

Increased sleep latency and reduced sleep duration in children with asthma

Megan E. Jensen · Peter G. Gibson · Clare E. Collins ·
Jodi M. Hilton · Fiona Latham-Smith · Lisa G. Wood

Received: 30 August 2011 / Revised: 18 January 2012 / Accepted: 5 March 2012 / Published online: 13 March 2012
© Springer-Verlag 2012

Abstract

Study objective Sleep disturbance is reported to be more prevalent in children and adolescents with asthma than those without. However, this has not been described adequately using objective measures. The aim of this study was to objectively characterise sleep disturbance in asthmatic and non-asthmatic children and adolescents.

Megan E. Jensen is supported by an Asthma Research Centre University of Newcastle RHD Scholarship and a Hunter Medical Research Institute Donor Grant.

This work was carried out at the Hunter Medical Research Institute John Hunter Hospital, Newcastle, Australia, in partnership with the University of Newcastle, Australia.

M. E. Jensen · P. G. Gibson · L. G. Wood
Department of Respiratory and Sleep Medicine,
Hunter Medical Research Institute, John Hunter Hospital,
Locked Bag 1, Hunter Region Mail Centre,
Newcastle, NSW 2310, Australia

M. E. Jensen · P. G. Gibson · L. G. Wood (✉)
Centre for Asthma and Respiratory Diseases,
University of Newcastle,
Newcastle, NSW 2308, Australia
e-mail: lisa.wood@newcastle.edu.au

C. E. Collins
School of Health Sciences, Faculty of Health,
University of Newcastle,
Newcastle, NSW 2308, Australia

J. M. Hilton
Paediatric Respiratory and Sleep Medicine,
John Hunter Children's Hospital,
Newcastle, NSW 2305, Australia

F. Latham-Smith
Paediatric Sleep Unit,
John Hunter Children's Hospital,
Newcastle, NSW 2305, Australia

Methods A retrospective analysis of polysomnography recordings from children aged 5–17 years old, with ($n=113$) and without asthma ($n=104$), referred for a sleep study over the period 2005–2010 at the Paediatric Sleep Unit, John Hunter Children's Hospital in Newcastle, NSW Australia, was carried out.

Results Polysomnographic recordings were analysed to compare sleep quality and quantity between asthmatic and non-asthmatic children. Sleep latency was significantly longer in asthmatic children compared to controls. However, this result was significant for females only (46.2 (5.6) vs 33.2 (2.7) min, $p<0.05$). Male asthmatics had significantly shorter sleep duration (425.9 (5.4) vs 441.8 (5.4) min, $p<0.05$) than male controls.

Conclusions Sleep disturbance exists in children with asthma and manifests differently in males and females. Further investigation into the clinical implication of increased sleep latency and reduced sleep duration upon daytime functioning and lifestyle behaviours in children and adolescents with asthma is warranted.

Keywords Adolescent · Asthma · Child · Polysomnography · Sleep

Abbreviations

AI	Arousal index
BMI	Body mass index
CVD	Cardiovascular disease
ECG	Electrocardiogram
EEG	Electroencephalogram
EMG	Electromyogram
FEV ₁	Forced expiratory volume in one second
FVC	Forced vital capacity
IQR	Interquartile range
PSG	Polysomnography

PEF	Pulmonary expiratory flow
PSU	Paediatric sleep unit
RDI	Respiratory disturbance index
REM	Rapid eye movement
SEM	Standard error mean
TST	Total sleep time
TTA	Total time awake
OSA	Obstructive sleep apnea

Introduction

Asthma affects one in six children in Australia and is one of the most common chronic conditions affecting children and adolescents worldwide [1]. Asthmatic children experience more disturbed sleep than children without asthma according to parental report [2], with approximately 20 % of children estimated to be woken by their asthma [3]. Children with asthma also rated significantly higher on a self-report scale for morning tiredness and difficulty waking [4]. The nature of this sleep disturbance has not been defined well, and objective data are required to better understand sleep disturbance in this group.

Not surprisingly, risk of nocturnal awakenings in children has been reported to be higher for those with more severe asthma, a lower forced expiratory volume in 1 s, forced vital capacity and greater atopy [5]. Morning and evening peak expiratory flow measurements have also been negatively associated with frequent night awakenings [4]. In addition, increased daytime sleepiness, self-reported depression and significantly poorer parental reported mood and behavioural functioning, memory recall and learning difficulty are evident in children suffering nocturnal asthma compared to healthy controls [6]. In adults, poor sleep quality is a significant predictor of poor asthma control in both severe and non-severe asthmatics [7]. However, interestingly, children with asthma reportedly experience more disturbed sleep compared to healthy controls even when their asthma is clinically stable [8].

Poor sleep quality and reduced duration has been linked to poorer cognitive performance and executive functioning in non-asthmatic children [9, 10]. Documented disturbances in key metabolic parameters in children self-reporting reduced sleep duration may also infer risk for chronic diseases [11]. Indeed reduced sleep duration in children has been associated with a significantly increased risk of overweight and obesity later in childhood [12] and is a risk factor for cardiovascular disease (CVD) [13–15].

Sleep disturbance has not been described well using objective measures in children with asthma. The majority of studies reporting sleep habits in children have used parental or self-report [5, 8, 16–18], while others have used actigraphy [4, 16] and one has used electroencephalographic

studies [19]. Only two have used the gold-standard technique of polysomnography (PSG) to study children with asthma, and these were confounded by the presence of obesity, obstructive sleep apnea (OSA) and uncontrolled asthma within the population studied [6, 20]. Therefore, the aim of this study is to compare sleep quality and quantity in children with and without asthma, using overnight PSG data, to assess whether sleep disruption is higher in children with asthma compared to non-asthmatic children.

Methods

This was a retrospective cross-sectional study comparing sleep quality and quantity, as measured by PSG, in non-obese children and adolescents aged 5–17 years old, with and without asthma. Sleep quality may be defined by electroencephalogram (EEG) arousals with (respiratory disturbance index (RDI)) or without (arousal index, (AI)) a respiratory event, sleep efficiency (%) and rapid eye movement (REM) sleep (%). For this study, the primary outcome measure of sleep quality was sleep efficiency (%) while sleep quantity was determined by the primary outcome measure, total sleep time (TST).

Subjects

De-identified data from patients aged 5–17 years old referred during the period January 2005–December 2010 to the Paediatric Sleep Unit (PSU) at the John Hunter Children's Hospital, Newcastle NSW, Australia, were extracted from the PSU database. Patients were referred to the PSU for investigation of possible sleep disturbance including snoring, suspected apnoeas, daytime somnolence, delayed initiation and/or maintenance of sleep and daytime behavioural issues. Data were categorised into those with a diagnosis of asthma ($n=113$) and those without asthma ($n=104$). Asthma was defined as respiratory physician diagnosis as determined by written documentation in the medical notes. Demographic and anthropometric data were obtained from the PSG report and confirmed from the medical records. These data were collected as part of the quality assurance procedures in place at the PSU and considered an exempt protocol by the Hunter Area Research Ethics Committee. Exclusion criteria included obesity (BMI z -score ≥ 1.64); respiratory disease other than asthma; conditions which may cause pain or discomfort and potentially affect sleeping patterns, e.g. current eczema; mood disorders and medications known to affect sleep patterns; diagnosed sleep disorders or obstructive sleep apnoea; patients receiving continuous positive airway pressure; genetic conditions, e.g.

Trisomy 21; severe developmental delay or behavioural problems; or cranio-facial abnormalities (Fig. 1).

Polysomnography

Overnight PSG was performed on patients using the modified 10–20 application system (E-Series, Compumedics Ltd., Victoria, Australia). Monitoring included ECG; EEG; pulse rate; oximetry (SpO₂); airflow, via nasal thermistor and nasal prong pressure transducer where tolerated; transcutaneous CO₂ (tcCO₂); and diaphragmatic and respiratory effort via diaphragmatic EMG and Peizo respiratory bands located on the chest and abdomen. Scoring of polysomnographic recordings was carried out by a PSG technician using Profusion PSG 2 software (Compumedics Ltd., Victoria, Australia, 2001–2007). Sleep–wake state was scored using R & K Rules criteria for children >6 months of age [21]. EEG arousals with (RDI, RDI in REM) and without (AI) a respiratory event were scored using modified American Thoracic Society criteria [22, 23]. The use of these scoring guidelines was under the direction of the paediatric respiratory and sleep physicians. The PSU meets accreditation by the Thoracic Society of Australia and New Zealand and the Australasian Sleep Association.

Data storage and analysis

De-identified data were analysed using Intercooled Stata Release 11.1 for Windows (StataCorp, College Station, TX, USA 2009). Variables were assessed for normality

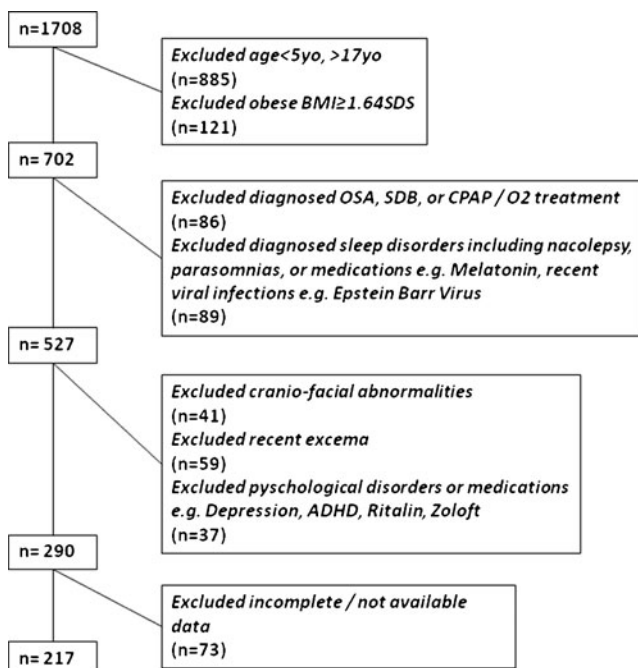


Fig. 1 Flow chart of initial studies available between the years 2005–2010 and the main exclusion criteria applied to obtain the final sample size

before performing the Students *t*-test or Wilcoxon rank sum test. Results are presented as mean (standard error mean, SEM) or median [interquartile range, IQR] and considered statistically significant if $p \leq 0.05$.

Results

The asthmatic ($n=113$) and control ($n=104$) groups were similar in age (7.4 [5.9, 8.8] vs 6.6 [5.9, 9.1] years, $p=0.46$), gender distribution and anthropometric measures ($p>0.05$) (Table 1). Valid data on medication use were available on 25 % of the asthmatic group. Of these children, 54 % were prescribed short acting beta-2 agonists (SABA) and 54 % were prescribed an inhaled corticosteroid (ICS) with an average daily dose of 90 beclomethasone equivalents.

Sleep latency was significantly longer in the asthmatic group compared to controls (35.5 [26.5, 57] vs 29.5 [20.5, 44] min, $p=0.01$) (Table 2). However, TST, total time awake (TTA) and sleep efficiency did not differ between children with and without asthma.

Gender analysis revealed that male asthmatics had significantly shorter TST (425.9 (5.4) vs 441.8 (5.4) min ($p=0.04$; Fig. 2) and lower sleep efficiency (79.9 (0.9) vs 82.9 (0.9)%, $p=0.02$) compared to male controls (Table 3). Sleep latency was significantly longer in females with asthma compared to female controls (46.2 (5.6) vs 33.2 (2.7) min, $p=0.03$) (Fig. 3). Female asthmatics also had significantly shorter TTA compared to female controls (49.9 (4.9) vs 66.7 (5.4) min, $p=0.03$) (Table 4).

There were no differences in REM latency, REM sleep, the proportion or absolute time in the sleep stages or in the number of arousals with or without a respiratory event (RDI, RDI in REM, AI) between the asthmatic and control groups or within the groups between genders ($p>0.05$).

Table 1 Characteristics of children with and without asthma referred to the sleep clinic for single overnight polysomnography

Characteristic	Asthma	Control
Number of children, <i>n</i>	113	104
Age (years), median [IQR]	7.3 [5.9, 8.8]	6.6 [5.9, 9.1]
Gender (M/F), <i>n</i>	72:41	54:50
Weight (kg), median [IQR]	26 [21.3, 33.3]	25 [20.9, 33.4]
Height (cm), median [IQR]	122.5 [116.1, 134.2]	122.1 [115.0, 132.2]
BMI z-score (SDS), mean (SEM)	0.55 (0.10)	0.56 (0.10)

SDS standard deviation score

Table 2 Results from single overnight polysomnography: asthmatics vs controls

Sleep indices	Asthma (n=113)	Control (n=104)
TST (min), mean (SEM)	430.9 (4.0)	436.9 (4.2)
Total awake time (min), mean (SEM)	58.7 (3.4)	60.3 (3.1)
Sleep efficiency (%), mean (SEM)	80.9 (0.7)	82.0 (0.7)
Sleep latency (min), median [IQR]	35.5 [26.5, 57] ^a	29.5 [20.5, 44]
Stage 1 (%), median [IQR]	0.5 [0, 1.5]	0.75 [0.1, 2.4]
Stage 2 (%), mean (SEM)	44.0 (0.8)	45.2 (0.9)
Stage 3 (%), mean (SEM)	17.1 (0.6)	16.7 (0.6)
Stage 4 (%), mean (SEM)	19.5 (0.6)	19.0 (0.7)
REM latency (min), mean (SEM)	163.7 (5.6)	172.8 (6.1)
REM sleep (%), mean (SEM)	20.5 (1.9)	17.4 (0.5)
AI (n/h), mean (SEM)	4.6 (0.3)	4.9 (0.4)
RDI (n/h), median [IQR]	0.5 [0.1, 0.9]	0.3 [0, 1.5]
RDI in REM, median [IQR]	0.8 [0, 2.5]	0.9 [0, 3.5]

TST total sleep time, REM rapid eye movement, AI arousal index, RDI respiratory disturbance index

^a Significantly different between asthma and control group ($p \leq 0.05$)

Discussion

The presented study objectively describes sleep disruption in children with and without asthma who were referred for sleep assessment. Of interest, the results suggest that gender may be an important factor in the relationship between sleep disturbance and asthma. Asthmatic children had an extended time to sleep onset compared with controls. This trend was driven by females only. Surprisingly, females with asthma spent significantly less time awake after sleep onset than

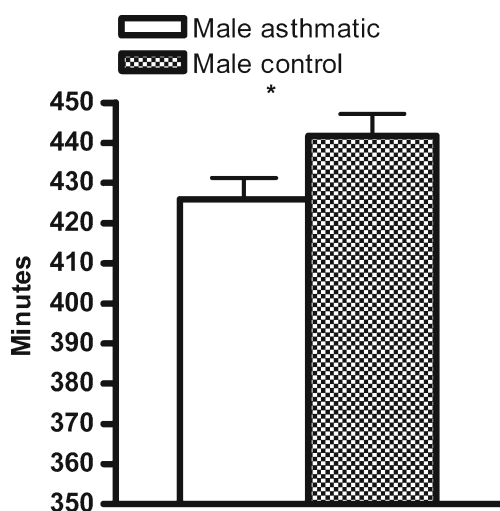


Fig. 2 Total sleep duration (minutes) in male asthmatics versus male controls measured using overnight PSG. *, significantly different between asthma and control group ($p \leq 0.05$)

Table 3 Results from single overnight polysomnography: male asthmatics vs controls

Sleep indices	Male asthma (n=72)	Male control (n=54)
TST (min), mean (SEM)	425.90 (5.35) ^a	441.81 (5.35)
Total awake time (min), mean (SEM)	63.66 (4.44)	54.33 (2.98)
Sleep efficiency (%), mean (SEM)	79.95 (0.92) ^a	82.89 (0.84)
Sleep latency (min), mean (SEM)	40.49 (2.76)	35.95 (2.97)
Stage 1 (%), median [IQR]	0.5 [0, 1.55]	0.7 [0.2, 2.6]
Stage 2 (%), mean (SEM)	43.53 (1.02)	44.82 (1.15)
Stage 3 (%), mean (SEM)	17.24 (0.79)	16.69 (0.88)
Stage 4 (%), mean (SEM)	19.16 (0.82)	18.65 (1.02)
REM latency (min), mean (SEM)	166.89 (7.08)	165.80 (7.32)
REM sleep (%), mean (SEM)	21.52 (2.76)	17.76 (0.59)
AI (n/h), mean (SEM)	4.82 (0.39)	5.01 (0.48)
RDI (n/h), median [IQR]	0.4 [0.1, 0.9]	0.35 [0.1, 1.2]
RDI in REM, median [IQR]	0.7 [0, 2.25]	1.15 [0, 3.20]

TST total sleep time, REM rapid eye movement, AI arousal index, RDI respiratory disturbance index

^a Significantly different between asthma and control group ($p \leq 0.05$)

female controls. On the other hand, asthmatic males had significantly reduced sleep duration and marginally lower sleep efficiency than males without asthma.

Previous studies using PSG data to describe sleep patterns in children with and without asthma [6, 20] were

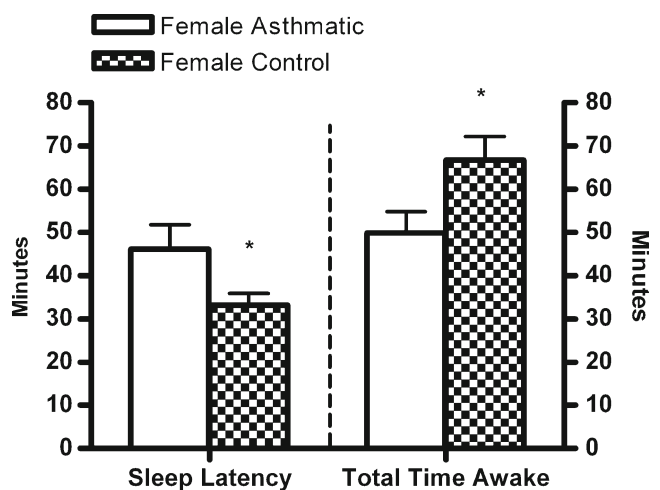


Fig. 3 Sleep onset latency (minutes) and total time awake after sleep onset (minutes) in female asthmatics versus female controls measured using overnight PSG. *, significantly different between asthma and control group ($p \leq 0.05$)

Table 4 Results from single overnight polysomnography: female asthmatics vs controls

Sleep indices	Female asthma (n=41)	Female control (n=50)
TST (min), mean (SEM)	439.71 (5.67)	425.61 (8.49)
Total awake time (min), mean (SEM)	49.87 (4.91) ^a	66.71 (5.43)
Sleep efficiency (%), mean (SEM)	82.54 (1.02)	80.95 (1.07)
Sleep latency (min), mean (SEM)	46.16 (5.59) ^a	33.16 (2.69)
Stage 1 (%), median [IQR]	0.5 [0, 1.5]	0.9 [0, 2]
Stage 2 (%), mean (SEM)	44.74 (1.16)	45.50 (1.34)
Stage 3 (%), mean (SEM)	16.92 (1.08)	16.69 (0.94)
Stage 4 (%), mean (SEM)	20.19 (0.83)	19.3 (0.88)
REM latency (min), mean (SEM)	158.13 (9.24)	180.38 (9.95)
REM sleep (%), mean (SEM)	18.57 (1.76)	17.01 (0.83)
AI (n/h), mean (SEM)	4.28 (0.50)	4.70 (0.66)
RDI (n/h), median [IQR]	0.6 [0.1, 0.8]	0.1 [0, 0.5]
RDI in REM, median [IQR]	1.2 [0, 3.2]	0.65 [0, 2.7]

TST total sleep time, REM rapid eye movement, AI arousal index, RDI respiratory disturbance index

^a Significantly different between asthma and control group ($p \leq 0.05$)

limited by their sample size, the use of self-reported asthma and the presence of confounding factors, particularly obesity and OSA. Furthermore, gender comparisons were not reported and may have overlooked important differences. One study reported a significantly increased number of awakenings and poorer sleep efficiency in children with poorly controlled nocturnal asthma compared to normative data [6]. However, it is unknown whether these differences remained following improvements in asthma control as the control group was not measured at the post-intervention stage [6]. Despite a statistically significant difference in sleep efficiency between male asthmatics and controls in our study, both groups were at the lower limit of normal and the difference between the values was not clinically relevant. In contrast to our study, a previous retrospective analysis found a significantly increased AI in asthmatic children compared to controls [20]. However, this may be attributed to the high prevalence of obesity and diagnosed OSA in their dataset. Clinical abnormalities indicative of sleep disorders, OSA or uncontrolled asthma were not identified in our dataset.

Our study demonstrated a significantly lower TST in male asthmatics compared to controls. Although the magnitude of this difference in sleep quantity between the males in our study is comparable to previous reports of sleep duration in asthmatics versus controls [6, 20], the clinical significance is unknown. An inverse association exists between sleep duration and childhood obesity [11, 24, 25], which is

particularly prominent in males [12]. Specific risk factors for weight gain, including frequent consumption of energy-dense foods and soft drink, reduced resting energy expenditure and lower levels of the appetite-suppressing hormone, adiponectin [26, 27], have been inversely associated with sleep duration in boys only, while increased media usage and reduced physical activity are reportedly higher in both male and female short sleepers [26]. Interestingly, these risk factors are highly prevalent in children with self-reported sleep duration up to 1 h below the recommended 9–10 h. Marginal reductions in sleep duration have also been linked with poorer mathematical, neurodevelopmental and behavioural test scores [28, 29]. In the presented study, sleep duration was approximated to be 7 h for all children, which is substantially below recommended levels. This indicates that these children may be vulnerable to significant lifestyle disease risk factors and poorer cognitive performance, and further investigation is required.

Contrasting previous studies, sleep onset was considerably delayed in asthmatic children compared to controls. However, this remained significant for females only, with the onset of sleep taking approximately 13 min longer in asthmatics. While the clinical relevance of increased sleep latency in children has not been assessed, a difference in sleep onset of 19 min was associated with significantly poorer cognitive performance in asthmatic versus non-asthmatic adults [30]. Increased sleep latency in non-asthmatic children has been linked with decreased physical activity, increased sedentary behaviour and poorer mental health and eating patterns [31–33]. Sleep latency reportedly increases by 3 min and decreases by 5.7 min for every respective hour of sedentary and vigorous activity [33]. It has also been suggested that delayed sleep initiation may lead to anxiety and consequent emotional eating, thereby increasing lifestyle disease risk [32]. Investigation into the clinical significance and potential health impact of delayed sleep onset in asthmatic children requires further investigation.

Our results demonstrated a shorter TTA in female asthmatics versus controls, of which the clinical significance is unknown. This may suggest that female asthmatics have difficulty initiating sleep but nil difficulty in maintaining sleep after onset. Alternatively, it is possible that the observed differences are the result of chance and multiple statistical comparisons, and as such, further investigation is warranted.

The presented study extends previous research through the use of an objective gold-standard sleep assessment technique, unique access to a large sample with documented evidence of physician-diagnosed asthma and the appropriate minimisation of potentially confounding factors. The PSG procedure (where the opportunity to sleep may be impeded by the laboratory hours and the unfamiliar environment) and

the measurement of a small time period to estimate usual sleep patterns, is a limitation common to the research and diagnostic setting. Although the acquisition of data from a referral-based population with suspected sleep disturbances is not ideal, collection of PSG data from children without clinical indication is not practical.

The aim of the presented study was to examine the effects of asthma upon sleep, and therefore patients with potentially confounding co-morbidities, such as OSA, were excluded. Indeed in adults, severe and non-severe asthma is associated with poorer sleep quality and extended sleep latency after accounting for asthma co-morbidities [7]. Being inflammatory diseases of the respiratory system, certain characteristics such as increased bronchial wall thickness and airway hyper-reactivity are reportedly common to both asthma and OSA, and a recent review suggests that an interaction effect upon sleep may exist [34, 35]. Given the co-presentation of these diseases, it is possible that asthmatic patients were excluded from our sample group due to the presence of OSA. Although an important area of research, due to the likely compounding effects of asthma and OSA upon sleep disturbance, the inclusion of subjects with OSA was deemed outside the aim of the present study.

The retrospective design of this study is the primary methodological limitation to the interpretation of results. Comprehensive characterisation of lung function, asthma control and medication use was not possible. However, it can be confirmed that all subjects with asthma were stable as, under PSU guidelines, subjects are rescheduled if currently exacerbating or unwell. Secondly, the method of statistical analysis involved multiple comparisons and it is possible that observed differences may be due to chance. Therefore, follow-up studies using a prospective design with attention to the thorough characterisation of asthma status and consideration of age and gender effects are warranted.

The presented study used an adequately characterised dataset, otherwise unavailable outside the clinical setting, to extend our knowledge of sleeping patterns in children with physician-diagnosed asthma. Results suggest that asthma in childhood may impact the quantity, rather than the quality, of sleep and provides the first indication that gender-based differences in sleep disruption may exist between children with and without asthma. Males with asthma have shorter sleep duration and females with asthma have significant difficulty initiating sleep compared to non-asthmatic children. The clinical implications of the presented findings require investigation using a prospective design that includes assessment of lifestyle factors and daytime performance, the characterisation of asthma status and medication use and the thorough consideration of gender and age effects.

Acknowledgements The authors acknowledge the work of the John Hunter Children's Hospital Paediatric Sleep Unit staff for the collection, extraction, and scoring of the polysomnography data.

Conflict of interest The authors declare that they have no conflict of interest.

References

- Masoli M, Fabian D, Holt S, Beasley R (2004) Global burden of asthma: a report prepared for the global initiative for asthma. GINA: 1-12
- Australian Centre for Asthma Monitoring (2009) Asthma in Australian children: findings from Growing Up in Australia, the longitudinal study of Australian children. Australian Institute of Health and Welfare, Canberra
- Australian Centre for Asthma Monitoring (2008) Asthma in Australia. In: AIHW asthma series no. 3. Australian Institute of Health and Welfare, Canberra.
- Sadeh A, Horowitz I, Wolach-Benodis L, Wolach B (1998) Sleep and pulmonary function in children with well-controlled, stable asthma. *Sleep* 21(4):379–384
- Strunk RC, Sternberg AL, Bacharier LB, Szefer SJ (2002) Nocturnal awakening caused by asthma in children with mild-to-moderate asthma in the childhood asthma management program. *J Allergy Clin Immunol* 110(3):395–403
- Stores G, Ellis A, Wiggs L, Crawford C, Thomson A (1998) Sleep and psychological disturbance in nocturnal asthma. *Arch Dis Child* 78(5):413–419
- Luyster F, Teodorescu M, Bleecker E, Busse W, Calhoun W, Castro M, Chung K, Erzurum S, Israel E, Strollo P, Wenzel S (2011) Sleep quality and asthma control and quality of life in non-severe and severe asthma. *Sleep Breath*. doi:10.1007/s11325-011-0616-8
- Chugh IM, Khanna P, Shah A (2006) Nocturnal symptoms and sleep disturbances in clinically stable asthmatic children. *Asian Pac J Allergy Immunol* 24(2):135–142
- Paavonen JE, Raikkonen K, Pesonen A-K, Lahti J, Komsu N, Heinonen K, Jarvenpaa A-L, Strandberg T, Kajantie E, Porkka-Heiskanen T (2010) Sleep quality and cognitive performance in 8-year-old children. *Sleep Med* 11:386–392
- Anderson B, Storfer-Isser A, Taylor HG, Rosen CL, Redline S, Anderson B, Storfer-Isser A, Taylor HG, Rosen CL, Redline S (2009) Associations of executive function with sleepiness and sleep duration in adolescents. *Pediatrics* 123(4):e701–e707
- Spruyt K, Molfese DL, Gozal D (2011) Sleep duration, sleep regularity, body weight, and metabolic homeostasis in school-aged children. *Pediatrics*. doi:10.1542/peds.2010-0497
- Chen X, Beydoun MA, Wang Y (2008) Is sleep duration associated with childhood obesity? A systematic review and meta-analysis. *Obesity* 16:265–274
- Bayer O, Neuhauser H, von Kries R, Bayer O, Neuhauser H, von Kries R (2009) Sleep duration and blood pressure in children: a cross-sectional study. *J Hypertens* 27(9):1789–1793
- Gangwisch JE, Malaspina D, Babiss LA, Opler MG, Posner K, Shen S, Turner JB, Zammit GK, Ginsberg HN, Gangwisch JE, Malaspina D, Babiss LA, Opler MG, Posner K, Shen S, Turner JB, Zammit GK, Ginsberg HN (2010) Short sleep duration as a risk factor for hypercholesterolemia: analyses of the National Longitudinal Study of Adolescent Health. *Sleep* 33(7):956–961
- Wells JC, Hallal PC, Reichert FF, Menezes AM, Araujo CL, Victora CG, Wells JCK, Menezes AMB, Araujo CLP (2008) Sleep patterns and television viewing in relation to obesity and blood

- pressure: evidence from an adolescent Brazilian birth cohort. *Int J Obes* 32(7):1042–1049
16. Kieckhefer GM, Ward TM, Tsai S-Y, Lentz MJ (2008) Nighttime sleep and daytime nap patterns in school age children with and without asthma. *J Dev Behav Pediatr* 29(5):338–344
 17. Hanson MD, Chen E (2008) The temporal relationships between sleep, cortisol, and lung functioning in youth with asthma. *J Pediatr Psychol* 33(3):312–316
 18. Tirosh E, Scher A, Sadeh A, Jaffe M, Lavie P (1993) Sleep characteristics of asthmatics in the first four years of life: a comparative study. *Arch Dis Child* 68(4):481–483
 19. Kales A, Kales JD, Sly RM, Scharf MB, Tan T-L, Preston TA (1970) Sleep patterns of asthmatic children: all-night electroencephalographic studies. *J Allergy* 46(5):300–308
 20. Ramagopal M, Scharf SM, Roberts DW, Blaisdell CJ (2008) Obstructive sleep apnea and history of asthma in snoring children. *Sleep Breath* 12:381–392
 21. Rechtschaffen A, and Kales A. (1968) A manual of standardized terminology, techniques and scoring system for sleep stages of human subjects. US Dept. of Health, Education, and Welfare, Public Health Services—National Institutes of Health, National Institute of Neurological Diseases and Blindness, Neurological Information Network, Los Angeles.
 22. Loughlin G, Brouillette R et al (1996) American Thoracic Society: standards and indications for cardiopulmonary sleep studies in children. *Am J Respir Crit Care Med* 153:866–878
 23. Thoracic Society of Australia and New Zealand (1994) Guidelines for respiratory sleep studies. TSANZ, Australia
 24. Padez C, Mourao I, Moreira P, Rosado V (2009) Long sleep duration and childhood overweight/obesity and body fat. *Am J Hum Biol* 21(3):371–376
 25. Patel SR, Hu FB (2008) Short sleep duration and weight gain: a systematic review. *Obesity* 16(3):643–653
 26. Hitze B, Bosy-Westphal A, Bielfeldt F, Settler U, Plachta-Danielzik S, Pfeuffer M, Schrezenmeir J, Monig H, Muller MJ (2009) Determinants and impact of sleep duration in children and adolescents: data of the Kiel Obesity Prevention Study. *Eur J Clin Nutr* 63(6):739–746
 27. Westerlund L, Ray C, Roos E (2009) Associations between sleeping habits and food consumption patterns among 10–11-year-old children in Finland. *Br J Nutr* 102(10):1531–1537
 28. Ng EP, Ng DK, Chan CH (2009) Sleep duration, wake/sleep symptoms, and academic performance in Hong Kong secondary school children. *Sleep & Breathing* 13(4):357–367
 29. Touchette E, Petit D, Seguin JR, Boivin M, Tremblay RE, Montplaisir JY, Touchette E, Petit D, Seguin JR, Boivin M, Tremblay RE, Montplaisir JY (2007) Associations between sleep duration patterns and behavioral/cognitive functioning at school entry. *Sleep* 30(9):1213–1219
 30. Fitzpatrick M, Engleman H, Whyte K, Deary I, Shapiro C, Douglas N (1991) Morbidity in nocturnal asthma: sleep quality and daytime cognitive performance. *Thorax* 46:569–573
 31. Forbes EE, Bertocci MA, Gregory AM, Ryan ND, Axelson DA, Birmaher B, Dahl RE, Forbes EE, Bertocci MA, Gregory AM, Ryan ND, Axelson DA, Birmaher B, Dahl RE (2008) Objective sleep in pediatric anxiety disorders and major depressive disorder. *J Am Acad Child Adolesc Psychiatr* 47(2):148–155
 32. Nguyen-Rodriguez ST, McClain AD, Spruijt-Metz D, Nguyen-Rodriguez ST, McClain AD, Spruijt-Metz D (2010) Anxiety mediates the relationship between sleep onset latency and emotional eating in minority children. *Eat Behav* 11(4):297–300
 33. Nixon GM, Thompson JM, Han DY, Becroft DM, Clark PM, Robinson E, Waldie KE, Wild CJ, Black PN, Mitchell EA, Thompson JMD, Becroft DMO (2009) Falling asleep: the determinants of sleep latency. *Arch Dis Child* 94(9):686–689
 34. Malakasioti G, Gourgoulis K, Chrousos G, Kaditis (2011) Interactions of obstructive sleep-disordered breathing with recurrent wheezing or asthma and their effects on sleep quality. *Pediatric Pulmonology* 46(11):1047–1054
 35. Sarıman N, Levent E, Çubuk R, Yurtlu Ş, Benli Aksungar F (2011) Bronchial hyperreactivity and airway wall thickening in obstructive sleep apnea patients. *Sleep Breath* 15(3):341–350