

GENERAL SLEEP PARAMETERS AND SLEEP ARCHITECTURE IN CHILDREN  
WITH ATTENTION-DEFICIT/HYPERACTIVITY DISORDER AND THEIR  
TYPICALLY DEVELOPING PEERS

by

Andre Benoit

Submitted in partial fulfilment of the requirements  
for the degree of Master of Science

at

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**DALHOUSIE UNIVERSITY**

DEPARTMENT OF PSYCHOLOGY

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Dated: August 7<sup>th</sup>, 2012

Supervisor: \_\_\_\_\_

Readers: \_\_\_\_\_

\_\_\_\_\_

Department Representative: \_\_\_\_\_

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## **DEDICATION PAGE**

This thesis is dedicated to my family, friends and all who have supported me throughout the completion of my Masters thesis.

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## ABSTRACT

Polysomnography (PSG) sleep studies that compare the sleep of children with attention-deficit/hyperactivity disorder (ADHD) to their typically developing (TD) peers have historically been highly inconsistent. Not only has there been sporadic control over potential confounding variables (e.g., age, medication-status), but no studies have compared sleep between ADHD subtypes. Therefore, this thesis compared the sleep parameters (sleep onset, duration) and sleep architecture (e.g., REM latency, % of REM and NREM sleep]) between a medication-naïve sample of 25 children diagnosed with ADHD, to an age- and sex-matched sample of 25 of their TD peers. Statistical analyses revealed that the ADHD group took longer to fall asleep and slept less than the TD group. However, no significant sleep architecture differences were found between the ADHD and TD groups, or between the ADHD subtypes. Results suggest that ADHD does not relate to intrinsic differences in sleep architecture in children.



## LIST OF ABBREVIATIONS USED

ACC	Anterior Cingulate Cortex
ADD	Attention-deficit disorder
ADHD	Attention-deficit/hyperactivity disorder
ADHD-C	Attention-deficit/hyperactivity disorder- combined type
ADHD-HI	Attention-deficit/hyperactivity disorder- hyperactive/impulsive type
ADHD-IA	Attention-deficit/hyperactivity disorder- inattentive type
APA	American Psychiatric Association
ASD	Autism Spectrum Disorder
CBT	Cognitive Behavioural Therapy
CNS	Central Nervous System
CV	Covariate
CPT	Continuous Performance Task
CRS	Conners Rating Scales
CSHQ	Children's Sleep Habits Questionnaire
DA	Dopamine
DLPFC	Dorsal lateral prefrontal cortex
DSM	Diagnostic and Statistical Manual
DV	Dependent Variable
EEG	Electroencephalogram
EF	Executive Function
EMG	Electromyogram
EOG	Electrooculography
GAD	Generalized Anxiety Disorder
IV	Independent Variable
LC	Locus Coeruleus
LD	Learning Disability
MANOVA	Multiple analyses of variance
MDD	Major Depressive Disorder
MPH	Methylphenidate
NE	Norepinephrine
NREM	Non rapid eye movement
OSA	Obstructive Sleep Apnea
PFC	Prefrontal Cortex
PLMS	Periodic Limb Movement Syndrome
PSG	Polysomnography
QEII	Queen Elizabeth Health Science Centre
REM	Rapid Eye Movement
RLS	Restless Leg Syndrome
SCN	Suprachiasmatic nucleus
SCT	Sluggish Cognitive Tempo
SES	Socioeconomic Status
SOL	Sleep Onset Latency
SSRI	Selective Serotonin Reuptake Inhibitor
TD	Typically developing children

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# CHAPTER 1 INTRODUCTION

## 1.1 Chapter Overview

This chapter begins with a historical overview of attention-deficit/hyperactivity disorder (ADHD); namely its development from a disorder of hyperactivity, to its current classification with two symptoms clusters; hyperactive/impulsive and inattentive. Diagnostic techniques and evidence-based treatments for ADHD (e.g., stimulant medications) are then reviewed, followed by a discussion of theoretical models and neurobiology of ADHD. Second, information pertaining to sleep, sleep stages, and sleep architecture is presented. Key sleep measurement techniques are discussed, with a particular focus on polysomnography (PSG). Next, both the circadian and homeostatic regulation of sleep, and the key factors which can impact children's general sleep parameters and sleep architecture (e.g., age, medications) are discussed.

Third, in the subsection entitled ADHD and Sleep, a literature review and critical analysis of empirical PSG sleep studies which have examined general sleep parameters and sleep architecture between children with ADHD and their typically developing (TD) peers are presented. In this section several key findings are highlighted that contribute to the varied results of this literature. Lastly, a research introduction (i.e., overview, hypotheses) is presented.

## 1.2 ADHD

Attention-deficit/hyperactivity disorder (ADHD) is a highly prevalent neurodevelopmental disorder, which affects roughly 5 to 10% of school-age children (Diagnostic and Statistical Manual- Fourth Edition, [DSM-IV-TR], APA, 2000). Children diagnosed with ADHD display age-inappropriate symptoms of hyperactivity, impulsivity

and/or inattention, which negatively impact their overall psychosocial functioning (APA, 2000). Although once believed to primarily be a disorder of childhood, ADHD symptoms persist into adulthood in roughly 60% of cases (APA, 2000). ADHD is more often diagnosed in boys than girls, with estimates of ADHD diagnoses in boys versus girls varying from roughly 3:1 to 9:1 (e.g., Brown et al., 2001). However, Reid et al. (2000) notes that boys are over-diagnosed while girls are under-diagnosed due to several key factors, including: a) that behaviours used to define the symptomology of ADHD in the DSM-IV were developed from a sample predominately composed of males; and b) there is a potential bias for teachers to more easily recognize hyperactive symptoms, which are more often displayed by boys compared to girls.

A wealth of research has focused on ADHD with the intent of gaining a clear understanding of: (1) the key symptoms, impairments, and comorbidities, (2) how ADHD should be diagnosed and treated, as well as (3) conceptual models of ADHD. Presented below is an overview of these key topics.

### 1.2.1 *Historical Overview of ADHD.*

ADHD is certainly not a novel disorder within psychology, education, or medicine. In some form, symptoms of inattention, and/or hyperactivity/impulsivity have been well documented in children since the early 1900's. For a comprehensive account of the historical development of ADHD, see reviews by Barkley (2006) or Conners (2000). The first individual to document "ADHD-like" symptoms in children was a British physician, Sir George Still in 1902. In a series of lectures, Still described a group of twenty children who showed marked deficits in "volitional control and attention" (Still, 1908; as cited in Conners, 2000). Still described a syndrome associated with the core

symptoms of “passionateness”; “over-activity”, and/or an “intense impulsivity regarding some immediate goal...”, which was roughly three times more prevalent in boys than girls (Conners, 2000).

Researchers in the early 1920’s, following the worldwide outbreak of encephalitis, noted “ADHD-like” symptoms in numerous children who had suffered organic brain injuries following infection, or other traumas (e.g., Barkley, 2006). For example, children diagnosed with “Postencephalitic behavior disorder” (e.g., Ebaugh, 1923; as cited in Barkley, 2006) displayed symptoms of over-activity, attentional dysregulation, social disruptiveness, and impulsivity. Throughout the early 1920’s and well into the late 1950’s there were clinical descriptions of children that displayed symptoms of hyperactivity, over-activity, and restlessness (e.g., Childers, 1935; as cited in Barkley, 2006). Initially, the prevailing explanations for these children’s “ADHD-like” hyperactive symptoms were some type of brain damage (e.g., infection, birth trauma, head injury, frontal lobe lesions) (Blau, 1936, cited in Barkley 2006).

In 1957, Strauss and Lehtinen posited that the “ADHD-like” hyperactivity demonstrated by children was not necessarily due to neurological issues. Specifically, Strauss and Lehtinen (1957; as cited in Barkley, 2006) described the concept of the “brain-injured child”, who, “demonstrated a variety of hyperactive behaviours, [which appeared], despite insufficient or no evidence of brain pathology”. This research ultimately led to the conceptualization of a disorder known as “hyperactivity syndrome” (e.g., Chess, 1960; Laufer & Denhoff, 1957, as cited in Barkley, 2006). The defining features of hyperactivity syndrome were symptoms of motor over-activity. For example, Chess (1960) described symptoms of impulsivity, aggression, and short attention span, in

a sample of 36 children diagnosed with “physiological hyperactivity” (as cited in Barkley, 2006).

In 1968, the second edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-II; American Psychiatric Association, 1968), first described an “ADHD-like” disorder of childhood, “Hyperkinetic Reaction of Childhood” (DSM-II, APA, 1968). Although no specific symptom list was outlined, Hyperkinetic Reaction of Childhood was characterized by “over-activity, restlessness, distractibility, short attention span, especially in young children; [where] the behavior usually diminished in adolescence; and did not have to be related to an organic brain injury” (DSM-II, APA, 1968). The inclusion of the Hyperkinetic Reaction of Childhood in the DSM-II represented the consolidation of roughly 50 years of research that examined hyperactivity and its relation to impaired behavioral functioning in children.

Pivotal to our current conceptualization of ADHD (as a disorder with both hyperactive/impulsive and inattentive symptoms) has been the work of Canadian psychologist Virginia Douglas, and her colleagues at McGill University. By conducting research that employed a myriad of objective measures of behavioral and cognitive performance, Douglas and her colleagues provided strong evidence that the behavioral impairments presented by children were not exclusively due to the result of their hyperactive symptoms (e.g., Douglas, 1972; as cited in Barkley, 2006). Specifically, Douglas demonstrated that poor sustained attention, as well as poor impulse control, were more likely than hyperactivity alone to contribute to the difficulties presented by these children (e.g., Douglas, 1972; as cited in Barkley, 2006). For example, Douglas found that children who presented with symptoms of hyperactivity experienced the most

difficulty on objective measures of sustained attention (e.g., continuous performance task ([CPT]; e.g., Conners, Sitarenios, Parker, & Epstein, 1998). Furthermore, Douglas (1972) found that children with hyperactivity did not always demonstrate more distractibility than their healthy peers, and that sustained attention problems could still emerge even when no clear distractions were present (Douglas, 1972; as cited in Barkley, 2006). The major implication from these findings was that children's behavioural impairments, which were previously attributable primarily to hyperactivity, were now found to relate to issues with sustained attention. In other words, Douglas and her colleagues sparked a paradigm shift away from hyperactivity to a focus on attentional issues.

Prompted by pioneering research like that of Virginia Douglas and her colleagues, Hyperkinetic Disorder of Childhood (DSM-II, APA, 1968) was re-conceptualized as "Attention Deficit Disorder" (ADD) in the publication of the DSM-III (American Psychiatric Association, 1980). There were two subtypes; ADD-with hyperactivity and ADD-without hyperactivity. The DSM-III definition provided clinicians with more overt diagnostic criteria by including three separate symptom checklists. Specifically, in order for a child to be diagnosed with ADD, he/she had to present with at least three out of five *inattentive* symptoms (e.g., "often does not seem to listen"), along with at least three out of six *impulsive* symptoms (e.g., "often acts before thinking"). Further, for a child to be diagnosed as ADD with hyperactivity, he/she must also have presented with at least two out of five *hyperactive* symptoms (e.g., "has difficulty staying seated"). Onset of symptoms must have been before age seven; been present for at least six months, and not be better accounted for by another psychiatric condition (DSM-III, APA, 1980). Most notable in this revision was a clear shift towards attention deficits as being the core

symptoms of the disorder, with less emphasis on symptoms of hyperactivity. Interestingly the DSM-III included a symptom related to children's sleep patterns in the hyperactive symptom grouping. (i.e., "moves about excessively during sleep"). This was the first and only time that sleep was mentioned as symptom of ADHD. This symptom was removed in subsequent revisions of DSM.

The DSM-III definition of ADHD was revised in 1987 (DSM-III-R, APA, 1987) and ADD was re-conceptualized as "Attention-Deficit/Hyperactivity Disorder" (ADHD). Unlike DSM-III, the three symptom lists for inattention, impulsivity, and hyperactivity were collapsed into a single symptom list. To meet DSM-III-R diagnostic criteria for ADHD, a child would have presented with at least eight out of 14 symptoms listed.

In the most recent edition of the DSM, the DSM-IV TR (American Psychiatric Association [APA], 2000), similar to DSM-III, ADHD is reorganized into three distinct subtypes. According to DSM-IV-TR criteria, diagnoses of ADHD can be made when a child *often* experiences six or more symptoms of inattention (ADHD- Predominantly Inattentive subtype [ADHD-IA]; e.g., difficulty organizing tasks, easily distracted), six or more symptoms of hyperactivity/impulsivity (ADHD- Predominantly Hyperactive/Impulsive subtype [ADHD-HI]; e.g., fidgets), or six or more symptoms in both domains (ADHD- Combined subtype [ADHD-C]). Symptoms must persist for at least six months, be maladaptive, and be inconsistent with developmental level. Evidence of symptoms *causing impairment* must appear before the age of 7 years (Criterion B), impairment must be present in two or more settings (e.g., school and home; Criterion C), and evidence of clinically significant impairment in social, academic, or occupational functioning as a result of ADHD symptoms must be clear (Criterion D). ADHD-related



impairments in children include difficulties interacting with parents, teachers, and peers; poor emotion regulation, difficulties in daily living and adaptive skills, and poorer academic achievement (e.g., APA, 2000). Finally, ADHD symptoms should not occur exclusively during the course of a pervasive developmental disorder or psychotic disorder, or be better accounted for by another disorder (Criterion E).

The next edition of the Diagnostic and Statistical Manual (DSM-V) is scheduled for release by the APA in May, 2013. A task force of leading clinicians and researchers established by the APA has conducted quantitative research to determine if/how ADHD symptomatology and/or diagnostic criteria should be revised in the DSM-V. Most recently the APA DSM-V Task Force conducted a comprehensive literature review and meta-analyses, to determine if the DSM-IV “subtype” model of ADHD should be retained in DSM-V (e.g., Willcutt et al., 2012). Based on a meta-analysis, Willcutt and colleagues found only weak evidence for the validity of the ADHD-HI subtype, as well as minimal evidence to justify distinction between ADHD-IA and ADHD-C subtypes. Overall these authors concluded that “... [although] DSM-IV subtypes provide convenient clinical shorthand to describe the functional and behavioral correlates of current levels of inattentive and hyperactive/impulsive symptoms... [they] do not identify discrete subgroups with sufficient long-term stability to justify the classification of distinct forms of [ADHD] disorder”. The APA Task Force, suggest DSM-V include dimensional modifiers for ADHD-HI, and ADHD-IA symptoms, in lieu of DSM-IV subtypes. The dimensional modifiers (also described by APA Task Force as “Specifiers for Presentation”) proposed would primarily describe the number of inattentive and/or hyperactive symptoms present at the time of global ADHD diagnosis, rather than

segment ADHD as a disorder with distinct subtypes. These would include (1) “Combined Presentation”, “Predominately Inattentive Presentation”, and “Predominately Hyperactive-Impulsive Presentation”.

### *1.2.2 Best Practices for Diagnosis ADHD.*

ADHD is a complex neurodevelopmental disorder, which can give rise to different presentations in terms of symptomology and impairments. In order to diagnose ADHD, multiple methods are suggested. For example, in 2000, the American Academy of Pediatrics (AAP) established a committee of primary care physicians, researchers and clinicians to develop a list of “best practice” guidelines, along with a visual clinical algorithm to aid diagnosis of ADHD in school-aged children (6 – 12 years).

First, the AAP advocates the completion of a comprehensive health assessment (i.e., medical history and physical/neurological examination), along with in-depth family and school assessments. Both family and school assessments typically follow a similar pattern. First, parents and teachers complete behavioral checklists, which assess the type, frequency, and onset of the child’s inattentive, hyperactive, and/or impulsive symptoms. For example, one of the most commonly used clinical scales is the Conners’ ADHD Rating Scales. The Conners’ Rating Scale (CRS; Conners et al., 1998) is an 80 item self-report measure, most often used as a screener for various childhood behavioural problems including ADHD. The CRS (Parent and Teacher versions) have been shown to demonstrate excellent validity and reliability as respective values have ranged from 0.80 to 0.90 (Conners et al., 1998). However, clinically significant scores on the behavioural rating scales alone do not adequately support whether or not a child should be diagnosed with ADHD. For example, in addition to the AAP, numerous authors (e.g., Brown et al.,

2001; Reid et al., 2000) caution clinicians against the sole use of behavioural rating scales for diagnosis of ADHD in children. Instead, information from the parent- and teacher-report behavioural rating scales should supplement information received from (1) semi-structured or structured diagnostic interviews conducted with the parent/guardian and teacher, as well as (2) direct observation of the child's behaviour in school and/or home environment.

Second, once thorough information about the child's symptomology, academic performance and/or impairments, and overall psychosocial functioning are obtained, the clinician is recommended to review, and rule in/rule out any co-morbid and/or differential psychiatric conditions (other than ADHD) that may also be significantly impacting the child's functioning. It is imperative that these procedures are similarly applied to research involving clinical samples of children with ADHD.

This final step is important as most children diagnosed with ADHD often have at least one comorbid psychological disorder (for review see Barkley, 2006; Millberger, Biederman, Faraone, Murphy, & Tsuang, 1995). For example, 25% of children diagnosed with ADHD also experience depression or other mood disorders (e.g., Millberger et al., 1995). Meanwhile, anxiety and conduct disorders co-occur in roughly 25% to 40%, and 30% to 50% of children, respectively (e.g., DSM-IV, [APA] 2000; Millberger et al., 1995). In addition, roughly 50 to 60% of children diagnosed with ADHD also have some form of learning disability (e.g., Sprafkin, Gadow, Weiss, Schneider, & Nolan, 2007). Along with these co-morbid psychological conditions, many children with ADHD often experience sleep problems, with approximately 50% to 75% of children with ADHD having significant difficulties falling asleep, disrupted sleep, and/or overall shorter sleep

duration (e.g., Bullock & Schall, 2005; Cohen-Zion & Ancoli-Israel, 2004; Corkum, Tannock, & Moldofsky, 1998; Owens et al., 2009)

After the clinician has conducted a comprehensive assessment and diagnosed ADHD and any potential co-morbid conditions, the next step is the development of an effective treatment plan for the child's symptoms and functional impairments. Extensive research over the last 50 years has identified a number of pharmacological and non-pharmacological interventions for ADHD symptoms. For an in-depth review, see Chronis, Jones, & Raggi, 2006 or Toplak, Connors, Shuster, Knezevic, & Parks, 2008.

### *1.2.3 Best Practices for the Treatment of ADHD.*

Children's hyperactive/impulsive and or inattentive symptoms are typically treated with a combination of psychostimulant medication along with some form of behavioural or psychosocial intervention. Roughly 85% of children diagnosed with ADHD receive stimulant medication for treatment of their ADHD symptoms (e.g., Barkley, 2006). The most common type of stimulant medication is methylphenidate (MPH) (i.e., Ritalin). Numerous randomized control trials have documented the efficacy of stimulant medications for reducing ADHD symptoms in children. Specifically, one of the most comprehensive and influential studies was a study by the Multimodal Treatment Study of children with ADHD (MTA Cooperative Group, 1999). The MTA study included a sample of 579 children (aged 7 to 10 years) diagnosed with ADHD-Combined subtype, who were subsequently randomized into groups who received either: (1) stimulant medication, (2) behavioral interventions, or (3) stimulant medication and behavioral intervention. The MTA Group (1999) found that at the 12 month follow-up stimulant medication significantly decreased ADHD symptoms compared to the

behavioral interventions alone. Also, the combined stimulant medication and behavioral treatment groups were not significantly better at ADHD symptom reduction than stimulant medication alone, or behavioral treatments alone (MTA Group, 1999). Overall, the MTA Group concluded that stimulant medication was superior to behavioural interventions for symptom reduction of core ADHD symptoms (i.e., hyperactivity, impulsivity, inattention). However, a combined treatment protocol (medication plus behavioural intervention) was recommended to clinicians as combined treatments showed additional reductions in related impairments inherent in ADHD (e.g., oppositional/aggressive, internalizing symptoms).

Stimulant medications are generally considered to be easy-to-use, and effective with few side effects (i.e., decreased appetite, weight loss). However, several considerations must be considered regarding the use of stimulant medication with children diagnosed with ADHD. Treatment gains (i.e., reduced ADHD symptomology) last only as long as the child is taking the medication and 20 to 30% of children do not receive any positive treatment effects with stimulant medication alone. Research has also found that stimulant medications are not effective at ADHD symptom reduction over the long-term (e.g., Toplak et al., 2008). Furthermore, evidence also suggests that stimulant medications negatively impact sleep of children with ADHD. Specifically, stimulant medication has also been shown to relate to delayed sleep onset, as well as an overall reduction in total sleep time, and poorer sleep efficiency in children (e.g., Cohen-Zion & Ancoli-Israel, 2004). However, the exact nature of how sleep, ADHD, and stimulant medications are related remains unclear (e.g., Sadeh, 2000).

### *1.2.3. Conceptual Models and Neurobiology of ADHD.*

Numerous conceptual models of ADHD have been developed. These models vary in the purported relationship between top-down (i.e., executive) and/or lower-level bottom-up cognitive processes, and the observed impairments and symptomology in children with ADHD. However, for the purposes of this thesis, the cognitive-energetic model (Sergeant, Geurts, Huijbregts, Scheres, & Oosterlaan, 2003) was thought to be the most relevant model to the examination of the potential relationship between ADHD and sleep. For a discussion of different models of ADHD, see Sergeant et al., 2003. The cognitive-energetic model posits that children with ADHD demonstrate hyperactive/impulsive, and inattentive symptoms due to impairments in three key areas. First, ADHD is thought to be, in part, the result of deficits in top-down executive (EF) control systems (e.g., planning, set-shifting, response inhibition), as well as, bottom-up cognitive mechanisms (e.g., response output). The defining feature of the cognitive-energetic model is the idea that children's ADHD symptoms also relate to deficits in various energetic mechanisms (e.g., activation, effort) (e.g., Sergeant et al., 2003). Essentially, deficits in energetic mechanisms lead to children with ADHD being psychophysiologicaly under-aroused. Therefore, the cognitive-energetic model of ADHD proposes that children's hyperactive/ impulsive and inattentive behaviours represent children's attempts to counter their intrinsic psychophysiological under-arousal (e.g., Sergeant et al., 2003).

Evidence from neuroanatomical research suggests that these energetic processes, including arousal regulation, are associated with multiple brain areas. Specifically, these include the anterior cingulate cortex (ACC), dorsolateral prefrontal cortex (DLPFC), the

thalamus, and the locus coeruleus (LC) (e.g., Berger & Posner, 2000, as cited in Sergeant et al., 2003). Furthermore, researchers have also provided evidence to implicate key neurochemical correlates that may underlie impaired regulation of energetic resources (i.e., under-arousal) in ADHD.

More specifically, dopamine (DA) and norepinephrine (NE) are believed to be central in arousal modulation in children with ADHD (e.g., Sergeant et al., 2003). For example, it is believed that the efficacy of stimulant medications used to reduce ADHD symptoms in children is based on the medication's abilities to increase the release of, as well as inhibit the reuptake of, such neurotransmitters as DA and NE (e.g., McDowell, Whyte, & D'Esposito, 1998). It is hypothesized that the increased availability of these neurotransmitters (i.e., DA, NE) through psychostimulant medication improves overall PFC functioning in children with ADHD via increased overall CNS arousal (e.g., Barkley, 2006).

### **1.3 Sleep**

Sleep is essential for multiple human functions, including survival, growth, development, and overall physical and psychological health (Carskadon, Dement, & William, 2011). Sleep has been a topic that has fascinated philosophers for millennia, though recent advances in electrophysiological and neuroimaging technologies have facilitated our understanding of the general structure of sleep and sleep architecture (e.g., Kryger, Roth, & Dement, 2011).

#### *1.3.1 Measurement of Sleep and Sleep Architecture*

Researchers who investigate central aspects of children's sleep and sleep architecture have primarily used measures that can be classified as *objective* or *subjective*

(e.g., Owens, 2009). The main *objective* measure of sleep architecture is polysomnography (PSG) or a sleep study. A sleep study is typically conducted in a sleep laboratory where, under the supervision of a trained sleep technician, electrodes are attached to the participant in order to record electroencephalographic activity (EEG), respiration, and movement during their sleep. Specifically, PSG captures information about children's general sleep parameters, as well as their overall sleep architecture variables. The general sleep parameters recorded by PSG include children's (1) Lights out ("Bedtime"; time of lights out represents the time that the child is suppose to try to fall asleep), (2) Lights on ("Wake-time"; this is the time the child either spontaneously awakens in the morning or is awoken by an adult), (3) Sleep onset latency (SOL; Time from wakefulness to the first episode of Stage 1 NREM sleep), and (4) Total Sleep duration ("Mins."). In addition, PSG is the gold standard method for capturing information about children's sleep architecture (i.e., sleep stages, NREM-REM sleep cycle). The primary sleep architecture variables measured through PSG include children's (1) Sleep Efficiency % (i.e., child's time in bed versus time asleep), (2) REM Latency (i.e., time from wakefulness to first REM sleep episode), (3) Number of REM periods, (4) Stage 1 NREM sleep (%), (5) Stage 2 NREM sleep (%), (6) Stage 3/4 NREM sleep (%), and (7) REM Sleep (%).

Over the last 15 years PSG technologies and sleep lab-monitoring techniques have been revolutionized. Many sleep laboratories are equipped with in-room microphones and infrared video cameras, which allow for the visual coding and analyses of children's sleep behaviors (e.g., Kryger et al, 2011). In addition, portable "Home PSG" technologies has been developed, which allow researchers to gain information about the



children's sleep and sleep architecture in the child's bedroom, as opposed to a sleep laboratory. Although home-PSG studies have been conducted in research using school-aged children as participants (i.e., Gruber et al., 2009), the majority of empirical PSG research has been conducted within sleep laboratories.

The second type of *objective* sleep measure is actigraphy. An actigraph is a wristwatch-like device, which uses information about the child's total overall activity and/or gross body movements throughout the night, as indices of different sleep variables. One of the main advantages of actigraphy as a measure of sleep is that it is a highly unobtrusive measure of children's general sleep parameters, and some sleep architecture variables. For example, actigraphy captures information about children's (1) Lights out "Bedtime", (2) Lights on "Wake-time", (3) Sleep Onset, (4) Sleep Duration, and (5) Sleep Efficiency (%). However, actigraphy cannot capture information about children's sleep stages, or NREM-REM cycles.

In addition to *objective* sleep measures, numerous *subjective* parent-and child-report measures (e.g., questionnaires, rating scales) of children's sleep, and sleep habits have been developed. For example, the Child Sleep Habits Questionnaire (CSHQ) (Owens, 2000) is often completed by parents, to assess their perceptions of their child's sleep. Comprised of eight subscales, the CSHQ assesses a variety of factors including children's (1) sleep hygiene (routines, diet, parent/child interactions), (2) sleep quality (onset, duration), and (3) potential sleep disorders.

Sleep can broadly be defined as a, "natural state characterized by a reduction in voluntary motor activity, a decreased response to stimulation, and stereotypical posture" (e.g., Carskadon et al., 2011). Furthermore, sleep is dissociated from other altered

conscious states (i.e., coma, and/or anesthesia), in that sleep is easily reversible and self-regulating (e.g., Carskadon et al, 2011; Fuller, Gooley, & Saper, 2006). Using electroencephalogram (EEG) technology, researchers have demonstrated that normal human sleep is comprised of two alternating sleep states; rapid eye movement (REM, or “dreaming sleep”) and non-rapid eye movement (NREM) sleep. NREM sleep is subdivided into three distinct sleep stages (i.e., NREM Stage 1, 2, 3/4) (e.g., Carskadon et al., 2011; Fuller et al., 2006). NREM and REM sleep states are also each typified by dissociable EEG activation patterns.

During wakefulness, cortical EEG typically present as desynchronized, high frequency, low amplitude waves in the 14-30 Hz range (i.e., “beta waves”). These patterns of activity are presumed to reflect differences in the timing of different cognitive, motor, and perceptual functions (e.g., Fuller et al, 2006). When an individual enters a quiet rest period, with his/her eyes closed, EEG oscillations typically are within the 8-12 Hz range (i.e., “alpha waves”). Next, when an individual transitions into NREM Stage 1 sleep, conscious awareness of the external environment gradually disappears, and EEG waves become larger in amplitude and slower, with oscillations predominantly in the 4- to 7 Hz (“theta”) range. Next, Stage 2 NREM sleep is associated with the complete loss of conscious awareness as well as the appearance of “sleep spindles” and “K-complexes”. Sleep spindles and k-complexes originate in the thalamus, and represent bursts of cortical activity, which are thought to relate to maintaining NREM sleep continuity (Siegel, 2011). These stereotypic patterns appear on the computerized output of the EEG record. An individual will then progress into Stage 3/4 NREM sleep where EEG oscillations are in the 1- to 3- Hz range (“delta waves”). Stage 3/4 NREM sleep is also referred to as slow

wave sleep (SWS), due to the structure of the EEG waveforms in the EEG output (Seigel, 2011). Specifically, cortical EEG oscillations resemble low frequency, high amplitude waveforms during SWS. However, when an individual transitions from SWS to REM sleep, waveforms resemble those generated during wakefulness (i.e., high frequency, low-amplitude). Also, during REM sleep, the electrooculogram (EOG) reflects rapid eye movements, and the electromyogram (EMG) displays profound atonia of the skeletal muscular tissue. It has been hypothesized that muscle atonia in REM sleep limits individuals from acting out their dream content (Seigel, 2011).

### *1.3.2 Circadian and Homeostatic Regulation of Sleep*

Regulation of human sleep and sleep architecture patterns is achieved through both circadian and homeostatic systems. A myriad of theoretical models have been developed to explain how circadian and homeostatic processes are involved in sleep regulation. Below is a discussion of Borbely et al., (1989) *Two-process model* of sleep regulation. For a more extensive review see Krygier, Roth, & Dement (2010).

Borbely (1982) posited that human sleep propensity is determined by both *circadian* (“Process C”) and *homeostatic* (“Process S”) processes. First, a *circadian* (C) process regulates sleep in humans by an internal circadian pacemaker, or “biological clock”. This circadian pacemaker regulates different aspects of psychophysiological arousal in order to maintain a sleep-wake cycle that corresponds to roughly a 24-hour time period (Borbely, 1982). Second, sleep is also regulated via a *homeostatic* (S) process. For example, when an individual stays awake longer, his/her need for sleep (“sleep drive”) increases. However, the longer he/she subsequently sleeps, the more their overall sleep drive decreases. Overall, *homeostatic* processes (Process S) rise during

waking and decline during sleep, while interacting with circadian processes (Process C) to regulate sleep-wake patterns in humans (e.g., Borbely et al., 1982).

In addition, extensive research on sleep and psychophysiology have illuminated some key neuroanatomical and electrophysiological (EEG) correlates, which potentially underlie these *circadian* and *homeostatic* sleep regulation processes (a comprehensive discussion can be found in Krygier, Roth, & Dement, 2011.) First, researchers generally believe that the internal *circadian* pacemaker, or biological clock, which regulates the sleep-wake cycle, is located within the anterior region of the hypothalamus. This compact structure, comprised of 20,000 neurons, is known as the suprachiasmatic nucleus (SCN) (e.g., Stephan & Zucker, 1972). Specifically, researchers have demonstrated that lesions to SCN increase dysregulation of key physiological and behavioral processes including sleep-wake cycles (e.g., Rosenwasser & Turek, 2011).

Second, researchers have used EEG technologies to demonstrate the existence of the *homeostatic* process of sleep regulation in humans. For example, numerous sleep researchers (e.g., Borbely et al., 1981) have shown that sleep deprivation for a duration of 24 hours or more significantly increases the overall amount of Stage 3/4 NREM (SWS) sleep. SWS is thought to be the most restorative type of sleep. Therefore, these findings were interpreted as evidence for sleep drive and support for homeostatic sleep regulation theories.

### *1.3.3 Factors affecting Children's Sleep Architecture*

Children's sleep and sleep architecture can be greatly affected by a myriad of intrinsic and extrinsic factors. The most salient factors include:

*Age.* The strongest and most consistent factor that can affect general sleep parameters and sleep architecture is age (e.g., Kryger et al., 2011; Roethlis, 2011). For example, Stage 3/4 NREM sleep is maximal in young children, and decreases progressively with age (e.g., Kryger, Roth, Dement, 2011). In a comprehensive meta-analysis, Ohayon, Carskadon, Guilleminault, and Vitiello (2004) reported significant age trends on PSG sleep variables in children and adolescents. Specifically, based on a sample size of 1360 children, Ohayon et al. (2004) found that percentage of Stage 3/4 NREM sleep and REM latency were negatively correlated with children's age and that percentage of Stage 2 NREM sleep was positively correlated with children's age. Effect sizes reported by Ohayon et al. (2004) ranged from small to medium, which indicates that there is stability in the age-related developmental changes in sleep in children.

*Psychiatric disorders.* A myriad of mental health disorders have been inexorably linked with sleep problems. First, individuals diagnosed with depression or other mood disorders often have a myriad of sleep issues. For example, individuals with Major Depressive Disorder (MDD, DSM-IV-APA, 2000) often have difficulties falling asleep (i.e., delayed sleep onset), more night-wakings, and a shorter overall sleep duration (e.g., Sinton, & McCarley, 2010). Specifically, some of the key distinguishing symptoms of MDD are sleep difficulties, whereby patients are either unable to sleep (i.e., insomnia), or sleep too much (i.e., hypersomnia). Differences in underlying sleep architecture have also been documented in individuals diagnosed with depression. The most consistent finding from PSG research is that individuals diagnosed with MDD have significantly shorter latencies to REM sleep, higher overall percentage of NREM sleep, and less REM sleep compared to healthy controls (e.g., Kupfer, 1982; Rietman & Berger, 2001).

Second, similar to depression, one of the hallmark symptoms of anxiety, and anxiety disorders, are sleep issues. For example, in the DSM-IV-TR, (APA, 2000), generalized anxiety disorder (GAD) is characterized by rumination, or excessive worry, which often leads to sleep issues. For example, individuals with anxiety disorders experience disruptions to general sleep parameters including delayed sleep onset and reduced sleep duration (e.g., Gillin & Borbely, 1985). PSG research has suggested that children with anxiety disorders also have less NREM sleep and experience changes to the structure of their REM sleep, relative to TD children (e.g., Fuller, Walters, Binks, & Anderson, 1997; Gillin & Borbely, 1985).

Third, sleep problems have also been reported in children diagnosed with autism spectrum disorder (ASD). For example, Malow et al. (2006) found that parents of school-aged children diagnosed with ASD reported a significantly higher number of sleep issues (e.g., insomnia, delayed sleep onset) than parents of TD children. Interestingly, children with ASD did not differ from their TD peers in sleep architecture variables (e.g., sleep efficiency [%]).

Fourth, based on subjective parent-report data, sleep problems have also been found in children diagnosed with ADHD (e.g., Owens, 2009). The relationship between sleep and ADHD is further reviewed in section entitled “ADHD & Sleep”.

*Drug therapies for Psychiatric Disorders.* Pharmacological treatments have been developed to treat a range of mental health disorders (e.g., depression, anxiety, and ADHD) in children. Although effective at symptom reduction, empirical research suggests that many of these drug treatments have sleep related side-effects (i.e., sleep quality and sleep architecture). For example, physicians will often prescribe some variant

of a selective serotonin reuptake inhibitor (SSRI) to patients suffering from depression and/or anxiety. It is hypothesized that deficits in the neurotransmitter serotonin correlates with patient's overt symptoms of depressed and/or anxious mood (e.g., Carkasdon & Dement, 2011). Therefore, inhibition of serotonin reuptake (i.e., increase serotonin availability) within the brain is thought to improve depressed or anxious mood and reduce symptoms. However, SSRIs have been shown to significantly impact sleep and sleep architecture (e.g., Carkasdon & Dement, 2011). For example, participants diagnosed with depression and who were administered an SSRI (Fluoxetine) were found to have (1) higher overall sleep latency, (2) higher latency to REM sleep, and (3) more Stage 1 NREM sleep than controls. In addition, patients with depression also showed significantly fewer REM periods throughout the night (e.g., Carkasdon & Dement, 2011).

Second, the most common pharmacological intervention for reduction of ADHD symptoms in children are psychostimulant medications (i.e., methylphenidate; Ritalin). The efficacy of methylphenidate in symptom reduction in children with ADHD is believed to be via the inhibition of the reuptake of the neurotransmitters dopamine (DA) and norepinephrine (NE). Deficits in DA and NE are thought to be highly correlated with under-arousal, particularly in areas that mediate higher-level, executive functions (i.e., PFC) (e.g., Barkley., 1999). Psychostimulant medications have been linked with a variety of sleep issues. For example, Schwartz et al., (2004) completed a within-subjects double-blind, randomized controlled administration of methylphenidate (MPH). Specifically, Schwartz and colleagues, (2004) collected actigraph data during medication and placebo weeks for 44 children diagnosed with (DSM-IV) ADHD. Schwartz et al. (2004) found that children with ADHD had significantly longer sleep onset latency

(SOL), reduced total sleep time (TST), as well as significantly lower sleep efficiency while on MPH. Also, Corkum, Panton, Ironside, MacPherson, & Williams (2008) conducted a randomized control trial to determine how MPH affected the sleep of n=21 children diagnosed with ADHD. Specifically, Corkum et al., (2008) found that although MPH was successful at significantly reducing children's ADHD symptoms, compared to placebo condition, children with ADHD experienced significant delays in their sleep onset latency, and subsequent reduction in their overall sleep duration. Taken together, findings from Corkum et al., (2008) and Schwartz et al., (2004) suggest that stimulant medications used to treat symptoms of ADHD can significant affect the quality of children's sleep. Therefore, sleep scientists who are interested in researching the sleep quality and sleep architecture of children with ADHD must consider the role of stimulant medication as a potential confounding factor.

## **1.4 ADHD & SLEEP**

### *1.4.1 General Overview*

Compelling clinical, theoretical, behavioral, and neurobiological evidence suggests a relationship between sleep and ADHD. First, both sleep and ADHD have been conceptually linked to the construct of *arousal*. Namely, circadian and homeostatic factors mediate psychophysiological arousal of human sleep and sleep-wake cycles. Also, the cognitive-energetic model (Sergeant et al., 2003) posits that psychophysiological under-arousal underlies ADHD symptomology and functional impairments. Second, behaviorally, both insufficient sleep and ADHD appear to relate to similar constellations of symptoms and impairments in children's cognition, learning, and behavior regulation. Third, neurobiological research has implicated common neuroanatomical structures (i.e.,



locus coeruleus, PFC), and neurotransmitters (DA, NE) as mediators of both sleep and ADHD. These three factors have typically led pediatric sleep researchers and clinicians to adopt one of three common positions regarding *how* sleep and ADHD may be related (e.g., Owens, 2009).

First, some believe that underlying sleep problems cause (or mimic) ADHD symptoms in children (e.g., Owens, 2009). For example, Piccetti and Watters (1993) found 93% of children diagnosed with RLS also met DSM-IV criteria for ADHD. Similarly, other researchers have reported that children diagnosed with obstructive sleep apnea (OSA), and other forms of sleep-disordered breathing present with, were clinically significant levels of hyperactivity and impulsivity (e.g., Ali, Pitson, Stransling, 1996; Gaultney, Terrel, & Gingras, 2005; Melendres et al, 2004). These results are interpreted as evidence that ADHD symptoms are simply manifestation of an underlying organic sleep disorder (i.e., OSA/RLS). From this perspective, treatment of a sleep disorder should mitigate ADHD symptoms and impairments (Owens, 2009; Sadeh, 2004). However, at the current time, more research is needed to clarify this possibility.

Second, others believe that ADHD causes sleep disruptions in children (e.g., Owens, 2009). Specifically, it is thought that ADHD causes disruptions to children's general sleep parameters (i.e., sleep onset, sleep duration), and their overall sleep architecture (i.e., sleep efficiency [%]; NREM Sleep [%]). Empirical evidence has suggested that children's general sleep parameters and sleep architecture may be affected by having a diagnosis of ADHD. Subjective parent-report data suggests that children's sleep (i.e., sleep onset and duration) are impacted significantly by hyperactivity/impulsivity and/or inattention in ADHD (e.g., Ball, 1995). For example, children with

ADHD take longer to fall asleep, have more night-wakings, and sleep less than TD peers. However, PSG studies have failed to reveal consistent differences in sleep architecture between children with ADHD and their TD peers.

Third, others believe sleep and ADHD are influenced by an unknown third variable (e.g., Owens, 2009). For example, numerous potential “third” variables have been put forward by pediatric sleep researchers to unify or explain both sleep issues and ADHD symptomology. These include deficits in neurotransmitters (DA, NE), neuroanatomical issues in the PFC, and/or dysregulation of psychophysiological arousal (e.g., Sergeant et al., 2003). However, although these factors have been conceptually linked to both ADHD and sleep, there has not been significant empirical evidence to support a specific third variable that underlies both sleep and ADHD (e.g., Owens, 2009).

The current thesis examines if and how ADHD is related to sleep disruptions in children. For example, there has been a significant debate between pediatric sleep researchers as to whether or not children with ADHD present dissociable sleep profiles (i.e., general sleep parameters, sleep architecture) compared to their TD peers. A critical review of empirical studies that have used PSG to compare sleep in school-aged children (6 – 12 years) diagnosed with ADHD to their TD peers has been provided in the following sections. Displayed in Tables 1a and 1b, are 28 empirical studies published between 1971 and 2011, which compared whether children with ADHD differed significantly from TD peers on general sleep parameters (sleep onset, duration), or sleep architecture (e.g., sleep efficiency %).

Results are discussed according to the following format. First, each sleep variable of interest is presented along with a listing of the relative number of studies which

reported either non-significant (i.e., ADHD = TD) or significant differences (i.e., ADHD > TD; ADHD < TD) between ADHD and TD groups. Second, PSG results are then critically reviewed to determine how well researchers controlled for confounds including age- and sex-matching, medication status, diagnosis subtype, and co-morbidities. (Please note that the studies included in this critical analyses are indicated by an asterisk in the reference section of this thesis.)

#### *1.4.2. Literature Review Results*

As shown in Tables 1a and 1b, the majority of empirical PSG studies failed to find significant differences between children with ADHD and their TD peers in terms of general sleep parameters. For example, 24 studies examined whether children with ADHD differed significantly from their TD peers in sleep onset latency. Overall, 75% of these studies reported no significant group differences for sleep onset latency between children with ADHD and their TD peers. Only four of 24 studies reported significantly longer sleep onset latencies, while just two of 24 studies reported shorter sleep onset latencies for children with ADHD compared to their TD peers. Similarly, 25 studies examined whether children with ADHD differ in their total sleep duration compared to their TD peers. Of these studies, 68% of studies reported no significant differences in total sleep duration between ADHD and TD groups (ADHD = TD). Of those studies that reported group differences, six found children with ADHD had shorter sleep than TD peers); two reported children with ADHD had longer sleep than TD peers.

As presented in Tables 1a and 1b, the majority of empirical PSG studies also failed to find significant differences between children with ADHD and their TD peers in terms of sleep architecture. For example, there have been two empirical studies

comparing the number of REM periods between children with ADHD and their TD peers and both found no differences between groups.

Twenty-three empirical PSG studies examined whether children with ADHD and their TD peers differed in their sleep efficiency percentage. Overall, 73.9% of these studies found no difference in sleep efficiency percentage between ADHD and TD groups. Six studies reported differences; five studies found that children with ADHD had lower sleep efficiency percentages) and but one study found children with ADHD had higher sleep efficiency percentages.

Investigation of potential differences in total NREM sleep percentages between children with ADHD and their TD has been investigated in 11 studies. Overall, 82% of these studies found no difference in total NREM sleep between ADHD and TD groups. Further, only two studies found children with ADHD had significantly less NREM sleep percentage than TD peers. Examination of potential discrepancies in total Stage 1 NREM Sleep percentages between children with ADHD and their TD has been investigated in nine studies. Of these studies, 89% failed to find differences in Stage 1 NREM Sleep percentages between ADHD and TD groups. Only one study found children with ADHD had significantly less Stage 1 NREM sleep percentages than TD peers (ADHD < TD).

Within the empirical PSG literature some inconsistencies were noted regarding other sleep architecture variables. More specifically, 58.4% of studies examining differences in Stage 2 NREM sleep found no significant group differences between children with ADHD and their TD peers. Similarly, 53.8% of studies also reported a lack of significant differences between children with ADHD and their TD peers for Stage 3/4 NREM sleep. The majority (58.8%) of empirical studies investigating differences

between children with ADHD and their TD peers for latency to REM sleep found no significant differences between groups (ADHD = TD). Finally, investigations of REM sleep percentage between children with ADHD and their TD peers similarly found no group differences in 56% of studies.

Overall, results from the current literature review are consistent with past meta-analytic (Cortese et al., 2003; Sadeh, 2006) and qualitative reviews (e.g., Corkum et al., 1998) in demonstrating that there is a lack of consistent differences in general sleep parameters and architecture between children with ADHD and their TD peers. These highly inconsistent results obtained from PSG research have led paediatric sleep researchers to examine what factors relate to, or cause, these highly inconsistent findings. Numerous authors (e.g., Corkum et al., 1998; Sadeh, Pergamin, & Bar-haim, 2006) have posited that various methodological issues may account for variability in these empirical PSG studies.

#### *1.4.3 Critical Review of PSG Results*

Previous qualitative (Corkum, Tannock, & Moldofsky, 1998; Hoban, 2008) and meta-analytic reviews (Cortese, Faraone, Konofal, & Lecendreux, 2009; Sadeh, Pergamin, & Bar-haim, 2006) have demonstrated that children's sleep architecture can be impacted by different confounding factors such as their sex, age, medication-status, and prevalence of co-morbid conditions. The following will present a critical review of the empirical PSG literature to examine control over five key potential confounding factors (i.e., age-matching, sex-matching, medication-status, ADHD diagnostic techniques, co-morbid conditions).

First, despite the fact that empirical research has found that both children's age and their sex can significantly impact their general sleep parameters and sleep architecture (e.g., Krygier, 2011), only 16 of the 28 studies presented in Table 1a matched ADHD and TD groups according to *age*, and in only six of 28 studies were ADHD and TD groups matched according to *sex*. Second, evidence from pharmacological research has demonstrated how stimulant medications (i.e., methylphenidate) can significantly delay sleep onset, as well as impact sleep architecture (e.g., significantly reduce REM latency; Stage 3/4 NREM sleep) in children with ADHD (Schwartz et al., 2004). However, review of empirical results found that in only 13 of 28 studies were ADHD participants *stimulant medication-naïve*. Third, extensive research has found that a variety of co-morbid conditions can impact children's sleep and sleep architecture- namely depression and anxiety (e.g., Owens, 2009). However of the 28 empirical PSG studies, only 13 studies provided clear *exclusionary criteria* regarding medication. In the remaining 15 studies, exclusionary criteria were either not addressed or described, or only vague descriptions were presented. One positive note is that throughout the empirical PSG research, investigators have employed *robust diagnostic techniques* for assessment of children with ADHD. In other words, researchers did not simply assign children to ADHD condition based simply on clinically significant scores on an ADHD symptom rating scale, but instead the research participants were diagnosed with ADHD following a comprehensive diagnostic assessment. Specifically, in 18 out of 24 studies children were assessed by at least one psychiatrist or clinical psychologist. Also, in only three PSG research studies did researchers diagnose ADHD solely via rating scales.

Out of the 28 empirical PSG studies, only three matched ADHD and TD participants according to age and sex, as well as included only children with ADHD that were stimulant medication-naïve prior to PSG testing. Furthermore, only one study (Prihodova et al., 2010) controlled for all potential confounds discussed (e.g., age, sex, medication-status, exclusionary criteria). Following these stringent controls, Prihadova et al. (2010) did not find differences in sleep parameters or architecture between ADHD and TD groups.

### 1.5 *Research Introduction*

In light of the methodological concerns raised in previous studies, the primary goal of the current thesis was to conduct PSG research to determine whether a rigorously diagnosed, medication-naïve, age- and sex-matched sample of children with ADHD differs from a TD sample in general sleep parameters and/or sleep architecture. Additionally, the secondary goal was to compare general sleep parameters and sleep architecture between ADHD-C/HI, ADHD-IA, and their TD peers. Of the 28 empirical PSG studies in Table 1a, only one study (Ramos Platon, M.J., Bueno, A.V., Sierra, J.E., & Kales, 1990) examined general sleep parameters and sleep architecture within different ADHD subtypes. Ramos Platon et al (1990) found children diagnosed with the attention deficit disorder with hyperactivity subtype (ADD/H) (DSM-III, APA 1987) had significantly shorter sleep onset and lower sleep efficiency values than children with attention deficit disorder without hyperactivity (ADD/WO). However, there have been no published studies that have compared general sleep parameters and sleep architecture between subtypes of ADHD according to DSM-IV-TR (APA, 2000) classification. Based on empirical PSG results the following hypothesis were generated:

- 1. Children with ADHD would not differ from their TD peers in terms of general sleep parameters (i.e., bedtime, wake-time, sleep onset, sleep duration).*
- 2. Children with ADHD will not differ from their TD peers in terms of sleep architecture variables (e.g., REM Latency)*
- 3. There will be no significant differences in general sleep parameters, and/or sleep architecture between ADHD-C/HI, ADHD-IA, or TD peers.*



## CHAPTER 2: METHODS

### 2.1 Participants

The overall sample initially consisted of 30 children diagnosed with ADHD and 30 TD children. However, after participant matching according to *sex* and *age*, the final sample included 25 children in the ADHD group and 25 children in the TD group. Boys and girls who comprised the ADHD and TD groups were between 6 and 12 years of age.

Participants were recruited using a variety of methods. For example, children in the ADHD group had previously completed a study that examined the impact of stimulant medication on sleep in children with ADHD. Prior to participation in the medication trial, each child received a comprehensive diagnostic assessment by a clinical psychologist at either the Colchester ADHD Clinic in Truro or via private practice assessment (i.e., Corkum and Associates). Both the private practice and the ADHD Clinic used similar diagnostic tools and techniques. Specifically, each child, (1) had a new DSM-IV (APA, 2000) diagnosis of either ADHD-C or ADHD-IA subtypes based on a comprehensive clinical diagnostic assessment, (2) were stimulant medication-naïve, and (3) were free of any chronic or impairing medical illness (e.g., diabetes) and/or or co-morbid psychiatric conditions known to impact sleep (i.e., depression, anxiety). However, children in the ADHD group were not excluded for having a learning disability (LD), given the high comorbidity of these two disorders (e.g., Barkley, 1999; Milberger et al., 1995), and lack of empirical research linking LDs and sleep issues. Prior to the medication trial, all children in the ADHD group completed a one-week assessment of their sleep and behaviour and on the last night of participated in an overnight PSG testing session. The PSG testing was done to confirm children with ADHD had no intrinsic sleep disorders (e.g., OSA, RLS).

Approximately two-thirds of the typically developing children were recruited from a previous study that examined the impact of sleep manipulation on daytime functioning in TD children. Of note, children completed no PSG testing during their participation in the sleep manipulation study. The remaining TD participants were recruited from the community via web advertisements, word-of-mouth, and newsletters (e.g., Dalhousie Notice Digest, IWK). Participants were excluded from the TD group if screening indicated (1) a chronic or impairing medical illness (e.g., diabetes), (2) a history of neurological impairments, (3) any mental health, or (4) primary sleep disorder. This was done through the use of parent-report rating scales, as well as a screener form administered via telephone by a research assistant. Prior to the completion of the PSG testing, all children in the TD group completed a one-week assessment of their sleep and behaviour.

## **2.2 Procedure**

Ethical approval for this study was obtained from the Research Ethics Board of the IWK Health Centre. Data collection for the present study involved two key parts. First, the primary researcher confirmed that parents of the children in the ADHD group consented to the use of his/her child's PSG data (obtained during baseline night of stimulant medication trial), for use in subsequent research. Only data from the children's baseline PSG testing session completed were used in the current thesis. The primary researcher also confirmed that parents of the children in the TD group (who previously completed sleep manipulation study) consented to be recruited for their child's participation in subsequent sleep research.

Second, the primary researcher then contacted interested parents of TD children via telephone to describe the current study, as well as administer a screening questionnaire. If criteria inclusion were met, a PSG testing session was scheduled for a single overnight sleep study on a Friday or Saturday evening. Each child, under the supervision of his/her parent, was asked to closely follow his/her typical sleep schedule for the week prior to scheduled PSG test date. Upon arrival at sleep lab facility for PSG testing (2 hours prior to child's typical bedtime), the child and parent were introduced to the primary researcher as well as the research assistant (RA) who provided a brief tour of the facility, including child and parent bedrooms, bathrooms and control room. Next, information about the child's sleep schedule over the past seven days, as well as the parent-report demographic were collected, and the primary researcher obtained parent consent and assent from children. Simultaneously, a research assistant entertained (e.g., played a board game) the child before electrode placement began and selected an age-appropriate DVD for child to watch while the PSG hook-up (e.g., electrodes, belts) was completed. The child's "lights out" and "lights on" times were set based on parent-report information regarding the child's typical weeknight bed and wake time. Throughout the night the primary researcher monitored each child's sleep patterns via video recording and in-room microphone to ensure no wires become entangled, detached or loose, and to troubleshoot any issues. In addition to electronically recording information directly on the PSG program, the researcher also completed hourly documentation of relevant information on a sleep log (e.g., position changes). In the morning, the researcher removed electrodes from the child and administered questionnaires of child- and

parental-perceptions of sleep quality during PSG testing (not used in the current study).  
PSG data was processed by a registered sleep technologist.

### **2.3 Measures**

For both the ADHD and TD groups all PSG testing was performed at the Chronobiology Laboratory in the Department of Psychiatry at the QEII HSC in Halifax. The lab is a two-bedroom facility, equipped with Sandman® polysomnography system which recorded four electroencephalogram (EEG) channels, left and right electrooculogram (EOG), two electromyogram (EMG) channels, and electrocardiogram. The child's respiratory effort and oxygen saturation were captured via oral nasal cannula, and a finger-probe pulse oximeter, respectively. A room microphone detected snoring, and an infrared camera recorded any changes in body position the child made throughout the night. PSG captured the following general sleep parameters and sleep architecture variables. First, general sleep parameters included children's, (1) Light-outs "Bedtime", (2) Lights-on "Wake-time", (3) Sleep Onset Latency (mins), and (4) Total Sleep Duration (mins). Second, sleep architecture variables included children's (1) Sleep Efficiency (%), (2) Number of REM periods, (3) Latency to REM sleep (mins), (4) % of Stage 1 NREM sleep, (5), % of Stage 2 NREM sleep, (6) % of Stage 3/4 NREM sleep, and (7) % of REM sleep.

Prior to the PSG testing, each parent completed the Conners' Parent Ratings Scale (CPRS; Conners', 1997). The CPRS is an 80-item parent-report measure, most often used as a screener for various childhood behavioural problems including ADHD. The CPRS has been shown to demonstrate excellent validity and reliability as respective values have ranged from 0.80 to 0.90 (e.g., Conners et al., 1997). CPRS scores were used to confirm

that TD participants did not have clinically significant ADHD symptoms. Parents also completed a demographics questionnaire, which captured salient information pertaining to child, parent, and family variables. This included information on family socioeconomic status (SES; Hollingshead, 1975), family composition, as well as age, gender, and overall medical history of his/her child.

## **2.4 Statistical Analyses**

First, independent t-tests were conducted to ensure ADHD and TD groups differed significantly on key behavioural variables (i.e., CPRS Scores), but equivalent on demographic variables (i.e., age, sex, SES, total number of children in household, family income).

The first research question examined whether children with ADHD differed from their typically developing (TD) peers on key general sleep parameters, using a between-subjects multiple analysis of variance (MANOVA). The independent variable (IV) *Group*, had two levels, (ADHD, TD) and there were four dependant variables (DV's) including children's: (1) Lights out "Bedtime", (2) "Lights on" Wake-time, (3) Sleep Onset Latency (mins), and (4) Total Sleep Duration (mins),

The second research question examined whether children with ADHD differed from their typically developing (TD) peers on key sleep architecture variables, using a between-subjects multiple analysis of variance (MANOVA). The IV *Group*, had two levels, (ADHD, TD) and there were seven dependent variables (DV's) including children's: (1) Sleep Efficiency (%), (2) Number of REM periods, (3) Latency to REM sleep (mins), (4) % of Stage 1 NREM sleep, (5), % of Stage 2 NREM sleep, (6) % of Stage 3/4 NREM sleep, and (7) % of REM sleep.

The third research question examined whether there were significant differences in general sleep parameters or sleep architecture variables between ADHD-C/HI and ADHD-IA groups. Separate analyses of covariance (ANCOVA's) including Helmert's contrasts were conducted- the first for general sleep parameters (e.g., sleep onset, duration), the second for sleep architecture (e.g., sleep efficiency). In both ANCOVA's the IV *Group* had three levels (ADHD-IA, ADHD-C/HI, TD), with children's age entered as covariate.

In order to determine the likelihood of detecting true population differences based on the sample size of the current study, a power analysis was conducted using G\*power technology. Specifically, based on a sample size of 25 participants in both the ADHD and TD groups, and nine total dependent variables, power to detect a medium to large effect size (i.e., 0.30 or greater) was determined to be 0.95. As such, the results from G\*power analysis suggests that any statistically significant in sleep variables between ADHD and TD groups are likely to be detected based on the current studies final sample.

## CHAPTER 3: RESULTS

### 3.1 Demographic Characteristics

Demographic variables are presented for both groups in Table 2. Independent sample t-tests confirmed that the 25 children in the ADHD group (22 boys, 3 girls) and the 25 children in the TD group (22 boys, 3 girls) did not significantly differ in terms of their mean age (months)  $t(1,46) = 0.292, p = 0.77$ . The mean age of children in the ADHD group was 105.72 months (SD= 22.57 months), whereas the mean age of children in TD group was 103.92 months (SD= 21.07 months). However, as expected, independent samples t-test did confirm that the average CPRS ADHD indices for the children in the ADHD group ( $M_{ADHD} = 73.32, SD = 8.11$ ) was significantly higher than the CPRS indices for children in TD group ( $M_{TD} = 47.17, SD = 7.06$ ) for ADHD DSM-IV symptomology  $t(1,46) = 11.86, p < 0.001$ . Finally, the ADHD and TD groups did not differ on key family characteristics, including (1) socioeconomic status (SES; Hollingshead, 1975),  $t(1,46) = 0.277, p = 0.78$ , (2) average household income  $t(1,46) = 1.17, p = 0.25$ , and (3) total number of children in the household  $t(1,46) = 1.07, p = 0.28$ . Overall, these results confirm that the matching procedures were successful.

### 3.2 First Research Hypothesis: General Sleep Parameters

MANOVA analyses examining general sleep parameters revealed significant effects for *Group*,  $F(1,44) = 3.74, p = 0.01$ . As presented in Table 3, there were statistically significant differences between the ADHD group and the TD group on (1) Sleep Onset  $F(1,40) = 7.95, p = 0.007, d = 0.63$ , and (2) Sleep Duration  $F(1,40) = 4.14, p = 0.047, d = 0.74$ . The respective effects sizes are considered to demonstrate “medium” to “large” effects (Cohen, 1992).

In terms of *Sleep Onset*, the children in the ADHD group had a mean value of 47.42 minutes (SD=38.61 mins), which was significantly higher than the children in the TD who had a mean value of 24.07 minutes (14.01 mins). The ADHD group had a mean value of 466.01 minutes (SD= 69.02 mins) for *Sleep Duration*, which was significantly lower than the TD Group who had a mean value of 500.79 minutes (SD= 50.42 mins). However, as presented in Table 3, there were no significant differences between ADHD and TD groups for Lights out “Bedtimes, or Lights on “Wake times”. For example, the average bedtime for children in ADHD was 9:33pm (SD= 40 mins), the average bedtime for children in the TD group was 9:38pm (SD= 34 mins). Also, the average wake-time for children in the ADHD was 6:44am (SD= 47 mins), while children in the TD group’s wake time was 7:05am (SD= 45 mins). However, it did appear that children in the ADHD group woke earlier than TD children as p-value for Lights on “Wake times” approached statistical significance (i.e.,  $p = 0.11$ ).

### **3.3 Second Research Hypothesis: Sleep Architecture**

MANOVA analysis examining sleep architecture variables did not reveal statistically significant effects for *Group*,  $F(1,42) = 0.993$ ,  $p = 0.449$ . For example, as presented in Table 4, non-significant p-values ranged from 0.219 for REM sleep % to 1.0 for number of REM periods.

### **3.4 Third Research Hypothesis: ADHD Subtype**

The final group of analyses examined differences between the subtypes of ADHD as well as TD children. Key demographic characteristics of the ADHD-C/HI, ADHD-IA, and TD groups are presented in Table 5. Although there were no statistically significant differences in age between the three groups ( $p=.06$ ), there were some potential



meaningful discrepancies, which could have impacted results. For example, mean age ranged across the groups (ADHD-IA group:  $M= 114.33$  months,  $SD = 24.22$ ), ADHD-C/HI group:  $M= 97.77$ ,  $SD = 18.38$ , TD group:  $M= 103.92$ ,  $SD = 21.07$ ), which represented a mean difference of 16 months. As empirical research (e.g., Ohayon et al., 2004) has found that age can be a significant moderator of children's sleep architecture, in the two subsequent MANOVA analyses children's age was included as a covariate.

First, in terms of general sleep parameters, results from a between-subjects MANCOVA revealed significant effects of Group ( $F(2,46) = 3.54$ ,  $p = 0.010$ ). As presented in Table 6 results from Helmert contrasts revealed the mean sleep onset latency for the TD group (24.07,  $SD=14.01$ ) was significantly shorter than both the ADHD-IA (39.32,  $SD= 28.62$ ), and ADHD-C/HI (54.54,  $SD= 45.93$ ) groups (contrast estimate = -21.35,  $p = 0.01$ ). There were however no significant differences between ADHD-C/HI, and ADHD-IA subtypes (contrast estimate = -19.8,  $p = 0.09$ ). Helmert contrasts also revealed that though the mean sleep duration for the TD group (500.79,  $SD= 50.43$ ) was significantly longer than both ADHD-IA (443.84,  $SD= 81.11$ ), and ADHD-C/HI (486.47,  $SD= 50.51$ ) groups (contrast estimate = -32.8,  $p = 0.04$ ), there were no significant differences between ADHD-C/HI and ADHD-IA groups for total sleep duration (contrast estimate = -4.18,  $p = 0.85$ ).

Next, regarding differences in sleep architecture between ADHD-C/HI, ADHD-IA, and TD groups, results from a between-subjects MANOVA failed to reveal significant effects for Group ( $F(2,46) = 0.912$ ,  $p= 0.550$ ). Finally, results from an additional MANOVA following outlier analysis is described in Footnote<sup>1</sup>.

## CHAPTER 4: DISCUSSION

### 4.1 Summary of Research Hypotheses

The aim of this thesis was to objectively examine general sleep parameters and sleep architecture in a rigorously diagnosed, stimulant medication-naïve, sample of children with ADHD, compared to an age- and sex-matched sample of their typically developing (TD) peers.

Although children with ADHD took longer to fall asleep and slept less than their typically developing peers, they did not have any differences in sleep architecture. The results of the present study do not support the notion that children with ADHD have differences in their sleep architecture. Our data did not demonstrate differences between ADHD subtypes, once age was controlled for sleep variables. ADHD subtype groups did not differ from each other.

In terms of general sleep parameters, results from PSG testing revealed children in ADHD group took significantly longer to fall asleep and slept significantly less than their TD peers. These results were inconsistent with the majority of empirical PSG studies, which have found no differences between children with ADHD and their TD peers for sleep onset or sleep duration (e.g., Cortese, 2006; Sadeh, 2006). However, results are consistent with empirical subjective, parent-report sleep measures, which have generally reported more difficulties with sleep onset, and shorter sleep duration in children with ADHD, than their TD peers (e.g., Cortese, 2009). One explanation for why thesis results coincide with parent-report sleep research is that in the current thesis a concerted effort was used in order to help ensure that children's sleep routines at the sleep laboratory (i.e., bedtime routines; bed and wake-times) were as similar as possible

to their home routines. This is in contrast to previous empirical PSG studies (e.g., Busby & Pivik., 1981; Cooper et al., 2004; O'brien et al., 2003) that imposed standardized bed and wake-times for both ADHD and control groups. For example, in a paper published by O'Brien et al. (2003) all children (whether in the ADHD or TD group) were awoken at 7:00 am, if the child had not spontaneously awoken prior to this time, so that PSG electrodes could be removed. Prescribing pre-set bed or wake-times could have masked differences in sleep onset or duration between ADHD and TD groups in empirical PSG research.

There are two reasons for the shorter sleep duration in children with ADHD found in this current study. First, are the observed differences in the wake-times between children with ADHD compared to their TD peers. Namely, although not statistically significant, children in the ADHD group woke earlier, on average, than their TD peers. Second, the shortened sleep duration in children with ADHD is also due to longer sleep onset latencies in children with ADHD compared to their TD peers.

Based on the cognitive-energetic model of ADHD (Sergeant et al., 2003), it seems plausible that children in the ADHD group took longer to fall asleep and woke earlier than their TD peers because of potential difficulty with psychophysiological arousal regulation. Specifically, children with ADHD likely had underlying issues with regulation of activity or arousal, which could also explain why children woke earlier than their TD peers. Specifically, children with ADHD may not have been able to moderate their arousal levels so that they could fall back to sleep until their scheduled wake-time.

Another potential explanation for differences in sleep parameters is related to the fact that children's sleep was measured in a sleep lab, rather than in their home

environment. Various authors (e.g., Prihadova; Sadeh et al., 2006) have suggested that factors such as being in a novel environment, as well as being attached to electrodes, may potentially disrupt children's typical sleep patterns. For example, sleep lab "first night" effects have been demonstrated in a meta-analysis by Sadeh and colleagues (2006). Specifically, Sadeh et al. (2006) reported that children's total sleep duration is significantly longer in studies where multiple nights of PSG data was recorded, compared to studies which collect only a single night of PSG data. Given that this current thesis consisted of only included a single night of PSG data collection, this could have impacted children's total sleep duration. However, there is no evidence that children with ADHD are differentially affected by first night effects (Bessey & Corkum, 2012), so it is likely that both the ADHD and TD group would have been affected similarly.

Many of the other potential confounding factors can be ruled out. Both stimulant medication effects, as well as co-morbid psychiatric disorders, can be ruled out as potential explanations for the differences in delayed sleep onset latencies and shorter sleep duration for children with ADHD. For example, previous empirical literature has found that both stimulant medications (e.g., methylphenidate), and co-morbid psychiatric conditions (i.e., anxiety, depression) can significantly delay sleep onset in children with ADHD (e.g., Barkley, 2006; Corkum et al., 2008). However, in our sample, the children were medication-naïve and did not have significant levels of comorbidity. Therefore, it is possible that delayed sleep onset and shorter sleep duration is part of the presentation of children with ADHD and this could be further affected by treatment with stimulant medications and/or by other co-morbid conditions. Further research is needed to clarify the exact nature of the relationships between ADHD, co-morbid conditions, the use of

stimulant medications, and potential differences in general sleep parameters (i.e., sleep onset, duration) between children with ADHD and their TD peers.

With regard to sleep architecture, we did not find any significant differences between children with ADHD and their TD peers in their underlying sleep architecture as assessed by PSG. These results support our initial research hypothesis, previous empirical research, and conclusions from the meta-analyses, which generally have found no differences between ADHD and TD peers (e.g., Sadeh, 2006). A comparison of PSG sleep architecture values from the current thesis for the ADHD and TD groups group revealed that these values are consistent with published developmental norms (e.g., Ohayon et al., 2004). For example, values for percentages for NREM sleep and REM sleep for children with ADHD fall within developmental levels.

It is possible that previous studies, which found differences in sleep architecture in ADHD, may have suffered from methodological problems. Our study had rigorous methodological controls, including (1) closely matched ADHD and TD groups according to age and sex, (2) ensured that children in the ADHD group were stimulant medication-naïve, and (3) excluded children from the ADHD group who had co-morbid psychiatric conditions which are known to impact sleep architecture (e.g., anxiety, depression).

The lack of significant differences in sleep architecture between ADHD and TD groups has potential implications for conceptual models of ADHD and their relation to sleep. Specifically, the cognitive-energetic model (e.g., Sergeant et al., 2003) posits that children's hyperactive/impulsive and/or inattentive ADHD symptoms are the result of psychophysiological arousal dysregulation. Although this dysregulation of arousal may impact children with ADHD's ability to fall asleep (i.e., delay sleep onset latency), this

potential arousal dysregulation does not appear to affect sleep-wake cycles or sleep architecture. Multiple sleep latency tests (the gold standard method for evaluation of children's arousal, and overall daytime and behavioural functioning) would be ideal tools for evaluating these issues in future research studies.

Our analyses comparing sleep in ADHD subtypes indicated that there were no significant differences in general sleep parameters or sleep architecture variables between children diagnosed with ADHD-C/HI and ADHD-IA subtypes (DSM-IV, [APA], 2000). The sleep profiles of the different subtypes of ADHD are not well studied. For example, differences in sleep between ADHD subtypes have been empirically investigated in two known studies; one which used PSG (Ramos Platon et al., 1990) and one that employed subjective, parent-report ratings (Mayes et al., 1999). The finding from these two studies suggested the ADHD hyperactive subtype was associated with more sleep problems (i.e., delayed sleep onset; lower sleep efficiency [%]) than children in the inattentive subtype. Given that ADHD subtypes tend to differ in terms of some important demographic variables (age, sex), it was important to control for these factors. Once these factors were controlled for there were no significant differences in sleep variables between the subtypes observed in the current thesis. One explanation for why Ramos Platon et al. (1990) found significantly lower sleep efficiency in children with ADD with hyperactivity, compared to children with ADHD without hyperactivity, is perhaps because their groups were not matched for age and they did not control for age in statistical analyses.

It is important to note that recent empirical research has generated only minimal support for validity of the DSM-IV ADHD subtypes (e.g., Willcutt et al, 2012). For

example, a meta-analysis by Willcutt and colleagues (2012) found only weak evidence for the validity of the ADHD-HI subtype, as well as minimal evidence to justify distinction between ADHD-IA and ADHD-C subtypes. Based on these findings, it is not surprising that no significant differences in sleep were observed between ADHD subtypes in the current thesis. Another potential explanation for the lack of differences between ADHD subtypes on sleep architecture variables could be related to the small sample sizes for each subtype in the current thesis.

#### **4.2 Study Limitations and Strengths**

The current thesis had a number of potential limitations, which could have impacted the study's results. First, the scope of this thesis allowed for the collection of only one night of PSG data from each participant in both the ADHD and TD groups. Therefore, sleep onset and/or duration could have been affected by "first night" effects (e.g., Prihadova et al., 2010). However, there is no evidence that children with ADHD are differentially affected by first-night effects (Bessey & Corkum, 2012), and as such, there is no reason to believe that our results would be different if an adaptation night was incorporated.

Second, the relatively small number of children included in the ADHD subtype analyses reduced the overall statistical power of these analyses. However, sufficient statistical power was still achieved. Results from a G\*power analysis indicated that the achieved power for the analyses between ADHD-C/HI and ADHD-IA subtypes was 0.71. (Power analysis is further described in Footnote<sup>2</sup>.)

Third, generalizability of the study's results to the larger population of children diagnosed with ADHD may be limited to due to our stringent inclusionary criteria. For

example, in order to participate in the ADHD group, children must have been stimulant medication-naïve, as well as free from any co-morbid psychiatric conditions known to impact sleep (i.e., depression, anxiety). However, extensive clinical research (e.g., Barkley, 2000) has shown that in roughly 90 percent of cases, stimulant medications (e.g., methylphenidate) are the primary treatment used to treat ADHD in children. Also, although prevalence rates vary, roughly 75% of children with ADHD will also receive a diagnosis for at least one co-morbid disorder (e.g., anxiety and/or depression) (Millberger et al., 2003). Therefore, our results are only generalizable to medication-naïve children with ADHD without comorbidity.

Despite these limitations, the present study also had a number of strengths. First, children's sleep architecture was examined using polysomnography, the gold standard measure of children's sleep (e.g., Krygier et al., 2011). Second, PSG data was collected while children followed their typical home bedtime routines. This is in contrast to many previous studies that have imposed an arbitrary bedtime and wake-time. Third, we employed rigorous controls over many potential confounds that could have impacted children's general sleep parameters and/or sleep architecture. The internal validity of the current thesis was strengthened significantly by matching procedures, ADHD diagnostic techniques, and control over medication-status and co-morbidities. Fourth, this is the first known study to investigate whether there were differences in sleep architecture across the current DSM-IV ADHD subtypes. The only other study (Ramos Platon et al, 1990) examined sleep architecture between children diagnosed based on DSM-III-TR criteria of ADD with or without hyperactivity.



### 4.3 Conclusions and Future Directions

Results from the current thesis suggest that children with ADHD who are medication-naïve do not have differences in their sleep architecture relative to their typically developing peers. Rather, it was found that children with ADHD took longer to fall asleep and slept less than their TD peers, and it is posited arousal regulation difficulties may explain delayed sleep onset latencies of children with ADHD compared to their TD peers.

Given that there is now a relatively large body of research demonstrating that children with ADHD do not differ on the typical variables used to examine sleep architecture, future research should examine different aspects of sleep architecture. For example, the majority of empirical PSG research has examined sleep macrostructure (e.g., sleep efficiency, % of REM sleep). One alternative would be to compare children with ADHD and TD peers for differences in their sleep microstructure- namely, cyclic alternating patterns (CAP) (e.g., Miano et al., 2006). CAP's are characterized by sequences of transient electro cortical events, which are distinct from background EEG activity and recur at up to 1-minute intervals (e.g., Miano et al., 2006). These EEG sequences are thought to typify cortical arousals, which are subsequently believed to relate to sleep organization or fragmentation. Since some authors posit ADHD to be a disorder of arousal (e.g., Sergeant et al., 2003), CAP analyses may be a more effective research method for finding differences in sleep architecture between children with ADHD and their TD peers.

Not only were there statistically significant mean differences between the ADHD and TD groups for sleep onset and sleep duration, but there were also large variability

with the data from the children in the ADHD group. Therefore, future sleep research should attempt to identify the key factors as to why children with ADHD present so much variability in their sleep profiles. Specifically, although some researchers (e.g., Gruber et al., 2000) have suggested that children with ADHD have higher instability in their overall sleep patterns, there is a lack of understanding for why such variability occurs.

Another potential area of investigation for pediatric sleep researchers would be to compare the general sleep parameters and sleep architecture between children diagnosed with ADHD-combined, or hyperactive/impulsive subtypes, to children who present with sluggish cognitive tempo (SCT) (e.g., Carlson & Mann, 2002). SCT is a construct thought to characterize children who present with DSM-IV symptoms of inattention, but do not show any symptoms of hyperactivity/impulsivity (e.g., Carlson & Mann, 2002). For example, Penny, Waschbusch, Klein, & Corkum, (2009) developed and provided initial psychometric validation for a 14-item measure (Likert scale range 0 – 3) of SCT in school-aged children. Based on administration to 335 parents and teachers of children aged four to 13 years, Penny et al. (2009) found evidence for three SCT subtypes. These include, *slow* (“efforts on tasks fade quickly”), *sleepy* (“appears tired, drowsy”), and *daydreamer* (“gets lost in his/her thoughts”). Specifically, because children diagnosed with SCT demonstrate only inattentive symptoms and no hyperactive/impulsive symptoms, use of items that measure SCT, in lieu DSM-IV ADHD-IA subtype criteria, may more robustly facilitate examination of sleep differences between ADHD subtypes.

Regardless of whether future pediatric sleep researchers examine the macro- and/or microstructure of children’s sleep architecture patterns in ADHD, or ADHD

subtypes, it will be important to control key confounding factors (e.g., age, medication status, co-morbidities) known to impact sleep in school-aged children.

In conclusion, results from the current thesis have important theoretical and clinical implications for pediatric sleep researchers and clinicians who diagnose and treat ADHD in school-aged children. First, regarding theoretical implications, unlike previous empirical PSG research, which has largely been atheoretical in nature, this current study proposes a psychological theory to explain why potential differences in sleep architecture should be expected. For example, rarely in previous PSG sleep research have sleep scientists overtly communicated what theoretical basis suggest that children with ADHD would show significant differences in their sleep compared to their TD peers. The current thesis provides partial support for the cognitive-energetic model of ADHD (Sergeant et al., 2003). Namely that arousal dysregulation may explain the observed differences in sleep onset and total sleep time between children with ADHD and their TD peers. Therefore, to generate converging evidence in support for arousal theories of ADHD, it is important for pediatric sleep researchers to collect data on measures of psychophysiological arousal, as well as circadian preferences. One such measure of psychophysiological arousal available to researchers is EMG heart rate variability. For example, Borger et al, (1999) found that children with ADHD had significantly higher baseline heart rate variability than their TD peers. Therefore, if children with ADHD demonstrate delayed sleep onset latencies, along with increased heart rate variability, it is plausible that children have arousal regulation issues.

There has also been a paucity of research pertaining to the circadian preferences (i.e., either evening- or morning-orientated) of children with ADHD. For example, there

has been only one published study (Caci, Bouchez, & Bayle, 2009), which found that adults with ADHD demonstrated more evening-oriented circadian preferences compared to TD peers. If children with ADHD are found to be more evening-oriented, then this circadian preference could be a factor which potential explains why children with ADHD show delayed sleep onset or shortened sleep duration. However, more research is required to examine both physiological arousal and circadian preferences in children with ADHD.

Overall the major theoretical contribution from the current thesis is that there does not appear to be sleep architecture differences between (1) children diagnosed with ADHD and their TD peers, or (2) between the ADHD subtypes. Results from the current thesis also have important clinical implications. First, clinicians should be advised that children with ADHD demonstrate longer sleep onset latencies and shorter sleep duration compared to TD peers. Also, past researchers (e.g., Corkum et al., 2008) have demonstrated that stimulant medications used to treat ADHD symptoms can significantly delay children's circadian phase. Therefore, clinicians should advise parents of these potential sleep side effects prior to starting a stimulant medication trial for their child's ADHD symptomology. There is a concern that treatment gains in terms of ADHD symptom reduction by stimulant medications may be undercut by sleep side effects (i.e., sleep onset, sleep duration).

The key finding from the current thesis was that children with ADHD take longer to fall asleep than their TD peers, and as such sleep less each night. Therefore, parents of children with ADHD should be advised to try to control for external factors that could impact their child's ability to settle, and subsequently to fall asleep. For example, such

factors include ensuring bed- and wake-times are consistent across the week, television and other electronic devices are turned off well prior to the child's bedtime, as well as children should abstain from caffeine and sugary foods, as well as rigorous physical activity prior to bed-time. Ensuring good sleep practices could influence their child's sleep routines and overall sleep so that positive sleep behaviours are subsequently forged as early as possible in development.

## ENDNOTES

<sup>1</sup>An additional MANOVA was conducted between the ADHD and TD groups and included both (1) the general sleep parameters (e.g., Sleep Onset, Sleep Duration), as well as the sleep architecture variables (e.g., Sleep Efficiency %; Latency to REM sleep) following the removal of any univariate or multivariate outlier scores. Specifically, three participants in the ADHD group were excluded from reanalyses as they had standardized scores greater than 3.29 SD's from group mean (Tabachnick & Fidell, 2007). In addition, the three typically developing (TD) participants whom they were matched with were also excluded from reanalyses.

Results from omnibus MANOVA failed to reveal significant effects for *Group* (ADHD vs TD)  $F(1,33) = 1.90, p = 0.081$ , following the removal of participants with outlier scores. Due to the lack of omnibus MANOVA effects, no additional analysis was conducted between ADHD-C/HI, ADHD-IA, TD groups.

<sup>2</sup>G\*power software was used to conduct an analyses of achieved statistical power between ADHD-C and ADHD-IA groups for sleep variables. Results for power analyses revealed that the power to detect a large effect (0.40), based on a total sample size of 25 participants, between two groups (ADHD-IA, ADHD-C/HI), was 0.71.

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## APPENDIX: Tables

**Table 1a**

*PSG Studies examining General Sleep Parameters and Sleep Architecture between children with ADHD and their TD peers*

Authors, year published	Sample Characteristics  ADHD Group	TD Group	Sleep Onset (Mins.)	Sleep Duration (Mins.)	REM periods	REM Latency (Mins.)	Sleep Efficiency (%)	*% NREM Sleep % Stage 1 Sleep <sub>1</sub> % Stage 2 Sleep <sub>2</sub> % Stage 3/4 Sleep <sub>3</sub>	REM Sleep %
Busby et al., 1981	11 Males Mean age: 10.6 years (SD= 1.7) ADHD Diagnosis based on rating scale scores from CPRS, CTRS, DSM-III criteria Exclusionary Criteria: Any reported health, sleep, or psychiatric conditions  ADHD participants medication-naïve prior to PSG testing; 5 PSG nights recorded for each child, total sleep time limited to 9.5 hours/nightly for each child	11 Males Mean age: 10.6 years (SD= 1.3)	ADHD = TD	ADHD = TD	--	ADHD > TD	ADHD = TD	ADHD > TD <sub>2</sub> ADHD < TD <sub>3</sub>	ADHD < TD
Busby & Pivik., 1985	8 Males Mean age: 11.0 years (SD= 1.4) ADHD Diagnosis based on rating scale scores from CPRS, CTRS, DSM-III criteria Exclusionary Criteria: Any reported health, sleep, or psychiatric conditions  4 consecutive nights of PSG completed with first night as “adaptation” night; 7 of 8 ADHD children on MPH during PSG	8 Males Mean age: 10.4 years (SD= 2.0)	ADHD > TD	ADHD = TD	--	ADHD = TD	--	--	--
Cooper et al., 2004	N= 18 (15 Males, 3 Females) Mean age: 10.5 years (SD= 3.0) ADHD diagnosis made by clinical psychologist using standardized psychometric testing (e.g., WISC-III, CBCL) Exclusionary Criteria: Not available  Single overnight PSG session, medication-naïve 24 hours prior to PSG testing	N=20 (11 Males, 9 Females) Mean Age: 10.0 years (SD= 3.9)	ADHD = TD	ADHD = TD	--	ADHD > TD	ADHD = TD	ADHD = TD <sub>1,2,3</sub>	ADHD = TD
Feinberg et al., 1974	8 Males Mean age: 8.8 years (7.7 – 10.5) Exclusionary Criteria: Not available	20 Children Mean age: 8.9 years (6.7 – 10.6)	ADHD = TD	ADHD = TD	--	--	ADHD = TD	--	ADHD = TD

Table 1a (continued)

Authors, year published	Sample Characteristics		Sleep Onset (Mins.)	Sleep Duration (Mins.)	REM periods	REM Latency (Mins.)	Sleep Efficiency (%)	*% NREM Sleep		
	ADHD Group	TD Group						% Stage 1 Sleep <sub>1</sub>	% Stage 2 Sleep <sub>2</sub>	% Stage 3/4 Sleep <sub>3</sub>
Galland et al., 2011	N=28 (22 Males, 6 Females) Mean age: 10.1 years (6.7 – 12.4) ADHD diagnosis made by clinical psychologist using standardized psychometric testing Exclusionary Criteria: Not available ADHD and TD children matched according to Sex and Age; Children in ADHD group were medication-naïve for 48 hours prior to PSG testing; 2 nights of PSG data collected, PSG data from TD group was compared with ADHD medication-naïve data	N=28 (22 Males, 6 Females) Mean age: 10.2 years (6.6 – 12.3)	ADHD >TD	ADHD = TD	--	ADHD = TD	ADHD > TD	ADHD < TD		ADHD = TD
Golan et al., 2004	N=34 (26 Males, 8 Females) Mean age: 12.4 years (SD= 4.6) ADHD diagnosis made by clinical psychologist using standardized psychometric testing Exclusionary Criteria: Not available Children in ADHD group were medication-naïve for at least 72 hours prior to PSG testing; All children completed a single overnight PSG testing session	N=32 (21 Males, 11 Females) Mean age: 12.0 years (SD= 3.6)	ADHD = TD	ADHD = TD	--	--	ADHD = TD	ADHD = TD <sub>1,2,3</sub>		ADHD > TD
Greenhill et al., 1983	12 Males Mean Age: 8.6 years (6.7 – 10.7) Diagnoses of ADDH based on DSM-III scales from CPRS and CTRS rating scales Exclusionary Criteria: Any reported health, sleep, or psychiatric conditions Children in ADHD group medication-naïve for at least 14 days prior to PSG testing; 2 consecutive overnight PSG testing sessions	N=11 (4 Males, 7 Females) Mean Age: 9.3 years (8.3 – 11.8)	ADHD = TD	ADHD = TD	--	--	ADHD = TD	ADHD = TD		ADHD < TD
Grissom et al., 2009	N=13 Age range: 6 – 10 years Children had previously been diagnosed with ADHD using methods unreported by authors Exclusionary Criteria: Co-morbid health issues	N=16 Age range: 6 – 10 years	ADHD = TD	ADHD = TD	ADHD = TD	ADHD = TD	ADHD = TD	ADHD < TD <sub>2</sub> ADHD > TD <sub>3</sub>		ADHD = TD

**Table 1a** (continued)

<b>Authors, year published</b>	<b>Sample Characteristics</b>		<b>Sleep Onset (Mins.)</b>	<b>Sleep Duration (Mins.)</b>	<b>REM periods</b>	<b>REM Latency (Mins.)</b>	<b>Sleep Efficiency (%)</b>	<b>*% NREM Sleep % Stage 1 Sleep<sub>1</sub> % Stage 2 Sleep<sub>2</sub> % Stage 3/4 Sleep<sub>3</sub></b>	<b>REM Sleep %</b>
	<b>ADHD Group</b>	<b>TD Group</b>							
Gruber et al., 2009	N=15 (10 Males, 5 Females) Mean Age: 8.93 years (SD= 1.39) Children diagnosed with ADHD following parental completion of diagnostic interview (DISC-IV), and ADHD rating scales (e.g., CPRS) Exclusionary Criteria: Not Available Children in ADHD sample were stimulant-medication free for at least 7 days prior to PSG testing. PSG evaluation was performed using portable PSG device, with each child slept in their own bedroom, instead of at sleep lab	N=23 (13 Males, 10 Females) Mean Age: 8.61 years (SD= 1.27)	ADHD = TD	ADHD < TD	--	--	ADHD = TD	ADHD = TD <sub>1,2,3</sub>	ADHD < TD
Haig et al., 1974	6 Males Mean Age range: 8.7 – 14.6 years Children in experimental group defined as behaviorally as “severely hyperactive”, following assessment in an interdisciplinary clinic Exclusionary Criteria: Not Available PSG testing was conducted over 5 consecutive nights in sleep lab, where first two night’s considered adaptation nights	6 Males Mean Age Range: 6.5 – 12 years	ADHD > TD	ADHD = TD	--	ADHD > TD	--	ADHD = TD	ADHD = TD
Huang et al., 2004	N= 88 (77 Males, 11 Females) Mean Age: 8.46 years (SD= 1.85) Children diagnosed with ADHD following structured clinical interview with two psychiatrists Exclusionary Criteria: Children assessed by pediatrician and neurologist with co-morbid pervasive developmental disorder, seizure, anxiety disorder, CNS disorders or physical health conditions Children in ADHD group had been stimulant medication-free for at least 9 days prior of PSG testing. All PSG testing completed in hospital sleep lab facility	N= 27 (21 Males, 6 Females) Mean Age: 9 years (SD= 1.96)	ADHD = TD	ADHD < TD	--	ADHD < TD	ADHD < TD	ADHD < TD <sub>3</sub>	ADHD > TD

**Table 1a** (continued)

<b>Authors, year published</b>	<b>Sample Characteristics</b>		<b>Sleep Onset (Mins.)</b>	<b>Sleep Duration (Mins.)</b>	<b>REM periods</b>	<b>REM Latency (Mins.)</b>	<b>Sleep Efficiency (%)</b>	<b>*% NREM Sleep % Stage 1 Sleep<sub>1</sub> % Stage 2 Sleep<sub>2</sub> % Stage 3/4 Sleep<sub>3</sub></b>	<b>REM Sleep %</b>
	<b>ADHD Group</b>	<b>TD Group</b>							
Khan &Rechtshaffen., 1978	5 Males Mean Age Range: 6.1 – 8.5 years Exclusionary Criteria: Not Available	N=7 (5 Males, 2 Females) Mean Age Range: 6.7 – 8.8 years 3 children in ADHD group were taking stimulant medication during PSG testing; PSG testing conducted on 3 nights in sleep lab; including first night as adaptation night	--	ADHD = TD	--	--	--	ADHD < TD	--
Kirov et al., 2007	N=19 (18 Males, 1 Female) Mean Age: 11.07 years (SD= 2.26) ADHD diagnosis made by clinical psychologist using standardized psychometric testing Children in ADHD group diagnosed following comprehensive assessment with two independent psychiatrists. Exclusionary Criteria: Children excluded from participation if, “presence of internal or neurological problems associated with ADHD.” Children in ADHD group with medication-free for at least 7 days prior to PSG testing. ADHD and TD participants matched according to Age, Gender, and IQ, with PSG testing conducted over 2 nights in sleep lab facility that included first night as adaptation night	N=19 (17 Males, 2 Females) Mean Age: 11.09 years (SD= 2.23)	ADHD = TD	ADHD > TD	--	ADHD < TD	ADHD = TD	ADHD = TD	ADHD > TD
Konofal et al., 2001	N=30 Mean Age: 7.8 years (SD= 1.6) Children in ADHD group diagnosed following comprehensive assessment by four independent psychiatrists. Exclusionary Criteria: Any co-morbid health, psychiatric disorders including anxiety and depression, or sleep disorders ADHD group medication-naïve prior to PSG testing; ADHD and TD groups matched according to Age and Gender. PSG testing in hospital sleep lab for three nights before PSG data recorded	N=19 Mean Age: 8.4 years (SD= 1.4)	ADHD = TD	ADHD = TD	--	ADHD = TD	ADHD = TD	ADHD = TD <sub>1,2,3</sub>	ADHD = TD

Table 1a (continued)

Authors, year published	Sample Characteristics ADHD Group	TD Group	Sleep Onset (Mins.)	Sleep Duration (Mins.)	REM periods	REM Latency (Mins.)	Sleep Efficiency (%)	*% NREM Sleep % Stage 1 Sleep <sub>1</sub> % Stage 2 Sleep <sub>2</sub> % Stage 3/4 Sleep <sub>3</sub>	REM Sleep %
Lecendreux et al., 2000	N=30 Mean Age: 7.8 years (SD= 1.6) Children in ADHD group diagnosed following comprehensive assessment by four independent psychiatrists. Exclusionary Criteria: Any co-morbid health, psychiatric disorders including anxiety and depression, or sleep disorders All children in ADHD group medication-naïve prior to PSG testing; ADHD and TD groups matched according to Age and Gender. PSG testing in hospital sleep lab where children had slept in bedroom for three nights before PSG data recorded	N=22 Mean Age: 8.4 years (SD= 1.4)	ADHD = TD	ADHD = TD	--	ADHD = TD	ADHD = TD	ADHD = TD <sub>1,2,3</sub>	ADHD = TD
Miano et al., 2006	N=20 (18 Males, 2 Females) Mean Age: 9.3 years (6 – 13 years) ADHD diagnosis made by clinical psychologist using semi-structured clinical interview, along with parent-report rating scales Exclusionary Criteria: Any co-morbid neurological disorders, including epilepsy. ADHD and TD children matched according to Age; All children medication-naïve prior to PSG testing, PSG collected over two nights in sleep lab facility; first night served as adaptation night	N=20 (11 Males, 2 Females) Mean Age: 8.4 years (6 – 13 years)	ADHD = TD	ADHD < TD	--	ADHD =TD	ADHD = TD	ADHD = TD <sub>1,2,3</sub>	ADHD = TD
Nahas & Krynicki, 1977	4 Males Mean Age: 8.4 years (6.1 – 8.5) Exclusionary Criteria: Not Available Children in ADHD group stimulant medication-free for 21 days prior to PSG testing; PSG collected over 1 or 2 nights in sleep lab	“Normative Data”	--	--	--	--	--	ADHD > TD <sub>2</sub> ADHD < TD <sub>3</sub>	ADHD < TD
O’Brien et al., 2003a	N=47 (35 Males, 12 Females) Mean Age 8.0 years (SD= 1.6) Children diagnosed with ADHD following assessment with clinical psychologists	N=49 (22 Males, 27 Females) Mean Age: 6.7 years	ADHD = TD	--	--	ADHD > TD	ADHD = TD	ADHD = TD	ADHD < TD

**Table 1a** (continued)

Authors, year published	Sample Characteristics		Sleep Onset (Mins.)	Sleep Duration (Mins.)	REM periods	REM Latency (Mins.)	Sleep Efficiency (%)	% NREM Sleep % Stage 1 Sleep <sub>1</sub> % Stage 2 Sleep <sub>2</sub> % Stage 3/4 Sleep <sub>3</sub>	REM Sleep %
	ADHD Group	TD Group							
O'Brien et al., 2003b	N=34 (18 Males, 16 Females) Mean Age 6.5 years (SD= 1.5) Children diagnosed with ADHD following assessment with clinical psychologists and psychiatrists Exclusionary Criteria: Any co-morbid psychiatric diagnosis that could impact sleep, as assessed by psychologist and pediatrician Children in ADHD group medication-naïve prior to PSG testing; PSG testing conducted in a sleep lab facility over a single night session	N=53 (23 Males, 30 Females) Mean Age: 6.6 years (SD= 0.6)	ADHD = TD	ADHD < TD	--	ADHD = TD	ADHD = TD	ADHD = TD	ADHD < TD
Palm et al., 1992	N=10 (8 Males, 2 Females) Mean Age: 9.8 years (6.9 – 12.3) Children diagnosed with ADDH following assessment by independent physician and clinical psychologist Exclusionary Criteria: Children diagnosed with severe neurotic or psychotic disorders All children medication-naïve prior to PSG testing; PSG evaluation was performed using portable PSG device, with each child slept in their own bedroom, instead of at sleep lab over 2 nights	N=18 (9 Males, 9 Females) Mean Age: 10.7 years (8.8 – 12.6)	ADHD > TD	ADHD = TD	--	ADHD > TD	ADHD < TD	ADHD = TD <sub>1,2,3</sub>	ADHD = TD
Pichietti et al., 1999	N=14 (13 Males, 1 Female) Mean Age: 8.2 years (5 – 12 years) ADHD diagnoses made by a single pediatric neurologist at a sleep clinic Exclusionary Criteria Children diagnosed with seizure disorders, sleep apnea, or another chronic childhood disease All ADHD children medication-naïve prior to PSG testing. PSG testing conducted over a single session at sleep lab facility	N=10 (5 Boys, 5 Girls) Mean Age 11.1 years (9.9 – 12.7 years)	ADHD = TD	ADHD < TD	--	--	ADHD = TD	ADHD = TD	ADHD = TD
Poitras et al., 1981	4 Males Mean Age Range: 8 – 12 years	4 Males Mean Age	--	--	--	--	--	ADHD > TD <sub>2</sub>	--

Table 1a (continued)

Authors, year published	Sample Characteristics		Sleep Onset (Mins.)	Sleep Duration (Mins.)	REM periods	REM Latency (Mins.)	Sleep Efficiency (%)	*% NREM Sleep % Stage 1 Sleep <sub>1</sub> % Stage 2 Sleep <sub>2</sub> % Stage 3/4 Sleep <sub>3</sub>	REM Sleep %
	ADHD Group	TD Group							
Prihadova et al., 2010	N=31 (26 Males, 5 Females) Mean Age: 9.3 years (SD= 1.7) ADHD diagnoses made by psychiatrist who collected parent-rating scales (e.g., CPRS), as well as completed structured diagnostic interview Exclusionary Criteria: Free from any chronic physical health, sleep, neurological, or psychiatric conditions All ADHD children medication-naïve prior to PSG testing. PSG testing conducted over a two consecutive nights at sleep lab facility, where first night served as adaptation night.	N=26 (22 Males, 4 Females) Mean Age: 9.2 years (SD- 1.5)	ADHD = TD	ADHD = TD	--	ADHD = TD	ADHD = TD	ADHD = TD <sub>1</sub>	ADHD = TD
Ramos Platon et al., 1990	N=13 (9 Males, 4 Females) Mean Age: 9 years (SD=1.73) Children diagnosed with ADD/H or ADD/WO following rigorous physical, neurological, and psychological assessment. Exclusionary Criteria: Evidence of psychosis, severe affective disorder, or neurological disturbance 10 children met DSM-III criteria for ADD/H; 3 with ADHD/WO; All children in experimental group were stimulant medication free for at least one month prior to PSG testing. Each child completed 2 overnight stays in sleep lab, where first night adaptation night, second, PSG data obtained	43 Normative sleep data from TD participants who completed another study by authors	ADHD < TD	ADHD > TD	ADHD = TD	--	ADHD < TD	ADHD < TD <sub>1</sub> ADHD > TD <sub>3</sub>	ADHD < TD
Sangal et al., 2005	N=40 Age Range: 6 – 14 years ADHD diagnosis made by clinical psychologist using standardized psychometric testing Exclusionary Criteria: Any co-morbid primary health, sleep disorders or psychiatric disorders ADHD children medication-naive	Normative sleep data	ADHD = TD	ADHD = TD	--	ADHD = TD	ADHD = TD	ADHD = TD	ADHD = TD



Table 1a (continued)

Authors, year published	Sample Characteristics		Sleep Onset (Mins.)	Sleep Duration (Mins.)	REM periods	REM Latency (Mins.)	Sleep Efficiency (%)	*% NREM Sleep % Stage 1 Sleep <sub>1</sub> % Stage 2 Sleep <sub>2</sub> % Stage 3/4 Sleep <sub>3</sub>	REM Sleep %
	ADHD Group	TD Group							
Silvestri et al., 2009	N=55 (47 Males, 8 Females) Mean Age: 8.9 years (SD= 2.7 years) ADHD diagnosis made by clinical psychologist using semi-structured clinical interview, along with parent-report rating scales Exclusionary Criteria: Any co-morbid primary health, sleep disorders or psychiatric disorders  Children in ADHD group were medication-naïve prior to PSG testing and age-, and sex matched with a sample of 20 of their TD peers. All children completed a single overnight PSG session at sleep lab facility	N=20	ADHD = TD	ADHD < TD	--	ADHD > TD	ADHD < TD	ADHD > TD <sub>2,3</sub>	ADHD < TD
Small et al., 1971	3 Males Mean Age: 8.2 years (7.8 – 8.11) Exclusionary Criteria: Not Available  Researchers compared PSG data from experimental group's placebo nights to TD group PSG data. Study included adaptation night and 5 placebo nights.	N=7 Mean Age: 8.4 (4.8 – 10.9)	ADHD < TD	ADHD = TD	--	--	ADHD = TD	ADHD = TD	ADHD = TD
Stahl et al., 1979	N=5 Mean Age Range: 6 – 12 years Exclusionary Criteria: Not Available  One child in experimental group on stimulant medication with other children medication-free for 2 and 3 weeks respectively. PSG sessions over 3 or 4 nights in sleep lab, included adaptation night	N=5 Mean Age Range: 6 – 12 years	--	ADHD = TD	--	--	ADHD < TD	ADHD = TD	ADHD = TD

\*Indicates that potential differences in overall NREM sleep between ADHD and TD groups were investigated. Subscripts indicate results from specific NREM sleep stages.

**Table 1b***Summary of 28 empirical PSG studies examining differences between children with ADHD and TD peers*

	<b>PSG Sleep Onset</b>	<b>PSG Sleep Duration</b>	<b>REM Periods</b>	<b>REM Latency</b>	<b>Sleep Efficiency (%)</b>	<b>Total NREM Sleep (%)</b>	<b>Stage 1 NREM Sleep (%)</b>	<b>Stage 2 NREM Sleep (%)</b>	<b>Stage 3/4 NREM Sleep (%)</b>	<b>REM Sleep (%)</b>
<b>ADHD = TD</b>	18 (75%)	17 (68%)	2 (100%)	10 (58.8%)	17 (73.9%)	9 (82%)	8 (89%)	7 (58.4%)	7 (53.8%)	14 (56%)
<b>ADHD &gt; TD</b>	4 (16.7%)	2 (8%)	0	5 (29.5%)	1 (4.4%)	0	0	4 (33.3%)	3 (23%)	3 (12%)
<b>ADHD &lt; TD</b>	2 (8.3%)	6 (24%)	0	2 (11.7%)	5 (21.7%)	2 (18%)	1 (11%)	1 (8.3%)	3 (23%)	8 (32%)
<b>Total</b>	<i>24</i>	<i>25</i>	<i>2</i>	<i>17</i>	<i>23</i>	<i>11</i>	<i>9</i>	<i>12</i>	<i>13</i>	<i>25</i>

Note: Values in brackets represent number of studies in percentages out of total

**Table 2**

*Primary research question: Demographic Characteristics of ADHD versus TD participants*

Source	ADHD Group (n=25; 22 males, 3, females)	TD Group (n=25; 22 males, 3, females)	t	p-value
Age (Months)	105.72 (22.57)	103.92 (21.07)	0.292	.772
CPRS Score	73.32 (8.11)	47.17 (7.06)	11.86	.012*
Total number of children in household	2.24 (0.83)	2.50 (1.00)	1.07	.288
<sup>A</sup> Hollingshead SES	62.46 (21.84)	63.94 (15.41)	0.277	.783
<sup>B</sup> Family Income	5.6 (3.1)	6.6 (2.96)	1.17	.249

\*Significant effect at a  $p < .05$  level

<sup>A</sup>Indicates average parental value for Hollingshead Socioeconomic (SES)

<sup>B</sup>Family Income was reported by parents according to nominal scale where a value of 4 = \$51,000 – \$60,000, 5 = \$61,000 - \$70,000, 6 = \$71,000 - \$80,000, 7 = \$81,000 - \$90,000, 8 = \$91,000 - \$100,000, 9 = \$100,000+

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**Table 3***General Sleep Parameters between ADHD & TD Groups*

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Source	ADHD Group (22 Boys, 3 Girls)	TD Group (22 Boys, 3 Girls)	p-value
PSG “Lights Out” Time	9:33 PM (40 mins)	9:38 PM (34 mins)	.632
PSG “Lights On” Time	06:44 AM (47 mins)	07:05 AM (45 mins)	.111
Sleep Onset Latency in minutes	47.24 (38.61)	24.07 (14.01)	.007**
Sleep Duration in minutes	466.01 (69.01)	500.79 (50.43)	.047*

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\*Indicates a significant effect at a  $p < 0.05$  level; \*\*Indicates a significant effect at  $p < 0.01$  level  
NOTE: Numbers presented in brackets represent SD

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**Table 4***Sleep Architecture between ADHD and TD Groups (MANOVA)*

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Source	ADHD Group	TD Group	p-value
Sleep Efficiency (%)	84.06 (9.13)	84.46 (6.37)	.861
Latency to REM Sleep	138.44 (59.31)	131.86 (47.17)	.666
Number of REM Periods	4.72 (1.31)	4.72 (0.93)	1.00
Percentage of Stage 1 Sleep	5.78 (2.66)	4.99 (2.15)	.253
Percentage of Stage 2 Sleep	43.68 (9.2)	45.44 (8.84)	.493
Percentage of Stage 3/4 Sleep	27.79 (9.16)	28.44 (8.6)	.796
Percentage of REM Sleep	22.71 (5.21)	21.08 (3.92)	.219

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NOTE: Numbers presented in brackets represent SD

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**Table 5***Demographic Characteristics of ADHD-IA, ADHD-C, TD Groups*

Source	ADHD-IA Group (n=12)	ADHD-C/HI Group (n=13)	TD Group (n=25)	p-value
Age in months	114.33 (24.22)	97.77 (18.38)	103.92 (21.07)	0.06
Total number of children in household	2.25 (0.86)	2.23 (0.83)	2.52 (1.00)	.955
<sup>A</sup> Hollingshead SES	66.50 (20.72)	58.73 (23.01)	63.94 (15.41)	.386
<sup>B</sup> Family Income	6.33 (2.64)	4.92 (3.42)	6.60 (2.96)	.264

<sup>A</sup>Indicates average parental value for Hollingshead Socioeconomic (SES)

<sup>B</sup>Family Income was reported by parents according to nominal scale where a value of 4 = \$51,000 – \$60,000, 5 = \$61,000 - \$70,000, 6 = \$71,000 - \$80,000, 7 = \$81,000 - \$90,000, 8 = \$91,000 - \$100,000, 9 = \$100,000+

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**Table 6**

*General Sleep and Sleep Architecture between ADHD subtypes and TD peers (MANCOVA)*

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Source	ADHD-IA n=12	ADHD-C/HI n=13	T n=25
*Sleep Onset Latency in minutes	39.32 (28.62)	54.54 (45.93)	24.07 (14.01)
*Sleep Duration in minutes	443.84 (81.11)	486.47 (50.51)	500.79 (50.43)

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TABLE NOTE: Numbers presented in brackets represent SD

\*Indicates statistically significant difference at a  $p < 0.05$  level between TD group and ADHD subtypes. Helmert contrasts revealed no significant differences between ADHD-IA, and ADHD-C/HI groups for any of the general sleep parameters or sleep architecture variables

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