Sleep and Cognitive Functioning: The Moderating Role of Vagal Indices

by

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Abstract

This dissertation contains two studies. In the first study, vagal tone and vagal reactivity (indexed by respiratory sinus arrhythmia; RSA) were examined as possible moderators of the associations between both actigraphy and subjective sleep disruptions and multiple aspects of children's cognitive performance. Children (*M* age = 8.70 years; *N* = 166; 3rd grade) wore actigraphs for seven consecutive nights and completed a subjective measure of sleep via interview. To evaluate cognitive functioning, children completed the Woodcock-Johnson III Tests of Cognitive Abilities (WJ III; Woodcock, McGrew, & Mather, 2001) and a computerized reaction time task (Psych/Lab; Abrams, 2004) in the laboratory. Vagal indices were assessed in the lab; RSA was obtained during a three minute baseline and RSA reactivity (RSA-R) was derived as the difference between RSA during a mild cognitive stressor (i.e., star tracing task) and initial RSA. Results suggest that children who exhibited RSA withdrawal to the laboratory stressor (lower RSA during stressor than baseline), and to a lesser extent those who had higher levels of baseline RSA, were protected from the negative effects of sleep disruptions on various executive functioning tests.

Building on the first study and following the same procedures, the second study further delineated the role of the parasympathetic nervous system by examining children's RSA activity (i.e., baseline levels, reactivity to a lab challenge and/or recovery following the challenge) as potential moderators in the link between children's sleep (self-report and actigraphy-based) and multiple domains of children's cognitive functioning both cross-sectionally (*N* = 132; 80 boys; 5th grade) and longitudinally over two years (from 3rd to 5th grade). Cross-sectional results indicate that actigraphy-based sleep variables (i.e., sleep minutes, sleep efficiency, and long wake

minutes) interact with RSA reactivity (RSA-R) to a problem-solving task to predict children's Decision Speed scores on the WJ III; cross-sectional findings did not yield significant moderation effects for baseline RSA or RSA recovery. Longitudinal findings show that RSA-R, and to a lesser extent baseline RSA, interact with both self-reported and actigraphy-based sleep parameters to predict multiple WJ III test scores two years later. Results build on the scant literature examining individual differences in the sleep-cognitive functioning link and suggest that interactions between physiological and biological systems are important for the prediction of children's cognitive functioning.

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List of Abbreviations

RSA	Respiratory Sinus Arrthymia
ANS	Autonomic Nervous System

Introduction

Sleep disruptions, such as short sleep duration, trouble falling asleep, or prolonged night wakings are common occurrences in Americans' lives, and this issue has been recognized by the medical community and the media. Although this is the case, much of the sleep research has focused on clinical sleep problems (e.g., obstructive sleep apnea) or has been conducted with samples of adolescents or adults. Recent estimates by the National Sleep Foundation (2004) report about 20 - 40% of children have sleep disruptions, but there is a scant amount of information available regarding the role of sleep in typically developing school-aged children's adaptation and maladaptation. The limited research conducted with school-aged children indicates that children get less than the recommended amount of sleep for optimal functioning (National Sleep Foundation, 2004; 2006; 2008). Specifically, the National Sleep Foundation recommends that children in middle childhood get 10-11 hours of sleep. However, most studies report that children are getting one to two hours less than the recommended amount of sleep per night (Buckhalt, El-Sheikh, & Keller, 2007; Sleep in America Poll, 2004). Even though this discrepancy may seem insignificant, recent research conducted with children indicates that even small restrictions in sleep amount (e.g., 1 hour per night), and/or mildly disrupted sleep, can have deleterious effects on children's overall functioning (Sadeh, Gruber, & Raviv, 2003), including reductions in cognitive performance (Buckhalt, El-Sheikh, & Keller, 2007). This, in combination with the high rate of sleep disruptions, suggests that American children may be at an increased risk for adjustment, academic, and cognitive problems. It also brings to the forefront the importance of studying how sleep problems impact school-aged children's outcomes, especially cognitive functioning domains.

Sleep

The definition of sleep has changed drastically over time, with earlier perceptions of sleep encompassing the notion that it is a state of inactiveness for the body and mind. Today, we know that this is not the case; instead, sleep can be defined as an active, reversible state of unresponsiveness and disengagement with the environment (Carskadon & Dement, 2005). In this paper, sleep problems are conceptualized on a continuum and are evidenced by shorter sleep durations, restlessness or high activity during sleep, multiple and extended night waking, and/or highly variable sleep schedules. These kinds of sleep problems may be the most likely forms of sleep disruptions experienced by typically developing children, and addressing sleep using a continuum can contribute substantially to the understanding of developmental outcomes. The terms sleep disruptions and sleep problems will be used interchangeably, however, it is important to note that these terms do not refer to clinical levels of sleep disorders (e.g., sleep apnea) unless noted.

Sleep and Cognition

The importance of sleep in learning and memory is well established in the literature across the lifespan (Sadeh et al., 2003) and in recent years the connection between sleep and memory has gained increased interest from the scientific community. Evidence suggests that memory is improved following sleep (Kopasz, Loessl, Hornyak, Riemann, Nissen, Piosczyk, & Voderholzer, 2010), and that attention, cognition, and memory are significantly altered following episodes of sleep deprivation (Adam, Retey, Khatami, & Landolt, 2006; Drummond, Brown, Gillin, Stricker, Wong, & Buxton, 2000; Durmer & Dinges, 2005; Pilcher & Huffcut, 1996). Recent work incorporating imaging techniques (i.e., fMRI) suggests that one function of sleep is the consolidation of novel material into memory (Gais et al., 2007; Kopasz et al., 2010; Walker & Stickgold, 2006). Thus, studying sleep and the effects it has on cognitive functioning, particularly during the school-years when the brain is still developing and basic building blocks of knowledge are being acquired is of utmost importance.

The role of sleep seems to be particularly important for declarative or explicit memory which is defined as factual memories that are consciously accessible (Kopasz et al., 2010). For example, fMRI sleep studies link the connection between the hippocampus and the prefrontal cortex, especially during slow-wave sleep, to improved learning or memory of novel information (Born, Rasch, & Gais, 2006). However, if sleep deprivation occurs following a learning task, it is hypothesized that poorer memory of the novel material may be a result of a malfunction in the communication between these two brain regions (Born, Rasch, & Gais, 2006). Another possible explanation is that increased disruptions of sleep or shorter sleep amounts may reduce the brain activity that occurs overnight and that is essential for neurocognitive functioning, such as the integration of novel material (Dewald, Meijer, Oort, Kerkhof, & Bogels, 2009). These higher order cognitive functions are heavily influenced by the prefrontal cortex, which is especially sensitive to poor sleep quality and quantity (Curcio, Ferrara, & De Gennaro, 2006; Harrison & Horne, 1998).

It is important to note that the aforementioned imaging research refers to adult samples, and it is unclear if these relations would hold true for children. Specifically, it is known that children and adult's sleep differ in both the duration of sleep as well as the percentage of time spent in each stage. For example, children sleep longer and cycle into slow-wave sleep (SWS) and rapid eye movement (REM) sleep more often than adults (Anders, 1994; Hill, Hogan, & Karmiloff-Smith, 2007; Ohayon, Carskadon, Guilleminault, & Vitiello, 2004). The specific reasons for these differences throughout stages of development are unclear. But, taking into account the large amount of knowledge children are processing, especially at young ages when the basic building blocks of knowledge are forming, it is likely that sleep may play a different or more significant role in the development of short and long term cognitive functioning for children in comparison to adults.

Multiple studies report links between sleep disruptions (e.g., low sleep amount, increased night wakings) and poorer cognitive performance, including difficulties in executive

functioning, working memory and attention (Blunden, Lushington, & Kennedy, 2001; Curcio, Ferrara, & De Gennaro, 2006; Gozel, 1998; Huntley & Lewin, 2006; Sadeh, 2007; Sadeh, Gruber, & Raviv, 2002; Sadeh et al., 2003; ~ 2 – 7% of the variance in cognitive functioning is accounted for by sleep in non-clinical populations). Two primary components of executive functions are sustained attention (i.e., ability to maintain focus on a task for extended periods; Johnsen, Laberg, Eid, & Hugdahl, 2002) and working memory (i.e., continuous updating and prolonged storage of information; Logie, Zucco, & Baddeley, 1990). Executive functions stem from the neural systems of the prefrontal cortex and are indicated as important contributors to behavior regulation (Cole, Usher, & Cargo, 1993; Pennington & Ozonoff, 1996). The prefrontal cortex coordinates executive functions and it is particularly susceptible to sleep disruptions (Curcio, Ferrara, & De Gennaro, 2006; Dahl, 1996; Drummond & Brown, 2001; Harrison, & Horne, 1998).

A meta analysis of research with adults concluded that: (1) cognitive performance was affected more than motor skills when sleep deprivation occurred; (2) as the task lengthened, sleep deprived participants showed marked decreases in performance levels and (3) the range of effects from sleep debt suggests that there are a number of individual differences yet to be explored (Pilcher & Huffcutt, 1996). An experimental study indicated that children (*M* age = 10.6 years) with extended sleep (i.e., an additional one hour of sleep per night for three nights) had improved performance on a memory test and a continuous performance task (baseline to post), whereas sleep-restricted children (i.e., one less hour of sleep per night for three nights) had a significant decline in their performance on a simple reaction time task (Sadeh et al., 2003). A study conducted by Huntley and Lewin (2006) found acute sleep restriction (one night of four hours of sleep) had a significant impact on children's (*N* = 93; *M* age = 7.9, *SD* =1.4 years) neurocognitive functioning. Specifically, more errors of omission, reduced signal detection, and longer response times were recorded during the neurocognitive task following the sleep restriction night in comparison to the baseline measure taken two weeks prior. These results

suggest that sleep impacts children's attention and processing abilities. Another study with 11- to 13- year- olds found prolonged sleep restriction (6.5 hours per night for 6 nights) was related to attention impairments (Fallone, Seifer, Acebo, & Carskadon, 2000). Acute sleep restriction during one night (5 hours sleep) for 10- to 14- year-olds was found to impair higher cognitive functions related to attention (Randazzo, Muehlbach, Schweitzer, & Walsh, 1998).

Overall, there are more than 30 adult and child studies addressing sleep problems (defined and measured in a variety of ways) and cognitive or academic performance (Buckhalt, Wolfson, & El-Sheikh, 2009; Curcio, Ferrara, & De Gennaro, 2006), with the majority, but not all, reporting links between sleep disruptions and poorer cognitive performance. For example, Steenari and colleagues (2003) studied 60 children (6- to 13- years-old) and found that actigraphically derived sleep variables were related to auditory and visual tasks assessed on separate days. Specifically, reduced sleep efficiency and increased sleep latency was related to more incorrect responses on both the visual and auditory tasks, and shorter sleep times were only related to reduction in working memory performance at the highest memory load level. From these findings, the authors speculate that sleep quality more than sleep duration is more closely related to working memory performance.

On the other hand, Meijer and colleagues (2000) sampled 449 Dutch children (9-to 14-years-old) and found no relation between subjective reports of sleep and scores on an attention task. Considering that attention is crucial for successful school performance, and that insufficient, poor, or disorganized sleep has been linked to academic achievement (Epstein, Chillag, & Lavie, 1998; Gozal, 1998; Randazzo et al., 1998; Wolfson & Carskadon, 1998), it is important to delineate specific aspects of both objective and subjective sleep problems related to problems in cognition (Sadeh, 2007), including executive functions, working memory, and processing speed difficulties.

Recent research with children indicates links between insufficient or poor quality sleep and impaired neurobehavioral functions, especially executive functions, such as sustained

attention and working memory (Blunden, Lushington, Lorenzen, Martin, & Kennedy, 2005; Sadeh et al., 2003). These cognitive functions are essential to learning and may be especially important at ages when skills are being acquired (Van Leijenhorst, Crone, & Van der Molen, 2007). Understanding how and why sleep is related to children's cognitive functioning is essential for children's future academic achievement. Specifically, sleep debt or deprivation, created when the optimal and actual amounts of sleep are not congruent (Van Dongen, Rogers & Dinges, 2003), has lasting effects on functioning and adjustment (Carskadon, 2002). Reviews of the literature (Buckhalt, Wolfson, & El-Sheikh, 2009; Curcio, Ferrara, & De Gennaro, 2006; Mitru, Millrood, & Mateika, 2002; Sadeh, 2007; Wolfson & Carskadon, 2003) show reduced amounts of sleep, varying sleep schedules, delayed bedtimes and wake times, and active or fragmented sleep influence many aspects of cognitive performance and functioning, including those incorporated in comprehensive intelligence tests and narrow tests of neurocognitive functioning.

Further evidence of associations between sleep problems and poor cognitive functioning stems from research conducted with clinical samples. Over 50 studies have been reported with sleep disordered children since 2000, and studies have linked sleep-disordered breathing with a variety of cognitive and academic problems in children (Beebe, 2006; Blunden & Beebe, 2006; Blunden, Lushington, Lorenzen, Martin, & Kennedy, 2005; Halbower & Mahone, 2006). Recently, studies have also linked treatments of sleep disordered breathing (i.e., adenotonsillectomy) with improvements in attention and cognition one year following the surgery (Chervin et al., 2006). In addition, children diagnosed with learning problems, such as mental retardation and autism spectrum disorders, have higher incidences of sleep problems than their typically developing counterparts (Richdale, Francis, Gavidia-Payne, & Cotton, 2000; Stores & Wiggs, 2001; Williams, Sears, & Allard, 2004).

Although sleep restrictions and disrupted sleep patterns are linked to deficits in cognitive performance, not all children with restricted or disrupted sleep display these negative outcomes.

Further, in the clinical research realm, investigators have indicated that clinical sleep interventions work well for some individuals but not at all for others. This brings to the forefront the importance of delineating possible individual difference variables that may contribute to these variations.

Parasympathetic Nervous System

Researchers have begun to recognize the importance of individual differences in psychophysiology and how those differences potentially influence child adjustment (Cicchetti & Dawson, 2002), including cognitive performance. One such individual difference that may be an important factor in the association between sleep and cognitive performance is parasympathetic nervous system (PNS) activity. The PNS is the down-regulating branch of the autonomic nervous system (ANS) and it works to maintain homeostasis under normal conditions (Porges, 1991). The term vagal tone is used as an indicator of the influence of the PNS on the heart. Specifically, the vagus nerve is the 10th cranial nerve that provides an inhibitory influence on the heart (i.e., decrease in heart rate). Respiratory sinus arrthymia (RSA), the changes in heart rate associated with the normal cycles of respiration (heart rate decreases during exhalation and increases during inhalation), is used as a marker of vagal tone. The vagus nerve's influence on RSA has been demonstrated through its significant attenuation or elimination by pharmacological blockade (Berntson et al., 1997; Hayano et al., 1991; Sherwood, Allen, Obrist, & Langer, 1986). Within a wide range of normal physiological functioning, RSA is an acceptable measure of vagal tone (Grossman &Taylor, 2007). Baseline RSA, also termed resting RSA, reflects the status of the PNS at rest, the ability to maintain homeostasis under normal circumstances, and perhaps the ability to sustain attentional focus (Porges, 1991). Vagal pathways may reduce (vagal suppression) or increase (vagal augmentation) cardiac output to promote regulatory and goal-directed behavior (Porges, 1995, 2007; Thayer & Lane, 2000), referred to as RSA regulation. Specifically, RSA typically decreases during stressors and engagements that require increases in attention (Porges, 2007). RSA is an appropriate index of

vagal activity for research with normally developing children (Grossman &Taylor, 2007) and can be obtained non-invasively.

The Polyvagal Theory's propositions (Porges, 2007) link the ANS with emotion regulation, cognition, and behavior, and will be used as a guiding theory for the potential role of vagal functioning in the relation between sleep parameters and facets of cognitive performance. Empirical research supporting the theory indicates that higher levels of baseline vagal tone positively correlate with optimal adjustment and health outcomes (Rottenberg, 2007). That is, higher vagal tone maintains a low heart rate (Porges, 1991) under normal conditions, which is optimal for health and adaptation. Stability of vagal tone has been examined at different points in childhood. Specifically, Calkins and Keane (2004) conducted a two-point longitudinal study with preschoolers (age 2 at T1; age 4.5 at T2) and found high stability (r = .57) in baseline vagal tone. Kennedy, Rubin, and Hastings (2004) also reported stability in preschool children's cardiac vagal tone over two years (r = .47). In addition, El-Sheikh (2005) examined stability of vagal tone in middle childhood and reported that baseline vagal tone was a fairly stable (r =. 49) individual difference variable over a two year span.

The reactivity of the PNS in response to a challenge is also an important aspect of PNS activity and is typically referred to as vagal regulation or vagal reactivity (Porges, 2007). When faced with a challenge, the PNS responds in one of two ways: (1) it can be withdrawn or suppressed, frequently referred to as vagal suppression or vagal withdrawal, which leads to an increase in heart rate, or (2) it can be activated or augmented, frequently termed vagal augmentation, which leads to a decrease in heart rate. Vagal suppression is thought to be an optimal response when confronted with a challenge because it allows for the potential to mobilize attentional resources when needed to respond to environmental demands (Huffman et al., 1998). Research indicates that vagal suppression likely facilitates a shift in metabolic resources that support information processing (Bornstein & Suess, 2000). However, it has been noted in the literature that while moderate vagal suppression appears adaptive, vagal

withdrawal that is extreme or prolonged may index over-reactivity (Beauchaine, 2001), which could have implications on the ability to focus attention. Therefore, more clarification is needed to truly understand the process associated with attention and other aspects of cognition, particularly the role of vagal regulation.

Vagal regulation has shown stability across tasks occurring within one session (preschoolers; Calkins & Keane, 2004) and across time points (middle childhood; El-Sheikh, 2005). Vagal regulation in response to cognitive tasks (i.e., star-tracing) has shown some stability (r = -.23) over two years (El-Sheikh, 2005). Although vagal tone and vagal regulation are often moderately correlated, each vagal marker can have differential relations to child outcomes (Calkins, 1997). For example, higher levels of vagal augmentation to a reaction time task have been linked with increases in both objective and subjective sleep problems, whereas baseline RSA with the same sample was correlated only with subjective sleep measures (El-Sheikh & Buckhalt, 2005). In a paper published with the current sample, vagal suppression in response to listening to an argument between two adults interacted with sleep parameters and functioned as a moderator of risk in the link between sleep problems and adjustment problems. Specifically, higher levels of vagal suppression functioned as a protective factor in the sleep-child adjustment link (i.e., externalizing and depression). Conversely, lower levels of vagal suppression or augmentation to the stressor acted as a vulnerability factor in the link between sleep problems and children's adjustment.

Lastly, the Polyvagal Theory postulates that once the challenge has subsided, PNS activity needs to return to baseline levels, and the percentage or recovery time is termed vagal recovery. The theory posits that a faster return to baseline is more optimal (i.e., less wear and tear on the heart), but few studies have addressed this assumption empirically. Adult studies have linked slower vagal recovery times to an increase in cardiovascular disease (Mezzacappa, Kelsey, Katkin, & Sloan, 2001). However, only one study to our knowledge has examined vagal

recovery in children (ages 4 to 7), with results indicating that less vagal recovery predicted reduced attention (Santucci, Silk, Shaw, Gentzler, Fox, & Kovacs, 2008).

Parasympathetic Nervous System and Cognition

RSA indices are recognized as both a correlate and predictor of cognitive functioning (Morgan, Aikins, Steffian, Coric, & Southwick, 2007; Porges, 2007). Specifically, Beauchaine (2001) found those with a greater capacity to suppress vagal tone had more optimal cognitive functioning. Additionally, better performance on working memory and attention tasks have been noted for persons with higher variability in baseline RSA levels (Hansen, Johnsen, Thayer, 2003). A dynamic range of vagal tone (i.e., high resting levels of vagal tone and the ability to suppress to low levels when met with a stressor) is a correlate of better cognitive performance, and it is believed that these persons have a greater ability to appropriately engage and disengage with the environment as needed (Thayer & Lane, 2000), as well as exhibit a more rapid recovery pattern (Porges, 2001).

Of importance to note is that the majority of the literature in this area has been conducted with adults or young children. Low intensity stimulation to the vagus system is shown in adults to be related to increased verbal memory storage (Clark, Naritoku, Smith, Browning, & Jensen, 1999) and improvements in alertness (Rizzo, Beelke, De Carli, Canovaro, Nobili, Robert, et al., 2003). Further, when examinations are conducted regarding focused and divided attention, results show a reduction in PNS activity as attention load increases (Backs & Ryan, 1992). Attention processes are found to be mediated by vagal suppression (Suess, Newlin, & Porges, 1997; Weber, Van der Molen, & Molenaar, 1994).

The limited literature based on infants and children overall support the association between RSA and cognitive functioning. For example, Richards (1985) linked basal vagal tone and vagal suppression with infants' ability to sustain attention during a continuous performance task. This study also found higher levels of baseline RSA to be correlated with more "mature" visual fixation lengths. Similarly, infants that were more distracted during tasks had lower basal

RSA than their more focused counterparts (Richards, 1987). More pronounced vagal suppression is associated with taking less time to orient to a visual stimulus or reach a learning criterion than those infants with less pronounced vagal suppression.

Scant information is available regarding RSA and cognitive performance in typically developing children beyond infancy, and of those that have been conducted the range of cognitive outcomes and/or the sample size has been limited. Preschool aged children (*N*= 42) with more RSA suppression had better on-task behaviors in the classroom as reported by their teachers, which was significantly correlated with better executive functioning (Blair, 2003). A small study (*N* = 32) with fourth and fifth grade children found links between higher baseline RSA and a greater ability to sustain attention on a continuous performance task than children with lower levels of basal RSA (Suess, Porges, & Plude, 1994). Additionally, Mezzacappa and colleagues (1998) conducted a study with 10-year-olds (41 of 42 participants were boys) and results indicated that those children with greater vagal variability showed more competence in executive control. A pilot study conducted by this author (Staton, El-Sheikh, & Buckhalt, 2009) with a small sample (*N* = 41) of children ages 6- to 13-years old (*M* age = 10.06; *SD* = 1.74) addressed links between RSA and RSA regulation and cognitive outcomes derived through the Woodcock-Johnson III Tests of Cognitive Ability (WJ III; Woodcock, McGrew, & Mather, 2001) and a version of the Sternberg memory scanning paradigm (Sternberg, 1966). Results indicated that even after controlling for child age, sex, and pubertal status, RSA was a robust predictor of multiple aspects of cognitive functioning (i.e., accounted for 5 – 11% of the variance in cognitive measures). Specifically, those with higher levels of baseline RSA had higher scores of intellectual ability, processing speed, working memory, and cognitive efficiency. In addition, these children had better reaction time scores than their counterparts with lower levels of basal RSA. On the other hand, RSA reactivity was not found to be a significant predictor of WJ III scores, and only approached statistical significance for Brief Intellectual Ability and reaction time. The reason for the lack of association between RSA reactivity and cognitive functioning

variables was unclear, however it is possible that the challenge task (i.e., reaction time task) did not function as a stressor since there was no consequence based on their performance. Specifically, the mean level of RSA during the baseline and challenge task did not vary significantly, and a large variation of individual differences in RSA reactivity was observed.

Parasympathetic Activity as a Moderator

Sleep problems can disrupt neurobiological regulatory systems, especially those largely governed by the prefrontal cortex (PFC; Archbold, Giordani, Ruzicka, & Chervin, 2004; Dahl, 1996; Horne, 1993; Sadeh et al., 2003), such as executive functions and PNS regulatory functions (El-Sheikh & Buckhalt, 2005, El-Sheikh, Erath, & Keller, 2007a; Irwin, 2008). Although sleep may influence vagal functioning, the neural networks impacted by sleep are not the only influence on vagal functioning. Thus, it is likely that some variability in vagal functioning is independent of sleep. Therefore, vagal functioning may not only act as a mechanism through which sleep influences cognitive functioning, but it may also intensify or weaken the impact of sleep problems on children's cognitive functioning. Higher vagal tone and suppression have been found to function as protective factors against a number of adjustment outcomes, including cognitive impairments, in the context of family stress (El-Sheikh, 2005). Similarly, optimal vagal functioning may also serve as a protective factor in the context of biological stress, such as sleep disruptions. The moderation hypothesis is based on the mutual influence of sleep and vagal functioning on the regulation of attention. Dahl (1996) posits that sleep is affected by not only the levels of arousal and vigilance, but also the ability to regulate arousal and emotions during waking hours. Furthermore, the Polyvagal Theory (Porges, 2007), states that vagal functioning provides one with the ability to efficiently regulate attention to meet with the demands of the environment. Thus, both sleep and vagal functioning assist or obstruct the regulation of attention. Due to the similar influences on attention, more optimal vagal functioning could compensate for the lack of attention due to sleep disruptions, whereas less adaptive vagal functioning could exacerbate the attention problems related to sleep disruptions. In turn,

regulatory capacities have implications for children's cognitive functioning, but this has not been addressed in the context of sleep disruptions. Recent empirical evidence based on the present sample indicated that children with less optimal vagal functioning (i.e., lower baseline vagal tone, less vagal suppression, or vagal augmentation) were at an increased risk for externalizing and internalizing symptoms when their sleep was disrupted (increased wake minutes and reduced sleep efficiency) in comparison to children with more optimal vagal regulation (El-Sheikh et al., 2007a). Of note that in the aforementioned study there were no direct relations apparent between the various sleep parameters and children's vagal tone or reactivity to the lab stressor. The present investigation will build on this literature by examining not only vagal tone and regulation, but also vagal recovery in the context of sleep disruptions and by focusing on the effects of sleep on multiple facets of cognitive functioning.

Sleep Measurement

Information regarding the quality and quantity of sleep can be obtained in two ways: (1) objectively (e.g., polysomnography-PSG; actigraphy) or (2) subjectively (e.g., questionnaires, sleep diaries). Historically, sleep research has been conducted in medical settings in which physicians asked patients questions regarding their sleep quality and quantity. If sleep problems were significant, physicians usually referred patients to a sleep lab, which uses PSG (invasive and time-consuming method) to assess a wide range of clinical sleep problems, such as obstructive sleep apnea. Recently, new technological developments have allowed for the assessment of sleep activity (i.e., movement) via a non-invasive wristwatch (i.e., actigraphy). This small device has been validated against PSG (agreements ranging from 85-90% between the two methods; Sadeh, Sharkey, & Carskadon,1994; Sadeh, Acebo, Seifer, Aytur, & Carskadon,1995), and found to be an acceptable proxy for sleep (Sadeh, 2008). The actigraphy watch is a great research tool for a more in depth study of sleep in the natural setting (Sadeh, et al., 1995).

Subjective reports of sleep of children are typically completed by either the parent or the child, depending on the developmental stage of the child. Although parents tend to be fairly reliable in their reporting of children's sleep schedules (Tikotzky & Sadeh, 2001), they do not always know the number of night wakings, how long it takes the child to go back to sleep after he or she has awoken, or how much sleep activity occurred, leading to discrepancies in sleep quality measures between objective and subjective assessments (Sadeh, Sharkey, & Carskadon, 1994). Children, on the other hand, may be more accurate regarding how many times they get up during the night, but they may not be good at reporting how long it took them to fall asleep or their sleep schedule (e.g., time perception may be more difficult for them). Child and adolescent reports of subjective sleep quality have been found to have low or non-significant relations with objective measures of sleep (Liu & Zhou, 2002; Meijer, Habekothe, & Van Den Wittenboer, 2000), however both types of sleep assessments have been found to be related to child adjustment and health (Dewald et al., 2009; Sadeh, 2008; Sadeh, Gruber, & Raviv, 2002; Wolfson & Carskadon, 2005). This provides evidence for the idea that subjective and objective measures represent two separate domains (Dewald et al., 2009). Although actigraphy provides more precise information (e.g., sleep onset) and is related to a number of adjustment and cognitive outcomes, subjective reports have been found to show stronger associations with behavior and cognitive function (Mitru, Millrood, & Mateika, 2002; Moore & Meltzer, 2008). Thus, subjective reports, especially those related to the amount of sleepiness, could be tapping into an aspect of sleep that objective assessments currently cannot access, such as differences in individual needs of sleep amounts. The inclusion of both objective and subjective sleep measures is therefore essential given their differing links with child outcomes (Sadeh, 2008; Sadeh, Gruber, & Raviv, 2002; Wolfson & Carskadon, 2005). Therefore, in the following studies we will use both subjective child reports and objective actigraphy measures to provide better estimates of sleep parameters than reliance on either measure alone.

Potential Influential Variables

Study Variables and Age. Sleep parameters, including amount (duration) and quality (number of night wakings) change with age (Sadeh, Raviv, & Gruber, 2000). According to the National Sleep Foundation, infants need, on average, a total of 15 hours of sleep a day, but children in middle childhood typically require about 10 hours of sleep, whereas adults require much less (~ 8 hrs). By the time children attend school, they would have spent more time asleep than in any other waking activity (Dahl, 1996). Sleep is an important regulatory process for individuals of all ages, but it seems especially fundamental to brain development at young ages. Research on sleep patterns of typically developing school-aged children is growing but small, and suggests that sleep problems at early ages have a significant impact on developmental outcomes at later ages. For example, sleep difficulties at age 4 predicted behavioral, emotional, and attention problems at age 15 (Gregory & O'Connor, 2002). Additionally, Buckhalt and colleagues (2009) found sleep at age 8 to significantly predict cognitive functioning a year later, controlling for children's initial cognitive functioning.

In addition to age influencing aspects of children's sleep, age also plays a role in levels of understanding and cognitive functioning abilities. Throughout life, but particularly during the younger years of development when the basic building blocks of knowledge (e.g., vocabulary, addition) are being developed, neural plasticity and refinement are constantly occurring in response to challenges presented by the environment (Kopasz et al., 2010).

Furthermore, relations between age and vagal suppression have been reported in the literature. Older school-aged children have been found to show less RSA suppression to laboratory tasks in comparison to their younger counterparts (El-Sheikh, 2005).

Both studies included in this dissertation focused on children of a narrow age range (8 to 9 yrs at T1; 10 to 11 yrs at T2) to minimize potential confounds associated with developmental and sleep requirement differences among participants. Further, I consider this investigation an initial step towards understanding associations among sleep, vagal regulation, and children's

cognitive functioning. Age is used as a control variable in all path models in which age is a correlate of any of the main study variables.

Study Variables and Ethnicity. Ethnic practices impact sleep (Redline et al., 1999), but variations in sleep patterns among ethnic groups are relatively unexplored. For example, the majority of African American (AA) sleep literature has appeared within the last 5 years. In a review of 30 AA adult sleep studies, Durrence and Lichstein (2006) generally concluded that: (1) AAs take longer to fall asleep, (2) nap longer, (3) have higher rates of sleep-disordered breathing (SDB), and (4) have basic differences in their sleep architecture (e.g., depth of sleep). Furthermore, studies have reported that AA adults were more likely than European American (EA) adults to have short sleep duration or sleep less than seven hours per night (Stamatakis, Kaplan, & Roberts, 2007; Krueger & Friedman, 2009).

Sleep and ethnicity research conducted with children is sparse, but suggests that asthma and SDB are more prevalent in AA children than EA children (Redline et al., 1999; Rosen et al., 2003). A recent study (Hale, Berger, LeBourgeois, & Brooks-Gunn, 2009) found that even after controlling for potential SES and family composition confounds, African-American children went to bed later and had less regular bedtimes or bedtime routines than European-American children. A longitudinal study (N = 1,043) conducted with 2- to 8-year-olds examined ethnic differences in parent reported sleep (Crosby, LeBourgeois, & Harsh, 2005). Results indicated that the AA children (26% of the sample) had shorter nighttime sleep, more napping, and more weekend “oversleep” than their EA counterparts, the latter of which is considered a marker of sleep debt (Crosby et al., 2005). Crosby and colleagues (2006) conducted another study with 715 caregiver reports regarding the sleep behaviors of children ages 2- to 12-years old. Results indicated differences in EA and AA children’s sleep patterns. For example, EA children were found to quit napping by age 6, whereas 64% of the AA children continued to nap between the ages of 6 and 12. In addition, EA children began to sleep about 25 minutes longer on the weekends compared to the weekdays around the age that school

attendance began. For AA children, this weekend “oversleep” began at age 3. Further, El-Sheikh and colleagues have reported AA children to have significantly shorter sleep durations, less sleep activity, and more variability in their sleep onset time in comparison to their EA counterparts. For example, AA children slept on average 23 minutes less a day and got up about 17 minutes earlier on the weekdays than EA children (Buckhalt, El-Sheikh, & Keller, 2007). Although I would usually examine ethnicity as a moderating variable because of the aforementioned differences, it was determined that it would be best to control for race in these studies due to issues associated with power.

Study Variables and SES. According to the National Sleep Foundation (www.sleepfoundation.org), sleep hygiene (recommendations for obtaining good sleep), includes practices such as maintaining a cool, comfortable sleep environment and having a consistent sleep-wake schedule. These recommendations may be harder to obtain for those of lower SES than higher SES homes. For example, chaotic living conditions of economically disadvantaged preschool children, such as less structured routines, overcrowding, and family instability, is significantly related to mothers’ reports of their children’s subjective sleep problems (Brown & Low, 2008). Furthermore, smoking is more common in lower SES homes (Barbeau, Krieger, & Soobader, 2004) and children from lower SES homes are at an increased risk of second-hand smoke exposure especially when the size of the home and the ventilation system is accounted. Research indicates that children from low-income families have higher rates of sleep disordered breathing, shorter sleep durations, reduced sleep quality, and more frequent weekend napping in comparison to their more affluent counterparts (Crosby, LeBourgeois, & Harsh, 2005; Redline et al., 1999; Rosen et al., 2003; Stepanski, Zayyad, Nigro, Lopata, & Basner, 1999). Other SES factors that could contribute to a reduction in optimal sleep are: resources (money) or the capability (central heat and air) to adjust home temperature settings, increased home allergens, room sharing, school/work schedules (parents may work swing

shifts; children may have to wake early for the school), low quality mattresses and bedding, as well as the added stressors that tend to aggregate disproportionally in poor populations.

The potential role of SES in children's sleep is important to address, yet few studies have examined these effects (Buckhalt et al., 2007; Buckhalt & Staton, in press). Of those that have, results suggest that children from higher SES homes report fewer sleep-wake problems and slept longer in comparison to those of lower SES homes (El-Sheikh, Buckhalt, Keller, Cummings, & Acebo, 2007b). Cross-sectional examinations conducted with the current sample indicated that when children were in third grade, SES moderated the link between sleep and cognitive performance. Specifically, when sleep patterns and quality were optimal, no differences were found in cognitive functioning of lower and higher SES children. However, when sleep problems were present, children from lower SES homes performed more poorly than their higher SES counterparts (Buckhalt, et al., 2007). When examinations were conducted longitudinally, SES, particularly paternal education, was again found to be a moderator of the sleep-cognitive link (Buckhalt, et al., 2009). However, the extent to which specific ecological risks predict child sleep disruptions is unknown.

Cognitive performance is greatly impacted by poverty and lower SES (Pungello, Kupersmidt, & Burchinal, 1996). A "gap" in cognitive functioning between those of higher and lower SES has consistently been recognized in the literature (Buckhalt et al., 2007; Buckhalt & Staton, in press). Children from lower SES homes are more likely to have poor school performance (Duncan, Brooks-Gunn, & Klebanov, 1994; Sirin, 2005), are referred for special education services more frequently (Blair & Scott, 2002), and are at a greater risk to repeat grades or drop out of school than their more economically advantaged peers (Alexander, Entwisle, & Dauber, 1996; Sirin, 2005), although the role of sleep in the SES-cognitive link has relatively been unexplored (Buckhalt & Staton, in press). One of the few studies examining the role of sleep in the SES-cognitive functioning link was conducted with children (Bates, Viken, Alexander, Beyers, & Stockton, 2002), and findings suggest that variability in sleep schedule is

related to academic performance only for preschoolers (90% EA) from low income homes. Due to the relations of the study variables with SES, this demographic variable was controlled in all analyses.

Study Variables and Sex. Sex differences have been reported in the sleep literature (El-Sheikh, Erath, & Keller, 2007; Sadeh et al., 2000). For example, the 2005 *Sleep in America* poll conducted by the National Sleep Foundation found that women were more likely than men to experience problems falling and staying asleep and to report more daytime sleepiness. In a study with adults conducted by Lindberg and colleagues (1997), women reported longer sleep times than men, but difficulties with maintaining sleep and daytime sleepiness were more common among women. Sleep disordered breathing is more likely to occur in men than women (Redline, Kump, Tishler, Browner, & Ferrette, 1994).

Although few studies have examined gender differences with typically developing children, some have found girls to have longer sleep duration and less active sleep than boys (Gaina, Sekine, Hamanishi, Chen, & Kagamimori, 2005; Sadeh, Raviv, & Gruber, 2000; Ito, Kondo, Kanbayashi, Ohnuma, Kanayama, Aizawa, Kaneko, Shimizu, & Manber, 2006). In the current sample, initial analyses indicated that girls slept longer (*M* = 7:34 hrs:min) than boys (7:05), and had better sleep efficiency (88%) and less activity during their sleep (41) than boys (*M* sleep efficiency = 85%; sleep activity = 50) at T1. During the second wave of data collection, girls were still found to sleep longer (7:32) than boys (7:16) and have less sleep activity (*M* = 37) than boys (*M* = 43).

Sex differences have also been noted in the vagal literature although results have not been consistent across studies. Specifically, some studies have found boys to have higher baseline RSA than girls (El-Sheikh, 2005; Salomon, 2005), whereas others have reported higher baseline levels in girls (Fabes, Eisenberg, Karbon, Troyer, & Switzer, 1994). In addition, others have found no gender differences (Suess et al., 1994). Due to the relations between gender and the study variables, gender was considered a covariate in all path models.

Study Variables and Puberty. A change in circadian rhythm, known as sleep phase delay, has been noted to occur during puberty (Carskadon, Vieira, & Acebo, 1993). This change delays the time that adolescents feel sleepy, which in turn alters their sleep patterns (Wolfson & Carskadon, 1998) and can reduce their sleep duration especially during the school year when wake times are more consistent. Additionally, research has shown that even when teens obtain optimal amounts of sleep, they show an increase in daytime sleepiness (Carskadon, Vieri, & Acebo, 1993).

In addition, pubertal development has been found to be related to RSA suppression, in that more physically mature children have been found to have less RSA suppression to laboratory tasks (El-Sheikh, 2005). To determine if puberty should be controlled in our analyses, we examined correlations between children's pubertal status and all study variables. Results indicated that there were no significant relations between pubertal status and the study variables at T1 or T2; therefore, puberty was not used as a covariate.

Study Variables and Body Mass Index. Sleep problems and body mass index (BMI) have been associated positively both cross-sectionally (Chervin et al., 2003; Gupta, Muller, Chan, & Meininger, 2002; Von Kries, Toschke, Wurmser, Sauerwald, & Koletzko, 2002; Sekine et al., 2002) and longitudinally (Agras, Hammer, McNicholas, & Kraemer, 2004; Reilly et al., 2005; Sugimori et al., 2004). Overweight individuals have more symptoms of sleep disordered breathing, later sleep times, shorter sleep durations, and more fragmented sleep than healthy controls (Beebe et al., 2007). Further, in comparison to lean children, children with weight problems are at a significantly higher risk of having obstructive sleep apnea (Chay et al., 2000; Marcus, 1996; Wing et al., 2003).

Obesity is linked to reductions in heart rate variability, a marker of PNS influence on the heart (Connolly, Kraus, Short & Carno, 2006). Specifically, researchers examined 54 participants (24 normal BMI; 10 at risk for obesity; 20 overweight) with a mean age of 9.2 years (*SD* = 4.1 yrs). Results demonstrated that the degree of obesity was more closely linked to

decreases in heart rate variability associated with sympathetic over-drive than the presence of obstructive sleep apnea syndrome.

Additionally, relations between children's BMI and baseline RSA have been noted in the literature. El-Sheikh and colleagues (2007a) found a significant negative association between BMI and RSA, in that school-aged children with a higher body mass index displayed lower baseline RSA. The links between BMI and the variables of interest in this study indicate a need to control for children's BMI in all analyses.

Study Variables and Season of Sleep Assessment. The change of seasons brings about alterations in the length of daylight during a given 24-hour period. Considering that light patterns impact the release of melatonin, it is important to consider the season of the year when examining children's sleep parameters (Ozaki, Ono, Ito, & Rosenthal, 1995). Season was dichotomized into two; winter and all other seasons. The season of actigraph watch wear was found to be related to the objective sleep variables at T1, however not at T2; therefore, in all regression equations at T1 the season of watch wear was controlled.

The aforementioned child and demographic variables were controlled for or examined as possible controls to increase the rigor in testing the proposed research questions.

Research Objectives

This dissertation contains two studies. In Study 1 (manuscript 1), I will test whether the objective or subjective sleep markers (Sleep Minutes, Long Wake Episodes, Sleep Efficiency, Sleepiness, or Sleep/Wake Problems) interact with either vagal tone or vagal reactivity cross-sectionally to predict one of the following cognitive outcomes: Concept Formation, Auditory Working Memory, Numbers Reversed, and Decision Speed assessed via the Woodcock-Johnson III Tests of Cognitive Abilities (WJIII; Woodcock et al., 2001). The aim of the study is to determine if vagal tone or vagal reactivity is a potential individual difference variable that should be considered in future sleep research and in the development of treatment and

intervention programs aimed at lessening the effects of sleep disruptions on child outcomes, specifically cognitive performance.

Building on the first study, the second study (manuscript 2) addresses the aforementioned research question not only cross-sectionally at T2, but also longitudinally (3rd to 5th grade) while controlling for the autoregressive effects. In addition, I add vagal recovery as a third marker of vagal functioning with a potential to moderate the sleep disruptions-poor cognitive functioning connection cross-sectionally. Of note, this is the first study to examine links between vagal recovery and sleep in children. My aims for the second manuscript are to follow up on significant moderation findings presented in manuscript 1 two years later. This follow up could provide insight into possible critical stages in which the interaction of sleep and vagal functioning may be most influential in the prediction of cognitive functioning. Further, the longitudinal component of the study will allow for clarification of a possible cumulative effect in cognitive functioning associated with the interaction of two biological systems, namely sleep and parasympathetic nervous system functioning.

Based on available evidence, I hypothesize that higher vagal tone, more pronounced vagal suppression to the challenge, and/or faster vagal recovery will act as a protective factor against reduced cognitive functioning in the context of sleep disruptions (Sleep Minutes, Long Wake Episodes, Sleep Efficiency, Sleepiness, or Sleep/Wake Problems). That is, I expect an interaction between sleep and vagal functioning in that children with more optimal vagal functioning indices will be protected from reduced cognitive functioning (i.e., these children will perform better on the WJIII tests) in the context of sleep disruptions. Conversely, children with lower levels of baseline vagal tone, less pronounced vagal suppression, and/or slower recovery from stress are expected to show less optimal cognitive performance scores when sleep disruptions are present. That is, I expect children with less optimal vagal functioning (i.e., low baseline vagal tone, less vagal suppression or vagal augmentation, and/or slower vagal recovery) to have reduced cognitive functioning when sleep disruptions are present than their

more optimally regulating counterparts. These studies are the first of their kind and therefore no hypotheses regarding which sleep indices (quality, quantity, sleepiness, or sleep/wake problems) will interact with which vagal parameter (vagal tone, vagal reactivity, or vagal recovery) most strongly to influence cognitive functioning. However, I do anticipate that the cognitive outcomes that are most tied to vagal functioning (executive functions and working memory) will be the most likely candidates for the moderation hypothesis.

For a more thorough examination of the research question, the objective sleep variables were examined in separate models for each of the cognitive outcomes. The subjective reports of sleep were examined together for each cognitive outcome. These decisions were made due to multi-collinearity of the objective sleep variables and because this is a first step in understanding what types of cognitive functions are impacted by the interaction of sleep and vagal indices. Further, a call in the sleep literature has been made to examine quality, quantity and sleepiness separately because they are considered to represent separate domains of sleep (Dewald et al., 2009). In addition, it was suggested that research compare the effects of subjective and objective assessments within the same investigation to examine possible parameter assessment differences (Sadeh, 2007).

In the two studies presented in this document, reference will be made to vagal functioning when no specificity is required regarding the various facets of vagal activity. However, when more specificity is needed, we use the terms vagal tone, vagal regulation (suppression or augmentation for added precision regarding the direction of vagal regulation), and vagal recovery as pertinent. Vagal tone and regulation, that is higher vagal tone and vagal suppression to tasks, has been established in the literature regarding social (e.g., Eisenberg, Fabes, & Murphy, 1996), emotional (e.g., Calkins, 1997), and behavioral (e.g., Pine et al., 1996) domains. However, vagal recovery literature is scant, especially with children. This will be the first study to our knowledge to examine the role of vagal tone, vagal regulation, and vagal recovery (the latter used only at T2) in the relation between sleep parameters (i.e., quality,

quantity, subjective reports) and several cognitive performance measures (e.g., Concept Formation, Auditory Working Memory, Numbers Reversed, and Decision Speed).

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Abstract Manuscript 1

The role of children's vagal tone and vagal reactivity (indexed by respiratory sinus arrhythmia; RSA) as a possible moderator of the associations between both actigraphy and subjective sleep disruptions and multiple aspects of children's cognitive performance was examined. Children (*N* = 166; *M* age = 8.70 years old) wore actigraphs for seven consecutive nights and completed a subjective measure of sleep via interview. To evaluate cognitive functioning, children completed several tests in the the Woodcock-Johnson III Tests of Cognitive Abilities (WJ III; Woodcock, McGrew, & Mather, 2001) and a computerized reaction time task (Psych/Lab; Abrams, 2004) in the laboratory. Vagal indices were assessed in the lab; RSA was obtained during a three minute baseline and RSA reactivity (RSA-R) was derived as the difference between RSA during a mild cognitive stressor (i.e., star tracing task) and initial RSA. Results suggest that children who exhibited RSA withdrawal to the laboratory stressor (lower RSA during stressor than baseline), and to a lesser extent those who had higher levels of baseline RSA, were protected from the negative effects of sleep disruptions on various executive functioning tests. Findings demonstrate the importance of examining individual difference variables, such as physiological regulation, when considering the effects of sleep disruptions on children's cognitive functioning.

Sleep and cognition: The moderating role of RSA and RSA reactivity

Non-clinical sleep disruptions, such as reductions in sleep quality and duration, have become causes of concern due to their high prevalence (25 to 50%) in typically developing children (Mindell, Owens, & Carskadon, 1999; Stein, Mendelsohn, Obermeyer, Amromin, & Benca, 2001). Recommendations of pediatric specialists (Mindell & Owens, 2009) suggest that children in middle childhood get approximately 10 hours of sleep per night, and recent studies indicate they are obtaining much less than the recommended amount (e.g., Buckhalt, El-Sheikh, & Keller, 2007; National Sleep Foundation, 2004; Spilsbury et al., 2005). Reductions in sleep as small as one hour per night and mild disruptions in sleep quality are linked to poorer cognitive performance in children, including slower reaction times (Buckhalt et al., 2007) and reduced attention (Sadeh, Gruber, & Raviv, 2003). These findings, in conjunction with the high rate of chronic sleep reduction in American children, suggest that many American children may be at risk for cognitive and academic problems associated with sleep problems. Further, although sleep disruptions do not affect all persons in the same way (Buckhalt et al., 2007), little is known about individual difference variables that may act as protective or vulnerability factors in these relations. The goal of this study is to explicate the possible moderating role of individual differences at the physiological level, namely vagal indices (i.e., resting respiratory sinus arrhythmia [RSA] and changes in RSA from baseline to challenge conditions [RSA reactivity]).

This study focuses on relations between non-clinical sleep disruptions, characterized by reduced sleep duration or increased sleep activity and awakenings (indicative of poor quality) and non-clinical problems in children's cognitive performance (e.g., slower reaction time responses, inattention, short term memory lapses), unless otherwise noted. Sleep and cognitive functioning problems are defined on a continuum and in relation to other children within this community sample.

In recent years, researchers have begun to ask whether children's sleep disruptions show similar effects on cognitive functioning as those that have been extensively documented with adults. Now more than 30 studies link sleep parameters of typically developing children and aspects of cognitive functioning and or achievement (Buckhalt, Wolfson, & El-Sheikh, 2009b). Reviews of the science indicate that multiple sleep parameters, including reduced sleep duration, poor sleep quality and daytime sleepiness are related to an array of cognitive functioning outcomes, including difficulties in executive functioning and slower reaction times (Buckhalt et al., 2009b; Curcio, Ferrara, & De Gennaro, 2006; Sadeh, 2007). For example, insufficient or highly disrupted sleep in children is known to impair aspects of executive functioning, such as working memory and the ability to sustain attention (Blunden, Lushington, Lorenzen, Martin, & Kennedy, 2005; Sadeh et al., 2003). Low sleep efficiency (a marker of sleep quality) and longer sleep latency (time it takes to fall asleep) in typically developing elementary school-aged children correlate with higher error rates on both visual and auditory tasks (Steenari, et al., 2003). Furthermore, experimental studies indicate that acute and prolonged sleep restriction in children are related to significant declines in reaction time (Sadeh et al., 2003) and deficits in attention (Fallone, Seifer, Acebo, & Carskadon, 2000).

Although sleep disruptions are associated with poorer cognitive performance, not all children or adults who experience sleep problems are at an equal risk for reduced cognitive outcomes (Buckhalt et al., 2007; Buckhalt, El-Sheikh, Keller, & Kelly, 2009a; Dorrian, Rogers, & Dinges, 2005; Van Dongen, 2006). This heterogeneity in outcomes highlights the importance of moderators of effects in the association between sleep and cognitive performance. For example, Van Dongen (2006) concluded that basal level of alertness, the amount of sleep needed to optimally function, and the timing of the circadian process were all individual differences that may influence the extent of reduced cognitive performance otherwise associated with sleep deprivation in adults. For children, recent studies examined social class (e.g., father education level) and minority status (Buckhalt et al., 2007; Buckhalt et al., 2009a) or

daytime sleepiness (Geiger, Achermann, & Jenni, 2010) as possible individual differences variables that may explain the conditions to which poor sleep is most highly associated with poor cognitive functioning. For example, Buckhalt et al. (2007, 2009a) found ethnic minority children and those from more disadvantaged socioeconomic situations to be more vulnerable than their counterparts to the effects of sleep disruptions on cognitive functioning. Differences in psychophysiology are recognized as important individual differences that can potentially influence child adjustment (Cicchetti & Dawson, 2002). For example, El-Sheikh and colleagues (2007) found that individual differences at the physiological level moderated the effects of disrupted or shortened sleep on children's internalizing and externalizing problems. Yet, physiological indices as moderators of the association between sleep disruptions and cognitive functioning have not been investigated.

Vagal Tone and RSA

The parasympathetic nervous system (PNS) is the down-regulating component of the autonomic nervous system (ANS), which works to maintain homeostasis under normal conditions (Porges, 1991). An indicator of the PNS's influence on the heart is termed vagal tone, which refers to the role of the vagus nerve, the 10th cranial nerve that provides an inhibitory influence on the heart (i.e., decreases in heart rate). Vagal tone in this study is indexed by respiratory sinus arrhythmia (RSA), which represents the rhythmic changes in heart period related to the respiratory cycle (Grossman, Karemaker, & Wieling, 1991). Specifically, heart rate decreases during exhalation and increases during inhalation. RSA is an appropriate index of vagal activity for research with normally developing children (Grossman & Taylor, 2007) and can be obtained non-invasively. The vagus nerve has been demonstrated to influence RSA through its significant attenuation or elimination by pharmacological blockade (Bernston, et al., 1997; Hayano et al., 1991; Sherwood, Allen, Obrist, & Langer, 1986).

The Polyvagal Theory's (Porges, 2007) propositions connect the ANS with emotion regulation, cognition, and behavior. Specifically, the Polyvagal Theory proposes that when a

mammal is confronted with a challenge, it automatically responds by disengaging the vagal brake (i.e., vagal suppression/withdrawal) which inhibits parasympathetic influence (i.e., increased heart rate). This response allows the individual to focus attention on the environment, obtain information, and/or engage social strategies to reduce the challenge/stress. Once the challenge has subsided, the PNS influence can reengage to reduce arousal. The ability to quickly engage and disengage as the environmental conditions become more challenging and to be able respond to metabolic demands, including increased attention and information processing (Suess, Porges, & Plude, 1994), are adaptive processes vital for behavior. We utilize the Polyvagal Theory as a guiding framework for our assessment of vagal functioning as a moderator in the link between disrupted sleep in children and multiple facets of their cognitive functioning.

Baseline vagal tone (i.e., RSA) indexes the activity of the PNS at rest, and is posited to reflect the ability to sustain attention and maintain homeostasis under normal conditions (Porges, 1991). Empirical research supporting the Polyvagal Theory has indicated that higher levels of baseline vagal tone are generally positively associated with more optimal adjustment outcomes (Rottenberg, 2007). That is, higher vagal tone maintains a low heart rate under normal conditions (Porges, 1991), which reduces the wear and tear on the heart and leads to more optimal health and adaptation.

The reduction or increase in heart rate by vagal pathways to promote regulatory and goal-directed behavior is referred to as vagal reactivity (Porges, 1995, 2007; Thayer & Lane, 2000). More specifically, RSA typically decreases during challenges that require focused attention (Porges, 2007). When a challenge is encountered, the PNS can respond in one of two ways: it can be (1) withdrawn or suppressed, frequently referred to as vagal withdrawal, which leads to an increase in heart rate; or (2) activated or augmented, which leads to a decrease in heart rate. Vagal withdrawal is thought to be the most optimal response when confronted with a challenge because it mobilizes attentional resources when the environment demands a

response (Huffman et al., 1998). Vagal withdrawal likely facilitates a shift in metabolic resources that support information processing (Bornstein & Suess, 2000). However, there is a small amount of literature, specifically with infants, indicating that vagal augmentation in response to a surprising stimulus (i.e., jack-in-the-box) leads to more attentiveness than vagal suppression (DiPietro, Porges, Uhly, 1992); very high levels of vagal withdrawal have also been associated with adjustment problems (Beauchaine, 2001). Research on vagal reactivity (i.e., RSA-R) and cognitive functioning in children is scant, and further clarification of this association is warranted. Although vagal tone and vagal reactivity are often moderately correlated, each vagal marker can have differential relations to child outcomes (Calkins, 1997), which supports the assessment of both forms of vagal activity.

Sleep and RSA Functioning

Few investigations have examined links between sleep and RSA functioning in children. Of those that have, vagal augmentation to a laboratory challenge has been shown to be a risk factor, while vagal withdrawal to a stressor functioned as a protective factor. For example, increases in sleep problems are linked to higher levels of vagal augmentation (less optimal response) during a reaction time task (El-Sheikh & Buckhalt, 2005). In a previous paper with the current sample, increased vagal withdrawal to an inter-adult argument served as a protective factor against externalizing and depression symptoms in the context of increased sleep disruptions (El-Sheikh et al., 2007). Conversely, children who responded to the interadult argument with reduced vagal withdrawal or increased vagal augmentation were at greater risk for externalizing and depression symptoms in the context of sleep problems (El-Sheikh et al., 2007).

RSA and Cognition

More optimal vagal functioning (e.g., higher RSA and increased RSA withdrawal) is related to a greater ability to engage and disengage with the environment as needed (Porges, 2001; Thayer & Lane, 2000), which is necessary for optimal cognitive functioning. The majority

of the studies that have examined the associations between vagal functioning and cognitive performance have been conducted with infants or adults; however, a few small studies have addressed this question with school- aged children. Specifically, higher baseline RSA was significantly correlated with greater sustained attention during a continuous performance task in a sample (*N* = 32) of fourth and fifth graders (Suess et al., 1994). Further, 10-year-olds (*N* = 42) with greater vagal variability (i.e., wider range of responses; higher baseline and greater vagal withdrawal) exhibited more competence in executive control than their counterparts with lower vagal variability (Mezzecappa, Kindlon, Saul, & Earls, 1998). Staton, El-Sheikh, and Buckhalt (2009) found that, higher baseline RSA was associated with better Woodcock-Johnson III (WJIII; Woodcock et al., 2001) scores of overall intellectual ability, processing speed, working memory, and cognitive efficiency with a sample of 6- to 13-year olds (*N* = 41; *M* age = 10.06; *SD* = 1.74). However in the latter study, RSA reactivity (derived during a memory task) was not predictive of cognitive functioning as measured with the WJ III (Woodcock et al., 2001) or the memory task (a variation of the Sternberg Memory Task; Sternberg, 1966).

New Directions

In the present study, we advance prior work conducted in the sleep disruptions-cognitive performance literature by examining the moderating role of two physiological individual difference variables: vagal tone (RSA) and vagal reactivity (RSA-R). Sleep problems can disrupt neurobiological regulatory systems, especially those governed largely by the prefrontal cortex (PFC; Archbold, Giordani, Ruzicka, & Chervin, 2004; Dahl, 1996; Horne, 1993; Sadeh et al., 2003), such as executive functions and PNS regulatory functions (El-Sheikh & Buckhalt, 2005; El-Sheikh et al., 2007; Irwin, 2008). Although sleep may influence vagal functioning, the neural networks impacted by sleep are not the only influence on vagal functioning. Therefore, some variability in vagal functioning exists independent of sleep. In addition, baseline RSA and to a lesser extent RSA reactivity (RSA-R) are considered stable individual difference variables for

school-aged children (El-Sheikh, 2005; Salomon, 2005), which supports the examination of RSA indices as moderators of effects.

It is our hypothesis that vagal functioning may strengthen or weaken the influence of sleep problems on children's cognitive functioning (see El-Sheikh et al., 2007). Dahl (1996) posits that sleep is affected not only by the levels of arousal and vigilance at night, but also by the ability to regulate arousal and emotions during waking hours. Furthermore, the Polyvagal Theory (Porges, 2007) states that vagal functioning facilitates efficient regulation of attention in response to environmental demands. Thus, both sleep and vagal functioning can assist or obstruct the regulation of attention.

We hypothesize that both RSA and RSA-R will function as moderators of effects in the association between children's sleep disruptions and their cognitive performance. Based on the extant literature, we expect higher levels of RSA and RSA withdrawal to serve as protective factors in the context of sleep disruptions, as it was shown to do among other areas of adjustment (e.g., externalizing and internalizing problems; El-Sheikh et al., 2007), thereby attenuating the connection between sleep problems and reductions in cognitive functioning. Conversely, and consistent with a conceptualization of protection and vulnerability along a continuum (Brown & Barlow, 2005), low RSA and RSA augmentation are expected to function as vulnerability factors and exacerbate the associations between sleep disruptions and poor cognitive performance. In the context of this emerging literature, no specific hypotheses are advanced regarding the moderating role of vagal indices in relation to specific forms of sleep disruptions (e.g., duration, quality). However, based on literature supportive of vagal indices as markers of attention (Porges, 2007), and the role of the prefrontal cortex in executive functioning (Dahl, 1996), we expect that those tests tapping into higher levels of executive functioning (e.g., working memory) will be the best candidates for the moderation hypothesis. Of note, all of our cognitive outcomes tested in this study require focused attention to perform well, and all of the cognitive tests tap varying levels of executive functioning.

In the present study, we examine both actigraphy and children's self-reported sleep (Sadeh, 2008) and assess two important sleep parameters (duration, quality). Both actigraphy (Buckhalt et al., 2007; Buckhalt et al., 2009a; Sadeh, Raviv, & Gruber, 2000) and subjective (Buckhalt et al., 2007; Buckhalt et al., 2009a; Wolfson & Carskadon, 2003) sleep problems have been linked to reduced cognitive performance and thus their assessment in the context of vagal functioning was deemed important. Similarly, for a more comprehensive explication of children's cognitive functioning in relation to vagal functioning and sleep, we administered an extensive and well-validated battery of cognitive abilities and a well-established reaction time task. The examination of multiple sleep parameters (e.g., actigraphy-based duration and quality, and subjective reports of sleep quality and daytime sleepiness) as they interact with two vagal functioning indices (RSA, RSA-R) is imperative for a more nuanced explication of physiological and biological domains linked with various cognitive performance parameters. The proposed conceptual model is depicted in Figure 1.

Method

Participants

The sample consisted of 166 children (92 girls) and their mothers and fathers from the Southeastern United States. To recruit participants, an agreement with three local school systems was obtained. The school systems provided researchers with names, phone numbers, and addresses of children in the third grade. Researchers then contacted parents via letter and phone. Of the 254 families contacted that were eligible to participate, 66% participated in the study, 28% declined participation, and 6% were interested but said they were too busy at the time. Children were unable to participate in the study if they had a diagnosed chronic illness, mental retardation, attention-deficit/hyperactivity disorder (ADHD), or a diagnosed sleep problem. If children were suffering from an acute illness, such as the flu, their participation was postponed until they were well. If children became sick during the week of actigraphy and took

medications that influence sleep (e.g., Benadryl), the nights they took medications were excluded from actigraphy analyses.

Participants were eight-years-old (*M* = 8.70; *SD* =.30), in the third grade, and from two-parent homes when they were contacted to participate in the study. The majority of children (77%) lived with both biological parents; 21% lived with their biological mother and a step-father; 2% lived with their biological father and a step-mother. Parents of these children had been living together for an average of 10.2 years (*SD* = 5.3). According to parental reports, the ethnic composition of the sample was similar to the community from which they were drawn. Specifically, 69% were European-American (EA) and 31% were African-American (AA). We used oversampling procedures to obtain AA and EA participants across all socio-economic backgrounds. Based on the Hollingshead (1975) criteria, 30% of the families were classified in level 4 or 5 (e.g., professional), 44% in level 3 (e.g., skilled labor), and 26% in level 1 or 2 (e.g., unskilled or semi-skilled laborers). Mothers reported the annual family income at the following percentages: 4% less than $10,000; 7% between $10,000 and $20,000; 25% between $20,000 and $35,000; 28% between $35,000 and $50,000; 25% between $50,000 and $75,000 and 11% more than $75,000. Parental education attainment was as follows for fathers and mothers, respectively: 12% and 8% partial high-school or less; 47% and 36% high school diploma; 30% and 43% partial college or specialized training; 9% and 12% bachelor's degree; and 3% and 2% graduate degree.

Procedures

This study is part of a larger longitudinal study examining the role of the family environment (e.g., marital conflict, parent-child attachment) in relation to children's adjustment, sleep, and health. Only the procedures pertinent to the current study are detailed. The study was approved by the institution's Internal Review Board (IRB), and parents provided consent and children provided assent for participation. Families received monetary compensation for participating in the study.

To reduce confounds associated with variability in sleep schedules, all objective sleep data (actigraphy) were obtained during the regular school year (i.e., not during breaks or summer months). Actigraphs, watch-like devices that measure movement, were used to obtain objective measures of sleep duration and quality. The watches were delivered by researchers to the participants' homes. Parents were instructed to place the actigraph on the child's non-dominant wrist just prior to bedtime and to remove it upon waking. Actigraphy took place over seven consecutive nights, which is considered to be more than acceptable for obtaining reliable measurements of typical sleep patterns (Acebo et al., 1999). During the week of actigraphy assessment researchers called parents daily to obtain information regarding children's sleep and wake times, which was used to validate actigraphic recordings (Acebo & Carskadon, 2001).

Typically, on the day after the actigraph data collection children and their parents came to our research laboratory located on a university campus. Children were administered the brief version of the WJ III. Immediately following the WJIII, children completed a 15-minute reaction time task on the computer. After a short break, children were accompanied into another lab room for a physiological assessment session.

To reduce any anxiety associated with attaching electrodes to obtain RSA data, researchers conversed with the child and a parent (typically the mother) while the electrodes and a pneumatic bellows belt were attached. This process took approximately ten minutes, which allowed a sufficient amount of time for the child to relax prior to the collection of physiological data. The parent and researcher then left the room and the child was given two additional minutes to acclimate to the setting without others present before data collection began (the child was asked to relax while sitting down quietly). Immediately following, a three minute baseline (resting level) was obtained. To accurately assess RSA activity, the duration of the conditions must be sufficient to characterize the signal and three minutes are considered more than adequate for assessment of RSA (Bar-Haim, Marshall, & Fox, 2000). To assess RSA-R, children completed a mildly stressing problem-solving task, namely the star-tracing task

(Matthews, Woodall, & Stoney, 1990). This task requires the child to trace the design of a star on a piece of paper by only seeing the star in a mirror (Lafayette Instrument Company, Lafayette, Indiana, United States). In addition to the difficulty associated with the reverse properties that the mirror adds, children were only given three minutes to complete the task. This task has been used in numerous investigations to evoke ANS reactivity in children (El-Sheikh, et al., 2009; Matthews, Rakaczky, Stoney, & Manuck, 1987; Matthews, Woodall, & Stoney, 1990). Following the physiological session, children were given a break and a snack and were then interviewed by a research assistant regarding their sleep habits.

Measures

Children's Objective Sleep. The actigraph watch used was the Motionlogger Octagonal Basic (Ambulatory Monitoring, Inc., Ardsley, NY). This device is small (36 mm X 38 mm X 13 mm) and lightweight (45 g) and was worn like a wristwatch on the non-dominant arm. Movement during sleep was continuously recorded and raw data obtained from the actigraph were processed with the Octagonal Motionlogger Interface with ACTme Software, and the Analysis Software Package (ActionW2 or AW2). Using the Sadeh algorithm, which has established validity with children (Sadeh, Sharkey, & Carskadon, 1994), one-minute epochs using zero crossing mode were scored as either sleep or wake. Guidelines for determining sleep onset were developed at the E.P. Bradley Sleep Laboratory at Brown University (Acebo & Carskadon, 2001). Good reliability has been established with this actigraph and the Sadeh algorithm (Acebo et al., 1999).

Sleep variables derived via actigraphy included: (a) Sleep Minutes - total minutes scored as sleep during the Sleep Period (time in bed), providing a measure of sleep duration; (b) Sleep Efficiency - percentage of minutes in bed that are scored as sleep, providing a measure of sleep quality; and (c) Long-Wake Episodes- number of wake episodes during the night that last at least 5 minutes, providing a second measurement of sleep quality.

Stability of sleep variables was examined through intraclass correlations. Analyses indicated high levels of stability for sleep duration and sleep quality (.78 for Sleep Minutes, .86 for Sleep Efficiency, .85 Long Wake Episodes) over the week of actigraphy. Thus, the reliability for these measures exceeded recommended minimum levels (.70; Acebo et al., 1999).

The majority of children (75%) had all seven nights of actigraphic data. However, due to a malfunctioning actigraph, forgetting to wear the actigraph, or exclusion of nights during which children took medication, 13% of children had six nights of data, 7% had five nights of data, 2% had four nights of data, 1% had either two or three nights of data, and 2% of children had no actigraphy data. This proportion of valid data for children is comparable to similar studies and is considered very good (Acebo et al., 1999). Sleep parameters were based on averaged data across all available nights of actigraphic assessments.

Children's Subjective Sleep. Researchers interviewed children during the lab session to obtain children's perceptions of their sleep quality and daytime sleepiness. Specifically, children completed the Sleep Habits Survey (SHS; Wolfson & Carskadon, 1998), which inquires about sleep during the past two weeks. This questionnaire is commonly used with children (Acebo & Carskadon, 2002), and has good reliability and validity (Carskadon, Seifer, & Acebo, 1991; El-Sheikh & Buckhalt, 2005; Wolfson et al., 2003). Two subscales were used in analyses: (1) the Sleep/Wake Problems Scale, which is composed of 10 items that assess the frequency of sleep problems (e.g., oversleeping and staying up late at night); and (2) the Sleepiness scale (comprised of 10 items), which measures the extent to which children struggle to stay awake during the day. One item (i.e., sleepiness while driving) on the Sleepiness scale was deleted due to the child's age. Items were rated on a 5-point Likert scale, and sums for each subscale were computed. Higher scores are indicative of more sleepiness and sleep-related problems. In the current sample, internal reliability coefficients were acceptable for both scales: .67 for Sleep/Wake Problems and .70 for Sleepiness.

RSA and RSA-R. To obtain heart rate, electrodes were placed axially on the left and right rib cage, approximately 10 to 15 cm below the armpits. A pneumatic bellows was placed around the chest and held in place using a metal beaded chain. Physiological acquisition equipment and software were from James Long Company. The bioamplifier was set for band-pass filtering with half power cut-off frequencies of 0.1 and 1000 hertz, and the signal was amplified with a gain of 500 for electrocardiography (ECG). The pneumatic bellows, used to assess respiration, was attached to a pressure transducer with a band-pass of direct current to 4000 hertz, to minimize phase or time shifts in the measurement. Data were digitized at a sampling rate of 1000 Hz with a 12-bit analog-to-digital board in a laboratory computer.

An automated algorithm was used to identify the R-waves in the ECG data. In the rare case it was needed, manual correction of missed or misidentified R-waves was conducted using an interactive graphical program. R-wave times were converted to inter-beat intervals (IBIs) and resampled into equal time intervals of 125 ms. Prorated IBI values were used for analysis of the mean and variance of heart period as well as the processing of heart period variability due to RSA. The mean RSA was calculated over the entire 3-min of baseline and the 3-min of the star tracing challenge task. To derive RSA-R in response to the star tracing task, a change score between RSA during the star tracing task and RSA at baseline (i.e., task minus baseline) was computed. Lower scores during the star tracing as compared to the baseline indicate RSA suppression to the challenge task (i.e., negative score for RSA-R), whereas RSA augmentation was indicated by a higher task score as compared to baseline (i.e., positive score for RSA-R). RSA was controlled in all assessments of RSA-R.

The peak-valley method was used to compute RSA; RSA measurement units reported in this paper are in seconds. The peak-valley method is one of several acceptable procedures for quantifying RSA (Berntson et al., 1997) and this procedure highly correlates with spectrally derived measures of RSA (Galles, Miller, Cohn, & Fox, 2002). To identify inspiration and expiration onset times and relative tidal volume, the respiration signal was used. RSA was

calculated as the difference in IBI between inspiration and expiration onset for consecutive respiratory cycles. Specifically, RSA was computed twice per breath, once from inspiration to expiration and again from expiration to inspiration, and then averaged across respiratory cycles to ensure that any underlying trends in heart period had little impact on the computed mean RSA.

Cognitive Functioning. The Woodcock-Johnson III (WJIII; Woodcock et al., 2001) is a well-normed measurement of general intellectual ability (*g*), crystallized intelligence (*Gc*), fluid reasoning (*Gf*), and processing speed (*Gs*). General intellectual ability refers to non-specific abilities that affect the performance of most tasks, whereas *Gc* is thought to reflect skills acquired through knowledge and experience (e.g., verbal ability). Fluid reasoning is the capacity to think logically and solve novel problems, and processing speed involves the ability to automatically and fluently perform tasks. In the current study, the following tests were used: Concept Formation (CF); Numbers Reversed (NR); Auditory Working Memory (AWM); and Decision Speed (DS). CF is a novel controlled-learning task that requires categorical reasoning based on principles of inductive logic. It is a test of fluid reasoning and also measures an aspect of executive functioning, flexibility in shifting mental set. NR is a test of short-term memory span and also engages working memory, as the numbers must be held in working memory while performing a mental operation (reversing the sequence). AWM also measures short-term auditory memory and working memory while also requiring the use of divided attention. A series of numbers mixed with words is presented, and one must recall the series reporting the words in correct order first, then the numbers. DS measures visual processing speed by requiring that two conceptually similar items are selected on multiple trials presenting rows of alternatives. These four tests were chosen to obtain scores reflecting simple processing speed, attention and varying levels of executive functioning, which allowed for a comprehensive assessment of cognitive functioning domains that may be impacted by disruptions in sleep.

Reaction Time. Speed and variability of response speed across a number of trials was measured via a computerized reaction time task based on the classic Donders task adapted by Sternberg (1969) (Psych/Lab; Abrams, 2004). Specifically, the child was seated at a computer and a researcher instructed the child to focus on a "1" in the center of the computer screen. The child placed the right index finger on the "/" key, and the left index finger was placed on the "Z" key. The child was told to follow the instructions presented on the screen, which were also read to the child by the researcher. If the child responded too early, too late, not at all, or if a key was pressed when one was not required, a short tone was sounded.

To assess simple reaction time (Donders Type A), the child was told to press the "/" key as quickly as possible when the "x" appears in the box on the right. For the assessment of choice reaction time (Donders Type C), two boxes were presented and either could have the "x" appear, but children were told to only respond (by pressing "/") when the "x" appears in the right box. The difficulty is increased in this task due to the monitoring of two boxes and having to inhibit a response with the left finger. In a third condition, choice reaction time 2 (Donders Type B), two boxes were presented and the "x" could appear in either box. Children were instructed to press the "/" key with the right finger when the "x" appears in the right box or press the "z" key with the left finger if the "x" appears in the left box. Conditions were presented in a fixed order with the following parameters: 10 practice trials per condition; 20 test trials per condition; a 500 ms inter-trial interval; 100 ms minimum allowed RT; and 1,500 ms maximum allowed RT. Trials were repeated if responses were faster or slower than the criteria. Test trial averages and standard deviations were computed for each condition and were derived for analyses. The mean scores represent the average response time, whereas the standard deviation scores represent vigilance to the task. Examination of children's responses during the three conditions (types A, B, and C) indicated that both the means (*rs* = .66 – .76, *ps* < .001), and *SD*s (*rs* = .37 – .42, *ps* < .001) were significantly correlated. Thus, to reduce the number of analyses, one averaged mean and one *SD* were derived and used in all subsequent analyses.

Body Mass Index. Children's body mass index was calculated using height and weight measurements obtained by a researcher during the laboratory session. A Sunbeam scale was used to obtain the child's weight. A standard formula was used to compute BMI [703 X (weight/ height^2); http://www.cdc.gov]. Based on the Centers for Disease Control criteria, 1% of the sample was classified as underweight, 61% were of normal weight, 23% were overweight, and 15% were considered to be obese.

Demographic Information. Children's gender, ethnicity, age and parental education and occupation were obtained by phone from a parent prior to participation. Parental education and occupation were used to derive a socioeconomic status index via the Hollingshead Index (Hollingshead, 1975).

Season of Assessment. The change of seasons brings about alterations in the length of daylight during a given 24-hour period. Considering that light patterns impact the release of melatonin, it is important to consider the season of the year when examining children's sleep parameters (Ozaki, Ono, Ito, & Rosenthal, 1995). Season was dichotomized into two; winter and all other seasons.

Results

Preliminary Analyses

Table 1 displays means and standard deviations of study variables. Descriptive analyses indicated that on average, children had a fairly low sleep efficiency of 87% (≥ 90% is considered optimal; Sadeh, Raviv, & Gruber, 2000). Similarly, children's average sleep duration was 7.42 hours per night, well below the recommended average for children of this age (Mindell & Owens, 2009). The majority of the children (69%) exhibited RSA withdrawal in response to the star tracing task; 13% showed no change and 18% exhibited RSA augmentation to the task. Of the WJ III tests examined, children in this sample scored the lowest on concept formation and the highest on auditory working memory, but scores on all tests were in the normative range.

Additional analyses explored relations between the main study variables (sleep, vagal indices, cognitive functioning and reaction time) and several possible confounding variables: BMI, child age, gender, ethnicity, socioeconomic status (SES), season of actigraph assessment, and pubertal status. Higher baseline RSA was related to lower BMI. In comparison to younger children, older children showed better cognitive performance on all WJ III tests (*rs* = .16 - .21, *ps* < .05), with the exception of decision speed. Further, in comparison to boys, girls had higher sleep efficiency, fewer long wake episodes, more sleep minutes, and less self-reported sleepiness (*rs* = .15 - .24, *ps* < .05). Boys had significantly lower decision speed scores (*r* = -.17, *p* < .05) and a faster mean reaction time (*r* = -.31, *p* < .01) than girls. African-Americans were more likely to be from lower SES backgrounds (*r* = -.25, *p* < .01), to have less long wake episodes (*r* = - .19, *p* < .05), and to report greater sleepiness (*r* = .16, *p* < .05) and sleep/wake problems (*r* = .25, *p* < .01) than European-Americans. African-Americans also scored lower on the WJ III concept formation (*r* = -.19, *p* < .05) and numbers reversed tests (*r* = -.15, *p* < .05) than their counterparts. The season of actigraph assessment was related to sleep duration (*r* = .17, *p* < .05). Pubertal status was not found to be related to any of the study variables and was therefore not considered in any further analyses.

Table 2 denotes relations among study variables. As expected, actigraphy-based sleep variables were strongly related to each other; so were self-reported measures of sleep. Similarly, cognitive functioning variables (WJ III and reaction time measures) were highly correlated. Neither sleep nor cognitive variables were associated with either RSA or RSA-R.

Analysis Plan

A series of path models were fit using AMOS 17.0 (Arbuckle, 2007). The use of path analysis in AMOS improves the power (i.e., uses the full sample size) by employing maximum likelihood estimation to deal with missing data, unlike multiple regression. Listwise deletion is typically used in multiple regression software such as SPSS and it relies on the assumption that

the data are missing completely at random (MCAR; Little & Rubin, 2002). The assumption regarding missing at random (MAR) is more relaxed in path analysis than in multiple regression.

In each path model, error terms of endogenous variables and significantly related exogenous variables were allowed to correlate. Covariates for each model included child gender, race/ethnicity, age, body mass index, familial socioeconomic status, and season of actigraph assessment (these were the last paths included in each model; Keiley, personal communication). Sleep parameters, RSA, RSA-R or cognitive performance data that exceeded 4 *SDs* (*n* = 4) were considered outliers and were removed from analyses. Of note, sensitivity analyses were conducted and results with and without outlier removal were similar. An example of the path models is presented in Figure 2.

Separate models were fit for each objective sleep parameter (sleep efficiency, long wake episodes, sleep minutes) and each cognitive outcome (concept formation, numbers reversed, auditory working memory, decision speed, Donders *M*, Donders *SD*) resulting in 18 models. Additionally, subjective sleep parameters were examined simultaneously (Sleep/Wake Problems, Sleepiness) for each cognitive outcome; a total of six models. All models contained the two vagal indices (RSA, RSA-R), the main effects and the associated interactions with the sleep parameter(s).

Separate analyses of the actigraphic sleep variables were conducted based on recommendations in the pediatric sleep literature (Berger, Wielgus, Young-McCaughan, Fischer, Farr, & Lee, 2008; DeWald, Meijer, Oort, Kerkhof, & Bogels, 2010) and towards a more nuanced understanding of associations between sleep parameters and child functioning. Further, a call in the sleep literature has been made to examine duration, quality and sleepiness separately because they are considered to represent separate sleep domains (Dewald et al., 2010). In addition, it is recommended that researchers examine multiple sleep parameters including actigraphy-based and self-report measures of sleep (Sadeh, 2011). Thus, due to multicollinearity and power considerations (Babyak, 2004; Whisman & McClelland, 2005), each

cognitive outcome was fit separately. Due to the large number of models fit (*n* = 24) and to decrease the likelihood of Type 1 error, we only report and interpret moderation findings when significant interactions represented at least 10% of the number of interactions examined for that cognitive outcome (e.g., 10% of significant findings across all models associated with Concept Formation). Models were considered an acceptable fit if at least two of the three following criteria were met: χ^2/df < 2, CFI > .95, and RMSEA < .08 (Arbuckle & Wothke, 1999; Browne & Cudeck, 1993). All fitted models reported in this study satisfied these criteria.

As recommended by Aiken and West (1991), we plotted significant interactions at +1 *SD* and -1 *SD* of the predictor and the moderator to understand the influence of the moderator. Of note, the majority of the children exhibited RSA augmentation to the task. Thus, the depiction of the less optimal RSA reactivity group (i.e., Low RSA Withdrawal) includes children who exhibited no change, augmentation, or less vagal withdrawal in comparison to their counterparts. The interaction utility developed by Preacher, Curran, and Bauer (2006) was used to plot the interactions; estimates were obtained from the fitted models. Significant interactions signify a statistically significant difference between the plotted lines (e.g., the association between sleep and cognitive functioning at high levels of the moderator is significantly different from the link at low levels of the moderator). We also examined whether each slope representing high and low levels of the moderator was significantly different from zero; significant slopes are indicated in the figures.

Examination of RSA Indices as Moderators of Associations between Children's Sleep and Concept Formation Scores

To examine whether RSA or RSA-R to the cognitive laboratory task moderated the paths among children's sleep parameters (actigraphy-based and self-reported) and their scores on the WJ III concept formation test, interaction terms were added to the path models after the inclusion of the main effects. Three of the four models tested (75% of possible relations)

indicated a significant moderating role of RSA-R in the relation between sleep problems and performance on the concept formation test; no moderation effects were found for RSA.

The path between sleep efficiency and concept formation was moderated by RSA-R (β = .20, $p < .05$) and explained 3% of unique variance in concept formation ($\chi^2 = 45.45$, $p = .29$, $\chi^2/df = 1.10$, CFI = .96, RMSEA = .03). The full model accounted for 16% of the variance in concept formation. As depicted in Figure 3A, in the context of low sleep efficiency, children who exhibited RSA withdrawal to the star tracing task showed better performance on the concept formation test than those who exhibited RSA augmentation to the lab challenge. Furthermore, for children who exhibited RSA withdrawal, sleep efficiency was negatively associated with concept formation and the slope representing this association was significantly different from zero. Children who exhibited RSA augmentation had lower concept formation scores regardless of their sleep efficiency.

Similar to the pattern of effects observed for sleep efficiency, RSA-R moderated the association between sleep minutes and concept formation (β = .14, p = .09) and explained 2% of the unique variance in concept formation ($\chi^2 = 40.28$ p = .41, $\chi^2/df = 1.03$, CFI = .99, RMSEA = .01). The full model accounted for 15% of the variance in concept formation. Specifically, in the context of reduced sleep minutes, children who exhibited RSA withdrawal performed better on the concept formation test than those who exhibited RSA augmentation (Fig. 3B). Furthermore, a significant slope indicated a negative association between sleep minutes and concept formation scores for children who exhibited RSA withdrawal. Conversely, children who showed RSA augmentation had similar scores on concept formation regardless of the amount of sleep minutes they obtained.

RSA-R also played a significant moderating role in the link between long wake episodes and concept formation scores (β = .24, $p < .01$) and explained 2% of the unique variance in concept formation ($\chi^2 = 68.75$, $p < .01$, $\chi^2/df = 1.56$, CFI = .74, RMSEA = .06). The full model accounted for 15% of the variance in concept formation. As shown in Figure 3C, in the context

of increased long wake episodes, children who exhibited RSA withdrawal to the laboratory task performed better than their counterparts who exhibited RSA augmentation. Further, a significant positive association between long wake episodes and concept formation was observed for children who showed RSA withdrawal task. However, for those who exhibited RSA augmentation, cognitive performance was similar regardless of sleep problems.

Neither RSA nor RSA-R moderated the link between subjective measures of children's sleep (sleepiness; sleep/wake problems) and concept formation.

Examination of RSA Indices as Moderators of Associations between Children's Sleep and Numbers Reversed Scores

Analyses of children's performance on the WJ III numbers reversed test indicated that RSA was a significant moderator in one of the four models examined (25% of possible relations), and RSA-R was a significant moderator in one of the four models (25% of the possible relations).

Neither RSA nor RSA-R moderated the paths between actigraphy-based sleep parameters (i.e., sleep efficiency, sleep minutes, or long wake episodes) and numbers reversed.

RSA moderated the pathway between sleepiness and numbers reversed (β = -.17, p < .05) and explained 2% of the unique variance in numbers reversed (χ^2 = 112.63, p < .01, χ^2/df = 1.56, CFI = .81, RMSEA = .06). A total of 18% of the variance in numbers reversed was accounted for by the full model. Specifically, children who showed low RSA had average and stable performance regardless of their sleepiness (Figure 4A). However, children who exhibited high RSA and experienced low sleepiness, performed much better than all other children.

RSA-R also moderated the association between sleepiness and numbers reversed (β = -.23, p < .05) and explained 3% of the unique variance (χ^2 = 112.63, p < .01, χ^2/df = 1.56, CFI = .81, RMSEA = .06). The full model accounted for 18% of the variance in numbers reversed. As shown in Figure 4B, children who exhibited RSA withdrawal performed similarly regardless of level of sleepiness. The pattern of effects was only partially consistent with expectations: at high

levels of sleepiness, those exhibiting RSA withdrawal performed better than children who exhibited augmentation; yet, the pattern was reversed at lower levels of sleepiness.

RSA did not moderate the link between sleep/wake problems and numbers reversed, but RSA-R did (β = .24, p < .01) and explained 4% of the unique variance (χ^2 = 112.63, p < .01, χ^2/df = 1.56, CFI = .81, RMSEA = .06) in numbers reversed. A total of 18% variance in numbers reversed was accounted for by the full model. Again, this relation was not consistent with expectations. Specifically, children who showed RSA withdrawal performed significantly worse in the context of high sleep/wake problems than in the context of low sleep/wake problems. Further, there was no significant difference in children's performance on the numbers reversed test for those children who exhibited RSA augmentation at either high or low levels of sleep/wake problems.

Examination of RSA Indices as Moderators of Associations between Children's Sleep and Auditory Working Memory Scores

Analyses of children's performance on the WJ III auditory working memory test indicated that RSA was a significant moderator in one of the four models examined (25% of possible relations), and RSA-R was a significant moderator in two of the four models (50% of the possible relations).

RSA-R moderated the link between sleep efficiency and auditory working memory (β = .15, p < .09), which explained 1% of unique variance (χ^2= 45.22, p < .30, χ^2/df = 1.10, CFI = .96, RMSEA = .03) and 11% of the variance in auditory working memory was accounted for by the full model. As depicted in Figure 5A, when sleep efficiency was low, children who exhibited RSA withdrawal performed better than their counterparts. On the other hand, those children who exhibited RSA augmentation performed better in the context of high sleep efficiency than children who showed RSA withdrawal.

RSA moderated the link between sleep minutes and auditory working memory (β = - .17, p = .05) and explained 2% of the unique variance in auditory working memory (χ^2 = 40.26,

$p < .42$, χ^2/df = 1.03, CFI = .99, RMSEA = .01). The full model accounted for 12% of the variance in auditory working memory. In the context of short sleep, children with higher levels of RSA performed better on the WJ III auditory working memory test than those who exhibited lower baseline RSA (Figure 5B). Those who exhibited high RSA did not significantly differ on the auditory working memory test regardless of their sleep duration. However, for those who with lower RSA, sleep minutes was negatively associated with auditory working memory, with the worst performance in the context of shortened sleep. No moderation effects were found for RSA-R in the association between sleep minutes and auditory working memory.

RSA did not moderate the link between long wake episodes and auditory working memory, however RSA-R was a moderator of this pathway (β = -.15, p = .06; 1% of the unique variance explained; χ^2 = 68.77, p = .01, χ^2/df = 1.56, CFI = .73, RMSEA = .06; the full model accounted for 12% of the variance in auditory working memory). As shown in Figure 5C, when sleep was poor, children who exhibited RSA withdrawal performed better than their counterparts who showed RSA augmentation; the opposite pattern was observed when sleep was more optimal.

Neither RSA nor RSA-R moderated the link between subjective measures of children's sleep and auditory working memory.

Examination of RSA Indices as Moderators of Associations between Children's Sleep and Decision Speed Scores

Examination of children's scores on the WJ III decision speed test yielded two significant moderation effects for RSA-R (50% of possible relations); no moderation effects were found for RSA.

Neither RSA nor RSA-R moderated associations between sleep efficiency or sleep minutes and decision speed.

RSA-R moderated the link between long wake episodes and decision speed (β = -.15, p < .05), which explained 4% of unique variance (χ^2 = 68.71, p = .01, χ^2/df = 1.56, CFI = .74,

RMSEA = .06) and 17% of the variance in decision speed was accounted for by the full model. When sleep was optimal (fewer long wake episodes) all children had similar predicted means on decision speed (Figure 6A). However, when sleep was less optimal, children who exhibited RSA withdrawal performed much better than children who expressed RSA augmentation.

RSA-R also acted as a moderator in the pathway between sleepiness and decision speed (β = - .28, p < .01) and explained 4% of unique variance in decision speed (χ^2 = 111.83, p < .01, χ^2/df = 1.55, CFI = .82, RMSEA = .06). The full model accounted for 19% of the variance in decision speed. Figure 6B depicts that when sleepiness is high, children who exhibited RSA withdrawal performed much better on the WJ III decision speed test than those who showed RSA augmentation. Further, for children exhibiting RSA augmentation, a negative association was observed between sleepiness and cognitive performance; however, this pattern was reversed for those exhibiting RSA withdrawal.

No moderation effects were found for the link between sleep/wake problems and decision speed.

Examination of RSA Indices as Moderators of Associations between Children's Sleep and Reaction Time Means and SDs

Analyses revealed that neither RSA nor RSA-R moderated the link between children's sleep and their performance on the reaction time task (*M* or *SD* scores).

Discussion

We examined whether vagal tone and vagal reactivity, as indexed by RSA, moderated associations between disruptions in children's sleep and varying domains of cognitive performance in a large sample of healthy elementary school-age children. Results are among the first to indicate that vagal functioning interacts with sleep disruptions (objective and subjective) to statistically predict children's cognitive performance. Specifically, in the context of poor sleep, children with more optimal vagal functioning characterized by either higher RSA or more pronounced RSA withdrawal to lab challenges tended to perform better on several

cognitive functioning measures (i.e., WJIII) than children who exhibited lower RSA or less optimal RSA reactivity. Although the amount of variance accounted for by the interaction term was small, the consistency of the findings strengthens confidence in the results. Further, the percent of variation explained is similar to studies that have investigated interactions between sleep and vagal activity in the prediction of child adjustment (e.g., El-Sheikh, Erath, & Keller, 2007). Findings highlight the importance of individual differences in physiological functioning and the aggregation of risk across physiological and biological domains.

The impact sleep problems have on the prefrontal cortex's ability to exercise executive control over behaviors and thought was first suggested by Horne (1988, 1993) and based on observations in the lab (i.e., novel language and creativity were significantly impaired by sleep loss). Dahl (1996) extended this model by proposing that sleep loss directly affects cognitive tasks that require efficient working memory and executive functioning, and/or the ability to synchronize attention and arousal to perform abstract or complex goals. Here we found that the direct relations between either the objective or subjective sleep parameters and cognitive performance were not robust. However, some significant associations in the expected directions were observed: sleep minutes and long wake episodes were related to decision speed, sleepiness was associated with auditory working memory, and sleep-wake problems were related to numbers reversed, auditory working memory and a higher variability in reaction time. Further, using the same sample, Buckhalt and colleagues (2007) reported similar associations between sleep problems and cognitive performance albeit with different cognitive measures. The direct relations observed in the current study, as well as those reported by Buckhalt and colleagues, provide some support for Dahl's (1996) hypothesis that facets of executive functioning such as working memory are directly impacted by sleep disruptions.

A major contribution of the present investigation is the delineation of RSA and RSA-R as moderators in the link between sleep problems and cognitive performance. While the pattern of effect varied based on the sleep parameter, the vagal activity index, and the cognitive

performance domain, one general conclusion can be made: in the context of poor sleep, children with more optimal vagal functioning (higher RSA or more pronounced RSA withdrawal) tended to perform better than their counterparts with less optimal vagal functioning. For example, in the context of reduced sleep duration or increased reports of daytime sleepiness children with lower RSA were at a greater risk for poor cognitive performance especially on tasks requiring working memory (i.e., auditory working memory, numbers reversed). This pattern of effects illustrates aggregation of risk in that children who have poor sleep in conjunction with lower levels of RSA are the ones most at risk for poorer cognitive performance. Overall, results are consistent with those of El-Sheikh and colleagues (2007) conducted with the present sample in that RSA interacted with multiple sleep parameters to predict children's adjustment and body mass index; children at most risk were those who had poor sleep and lower levels of RSA.

Similarly, children with less optimal RSA-R (i.e., augmentation) were particularly at risk for poorer cognitive performance in the context of sleep disruptions. Findings pertaining to reactivity were much more robust than those found with RSA. Specifically, children who had higher levels of sleep problems (actigraphy-based duration and quality as well as subjective reports of sleep/wake problems and sleepiness), and exhibited RSA augmentation were at increased risk for cognitive problems in comparison to their more physiologically regulated counterparts (i.e., RSA withdrawal). However, neither RSA nor RSA-R interacted with any of the sleep variables to predict children’s reaction time or reaction time variability. This finding was unexpected given that vagal activity is considered to be a marker of attention (Porges, 2007), which is essential to perform optimally on the reaction time task; replication is necessary and caution should be exercised not to accept the null hypotheses. Overall, results are novel and suggest an interaction between biological (sleep) and physiological (PNS activity) systems in the prediction of children’s ability to store novel information while performing a task (i.e.,

numbers reversed, auditory working memory) and to apply previously learned rules about grouping a class of objects or ideas (i.e., concept formation, decision speed).

Children with poor vagal functioning (i.e., low RSA and/or RSA augmentation) may have trouble obtaining good sufficient sleep (Dahl, 1996; El-Sheikh et al., 2007). Of note, in the current sample there were no direct relations between any of the sleep variables and RSA or RSA-R. Lack of significant associations contradicts findings reported by El-Sheikh and Buckhalt (2005) in which multiple objectively and subjectively derived sleep parameters were associated with children's RSA-R to a continuous performance task. It is not evident why discrepant results were observed and sample characteristics (i.e., children in El-Sheikh and Buckhalt 's study were older) or study design (i.e., RSA-R was derived during task performance in El-Sheikh and Buckhalt, whereas RSA and cognitive performance were examined during different time periods in the present study) may have contributed to the inconsistencies. Further, it is not unusual that vagal indices have no direct relations with study outcome variables (e.g., externalizing and internalizing behaviors), but rather act as moderators of relations (e.g., El-Sheikh et al., 2007; El-Sheikh & Whitson, 2006; Leary & Katz, 2004). In addition, the lack of association among sleep and vagal indices may be due to the normative sample used in this study. It is possible that samples of children with clinically diagnosed problems in sleep or vagal functioning may show direct links between the two biophysiological systems. Examinations of associations between vagal indices during non-nocturnal conditions and sleep parameters are scarce. Thus, further testing of these relations among diverse populations of children is likely to delineate the conditions under which RSA and sleep problems are related.

The protective function of higher RSA and increased RSA withdrawal to stressors are consistent with the Polyvagal Theory (Porges, 1998), which proposes that vagal functioning influences the regulation of arousal, affect and attention (Porges, 2007). An adaptive physiological response when confronted with stress (e.g., sleep disruptions), is considered to be a disengagement of the vagal brake (i.e., vagal withdrawal), which inhibits PNS influence (i.e.,

increased heart rate). This response allows the individual to focus attention on the environment, obtain information, and/or reduce the challenge by using social strategies to regulate emotions. Once the challenge has subsided, the PNS influence can reengage to reduce arousal. The ability to quickly engage and disengage as the environmental conditions become more challenging and to be able respond to metabolic demands, including increased attention and information processing (Suess et al., 1994), are adaptive processes vital for behavior.

Several of the moderation effects were difficult to comprehend especially those pertaining to either subjective sleep measures or the pattern of effects observed in the context of more optimal sleep. For example based on the large literature regarding RSA withdrawal, one would expect it children who exhibited this pattern of reactivity to a stressor would obtain more optimal outcomes than those who showed augmentation regardless of the context. However, in five of the ten significant moderation findings children who exhibited higher levels of vagal withdrawal performed significantly better on several cognitive assessments when sleep was poor than when it was more optimal. While the meaning of these inconsistent effects is not apparent, we conducted post-hoc analyses to examine some possible three-way interaction effects. Child gender and ethnicity were examined through three-way interactions involving sleep, vagal functioning, and cognitive performance; no significant interactions emerged. The meaning of these unexpected findings remains unclear and warrants further investigation.

Although the results suggest that more optimal PNS activity may play an important role in the link between sleep disruptions and children's cognitive performance, the PNS is one branch of the ANS and the second branch, namely the sympathetic nervous system (SNS) is an important physiological system associated with sleep (El-Sheikh & Arsiwalla, 2011). Furthermore, PNS and SNS activity can have additive and interactive effects in the prediction of child functioning (e.g., El-Sheikh et al., 2009). Thus, future research would benefit from examining the potential influence of both branches of the ANS in relation to sleep and cognitive functioning. Similarly, vagal recovery (the change in RSA once a challenge has subsided) is a

third component of vagal activity that was not measured in this study. According to the Polyvagal Theory (Porges, 2007), once the stressful event has ended, it is pertinent for heart rate to return to normal levels to reduce wear and tear on the heart, which would correspond with a return of RSA to baseline or pre-stress levels. The inclusion of this third index of PNS functioning (i.e., recovery) may also assist in further explication of the role of the PNS in the sleep-cognitive functioning link.

The reliance on cross-sectional data did not allow us to determine the causality or examine the possible meditational role of vagal functioning in the link between sleep problems and cognitive performance; note however, that sleep was not directly associated with vagal activity limiting mediation or intervening variable pathways in this cross-sectional study. Results should be interpreted with caution and considered as a first step in explicating how sleep is differentially associated with cognitive performance based on an individual's physiological reactivity. Additionally, these results must be interpreted in relation to the developmental age of the children. It may be that the role of vagal functioning in the link between sleep disruptions and cognitive functioning may be more or less pronounced for individuals at different stages of development (e.g., infancy, adolescence). This sample contained only children from the third grade, which allowed for a more focused examination; however, the limited age range reduces the generalizability of the findings to a more diverse population. Further, the normative level of sleep and physiological functioning in this community sample, and exclusion/inclusion criteria (e.g., two-parent home, lack of children with mental retardation, developmental delays, ADHD, chronic illnesses) does not allow for understanding examined questions in the context of more severe or clinically significant problems.

Although this study used both actigraphy and subjective measures of children's sleep, it is important to note that daytime naps were not taken into consideration. Sleeping during the day may compensate for some of the problems associated with reduced sleep duration and we recommend that future studies incorporate napping into the total daily sleep duration to further

delineate the relations between sleep and cognition. Although the use of actigraphy and subjective sleep measures constitute an advance in this field of inquiry, and are more likely to explicate relations between sleep problems and cognitive outcomes (Sadeh, 2008), these assessments do not have the ability to assess sleep staging. An important next step for researchers is to investigate sleep staging through polysomnography (PSG) and its association with both cognitive outcomes and physiological reactivity. The amount of time spent in different stages of sleep may play a role in the type of cognitive functioning that is impaired by disruptions in the duration and quality of children's sleep. For example, research has shown that slow-wave sleep (stages 3 and 4) is implicated in declarative memory consolidation, and rapid eye movement sleep (REM) is implicated in procedural memory consolidation (Hornung, Danker-Hopfe, Regen, Schredl, & Heuser, 2007). Thus, the use of PSG in future studies is warranted.

In summary, support for interactions between biological (e.g., sleep) and physiological (e.g., RSA) regulatory systems in predicting children's cognitive performance abilities was found with a large sample of typically developing third grade children. Results extend work in the area of sleep and cognitive outcomes of typically developing school-aged children by (1) assessing possible individual differences variables; (2) incorporating both actigraphy and self-reports as a measure of sleep; and (3) examining multiple aspects of executive functioning using standardized tests. The multifaceted nature of this study lends strength to the moderation effects. Results suggest that poor physiological regulation (i.e., low baseline RSA or RSA augmentation) may undermine children's cognitive performance, namely executive functions, in the context of normative levels of sleep problems.

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Table 1. Means, Standard Deviations and Range of Study Variables.

	Mean	*SD*	*Range*
Child Age-years	8.72	.30	8.08 - 9.83
Family Socioeconomic Status	35.95	9.35	17 - 63
Body Mass Index	20.34	4.63	12.87 - 41.70
Sleep Efficiency-actigraphy	86.78	7.94	48.70 - 97.86
Long Wake Episodes-actigraphy	3.92	2.20	.43 - 10.57
Sleep Minutes-actigraphy	445.10	54.78	254.00 - 539.71
Sleepiness-child report	14.11	4.50	0 - 27
Sleep/WakeProblems-child report	17.04	4.95	10 - 34
RSA	.13	.06	.02 - .36
RSA-R	-.02	.05	-.16 - .31
Concept Formation-WJ III	99.32	12.67	54 - 142
Numbers Reversed- WJ III	104.03	15.92	52 - 150
Auditory Working Memory- WJ III	112.01	13.08	75 - 151
Decision Speed- WJ III	107.65	13.12	76 - 145
Reaction Time *M*-Donders (ms)	464.74	75.44	327.04 - 754.30
Reaction Time *SD*-Donders (ms)	140.11	37.16	66.72 - 227.39

Note: *N* ranges from 159 to 165 due to missing data. WJIII = Woodcock Johnson III; *M* = mean; *SD* = standard deviation; ms = milliseconds.

Table 2. Correlations among study variables.

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.
1. Child Age	-										
2. Child Gender; 1 = Boy; 0 = Girls	-.02	-									
3. Ethnicity; 1 = AA, 0 = EA	-.10	-.08	-								
4. Family Socioeconomic Status	.18*	.05	- .24**	-							
5. Body Mass Index	-.11	-.09	.07	-.13	-						
6. Season of actigraphy	.36**	-.06	.05	.20**	.04	-					
7. Sleep Efficiency-actigraphy	.17*	-.20	.09	.10	-.18*	.15*	-				
8. Long Wake Episodes-actigraphy	-.19*	.18*	-.19*	-.04	.11	.07	-.85**	-			
9. Sleep Minutes-actigraphy	.18*	-.24**	-.09	.20**	-.21**	-.17*	.83**	-.61**	-		
10. Sleepiness-child report	-.10	.15*	.16*	-.10	-.01	-.07	-.07	.01	-.08	-	
11. Sleep/Wake Problems-child report	-.09	-.06	.25**	-.16*	.06	-.02	-.07	.02	-.12	.43**	-
12. RSA	-.08	.07	.05	-.03	-.24**	-.02	.03	-.05	.09	.04	.10
13. RSA-R	.02	.05	.04	-.02	.04	-.05	-.04	-.02	-.08	-.03	.02
14. Concept Formation-WJ III	.16	-.03	-.19*	.25**	-.03	.19*	-.04	.04	.00	-.05	-.08
15. Numbers Reversed-WJ III	.20**	.01	-.15*	.15*	-.07	.11	.02	-.07	.03	-.13	-.16*
16. Auditory Working Memory-WJ III	-.26**	.06	-.11	.23**	-.10	.05	.10	-.12	.11	-.16*	-.18*
17. Decision Speed- WJ III	.12	-.17*	-.01	.20**	-.05	.03	.13	-.15*	.16*	-.03	.02
18. Reaction Time *M*-Donders	-.24**	-.31**	.07	-.11	.13	-.04	.01	-.04	.04	-.01	.12
19. Reaction Time *SD*-Donders	-.24**	-.10	.08	-.13	.08	-.05	-.04	-.02	-.04	.05	.17*

Note: Season of watch wear, 1= winter, 0= fall, spring; RSA = respiratory sinus arrhythmia; RSA-R = respiratory sinus arrhythmia reactivity; WJ III = Woodcock Johnson III.
*p < .05; **p < .01; ***p < .001.

Table 2 continued.

	12.	13.	14.	15.	16.	17.	18.	19.
13. RSA-R	-.13	-						
14. Concept Formation-WJ III	-.02	-.01	-					
15. Numbers Reversed-WJ III	.07	-.03	.37**	-				
16. Auditory Working Memory-WJ III	-.08	.06	.33**	.38**	-			
17. Decision Speed- WJ III	-.11	-.06	.24**	.20**	.21**	-		
18. Reaction Time *M*-Donders	.01	.01	-.21**	-.24**	-.24**	-.23**	-	
19. Reaction Time *SD*-Donders	.05	.09	-.28**	-.28**	-.20*	-.20*	.72**	-

Note: Season of watch wear, 1= winter, 0= fall, spring; RSA = respiratory sinus arrhythmia; RSA-R = respiratory sinus arrhythmia reactivity; WJ III = Woodcock Johnson III. *$p < .05$; **$p < .01$; ***$p < .001$.

Figure 1.

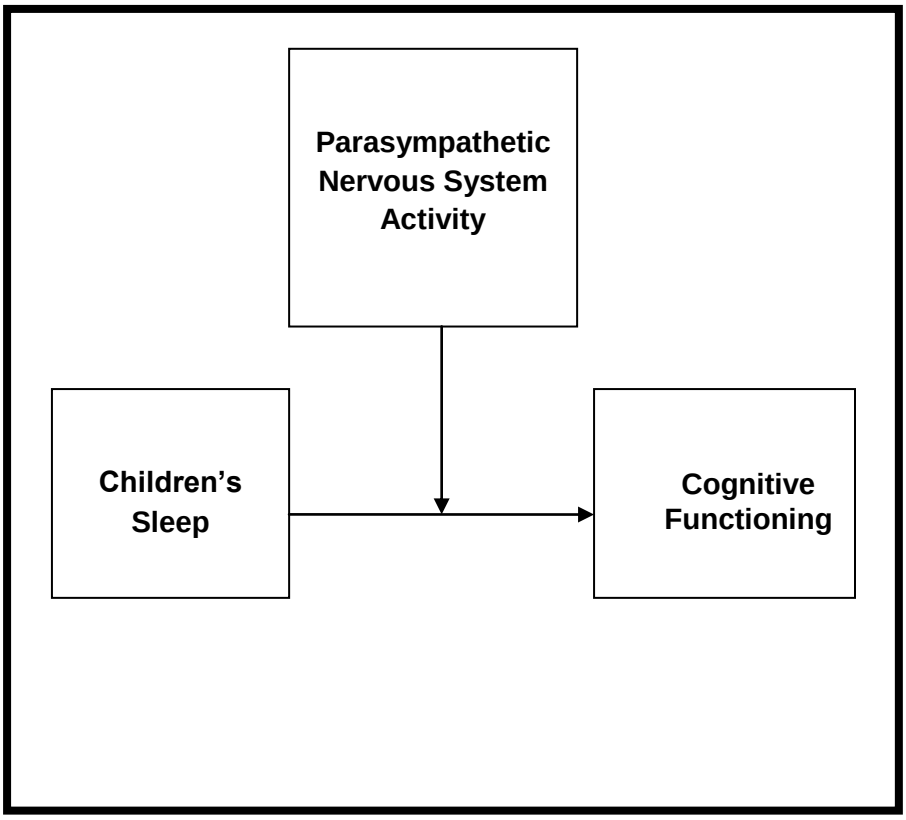

Figure 1. Conceptual model depicting children's parasympathetic nervous system activity as a moderator of the association between children's sleep and their cognitive functioning.

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Figure 2.

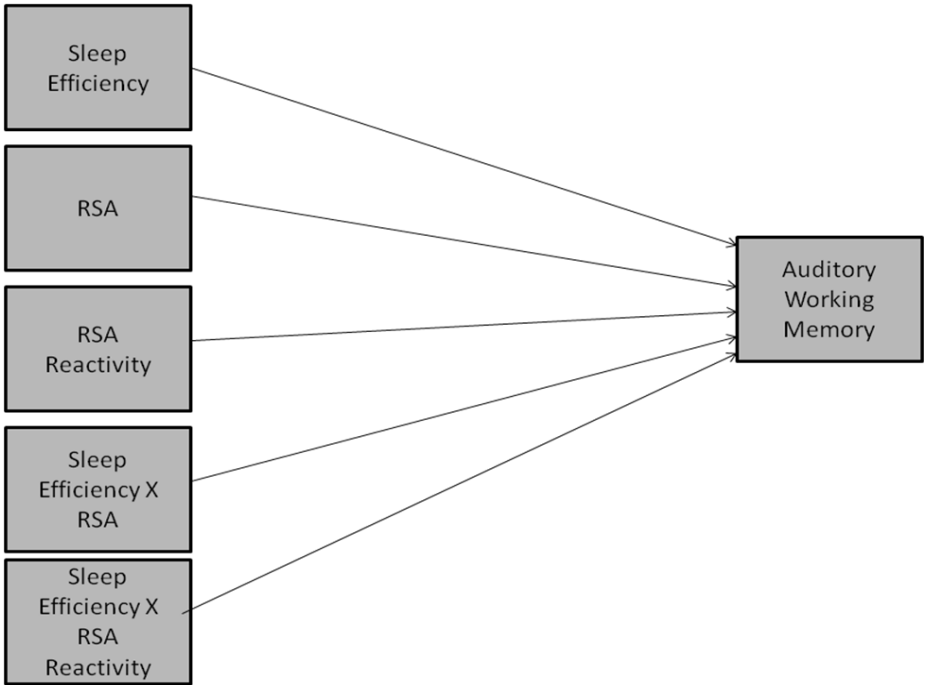
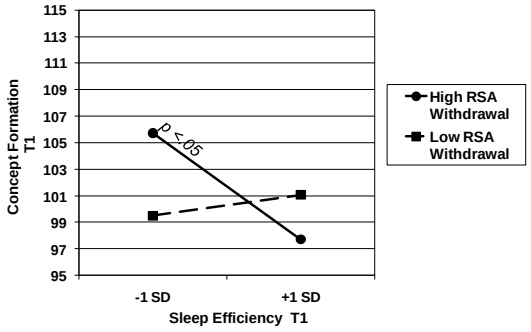


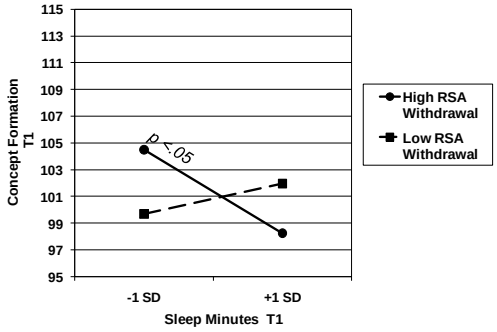
Figure 2. An example of the path models that examined the possible moderating role of RSA and RSA-R in the sleep-cognitive functioning link. Of note, control variables are not included in the model above however all controls (sex, race/ethnicity, age, body mass index, and season of watch wear) were entered as the last paths in the model.

Figure 3.

A.



B.



C.

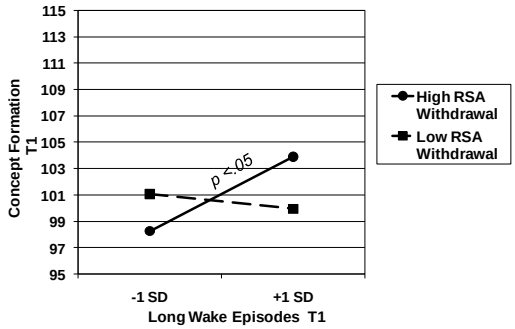
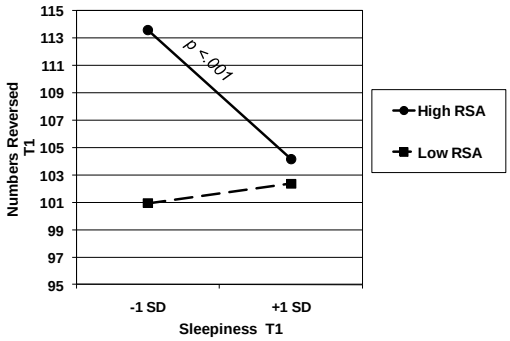


Figure 3. (A) Interaction between Sleep Minutes and RSA-R in the prediction of children's Concept Formation. (B) Interaction between Sleep Efficiency and RSA-R in the prediction of children's Concept Formation. (C) Interaction between Long Wake Episodes and RSA-R in the prediction of children's Concept Formation.

Figure 4.

A.



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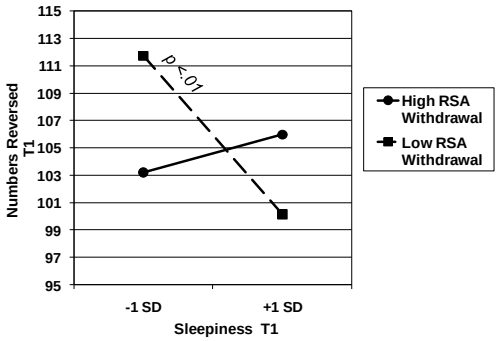


C.

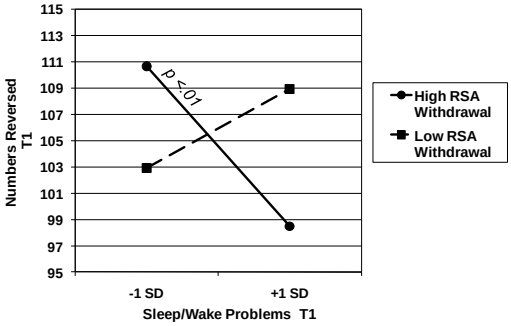
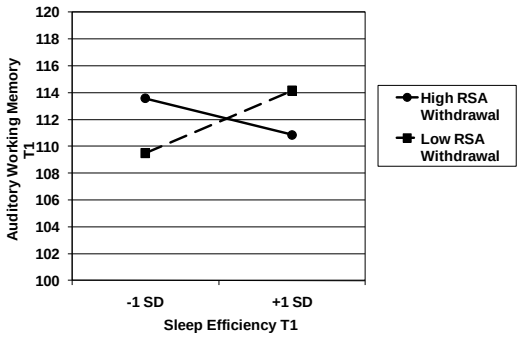


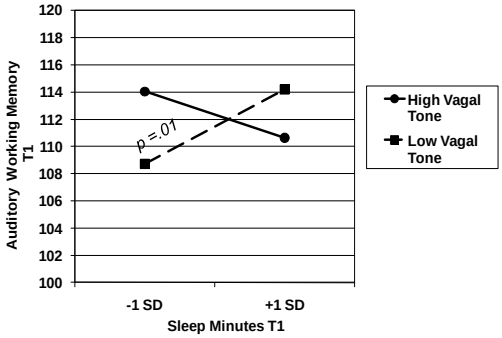
Figure 4. (A) Interaction between Sleepiness and RSA in the prediction of children's Numbers Reversed scores. (B) Interaction between Sleepiness and RSA-R in the prediction of children's Numbers Reversed scores. (C) Interaction between Sleep/Wake Problems and RSA-R in the prediction of children's Numbers Reversed scores.

Figure 5.

A.



B.



C.

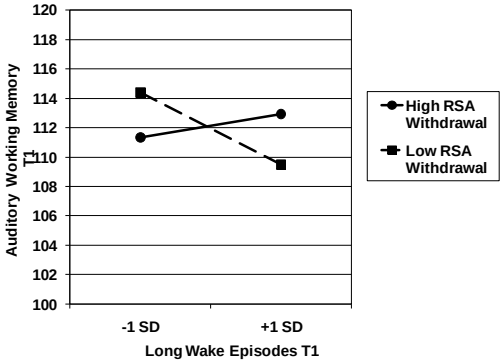
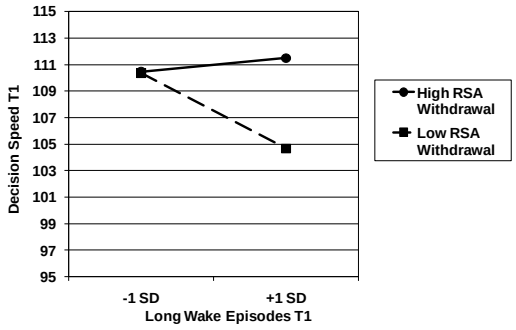


Figure 5. (A) Interaction between Sleep Efficiency and RSA-R in the prediction of children's Auditory Working Memory scores. (B) Interaction between Sleep Minutes and RSA in the prediction of children's Auditory Working Memory scores. (C) Interaction between Long Wake Episodes and RSA-R in the prediction of children's Auditory Working Memory scores.

Figure 6.

A.



B.

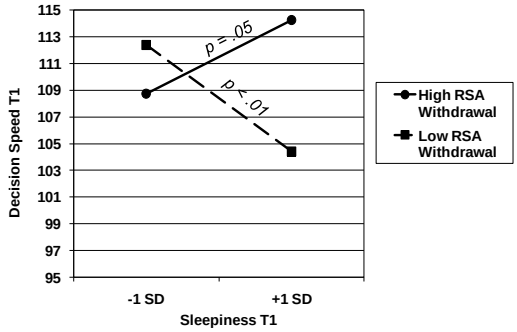


Figure 6. (A) Interaction between Long Wake Episodes and RSA-R in the prediction of children's Decision Speed scores. (B) Interaction between Sleepiness and RSA-R in the prediction of children's Decision Speed scores.

Abstract Manuscript 2

Children's respiratory sinus arrhythmia (RSA) indices (i.e., baseline levels, reactivity to a lab challenge and/or recovery following the challenge), markers of the parasympathetic nervous system activity, were examined as potential moderators of the association between sleep parameters (self-report and actigraphy-based) and multiple domains of children's cognitive functioning (i.e., concept formation, working memory, processing speed, attention) both cross-sectionally and longitudinally. African- and European-American third grade children (*N* = 166; 55% boys; *M* age = 8.72) participated at T1 and again two years later (T2; *N* = 132; 80 boys; *M* age = 10.79). At both time points: (1) children's sleep was assessed via seven nights of actigraphy and by interview; (2) cognitive abilities were derived from the Woodcock-Johnson II Tests of Cognitive Abilities (WJ III) and a reaction time task administered individually; and (3) RSA indices (tone, reactivity, and/or recovery) were assessed in the lab. Cross-sectional results indicate that actigraphy-based sleep variables (i.e., sleep minutes, sleep efficiency, and long wake minutes) interact with RSA reactivity (RSA-R) to a problem-solving task to predict children's decision speed scores on the WJ III; cross-sectional findings did not yield significant moderation effects for baseline RSA, RSA recovery. Longitudinal findings show that RSA-R, and to a lesser extent baseline RSA, interact with both self-reported and actigraphy-based sleep parameters to predict multiple WJ III test scores two years later. Results build on the scant literature examining individual differences in the sleep-cognitive functioning link and suggest that interactions between physiological and biological systems are important for the prediction of children's cognitive functioning.

Sleep and cognition: The moderating role of respiratory sinus arrhythmia activity

The effects of sleep on children's and adolescents' functioning are receiving increased attention in both the research and public media realms. Studies have provided strong evidence that sleep problems, both clinical and non-clinical, can lead to a number of problems, including emotion dysregulation, behavior problems, and trouble performing cognitive tasks (Sadeh, 2007). In addition, adult and child research has linked sleep problems with psychiatric disorders and physical health problems (Cohen-Zion & Ancoli-Israel, 2004; Dahl & Harvey, 2007; Williams, Sears, & Allard, 2004). However, in the context of sleep disruptions, not everyone shows the same cognitive functioning decrements (Buckhalt, El-Sheikh, & Keller, 2007; Buckhalt, El-Sheikh, Keller, & Kelly, 2009), yet we know little about individual differences that may modulate these associations. Respiratory sinus arrhythmia (RSA), a marker of parasympathetic nervous system activity, is an important individual difference variable that is linked to children's cognitive functioning (Staton, Buckhalt, & El-Sheikh, 2009). RSA interacts with sleep problems to predict child adjustment outcomes (El-Sheikh, Erath, & Keller, 2007), including cognitive functioning (manuscript 1). Thus, in this study we aim to examine how indices of biological regulation (i.e., sleep) and physiological activity (i.e., RSA) interact to predict various parameters of children's executive functioning (i.e., concept formation, numbers reversed, auditory working memory, decision speed) and reaction time (i.e., reaction time, variability in reaction time), both cross-sectionally and longitudinally two years later. Figure 1 depicts the conceptualization of the moderating role of vagal activity in the association between sleep disruptions and cognitive performance.

The majority of sleep research with children has been conducted with clinical samples; however, there has been a recent focus on typically developing children. Non-clinical sleep disruptions such as reduced sleep quality and duration in typically developing school-aged children are estimated at rates ranging from 20% to 50% (Kahn et al., 1989; Mindell, Owens, & Carskadon, 1999; Smaldone, Honig, & Byrne, 2007; Stein, Mendelsohn, Obermeyer, Amromin,

& Benca, 2001). Epidemiologic and laboratory studies suggest that an adequate amount of sleep for school-aged children is around 9 to 10 hours per night (Anders, Sadeh, & Appareddy, 1995; Heussler, 2005; Mindell & Owens, 2009). However, studies with American school-aged children show that they are getting less than the recommended amount (Buckhalt, El-Sheikh, & Keller, 2007; Buckhalt, El-Sheikh, Keller, & Kelly, 2009; Sleep in America Poll, 2004; Spilsbury, Storfer-Isser, Drotar, Rosen, Kirchner, & Redline, 2005). Given consistent links between sleep problems (i.e., short sleep duration, reduced sleep quality) and numerous domains of psychological adjustment (Gregory, Caspi, Eley, Moffitt, O'Connor, & Pouton, 2005), physical health (Redwine, Hauger, Gillin, & Irwin, 2000), social problems (Bates, Viken, Alexander, Beyers, & Stockton, 2002) and cognitive functioning (Buckhalt, El-Sheikh, & Keller, 2007; Sadeh, 2007), sleep of typically developing children including predictors and sequelae warrants further study. Thus, in this study we focus on associations between non-clinical sleep disruptions (i.e., reduced sleep amount and worse sleep quality) and non-clinical problems in children's cognitive performance (e.g., inattention, reduced reaction time responses, short term memory lapses). That is, sleep and cognitive functioning problems in this paper are defined along a continuum and in comparison to other children within this community sample.

Multiple studies conducted with adults and children report links between sleep disruptions (e.g., short sleep, increased night wakings) and poorer cognitive performance, including difficulties with executive functioning (Blunden, Lushington, & Kennedy, 2001; Gozal, 1998; Huntley & Lewin, 2006; Sadeh et al., 2002; Sadeh et al., 2003). Two primary components of executive functioning are sustained attention (i.e., ability to maintain focus on a task for extended periods; Johnsen et al., 2002) and working memory (i.e., continuous updating and prolonged storage of information; Logie, Zucco, & Baddeley, 1990). Executive functions stem from the neural systems of the prefrontal cortex, a part of the brain that is particularly susceptible to sleep disruptions (Curcio, Ferrara, & De Gennaro, 2006; Dahl, 1996; Drummond

& Brown, 2001; Horne, 1988; Harrison, & Horne, 1998), and are important contributors to behavior regulation (Cole, Usher, & Cargo, 1993; Pennington & Ozonoff, 1996).

Research conducted with adults consistently links impairments in attention and cognition with restricted or fragmented sleep (Pilcher & Huffcut, 1996). Adult fMRI studies suggest that sleep may play an important role in the consolidation of newly formed memories (Walker & Stickgold, 2006). In regards to links between children's sleep and cognitive functioning, clinical research indicates improvements in attention and working memory when sleep disorders are treated (Chervin, Ruzicka, Giordan, Weatherly, Dillon, Hodges et al., 2006). Further, research with non-clinical child populations suggests that small reductions in sleep duration or mildly disrupted sleep can impact children's attention, processing speed, working memory, and response times (Buckhalt, El-Sheikh, & Keller, 2007; Sadeh, Gruber, & Raviv, 2003). Further, poor sleep may impact cognitive functioning two-years later (Buckhalt, El-Sheikh, Keller, & Kelly, 2009), and worsening of sleep problems over time predict poorer cognitive functioning later in development (Bub, Buckhalt, & El-Sheikh, in press). Sleep debt or deprivation, occurring when the optimal and actual amounts of sleep are not congruent (Van Dongen, Rogers & Dinges, 2003), has lasting effects on functioning and adjustment (Carskadon, 2002). Reviews of the literature suggest that reduced amounts of sleep and active or fragmented sleep influence many aspects of cognitive functioning, including tests of neurocognitive functioning and reaction time (Buckhalt, Wolfson, & El-Sheikh, 2009; Curcio, Ferrara, & De Gennaro, 2006; Mitru, Millrood, & Mateika, 2002; Sadeh, 2007; Wolfson & Carskadon, 2003). Problems with neurobehavioral functions such as sustained attention are particularly important at ages when skills are being acquired (Van Leijenhorst, Crone, & Van der Molen, 2007). Thus, understanding how and why sleep is related to children's cognitive functioning is essential for children's future achievement.

Experimental sleep-cognitive studies conducted with non-clinically diagnosed children are limited, yet results are similar to the findings with adults. Specifically from pre- to post-test,

children (*M* age = 10.6 years) in the extended sleep group (i.e., an additional one hour of sleep per night for three nights) showed memory and attention improvement, whereas the children in the sleep restriction group (i.e., one less hour of sleep per night for three nights) exhibited significantly slower response times on a reaction time task (Sadeh, et al., 2003). In addition to reduced reaction times, acute sleep restriction (i.e., one night of four hours of sleep) in children (*N* = 93; *M* age = 7.9, *SD* =1.4 years) is linked to increased omission errors and reduced signal detection on a neurocognitive task as compared to a baseline taken two weeks prior to the sleep restriction (Huntley & Lewin, 2006). Both acute (i.e., 5 hours of sleep one night) and prolonged (6.5 hours per night for 6 nights) sleep restriction in children are associated with attention impairments (Fallone, Seifer, Acebo, & Carskadon, 2000; Randazzo, Muehlbach, Schweitzer, & Walsh, 1998).

Although a growing literature with typically developing school-aged children consistently links disrupted or shortened sleep with reductions in cognitive functioning (Buckhalt, Wolfson, & El-Sheikh, 2009; Curcio, Ferrara, & De Gennaro, 2006), the relation between specific facets of sleep (e.g., quality, duration) and cognitive domains (e.g., reaction time, working memory load) is less understood. Further, findings stemming from actigraphy do not yield the same associations found with self-reported sleep measures. For example, actigraphic measures of sleep including reduced sleep efficiency (a measure of sleep quality) and increased sleep latency (how long it takes to fall asleep once actively trying) in children (*N* = 60; 6- to 13- years-old) were related to incorrect responses on visual and auditory cognitive tasks; short sleep duration was associated with a reduction in working memory at the highest memory load (Steenari et al., 2003). From these findings, the authors speculate that sleep quality may be more closely related to working memory performance than sleep duration. However, Meijer and colleagues (2000) sampled 449 Dutch children (9-to 14- years-old) and found no relation between subjective reports of sleep quality or duration and scores on a short attention task. Using polysomnography (PSG), Randazzo and colleagues (1998) found a single night of sleep

restriction for children ages 10 to 14 years impaired their verbal creativity and abstract thinking; no effect was found for less complex cognitive tests assessing attention and reaction time. These variations in findings suggest that studies should incorporate objective (i.e., actigraphy; PSG) and self-reports of sleep, while also examining a wide array of cognitive outcomes with varying levels of difficulty.

Adult and child research indicates individual difference variables contribute to the strength of the connection between sleep and cognitive performance. For example, VanDongen (2006) found the association between adult sleep deprivation and poor cognitive performance to be moderated by the individual amount of sleep need, the timing of the circadian process, and the basal level of alertness. For children, research examining possible moderators of this relation are limited to race/ethnicity and socio-economic status (SES) (e.g., Buckhalt, Keller, & El-Sheikh, 2007; Buckhalt, Keller, Kelly, & El-Sheikh, 2009), gender (Bub et al., in press) and daytime sleepiness (Geiger, Achermann, & Jenni, 2010). For example, Buckhalt and colleagues (2007; 2009) examined demographic characteristics (i.e., multiple markers of socioeconomic status, race) as possible moderators in the sleep-cognitive link and results show that children from economically disadvantaged backgrounds or from African-American ethnicity were at a greater risk for cognitive problems (crystallized and fluid intelligence) when sleep disruptions were present than did their more advantaged and European-American counterparts. In Bub et al. (in press), girls were found to be more susceptible than boys to the negative impact of increases in sleep problems over time on cognitive performance.

The evaluation of possible individual difference variables at the physiological level are important to consider because they can modulate the type and degree of maladjustment associated with stress exposure (Steinberg & Avenevoli, 2000); chronic sleep disruptions are considered a significant biological stressor (McEwen, 2006). It is important to understand how physiological regulation influences adjustment and cognitive outcomes (e.g., Cicchetti & Dawson, 2002) to develop appropriate intervention and prevention strategies. Parasympathetic

nervous system activity (PNS), the down-regulating component of the autonomic nervous system (ANS), has been established as a correlate and predictor of developmental outcomes (Beauchaine, 2001; Calkins & Dedmon, 2000; El-Sheikh, 2005; El-Sheikh & Whitson, 2006), the), including those related to sleep disruptions (see El-Sheikh, Erath, & Keller, 2007).

PNS Activity

The ANS is comprised of two branches, the PNS and the sympathetic nervous system (SNS; Porges, 1991). The PNS provides an inhibitory influence on the cardiac muscle (i.e., slows heart rate) via the vagus or 10th cranial nerve. Respiratory sinus arrhythmia (RSA), a measure of vagal tone, is used in this study to index the PNS's influence on the heart. RSA represents the changes in heart rate associated with normal cycles of respiration (i.e., heart rate decreases during exhalation and increases during inhalation). RSA is considered an appropriate index of vagal nerve activity under normal conditions (e.g., sitting, breathing normally; Porges, 2007) and is a suitable measure for research with typically developing children because it can be assessed non-invasively (Grossman & Taylor, 2007). RSA is a well-validated physiological marker of emotion regulation (Beauchaine, 2001; El-Sheikh & Erath, 2011; Porges, 2007; Thayer & Lane, 2009), is a correlate of a number of child adjustment outcomes, including cognitive functioning variables (Staton, El-Sheikh, & Buckhalt, 2009), and is often found to function as a physiological risk or protective factor during childhood (Beauchaine, 2001; Calkins & Dedmon, 2000; El-Sheikh, 2005; El-Sheikh & Whitson, 2006).

The Polyvagal Theory (Porges, 2007), the guiding theory for this study, is the prominent theory in the area of vagal research and it posits connections between the ANS and cognition; also similar to the neuroanatomical integration of neural networks developed by Thayer and Lane (2007). Specifically, the Polyvagal Theory postulates that mammal's first response when confronted with a challenge is to disengage the vagal brake (i.e., vagal withdrawal), which allows the individual to focus attention on the environment, obtain information, and/or engage social strategies to reduce the challenge/stress. Once the challenge is over, the PNS influence

can reengage the vagal brake thus reducing arousal. The ability to physiologically engage and disengage as the environment demands (e.g., the need for focused attention; Suess, Proges, & Plude, 1994) is important for optimal cognitive performance. We utilize the Polyvagal Theory and our extension of the theory to consider vagal tone as a moderator of effects (El-Sheikh & Erath, 2011) as guiding frameworks for our examination of vagal functioning as it interacts with child sleep disruptions to predict cognitive functioning.

Baseline vagal tone reflects PNS activity at rest and the ability to sustain attention and maintain homeostasis under normal conditions (Porges, 1991). Supporting empirical research indicates that higher levels of baseline vagal tone are positively associated with more optimal adjustment outcomes (Rottenberg, 2007). That is, higher vagal tone maintains a low heart rate under normal conditions (Porges, 1991), which reduces the wear and tear on the heart and leads to more optimal health and adaptation (Whitson & El-Sheikh, 2003).

Increases and reductions in heart rate by vagal pathways to promote regulatory and goal-directed behavior are referred to as RSA reactivity (Porges, 1995, 2007; Thayer & Lane, 2000). When a challenge is encountered, the PNS can respond in one of two ways: (1) it can be withdrawn or suppressed, frequently referred to as vagal withdrawal, which leads to an increase in heart rate; or (2) it can be activated or augmented, which leads to a decrease in heart rate. Vagal withdrawal facilitates focused attention when needed (Huffman et al., 1998; Porges, 2007) and the shift in metabolic resources to support information processing (Bornstein & Suess, 2000). Thus, vagal withdrawal is generally considered the most optimal response when confronted with a challenge. However, infants' attentiveness has been associated with vagal augmentation (i.e., jack-in-the-box play; DiPietro et al., 1992), and very high levels of vagal withdrawal have been linked with adjustment problems (Beauchaine, 2001). Examinations of associations between vagal reactivity and cognitive outcomes in children are few and warrant further investigation. Although vagal tone and vagal reactivity are often moderately correlated, each has shown differential relations to child outcomes (Calkins, 1997), therefore supporting the

inclusion of both forms of vagal activity towards a more thorough examination of child functioning.

Vagal recovery, the change in RSA once a challenge has subsided, is a third component of vagal activity. The Polyvagal Theory proposes that a faster return to baseline levels following a challenge is an optimal response and empirical research on health and adaptation is supportive. Vagal recovery has not received much attention in the literature; however, adult research indicates low recovery (i.e., slower return to baseline levels) is associated with an increase in cardiovascular disease (Mezzacappa, Kelsey, Katkin, & Sloan, 2001). Only one study to our knowledge has examined vagal recovery in children (ages 4 to 7), and results indicate low vagal recovery predicts the inability to focus attention (Santucci, Silk, Shaw, Gentzler, Fox, & Kovacs, 2008). Further examination of vagal recovery and cognitive functioning is needed and our assessment of three indices of vagal functioning in this study is more likely to explicate relations examined than any single vagal index alone.

Sleep and RSA Functioning

Few child studies have investigated associations between sleep and RSA functioning. Of those few investigations, vagal withdrawal to a lab stressor functioned as a protective factor, while vagal augmentation was shown to be a risk factor. For example, vagal augmentation during a reaction time task was linked to a higher occurrence of sleep problems (El-Sheikh & Buckhalt, 2005). In a previous paper with the first wave of this sample, vagal withdrawal to an inter-adult argument was a protective factor against child externalizing and depression symptoms in the context of sleep problems (El-Sheikh, Erath, & Keller, 2007). Further, children who exhibited less vagal withdrawal or increased vagal augmentation were more at risk for adjustment problems (e.g., externalizing symptoms) in the context of increased sleep disruptions (El-Sheikh et al., 2007).

RSA and Cognition

A greater ability to engage and disengage with the environment as needed is associated with more optimal vagal functioning (Porges, 2001; Thayer & Lane, 2000) and is necessary for optimal cognitive performance. Researchers who have examined associations between vagal indices and cognitive performance have addressed this question with infant and adult samples; few have assessed these relations with typically developing school-aged children. Of those few studies, higher baseline RSA was linked to greater sustained attention (Suess, Porges, & Plude, 1994) and better cognitive performance on standardized tests of cognitive abilities (Staton, El-Sheikh, & Buckhalt, 2009). Additionally, greater vagal variability (i.e., wider range of responses; higher baseline and greater vagal withdrawal) was predictive of better executive control (Mezzecappa, Kindlon, Saul, & Earls, 1998). However, Staton and colleagues (2009) did not find vagal reactivity during a continuous performance task to predict cognitive performance on the task or on the standardized cognitive assessments (i.e., WJ III). Only one study to our knowledge has demonstrated relations between longer vagal recovery and worse cognitive outcomes (i.e., inattention; Santucci et al., 2008). Thus, more examinations are needed to delineate relations among vagal indices and cognitive functioning parameters in school-aged children.

New Directions

This study advances prior sleep-cognitive literature by examining the moderating role of three important physiological regulation indicators: vagal tone (RSA), vagal reactivity (RSA-R), and vagal recovery (RSA recovery). Executive functions and PNS regulatory functions are known to be disrupted by sleep problems (El-Sheikh & Buckhalt, 2005; El-Sheikh, Erath et al., 2007; Irwin, 2008), however sleep is not the only contributor to the neural networks that influence vagal functioning. Supportive of our examination of RSA indices as moderators of effects, variability in vagal functioning exists independent of sleep and RSA parameters are considered stable individual difference variables for school-aged children (El-Sheikh, 2005;

Salomon, 2005). Polyvagal Theory states optimal vagal functioning facilitates efficient regulation of attention and Dahl (1996) considers daytime regulation of arousal and emotion to affect sleep. In accordance with the Polyvagal Theory (Porges, 1998; Porges, 2007) and Dahl (1996), and our extension of these views, we expect vagal functioning may exacerbate or attenuate the effect of sleep problems on children's cognitive functioning (see El-Sheikh et al., 2007; manuscript 1).

It is our hypotheses that lower RSA, RSA augmentation (or reduced vagal withdrawal) in response to the challenge task, or a reduced RSA recovery following the lab task will interact with sleep problems to exacerbate the risk of poor cognitive functioning. Conversely, and consistent with a conceptualization of protection and vulnerability along a continuum (Brown & Barlow, 2005), higher RSA, RSA withdrawal, and a greater RSA recovery are expected to attenuate the association between sleep problems and poor cognitive performance. Given the scarcity of this literature, no specific hypotheses are advanced regarding the moderating role of the various vagal indices in relation to specific types of sleep disruptions (e.g., duration, quality). Of note, all of the cognitive outcomes that we examine involve executive functioning and require attention for better performance. We expect that cognitive tests tapping higher levels of executive functioning (e.g., highest working memory load) will be most likely influenced by vagal tone and its moderating effects (Staton et al., 2009).

To conduct a rigorous test of our hypotheses, we examine actigraphy and children's self-reported sleep problems and assess multiple sleep parameters (i.e., duration, quality) as recommended in the literature (Sadeh, 2008). Actigraphy (Buckhalt et al., 2007; 2009; Sadeh, Gruber, & Raviv, 2000) and subjective reports of sleep (Buckhalt et al., 2007; 2009; Wolfson & Carskadon, 2003) have been linked to cognitive performance and thus their examination in the context of vagal functioning was considered important. Similarly, we administered an extensive and well-validated cognitive abilities test and a well-established reaction time task to allow for a more thorough examination of cognitive functioning in relation to sleep and vagal activity.

Examinations of multiple sleep parameters as they interact with three vagal functioning indices is imperative for a more nuanced explication of physiological and biological domains linked with various cognitive performance parameters.

Method

Participants

Three school systems in the Southeastern United States provided contact information for children enrolled in the third grade. Researchers then contacted parents by letter or phone. Via initial phone contact, researchers assessed if the children were eligible for participation in the study based on the following criteria: (1) they lived in a two-parent home for at least two years; (2) had no chronic physical illness; (3) were not diagnosed with ADHD, learning disability or mental retardation; or (4) did not have a history of diagnosed sleep problems. Of those eligible, 66% agreed to participate. Children with acute illnesses (e.g., flu) were asked to postpone their participation until they were well.

The sample at T1 consisted of 166 third grade children (45% boys). Children had a mean age of 8.72 (*SD* = .30), with 94% of the sample considered prepubertal and 6% early pubertal (Petersen et al., 1988). The majority of the children (77%) lived with both biological parents; 21% lived with their biological mother and a step-father; 2% lived with their biological father and a step-mother and marital partners had been living together for an average of 10.20 years (*SD* = 5.30 years). Consistent with the recruiting school districts' demographic characteristics, 69% of parents reported being European-American (EA) and 31% reported being African-American (AA). Oversampling methods were used to ensure that families from both ethnic groups represented a wide range of socioeconomic levels. The sample was diverse in relation to socioeconomic status: 26% of the families were unskilled or semiskilled workers, 44% were skilled workers, and 30% were minor professionals or professionals (based on Hollingshead 1975 criteria). Mothers reported the annual familial income at the following ranges: 4% less than 10,000; 7% between $10,000 and $20,000; 25% between $20,000 and $35,000;

28% between $35,000 and $50,000; 25% between $50,000 and $75,000; and 11% more than $75,000. In terms of years of parental education, mothers had an average of 13.80 years (*SD* = 2.82) and fathers had an average of 13.20 years (*SD* = 2.82), indicative of one year of post high school education or specialized training.

Of the original 166 participants, 132 children and their families (80 boys) participated two years later (T2; 80% of the original sample). Attrition was due to the inability to be located, hectic schedule, lack of interest, and geographic relocation. At T2, children's mean age was 10.80 years (*SD* = .36). The ethnic composition of the sample was 70% EA and 30% AA. Pubertal status was 2.07 (*SD* = .56) for girls, which is indicative of an early pubertal status (Petersen et al., 1988). Boys were considered to be in prepubertal stages, with a mean pubertal status score of 1.49 (*SD* = .42).

In comparison to children who participated in the longitudinal follow up, those who dropped out of the study had lower scores on the Woodcock Johnson Test of Cognitive Abilities (WJ III, Woodcock, McGrew, & Mather, 2001) for Cognitive Efficiency, $t(164) = -2.73$, $p < .01$, and came from families characterized by lower overall SES, $t(164) = -3.37$, $p < .001$. Missing and retained participants did not differ in terms of ethnicity, gender, sleep parameters, or vagal indices.

Procedures

This study is part of a larger longitudinal investigation of the role of the family environment (e.g., marital conflict) in relation to children's adjustment, sleep, and health. Only the pertinent procedures are detailed. The study was approved by the institution's Internal Review Board (IRB), and parents provided consent and children provided assent for participation. Families received monetary compensation for their participation. All procedures were identical at T1 and T2 unless otherwise indicated.

During both waves of data collection, research assistants delivered the actigraph (watch-like devices that measure movement) to the child's home and instructed the parents to place the

watch on the child's non-dominant wrist just prior to bedtime and remove it upon waking for seven consecutive nights. A week of actigraphic assessments exceeds the recommended number of nights for the reliable measurement of typical sleep parameters (Acebo et al., 1999). During the week of actigraphy, researchers called parents daily to obtain children's bed and wake times, which were used to corroborate actigraphy measures. Actigraphic data were collected only during the regular school year to avoid confounding variables (i.e., irregular sleep patterns due to less structured wake times). Due to the influence medications have on sleep (Qureshi & Lee-Chong, 2004), only data from medication-free nights were used in analyses.

Typically on the day following the last night of actigraphic sleep assessment, children and their families came to the university laboratory where children were individually administered the brief version of the Woodcock-Johnson III Cognitive Abilities Battery (WJ III; Woodcock et al., 2001), completed a 15-minute computerized reaction time task (i.e., Donders), participated in a physiological assessment session and completed an interview.

To reduce anxiety associated with attaching electrodes to obtain RSA data, researchers conversed with the child and a parent (typically the mother) while the electrodes and a pneumatic bellows belt were attached. This process took approximately ten minutes, which allowed a sufficient amount of time for the child to relax prior to the collection of physiological data. The parent and researcher then left the room and the child was given two additional minutes to acclimate to the setting without others present before data collection began (the child was asked to relax while sitting down quietly). Immediately following, a three minute baseline (resting level) was obtained. Three minutes are considered more than adequate for assessment of RSA (Bar-Helm et al., 2000). To assess RSA reactivity, children completed a mildly stressing problem-solving task, namely the star-tracing task (Matthews, Woodall, & Stoney, 1990). This task requires the child to trace a star on a sheet of paper while only seeing the star in a mirror. In addition to the difficulty associated with the reverse properties that the mirror adds, children were only given three minutes to trace the star, which increases task demands. This task has

been used in numerous investigations to evoke ANS reactivity in children (El-Sheikh, et al., 2009; Matthews, Rakaczky, Stoney, & Manuck, 1987; Matthews, Woodall, & Stoney, 1990). At T2, an additional three minute assessment of RSA following the star tracing task allowed for the examination of RSA recovery. Following the physiological session, children were given a break and a snack and were then interviewed by a research assistant regarding their sleep habits.

Measures

Children's Objective Sleep. The actigraph wristwatch, Motionlogger Octagonal Basic (Ambulatory Monitoring, Inc., Ardsley, NY), is small (36 mm X 38 mm X 13 mm) and lightweight (45 g). Movement during sleep was continuously recorded and collected in 1-min epochs using zero crossing mode. The raw data was processed with the Octagonal Motionlogger Interface with ACTme Software, and the Analysis Software Package (ActionW2 or AW2). Using the Sadeh algorithm, which has established validity with children (Sadeh, Sharkey, & Carskadon, 1994) and good reliability with this actigraph (Acebo, Sadeh, Seifer, Tzischinsky, Wolfson, Hafer, et al., 1999), one-minute epochs were scored as either sleep or wake. Guidelines used for determining sleep onset were developed at the E.P. Bradley Sleep Laboratory at Brown University (Acebo & Carskadon, 2001).

Sleep variables derived via actigraphy included: (a) Sleep Minutes - total minutes scored as sleep during the Sleep Period, providing a measure of sleep duration; (b) Sleep Efficiency - percentage of minutes scored as sleep, providing a measure of sleep quality; and (c) Long Wake Minutes – number of wake episodes during the night at least 5 minutes in duration, providing a second measurement of sleep quality.

Cronbach's alphas were used to assess the stability of children's sleep over the week of actigraphy. The reliability coefficients for T1 and T2 were: Sleep Minutes = .78 and .86; Sleep Efficiency = .86 and .93; Long Wake Episodes = .85 and .88. All of the actigraphy variables exceed the recommendation of Acebo and colleagues (1999) for achieving a stability of .70 or greater.

At T1, 74% of the children had actigraphy data for all seven nights. Due to actigraph malfunctions, four children (2%) did not have any actigraphy data. The remaining 24% had data for fewer than seven nights (*M* = 5.33 days; *SD* = 0.97) due to use of allergy medication, forgetting to put the watch on prior to bed, or sleeping while riding in a car. At T2, the majority of the children (56%) had all seven nights of actigraphy data, 25% had data for six nights, 14% had data for five nights, and 5% had data for four or fewer nights. These percentages of valid actigraphy data are considered to be very good (Acebo et al., 1999) and are comparable to similar studies. Sleep parameters used in analyses were based on averaged data across all available nights of actigraphic assessments.

Children's Subjective Sleep. Children completed the Sleep Habits Survey (SHS; Wolfson & Carskadon, 1998) via interview, which assesses children's perceptions of their sleep within the last two weeks. This questionnaire is commonly used with children and adolescents (Acebo & Carskadon, 2002; Buckhalt, El-Sheikh, & Keller, 2007; Buckhalt, El-Sheikh, Keller, & Kelly, 2009), and has good reliability and validity (Carskadon, Seifer, & Acebo, 1991; El-Sheikh & Buckhalt, 2005; Wolfson, Carskadon, Acebo, Seifer, Fallone, Labyak, et al., 2003). Two subscales were used in analyses: (1) the Sleep/Wake Problems Scale, which is composed of 10 items that assess the frequency of sleep problems (e.g., oversleeping and staying up late at night); and (2) the Sleepiness scale, comprised of 10 items, which measures the extent to which children struggle to stay awake during their daily activities. One item (i.e., sleepiness while driving) on the Sleepiness scale was deleted due to the child's age. Items were rated on a 5-point Likert scale, and sums for each subscale were computed. Higher scores are indicative of more sleep-related problems and daytime sleepiness. For this sample, the Sleep/Wake Problems Scale coefficient alphas were .67 and .94, respectively. The Sleepiness Scale had a reliability coefficient of .70 at T1 and .79 at T2.

RSA, RSA-R, and RSA Recovery. To obtain heart rate, electrodes were placed axially on the left and right rib cage, approximately 10 to 15 cm below the armpits. A pneumatic bellows

was placed around the chest and held in place using a metal beaded chain. Physiological acquisition equipment and software were from James Long Company. The bioamplifier was set for band-pass filtering with half power cut-off frequencies of 0.1 and 1000 hertz, and the signal was amplified with a gain of 500 for electrocardiography (ECG). The pneumatic bellows, used to assess respiration, was attached to a pressure transducer with a band-pass of direct current to 4000 hertz, to minimize phase or time shifts in the measurement. Data were digitized at a sampling rate of 1000 Hz with a 12-bit analog-to-digital board in a laboratory computer.

An automated algorithm was used to identify the R-waves in the ECG data. Manual correction of missed or misidentified R-waves occurred in the rare event that it was needed using an interactive graphical program. R-wave times were converted to inter-beat intervals (IBIs) and resampled into equal time intervals of 125 ms. Prorated IBI values were used for analysis of the mean and variance of heart period as well as the processing of heart period variability due to respiratory sinus arrhythmia (RSA).

The peak-valley method was used to compute RSA, and RSA is reported in seconds. The peak-valley method is one of several acceptable procedures for quantifying RSA (Berntson et al., 1997) and this procedure highly correlates with spectrally derived measures of RSA (Galles, Miller, Cohn, & Fox, 2002). To identify inspiration and expiration onset times and relative tidal volume, the respiration signal was used. RSA was calculated as the difference in IBI between inspiration and expiration onset for consecutive respiratory cycles. Specifically, RSA was computed twice per breath, once from inspiration to expiration and again from expiration to inspiration, and then averaged across respiratory cycles to ensure that any underlying trends in heart period had little impact on the computed mean RSA. To derive RSA-R in response to the star tracing task, a change score between RSA during the task and RSA at baseline (i.e., task minus baseline) was computed. Negative RSA-R scores indicate RSA withdrawal to the task, whereas positive RSA-R scores represent augmentation to the star tracer. In all analyses examining RSA-R, we controlled for RSA. RSA recovery was calculated

as the difference between RSA during the three minutes after the star tracing task and RSA during the initial baseline (Santucci, Silk, Shaw, Gentzler, Fox, & Kovacs, 2008). A higher RSA recovery score represents a faster return to individual baseline levels of vagal tone after the challenge had subsided, whereas a lower recovery score represents slower return to basal RSA levels.

Cognitive Functioning. The Woodcock-Johnson III Tests of Cognitive Abilities (WJ III; Woodcock et al., 2001) is a well-normed measure of general intellectual ability (*g*), crystallized intelligence (*Gc*), fluid reasoning (*Gf*), and processing speed (*Gs*). General intellectual ability refers to non-specific abilities that affect the performance of most tasks, whereas *Gc* is thought to reflect skills acquired through knowledge and experience (e.g., verbal ability). Fluid reasoning is the capacity to think logically and solve novel problems and processing speed involves the ability to automatically and fluently perform tasks. The following tests were administered: Concept Formation (CF); Numbers Reversed (NR); Auditory Working Memory (AWM); and Decision Speed (DS). CF is a novel controlled-learning test that requires the use of categorical reasoning based on principles of inductive logic. This test assesses fluid reasoning and one's flexibility in shifting mental sets; the latter is an aspect of executive functioning. NR requires that numbers be held in working memory while executing a mental operation (i.e., reversing the sequence), and examines short-term memory. AWM is another test of short-term and working memory, but it also requires the use of divided attention. Specifically, a series of numbers and words are presented in a random order, and the respondent is asked to recall the words in the correct order they were stated, and then the numbers. For the last test, DS, one is asked to select two conceptually similar items from a row of alternatives. Multiple rows of items are presented and one is only given 3 minutes to complete as many rows as possible, thus assessing visual processing speed. All tests started at a basic skill level and increased in complexity as the test progressed. These tests were chosen to comprehensively examine

cognitive functioning processes that may be impacted by sleep disruptions, specifically attention, working memory and simple processing speed.

Reaction Time. Speed and variability of response speed across a number of trials was assessed using a computerized reaction time task known as Donders (Psych/Lab; Abrams, 2004). Multiple conditions were used to obtain reaction times. Specifically, the child was seated at a computer and a researcher instructed the child to focus on a "1" in the center of the computer screen. The child placed the right index finger on the "/" key, and the left index finger was placed on the "Z" key. The child was told to follow the instructions presented on the screen, which were also read to the child by the researcher. A short tone sounded if the child responded too early, too late, not at all, or if a key was pressed when one was not required, a short tone was sounded.

To assess simple reaction time (Donders Type A), the child was told to press the "/" key as quickly as possible when the "x" appears in the box on the right. For the assessment of choice reaction time (Donders Type C), two boxes were presented and either could have the "x" appear, but children were told to only respond (by pressing "/") when the "x" appears in the right box. The difficulty is increased in this task due to the monitoring of two boxes and having to inhibit a response with the left finger. In a third condition, choice reaction time 2 (Donders Type B), two boxes were presented and the "x" could appear in either box. Children were instructed to press the "/" key with the right finger when the "x" appears in the right box or press the "z" key with the left finger if the "x" appears in the left box. Conditions were presented in a fixed order with the following parameters: 10 practice trials per condition; 20 test trials per condition; a 500 ms inter-trial interval; 100 ms minimum allowed RT; and 1,500 ms maximum allowed RT. Trials were repeated if responses were faster or slower than the criteria. Test trial averages and standard deviations were computed for each condition and were derived for analyses. The mean scores represent the average response time, whereas the standard deviation scores represent vigilance to the task. Examination of children's responses during the three conditions

(types A, B, and C) indicated that both the means (*rs* = .66 – .76, *ps* < .001), and *SD*s (*rs* = .37 – .42, *ps* < .001) were significantly correlated. Thus, to reduce the number of analyses, one averaged mean and one *SD* were derived and used in all subsequent analyses.

Body Mass Index. Children's height and weight (via a Sunbeam scale) were measured during the laboratory visit and used to calculate BMI [703 x (weight/height^2); http://www.cdc.gov]. Based on the Center for Disease Control's classifications, 1% of the T1 sample was categorized as underweight, 61% was of normal weight, 23% were overweight, and 15% were obese. At T2, 2% were classified as underweight, 72% were of normal weight, 13% were overweight, and 13% were obese.

Demographic Variables. Child age, ethnicity, sex, and familial SES were obtained from a parent over the phone prior to participation. Familial SES was derived using parental education and occupation as indexed via the Hollingshead Index (Hollingshead, 1975).

Results

Preliminary Analyses

Means and standard deviations for primary study variables are presented in Table 1. Descriptive analyses indicated on average, children had fairly low sleep efficiency (87% at T1; 89% at T2); ≥ 90% is optimal (Sadeh et al., 2000). Similarly, at both time points children obtained well below the recommended average amount of sleep for children of this age (~10 hours; www.cdc.gov/features/sleep/), with the average sleep duration similar across time points (~7.25 hours per night). Most children exhibited RSA withdrawal to the star tracing task (69% at T1; 49% at T2); 13% at T1 and 12% at T2 showed no change and 18% at T1 and 39% at T2 exhibited RSA augmentation to the task. At T2 (recall that recovery data were not derived at T1), 45% of children had RSA levels during the post-task baseline that were lower than those during the initial baseline; 16% fully recovered (the difference between the initial and post-task baselines was zero); and 38% had RSA levels higher during the post-task than the initial baseline. Children in the sample scored the lowest on Concept Formation at both waves and the

highest on Auditory Working Memory at T1 and Decision Speed at T2; all scores were in the normative range.

Relations between the main study variables (sleep, vagal indices, cognitive functioning, reaction time) and several confounding variables (BMI, child age, gender, ethnicity, socioeconomic status, season of actigraph collection, and pubertal status) were examined. Lower BMI was associated with higher RSA at both time points. Child age was associated with most of the cognitive functioning variables at T1 (i.e., older children typically performed better than younger children), however this association was not found at T2. Gender differences in actigraphic and self-reported sleep patterns were found at T1, with boys having more sleep problems than girls. AT T2, these gender differences were not apparent. At both assessments, AA children reported more sleepiness and sleep/wake problems than their EA counterparts. Actigraphic results indicated that AA children had fewer long wake episodes than EA children at T1 and less sleep duration at T2. At T2, girls had higher decision speed scores and a slower mean reaction time than boys at T1, with reaction time associations holding across waves.

Table 2 shows associations among study variables. As expected at cross-sectionally and longitudinally, actigraphy-based sleep variables were highly intercorrelated and so were the self-report measures of sleep. Although sleep efficiency and long wake minutes were related, these are considered to be separate constructs in that long wake minutes can be used as an index of insomnia, whereas sleep efficiency is percentage of active versus non-active sleep over the course of the sleep onset to sleep offset. The WJ III variables and the reaction time measures were also highly correlated within and across time points. RSA indices were correlated within each timepoint, however there were no significant associations between RSA-R at T1 and RSA-R at T2; RSA was correlated over time with itself. RSA and RSA-R were not related to either sleep or cognitive variables at T1, however at T2 RSA-R was cross-sectionally related to children's sleep efficiency, sleep minutes, and self-reported sleepiness.

Analysis Plan

AMOS 17.0 (Arbuckle, 2007) was used to fit a series of path models. Path analysis was chosen because unlike multiple regression it employs maximum likelihood estimation to handle missing data, which improves the power. Additionally, the assumption that the data are missing completely at random (MCAR; Little & Rubin, 2002) is more relaxed in path analysis than in multiple regression.

In each path model, residuals of endogenous variables and significantly related exogenous variables were allowed to correlate. The covariates for each model were entered as the last paths in the model. Covariates for each model included: season of actigraphic assessment, child gender, ethnicity, age, BMI, and familial SES; longitudinal models also controlled for the autoregressive effects. Data points that were considered to be outside the normal distribution (i.e., *SD*s greater than 4) were removed from the dataset (n = 2 at T1; n =1 at T2). After removing the univariate outliers, variables were also checked for multivariate outliers. Results did not indicate any multivariate outliers.

Cross-sectional models were fit for each objective sleep parameter (sleep efficiency, long wake episodes, sleep minutes) and each cognitive outcome (concept formation, numbers reversed, auditory working memory, decision speed, reaction time *M*, reaction time *SD*) resulting in 18 models. Moreover, self-reported sleep parameters were examined simultaneously (sleepiness, sleep/wake problems) for each of the six aforementioned cognitive outcomes; a total of six models. All models contained three vagal indices (RSA, RSA-R, RSA recovery), the main effects and the associated interactions with the sleep parameter(s). Control variables were added to the models: season of sleep assessment, child age, sex, ethnicity, BMI, and SES status. All predictor and control variables were centered prior to inclusion in the path model.

Longitudinal models were also fit separately for each objective sleep parameter at T1 predicting each cognitive outcome variable at T2; a total of 18 models. An additional six models

were fit in which sleepiness and sleep/wake problems at T1 predicted T2 concept formation, numbers reversed, auditory working memory, decision speed, reaction time mean, and reaction time variability. All longitudinal models included two T1 vagal parameters (RSA, RSA-R), the main effects, the associated interactions with the sleep parameter(s), and the autoregressive effect (T1 cognitive outcome). Controlling for prior levels of the predicted outcome helps to reduce bias in parameter estimates, allows for conclusions about change in the predicted variable, and provides knowledge about the direction of effects (Cole & Maxwell, 2003). An example of the models is presented in Figure 2.

Actigraphy-based sleep variables were examined separately based on calls in the pediatric sleep literature to include multiple specific sleep variables in research to facilitate comparisons across studies (Berger, Wielgus, Young-McCaughan, Fischer, Farr, & Lee, 2008; DeWald, Meijer, Oort, Kerkhof, & Bogel, 2010) and to further delineate associations between sleep parameters and child functioning. Additionally, separate examinations of duration, quality and sleepiness have been recommended because research indicates that these are separate sleep domains (DeWald et al., 2010). Further, it is suggested that researchers examine multiple sleep parameters including actigraphy-based and self-reported measures of sleep (Sadeh, 2011). Due to multicollinearity and power considerations (Babyak, 2004; Whisman & McClelland, 2005), each cognitive outcome was fit separately.

Considering the large number of models fit (n = 24 cross-sectional; n = 24 longitudinal) and to reduce the likelihood of Type 1 error, we only report and interpret findings when significant interactions represented at least 10% of the number of interactions examined for that cognitive outcome (e.g., ≥ 10% of significant findings across all models associated with concept formation). Models were considered an acceptable fit if at least two of the three following criteria were met: $\chi^2/df < 2$, CFI > .95, and RMSEA < .08, p <.05 (Arbuckle & Wothke, 1999; Browne & Cudeck, 1993). All fitted models reported satisfied these criteria.

Significant interactions were plotted at high (+1 SD) and low (-1 SD) levels of the predictor and moderator as recommended by Aiken and West (1991). Of note, a very small percentage of the sample exhibited RSA augmentation to the task. Thus, the depiction of the less optimal RSA reactivity group (i.e., Low RSA Withdrawal) includes children who exhibited no change, augmentation, or less vagal withdrawal in comparison to their counterparts. The interaction utility developed by Preacher, Curran, and Bauer (2006) was used to graph the interactions and estimates plotted were obtained from the fitted models. Significant interactions indicate that there is a statistically significant difference between the plotted lines (e.g., the association between sleep and cognitive functioning at high levels of the moderator is significantly different from the link at low levels of the moderator). We also tested each slope to determine whether it was significantly different from zero; significant slopes are indicated in the figures.

Cross-Sectional Examinations of T2 RSA Indices as Moderators of Associations between Children's Sleep and their Cognitive Performance

Concept Formation. To examine whether RSA, RSA-R, or RSA recovery following the task moderated the cross-sectional pathway between children's sleep parameters and their CF scores, interaction terms were added to the model. None of the four models fit indicated a significant moderating role of any of the three RSA indices.

Numbers Reversed. Analyses yielded no significant moderation effects for RSA, RSA-R, or RSA recovery in the link between children's sleep and their cognitive performance indexed by NR scores.

Auditory Working Memory Scores. No moderation effects were observed for any of the vagal indices.

Decision Speed Scores. Analyses of children's performance on the WJ III decision speed test indicated that RSA-R was a significant moderator in three of the four models

examined (75% of possible relations); no moderation effects were found for either RSA or RSA recovery.

RSA-R moderated the link between sleep efficiency and DS (β = - .33, p < .05), explained 6% of unique variance in DS (χ^2 = 59.45, p < .06, χ^2/df = 1.35, CFI = .92, RMSEA = .00). The full model explained 14% of the variance in DS. As depicted in Figure 3A, regardless of sleep efficiency, children who exhibited RSA withdrawal to the star tracing task showed better performance on the DS test than those who exhibited RSA augmentation. Further, for children who exhibited RSA augmentation, sleep efficiency was negatively associated with DS.

Similar to the pattern of effects observed for sleep efficiency, the association between long wake episodes and DS was moderated by children's RSA-R (β = .31, p < .05), which explained 4% of unique variance in DS (χ^2 = 62.29 p = .26, χ^2/df = 1.11, CFI = .96, RMSEA = .00); the full model accounted for 17% of the variance in DS. Specifically, regardless of the quality of sleep, children who exhibited RSA withdrawal to the lab challenge performed better than their counterparts on the DS test (Fig. 3b). Children who showed RSA augmentation during the lab challenge had a positive association between long wake episodes and DS and the slope representing this association was significantly different from zero.

RSA-R also moderated the relation between sleep minutes and DS (β = - .29, p = .05) and explained 4% of unique variance in DS (χ^2 = 61.78 p = .04, χ^2/df = 1.40, CFI = .89, RMSEA = .04). The full model accounted for 14% of the variance in DS. As shown in Figure 3C, in the context of longer sleep duration children who exhibited RSA withdrawal to the star tracing task performed better on the DS test than those who exhibited RSA augmentation.

RSA activity did not moderate relations between Sleepiness or Sleep/Wake Problems and DS.

Reaction Time Means and SDs. RSA parameters did not moderate the link between children's sleep (objective and subjective) and their performance on the reaction time task (*M* or *SD* scores).

Longitudinal Examinations of T1 RSA Indices as Moderators of Associations between Children's Sleep at T1 and their Cognitive Performance at T2

Concept Formation. To examine whether RSA or RSA-R moderated the longitudinal pathway between children's sleep parameters (both objective and subjective) and their CF scores, interaction terms were added to the path models after the inclusion of the main effects. One of the four models (25%) was significant for RSA and 50% were significant for RSA-R.

The relation between sleep efficiency at T1 and children's scores on the CF test at T2 varied based on children's RSA (β = - .14, p = .05) and explained 2% of unique variance in CF (χ^2 = 74.85, p = .02, χ^2/df = 1.43, CFI = .88, RMSEA = .05). A total of 45% of the variance in CF was accounted for by the full model. Specifically, in the context of low sleep efficiency, children who had high levels of RSA performed better on concept formation two years later than children who exhibited low RSA (Fig. 4A).

RSA did not function as a moderator in the longitudinal pathway between long wake episodes at T1 and children's CF at T2, but RSA-R did (β = - .20, p < .01) and explained < 1% of unique variance in CF (χ^2 = 94.13 p = .001, χ^2/df = 1.71, CFI = .76, RMSEA = .07). The full model accounted for 44% of the variance in CF. Specifically, children who expressed RSA withdrawal to the lab task had a significant positive association between T1 long wake episodes and T2 CF (Fig. 4B). The pattern of effects was only partially consistent with expectations: at high levels of sleep problems, children who showed RSA withdrawal performed better than children who exhibited augmentation; yet the no clear pattern was evident at lower levels of sleep problems.

The association between sleep minutes at T1 and CF at T2 was not moderated by either RSA or RSA-R.

RSA-R (but not RSA) moderated the link between self-reported sleepiness at T1 and CF scores at T2 (β = - .31, p < .001) and explained 5% of unique variance in CF (χ^2 = 134.92 p = .001, χ^2/df = 1.56, CFI = .83, RMSEA = .06); a total variance of 51% was accounted for by the

full model. As depicted in Figure 4C, children who exhibited RSA withdrawal had similar CF scores at T2 regardless of their level of sleepiness reported at T1. However, consistent with expectations, a negative association was found between sleepiness and CF for children who showed RSA augmentation and the slope representing this relation was significantly different from zero.

No moderation effects were found between children's self-reported sleep/wake problems at T1 and their concept formation scores at T2.

Numbers Reversed. Analyses of children's performance on the WJ III NR test indicated that RSA-R, not RSA, moderated the longitudinal pathway between sleep efficiency and NR (β = .20, p < .05) and explained < 1% of unique variance in NR (χ^2 = 67.98 p = .07, χ^2/df = 1.31, CFI = .89, RMSEA = .04); a total of 34% of the variance in NR was accounted for by the full model. Results indicated a negative longitudinal association between children's sleep problems and NR test scores for children who exhibited RSA withdrawal and the slope representing this association was significantly different from zero (Fig. 5A). The slope representing the association between sleep efficiency and NR was not significantly different from zero for children who showed RSA augmentation.

The longitudinal relation between long wake episodes and NR was not moderated by RSA, but RSA-R did act as a moderator of this association, (β = - .21, p < .001) and explained 2% of unique variance in NR (χ^2 = 90.05 p = .00, χ^2/df = 1.63, CFI = .73, RMSEA = .06). The full model accounted for 6% of the variance in NR. Specifically, in the context of high sleep disruptions at T1, children who showed RSA withdrawal to the laboratory challenge performed better on the NR scores than their counterparts with RSA augmentation (Fig. 5B). Further, the longitudinal association between long wake episodes and children's cognitive performance was only significant for those exhibiting RSA withdrawal.

The link between sleep minutes at T1 and children's scores on the NR test T2 was not moderated by either RSA or RSA-R.

RSA did not moderate the longitudinal association between child-reported sleepiness and NR. However, RSA-R did moderate the relation, (β = - .27, p < .001) and explained 2% of unique variance in NR (χ^2 = 144.86 p = .00, χ^2/df = 1.68, CFI = .77, RMSEA = .06). A total of 37% of the variance in NR was accounted for by the full model. As depicted in Figure 5C, in the context of high rates of sleepiness at T1, children who exhibited RSA withdrawal to the task performed better showed better cognitive performance two years later than their counterparts who showed RSA augmentation. For those children who showed RSA augmentation, there was a negative association between child-reported sleepiness and NR scores and this slope was significantly different from zero.

No moderation findings were evident for RSA in the longitudinal pathway between sleep/wake problems and NR. Conversely, RSA-R did moderate the sleep-cognitive link, (β = .16, p = .05) and explained 1% of unique variance in NR (χ^2 = 40.28 p = .41, χ^2/df = 1.68, CFI = .77, RMSEA = .06); 37% of the total variance in NR was accounted for by the model. The pattern of effects was not consistent with our hypotheses (Fig. 5D).

Auditory Working Memory. Examination of children's scores on the WJ III AWM test yielded one significant moderation effect for RSA-R (25% of the possible relations); no moderation effects were found for RSA.

Neither RSA nor RSA-R moderated the association between any of the actigraphy-based sleep measures (sleep efficiency, long wake episodes, sleep minutes) at T1 and children's AWM scores two years later.

RSA-R moderated the link between subjective reports of sleepiness and AWM, (β = - .32, p < .001) and explained 6% of unique variance in AWM (χ^2 = 136.745 p = .00, χ^2/df = 1.59, CFI = .78, RMSEA = .06); the full model accounted for 28% of the variance in AWM. Regardless of the amount of self-reported sleepiness, children who exhibited RSA withdrawal to the star tracing task performed similarly on the AWM test two years later (Fig. 6A). However, consistent with hypotheses, a negative association between sleepiness and AWM was found for children

who showed RSA augmentation and the slope representing this relation was significantly different from zero.

RSA-R also acted as a moderator in the longitudinal pathway between sleep/wake problems and AWM scores, (β = .31, p < .001) and explained 7% of unique variance in AWM (χ^2 = 136.745 p = .00, χ^2/df = 1.59, CFI = .78, RMSEA = .06); the full model accounted for 28% of the variance in AWM. Figure 6B depicts that when sleep/wake problems are high, children who exhibited RSA withdrawal performed worse on the working memory task than those who showed RSA augmentation. Further, for children exhibiting RSA augmentation, a positive association was observed between sleep problems and cognitive performance; however, this pattern was reversed for those exhibiting RSA withdrawal. Both slopes representing these associations were significantly different from zero.

Decision Speed Scores. Analyses revealed that RSA was a significant moderator in one of the four models examined (25% of possible relations); RSA-R did not moderate the link between children's sleep and DS scores over time.

The pathway between sleep efficiency at T1 and children's scores on the WJ III DS test at T2 was not moderated by either RSA or RSA-R.

The longitudinal association between long wake episodes and DS was moderated by children's RSA (β = - .18, p < .05), which explained 3% of unique variance in DS (χ^2 = 94.80 p = .02, χ^2/df = 1.72, CFI = .70, RMSEA = .07). The full model accounted for 34% of the variance in DS. As depicted in Figure 7A, a positive association between children's disrupted sleep and their DS was found for children who exhibited low RSA and the slope representing that relation was significantly different from zero. The performance of children with higher RSA did not seem to be impacted by sleep problems.

RSA activity did not moderate relations between sleep minutes, sleepiness or sleep/wake problems at T1 and DS scores two years later.

Reaction Time Means and SDs. RSA activity did not moderate in the link between children's sleep (objective and subjective) at T1 and their performance on the reaction time task (*M* or *SD* scores) at T2.

Discussion

We investigated whether multiple indices of vagal activity as indexed by RSA (baseline, reactivity, and/or recovery), moderated the associations between children's sleep disruptions and multiple domains of cognitive performance cross-sectionally and longitudinally in a large sample of healthy elementary school-age children. Cross-sectionally, RSA-R was the only index of vagal activity that functioned as a moderator and all significant moderation effects were for associations between actigraphy-based sleep parameters and children's decision speed. Consistent with hypotheses, RSA withdrawal to the star tracing task functioned as a protective factor, in that, regardless of the sleep context children who expressed withdrawal performed better on the decision speed test than their counterparts who exhibited RSA augmentation. This protective function was observed in relation to multiple sleep parameters (actigraphyically-derived sleep efficiency, long wake episodes, and sleep minutes). Although the amount of variance accounted for by the interaction term was small, the consistency of the findings strengthens confidence in the results. In addition, the amount of variance accounted for in this study is similar to other studies that have examined interactions between sleep and vagal activity in the prediction of child adjustment (e.g., El-Sheikh, Erath, & Keller, 2007). Taken together, these findings are novel and build on the literature by identifying vagal withdrawal as a protective factor for cognitive functioning in the context of sleep disruptions.

Longitudinal analyses revealed that vagal tone and reactivity moderated relations between children's sleep problems and their cognitive functioning as indexed by multiple WJ III test scores. In 10 out of the 18 models examined, vagal activity was a significant moderator; RSA-R was the most robust moderating vagal index (8 of the 10 significant models examined were moderated by RSA-R). Although the overall pattern of effects indicates that children who

exhibit RSA withdrawal, and to a lesser extent higher resting RSA, perform better than those who show RSA augmentation or exhibit lower resting RSA in the context of poor sleep, the pattern of effects is not clear when sleep is more optimal. The fact that RSA-R emerged as a more robust moderator of relations between children's sleep and their cognitive functioning is similar to previous studies examining these moderation effects in relation to children's cognitive performance (manuscript 1) and psychological adjustment (El-Sheikh, Erath, & Keller, 2007). Overall, findings highlight the importance of individual differences in physiological functioning and the aggregation of risk across physiological and biological domains.

Horne (1988, 1993) was the first to suggest sleep impacted cognitive functions, as his lab experiments indicated novel language and creativity were significantly impaired by sleep loss. This notion was extended by Dahl's research (1996), which suggested a direct effect of sleep on executive functions and/or the synchronization of attention and arousal to perform abstract or complex tasks. In this study we found few direct relations between objective or subjective sleep parameters and cognitive performance. However, some significant associations in the expected directions were observed. Specifically, greater sleep duration and reduced night waking were associated with better decision speed performance at T1. At T2, children's self-reported sleep problems, particularly sleepiness, was related to poor performance on the WJ III tests.

The novel contribution of this study is the examination of vagal activity indices as moderators of the link between reductions in sleep duration and quality and cognitive performance cross-sectionally and longitudinally. In the cross-sectional investigation three aspects of vagal functioning were examined as moderators of effects: vagal tone, reactivity, and recovery. Results indicate that both objective sleep duration and quality measures interacted with RSA-R to predict children's decision speed. That is, regardless of the sleep context children who exhibited vagal withdrawal performed had better decision speed scores than their counterparts. For children who exhibited vagal augmentation to the challenge, the pattern was

unexpected in that under optimal sleep conditions decision speed scores were lower than when sleep quality was less optimal. In regards to sleep duration, children who showed RSA augmentation performed similarly on the decision speed test regardless of the amount of sleep they received. No other cross-sectional moderation patterns emerged with any other WJ III tests or vagal activity (RSA, RSA recovery).

While the pattern of effect for the longitudinal findings varied based on the sleep parameter, the vagal activity index, and the cognitive performance domain, in 7 of the 10 significant moderation models it was more optimal vagal functioning (RSA withdrawal and to a lesser extent high RSA) that functioned as a protective factor in the context of poor sleep. For example, in all three significant models involving sleepiness, children who showed RSA withdrawal to the task performed similarly on the three tests (i.e., concept formation, auditory working memory, numbers reversed) regardless of the level of reported sleepiness. On the other hand, children who exhibited RSA augmentation had a significant negative association between sleepiness and cognitive performance, indicating an aggregation of risk. That is, the pattern suggests that children who have poor sleep in conjunction with less optimal vagal reactivity (augmentation or lower levels of vagal withdrawal) are the ones most at risk for poorer cognitive performance two years later, particularly tasks involving working memory. Of note, there were three models in which this pattern of effects were not observed and it is not evident why this is the case. Thus, although there were some consistent results supportive of vagal withdrawal as a protective factor in the context of poor sleep, findings should be viewed as tentative pending replication. Overall, results are somewhat consistent with the few studies that have examined interactions between sleep and vagal activity in the prediction of healthy children's adjustment; children at most risk were those who had poor sleep and less optimal vagal functioning (manuscript 1; El-Sheikh et al., 2007).

Children with less optimal vagal functioning (i.e., low RSA and/or RSA augmentation) may have difficulty obtaining sufficient, good quality sleep (Dahl, 1996; El-Sheikh, Erath, &

Keller, 2007). There were no direct relations found with this sample between the objective or self-reported sleep and RSA parameters at T1, however RSA-R at T2 was cross-sectionally related to all actigraphy-based sleep parameters and self-reported sleepiness. Specifically, RSA-R was negatively associated with sleep minutes and sleep efficiency and positively associated with long-wake episodes and child-reported sleepiness. The cross-sectional relations are consistent with results from El-Sheikh and Buckhalt's (2005) indicating multiple actigraphy-based and subjective measures of sleep to be related with children's vagal withdrawal during a continuous performance task, with better sleep associated with increased vagal withdrawal. It is unclear as to why these direct effects were observed at T2 but not T1. Sample characteristics (i.e., children in El-Sheikh & Buckhalt's study were older than those at T1) or the study design (i.e., parent-reported vs. child-reported sleep problems) may have contributed to the discrepant findings. However, it is not unusual that vagal activity measures are not directly related to the study outcome variables (e.g., internalizing and externalizing behaviors), but rather function as moderators of the associations (e.g., El-Sheikh et al., 2007; El-Sheikh & Whitson, 2006; Leary & Katz, 2004). Further, the normative sample used in this study may have contributed to the lack of association among sleep and vagal indices. Children with clinically diagnosed sleep or vagal functioning problems may show stronger direct correlations between the two biophysiological systems (Hsieh, Chen, McAfee, & Kifle, 2008; Marzec, Edwards, Sagher, Fromes, & Malow, 2003).

Consistent with Polyvagal Theory (Porges, 1998) propositions that vagal functioning impacts arousal, affect and attention (Porges, 2007), increased RSA withdrawal and to a lesser extent higher baseline levels of RSA served a protective function in the link between sleep problems and cognitive functioning. According to the theory, an adaptive physiological reaction to stress (e.g., sleep disruptions), is considered to be a disengagement of the vagal break (i.e., vagal withdrawal), which suppresses the PNS's influence on the heart and increases heart rate. This response allows one to become focused on the task at hand, obtain information, and/or

reduce the challenge by engaging social strategies that regulate emotions. After the challenge has subsided, the PNS influence can reengage to reduce arousal. The ability to engage and disengage as environmental demands change and to respond to metabolic demands, including increased attention and information processing (Suess, Porges, & Plude, 1994) are processes vital for adaptive behavior.

A novel aspect of this study was the inclusion of vagal recovery as a possible moderator in the pathway between children's sleep disruptions and cognitive performance during the fifth grade. Vagal recovery was not a moderator in this study; however we were only able to examine this index of vagal functioning cross-sectionally at T2. In the current study, 45% of children had RSA levels during the post-task baseline that were lower than those during the initial baseline; 16% fully recovered (the difference between the initial and post-task baselines was zero); and 38% had RSA levels higher during the post-task than the initial baseline. Although there was variation in the amount of recovery, it is important to note that vagal recovery can be computed in a number of ways (e.g., amount of time before baseline levels are reached, percentage of recovery that has occurred within a specified timeframe) and it is possible that findings could differ based on how the variable was computed. Future research is encouraged to incorporate multiple calculations of vagal recovery into studies investigating physiological activity.

Similar to the first manuscript of this dissertation, vagal markers did not interact with sleep parameters to predict children's reaction time mean or the variability in response time. Considering the importance of attention and vigilance needed to perform well on the reaction time task, and that vagal activity is a marker of attention (Porges, 2007) and vigilance is hard to obtain when sleepy (Dahl, 1996), this finding was unexpected. The study design may have contributed to the results (e.g., no incentives for performance were given), thus caution should be exercised to not accept the null hypotheses.

Several of the moderation effects were hard to interpret and inconsistent with expectations. Specifically, and consistent with the cross-sectional findings from manuscript 1,

three of the ten longitudinal plots revealed that children who exhibited higher levels of vagal withdrawal performed significantly better on several cognitive assessments when sleep was poor than when it was more optimal. While findings supportive of our hypotheses suggest that physiological regulation may play an important role in understanding how sleep impacts children's cognitive functioning, we only examined one component ANS activity. The second branch, the sympathetic nervous system (SNS), has also been linked to sleep (El-Sheikh & Arsiwalla, 2011) and interactions between the PNS and SNS have additive and interactive effects in the prediction of child adaptation (e.g., El-Sheikh, Kouros, Erath, Cummings, Keller, & Staton, 2009). Therefore, for a more thorough explication of research questions, it may be fruitful for future studies to examine both branches of the ANS in relation to sleep and cognitive functioning.

The results must be interpreted in relation to the age of the children. Cross-sectional findings when children were in the third grade (manuscript 1) were more robust than when the children were in the fifth grade. It is plausible that the interaction between sleep and vagal functioning may be more important at an early age when knowledge is being acquired than later on in development when the foundation of knowledge has been formed; it is also plausible that this association would be more apparent for adolescents who experience a higher rate of sleep problems (El-Sheikh, 2011). Thus, examination of our research questions with children across various developmental ages is imperative. Further, the community sample in conjunction with the exclusion/inclusion criteria (e.g., two-parent home, lack of children with mental retardation, developmental delays, ADHD, chronic illnesses, and diagnosed sleep problems) did not allow for understanding the research questions in the context of more severe or clinically significant problems.

Although we had a thorough assessment of sleep (actigraphy and self-reports; Sadeh, 2008) we did not take into account daytime naps nor did we have the ability to assess sleep staging. Sleeping during the day may compensate for sleep problems at night and it is

recommended that future research includes napping in the calculation of total sleep duration to further delineate the associations between sleep and cognition. An important next step in the field is the investigation of sleep staging through polysomnography (PSG) and its association with physiological activity and cognitive functioning. Adult research has implicated slow-wave sleep in declarative memory consolidation and REM sleep in the consolidation of procedural memory (Hornung, Danker-Hopfe, Schredl, & Heuser, 2007). Thus, the integration of PSG in child development studies is warranted.

In summary, we found cross-sectional and longitudinal support for interactions between biological (i.e., sleep) and physiological (i.e., RSA) regulatory systems in predicting children's cognitive performance with a large normative sample of school-aged children. Results extend the sleep-cognitive literature by (1) suggesting PNS indicators, primarily RSA-R, are important individual difference variables to be considered when assessing the role of sleep on child cognitive functioning; (2) including both actigraphy and self-reported assessments of sleep given the differential outcomes with the various sleep parameters; (3) assessing the moderating role of vagal indices both cross-sectionally and longitudinally in the link between sleep and cognitive functioning; and (4) examining multiple aspects of executive functioning using standardized tests and controlling for autoregressive effects. The moderation results consistent with hypotheses are strengthened by the multifaceted nature of the study. Results suggest that children who exhibit poor physiological regulation (i.e., low baseline RSA or RSA augmentation) may be at an increased risk for cognitive functioning problems otherwise associated with normative levels of poor sleep.

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Table 1. Means and standard deviations of study variables at T1.

Variable Name	*M*	*SD*
1. Sleep Minutes	7hrs:21min	0:59min
2. Sleep Efficiency	86.78	7.94
3. Long Wake Minutes	3.92	2.20
4. Sleep/Wake Problems	17.04	4.95
5. Sleepiness Scale	14.11	4.50
6. RSA	.13	.06
7. RSA-R	-.02	.05
8. Concept Formation WJ III	99.32	12.67
9. Numbers Reversed WJ III	104.03	15.92
10. Auditory Working Memory WJ III	112.01	13.08
11. Decision Speed WJ III	107.65	13.12
12. Reaction Time *M* Donders (ms)	464.74	75.44
13. Reaction Time *SD* Donders (ms)	140.11	37.16

Note: RSA = baseline vagal tone; RSA-R = vagal reactivity; Woodcock-Johnson III; *M* = mean; *SD* = standard deviation; ms = milliseconds.

Table 2. Means and standard deviations of study variables at T2.

Variable Name	*M*	*SD*
1. Sleep Minutes	7hrs:25min	0:47min
2. Sleep Efficiency	89.12	9.34
3. Long Wake Minutes	3.09	2.22
4. Sleep/Wake Problems	15.93	4.67
5. Sleepiness Scale	12.63	3.53
6. RSA	.14	.07
7. RSA-R	-.02	.07
8. Concept Formation WJ III	100.46	12.94
9. Numbers Reversed WJ III	105.81	15.20
10. Auditory Working Memory WJ III	106.24	12.57
11. Decision Speed WJ III	108.73	14.17
12. Reaction Time *M* Donders (ms)	124.14	39.93
13. Reaction Time *SD* Donders (ms)	414.80	70.36

Note: RSA = baseline vagal tone; RSA-R = vagal reactivity; Woodcock-Johnson III; *M* = mean; *SD* = standard deviation; ms = milliseconds.

Table 3. Correlations among study variables.

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.
1. Age T1	-													
2. Gender; 1 = Boys, 0 = Girls	-.02	-												
3. Ethnicity; 1 = AA, 0 = EA	-.10	-.08	-											
4. Family Socioeconomic Status T1	.18*	.05	-.24**	-										
5. Body Mass Index T1	-.11	-.09	.07	-.13	-									
6. Sleep Efficiency T1 [a]	.17*	-.20	.09	.10	.04	-								
7. Long Wake Episodes T1 [a]	-.19*	.18*	-.19*	-.04	.11	-.85**	-							
8. Sleep Minutes T1 [a]	.18*	-.24**	-.09	.20**	-.21**	.83**	-.61**	-						
9. Sleepiness T1 [b]	-.10	.15*	.16*	-.10	-.01	-.07	.01	.08	-					
10. Sleep/Wake Problems T1 [b]	.09	-.06	.25**	-.16*	.06	-.07	.02	-.12	.43**	-				
11. RSA T1	-.08	.07	.05	-.03	-.24**	.03	-.05	.09	.04	.10	-			
12. RSA-R T1	.16	.05	.04	-.02	.04	-.04	-.02	-.08	-.03	.02	-.13	-		
13. Concept Formation T1 [c]	.20**	-.03	-.19*	.25**	-.03	-.04	.04	.00	-.05	-.08	-.02	-.01	-	
14. Numbers Reversed T1 [c]	.20**	.01	-.15*	.15*	-.07	.02	-.07	.03	-.13	-.16*	.07	-.03	.37**	-
15. Auditory Working Memory T1 [c]	-.26**	.06	-.11	.23**	-.10	.10	-.12	.11	-.16*	-.18*	-.08	.06	.33**	.38**
16. Decision Speed T1 [c]	.12	-.17*	-.01	.20**	-.05	.13	-.15*	.16*	-.03	.02	-.11	-.06	.24**	.20**
17. Reaction Time Mean T1 [d]	-.24**	-.31**	.07	-.11	.13	.01	-.04	.04	-.01	.12	.01	.01	-.21**	-.24**
18. Reaction Time *SD* T1 [d]	-.24**	-.10	.08	-.13	.08	-.04	-.02	-.04	.05	.17*	.05	.09	-.28**	-.28**
19. Age T2	.69**	-.08	-.16	.17	-.06	.00	-.00	-.03	-.12	.02	-.08	.07	.08	.10
20. Family Socioeconomic Status T2	.11	.06	-.25**	.99***	-.18*	.11	-.04	.24**	.02	-.10	.05	-.01	.31**	.10
21. Body Mass Index T2	-.05	.03	.02	-.12	.83**	-.27**	.18*	-.25**	-.05	.05	-.26**	.02	-.01	-.07
22. Sleep Efficiency T2 [a]	-.01	-.14	.04	.05	-.12	.40**	-.42**	.34**	.05	-.03	.05	.04	.09	.22**
23. Long Wake Episodes T2 [a]	-.05	.02	-.05	-.13	.09	-.54**	.55**	-.42**	.01	.09	-.13	.03	.01	-.09
24. Sleep Minutes T1 [a]	.02	-.17	-.21*	.23**	-.16	.46**	-.39	.57**	.05	-.01	.11	-.05	.05	.06
25. Sleepiness T2 [b]	.01	.14	.20*	-.01	.04	.12	-.13	.07	.20*	.21*	-.05	.02	-.31**	-.16
26. Sleep/Wake Problems T2 [b]	-.06	.07	.01	-.15	.08	-.09	.03	-.12	.20*	.43**	.11	-.08	-.12	-.13
27. RSA T2	-.09	.12	-.03	.00	-.18	-.04	.05	.02	.03	.12	.36**	-.18	-.08	.11
28. RSA-R T2	.06	-.02	.15	-.07	.12	-.09	-.04	-.18	.15	.16	-.11	.09	-.12	-.16
29. Concept Formation T2 [c]	.10	.04	-.31**	.22*	-.17	.05	.06	.15	-.21*	-.29**	-.02	.10	.64**	.39**
30. Numbers Reversed T2 [c]	-.01	-.01	-.06	.09	-.11	.07	-.07	.13	-.10	.07	.18*	.07	.31**	.54**
31. Auditory Working Memory T2 [c]	.01	.06	-.10	.05	-.04	-.04	.05	.01	-.22*	-.13	.14	-.01	.37**	.40**
32. Decision Speed T2 [c]	-.03	-.16	-.03	.08	-.17	.08	-.09	.10	-.07	-.16	-.04	.01	.17	.15
33. Reaction Time Mean T2 [d]	-.02	-.26**	.01	-.08	.01	-.04	.02	-.03	.02	.22*	-.13	-.01	-.23	-.15
34. Reaction Time *SD* T2 [d]	-.01	-.43**	.03	-.17	.03	.02	-.01	.03	.01	.24*	-.12	.03	-.10	-.15

Table 3 continued.

	15.	16.	17.	18.	19.	20.	21.	22.	23.	24.	25.	26.	27.	28.
1. Age T1														
2. Gender; 1 = Boys, 0 = Girls														
3. Ethnicity; 1 = AA, 0 = EA														
4. Family Socioeconomic Status T1														
5. Body Mass Index T1														
6. Sleep Efficiency T1 [a]														
7. Long Wake Episodes T1 [a]														
8. Sleep Minutes T1 [a]														
9. Sleepiness T1 [b]														
10. Sleep/Wake Problems T1 [b]														
11. RSA T1														
12. RSA-R T1														
13. Concept Formation T1 [c]														
14. Numbers Reversed T1 [c]														
15. Auditory Working Memory T1 [c]	-													
16. Decision Speed T1 [c]	.21**	-												
17. Reaction Time Mean T1 [d]	-.24**	-.23**	-											
18. Reaction Time *SD* T1 [d]	-.20*	-.20*	.72**	-										
19. Age T2	.08	.19*	-.09	-.10	-									
20. Family Socioeconomic Status T2	.24**	.30**	-.15	-.21*	.18*	-								
21. Body Mass Index T2	-.08	-.18*	.14	.07	-.05	-.14	-							
22. Sleep Efficiency T2 [a]	.10	.09	.17	.10	-.02	.06	-.13	-						
23. Long Wake Episodes T2 [a]	-.11	.03	-.14	.00	.01	-.14	.10	-.63**	-					
24. Sleep Minutes T2 [a]	.14	.14	.18	.01	-.11	.23*	-.19*	.45**	-.60**	-				
25. Sleepiness T2 [b]	-.19*	.01	-.04	.01	-.04	-.03	.01	.06	-.03	.02	-			
26. Sleep/Wake Problems T2 [b]	-.18*	-.07	-.04	.09	-.02	-.14	.05	-.03	.08	-.13	.48**	-		
27. RSA T2	-.16	-.09	.05	.15	-.05	-.00	-.22*	.08	-.12	.08	.04	.06	-	
28. RSA-R T2	-.29**	.01	.02	-.01	.08	-.06	.13	-.23*	.20*	-.26**	.26**	.13	-.30**	-
29. Concept Formation T2 [c]	.35**	.21*	-.25**	-.31**	.13	.23*	-.12	.10	.00	-.01	-.31**	-.21*	-.13	-.15
30. Numbers Reversed T2 [c]	.38**	.14	-.33**	-.31**	-.01	.09	-.01	.07	-.00	-.02	-.20*	-.15	-.01	.08
31. Auditory Working Memory T2 [c]	.40**	.03	-.21**	-.23**	-.01	.06	.04	.01	-.00	.01	-.21*	-.17	-.10	.06
32. Decision Speed T2 [c]	.10	.50**	-.10	-.13	-.14	.09	-.12	-.02	.07	.09	-.01*	-.14	-.14	.10
33. Reaction Time Mean T2 [d]	-.09	-.15	.44**	.48**	-.06	-.09	-.10	.08	.05	.05	-.03	.02	-.03	.04
34. Reaction Time *SD* T2 [d]	-.21*	-.16	.62**	.47**	-.08	-.17	-.04	.16	-.06	.10	-.01	.04	.50	.02

Table 3 continued.

	29.	30.	31.	32.	33.	34.
1. Age T1						
2. Gender; 1 = Boys, 0 = Girls						
3. Ethnicity; 1 = AA, 0 = EA						
4. Family Socioeconomic Status T1						
5. Body Mass Index T1						
6. Sleep Efficiency T1 [a]						
7. Long Wake Episodes T1 [a]						
8. Sleep Minutes T1 [a]						
9. Sleepiness T1 [b]						
10. Sleep/Wake Problems T1 [b]						
11. RSA T1						
12. RSA-R T1						
13. Concept Formation T1 [c]						
14. Numbers Reversed T1 [c]						
15. Auditory Working Memory T1 [c]						
16. Decision Speed T1 [c]						
17. Reaction Time Mean T1 [d]						
18. Reaction Time *SD* T1 [d]						
19. Age T2						
20. Family Socioeconomic Status T2						
21. Body Mass Index T2						
22. Sleep Efficiency T2 [a]						
23. Long Wake Episodes T2 [a]						
24. Sleep Minutes T1 [a]						
25. Sleepiness T2 [b]						
26. Sleep/Wake Problems T2 [b]						
27. RSA T2						
28. RSA-R T2						
29. Concept Formation T2 [c]	-					
30. Numbers Reversed T2 [c]	.27**	-				
31. Auditory Working Memory T2 [c]	.40**	.56**	-			
32. Decision Speed T2 [c]	.13	.17	.13	-		
33. Reaction Time Mean T2 [d]	-.37**	-.16	-.08	-.05	-	
34. Reaction Time *SD* T2 [d]	-.22*	-.22*	-.10	-.10	.75**	-

Note: T1 refers to cross-sectional analyses during the first wave of data collection; T2 refers to cross-sectional during the second wave of data collection. [a] = actigraphy; [b] = self-reported; [c] = Woodcock Johnson III; [d] = Donders; RSA = Respiratory Sinus Arrhythmia; RSA-R = RSA reactivity. RSA recovery was not found to be related to any of the study variables and thus was not included in the table.
*p < .05; **p < .01; ***p < .001.

Figure 1.

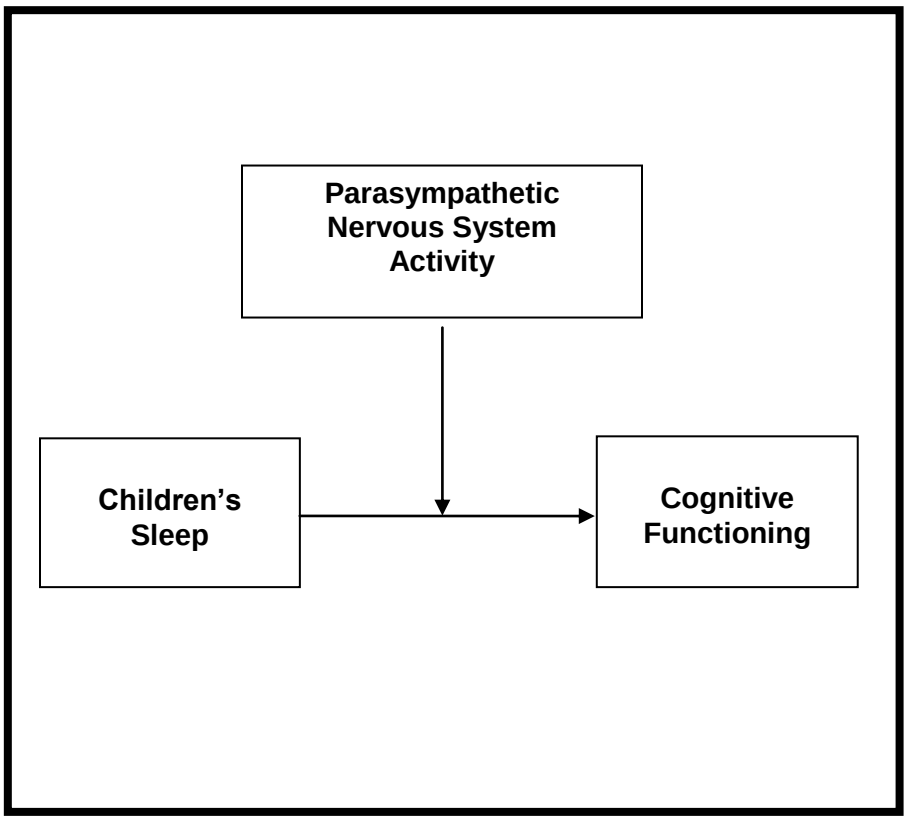


Figure 1. Conceptual model depicting children's parasympathetic nervous system activity as a moderator of the association between children's sleep and their cognitive functioning.

Figure 2.

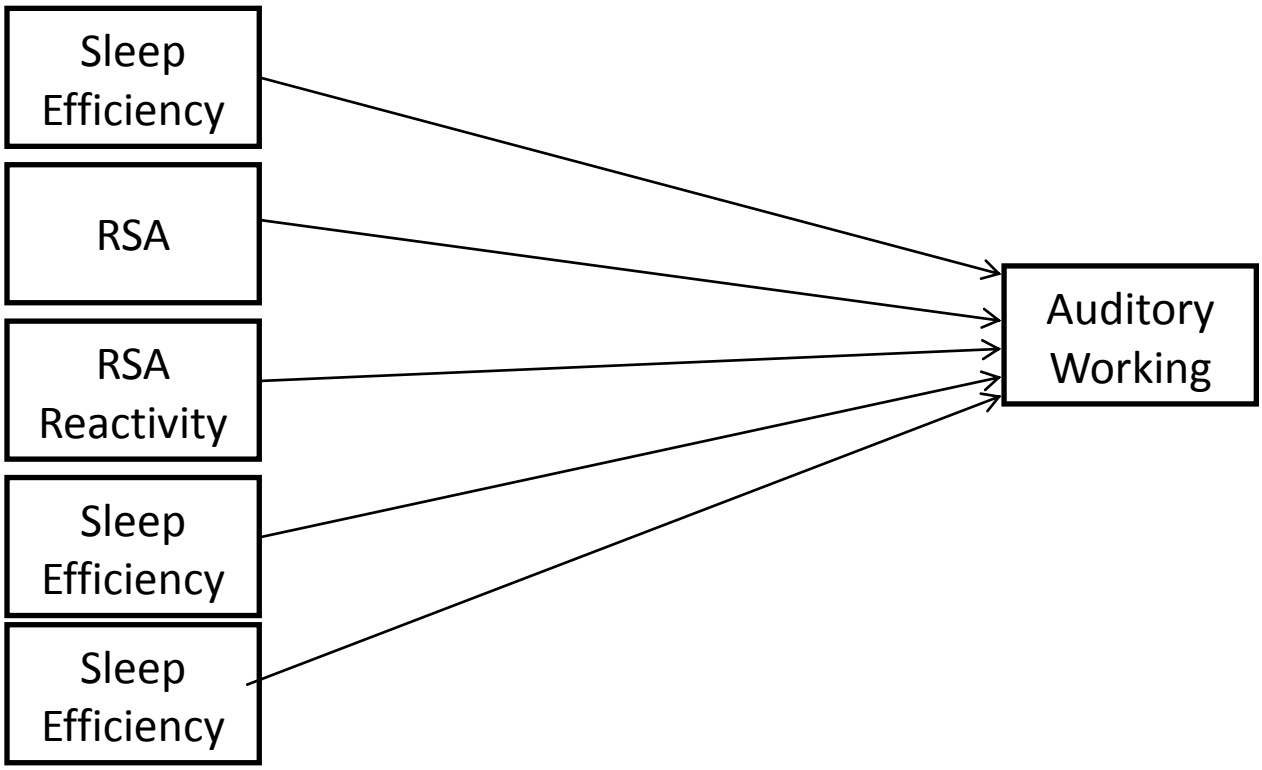
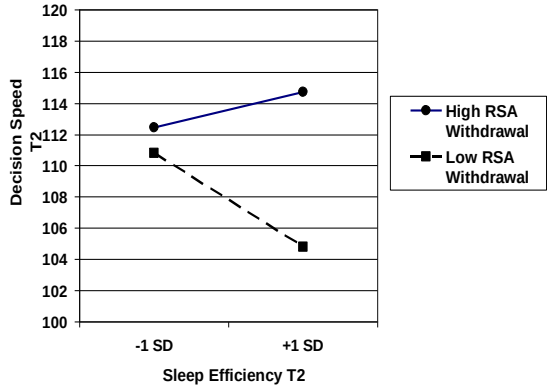


Figure 2. An example of the path models that examined the possible moderating role of RSA and RSA-R in the cross-sectional link sleep-cognitive functioning link. Of note, control variables are not included in the model above however all controls (sex, race/ethnicity, age, body mass index, and season of watch wear) were entered as the last paths in the model. Longitudinal models also included the pertinent T1 cognitive outcome (autoregressive effects).

Figure 3.

A.



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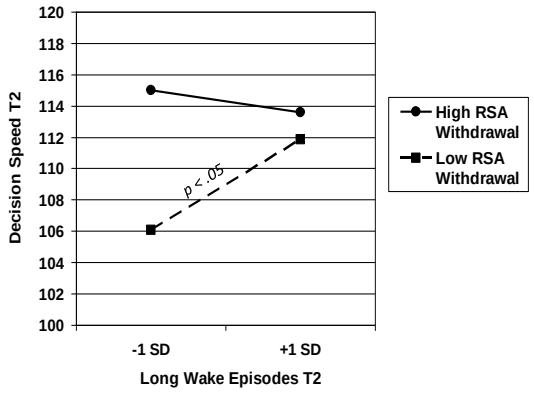


C.

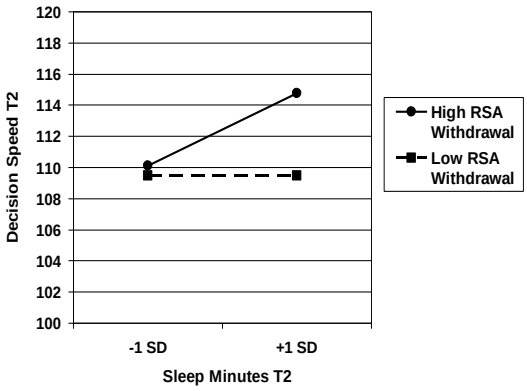
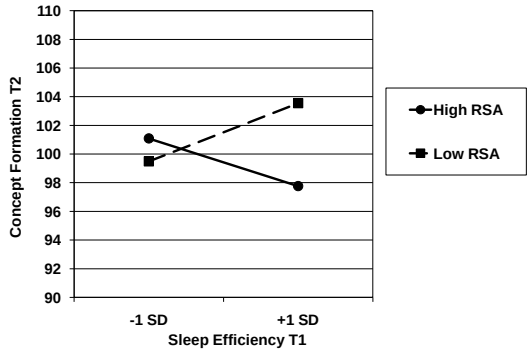


Figure 3. (A) Cross-sectional interaction between Sleep Efficiency and RSA-R in the prediction of children's Decision Speed. (B) Cross-sectional interaction between Long Wake Episodes and RSA-R in the prediction of children's Decision Speed. (C) Cross-sectional interaction between Sleep Minutes and RSA-R in the prediction of children's Decision Speed.

Figure 4.

A.



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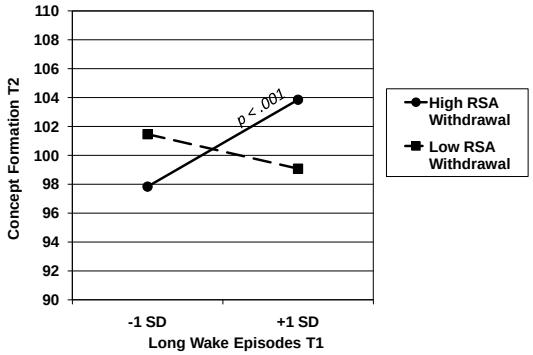

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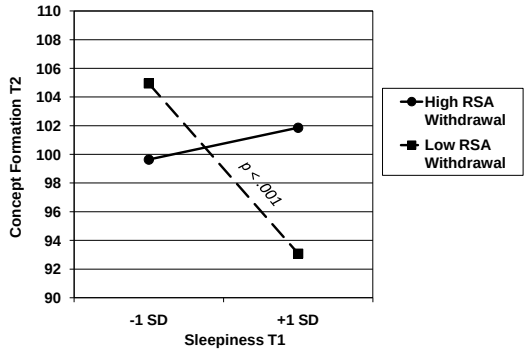
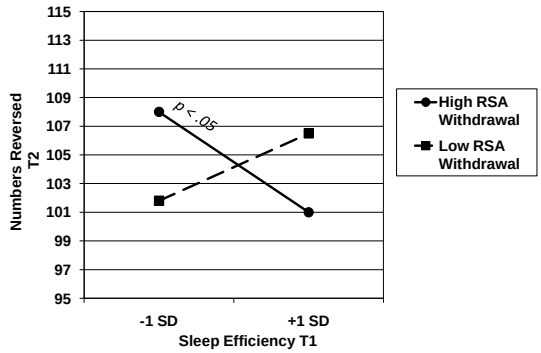


Figure 4. (A) Longitudinal interaction between Sleep Efficiency and RSA-R in the prediction of children's Concept Formation. (B) Longitudinal interaction between Long Wake Episodes and RSA-R in the prediction of children's Concept Formation. (C) Longitudinal interaction between Sleepiness and RSA-R in the prediction of children's Concept Formation.

Figure 5.

A.



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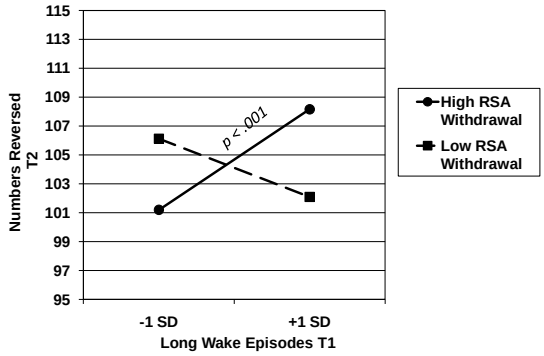
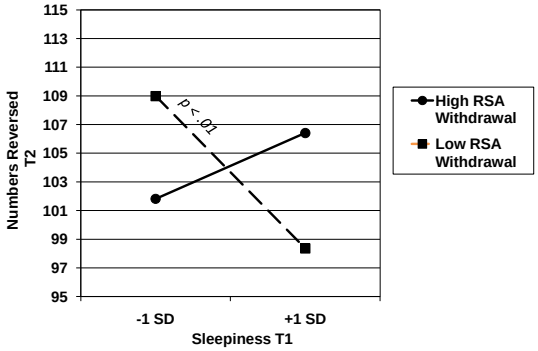


C.



D.

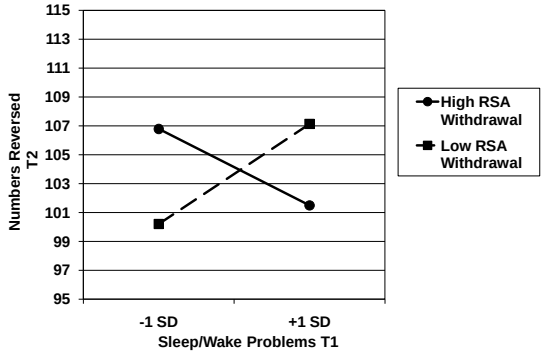
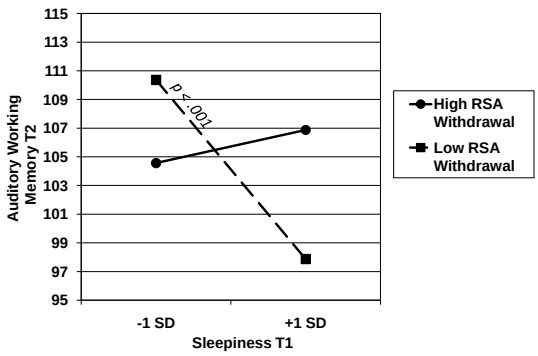


Figure 5. (A) Longitudinal interaction between Sleep Efficiency and RSA-R in the prediction of children's Numbers Reversed. (B) Longitudinal interaction between Long Wake Episodes and RSA-R in the prediction of children's Numbers Reversed. (C) Longitudinal interaction between Sleepiness and RSA-R in the prediction of children's Numbers Reversed. (D) Longitudinal interaction between Sleep/Wake Problems and RSA-R in the prediction of children's Numbers Reversed.

Figure 6.

A.



B.

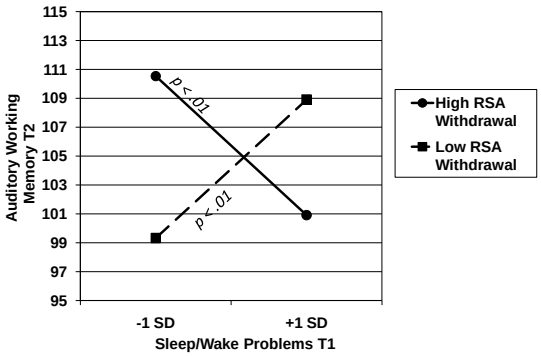


Figure 6. (A) Longitudinal interaction between Sleepiness and RSA-R in the prediction of children's Auditory Working Memory. (B) Longitudinal interaction between Sleep/Wake Problems and RSA-R in the prediction of children's Auditory Working Memory.

Figure 7.

A.

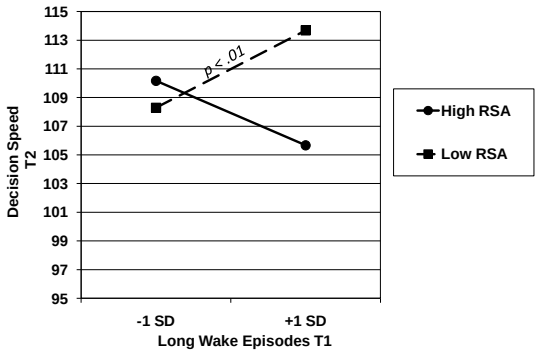


Figure 3. (A) Longitudinal interaction between Long Wake Episodes and RSA in the prediction of children's Decision Speed.