive direct sleep intervention, but the intervention addressed many elements that are important to obtaining quality sleep, it was unclear whether the TFC group would show similar sleep disruption to the regular foster care children or whether their sleep would be improved to look more like the community groups. Based on past research of the MTFC-P intervention there is suggestion that the TFC group may have shown improvements in some areas of sleep, although it was unclear whether these prior findings could be extended to sleep behavior. Sleep in the TFC group was compared with all other groups as an exploratory means of determining the extent of any treatment effect. Three sets of contrasts tested these hypotheses with the following comparisons: 1. TFC and RFC; 2. TFC and LIC; and 3. TFC and UMC. Sleep

improvement in the TFC group over the RFC group may indicate potential intervention effects of sleep in foster children and that the TFC group's sleep may look more like the community comparison. Furthermore, differences among the TFC group and the community comparison groups were investigated to identify if the TFC children's sleep is most similar to the RFC children or whether they showed improved sleep beyond that of either community group.

Methods

Participants

Seventy-nine children (41 females) between the ages of 3- and 7-years-old (M=5.25, SD=1.05) were recruited to participate in the study. Subsequently, four children were excluded from analyses due to actigraph equipment malfunction. The sample was demographically representative of Eugene, Oregon and was primarily comprised of European American ethnicities (82.3%, n=65), while the remaining children represented Latino (7.6%, n=6), Native American (6.3%, n=5), and African American (3.8%, n=3) ethnicities. Fifty of these children were concurrently participating in Early Intervention Foster Care (EIFC) ongoing longitudinal study, which is a randomized clinical trial to evaluate the MTFC-P intervention. The remaining 29 children were recruited from the community and responded to flyers posted in day cares, athletic facilities, and local businesses.

The sample was comprised of four groups of children: 1. Children living with a foster care provider receiving care as usual (RFC; n=15); 2. Children living with a foster

care provider and receiving the Multidimensional Treatment Foster Care for Preschoolers (MTFC-P) intervention (TFC; n=17); 3. Children living with their biological parents in a low-income community household (LIC; n=18); and 4. Children living with their biological parents in a upper middle-income community household (UMC; n=29).

The inclusion criteria for the LIC group were socioeconomic status (SES) indicators that did not exceed that of the biological parents of the foster children, (i.e., if either of their parents had a bachelor's degree or greater and if the combined gross household income exceeded $30,000 annually). Fifty-five percent of the LIC households received government food stamps.

Children were included in the UMC group if their family's gross household income exceeded $60,000 annually. Income ranges in the UMC group were $60,000 to $79,000 ($n$=12), $80,000 to $99,000 ($n$=7), and greater than $100,000 ($n$=10). There were no exclusion criteria for education. None of the UMC families received government aid.

Children in both community comparison groups (LIC and UMC) were excluded from participation if they had any history of caregiver transitions or maltreatment. Reports of maltreatment were determined by Department of Human Services (DHS) records.

Children in the TFC group were placed with a caregiver who had undergone training to be an MTFC-P foster care provider. As part of the MTFC-P intervention, a team of treatment providers worked with the foster child, the child's foster care provider, and the child's permanent placement resources. The intervention targeted family interactions to enhance warmth and consistency in the home in order to provide an

environment that facilitates learning and development. Foster care providers were provided with ongoing support to meet intervention goals though 24-hour staff access, foster parent support meetings, and daily telephone contact. The children received individual instruction that addressed problem behaviors across home, school, and community settings and they also participated in therapeutic playgroups that targeted school readiness competencies. Children generally received intervention for 6 to 9 months, although some children in long-term foster care continued receiving the intervention until their behaviors stabilized.

Children in the RFC group received services-as-usual, which typically included weekly psychotherapy sessions.

Materials

Actigraphy. Actigraphy is a well-validated measure of sleep quality and duration that can be collected at home. Actigraphs record movement-generated data, which is subsequently scored by computer-generated algorithms (e.g. Sadeh and Cole-Kripke). These scoring algorithms differentiate activity into periods of sleep and wakefulness. In recent studies, actigraphy has been validated against polysomnography (PSG), with reported correspondence of the measures up to 93 % in adults and 89.9% in children (Sadeh, Hauri, Kripke, & Lavie, 1995; Sadeh, Lavie, Scher, Tirosh, & Epstein, 1991; Sadeh, Sharkey, & Carskadon, 1994).

The actigraphs used in the current study were Basic Mini Motionlogger models (Ambulatory Monitoring, Inc., see Appendix A for a photo), which were approximately the size of a wristwatch. The actigraphs were fastened with a strap around the child's

non-dominant wrist as is recommended by Sadeh and Acebo (2002). To make the device more child-friendly, it was placed in a soft sleeve that was shaped like a sea-creature (see Appendix B for a photo).

Data were collected in 1-min epochs and at data amplification of 18, which is the default acquisition setting. After acquisition, actigraphic sleep data was downloaded to a PC using ACT Millenium software and subsequently scored on Action W software (Ambulatory Monitoring, Inc.) using the Sadeh algorithm (Sadeh, Sharkey, & Carskadon, 1994). Sleep variables used in the current study that were acquired via actigraphy and scored with Action W software included measures of sleep *quantity*: *(a)* sleep duration scored as total minutes between sleep onset to wake onset and *(b)* true sleep time scored as total sleep minutes excluding any periods of wakefulness; and measures of sleep *quality: (c)* sleep percentage scored as the ratio of true sleep time and total duration, *(d)* nighttime activity scored as the percentages of sleep epochs with detected motion, and *(e)* number of wake minutes

Actigraphic measure of sleep *schedule,* sleep onset, wake onset, lights out, and rise time, were manually indicated in each of the data files. Sleep onset was defined as the beginning of the first 15-min epoch of uninterrupted sleep and wake onset was defined as the last 15-min epoch of uninterrupted sleep. Caregivers were trained to indicate lights out time and rise time using an event mark button on the actigraph. The total time in bed was scored as the difference between lights out time and rise time. Sleep latency was scored as the number of minutes between lights out time and sleep onset time. The number of night wake episodes was a manually scored sleep quality measure

and was defined as any 5 consecutive minutes of wake bounded by 15-min of uninterrupted sleep epochs.

Prior to conducting analyses, each file was cleaned to ensure data integrity. This involved checking actigraphy data against parent reported bedtime and wake times in a sleep diary (see below) to ensure that parents accurately indicated sleep time and rise time with the actigraphic event marks, and to determine whether the actigraph was removed at any time during the night. Nights in which the actigraph was removed (n=7) and when there was non-compliance with the study protocol (n=7) were excluded from the analyses. All children included in analyses had at least four nights of data.

Sleep Diary. The 15-item Sleep Diary was created by Sadeh (1994). Caregivers were asked to complete daily diary entries after children went to bed and again upon wake (e.g. lights out time, times and lengths of daytime naps, episodes of nighttime waking, morning rise time). Caregivers also reported subjective impressions of how tired their children appeared at bedtime and upon wake, overall health of the child and activity during the day, and any unusual circumstances that occurred during the night that may have interfered with sleep or the acquisition of sleep data (e.g. actigraph was removed). Sleep data were checked against the diary entries to ensure compliance with the protocol and to identify potential external sources of sleep anomalies.

Procedure

A home visit was scheduled with each family so that the study could be explained fully to caregivers and the participating child. For children who were in foster placements, caseworker consent was obtained prior to contacting foster providers. After

consent for participation was received from caregivers, the study materials were introduced to both parents and the child.

Sleep data were obtained on five consecutive nights, as recommended for adequate reliability (Acebo et al., 2005; Acebo et al., 1999). The majority of the sample wore the actigraph on Sunday through Thursday nights, although seven children participated on Monday through Friday due to scheduling conflicts. Parents were instructed to maintain a normal sleep routine and were not discouraged from allowing daytime naps or from engaging in bedtime routines (e.g. stories or songs). After the bedtime routine was completed, parents were asked to secure the actigraph to the child's non-dominant wrist on each study night prior to turning the lights out and then complete the evening sleep diary. The time that the child went to bed was recorded by pressing an event marker button on the actigraph. When the child woke, parents were instructed to remove the actigraph and press the event marker button to indicate rise time. The sleep diary was again completed in the morning after the child was awake. In the event that the actigraph was removed during the night, parents were asked to refasten the device as soon as possible.

Children were rewarded with stickers after nights of successfully wearing the actigraph. Upon completion of the study, they were given a bath mitt that was a larger version of the actigraphy sleeve and parents were compensated with $100.00.

Results

Once files were cleaned, all sleep variables (i.e. measures of sleep quantity, quality, and schedule) were aggregated over the five days of actigraphy collection. Variability of sleep measures was computed as the standard deviation across the five days of data collection. Stability of actigraph measures across nights generally reached the suggested intraclass (ICC) correlation level of.70, (values ranged from .70 to .89), indicating adequate stability by (Acebo et al., 1999). Other variables, sleep duration and number of nighttime wakings, approached the recommended values with reliability estimates ranging from .60 to .69.

Since there were no group differences in age, $F(3, 75)=.56$, $p>.05$, or across the gender distribution, $\chi^2(3)=1.10$, $p>.05$, these variables were excluded from further analysis.

Sleep Differences between Groups

A multivariate analysis of variance (MANOVA) was computed on averaged sleep measures and sleep variability measures to address differences among care groups and income levels. The omnibus MANOVA using the Wilks' Lambda criteria was significant, indicating differences between the groups, $F(75,141.37)=1.57$, $p=.01$ (See Table 1 for descriptives).

Due to power limitations and to guard against Type I error, specific contrasts (vs. all possible contrasts) were selected to test study hypotheses. Pairwise comparisons were used to determine differences between the groups on sleep variables for which the between subjects test reached or approached significance (See Table 1 for between

subjects results). The follow-up contrasts first tested whether the RFC group differed from the LIC comparison group. Differences of these comparisons may be attributable to placement in foster care since the groups were comparable on SES. Secondly, the influences of SES were examined in comparisons of the LIC and UMC groups. There were no prior hypotheses for the TFC group because it was unknown how much the intervention may affect sleep patterns of children in this group. To more clearly understand how the intervention may have impacted sleep patterns in this group, pairwise comparisons of the TFC group with all the other groups were investigated. Alpha inflation was controlled with Bonferroni corrections and values were compared against $p<.01$. Only results reaching significance are discussed below.

Sleep Quantity. Although it was hypothesized that children in the TFC group would sleep for shorter sleep durations than children in both community comparison groups, this was not supported. Results did suggest, however, that children in the TFC group had less disrupted sleep in terms of quantity than the RFC group. The TFC group slept for a significantly longer nightly duration than the RFC group, $F(1,71)=8.50$, $p<.01$, or the LIC group, $F(1,71)=8.74$, $p<.01$, (see Figure 1).

Similarly, the TFC group also showed a trend toward obtaining more true sleep than LIC children, $F(1, 71)=5.64$, $p=.02$ (see Figure 2). Overall, results suggest that children in the treatment group were sleeping more on average than children in the low-income and RFC groups. The TFC children did not differ from UMC children in either measure of sleep quantity.

Table 1

Descriptives of Sleep Variables and Results of Univariate MANOVA Tests in RFC, TFC, LIC, and UMC Goups

	RFC (n=14)		TFC (n=17)		LIC (n=18)		UMC (n=26)		
Actigraphy-Derived Sleep Measures	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*F*
Sleep Quantity									
Sleep duration (min)	557.45	35.28	597.23	43.32	559.43	29.57	571.40	40.16	3.88(3, 71) **
True sleep time	471.70	54.17	514.66	53.08	472.77	52.13	491.43	50.53	2.48(3,71) [b]
Variability of sleep duration	52.11	20.48	62.17	34.67	44.57	21.82	39.35	17.24	3.42(3, 71) *
Variability of true sleep time	50.10	23.16	48.52	24.42	47.17	22.31	42.63	18.42	.46(3, 71)
Sleep Quality									
Sleep percentage	84.56	6.77	86.41	5.62	84.53	7.32	85.97	5.34	.43(3, 71)
Sleep activity	53.66	11.35	47.18	12.09	54.07	10.39	46.91	7.69	2.81(3, 71) *
Night wakings	3.84	1.59	3.62	1.13	3.96	1.10	3.68	1.12	.29(3, 71)
Wake minutes	85.76	38.18	82.56	34.52	86.67	40.19	79.97	29.40	.16(3, 71)
Variability of sleep percentage	4.92	2.48	5.21	2.78	5.51	4.73	5.23	3.00	.08(3, 71)
Variability of sleep activity	6.11	2.40	6.84	2.59	6.13	3.20	6.28	3.31	.22(3, 71)
Variability of night wakings	1.54	0.93	1.41	0.54	1.43	0.41	1.25	0.41	.88(3, 71)
Variability of wake minutes	28.90	16.94	36.35	22.81	32.26	27.65	31.02	18.11	.34(3, 71)

Note. SD= standard deviation; * p<.05, ** p<.01, *** p<.001, [a] p<.07, [b] p=.06

Table 1 (continued).

	RFC (n=14)		TFC (n=17)		LIC (n=18)		UMC (n=26)		
Actigraphy-Derived Sleep Measures	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*F*
Sleep Schedule									
Time of sleep onset	21:10	51 min	20:43	36 min	21:26	62 min	21:15	38 min	2.51(3, 71) [a]
Time of wake onset	6:26	51 min	6:41	55 min	7:01	50 min	6:45	27 min	1.56(3, 71)
Lights out time	20:21	46 min	20:00	33 min	21:11	47 min	20:48	38 min	9.60(3, 71) ***
Rise time	7:09	55 min	7:17	60 min	7:32	46 min	7:14	27 min	.81(3, 71)
Total time in bed	647.76	31.07	676.99	58.40	625.86	33.62	625.98	35.31	6.57(3, 71) ***
Sleep Latency	51.63	16.85	43.31	19.28	30.41	18.58	26.76	10.59	9.19(3, 71) ***
Variability of time of sleep onset	35 min	17 min	41 min	19 min	61 min	135 min	25 min	15 min	1.04(3, 71)
Variability of time of wake onset	37 min	23 min	40 min	24 min	38 min	27 min	34 min	17 min	.24(3, 71)
Variability of lights out time	31 min	16 min	38 min	22 min	35 min	21 min	26 min	15 min	1.70(3, 71)
Variability of rise time	22 min	14 min	28 min	21 min	26 min	23 min	21 min	10 min	.73(3, 71)
Variability of total time in bed	39.57	12.54	44.79	40.34	43.6	34.37	28.52	14.85	1.64(3, 71)
Variability of sleep latency	28.92	10.33	23.66	14.56	17.56	15.25	14.85	10.74	4.35(3, 71) **

Note. SD= standard deviation; * p<.05, ** p<.01, *** p<.001, [a] p<.07, [b] p=.06

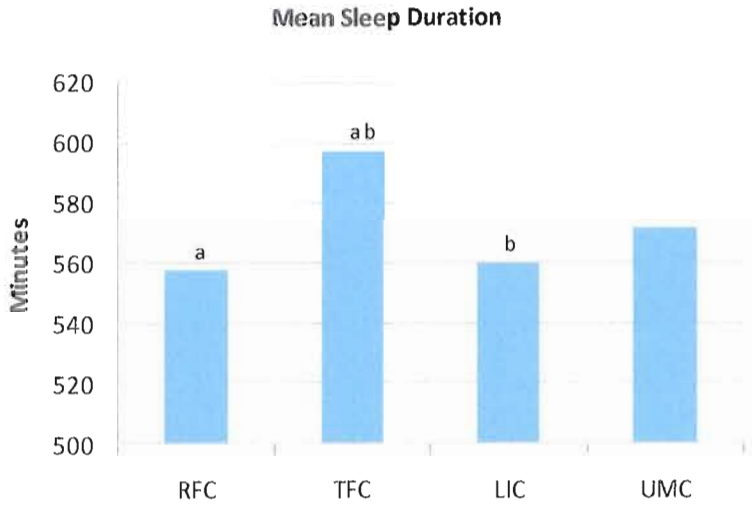

Figure 1. Differences in mean sleep duration between groups. *a* denotes $p<.01$, *b* denotes $p<.01$.

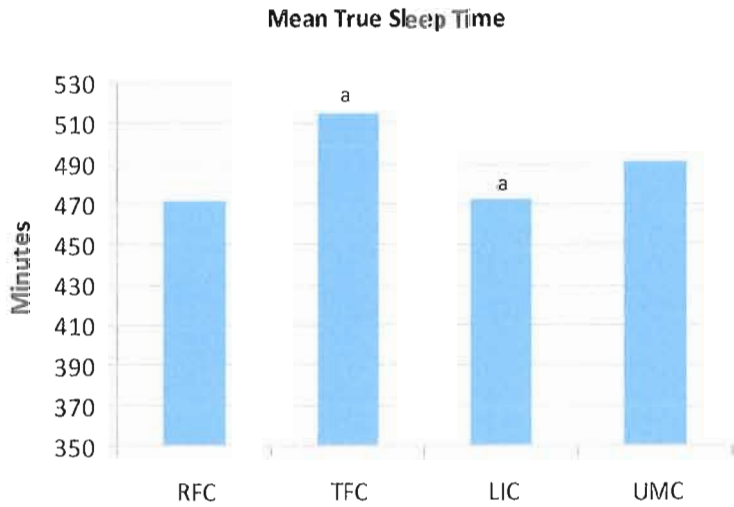

Figure 2. Differences in mean true sleep time between groups. *a* denotes $p<.02$.

Sleep Quality. Although the univariate test for nighttime activity was significant, none of the pairwise comparisons reached significance.

Sleep Schedule. There were unexpected sleep schedule differences between the groups. Parents put children in the RFC group to bed at an earlier time than parents in the

LIC group, $F(1,71)=11.23$, $p<.001$. The TFC group went to bed at a significantly earlier time than the LIC group, $F(1,71)=24.96$, $p<.001$, and the UMC group, $F(1,71)=13.40$, $p<.001$ (see Figure 3). The TFC and RFC groups did not differ in the average lights out time. This suggests that foster parents put children to bed significantly earlier than did the community parents.

The TFC group also spent more time in bed on average than those children in the LIC, $F(1,71)=13.81$, $p<.001$, and UMC groups, $F(1,71)=16.16$, $p<.001$ (see Figure 4). Correspondingly, the time in which the TFC group was able to initiate sleep was also significantly earlier than the LIC group, $F(1,71)=6.91$, $p<.01$ (see Figure 5).

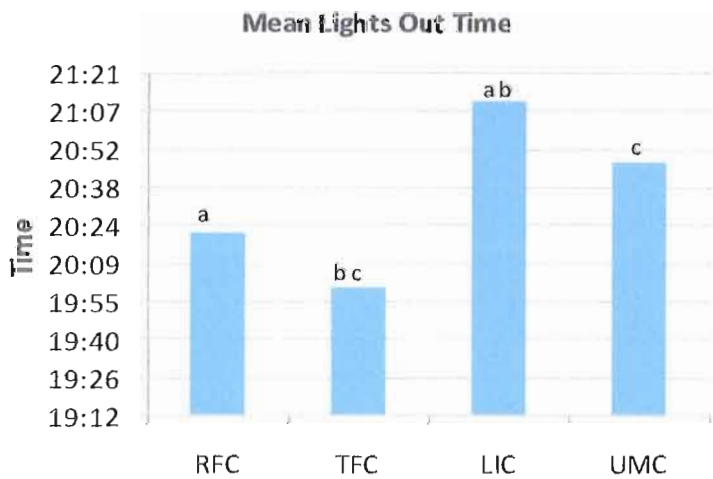

Figure 3. Differences in mean lights out time between groups. *a* denotes $p<.001$, *b* denotes $p<.001$, *c* denotes $p<.001$.

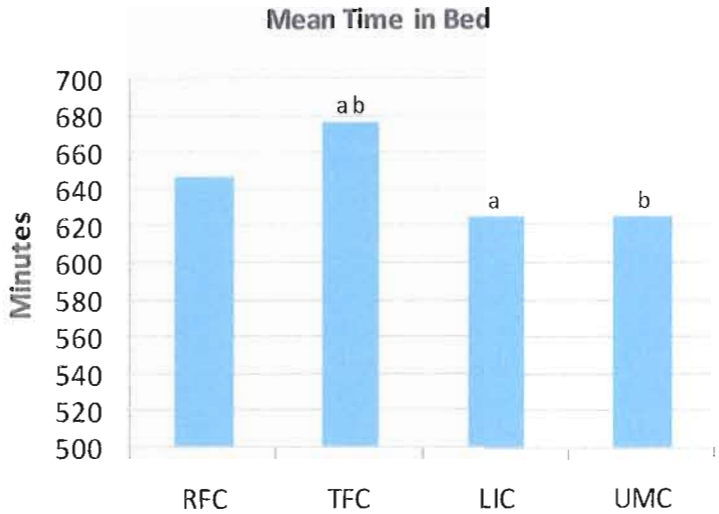

Figure 4. Differences in mean time in bed between groups. *a* denotes $p<.001$, *b* denotes $p<.001$.

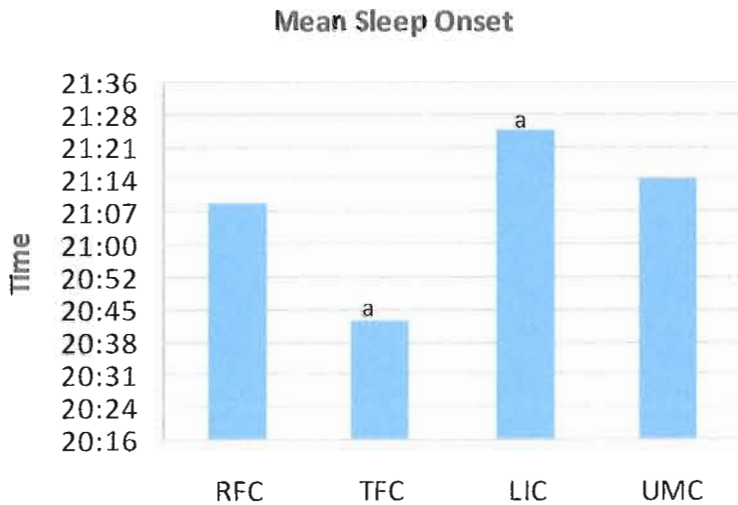

Figure 5. Differences in mean sleep onset time between groups. *a* denotes $p<.01$.

There were also significant differences in sleep latency across the groups in the expected direction. Specifically, the RFC group spent a longer time in bed prior to falling asleep than the LIC group, $F(1,71)=13.75$, $p<.001$. The TFC group took a longer time to fall asleep once in bed than did the UMC group and marginally more time than the LIC

group, $F(1,71)=10.92$, $p<.001$ and $F(1,71)=5.65$, $p<.02$, respectively (see Figure 6). The foster groups did not significantly differ in latency of sleep onset.

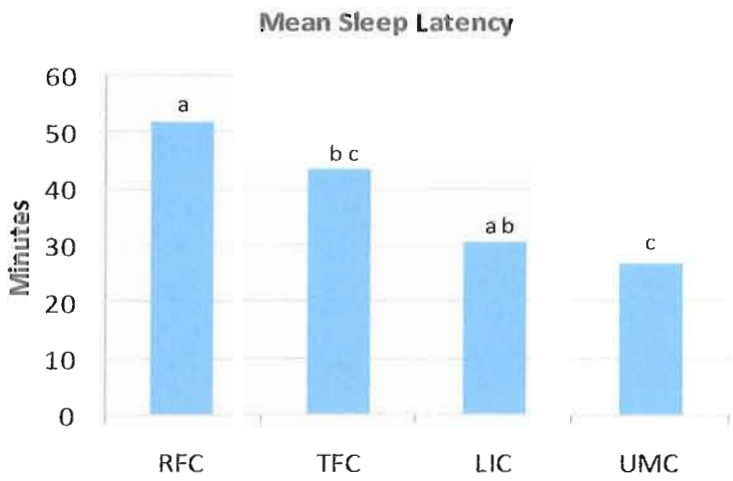

Figure 6. Differences in mean sleep latency between groups. *a* denotes $p<.001$, *b* denotes $p<.02$, *c* denotes $p<.001$.

Sleep Variability. Although the TFC group slept significantly longer than the RFC and LIC groups, pairwise comparisons of the variability of sleep duration indicated that the TFC group's sleep duration was less consistent than the UMC group, $F(1, 71)=9.45$, $p<.01$ (see Figure 7). In addition, the RFC group was marginally more variable in the latency time prior to sleep onset than was the LIC group, $F(1,71)=6.21$, $p=.015$ (see Figure 8).

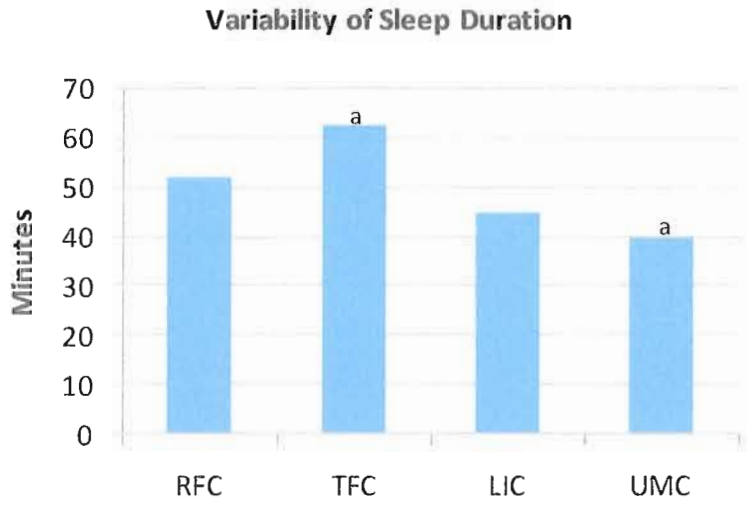

Figure 7. Differences in variability of sleep duration between groups. *a* denotes $p<.01$.

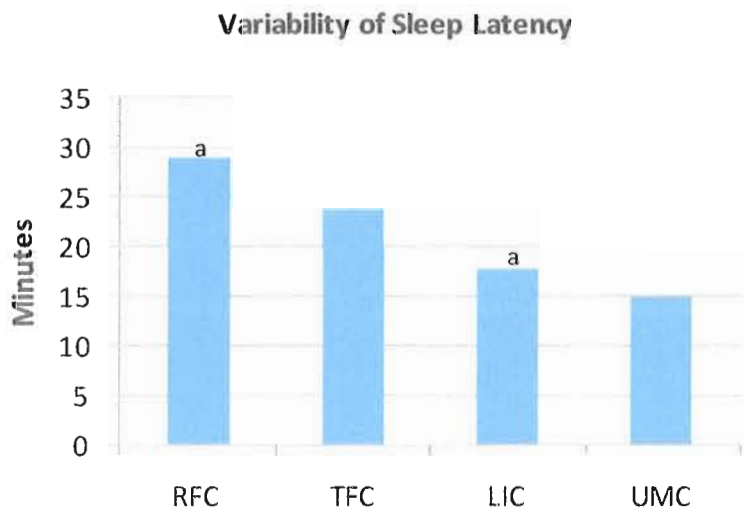

Figure 8. Differences in variability of sleep latency between groups. *a* denotes $p=.015$.

Discussion

A number of noteworthy results emerged from the analyses. The community groups were expected to show less sleep disruption in terms of sleep schedule, sleep

quality, and sleep quantity than either of the foster groups. Overall, results suggested that differences among care groups (foster vs. community) were more pronounced than were SES differences (LIC vs. UMC). However, across many of the sleep measures, specifically in measures of sleep quality, no group differences emerged. Among the hypothesized effects, the foster groups displayed some indication of disruption of sleep initiation relative to the community groups. Specifically, the TFC group had greater difficulty initiating sleep, as measured by longer sleep latency, than either of the community groups. Furthermore, the contrasts suggested that the RFC group also spent more time in bed awake than the LIC group prior to sleep initiation.

There were also differences between groups that suggested treatment effects for the TFC group. In particular, the TFC group slept longer than both the RFC and LIC groups in terms of sleep duration and true sleep time. Furthermore, this group went to bed earlier, spent more time in bed on average, and fell asleep at an earlier hour than both community groups. This is an especially relevant finding for the MTFC-P intervention as prior research has consistently suggested that sleep duration is highly important for the acquisition of restorative sleep, which decreases the probability of impaired cognitive functioning, emotion regulation, and behavior problems (Aronen, Paavonen, Fjallberg, Soininen, & Torronen, 2000; Lavigne et al., 1999; Sadeh, Gruber, & Raviv, 2003; Steenari et al., 2003). This may be particularly important for foster children due to the high occurrence of cognitive, behavioral, and emotional problems that have been observed in this population (Clausen, Landsverk, Ganger, Chadwick, & Litrownik, 1998; Klee, Kronstadt, & Zlotnick, 1997; Pears & Fisher, 2005a, 2005b). These complex

problems are challenging for foster care providers and may be an impetus for placement transitions. Moreover, targeting these problems directly may be very challenging and resource intensive. In contrast, addressing these problems at least in part via the indirect mechanism of improved sleep may be a very efficient intervention approach. Along these lines, past evidence suggests that increases in sleep duration may provide an opportunity for reducing the intensity or frequency of these difficult behaviors (Lavigne et al., 1999; Weissbluth, Davis, Poncher, & Reiff, 1983).

It is important to acknowledge that sleep was not specifically targeted in the intervention. However, the MTFC-P intervention does directly intervene on many areas that are central to adequate sleep hygiene. MTFC-P foster care providers are trained to provide highly consistent care, which includes establishing predictable daily routines. Furthermore, the intervention emphasizes high rates of reinforcement in concert with brief, but effective, corrective feedback, which helps facilitate a warm and instructional environment. Prior research on MTFC-P outcomes have found that the multi-layered treatment approach is associated with changes that are central to adequate sleep. For example, Fisher and Kim (2007) found that children in the foster care as usual group displayed increasingly greater insecure attachment relationships over time, while the children in the MTFC-P intervention group displayed increases in secure attachment behaviors and decreases in insecure behaviors. Although the intervention did not specifically target attachment relationships, children began to utilize their caregivers for help or protection more frequently than those children who did not receive the intervention. This may also have implications for better quality sleeping behavior as it

may be an indicator that children feel more protected in the treatment foster home and find their foster care provider to be a safe and reliable resource when they need assistance.

Due to the intervention training, caregivers in the TFC group may be especially sensitive to the difficulties that foster children have around bedtime as well as to the importance of establishing consistent routines. The likelihood that the foster children obtained sufficient sleep duration was increased by the TFC care providers initiating bedtime over an hour earlier than the LIC children, 45 min earlier than the UMC children, and over 20 min earlier than the RFC children. This earlier bedtime allowed for the TFC children to take an average of 43 min to initiate sleep, and still obtain the most sleep of any group.

The RFC group also was put to bed earlier than the low-income community group, but they required a longer latency period prior to initiating sleep and spent the most time in bed awake, thereby obtaining the least amount of sleep of any group. The RFC group obtained less overall sleep than the TFC group, due to slightly less time spent in bed, greater length of time elapsed prior to sleep onset, and later time of sleep onset.

Although the TFC group slept longer on average than the LIC and RFC groups, the sleep lengths were more variable, and this inconsistency was comparable to the RFC group. As predicted, the UMC group had the most stable sleep, fluctuating approximately 40 min, while the TFC group had night-to-night fluctuations of over 60 minutes. The RFC group was also inconsistent in sleep duration and sleep latency, suggesting that on some nights, sleep was significantly shortened while other nights, sleep was less

impacted, although still poor in comparison to the other groups. The finding that children in the TFC group slept longer, although inconsistently longer, may be a positive indication that new sleep patterns are beginning to be established and these developing sleeping patterns may stabilize over time. Prior studies of the MTFC-P intervention investigating other regulatory functions such as cortisol, the stress hormone end-product of the hypothalamic-pituitary-adrenocortical (HPA) axis, have observed similar processes as a result of the intervention. Fisher, Stoolmiller, Gunnar and Burraston (2007) found that after beginning the intervention, cortisol levels stabilized in the TFC group and decreased in variability over time. Cortisol in the RFC group continued to show increased dysregulation and variability in the longitudinal study. Although speculative, these findings may be extended to the current study to suggest that the TFC group may be benefitting from the consistent nature of the environment, thereby positively impacting the nightly sleep duration and possibly impacting stability of these patterns over time.

A commonality of the foster groups was the expected difficulties with initiating sleep. This is one of the most frequently reported sleep problems in young children (Beltramini & Hertzig, 1983), and for children with histories of maltreatment, this problem may be exacerbated. Past research has shown associations with maltreatment and prolonged sleep latency (Glod, Teicher, Hartman, & Harakal, 1997), which is supported by the results of the current study. The delay in sleep onset seen in these groups may be a precursor to more problematic sleeping problems in later years. However, the earlier bedtime in the TFC group seemed to be associated with amelioration of the potential negative effects of reduced sleep duration.

There were also surprisingly few sleep differences between SES groups (LIC vs. UMC). Such differences were expected based on past research (El-Sheikh, Buckhalt, Mark Cummings, & Keller, 2006; Rona, Li, Gulliford, & Chinn, 1998). One explanation for this discrepant finding is that, unlike past research, the current study parses the experience of maltreatment from the measure of SES when looking at sleep outcomes. As previously noted, the experience of maltreatment is more common among low-income children than upper middle-income children, so differences in maltreatment experiences may have inflated SES differences in prior studies. Another potential difference in the current study is that the income level of the low-income sample was set above the poverty line in order to provide a comparison with the income levels of the families of origin of the foster groups. Children living in poverty may experience greater sleep disruption than other low-income children, and these differences may have been diluted by the selected income threshold.

Another unexpected outcome of the study was the absence of group differences in sleep quality. Sleep quality has been widely recognized as important for the initiation of deep stages of sleep and obtaining adequate durations of sleep at these stages (Robins, Norem, & Cheek, 1999; Wesensten, Balkin, & Belenky, 1999). Fragmented sleep and sleep with frequent activity may indicate increased restless sleep or more time spent in sleep stages 1 and 2. Although interesting group differences in sleep schedule and duration emerged, overall results suggest that, to a certain extent, sleep may be protected in all children regardless of risk exposure in this early period. Dahl (1996) proposes that sleep is a regulatory process that is difficult to disrupt throughout early childhood. He

argues that it is in adolescence when sleeping patterns are no longer protected, and thus that early disruptions may have significant impacts on functioning at later developmental periods. The current study supports this idea as the four groups in different care environments did not differ on most measures of sleep quality (e.g. number of nighttime wake episodes, nighttime activity, sleep efficiency). This supports the notion of a protective mechanism for young children related to sleep, and may indicate the presence of a viable prevention window where the establishment of regular and adequate sleep routines may be most beneficial before entry into puberty. The MTFC-P outcomes of increased sleep duration are a promising step in ensuring that young foster children obtain adequate sleep prior to adolescence. However, the MTFC-P intervention may be bolstered by including a sleep hygiene component to ensure that all children receiving the intervention adhere to a bedtime routine that enhances the likelihood of obtaining sufficient sleep.

Limitations

Although this study on foster care differences in sleep produced a number of interesting results, a number of limitations also exist. One important shortcoming of the present study is the relatively small size of the groups. Follow-up studies of larger scale would benefit from a larger sample size to increase power to detect group differences. It is encouraging that group differences were detected in the underpowered study and suggests that the effects may increase in magnitude with a larger sample.

A main limitation of the current study is the lack of pre and post measures within the treatment outcome study. Furthermore, the length of time in treatment was not controlled in analyses. Although these limitations are warranted given the exploratory nature of the study, they make interpretation of the findings difficult and require further exploration to fully unpack potential intervention effects.

The small sample size also precluded further investigation of potential heterogeneity within the foster samples. Bruce and colleagues (2007) found that foster children who have had greater than four placement transitions, who were placed in the foster care system in infancy, and who experienced severe neglect are those at greatest risk for regulatory problems. Other studies have also found that frequency and type of prior maltreatment may impact outcomes of foster children (Pears & Fisher, 2005a). An important direction for future studies will be to investigate placement history (e.g., age of placement, number of placement transitions) and maltreatment type as possible moderators of sleep differences.

Aside from differences in nighttime activity, the current study did not support prior findings of sleep differences among socioeconomic groups. SES group differences may have been obscured by the homogeneity of ethnicity and relatively low-risk nature of the sample. The sample was collected in Eugene, Oregon, which is a rural and primarily Caucasian community. Many prior studies indicating socioeconomic effects associated with impaired sleep have primarily sampled from urban communities. In these types of communities, environmental factors such as neighborhood violence, noise, and home crowding have been implicated in sleep disruption, but these factors may be less

prevalent in rural neighborhoods. Additionally, studies have found ethnicity differences beyond the effects of SES (Buckhalt, El-Sheikh, & Keller, 2007), which could not be examined due to the predominantly Caucasian community from which the study samples were recruited. In larger-scale future studies, it will be important to continue to distinguish between maltreated groups, by excluding children with maltreatment histories from community groups, but will also be important to identify differences among children from both rural and urban communities.

Future Directions

The potential treatment implications suggested by this study's findings warrant further exploration in future studies. One potential mechanism that may account for sleep differences among the foster groups is attachment-related behaviors. To better understand how caregiver relationships may impact sleep, future studies should assess the attachment relationship between child and caregiver and the way in which this relationship may predict sleeping behavior. Specifically, since increases in secure attachment behaviors have been outcomes of the MTFC-P intervention, it is important to better understand how these changes in these mechanisms over time may be related.

Furthermore, it is important that future studies investigate longitudinal changes in sleep as a function of involvement in the MTFC-P program. A longitudinal study is important to better understand the impact of the intervention on sleep, investigate potential stabilization of sleep over time, and to understand how early prevention efforts may impact sleep after pubertal onset. A study of this nature would additionally allow for

investigation of how changes in sleep over time may also correspond with other changes in regulatory functioning (e.g. problem behaviors) so that sleep can be better understood in the context of broader regulation.

Although comparison groups were chosen across SES levels, the question of whether placement in foster care may be associated with sleep problems beyond what is attributable to maltreatment remains. Future studies may disentangle these effects by looking at sleep differences among children who have experienced different types and frequencies of maltreatment as well as differences between children who have experienced longer periods of time in foster care or greater number of placement disruptions while in the foster care system. These follow-up studies may be helpful in understanding the extent to which prior stressful experiences may be attributable to sleep disruption and the extent to which variables associated with placement in foster care can maintain or enhance these sleep disruptions.

Aside from the study limitations and need for future research, this initial study investigating actigraphy-derived sleeping behavior in foster and community groups suggested promising treatment effects that were encouraging for prevention future efforts. Acknowledging that sleep difficulties may be more prevalent in children in foster care is an important step toward ensuring that sleep needs are addressed in this group of children prior to puberty.

CHAPTER III

STUDY 2: ASSOCIATIONS BETWEEN SLEEP AND REGULATION OF BEHAVIOR AND CORTISOL LEVELS IN EARLY CHILDHOOD

Introduction

Due to the close relationships between sleep and other physiological measures, it has been regarded as a "window to the central nervous system" (Halpern, Maclean, & Baumeister, 1995). Insufficient or inconsistent sleep is predictive of regulatory problems in children across developmental periods including failure to thrive in infancy, poor neurobehavioral functioning in early childhood, and poor academic performance in school-aged children and adolescents (Gruber & Sadeh, 2004; Halpern, Maclean, & Baumeister, 1995; Meijer, Habekothe, & Van Den Wittenboer, 2000; Owens-Stively et al., 1997; Wolfson & Carskadon, 1998). Sleep impacts cognitive functioning and behavior via two primary mechanisms. First, sleep provides a restorative mechanism that decreases daytime sleepiness and increases daytime alertness, behavioral regulation, and cognitive functioning (Dahl, 1996; Davis, Parker, & Montgomery, 2004; Sadeh, 2007). Second, sleep is an active state that is integral to brain functions including memory consolidation, learning, mood regulation, brain development, and hormonal regulation (Dahl & Harvey, 2007; Maquet, 2001; Sadeh, 2007). Early childhood is a developmental period in which children are highly vulnerable to sleep disruption due to the complexity

of the process. However, early disturbance, identified as early as in infancy, has been predictive of long-term sleeping problems, which have negative implications for cognitive, emotional, behavioral, and physiological regulatory processes (Dahl, 1996).

Sleep disturbance has been clearly associated with poor regulation of both physiological and behavioral processes. Disturbance has been measured in a variety of ways, including reduced quantity of sleep, increased fragmentation of sleep, problems initiating sleep, and night-to-night inconstancy of these measures. However, associations between sleep disturbance and regulation have been much stronger when subjective (e.g. sleep diaries or retrospective report) rather than objective measures of sleep (e.g. polysomnography or actigraphy) were used (Corkum, Tannock, Moldofsky, Hogg-Johnson, & Humphries, 2001; Sadeh, Pergamin, & Bar-Haim, 2006). One explanation for these discrepant findings is that studies using objective measures typically average sleep data over the course of multiple nights, consequently eliminating potentially informative night-to-night variability. There is evidence that marked sleep disturbance on only a few nights of the week may color parents' perceptions of children's overall sleep. On the other hand, when researchers simply investigate mean levels of sleep, outcomes associated with nights of highly disrupted sleep may be washed out. Assessing variability in sleep rather than average sleep may provide an opportunity to observe the "pendulum effect" that has been considered a marker of systemic instability (Gruber, Sadeh, & Raviv, 2000).

Inconsistent sleep behavior has been described as a stressor to the system. When sleep is irregular, it is difficult to overcome sleep debt, which is accumulated lost sleep.

Bates and colleagues (2002) likened the impact of high variability in sleep to extreme fatigue and cognitive impairment associated with the stress of jet lag. Other groups have found that variability in sleep is associated with poor developmental outcomes as early as infancy. Outcomes of these studies show that the proportion of time spent in sleep versus wake, or average amount of sleep, is not predictive of subsequent developmental status (Halpern, Maclean, & Baumeister, 1995). A more successful predictive measure has been the degree of stability within infant state profiles, specifically, the amount of time spent in sleep or wake states over the course of each 24-hour period. This measure of regulatory ability is associated with lower behavioral and medical problems within the first 30 months, lower mortality rates, and psychomotor and mental abilities as measured on the Bayley scale (Halpern, Maclean, & Baumeister, 1995). The current study investigates how variability of sleep in early childhood impacts both aspects of sleep benefits, behavioral and neurophysiological functioning. Although there is evidence that poor sleep may drive changes in stress hormones and problem behavior in children, there are very few studies that have examined these relationships. The current study will investigate associations between within-individual sleep variability and hormonal stress response and behavior in early childhood using both descriptive and predictive approaches.

Sleep and the HPA Axis

Sleep disruption may be an indicator of neurophysiological dysregulation. Inadequate sleep has been considered to be a systemic stressor, which in turn triggers the

hypothalamic-pituitary-adrenocortisol (HPA) axis-initiated neurohormonal stress response (Weissbluth, 1989). Cortisol, an HPA end product, is a stress hormone that is secreted in both a diurnal pattern as well as in response to environmental and physiological stressors (Sapolsky, Romero, & Munck, 2000). This diurnal pattern is closely tied to the circadian sleep and arousal cycle. It peaks just after the awakening response at approximately 9:00 AM and slowly declines throughout the day until its lowest point, which occurs around midnight (Buckley & Schatzberg, 2005). Cortisol increases rapidly just prior to wake onset, which serves as a trigger for the system to transition into a wake state (Buckley & Schatzberg, 2005). Cortisol secretion is a complex measure to interpret because of the difficulty associated with disentangling the diurnal cortisol level from reactivity bursts of cortisol after exposure to a stressor. Although cortisol secretion plays a key role in learning, memory, immune functioning, and is a necessary component for human survival, dysregulation in this system can also have deleterious implications for neurophysiological functioning and health (Payne, 2004; Sapolsky, Romero, & Munck, 2000). Cortisol hyper-secretion has been linked to heart disease, hyperglycemia, hyperactivity, and internalizing problems (Hatzinger et al., 2007; Schmidt et al., 1997; Sondeijker et al., 2007) and cortisol hypo-secretion with aggressive and delinquent behavior (McBurnett, Lahey, Rathouz, & Loeber, 2000; Shoal, Giancola, & Kirillova, 2003; Smider et al., 2002).

Cortisol has been a promising mechanism for measuring stress in young children due to the relative ease of collection and its consistent associations with problem behavior (Stansbury & Gunnar, 1994). Numerous studies have found associations

between elevations in morning as well as evening cortisol and problem behaviors in both clinical and non-clinical samples of children (Tout, de Haan, Campbell, & Gunnar, 1998). Elevated cortisol is an indicator of physiological dysregulation and implicates a heightened stress response. Elevations in evening cortisol are especially problematic since this is a time when free cortisol should be nearly depleted. Other studies have indicated a blunted response of cortisol as another indication of physiological dysregulation. Blunted cortisol patterns lack the morning peak and are characteristic of low cortisol levels throughout the day. This pattern has been most commonly associated with prolonged exposure to high intensity stressors and is thought to be a compensatory mechanism to protect the system from a flooding of cortisol (Oquendo et al., 2003).

Cortisol has come to be of interest to sleep researchers because of its diurnal secretion that follows circadian rhythms. Furthermore, dysregulation of cortisol secretion and sleep have both shown strong associations with wide-ranging problem behaviors. There is reason to believe that the relationship between HPA functioning and sleep is bidirectional. Sleep plays a key role in down-regulating the secretion of cortisol, while activation of the HPA axis in response to stressors can interfere with the acquisition of prolonged and adequate sleep. Although the relationship between sleep and cortisol secretion is multi-faceted, deep slow wave sleep (SWS) has generally been found to have a suppressive effect on cortisol secretion (Vgontzas et al., 1999). Animal and human sleep deprivation studies have shown that prolonged and acute sleep interference has stimulatory effects on the HPA axis stress response, thereby increasing cortisol and its precursors (Meerlo, Koehl, van der Borght, & Turek, 2002; Spiegel, Leproult, & Van

Cauter, 1999; Vgontzas et al., 1999). A rebound effect of cortisol down-regulation has been noted following post-deprivation recovery sleep, corresponding with an increased percentage of SWS (Buckley & Schatzberg, 2005).

Aside from sleep deprivation, sleep disruption due to fragmentation may also have a stimulating effect on the HPA axis. Adult and animal studies have identified pulsatile releases, or bursts, of cortisol secretions during episodes of wakening during sleep. When sleep is highly fragmented, precluding sufficient SWS, cortisol levels continue to rise throughout the night leaving morning cortisol levels higher than if sleep was undisturbed (Buckley & Schatzberg, 2005). Slow wave sleep cycles are greatly reduced when sleep is fragmented and instead, the sleep period is comprised of a higher percentage of "light" sleep (Stages 1 and 2). Cortisol levels have been found to be higher after fragmented sleep due to reduction of SWS inhibitory effects and increases in cortisol releases associated with wakening (Wesensten, Balkin, & Belenky, 1999).

There have been very few studies that have investigated the effects of fragmentation or variable sleep on HPA axis functioning in children. In animals, and potentially in young humans, there is a hypo-responsive period of the HPA axis in early life that functions to protect the developing brain from high elevations of cortisol (Gunnar & Donzella, 2002). However, animal studies of neonate rats have demonstrated heightened levels of cortisol following sleep deprivation even during this hypo-secretory protected period (Hairston et al., 2001). This suggests that sleep is a key mechanism for the regulation of diurnal cortisol and the tight linkage is maintained even when the stress response is less strongly coupled with cortisol increases. Furthermore, there is evidence

that sleep duration may have less of an impact on HPA functioning than sleep quality, suggesting that fragmentation or irregularity may have more influence over HPA functioning than the length of time spent in sleep (Capaldi II, Handwerger, Richardson, & Stroud, 2005).

Other studies have found that both quality and quantity are important considerations since increased sleep duration also increases the likelihood of engaging in SWS. El-Sheikh and colleagues (2008) investigated potential directional relationships between actigraphy-derived sleep measures and afternoon cortisol secretion. They found that sleep problems related to impaired quality (e.g., increased sleep activity, more wake time after sleep onset, and decreased sleep efficiency) predicted increases in cortisol, while total sleep minutes, or duration, predicted decreased afternoon cortisol (El-Sheikh, Buckhalt, Keller, & Granger, 2008). In addition, they found evidence for a bidirectional relationship as higher afternoon cortisol values predicted the same sleep variables. One limitation of this study is the reliance on sleep and cortisol values averaged over multiple days. This analytic approach precludes the ability to identify influences of variability in the systems.

Sleep and Behavior

Sleep researchers have also begun to investigate variability in sleep as a measure of overall regulation and predictor of behavior problems (Bates, Viken, Alexander, Beyers, & Stockton, 2002; Gruber & Sadeh, 2004; Gruber, Sadeh, & Raviv, 2000; Halpern, Maclean, & Baumeister, 1995). They argue that circadian regularity, or

regulation of sleep and arousal states, is a marker of developmental maturation and is essential for self-regulation. If arousal is poorly regulated, individuals are not able to effectively modulate and orient attention. Other associated functions such as information processing, learning, and memory are also compromised (Gruber & Sadeh, 2004). As an example, Gruber & Sadeh (2004) found that within-child irregularities in sleep quality, sleep quantity, and sleep schedule were related to poor outcomes on both simple and complex neurobehavioral tasks in non-disordered children. They also found some relationships between mean levels of sleep and neurobehavioral functioning, but the relationships were not nearly as consistent and only reached significance for the complex neurobehavioral tasks.

Compromised sleep and sleep inconsistency have been associated with internalizing, externalizing, and inattentive problem behaviors (Chervin, Dillon, Archbold, & Ruzicka, 2003; Dahl, 1996; Ivanenko, Crabtree, & Gozal, 2004; Owens, 2005). In fact, sleep disruption is so commonly observed in affective and anxiety disorders (e.g., major depression, post-traumatic stress disorder, adjustment disorder), that it is a diagnostic criterion in the DSM-IV (American Psychiatric Association, 2000). Correspondingly, there may be a stronger association in young children between sleep disruption and angry, aggressive, or delinquent behaviors rather than sadness, fear, and withdrawal problem behaviors (Aronen, Paavonen, Fjallberg, Soininen, & Torronen, 2000; Lavigne et al., 1999). Observations gained from parent reports or clinical records have described irritability and oppositionality as frequently occurring after a night of disrupted sleep. In a study investigating differences between "good sleepers" and "poor

sleepers", as assessed by actigraphic evaluation of nighttime awakenings and sleep efficiency, Sadeh, Gruber, and Raviv (2002) found that poor sleepers had elevated scores on delinquent behavior and thought problems, indicating that sleep disruption in young children may be specifically related to increases in disruptive problem behaviors. Dahl (1996) suggests that sleep impairment creates a disinhibited effect associated with poor self-monitoring of behavior. It may be this weakening of self-regulation paired with the decreased threshold for negative affectivity that may underlie these aggressive and oppositional symptoms.

Other researchers have presented strong cases for night-to-night variability as contributing to behavior problems in nonclinical samples. In a community preschool sample, Bates and colleagues (2002) found inverse relationships between within-child variability of the duration of parent-reported nightly sleep and adjustment to preschool, as measured by school-based non-compliant behaviors. The authors found no relation between school adjustment and total night sleep or lateness of bedtime. They acknowledged that a weakness of sleep research has been its reliance on correlational designs rather than testing causal hypotheses. They presented an argument that sleep variability in fact may cause compromised socioemotional outcomes, but conceded that this hypothesis remains untested in the literature. The current study will extend this work and will further investigate the associations with sleep variability on disruptive behaviors (e.g. non-compliance).

There is some evidence of a causal relationship between variable sleep disruption and difficulties regulating behavior, emotion, and attention. However, these types of

associations have very rarely been tested as the majority of sleep studies have employed correlative approaches. One study investigating changes in behaviors after medical intervention for sleep disordered breathing, found a causal relationship between fragmented sleep and inattentive/hyperactive behaviors. One of the study groups received a tonsillectomy or other indicated medical intervention to address the night breathing problems, while the comparison group did not receive medical treatment. After the procedure, behavior problems and inattention decreased and school grades increased from baseline measures in the treatment group (Gozal, 1998). The striking improvements did not appear to be due to a developmental trend as comparison groups, children without sleep disordered breathing and children with untreated sleep disordered breathing, did not show any behavioral or academic improvement.

A variety of indicators of sleep disruption have been associated with inattentive and hyperactive problem behaviors. For example, increased sleep latency, decreased sleep duration, and decreased sleep efficiency have all been linked with greater inattention and hyperactivity (Owens, 2005; Sadeh, Pergamin, & Bar-Haim, 2006; Shur-Fen Gau, 2006). The most consistent finding across studies has been increased nighttime activity, indicating increased restless sleep (Owens, 2005; Sadeh, Pergamin, & Bar-Haim, 2006). Studies have also found significant associations with total sleep duration and inattentive or hyperactive behaviors, although these findings have varied with age. In a meta-analysis, Sadeh and colleagues identified that younger children diagnosed with ADHD slept for a shorter duration, while older children diagnosed with ADHD slept for a longer duration than age-matched controls (Sadeh, Pergamin, & Bar-Haim, 2006). This

may indicate that some children with early-diagnosed ADHD may instead be experiencing chronic sleep deprivation that contributes to the intensity of their symptoms.

The current study has the potential to contribute to the literature on sleep variability by examining how variability of sleep in young children is related to problem behaviors as well as HPA axis functioning. Sleep variability was included as a measure of a broadened definition of "good" sleep that moves beyond prior definitions including only quality and quantity variables. Furthermore, the current study investigated how shifts in sleep variables impact daily changes in both behavior and neurohormonal response. Behavior was measured using a daily checklist approach to allow for detection of daily changes in child behaviors. This approach allowed for clarification of how a night of "poor" sleep may impact behavior or cortisol levels the following day.

Secondly, the current study investigated how associations between sleep and cortisol or behavior may be different in high risk children in comparison with their lower risk peers. As was found in Study 1, children who experience greater amounts of stress may be the most vulnerable to sleep disruption. Specifically, it was found that children in the foster system had greater difficulty falling asleep and that their sleep duration and latency was more variable than children from upper middle-income homes. There is also a great deal of evidence that children in the foster care system are most vulnerable to behavioral and emotional disturbances and many meet criteria for multiple psychiatric diagnoses at early ages. Children living with their biological parents were compared with children living with foster care providers to investigate whether sleep disruption impacted neurohormonal and behavioral functioning of one group to a greater extent than the other.

In the current study of young children ages 3- to 7-years-old, it was expected that increased variability of sleep, measured by actigraphy, would be associated with elevations in both morning and afternoon cortisol as well as elevations in various problem behaviors. Secondly, it was expected that impairments in sleep (e.g. decreased quantity or quality) would predict increases in morning and afternoon cortisol as well as increases in problem behaviors. These analyses investigated how daily changes in sleep might impact functioning on the following day. Based on prior findings, variables reflecting sleep quality such as nighttime awakenings, nighttime activity, and sleep latency as well as sleep duration, a measure of quantity were of specific interest, although other variables were included in analyses for exploratory purposes.

Methods

Participants

Participants were 79 children (41 females) between the ages of 3- and 7-years-old (M=5.25, SD=1.05). The ethnic makeup of the sample was representative of the community from which it was drawn (Eugene, Oregon). The children were primarily from European-American descent (82.3%), with Latino (7.6%), Native American (6.3%), and African American (3.8%) ethnicities also represented. Study children were from two care groups as 32 children were in foster care and the other 47 community children were living with their biological parents. Please see Study 1 for a detailed discussion of recruitment procedures and study groups.

Measures

Actigraphy. Sleep data was collected using actigraphy over five consecutive nights as described in Study 1. All sleep variables of interest were averaged over the five study days to compute mean values. Furthermore, the variability of sleep measures was computed by taking the standard deviation over the five study days. Variables of interest were indicators of sleep quantity (i.e. sleep duration), quality (i.e. sleep percentage, nighttime activity, number of wake episodes), and schedule (time of sleep onset, time of wake onset, sleep latency).

Sleep Diary. Caregivers reported subjective impressions of their child's sleep quality and quantity, daytime activity, and daytime sleepiness on the 15-item Sleep Diary (Sadeh, 1994). The diary was completed by parents nightly after the child was asleep and again in the morning after the child woke.

Parent Daily Report (PDR). PDR is a 53-item checklist in which parents report whether a series of problem behaviors occurred and the extent to which the behaviors were stressful (Chamberlain & Reid, 1987). The current study used a version of the PDR modified to reflect frequency and intensity of behaviors. Parents were asked to record a 0 if a specific behavior did not occur in the prior 24 hour period, a 1 if the behavior occurred but was not stressful, and a 2 if the behavior occurred and was stressful. At the end of the checklist, parents were asked to assign a subjective grade to best describe the day from A through F. PDR has been reported to have good test-retest reliability and concurrent validity with other known problem behaviors (Chamberlain & Reid, 1987). In the current study, PDR scales of anxious/depressed problem behavior, disruptive problem

behavior, and inattentive/hyperactive problem behavior were used. Due to the low endorsement of anxious/depressed problem behavior in the sample, this scale could not be used in subsequent analyses.

Salivary Cortisol. On study days 3 through 5, parents were also instructed to collect morning and evening saliva for cortisol samples within 30-min of waking and bedtime, respectively. Parents were asked to complete the collection before the child brushed his or her teeth and no less than an hour after eating in order to prevent residual food and/or blood in the saliva from interfering with cortisol readings.

Children were instructed to chew Trident Original sugarless gum for 1-min prior to the cortisol collection to stimulate available saliva. Other brands and flavors of gum were forbidden because they have been previously shown to interfere with cortisol values (Schwartz, Granger, Susman, Gunnar, & Laird, 1998). Parents were trained to insert a plain cotton salivette (Sarstedt, Inc., Newton, NC) into the child's mouth using the collection tube. The child was asked to chew on the salivette for 1-min and then deposit the salivette into the pre-labeled plastic collection tube when finished. If the collection was spoiled by touching another surface (e.g. fell on the floor, touched by a hand), parents were asked to collect another saliva sample. Immediately after collection, parents recorded the exact time of collection on the label. Saliva samples were immediately stored in the refrigerator to prevent degradation until they were transported to the laboratory by a staff member where they were stored at -5°F until assayed.

Cortisol samples were assayed in the laboratory using High Sensitivity Salivary Cortisol Enzyme Immunoassay Kits (Salimetrics LLC, State College, PA). Each

participant's samples were assayed together in duplicate to reduce within-subject variability. Duplicate samples were checked for consistency and were then averaged and were re-assayed if the two samples differed by more than 15%. The intraassay and interassay coefficients of variance were 2.62% and 11.16%, respectively.

Caregivers completed a brief questionnaire following morning and evening cortisol samples that included sampling times, medications, and eating and sleeping behavior, since cortisol levels can be affected by fluctuations in these variables (de Kloet, 1991). Children using steroid-based medications (e.g. asthma inhalers) on a regular basis were excluded from the study and parents were instructed to refrain from using short-term steroid-based medications during the study period. The questionnaires were inspected to ensure compliance with sampling during the specified window (i.e. within 30 min of waking and bedtime).

Cortisol data was initially investigated to ensure that each participant did not have more than one day of missing data. Several participants had missing cortisol data due to lack of available saliva, tainted samples (e.g. taken too late after wake, contaminated by contact with surface), or levels that were biologically implausible (i.e. >2.0 μg/dl). For the wake samples, there were 8 missing samples and 10 missing cortisol samples at bedtime. One participant did not have any valid cortisol samples and was excluded from all analyses involving cortisol. All other participants had at least two days of wake and bedtime cortisol data.

Procedure

After completing recruitment and consent procedures (as described in Study 1), caregivers were trained to use the actigraphs and on appropriate procedures for cortisol collection. Parents were provided with a binder of materials (e.g. Sleep Diary, PDR, cortisol collection forms) to be completed twice daily, after evening lights out and after morning wake. The Sleep Diary was completed on all five study days and provided a subjective report of sleep quality, daily naps and nighttime sleep duration. Children were given a 2-day acclimation period to become comfortable with wearing the actigraph at night prior to collecting cortisol or behavior data. Therefore, the cortisol collection diary was completed on study days 3 through 6. The parent daily report (PDR) questionnaire was completed in the morning of study days 4 through 6. The PDR rating of the previous day's behaviors was completed in the morning so that nighttime behaviors could also be captured.

Upon completion of the six-day study period participating children were compensated with a bath mitt version of their actigraphy sleeve and caregivers were compensated with $100.

Results

Reliability of Measures

Stability of the repeated actigraphy, behavior and cortisol measures was examined through intraclass correlations (ICC). For most actigraphy variables, reliability estimates ranged from .70 to .89, which is considered to exceed the night-to-night reliability

standards (intraclass correlations greater than .70) proposed by Acebo et al., (1999). The number of nighttime waking and sleep duration had less night-to-night stability, .60 and .69, but approached the recommended values. Reports of behavior were highly stable across days of data collection for disruptive and inattentive/hyperactive scales, with reliability estimates ranging from .81 to .83. As expected, cortisol was less reliable, yet still adequate (reliability estimates for morning and evening measures of cortisol were .68 and .66 respectively). See Table 2 for descriptives of sleep, behavior, and cortisol measures.

Preliminary Analyses

Prior to the study's central analyses, age and gender were investigated to determine whether they are related to measures of sleep, behavior, and cortisol. Gender differences were tested through a series of *t*-tests, and Pearson correlations were used to determine the extent to which age was related to sleep, behavior, and cortisol. There were no gender differences or age associations for morning or evening cortisol. There were some expected gender differences in the measures of disruptive and inattentive/ hyperactive behavior. In accordance with prior research, boys displayed greater frequency of inattentive/hyperactive problem behaviors, $t(76)=2.61$, $p<.05$, and greater amounts of disruptive behavior, $t(76)=2.27$, $p<.05$, than girls. The *M*s and *SDs* for boys and girls were, respectively, .97 (1.23) and .41 (.83) for inattention/hyperactivity and 5.58 (4.77) and 3.54 2.54) for disruptive behavior. Neither of the behavior variables was associated with age.

Table 2

Descriptives for Measure of Sleep, Cortisol, and Problem Behavior

Actigraphy-Derived Sleep Measures	*M*	*SD*
Sleep Quantity		
Sleep duration (min)	571.78	39.94
Variability of sleep duration	48.16	24.93
Sleep Quality		
Sleep percentage	85.46	6.12
Sleep activity	49.94	10.53
Wake episodes	3.76	1.20
Variability of sleep percentage	4.92	2.48
Variability of sleep activity	6.11	2.40
Variability of night wakings	1.54	0.93
Sleep Schedule		
Sleep Latency	36.03	18.53
Time of sleep onset	21:09	48 min
Time of wake onset	6:44	46 min
Variability of sleep Latency	20.12	13.63
Variability of time of sleep onset	39 min	68 min
Variability of time of wake onset	37 min	22 min
Cortisol		
Morning	0.45	0.22
Evening	0.06	0.09
Problem Behavior		
Disruptive	4.82	4.23
Inattentive/Hyperactive	0.36	0.65

Note. All actigraphy variables are composites for five nights of sleep

There were very few associations of age with average sleep or variability of sleep, and no differences among any of the variables as a function of child gender. Younger children tended to have a longer sleep latency, r=-.25, p<.05, increased nightly activity, r=-.23, p<.05, and a trend toward a lower sleep percentage, r=.21, p=.06, indicating that sleep became less disrupted with age. Overall, higher variability of sleep was negatively

related to age. Specifically, increased variability of wake time, r=-.31, p<.05, nighttime activity, r=-.33, p<.05, and sleep percentage, r=-.43, p<.05, was associated with younger ages, indicating that sleep schedule and quality became more consistent with age. Due to the age and gender differences observed, these variables were controlled for in all subsequent analyses. Additionally, because Study 1 demonstrated that placement in either foster or community settings also was associated with sleep differences, care setting was controlled in the following analyses.

Associations among Sleep and Measures of Behavior and Cortisol

Mean level associations were first investigated to determine how sleep may be associated with behavior and neurohormonal activity (see Table 3 for correlations). The only averaged sleep variable that showed any association with behavior and cortisol was sleep duration. There was a trend for evening cortisol to be lower when sleep duration was greater, r=-.23, p=.06. There were no other significant mean level associations.

There were also very few significant associations between variability of sleep and problem behavior or cortisol values. Notable, however, children who had higher night-to-night variability of sleep duration also exhibited inattentive/hyperactive problem behaviors more frequently than children with more consistent sleep, r=-.27, p<.05. Furthermore, children who had greater instability of wake times also showed a trend toward lower cortisol, r= -.23, p<.06.

Table 3

Correlations between Actigraphy-Derived Sleep, Problem Behavior, and Cortisol

	Problem Behavior		Cortisol	
	Disruptive Behavior	Inattention/ Hyperactivity	Morning	Evening
Mean Sleep (N=71)				
Sleep Duration	0.13	-0.10	0.13	-0.22[a]
Sleep Latency	-0.20	-0.08	-0.08	0.02
Activity during Sleep	-0.04	0.01	-0.12	-0.10
Wakings	0.01	-0.05	-0.09	-0.11
Percent Sleep	0.09	0.01	0.07	0.07
Sleep Onset Time	-0.21	0.01	-0.19	0.09
Wake Onset Time	-0.11	-0.15	-0.01	0.06
Sleep Variability (N=71)				
Sleep Duration	-0.08	0.27*	-0.17	-0.02
Sleep Latency	-0.19	0.06	-0.21	0.02
Activity during Sleep	-0.16	0.09	0.05	-0.05
Wakings	0.01	-0.10	-0.12	-0.19
Percent Sleep	-0.09	0.03	-0.15	-0.14
Sleep Onset Time	-0.04	-0.09	0.07	0.20
Wake Onset Time	-0.10	0.18	-0.23[a]	-0.07

Note: $*p<.05$ ^{a} $p<.06$

Prediction of Behavior and Cortisol Outcomes from Sleep Measures

Mixed-effects linear models with autoregressive error structures were used a means for investigating potential associations between sleep and morning cortisol, evening cortisol and disruptive behavior. Mixed models are advantageous for repeated data since they allow for estimation of participant-specific fixed effects (e.g. regression coefficients) and participant-specific random effects (e.g. time-varying). In this modeling approach, both the means and variances are estimated for each participant. Furthermore,

mixed linear models can easily manage missing data due to the maximum likelihood fit approach, so participants who have missing data points can be retained in the model. Refer to Bagiella, Slone, and Heitjan (2000) for a detailed discussion of the use of mixed-effects models.

For the cortisol data and disruptive problem behavior data, the mixed-effects models were estimated using SAS PROC MIXED, version 9.1.3 (SAS Institute Inc., 2006). A first-order autoregressive variance structure was selected due to the superior fit for the data. A variety of error structures were compared (e.g. unstructured, compound symmetry, etc.), and the -2 times the residual log likelihood indicator (-2RLL) was the smallest for the autoregressive structure, indicating best fit. Autoregressive error structures assume that data points closest in time will be most highly correlated and that correlations between repeated measures will decrease as the lag time between measurements increases. This approach allows for estimation of different variances at each time point by not fixing the variances to be equal.

In these three models, sleep on study days 2, 3, and 4 predicted change in behavior or change in cortisol on study days 3, 4, and 5, by controlling for associations with the previous day. Three sleep variables, sleep latency, duration, and nighttime activity, were selected as predictors in the model due to their low inter-correlations and significant associations with the variables in prior studies. Gender, age (median split), and care group (e.g. foster care or community care) were entered in the model as independent variables to determine whether some children may be more vulnerable to behavioral or cortisol dysregulation after disrupted sleep than others. None of the sleep

predictors were significant in any of the models indicating that changes in sleep on measures of nighttime activity, duration, or sleep latency over the three days were not associated with changes in either disruptive behavior or either cortisol measure.

For the prediction of inattentive/hyperactive behavior, a generalized linear mixed-effects model for binomial data with an autoregressive error structure was used due to the dichotomization of the measure. This model was identical to the previously described models, although it used PROC GLIMMIX in SAS version 9.1.3 (SAS Institute Inc., 2006), which is designed to be used with binary data. In this model, an autoregressive error structure also was the best fit for the data as indicated by the lowest -2 times the residual log pseudo-likelihood indicator. The binomial model was also set up so that sleep latency, duration, and nighttime activity were the predictors of presence or absence of inattentive and hyperactive behaviors. Age, group, and gender variables were also included as independent variables as in the continuous model.

In the binomial model, changes in sleep duration significantly predicted changes in inattentive/hyperactive behavior, $F(1, 203.1)=5.36$, $p<.05$. Overall, at lower sleep durations, inattentive and hyperactive behaviors increased. Furthermore, the probability of inattentive/hyperactive behaviors differed depending upon the study group and child's gender, $F(1, 114.5)=23.97$, $p<.001$ and $F(1, 101.3)=7.13$, $p<.01$, respectively. Specifically, this indicated that children in the foster care group and males were more likely to display problem behaviors at low sleep durations than were children in the community group or females.

The foster group showed much greater vulnerability for problem behaviors at low sleep durations than did the community group (see Figure 9). At 400 min of sleep, the odds ratio was 4.6 for problem behavior in foster children while it was only .20 in community children. This indicates a significant difference between groups as foster children were over five times more likely to display inattentive and hyperactive behaviors after shortened sleep durations than community comparison children, $t(73)=4.22$, $p<.001$. At longer durations of sleep (e.g. 700 min), the groups converged and occurrence of problem behaviors in both groups decreased and were no longer significantly different, $t(73)=.35$, $p>.05$. The odds ratio for problem behaviors at 700 min in the foster group was .35 and .02 for the community group. The model was also run with four care groups (RFC, TFC, LIC and UMC). Since the odds ratios did not significantly differ between the RFC and TFC foster groups or the LIC and UMC community groups, it was determined that the model with the collapsed care groups (foster vs. community) fit the data best due to the increased parsimony.

A similar pattern was seen in males who generally exhibited a greater probability of inattentive/hyperactive behavior than did females. Across shortened sleep durations, males had a marginally higher probability of exhibiting inattentive/hyperactive behaviors than did females, $t(73)=1.40$, $p=.08$, although at longer sleep durations, inattentive and hyperactive behaviors converged to be non-significant, $t(73)=.12$, $p>.05$, (see Figure 10). At 400 min of sleep, males had an odds ratio of the occurrence of inattentive/hyperactive behaviors of 1.89, while the odds ratio for females was .49. This suggests that males were three times more likely to exhibit inattentive and hyperactive behaviors following

shortened sleep durations. At 700 minutes of sleep, the odds ratios decreased for both genders and became non-significantly different, (odds ratio was .14 for males and .04 for females).

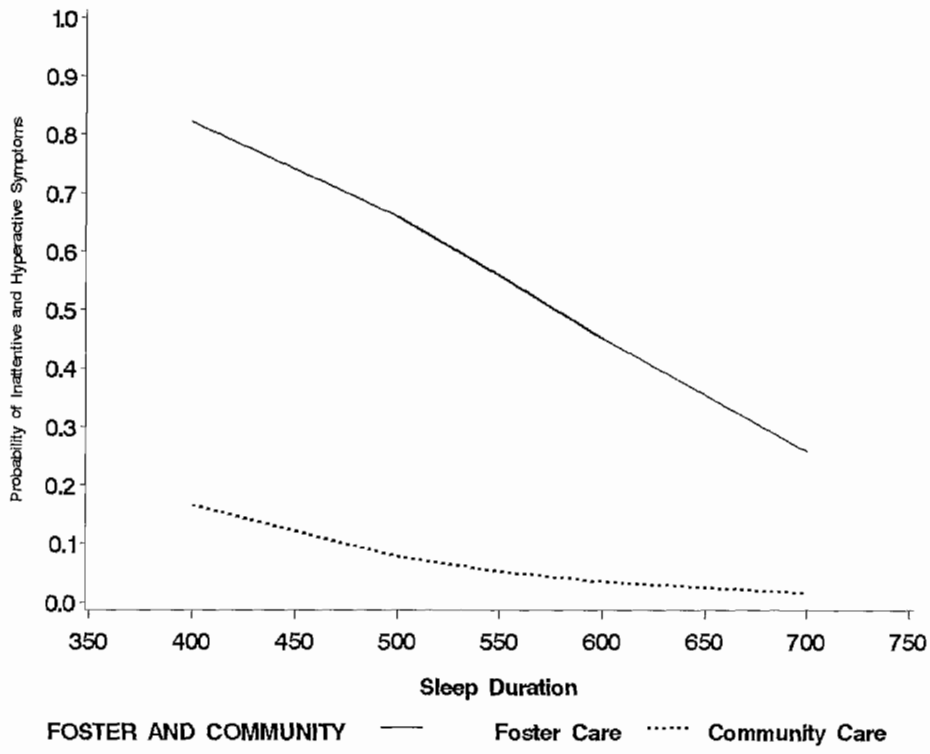

Figure 9. Probability of Hyperactive/Inattentive Behavior as a Function of Sleep Duration and Care Group

In summary, inattentive/hyperactive problem behaviors were reported less frequently for all children with increased duration of sleep. However, some children were especially vulnerable to decreased sleep and were more likely to display inattentive/ hyperactive behaviors after inadequate sleep durations.

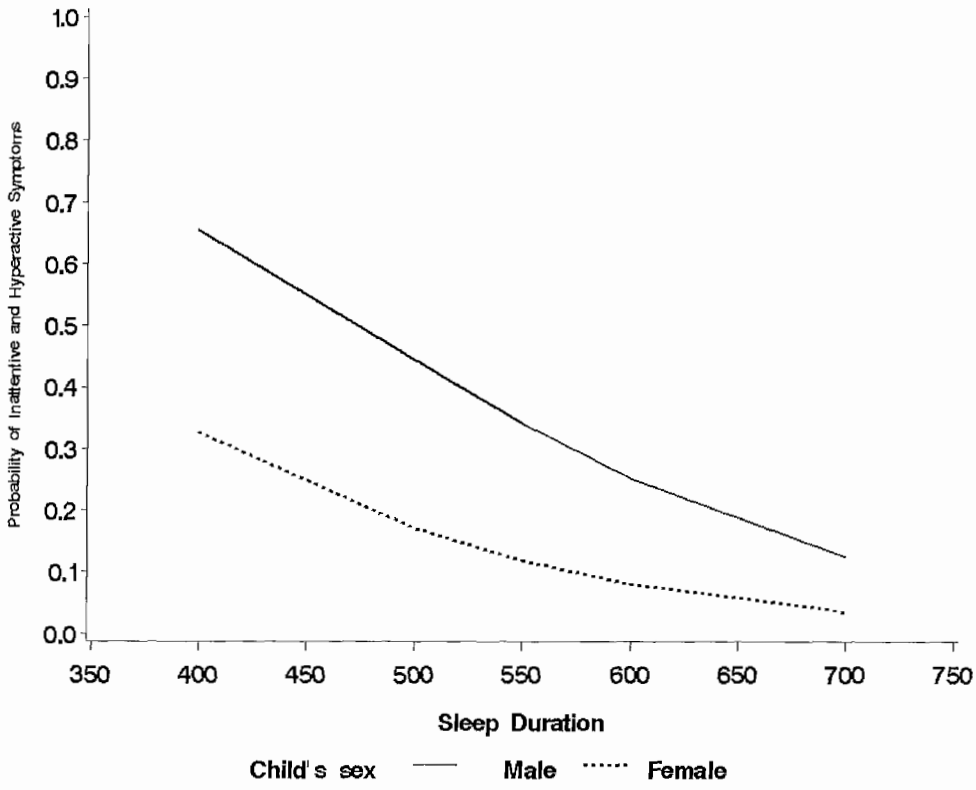

Figure 10. Probability of Hyperactive/Inattentive Behavior as a Function of Sleep Duration and Gender

Discussion

The current study used actigraphy and varied analytic approaches in an effort to tease apart potential associations among sleep and behavioral or neurohormonal processes. Furthermore, in the current study, differences between foster and community children as well as between genders were investigated as potential individual difference factors associated with increased risk for negative outcomes following sleep disruption.

The current study addresses the shortcomings of the current literature by investigating the impact of sleep on measures of behavior and cortisol using actigraphic measurement of sleep. One of the study's primary strengths is the inclusion of both

correlational and predictive approaches to understanding the relations between variables. Prior literature has indicated that there may be a bidirectional relationship between the variables and while changes in sleep may drive changes in both cortisol and behavior, the reverse relationships may also exist. Correlational analyses were important for an initial descriptive investigation of potential bidirectional relationships in this sample. However, in this study, the use of predictive models was a more informative mechanism for clarifying associations between variables and directionality of effects. These models allowed for investigation of differences in these associations among the study groups.

Most of the hypothesized bidirectional relationships were not supported by the data. However, greater sleep durations were marginally related to lower levels of evening cortisol, as was expected. Although the effect was small, this finding corresponds with El Sheikh and colleagues' bidirectional findings that sleep disruption (e.g. shorter duration) predicted increases in mean afternoon cortisol values and afternoon cortisol values predicted increases in sleep disruption (El-Sheikh, Buckhalt, Keller, & Granger, 2008). This is consistent with Hatzinger and colleagues' (2008) report that disrupted sleep was not only associated with increases in diurnal cortisol, but also was associated with increased cortisol responsivity to stressors. Although this association is only cautiously considered as a potential link between sleep-arousal and systems due to the marginal effect, the correspondence with past literature suggests that it may warrant further investigation.

Although it was expected that higher sleep variability across measures would be associated with higher morning and evening cortisol, there was very little support for

these hypotheses. One marginally significant finding was the positive association between the variability of wake onset time and lower morning cortisol. Although this association was not in the predicted direction, it may suggest that some children with variable wake times may fail to signal their caregivers upon waking, thereby prolonging the time between the morning cortisol peak and collection time. This is supported by mean wake latency values exceeding 30 minutes. Caregiver reports of wake times were closely related to the time at which they indicated removal of the actigraph, thereby suggesting that some caregivers were unaware of their child's actual wake time. This finding may have implications for future studies collecting cortisol in children. Specifically, actigraphy may be a useful tool to determine true wake time, which may add to the accuracy of morning cortisol values.

There were also very few associations between variability of sleep and mean levels of cortisol and behavior. One expected finding supported by the data was the positive association between variability of sleep duration and endorsement of inattentive/hyperactive problem behaviors. Past research suggests that inattentive and hyperactive symptoms may be most strongly related to sleep inconsistency rather than the average quantity or quality of sleep over time (Gruber, Sadeh, & Raviv, 2000).

Although there were no significant effects in the models predicting cortisol and disruptive problem behavior from sleep, there were intriguing effects in the logistic model predicting inattentive/hyperactive problem behaviors from sleep. Building upon the results from the correlation analyses of sleep variability, the logistic model indicated that when sleep duration was shortened, the probability of occurrence of inattentive and

hyperactive behaviors increased on the following day. The autoregressive models in this study provided a mechanism to examine changes in behavior from day-to-day variations in sleep. This relationship supports prior findings in the sleep deprivation literature linking increased sleep debt with inattentive and hyperactive behaviors. The strength of the current study is that it suggests that normative variation in young children's sleep duration, rather than experimentally restricting sleep duration, can be sufficient to be associated with inattentive behaviors.

The current study also builds on prior findings that some children are more susceptible to sleep and behavior problems than others. The foster children in this sample were five times more likely to display inattentive and disruptive problem behavior after shorter sleep durations than were community children living with their biological parents. The probability of inattentive and hyperactivity problem behaviors exceeded 80% at very low sleep durations (i.e. 400 minutes), while community children had less than a 20% probability of problem behaviors after obtaining the same amount of sleep. At greater sleep durations, the probability of behavior problems converged significantly, although corresponding to prior literature, the foster children still displayed more behavior problems. This finding emphasizes the importance of sleep in this vulnerable group of children as a means of regulating behavior. Furthermore, it implicates negative outcomes in domains of learning, school readiness, and behavior management for foster children whose caregivers do not emphasize good sleep hygiene and consistent bedtime routines.

Past findings have suggested that foster children have regulatory problems (e.g. behavioral, emotional, and physiological) at a much higher rate than do community

children (Clausen, Landsverk, Ganger, Chadwick, & Litrownik, 1998). The results of Study 1 also indicate that foster children have problems with sleep at a higher frequency than do community children. Together, results of these studies imply that sleep may be an important avenue for intervention on inattentive and hyperactive behavioral problems in foster children. This has important implications because children with difficult to manage behavioral problems are also at heightened risk for increased placement instability, which also increases risk for negative psychosocial outcomes (Newton, Litrownik, & Landsverk, 2000). Ensuring that young foster children receive adequate sleep may be an important first step for decreasing the frequency and intensity of inattentive and hyperactive problem behaviors.

Gender was also another determinant of children at-risk for increased inattentive and hyperactive behaviors following decreased sleep duration. This gender discrepancy has been reported previously in the ADHD literature as it has been estimated that boys are nine times more likely than girls to receive a diagnosis of ADHD (American Psychiatric Association, 2000) and are also more likely to display sub-clinical symptoms. In this study, at shorter sleep durations, males were more likely than were girls to display inattentive and hyperactive problem behaviors. In addition, there were no gender differences across any of the sleep measures, implying that the gender differences were not a function of males obtaining less overall sleep. However, there was very little difference between genders in the likelihood of displaying inattentive or hyperactive symptoms at longer sleep durations. This indicates that males may be more susceptible to

problem behaviors at shortened sleep durations, which may be a potential contributor to the gender differences in frequency of ADHD symptoms reported in the literature.

The results suggest that the current literature on inattentive and hyperactive behaviors in young children should be critically examined to disentangle the potential impacts of disrupted sleep on these behaviors. It is currently unclear how much of the reported inattentive and hyperactive behaviors may be attributable to problematic sleep at these early ages. This point is supported by the growing trend for children in the United States to sleep for a shorter nightly duration than is recommended. In the National Sleep Foundation's 2004 nation-wide poll, it was found that toddler and preschoolers slept approximately one to two hours less than the recommended 11 to 13 hours (National Sleep Foundation (NSF), 2004). In the current study, objective measures of average sleep indicated that children received even less sleep than the caregiver-reported national average. This is most likely a function of poor reliability of subjective reports of sleep, since the sleep durations reported in the sleep diaries were similar to what was reported in the national poll.

Although prior research has found that sleep is related to increases in disruptive behavior, there was no evidence for similar associations in this study. These results support Hatzinger and colleagues' study of preschool-aged children that found that children who were poor sleepers self-reported increases in impulsive behavior, but there were no associations between objectively-reported sleep and teacher-report of behavior problems in externalizing domains (Hatzinger et al., 2008). The use of objective sleep measures was an important similarity between the current study and Hatzinger and

colleagues' work that may account for the discrepant findings. It is important to distinguish studies using objective measures of sleep from studies relying upon caregiver-reported measures of sleep because the associations between daytime behavior and sleep have been found to be inflated when caregiver reports are used due to caregiver reporting bias. Although caregiver reports have been found to be somewhat reliable reports of nightly sleep, they typically are not sensitive to the intensity of sleep disturbance. For example, Hatzinger and colleagues also collected caregiver reports of nightly sleep and found that caregiver-reported sleep was associated with objective reports of sleep, although caregiver-report was unrelated to classification of children as "poor", "normal", or "good" sleepers by objective measures. Therefore, the use of actigraphy in the current study may have been a useful tool to reduce the effects of caregiver bias on the associations between disruptive behavior and sleeping patterns.

It was also anticipated that sleep would drive changes in both morning and evening cortisol. However, neither relationship was supported in the mixed-effects models. One potential explanation between results of the current study and past findings may be due to the home-based assessment methodology. Other studies that detected these associations have primarily been polysomnography studies in which cortisol was measured in the laboratory immediately after wake. Very few studies have measured these associations in children, due to the difficulty of collecting polysomnography data in these age groups. It is more feasible to collect actigraphy data in younger children because it is unobtrusive and it does not interfere with children's typical bedtime routines or sleeping patterns. However, morning cortisol collections in actigraphy studies may not

be an accurate representation of children's peak cortisol levels due to the variability with which children alert their caregivers upon wake. Therefore, the time lapse between wake and collection of morning cortisol in the present study may have been too extensive or too inconsistent to detect the expected associations.

In addition to the relatively few studies investigating morning cortisol and sleep, there have been even fewer studies investigating associations between evening cortisol and sleep in children. In one of the few relevant studies in this area, El-Sheikh and colleagues found that sleep was associated with afternoon cortisol in children (El-Sheikh, Buckhalt, Keller, & Granger, 2008). Based on these findings, it was expected that sleep may drive changes in evening cortisol, although the current study did not support this hypothesis. One potential explanation for the marginal correlational effect and lack of predictive effects in the current study is that cortisol was collected in the evening before bed rather than the afternoon. Since bedtime is a stressful time for many children and bedtime resistance is among the most common sleep problem in young children (Mindell, 1993), the added stress may have enhanced evening cortisol levels, thereby decreasing the overall effect. Perhaps measures of afternoon cortisol, as were employed by El-Sheikh's group, may be less likely to be impacted by bedtime stressors and would be a better indicator of the diurnal pattern of cortisol secretion.

Another potentially important difference between the current study and past literature on sleep and cortisol associations is the age of the sample. Children in the current study ranged between three and seven years old, which may be a developmental period in which cortisol secretion is hyporesponsive (Gunnar & Fisher, 2006). There is

evidence gained from animal models that at early ages, cortisol levels may be less responsive to stressors in order to protect the developing brain from negative effects of cortisol hypersecretion. It is possible that during early periods of development, cortisol levels are less impacted by sleep disruption than at later periods. This rationale may be supported by outcomes of a recent study of preschool-aged children where cortisol was related to sleep in only the most extreme groups of children, but not in the normal range of sleep (Hatzinger et al., 2008). This may indicate that at young ages, cortisol may be less responsive to changes following sleep disruption, unless this disruption is very extreme.

Limitations and Future Directions

Although the use of a parent daily report of behavior was important for detecting day-to-day variability in behavior, a potential limitation of the current study is the lack of a diagnostic behavioral measure. This may be one reason for the minimal association between variability of sleep measures and behavior. Prior studies using diagnostic indicators of ADHD or externalizing disorders have reported much stronger associations between variables. There may have been important heterogeneity of behavior within the study sample due to the inclusion of children in the foster care system, who may have exhibited clinical levels of behavior problems. A valuable next step for future studies would be to examine differences in associations between disrupted sleep and disruptive or hyperactive/inattentive behavior between children who meet diagnostic criteria for externalizing-type disorders or ADHD and those children exhibiting disruptive or

inattentive/hyperactive behaviors within the normal range. Specifically, it will be important to more thoroughly investigate whether there is an increased vulnerability for inattentive and hyperactive symptoms following impaired sleep in children diagnosed with ADHD. These types of studies would provide valuable information to caregivers and treatment providers regarding the value of adequate sleep for decreasing behavior problems.

Furthermore, the low parental endorsement of anxious/depressed problem behaviors precluded investigation of these types of problem behaviors in the current studies. This is likely to be attributable to the young age of participants, as internalizing-type problem behaviors occur less frequently in this age group. Furthermore, parents are typically less accurate at reporting on anxious/depressed problem behaviors than disruptive problem behaviors. As internalizing-type behaviors are closely related to sleep disruption in adolescents and adults, future studies are needed to investigate at what period these types of behaviors become linked with sleep disruption.

Due to design limitations of the current study, models in which sleep disruption was predicted from cortisol or behavior could not be tested. Instead, the models solely tested the predictive ability of sleep disruption, although prior literature also supports these alternative associations. Future studies would benefit from understanding more about directionality of effects by incorporating both types of models in analyses.

Another limitation of the current study may be the timing and frequency of cortisol collections. As previously discussed, the relative lack of significant associations between sleep and cortisol within this sample may have been due to latency between

wake and collection time in the morning and the increased stress at associated with bedtime. Other studies that have found significant relationships have collected multiple samples of cortisol at each time of day and then averaged those samples. This may add to the reliability of cortisol across days because the potential effects of a time lag between wake and the collection period may be reduced. Furthermore, future studies may want to investigate the diurnal pattern of cortisol by collecting samples in the morning, afternoon, and evening. More frequent sampling of cortisol may be helpful in understanding how disrupted sleep may impact or be impacted by diurnal cortisol rhythms.

Another important future study will be to investigate the impact of bedtime resistance and stressful nighttime interactions with parents on cortisol and sleep. Although the current study did not include a descriptive measure of the intensity of bedtime resistance and family stress, this type of information may be especially important in future studies investigating associations between sleep and cortisol.

Although the current study did not find many of the anticipated associations between cortisol and sleep, the results may have opened up for new avenues of study of these relationships in young children. Furthermore, it contributed significantly to our understanding of inattentive and hyperactive behaviors following disrupted sleep. Results further clarified that some children, namely boys and foster children, may be especially vulnerable to negative effects following restricted sleep durations. Although more research is needed, results of this study suggest that sleep interventions may be an indicated first line approach to the treatment of inattentive and hyperactive problem behaviors.

CHAPTER IV

GENERAL DISCUSSION

These dissertation studies contribute to our knowledge of sleep patterns in foster care and highlight potential individual difference factors that may contribute to vulnerability for negative outcomes following disrupted sleep. One especially intriguing finding of these studies was that foster children were at risk for some aspects of sleep disruption and negative outcomes of sleep, but this was not true across all variables. It is encouraging that foster children's early experiences were not attributable to widespread and serious sleep disruption. This may be an indication of a protected period in development where sleep is difficult to disrupt. The EIFC study has identified similar patterns across other basic neural functions, such as cortisol. If basic processes, such as sleep, are protected in early childhood, the questions then become about timing of protected periods and potential for malleability of systems during those periods. In fact, the absence of differences among groups in the current studies may be an indication of an optimal time for intervention because these are periods of significant ongoing development. It is conceivable that the extent of dysregulation may not be completely evident until after these functions are no longer protected, which may be as late as adolescence.

The indications of positive intervention effects of the MTFC-P study on the foster children in the study may be evidence that sleep patterns are still highly malleable in early childhood, which would suggest an ideal period for intervention. At the very least, Study 1 indicated that treatment foster care providers may be more aware of their children's needs around bedtime. The treatment foster group looked very similar to the regular foster group in their difficulty initiating sleep, which is possibly attributable to increased vigilance. However, treatment foster care providers seemed to acknowledge this difficulty and began putting children to sleep earlier than the other groups. This basic change in sleep schedule addressed the foster children's vulnerability toward insufficient duration of sleep and children in the treatment group obtained the most sleep of any group. This finding is quite applicable to the MTFC-P philosophy of initiating prevention efforts before problems arise. At least for some children, the MTFC-P intervention may have altered the trajectory of sleep problems so that they are less at risk for developing sleep disorders at later developmental periods.

Another aspect of these dissertation studies that may be especially important when studying sleep and foster children is the idea of negative impacts of systemic instability. It is common to investigate individual differences in variables averaged over multiple timepoints. In the EIFC studies on cortisol patterns and in the current studies on sleep, there is some suggestion that instability may be equally or more informative than stability across systems. Specifically, the outcomes of Study 2 indicated that the night-to-night changes in sleep were the most indicative of negative outcomes. In fact, mean levels of sleep were not associated with any measures of behavior or cortisol levels. The

variability of sleep duration was the more informative measure. Study 1 suggested that the foster groups had the most variable sleep duration. Study 2 built upon this finding and showed that the foster groups were also five times more likely to display behavior problems following shorter sleep durations than community comparison children. Together these studies indicate that a marker of vulnerability in the foster groups may be the instability of their nightly sleep rather than overall sleep.

Although it was not explicitly tested in either of these dissertation studies, the MTFC-P intervention may address sleep instability. Consistency is a major emphasis of the treatment program. This theme is taught across settings and situations. For example, treatment foster care providers are trained on how to respond to positive and negative child behaviors in a manner that is supportive and predictable for the children. Furthermore, they are encouraged to help the foster children understand their environment by keeping consistent routines and preparing children prior to any deviation from those routines. Keeping the environment as consistent as possible may be important means for improving overall stability in foster children's regulatory processes. An important next step may be to investigate whether teaching foster care providers about the importance of sleep and emphasizing good sleep hygiene (e.g., consistent bedtimes, sufficient sleep duration) as part of the MTFC-P intervention may augment the treatment effects seen in Study 1.

The general consensus of the sleep literature, regardless of the assessment approach, has been that young children are prone to sleep disruptions. Sadeh, Raviv, and Gruber (2000) suggested that an appropriate indicator of "poor sleep" in young children

may be waking more than three times per night or a sleep percentage (the ratio of true sleep time and total sleep duration) of less than 90%. In the current sample, over 77% of the children met either of the criteria, and there was little difference among groups in the frequency with which children met the criteria. These numbers are much higher than reported by Sadeh and his colleagues, which may be a function of the older ages of children in their sample or another currently unexplored difference.

Although preliminary, results of Study 2 imply that children with disrupted sleep duration at these early ages may be at increased risk for inattentive and hyperactive problem behavior. This may be an especially problematic outcome during early years because of the negative implications for school readiness. Children who experience shorter nightly sleep durations, may be at risk for greater school failures than those children who obtain adequate amounts of sleep on a consistent basis. A future direction of study may be to further explore potential impacts of sleep disruption in foster care children on their school readiness. This is especially important in foster groups because these children tend to experience less school success than their peers.

Furthermore, Study 2 suggests that some early problem behaviors associated with inattention and hyperactivity may be attributable to sleep problems. Children exhibiting these behaviors may benefit from a sleep evaluation as a first line approach to treatment. Sleep intervention may be a less-costly and more direct means of addressing the inattentive/hyperactive problem behavior than other potential intervention approaches.

Although this study addressed only the initial questions of whether foster children's sleep is different from community comparison children and whether the

MTFC-P program may have any effect on sleep, the next steps will be to address potential mechanisms associated with sleep improvement in foster children. One important direction of study may be to more clearly understand the chronicity and trajectories of sleep problems in foster children and whether they differ from community children. Specifically, the idea of a protective period for sleep in early development is intriguing and deserves further study. It may be important to study sleep behavior in foster children longitudinally in order to understand whether there is a specific time period when sleep in these children deviate from their community peers. Specifically, this will be important to study in school-aged and adolescent children. In these older age groups, sleep is dictated by school schedules, and as children get older, these school schedules dictate earlier rise times. Specifically for adolescent children, this earlier school routine makes it difficult to obtain sufficient sleep and many endorse feeling sleepy much of the time. If foster children continue to show disrupted sleep throughout this time period, they may be at greater risk for increased sleep debt.

Furthermore, potential next steps will be to investigate how sleep may improve in foster children as a function of placement with an MTFC-P care provider. Some possibly associated outcomes might be changes in attachment status and decreases in problem behaviors. EIFC outcomes already indicate increases in secure attachment behaviors and decreases in negative behaviors in the treatment foster children. Better understanding of attachment as a potentially related process will help to clarify the role of perceptions of safety and protection in the acquisition of adequate sleep. Further definition of the relationship between disrupted sleep and inattentive/hyperactive behaviors in foster

children may also open up new doors for behavioral intervention. Although follow-up work is needed, the results of Study 2 implicate that sleep may be an important factor to consider as a driving force for foster children's problem behaviors.

This dissertation was an initial step toward understanding sleep in children in foster care. Overall, there was indication that foster children were more at risk for sleep disruption and negative outcomes following this disruption than community children. However, there was also suggestion that these early patterns could be altered with appropriate intervention. These studies opened new avenues for research that will certainly inform our understanding of the widespread negative outcomes observed in children in the foster care system.

APPENDIX A

BASIC MINI MOTIONLOGGER MODEL ACTIGRAPH

(AMBULATORY MONITORING, INC.)

APPENDIX B

EXAMPLES OF ACTIGRAPH SLEEVES

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