POSITION STATEMENT ON PEDIATRIC SLEEP

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EXECUTIVE SUMMARY

INTRODUCTION: Sleep is an essential component of healthy development and is required for physical and mental health. Sleep deprivation and sleep disorders are highly prevalent among Canadian children, and these problems are under-reported, under-recognized, under-diagnosed, and often untreated. The objectives of this consensus statement are to: 1) describe the role of sleep in the physical and mental health of children and adolescents (0-19 years), 2) review normal sleep development and knowledge of the effects of sleep deprivation on children and adolescents (0-19 years), 3) provide health care practitioners with counseling strategies to prevent sleep deprivation, and 4) provide health care practitioners with existing evidence-based guidelines and resources regarding the evaluation and treatment of pediatric sleep disorders The rationale for developing this statement is that sleep deprivation and sleep disorders have a pervasive negative impact on the health, cognitive function and socio-emotional regulation, quality of life, and future health trajectories of children and adolescents, and greater resources are necessary to optimize the care of children with sleep deprivation and disorders. The intended audience for this statement includes pediatric healthcare practitioners, particularly pediatricians, family physicians and psychiatrists. This statement represents the outcome of a Canadian Institute of Health Research (CIHR) funded workshop held at McGill University in Montreal on May 4, 2012 which was attended by over 30 thought leaders in the areas of pediatric sleep, psychology, pediatrics, and education. Individuals in attendance represented a broad range of professional organizations including the Canadian Pediatric Society (CPS), Canadian Psychological Association (CPA), Canadian Sleep Society (CSS), Canadian Academy of Child and Adolescent Psychiatry (CACAP), Québec National Institute of Public Health (INSPQ), the Riverside School Board, as well as a number of academic institutions in North America.

CURRENT STATE OF SLEEP HEALTH IN CANADIAN CHILDREN AND ADOLESCENTS

An extensive scientific literature confirms the importance of healthy sleep to human growth and development, metabolism and weight regulation, immune function, accident risk, learning, memory and executive function, and emotional health and regulation. Inadequate or unhealthy sleep is associated with decreased performance or function across these domains, which translates into significant economic and societal costs.

S**leep deprivation**: Despite very strong evidence indicating the importance of sleep, the past century has seen consistent and rapid declines in the sleep duration of children and adolescents. Cross-sectional surveys of Canadian high school students have revealed that as many as 70% of students get less than the recommended amount of sleep for their age, they tend to show an increasing gap in sleep on school days versus non-school, and more than half report feeling excessively tired or sleepy during the day. Modern lifestyle factors that affect sleep and sleep regulation include electronic media, excessive light exposure late in the evening, inappropriate caffeine consumption, and the low priority given to sleep by families and society in general. Effective strategies to address sleep deprivation include using routine health visits to provide

parental education on normative sleep habits, identification of unhealthy sleep patterns, counseling on the benefits of healthy sleep, and identification of factors contributing to unhealthy sleep. Motivational interviewing may help elicit and improve the probability for positive change.

Sleep disorders: Common categories of sleep disorders in children include insomnias, sleep-related breathing disorders, hypersomnias, circadian rhythm sleep disorders, and abnormal movements and behaviors in sleep (parasomnias). These problems are common in the general pediatric population, and are often under-recognized and untreated. Evaluation consists of a thorough history and physical examination by a knowledgeable clinician, and in some cases, overnight polysomnography (sleep study) to record physiological data. Sleep disorders in children require an individualized treatment plan developed by a knowledgeable clinician; some sleep disorders are time-limited and resolve uneventfully while others are best managed using a chronic disease model with longitudinal follow-up.

Evaluation and management issues: A thorough history and physical examination provides the foundation for evaluation and diagnosis. Many children with sleep issues can be managed effectively by the primary care provider, but children with more complex disorders or those who have not responded well to initial management may require referral to a sleep center and/or a mental health professional. Primary care providers are in an ideal position to promote healthy sleep by providing parents and children with basic information about sleep, sleep hygiene, and the impact of sleep deprivation. When there is concern regarding sleep-related breathing disorders, polysomnography is often indicated, and referral to a qualified sleep center is necessary. Whether every child should undergo diagnostic polysomnography prior to adenotonsillectomy is controversial, and in some communities polysomnography is unavailable or associated with long wait times. In these situations, surgical intervention such as adenotonsillectomy should not be delayed when there is strong clinical suspicion for severe obstructive sleep apnea. There is increased recognition of the role of positive airway pressure (PAP) use in children with residual upper airway obstruction following adenotonsillectomy, or in children with obesity and no adenotonsillary hypertrophy.

Barriers to sleep health in Canada: Barriers to prevention of sleep deprivation and treatment of pediatric sleep disorders in the current system include 1) inadequate awareness or knowledge deficits by parents, healthcare providers, and policy-makers regarding sleep in children, 2) under-utilization of existing evidence-based recommendations or guidelines regarding sleep disorders in children, and 3) uneven distribution of sleep centers with experience in pediatric sleep and tertiary care centers to provide comprehensive evaluation and management.

RECOMMENDATIONS AND EVIDENCE-BASED RESOURCES:

Health care professions should screen for sleep deprivation and sleep disorders as part of their routine interactions with children and adolescents. Polysomnography should be considered whenever there is concern for significant respiratory disturbance during sleep, atypical or potentially injurious parasomnias, and as part of the evaluation of suspected narcolepsy. Consultation with mental health professions should be initiated when children with insomnia have not responded to initial interventions, or when there are significant co-morbidities such as anxiety, mood disorders, post-traumatic stress disorder, or autistic spectrum disorders. Health

care professionals should follow available evidence-based guidelines for healthy sleep in children and families, and should advocate for improved awareness and promotion of sleep-friendly activities and schedules for children, inclusion of sleep health into the school curriculum, and integration of sleep health into programs and interventions that target obesity. Professionals involved with medical education and health policy decisions should make sleep health a priority through improved education at the medical school and residency levels, and through expansion of fellowship programs in sleep medicine. The high prevalence of sleep deprivation and disorders in children, coupled with limited resources in many communities, indicates that sleep must become a high priority for health care professionals, parents, educators, public policy-makers, and society-at-large.

CONSENSUS STATEMENT ON PEDIATRIC SLEEP:
GUIDELINES AND RECCOMENDATIONS FOR HEALTH CARE PROFESSIONALS` PROMOTION OF OPTIMAL CHILD AND ADOLESCENT DEVELOPMENT

Sleep is an essential component of healthy development and is required for physical and mental health. Unfortunately, sleep deprivation and sleep disorders are highly prevalent among Canadian children and adolescents (0-19 years). A recent cross-sectional survey of 3235 Canadian adolescents revealed that 70% attain less than the recommended number of hours of sleep per night (Gibson et al., 2006). In addition, 25-50% of youth are affected by some type of sleep disorder during infancy, childhood and/or or adolescence (Davis, Parker, & Montgomery, 2004; Mindell et al., 1997). Despite being associated with numerous physical and mental health problems and injury risks across the lifespan, as well as the loss of productivity and mortality in the adult years, chronic sleep insufficiency is under-recognized as a public health problem. Although the etiology of sleep deprivation and sleep disorders is multifactorial, it is known that environmental factors and daily habits that interfere with sleep play a key role. As well, there is currently a lack of access to evidence-based tools for preventing sleep deprivation and treating pediatric sleep disorders, as well as a lack of both clinician and public awareness of the importance of sleep, of habits that interfere with sleep, and of information about how to prevent sleep deprivation.

Despite the wealth of strong empirical evidence indicating that sleep disorders are very common within the pediatric population and have serious consequences for both children and their families, such disorders are commonly under-diagnosed (Meltzer et al., 2010). Furthermore, even when sleep issues are identified, healthcare professionals frequently lack the knowledge required to use effective evidence-based behavioral approaches to treat pediatric sleep disorders. For example, healthcare professionals often prescribe pharmacological interventions that are not approved by the (US) Food and Drug Administration for the pediatric age range or the specific sleep disorder in question. Stojanovski et al. (2007) examined the patterns of physicians prescribing medications for children with sleep difficulties in outpatient settings, and found that 81% of children with pediatric insomnia were given sleep medication (Stojanovski et al., 2007). This is particularly remarkable given that these medications are not approved for use in this context and have been shown to be ineffective in the long term.

This occurs because most healthcare professionals receive very limited training in sleep medicine. For example, preclinical medical students receive only ~ 1 hour of sleep education on average. Moreover, 37% of medical schools reported offering no training on sleep or sleep disorders, and even less training is offered at the internship and residency levels, with only 23% of programs featuring at least 1 hour of training on sleep (Mindell et al., 1994; Rosen et al., 1993; Moline & Zendell, 1993). It is therefore essential that we improve the knowledge of healthcare providers regarding the availability and appropriate use of evidence-based treatment options for sleep disorders, in order to ensure that patients receive safe, effective, and comprehensive care. Significant improvement is possible if healthcare professionals can be given adequate information and tools that allow for evidence-based diagnosis, prevention, and interventions related to pediatric sleep problems. Thus, the ***objective*** of this statement is to provide pediatric-sleep-related information, tools and resources to physicians, especially

pediatricians, family physicians and psychiatrists, who frequently see youths with such issues in their routine practice. Such an effort will facilitate the integration of effective evidence-based assessment, prevention and intervention into current practice.

The objectives of this statement are to: 1) describe the role of sleep in the physical and mental health of children and adolescents, 2) review normal sleep development and knowledge of the effects of sleep deprivation on children and adolescents; 3) provide health care practitioners with counseling strategies to prevent sleep deprivation, and 4) provide health care practitioners with existing evidence-based guidelines and resources regarding the evaluation and treatment of pediatric sleep disorders. The intended audience for this statement includes pediatric healthcare practitioners, particularly pediatricians, family physicians and psychiatrists.

At the onset of this review, it is useful to clarify some of the key terminologies used throughout the text. As such, the following paragraph will make a distinction between the terms "sleep deprivation", "sleep disorders", or "sleep difficulties".

Sleep deprivation refers to the inability to obtain a sufficient amount of sleep. There are many reasons why a person may not get enough sleep at any given moment; however, sleep deprivation (without the presence of a sleep disorder) is caused by lifestyle factors, and thus, can be addressed through lifestyle modification alone. Sleep-disrupting lifestyle factors, if not changed, can also lead to the development of a *sleep disorder* (for example, the habitual consumption of caffeine at bedtime can cause insomnia); however, a sleep disorder can only be remedied through clinical intervention. Sleep disorders are associated with sleep difficulties, including long sleep-onset latency, restless sleep, frequent awakenings, and morning fatigue. Sleep deprivation may not only trigger a sleep disorder, it can also be the *consequence* of having a sleep disorder. Overall, though sleep deprivation and sleep disorders may lead to similar health detriments, they require different interventional strategies (i.e., lifestyle modifications vs**.** clinical intervention) to be addressed.

1. THE BENEFITS OF SLEEP AND THE HARMFUL EFFECTS OF SLEEP DEPRIVATION ON CHILDREN'S PHYSICAL AND MENTAL HEALTH

The World Health Organization (WHO) defines health as a state of complete physical, mental and social well-being. Healthy and productive days are dependent on healthy nights. This section will review the scientific evidence showing that sleep is essential for optimal physical and mental health, as well as provide evidence that poor sleep is implicated in the development and persistence of several childhood disorders affecting mental and physical health.

1.1 SLEEP AND PHYSICAL HEALTH

Sleep is essential for adequate weight control, efficient immune responses, cardiovascular health, and prevention of injuries.

Body Weight. Sleep modulates levels of the hormones leptin and ghrelin, which are involved in regulating metabolism and controlling appetite (Bassett et al., 2004; K. Spiegel et al., 2004). Shortened, more variable sleep patterns have been linked to an increased risk of overweight and obesity among children and adolescents (Cappuccio et al., 2008; Chaput et al., 2011; Magee, Huang, Iverson, & Caputi, 2010). Direct and indirect pathways have been proposed to explain this relationship. In terms of a direct pathway, leptin and ghrelin regulate appetite in opposing directions (Taheri, Lin, Austin, Young, & Mignot, 2004); with reduced sleep, decreased leptin levels and increased ghrelin levels cause individuals to feel hungrier and eat more throughout the day, leading to obesity (Gunther et al., 2004; Spiegel, Tasali, Penev, & Van Cauter, 2004; Taheri et al., 2004). Regarding an indirect pathway, insufficient sleep may increase daytime fatigue or sleepiness, leading children to reduce physical activity and increase sedentary activities, thereby burning fewer calories.

Immune Response. Sleep enhances the antigen-specific immune defense system. This has been demonstrated by the increased production of antibodies following vaccination among well-rested, compared to sleep-derived, individuals (Lange, Perras, Fehm, & Born, 2003). Sleep deprivation significantly reduces cellular immunity and impairs cytokine function.(Bryant, Trinder, & Curtis, 2004; Irwin, 2002; Kamdar, Needham, & Collop, 2012).

Risk of Injury. In young children, lack of sleep increases the risk of injury from accidental falls (Boto et al., 2012) and is associated with a higher number of medically-attended injuries in general (Koulouglioti, Cole, & Kitzman, 2008). The single largest cause of mortality in adolescence is death due to unintentional injuries, particularly fatal automobile crashes. Compelling evidence shows that sleep deprivation (e.g., 18-24 hours of continuous wakefulness) impairs attention, reaction time, and judgment to levels that are comparable to those seen with alcohol intoxication over the legal limit (Arnedt, Wilde, Munt, & MacLean, 2000; Falleti, Maruff, Collie, Darby, & McStephen, 2003; Powell et al., 2001; Williamson & Feyer, 2000; Williamson, Feyer, Mattick, Friswell, & Finlay-Brown, 2001). Moreover, young people are particularly vulnerable to sleepiness and nighttime crashes, with growing evidence suggesting that the majority of drowsy-driving-related crashes are caused by teenagers and young adults (Horne & Reyner, 1995; Lyznicki, Doege, Davis, & Williams, 1998; McConnell, Bretz, & Dwyer, 2003; Pack et al., 1995). Sleep deprivation in adolescents may contribute to driving risk in at least four ways: (1) lapses in attention/falling asleep while driving; (2) impaired judgment and decision making, leading to impulsive and risky behavior; (3) the negative additive effects of alcohol and sleep deprivation; and (4) reduced impulse control and increased reactive aggression when conflictual situations arise. In contrast, sleep extension has been shown to decrease the risk of car accidents amongst adolescents (Danner & Phillips, 2008).

1.2 SLEEP AND MENTAL HEALTH

According to the World Health Organization (WHO), mental health is defined as a state of well-being in which an individual realizes his or her own potential, can cope with the normal stresses of life, can work productively and fruitfully, and can contribute to his or her community ("Mental Health: A State of Well-being," 2011). Sleep plays a significant role in a child's realization of his or her academic potential and ability to cope with stress, regulate emotions, socialize and be productive.

1.2.1 Realization of academic potential

Sleep deprivation compromises the function of specific brain areas involved with key processes and skills required for academic success, including learning, memory, intelligence, executive functions (EFs), and emotional regulation.

Learning and memory. Sleep plays an integral role in "off-line" memory processing among children and adults, especially in the consolidation of memories, which is essential for retaining new information (Kopasz et al., 2010; Stickgold & Walker, 2005). Inadequate, irregular, and poor quality sleep can negatively impact neurobehavioral functioning, learning and memory in children (Dworak, Schierl, Bruns, & Struder, 2007; Sadeh, Gruber, & Raviv, 2002), and sleep-deprived subjects perform poorly on learning and memory tasks, compared with well-rested individuals (Mograss, Guillem, Brazzini-Poisson, & Godbout, 2009; Smith, 1995).

Executive functions. The executive regulatory system comprises a set of interrelated cognitive processes that organize and regulate information processing and behavior in response to the demands of the environment (Drummond et al., 1999; Harrison & Horne, 2000; Harrison & Horne, 1998; J. A. Horne, 1988; Mesulam, 1990). These processes include working memory, inhibitory control, attention-shifting or flexibility, planning, problem-solving, and reasoning. The neural areas governing executive function include structures in the dorsolateral, prefrontal, anterior cingulate, and parietal cortices. The executive regulatory system plays a fundamental role in the ability of an individual to achieve success and is closely associated with academic achievement throughout a child's schooling, whereas poor executive functions are associated with academic failure and behavior problems. Numerous studies have demonstrated that sleep has important impact on these brain functions. Similarly, multiple studies have shown that sleep loss impairs performance on measures of executive function including tasks requiring abstract thinking, creativity, integration, planning (Dahl, 1996b), problem-solving, decision-making (Killgore, Balkin, & Wesensten, 2006), divergent thinking capacity (Horne, 1988; Linde & Bergstrom, 1992), working memory (Nilsson et al., 2005), flexibility (Alhola & Polo-Kantola, 2007), attention and vigilance (Durmer & Dinges, 2005), behavioral inhibition (Harrison & Horne, 1998), and cognitive set shifting (Wimmer, Hoffmann, Bonato, & Moffitt, 1992). These changes may act in combination to impair the regulation of behavior and academic performance.

Academic performance. Sleep disorders, sleep deprivation and daytime sleepiness have been linked to school tardiness and absenteeism (Drake et al., 2003; Gozal, 1998; Wahlstrom, 2002). Short sleep duration and low sleep quality are related to poorer academic performance, as measured by Stanford Achievement Test (SAT) scores and academic grades (Buckhalt, El-Sheikh, Keller, & Kelly, 2009; El-Sheikh, Buckhalt, Mark Cummings, & Keller, 2007; Meijer, 2008; Meijer, Habekothe, & Van Den Wittenboer, 2000; Wolfson, Spaulding, Dandrow, & Baroni, 2007). IQ is strongly related to academic achievement (Glutting, McDermott, Prifitera, & McGrath, 1994; Keith, 1993; Neisser et al., 1996; Thorndike, 1994). Among young children, short sleep duration approximately triples the risk of low performance on neurodevelopmental tests at school-entry (Touchette et al., 2007), whereas longer habitual sleep duration in healthy school-age participants is associated with better performance on measures of perceptual reasoning and overall IQ (Gruber et al., 2010).

1.2.2 Emotional health and psychopathology

Evidence in humans has shown an intimate and causal relationship between sleep and affective brain regulation, with maladaptive consequences following the absence of sleep and beneficial effects following restorative sleep. Studies assessing physiological and neural measures have objectively verified the relationship between emotional dysregulation and sleep deprivation. These findings collectively support a framework in which sleep deprivation exaggerates subcortical limbic and striatal reactivity to both positive and negative affective stimuli, both of which are associated with impoverished prefrontal cortex connectivity (Gujar, Yoo, Hu, & Walker, 2011; Yoo, Gujar, Hu, Jolesz, & Walker, 2007).

In contrast to affective dysregulation caused by the absence of sleep (El-Sheikh, Buckhalt, Keller, Cummings, & Acebo, 2007), beneficial influences on emotional perception and regulation have been described with adequate sleep. A daytime nap has been shown to dissipate the intensity ratings of negative, threat-relevant facial expressions (fear or anger) and increase responsivity towards positive (happy) facial images (Gujar, McDonald, Nishida, & Walker, 2011). These studies demonstrate that sleep facilitates the neural dissipation of limbic reactivity to prior emotional memories, while sleep loss promotes reactivity even after several nights of recovery sleep. Sufficient sleep is therefore crucial for the maintenance of emotional health among typically developing children and adolescents.

Sleep and psychopathology. Sleep disorders often accompany psychiatric disorders (Benca, Obermeyer, Thisted, & Gillin, 1992; Legenbauer, Heiler, Holtmann, Fricke-Oerkermann, & Lehmkuhl, 2012; Wulff, Gatti, Wettstein, & Foster, 2010), and have been found to predict the onset of both depressive episodes (Chang, Ford, Mead, Cooper-Patrick, & Klag, 1997) and mania (Plante & Winkelman, 2008). The available data suggest a close reciprocal link between sleep disorders and psychiatric symptoms. Parallel findings of anatomical dysfunction in the brain (e.g., altered activity in limbic areas and limbic-prefrontal cortex connectivity) have been reported in a number of psychiatric mood and anxiety disorders that express co-occurring sleep abnormalities, including major depression, bipolar disorder and posttraumatic stress disorder (Davidson, Pizzagalli, Nitschke, & Putnam, 2002; Drevets, Price, & Furey, 2008; Etkin, 2010; Pezawas et al., 2005; Rauch et al., 2000; Rich et al., 2006; Shin, Rauch, & Pitman, 2006). Also, the beneficial effects of psychoactive drugs on the neurotransmission systems of those experiencing psychiatric illnesses are reflected in polysomnographic recordings demonstrating altered sleep continuity and architecture (Staner, 2005). The sleep of individuals with mental illness may therefore offer important insights into the neurobiology of these disorders. Considering the known disruption of sleep in a number of addiction disorders, sleep loss has been suggested as a predisposing risk factor and therapeutic target in the vulnerability of addiction to reward-stimulating drugs. Sleep disorders are particularly prevalent in children and adolescents with anxiety disorders, depression, ADHD, bipolar disorder, Post Traumatic Stress Disorder (e.g., child abuse and neglect), and autism. In fact, sleep disruptions are one on the primary symptoms noted by parents of children with autistic spectrum disorders and other neurodevelopmental disorders (Williams, Sears, & Allard, 2004). Similarly, adolescents who report sleep difficulties are significantly more likely to also report symptoms of depression, anxiety, tension, lethargy, irritability, poor self-esteem, daytime stress, worry, negative thoughts,

and emotional lability. Finally, sleep problems in early childhood predict earlier onset in the use of alcohol, cigarettes, marijuana, and illicit drugs (Teotia & Gupta, 2002).

Parenting and family stress. Pediatric sleep disorders are a "family affair." When a child cannot sleep well, the parents' sleep is often disturbed (Meltzer & Mindell, 2007). Parents may allow children with sleep difficulties, and/or psychiatric difficulties such as anxiety, to sleep with them if they cannot fall asleep on their own. This is highly problematic as bed-sharing has been associated with increased risk for Sudden Infant Death Syndrome (SIDS; Leduc, Cote, Woods, & 2004). It may also lead to further difficulties because the child will develop habitual dependence on this habit and not learn how to fall asleep independently. This can subsequently affect the a parent's own abilities to deal with stress, low mood, and sense of fatigue (Meltzer & Mindell, 2007; Mindell & Owens, 2010), leading to increases in family conflict and negative interactions.

1.3 SOCIETAL AND ECONOMIC COSTS

Sleep loss and sleep disorders have significant public health and economic impacts (AlGhanim, Comondore, Fleetham, Marra, & Ayas, 2008; Colten & Altevogt, 2006; Hillman, Murphy, & Pezzullo, 2006; Reuveni, Simon, Tal, Elhayany, & Tarasiuk, 2002). Sleep-deprived individuals are likely to be less productive and have an increased need for health care services (Colten & Altevogt, 2006). In the United States, the medical costs associated with sleep-related doctor's consultations, hospital services, and prescriptions are estimated to amount to hundreds of billions of dollars a year (Colten & Altevogt, 2006). To date, there are no comparable data in Canada. In addition, parents of children who do not sleep tend to be sleep deprived and, therefore, less productive.

Given the pervasive impact of sleep on mental and physical health, medical costs are probably only a small percentage of the real cost of sleep disorders. Additional costs are likely to be incurred due to individuals' loss of productivity, and the increased negative contributions to society that a chronically sleep deprived individual imposes.

2. NORMAL SLEEP DEVELOPMENT AND THE CURRENT STATE OF SLEEP DEPRIVATION IN CHILDREN AND ADOLESCENTS

Two important aspects of sleep are sleep duration (how much sleep), and the timing of sleep (when sleep occurs). These aspects of sleep are regulated by two distinct physiological processes (Borbely, 1982; Czeisler et al., 1986; Moore, 1999). A *homeostatic* process regulates sleep onset by creating "sleep pressure" as the wake time lengthens and by dissipating this pressure as sleep is initiated and sustained; a *circadian* process regulates the sleep onset and awakening by realigning the "internal" clock (circadian pacemaker) each day with the light-dark cycle using input from the environment (Allada, White, So, Hall, & Rosbash, 1998; Blau & Young, 1999; Ebadi & Govitrapong, 1986; Moore, 1999). The major environmental "time giver," therefore, is light (Borbely, 1982; Czeisler et al., 1986). These processes interact to determine sleep quality, quantity, and timing. Developmental changes in physiological, chronobiological, neurologic, and social/environmental inputs influence sleep patterns in important ways.

2.1 SLEEP CHANGES FROM CHILDHOOD TO ADOLESCENCE

Infants (0-1 years). During the first month of life, an infant's sleep is distributed almost equally across the night and day (Kahn, Dan, Groswasser, Franco, & Sottiaux, 1996). Within the first 6 months, most infants develop the ability to sustain longer episodes of sleep and begin to consolidate sleep at night, gradually assuming a sleep pattern similar to that of adults (Peirano, Algarin, & Uauy, 2003). By around 10-12 weeks of age, a circadian rhythm begins to emerge and the infant's sleep becomes increasingly nocturnal, with longer bouts of night-time sleep complemented by three or four naps during the day. A major developmental milestone achieved by most infants by age 6-9 months is the ability to "sleep through the night" (i.e., to sleep for at least 8 hours per night). This sleep period is typically accompanied by one morning nap and one afternoon nap.

Toddlers (2-4 years). After the first year, the sleep-wake system continues to develop at a slow and steady rate. A gradual decline in daytime napping occurs, with children typically dropping their morning nap by 18 months, leaving one afternoon nap and one long nocturnal period of sleep (Iglowstein, Jenni, Molinari, & Largo, 2003). The average length of a toddler's afternoon nap ranges from 1.5 to 2 hours (Iglowstein et al., 2003; Kahn et al., 1996). By 3 years of age, 50% of children are still napping in the afternoon; 35% continue to nap until ~ 4 to 5 years of age (Iglowstein et al., 2003). By age 5, most but not all children stop napping and sleep becomes consolidated into a single nighttime period (Weissbluth, 1995). Night-time awakening is common, with 20% of children waking at least once per night and about 50% waking at least one night per week (Acebo et al., 2005). Thus, night wakening is common, and the ability of the child to fall back to sleep without parental intervention is an indication of healthy sleep development. If the child does not acquire this skill, a form of insomnia (sleep disorder) develops.

School-age children (5-12 years). A gradual shift to a later bedtime and sleep onset time begins in middle childhood and accelerates in early-to-mid adolescence. Children's total sleep time and sleep efficiency (i.e., the ratio of total sleep time to time spent in bed) decrease with increasing age, such that the evening bedtime is delayed and total sleep time is reduced (Challamel, 2001).

Adolescents (13-19 years). The characteristic maturational change of puberty is a delayed sleep phase, with adolescents showing an endogenous shift to a much later bedtime compared with children and adults (Carskadon, 2002). Given socio-environmental pressures in modern society, this phase delay frequently results in insufficient sleep during the school week and the need for "catch-up" sleep on weekends (Andrade, Benedito-Silva, Domenice, Arnhold, & Menna-Barreto, 1993; Carskadon, 2005; Carskadon & Acebo, 2002; Dahl & Lewin, 2002). Increasingly irregular sleep-wake patterns, larger discrepancies between school night and non-school night bedtimes and wake times, and increased weekend oversleep are seen from middle childhood through adolescence and beyond.

2.2 RECOMMENDED AMOUNT OF SLEEP AT DIFFERENT AGES

The reported sleep duration varies widely in infancy and shows a strong inverse relationship with age, with the fastest rate of decline occurring over the first 6 months of life. Sleep patterns show consistent developmental trends in sleep duration (decreases from 0 to 12 years), number of night wakings (decreases from 0 to 2 years), longest sleep period (increases from 0 to 2 years), and number of daytime naps (decreases up to age 5). Because significant individual differences

in the “right amount” of sleep are apparent, there is no “correct” amount of sleep that each child of a particular age should get each night. However, the average sleep duration and number of naps taken by specific age groups have been calculated and are shown below (Iglowstein, Jenni, Molinari, & Largo, 2003). A generally valid assumption is that a child obtains the “right” amount of sleep if he or she wakes feeling well-rested.

In summary, sleep duration varies widely in childhood, with the greatest rate of change occurring within the first 6 months of life.

Table # 1: Developmental changes in total sleep duration (hours), number of night wakings, daytime nap frequency, and daytime sleep duration (hours) during childhood and early adolescence

	Age	Mean (SD)	Min	Max	Minimal Acceptable Sleep Duration*
Total sleep duration: (Galland et al., 2012)					
	0-2 months	14.6	9.3	20.0	
	≈3 months	13.6	9.4	17.8	
	≈6 months	12.9	8.8	17.0	10.4
	≈9 months	12.6	9.4	15.8	10.5
	≈12 months	12.9	10.1	15.8	11.5
	1-2 years	12.6	10.0	15.2	11.2
	2-3 years	12.0	9.7	14.2	10.8
	4-5 years	11.5	9.1	13.9	10.3
	≈6 years	9.7	8.1	11.4	9.8
	≈7 years	9.4	7.9	10.9	9.6
	≈8 years	9.3	7.8	10.8	9.4
	≈9 years	9.3	7.8	10.8	9.2
	≈10 years	9.1	7.8	10.7	9
	≈11 years	9.0	7.3	10.6	8.9

	≈12 years	8.9	7.3	10.6	8.7
(Iglowstein et al., 2003)	13 years	9.0	(0.7)		7.6
	14 years	8.7	(0.7)		7.3
	15 years	8.4	(0.7)		7
	16 years	8.1	(0.7)		6.7
Number of nightwakings:					
(Galland et al., 2012)					
	0-2 months	1.7	0	3.4	
	3-6 months	0.8	0	3	
	7-11 months	1.1	0	3.1	
	1-2 years	0.7	0	2.5	
Daytime nap frequency:					
(Galland et al., 2012)					
	0-5 months	3.1	1.2	5.0	
	6-11 months	2.2	0.9	3.5	
	1-2 years	1.7	0.6	2.8	
Daytime sleep duration:					
(Iglowstein et al., 2003)					
	0.5 years	3.4	(1.5)		
	0.75 years	2.8	(1.2)		
	1 years	2.4	(1.1)		
	1.5 years	2.0	(0.7)		
	2 years	1.8	(0.5)		
	3 years	1.7	(0.4)		
	4 years	1.5	(0.4)		

*Minimal acceptable sleep durations represent two standard deviations below the mean, and were calculated using mean values obtained by Iglowstein et al. (2003).

3. SLEEP DEPRIVATION IN CHILDREN AND ADOLESCENTS

3.1 PREVALENCE AND DEFINITION

Despite very strong evidence indicating the great importance of sleep, the sleep of children and adolescents has declined over time (Matricciani, Olds, & Petkov, 2011). A large systematic review of data from 690,747 children from 20 countries found that the sleep duration of Canadian children and adolescents (n = 6670; aged 5 to 18 years) has consistently and rapidly decreased over the past century (Matricciani et al., 2011). This information is consistent with data obtained from the Sleep in America Polls conducted by the National Sleep Foundation (2004, 2006), which showed that 34% of toddlers, 32% of preschoolers and 27% of school-aged children sleep fewer hours than their parents think they need .

Although the optimal amount of sleep required in adolescence is reported to be at least 8.5 hours per night (Carskadon, Acebo, & Seifer, 2001), data obtained by Eaton et al. suggests that over 69% of teenagers sleep less than 7 hours a night (Eaton et al., 2010). Cross-sectional surveys of 3235 Canadian high school students revealed that as many as 70% of students get less than the recommended amount of sleep (Gibson et al., 2006). As adolescents age, they tend to show an increasing gap in sleep on school days versus non-school days (Olds, Blunden, Petkov, & Forchino, 2010), and more than half report feeling excessively tired or sleepy during the day (Spilsbury et al., 2004).

The manifestations of sleep deprivation may vary depending on age and developmental status. Many of the symptoms are related to the impact of sleep deprivation. Unlike sleepiness experienced by adults, sleep deprivation in children more often produces “paradoxical” manifestations, such as hyperactivity, aggression or risk-taking behavior.

Signs of fatigue or sleepiness in sleep-deprived children and adolescents may include some combination of the following: routinely falling asleep in class, in the car, or in front of the TV; increased instances of “sleepy behavior,” such as yawning or rubbing eyes; being “on the go” and running around; moodiness, irritability, crankiness and frustration; inattention, difficulty concentrating, and/or difficulty starting and completing tasks; and non-compliance (Dahl, 1996a).

It is therefore important to identify modifiable factors that influence the amount and quality of sleep in children and adolescents. Recognition of these factors allows caregivers to target these factors to optimize sleep and improve daytime function and health. A number of lifestyle habits have been shown to affect sleep quality. The sections to follow will review common lifestyle and environmental factors that negatively affect sleep in children and adolescents.

3.2 THE CAUSES OF SLEEP DEPRIVATION IN CHILDREN AND ADOLESCENTS

Sleep processes are affected by external cues and stimuli. Modern lifestyle factors that affect the mechanisms involved in sleep regulation or encourage later bedtimes and longer hours of nighttime arousal can disrupt and shorten sleep. These include modern technologies associated

with excessive light exposure and over-stimulation, consumption of wake-promoting substances such as caffeine, aspects of the physical environment (e.g. air quality, excessive noise, and temperature extremes) that hinder sleep, and the low priority given to sleep by families and society in general. .

Electronic media in the bedroom. The average screen time of young Canadians (Grades 6 to 12) today, is alarmingly high at 7.8 hours per day (Leatherdale & Ahmed, 2011). Moreover, a large percentage of children and adolescents have electronic media in their bedrooms and use these technologies late at night. A recent large study conducted in Alberta found that half of parents reported that their grade 5 child had a TV, DVD player and/or video game console in his or her bedroom, while 21% had computers and 17% had cellular phones (Chahal, Fung, Kuhle, & Veugelers, 2013). Observational studies have consistently shown associations between media use and child sleep difficulties (Garrison, Liekweg, & Christakis, 2011; Li et al., 2007; Nixon et al., 2008; Oka, Suzuki, & Inoue, 2008; Owens et al., 1999; Paavonen, Pennonen, Roine, Valkonen, & Lahikainen, 2006; Thompson & Christakis, 2005). These effects have been observed across cultures and in all media formats, including TV (Alexandru et al., 2006; J.A. Mindell, Meltzer, Carskadon, & Chervin, 2009; Mistry, Minkovitz, Strobino, & Borzekowski, 2007; Paavonen et al., 2006), video games (Alexandru et al., 2006; Dworak et al., 2007; Van den Bulck, 2004), and computers (Eggermont & Van den Bulck, 2006; Mesquita & Reimao, 2007; Van den Bulck, 2004). The effects are also evident across the age spectrum, including preschoolers (Garrison et al., 2011; Mindell et al., 2009; Mistry et al., 2007; Thompson & Christakis, 2005), school-aged children (Li et al., 2007; Nixon et al., 2008; Oka et al., 2008; Owens et al., 1999; Paavonen et al., 2006), adolescents and adults (Eggermont & Van den Bulck, 2006; Johnson, Cohen, Kasen, First, & Brook, 2004; Mesquita & Reimao, 2007).

Access to and night-time use of electronic media has been associated with shortened sleep duration and excess body weight (Chahal et al., 2013). Children with increasingly more electronic media devices in their bedrooms reported shorter sleep durations, and students who used electronic devices on most or all nights reported sleeping less and having more sleep difficulties. The effect of night-time media use on sleep duration and quality is the result of: 1) the use of devices after bedtime at the expense of sleep; 2) the strong effect of light exposure on the circadian timing system (Higuchi, Motohashi, Liu, Ahara, & Kaneko, 2003). Bright light emanating from electronic screens during the night suppresses melatonin, leading to circadian desynchrony, disrupted sleep and delayed sleep phase (Cajochen et al., 2011); 3) the media content (exposure to violent media and games can lead to over-stimulation and difficulty initiating and maintaining sleep); 4) sleep interruptions (cell phones and texting awakens children, and their content can increase arousal and make it difficult to disengage and return to sleep); and 5) poor parental control. In the latter context, Van den Bulck (2004, 2010) has referred to electronic media exposure as an unstructured and boundless leisure activity with no clear endpoint, unlike other hobbies or sports activities. It has been suggested that the presence of a media device in the bedroom may indicate low parental control, contributing to increased exposure.

Caffeinated beverages. Caffeine is a widely consumed psychoactive substance that activates dopaminergic reward circuits, and produces behavioral effects less potent than but similar to other dopaminerically mediated substances such as cocaine and amphetamine (Cauli & Morelli,

2005) . Adenosine is a sleep-inducing neurochemical that decreases sensitivity to dopamine (D2) receptors, and helps promote sleep. Caffeine is an adenosine antagonist that blocks the adenosine receptor, thereby increasing the effect of dopamine on the D2 receptor and enhancing the availability of dopamine, thus creating a stimulating effect (Stahl, 2008). As an adenosine antagonist, caffeine has been shown to attenuate electroencephalographic (EEG) markers associated with increased homeostatic sleep pressure, and thus promote wakefulness in humans (Roehrs & Roth, 2008). Energy drinks are particularly problematic, as they are treated as "dietary supplements" and are not subject to the same rules as soft drinks. For example, the U.S. Food and Drug Administration (FDA) allow a 12-ounce can of soda to contain up to 65 mg of caffeine. In contrast, the energy drink Red Bull contains 80 mg of caffeine in an 8.4-ounce can, while Full Throttle® (original) has 144 mg of caffeine in a 16-ounce can (Babu, Church, & Lewander, 2008). In a recent study, 75% of children surveyed reported that they consumed caffeine on a daily basis, with an inverse relationship between increased caffeine use and decreased sleeping times. Children aged 5 to 7 years old consumed approximately 52 mg of caffeine per day, and children aged 8 to 12 years old consumed approximately 109 mg (Warzak, Evans, Floress, Gross, & Stoolman, 2011). As stipulated by Health Canada (2012), the maximum recommended caffeine intake level for children aged 10-12 years is 85 mg/day, and even less is recommended for younger children (62.5 mg/day for ages 7-9 years, and 45 mg/day for ages 4-6 years). Habitual daily caffeine consumption has been related to sleep disruption, sleepiness (Orbeta, Overpeck, Ramcharran, Kogan, & Ledsky, 2006; Pollak & Bright, 2003; Roehrs & Roth, 2008), and impaired daytime functioning (Calamaro, Mason, & Ratcliffe, 2009). This is likely related to the long half-life of caffeine. The half-life of a single dose of caffeine ranges from 3 to 7 hours (Roehrs & Roth, 2008); Thus, caffeine consumption during the afternoon or evening, such as at dinnertime, is likely to last well into the night and have an effect on the arousal system even at bedtime, hindering children's ability to fall asleep.

Low priority given to sleep. A healthy diet, physical activity, and the proper amount of sleep are all interrelated and important for a child's health. Unfortunately, sleep is often neglected in this regard. Many perceive time spent asleep has lost time that could be spent more productively. In some ways, society's current view of sleep deprivation is similar to our past attitude toward smoking, which was characterized by ignorance, lack of concern regarding the serious consequences, and even humor. Our socio-cultural environment (long store hours, late-night sports events, energy drinks, screen time and artificial light exposure at night) does not promote healthy sleep habits, and people living in a "24/7 society" place sleep low on their priority list. Parents' busy schedules and late work hours may push dinner and family activities to a later time. Children have busy schedules too, as they are often enrolled in multiple extracurricular activities and attend late-ending social, sporting and school events that contribute to delayed bedtimes and short sleep duration. Sleep health is often neglected as a crucial component of a healthy lifestyle, and sleep deprivation is not currently considered a public health concern by most education or public policy makers.

Sleep is rarely integrated into programs and interventions designed to target and improve health issues, such as weight regulation and obesity. The majority of these programs are focused on healthy eating and active living. Healthy sleep is typically not a focus for government policy or in pediatric practice, and the importance of sleep and its relevance to academic success is rarely addressed in educational programs aimed at optimizing academic performance.

3.3 GUIDELINES AND STRATEGIES FOR THE PREVENTION OF SLEEP DEPRIVATION

Although health care providers are in a position to offer advice regarding sleep and ask about the sleep health of children, this is often not part of the routine due to time constraints, insufficient knowledge, and inadequate reimbursement for care requiring conversation, questioning and counseling[1]. To prevent sleep deprivation caused by lifestyle factors, routine health assessments must include:

1) Parental education on normative sleep expectations using a developmental perspective and highlighting the importance of age-appropriate sleep duration, habits, timing, and day/night distribution;

2) Identification of unhealthy sleep patterns;

3) Counseling on the benefits of healthy sleep; and

4) Identification of factors contributing to unhealthy sleep, barriers to lifestyle changes, and the patient's self-efficacy in making the needed change. For example, the importance of a sleeping environment (including floor coverings and bedding) that is clean, well-ventilated, quiet, dark, and pet-free can be emphasized. Motivational interviewing, which is a person-centered, goal-oriented method of communication, may help elicit and improve the probability for positive change.

Families or family members who are not prepared to change should be asked about possible barriers and offered potential solutions. Once a family is ready to begin implementing strategies toward healthy sleeping, a personal sleep "prescription" should be written and posted in the home (See Appendix 3 for a sample sleep prescription, and Appendix 4 for sleep recommendations that can be provided to expecting parents and/or parents of newborns). This strategy takes advantage of a familiar medical model, and thus reinforces the importance of sleep for optimal health. The targeted sleep duration, bedtime, and desired behavior (e.g., the removal of electronics from the bedroom) should be included in this family plan. These choices should be integrated into the family's routine in a consistent way, making them a natural and predictable part of family life. If we are to help young children, the parents must be included as agents of change.

[1]. Public Health Nurses do not face the same problems as busy general practitioners and could thus potentially contribute to promoting healthy sleep.

Physicians and healthcare professionals should promote healthy sleep by:

1) Providing information to parents on:

- Basic information on sleep processes and age-appropriate information on normative sleep needs and patterns
- Environmental factors that might affect their child's sleep

- The importance of sleep for healthy and successful development
- The importance of their role in optimizing their child's sleep and making a significant positive impact on their child's health and success

2) Counseling parents, and helping them to:

- Identify necessary changes in family/child routines that will maximize healthy sleep behavior
- Make necessary changes by providing concrete strategies
- Promote regular nocturnal sleep patterns by facilitating social cues to sleep (e.g., by implementing consistent meal times and bed-time routines)
- Minimize the use of screen-based activities for children under 2 years of age and limit recreational screen time to < 1 hour/day for children 2-4 years of age and to < 2 hours/day for older children
- Encourage the watching of pro-social content rather than aggressive or scary content in general and particularly before bedtime
- Discourage the watching of movies or TV at bedtime
- Prioritize sleep when scheduling extracurricular activities for their child and making family plans
- Set clear and consistent bedtimes for children at all ages
- Keep TV sets, video games, cell phones and computers out of their child's bedroom
- Identify barriers to the adoption of healthy sleeping as part of the family routine
- Support their child's preferences in sport and recreational activities, provided that they are safe, age-appropriate, and not scheduled too close to bedtime
- Support healthy eating, avoid heavy meals before bedtime, and remove energy drinks and caffeine-containing beverages and foods from the child's diet

3) And by:

- Conducting or providing for follow-up after a visit by the parents and child
- Asking about the sleep of family members at regular healthcare visits, and promoting healthy sleep at every well-baby, well-child or adolescent visit
- Providing age-appropriate napping schedules and durations for children ages 0-5, advising parents and caregivers that toddlers and preschoolers should nap, and recommending that caregivers should check the daycare nap policy if the child attends daycare
- Encouraging older school-aged children to become healthy sleep role models for younger schoolmates
- Being active role models themselves
- Working with community-based organizations and/or centres (e.g., CLSCs, YMCAs) to educate different community members about sleep (e.g., being available for community-based workshops and parent education sessions on sleep as well as the prevention of sleep disorders)
- Working with schools and school boards to increase personnel's understanding of the interconnections between sleep, physical activity and a healthy diet (i.e., advocating for

increased physical activity, healthy foods, and the banning of caffeinated beverages to promote healthier sleep).
- Exercising caution before prescribing pharmacological agents to treat sleep disorders in young children as many medications have not been approved by the (US) Food and Drug Administration and/or Health Canada for use in the pediatric age range.
- Employing approved pharmacological interventions as only a short-term solution for the treatment of sleep disorders.

For additional information:

Gruber, R., Cassoff, J., & Knauper, B. (2011). Sleep health education in pediatric community settings: rationale and practical suggestions for incorporating healthy sleep education in pediatric practice. *Pediatric Clinics of North America, 58,* 735-754.

4. PEDIATRIC SLEEP DISORDERS

4.1 PREVALENCE RATES AND DEFINITIONS

Parent reported sleep disorders occur in 25% to 50% of pre-school-aged children (i.e., 3 to 5 years) (Mindell et al., 1997; Scala-Foley & Bryant, 2004). Such problems can affect not only children's behavior, cognition, emotions and academic functioning, but also their parents' functioning and family life. It is therefore crucial to effectively identify and treat these problems. Pediatric sleep disorders are classified by the American Academy of Sleep Medicine into eight major categories:

- Behavioural Insomnias,
- Sleep-related breathing disorders,
- Hypersomnias,
- Circadian rhythm sleep disorders,
- Parasomnias,
- Sleep-related movement disorders,
- Isolated symptoms and normal variants,
- Other sleep disorders.

(American Academy of Sleep Medicine, 2005)

Table 2 gives brief definitions and prevalence rates for common pediatric sleep disorders. The clinical manifestations of sleep disorders may vary by age and developmental level, and both sleep and sleep disorders in children are strongly influenced by cultural factors, parental expectations, and parental responses.

Because of the high prevalence of sleep disorders in pediatric populations, their scope, and their negative impact on children and families, effective screening interventions are needed and should be implemented early. Unfortunately, pediatric sleep disorders are under-diagnosed and under-treated, and many families have trouble accessing sleep services. Thus, health care professionals should: 1) screen for the presence of pediatric sleep disorders; 2) determine when a

sleep study is indicated; 3) offer intervention; and 4) make appropriate referrals for sleep disorders s that require more specialized care. To facilitate this, the following sections offer practical guidelines for: 1) taking a sleep history; 2) assessing sleep disorders; 3) making a diagnosis; and 4) using evidence-based information to treat pediatric sleep disorders.

4.2 GUIDELINES FOR DIAGNOSING PEDIATRIC SLEEP DISORDERS

4.2.1 Take the history with a differential diagnosis in mind

A thorough sleep and medical history, taken with an understanding of normal sleep physiology, provides the foundation for the diagnosis and management of sleep disorders. The clinician should evaluate the patient's sleep/wake schedule, difficulties initiating or maintaining sleep, abnormal movements or behavior during sleep, and daytime associations (e.g., sleepiness, inattentiveness, or irritability). The history should include details about the duration and frequency of the problem, the temporal profile of onset (abrupt, gradual, or intermittent), and the degree of variability from night to night. Most sleep complaints can be distilled into one (or more) of four categories: 1) difficulty initiating or maintaining sleep, 2) excessive daytime sleepiness, 3) snoring, or other breathing problems during sleep, and 4) abnormal movements or behaviors during sleep (Wise & Glaze, 2013).

Difficulty initiating or maintaining sleep. A useful way to gather history regarding a child with difficulty initiating sleep is to review the child's hour-by-hour activity pattern and sleep schedule from their arrival home after school or daycare until sleep onset. It is important to identify behavioral and physiological factors that contribute to the child's difficulty in initiating or maintaining sleep. These could be related to the sleeping environment, the consumption of caffeine or exposure to bright light at bedtime, an inconsistent bedtime routine, and/or the parents' response to nighttime awakenings. In addition, the clinician should probe: 1) the psychosocial history of the family, including the presence of marital discord, drug or alcohol use, and the possibility of child abuse; and 2) psychiatric or emotional problems, including anxiety, depression, ADHD and post-traumatic stress disorder (all are relatively common causes of insomnia in children). Difficulty initiating sleep can be an important sign of anxiety disorders, even before it is recognized as such. It is also important to determine whether there is a physiological factor contributing to difficulty initiating sleep, such as a circadian sleep disorder, or stimulant effects from caffeine or medications.

Excessive daytime sleepiness. When a clinician evaluates a child with excessive daytime sleepiness, the goal is to identify potential causes. If parents are not aware of age-appropriate norms for nighttime sleep and daytime napping, they may fail to recognize poor sleep hygiene or chronic sleepiness in their child. Furthermore, a sleepy child may not appear sleepy to parents or clinicians. Instead, these observers may notice attentional difficulties, hyperactivity secondary to the child's efforts to stay awake, and/or aggressive and disruptive behaviors that reflect the inability of a sleep-deprived frontal cortex to regulate emotion.

Common causes of daytime sleepiness include insufficient nocturnal sleep, inadequate sleep hygiene, and the side effects of medication (e.g., antidepressants, atypical anti-psychotics, or anti-seizure medications). Less common but important causes include obstructive sleep apnea (OSA), narcolepsy, idiopathic hypersomnia, periodic limb movement disorder, and a variety of

toxic, endocrine, and metabolic problems (Wise & Glaze, 2013). OSA can present with daytime sleepiness or associated behavioral problems; complaints of excessive snoring or abnormal breathing during sleep are usually, but not always, present. Sleepiness should be differentiated from chronic fatigue, which often involves somatic complaints, such as weakness, listlessness, malaise, non-restorative sleep patterns and emotional disturbances (Wise & Glaze, 2013). These latter problems often suggest a medical problem such as anemia, thyroid disease or other metabolic problems, rheumatological processes, or malignancy, or psychiatric problems such as depression or anxiety.

Other sources of hypersomnia include post-traumatic hypersomnia, recurrent hypersomnia (Kleine-Levin syndrome), menstruation-associated hypersomnia, pregnancy-associated hypersomnia, and circadian rhythm disorders such as Delayed Sleep Phase Disorder (DSPD) and Advanced Sleep Phase Disorder (ASPD) (American Academy of Sleep Medicine, 2005; Huang, Lakkis, & Guilleminault, 2010; Lisk, 2009).

Snoring or breathing problems. Sleep-related breathing disorders may be associated with snoring and other sounds while sleeping. Night-time signs of OSA may also include difficulty breathing, paradoxical chest-abdominal movements, retractions, observed obstructive apneas, restless sleep, excessive sweating and cyanosis. Daytime symptoms may include nasal obstruction, mouth breathing, poor attentiveness, irritability, behavior problems and sleepiness (Wise & Glaze, 2013). The most common cause of pediatric OSA is adenotonsillar hypertrophy but OSA may also be associated with craniofacial, genetic, neurologic abnormalities and with morbid obesity.

Abnormal movements or behaviors during sleep. Abnormal movements or behaviors (e.g., respiratory changes, parasomnias and nocturnal seizures) occur in a variety of sleep disorders. Nocturnal events associated with high-amplitude (or vigorous) movements may present a significant risk of injury to the child, and protective measures may be required. A thorough history is adequate to characterize most nocturnal events and establish a diagnosis. Recordings of representative clinical events with a home video camera may also provide useful information. In some cases, additional diagnostic evaluation, such as electroencephalography or prolonged EEG/video/polygraphic monitoring, is necessary (Wise & Glaze, 2013).

There is significant overlap between periodic limb movement disorder (PLMD) and restless leg syndrome (RLS) in children and adults. PLMD is a disorder characterized by repetitive, highly stereotyped limb movements occurring during sleep (defined by a rate of period limb movements of sleep greater than 5 per hour; PLMI $\geq$ 5). Periodic leg movements are also often associated with Restless Leg Syndrome (Aurora et al., 2012). RLS is an independent sensorimotor disorder characterized by the complaint of a strong, almost irresistible urge to move the legs, which is often accompanied by symptoms of discomfort or pain. These sensations are worse at rest and tend to occur more frequently in the evening or throughout the night-time. Full or partial relief of the unpleasant sensations is attained through movement (Thorpy, 2012). Children with RLS often have depressed serum ferritin levels, indicating reduced iron stores (Allen & Earley, 2007; Durmer & Quraishi, 2011; Kotagal & Silber, 2004; Picchietti, 2007; Picchietti & Stevens, 2008). In this case, the disorder may improve with iron supplementation.

Additional relevant information. When evaluating a child with sleep difficulties, the clinician should also perform a thorough medical review, paying attention to possible neurodevelopmental or medical problems. Chronic conditions, such as reactive airways disease, gastroesophageal reflux, congenital heart disease, arthritis, and other causes of chronic pain, may predispose a child to sleep problems. Neurological disorders, such as cerebral palsy, nocturnal seizures, developmental delay, blindness, conditions with poor oropharyngeal function, as well as autism and related disorders, are also associated with elevated risks for sleep disorders (Brown, Maistros, & Guilleminault, 1995). The clinician should identify chronic medical problems that may influence sleep latency and continuity, including chronic or recurrent pain, symptoms suggestive of gastroesophageal reflux, breathing problems during wakefulness or sleep, and the medication history. The latter is important because many medications used in pediatrics can affect sleep physiology, and those with sedating effects can cause sleep-related airway obstruction and daytime sleepiness (Nicholson, Bradley, & Pascoe, 1994; Paykel, Fleminger, & Watson, 1982).

Pediatric clinicians, especially those addressing mental health issues in children and adolescents, should be aware of drugs causing *insomnia*, on the one hand, and those agents inducing excessive sleepiness, on the other. If given too close to bedtime, psychostimulants may cause some degree of insomnia, delaying sleep onset up to an hour with immediate release methylphenidate (Corkum et al., 2008) and even longer with slow release preparations (Weiss, Childress, Pucci, & Hectman, 2011). In contrast, many sedating antidepressants and anti-psychotics, by producing daytime somnolence and excessive night-time sleep, may interfere with academic performance and quality of life in pediatric patients.

For adolescents, clinicians should inquire about any type of abused drug (either diverted or illegal), which may affect sleep patterns and interact with prescribed psychotropics to exacerbate insomnia or hypnotic effects. In patients with medical conditions, the clinician needs to inquire about pharmacological treatments that can affect sleep (e.g., systemic corticosteroids or albuterol, decongestants, diet pills). At present, little is known about the long term impact of these medications on child and adolescent development through their effect on altered sleep stage physiology.

In any evaluation to determine the presence of a sleep disorder, the family and the child should be asked to complete a sleep log during the two weeks prior to evaluation (See Appendix 1 for samples of a parent-report and child self-report sleep log). This may provide important information regarding their sleep/wake pattern and nocturnal events. The log should include bed time, time of sleep onset, awakenings, rising time, nocturnal events, feeding pattern, naps, perceived quality of sleep, degree of alertness or sleepiness during the day, and observations regarding medical or psychological stressors. The child's sleep patterns can then be compared to typical sleep patterns for his or her age group (Table 1), although it should be recognized that the average sleep time of children in a given age group can vary by as much as 2 hours.

4.2.2 Physical examination

The physical examination is directed towards identifying the causes of sleep disorders or the sequelae associated with sleep pathology. The clinician's general observations should include the child's level of alertness (and any fluctuations) during the examination. Repetitive yawning,

droopy eyelids, a blank facial expression, frequent changes in position, over-activity and irritability may all indicate excessive sleepiness (Wise & Glaze, 2013). The general examination should include assessment for dysmorphic features that may occur in children with genetic disorders (e.g. Trisomy 21, Prader-Willi and others), craniofacial anomalies, or abnormalities of head size (i.e. macro or microcephaly). Inspection for signs of scoliosis and neuromuscular disease should be performed (Wise & Glaze, 2013). Developmental milestones should be evaluated. Evaluation of growth parameters may indicate failure to thrive, evolving obesity, or obesity.

Examination of the oropharynx may reveal evidence of tonsillar or adenoidal hypertrophy, an abnormally small upper airway, mandibular hypoplasia, retrognathia, or bulbar dysfunction. Bulbar dysfunction can manifest with decreased or absent gag reflex, poor movement of the soft palate, or swallowing problems. Persistent mouth breathing or noisy breathing may suggest nasal obstruction. Clubbing, cyanosis, or edema may suggest heart failure. Lung examination may suggest chronic lung disease or reactive airways disease (Wise & Glaze, 2013).

4.2.3 Ancillary testing of sleep or sleep-wake patterns

Ancillary sleep assessments can include subjective and objective measures. Subjective measures generally take the form of questionnaires filled out either by the patient or a proxy (i.e., a parent, caregiver, bed-mate, etc.), and may require some degree of judgment or interpretation of the patient's behavior, symptoms, and condition (See Appendix 2 for sample sleep questionnaires). A two-week sleep log can be very useful in understanding sleep hygiene, sleep duration and circadian patterns. In addition, a number of validated and normed questionnaires are available at no cost, and these can be used to screen for the common pediatric sleep disorders.

Objective measures directly record physiological events (nocturnal polysomnography, or PSG), activity patterns (actigraphy), or performance of certain cognitive or behavioral tasks. Objective characterization of daytime sleepiness can be performed using the Multiple Sleep Latency Test (MSLT). These procedures are described below.

Polysomnography (PSG). PSG consists of an all-night recording performed in a sleep laboratory with the attendance of a sleep technologist. PSG is performed to characterize sleep architecture and sleep pathology such as sleep-related respiratory disturbance or periodic limb movements during sleep. Recordings include a combination of the electroencephalogram (EEG or brain wave recordings), electro-oculogram (EOG or eye movements), and sub-mental muscle tone (EMG) in order to characterize sleep/wake states, arousals and awakenings, and sleep architecture. Respiratory function is assessed using registration of air flow at the nose and mouth, respiratory movements of the chest and abdomen, and oximetry. The electrocardiogram (EKG) measures heart rate and rhythm, and limb movements are monitored using limb EMG sensors. Audio recordings detect sounds such as snoring or vocalizationsm and video recordings help characterize movements or behaviors during sleep (Iber, Ancoli-Israel, Chesson, & al., 2007; Mindell & Owens, 2010). A standardized scoring manual provides guidelines and criteria for analysis of PSG results in adults and children (American Academy of Sleep Medicine, 2005). Specific guidelines are also available for infants (*A manual of standardized terminology, techniques and criteria for scoring of states of sleep and wakefulness in newborn infants*, 1971). At present, there are 132 sleep centers in Canada which offer in-laboratory, technologist attended

polysomnography, according to the website for the Canadian Sleep Society. However, pediatric PSG requires special skill and experience in children and not all sleep centers have personnel or equipment suitable for performing PSG in infants or children.

For more information, please see:

http://www.canadiansleepsociety.ca/publisher/articleview/?PHXSESSID=1e2abcd3ea241f7e4c1b285983ae1db1&/1/frmArticleID/370/

When is PSG indicated? Laboratory-based PSG performed in the presence of a sleep technologist is indicated for : 1) establishing the diagnosis of a sleep-related breathing disorder (e.g., OSA), 2) evaluating for narcolepsy (in conjunction with the MSLT); 3) verifying a strong clinical suspicion of periodic limb movement disorder; 4) initiating and titrating continuous positive airway pressure (CPAP) therapy, or for titrating non-invasive positive pressure ventilation (NIPPV); 5) evaluating patients with neuromuscular disorders and sleep-related symptoms; 6) testing for parasomnias associated with clinical suspicion of a sleep-related breathing disorder or periodic limb movement disorder; 7) confirming an atypical or potentially injurious parasomnia; and 8) establishing sleep-related epilepsy. PSG with an expanded EEG montage may be used to evaluate patients with suspected sleep-related epilepsy when the initial clinical evaluation and standard EEG are inconclusive; this may help distinguish the disorder from a parasomnia.

When is PSG not indicated? Polysomnography is NOT routinely indicated for the evaluation of difficulty initiating or maintaining sleep (insomnia), circadian rhythm disorders, non-epileptic parasomnias, chronic lung disease, depression, bruxism, or behavior-based sleep disorder.

Ambulatory (out of center, unattended) PSG monitoring has been used in children but it remains primarily a research tool at this time.

For additional information:

Aurora, R. N., Zak, R. S., Karippot, A., Lamm, C. I., Morgenthaler, T. I., Auerbach, S. H., Bista, S. R., Casey, K. R., Chowdhuri, S., Kristo, D. A., Ramar, K. (2011). Practice parameters for the respiratory indications for polysomnography in children. *Sleep, 34*(3), 379-388.

Wise, M. S., Nichols, C. D., Grigg-Damberger, M. M., Marcus, C. L., Witmans, M. B., Kirk, V. G. D'Andrea, L. A., Hoban, T. F. (2011). Respiratory indications for polysomnography in children: An evidence-based review. *Sleep, 34*(3), 389-398.

Aurora, R. N., Lamm, C. I., Zak, R. S. Kristo, D. A., Bista, S. R., Rowley, J. A., & Casey, K. R. (2012). Practice Parameters for the Non-Respiratory Indications for Polysomnography and Multiple Sleep Latency Testing for Children. *Sleep, 35*(11), *1467-1473.*

Kotagal, S., Nichols, C. D., Grigg-Damberger, M. M., Marcus, C. L., Witmans, M. B., Kirk, V. G., D'Andrea, L. A., Hoban, T. F. (2012). Non-respiratory indications for polysomnography and related procedures in children: an evidence-based review. *Sleep, 35*(11), 1451-1466.

Actigraphy. Sleep-wake patterns and circadian rhythms can be investigated by assessing movement (Littner et al., 2003). In actigraphy, a small device is attached to the wrist of the non-dominant hand, or to an ankle, where it records data from accelerometers several times per second. If actigraphy is performed alone, the individual under study is usually asked to wear the device continuously for about a week. During the observation period, a sleep diary is commonly kept to assist in determining times of lights on and off (Littner et al., 2003). The data are uploaded to a computer and processed to provide information such as average activity during different periods, estimated wake and sleep periods based on pre-defined cut-off values, circadian rhythm patterns, and average times of peak activity. Actigraphy is commonly used to empirically study sleep, and has also been increasingly integrated into clinical care. Its use in clinical settings largely depends upon health care professionals' expertise and the availability of the equipment.

When is actigraphy indicated? This method is used to evaluate insomnia, circadian rhythm sleep disorders and excessive sleepiness, and to assess the effectiveness of treatments.

When is actigraphy not indicated? Actigraphy cannot provide sleep-staging information and is not used alone for the diagnosis or management of sleep disorders.

4.3 GUIDELINES FOR EVIDENCE-BASED INTERVENTIONS FOR PEDIATRIC SLEEP DISORDERS

A description of the most common sleep disorders of childhood, as well as their estimated prevalence rates, are provided in Table 2. Evidence-based interventions are available for some pediatric sleep disorders. The available best practice guidelines and systematic reviews are also listed.

4.3.1 Behavioral Insomnias

Behavioral interventions for the treatment of behavioral insomnias of childhood have been empirically validated (Mindell, Kuhn, Lewin, Meltzer, & Sadeh, 2006). Behavioral interventions are those that clinically apply the fundamental principles of learning in order to affect change in the child's sleep habits and behaviours, with parents typically acting as the "agents of change" (Kuhn & Roane, 2012). They begin with a thorough sleep assessment and proceed through improvements in sleep hygiene, standard extinction and graduated extinction. "Extinction" in this context is best described as the removal of any inappropriate parental attention that may reinforce the problem sleep behavior (i.e. "ignoring" the behavior). Such interventions are not recommended for infants less than 6 months of age because they may interrupt feeding practices, and no sleep program should be given to children who are ill. Also parents need to become *fully* acquainted with the correct use of a program in order for it to be successful. As well, the *prevention* of sleep problems in young children through early intervention is an appealing alternative to the treatment of sleep disorders once they have already become well established (Mindell, et al., 2006). One of the most effective, and efficient approaches to the prevention of sleep problems involves educating soon-to-be, or new, parents how to encourage healthy sleep habits in their children (for example, through the development of healthy sleep routines, schedules, and appropriate sleep associations; (Kuhn & Roane, 2012; Mindell et al., 2006). Typically, parent education aims to teach children how to initiate sleep autonomously at bedtime.

For more information see:

Mindell, J., Kuhn, B., Lewin, D. S., Meltzer, L. J., Sadeh, A. (2006). Behavioral treatment of bedtime problems and night wakings in infants and young children. *Sleep*, *29*, 1263-1276.

Vriend, J. & Corkum, P. (2011). Clinical management of behavioral insomnia of childhood. Psychology Research and Behavior Management, 4, 69-79.

Kuhn, B. R., Roane, B. M. (2012). Pediatric insomnia and behavioral interventions. In T. J. Barkoukis, J. K. Matheson, R. Ferber, & K. Doghramji (Eds.), Therapy in Sleep Medicine (1st Ed., pp. 448-456). Philadelphia, PA: Elsevier Inc (Saunders).

Pharmacologic agents. In general, the use of medications should be relatively uncommon in children, and whenever possible restricted to a short term strategy to re-establish healthy sleep patterns. Medication use should almost always be accompanied by a rigorous sleep hygiene program. Weiss and colleagues (2006), for example, have demonstrated that while melatonin (5 mg) reduced insomnia by 16 minutes, insomnia was reduced by 60 minutes if combined with sleep hygiene intervention. While there are many agents available, only a few have been tested with a sound experimental methodology. Alpha agonists, such as clonidine and guanfacine, are widely used; however, clonidine has been associated with rebound hypertension and cardiotoxicity because of its short half-life. Sedating antidepressants (i.e., mirtazapine and trazadone) are used for their sedating properties at bedtime. They may also be administered to improve daytime mood, and lead to better sleep. Antihistamines are popular over-the-counter medications to treat insomnia, but they have been found to be potentially no better than placebo in controlled trials. Benzodiazepine and other GABA agonists can have many side effects (e.g., a hangover effect, potential anterograde amnesia, etc.). Non-benzodiazepine GABA agonists (i.e., zalaplon and zolpidem) are more frequently used now in child and adolescent inpatient units, but a single published trial in children failed to show effectiveness (for a review of pharmacologic treatments for pediatric insomnia, see Owens & Mindell, 2011). Little is currently known about the long-term effects of these drugs on the physiology of different sleep stages, although some antidepressants suppress REM and increase latency to REM sleep (Owens & Mindell, 2011).

4.3.2 Sleep-related Breathing Disorders

The most common sleep-related breathing disorder in children is obstructive sleep apnea (OSA) syndrome. The most common etiology is adenotonsillar hypertrophy, but increasingly, obese children have significant OSA not due to adenotonsillar hypertrophy. Adenotonsillectomy is the first-line treatment of choice for most children with significant OSA. Nasal inflammation is often present in children with OSA (Friedman & Goldman, 2011). Intranasal corticosteroids can therefore be an effective therapeutic option in mild or mild to moderate OSA if there is nasal inflammation and/or obstruction (Brouillette et al., 2001; Friedman & Goldman, 2011; Kheirandish-Gozal & Gozal, 2008). Simple snoring, a sign of increased upper airway resistance without proven hypopnea or obstructive apnea, usually requires no specific treatment, but children with simple snoring should be monitored for worsening in respiratory function or daytime symptoms over time.

Continuous positive airway pressure (CPAP). CPAP therapy is a well-recognized treatment for OSA in adults and it is an increasingly utilized option for treatment of pediatric OSA. Recent data indicate that even with suboptimal use, CPAP confers benefits in neurobehavioral outcomes in children with OSA when adenotonsillectomy is not indicated or has not resolved the problem (Marcus et al., 2012). However, while CPAP is effective in lowering the apnea/hypopnea index (AHI), and it is associated with improvements in several areas of function, its therapeutic use requires parents who are committed to supporting the child and close follow-up with the sleep specialist to address compliance issues.

Adenotonsillectomy and other surgical procedures. The surgical removal of tonsils and adenoids is frequently used to treat OSA in children. Most, but not all, patients are cured or significantly improved (e.g., Marcus et al., 2013). In patients with underlying medical conditions such as obesity, craniofacial anomalies, or neuromuscular disorders, surgery alone may not be curative. These patients may also require CPAP to control their sleep apnea. Several treatment options other than adenotonsillectomy are available but most are applicable only to special populations. Patients with certain craniofacial anomalies may benefit from specific craniofacial surgical procedures which are generally available only in tertiary medical centers. Treatment such as rapid maxillary expansion may be effective in some children (Pirelli, Saponara, & Guilleminault, 2004; Villa et al., 2007). In cases of severe upper airway obstructive during sleep, tracheostomy may be required to restore adequate ventilation.

Orthodontic approaches. Orthodontic approaches, particularly rapid maxillary expansion (RME), are increasingly reported as aiding in the treatment of OSA in preschool (Marino, Ranieri, Chiarotti, Villa, & Malagola, 2012) and school-aged children (Villa, Rizzoli, Miano, & Malagola, 2011) with narrow, arched palates, retrusive bites or crossbites. Improvements have been maintained 2 years after intervention, both alone and in combination with adenotonsillectomy. Despite the near normalization of sleep architecture and significantly improved AHI one year after intervention, however, some aspects of sleep microstructure remained disturbed, suggesting an incomplete response to therapy (Miano et al., 2009).

Other treatment options. Weight loss may be effective in obese patients with OSA, but is difficult to achieve and relapse is probably common (Kalra & Inge, 2006). Anti-inflammatory agents, such as nasal steroids or leukotriene antagonists, may be beneficial in the treatment of mild or borderline OSA (Kheirandish, Goldbart, & Gozal, 2006). Although not a definitive treatment, positional therapy such as having the patient sleep on his or her side, or with elevation of the head and trunk, may represent another option when necessary. Supplemental oxygen will likely blunt the degree of oxygen desaturation associated with moderate or severe OSA, but it does not address the upper airway obstruction with associated ventilatory abnormality.

For detailed reviews of the recommendations for diagnosis and treatment of sleep-disordered breathing and OSA:

Marcus, C. L., Brooks, L. J., Davidson-Ward, S. Draper, K. A., Gozal, D., Halbower, A. C., Jones, J., Lehmann, C., Schechter, M. S., Sheldon, S., Shiffman, R. N., & Spruyt, K. (2012). Diagnosis and management of childhood obstructive sleep apnea syndrome. *Pediatrics,130,* e714 – e755.

Friedman, N. R., Perkins, J. N., McNair, B., & Mitchell, R. B. (2013). Current practice patterns for sleep-disordered breathing in children. *Laryngoscope, 123*, 1055-1058.

4.3.3 Hypersomnias

Narcolepsy

Treatment goals for children with narcolepsy include symptom control and the optimization of lifestyle and psychosocial function. Successful treatments typically include both behavioral aspects (i.e., developing healthy sleep habits and avoiding sleep deprivation) and pharmacological intervention aimed at treating daytime sleepiness and cataplexy. Central nervous system stimulants are generally successful and widely used medications in the treatment of sleepiness due to narcolepsy. However, adverse side effects such as appetite suppression, tremor, and emotional lability may limit the use of stimulants in some children. More recently, modafinil, armodafinil, and sodium oxybate have become available, although published data involving pediatric usage are very limited for these agents. The management of narcolepsy in children requires a comprehensive approach which is typically available only in concert with a pediatric sleep medicine specialist.

For more information:

Wise, M. S., Arand, D. L., Auger, R. R., Brooks, S. N. & Watson, N. F. (2007). Treatment of narcolepsy and other hypersomnias of central origin: An American Academy of Sleep Medicine review. *Sleep, 30*(12), 1712–1727.

4.3.4 Circadian Rhythm Sleep Disorders (CRSDs)

CRSD therapies seek to synchronize the individual's circadian clock with the environmental light-dark cycle using various strategies that target either the schedule itself (chronotherapy) or the mechanisms that can reset the circadian timing system (i.e., light, melatonin, and other non-photic time cues). Changing the circadian rhythm implies major lifestyle changes, and requires the patient to sleep at times when he or she was previously alert. Therefore, treatment can be more effective if the therapist can identify motivating factors that will assist the patient in making such lifestyle changes.

Chronotherapy targets the sleep-wake schedule by progressively delaying the sleep and wake times by approximately 2-3 h every two days, until an appropriate earlier bedtime and wake time has been reached and is thereafter maintained (Weitzman et al., 1981).

Phototherapy. Bright light has been successfully used to realign the circadian phase, effectively shifting the phase both forward (phase advance) and backward (phase delay). In general, Delayed Sleep Phase Disorder (DSPD; See Table 2) patients should undergo light exposure in the morning to advance circadian rhythms, whereas Advanced Sleep Phase Disorder (ASPD; See Table 2) patients should undergo light exposure in the evening to delay the rhythms and achieve a more appropriate sleep-wake timetable (Campbell, Dawson, & Anderson, 1993; Chesson et al., 1999; Lack & Wright, 1993; Lack, Wright, Kemp, & Gibbon, 2005; Murphy & Campbell, 1996). Phototherapy can, however, be associated with compliance problems, as it is a demanding treatment for many children and parents. Therefore, a phototherapy treatment plan should

include a behavioral component that addresses the child's motivation and makes the treatment a collaborative effort.

Melatonin administration has been used to shift the sleep phase and as a hypnotic to relieve sleep-onset insomnia in DPSD patients (Arendt, 2006; Lewy, Ahmed, & Sack, 1996; Skene & Arendt, 2006). Melatonin (3-5 mg) can be administered 2 hours prior to the estimated DLMO (dim light melatonin onset), or 4 hours prior to the average sleep onset time. Despite its potential for treating some CRSDs, however, the clinical effectiveness and guidelines for melatonin use (e.g., length of treatment, dosing parameters and timing of administration) have not been firmly established. The studies examining the use of exogenous melatonin to advance the circadian phase in children and adults with DSPD have suffered from methodological flaws and have yielded limited and variable results (Buscemi et al., 2006; Dahlitz et al., 1991; van Geijlswijk, Korzilius, & Smits, 2010). In the US, Melatonin has not been approved by the FDA for the treatment of CRSD, and its production is largely unregulated. Similarly, Health Canada only recommends the use of melatonin to treat sleep disorders in adults, while it is considered "off-label" for the treatment of sleep difficulties in younger patients (Cummings, 2012). Clearly, more studies on the long-term efficacy and safety of melatonin are required, especially in the context of children and adolescents.

4.3.5 Parasomnias

The most common parasomnias of childhood include sleep walking, nightmares, sleep terrors, confusional arousals, and nocturnal enuresis (i.e., bedwetting) (Bansal & Sheldon, 2008). When parasomnias are not excessively bothersome and do not pose any significant danger, the usual management is to educate the parent and child that the experiences are normal and that the child will likely "grow out of them" (Clore & Hibel, 1993; Mason & Pack, 2007). It is also important to teach good sleep hygiene to minimize potential triggers for parasomnias (Markov, Jaffe, & Doghramji, 2006). This can include creating a consistent sleep schedule that ensures adequate rest, avoiding caffeine, exercising early in the day, and creating a relaxing bedtime ritual (e.g., a bath, reading, etc.). It is important for the family to institute safety interventions to minimize the physical risks posed by these episodes. These interventions could include alarms on the bed or door to alert family members if the child gets up and begins to walk, locks on the windows and entry doors, and barriers in front of stairs or other areas where the child could fall or trip. Parents should be made aware that children with habitual sleepwalking who fall asleep during car rides could unintentionally try to get out of the car. Also, sleepwalking may occur more frequently when children are away from home or on an altered schedule, such as while attending camp or sleep-overs. In rare cases, highly recurrent or problematic sleepwalking can be treated with clonazepam given at bedtime.

Nocturnal enuresis

Nocturnal enuresis is a very common problem in children, and it can be disruptive for both children and their families (Thiedke, 2003). Fortunately, effective non-pharmacologic and pharmacologic treatment options are available. Continence training is a very important element of any treatment regimen. In particular, the use of a bed-wetting alarm has been found to have the highest success rate (in terms of both relieving bedwetting and preventing relapse); however, some families may have difficulty with this treatment approach. Medications such as

desmopressin and imipramine have also been used to treat nocturnal enuresis, although both have also been associated with relatively high rates of relapse (Thiedke, 2003).

The treatment of nocturnal enuresis in childhood is beyond the scope of this consensus statement.

For more information:

Bloomfield, E. R., & Shatkin, J. P. (2009). Parasomnias and movement disorders in children and adolescents. *Child and Adolescent Psychiatric Clinics of North America, 18* (4), 947-965**.**

Kotagal, S. (2012). Treatment of dyssomnias and parasomnias in childhood. *Current Treatment Options in Neurology, 14,* 630–649.

4.3.6 Sleep-related Movement Disorders

Children with RLS often have depressed serum ferritin levels, indicating reduced iron stores (Durmer & Quraishi, 2011; Kotagal & Silber, 2004; Picchietti, 2007; Picchietti & Stevens, 2008); in this case, the disorder may improve with iron supplementation (i.e., ferrous sulfate, 6 mg/kg/day mixed with orange juice and taken on an empty stomach) (Davis, Rajput, Rajput, Aul, & Eichhorn, 2000; Kryger, Otake, & Foerster, 2002; Mohri et al., 2012). The objective is to raise serum ferritin levels above 50 ug/L, as lower levels have been associated with periodic limb movements in childhood (Durmer & Quraishi, 2011; Kotagal & Chopra, 2012). The treatment may need to last several months, with periodic monitoring of serum ferritin levels (Sullivan, 2012).

Sleep initiation and maintenance may be difficult when there is significant discomfort in the extremities. In such instances, gabapentin (3-12 years: 10-50 mg/kg/day; > 12 years: 300 -3600 mg/day about an hour prior to bedtime; ("PDR.net," 2013) can be used to alleviate the sensory discomfort. Gabapentin enacarbil is a recently introduced extended-release formulation that can be given to older teens (≥18 years: 600 mg once daily with supper (Garcia-Borreguero et al., 2002; Happe, Sauter, Klosch, Saletu, & Zeitlhofer, 2003).

Agents that have been approved for treatment of RLS in adults such as ropinirole (0.25-6 mg/day at bedtime; Kushida, 2006) and pramipexole (0.125-0.375 mg/day at bedtime; “PDR.net”, 2013) can also be tried in older children (Muhle et al., 2008; M. A. Picchietti & Picchietti, 2010; Walters et al., 2000), especially when symptoms are refractory to iron supplementation and gabapentin. The safety and efficacy of these agents has not been established in younger children (Sullivan, 2012).

For more information:

Picchietti, M. A., & Picchietti, D. L. (2010). Advances in pediatric restless legs syndrome: Iron, genetics, diagnosis and treatment. *Sleep Medicine*, *11*(7), 643-651.

Allen, R. P., Picchietti, D., Hening, W. A., Trenkwalder, C., Walters, A. S., & Montplaisir, J. (2003). Restless legs syndrome: Diagnostic criteria, special considerations, and epidemiology. A report from the Restless Legs Syndrome Diagnosis and Epidemiology Workshop at the National

Institutes of Health, International Restless Legs Syndrome Study Group. *Sleep Medicine, 4*(2), 101-119.

Chesson, A. L., Anderson, W. M., Littner, M., Davila, D., Hartse, K., Johnson, S., Wise, M., & Rafecas, J. (1999). Practice parameters for the nonpharmacologic treatment of chronic insomnia: An American Academy of Sleep Medicine report. *Sleep, 22*(8), 1128-1133.

5. CONCLUSION

Healthy sleep is the goal for all infants, children and adolescents. Essential steps toward achieving this goal include: 1) modifying lifestyle habits that contribute to sleep deprivation; 2) increasing the awareness of parents, educators and healthcare providers with respect to the impact of sleep on the physical and mental health of children and adults; 3) increasing the priority given to sleep in everyday life, educational policies and public health policies; and 4) reinforcing the right of every person to obtain sufficient sleep throughout life. Active management may be needed to overcome critical barriers, such as: 1) the lack of knowledge and awareness regarding the importance of healthy sleep and normative sleep needs; 2) overexposure to electronic media in the evening; 3) consumption of caffeinated beverages; and 4) inadequate recognition and treatment of sleep disorders. Insufficient training in pediatric sleep disorders among healthcare professionals can hinder not only the proper diagnosis and treatment of pediatric sleep disorders, but also the provision of effective anticipatory guidance for their prevention. Thus, the development and maintenance of physical and social environments that encourage healthy sleep in safe settings should be a priority for governments and communities. Sleep hygiene should be a key element of any healthy living school policies and interventions along with active living and healthy nutrition. Parents and caregivers should participate in school-led healthy living initiatives and sustain these efforts at home. In addition, medical schools and training programs for nurses, psychologists and other health care providers must provide sufficient training on prevention and diagnosis, as well as evidence-based interventions for pediatric sleep disorders.

6. RECOMMENDATIONS

Because of the high prevalence of sleep disorders and deprivation in pediatric populations, their scope, and their negative impact on children, adolescents and families, and because pediatric sleep disorders are underdiagnosed and undertreated, and many families have trouble accessing sleep services, the *College of Family Physicians of Canada, Canadian Paediatric Society, Canadian Sleep Society,* and the *Canadian Psychological Association,* in order to improve healthy sleep for children and adolescents, recommend the following:

That health care professionals:

1) Screen for the presence of pediatric sleep deprivation and disorders;

- Review the child's sleep patterns and help identifying the consequences of sleep deprivation (e.g., inattention, hyperactivity, impulsivity, poor concentration, emotional lability, negative mood, weight gain, etc.) at well-baby, well-child or adolescent visits

2) Evaluate sleep disorders and deprivation and search for causes starting with an appropriate history and physical examination. For infants 0-5 years, health-related information may be collected using the Rourke Baby Record, while the Greig Health Record may be used for children and adolescents aged 6-17 years (Rourke, Leduc & Rourke, 2011; Greig, 2010);

3) Determine when investigations such as a sleep study need to be conducted;

4) Offer intervention and counseling;

5) Make appropriate referrals for sleep disorders that require more specialized care.

That health care professionals and their professional organizations advocate for:

- The development of Canadian Healthy Sleep Guidelines (as part of the Healthy Living Guidelines) that reflect current, evidence-based recommendations for healthy sleep among children and adolescents;
- Social awareness and promotion of sleep-friendly activities for children, such as age-appropriate timing of evening TV shows and extracurricular activities for children and adolescents.
- The establishment of a school wellness council that involves qualified resource people. Local physicians should be available to help schools in defining their interventions;
- Advocate public health promotion and disease prevention in the school context that promote educational and environmental interventions in favor of healthy living which take into account healthy sleeping (e.g., protect children's evening time and mitigate heavy work load, exams and extracurricular activities schedules that are not conductive to sufficient sleep).
- The integration of healthy sleeping as a core pillar of healthy living policies;
- The integration of topics in sleep physiology / pathology / screening / intervention / prevention into the curricula of all Canadian medical schools and training programs for other health care professionals, including psychologists and nurses
- The funding of quality research on the promotion of healthy sleep and its relationship with active living, healthy nutrition, and educational success.
- The integration of healthy sleeping habits and counseling in preventive clinical practices (les pratiques cliniques preventives; PCP); and

- The integration of notions of sleep and sleep deprivation (i.e., normative sleep, benefits of sleep, harmful effect of sleep deprivation, etc.) in group training interventions on parenting skills.

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Appendix 1: Sample

Sleep Logs

1- *Parent-Report Sleep Log 1 (Attention, Behavior and Sleep Lab, Douglas Mental Health University Institute)*

2- *Child Self-Report Sleep Log 1(Attention, Behavior and Sleep Lab, Douglas Mental Health University Institute)*

3- *Self-Report Sleep Log 3 (Montreal Children's Hospital, McGill University Health Centre)*

From Gruber et al.

Gruber R, Weiss S, Frappier J, et al. Position statement on pediatric sleep. 2013. http://www.canadiansleepsociety.ca/publisher/articleview/frmArticleID/396.

SLEEP AND ACTIVITY LOG

Produced by: Dr. Gruber's Attention, Behavior, and Sleep Lab,
Douglas Mental Health university Institute, Montreal (QC)

This sleep log belongs to: _____

Date: _____Sunday / Monday / Tuesday / Wednesday / Thursday / Friday / Saturday
Today is: School day / P.E.D, day / Holiday Filled by: Mother/ Father/ Other_____

1) Medication

- Today my child took medication? **Yes / No**
- If yes, please indicate: What medication?

  _____
- The dosage per day:_____
- How many times did your child take it? _____At what time? _____

2) Morning

- My child woke today at:_____
- When waking up he/she was : **Alert / Sleepy**
- How difficult was it for your child to get out of bed? **Very difficult / Difficult / Easy**
- My child is feeling **Well / Sick**: (please describe)_____
- How long did my child sleep? _____hours and _____minutes.
- Did my child wake up during the night? **Yes / No** How many times?_____

1st Wake up: Time: _____Why? _____For how long? _____
2nd Wake up: Time: _____ Why? _____For how long? _____
3rd Wake up: Time: _____ Why? _____For how long? _____
4th Wake up: Time: _____ Why? _____For how long? _____
5th Wake up: Time: _____ Why? _____For how long? _____

- Did my child brush his/her teeth this morning? **Yes / No**

3) Evening

- My child was in bed at:_____
- How long did it take my child to fall asleep? _____
- Any problems at bedtime? _____
- How did my child fall asleep? **By him/herself / With a toy / With a parent in the room / Other (please describe if other)** _____
- How was my child's behavior today?
  As usual / Better than most days / Worse than most days
  (please describe): _____
- How was my child's attention today?
  As usual / Better than most days / Worse than most days
- How was my child's mood today?
  As usual / Better than most days / Worse than most days
- Did my child make it to school on time today? **Yes / No**
  (**If no, please explain**) _____
- How much activity did my child do today (include in-school activities)? _____**minutes**
- Did my child exercise within 1 hour of his/her bedtime? **Yes / No**
- Did my child eat healthily today? **Yes / No**
  (**Please describe**) _____
- Did my child have a large meal within 1 hour of his or her bedtime? Yes / No
- Did my child brush his or her teeth before going to bed? Yes / No

My Sleep Log

Produced by:

Dr. Gruber's Attention, Behavior, and Sleep Lab,
Douglas Mental Health university Institute, Montreal (QC)

Today is : _____

I woke up at: _____
I went to bed at: _____

I took medication: Yes / No
If yes, what kind? _____

Today is : _____

I woke up at: _____
I went to bed at: _____

I took medication: Yes / No
If yes, what kind? _____

Today is : _____

I woke up at: _____
I went to bed at: _____

I took medication: Yes / No
If yes, what kind? _____

Today is : _____

I woke up at: _____
I went to bed at: _____

I took medication: Yes / No

If yes, what kind? _____

Today is : _____

I woke up at: _____

I went to bed at: _____

I took medication: Yes / No

If yes, what kind? _____

Today is : _____

I woke up at: _____

I went to bed at: _____

I took medication: Yes / No

If yes, what kind? _____

Today is : _____

I woke up at: _____

I went to bed at: _____

I took medication: Yes / No

If yes, what kind? _____

Today is : _____

I woke up at: _____

I went to bed at: _____

I took medication: Yes / No

If yes, what kind? _____

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* F M U - 5 3 9 9 *

JEREMY RILL SLEEP LABORATORY

SLEEP DIARY

Numéro de dossier / Unit Number / Nom du patient / Patient's Name

Date(yymmdd)		☾ Midnight	☾ 2:00	☾ 4:00	☼ 6:00	☼ 8:00	☼ 10:00	☼ Noon	☼ 2:00	☼ 4:00	☾ 6:00	☾ 8:00	☾ 10:00	COMMENTS*
	Mon													
	Tues													
	Wed													
	Thur													
	Fri													
	Sat													
	Sun													
	Mon													
	Tues													
	Wed													
	Thur													
	Fri													
	Sat													
	Sun													

Legend (see reverse for instructions)

↓ -you get into bed ↑-you get out of bed shade in the boxes that correspond to your sleep times

C -caffeinated drinks E -exercise for at least 20 mins M -medications (please specify in COMMENTS column)

* **COMMENTS** – please write why your sleep was disturbed and which medications you took. use another sheet if necessary, write the date beside each entry.

Instructions

Please give your best guess in filling out this diary. Do not look at the clock in the middle of the night; just give your best guess the next morning. If you have any questions with this sleep diary, please call the Jeremy Rill Sleep Laboratory of the Montreal Children's Hospital (514) 412-4321.

Legend (what to write)

Draw an ↓ when you get into bed. Draw an ↑ when you get out of bed. Shade in the boxes corresponding to the time that you are asleep. Write a **C** if you take caffeinated drinks. Write an **E** for exercise you do that lasts more than 20 minutes. Write an **M** for medications you take. Write the reasons why your sleep was disturbed and what medications you took in the **COMMENTS** column.

Example:

Bedtime: Nine thirty on Monday night. Go to 9:30 p.m. on Monday night and draw a down-arrow ↓ to show you got into bed at this time.

First fell asleep: Ten o'clock Monday night. You think that you fell asleep at ten o'clock Monday night. Go to 10:00 o'clock Monday night and shade in from that point until you wake up.

Awakening from sleep: Four o'clock for two hours. You wake up during your sleep and are awake for two hours from 4:00 to 6:00 a.m.. There should be no shading between 4:00 and 6 a.m. Tuesday morning. No arrow would be placed if you stayed in bed during this wake period.

Final awakening: Ten o'clock Tuesday morning. You woke up for the day at ten o'clock on Tuesday morning. Based on the above there would be shading from 6:00 a.m. until 10:00 a.m.

Out of bed: Ten thirty a.m. Tuesday. Go to 10:30 a.m. on Tuesday morning and draw an up-arrow ↑ signifying you got out of bed at this point.

Nap: Tuesday afternoon from one thirty to four o'clock. Go to noon on Tuesday afternoon and draw a down arrow ↓ signifying when you got into bed. You think that you fell asleep at 2 o'clock so you shade in from 2:00 to 4:00. You draw an up-arrow ↑ to show when you got out of bed.

Caffeinated drinks (C): You drank one caffeinated drink at 3:00 o'clock on Monday afternoon. Go to 3:00 on Monday afternoon and write a **C**.

Exercise (E): You exercised for at least 20 minutes at 7:30 o'clock on Monday evening. Go to 7:30 on Monday evening and write an **E**.

Medications (M): You took your medications at 11:00 o'clock on Tuesday night. Go to 11:00 o'clock on Tuesday night and write an **M**.

Comments: Write the reasons your sleep was disturbed and what medications were taken.

Date (yymmdd)		☾ Midnight	☾ 2:00	☾ 4:00	☼ 6:00	☼ 8:00	☼ 10:00	☼ Noon	☼ 2:00	☼ 4:00	☾ 6:00	☾ 8:00	☾ 10:00	COMMENTS
04-03-01	Mon									C		E	↓	
04-03-02	Tues						↑	↓		↑			M	4:00 a.m. barking dog M-ventolin

Appendix 2:

Subjective Sleep Questionnaires:

1- BEARS

Owens, J. A., & Dalzell, V. (2005). Use of the 'BEARS' sleep screening tool in pediatric residents' continuity clinic: a pilot study. *Sleep Medicine, 6*, 63-69.

2- Modified Pediatric Epworth Sleepiness Scale

Melendres, C. S., Lutz, J. M., Ruban, E. D., & Marcus, C. L. (2004). Daytime sleepiness and hyperactivity in children with suspected sleep-disordered breathing. *Pediatrics, 114*, 768-775.

Johns, M. W. (1991). A new method for measuring daytime sleepiness: The Epworth Sleepiness Scale. *Sleep 14*, 540–545.

Johns, M. W. (1992). Reliability and factor analysis of the Epworth Sleepiness Scale. *Sleep, 15*, 376–381.

3- Child Sleep Habits Questionnaire

Owens, J. A. Spirito, A., & McGuinn, M. (2000). The children's sleep habits questionnaire (CSHQ): Psychometric properties of a survey instrument for school-aged children, Sleep, 23(8), 1-9.

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Special Section: Sleep Medicine Education based on NIH Sleep Academic Award Program

Use of the 'BEARS' sleep screening tool in a pediatric residents' continuity clinic: a pilot study

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Abstract

Objective: To assess the effectiveness of a simple, 5-item pediatric sleep screening instrument, the BEARS (B = Bedtime Issues, E = Excessive Daytime Sleepiness, A = Night Awakenings, R = Regularity and Duration of Sleep, S = Snoring) in obtaining sleep-related information and identifying sleep problems in the primary care setting.

Setting: Pediatric residents' continuity clinic in a tertiary care children's hospital.

Methods: BEARS forms were placed in the medical records of a convenience sample of 2 to 12 year old children presenting for well child visits over the 5 month study period. Sleep-related information recorded in the BEARS visit and in the pre-BEARS visit, which was the subject's most recent previous well child check (WCC), was coded with respect to whether or not a sleep problem was indicated, and whether sleep issues were addressed.

Results: A total of 195 children had both a documented pre-BEARS and BEARS WCC visit. BEARS visits were significantly more likely than the pre-BEARS visits to have any sleep information recorded (98.5% vs. 87.7%, $p<0.001$), and to have information recorded about bedtime issues (93.3% vs. 7.7%, $p<0.001$), excessive daytime sleepiness (93.9% vs. 5.6%, $p<0.001$), snoring (92.8% vs. 7.2%, $p<0.001$), nighttime awakenings (91.3% vs. 29.2%, $p<0.001$), and regularity and duration of sleep (65.3% vs. 31.5%, $p<0.001$). Significantly more sleep problems were identified during the BEARS visits in the domains of bedtime issues (16.3% vs. 4.1%, $p<0.001$), nighttime awakenings (18.4% vs. 6.8%, $p<0.001$) and snoring (10.7% vs. 4.6%, $p=0.012$). Finally, almost twice as many BEARS charts had sleep mentioned in the Impression and Plan (13.1% vs. 7.3%), which approached significance ($p=0.07$).

Conclusions: The BEARS appears to be a user-friendly pediatric sleep screening tool which significantly increases the amount of sleep information recorded as well as the likelihood of identifying sleep problems in the primary care setting.

Keywords: Sleep; Screening tools; Primary care

Sleep disturbances are among the most common issues raised by parents during health supervision, and it is estimated that upwards of 25% of children experience a significant sleep problem at some point during childhood [1]. Snoring, for example, the most common symptom of sleep-disordered breathing, has a high prevalence in childhood, affecting some 3–12% of preschool-aged children [2], and obstructive sleep apnea syndrome is conservatively estimated to affect 1–3% of the pediatric population [3]. Other studies have reported an overall prevalence of a variety of parent-reported sleep problems ranging from 37% in a community sample of 4–10-year-olds [4] to 25–50% in pre-school aged samples [5]. Although many sleep problems in infants and children are transient and self-limited in nature, certain intrinsic and extrinsic risk factors such as difficult temperament [6], chronic illness [7], and maternal depression [8] may predispose some children to develop more chronic sleep disturbances. Inadequate or poor sleep in children may have negative consequences on a host of functional domains, including mood [9], behavior [10,11], school performance [12,13], and health outcomes [14].

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The impact of childhood sleep problems is further intensified by their direct effect on parents' sleep, resulting in parental daytime fatigue, mood disturbances, and a decreased level of effective parenting [15]. Furthermore, the financial burden of childhood sleep problems is considerable; it has been estimated that the economic cost of health professional contacts for infant crying and sleeping problems, for example, is the equivalent of 104 million US dollars per annum [16]. However, a number of empirically supported behavioral [17] and medical treatments for childhood sleep disorders exist and have been found to result in improved health-related and behavioral and academic outcomes [18,19].

It is clear from the above considerations, that pediatric sleep problems meet most if not all of the criteria for clinical conditions that warrant the implementation of screening procedures, including high prevalence, significant clinical impact, a natural history that may be affected by screening and intervention, and the availability of acceptable and effective treatments [20]. Therefore, it is especially important for pediatricians both to screen for and identify these treatable sleep disorders in children and adolescents during routine health encounters. The recent American Academy of Pediatrics clinical guidelines for the assessment and management of obstructive sleep apnea in children [21], for example, recommends that all children should be regularly screened for snoring in order to prevent and minimize the morbidity associated with sleep-disordered breathing. In addition, the screening process presents an opportunity during the well child visit to educate parents about normal sleep and the consequences of inadequate sleep in children, and to teach parents both primary and secondary prevention strategies. The recognition and evaluation of sleep problems in children by primary care providers requires not only familiarity with the developmentally appropriate differential diagnoses of common presenting sleep complaints (difficulty initiating and maintaining sleep, episodic nocturnal events, etc.), but also an understanding of the association between sleep disturbances and daytime consequences, such as irritability, inattention, and poor impulse control.

Despite the magnitude and clinical importance of sleep issues, several studies have documented that there is a low level of recognition of sleep disorders by primary care physicians in both adults [22–24] and children [25,26]. For example, in a recent survey of over 600 community-based pediatricians, over 20% of the respondents did not routinely screen for sleep problems in school-aged children in the context of the well-child visit, only about one quarter of routinely screened toddlers and preschoolers for snoring, and less than 40% questioned adolescents directly about their own sleep habits, despite the respondents' acknowledgement of the importance of sleep's impact on health, behavior, and school performance [25]. The supposition that parents would spontaneously volunteer the presence of any sleep problems and lack of time were cited as the primary reasons for not screening by the sample. Another recent study [26] used a validated pediatric sleep questionnaire to identify a series of children with sleep-related symptoms at two community-based general pediatrics clinics and reviewed medical chart notes for the previous 2 years to determine how often sleep problems had been addressed. Fewer than 15% of patients had current chart notes that mentioned any of the questionnaire-defined sleep problems; diagnoses were mentioned for two of 86 patients and no treatments were discussed.

A number of studies have suggested that both education about screening [27] and the use of brief screening tools, including simple chart reminders, are cost-effective methods of increasing compliance with screening and preventive health care measures by health care providers [28,29]. Several studies have demonstrated that the use of simple screening tools, such as three question chart prompts and algorithms, was found to be associated with increased detection of obstructive sleep apnea in adults [24,30]. Because no similar pediatric sleep screening tools have been empirically tested, the purpose of the following study was to evaluate the effectiveness of a simple pediatric sleep screening instrument, the BEARS, in eliciting information and identifying sleep problems in a primary care setting. In order to be an effective screening tool, the instrument needed to be 'user-friendly', brief and easy to remember, acceptable to practitioners and parents, and had to screen for the most common pediatric sleep complaints across a range of ages in a diverse patient population. In this pilot study, we compared the amount and type of sleep information obtained and the likelihood of identifying sleep problems in a sample of pediatric primary care patients during the well child encounter, using both a standard, single, chart sleep prompt and the BEARS screening tool.

1. Methods

1.1. Subjects

This study was conducted in a pediatric residents' continuity clinic in a children's teaching hospital in Rhode Island, which serves a multi-ethnic, primarily low-income population. The clinic has approximately 21,000 primary care visits per year. Patients are primarily seen for clinic visits by pediatric residents, as well as by pediatric nurse practitioners on the clinic staff and occasionally by pediatric attending faculty. Because of resident turnover and scheduling considerations, patients may be seen by multiple different practitioners for well childcare (WCC).

Study subjects were a convenience sample of patients between the ages of 2 and 12 years presenting for a routine WCC visit on designated study days over the 5-month study period between September and January. Subjects were included if there was a BEARS form (explained below) for that WCC visit ('BEARS visit') in the chart and if

the subject had had at least one previous WCC documented on the standard clinic form in the medical record ('pre-BEARS visit'). The subjects' most recent previous well child visits recorded on the standard clinic form was used as an historical control group.

1.2. Screening procedure

The standard WCC clinic form contained a series of brief one- or two-word prompts (such as 'School,' and 'Development') to direct residents in obtaining and recording medical information during the clinical interview. A single word prompt 'Sleep' was included as part of the standard clinic form. The standard form also included separate sections to record physical exam findings as well as an Impression and Plan section.

The BEARS is a screening tool developed by the investigators, which was designed to address the most common sleep issues in toddlers, preschoolers, and school-aged children. It incorporates five basic sleep domains: *B*edtime Problems, including difficulty going to bed and falling asleep; *E*xcessive Daytime Sleepiness, which includes behaviors typically associated with daytime somnolence in children; *A*wakenings during the night; *R*egularity of sleep/wake cycles (bedtime, wake time) and average sleep duration; and *S*noring. These domains are felt to reflect the most common presenting sleep complaints in children. This screening tool prompts clinicians to ask parents an initial screening question about possible problems in each domain, eliciting a yes or no response. If the answer is 'yes' then the parents are asked to describe the problem. For example, if a parent responded 'yes' to snoring, the parents would be asked to describe how often the child snored and whether apnea accompanied the snoring.

During each clinic session in the 2 weeks preceding the study period, the investigators conducted brief (10 min) group orientation sessions with all the residents to explain the BEARS screen and inform them of placement of the BEARS forms in patient charts. No additional didactic information about sleep and/or sleep problems in children was included in these orientation sessions. Half-page forms with the BEARS screen were placed in the medical records of WCC visits in the appropriate age range by the certified nursing assistants at the time of the visit. Charts were collected after each visit of each clinic day. The medical record for the BEARS visit and the pre-BEARS visit were copied. The BEARS was initially test piloted by the investigators in several pediatric primary care settings to assess its adaptability to different age groups. The project was reviewed and approved by the hospital institutional review board.

1.3. Data collection

Charts were then reviewed and demographic information recorded. The professional status of the practitioner who saw the patient at each visit (nurse practitioner, attending, resident) and, when applicable, the resident's training level was also recorded. The medical records for each BEARS and pre-BEARS WCC visit for each patient were then independently coded by two reviewers for the following information: (1) whether or not any sleep information was recorded for the visit in the five BEARS domains, and (2) whether the sleep information recorded for the visit indicated a definite sleep problem, a probable sleep problem, no problem, or insufficient information to make a determination. In order to assess whether the use of the BEARS screen was more likely to result in documentation of other sleep issues as well, additional sleep-related variables not included in the five BEARS domains, such as parasomnias, napping, co-sleeping, and presence of a TV in the bedroom, were also coded for each visit. In addition, the Impression and Plan section of the medical record for each visit was coded for (1) whether or not a sleep problem was mentioned and, if so, in what domain(s) and (2) whether a sleep-related diagnostic test (e.g., lateral neck radiograph, overnight sleep study) was ordered. In the event of a coding discrepancy between reviewers, each chart was re-reviewed and a consensus was reached. Visits were included even if the resident chose not to fill out the BEARS form.

1.4. Analyses

Data were entered into the SPSS version 9.0. Descriptive statistics were used to describe the sample as a whole including frequency counts and means. A McNemar test was used to compare the pre-BEARS and BEARS visits with respect to the following categorical variables: presence or absence of any sleep information, presence or absence of sleep information in each of the five BEARS domains, presence or absence of a definite or probable sleep problem (two problem categories combined) in each domain, and presence or absence of a sleep problem mentioned in the Impression and Plan section of the WCC. The total number of other sleep issues documented in the medical record for both the BEARS and pre-BEARS visits were also compared using a paired sample *t*-test.

Pearson correlations were used to examine the association between frequency of sleep problems and the age of the patient at the time of the WCC visit.

2. Results

A total of 195 children had both a documented pre-BEARS and BEARS WCC visit. As expected, the average age at the BEARS visit was significantly older at 5.60 SD 2.85 years than the average age at the pre-BEARS visit of 4.35 SD 2.77 years ($t = -20.586$, $P < 0.001$). Half (52%) of the sample was male, 44% was Hispanic, 27% was African-American, 16% Caucasian, 1% Asian, and 12% other.

Table 1
Comparison of percentage of medical records with sleep information recorded between Pre-BEARS and BEARS WCC Visits

	Pre-BEARS (%)	BEARS (%)	*P* value
General sleep	87.7	98.5	<0.001
Bedtime issues	7.7	93.3	<0.001
Excessive day sleepiness	5.6	93.9	<0.001
Awakenings at night	29.2	91.3	<0.001
Regularity/duration	31.5	65.3	<0.001
Snoring	7.2	92.8	<0.001
Parasomnias	3.1	7.7	0.035

Eighty percent was at poverty or low-income level, based on Rhode Island zip code information.

Table 1 compares pre-BEARS and BEARS visits with respect to whether any information was recorded about sleep in general, and whether there was any information recorded in each of the sleep domains. Significantly more BEARS visits had *any* sleep information in general recorded; BEARS WCC visits were over 10 times more likely than the pre-BEARS visits to have information recorded about bedtime issues and excessive daytime sleepiness, three times more likely to have had information recorded about nighttime awakenings, and twice as likely to have had information recorded about regularity and duration of sleep. Finally, over 10 times as many BEARS charts had information recorded about snoring.

In terms of other sleep-related information recorded, although parasomnias were not directly addressed in the BEARS screen, they were still twice as likely to be mentioned in the BEARS visits charts. Using a paired *t*-test comparison, the difference between the total number of sleep-related (non BEARS domains) variables recorded in the BEARS WCC visits (mean = 0.99 ± 0.95) compared to the pre-BEARS visits (mean = 0.59 ± 0.88) was highly significant ($t = 4.791$, $P < 0.001$).

Table 2 compares the presence of a probable or definite problem in each of the BEARS sleep domains and parasomnias between the pre-BEARS and BEARS WCC visits. Significantly more probable or definite problems were identified during the BEARS visits compared to the pre-BEARS visits in the domains of bedtime issues (four-fold), nighttime awakenings (almost three-fold), and snoring (more than twice the number). The BEARS visits were not significantly more likely to identify a problem with excessive daytime sleepiness. A regular bedtime of later than 10 p.m. was recorded and used to define a probable or definite problem with sleep regularity and duration. Although the BEARS visits were more likely to identify a problem in this domain, this was not statistically significant ($P = 0.454$). Twice as many parasomnias were reported during the BEARS visits but this difference was also not significant ($P = 0.219$).

Table 2
Comparison of percentage of medical records with identified sleep problems (definite or probable) between Pre-BEARS and BEARS WCC visits

	Pre-BEARS (%)	BEARS (%)	*P* value
Bedtime issues	4.1	16.3	<0.001
Excessive day sleepiness	4.1	5.6	0.629
Awakenings at night	6.8	18.4	<0.001
Regularity/duration	3.6	5.7	0.454
Snoring	4.6	10.7	0.012
Parasomnias	2.0	4.1	0.219
Sleep in impression/plan	7.3	13.1	0.071

Table 2 also compares the percentage of pre-BEARS and BEARS visits that mention sleep-related issues in the Impression and Plan section of the medical record. Almost twice as many of all BEARS charts had sleep mentioned in the Impression and Plan (13.1 vs. 7.3%); this difference approached significance ($P = 0.071$). There was no difference in the likelihood of ordering a sleep diagnostic test (e.g. overnight sleep study, lateral neck film) between groups, but very few sleep-related diagnostic studies were ordered by either group. Behavioral interventions mentioned in the Plan section were largely general recommendations regarding behavior (setting limits, providing positive reinforcement), but also included some specific sleep strategies such as limiting television viewing, setting a bedtime routine, shifting the sleep-wake schedule, and limiting naps. One patient was referred to otolaryngology, but no patients were referred to a sleep clinic.

In order to assess the impact of potential confounding factors, the following additional analyses were conducted. Given that the BEARS sample was conducted when the group was older, it was possible that increasing age accounted for the increased likelihood of identifying a sleep problem. However, the number of sleep problems identified did not significantly correlate with age at either of the visits ($R = 0.011$, $P = -0.953$, pre-BEARS and $R = -0.072$, $P = 0.953$ BEARS). In order to assess the possible impact of resident training level (more experienced residents more likely to identify a sleep problem), training levels for resident-conducted visits were dichotomized into post-graduate level one (PL-1) or post-graduate level two or greater and compared using the McNemar test. The percentage of less experienced PL-1 residents conducting BEARS (34%) and pre-BEARS visits (31%) was not significantly different ($P = 0.724$). Finally, it was possible that the BEARS visits were more likely to have more medical information in general recorded that was not just limited to sleep-related information than the pre-BEARS visits. In order to further examine this possibility, we compared information recorded about another behavioral/developmental domain, school problems, between pre-BEARS and BEARS visits. As mentioned above, 'School' was another one of the single word prompts in the standard well child form. School problems were not significantly more likely to be documented during the BEARS vs. the pre-BEARS visits ($P = 0.115$).

3. Discussion

The results of this study suggest that the use of a simple 5-question screening tool for pediatric sleep problems is significantly more likely than the use of a standard single chart prompt to yield sleep information in general, as well to yield information about specific sleep domains. There was a 2–ten-fold difference in the amount of information recorded during the BEARS visits in each of the five sleep domains and parasomnias. In addition, the information obtained with the BEARS screen was significantly more likely to result in sleep problems being identified in the chart for bedtime issues, night wakings, and snoring. Increases in the percentage of problems in the individual sleep domains identified at the BEARS visits ranged from more than two-fold for snoring and almost three-fold for night wakings, to four-fold for bedtime issues. The finding that the BEARS was more effective in eliciting information is even more significant when consideration is given to the fact that, in most clinical settings, well child encounter forms do not include any sleep prompts at all and there was such a prompt included in the pre-BEARS visits. The BEARS is therefore likely to have even more impact when compared to usual clinical practice.

Furthermore, the percentage of patients identified as having sleep problems in the various domains during the BEARS visits was similar in many cases to the prevalence of those same problems cited in the literature. For example, a number of studies have suggested that the prevalence of bedtime resistance in early school-aged children, the same age group as the sample population, is in the range of 15% [4] to 27% [31], which is much higher than the 4% identified in the control visits and closer to the 16% prevalence identified at the BEARS visits. Similarly, the percentage of children identified by the BEARS as having significant snoring (11%) was very similar to the prevalence of frequent snoring for that age group reported in previous studies [2, 32]. This further supports the suggestion that the use of a standard single sleep question may fail to elicit adequate clinical information to determine the presence of a potentially serious sleep problem, particularly in the realm of sleep-disordered breathing.

The use of the BEARS screen was also more likely to result in documentation of additional sleep-related information, including sleeping arrangements, presence of a television in the child's bedroom, naps, and co-sleeping. Such information may not only be useful in elucidating the context of and factors contributing to existing sleep problems, but may be important in identifying potential intervention points to prevent future sleep problems from developing. For example, the use of prevention strategies, such as suggesting that parents begin to put infants to bed 'drowsy but awake' at around 4 months of age in order to avoid dependence on parental presence at sleep onset and to foster the infants' ability to 'self-soothe', have been shown to be highly effective in reducing the likelihood of prolonged night wakings [33]. An increased focus during the well child encounter on sleep issues allows for the opportunity to provide additional anticipatory guidance, such as educating parents of newborns about normal sleep amounts and patterns, discussing the importance of regular bedtimes, bedtime routines, and transitional objects for toddlers, and providing parents and children with basic information about good 'sleep hygiene' and adequate sleep amounts.

Although sleep problems were more likely to be identified in the BEARS visits, this did not appear to have as significant an effect on the likelihood of having a specified diagnostic and/or treatment plan documented in the medical record. Previous chart review studies of sleep histories in adults have reported similar findings [30] regarding of lack of impact on patient management. One possible explanation for this finding is that residents may not feel comfortable and/or knowledgeable enough about sleep problems in their patients to appropriately address them. A recent survey study of community-based practicing pediatricians reported that less than a third of the respondents rated themselves as very confident or confident of their own ability to evaluate sleep problems in children and only one quarter rated themselves as very confident or confident in treating pediatric sleep disorders [25]. The relative lack of attention paid to sleep disorders in post-graduate pediatric education programs [34] may be in part responsible for this clinical knowledge gap.

There were a number of limitations in this pilot study, which should be addressed. First, because of the study design, we were unable to separate out the effectiveness of the BEARS instrument as a screening tool independent of several related factors, including the impact of incorporating the BEARS as a chart reminder into the medical record and the role played by instruction provided to residents on use of BEARS. Although residents were not explicitly informed of the purpose of the study, they may have been somewhat more likely to record information during the BEARS visits because of the attention focused on sleep issues by the orientation sessions. However, these sessions were felt to be necessary in order to provide uniform clarification on the use of the BEARS and specifically did not include any educational component regarding sleep issues in children. Due to logistical constraints in the clinic setting, we were unable to monitor on a daily basis if blank or incomplete BEARS forms were removed from the charts by residents; however, of the BEARS forms collected, only 5% had not been filled out, suggesting a high rate of compliance. Because of concern regarding possible contamination of information about the BEARS across residents, particularly given the fact that residents frequently saw their patients on continuity clinic days other than the one to which they were regularly assigned, we elected to use a design that incorporated historical rather than concurrent controls. We were also unable to follow the residents longitudinally after the study period was

concluded so that we could assess their continued use of the BEARS screen during subsequent well child encounters and thus cannot comment on the longer-term sustainability of the behavioral change. This would clearly be a key issue to study in the future. Finally, as with all chart review studies, the written documentation of the clinical encounter may not have been a complete record of the information actually obtained by the resident during the clinical interview, although this factor was unlikely to be substantially different across the two conditions.

The differences found in information and prevalence and types of sleep problems recorded between the BEARS and control visits may in part have been related to variables other than the sleep screening method employed, including provider- and patient-related factors [35]. For example, because of the study design, the patients were older at the time of the BEARS visit than at the control well child visit, and the increase in sleep problem prevalence may have been a factor of increasing age. However, studies have suggested that sleep problems in general are more prevalent in younger children than in school-aged children [36], and that parents are also more likely to both be aware of and to report sleep problems in younger children as well [4]. Furthermore, we did not find a significant correlation in our sample population between sleep problems and age. It is also possible that the BEARS visits were more likely to be conducted by upper level and thus more experienced residents who were more likely to note and record sleep problems in their patients. However, there was not a significant difference between the BEARS and control visits in the percentage of patients seen by a first-year compared to an upper-level resident.

In conclusion, this study suggests that the use of a simple brief screening tool for pediatric sleep problems is a cost-effective tool for identifying parents' concerns about their children's sleep, particularly in domains such as snoring that may not have been otherwise assessed. Future studies should evaluate the effectiveness of the BEARS screen with both experienced practitioners, such as community-based pediatricians, and with other types of health care professionals, such as family medicine practitioners, nurse practitioners, and mental health providers, in order to assess the generalizability of our results. The BEARS should also be compared to accepted 'gold standards' for the diagnosis of pediatric sleep disorders (International Classification of Sleep Disorders criteria, polysomnography, other pediatric sleep screening tools [37], etc.) in order to assess the validity as well as sensitivity and specificity of the instrument. Finally, combining the use of the BEARS with sleep curriculum materials and ongoing educational efforts may be necessary in order to more definitively impact physician behavior, including optimal management of sleep problems in the primary care setting, and is worthy of further study.

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Appendix A

The 'BEARS' is designed to provide a practical and user-friendly vehicle for teaching medical students and residents to incorporate a pediatric sleep history into the standard history and physical in both ambulatory and inpatient settings. The 'BEARS' instrument is divided into five major sleep domains, which provides a comprehensive screen for the major sleep disorders affecting children in the 2–18-year old age range. Each sleep domain has a set of age-appropriate 'trigger questions' for use in the clinical interview.

Examples of developmentally appropriate trigger questions:

	Preschool (2–5 years)	School-aged (6–12 years)	Adolescent (13–18 years)
*B*edtime problems	Does your child have any problems going to bed? Falling asleep?	Does your child have any problems at bedtime? (P) Do you have any problems going to bed? (C)	Do you have any problems falling asleep at bedtime? (C)
*E*xcessive daytime sleepiness	Does your child seem over tired or sleepy a lot during the day? Does she still take naps?	Does your child have difficulty waking in the morning, seem sleepy during the day or take naps? (P) Do you feel tired a lot? (C)	Do you feel sleepy a lot during the day? in school? while driving? (C)
*A*wakenings during the night	Does your child wake up a lot at night?	Does your child seem to wake up a lot at night? Any sleepwalking or nightmares? (P) Do you wake up a lot at night? Have trouble getting back to sleep? (C)	Do you wake up alot at night? Have trouble getting back to sleep? (C)
*R*egularity and duration of sleep	Does your child have a regular bedtime and wake time? What are they?	What time does your child go to bed and get up on school days? weekends? Do you think he/she is getting enough sleep? (P)	What time do you usually go to bed on school nights? Weekends? How much sleep do you usually get? (C)

(*continued on next page*)

	Preschool (2–5 years)	School-aged (6–12 years)	Adolescent (13–18 years)
*S*leep-disordered breathing	Does your child snore a lot or have difficulty breathing at night?	Does your child have loud or nightly snoring or any breathing difficulties at night? (P)	Does your teenager snore loudly or nightly? (P)

B, bedtime problems; E, excessive daytime sleepiness; A, awakenings during the night; R, regularity and duration of sleep; S, sleep-disordered breathing; P, Parent C, Child.

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Daytime Sleepiness and Hyperactivity in Children With Suspected Sleep-Disordered Breathing

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ABSTRACT. *Objectives.* **Excessive daytime sleepiness (EDS) is seen less frequently as a presenting complaint in children with sleep-disordered breathing than in adults. Instead, symptoms of hyperactivity are often described. We hypothesized that children with suspected sleep-disordered breathing (S-SDB) were both sleepier and more hyperactive than control subjects. Furthermore, we hypothesized that overnight polysomnographic parameters correlated with sleepiness and hyperactivity.**

Methods. **A cross-sectional study was conducted at a university-affiliated hospital and a community-based pediatric clinic. A total of 108 patients with S-SDB (mean [standard deviation] age: 7 ± 4 years) and 72 control subjects (8 ± 4 years) were recruited. A modified Epworth Sleepiness Scale (ESS) and the Conners Abbreviated Symptom Questionnaire were administered. Polysomnography was performed in patients with S-SDB.**

Results. **Patients with S-SDB had a higher ESS (8.1 ± 4.9 vs 5.3 ± 3.9) and a higher Conners score (12.8 ± 7.6 vs 9.0 ± 6.2) than control subjects. On the basis of adult criteria, 28% of patients had EDS. There was no difference in the ESS and Conners scores of patients with primary snoring and patients with obstructive sleep apnea. The ESS had weak correlations with polysomnographic parameters.**

Conclusions. **Although the ESS score of children with S-SDB was within the normal range for adults, these children were sleepier and more hyperactive than control subjects. However, these data should be confirmed by a population-based study.** *Pediatrics* **2004;114:768–775;** ***obstructive sleep apnea, Epworth score, polysomnography.***

ABBREVIATIONS. OSAS, obstructive sleep apnea syndrome; EDS, excessive daytime sleepiness; SDB, sleep-disordered breathing; S-SDB, suspected sleep-disordered breathing; PSG, polysomnography; ESS, Epworth Sleepiness Scale; Sao_2, arterial oxygen saturation; $ETco_2$, end-tidal carbon dioxide tension; PS, primary snoring; PLM, periodic limb movement; REM, rapid eye movement; UARS, upper airway resistance syndrome; EEG, electroencephalogram.

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The childhood obstructive sleep apnea syndrome (OSAS) is a disorder of breathing during sleep characterized by prolonged partial upper airway obstruction and/or intermittent complete obstruction that disrupts normal ventilation during sleep and normal sleep patterns.[1] It has an estimated prevalence of 1% to 2% among young children[2,3] and can lead to serious morbidity and even mortality if left untreated.[4–7]

In contrast to adults, excessive daytime sleepiness (EDS) is seen less frequently as a presenting complaint in children with sleep-disordered breathing (SDB).[4,8–10] Symptoms of inattention and hyperactivity are often described.[8] The prevalence of EDS in children with SDB has been shown to vary over a wide range, from as low as 8% to as high as 84%.[7,9–12] The significantly differing values may be attributable in part to a lack of standard assessment techniques for sleepiness in children. Previous studies have used different subjective criteria such as the use of parental report. Only 1 previous study used objective criteria to evaluate sleepiness in children with SDB.[13] The present study aimed to use a simple and inexpensive instrument, which has been validated in adults,[14,15] to evaluate EDS in children with suspected SDB (S-SDB).

We hypothesized that children with S-SDB were both sleepier and more hyperactive than control subjects. Furthermore, we hypothesized that parameters on overnight polysomnography (PSG) correlated with EDS and hyperactivity in these children.

METHODS

The study protocol was approved by the Institutional Review Board of Johns Hopkins University. Informed consent was secured from the subjects' parents or legal guardians. Assent was obtained from all subjects ≥5 years of age. Patients with S-SDB and control subjects were studied. Control subjects were screened using Brouillette's scoring system.[16] A modified Epworth Sleepiness Scale (ESS)[14] and the Conners Abbreviated Symptom Questionnaire[17] for hyperactivity were administered to all subjects. Patients with S-SDB then underwent overnight PSG.

Study Population

S-SDB patients were recruited sequentially and prospectively from all new patients who were referred to the Pediatric Sleep Disorders Clinic at Johns Hopkins Hospital for evaluation of clinically S-SDB secondary to adenotonsillar hypertrophy. Children were included when they were aged 2 to 18 years. They were excluded when they had other medical or neurologic conditions, had craniofacial abnormalities, had undergone adenotonsillectomy or other upper airway surgery, or were on medications that could affect their level of alertness. All patients were seen by a pediatric sleep specialist, and those with possible sleep disorders

other than OSAS were excluded. Control subjects were recruited from a general pediatric clinic in a primary care setting and from the Dermatology Clinic at Johns Hopkins Hospital.

Screening of Control Subjects

Control subjects were screened for OSAS using Brouillette's scoring system. Brouillette et al[16] formulated a scoring system to determine the likelihood of OSAS on the basis of history alone. Three variables were included in this score: difficulty breathing during sleep, observed apnea, and snoring. A child with a score of <-1 had no OSAS, whereas a child with a score >3.5 had OSAS; scores between -1 and 3.5 were indeterminate. In our study, control subjects with an OSAS score ≥ -1 were excluded.

Questionnaires

Two questionnaires (a modified ESS and the Conners Abbreviated Symptom Questionnaire for hyperactivity) were administered to the child's caregiver by a trained research assistant. In addition, questions were asked directly to the first 46 children ≥6 years of age.

The ESS is a measure of a person's general level of daytime sleepiness.[14] It is an 8-item questionnaire detailing an individual's propensity to fall asleep during commonly encountered situations. Scores can range from 0 to 24. In adults, an ESS score >10 is taken to indicate increased daytime sleepiness.[14] The ESS was modified slightly in this study to be more applicable to children. The mention of alcohol was deleted in question number 7. In addition, question 8 was taken to indicate that the subject was a passenger in the car (Appendix 1).

The Conners Abbreviated Symptom Questionnaire for hyperactivity is a 10-item index from the Revised Conners Parent Rating Scale.[17] This is used in rating children who are aged 3 to 17 years for the presence of inattention, distractibility, and overactivity. Raw scores may range from 0 to 30, with a score of 15 considered clinically relevant (Appendix 2).

PSG

Patients with S-SDB underwent an overnight polysomnogram. Control subjects did not undergo PSG. Standard PSG consisted of electroencephalogram (C3A2/C3O1); electromyogram (submental and tibial); electrooculogram (right, left); arterial oxygen saturation (Sao_2), oximeter pulse wave form, and end-tidal carbon dioxide tension ($ETco_2$); oronasal airflow using a thermistor; and thoracic and abdominal wall motion (piezo belts or respiratory inductance plethysmography). Sleep was staged based on the criteria of Rechtschaffen and Kales.[18] Arousals were scored according to the American Sleep Disorders Association criteria.[19] Standard pediatric scoring criteria were used for respiratory events.[1] Hypopneas were scored when there was a decrease in airflow ≥50% associated with either a 3% desaturation or an arousal.[1] The apnea-hypopnea index was defined as the total number of obstructive apneas, hypopneas, and mixed apneas per hour of sleep. OSAS was defined as an obstructive apnea index ≥1/hour.[1,7] Severity of OSAS was classified on the basis of the obstructive apnea index, with mild OSAS having an obstructive index between 1 and 4/hour, moderate OSAS 5 and 9/hour, and severe OSAS ≥10/hour.[20] Primary snoring (PS) was defined as snoring without episodes of apnea, desaturation, hypoventilation, or excessive arousals.[21] Periodic limb movements (PLMs) were scored using the International Classification of Sleep Disorders (Revised) criteria.[21] The scorer was blinded to the results of the questionnaires.

Statistical Analysis

Data were expressed as means and standard deviations, where appropriate. χ^2 analysis was used for categorical variables. The unpaired t test was used to compare ESS scores of patients with S-SDB versus control subjects, as well as the Conners scores of patients with S-SDB versus control subjects. The ESS scores of children who were younger than 5 years were also analyzed separately as children in this age group usually take daytime naps. To assess for the contribution of puberty to the degree of daytime sleepiness in our subjects, we analyzed separately the ESS scores of children who were older than 12 years (which was arbitrarily set as the cutoff age for puberty). One-way analysis of variance was performed to test the difference in ESS scores of patients with mild, moderate, and severe OSAS. Similarly, one-way analysis of variance was used to test the difference in Conners scores between patients with mild, moderate, and severe OSAS. As the Conners score has been studied only in children 3 to 17 years of age, data were reanalyzed with subjects younger than 3 years excluded. The Spearman correlation coefficient was used to describe the relationship between the ESS score and PSG variables, as well as between the Conners score and PSG variables. PSG variables evaluated include sleep efficiency, arousal index, apnea-hypopnea index, apnea-hypopnea index during rapid eye movement (REM) sleep, Sao_2 nadir, duration of Sao_2 ≤92%, mean $ETco_2$, mean $ETco_2$ during REM sleep, peak $ETco_2$, duration of $ETco_2$ ≥50 mm Hg, and PLM index. A Bonferroni correction for multiple comparisons was used.

RESULTS

Study Group

Of all subjects who were approached to join the study, only 1 family refused. A total of 203 consecutive children were recruited; 23 were excluded. Among the patients with S-SDB, 18 were excluded. Seventeen of these children failed to undergo a sleep study, and 1 had a sleep study done at another institution. Among the control subjects, 5 were excluded because of an OSAS score >-1. Therefore, 108 patients with S-SDB and 72 control subjects composed the study population. The 2 groups were not statistically different on the basis of age, gender, race, and type of insurance (Table 1). The type of insurance was used as a surrogate measure of socioeconomic status. Snoring was present in all but 1 of the patients with S-SDB. Twenty-two patients had a history of daytime sleepiness by parental report.

ESS and Conners Score

The first 46 children who were ≥6 years of age gave separate ESS scores from their parents. As the correlation between parent and child ESS score was good ($r = 0.71$, $P < .001$), subsequent ESS scores were obtained from the accompanying caregiver alone. The mean ESS score given by parents of patients with S-SDB was significantly higher than that of control subjects (8.1 ± 4.9 vs 5.3 ± 3.9; $P < .001$; Fig 1). Taking an ESS score >10 as the cutoff for increased daytime sleepiness, 32 (28%) patients with S-SDB and 9 (12%) control subjects had EDS ($P < .007$). There was no significant difference in the ESS scores of patients who had S-SDB and were younger than 5 years and those who were 5 years and older (8.2 ± 4.2 vs 8.1 ± 5.2, respectively). Likewise, the ESS scores of children who had S-SDB and were ≤12

TABLE 1. Demographic Data of Study Population

	Patients With S-SDB	Controls
N	108	72
Age, y, mean ± SD (range)	7 ± 4 (2–16)	8 ± 4 (2–17)
Female gender, *n* (%)	58 (55)	43 (60)
Race, *n* (%)		
White	26 (24)	26 (36)
Black	79 (73)	46 (64)
Other	3 (3)	0 (0)
Private insurance, *n* (%)	37 (34)	28 (39)

There was no statistical difference between patients with S-SDB and control subjects on the basis of age, gender, race, and type of insurance. The type of insurance was used as a surrogate measure of socioeconomic status.

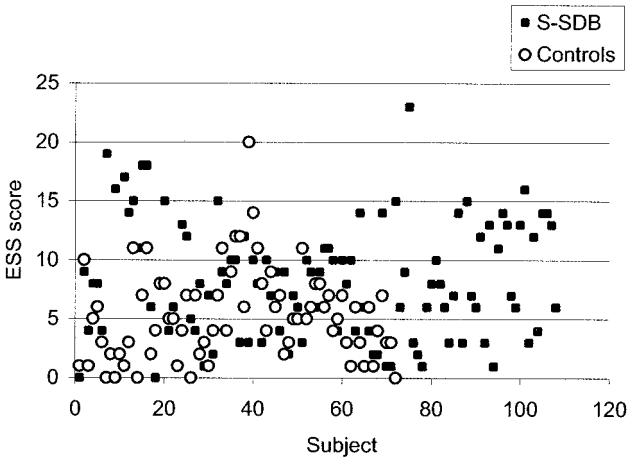

Fig 1. The ESS score of patients with S-SDB was higher than the score of control subjects ($P < .001$).

years (8.1 ± 4.8) were not statistically different from the ESS scores of those who were older than 12 years (8.4 ± 6.6). The ESS scores of patients with S-SDB and control subjects who were younger than 5 years (8.2 ± 4.2 vs 8.0 ± 4.2) were not significantly different, but the ESS scores of patients who had S-SDB and control subjects who were older than 12 years (8.4 ± 6.6 vs 4.3 ± 2.6) were ($P < .05$).

The Conners score of patients with S-SDB (12.8 ± 7.6) was significantly higher ($P < .001$) than the score of control subjects (9.0 ± 6.2; Fig 2), although it was lower than the score considered clinically relevant. Excluding children <3 years of age, the significant difference between the Conners score of patients with S-SDB and controls persisted (13.0 ± 7.5 vs 9.0 ± 6.3, respectively).

Subgroup Analysis

The PSG results are shown in Table 2. Of the 108 patients with clinically S-SDB, 63 (58%) received a diagnosis of PS on overnight PSG. The remaining 45 (42%) had various degrees of OSAS. Twenty-one had mild, 8 had moderate, and 16 had severe OSAS. None of the patients with S-SDB fulfilled the criteria for PLM disorder. The ESS score of patients with

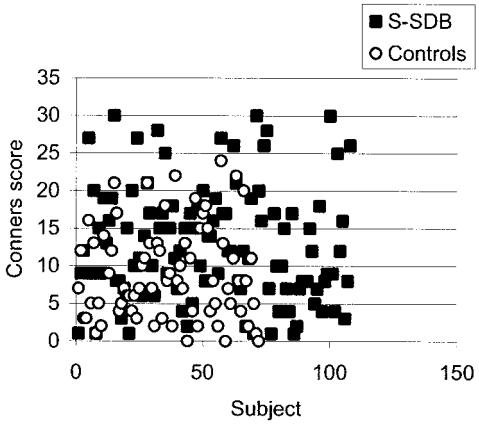

Fig 2. The Conners score of patients with S-SDB was higher than the score of control subjects ($P < .001$).

TABLE 2. PSG Results of Children With S-SDB

PSG Parameter	Mean ± SD (range)
Sleep efficiency, %	83 ± 12 (39–97)
Arousal index, *n*/h	10 ± 8 (1–53)
Apnea-hypopnea index, *n*/h	8 ± 18 (0–48.2)
REM apnea-hypopnea index, *n*/h REM sleep	17 ± 30 (0–148.2)
REM obstructive index, *n*/h REM sleep	10 ± 20 (0–107.5)
Peak $ETco_2$, mmHg	52 ± 5 (41–67)
Sao_2 nadir, %	88 ± 10 (48–98)
Mean Sao_2, %	98 ± 2 (88–100)
Mean Sao_2 (REM sleep), %	97 ± 3 (73–100)
PLM index, *n*/h	0.3 ± 0.8 (0–3.3)

OSAS (8.3 ± 5.6) was not statistically different from the ESS score of those who had a diagnosis of PS alone (8.0 ± 4.5). There was also no difference between the ESS score of patients with mild, moderate, and severe OSAS (Fig 3). Similarly, the Conners score was not statistically different between patients with OSAS and those with PS (12.6 ± 8.3 and 13.0 ± 7.0 for OSAS and PS respectively). There was also no statistically significant difference in the Conners scores of patients with mild, moderate, and severe OSAS (Fig 4).

In children with OSAS, the ESS score had a statistically significant but weak correlation with the mean Sao_2 during REM sleep ($r = -0.41$, $P < .05$), PLM index ($r = 0.40$, $P < .05$), apnea-hypopnea index ($r = 0.32$, $P < .05$), mean Sao_2 ($r = -0.31$, $P < .05$), and Sao_2 nadir ($r = -0.31$, $P < .05$) but not with other parameters tested (Table 3). However, when the *P* value was adjusted for multiple comparisons using the Bonferroni correction factor, these correlations failed to reach significance. There was no significant correlation between the Conners score and PSG parameters.

As most children with OSAS obstruct primarily during REM sleep,[22] data were reanalyzed using the apnea-hypopnea index during REM sleep. No significant correlation was found between the ESS or Conners scores and the REM apnea-hypopnea index. However, ESS scores correlated with the mean Sao_2 during REM sleep.

DISCUSSION

To our knowledge, this is the first study to evaluate the use of the ESS score in children. We have shown that children with S-SDB were sleepier than age-, gender-, and race-matched control subjects. Our data also confirmed previous reports that children with S-SDB exhibit more symptoms of attention-deficit/hyperactivity disorder than normal children. PSG parameters correlated only weakly with the ESS score and had no significant correlation with hyperactivity.

Daytime Sleepiness

EDS is a cardinal feature of adult OSAS.[21] In children, it is relatively uncommon, but its reported prevalence has varied over a wide range.[7,9–12] Although the mean ESS score of children with S-SDB

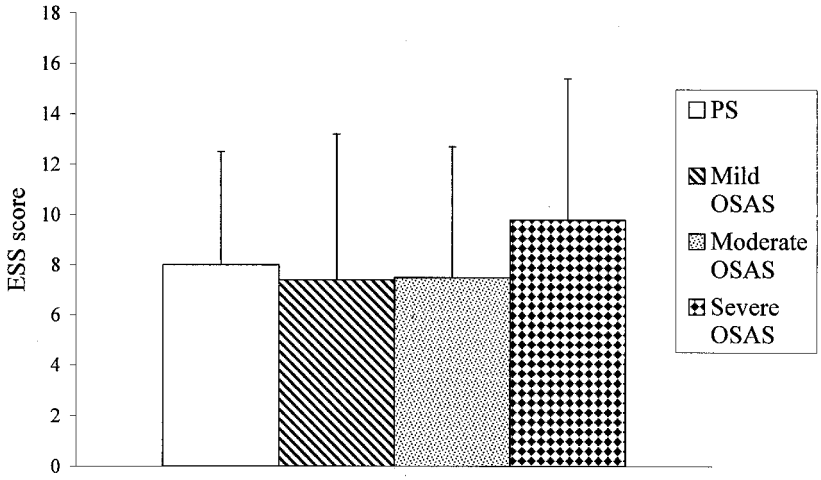

Fig 3. One-way analysis of variance revealed that the ESS scores of patients with mild, moderate, and severe OSAS did not differ significantly.

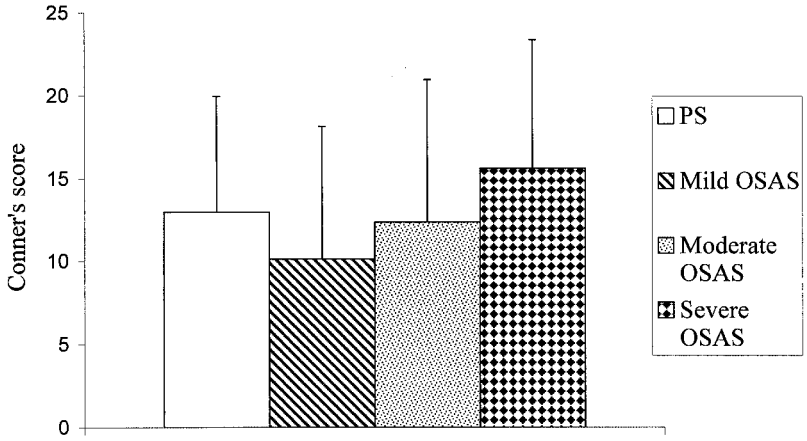

Fig 4. One-way analysis of variance revealed that the Conners scores of patients with mild, moderate, and severe OSAS did not differ significantly.

TABLE 3. Correlation Between PSG Parameters and ESS and Conners Scores

PSG Parameter	Correlation With ESS Score (*r*)	Correlation With Conners Score (*r*)
Sleep efficiency, %	−0.05	−0.09
Arousal index, *n*/h	0.25	0.04
Apnea-hypopnea index, *n*/h	0.32*	0.24
REM apnea-hypopnea index, *n*/h	0.26	0.13
REM obstructive apnea index, *n*/h	0.24	0.19
Peak $ETco_2$, mm Hg	0.19	0.29
Mean $ETco_2$, mm Hg	0.04	0.12
Mean $ETco_2$, REM sleep, mm Hg	0.13	0.19
Duration $ETco_2$ ≥50 mm Hg, min	−0.06	0.03
Sao_2 nadir, %	−0.31*	−0.22
Mean Sao_2, %	−0.31*	−0.16
Mean Sao_2, REM sleep, %	−0.41*	−0.27
Duration Sao_2 ≤92%, min	0.13	−0.03
PLM index, *n*/h	0.40*	0.25

* $P < .05$.

did not reach the level generally set for EDS based on adult studies (ESS >10),[14] it was statistically higher than the ESS score of control subjects. Therefore, as a group, we have shown that children with S-SDB are relatively sleepy compared with nonsnoring control subjects.

An interesting finding was the lack of a significant difference between the ESS scores of children with S-SDB and control subjects who were younger than 5 years. That children in this age group usually nap during the day may account for this lack of difference in parental perception of sleepiness.

After classifying children who were referred for S-SDB into those with PS and those with OSAS, there was no significant difference in daytime sleepiness between these 2 groups. Daytime symptoms are traditionally thought to be absent in patients with PS.[21] Our results, however, suggest that snoring may in

itself be associated with sleepiness in children. Similar findings have been shown in adult studies.[23,24] In a cross-sectional cohort of 5777 adults, the authors found that snoring was independently associated with excess sleepiness.[23] In another adult study, daytime sleepiness, as measured by both ESS and multiple sleep latency test, was compared between normal controls and patients with either upper airway resistance syndrome (UARS), sleep hypopnea syndrome, or OSAS.[25] There was a significant difference between the ESS scores of the controls versus the patients with SDB. However, the ESS and multiple sleep latency test were similar for the 3 patient groups.

Classifying the children with OSAS in our study according to severity did not yield a significant relationship between the severity of OSAS and the degree of daytime sleepiness. It is possible that children may have varying susceptibility to the effects of OSAS severity, accounting for the lack of relationship. Previous studies have shown conflicting results in this area. Some adult studies have similarly shown this lack of relationship,[26–28] whereas others have shown a positive relationship between OSAS severity and the degree of sleepiness.[13,29] As there were only a small number of children with moderate to severe OSAS, it is also possible that our study was underpowered to detect a significant relationship.

Relationship Between PSG Parameters and Daytime Sleepiness

We found weak correlations between daytime sleepiness and the mean Sao_2 during REM sleep, PLM index, the apnea-hypopnea index, and the lowest recorded Sao_2. There was no significant relationship between daytime sleepiness and sleep efficiency or the arousal index.

The link between the ESS score and PSG parameters is unclear. Some studies have shown significant correlation between these, whereas others have not. In adults with OSAS, sleep fragmentation as a result of recurrent arousal is thought to be the primary reason for EDS.[30,31] Other factors have also been shown to correlate with daytime sleepiness, such as nocturnal hypoxemia and the apnea-hypopnea index.[32,33] In our study, none of the PSG parameters could be used to predict daytime sleepiness, as the correlations were weak. Similar findings have been shown in a number of adult studies.[26,27,34]

The PLM disorder, defined as a PLM index ≥ 5, is believed to cause daytime sleepiness due to recurrent arousals, resulting in sleep fragmentation.[21] Although none of the patients in the present study satisfied the criteria for this disorder, the PLM index showed a weak correlation with daytime sleepiness (Table 2). With this weak correlation, it is unlikely that PLMs contribute significantly to the daytime sleepiness in children with S-SDB. Using both objective and subjective measures of daytime sleepiness, a recent study also showed comparable findings.[35]

The absence of a strong relation between PSG parameters and daytime sleepiness in this study brings up the possibility either that we are not measuring the right parameter during routine PSG or that the commonly measured parameters in PSG are not sensitive determinants of daytime sleepiness. Sleep fragmentation seems not to be a major factor in the development of daytime sleepiness in children with OSAS, as there was no significant correlation between daytime sleepiness and the arousal index. In fact, previous studies have shown that apneas in children are terminated by arousal less often than in adults, leading to less fragmented sleep.[36] Theoretically, measures of increased upper airway resistance, such as esophageal pressure swings, may be more sensitive determinants of daytime sleepiness.

UARS, which is part of the spectrum of SDB, is characterized by EDS as a result of fragmented sleep caused by brief arousals not associated with discrete apneas or gas exchange abnormalities.[25] It is diagnosed by demonstrating an association between esophageal pressure swings and arousals.[37] As esophageal pressure was not measured in this study, it is possible that some of the subjects who were labeled as PS may actually have had UARS. However, in that case, we would have expected to see a correlation between the arousal index and the ESS score.

Alternatively, subcortical arousals that have been shown to be common in children may also be contributing to sleepiness.[38] The pulse transit time, which is a noninvasive measure of subcortical arousal, was found to be a more sensitive measure of sleep disruption than visible electroencephalogram (EEG) arousals.[39] In addition, significant changes in spectral EEG characteristics have been shown in obstructive events not terminated by EEG arousal.[40] These measures of subcortical arousal, which are not part of the routinely measured parameters on PSG and were not analyzed in this study, may perhaps have a stronger relation with daytime sleepiness. This may be an area for future research. With the correlation, although weak, between sleepiness and oxygen saturation, hypoxemia may play a bigger role in the cause of sleepiness in children with SDB than EEG arousals.

Attention Deficit and Hyperactivity

On the basis of the Conners score, we found that children with S-SDB had more symptoms of attention-deficit/hyperactivity disorder than control subjects (Fig 2). There was no difference in attention-deficit/hyperactivity symptoms between the PS and OSAS groups.

These data confirm previous reports that children with SDB commonly manifest neurobehavioral complications, specifically hyperactivity and inattention.[2,5,41–43] It is interesting that the Conners score of children with OSAS was not statistically different from the Conners score of children with PS alone. Similar findings were shown in a recent study of 113 children who were referred for S-SDB.[42] There was no difference in the hyperactivity scores of children

who subsequently received a diagnosis of SDB by polysomnography and those without. These findings suggest that snoring by itself may affect a child's daytime behavior.

None of the PSG parameters measured in our study correlated to a significant degree with symptoms of attention-deficit/hyperactivity disorder. Likewise, Chervin et al[42] found no correlation between hyperactivity and respiratory parameters on PSG. However, they found a correlation between hyperactivity and the PLM index. This association was found only in patients with SDB.

The absence of a relation between PSG parameters and symptoms of attention deficit and hyperactivity again indicates that we may be measuring the wrong parameters or that commonly measured parameters on nocturnal PSG are not sensitive determinants of hyperactivity. Hyperactivity has been proposed to be a child's way of acting out daytime sleepiness. As such, measures of subcortical arousal associated with respiratory events may give better correlations with hyperactivity.

It is important to note that we excluded children who were taking medications that could affect their level of alertness, including those who were taking drugs used for attention-deficit/hyperactivity disorder. As such, our data may have underestimated attention-deficit and hyperactivity in children with S-SDB. In addition, differences in birth history and other medical conditions may have played a role. However, as we excluded those with significant medical conditions, it is unlikely that these factors played a major role.

Study Limitations

This study compared clinically referred children with S-SDB with control subjects from other clinics. Thus, clinical referral bias may account for the difference in ESS and Conners scores between patients and control subjects. The ESS and the Conners score are subjective methods of assessing sleepiness and hyperactivity and as such may also be prone to report bias. Of note, however, is that only 22 of the 108 patients with S-SDB had a history of daytime sleepiness by parental report. Most children presented with chief complaints of snoring and witnessed apnea during sleep. Furthermore, all children were seen by a sleep specialist, and children with other types of sleep disorders were excluded. Nevertheless, it would be important to confirm these results with a population-based study.

Our control subjects did not undergo a sleep study but were screened for OSAS on the basis of history using the OSAS score developed by Brouillette et al.[16] This score has been shown to differentiate am individual with no SDB from one with OSAS, although it has not been shown to be effective at differentiating PS from OSAS.

The use of a thermistor to measure nasal airflow has its limitations. Being a qualitative measurement, it is not as sensitive in detecting flow limitation, which may be associated with increased upper airway resistance or hypopneas.[1] Nasal cannula pressure measurements, which have been validated in adults,[44] have been shown to detect apneas, hypopneas, and flow-limited events not identified by thermistors in children.[45,46] However, as this detects only nasal airflow, it may miss events in children who frequently mouth-breathe[45,46] and in those who experience nasal obstruction.[47] As such, to minimize the limitations associated with the use of a thermistor, we used the $ETco_2$ wave form as an additional means of measuring airflow.

It should be noted that the majority of subjects in both the S-SDB and control groups were black, reflecting the ethnic mix of our hospital's clinical population. Thus, the study sample may not be representative of the rest of the United States.

CONCLUSIONS

In summary, we have shown that although the mean ESS score of patients was within the normal range for adults, children with S-SDB were sleepier and more hyperactive than control subjects. These findings have important implications in clinical practice as we have shown that even the mildest form of SDB may be associated with daytime symptoms. The long-term consequence of this is currently unknown, but it is possible that the neurocognitive functioning of a child may be affected. We have also shown that the ESS is a simple and useful test to administer to children. However, the results of this study need to be confirmed by a population-based study with a more representative population. Additional studies are needed to elucidate the causative factors for EDS and hyperactivity in children with S-SDB.

APPENDIX 1: MODIFIED EPWORTH SLEEPINESS SCALE

How likely are you/your child to doze off or fall asleep in the following situations, in contrast to feeling just tired? This refers to your usual way of life in recent times. Even if you/your child have not done some of these things recently, try to work out how they would have affected you/your child. Use the following scale to choose the most appropriate number for each situation:

0 = no chance of dozing
1 = slight chance of dozing
2 = moderate chance of dozing
3 = high chance of dozing

Situation	Chance of Dozing
Sitting and reading	
Watching TV	
Sitting inactive in a public place (eg, movie theater or a meeting)	
As a passenger in a car for an hour without a break	
Lying down to rest in the afternoon when circumstances permit	
Sitting and talking to someone	
Sitting quietly after lunch	
In a car, while stopped for a few minutes in traffic	

APPENDIX 2: CONNERS ABBREVIATED SYMPTOM QUESTIONNAIRE

Observation	Not at All	Just a Little	Pretty Much	Very Much
1. Restless or overactive				
2. Excitable, impulsive				
3. Disturbs other children				
4. Fails to finish things he/she starts–short attention span				
5. Constantly fidgeting				
6. Inattentive, easily distracted				
7. Demands must be met immediately–easily frustrated				
8. Cries often and easily				
9. Mood changes quickly and drastically				
10. Temper outbursts, explosive and unpredictable behavior				

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EUROPEAN ENVIRONMENTAL RULES PROPEL CHANGE IN U.S.

"Generally stricter European laws reflect a different philosophical approach to regulation, says Dr Indra Spiecker, a lawyer specializing in comparative law and assistant professor for American law at the University of Osnabruck in Germany. American lawmakers primarily look to cost-benefit analysis, which holds that the benefit of imposing regulation should outweigh its cost. European nations have more readily embraced what is called the precautionary principle. Essentially, Europeans emphasize the cost of inaction, while Americans tend to focus on the cost of action. . . . The EU is now considering sweeping new regulation of its chemical industry that has unleashed what analysts here say is the biggest lobbying effort in Brussels ever mounted by American industry. The new law, known as Reach, would place the burden of proof of safety on the producers before its sale, rather than waiting for the problems to spur regulation later. It would force American chemical companies to comply with the legislation in order to continue exporting to Europe—and raises the fear of similar legislation in the United States. The chemical industry points out that few if any of the unregulated chemicals are causing obvious health crises and says the legislation is overly bureaucratic and expensive. The American Chemical Council has marshaled its members to alter or derail the legislation."

Pohl O. *New York Times*. July 6, 2004

Noted by JFL, MD

Daytime Sleepiness and Hyperactivity in Children With Suspected Sleep-Disordered Breathing

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A New Method for Measuring Daytime Sleepiness: The Epworth Sleepiness Scale

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Summary: The development and use of a new scale, the Epworth sleepiness scale (ESS), is described. This is a simple, self-administered questionnaire which is shown to provide a measurement of the subject's general level of daytime sleepiness. One hundred and eighty adults answered the ESS, including 30 normal men and women as controls and 150 patients with a range of sleep disorders. They rated the chances that they would doze off or fall asleep when in eight different situations commonly encountered in daily life. Total ESS scores significantly distinguished normal subjects from patients in various diagnostic groups including obstructive sleep apnea syndrome, narcolepsy and idiopathic hypersomnia. ESS scores were significantly correlated with sleep latency measured during the multiple sleep latency test and during overnight polysomnography. In patients with obstructive sleep apnea syndrome ESS scores were significantly correlated with the respiratory disturbance index and the minimum SaO_2 recorded overnight. ESS scores of patients who simply snored did not differ from controls. **Key Words:** Sleepiness—Questionnaire—Sleep propensity—Insomnia—Obstructive sleep apnea syndrome.

A large proportion of adult patients who present to sleep disorder centers have disorders associated with excessive daytime sleepiness. These include obstructive sleep apnea syndrome (OSAS), periodic limb movement disorder (PLMD), narcolepsy, idiopathic hypersomnia and other miscellaneous disorders (1). The severity of their chronic daytime sleepiness is an important aspect of each patient's assessment. Thus, there is a great need for a simple standardized test for measuring a patient's general level of sleepiness, which is independent of short-term variations in sleepiness, with the time of day and from day to day.

The multiple sleep latency test (MSLT) is widely used and is generally believed to provide a valid measurement of sleepiness on the particular day of the test (2,3). It is based on the premise that the sleepier the subject, the quicker he will fall asleep when encouraged to do so while lying down in a nonstimulating environment. The MSLT has a reasonably high test–retest reliability over periods of months in normal subjects (4). Assuming the same reliability holds true for patients, the MSLT must be considered the standard method for measuring their chronic daytime sleepiness. However, the MSLT is very cumbersome, time-consuming and expensive to perform. It takes all day, both for the subject and the polysomnographer and is not easy to justify as a routine test for all patients.

Other measures of sleepiness have been devised (5,6). In the maintenance of wakefulness test (MWT) the latency to sleep onset is measured with the subject sitting in a dimly lit, warm, quiet room, trying to stay awake rather than to fall asleep (5). However, all such tests share the disadvantage of the MSLT in being cumbersome and expensive. Similar criticisms can be levelled at tests of sleepiness based on pupillometry (7), or cerebral evoked potentials (8). Other assessments of sleepiness have involved prolonged psychomotor performance tests, the results of which are not related in any simple or consistent way to sleepiness in different subjects (9).

By contrast, the Stanford sleepiness scale (SSS) is a quick and simple test (10). It involves the subject's own reports of symptoms and feelings at a particular time. Visual analogue scales (VAS) of sleepiness/alertness have also been used in this context (11). However, these tests do not attempt to measure the general level of daytime sleepiness, as distinct from feelings of sleepiness at a particular time. Nor, it appears, is the subjective sleepiness that they measure the same as the objective sleepiness measured by the MSLT (3,7). Scores on the SSS or on a VAS of sleepiness are not significantly correlated with sleep latency in the MSLT,

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TABLE 1. *The Epworth sleepiness scale*

THE EPWORTH SLEEPINESS SCALE

Name: _____
Today's date: _____ Your age (years): _____
Your sex (male = M; female = F): _____

How likely are you to doze off or fall asleep in the following situations, in contrast to feeling just tired? This refers to your usual way of life in recent times. Even if you have not done some of these things recently try to work out how they would have affected you. Use the following scale to choose the *most appropriate number* for each situation:

0 = would *never* doze
1 = *slight* chance of dozing
2 = *moderate* change of dozing
3 = *high* chance of dozing

Situation	Chance of dozing
Sitting and reading	
Watching TV	
Sitting, inactive in a public place (e.g. a theater or a meeting)	
As a passenger in a car for an hour without a break	
Lying down to rest in the afternoon when circumstances permit	
Sitting and talking to someone	
Sitting quietly after a lunch without alcohol	
In a car, while stopped for a few minutes in the traffic	

Thank you for your cooperation

even when measured at virtually the same time (12). These subjective reports may be related more to tiredness and fatigue than to sleep propensity, as manifested by the tendency to fall asleep.

The present report describes the development and use of a new questionnaire, the Epworth sleepiness scale (ESS), designed to measure sleep propensity in a simple, standardized way. The scale covers the whole range of sleep propensities, from the highest to the lowest.

Development of the ESS

The concept of the ESS was derived from observations about the nature and occurrence of daytime sleep and sleepiness. Some people who suffer from excessive daytime sleepiness keep themselves busy and choose not to lie down nor to sit and relax during the day, thereby purposely avoiding daytime sleep. Others who may be bored, with spare time or who are socially withdrawn but who may not be very sleepy, choose to lie down and sleep during the day. About 50% of ostensibly healthy medical students usually sleep during the day at least once in an average week (13). Among 17–22-year-old recruits entering the French army, 19% reported sleeping during the day, regularly or occasionally. But only 5% complained of daytime sleepiness (14). Thus, knowing how frequently or for how long subjects usually sleep during the day will probably not provide a useful measurement of their sleepiness.

By contrast, sleepy people often describe how they doze off inadvertently while engaged in activities that involve low levels of stimulation, relative immobility and relaxation, such as sitting and watching TV. Earlier questionnaire surveys have indicated which situations, commonly encountered in daily life, are the most soporific (15). A large survey among adults in New Mexico asked about their frequency of falling asleep in five situations (16). The authors derived a score from the three "most sleepy" questions, which referred to falling asleep while "inactive in a public place", "at work", and "in a moving vehicle as passenger or driver". MSLTs on 116 of these subjects showed a statistically significant correlation between their sleep latency (SL) and their answers to those three questions ($r = -0.32$, $p < 0.001$).

The ESS is based on questions referring to eight such situations, some known to be very soporific; others less so. The questionnaire, which is self-administered, is reproduced in Table 1. Subjects are asked to rate on a scale of 0–3 how likely they would be to doze off or fall asleep in the eight situations, based on their usual way of life in recent times. A distinction is made between dozing off and simply feeling tired. If a subject has not been in some of the situations recently, he is asked, nonetheless, to estimate how each might affect him.

The ESS tries to overcome the fact that people have different daily routines, some facilitating and others inhibiting daytime sleep. For example, the ESS does not ask how frequently the subject falls asleep while watching TV. That would depend on how frequently he watched TV as much as on his sleepiness. Instead, the subject rates the chances that he would doze off whenever he watches TV.

One question asks how likely the subject would be to doze off while lying down to rest in the afternoon when circumstances permit. It was felt that normal people probably would, and sleepy people certainly would tend to doze off in that situation. Never to do so would indicate an unusually low level of sleepiness, as described by some insomniacs. Some other situations were included in the questionnaire because it was believed that only the most sleepy people would doze in them—while sitting and talking to someone, and in a car while stopped for a few minutes in traffic. These suppositions proved correct.

The numbers selected for the eight situations in the ESS were added together to give a score for each subject, between 0 and 24. These ESS scores proved capable of distinguishing individuals and diagnostic groups over the whole range of daytime sleepiness.

TABLE 2. *The groups of experimental subjects, their ages and ESS scores*

Subjects/diagnoses	Total number of subjects (M/F)	Age in years (mean ± SD)	ESS scores (mean ± SD)	Range
Normal controls	30 (14/16)	36.4 ± 9.9	5.9 ± 2.2	2–10
Primary snoring	32 (29/3)	45.7 ± 10.7	6.5 ± 3.0	0–11
OSAS	55 (53/2)	48.4 ± 10.7	11.7 ± 4.6	4–23
Narcolepsy	13 (8/5)	46.6 ± 12.0	17.5 ± 3.5	13–23
Idiopathic hypersomnia	14 (8/6)	41.4 ± 14.0	17.9 ± 3.1	12–24
Insomnia	18 (6/12)	40.3 ± 14.6	2.2 ± 2.0	0–6
PLMD	18 (16/2)	52.5 ± 10.3	9.2 ± 4.0	2–16

METHODS

Subjects

A total of 180 adult subjects completed the questionnaire. There were 30 controls who were mainly hospital employees, working during the day, who gave a history of normal sleep habits without snoring. There were 150 patients with various sleep disorders, whose ages, sex and diagnostic categories are shown in Table 2. Every new patient who presented to the Epworth Sleep Disorders Unit answered the ESS at their first consultation. After investigation, all patients with the diagnoses listed in Table 2 were included in the study until there were 150. The ages of patients ranged from 18 to 78 years. The mean age within diagnostic groups varied from 36 to 52 years. Men greatly outnumbered women in the snoring, OSAS and PLMD groups. The sexes were about equal in the other groups, apart from the insomniacs where women outnumbered men.

A total of 138 patients had overnight polysomnography, but another 12 who were clearly suffering from either chronic psychophysiological or idiopathic insomnia did not. The latter diagnoses were made on the basis of each patient's history, using the criteria set out in the International Classification of Sleep Disorders (1). Other insomniacs, with mood disorders or drug effects, were excluded.

Twenty-seven patients had MSLTs after overnight polysomnography. They had four naps, at 1000, 1200, 1400 and 1600 hours. Sleep latency was measured from the time lights were switched off until the onset of stage 1 sleep of at least 1 minute duration, or the onset of either stage 2 or rapid eye movement (REM) sleep. The early onset of REM sleep was indicated by the occurrence of REM sleep within 20 minutes of sleep onset. Of the 27 patients, 11 had narcolepsy diagnosed from the patient's history, particularly of cataplexy, associated with an SL of less than 10 minutes and the early onset of REM sleep in two or more naps (10 patients) or in one nap (1 patient with cataplexy). Fourteen of the 27 patients had idiopathic hypersomnia, diagnosed from their excessive daytime sleepiness in the absence of either cataplexy or the early onset of REM sleep in the MSLT. The remaining two patients had excessive daytime sleepiness due to OSAS. The ESS scores for the 27 patients who had MSLTs ranged from 11 to 24.

All patients with primary snoring had presented initially because of the intensity and persistence of their snoring, on most nights at least. Many had been observed at home to pause in their breathing at night, suggesting that they may have had sleep apnea, but this was found not to be of clinical significance by polysomnography. The respiratory disturbance index (RDI) was calculated as the number of apneas and hypopneas causing a drop of >3% in the arterial oxygen saturation per hour of sleep. The RDI for primary snorers was ≤5. The 55 patients with OSAS were divided into three subcategories according to their RDI, regardless of their complaints about daytime sleepiness or insomnia (Table 3). The RDI for mild OSAS was within the range >5–15; for moderate OSAS the range was >15–30, and for severe OSAS it was >30.

A diagnosis of PLMD was made only if there were at least 90 separate movements in one or both legs per night. The mean periodic movement index for these subjects, calculated as the number of movement events per hour of sleep, was 43.6 ± 30.4 (SD). Patients who had both PLMD and OSAS were excluded from this study. However, 9 of the 18 subjects with PLMD snored during polysomnography without having OSAS.

Statistical methods

The ESS scores of male and female control subjects were compared by a Student's *t* test. Differences in ESS scores between the diagnostic groups were tested by one-way ANOVA and then by posthoc Scheffé tests. A separate ANOVA and posthoc Scheffé tests were

TABLE 3. *ESS scores in mild, moderate and severe OSAS*

	Mean RDI ± SD	Total number of subjects (M/F)	ESS scores (mean ± SD)	Range
Mild OSAS	8.8 ± 2.3	22 (21/1)	9.5 ± 3.3	4–16
Moderate OSAS	21.1 ± 4.0	20 (20/0)	11.5 ± 4.2	5–20
Severe OSAS	49.5 ± 9.6	13 (12/1)	16.0 ± 4.4	8–23

used to test the differences in ESS scores between primary snorers and the three categories of OSAS. The Scheffé test is conservative and is suitable for groups with unequal numbers of subjects (17). The distribution of sleep latencies ,measured in minutes, was highly skewed positively and was normalized by $\log_e$ transformation. The relationships between pairs of continuous variables, such as RDI and sleep latency during overnight polysomnography, were tested by Pearson correlation coefficients and linear regression. Statistical significance was accepted at $p < 0.05$ in two-tailed tests.

RESULTS

The mean ESS score for control subjects was 5.9 ± 2.2 (SD) and their modal score was 6. There was no significant difference in the scores between male and female controls (males = 5.64 ± 2.56; females = 6.06 ± 1.84, $t = 0.520$, $p = 0.607$). Consequently, no distinction was made between the sexes in other groups.

Patients suffering from disorders known to be associated with excessive daytime sleepiness reported the likelihood of dozing under circumstances that were not conducive to sleep in normal subjects. For example, 96% of the patients with either narcolepsy or idiopathic hypersomnia reported some chance, and often a high chance, of dozing while sitting and talking to someone, or in a car while stopped for a few minutes in the traffic. Only 6% of controls reported a slight chance of doing so.

Patients with persistent psychophysiological or idiopathic insomnia reported either a complete inability or only a slight chance of dozing while lying down to rest in the afternoon when circumstances permitted. By contrast, 94% of controls reported some likelihood of dozing then.

One-way ANOVA demonstrated significant differences in ESS scores between the seven diagnostic groups in Table 2 ($F = 50.00$; df = 6,173; $p < 0.0001$). Posthoc tests between paired groups showed that the ESS scores for primary snorers did not differ from controls ($p = 0.998$). Scores for OSAS, narcolepsy and idiopathic hypersomnia were significantly higher than for controls ($p < 0.001$) or primary snorers ($p < 0.001$). The insomniacs had significantly lower scores ($p < 0.01$) than all groups other than controls, for which the difference did not quite reach statistical significance ($p = 0.063$). The ESS scores of patients with PLMD did not differ significantly from controls ($p = 0.149$).

A separate one-way ANOVA for the ESS scores of primary snorers and the three subcategories of OSAS showed significant differences between these groups ($F = 23.11$; df = 3,82; $p < 0.001$). Posthoc tests then showed that ESS scores for each level of OSAS were significantly higher than for primary snorers ($p = 0.035$ for mild OSAS; $p < 0.001$ for moderate and severe OSAS). Scores for severe OSAS were higher than for moderate OSAS ($p < 0.001$), but the difference between mild and moderate OSAS did not reach statistical significance ($p = 0.085$).

Considering all 55 patients with OSAS together, there was a significant correlation, on the one hand, between ESS scores and RDI ($r = 0.550$, $p < 0.001$) and on the other hand, between ESS scores and the minimum SaO_2 recorded during apneas overnight ($r = -0.457$, $p < 0.001$). The RDI and the minimum overnight SaO_2 during apneas were also significantly correlated ($r = -0.687$, $p < 0.001$). The linear regression equations for these three relationships, in the form $Y = a + bx$, were as follows:

$$(\text{RDI}) = -0.674 + 2.006(\text{ESS score})$$
$$(\text{minimum } SaO_2\%) = 86.47 - 1.055(\text{ESS score})$$
$$(\text{minimum } SaO_2\%) = 84.15 - 0.440(\text{RDI})$$

Among the 138 patients who had overnight polysomnography there was a significant correlation between ESS score and (ln) sleep latency at night ($r = -0.379$, $n = 138$, $p < 0.001$). In the smaller group of patients who had MSLTs, the correlation between ln (SL) during the day and ESS score was also statistically significant ($r = -0.514$, $n = 27$, $p < 0.01$. The linear regression equation for this relationship was $\ln(\text{SL}) = 3.353 - 0.091(\text{ESS score})$.

Individual ESS scores of 16 or more, indicating a high level of daytime sleepiness, were found only in patients with narcolepsy, idiopathic hypersomnia or OSAS of at least moderate severity (i.e. RDI > 15). All patients with either narcolepsy or idiopathic hypersomnia had higher ESS scores than the controls (i.e. ESS > 10) as did 12 of 13 patients with severe OSAS. The remaining patient in the latter category had an ESS score of 8 and was clinically not much affected by his sleep apnea.

Within the group of patients with PLMD, the periodic movement index, which ranged from 16 to 122 movements per hours of sleep, was not significantly correlated with ESS scores ($r = 0.049$, $n = 18$, $p > 0.1$).

DISCUSSION

These results provide evidence that a questionnaire-based scale as brief and as simple as the ESS can give valid measurements of sleep propensity in adults. ESS scores significantly distinguished groups of patients who are known from other investigations to have differences in their levels of sleepiness, as measured by the MSLT (2,18). ESS scores were significantly correlated

with sleep latency measured during the day with MSLTs and at night with polysomnography. This is despite any effect of the first night in the laboratory. Others have found a significant positive correlation between the SL at night and during the day in the same subject (19).

ESS scores greater than 16, indicative of a high level of daytime sleepiness, were encountered only in patients with moderate or severe OSAS (RDI > 15), narcolepsy or idiopathic hypersomnia. These disorders are known to be associated with excessive daytime sleepiness as measured by the MSLT (2,18). Nevertheless, high ESS scores, by themselves, are not diagnostic of a particular sleep disorder, any more than is an SL of 5 minutes in an MSLT.

ESS scores were correlated with both the RDI and the minimum SaO_2 recorded during polysomnography in patients with OSAS of differing severity. In the past, these measures of the severity of OSAS have been found to be related to the SL in MSLTs in some, but not in all investigations (18,20). The finding that ESS scores can distinguish patients who simply snore from those with even mild OSAS is evidence for the sensitivity of the ESS. The questionnaire should be useful in elucidating the epidemiology of snoring and OSAS, and any associated cardiovascular or cerebrovascular risks. Previous investigations of this kind have tended to blur the distinction between primary snoring and OSAS (21).

In the patients with PLMD, the finding of an almost zero correlation between their periodic movement index and ESS scores suggests that whatever level of daytime sleepiness is associated with PLMD, it is not related simply to the frequency of limb movements. It may be more closely related to the frequency of those movements producing arousal rather than those that do not. This distinction was not made here and further investigation is required to clarify this relationship.

The low ESS scores of patients with idiopathic or psychophysiological insomnia are consistent with evidence that such patients have a low sleep propensity, even when they are able to relax (22). It must not be assumed, however, that this is necessarily so for other kinds of insomnia, such as with mood disorders.

The relatively wide range of ESS scores in the control subjects [2–10] is consistent with evidence that some healthy adults, without recognizable sleep disorders, remain sleepier than others during the day (23). Such differences persist in MSLTs, even after extending the hours of nocturnal sleep to overcome possible sleep deprivation (24). The sleep propensity of a subject on a particular day would be influenced by the quality and duration of prior sleep or of sleep deprivation, the time of day, the presence of various sleep disorders, drug effects, the level of interest and motivation induced by the situation at hand, as well as longer-term physiological differences. The ESS does not distinguish the nature of long-term physiological or pathological processes that produce a particular level of sleep propensity. Other investigations, including overnight polysomnography, are required for that.

The ESS assumes that subjects can remember whether or not and under what circumstances they have dozed off during the day as part of their "usual way of life in recent times". The present results suggest that most patients can give meaningful self reports about this aspect of their behavior and that their ESS scores provide a measurement of their general level of daytime sleepiness, from low to very high levels. This has not been achieved previously by any other published questionnaire.

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Reliability and Factor Analysis of the Epworth Sleepiness Scale

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Summary: The Epworth Sleepiness Scale (ESS) is a self-administered eight-item questionnaire that has been proposed as a simple method for measuring daytime sleepiness in adults. This investigation was concerned with the reliability and internal consistency of the ESS. When 87 healthy medical students were tested and retested 5 months later, their paired ESS scores did not change significantly and were highly correlated ($r = 0.82$). By contrast, ESS scores that were initially high in 54 patients suffering from obstructive sleep apnea syndrome returned to more normal levels, as expected, after 3–9 months' treatment with nasal continuous positive airway pressure. The questionnaire had a high level of internal consistency as measured by Cronbach's alpha (0.88). Factor analysis of item scores showed that the ESS had only one factor for 104 medical students and for 150 patients with various sleep disorders. The ESS is a simple and reliable method for measuring persistent daytime sleepiness in adults. **Key Words:** Sleepiness—Epworth—Questionnaire—Factor analysis—Reliability.

In the assessment of sleep habits and sleep disorders, a subject's general level of sleepiness during the day is an important characteristic that should be measured routinely. The Multiple Sleep Latency Test (MSLT) is widely used to measure daytime sleepiness, in the sense of the propensity to fall asleep when encouraged to do so in a nonstimulating environment (1). However, the time-consuming nature and expense of the MSLT are such that, all too often, such tests have not been done. There is great need for a simpler alternative.

Some earlier attempts to quantify daytime sleepiness on the basis of the subject's responses to one or more questions succeeded to some extent (2,3). However, each attempt involved different questions for which normative data have seldom been available. The lack of standardization has prevented comparisons being made between different studies. By contrast, other investigations have failed to distinguish different levels of daytime sleepiness on the basis of subjective reports (4–6). One can but speculate about the reasons for this failure, but the methods were not validated and the questions asked or the grading of responses cannot have been appropriate. The Stanford Sleepiness Scale (SSS) measures feelings of sleepiness or, perhaps more accurately, of tiredness at a particular time (7). However, the SSS has been found to be quite unsatisfactory when assessing sleep propensity as measured by the MSLT (8,9). A Sleep–Wake Activity Inventory (SWAI) has recently been developed with 35 items (10). Subjects respond to each item by marking a visual analogue scale at an appropriate position along its 10-cm length. In a factor analysis of item scores there were two factors, one of which was related to daytime sleepiness as measured by the MSLT, the other to "psychological distress". This supports the contention that appropriate questions in a standardized questionnaire can provide clinically useful measurements of a subject's general level of daytime sleepiness.

The Epworth Sleepiness Scale (ESS) has been proposed specifically for that purpose, as a simple method for measuring the general level of daytime sleepiness or sleep propensity in adults (11). The ESS is a brief, self-administered questionnaire that asks the subject to rate on a scale of 0–3 the chances that, over "recent times", he would have dozed in eight specific situations that are commonly met in daily life (0 = would never doze; 3 = high chance of dozing). Thus, the subject is asked to characterize, retrospectively, part of his usual behavior in a variety of situations that are more or less soporific. Subjects are asked to distinguish dozing behavior from feelings of tiredness. The ESS score is the sum of eight item-scores and can range from 0 to 24.

Evidence about the validity of ESS measurements has been reported previously (11). In patients with various sleep disorders ESS scores were correlated sig-

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nificantly with mean sleep latencies measured in MSLTs ($r = -0.514$, $n = 27$, $p < 0.01$).

Scores on the ESS significantly distinguished control subjects from several groups of patients with sleep disorders that are known to be associated with different levels of daytime sleepiness. In patients with obstructive sleep apnea syndrome (OSAS) ESS scores were related to the severity of that disorder as measured by the respiratory disturbance index and by the minimum arterial oxygen saturation measured overnight during apneas. Patients with chronic psychophysiological or idiopathic insomnia tend to have very low ESS scores, indicative of a low sleep propensity.

The present investigation was concerned, first, with the test–retest reliability of ESS scores and, second, with the internal structure and consistency of the questionnaire. Healthy medical students are shown here to have a wide range of ESS scores that did not change significantly over a 5-month period. By contrast, the high ESS scores indicative of excessive daytime sleepiness in patients with OSAS are shown to return to more normal levels, as expected, after a few months' treatment with nasal continuous positive airway pressure (CPAP). The internal consistency of the ESS questionnaire is demonstrated by item analysis in two different groups of subjects—medical students and patients with a variety of sleep disorders. Factor analysis of ESS item scores, performed separately in these same two groups of subjects, shows that the questionnaire measures only one main variable.

These results, in addition to those reported earlier (11), provide evidence that the ESS is a simple and reliable method for measuring the general level of daytime sleepiness in adults.

METHODS

Details of the ESS questionnaire have been published previously (11).

Subjects

All 104 third-year medical students who had assembled for a teaching session at Monash University Medical School, Melbourne, answered the ESS at the same time, without discussion. Their mean age was 20.9 ± 2.8 (SD) years. There were 55 men and 49 women, all of whom were ostensibly healthy although no attempt was made to investigate their general health or sleep habits in detail. The sleep habits of similar medical students at Monash University have been described previously using a sleep questionnaire (12). The mean duration of sleep on week nights was 7.7 ± 0.7 (SD) hours, increasing to 8.4 ± 1.2 (SD) hours on weekends, figures that do not suggest chronic sleep deprivation, at least for the majority of such students. Apart from sleeping about half an hour less per day by waking earlier in the morning in summer than in winter, most aspects of the sleep habits of such students do not change significantly over a year (12).

During the academic year in question these students were not involved with night calls. Their daytime sleepiness was not measured by means other than the ESS. It was assumed that although the daytime sleepiness of a few may increase or decrease over a period of months because of changes in their sleep habits, the sleepiness of most, and of the group as a whole, would remain constant, as others have found using MSLTs (13).

The students in the present investigation first answered the ESS at the beginning of May 1991, 2 months after the start of their academic year. The same class of students was asked, without warning, to answer the ESS a second time at the end of September, 5 months after their first response and 2 months after their winter vacation. Eighty-seven students were identified as having completed the questionnaire on both occasions. The ESS scores of these 87 did not differ significantly from those of the whole group of 104. The 87 paired ESS scores were used to assess the test–retest reliability of the questionnaire.

Over a 9-month period in the Sleep Disorders Unit at Epworth Hospital there were 54 patients with OSAS who were treated successfully with nasal CPAP for at least 3 months and who had answered the ESS before and after treatment. Their mean age was 53.1 ± 11.0 (SD) years with a range from 28 to 78 years. Their respiratory disturbance index (RDI) was defined as the number of apneas and hypopneas causing a fall in arterial oxygen saturation of at least 3% per hour of sleep. Apneas involved cessation of nasal and oral airflow, and hypopneas at least a 50% reduction in airflow for 10 seconds or more. The polysomnographic methods have been described previously (14). The mean RDI before treatment was 27.1 ± 15.5 (SD) (range 4–56). The minimum arterial oxygen saturation recorded for each patient during apneas overnight (before treatment) had a mean of 73 ± 10.5% (SD) (range 50–92%).

The optimum nasal CPAP pressure required to control snoring and apneas and to maintain the SaO_2 at ≥90% was determined for each patient during a second night's polysomnography. That pressure was then fixed in a CPAP pump that was used each night at home. Only patients who reported compliance with this treatment were included here.

The ESS scores of 150 consecutive patients with various sleep disorders have been reported previously (11). Their diagnoses included OSAS, primary snoring, narcolepsy, idiopathic hypersomnia, periodic limb

movement disorder and idiopathic or psychophysiological insomnia. Ages ranged from 18 to 78 years. Half of the patients with OSAS who are treated by nasal CPAP, as above, had their pretreatment ESS scores included in this group, which was finalized before the CPAP treatment group. These 150 patients' scores on each item of the ESS were submitted to factor analysis. A similar analysis was performed separately on the item scores of the 104 medical students.

Statistical methods

All statistical analysis was done on a personal computer using the commercially available package of programs called Statistica® (Statsoft Inc., Tulsa, OK). The tests included Student's t tests for dependent or independent samples, Pearson correlation coefficients and the measurement of skewness and kurtosis of an approximately normal distribution of ESS scores. Differences between the frequency distributions of item scores were tested by χ^2 tests (df = 3: no Yates correction). Statistical significance was accepted at $p < 0.05$ in two-tailed tests. The program "Reliability" was used for item analysis and the calculation of Cronbach's statistic, alpha, which gives a measure of the internal consistency of the questionnaire (15). Factor analysis of ESS item-scores was performed by the program "Factor". Communalities were calculated by multiple regression rather than being estimated from variances. As is usual, only factors with eigenvalues >1.0 were retained. Varimax or other rotation was not appropriate because there was only one main factor in each of the two separate factor analyses.

It is feasible to perform factor analysis on ordinal (ranked) data such as ESS item-scores that can only be 0, 1, 2 or 3 (16). However, such analysis assumes that the variables are normally distributed. In fact, the distribution of item-scores was significantly different from normal for all items except item 5 for the patients and all except items 5, 6 and 8 for the students ($p < 0.001$, tested by separate χ^2 tests, df = 3). It was not feasible to normalize the data separately for each item, and it was accepted that there may be some limitation in the accuracy of calculations as a result. If this were a practical problem it would presumably become apparent in the comparison of results from the two populations in which item-scores were distributed quite differently (see below).

RESULTS

ESS scores of medical students

The mean ESS score for the whole group of 104 medical students was 7.6 ± 3.9 (SD). The range was 0–18 and the modal score was 7 (Fig. 1). The distribution was approximately normal, but with a skewness of 0.32 and kurtosis of −0.53. These scores were slightly higher than those of 30 normal control subjects in a previous investigation in whom the mean ESS score was 5.9 ± 2.2 (SD), the modal score was 6 and the range was 2–10 (2). The difference was statistically significant ($t = 2.37$, df = 132, $p < 0.02$).

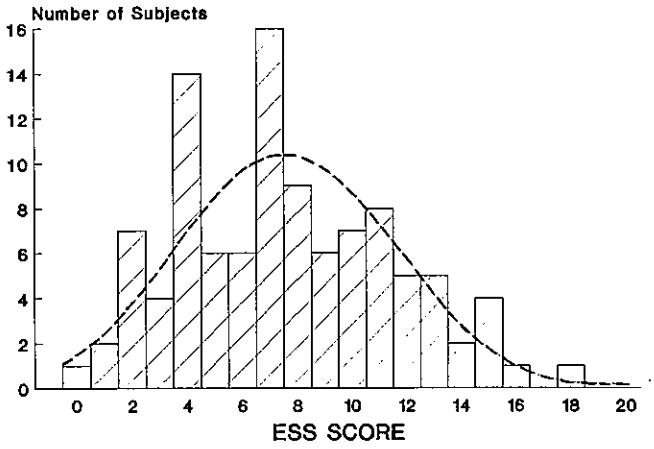

FIG. 1. The distribution of ESS scores from 104 medical students and the normal distribution (dotted).

Test-retest reliability

Comparisons between the ESS responses of 87 students on two separate occasions, 5 months apart, enabled an estimate to be made of the test–retest reliability of the questionnaire. On the first occasion their mean ESS score was 7.4 ± 3.9 (SD) and on the second occasion was 7.6 ± 3.8 (SD). The mean difference between these paired scores was 0.20 ± 2.3 (SD), which was not statistically significant ($t = 0.79$, df = 86, $p = 0.43$). The paired scores differed by no more than 1 in 51.7% of students, by no more than 2 in 81.6% and by no more than 4 in 96.6%. The Pearson correlation coefficient between the 87 paired scores was 0.822 ($p < 0.001$).

By contrast, the 54 patients with OSAS who were treated by nasal CPAP had ESS scores of 14.3 ± 3.6 (SD) with a range of 5–21 before treatment and 7.4 ± 4.1 (SD) with a range of 0–16 after treatment. The mean difference between these paired scores was 7.0 ± 5.2 (SD), which was statistically significant ($t = -9.59$, df = 53, $p < 0.001$).

Thus, ESS scores did not change significantly over several months under circumstances when daytime sleepiness was expected to remain constant but did change significantly as expected, after CPAP treatment for OSAS. These results indicate a reasonably high level of reliability for ESS scores.

TABLE 1. *The ESS item scores of (a) 40 sleepy patients and (b) 104 medical students and the significance of differences between those two groups for each item (χ^2 tests, 3 df)*

Item no.	Situation	Group	No. of subjects with item scores of 0	1	2	3	(p)
1	Sitting and reading	(a)	0	5	10	25	<0.001
		(b)	31	38	29	6	
2	Watching TV	(a)	0	5	7	28	<0.001
		(b)	41	46	12	5	
3	Sitting, inactive in a public place	(a)	1	8	19	12	=0.001
		(b)	40	38	18	8	
4	As a passenger in a car for an hour without a break	(a)	2	7	11	20	<0.001
		(b)	30	37	16	21	
5	Lying down to rest in the afternoon when circumstances permit	(a)	0	0	4	36	<0.001
		(b)	3	19	24	58	
6	Sitting and talking to someone	(a)	8	18	10	4	<0.001
		(b)	99	4	0	1	
7	Sitting quietly after a lunch without alcohol	(a)	0	10	12	18	<0.001
		(b)	38	38	20	8	
8	In a car, while stopped for a few minutes in the traffic	(a)	9	16	6	9	<0.001
		(b)	93	9	1	1	

Item analysis

Cronbach's statistic, alpha, was used as a measure of the internal consistency of the items in the questionnaire. This analysis was performed on item-scores from two separate groups of subjects—150 patients with various sleep disorders and 104 medical students. Alpha was 0.88 for the patients and 0.73 for the students. In neither group did the value of alpha increase after deleting any one of the items from the questionnaire. These results indicate a reasonably high level of consistency between the eight items of the ESS.

The eight situations described in the ESS are not all of the same soporific nature. Indeed, they were chosen on a priori grounds to be different. This is illustrated by comparing the item-scores of subjects in two different groups (Table 1). The first group was of 40 patients, described here as sleepy because they were all those with narcolepsy, idiopathic hypersomnia or severe OSAS (RDI > 30), selected from the group of 150 patients described previously (11). The mean ESS score for these 40 patients was 17.2 ± 3.7 (SD); much higher than for the 104 medical students (7.6 ± 3.9). These two groups also differed significantly in their item-scores for each item (Table 1). In all eight situations the 40 sleepy patients were much more likely than the students to doze. In both groups the situation in item 5 (lying down to rest in the afternoon when circumstances permit) was the most soporific. Every one of the 40 sleepy patients reported at least a moderate chance of dozing off then, of which 90% reported a high chance of doing so. However, more than three-quarters of the students also reported a moderate or a high chance of dozing in that situation. By contrast, the situation in items 6 and 8 (sitting and talking to someone, or in a car, while stopped for a few minutes in the traffic) were the least soporific for both groups. Thirty-five percent of the sleepy patients, compared with only 1% of students, reported at least a moderate chance of dozing when sitting and talking to someone. The other situations in items 1–4 and 7 were intermediate in their soporific nature for both groups.

TABLE 2. *The normalized factor loadings for items of the ESS reported by 150 patients and 104 students*

ESS item no.	Normalized factor loadings: Patients	Students
1	0.73	0.55
2	0.59	0.49
3	0.77	0.62
4	0.68	0.54
5	0.53	0.49
6	0.73	0.25
7	0.76	0.64
8	0.73	0.37

Factor analysis

Factor analysis was performed on the ESS item-scores of the 150 patients and the 104 students separately. This showed that there was only one main factor in each group (Table 2). The eigenvalue was 3.95 for the patients and 2.07 for the students. The normalized factor loadings were relatively high on all items for the patients, and on all but items 6 and 8 for the students. There were so few students dozing in those two situations (Table 1) that there was very little variance associated with those item-scores.

DISCUSSION

The results indicate, first, that the ESS questionnaire is reasonably reliable in the test–retest sense and, second, that it has a high level of internal consistency,

assessed by item analysis and factor analysis within two different groups of subjects.

ESS scores did not change significantly and were highly correlated when tested and retested 5 months later in students whose daytime sleepiness was expected to remain constant. By contrast, the high ESS scores of patients with OSAS, indicative of the excessive daytime sleepiness, which is a clinical feature of that disorder, were reduced to more normal scores, as expected, after 3 or more months' treatment with nasal CPAP.

The internal consistency of the ESS, measured by Cronbach's alpha, was high for the patients with a variety of sleep disorders (0.88) and a little lower, although still satisfactory (0.73), for the students. This consistency was not increased by deleting any of the items from either group. Factor analysis of item-scores showed that the ESS measured only one main variable within each group. Thus, the results support the use of the ESS as a sum-scale for which the derived measurement, the total ESS score, is simply the sum of eight item-scores. Scoring the results is therefore very quick and simple.

The ESS was designed to measure daytime sleepiness over the whole range, from very high to low levels. The items were chosen, therefore, to represent situations of a widely differing soporific nature. The results show that the relative soporific nature of those situations is about the same for very sleepy patients as it is for normal subjects. However, sleepy subjects doze more frequently and in more of those situations than do normal subjects.

Despite the similarities in the results from the patients and the students there were also minor differences. Cronbach's alpha and the normalized factor loadings indicated a level of internal consistency in responses to the ESS that was lower for the students than for patients. The reasons for this are not clear but may involve the nonnormal distribution of some item-scores or the fact that the range of ESS scores was higher among the patients (0–24) than the students (0–18).

The findings that ESS scores of the medical students were higher than those of healthy control subjects reported in a previous investigation (11) highlights some difficulties in establishing normal values. The control group in the previous investigation had only 30 subjects, all of whom were older than the present students. However, it may be that normal adults in their early 20s are sleepier, on average, than normal middle-aged adults, as their ESS scores suggest. There is corroborative evidence for this in MSLTs reported by others (17).

Nasal CPAP treatment for several months reduced the ESS scores of patients with OSAS to the same levels as those of the medical students, but still slightly higher than those of middle-aged, nonsnoring controls from the previous investigation, with whom comparison may be more appropriate. This difference did not quite reach statistical significance ($t = 1.89$, $df = 82$, $p = 0.06$). However, the daytime sleepiness of patients with OSAS, even after successful treatment with nasal CPAP, may remain slightly above normal, as indicated also by the results of Di Phillipo et al. (18) who used MSLTs to measure sleepiness under similar circumstances.

There was a wide range of ESS scores among the students and the patients here, as there had been within each diagnostic group of patients and the controls in the earlier investigation (11). At each end of the clinically normal range, particularly with scores greater than 9, there is a considerable overlap with scores that are abnormal in the sense that they are associated with sleep disorders that are known to affect daytime sleepiness. This overlap between normal and abnormal occurs also with daytime sleepiness measured by MSLTs. Some healthy adults fall asleep in less than 5 minutes, which is considered to represent pathological sleepiness in others (19).

When the ESS was developed it was intended that it should measure only that component of daytime sleepiness which persists from week to week and longer in a given subject, independent of changes with the time of day and from day to day. This is what the MSLT and its variants such as the Maintenance of Wakefulness Test (20) are believed to measure when the mean SL is determined from four to six naps on one day, so overcoming much of the time-of-day effect, and attempts have been made to normalize the preceding night's sleep and to remove the effects of drugs that would otherwise influence the results. This longer-term or persistent component of daytime sleepiness may be influenced by several factors such as a psychophysiological trait involving inherently different levels of sleep propensity in difficult subjects or the presence of one or more chronic sleep disorders such as obstructive sleep apnea, narcolepsy or periodic limb movement disorder. It was believed that this general level of daytime sleepiness would, over a period of time, become incorporated into each subject's way of life, modifying his chances of either dozing or staying awake when in various nonstimulating situations. It was assumed also that most adults would be able to report whether or not they dozed in those situations. The initial results with the ESS suggest that those suppositions were correct (11).

The ESS questionnaire is now shown to be reliable, to be internally consistent and to have only one main dimension in its variance. It is conceptually unique in measuring the whole range of sleep propensities, from very high to very low. It is simple and self-administered, taking a few minutes to complete and a few

seconds to score. With mounting evidence about its validity, it promises to provide a much-needed, simple, standardized method for measuring daytime sleepiness in adults. Nevertheless, the ESS cannot perform all the functions of the MSLT such as measuring changes in sleepiness from day to day or indicating the early onset of REM sleep, which is so important in the diagnosis of narcolepsy.

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The Children's Sleep Habits Questionnaire (CSHQ): Psychometric Properties of A Survey Instrument for School-Aged Children

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Study Objectives: To present psychometric data on a comprehensive, parent-report sleep screening instrument designed for school-aged children, the Children's Sleep Habits Questionnaire (CSHQ). The CSHQ yields both a total score and eight subscale scores, reflecting key sleep domains that encompass the major medical and behavioral sleep disorders in this age group.
Design: Cross-sectional survey.
Setting: Three elementary schools in New England, a pediatric sleep disorders clinic in a children's teaching hospital.
Participants: Parents of 469 school-aged children, aged 4 through 10 years (community sample), and parents of 154 patients diagnosed with sleep disorders in a pediatric sleep clinic completed the CSHQ.
Interventions: N/A
Measurements and Results: The CSHQ showed adequate internal consistency for both the community sample (=0.68) and the clinical sample (=0.78); alpha coefficients for the various subscales of the CSHQ ranged from 0.36 (Parasomnias) to 0.70 (Bedtime Resistance) for the community sample, and from 0.56 (Parasomnias) to 0.93 (Sleep-Disordered Breathing) for the sleep clinic group. Test-retest reliability was acceptable (range 0.62 to 0.79). CSHQ individual items, as well as the subscale and total scores were able to consistently differentiate the community group from the sleep-disordered group, demonstrating validity. A cut-off total CSHQ score of 41 generated by analysis of the Receiver Operator Characteristic Curve (ROC) correctly yielded a sensitivity of 0.80 and specificity of 0.72.
Conclusions: The CSHQ appears to be a useful sleep screening instrument to identify both behaviorally based and medically-based sleep problems in school-aged children.
Key words: Sleep habits; sleep survey; sleep disorders; pediatrics

INTRODUCTION

IN CONTRAST TO WHAT IS KNOWN ABOUT SLEEP HABITS AND SLEEP DISTURBANCES IN INFANTS AND TODDLERS[1-4] AND IN PRESCHOOL-AGED CHILDREN,[5-7] relatively few studies have addressed these issues in latency-aged children.[8-10] Those studies which have examined sleep behavior in middle childhood[11-13] have employed a variety of different interviews, brief questionnaires, and sleep survey instruments, many of which do not have reliability and validity data reported. This has led to considerable difficulties in comparing results across studies. Few of the sleep survey methods used in school-aged children examine both behaviorally-based (Limit Setting Sleep Disorder, Sleep Onset Association Disorder, etc.) and medical (Obstructive Sleep Apnea, Narcolepsy, etc.) sleep disorders, and most have not been formulated according to any of the standardized systems for categorization of clinical sleep disorders such as is contained in the *International Classification of Sleep Disorders (ICSD)* manual.[14] Finally, the definition of a sleep "disturbance" vs. a sleep "behavior" in these studies has been based on often arbitrary thresholds set by the authors and have not included parental definitions of sleep problems in the context of the individual family.

We present preliminary reliability and validity data on a parent-report sleep screening survey specifically

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designed for school-aged (4 years through 10 years) children, the Children's Sleep Habits Questionnaire (CSHQ). The design of the CSHQ is based on common clinical symptom presentations of the most prevalent pediatric *International Classification of Sleep Disorders* diagnoses.[14]

METHODS

Participants

The community sample population consisted of 1099 students aged 4—10 years inclusive, enrolled in three public elementary schools, each of which was comprised of grades kindergarten through fourth grade. The three schools were in a predominantly white, middle-income, English-speaking suburban school district in Southeastern New England and were selected both on the basis of accessibility and as representative of a typical suburban community school system. Participants from each of the three schools were surveyed separately during one of three periods during the school year (Spring, Fall, Winter), in order to minimize potential seasonal differences in sleep habits. Sixty parents also responded voluntarily to a request for test-retest completion of the survey approximately two weeks later.

Of the total of 1099 questionnaire packets mailed, 520 questionnaires were not returned; there were 54 refusals, and 10 subjects moved (response rate = 46.9%). Twenty-six 11-year-old children were eliminated because of the decision to restrict the sample age distribution in order to minimize possible pubertal influences on sleep. Twenty children were excluded from the final sample because of a parent-reported history of having been diagnosed with a psychiatric condition (such as ADHD or depression) that could impact on sleep onset or night wakings and/or were receiving medication with likely effects on sleep, such as psychostimulants, anticonvulsants, or antihistamines. The final sample consisted of 469 children. The mean age of the sample was 7.6 years ± 1.5 years. There were 240 boys (51.2%) and 229 girls (48.8%).[15] Socioeconomic status was determined using the Hollingshead Form Index of Social Status[15] which is based on occupation and education. The mean Hollingshead SES score was 45.5 (SD=11.3).

The clinical population consisted of 154 patients consecutively diagnosed with a behavioral sleep disorder, a parasomnia or sleep-disordered breathing in a pediatric sleep disorders clinic in a children's teaching hospital in Southeastern New England. The data was collected over a four year period due to presentation rates of the specific diagnostic entities in the selected age group. Patients were divided into the three primary diagnostic groups following an extensive evaluation in the sleep clinic. In addition, the diagnosis of Obstructive Sleep Apnea Syndrome (OSAS) in all patients in the sleep disordered breathing group was confirmed by standard one-night in-hospital polysomnographic (PSG) evaluation, including EEG monitoring for sleep staging, using a cut-off respiratory disturbance index of >1 to define OSA.[16] The mean age of the clinical sample was 6.8 years (SD=1.7 yr.). There were 91 boys (59.1%) and 63 girls (40.9%). The mean Hollingshead SES score was 33.0 (SD=13.8).

The characteristics of the three clinical sample diagnostic groups were as follows: Behavioral Sleep Disorders group (n=43) (including Limit Setting Sleep Disorders, Sleep Onset Association Disorder, and Adjustment Sleep Disorder); 22 M, 21 F; mean age = 6.6 years (SD=1.6); Parasomnias group (n=45) (including Sleepwalking, Night Terrors and Confusional Arousals); 25 M, 20 F; mean age = 7.1 years (SD=1.7); and Sleep-Disordered Breathing group (n=66); 44 M, 22 F; mean age = 6.7 years (SD=1.7).

MEASURE

The Children's Sleep Habits Questionnaire (CSHQ). The CSHQ is a retrospective, 45-item parent questionnaire that has been used in a number of studies to examine sleep behavior in young children.[17-19] The CSHQ includes items relating to a number of key sleep domains that encompass the major presenting clinical sleep complaints in this age group: bedtime behavior and sleep onset; sleep duration; anxiety around sleep; behavior occurring during sleep and night wakings; sleep-disordered breathing; parasomnias; and morning waking/daytime sleepiness. Parents are asked to recall sleep behaviors occurring over a "typical" recent week. Items are rated on a three-point scale: "usually" if the sleep behavior occurred five to seven times/week; "sometimes" for two to four times/week; and "rarely" for zero to one time/week. Some items were reversed in order to consistently make a higher score indicative of more disturbed sleep.

Reduction of Sleep Variables. For the purposes of further psychometric evaluation analysis, some of the CSHQ items were eliminated as redundant or ambiguous, and the remaining 35 were conceptually grouped into eight subscales reflecting the following sleep domains: 1) Bedtime Resistance, 2) Sleep Onset Delay, 3) Sleep Duration, 4) Sleep Anxiety, 5) Night Wakings, 6) Parasomnias, 7) Sleep-Disordered Breathing, 8) Daytime Sleepiness. Total Sleep Disturbance score included all items of the eight subscales, but consisted of only 33 items because two of the items on the Bedtime Resistance and Sleep Anxiety subscales were identical. Items contained in each of the subscales are listed in Table 1.

Statistical Analysis. The subscales were assessed for internal consistency using Cronbach's ∝-coefficients. Means and standard deviations of each item in the subscales and the total subscale scores are listed in Table 1. Test-retest reliability was calculated using Pearson's corre-

Table 1—Unadjusted Means, Standard deviations for individual items and subscales, N, F values, Test-retest, and Alpha coefficients for the Subscales of the CSHQ

Subscale Item	Control Sample Mean	SD	N	Clinic Sample Mean	SD	N	F	df	Z	Control Test-retest r[c]	Control Test-retest N	Control α	Control N	Clinic α	Clinic N
1. Bedtime Resistance	7.06	1.89	382	9.43	3.49	128	65.74	3, 506	--	0.676**	56	0.70	441	0.83	142
Goes to bed at same time	1.18	0.53	402	1.30	0.57	130			3.57	0.163	60	---	---	---	---
Falls asleep in own bed	1.21	0.57	400	1.52	0.81	129			5.10	0.335	58	---	---	---	---
Falls asleep in other's bed	1.21	0.52	401	1.48	0.76	129			4.53	0.580	59	---	---	---	---
Needs parent in room to sleep	1.17	0.48	390	1.57	0.82	129			6.67	0.886	58	---	---	---	---
Struggles at bedtime	1.13	0.41	392	1.70	0.86	130			9.50	0.265	58	---	---	---	---
Afraid of sleeping alone	1.19	0.49	388	1.85	0.91	128			9.60	0.597	59	---	---	---	---
2. Sleep Onset Delay Falls asleep in 20 minutes	1.25	0.53	403	1.80	0.88	128			7.58	0.620**	60	---	---	---	
3. Sleep Duration	3.41	0.93	398	4.94	1.98	122	102.68	3, 516		0.400	60	0.69	459	0.80	137
Sleeps too little	1.21	0.42	400	1.78	0.86	127			7.69	0.420	60	---	---	---	---
Sleeps the right amount	1.13	0.43	400	1.73	0.84	124			9.73	0.452	60	---	---	---	---
Sleeps same amount each day	1.07	0.34	398	1.42	0.63	125			8.84	0.062	60	---	---	---	---
4. Sleep Anxiety	4.89	1.45	374	7.09	2.44	119	114.13	3, 489		0.790**	56	0.63	432	0.68	132
Needs parent in room to sleep	1.17	0.48	390	1.57	0.82	129			6.67	0.886	58	---	---	---	---
Afraid of sleeping in the dark	1.38	0.68	387	2.08	0.87	129			9.23	0.585	59	---	---	---	---
Afraid of sleeping alone	1.19	0.49	388	1.85	0.91	128			9.60	0.597	59	---	---	---	---
Trouble sleeping away	1.17	0.44	386	1.56	0.79	120			6.10	0.551	58	---	---	---	---
5. Night Wakings	3.51	0.89	384	5.69	1.60	120	278.99	3, 500		0.634**	56	0.54	437	0.44	135
Moves to other's bed in night	1.17	0.44	392	1.76	0.82	126			9.58	0.584	59	---	---	---	---
Awakes once during night	1.31	0.55	393	2.13	0.76	121			11.45	0.682	58	---	---	---	---
Awakes more than once	1.03	0.16	385	1.86	0.83	126			15.19	0.018	57	---	---	---	---
6. Parasomnias	8.11	1.25	371	11.22	2.53	117	229.21	3, 484		0.618**	57	0.36	425	0.56	132
Wets the bed at night	1.12	0.43	380	1.30	0.61	125			4.48	1.000	58	---	---	---	---
Talks during sleep	1.22	0.44	393	1.72	0.77	127			7.97	0.392	58	---	---	---	---
Restless and moves a lot	1.37	0.58	390	2.26	0.83	127			11.30	0.572	59	---	---	---	---
Sleepwalks	1.04	0.22	384	1.36	0.65	128			8.76	1.000	58	---	---	---	---
Grinds teeth during sleep	1.25	0.52	386	1.50	0.70	124			5.07	0.668	57	---	---	---	---
Awakens screaming, sweating	1.02	0.12	385	1.50	0.77	125			12.03	1.000	58	---	---	---	---
Alarmed by scary dream	1.10	0.30	389	1.53	0.73	126			8.72	0.858	60	---	---	---	---

Table 1 Continued—Unadjusted Means, Standard deviations for individual items and subscales, N, F values, Test-retest, and Alpha coefficients for the Subscales of the CSHQ

Subscale Item	Control Sample Mean	SD	N	Clinic Sample Mean	SD	N	F	df	Z	Control Test-retest r[c]	Control Test-retest N	Control α	Control N	Clinic α	Clinic N
7. Sleep Disordered Breathing [d]	3.24	0.63	382	4.71	2.54	17	35.57	3, 395		0.688**	58	0.51	439	0.93	18[b]
Snores loudly	1.19	0.44	392	1.84	0.92	126			8.28	0.463	58	---	---	---	---
Stops breathing	1.01	0.13	385	1.46	0.83	24			8.14	1.000	58	---	---	---	---
Snorts and gasps	1.05	0.27	384	1.41	0.80	17			3.41	0.816	58	---	---	---	---
8. Daytime Sleepiness	9.64	2.80	381	11.99	3.39	119	1.59[a]	3,149[b]		0.649**	56	0.65	437	0.70	134
Wakes by himself	1.76	0.87	398	1.65	0.78	129			0.69[b]	0.666	59	---	---	---	---
Wakes up in negative mood	1.32	0.50	396	1.74	0.70	128			7.25	0.536	58	---	---	---	---
Others wake child	1.95	0.78	398	2.05	0.80	128			0.94[b]	0.543	60	---	---	---	---
Hard time getting out of bed	1.46	0.64	395	1.63	0.77	128			2.47[b]	0.415	59	---	---	---	---
Takes long time to be alert	1.25	0.49	393	1.55	0.72	127			5.42	0.607	59	---	---	---	---
Seems tired	1.23	0.43	392	1.85	0.69	123			10.76	0.291	60	---	---	---	---
Watching TV	0.19	0.53	400	0.65	0.75	124			8.57	0.451	60	---	---	---	---
Riding in car	0.50	0.81	401	0.77	0.81	124			4.46	0.637	60	---	---	---	---

a All F values for the subscale scores were significantly different at $P<0.001$ except where there are "a" superscripts.
b Z=Mann-Whitney U-test; all are significantly different at $P<0.001$ level except for those with "b" superscript.
c Subscale correlations are Pearson's r values; Item-by-item correlations are Spearman's r values.
d The items on this subscale were changed toward the end of clinic data collection, which accounts for the low number of subjects.

*** = significant at the 0.001 level
** = significant at the 0.01 level
* = significant at the 0.05 level

lation coefficients for the subscale scores and Spearman's correlation coefficients for the item scores. Because the subscale scores were not normally distributed, the scores were log transformed and then a Bonferroni correction was applied to the item analyses in order to correct for multiple comparisons. The control and clinical samples were compared using an analysis of covariance (ANCOVA), covarying age and SES. Individual items were compared on the Mann-Whitney U-test because age and SES varied across the samples, we also divided the samples by age (four to seven years/eight to ten years) and SES (low/high) and calculated Mann-Whitney U-tests on the items for each subdivided sample. Because the statistically significant findings changed little using these subsamples, only the findings for the total sample are presented.

PROCEDURES

Control Sample

A packet containing informed consent forms; a brief survey regarding parents' education and occupation and any significant medical problems and/or medication for the child; and the Children's Sleep Habits Questionnaire, were sent home with the student to be completed by the parent/guardian. A second mailing and reminder were sent to all parents who had not returned the questionnaire within two weeks of the initial mailing. This procedure was approved by the hospital Institutional Review Board as well as the town's school board. We were unable to obtain information on non-responders because of school board request for anonymity.

Clinical Sample

Patients referred to a pediatric sleep disorders clinic received the CSHQ in the mail to complete prior to the clinic appointment. Parents brought the CSHQ to their appointment at the sleep clinic and the responses were reviewed with the parents by staff conducting the clinical interview.

RESULTS

Preliminary Analyses

The community and clinical samples did not differ by gender, $\chi 2$ (1,623) = 2.92, ns. The two groups did differ by age, t (621) = 5.47, $p<0.001$, with the community sample (M = 7.6 years, SD = 1.5 years), significantly older than the clinic sample (M = 6.8 years, SD = 1.7 years). The two groups also differed by SES, t (538) = 10.47, $p<0.001$, with the community sample (M = 45.5, SD = 11.3) having a higher SES score than the clinic sample (M = 33.0, SD = 13.8).

There was no difference between the three clinical subgroups by gender, $\chi 2$ (2,154)) = 2.92, ns; or age, F (2,151) = 1.14, ns. There was a significant difference on Hollingshead SES, F (2,132) = 6.76, $p<0.01$. Post hoc testing with Tukey HSD revealed that the Sleep-Disordered Breathing group (28.6±11.7) had a lower SES score than either the Behavioral (38.5±15.4) or Parasomnia (34.0±13.1) groups.

Internal Consistency

The internal consistency of the entire CSHQ was 0.68 for the community sample and 0.78 for the clinical sample. The alpha coefficients for the subscales are listed in Table 1. Alphas ranged from 0.36 to 0.70 in the community sample. Items were systematically dropped from each of the subscales, with the exception of sleep onset delay which has only one item, to determine if this improved internal consistency. For the community subjects, dropping items 2 (goes to bed same time) and 11(struggles at bedtime) from the Bedtime Resistance subscale only increased the alpha from 0.70 to 0.73. Dropping items from the Sleep Duration subscale lowered the alpha coefficient on the Sleep Duration subscale. Dropping items 12 (afraid of sleeping in dark) and 29 (trouble sleeping away) increased the alpha slightly from 0.63 to 0.65 on the Sleep Anxiety subscale; dropping single items only lowered the score. Dropping item 34 (awoken more than once) from the Night Wakings subscale increased the alpha from 0.54 to 0.55. If items 19 and 25 (bedwetting and teeth grinding) are eliminated from the Parasomnias subscale, the alpha coefficient improves from 0.36 to 0.45. Dropping item 26 (snores loudly) improved the alpha coefficient to 0.58 from 0.51 on the Sleep-Disordered Breathing subscale. Finally, on the Daytime Sleepiness subscale, dropping five items increased the alpha coefficient to 0.76 from 0.65 (wakes negative mood, takes long time to be alert, seems tired, falls asleep watching TV and riding in the car).

For the clinical sample, alpha coefficients ranged from 0.44 to 0.83. Dropping four items from the Bedtime Resistance subscale increased the alpha from 0.83 to 0.86; dropping item 18 (sleeps same amount) increased the Sleep Duration subscale alpha from 0.79 to 0.89; dropping items 12 and 29 raised the Sleep Anxiety subscale from 0.68 to 0.77; dropping item 33 (awakes once) increased the alpha on the Night Wakings subscale from 0.44 to 0.48; dropping item 19 from the Parasomnias subscale increased the alpha from 0.56 to 0.61; and dropping the five items (Above) increased the alpha on the Daytime Sleepiness subscale from 0.70 to 0.80. Dropping items did not change the alpha coefficient on the Sleep-Disordered Breathing subscale.

Test-Retest Reliability

Test-retest reliability was assessed in a volunteer sample of 60 parents from the community sample who responded to a request to complete a second rating of the CSHQ at a

Table 2—Intercorrelation matrix among CSHQ subscales for both control (N = 469) and clinical (N = 154) samples

	1	2	3	4	5	6	7	8
1. BEDTIME RESISTANCE	-	.397	.170	.203	.430	.228	.813	.372
2. SLEEP DURATION	.431	-	.292	.262	.410	.626	.345	.495
3. PARASOMNIA	.212	.133	-	-.160	.396	-.325	.229	-.003
4. SDB	.126	.057	.202	-	-.270	-.001	.153	-.176
5. NIGHT WAKINGS	.337	.239	.340	.016	-	-.057	.460	.266
6. DAYTIME SLEEPINESS	.152	.156	.127	.182	-.029	-	.264	.519
7. SLEEP ANXIETY	.629	.250	.186	.066	.284	.081	-	.316
8. SLEEP ONSET DELAY	.140	.265	.119	.080	.126	.163	.035	-

two-week interval. The Pearson's correlations for three subscales and the Spearman's rank order correlations for each of the items are presented in Table 1. The correlations for the subscales ranged from 0.62 to 0.79, which is an acceptable level. T-tests between the subscales for the two administrations were all non-significant.

Interrelationships Among Subscales

The correlation matrix for each of the subscales of the CSHQ was calculated separately for the community and clinical samples. As can be seen at least in part in Table 2, because the two subscales had two items in common, bedtime resistance and sleep anxiety for both the clinical (r =0.81) and community (r =0.63) samples were the most highly correlated subscales. When these two items were dropped and correlations were recalculated, the coefficients dropped in both the clinical (r = 0.42) and community (r = 0.19) samples. In general, the intercorrelations among the subscales were higher for the clinical sample than the community sample.

Distribution of CSHQ Total Scores

The total scores for the community sample ranged from 6 to 83 (M = 56.2, SD = 8.9). For the clinical sample, the total scores ranged from 7 to 114 (M = 68.4, SD = 13.7). An effect size of 1.06 was calculated, suggesting this difference was clinically significant. Fifty-six percent (56%) of the clinical group had scores one standard deviation above the mean of the community sample. The distributions of the community and clinical samples are displayed in Figure 1. There was also a significant difference between the three clinical groups on the total CSHQ score, $F(2,143) = 5.44$, $p < .01$. Post-hoc tests revealed that the Behavioral Sleep Disorders group (M=74.4, SD = 9.7) had significantly higher scores in the CSHQ than either the Parasomnias (M=66.3), SD = 12.9) or Sleep-Disordered Breathing (M=66.1, SD = 16.4) groups. Effect size calculations indicated that the differences between the Behavioral Sleep Disorders Group and the Parasomnia and Sleep-Disordered Groups were moderate to large (d= .61, d = .70, respectively). There was a larger percentage of children in both the Parasomnia and Sleep-Disorder Breathing groups (60.9%) that had total CSHQ scores below the clinical sample mean of 68.4 compared to the percentage in the Behavioral Sleep Disorders group (27.5%).

Sleep Duration

The only item not listed on Table 1 is the number of hours of sleep per night. The mean (weekday) sleep duration as reported by parents in the community sample was 10.16 hours ± 44.48 minutes (median = 10 hours), with a range from 7 to 14.0 hours. This broad range is somewhat misleading in that there were several outliers at both ends of the distribution. Age and sleep duration were significantly but modestly negatively correlated ($r = -0.17$, $p<0.01$) in the control sample.

In the clinical sample, the mean weekday sleep duration reported by parents was 9.4 hours±3 hours (median = 10 hours) with a range from 3.5 to 14.0 hours. Age and sleep duration were not significantly related in the clinical sample, (r=-0.01, ns). An ANCOVA comparing two groups and covarying age and SES revealed the clinical sample slept significantly less than the community sample $F(3,520)=104.36$, $p<0.0001$.

Validity

Validity was investigated by comparing the clinical sample to the community sample for each of the items and the subscales of the CSHQ. Due to the large number of comparisons, statistical significance was set at $p<0.001$ for the individual items based on a Bonferroni correction. Each of the items was compared across groups using a Mann-Whitney U-test analysis. As can be seen in Table 1, the clinical group had higher (worse) scores than the community group on all items with the exception of item 38 (wakes by self). For 30 out of 33 items, the difference was statistically significant at the $p<0.001$. Only three items on the Daytime Sleepiness subscale were not significant at the $p<0.001$ level.

ANCOVAS, covarying age and SES, indicated that the

clinical sample had significantly higher (worse) scores than the community group (P<0.001) on all subscales (see Table 1). There was also a significant difference between the clinical (M = 68.4, SD = 12.1) and the control (M = 56.2, SD = 8.6) groups on the total score of the CSHQ, after controlling for age and SES, $F(3,531)=136.56$, $p<0.0001$.

Because there were age differences between the clinical and community samples, the Mann-Whitney U-tests were repeated separately for younger (four to seven year olds) and older (eight to ten year olds) samples. The findings were identical in the younger sample. There were four additional nonsignificant differences between the clinical and community samples: goes to bed at the same time, falls asleep in other's bed, falls asleep riding in car, and wets the bed at night. The sample was also divided using a median split on SES and Mann-Whitney U-tests repeated for high and low SES samples. The result were identical to the entire sample.

The three clinical groups were also compared on the subscales of the CSHQ using an analysis of variance. Statistically significant differences were found on all subscales with post hoc testing indicating that differences were in the predicted direction. For the Bedtime Resistance subscale, $F(2,139)=11.11$, $p<0.0001$, the Behavioral group had significantly higher scores than both the Sleep-Disordered and Parasomnias groups. The same pattern was also true for Sleep Duration, $F(2,139)=14.07$, $p<0.0001$, Sleep Anxiety, $F(2,131)=7.25$, $p<0.001$, and Sleep Onset subscales, $F(2,140) = 45.27$, $p<0.0001$. As expected, the Parasomnias group had higher scores on the Parasomnias subscale, $F(2,129) = 7.91$, $p<0.001$, than the other two groups. The Sleep-Disordered Breathing group has significantly higher scores than the other two clinical groups on the Sleep Disordered-Breathing subscale, $F(2,15) = 54.61$, $p<0.0001$. There was a smaller number of subjects for this latter analysis because two of the three items on this subscale were added to the scale towards the end of the study. However, on the snoring item of the subscale which had complete data, the Sleep-Disordered Breathing group had higher scores than the other two clinical groups. On the Night Wakings subscale, the Sleep-Disordered Breathing group had higher scores than both the Behavioral and the Parasomnias group, $F(2,132)=9.67$, $p<0.0001$. On the Daytime Sleepiness subscale, the Sleep-Disordered Breathing had higher (worse) scores than the Parasomnias group.

Sensitivity and specificity were examined using the Receiver Operator Characteristic (ROC) curve.[20] The estimated prevalence score of 40% for sleep problems was based on previous survey data in school-aged children.[10,21-23] These studies have reported a combined prevalence of bedtime struggles and night wakings of between 25% and 35% and an overall prevalence of "sleep" difficulties of 43%.[22] A cut-off score which maximized sensitivity was sought based on the belief that it was more important to avoid false negatives than false positives. The cut-off score with the best diagnostic confidence, as determined by the intersect point of sensitivity and specificity, was 41, which corresponded to the upper 23.2% of the control group's CSHQ total score. Using the cut-off score of 41, sensitivity was calculated at 0.80 and specificity at 0.72. This score correctly identifies 80% of the clinical group.

DISCUSSION

This paper reports the psychometric properties of a sleep screening questionnaire designed primarily for surveying sleep habits and sleep disturbances in community populations. The distribution of scores for the total score and the subscales suggest that these scores have an acceptable range of variability. Based on the criterion of 0.70,[24] the internal consistency coefficients of the entire scale are near (0.68) or above (0.78), acceptable standards for the community and clinical samples, respectively. There was a wider range of internal consistency coefficients among the subscales, with the alpha coefficients of the subscales for the clinical sample higher than those for the community sample. The subscales with the highest internal consistency coefficients in the clinical sample were Sleep Duration, Bedtime Resistance, Daytime Sleepiness and Sleep Anxiety. The stability of the CSHQ was demonstrated by acceptable test-retest reliability coefficients.

The subscale-to-subscale correlations were strongest in the clinical sample and highest between bedtime resistance and sleep anxiety, sleep duration and daytime sleepiness, and daytime sleepiness and sleep onset delay. Similarly, in the community sample, sleep duration and bedtime resistance, and sleep anxiety and bedtime resistance, were most highly correlated. These subscale intercorrelations suggest that the CSHQ taps the relatively distinct sleep behaviors described in the sleep medicine literature. That is, daytime sleepiness, bedtime resistance, sleep anxiety, sleep duration, and even the sleep onset delay subscales are related, although there may be different underlying sleep disorders causing these sleep symptoms. However, the other subscales—parasomnias, sleep disordered breathing, and night wakings—represent other types of sleep problems. It is important to note that in order to achieve subscales with greater discriminatory power between subscales, more items per subscale would be necessary. We feel that the brevity of the CSHQ is a strength, however, and thus we have chosen to keep the scale at its current length.

The validity of the CSHQ was demonstrated by the ability of the items, subscales, and total score of the CSHQ to consistently differentiate non-sleep disordered children from those seeking an evaluation due to a suspected sleep disorder, although there was overlap in the distribution of scores. Given the high prevalence of sleep problems reported in the literature[6,7,10] in children of a similar age distri-

bution to those in the community sample, it is not surprising that we found this overlap. The broad range of CSHQ scores of both the clinical and community samples reflects this overlap; in addition, because the construction of the subscales on the CSHQ is weighted toward items pertaining to difficulties with initiating and maintaining sleep, the total CSHQ scores tend to be scored (higher) for those children in either sample with behavioral sleep disorders. When the clinical group was subdivided into different sleep disorders, the subscales differed across the three clinical groups in the predicted directions, suggesting the scale has utility within clinical populations. Within the community sample, as previously reported, CSHQ total and subscale scores did not differ significantly by gender or SES, but there was a higher frequency of reported bedtime struggles and night wakings in younger compared to older (grades 3 and 4) children.[23]

We elected to group items together conceptually according to presenting symptom constellations rather than to rely on a statistical procedure to derive empirically related subscales. There have been a few factor analytic studies published in the literature to date of similar children's sleep scales. For example, a factor analysis of the Children's Sleep Behavior Scale,[25] using a community sample, resulted in five factors characteristic of parasomnias, bedtime resistance, activity during sleep, sleep anxiety, and positive affect. These factors did not correspond very well to clinical diagnostic categories. The Sleep Disturbance Scale for Children[11] has a factor structure closer to the clinical categories of disorders of initiating and maintaining sleep, sleep disordered breathing, disorders of arousal/nightmares, sleep wake transition disorders, disorders of excessive somnolence, and sleep hyperhydrosis. Although this factor analysis seems more clinically useful than that derived from the CSBS, the categories are rather broad when compared to the conceptually derived subscales presented here.

It should be noted that the CSHQ is designed primarily to be a screening tool. The sleep domains reflected in seven of the CSHQ subscales do parallel symptom constellations associated with *ICSD* (revised) classifications that represent the most common sleep disorders in this age group: Dyssomnias—Intrinsic and Extrinsic Sleep Disorders, including Sleep Onset Association Disorder, Limit Setting Sleep Disorder, Adjustment Sleep Disorder and Inadequate Sleep Hygiene, and Circadian Rhythm Sleep Disorders, including Delayed Sleep-Phase Syndrome, (CSHQ Subscales, Bedtime Resistance, Sleep Onset Delay, Sleep Duration, Sleep Anxiety and Night Wakings); Parasomnias; Obstructive Sleep Apnea (CSHQ Subscale, Sleep-Disordered Breathing). The Daytime Sleepiness Subscale reflects the daytime consequences common to many of these disorders. However, it should be noted that the subscale labels were not intended to be diagnostic or to define the underlying etiology of the presenting sleep symptoms. The items on the Sleep Anxiety Subscale, for example, could be associated with sleep onset delay related to such diagnostic entities as a circadian phase delay, nightmares, or a more generalized anxiety disorder, among others. The utility of the various subscale scores on the CSHQ is in both alerting the clinician of a potential sleep disorder and providing information that would serve as the starting point for a more detailed clinical evaluation. It is also important to note that clinical category systems for diagnosing childhood sleep disorders, including the *ICSD* classification, have yet to be sufficiently validated.

The limitations of this study must be considered in evaluating the suitability of this scale. As in any parent report measure, the role of both parental and retrospective bias in completing the scale must be considered. Despite data suggesting that parental report is reasonably accurate for identifying many types of sleep disturbances when compared to objective data such as actigraphy,[16] parents of older children, in particular, may not always be aware of any difficulties initiating and maintaining sleep. The survey was cross-sectional and asked only for the frequency of sleep behaviors over a week time period, which may have resulted in inaccurate assessments of the prevalence of more episodic sleep phenomena, such as sleepwalking or night terrors. In addition, the short time frame might have resulted in relative over-reporting of very transient sleep disturbances. However, the test-retest reliability results suggest that there was good consistency in the types and severity of sleep disturbances reported over at least a three-week period. The survey also did not significantly address possible irregularities in sleep-wake cycles related to differences in bedtimes on school vs. non-school nights. However, it would be important to specifically ask parents to clarify any discrepancies in using the survey in older children, in particular.

An additional limitation is the use of the 20 minute criteria for prolonged sleep onset latency, which may be an overly liberal definition for school-aged children. The validity of the CSHQ in the clinical setting, therefore, would be enhanced by the addition of a sleep log to some specifically delineate any sleep delay. Also, because of the importance of developmental factors in sleep disturbances, the results of this study may not be generalizeable to children older than the age range of the sample. We chose to limit the upper age range of the sample to 10 years in order to minimize the possible effects of pubertal changes on sleep behavior. More detailed results of the influence of age on sleep behavior from this study have been reported previously.[23] Finally, an additional limitation is that there may also have been children in the community sample who had undiagnosed sleep disorders.

The sensitivity and specificity analysis suggest that the CSHQ may have utility as a screening instrument for sleep

disorders in the clinical practice setting. Data from a recent survey of almost 500 pediatric health care providers[22] suggests that practicing physicians inadequately screen for sleep problems, especially in middle childhood. Thus, a brief parent-report survey such as the CSHQ could provide a relatively simple tool for identifying problematic sleep in the context of a well child encounter, for example. The eight CSHQ subscales roughly correspond to the most common presenting sleep complaints in pediatric practice —bedtime struggles; difficulty falling asleep, inadequate sleep, nighttime fears, sleepwalking/night terrors, night wakings, and difficulty getting up in the morning.[23] Although the CSHQ should not be used to make definitive sleep disorder diagnoses, both the cut-off total score and individual subscale score could be utilized to identify children with sleep disturbances, and highlight sleep domains which warrant further clinical evaluation. With that in mind, the cut-off was set at a level that maximized sensitivity and thus minimized false negatives. It should be noted, however, that because behavioral sleep disorders "drive" the CSHQ, even children with low total scores may have a sleep problem in a specific, "non-behavioral" (e.g., parasomnias) area. Therefore, it is important to examine the individual subscales in all children, regardless of the total score.

In summary, the CSHQ appears to be a useful sleep screening instrument to delineate sleep habits and identify problematic sleep domains in school-aged children. In particular, the CSHQ could be useful in identifying co-morbid sleep disturbances which might complicate the presentation of underlying medical or mental health concerns in children, including chronic illnesses such as juvenile rheumatoid arthritis,[24] and psychiatric diagnoses, such as attention deficit hyperactivity disorder.[25] Additional studies should address the use of the CSHQ in other populations, in order to provide further evidence of its utility in a variety of settings.

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Appendix 3:

Sample Sleep Prescription

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Child Sleep Prescription

Name (First, Last): _____

Age: _____years _____ months

Grade Level: _____

Recommended bedtime: _____ pm

Recommended wake time: _____ am

Goal: _____ hours of sleep

Important Reminders:

DO NOT:

- Use stimulating objects or electronic devices too close to bedtime (e.g., books, toys, video games, televisions, and computers).
- Complete homework right before bedtime, or within the bedroom. If homework is done on or near a child's bed, they may associate that area with working or stress and may not be able to fall asleep easily there.
- Have a heavy meal right before (i.e., within 1 hour of) bedtime.

DO:

- Have a regular and consistent bedtime routine. It should start at approximately the same time every night and be relaxing (e.g. bath, snack, brush teeth, put on pajamas, talk with parents, read).
- Have a light healthy snack to avoid going to bed hungry.

Created by Gruber et al.

Gruber R, Weiss S, Frappier J, et al. Position statement on pediatric sleep. 2013. http://www.canadiansleepsociety.ca/publisher/articleview/frmArticleID/396.

Appendix 4:

Sleep Recommendations for Expecting Parents and Families of Newborns

Created by Gruber et al.

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Sleep Recommendations for Expecting Parents and Families of Newborns

Health care providers can help parents learn how to promote their child's development of healthy sleep by:

1) Providing parents with information regarding normative sleep patterns across infancy and early childhood.

2) Suggesting concrete strategies to promote healthy sleep:

- Help parents help their infants to differentiate between day and night by recommending the following strategies:
  - Establish a bedtime and nap times for the child at approximately the same time each day.
  - Provide a sleeping environment that is darkened at night-time
  - Expose the infant to bright day light for several hours during the day every day
  - Ensure that interactions and playtime are concentrated during the daytime only

- Help parents promote the baby's development of self-soothing skills using the following strategies:
  - Explain to parents that infants need to learn to fall asleep at bedtime, independently, as well as how to fall back asleep after awakening during the night without their assistance.
  - Suggest putting the baby down when they appear to be drowsy, but are not yet asleep.

3) Emphasizing the importance of maternal (parental) sleep:

- Review potential challenges associated with sleep deprivation that can affect parenting quality; for example, negative mood, irritability, impulsivity, forgetfulness, inattentiveness, safety while driving.

- Offer strategies to minimize parental sleep deprivation including:
  - Taking naps whenever possible – for example, when the baby naps
  - Going to bed earlier
  - Prioritizing sleep
  - Practising relaxation techniques
  - Practising good "sleep hygiene"

Created by Gruber et al.

Gruber R, Weiss S, Frappier J, et al. Position statement on pediatric sleep. 2013. http://www.canadiansleepsociety.ca/publisher/articleview/frmArticleID/396.