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POSITION STATEMENT

Position Statement on Pediatric Sleep for Psychiatrists

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This article is an adaptation for psychiatrists of a consensus position statement aimed at paediatricians, family physicians and other health professionals working with youth that has been endorsed by the Canadian Academy of Child and Adolescent Psychiatry, College of Family Physicians of Canada, and Canadian Sleep Society.

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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,

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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, psychologists, psychiatrists, public policy-makers, and educators. 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.

Sleep 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 comorbidities 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 Recommendations 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 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.

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.

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. Acute sleep deprivation refers to no sleep or a reduction in the usual total sleep time, usually lasting for one or two days. In contrast, chronic sleep deprivation exists when the individual routinely sleeps less than required for optimal functioning (Pressman, 2014). 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 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. (For more information see: Knutson, Spiegel, Penev, & Van Cauter, 2012).

1.2 Sleep and Mental Health

According to the 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 (WHO, 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; 1998; 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, 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 post-traumatic 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, attention deficit/hyperactivity disorder (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. Bed-sharing has special significance in many cultures and is widely practiced in many countries. There is conflicting evidence regarding the safety and efficacy of bed sharing during infancy. Although it has been shown to facilitate breastfeeding and provide protection against hypothermia, it has been identified as a risk factor for Sudden Infant Death Syndrome (SIDS). Since bed sharing is strongly associated with both SIDS and unintentional sleep-related death in infants, the American Academy of Pediatrics recommends that infants share a room with their parents without sharing a bed when sleeping (Colson et al., 2013).

In older children, pediatric sleep disorders may become a "family affair." This can happen when parents share their beds in reaction to perceived child problems (i.e., "reactive bed-sharing"). 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 may lead to further difficulties, because the child will develop habitual dependence on this habit and not learn how to fall asleep independently. In addition, when a child cannot sleep well, the parents' sleep is often disturbed (Meltzer & Mindell, 2007), subsequently affecting the parents' abilities to deal with stress, low mood, and sense of fatigue (Meltzer & Mindell, 2007; Mindell & Owens, 2010). This may increase family conflicts 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 six 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 eight

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 (95% confidence limit)	Recommended sleep duration by the NSF*
Total sleep duration:			
(Galland, Taylor, Elder, & Herbison, 2012)	0-2 months	14.6 (9.3-20.0)	12-18
	≈3 months	13.6 (9.4-17.8)	14-15
	≈6 months	12.9 (8.8-17.0)	14-15
	≈9 months	12.6 (9.4-15.8)	14-15
	≈12 months	12.9 (10.1-15.8)	12-14
	1-2 years	12.6 (10.0-15.2)	12-14
	2-3 years	12.0 (9.7-14.2)	12-14
	4-5 years	11.5 (9.1-13.9)	11-13
	≈6 years	9.7 (8.1-11.4)	10-11
	≈7 years	9.4 (7.9-10.9)	10-11
	≈8 years	9.3 (7.8-10.8)	10-11
	≈9 years	9.3 (7.8-10.8)	10-11
	≈10 years	9.1 (7.8-10.7)	10-11
	≈11 years	9.0 (7.3-10.6)	8.5-9.5
	≈12 years	8.9 (7.3-10.6)	8.5-9.5
(Iglowstein et al., 2003)	13 years	9.0 (7.6-10.4)	8.5-9.5
	14 years	8.7 (7.3-10.1)	8.5-9.5
	15 years	8.4 (7-9.8)	8.5-9.5
	16 years	8.1 (6.7-9.5)	8.5-9.5
Number of nightwakings:			
(Galland et al., 2012)	0-2 months	1.7 (0-3.4)	
	3-6 months	0.8 (0-3.0)	
	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 (0.4-6.4)	
	0.75 years	2.8 (0.4-5.2)	
	1 years	2.4 (0.2-4.6)	
	1.5 years	2.0 (0.6-3.4)	
	2 years	1.8 (0.8-2.8)	
	3 years	1.7 (0.9-2.5)	
	4 years	1.5 (0.7-2.3)	

*NSF = National Sleep Foundation. Recommended sleep duration per age group obtained from the National Sleep Foundation (2014).

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 three years of age, 50% of children are still napping in the afternoon; 35% continue to nap until ~ four to five years of age (Iglowstein et al., 2003). By age five, most but not all children stop napping and sleep becomes consolidated into a single nighttime period (Weissbluth, 1995). Nighttime 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 six 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 five). 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 et al., 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 six months of life.

3. Sleep Deprivation in Children and Adolescents

3.1 Prevalence and Definition

Despite very strong evidence for the importance of sleep, sleep duration of children and adolescents has declined over time. Iglowstein et al. (2003) found about one hour decrease in sleep duration in young children between 1974 in 1986. A large systematic review of data from 690,747 children from 20 countries found that the sleep duration of Canadian children and adolescents has consistently and rapidly decreased over the past century (Matricciani, Olds, & Petkov, 2011). This information is consistent with data obtained from the Sleep in America Polls conducted by the National Sleep Foundation (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 . Because significant variability exist in sleep needs from child to child, and across age ranges there is no single "magic number" for the duration of sleep needed by children of a certain age. In addition, some authors have raised the question as to whether children are in fact sleeping less than in the past.

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 seven hours a night (Eaton et al., 2010). Cross-sectional surveys of 3,235 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 five 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; 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 five to seven 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 three to seven 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 psychiatrists 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, and insufficient knowledge.

To prevent sleep deprivation caused by lifestyle factors, that could worsen emotional, behavioral and cognitive functions routine psychiatric assessments must include:

1) Identification of unhealthy sleep patterns;
2) Counseling on the benefits of healthy sleep; and
3) 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 online Appendix 3 for a sample sleep prescription, and online Appendix 4 for sleep recommendations that can be provided to expecting parents and/or parents of newborns). 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 treatment plan in a consistent way.

Psychiatrists 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 emotional and behavioral functioning
   - The importance of their role in optimizing their child's sleep and making a significant positive impact on their child's mental health and treatment 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

For School Age Children:

- 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 two 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

For Older Children/Adolescents:

- The following sleep hygiene categories should be covered: 1) sleep routine; 2) sleep environment; and, 3) eating and drinking habits before bedtime.
- A detailed list of recommendations is provided below. It is important to note that the most important recommendations for teens will involve sleep hygiene aimed at evening de-arousal (e.g., reducing evening light and avoiding late caffeine, physical exercise, and use of electronic media), and plans for morning bright light therapy to advance sleep timing.
- Handouts that can be used by parents, adolescents and clinicians are available in Appendices 5, 6, and 7, respectively.

Sleep Hygiene Recommendations for Adolescents

Sleep Routine and Physical Activity

- *Keep a Regular Sleep Schedule.* Keeping a consistent wake time and bed time will promote better circadian cycling.
- In the morning: Exposure to bright lights in the morning is important. Sunlight helps an individual's biological clock to reset itself each day.
- In the evening: Adolescents should develop a relaxing routine before bedtime; ideas include bathing, listening to music, and reading.
- Exercise in the Late Afternoon or Early Evening

There is good evidence that aerobic exercise can deepen sleep. Deeper sleep may be more restorative and also protect against awakenings related to noise, pain, hot flashes, and other causes.

Timing of the exercise program is also important, as afternoon exercise is generally considered optimal, whereas exercise immediately before bedtime may be counterproductive. Adolescents are recommended to exercise for 20-30 minutes every day, at least three hours before going to bed.

Sleep Environment

- *Make the Sleep Environment Comfortable and Conducive to Sleep.* Subjects should have a comfortable mattress, with the bedroom kept generally cool and dark.
- The bedroom should be reserved for sleeping only. Cell phones, computers, and televisions should be kept out of the bedroom.

Eating and Drinking Habits before Bedtime

- The child should not go to bed feeling hungry, but should not eat a heavy meal immediately before bedtime.
- Avoid Caffeinated Products within Six Hours of Bedtime. Caffeine works as a stimulant and can keep an individual awake.
- Avoid Alcohol in the Evening. Although alcohol use before bedtime can help some people fall asleep more easily, it has been shown to result in more fragmented sleep and increased awakenings since it prevents the brain from getting into deep sleep and REM sleep.
- Avoid Smoking Cigarettes. Nicotine is a stimulant and should be avoided especially in the evening. Chronic cigarette smokers have been found to experience significantly improved sleep when they quit smoking.

3) And by:

- Promoting healthy sleep at regular visits
- Explain that the use of certain substances (e.g., marijuana) may complicate sleep and exacerbate arousal problems.
  Some Adolescents may certain substances before bedtime to help them relax and fall asleep more readily. It is therefore important to explain to them that although these substances may help them reduce sleep latency, they are likely to make it more difficult for them to get up on time in the morning.
- Exercising caution before prescribing pharmacological agents to treat sleep disorders in young children as many medications have not been approved by the US FDA 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 see Gruber, Cassoff, & Knauper, 2011).

4. Pediatric Sleep Disorders

4.1 Prevalence Rates and Definitions

Parent reported sleep disorders occur in 25% to 50% of preschool-aged children (i.e., three to five 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 undertreated, 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 antipsychotics, 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

Table 2 Description and prevalence of common sleep disorders in children

Sleep disorder*	Description	Prevalence estimates (% studied)
Insomnia		
Behavioural insomnias of childhood (BIC): • Limit setting sleep disorder: Bedtime resistance • Sleep-onset association disorder: inappropriate sleep associations • Inadequate Sleep Hygiene	Childhood insomnias are a group of sleep- related or bedtime problems involving difficulties initiating or maintaining sleep, and/or poor sleep quality. Problems may range in severity and include issues such as bedtime resistance, parent-child conflict at bedtime, and/or inappropriate sleep associations or habits. (Reid, Huntley, & Lewin, 2009). The term Inadequate Sleep Hygiene applies when individuals experience insomnia or excessive sleepiness which results from poor sleep habits (or 'sleep hygiene'; Stepanski & Wyatt, 2003). Such habits include: inconsistent wake times or bedtimes, frequent periods of extended amounts of time spent in bed, the consistent use of products containing sleep- disrupting agents before bedtime (such as caffeine), and engaging in stimulating or emotion-provoking activities close to bedtime (e.g., exercise, playing videogames, watching television), among others. To qualify for this diagnosis, a child must display only 1 habit that constitutes inadequate sleep hygiene (American Academy of Sleep Medicine [AASM], 2005).	Reports have indicated that approximately 9.4% of American adolescents (13-16 years; Johnson, Roth, Schultz, & Breslau, 2006) and 5.8% of European adolescents experience insomnia (Ohayon, Roberts, Zulley, Smirne, & Priest, 2000). 15% to 22% of children have frequent problems with sleep onset or maintenance (Petit, Touchette, Tremblay, Boivin, & Montplaisir, 2007). 20% to 30% of infants, toddlers and preschoolers display bedtime problems and frequent night waking – two key symptoms of BIC (Mindell, 1999; Mindell et al., 2006; Moore, 2012; Morgenthaler, et al., 2006).
Sleep-related breathing disorders		Approximately 2% to 4% of children and adolescents are affected by Sleep Disordered Breathing (Burg & Friedman, 2010).
Central apnea syndromes:	These disorders are characterised by a central nervous system dysfunction causing diminished or absent respiratory effort, occurring in an intermittent or a cyclical fashion. These disorders may be associated with anatomic or genetic abnormalities, or environmental causes (AASM, 2005).	Central sleep apnea is relatively rare in children.
Primary sleep apnea of infancy:		
Apnea of prematurity:	A disorder of respiratory control in infancy that causes respiratory pauses of 20 seconds or longer (AASM, 2005). It is most often diagnosed in preterm infants (apnea of prematurity), but can also be seen in infants with a predisposition (i.e., apnea of infancy; AASM, 2005). Apnea of prematurity is a developmental disorder which occurs in extremely preterm infants in a Neonatal Intensive Care Unit (NICU). It is often treated with caffeine or Continuous Positive Airway Pressure (CPAP). Apnea in older infants is not very well understood, but it is distinct from apneic pauses in extremely premature infants.	Apnea of prematurity is very frequent in extremely pre-term infants. Apnea in older infants is much less frequent.
Obstructive Sleep Apnea (OSA):	Obstructive sleep apnea occurs when there is either a full or partial blockage of the airway leading to inadequate ventilation and increased breathing effort. Obstructive sleep apnea in children is diagnosed when 1 or more obstructive event of at least 2 respiratory cycles of duration per hour. This results in snoring and sleep disruption. Although the features of pediatric OSA are similar to those seen in adults with the disorder, cortical arousals may not always occur. This may be due to a higher arousal threshold in children (AASM, 2005). Childhood OSA may also lead to impaired daytime functioning, including behavioural problems and excessive daytime sleepiness (Burg & Friedman, 2010).	It is estimated that 1% to 4% of otherwise healthy children are affected by OSA, with a higher prevalence in children with underlying medical issues such as obesity or genetic disorders (Ofer & Marcus, 2012). A recent systematic review (Marcus et al., 2012) has found a prevalence ranging from 0% to 5.7% in the general pediatric population.

continued

Table 2 (continued) Description and prevalence of common sleep disorders in children

Sleep disorder*	Description	Prevalence estimates (% studied)
Sleep-related hypoventilation/ hypoxemic syndromes:	Disorders associated with hypoventilation or hypoxemia during sleep as a result of a decreased alveolar hypoventilation, medical conditions of impaired lung function or chest wall mechanics, or of a failure of the central automatic control of breathing in infants (AASM, 2005). Congenital central hypoventilation syndrome is probably the most important dimension, and is caused by abnormalities in the PHOX2B gene.	
Obesity Hypoventilation Syndrome:	A syndrome referring to the combination of obesity and chronic hypercapnia (i.e., increased levels of carbon dioxide in the blood) that is not directly caused by an underlying cardio-respiratory condition, and is often associated with some form of sleep disordered breathing (i.e., apnea/hypopnea, sustained periods of hypoventilation; Berger, Goldring, Rapoport, 2009).	Though relatively uncommon in the general pediatric population, this disorder is increasingly identified among obese children and teenagers.
Hypersomnias	Hypersomnias are sleep disorders with excessive daytime sleepiness as a primary symptom when it is not associated with disturbed nocturnal sleep or misaligned circadian rhythms (AASM, 2005). Prader Willi syndrome (PWs) is the most frequent genetically-determined cause of hypersomnia in both children and adults (Parkes, 1999).	
Narcolepsy	Narcolepsy is a chronic neurologic disorder in which portions of rapid eye movement (REM) sleep intrude into wakefulness. It is characterized by recurrent "attacks" of uncontrollable daytime sleepiness, vivid dreams at sleep onset, sleep paralysis and/ or cataplexy (or the sudden loss of muscle tone in response to emotional triggers) (Kotagal, 2008). Narcolepsy without cataplexy is diagnosed in children when there is no cataplexy present, however there is sleep paralysis, hypnagogic hallucinations, and supportive evidence of the disorder in the form of a positive multiple sleep latency test (with a mean sleep latency of ≤8 minutes and 2 or more sleep-onset REM periods; AASM, 2005). Children with narcolepsy often experience extreme sleepiness, including falling asleep during meal times or social activities, and demonstrate poor academic performance (Nevsimalova, 2009).	There is no clear consensus regarding the prevalence of narcolepsy (with or without cataplexy) in children; however, most studies indicate a prevalence, across all age groups, of 0.025% to 0.05% (or 25 to 50 people in 100 000; Viorritto, Kureshi & Owens, 2012; Wise et al., 2007).
Circadian Rhythm Sleep Disorders (CRSD)	CRSDs are characterized by difficulty or failure to adjust the sleep-wake pattern to a socially appropriate schedule, leading to complaints of insomnia, and/or excessive daytime fatigue, as well as impairments in daytime functioning and quality of life (Gruber & Sheshko, 2008).	
Delayed Sleep Phase Syndrome (DSPS)	With DSPD, the sleep period is delayed in relation to the desired sleep and wake times. Individuals experience a chronic (or repeated) inability to fall asleep and rise according to a socially acceptable time schedule. Once sleep is achieved, however, the quality and quantity of sleep is normal if the individual is not awakened before his/her desired time. A typical clinical scenario would be an adolescent who goes to bed after midnight but must awaken at 6:30 am to be ready for school.	DSPS is the second most common sleep disorder among adolescents (with inadequate sleep hygiene being the most common), affecting an estimated 5% to 10% of the adolescent population (Moore, 2012).

continued

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

Table 2 (continued) Description and prevalence of common sleep disorders in children

Sleep disorder*	Description	Prevalence estimates (% studied)
Parasomnias	Parasomnias are defined as any undesired behaviours, emotions, experiences, abnormal movements or autonomic nervous system functioning occurring during sleep or the transition to or out of sleep. Parasomnias may be associated with transitions into sleep, partial arousals during sleep, or atypical arousals from sleep. The frequency of occurrence can range from a single night or span over many nights. Common parasomnias of childhood include: sleep walking, nightmares, sleep terrors (autonomic system activation and behavioral manifestation of intense fear such as a cry or scream), confusional arousals (episodes in which the child remains in a confused state upon awakening; Bansal & Sheldon, 2008) and nocturnal enuresis (involuntary loss of urine during sleep (Bansal & Sheldon, 2008).	The prevalence of parasomnias in childhood is variable. Research indicates that approximately 15% of children (aged 4-12 years) have one episode of sleep-walking, while 1% to 6% have frequent episodes (one to four per week; Bloomfield & Shatkin, 2009). In addition, about 17% of children (aged 3-13 years) experience occasional episodes of confusional arousals, while approximately 6% of children (aged 18 months to 10 years) experience sleep terrors (Bloomfield & Shatkin, 2009). Nightmares, bad dreams, and night-time fears are very common among children, with an estimated 75% of the pediatric population experiencing a bad dream or nightmare on at least one occasion (Moore, 2012). Nocturnal enuresis is estimated to affect 15% to 25% of children aged 5 yrs (Bansal & Sheldon, 2008).
Sleep-related movement disorders	These sleep disorders are characterized by simple stereotyped movements that disturb sleep.	
Periodic Limb Movement Disorder (PLMD):	PLMD consists of repetitive, highly stereotyped limb movements that occur during sleep. These movements can be associated with recurrent partial arousals, or full awakenings, leading to fragmented sleep. Children with PLMD report greater difficulties falling asleep, maintaining sleep, a higher frequency of nocturnal awakening, and increased difficulty falling back asleep after waking up during the night (Gingras & Gaultney, 2008).	Pediatric studies indicate a 5% to 25% prevalence rate of PLMD in children referred for sleep evaluation (Kirk & Bohn, 2004; Chervin, Archbold, 2001; Crabtree et al., 2003; Martinez, Guilleminault, 2004). In one study, 8.4% of referred children, and 11.9% of children from a community sample were identified with the disorder (Crabtree, Ivanenko, & O'Brien, et al., 2003).
Sleep related rhythmic movement disorder	The disorder consists of repetitive rhythmic motor behaviors occurring during a drowsy or light sleep state, and results in large movements of the head, body, or limbs. In many children, this is benign and is a form of self soothing. It can result in head and limb injuries caused by violent movements (AASM, 2005).	
Restless Leg Syndrome (RLS):	RLS is a neurologic, sensorimotor disorder characterized by: 1) a strong, irresistible urge to move the legs usually accompanied, or caused by, discomfort in the legs; 2) the urge to move or else uncomfortable sensations will worsen; 3) relief, or partial relief of unpleasant sensations, through movement (e.g., walking stretching); 4) undesirable sensations are worse, or only occur, throughout the night time compared to during the daytime. For a diagnosis of RLS, the ICSD-2 stipulates that the condition must not be more adequately explained by the presence of another sleep disorder, medical or neurological disorder, mental disorder, as well as any medication or substance use (AASM, 2005). Until recently, few studies examined RLS in children, and diagnostic criteria were only specified for adults (Bloomfield, & Shatkin, 2009). However, specific guidelines for children aged 2-12 years have been developed by Allen and colleagues (2003). For a definite diagnosis of RLS the child must meet all four of the adult criteria, and describe leg discomfort in their own words (i.e., using words such as "owies", "booboos", or "spiders), or the child must present with 2 or 3 supportive criteria, including: sleep disturbance for age, a biologic parent or sibling with RLS, and/or a PSG- documented Periodic Limb Movement Index of 5 or more per hour of sleep (Allen et al., 2003).	Approximately 1.9% to 2% of children and adolescents in the U.S. and U.K have pediatric RLS (Picchietti et al., 2007; Picchietti & Picchietti, 2010; Sullivan, 2012), with between 25% to 50% reporting moderate to severe symptoms (Sullivan, 2012)

continued

Table 2 (continued) Description and prevalence of common sleep disorders in children

Sleep disorder*	Description	Prevalence estimates (% studied)
Isolated symptoms and normal variants	*Sleep Length:* • Excessive sleepiness: 10h or more of sleep a day • Short sleep: 5h or less of sleep a day for adults and in children it is the equivalence of 3h or less than the norm for their age group. *Snoring (also referred to as primary snoring or simple snoring):* A respiratory noise that is disturbing for the patient, or another individual, and that is not associated with insomnia, repeated arousals, recurrent hypoxemia, or hypercapnea *Sleep talking:* vocalizations during sleep *Sleep starts:* Sudden and brief muscle contractions mostly associated with a sensation of falling or a sensory flash that usually occurs when sleep starts *Benign sleep myoclonus of infancy:* myoclonic jerks occurring during sleep in infants from birth till 6 months of age; the events are not epileptic in origin. (AASM, 2005)	The prevalence of parasomnias in childhood is variable. Research indicates that approximately 15% of children (aged 4-12 years) have one episode of sleep-walking, while 1% to 6% have frequent episodes (one to four per week; Bloomfield & Shatkin, 2009). In addition, about 17% of children (aged 3-13 years) experience occasional episodes of confusional arousals, while approximately 6% of children (aged 18 months to 10 years) experience sleep terrors (Bloomfield & Shatkin, 2009). Nightmares, bad dreams, and night-time fears are very common among children, with an estimated 75% of the pediatric population experiencing a bad dream or nightmare on at least one occasion (Moore, 2012). Nocturnal enuresis is estimated to affect 15% to 25% of children aged 5 yrs (Bansal & Sheldon, 2008).
Other sleep disorders	• Other physiological (organic) sleep disorders • Sleep disorders that are not due to substance or a known physiological condition • Environmental sleep disorders (AASM, 2005)	

* The specified categories are organized according to the nosology of sleep disorders presented in the 2nd edition of the International Classification of Sleep Disorders (ICSD-2; 2005).

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 five 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, Panton, Ironside, MacPherson, & Williams, 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 online 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 two 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 online 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, & Quan, 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 (2013). 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.

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 see: Aurora et al., 2011; 2012; Kotagal et al., 2012; Wise et al., 2011).

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).

Infant and Toddlers. 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 six 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: Kuhn & Roane, 2012; Mindell et al., 2006; Vriend & Corkum, 2011).

Behavioral Interventions for Older School Age Children and Adolescents. Sleep Hygiene Education is intended to provide information about lifestyle (diet, exercise, substance use) and environmental factors (light, noise, temperature) that may interfere with or promote sleep. Inadequate sleep hygiene refers to daily living activities that are inconsistent with the maintenance of good quality sleep and daytime alertness (Stepanski & Wyatt, 2003). Since the most common sleep issue experienced by adolescents is inadequate sleep, it is important to define and assess sleep hygiene practices in adolescents and use behavioral interventions to improve these practices.

These behavioral interventions include specific recommendations to improve sleep scheduling, sleep habits, sleep environment, and physiological practices conducive to sleep.

Two factors associated with sleep hygiene in adolescents are their use of caffeine and the presence in their bedrooms and use of technology (e.g. computer, television, cell phone, videogames), which have been clearly associated with decreases in sleep quantity and quality in children (National Sleep Foundation, 2004).

Assessment of Sleep Hygiene Practices of Adolescents. A detailed description of a child's bedtime routine, caffeine or alcohol consumption, drug use, exercise habits, and sleep environment should be used to determine the contribution of factors related to sleep hygiene to the presenting symptoms.

Sleep hygiene practices may be assessed in a face to face interview, with the physician reviewing the sleep hygiene of an adolescent to identify the targets for intervention. To save time, the physician can use a validated paper and pencil instrument to assess the extent to which sleep hygiene issues are operational, and which issues in particular represent problems for an adolescent.

The Adolescent Sleep Hygiene Scale (ASHS) is a 28-item self-reported questionnaire that assesses sleep-facilitating and -inhibiting practices in adolescents along nine domains: physiological, cognitive, emotional, sleep environment, daytime sleep, substance use, bedtime routine, sleep stability, and bed/bedroom sharing (Storfer-Isser, LeBourgeois, Harsh, Tompsett, & Redline, 2013). Participants report their sleep habits on a 6-point scale with an overall sleep hygiene score (internal consistency, Cronbach's α=0.80) obtained from the mean of the domain scores, where higher scores indicate better sleep hygiene (Storfer-Isser et al., 2013). The ASHS shows concurrent validity associations with the Adolescent Sleep Wake Scale (ASWS) (LeBourgeois, Giannotti, Cortesi, Wolfson, & Harsh, 2005) and was rated as "approaching well-established" in a recent review (Lewandowski, Toliver-Sokol, & Palermo, 2011). Furthermore, the ASHS is the only sleep hygiene measure appropriate for use with children older than 12 years of age (Lewandowski et al., 2011).

Intervention. The behavioral treatment of inadequate sleep hygiene is of particular importance in adolescence, given the high prevalence of poor sleep hygiene in this age group and its negative impact on sleep. An intervention would typically begin by educating parents and children/adolescents about appropriate sleep habits and the impact of sleep quality and quantity on all aspects of child and family functioning. Treatment may involve problem solving with the family to develop a consistent sleep schedule and bedtime routine, as well as reinforcing sleep promoting practices and conditions. Unhealthy habits such as falling asleep on the couch or with the television on may have to be gradually eliminated as part of the treatment plan.

Stimulus Control (SC). The goal of the intervention is to increase the frequency of quickly falling asleep by strengthening the cues for sleep as well as decreasing the cues for behaviors that are incompatible with sleep. SC (Bootzin, 1972; Bootzin & Epstein, 2000; Bootzin, Epstein, & Wood, 1991; Bootzin & Nicassio, 1978) is based on a conditioning analysis, in which falling asleep is conceptualized as an instrumental act designed to produce reinforcement (i.e., sleep). Thus, stimuli associated with sleep become discriminative stimuli for the occurrence of reinforcement. If bed and bedtime become cues for behaviors that are incompatible with falling asleep, such as watching television, reading, earing or worrying, difficulty in falling asleep will be due to inadequate stimulus control – i.e., strong discriminative stimuli for sleep may not have been established, and/or discriminative stimuli for activities incompatible with sleep may be present (Bootzin & Nicassio, 1978). SC consists of a set of instructions designed to: (1) establish a consistent sleep/wake rhythm; (2) strengthen the bed and bedroom as cues for sleep; and, (3) weaken them as cues for activities that might interfere or are incompatible with sleep.

The following instructions could be used when using SC (Bootzin 1972; Bootzin & Epstein, 2000; Bootzin et al., 1991; Bootzin & Nicassio, 1978):

1) Lie down to go to sleep only when you are sleepy.
2) Do not use your bed for anything except sleep; that is, do not read, watch television, eat, or worry in bed. Sexual activity is the only exception to this rule. On such occasions, the instructions are to be followed afterward when you intend to go to sleep.
3) If you find yourself unable to fall asleep, get up and go into another room. Stay up as long as you wish and then return to the bedroom to sleep. Although we do not want you to watch the clock, we want you to get out of bed if you do not fall asleep immediately. Remember, the goal is to associate your bed with falling asleep quickly! If you are in bed more than about ten minutes without falling asleep and have not gotten up, you are not following this instruction.
4) If you still cannot fall asleep, repeat step (3). Do this as often as is necessary throughout the night.
5) Set your alarm and get up at the same time every morning, irrespective of how much sleep you got during the night. This will help your body acquire a consistent sleep rhythm.
6) Do not nap during the day.

(For more information see: Bootzin & Epstein, 2000; Bootzin et al., 1991).

Cognitive Therapy. A few studies in older children (Paine & Gradisar, 2011) or adolescents (Gradisar et al., 2011) have reported using cognitive behavioral therapy (CBT) to treat behavioral insomnia. This intervention is directed at changing maladaptive attitudes and beliefs by using cognitive restructuring aimed at identifying and changing dysfunctional cognitions. These include, for example, misconceptions about the causes of insomnia, misattributions or amplifications of the consequences of poor sleep, and unrealistic sleep expectations.

Effectiveness of Behavioral Intervention for Older Children and Adolescents. Although few studies have examined this

approach in these groups, the results are promising (Paine & Gradisar, 2011). In addition, behavioral sleep interventions, utilizing combinations of sleep hygiene, cognitive therapy and stimulus control strategies, have been used to improve sleep in adolescents (Brown, Buboltz, & Soper, 2006; Taheri, 2006). These sleep hygiene studies have reported positive outcomes in various aspects of sleep quality and even drug use (Bootzin & Stevens, 2005).

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 (Weiss, Wasdell, Bomben, Rea, & Freeman, 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. In the treatment of depression, antidepressants may improve sleep by improving mood and other symptoms of depression. Antidepressants with sedation as a side effect have been administered at bedtime to minimize this side effect and improve sleep or can be used at low dose on an acute basis to treat insomnia (trazadone, mirtazapine, amitryptiline). 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., 2012b). 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 two 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 see: Friedman, Perkins, McNair, & Mitchell, 2013; Marcus et al., 2012a).

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 see: Wise, Arand, Auger, Brooks, & Watson, 2007).

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 hours 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., 1999b; 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 two hours prior to the estimated DLMO (dim light melatonin onset), or four 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 see: Bloomfield & Shatkin, 2009; Kotagal, 2012).

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, six 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; 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 see: Picchietti & Picchietti, 2010; Allen et al., 2003; Chesson et al., 1999a).

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 psychiatric 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 psychiatrists:

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.)
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. Begin with an empathetic but detailed inquiry into sleep patterns and the degree to which these individuals see themselves as having a problem with sleep. This should be followed by an appropriate differential diagnosis and subsequent specific treatment of the underlying condition(s), as well as the development of workable strategies to alleviate sleep problems.

   Implement healthy sleep habits as a first option even if the sleep complaints are thought to be medication-related.

   If, after some weeks (during which the negative effect of medication on sleep may spontaneously decrease), sleep hygiene is not effective, further options may include trying alternative dosages, dose regimens, formulations, or medications, or adding a sleep promoting agent (e.g., melatonin).

4) Offer intervention and counseling;

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

- 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 psychiatrists, psychologists and nurses.
- The funding of quality research on the promotion of healthy sleep and its relationship with emotional well-being.
- The integration of healthy sleeping habits and counseling in preventive clinical practices (les pratiques cliniques preventives; PCP).

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References

Acebo, C., Sadeh, A., Seifer, R., Tzischinsky, O., Hafer, A., & Carskadon, M. A. (2005). Sleep/wake patterns derived from activity monitoring and maternal report for healthy 1- to 5-year-old children. *Sleep, 28*, 1568-1577.

Alexandru, G., Michikazu, S., Shimako, H., Xiaoli, C., Hitomi, K., Takashi, Y.,...Sadanobu, K. (2006). Epidemiological aspects of self-reported sleep onset latency in Japanese junior high school children. *Journal of Sleep Research, 15*(3), 266-275. doi: 10.1111/j.1365-2869.2006.00530.x

AlGhanim, N., Comondore, V. R., Fleetham, J., Marra, C. A., & Ayas, N. T. (2008). The economic impact of obstructive sleep apnea. *Lung, 186*(1), 7-12. doi: 10.1007/s00408-007-9055-5

Alhola, P., & Polo-Kantola, P. (2007). Sleep deprivation: Impact on cognitive performance. *Journal of Neuropsychiatric Disease and Treatment, 3*(5), 553-567.

Allada, R., White, N. E., So, W. V., Hall, J. C., & Rosbash, M. (1998). A mutant Drosophila homolog of mammalian Clock disrupts circadian rhythms and transcription of period and timeless. *Cell, 93*(5), 791-804.

Allen, R. P., & Earley, C. J. (2007). The role of iron in restless legs syndrome. *Movement Disorders, 22 Suppl 18*, S440-448. doi: 10.1002/mds.21607

Allen, R. P., Picchiett, 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. *Sleep Medicine, 4*(2), 101-119.

American Academy of Sleep Medicine. (2005). *International classification of sleep disorders: Diagnostic and coding manual* (2nd ed.). Westchester, IL: American Academy of Sleep Medicine.

Anders, T. F., Emde, R. N., & Parmelee, A. H. (1971). *A manual of standardized terminology, techniques and criteria for scoring of states of sleep and wakefulness in newborn infants*. Los Angeles, CA: UCLA Brain Information Service, NINDS Neurological Information Network.

Andrade, M. M. M., Benedito-Silva, A. A., Domenice, S., Arnhold, I. J. P., & Menna-Barreto, L. (1993). Sleep characteristics of adolescents: A longitudinal study. *Journal of Adolescent Health, 14*, 401-406.

Arendt, J. (2006). Melatonin and human rhythms. *Chronobiology International, 23*(1-2), 21-37. doi: 10.1080/07420520500464361

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. doi: 10.5665/sleep.2190

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

Babu, K., Church, R., & Lewander, W. (2008). Energy drinks: The new eye-opener for adolescents. *Clinical Pediatric Emergency Medicine, 9*, 35-42.

Bansal, R., & Sheldon, S. H. (2008). Parasomnias in childhood. In A. Ivanenko (Ed.), *Sleep and psychiatric disorders in children and adolescents* (first edition, pp. 305-313). New York, NY: Informa Healthcare USA, Inc.

Benca, R. M., Obermeyer, W. H., Thisted, R. A., & Gillin, J. C. (1992). Sleep and psychiatric disorders. A meta-analysis. *Archives in General Psychiatry, 49*(8), 651-670.

Berger, K. I., Goldring, R. M., & Rapoport, D. M. (2009). Obesity hypoventilation syndrome. *Seminars in Respiratory and Critical Care Medicine, 30*(3), 253-261.

Blau, J., & Young, M. W. (1999). Cycling vrille expression is required for a functional Drosophila clock. *Cell, 99*(6), 661-671.

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.

Bootzin, R. R. (1972). A stimulus control treatment for insomnia. *American Psychological Association Proceedings*, 395-396.

Bootzin, R. R., & Epstein, D. R. (2000). Stimulus control instructions. In K. L. Lichstein & C. M. Morin (Eds.), *Treatment of late-life insomnia* (pp. 167-184). Thousand Oaks, CA: Sage.

Bootzin, R. R., Epstein D. R., & Wood, J. M. (1991). Stimulus control instruction. In P. Hauri (Ed.), *Case studies in insomnia* (pp. 19-28). New York, NY: Plenum.

Bootzin, R. R., & Nicassio, P. (1978). Behavioral treatments for insomnia. In M. Hersen, R. Eisler, & P. Miller (Eds.), *Progress in behavior modification* (Vol. 6, pp. 1-45). New York, NY: Academic Press.

Bootzin, R. R., & Stevens, S. J. (2005). Adolescents, substance abuse, and the treatment of insomnia and daytime sleepiness. *Clinical Psychology Review, 25(5)*, 629-644.

Borbely, A. A. (1982). A two process model of sleep regulation. *Human Neurobiology, 1*, 195-204.

Brown, F. C., Buboltz Jr., W. C., & Soper, B. (2006). Development and evaluation of the sleep treatment and education program for students (STEPS). *Journal of American College Health, 54(4)*, 231-237. doi: 10.3200/JACH.54.4.231-237

Brown, L. W., Maistros, P., & Guilleminault, C. (1995). Sleep in children with neurological problems. In R. Ferber & M. Kryger (Eds.), *Principles and Practice of Sleep Medicine in the Child* (pp. 135). Philadelphia, PA: WB Saunders.

Brouillette, R. T., Manoukian, J. J., Ducharme, F. M., Oudjhane, K., Earle, L. G., Ladan, S., & Morielli, A. (2001). Efficacy of fluticasone nasal spray for pediatric obstructive sleep apnea. *Journal of Pediatrics, 138*(6), 838-844.

Buckhalt, J. A., El-Sheikh, M., Keller, P. S., & Kelly, R. J. (2009). Concurrent and longitudinal relations between children's sleep and cognitive functioning: The moderating role of parent education. *Child Development, 80*(3), 875-892. doi: CDEV1303 [pii] 10.1111/j.1467-8624.2009.01303.x

Burg, C. J., & Friedman, N. R. (2010). Diagnosis and treatment of sleep apnea in adolescents. *Adolescent Medicine: State of the Art Reviews, 21*(3), 457-479.

Buscemi, N., Vandermeer, B., Hooton, N., Pandya, R., Tjosvold, L., Hartling, L.,...Baker, G. (2006). Efficacy and safety of exogenous melatonin for secondary sleep disorders and sleep disorders accompanying sleep restriction: Meta-analysis. *BMJ, 332*(7538), 385-393. doi: 10.1136/bmj.38731.532766.F6

Cajochen, C., Frey, S., Anders, D., Spati, J., Bues, M., Pross, A.,... Stefani, O. (2011). Evening exposure to a light-emitting diodes (LED)-backlit computer screen affects circadian physiology and cognitive performance. *Journal of Applied Physiology, 110*(5), 1432-1438. doi: 10.1152/japplphysiol.00165.2011

Calamaro, C. J., Mason, T. B., & Ratcliffe, S. J. (2009). Adolescents living the 24/7 lifestyle: Effects of caffeine and technology on sleep duration and daytime functioning. *Pediatrics, 123*(6), e1005-1010. doi: 10.1542/peds.2008-3641

Campbell, S. S., Dawson, D., & Anderson, M. W. (1993). Alleviation of sleep maintenance insomnia with timed exposure to bright light. *Journal of the American Geriatric Society, 41*(8), 829-836.

Canadian Sleep Society (2013). http://www.canadiansleepsociety.ca/publisher/articleview/?PHXSESSID=1e2abcd3ea241f7e4c1b285983ae1db1&/1/frmArticleID/370/

Carskadon, M. A. (2002). *Adolescent sleep patterns: Biological, social, and psychological influences*. Cambridge, MA: Cambridge University Press.

Carskadon, M. A. (2005). Sleep and circadian rhythms in children and adolescents: Relevance for athletic performance of young people. *Clinics in Sports Medicine, 24*, 319-328.

Carskadon, M. A., & Acebo, C. (2002). Regulation of sleepiness in adolescents: Update, insights, and speculation. *Sleep, 15*, 606-614.

Carskadon, M. A., Acebo, C., & Seifer, R. (2001). Extended nights, sleep loss, and recovery sleep in adolescents. *Archives Italiennes de Biologie, 139*(3), 301-312.

Cauli, O., & Morelli, M. (2005). Caffeine and the dopaminergic system. *Behavioural Pharmacology, 16*, 63-77.

Chahal, H., Fung, C., Kuhle, S., & Veugelers, P. J. (2013). Availability and night-time use of electronic entertainment and communication devices are associated with short sleep duration and obesity among Canadian children. *Pediatric Obesity, 8*(1), 42-51. doi: 10.1111/j.2047-6310.2012.00085.x

Challamel, M. J. (2001). Sleep in school-aged children. *Acta Paediatrica, 90*, 1365-1367.

Chang, P. P., Ford, D. E., Mead, L. A., Cooper-Patrick, L., & Klag, M. J. (1997). Insomnia in young men and subsequent depression: The Johns Hopkins precursors study. *American Journal of Epidemiology, 146*(2), 105-114.

Chervin, R. D., & Archbold, K. H. (2001). Hyperactivity and polysomnographic findings in children evaluated for sleep-disordered breathing. *Sleep, 24*(3), 313-320.

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

Chesson, A. L. Jr., Littner, M., Davila, D., Anderson, W. M., Grigg-Damberger, M., Hartse, K.,...Wise, M. (1999b). Practice parameters for the use of light therapy in the treatment of sleep disorders. Standards of Practice Committee, American Academy of Sleep Medicine. *Sleep, 22*(5), 641-660.

Clore, E. R., & Hibel, J. (1993). The parasomnias of childhood. *Journal of Pediatric Health Care, 7*(1), 12-16.

Colson, E. R., Willinger, M., Rybin, D., Heeren, T., Smith, L. A., Lister, G., & Corwin, M. J. (2013). Trends and factors associated with bed-sharing: The National Infant Sleep Position Study (NISP) 1993-2010. *JAMA Pediatrics, 167*(11), 1032-1037.

Colten, H. R., & Altevogt, B. M. (2006). Functional and Economic Impact of Sleep Loss and Sleep-Related Disorders. In *Sleep Disorders and Sleep Deprivation: An Unmet Public Health Problem*. Washington, DC: National Academies Press.

Corkum, P., Panton, R., Ironside, S., MacPherson, M., & Williams, T. (2008). Acute impact of immediate release methylphenidate administered three times a day on sleep in children with attention hyperactivity deficit disorder. *Journal of Pediatric Psychology, 33*(4), 368-379.

Crabtree, V., Ivanenko, A., O'Brien, L., & Gozal, D. (2003). Periodic limb movement disorder of sleep in children. *Journal of Sleep Research, 12*, 73-81.

Cummings, C. (2012). Melatonin for the management of sleep disorders in children and adolescents. *Paediatrics & Child Health, 17*(6), 331-333.

Czeisler, C. A., Allan, J. S., Strogatz, S. H., Ronda, J. M., Sanchez, R., Rios, C. D.,...Kronauer, R. E. (1986). Bright light resets the human circadian pacemaker independent of the timing of the sleep-wake cycle. *Science, 233*(4764), 667-671.

Dahl, R. (1996a). The impact of inadequate sleep on children's daytime cognitive function. *Seminars in Pediatric Neurology, 3*, 44-50.

Dahl, R. (1996b). The regulation of sleep and arousal: Development and psychopathology. *Development and Psychopathology, 8*, 3-27.

Dahl, R. E., & Lewin, D. S. (2002). Pathways to adolescent health sleep regulation and behavior. *Journal of Adolescent Health, 31*(6 Suppl), 175-184.

Dahlitz, M., Alvarez, B., Vignau, J., English, J., Arendt, J., & Parkes, J. D. (1991). Delayed sleep phase syndrome response to melatonin. *Lancet, 337*(8750), 1121-1124.

Davidson, R. J., Pizzagalli, D., Nitschke, J. B., & Putnam, K. (2002). Depression: Perspectives from affective neuroscience. *Annual Review of Psychology, 53*, 545-574.

Davis, B. J., Rajput, A., Rajput, M. L., Aul, E. A., & Eichhorn, G. R. (2000). A randomized, double-blind placebo-controlled trial of iron in restless legs syndrome. *European Neurology, 43*(2), 70-75. doi: 8138

Davis, K. F., Parker, K. P., & Montgomery, G. L. (2004). Sleep in infants and young children: Part two: Common sleep problems. *Journal of Pediatric Health Care, 18*(3), 130-137. doi: 10.1016/S0891

Drake, C., Nickel, C., Burduvali, E., Roth, T., Jefferson, C., & Pietro, B. (2003). The pediatric daytime sleepiness scale (PDSS): Sleep habits and school outcomes in middle-school children. *Sleep, 26*(4), 455-458.

Drevets, W. C., Price, J. L., & Furey, M. L. (2008). Brain structural and functional abnormalities in mood disorders: Implications for neurocircuitry models of depression. *Brain Structure and Function, 213*(1-2), 93-118. doi: 10.1007/s00429-008-0189-x

Drummond, S., Brown, G., Stricker, J., Buxton, R., Wong, E., & Gillin, J. (1999). Sleep deprivation-induced reduction in cortical functional response to serial subtraction. *NeuroReport, 10*, 3745-3748.

Durmer, J. S., & Dinges, D. F. (2005). Neurocognitive consequences of sleep deprivation. *Seminars in Neurology, 25*(1), 117-129. doi: 10.1055/s-2005-867080

Durmer, J. S., & Quraishi, G. H. (2011). Restless legs syndrome, periodic leg movements, and periodic limb movement disorder in children. *Pediatric Clinics of North America, 58*(3), 591-620. doi: 10.1016/j.pcl.2011.03.005

Dworak, M., Schierl, T., Bruns, T., & Struder, H. K. (2007). Impact of singular excessive computer game and television exposure on sleep patterns and memory performance of school-aged children. *Pediatrics, 120*(5), 978-985. doi: 10.1542/peds.2007-0476

Eaton, D. K., McKnight-Eily, L. R., Lowry, R., Perry, G. S., Presley-Cantrell, L., & Croft, J. B. (2010). Prevalence of insufficient, borderline, and optimal hours of sleep among high school students - United States, 2007. *Journal of Adolescent Health, 46*(4), 399-401. doi: 10.1016/j.jadohealth.2009.10.011

Ebadi, M., & Govitrapong, P. (1986). Neural pathways and neurotransmitters affecting melatonin synthesis. *Journal of Neural Transmission Supplement, 21*, 125-155.

Eggermont, S., & Van den Bulck, J. (2006). Nodding off or switching off? The use of popular media as a sleep aid in secondary-school children. *Journal of Paediatric Child Health, 42*(7-8), 428-433. doi: 10.1111/j.1440-1754.2006.00892.x

El-Sheikh, M., Buckhalt, J. A., Cummings, E. M., & Keller, P. (2007). Sleep disruptions and emotional insecurity are pathways of risk for children. *Journal of Child Psychology and Psychiatry, 48*(1), 88-96. doi: JCPP1604 [pii] 10.1111/j.1469-7610.2006.01604.x

El-Sheikh, M., Buckhalt, J. A., Keller, P. S., Cummings, E. M., & Acebo, C. (2007). Child emotional insecurity and academic achievement: The role of sleep disruptions. *Journal of Family Psychology, 21*(1), 29-38. doi: 10.1037/0893-3200.21.1.29

Etkin, A. (2010). Functional neuroanatomy of anxiety: A neural circuit perspective. *Current Topics in Behavioural Neuroscience, 2*, 251-277.

Friedman, B., & Goldman, R. D. (2011). Anti-inflammatory therapy for obstructive sleep apnea in children. *Canadian Family Physician, 57*(8), 891-893.

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.

Galland, B. C., Taylor, B. J., Elder, D. E., & Herbison, P. (2012). Normal sleep patterns in infants and children: A systematic review of observational studies. *Sleep Medicine Reviews, 16*(3), 213-222.

Garcia-Borreguero, D., Larrosa, O., de la Llave, Y., Verger, K., Masramon, X., & Hernandez, G. (2002). Treatment of restless legs syndrome with gabapentin: A double-blind, cross-over study. *Neurology, 59*(10), 1573-1579.

Garrison, M. M., Liekweg, K., & Christakis, D. A. (2011). Media use and child sleep: The impact of content, timing, and environment. *Pediatrics, 128*(1), 29-35. doi: 10.1542/peds.2010-3304

Gibson, E. S., Powles, A. C., Thabane, L., O'Brien, S., Molnar, D. S., Trajanovic, N.,...Chilcott-Tanser, L. (2006). "Sleepiness" is serious in adolescence: two surveys of 3235 Canadian students. *BMC Public Health, 6*, 116. doi: 1471-2458-6-116 [pii] 10.1186/1471-2458-6-116

Gingras, J. L., & Gaultney, J. F. (2008). Restless legs syndrome and periodic limb movement disorders: Association with ADHD. In A. Ivanenko (Ed.), *Sleep and psychiatric disorders in children and adolescents, first edition* (pp. 193-224). New York, NY: Informa Healthcare USA, Inc.

Glutting, J. J., McDermott, P. A., Prifitera, A., & McGrath, E. A. (1994). Core profile types for the WISC-III and WIAT: Their development and application in identifying multivariate IQ-achievement discrepancies. *School Psychology Review 23*, 619-639.

Gozal, D. (1998). Sleep-disordered breathing and school performance in children. *Pediatrics, 102*(3 Pt 1), 616-620.

Gradisar, M., Dohnt, H., Gardner, G., Paine, S., Starkey, K. Menne, A.,… Trenowden, S. (2011). A randomized controlled trial of cognitive-behavior therapy plus bright light therapy for adolescent delayed sleep phase disorder. *Sleep, 34(12)*, 1671-1680. doi: 10.5665/sleep.1432

Greig, A. (2010). *The Greig health record*. Retrieved from http://www.cps.ca/tools-outils/greig-health-record

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.

Gruber, R., Laviolette, R., Deluca, P., Monson, E., Cornish, K., & Carrier, J. (2010). Short sleep duration is associated with poor performance on IQ measures in healthy school-age children. *Sleep Medicine, 11*(3), 289-294. doi: S1389-9457(10)00042-0 [pii]10.1016/j.sleep.2009.09.007

Gujar, N., McDonald, S. A., Nishida, M., & Walker, M. P. (2011). A role for REM sleep in recalibrating the sensitivity of the human brain to specific emotions. *Cerebral Cortex, 21*(1), 115-123. doi: 10.1093/cercor/bhq064

Gujar, N., Yoo, S-S., Hu, P. T., & Walker, M. P. (2011). Sleep deprivation amplifies reactivity of brain reward networks, biasing the appraisal of positive emotional experiences. *Journal of Neuroscience, 31*(12), 4466-4474.

Happe, S., Sauter, C., Klosch, G., Saletu, B., & Zeitlhofer, J. (2003). Gabapentin versus ropinirole in the treatment of idiopathic restless legs syndrome. *Neuropsychobiology, 48*(2), 82-86.

Harrison, Y., & Horne, J. (2000). The impact of sleep deprivation on decision making: A review. *Journal of Experimental Psychology, 6*, 236-249.

Harrison, Y., & Horne, J. A. (1998). Sleep loss impairs short and novel language tasks having a prefrontal focus. *Journal of Sleep Research, 7*(2), 95-100.

Health Canada. (2012). Caffeine in food. Retrieved from http://www.hc-sc.gc.ca/fn-an/securit/addit/caf/food-caf-aliments-eng.php

Higuchi, S., Motohashi, Y., Liu, Y., Ahara, M., & Kaneko, Y. (2003). Effects of VDT tasks with a bright display at night on melatonin, core temperature, heart rate, and sleepiness. *Journal of Applied Physiology, 94*(5), 1773-1776. doi: 10.1152/japplphysiol.00616.2002

Hillman, D. R., Murphy, A. S., & Pezzullo, L. (2006). The economic cost of sleep disorders. *Sleep, 29*(3), 299-305.

Horne, J. A. (1988). Sleep loss and divergent thinking ability. *Sleep, 11*, 528-536.

Huang, Y. S., Lakkis, C., & Guilleminault, C. (2010). Kleine-Levin syndrome: Current status. *Medical Clinics of North America, 94*(3), 557-562. doi: 10.1016/j.mcna.2010.02.011

Iber, C., Ancoli-Israel, S., Chesson, A. L., & Quan, S. F. (2007). *The AASM manual for the scoring of sleep and associated events*. West Chester, IL American Academy of Sleep Medicine.

Iglowstein, I., Jenni, O. G., Molinari, L., & Largo, R. H. (2003). Sleep duration from infancy to adolescence: Reference values and generational trends. *Pediatrics, 111*(2), 302-307.

Johnson, E. O., Roth, T., Schultz, L., & Breslau, N. (2006). Epidemiology of DSM-IV insomnia in adolescence: Lifetime prevalence, chronicity, and an emergent gender difference, *Pediatrics, 117(2)*, e247-e256. doi: 10.1542/peds.2004-2629

Johnson, J. G., Cohen, P., Kasen, S., First, M. B., & Brook, J. S. (2004). Association between television viewing and sleep problems during adolescence and early adulthood. *Archives of Pediatric and Adolescent Medicine, 158*(6), 562-568. doi: 10.1001/archpedi.158.6.562

Kahn, A., Dan, B., Groswasser, J., Franco, P., & Sottiaux, M. (1996). Normal sleep architecture in infants and children. *Journal of Clinical Neurophysiology, 13*(3), 184-197.

Kalra, M., & Inge, T. (2006). Effect of bariatric surgery on obstructive sleep apnoea in adolescents. *Paediatric Respiratory Reviews, 7*(4), 260-267. doi: 10.1016/j.prrv.2006.08.004

Keith, T. Z. (1993). Latent variable structural equation models: LISREL in special education research. *Remedial and Special Education, 14,* 36-46.

Kheirandish, L., Goldbart, A. D., & Gozal, D. (2006). Intranasal steroids and oral leukotriene modifier therapy in residual sleep-disordered breathing after tonsillectomy and adenoidectomy in children. *Pediatrics, 117*(1), e61-66. doi: 10.1542/peds.2005-0795

Kheirandish-Gozal, L., & Gozal, D. (2008). Intranasal budesonide treatment for children with mild obstructive sleep apnea syndrome. *Pediatrics*, *122*(1), e149-e155.

Killgore, W. D., Balkin, T. J., & Wesensten, N. J. (2006). Impaired decision making following 49 h of sleep deprivation. *Journal of Sleep Research, 15*(1), 7-13. doi: JSR487 [pii] 10.1111/j.1365-2869.2006.00487.x

Kirk, V. G., & Bohn, S. (2004). Periodic limb movements in children: Prevalence in a referred population. *Sleep. 27(2)*, 313-315.

Knutson, K. L., Spiegel, K., Penev, P., & Van Cauter, E. (2012). The metabolic consequences of sleep deprivation. *Sleep Medicine Reviews, 11*(3), 163-178.

Kopasz, M., Loessl, B., Hornyak, M., Riemann, D., Nissen, C., Piosczyk, H. V., & Voderholzer, U. (2010). Sleep and memory in healthy children and adolescents – A critical review. *Sleep Medicine Reviews, 14*, 167-177.

Kotagal, S. (2008). Narcolepsy and idiopathic hypersomnia in childhood. In A. Ivanenko (Ed.), *Sleep and psychiatric disorders in children and adolescents, first edition* (pp. 163-174). New York, NY: Informa Healthcare USA, Inc.

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

Kotagal, S., & Chopra, A. (2012). Pediatric sleep-wake disorders. *Neurology Clinics, 30*, 1193-1212. doi: http://dx.doi.org/10.1016/j.ncl.2012.08.005

Kotagal, S., Nichols, C. D., Grigg-Damberger, M. M., Marcus, C. L., Witmans, M. B., Kirk, V. G.,…Hoban, T. F. (2012). Non-respiratory indications for polysomnography and related procedures in children: An evidence-based review. *Sleep, 35*(11), 1451-1466.

Kotagal, S., & Silber, M. H. (2004). Childhood-onset restless legs syndrome. *Annals of Neurology, 56*(6), 803-807. doi: 10.1002/ana.20292

Kryger, M. H., Otake, K., & Foerster, J. (2002). Low body stores of iron and restless legs syndrome: A correctable cause of insomnia in adolescents and teenagers. *Sleep Medicine, 3*(2), 127-132.

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).

Kushida, C. A. (2006). Ropinirole for the treatment of restless legs syndrome. *Neuropsychiatric Disease and Treatment, 2*(4), 407-419.

Lack, L., & Wright, H. (1993). The effect of evening bright light in delaying the circadian rhythms and lengthening the sleep of early morning awakening insomniacs. *Sleep, 16*(5), 436-443.

Lack, L., Wright, H., Kemp, K., & Gibbon, S. (2005). The treatment of early-morning awakening insomnia with 2 evenings of bright light. *Sleep, 28*(5), 616-623.

Leatherdale, S. T., & Ahmed, R. (2011). Screen-based sedentary behaviours among a nationally representative sample of youth: Are Canadian kids couch potatoes? *Chronic Diseases and Injuries in Canada, 31*(4), 141-146.

LeBourgeois, M. K., Giannotti, F., Cortesi, F., Wolfson, A. R., & Harsh, J. (2005). The relationship between reported sleep quality and sleep hygiene in Italian and American adolescents. *Pediatrics, 115*, 257-265. doi: 10.1542/peds.2004-0815H

Legenbauer, T., Heiler, S., Holtmann, M., Fricke-Oerkermann, L., & Lehmkuhl, G. (2012). The affective storms of school children during night time: Do affective dysregulated school children show a specific pattern of sleep disturbances? *Journal of Neural Transmission, 119*(9), 989-998. doi: 10.1007/s00702-012-0837-4

Lewandowski, A. S., Toliver-Sokol, M., & Palermo, T. M. (2011). Evidence-based review of subjective pediatric sleep measures. *Journal of Pediatric Psychology, 36(7)*, 780-793. doi: 10.1093/jpepsy/jsq119

Lewy, A. J., Ahmed, S., & Sack, R. L. (1996). Phase shifting the human circadian clock using melatonin. *Behavioral Brain Research, 73*(1-2), 131-134.

Li, S., Jin, X., Wu, S., Jiang, F., Yan, C., & Shen, X. (2007). The impact of media use on sleep patterns and sleep disorders among school-aged children in China. *Sleep, 30*(3), 361-367.

Linde, L., & Bergstrom, M. (1992). The effect of one night without sleep on problem-solving and immediate recall. *Psychological Research, 54*(2), 127-136.

Lisk, D. R. (2009). Kleine-Levin syndrome. *Practical Neurology, 9*(1), 42-45. doi: 10.1136/jnnp.2008.161950

Littner, M., Kushida, C. A., McDowell Anderson, W., Bailey, D., Berry, R. B., Davila, D. G.,...Johnson, S. F. (2003). Practice parameters for the role of actigraphy in the study of sleep and circadian rhythms: An update for 2002. *Sleep, 26*(3), 337-341.

Marcus, C. L., Brooks, L. J., Davidson-Ward, S. Draper, K. A., Gozal, D., Halbower, A. C.,…Spruyt, K. (2012a). Diagnosis and management of childhood obstructive sleep apnea syndrome. *Pediatrics, 130*, e714-e755.

Marcus, C. L., Moore, R. H., Rosen, C. L., Giordani, B., Garetz, S. L., Taylor, H. G.,…Redline S. (2013). Childhood Adenotonsillectomy Trial (CHAT): A randomized trial of adenotonsillectomy for childhood sleep apnea. *New England Journal of Medicine, 368*(25), 2366-2376. doi: 10.1056/NEJMoa1215881.

Marcus, C. L., Radcliffe, J., Konstantinopoulou, S., Beck, S. E., Cornaglia, M. A., Traylor, J.,...Meltzer, L. J. (2012b). Effects of positive airway pressure therapy on neurobehavioral outcomes in children with obstructive sleep apnea. *American Journal of Respiratory Critical Care Medicine, 185*(9), 998-1003. doi: 10.1164/rccm.201112-2167OC

Marino, A., Ranieri, R., Chiarotti, F., Villa, M. P., & Malagola, C. (2012). Rapid maxillary expansion in children with Obstructive Sleep Apnoea Syndrome (OSAS). *European Journal of Paediatric Dentistry, 13*(1), 57-63.

Markov, D., Jaffe, F., & Doghramji, K. (2006). Update on parasomnias. *Psychiatry (Edgmont), 3*(7), 60-76.

Martinez, S., & Guilleminault, C. (2004). Periodic leg movements in prepubertal children with sleep disturbance. *Developmental Medicine & Child Neurology, 46*(11), 765-770.

Mason, T. B., 2nd, & Pack, A. I. (2007). Pediatric parasomnias. *Sleep, 30*(2), 141-151.

Matricciani, L., Olds, T., & Petkov, J. (2011). In search of lost sleep: Secular trends in the sleep time of school-aged children and

adolescents. *Sleep Medicine Reviews, 16* (3), 203-211. doi: 10.1016/j.smrv.2011.03.005.

Meijer, A. M. (2008). Chronic sleep reduction, functioning at school and school achievement in preadolescents. *Journal of Sleep Research, 17*(4), 395-405. doi: JSR677 [pii] 10.1111/j.1365-2869.2008.00677.x

Meijer, A. M., Habekothe, H. T., & Van Den Wittenboer, G. L. (2000). Time in bed, quality of sleep and school functioning of children. *Journal of Sleep Research, 9*(2), 145-153. doi: jsr198 [pii]

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. doi: 10.1542/peds.2004-0730

Meltzer, L. J., & Mindell, J. A. (2007). Relationship between child sleep disturbances and maternal sleep, mood, and parenting stress: A pilot study. *Journal of Family Psychology, 21*(1), 67-73. doi: 2007-03149-008 [pii]10.1037/0893-3200.21.1.67

Mesquita, G., & Reimao, R. (2007). Nightly use of computer by adolescents: Its effect on quality of sleep. *Arquivos de Neuropsiquiatria, 65*(2B), 428-432.

Mesulam, M. (1990). Large-scale neurocognitive networks and distributed processing for attention, language, and memory. *Annals of Neurology, 28*, 597-613.

Miano, S., Rizzoli, A., Evangelisti, M., Bruni, O., Ferri, R., Pagani, J., & Villa, M. P. (2009). NREM sleep instability changes following rapid maxillary expansion in children with obstructive apnea sleep syndrome. [Controlled Clinical Trial]. *Sleep Medicine, 10*(4), 471-478. doi: 10.1016/j.sleep.2008.04.003

Mindell, J. A. (1999). Empirically supported treatments in pediatric psychology: Bedtime refusal and night wakings in young children. *Journal of Pediatric Psychology, 24*, 465-481.

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.

Mindell, J. A., Meltzer, L. J., Carskadon, M. A., & Chervin, R. D. (2009). Developmental aspects of sleep hygiene: Findings from the 2004 National Sleep Foundation Sleep in America Poll. *Sleep Medicine, 10*(7), 771-779.

Mindell, J. A., & Owens, J. A. (2010). *A clinical guide to pediatric sleep, diagnosis and management of sleep problems.* Second edition. Philadelphia, PA: Wolters Kluwer.

Mindell, J. A., Owens, J. A., Carskadon, M. A., Durand, V. M., Mindell, J., Mapstone, E.,...Nagel, K. (1997). *Developmental features of sleep: Concise guides*. New York, NY: American Psychiatric Press.

Mistry, K. B., Minkovitz, C. S., Strobino, D. M., & Borzekowski, D. L. (2007). Children's television exposure and behavioral and social outcomes at 5.5 years: Does timing of exposure matter?. *Pediatrics, 120*(4), 762-769. doi: 10.1542/peds.2006-3573

Mograss, M. A., Guillem, F., Brazzini-Poisson, V., & Godbout, R. (2009). The effects of total sleep deprivation on recognition memory processes: A study of event-related potential. *Neurobiology of Learning and Memory, 91*(4), 343-352.

Mohri, I., Kato-Nishimura, K., Kagitani-Shimono, K., Kimura-Ohba, S., Ozono, K., Tachibana, N., & Taniike, M. (2012). Evaluation of oral iron treatment in pediatric restless legs syndrome (RLS). *Sleep Medicine, 13*(4), 429-432. doi: 10.1016/j.sleep.2011.12.009

Moore, M. (2012) Behavioral sleep problems in children and adolescents. *Journal of Clinical Psychology in Medical Settings*, *19(1)*, 77-83.

Moore, R. Y. (1999). A clock for the ages. *Science, 284*(5423), 2102-2103.

Morgenthaler, T. I, Kapen, S., Lee-Chiong, T., Alessi, C., Boehlecke, B., Brown, T.,…Swick, T. (2006). Practice parameters for the medical therapy of obstructive sleep apnea. *Sleep, 29* (8)*,* 1031-1035.

Muhle, H., Neumann, A., Lohmann-Hedrich, K., Lohnau, T., Lu, Y., Winkler, S.,...Stephani, U. (2008). Childhood-onset restless legs syndrome: Clinical and genetic features of 22 families. *Movement Disorders, 23*(8), 1113-1121; quiz 1203. doi: 10.1002/mds.22016

Murphy, P. J., & Campbell, S. S. (1996). Enhanced performance in elderly subjects following bright light treatment of sleep maintenance insomnia. *Journal of Sleep Research, 5*(3), 165-172.

National Sleep Foundation (2004). *2004 Sleep in America Poll*. Retrieved from http://www.sleepfoundation.org/sites/default/files/FINAL%20SOF%202004.pdf

National Sleep Foundation (2006). *2006 Sleep in America Poll*. Retrieved from http://www.sleepfoundation.org/site/c.huIXKjM0IxF/b.2419037/k.1466/2006_Sleep_in_America_Poll.html

National Sleep Foundation (2014). *How much sleep do we really need?* Retrieved from http://sleepfoundation.org/how-sleep-works/how-much-sleep-do-we-really-need

Neisser, U., Boodoo, G., Bouchard, T. J., Boykin, A. W., Brody, N., Ceci, S. J.,…Urbina, S. (1996). Intelligence: Knowns and unknowns. *American Psychologist 51*, 77-101.

Nevsimalova, S. (2009). Narcolepsy in childhood. *Sleep Medicine Reviews, 13*(2), 169-180.

Nicholson, A. N., Bradley, C. M., & Pascoe, P. A. (1994). Medications: Effects on sleep and wakefulness. In M. H. Kryger, T. Roth & W. C. Dement (Eds.), *Principles and practice of sleep medicine* (2nd ed., pp. 364). Philadelphia, PA: WB Saunders.

Nilsson, J. P., Soderstrom, M., Karlsson, A. U., Lekander, M., Akerstedt, T., Lindroth, N. E., & Axelsson, J. (2005). Less effective executive functioning after one night's sleep deprivation. *Journal of Sleep Research, 14*(1), 1-6. doi: 10.1111/j.1365-2869.2005.00442.x

Nixon, G. M., Thompson, J. M., Han, D. Y., Becroft, D. M., Clark, P. M., Robinson, E.,...Mitchell, E. A. (2008). Short sleep duration in middle childhood: Risk factors and consequences. *Sleep, 31*(1), 71-78.

Ofer, D., & Marcus, C. L. (2012). CPAP treatment in children. In L.Kheirandish-Gozal, & D. Gozal (Eds.), *Sleep disordered breathing in children: A comprehensive clinical guide to evaluation and treatment (Respiratory medicine)* (pp. 531-540). New York, NY: Humana Press.

Ohayon, M. M., Roberts, R. E., Zulley, J., Smirne, S., & Priest, R. G. (2000). Prevalence and patterns of problematic sleep among older adolescents. *Journal of the American Academy of Child & Adolescent Psychiatry***,** *39*(12), 1549-1556.

Oka, Y., Suzuki, S., & Inoue, Y. (2008). Bedtime activities, sleep environment, and sleep/wake patterns of Japanese elementary school children. *Behavioral Sleep Medicine, 6*(4), 220-233. doi: 10.1080/15402000802371338

Olds, T., Blunden, S., Petkov, J., & Forchino, F. (2010). The relationships between sex, age, geography and time in bed in adolescents: A meta-analysis of data from 23 countries. *Sleep Medicine Reviews, 14*(6), 371-378.

Orbeta, R. L., Overpeck, M. D., Ramcharran, D., Kogan, M. D., & Ledsky, R. (2006). High caffeine intake in adolescents: Associations with difficulty sleeping and feeling tired in the morning. *Journal of Adolescent Health, 38*(4), 451-453. doi: 10.1016/j.jadohealth.2005.05.014

Owens, J., Maxim, R., McGuinn, M., Nobile, C., Msall, M., & Alario, A. (1999). Television-viewing habits and sleep disturbance in school children*. Pediatrics, 104*(3), e1-e8.

Owens, J., & Mindell, J. (2011). Pediatric insomnia. *Pediatric Clinics of North America, 58*, 555-569.

Paavonen, E. J., Pennonen, M., Roine, M., Valkonen, S., & Lahikainen, A. R. (2006). TV exposure associated with sleep disturbances in 5- to 6-year-old children. *Journal of Sleep Research, 15*(2), 154-161. doi: 10.1111/j.1365-2869.2006.00525.x

Paine, S., & Gradisar, M. (2011). A randomised controlled trial of cognitive-behaviour therapy for behavioural insomnia of childhood in school-aged children. *Behaviour Research and Therapy, 49*(6-7), 379-388. doi: 10.1016/j.brat.2011.03.008

Parkes, J. D. (1999). Genetic factors in human sleep disorders with special reference to Norrie disease, Prader-Willi syndrome and Moebius syndrome. *Journal of Sleep Research, 8* (S1)*,* 14-22.

Paykel, E. S., Fleminger, R., & Watson, J. P. (1982). Psychiatric side effects of antihypertensive drugs other than reserpine. *Journal of Clinical Psychopharmacology, 2*(1), 14-39.

PDR.net. (2013). PDR Network, LLC. Retrieved from http://www.pdr.net/

Peirano, P., Algarin, C., & Uauy, R. (2003). Sleep-wake states and their regulatory mechanisms throughout early human development. *Journal of Pediatrics, 143*, S70-S79.

Petit, D., Touchette, E., Tremblay, R. E., Boivin, M., & Montplaisir, J. (2007). Dyssomnias and parasomnias in early childhood. *Pediatrics, 119(5)*, e1016-e1025. doi:10.1542/peds.2006-2132

Pezawas, L., Meyer-Lindenberg, A., Drabant, E. M., Verchinski, B. A., Munoz, K. E., Kolachana, B. S.,...Weinberger, D. R. (2005). 5-HTTLPR polymorphism impacts human cingulate-amygdala interactions: A genetic susceptibility mechanism for depression. *Nature Neuroscience, 8*(6), 828-834. doi: 10.1038/nn1463

Picchietti, D. (2007). Is iron deficiency an underlying cause of pediatric restless legs syndrome and of attention-deficit/hyperactivity disorder? *Sleep Medicine, 8*(7-8), 693-694. doi: 10.1016/j.sleep.2007.06.007

Picchietti, D. L., & Stevens, H. E. (2008). Early manifestations of restless legs syndrome in childhood and adolescence. *Sleep Medicine, 9*(7), 770-781. doi: 10.1016/j.sleep.2007.08.012

Picchietti, M. A., & Picchietti, D. L. (2010). Advances in pediatric restless legs syndrome: Iron, genetics, diagnosis and treatment. *Sleep Medicine, 11*(7), 643-651. doi: 10.1016/j.sleep.2009.11.014

Pirelli, P., Saponara, M., & Guilleminault, C. (2004). Rapid maxillary expansion in children with obstructive sleep apnea syndrome. *Sleep, 27*(4), 761-766.

Plante, D. T., & Winkelman, J. W. (2008). Sleep disturbance in bipolar disorder: Therapeutic implications. *American Journal of Psychiatry, 165*(7), 830-843. doi: 10.1176/appi.ajp.2008.08010077

Pollak, C. P., & Bright, D. (2003). Caffeine consumption and weekly sleep patterns in US seventh-, eighth-, and ninth-graders. *Pediatrics, 111*(1), 42-46.

Pressman, M. R. (2014). Definition and consequences of sleep deprivation, from http://www.uptodate.com/contents/definition-and-consequences-of-sleep-deprivation

Rauch, S. L., Whalen, P. J., Shin, L. M., McInerney, S. C., Macklin, M. L., Lasko, N. B.,...Pitman, R. K. (2000). Exaggerated amygdala response to masked facial stimuli in posttraumatic stress disorder: A functional MRI study. *Biological Psychiatry, 47*, 769-776.

Reid, G. J., Huntley, E. D., & Lewin, D. S. (2009). Insomnias of childhood and adolescence. *Child and Adolescent Psychiatric Clinics of North America, 18*(4), 979-1000.

Reuveni, H., Simon, T., Tal, A., Elhayany, A., & Tarasiuk, A. (2002). Health care services utilization in children with obstructive sleep apnea syndrome. *Pediatrics, 110*(1), 68-72.

Rich, B. A., Vinton, D. T., Roberson-Nay, R., Hommer, R. E., Berghorst, L. H., McClure, E. B.,...Leibenluft, E. (2006). Limbic hyperactivation during processing of neutral facial expressions in children with bipolar disorder. *Proceedings of the National Academy of Sciences, 103*(23), 8900-8905.

Roehrs, T., & Roth, T. (2008). Caffeine: Sleep and daytime sleepiness. *Sleep Medicine Reviews, 12*(2), 153-162. doi: 10.1016/j.smrv.2007.07.004

Rourke, L., Leduc, D., & Rourke, J. (2011). *Rourke baby record: Evidence-based infant/child health maintenance*. Retrieved from http://www.rourkebabyrecord.ca/download_rbr.asp

Sadeh, A., Gruber, R., & Raviv, A. (2002). Sleep, neurobehavioral functioning and behavior problems in school-age children. *Child Development, 73*, 405-417.

Scala-Foley, M., & Bryant, N. (2004). Medicare fraud and abuse. *Issue Brief (Center for Medicare Education), 5*(6), 1-6.

Shin, L. M., Rauch, S. L., & Pitman, R. K. (2006). Amygdala, medial prefrontal cortex, and hippocampal function in PTSD. *Annals of the New York Academy of Sciences, 1071*, 67-79. doi: 10.1196/annals.1364.007

Skene, D. J., & Arendt, J. (2006). Human circadian rhythms: Physiological and therapeutic relevance of light and melatonin. *Annals of Clinical Biochemistry, 4*3(Pt 5), 344-353. doi: 10.1258/000456306778520142

Smith, C. (1995). Sleep states and memory processes. *Behaviour and Brain Research, 69*(1-2), 137-145.

Spilsbury, J. C., Storfer-Isser, A., Drotar, D., Rosen, C. L., Kirchner, L. H., Benham, H., & Redline, S. (2004). Sleep behavior in an urban US sample of school-aged children. *Archives of Pediatric and Adolescent Medicine*, *158*, 988-994.

Stahl, S. M. (2008). *Stahl's essential psychopharmacology: Neuroscientific basis and practical applications* (3rd Ed.). Cambridge, NY: Cambridge University Press.

Staner, L. (2005). Sleep disturbances, psychiatric disorders, and psychotropic drugs. *Dialogues in Clinical Neuroscience, 7*(4), 323-334.

Stepanski, E. J., & Wyatt, J. K. (2003). Use of sleep hygiene in the treatment of insomnia. *Sleep Medicine Reviews, 7*(3), 215-225.

Stickgold, R., & Walker, M. P. (2005). Sleep and memory: The ongoing debate. *Sleep, 28*(10), 1225-1227.

Storfer-Isser, A., LeBourgeois, M. K., Harsh, J., Tompsett, C. J., & Redline, S. (2013). Psychometric properties of the Adolescent Sleep Hygiene Scale. *Journal of Sleep Research, 2013*, 1-10. doi: 10.1111/jsr.12059

Sullivan, S. S. (2012). Current treatment of selected pediatric sleep disorders. *Neurotherapeutics, 9*(4), 791-800. doi: 10.1007/s13311-012-0149-2

Taheri, S. (2006). The link between short sleep duration and obesity: We should recommend more sleep to prevent obesity. *Sleep and Obesity, 91*(11), 881-884.

Teotia, S., & Gupta, M. N. (2002). Magnetite-alginate beads for purification of some starch degrading enzymes. *Molecular Biotechnology, 20*(3), 231-237. doi: 10.1385/MB:20:3:231

Thiedke, C. C. (2003). Nocturnal enuresis. *American Family Physician, 67*(7), 1499-1506.

Thompson, D. A., & Christakis, D. A. (2005). The association between television viewing and irregular sleep schedules among children less than 3 years of age. *Pediatrics, 116*(4), 851-856. doi: 10.1542/peds.2004-2788

Thorndike, R. L. (1994). 'g'. *Intelligence, 19*, 145-155.

Thorpy, M. J. (2012). Classification of sleep disorders. *Neurotherapeutics, 9*(4), 687-701.

Touchette, E., Petit, D., Seguin, J. R., Boivin, M., Tremblay, R. E., & Montplaisir, J. Y. (2007). Associations between sleep duration patterns and behavioral/cognitive functioning at school entry. *Sleep, 30*(9), 1213-1219.

Van den Bulck, J. (2004). Television viewing, computer game playing, and Internet use and self-reported time to bed and time out of bed in secondary-school children. *Sleep, 27*(1), 101-104.

Van den Bulck, J. (2010). The effects of media on sleep. *Adolescent Medicine: State of the Art Reviews, 21*(3), 418-429, vii.

van Geijlswijk, I. M., Korzilius, H. P., & Smits, M. G. (2010). The use of exogenous melatonin in delayed sleep phase disorder: A meta-analysis. *Sleep, 33*(12), 1605-1614.

Villa, M. P., Malagola, C., Pagani, J., Montesano, M., Rizzoli, A., Guilleminault, C., & Ronchetti, R. (2007). Rapid maxillary expansion in children with obstructive sleep apnea syndrome: 12-month follow-up. *Sleep Medicine, 8*(2), 128-134. doi: 10.1016/j.sleep.2006.06.009

Villa, M. P., Rizzoli, A., Miano, S., & Malagola, C. (2011). Efficacy of rapid maxillary expansion in children with obstructive sleep apnea syndrome: 36 months of follow-up. *Sleep and Breathing, 15*(2), 179-184. doi: 10.1007/s11325-011-0505-1

Viorritto, E. N., Kureshi, S. A., & Owens, J. A. (2012). Narcolepsy in the pediatric population. *Current Neurology and Neuroscience Reports*, *12*(2), 175-181.

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

Wahlstrom, K. (2002). Changing times: Findings from the first longitudinal study of later high school start times. *Sleep, 86*, 3-21.

Walters, A. S., Mandelbaum, D. E., Lewin, D. S., Kugler, S., England, S. J., & Miller, M. (2000). Dopaminergic therapy in children with restless legs/periodic limb movements in sleep and ADHD. Dopaminergic Therapy Study Group. *Pediatric Neurology, 22*(3), 182-186.

Warzak, W. J., Evans, S., Floress, M. T., Gross, A. C., & Stoolman, S. (2011). Caffeine consumption in young children. *Journal of Pediatrics, 158*(3), 508-509. doi: 10.1016/j.jpeds.2010.11.022

Weiss, M., Childress, A., Pucci, M., & Hechtman, L., (2011). Review of long-acting stimulant and non-stimulant ADHD pharmacotherapy in Canada. *Journal of the Canadian Academy of Child and Adolescent Psychiatry (Supplement), 20*(2).

Weiss, M., Wasdell, M., Bomben, M., Rea, K., & Freeman, R. (2006). Sleep hygiene and melatonin treatment for children and adolescents with ADHD and insomnia. *Journal of the American Academy of Child and Adolescent Psychiatry, 45*(5), 512-519.

Weissbluth, M. (1995). Naps in children: 6 months-7 years. *Sleep, 18*, 82-87.

Weitzman, E. D., Czeisler, C. A., Coleman, R. M., Spielman, A. J., Zimmerman, J. C., Dement, W.,...Pollak, C. P. (1981). Delayed sleep phase syndrome. A chronobiological disorder with sleep-onset insomnia. *Archives of General Psychiatry, 38*(7), 737-746.

Williams, P. G., Sears, L. L., & Allard, A. (2004). Sleep problems in children with autism. *Journal of Sleep Research, 13*(3), 265-268.

Wimmer, F., Hoffmann, R. F., Bonato, R. A., & Moffitt, A. R. (1992). The effects of sleep deprivation on divergent thinking and attention processes. *Journal of Sleep Research, 1*(4), 223-230. doi: jsr001004223 [pii]

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. *Sleep, 30(12)*, 1712-1727.

Wise, M. S., & Glaze, D. G. (2013). Assessment of sleep disorders in children. Retrieved from Up To Date website: http://www.uptodate.com/contents/assessment-of-sleep-disorders-in-children.

Wise, M. S., Nichols, C. D., Grigg-Damberger, M. M., Marcus, C. L., Witmans, M. B., Kirk, V. G.,…Hoban, T. F. (2011). Respiratory indications for polysomnography in children: An evidence-based review. *Sleep, 34*(3), 389-398.

Wolfson, A. R., Spaulding, N. L., Dandrow, C., & Baroni, E. M. (2007). Middle school start times: The importance of a good night's sleep for young adolescents. *Behavioral Sleep Medicine, 5*(3), 194-209. doi: 10.1080/15402000701263809

World Health Organization. (2011). *Mental Health: A State of Well-being*. Retrieved March 15 2013, 2013, from http://www.who.int/features/factfiles/mental_health/en/index.html

Wulff, K., Gatti, S., Wettstein, J. G., & Foster, R. G. (2010). Sleep and circadian rhythm disruption in psychiatric and neurodegenerative disease. *Nature Reviews Neuroscience, 11*(8), 589-599. doi: 10.1038/nrn2868

Yoo, S. S., Gujar, N., Hu, P., Jolesz, F. A., & Walker, M. P. (2007). The human emotional brain without sleep--a prefrontal amygdala disconnect. *Current Biology, 17*(20), R877-878. doi: 10.1016/j.cub.2007.08.007