

2016

Attention deficit hyperactivity disorder symptomatology and pediatric obesity: Psychopathology or sleep deprivation?

Alyssa Lundahl

University of Nebraska-Lincoln, alyssalundahl@yahoo.com

Timothy D. Nelson

University of Nebraska-Lincoln, tnelson3@unl.edu

Follow this and additional works at: <http://digitalcommons.unl.edu/psychfacpub>



Part of the [Psychology Commons](#)

Lundahl, Alyssa and Nelson, Timothy D., "Attention deficit hyperactivity disorder symptomatology and pediatric obesity: Psychopathology or sleep deprivation?" (2016). *Faculty Publications, Department of Psychology*. 771.

<http://digitalcommons.unl.edu/psychfacpub/771>

This Article is brought to you for free and open access by the Psychology, Department of at DigitalCommons@University of Nebraska - Lincoln. It has been accepted for inclusion in Faculty Publications, Department of Psychology by an authorized administrator of DigitalCommons@University of Nebraska - Lincoln.

Attention deficit hyperactivity disorder symptomatology and pediatric obesity: Psychopathology or sleep deprivation?

Alyssa Lundahl and Timothy D Nelson

University of Nebraska-Lincoln, USA

Abstract

The relationship between attention deficit hyperactivity disorder (ADHD) and obesity in children has received considerable attention in recent years. However, the literature currently overlooks the potential causal and maintaining role that sleep problems may play in this relationship. Using a biopsychosocial framework, this article highlights how sleep problems impact the biological, psychological, and social aspects of both ADHD symptomatology and obesity. An in-depth examination of this model illustrates the imperative need for future research and clinical practice to recognize and explore the role sleep has in the link between obesity and ADHD symptomatology.

Keywords

attention deficit hyperactivity disorder, biopsychosocial, child, obesity, sleep problems

The relationship between attention deficit hyperactivity disorder (ADHD) and obesity in children has received considerable attention in recent years. An emerging body of the literature suggests that youth diagnosed with ADHD have especially high rates of overweight/obesity (up to 57.7%; Agranat-Meged et al., 2005; Erermis et al., 2004) and weigh more, on average, than children without the diagnosis (e.g. Faraone et al., 2005; Lam and Yang, 2007). Some have suggested that this association between obesity and ADHD reflects an impulsive personality type that leads to overeating (Braet et al., 2007), deficits in the cerebral cortex that lead to a lack of control (Korner and Leibel, 2003), and/or a heightened need for immediate rewards (Noble, 2003). However, such explanations overlook a potentially important mechanism that could underlie the observed relationship between ADHD symptomatology and weight: sleep problems.

As we will argue in this article, disruptions in sleep could explain the association between ADHD symptomatology and overweight/obesity, as sleep problems are more common among overweight/obese children (e.g. Tauman and Gozal, 2006) and can cause or exacerbate ADHD symptomatology (e.g. Fallone et al., 2005; Gregory and O'Connor, 2002). Nevertheless, the literature currently lacks both empirical examination of this potential mechanism and a theoretical framework for such investigation. Thus, this article presents a biopsychosocial framework to better understand the relationship among sleep, overweight, and ADHD symptomatology. Implications in extending existing models

of pediatric obesity and ADHD to incorporate disrupted sleep patterns with regard to prevention and treatment are discussed and possible directions for future research are highlighted.

Research linking pediatric overweight/obesity and ADHD symptomatology

The diagnosis of ADHD consists of a number of symptoms and deficits that are heterogeneous in both etiology and presentation. Specifically, to be diagnosed, a child must present with developmentally inappropriate levels of inattention, hyperactivity, and/or impulsivity before the age of 12 years and functioning must be impaired in two or more settings (American Psychiatric Association, 2013). A diagnosis of ADHD, therefore, may result from predominantly inattentive symptoms, predominately hyperactive-impulsive symptoms, or a combination of inattention and hyperactive-impulsive symptoms. Moreover, children with ADHD often have impaired executive functioning, including deficits in behavioral inhibition, working memory, and attention, although such deficits are not required for diagnosis (Willcutt et al., 2005). Given the heterogeneity that is inherent to psychopathological diagnoses such as ADHD, it is important to examine the underlying mechanisms and symptomatology that make up the clinical diagnosis. Although much of the literature exploring the relationship between obesity and ADHD has focused on the *diagnosis* of ADHD, our discussion will emphasize the key underlying deficits associated with this diagnosis (i.e. inattention, hyperactivity, and executive functioning impairment) in an effort to better explicate the complex relationship between obesity, sleep, and ADHD-relevant symptomatology.

Numerous clinical and epidemiological studies have documented a link between obesity and ADHD. Agranat-Meged et al. (2005) found that over half (57.7%) of obese children hospitalized for their condition also had ADHD, a rate much higher than the general pediatric population. Others have found lower, but still significant rates of ADHD in overweight/obese populations. Specifically, Erermis et al. (2004) indicated that 13.3 percent of clinically obese children were diagnosed with ADHD compared to 3.3 percent of non-overweight children. Lam and Yang (2007) also found a significant relationship between ADHD and obesity (i.e. children with ADHD were more likely to be obese) after controlling for numerous covariates, including sex, number of siblings, snoring, and physical activity. Similarly, Waring and Lapane (2008) indicated that children with ADHD and not on medication are 1.5 times more likely to be overweight than children without the diagnosis, even after controlling for key demographics (i.e. age, gender, race, socioeconomic status) and comorbidity (i.e. depression and anxiety). Others examining the weight status of children diagnosed with ADHD concur; children with ADHD have a higher body mass index (BMI) than children without ADHD (Holtkamp et al., 2004; Hubel et al., 2006).

The results similar to those reported above are obtained when examining the prevalence of ADHD symptomatology in overweight children. For example, overweight boys have been found to be significantly more impulsive and less able to concentrate than normal weight children (Braet et al., 2007). Similarly, Cserjési et al. (2007) found that obese children were more likely than non-obese children to have problems with attention, while others suggest that overweight children exhibit more hyperactivity than their non-overweight counterparts (Lumeng et al., 2003). Children with excess weight have also been found to score higher on measures assessing disinhibition, an executive functioning deficit often implicated in children with ADHD (Maayan et al., 2011). Finally, Carey et al. (1988) found that maternally reported hyperactivity and low attention span predicted weight

gain throughout childhood, suggesting a possible causal mechanism between ADHD symptomatology and excess weight. Although the sample sizes in some of the studies were quite small (e.g. Cserjési et al., 2007; $n=12$) and thus, generalizability of findings may be limited, it is evident that a large number of studies confirm the presence of greater ADHD symptomatology in overweight children.

A variety of mechanisms have been posited to explain the relationship between obesity and ADHD symptoms. For example, Braet et al. (2007) hypothesize three explanations for the apparent relationship: (1) an impulsive personality may place individuals at risk for overeating, (2) having excess weight may result in personality changes, or (3) an interaction of the prior two mechanisms. Alternatively, others have taken a more brain-based explanatory approach, indicating that deficits in the cerebral cortex (Korner and Leibel, 2003) and hypothalamus (Schwartz et al., 2000) found in those with ADHD may also lead to a failure to control caloric intake. The reward deficiency syndrome has also been implicated as a potential mechanism linking obesity to ADHD, such that both obese individuals and those with ADHD may have abnormalities in their dopaminergic system that results in a heightened need for immediate rewards (e.g. impulsive behavior and overeating; Noble, 2003; Poston et al., 1998). Not one study, however, has considered the role that disrupted sleep patterns might play. In fact, only one of the aforementioned studies documenting the link between obesity and ADHD symptoms assessed for and controlled for disrupted sleep patterns (Lam and Yang, 2007). However, disrupted sleep patterns in this study were assessed via parent-report of snoring, which is highly subjective, a limitation that Lam and Yang (2007) recognized. As noted, not controlling for disrupted sleep patterns is problematic given that (1) sleep-related problems are quite prevalent in obese populations and (2) sleep problems are related to ADHD symptomatology.

Potential (and overlooked) Role of Sleep

Research has widely documented the increased risk that obese children are at for developing disrupted sleep patterns, especially sleep-disordered breathing (Guilleminault et al., 1981; Marcus et al., 1998; Redline et al., 1999; Tauman and Gozal, 2006; Young et al., 2002). In fact, rates of sleep-disordered breathing (ranging from snoring to obstructive sleep apnea) in overweight and obese pediatric populations range from 27percent to 55percent (Kalra et al., 2012; Lumeng et al., 2003; Mallory et al., 1989; Marcus et al., 1998; Wing et al., 2003; Young et al., 2002), whereas rates in the general pediatric population are as low as 1.2 percent (Bixler et al., 2009). Indeed, Redline et al. (1999) found that obese children are at a four- to five-fold risk for developing sleep-disordered breathing problems. Moreover, a meta-analysis by Cappuccio et al. (2008) indicates that a less severe disrupted sleep pattern, short-sleep duration, is also more common among obese children and adults. Although a primary risk factor for developing sleep-disordered breathing and associated disrupted sleep patterns is adenotonsillar hyperplasia/hypertrophy (Gozal et al., 2006; Marcus et al., 1998), other mechanisms are likely. For example, narrowing of the upper airway may occur from fatty infiltration of such areas, while fatty deposits in other neck regions may result in pharyngeal collapsibility (Horner et al., 1989; White et al., 1985). High levels of adipose tissue in the abdominal cavity and around the thorax may also increase respiratory load and make breathing difficult, especially during sleep (Mallory and Beckerman, 1992).

With regard to ADHD symptomatology, a diagnosis of ADHD is often accompanied by a comorbid sleep disorder or sleep disturbance, with 25 to 50 percent of youth with an ADHD diagnosis reporting disrupted sleep patterns (Corkum et al., 1998). The most

common disrupted sleep patterns in children with ADHD are sleep-disordered breathing and restless legs syndrome (see Cassoff et al., 2012 for a review), and both disorders have been associated with high levels of hyperactivity, inattention, and irritability (Chervin et al., 2002; Gottlieb et al., 2004; Lewin et al., 2002). Less severe sleep problems such as bedtime resistance, snoring, night-time waking's, and shortened sleep duration have also been associated with ADHD symptomatology (Chervin et al., 2002; Gregory and O'Connor, 2002). For example, Chervin et al. (2002) found snoring to be consistently linked with inattention and hyperactivity, while Gregory and O'Connor (2002) found that sleep disturbances in early childhood (age 4years) predicted attention problems in adolescence (age 15 years). Moreover, sleep restriction studies indicate that sleep disruptions worsen ADHD symptomatology (Fallone et al., 2005). However, treatment of disrupted sleep patterns results in significant improvements in both attention and hyperactivity and often leads to the discontinuation of ADHD stimulant medications (Fidan and Fidan, 2008; Guillemineault et al., 1981; Huang et al., 2007). As such, it is becoming quite evident that disrupted sleep patterns contribute to, or in some cases even cause, ADHD symptoms (Cassoff et al., 2012). In fact, Chervin et al. (1997) suggest that treatment of sleep disturbances (particularly sleep-related breathing disorders) would eliminate ADHD in 81percent of youth who are diagnosed with ADHD and snore and in 25 percent of all youth diagnosed with ADHD.

Given that research indicates both children with disrupted sleep patterns and children with excess weight have increased behavioral problems such as hyperactivity and inattention and/or an ADHD diagnosis, it would be reasonable to expect overweight/obese children with disrupted sleep patterns to have even more severe ADHD symptomatology. However, Rosen et al. (2004) found that the relationship between sleep-disordered breathing and hyperactivity did not change after controlling for BMI. Similarly, Gruber et al. (2012) found that sleep duration significantly predicted teacher-reported attention problems in children, again controlling for BMI, and Rudnick and Mitchell (2007) found no significant differences in ADHD symptomatology between obese youth with obstructive sleep apnea and normal weight youth with obstructive sleep apnea. Together, these studies indicate that disrupted sleep patterns account for ADHD symptomatology above and beyond body weight and highlight the need to account for sleep problems when examining the link between obesity and ADHD symptoms.

A biopsychosocial model for integrating sleep into models of ADHD and obesity

Engel's (1980) biopsychosocial model is a dynamic and interactional view of human functioning, stating that the presence of a disease or illness does not manifest simply from biological mechanisms. Rather, to fully understand the manifestation of a disease, one must consider the variety of factors at the biological, psychological, and social levels. Moreover, consideration of factors at all these levels is necessary for the most holistic and effective treatment of the disease in question. The biopsychosocial model, therefore, provides a logical framework for understanding how sleep problems may implicate the relationship between ADHD symptomatology and obesity. As outlined below, sleep problems impact the biological, psychological, and social aspects of both ADHD symptomatology and obesity. An in-depth examination of this model will illustrate how imperative it is for future research to recognize and empirically examine the role sleep has in the link between obesity and ADHD symptomatology.

Biological level

Numerous factors in the biological domain link disrupted sleep patterns to obesity and ADHD symptomatology (see Figure 1), including those at hormonal, neurological, and structural levels. First, excess adiposity is associated with reduced bioavailability of leptin, a hormone that both suppresses appetite and stimulates respiration (Aygun et al., 2005; Tankersley et al., 1998). Leptin resistance has been associated with reduced hypercapnic responses (e.g. increased breathing in an effort to increase access to oxygen), is implicated in mechanisms involved in hypoventilation, and appears to greatly impact respiration in obese individuals, especially during sleep (Polotsky et al., 2004; Tankersley et al., 1998). Moreover, the relationship between excess adiposity and respiratory problems is likely cyclical, given that (1) respiratory problems during sleep interfere with sleep duration, (2) short-sleep duration is associated with a reduction in leptin levels, and (3) low levels of leptin are associated with increased appetite (Taheri et al., 2004).

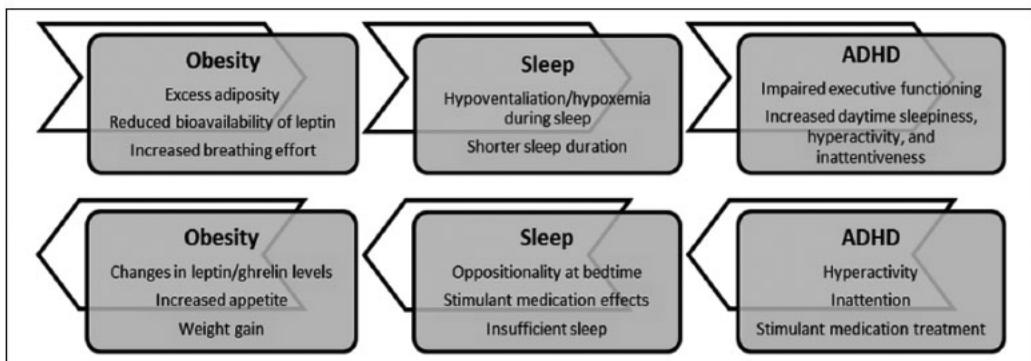


Figure 1. Potential mediating roles of sleep in the relationship between obesity and ADHD symptomatology.

ADHD: attention deficit hyperactivity disorder.

Respiratory problems during sleep, especially during childhood, also result in possible neuronal injury, specifically in the hippocampus, right frontal cortex, and pre-frontal cortex (Halbower et al., 2006). When airflow is intermittently obstructed while sleeping (i.e. hypoxemia) during key developmental periods (specifically the period in which there is surge of myelination in the pre-frontal cortex), injury to neuronal cells occurs, which may lead to dysfunction in pre-frontal regions and later manifest as behavioral dysfunction, much like that exhibited by those with ADHD (Beebe and Gozal, 2002; Rosen et al., 2004). Moreover, sleep restriction studies indicate that the functions most impacted by the sleep deprivation are those controlled by the pre-frontal cortex (e.g. executive functions) (Herscovitch et al., 1980; Randazzo et al., 1998). Specifically, sleep restriction effects dopaminergic activity (i.e. decreases dopamine levels and activity), which negatively impacts performance on attention tasks (Lal et al., 1981). Indeed, a deficiency in dopamine and dopamine receptors is hypothesized to be one etiological factor associated with the development of ADHD and is thus the primary target of medications used to ADHD (Vles et al., 2003). Taken together, substantial evidence points to the possibility that obesity may trigger sleep-related biological changes, which, in turn, contribute to ADHD-related symptoms.

Psychological level

As noted, sleep deprivation negatively impacts psychological processes (see Figure 1) such as executive functions, attention, and inhibitory control, and thus, mimics or exacerbates symptoms associated with ADHD (Chervin et al., 2002; Gottlieb et al., 2004; Lewin et al., 2002). Indeed, children with habitual snoring perform poorly on executive function measures assessing the ability to problem solve and develop future-oriented goals (Gozal et al., 2001). Moreover, both Gruber et al. (2012) and Sadeh et al. (2003) indicate that sleep deprivation results in significantly poorer performance on neurobehavioral tasks often used to assess inattention and impulsivity. Other research also indicates that disrupted sleep patterns are associated with attention deficits (Sadeh et al., 2002).

Sadeh et al. (2002) further suggest that poor sleep quality (i.e. fragmented sleep) is associated with behavioral inhibition problems. This is especially relevant in light of the association between sleep and an ADHD diagnosis. In fact, Russell Barkley, a leading ADHD researcher, posits that behavioral inhibition is necessary for higher executive control functions to both develop and later express (Barkley, 1977a, 1977b). He further suggests that the core problem in ADHD is impulsivity, which is the inability to inhibit behavioral responses. As such, it is evident that disrupted sleep patterns have far-reaching effects on neurocognitive processes associated with ADHD symptomatology. Given the documented connection between obesity and sleep problems, and the association between sleep problems and such psychological factors underlying ADHD, the mediating role of sleep in the obesity-ADHD relationship needs to be considered.

Social and Environmental Level

The behavior of children with disrupted sleep patterns appears to correspond and interact with the neurocognitive abnormalities (i.e. behavioral inhibition deficits and inattention) present in this population. Specifically, numerous studies indicate that hyperactive behavior and impulsivity are associated with disrupted sleep patterns, both of which are keystone behaviors of ADHD. A study examining the behavioral correlates of sleep-disordered breathing found that both snoring and other sleep-disordered breathing symptoms were associated with hyperactive behavior (Chervin et al., 2002). Likewise, Ali et al. (1994) indicate that among habitual snorers, the risk for daytime sleepiness (which often manifests as ADHD behaviors) is sixfold, while the risk for hyperactive behaviors is nearly threefold.

These findings are further strengthened by a twin study that examined the etiological factors associated with sleep and behavioral problems (Gregory et al., 2004). Gregory and colleagues found that sleep problems in early childhood (3-4 years old) predicted behavior problems (e.g. hyperactivity) in middle childhood (7 years old), and that genetic and non-shared environmental factors were less influential in this relationship than shared environmental factors. The researchers suggested that psychosocial factors, such as socioeconomic status or stress, may be at the root of these problems. Regardless, one such factor that does not seem to influence this relationship is body weight. In addition to the number of aforementioned studies that did not find an association between BMI and ADHD symptomatology when using it as a covariate in the examination of sleep problems and ADHD symptomatology, Paavonen et al. (2009) also found that sleep difficulties were associated with hyperactive behavior after controlling for BMI.

Environmental factors, such as obesogenic environments and strained peer relationships, may strengthen the mediating role of sleep in the relationships between

obesity and ADHD. Obesogenic environments are those in which an abundance of sedentary activities lead to a positive caloric balance (Chaput et al., 2011b). One such sedentary behavior that has stimulated much research is television viewing. The relationship between television viewing and obesity is well-established (e.g. Ekelund et al., 2006) and it is known that children with a television in their bedroom are more likely to be overweight than children without (Adachi-Mejia et al., 2007). Research further indicates that children with a television in their room also suffer from disrupted sleep (Van den Bulck, 2004). In fact, a number of studies (Dworak et al., 2007; Li et al., 2007; Owens et al., 1999) have found that having a television in the bedroom is associated with various disrupted sleep patterns. As aforementioned, short-sleep duration and similar sleep disturbances are associated with hyperactivity and other ADHD symptomatology (Touchette et al., 2007). Thus, it is possible that the role of sleep in the apparent relationship between obesity and ADHD symptomatology is strengthened by sedentary behaviors such as television viewing.

Weight-related teasing and other forms of emotional stress may also strengthen the mediating role of sleep. In fact, research indicates that short-sleep duration in obese individuals is accounted for, in part, by emotional stress (Vgontzas et al., 2008). Indeed, children who report being bullied also report sleep difficulties and bed-wetting (Williams et al., 1996). Given that overweight and obese children experience greater peer problems (especially weight-related teasing) (Hayden-Wade et al., 2005), such relational difficulties may contribute to more sleep disturbances, which may subsequently manifest as or exacerbate ADHD symptomatology. Although research has yet to examine such relationships, this appears to be a fruitful area of study.

The research presented above supports the hypothesis that the relationship between obesity and ADHD symptomatology may be largely accounted for by disrupted sleep patterns and factors that contribute to disrupted sleep patterns in children. However, studies examining the link between obesity and ADHD symptomatology have not controlled for disrupted sleep patterns and thus, empirical examination of this potential mechanism is currently lacking. Indeed, ample evidence suggests that multiple factors associated with disrupted sleep patterns in each of the biopsychosocial domains contributes to and accounts for the apparent relationship between obesity and ADHD symptomatology; however, studies comprehensively testing these sleep factors as mediators of the obesity-ADHD relationship are needed.

Clinical and Research Implications

Overlooking disrupted sleep patterns as an integral component of the relationship between obesity and ADHD symptomatology may have important treatment implications. As such, disrupted sleep patterns should be assessed regularly to best understand a child's presenting problems and to subsequently best tailor treatment. For example, for children presenting to treatment with comorbid obesity and ADHD, treating sleep problems may have beneficial effects for both conditions by (1) improving ADHD symptoms resulting from suboptimal sleep and (2) potentially assisting in more effective weight management, as improved sleep patterns promote health behaviors such as diet and physical activity (Chaput, 2011a). For children presenting to treatment with ADHD symptoms (but without comorbid obesity), it may be more helpful to treat the sleep problems (e.g. Fidan and Fidan, 2008) than to provide the standard treatment for ADHD (i.e. stimulant medication). Stimulant medications could exacerbate the child's existing sleep problems and, as a result, possibly undermine the treatment of ADHD symptoms (see Konofal et al., 2010 for

a review). Finally, for children presenting to treatment with obesity only, addressing sleep problems is appropriate both in the context of weight management and in the prevention of the development of attention-related issues, which could develop from chronically unaddressed sleep problems.

Clearly, more research is needed to delineate the relationships among sleep, obesity, and ADHD symptoms. Studies are needed to examine the relationship between obesity and ADHD symptoms, accounting for disrupted sleep patterns. In such studies, the use of a combination of objective measures of sleep (e.g. polysomnography, actigraphy) in addition to parent- and or self-report of sleep problems is recommended. ADHD subtypes (i.e. inattentive, hyperactive, and combination) should also be clearly specified and examined separately in order to best explicate the relationship between sleep, obesity, and ADHD. Moreover, some existing studies assessed for the presence of an ADHD diagnosis/symptomatology without administering validated screeners or conducting standardized clinical interviews, while others used vague terms – such as “ADHD tendency” – to define ADHD. As such, future studies should take greater care in defining and measuring the presence of ADHD in order to enhance the methodological rigor of their studies and the interpretability of their results. Likewise, much of the existing research examining the link between ADHD and obesity uses BMI as an indicator of adiposity. Though, BMI-for-age is a reasonably accurate method to detect obesity in children aged 2–19 years (Mei et al., 2002), future studies could use other methods in conjunction with BMI, such as skin-fold thickness or dual-energy X-ray absorptiometry, to more thoroughly and accurately assess obesity.

Studies examining the links between obesity, sleep, and ADHD should be designed in a manner that would allow for one or both of following questions to be answered: (1) to what extent does sleep duration, efficiency, or problems (e.g. snoring, limb movements, sleep apnea) mediate the relationship between obesity and ADHD symptoms and/or (2) does the prevalence of ADHD symptoms and comorbid obesity differ between children with diagnosed sleep problems and children without diagnosed sleep problems? Research is also needed on interventions addressing sleep problems in children who are obese and/or exhibit ADHD symptomatology. Interventions addressing sleep problems for children with both ADHD and comorbid obesity, ADHD only, and obesity only should be developed and evaluated, with a specific emphasis on the impact such interventions have on both ADHD symptomatology and weight management. Such interventions should be compared to standard methods of ADHD treatment and weight management, such as medication and/or behavioral treatment.

Conclusion

The relationship between ADHD and obesity in children has received considerable attention in recent years. However, the literature currently overlooks the potential causal and maintaining role that sleep problems may play in this relationship. Using a biopsychosocial framework, this article examined how sleep problems impact the biological, psychological, and social aspects of both ADHD symptomatology and obesity. Existing evidence regarding the impact that sleep has on ADHD symptomatology and obesity separately illustrates the imperative need for future research and clinical practice to recognize and empirically examine the role sleep has in the link between obesity and ADHD symptomatology. Such research may have important treatment implications for ADHD and comorbid obesity.

Declaration of conflicting interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/ or publication of this article.

Funding

The author(s) received no financial support for the research, authorship, and/ or publication of this article.

References

- Adachi-Mejia AM, Longacre MR, Gibson JJ, et al. (2006) Children with a TV in their bedroom at higher risk for being overweight. *International Journal of Obesity* 31(4): 644–651.
- Agranat-Meged AN, Deitcher C, Goldzweig G, et al. (2005) Childhood obesity and attention deficit/hyperactivity disorder: A newly described comorbidity in obese hospitalized children. *International Journal of Eating Disorders* 37(4): 357–359.
- Ali NJ, Pitson D and Stradling JR (1994) Natural history of snoring and related behaviour problems between the ages of 4 and 7 years. *Archives of Disease in Childhood* 71(1): 74–76.
- American Psychiatric Association (2013) *Diagnostic and Statistical Manual of Mental Disorders* (5th edn). Arlington, VA: American Psychiatric Publishing.
- Aygun AD, Gungor S, Ustundag B, et al. (2005) Proinflammatory cytokines and leptin are increased in serum of prepubertal obese children. *Mediators of Inflammation* 3: 180–183.
- Barkley RA (1997a) *ADHD and the Nature of Self-Control*. New York: Guilford Press.
- Barkley RA (1997b) Behavioral inhibition, sustained attention, and executive functions: Constructing a unifying theory of ADHD. *Psychological Bulletin* 121: 65–94.
- Beebe DW and Gozal D (2002) Obstructive sleep apnea and the prefrontal cortex: Towards a comprehensive model linking nocturnal upper airway obstruction to daytime cognitive and behavioral deficits. *Journal of Sleep Research* 11(1): 1–16.
- Bixler EO, Vgontzas AN, Lin HM, et al. (2009) Sleep disordered breathing in children in a general population sample: Prevalence and risk factors. *Sleep* 32(6): 731–736.
- Braet C, Claus L, Verbeken S, et al. (2007) Impulsivity in overweight children. *European Child & Adolescent Psychiatry* 16(8): 473–483.
- Cappuccio FP, Taggart FM, Kandala NB, et al. (2008) Meta-analysis of short sleep duration and obesity in children and adults. *Sleep* 31(5): 619–626.
- Carey WB, Hegvik RL and McDevitt SC (1988) Temperamental factors associated with rapid weight gain and obesity in middle childhood. *Journal of Developmental and Behavioral Pediatrics* 9: 316–327.
- Cassoff J, Wiebe ST and Gruber R (2012) Sleep patterns and the risk for ADHD: A review. *Nature* 4: 73–80.
- Chaput JP (2011a) Short sleep duration promoting overconsumption of food: A reward-driven eating behavior? *Sleep* 33(9): 1135–1136.
- Chaput JP, Klingenberg L, Astrup A, et al. (2011b) Modern sedentary activities promote overconsumption of food in our current obesogenic environment. *Obesity Reviews* 12: e12–e20.
- Chervin RD, Bassetti C, Ganoczy DA, et al. (1997) Pediatrics and sleep symptoms of sleep disorders, inattention, and hyperactivity in children. *Sleep* 20(12): 1185–1192.
- Chervin RD, Archbold KH, Dillon JE, et al. (2002) Inattention, hyperactivity, and symptoms of sleep-disordered breathing. *Pediatrics* 109(3): 449–456.
- Corkum P, Tannock R and Moldofsky H (1998) Sleep disturbances in children with

- attention- deficit/hyperactivity disorder. *Journal of the American Academy of Child and Adolescent Psychiatry* 37(6): 637–646.
- Cserjési R, Molnár D, Luminet O, et al. (2007) Is there any relationship between obesity and men- tal flexibility in children? *Appetite* 49: 675–678.
- Dworak M, Schierl T, Bruns T, et al. (2007) Impact of singular excessive computer game and tel- evision exposure on sleep patterns and memory performance of school-aged children. *Pediatrics* 120(5): 978–985.
- Ekelund U, Brage S, Froberg K, et al. (2006) TV viewing and physical activity are independently associated with metabolic risk in children: The European Youth Heart Study. *PLoS Medicine* 3(12): e488.
- Engel GL (1980) The clinical application of the biopsychosocial model. *American Journal of Psychiatry* 137(5): 535–544.
- Erermis S, Cetin N, Tamar M, et al. (2004) Is obesity a risk factor for psychopathology among adoles- cents? *Pediatrics International* 46(3): 296–301
- Fallone G, Acebo C, Seifer R, et al. (2005) Experimental restriction of sleep opportunity in children: Effects on teacher ratings. *Sleep* 28(12): 1561–1567.
- Faraone SV, Biederman J, Monuteaux M, et al. (2005) Long-term effects of extended-release mixed amphetamine salts treatment of attention- deficit/ hyperactivity disorder on growth. *Journal of Child and Adolescent Psychophar- macology* 15(2): 191–202.
- Fidan T and Fidan V (2008) The impact of adenoton- sillectomy on attention-deficit hyperactivity and disruptive behavioral symptoms. *The Eurasian Journal of Medicine* 40(1): 14–17.
- Gottlieb DJ, Chase C, Vezina RM, et al. (2004) Sleep- disordered breathing symptoms are associated with poorer cognitive function in 5-year-old chil- dren. *The Journal of Pediatrics* 145(4): 458–464.
- Gozal D, Holbrook CR, Mehl RC, et al. (2001) Correlation analysis between NEPSY bat- tery scores and respiratory disturbance index in snoring 6-year old children: a preliminary report. *Sleep* 24: A126. .
- Gozal D, Simakajornboon N, Holbrook CR, et al. (2006) Secular trends in obesity and parentally reported daytime sleepiness among children referred to a pediatric sleep center for snor- ing and suspected sleep-disordered breathing (SDB). *Sleep* 29: A74.
- Gregory AM and O'Connor TG (2002) Sleep prob- lems in childhood: A longitudinal study of developmental change and association with behavioral problems. *Journal of the American Academy of Child and Adolescent Psychiatry* 41(8): 964–971.
- Gregory AM, Eley TC, O'Connor TG, et al. (2004) Etiologies of associations between childhood sleep and behavioral problems in a large twin sample. *Journal of the American Academy of Child and Adolescent Psychiatry* 43(6): 744–751.
- Gruber R, Michaelsen S, Bergmame L, et al. (2012) Short sleep duration is associated with teacher- reported inattention and cognitive problems in healthy school-aged children. *Nature and Science of Sleep* 4: 33–40.
- Guilleminault C, Korobkin R and Winkle R (1981) A review of 50 children with obstructive sleep apnea syndrome. *Lung* 159(1): 275–287.
- Halbower AC, Degaonkar M, Barker PB, et al. (2006) Childhood obstructive sleep apnea asso- ciates with neuropsychological deficits and neu- ronal brain injury. *PLoS Medicine* 3(8): e301.
- Hayden-Wade HA, Stein RI, Ghaderi A, et al. (2005) Prevalence, characteristics, and correlates of teasing experiences among overweight children vs. non-overweight peers. *Obesity Research* 13(8): 1381–1392.

- Herscovitch J, Stuss D and Broughton R (1980) Changes in cognitive processing following short-term cumulative partial sleep deprivation and recovery oversleeping. *Journal of Clinical and Experimental Neuropsychology* 2(4): 301–319.
- Holtkamp K, Konrad K, Müller B, et al. (2004) Overweight and obesity in children with attention-deficit/hyperactivity disorder. *International Journal of Obesity* 28(5): 685–689.
- Horner RL, Mohiaddin RH, Lowell DG, et al. (1989) Sites and sizes of fat deposits around the pharynx in obese patients with obstructive sleep apnea and weight matched controls. *European Respiratory Journal* 2(7): 613–622.
- Huang YS, Guilleminault C, Li HY, et al. (2007) Attention-deficit/hyperactivity disorder with obstructive sleep apnea: A treatment outcome study. *Sleep Medicine* 8(1): 18–30.
- Hubel R, Jass J, Marcus A, et al. (2006) Overweight and basal metabolic rate in boys with attention-deficit/hyperactivity disorder. *Eating and Weight Disorders: EWD* 11(3): 139–146.
- Kalra M, Inge T, Garcia V, et al. (2012) Obstructive sleep apnea in extremely overweight adolescents undergoing bariatric surgery. *Obesity Research* 13(7): 1175–1179.
- Konofal E, Lecendreux M and Cortese S (2010) Sleep and ADHD. *Sleep Medicine* 11(7): 652–658.
- Korner J and Leibel RL (2003) To eat or not to eat— How the gut talks to the brain. *New England Journal of Medicine* 349(10): 926–928.
- Lal S, Thavundayil J, Nair NPV, et al. (1981) Effect of sleep deprivation on dopamine receptor function in normal subjects. *Journal of Neural Transmission* 50(1): 39–45.
- Lam LT and Yang L (2007) Overweight/obesity and attention deficit and hyperactivity disorder tendency among adolescents in China. *International Journal of Obesity* 31(4): 584–590.
- Lewin DS, Rosen RC, England SJ, et al. (2002) Preliminary evidence of behavioral and cognitive sequelae of obstructive sleep apnea in children. *Sleep Medicine* 3(1): 5–13.
- Li S, Jin X, Wu S, et al. (2007) The impact of media use on sleep patterns and sleep disorders among school-aged children in China. *Sleep* 30(3): 361–367.
- Lumeng JC, Gannon K, Cabral HJ, et al. (2003) Association between clinically meaningful behavior problems and overweight in children. *Pediatrics* 112(5): 1138–1145.
- Maayan L, Hoogendoorn C, Sweat V, et al. (2011) Disinhibited eating in obese adolescents is associated with orbitofrontal volume reductions and executive dysfunction. *Obesity* 19(7): 1382–1387.
- Mallory GB and Beckerman RC (1992) Relationships between obesity and respiratory control abnormalities. In: Beckerman RC, Brouillette RT and Hunt CE (eds) *Respiratory Control Disorders in Infants and Children*. Baltimore, MD: Williams & Wilkins, pp. 342–351.
- Mallory GB, Fiser DH and Jackson R (1989) Sleep-associated breathing disorders in morbidly obese children and adolescents. *The Journal of Pediatrics* 115(6): 892–897.
- Marcus CL, Curtis S, Koerner CB, et al. (1998) Evaluation of pulmonary function and polysomnography in obese children and adolescents. *Pediatric Pulmonology* 21(3): 176–183.
- Mei Z, Grummer-Strawn LM, Pietrobelli A, et al. (2002) Validity of body mass index compared with other body-composition screening indexes for the assessment of body fatness in children and adolescents. *The American Journal of Clinical Nutrition* 75(6): 978–985.
- Noble EP (2003) D2 dopamine receptor gene in psychiatric and neurologic disorders and its phenotypes. *American Journal of Medical Genetics. Part B: Neuropsychiatric Genetics*

- 116(1): 103–125.
- Owens J, Maxim R, McGuinn M, et al. (1999) Television-viewing habits and sleep disturbance in school children. *Pediatrics* 104(3): e27.
- Paavonen EJ, Rääkkönen K, Lahti J, et al. (2009) Short sleep duration and behavioral symptoms of attention-deficit/hyperactivity disorder in healthy 7-to 8-year-old children. *Pediatrics* 123(5): e857–e864.
- Polotsky VY, Smaldone MC, Scharf MT, et al. (2004) Impact of interrupted leptin pathways on ventilatory control. *Journal of Applied Physiology* 96(3): 991–998.
- Poston WS, Ericsson M, Linder J, et al. (1998) D4 dopamine receptor gene exon III polymorphism and obesity risk. *Eating and Weight Disorders: EWD* 3(2): 71–77.
- Randazzo AC, Muehlbach MJ, Schweitzer PK, et al. (1998) Cognitive function following acute sleep restriction in children ages 10–14. *Sleep* 21(8): 861–868.
- Redline S, Tishler PV, Schluchter M, et al. (1999) Risk factors for sleep-disordered breathing in children associations with obesity, race, and respiratory problems. *American Journal of Respiratory and Critical Care Medicine* 159(5): 1527–1532.
- Rosen CL, Storfer-Isser A, Taylor HG, et al. (2004) Increased behavioral morbidity in school-aged children with sleep-disordered breathing. *Pediatrics* 114(6): 1640–1648.
- Rudnick EF and Mitchell RB (2007) Behavior and obstructive sleep apnea in children: Is obesity a factor? *The Laryngoscope* 117(8): 1463–1466.
- Sadeh A, Gruber R and Raviv A (2002) Sleep, neu- robehavioral functioning, and behavior prob- lems in school-age children. *Child Development* 73(2): 405–417.
- Sadeh A, Gruber R and Raviv A (2003) The effects of sleep restriction and extension on school- age children: What a difference an hour makes. *Child Development* 74(2): 444–455.
- Schwartz MW, Woods SC, Porte D, et al. (2000) Central nervous system control of food intake. *Nature* 404: 661–671.
- Taheri S, Lin L, Austin D, et al. (2004) Short sleep duration is associated with reduced leptin, ele- vated ghrelin, and increased body mass index. *PLoS Medicine* 1(3): e62.
- Tankersley CG, O'Donnell C, Daood MJ, et al. (1998) Leptin attenuates respiratory compli- cations associated with the obese phenotype. *Journal of Applied Physiology* 85(6): 2261–2269.
- Tauman R and Gozal D (2006) Obesity and obstruc- tive sleep apnea in children. *Paediatric Respiratory Reviews* 7(4): 247–259.
- Touchette É., Petit D, Séguin JR, et al. (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, com- puter 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.
- Vgontzas AN, Lin HM, Papaliaga M, et al. (2008) Short sleep duration and obesity: The role of emo- tional stress and sleep disturbances. *International Journal of Obesity* 32(5): 801–809.
- Vles JSH, Feron FJM and Hendriksen JGM (2003) Dopamine receptor and transporter system in children with attention deficit hyperkinetic dis- order (ADHD). *Neuropediatrics* 34: 77–80.
- Waring ME and Lapane KL (2008) Overweight in children and adolescents in relation to attention- deficit/hyperactivity disorder: Results from a national sample. *Pediatrics* 122(1): e1–e6.
- White DP, Lombard RM, Cadieux RJ, et al. (1985) Pharyngeal resistance in normal humans: Influence of gender, age, and obesity. *Journal of Applied Physiology* 58(2): 365–371.