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Original Article

# Prevalence of insomnia symptoms in a general population sample of young children and preadolescents: gender effects ☆

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ABSTRACT

*Objective:* Our population-based study examined the prevalence of insomnia symptoms and its sociodemographic, subjective, and polysomnographic (PSG) sleep risk factors in young and preadolescent children.
*Methods:* We performed a cross-sectional study of 700 children, ages 5–12 years who underwent a 9-h PSG and parent-completed sleep and development questionnaires (Penn State Child Cohort). Insomnia symptoms were defined as parent report of difficulty falling or staying asleep and sleep-disordered breathing (SDB) as an apnea hypopnea index of $\geqslant 1$.
*Results:* The prevalence of insomnia symptoms was 19.3% and did not significantly change (20.2%) when children with SDB were excluded. A significant interaction between gender and age revealed that the prevalence of insomnia symptoms was highest in girls ages 11 to 12 years (30.6%). This gender difference was not associated with significant differences between girls and boys ages 11–12 years in anxiety and depressive symptoms. In contrast girls ages 11–12 years with insomnia symptoms, but not boys of the same group, demonstrated clinically significant PSG sleep disturbances compared to those without insomnia symptoms.
*Conclusions:* These data suggest that one out of five young children and preadolescents of the general population have insomnia symptoms. Importantly, the prevalence of insomnia symptoms peaks in girls ages 11 to 12 years and is associated with objective sleep disturbances which may be related to hormonal changes associated with the onset of puberty rather than anxiety and depression.
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## 1. Introduction

There is increasing recognition that sleep disturbances are important factors in child development. When children do not get enough sleep, aspects of their physical, emotional, cognitive, and social development are negatively affected and can impair both the parent and child's daytime functioning. It has been reported that approximately 40% of all children experience sleep problems, yet compared to adults, sleep disturbances including symptoms of insomnia in childhood are poorly studied and described.

Insomnia symptoms have been investigated in several community and clinical samples of young children and adolescents but have been idiosyncratically defined with differences in inclusion criteria, which makes comparisons across studies difficult. Consequently, prevalence estimates of insomnia symptoms in childhood have varied from 4% to 41% [1–9]. Insomnia symptoms of difficulty falling (DFA) and/or staying asleep (DSA) are the most common parent-reported sleep complaints in children, but little is known about the risk factors associated with insomnia symptoms in prepubescent children, with the exception of two population-based studies in China of children ages 6–13 years [3,10]. These studies did not find age or gender effects on the prevalence of insomnia symptoms. In contrast, a few studies on the risk factors associated with insomnia symptoms in adolescents have reported an increase in the prevalence of insomnia symptoms with age, [7,11] and an increase in the prevalence of insomnia symptoms in girls following menarche [4,8]. One study [4] reported a gender and age effect, with significantly increased risk for self-report of insomnia symptoms following onset of menses in girls ages 11–14 years, and

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similar results were found in a more recent study [8], which reported a 2.75 fold increased risk for insomnia following onset of menses in girls.

Although there is substantial published literature examining insomnia symptoms and its correlates in older adolescents and adults [12–18], as well as in children with psychiatric and medical problems such as autism [19], attention-deficit/hyperactivity disorder [20], and gastrointestinal regurgitation and headaches [2], few studies have been conducted on insomnia symptoms in young children and preadolescents from the US general population, and none assessing the risk factors of body mass index (BMI), PSG sleep disturbance, and anxiety and depressive symptoms on the prevalence of insomnia symptoms and the role of excessive daytime sleepiness (EDS) and objectively measured SDB. Thus the objectives of our study were to (1) report on the prevalence of insomnia symptoms and type of insomnia complaint in a general population sample of young children and preadolescents, (2) identify differences in objective sleep disturbance as measured with PSG in children with and without insomnia and (3) assess the effect of risk factors (e.g., gender, age, BMI, anxiety, depressive symptoms) and EDS and SDB, which have been demonstrated to be important in adult populations, on the prevalence of insomnia symptoms in the Penn State Child Cohort.

## 2. Methods

### 2.1. Sample

Our study was designed in 2 phases, with the first phase designed for collecting general information from the parents about their child's sleep and behavioral patterns. In the first phase, each grade in elementary schools (kindergarten through grade 5) with approximately 1500 students were selected. To establish a representative sample, a screening questionnaire based on the survey published by Ali et al. [21] was sent home to parents of every student in these school districts. Over the course of 5 years, we assessed all 18 public elementary schools within 3 school districts. We sent home 7312 questionnaires; 5740 were returned for a 78.5% response rate. The second phase of our study was initiated each year by randomly selecting 200 children based on a stratification of grade, sex, and risk for SDB from the returned questionnaires from the current year. We studied 704 children in this phase for a total response rate of 70.4%. Four children did not complete the PSG recording; thus 700 children were included in our study for a final response rate of 70%. We contrasted the subjects who completed the PSG recordings ($n = 700$) with those who completed the phase 1 questionnaire ($n = 5040$) and were not selected for phase 2. There were no significant differences between the two groups in gender, age, grade, BMI percentile, ethnicity, or race. We established compensatory weights to obtain estimates of the original target population and adjusted for the age and gender distribution of the nation as a whole. Please refer to our previous publications for further details regarding the sampling methods used in the Penn State Child Cohort [e.g., 22]. Thus our final sample for this study consisted of 700 children. The study was approved by the Institutional Review Board of Penn State College of Medicine. Informed consent was obtained from parents of all participants and assent was obtained from all children prior to participation.

### 2.2. Sleep laboratory

During their visit in the laboratory, all subjects underwent a series of subjective and objective measurements including parent-completed questionnaires and rating scales (e.g., behavior, sleep, child development) and measurement of height and weight were recorded for each child. BMI was calculated and expressed as BMI percentiles (BMI %), adjusted for age and gender using the formula and data of the National Health and Nutrition Examination Survey Centers for disease control and prevention growth charts [23].

All children underwent a 9-h PSG with a parent present in a sound-attenuated light- and temperature-controlled room in our General Clinical Research Center. Children's bedtime and waketime approximated their typical sleep times (average 2100–2200 to 0600–0700). Each child was monitored with an infrared video and a computerized system (24 analog channel and 10 dc channel TS amplifier using Gamma software, Grass Telefactor, Inc.), including four channels of electroencephalogram, two channel bilateral electrooculogram, and chin and anterior tibial electromyogram. Respiration was assessed throughout the night by use of a thermocouple at the nose and mouth (model TCT R, Grass Telefactor, Inc), nasal pressure (MP 45–871 + 2 cm $H_2O$, Validyne Engineering Corp), and Piezo thoracic and abdominal respiratory effort electric belts (model 1312, Sleepmate). We obtained an objective estimate of snoring during the PSG by monitoring breathing sounds with a microphone attached to the throat (model 1250, Sleepmate Technologies) and a separate room microphone. All night hemoglobin oxygen saturation was obtained by pulse oximeter (model 8800, Nonin Medical) attached to the finger. A single channel electrocardiogram also was recorded. All of the PSG records were double scored in accordance with a modified version of the American Thoracic Society standards for cardiopulmonary sleep studies in children [24]. These modifications included the addition of breathing-related arousals and elimination of central apneas in the calculation of the apnea-hypopnea index (AHI). Results from the overnight PSG evaluation were used to classify the children as having or not having SDB. Obstructive apnea was defined as a cessation of airflow with a minimum duration of 5 s and an out of phase strain gauge movement. A hypopneic event was defined as a reduction of airflow of approximately 50% with an associated decrease in oxygen saturation of at least 3% or an associated breathing related arousal. Based on these data an AHI was calculated ([apnea + hypopnea]/hours of sleep). Central apneas were not included in the AHI calculation. The scoring of snoring was based on the number of snoring-related arousals counted by the technician. SDB was defined as an AHI $\geqslant$ 1 events per hour of sleep.

### 2.3. Parent rating scales

One of the key determinants of insomnia in children as defined by the *International Classification of Sleep Disorders* is difficulty falling asleep, staying asleep, or both, as reported by the parent. In our study, we defined insomnia in a similar manner. A parent completed the Pediatric Behavior Scale (PBS) [25], a 165-item rating scale which has norm-referenced T scores for several subscales including problems with sleep. Each item is scored on a 0–3 point scale, with 0 indicating no problems and 3 indicating that a behavior is very often a problem. Children were classified as having insomnia symptoms within the past 2 months when the parent reported often or very often for either has trouble falling asleep or wakes up often in the night. In addition, parents rated their child on two additional subscales from the PBS related to anxiety and depressive symptoms. The PBS has been used in several studies by our group to assess sleep problems in children with autism and attention-deficit/hyperactivity disorder, as well as in the general population [2,19,20,26].

The Pediatric Sleep Questionnaire developed by Chervin [27] was completed by a parent to assess EDS in our study population. Children were classified as having EDS when the parent reported yes for either "Does your child have a problem with sleepiness during the day?" or "Has a teacher or other supervisor commented

that your child appears sleepy during the day?" The same definition of EDS was used in several recently published studies [28–30].

### 2.4. Data analysis

The $\chi^2$ test was used for categorical data and analyses of variance were used for continuous variables between children with and without insomnia symptoms. The prevalence of insomnia symptoms was calculated in two ways: for the total sample and when excluding all children with SDB (AHI $\geqslant$ 1). Logistic regression analysis was used to examine the level of interaction between age and gender while controlling for BMI, AHI, anxious and depressive symptoms, and medical complaints [2]. Based on the results of this interaction, we compared girls and boys 11–12 years of age on key variables such as anxiety, depression, EDS, and BMI; we also assessed 9 objective sleep variables, including AHI; total sleep time in minutes; sleep latency in minutes; rapid eye movement (REM) sleep latency (minutes from sleep onset to REM sleep); sleep efficiency (time asleep divided by total time in bed); and percentages of stages 1, 2, slow-wave sleep, and REM sleep. Furthermore, we conducted gender specific analyses comparing girls ages 11–12 years with and without insomnia symptoms on objective markers of sleep and then separately for boys ages 11–12 years with and without insomnia symptoms. We accounted for the sampling probability from phase 1 to phase 2 enrolments in all the analyses to generate population level estimates and to make inferences back to the population from which the phase 2 study participants were selected. All variables were normally distributed. With the exception of 9 missing cases for the total sample on parent reported anxiety and depression symptoms, complete data were available on all variables. The significance level was set at $P < .05$. Analyses were performed using PASW software, version 20.

## 3. Results

The demographic PSG sleep characteristics and subjective report of sleep and mood problems comparing children with and without insomnia symptoms are presented in Table 1. The prevalence of insomnia symptoms was 19.3% and did not significantly change (20.2%) when children with SDB ($n = 183$) were excluded. Within insomniacs, parents reported that 44.7% had DFA, 26.0% had DSA, and 29.3% had both. There were no significant differences between girls (20.0%) and boys (18.3%) or across age groups of 5–7, 8–10, and 11–12 years (21.2%, 16.5%, and 24.3%, respectively) in the prevalence of insomnia symptoms. PSG markers of sleep suggested that children with insomnia symptoms, as previously reported [2], had significantly increased sleep latency and stage 2 sleep, and decreased slow-wave sleep and REM sleep latency compared to children without insomnia symptoms. Children with insomnia symptoms had significantly more parent-reported symptoms of anxiety and depression ($P = .001$) and EDS ($P = .001$) than children without. When children with SDB were excluded from the analysis, anxiety and depression remained significant ($P < .001$), while EDS became non significant ($P = .22$). These excluded children with SDB were significantly heavier than the remaining children without SDB (BMI percentile, 65.4 ± 28.8 vs 59.5 ± 28.9; $P = .019$).

Logistic regression analysis revealed a significant interaction between age (5–10 vs 11–12 years) and gender on the prevalence of insomnia symptoms ($P = .03$). Specifically, girls ages 11–12 years showed a significantly higher prevalence of insomnia symptoms (30.6%) compared to girls ages 5–7 (20.8%) or 8–10 years (16.3%), whereas the prevalence of insomnia symptoms in boys was similar across all age groups (i.e., 5–7 [21.9%]; 8–10 [17.3%], or 11–12 years [16.7%]) as reported in Fig. 1. The significant interaction between age and gender on the prevalence of insomnia symptoms remained the same, even after controlling for BMI, AHI, anxiety and depression, sleep efficiency, and medical complaints ($P = .01$) In contrast, a two-way analysis of variance did not show a significant interaction between gender and age on anxiety and depression scores (gender effect, $P = .40$; age effect, $P = .002$; interaction, $P = .39$) or on other key clinical factors (see Supplementary Table 1).Therefore, no gender differences were found when comparing boys and girls ages 11–12 years on anxiety and depression scores or in BMI percentile, SDB, EDS, or PSG characteristics, with the exception of girls having slightly less % stage 1 and more % REM sleep than boys (Table 2). In addition, no gender differences were found when

**Table 1**
Demographic polysomnographic sleep characteristics and subjective report of sleep and mood problems of preadolescent children with and without insomnia.

| | No insomnia ($n = 565$) | Insomnia ($n = 135$) | *P* value |
|---|---|---|---|
| *Sociodemographics* | | | |
| Age | 8.8 ± 1.8 | 8.8 ± 1.7 | .94 |
| BMI percentile | 60.9 ± 30.6 | 61.2 ± 28.7 | .84 |
| % Male | 45.5 | 48.3 | .62 |
| % Nonprofessional | 46.6 | 44.2 | .70 |
| % Minority | 19.6 | 17.9 | .98 |
| *PSG characteristics* | | | |
| SL (min) | 28.1 ± 23.5 | 33.1 ± 26.0 | .04 |
| REML | 164.2 ± 86.6 | 148.1 ± 56.6 | .04 |
| TST (min) | 453.7 ± 49.8 | 453.3 ± 44.6 | .85 |
| SE (%) | 85.9 ± 8.6 | 85.8 ± 8 | .97 |
| Stage 1 (%) | 3.5 ± 3.3 | 3.3 ± 3 | .56 |
| Stage 2 (%) | 45.6 ± 11.6 | 48.7 ± 9.7 | .002 |
| SWS (%) | 31.1 ± 11.1 | 27.5 ± 8.8 | <.001 |
| REM (%) | 19.9 ± 5.7 | 20.6 ± 5.4 | .16 |
| AHI | 0.85 ± 1.7 | 0.72 ± 1.1 | .52 |
| *Subjective sleep* | | | |
| % DFA only | – | 44.7 | – |
| % DSA only | – | 26.0 | – |
| % Both DFA & DSA | – | 29.3 | – |
| % EDS | 11.7 | 21.4 | .001 |
| *Mood* | | | |
| Anxiety (T) | 50.4 ± 11.0 | 56.9 ± 14.1 | <.001 |
| Depression (T) | 50.6 ± 12.6 | 58.5 ± 19.8 | <.001 |

Abbreviations: BMI, body mass index; SL, sleep latency; REML, REM latency; TST, total sleep time; SE, sleep efficiency; SWS, slow-wave sleep; AHI, apnea-hypopnea index; EDS, excessive daytime sleepiness; DSA, difficulty staying asleep; DFA, difficulty falling sleep.

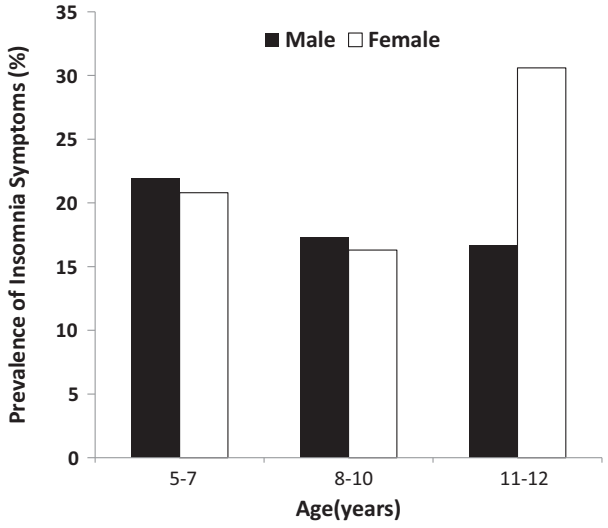


**Fig. 1.** Prevalence of insomnia by gender and age groups. Interaction between age and gender on insomnia prevalence was statistically significant ($P = .03$).

**Table 2**
Gender differences in children age 11–12 years old.

| | Girls ($n$ = 62) | Boys ($n$ = 56) | $P$ value |
|---|---|---|---|
| *PSG measures* | | | |
| SL (min) | 30.1 ± 18.2 | 31.8 ± 23.3 | .17 |
| REML | 150.9 ± 74.2 | 167.9 ± 64.3 | .54 |
| TST (min) | 456.3 ± 38.1 | 453.3 ± 52.3 | .17 |
| SE (%) | 86.8 ± 6.6 | 84.8 ± 8.3 | .11 |
| Stage 1 (%) | 2.6 ± 2.7 | 4.2 ± 3.3 | .001 |
| Stage 2 (%) | 51.0 ± 9.5 | 51.7 ± 10.0 | .51 |
| SWS (%) | 24.8 ± 8.8 | 25.0 ± 9.4 | .91 |
| REM (%) | 21.6 ± 5.3 | 19.0 ± 5.7 | .006 |
| % AHI ⩾ 1 | 25.0 | 38.7 | .12 |
| *Subjective sleep* | | | |
| % DFA only | 16.1 | 13.1 | .47 |
| % DSA only | 3.6 | 1.6 | .42 |
| % Both DFA & DSA | 10.7 | 1.6 | .03 |
| % EDS | 10.7 | 12.9 | .71 |
| *Mood* | | | |
| Anxiety (T) | 50.0 ± 12.6 | 50.3 ± 10.3 | .98 |
| Depression (T) | 52.5 ± 14.5 | 52.3 ± 19.3 | .70 |

Abbreviations: PSG, polysomnography; SL, sleep latency; REML, REM latency; TST, total sleep time; SE, sleep efficiency; SWS, slow-wave sleep; AHI, apnea-hypopnea index; EDS, excessive daytime sleepiness; DSA, difficulty staying asleep; DFA, difficulty falling sleep.

comparing boys and girls ages 5–7 or 8–10 years on any of the aforementioned variables (see Supplementary Tables 2 and 3).

Finally, gender-specific analyses showed that girls ages 11–12 years with insomnia symptoms ($n$ = 19) had significantly longer PSG sleep latency (40.4 ± 22.9 vs 24.2 ± 12.5 min; $P$ < .001), lower sleep efficiency (84.3 ± 6.1 vs 88.2 ± 5.9; $P$ = .017) and a trend towards shorter sleep duration (447.0 ± 35.9 vs 461.7 ± 38.5 min; $P$ = .12) than girls ages 11–12 years without insomnia symptoms ($n$ = 43). In contrast, no significant differences were found between boys ages 11–12 years with and without insomnia symptoms on any PSG markers of sleep.

## 4. Discussion

Our study demonstrates that approximately one-fifth of young and preadolescent children from the general population have insomnia symptoms. Moreover, our study showed that the prevalence of insomnia symptoms was highest in girls aged 11–12 years and was associated with objective sleep disturbances. Interestingly, this higher prevalence in girls was not associated with increased anxiety, depression, or SDB, but with objective sleep disturbance. Our novel findings suggest that the increased prevalence of insomnia symptoms in girls during the peripubertal stage of development may reflect biologic and psychosocial changes associated with this developmental stage.

Consistent with previous studies [4,6], the prevalence of insomnia symptoms in our population-based cohort was 19.3%, with DFA being the most frequent type of insomnia complaint (44.7%). Overall, insomnia symptoms were associated with greater reports of anxiety and depressive symptoms which is consistent with previous studies in young children [31], older adolescents, and adults [14,31–35]. To our knowledge, our study is the first study to report on objective measures of sleep, as assessed by PSG, in a population sample of young children and preadolescents with and without insomnia symptoms. We found that children with insomnia symptoms had significantly poorer objective sleep compared to children without insomnia overall. However, the magnitudes of the differences in PSG parameters were relatively small, which suggests that some children slept objectively better than others, even within those with parent-reported insomnia symptoms. Future studies should examine the association between the degree of objective sleep disturbance and clinically meaningful outcomes (e.g., behavioral and mood problems, stress system activation, blood pressure) in young children and preadolescents with insomnia symptoms.

Importantly, we found a significant interaction between gender and age on the prevalence of insomnia symptoms. The prevalence of insomnia symptoms was 10–14% higher in girls in the peripubertal stage of development (i.e., ages 11–12 years) as compared to girls ages 5–7 (20.8%) or 8–10 years (16.3%) or to boys in any of these age groups (∼19%), a finding that is consistent with a previous study reporting a similar prevalence of insomnia symptoms in girls aged 11–14 years old (30%) [4]. In contrast, we did not find a significant interaction between gender and age on anxiety and depressive symptoms, and therefore no significant differences between boys and girls aged 11–12 years in anxiety and depression. In fact, anxiety and depressive symptoms increased at ages 8–10 years and reached a plateau regardless of gender. These data suggest that the increased prevalence of insomnia symptoms in preadolescent girls is not likely explained by an increase in anxiety and depression per se and that other factors may play a role, e.g., biologic changes associated with puberty. Consistent with this hypothesis is the fact that we found clinically significant differences in objectively measured sleep latency, sleep efficiency, and sleep duration between girls ages 11–12 years with and without insomnia symptoms, while no PSG differences were found between boys with and without insomnia symptoms in the 11- to 12-year age range. Given that the onset of puberty occurs earlier in girls than in boys, together, these findings suggest that the increased prevalence of insomnia symptoms associated with objective sleep disturbances in preadolescent girls may be driven by hormonal changes occurring at the onset of puberty [4,8], rather than anxiety and depression per se. In fact, it is likely that insomnia symptoms associated with objective sleep disturbance in girls during the peripubertal stage of development may confer a risk factor for future onset of anxiety or depression. Another possible explanation for the increase in prevalence of insomnia in girls ages 11–12 years may be partially explained by a shift in circadian preference towards eveningness [36]. However, the fact that PSG defined sleep disturbances were found in girls ages 11–12 years and that they complained of both DFA and DSA indicates that the peak in insomnia symptoms in this age group is not just as a result of delayed sleep phase preference.

Finally, our study showed a lack of association between insomnia symptoms and SDB in a population-based sample of young children and preadolescents. In fact, the prevalence of insomnia symptoms remained similar after the exclusion of children with SDB, and interestingly the association between insomnia symptoms and EDS became non significant. These findings suggest that EDS is more closely linked to SDB through its association with obesity [22,28,29] than with insomnia symptoms, as in older adolescents and adults [13,14,37].

Several limitations should be considered when interpreting the results of our study. First, the objective sleep findings should be considered within the context of limitations that include the possible impact of the first-night effect. Future population-based studies should explore the use of multiple PSG nights or less expensive objective measures of habitual sleep, e.g., actigraphy. Second, the definition of insomnia was that of symptoms rather than a disorder or syndrome, as it did not include diagnostic criteria for behavioral insomnia of childhood. Future large, epidemiologic studies should use current diagnostic criteria for childhood insomnia and its subtypes. Third, we did not present a more detailed analysis of the electroencephalogram such as spectral analysis which could aid in explaining the increased prevalence of insomnia symptoms. Lastly, we did not collect data on pubertal status, and thus were not able to examine the effects of pubertal developmental status

on insomnia symptoms. Future studies should examine if the prevalence of insomnia symptoms increases in boys at a similar peripubertal stage. Despite these limitations, our study extends the limited previous knowledge on insomnia symptoms in young children by showing that the prevalence of insomnia symptoms peaks in girls ages 11–12 years is associated with objective sleep disturbances and may be related to hormonal changes associated with the onset of puberty rather than anxiety and depression per se. Furthermore, our study provides evidence that SDB does not significantly contribute to prevalent insomnia symptoms in children.

It is likely that multiple factors in the child, parent, family, and environment interacting overtime contribute to the development and maintenance of insomnia in children. These data have important implications for primary care physicians and pediatricians who generally are aware of sleep issues in infants and toddlers, but often fail to adequately screen young school-aged and preadolescent children for sleep problems in the clinical setting [38]. Given the high prevalence of insomnia symptoms in young children, the likelihood of the symptoms to persist over time [39], and the growing evidence on the importance of sleep in childhood health, future studies need to examine the physical and mental health consequences of insomnia in children.

## Disclosure statement

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## Conflict of interest

The ICMJE Uniform Disclosure Form for Potential Conflicts of Interest associated with this article can be viewed by clicking on the following link: http://dx.doi.org/10.1016/j.sleep.2013.08.787.

## References

[1] Archbold KH, Pituch KJ, Panabi P, Chervin RD. Symptoms of sleep disturbances among children at two general pediatric clinics. J Pediatr 2002;140:97–102.
[2] Singareddy R, Moole S, Calhoun S, Vocalan P, Tsaoussoglou M, Vgontzas AN, et al. Medical complaints are more common in young school aged children with parent reported insomnia symptoms. J Clin Sleep Med 2009;5:549–53.
[3] Zhang J, Li AM, Kong APS, Lai KYC, Tang NLS, Wing YK. A community based study of insomnia in Hong Kong Chinese children: prevalence, risk factors and familial aggregation. Sleep Med 2009;10:1040–6.
[4] Camhi SL, Morgan WJ, Pernisco N, Quan SF. Factors affecting sleep disturbances in children and adolescents. Sleep Med 2000;1:117–23.
[5] Fricke-Oerkermann L, Pluck J, Schredl M, Heinz K, Mitschke A, Wiater A, et al. Prevalence and course of sleep problems in childhood. Sleep 2007;30:1371–7.
[6] Paavonen EJ, Aronen ET, Moilanen I, Piha J, Rasanen E, Tamminen T, et al. Sleep problems of school aged children: a complementary view. Acta Paediatr 2000;89:223–8.
[7] Sprugt K, O'Brien LM, Cluydts R, Verleye GB, Ferri R. Odds, prevalence and predictors of sleep problems in school age normal children. J Sleep Res 2005;14:163–76.
[8] Johnson EO, Roth T, Schultz L, Breslau N. Epidemiology of DSM-IV insomnia in adolescence: lifetime prevalence, chronicity, and an emergent gender difference. Pediatrics 2006;117:e247–56.
[9] Liu X, Uchiyama M, Okawa M, Kurita H. Prevalence and correlates of self-reported problems among Chinese adolescents. Sleep 2000;23:27–34.
[10] Liu X, Sun Z, Uchiyama M, Shibui K, Kim K, Okawa M. Prevalence and correlates of sleep problems in Chinese schoolchildren. Sleep 2000;23:1053–62.
[11] Ohayon MM, Roberts RE, Zulley J, Smirne S, Priest RG. Prevalence and patterns of problematic sleep in older adolescents. J Am Acad Child Adolesc Psychiatry 2000;39:1549–56.
[12] Price VA, Coates TJ, Thorensen CE, Grinstead OA. Prevalence and correlates of poor sleep among adolescents. Am J Dis Child 1978;132:583–6.
[13] Basta M, Chrousos GP, Vela-Bueno A, Vgontzas AN. Chronic insomnia and the stress system. Sleep Med Clin 2007;2:279–91.
[14] Fernandez-Mendoza J, Vela-Bueno A, Vgontzas AN, Olavarrieta-Bernardino S, Ramos-Platon MJ, Bixler EO, et al. Nighttime sleep and daytime functioning correlates of the insomnia complaint in young adults. J Adolesc 2009;32:1059–74.
[15] Fernandez-Mendoza J, Calhoun S, Bixler EO, Pejovic S, Karataraki M, Liao D, et al. Insomnia with objective short sleep duration is associated with deficits in neuropsychological performance: a general population study. Sleep 2010;33:459–65.
[16] Liu X, Zhongtang Z, Cunxian J, Buysse D. Sleep patterns and problems among Chinese adolescents. Pediatrics 2008;121:1165–73.
[17] Vgontzas AN, Liao D, Bixler EO, Chrousos GP, Vela-Bueno A. Insomnia with objective short sleep duration is associated with a high risk for hypertension. Sleep 2009;32:491–7.
[18] Vgontzas AN, Liao D, Pejovic S, Calhoun S, Karataraki M, Bixler EO. Insomnia with objective short sleep duration is associated with type 2 diabetes: a population-based Study. Diabetes Care 2009;32:1980–5.
[19] Mayes SD, Calhoun SL. Variables related to sleep problems in children with autism. Res Autism Spectr Disord 2009;3:341–51.
[20] Mayes SD, Calhoun SL, Bixler EO, Vgontzas AN, Mahr F, Hillwig-Garcia J, et al. ADHD subtypes and comorbid anxiety, depression, and oppositional-defiant disorder: differences in sleep problems. J Pediatr Psychol 2009;34:328–37.
[21] Ali NJ, Pitson D, Stradling JR. Sleep disordered breathing: effects of adenotonsillectomy on behavior and psychological functioning. Euro J Pediatr 1996;155:56–62.
[22] Bixler EO, Vgontzas AN, Liao D, Calhoun SL, Vela-Bueno A, Fedok F, et al. Sleep disordered breathing in children from a general population sample: prevalence and risk factors. Sleep 2009;32:731–6.
[23] http://www.cdc.gov/ncdphp/growthcharts/SAS.html.
[24] American Thoracic Society. Standards and indications for cardiopulmonary sleep studies in children. Am J Respir Crit Care Med 1996;153:866–78.
[25] Lindgren SD, Koeppl GK. Assessing child behavior problems in a medical setting: development of the pediatric behavior scale. In: Prinz RJ, editor. Advances in behavioral assessment of children and families. Greenwich, CT: JAI; 1987. p. 57–90.
[26] Mayes SD, Calhoun SL, Bixler EO, Vgontzas AN. Nonsignificance of sleep relative to IQ and neuropsychological scores in predicting academic achievement. J Dev Behav Pediatr 2008;29:206–12.
[27] Chervin RD, Hedger K, Dillon JE, Pituch KJ. Pediatric sleep questionnaire (PSQ): validity and reliability of scales for sleep-disordered breathing, snoring, sleepiness, and behavioral problems. Sleep Med 2000;1:21–32.
[28] Tsaoussoglou M, Bixler EO, Calhoun SL, Chrousos GP, Sauder K, Vgontzas AN. Sleep disordered breathing in obese children is associated with prevalent EDS, inflammation, and metabolic abnormalities. J Clin Endocrinol Metab 2009;95:143–50.
[29] Calhoun SL, Vgontzas AN, Mayes SD, Fernandez-Mendoza J, Tsaoussoglou M, Basta M, et al. Prevalence and risk factors of excessive daytime sleepiness in a community sample of young children: the role of obesity, asthma, anxiety/depression and sleep. Sleep 2011;34(4):503–7.
[30] Calhoun SL, Fernandez-Mendoza J, Vgontzas AN, Mayes SD, Tsaoussoglou M, Bixler EO. Learning, attention, and conduct problems as sequelae of excessive daytime sleepiness in a general population of young children. Sleep 2012;35(5):627–32.
[31] Alfano CA, Zakem AH, Costa NM, Taylor LK, Weems CF. Sleep problems and their relation to cognitive factor, anxiety and depressive symptoms in children and adolescents. Dep Anx 2009;26:503–12.
[32] Johnson Eo, Roth T, Breslau N. The association of insomnia with anxiety disorders and depression: exploration of the direction of risk. J Psychaitr Res 2006;40(8):700–8.
[33] Roberts RE, Roberts ER, Duong HT. Chronic insomnia and its negative consequences for health and functioning in adolescents: a 12 month prospective study. J Adolesc Health 2008;42(3):294–302.
[34] Fernandez-Mendoza J, Calhoun SL, Bixler EO, Karataraki M, Liao D, Vela-Bueno A, et al. Sleep misperception and chronic insomnia in the general population: role of objective sleep duration and psychological profiles. Psychosom Med 2011;73:88–97.
[35] Vgontzas AN, Fernandez-Mendoza J, Bixler EO, Singareddy R, Shaffer ML, Calhoun SL, et al. Persistent insomnia: the role of objective short sleep duration and mental health. Sleep 2012;35:61–8.
[36] Crowley SJ, Acebo C, Carskadon MA. Sleep, circadian rhythms, and delayed phase in adolescence. Sleep Med 2007;8(6):602–12.
[37] Riedel BW, Lichstein KL. Insomnia and daytime functioning. Sleep Med Rev 2000;4:277–98.
[38] Owens JA, Spirito A, McGuinn M, Nobile C. Sleep habits and sleep disturbance in elementary school-aged children. Dev Behav Pediatr 2000;21:27–34.
[39] Zhang J, Lam SP, Li SX, Li AM, Lai KYC, Wing Y-K. Longitudinal course and outcome of chronic insomnia in Hong Kong Chinese children: a 5 year follow up study of a community based cohort. Sleep 2011;34:1395–402.