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Sleep hygiene and actigraphically evaluated sleep characteristics in children with ADHD and chronic sleep onset insomnia

KRISTIAAN B. VAN DER HEIJDEN[1,2], MARCEL G. SMITS[3] and W. BOUDEWIJN GUNNING[4]

[1]Department of Behavioural Research & Clinical Neuropsychology and Centre for Sleep and Wake Disorders, Epilepsy Centre Kempenhaeghe, Heeze, [2]Maastricht Institute of Brain & Behavior, Maastricht, [3]Centre for Sleep–Wake disorders and Chronobiology, Hospital Gelderse Vallei, Ede and [4]Department of Neurology, Epilepsy Centre Kempenhaeghe, Heeze, The Netherlands

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SUMMARY In the present study we investigated sleep hygiene and actigraphically evaluated sleep in 74 medication-naïve children, aged 6–12 years, with rigorously diagnosed attention-deficit/hyperactivity disorder (ADHD) and chronic sleep onset insomnia (ADHD-SOI) and 23 ADHD controls without insomnia (ADHD-noSOI). Between-group differences were analysed for lights out (sleep log), actigraphically evaluated sleep onset, sleep latency, total sleep duration, actual sleep time and sleep hygiene as measured with the Children's Sleep Hygiene Scale. We found a significant difference ($P < 0.001$) in mean ($\pm$SD) sleep onset between the ADHD-SOI group (21:49 $\pm$ 0:56 h) and ADHD-noSOI groups (20:41 $\pm$ 0:45 h). Sleep latency was significantly ($P < 0.001$) longer in ADHD-SOI (00:53 $\pm$ 0:25 h) compared to ADHD-noSOI (00:26 $\pm$ 0:25 h). The difference in total sleep duration between ADHD-SOI (9:42 $\pm$ 0:44 h) and ADHD-noSOI (10:09 $\pm$ 0:43 h) was not significantly different ($P = 0.18$). The group difference in actual sleep time was also not significant (8:43 $\pm$ 0:52 h in ADHD-SOI versus 9:13 $\pm$ 1:16 h; $P = 0.40$). There was no significant difference ($P = 0.17$) in mean ($\pm$SD) total sleep hygiene score between the ADHD-SOI (56.4 $\pm$ 10.5) and ADHD-noSOI groups (53.0 $\pm$ 10.6). We conclude that there were differences in sleep onset and sleep latency in ADHD children with chronic SOI and those without insomnia; however, sleep hygiene practices were similar and did not relate to sleep characteristics.

KEYWORDS attention-deficit/hyperactivity disorder, childhood insomnia, sleep hygiene

INTRODUCTION

Chronic sleep onset insomnia (SOI) often occurs in children with attention-deficit/hyperactivity disorder (ADHD) (Corkum *et al.*, 2001; Mick *et al.*, 2000; O'Brien *et al.*, 2003a,b; Owens *et al.*, 2000a; Smedje *et al.*, 2001) with reported rates of up to 28% in medication-free ADHD children (Corkum *et al.*, 1999) against 8–15% in the normal child population (Blader *et al.*, 1997; Meijer *et al.*, 2000; Owens *et al.*, 2000b). Childhood insomnia is a serious and severe disorder with a wide-ranging impact on the child's health, mood, behaviour, and cognition (Dahl, 1996; Tynjala *et al.*, 1993; Wiggs and Stores, 1999). Its daytime sequelae can be very similar to symptoms of ADHD and may therefore aggravate or even mimic ADHD (van der Heijden *et al.*, 2005a).

We recently showed that ADHD-related chronic SOI is associated with a delayed dim light melatonin onset and sleep–wake rhythm, while sleep maintenance was normal (van der Heijden *et al.*, 2005c). These typical characteristics of a circadian rhythm sleep disorder point towards an involvement of a disturbed circadian pacemaker, possibly due to clock gene disturbances (Archer *et al.*, 2003; Ebisawa *et al.*, 2001); however, the exact underpinnings are not yet known.

Since some behavioural aspects of ADHD such as hyperactivity and disorganized characteristics might be of influence on

Correspondence: Kristiaan B. van der Heijden, Department of Behavioural Research & Clinical Neuropsychology and Centre for Sleep and Wake Disorders, Epilepsy Centre Kempenhaeghe, P.O. Box 61, 5590 A.B. Heeze, The Netherlands. Tel.: +31 402279774; fax: +31 402265691; e-mail: heijdenk@kempenhaeghe.nl

the ability to fall asleep, we wanted to know the relationship of chronic SOI with sleep hygiene, which is defined as all behavioural and environmental factors that precede sleep and may interfere with sleep. It has been shown that inadequate sleep hygiene can deteriorate sleep (Brown *et al*., 2002) and sleep hygiene improving measures can increase sleep quality (Hryshko-Mullen, 2000; LeBourgeois *et al*., 2004).

In the present study we compared sleep hygiene in ADHD children with chronic SOI to that in ADHD children without sleep complaints. Our hypothesis was that ADHD children with SOI show poorer sleep hygiene measures compared to their counterparts without insomnia. None of the children in the study used psychotropic medication such as methylphenidate, clonidin, benzodiazepines to exclude a possible influence of these medicines on sleep. All children were subjected to a rigorous diagnostic evaluation of ADHD and chronic SOI to increase homogeneity of the population. Since ADHD exists in different subtypes, i.e. ADHD-inattentive, ADHD-hyperactive/impulsive, and ADHD-combined subtype, and often manifests with disruptive behaviour disorder (DBD), we also examined the relationship between sleep hygiene and these factors.

METHODS

Subjects

Between November 2001 and April 2004, 117 children (aged 6–12 years) were referred by seven Community Mental Health Institutions and three pediatric departments of non-academic hospitals in the Netherlands to an academic child psychiatry outpatient clinic (former affiliation of KH) for examination of ADHD. Children diagnosed with ADHD and SOI were invited to participate in a study on the efficacy of melatonin treatment. ADHD children without insomnia were eligible to participate in a study on the effect of methylphenidate on sleep. A further 21 children (aged 6–12 years) were recruited through advertisements in magazines for participation in one of the aforementioned studies. A total of 138 children took part in the current study.

The studies were approved by the Central Committee on Research Involving Human Subjects, conducted according to the European Guidelines for Good Clinical Research Practice in children (Committee for Proprietary Medicinal Products, 1997), and followed the 1983 revised provisions of the 1975 Declaration of Helsinki. Informed consent was obtained from the participating children's parents.

Exclusion criteria were: total IQ less than 80, pervasive developmental disorder, chronic pain, or used stimulants, melatonin, neuroleptics, benzodiazepines, clonidin, antidepressants, hypnotics, or beta blockers within 4 weeks before enrolment.

Diagnosis

Rigorous clinical diagnostic assessments were conducted on all children based on the *Diagnostic and statistical manual of mental disorders*, 4th edition (DSM-IV) (American Psychiatric Association, 1994). The following ADHD subtypes were determined using DSM-IV: (1) predominantly inattentive type (ADHD-I) assigned to children who displayed more than six inattentive symptoms, but less than six hyperactive/impulsive symptoms; (2) predominantly hyperactive/impulsive type (ADHD-HI) when children displayed more than six hyperactive/impulsive symptoms but less than six inattentive symptoms and (3) combined type (ADHD-C) for children with more than six symptoms of each set of symptoms.

Diagnostic procedures included clinical history, Diagnostic Interview Schedule for Children-Parent form (DISC-P), and the following behavioural questionnaires: Child Behaviour Checklist (Achenbach and University of Vermont Department of Psychiatry, 1991) and Teacher's Report Form (Achenbach and Achenbach, 1991). The criterion validity of the ADHD section of the DISC-P has recently been shown to be satisfactory (McGrath *et al*., 2004). Diagnostic procedures were carried out by a clinical psychologist (KH) and board-certified child and adolescent psychiatrist (BG). Additionally, the Weschler Intelligence Scale for Children-III (WISC-III) short form (Block Design, Vocabulary) was administered (duration $\pm$ 30 min) when IQ had not been assessed formerly and school performance had been poor in the last 3 years.

The definition of SOI used in the current study was one which has been used in other studies (van der Heijden *et al*., 2005b; Smits *et al*., 2001, 2003) and its criteria received extensive discussion (van der Heijden *et al*., 2005d). Children needed to meet all of the following criteria: (1) complaints of sleep onset (SO) problems by parents and/or child; (2) SO later than 20:30 hours for children at age 6, and for older children 15 min later per year; (3) sleep latency (SL) exceeding 30 min and (4) occurrence on at least 4 weekdays per week, for longer than 1 year. The diagnosis was based upon a thorough patient history, a 1-week 24-h actigraphy measurement, and a sleep hygiene questionnaire (Harsh *et al*., 2002) (see below).

Furthermore, the Dutch Sleep Disorders Questionnaire (SDQ; Centre for Sleep and Wake Disorders, Westeinde Hospital, The Hague, The Netherlands) was used to evaluate periodic limb movements disorder/restless legs syndrome (PLMS/RLS) and sleep apnea syndrome (OSAS). The SDQ is the Dutch translation and adaptation of the Sleep Disorders Questionnaire developed by Douglass *et al*. (1994) and has been proved reliable and valid (Sweere *et al*., 1998). The SDQ is a self-report questionnaire but was, in the present study, completed by parents and, where possible, in conjunction with the child. It encompasses 57 questions and has a 5-point response scale (1 = never, 2 = seldom, 3 = sometimes, 4 = often, and 5 = always).

Procedures

Sleep was evaluated using actigraphy (Actiwatch, Cambridge Neurotechnology Ltd, Cambridge, UK) worn on the non-dominant wrist 24 h a day, for 7 consecutive days. Actigraphy is a well-validated instrument to evaluate sleep and circadian

rhythm patterns in children (Littner *et al*., 2003b). Recordings of the amount of movement were made at 1-min epochs. A sleep log was completed by parents to mark lights out (when the child lies in bed, lights are dimmed, and the child is expected to go to sleep) and get-up time (time of leaving bed), and to provide supplemental data for identifying and rejecting actigraphy artefacts for example due to not wearing the device. SO was defined as the start of a period of at least 10 min of consecutively recorded immobile data following lights out; SL defined as the time from lights out until SO; total sleep duration (TSD) defined as the duration from SO until awake time; and actual sleep time (AST) as the actual amount of sleep, calculated as TSD minus estimated time awake in the period from SO until awake time. These sleep parameters were estimated from actigraphy data by a validated automatic scoring algorithm (Kushida *et al*., 2001) (Actiwatch), which compares favourable to other such packages (Chang *et al*., 1999), and subsequent manual verification.

Sleep hygiene was assessed using the Children's Sleep Hygiene Scale (CSHS) (Harsh *et al*., 2002). The CSHS comprises 25 short questions about sleep hygiene with a 6-point response scale (1 = never; 6 = always) to be completed by the parents. The scale has shown adequate internal consistency (Cronbach's alpha; $\alpha = 0.76$). The Adolescent Sleep Hygiene Scale was modified from the CSHS and has been proved valid and reliable (LeBourgeois *et al*., 2004, 2005).The CSHS was developed by the Sleep Research Laboratory of the University of Southern Mississippi and was translated into Dutch and back-translated to English by a certified translation centre.

Statistics

Reverse-coded items of the CSHS were recoded to align all responses in a positive direction (high score means poor sleep hygiene), and mean total sleep hygiene score was calculated for ADHD children with chronic sleep onset insomnia (ADHD-SOI) and ADHD children without sleep onset problems (ADHD-noSOI). For each subject, sleep parameters were averaged over 7 days.

Between-group differences (ADHD-SOI versus ADHD-noSOI) in sleep parameters and TSH were explored with General Linear Model (GLM) univariate analysis of variance with group as fixed factor and age as a covariate, or with Mann–Whitney *U*-test when data were found not to be normally distributed. Significance level was set at $P < 0.05$, two-tailed. Between-group differences in item scores were tested with Mann–Whitney *U* with adjusted significance level ($P < 0.01$, two-tailed), to account for multiple comparisons.

Relationship between TSH and sleep parameters was analysed for the total group and each group separately with linear regression analysis. Furthermore, the influence of gender, age, comorbid DBD, ADHD subtypes (ADHD-C, ADHD-I, and ADHD-HI, recoded into dummy variables), and severity of ADHD (defined as the number of ADHD symptoms shown out of nine per set of symptoms (i.e. ADHD-I and ADHD-HI)) on TSH was analysed with linear regression analysis or with Spearman's correlation for ordinal data.

Between-group (ADHD-SOI versus ADHD-noSOI) differences in severity of ADHD and in scores on the insomnia-, PLMS/RLS-, and OSAS scales of the SDQ were analysed with Mann–Whitney *U*-test. Significance level was set at $P < 0.05$, two-sided.

RESULTS

Subject characteristics

A total of 138 children were referred of which 29 (22%) were not diagnosed with ADHD, three (2%) had an IQ < 80, one (1%) used methylphenidate, and one (1%) used melatonin previously (Fig. 1). Seven (5%) children withdrew for a variety of reasons, e.g. including moving or necessity for an urgent start of therapy. The ADHD-SOI group consisted of 74 children (mean age 9.1 years ± 2.1 (SD)) and the ADHD-noSOI group of 23 children (mean = 7.9 ± 1.8 (SD)) ($P = 0.014$). Group characteristics regarding gender, age, ADHD subtype, and comorbidity are shown in Table 1.

There were no between-group differences in severity of inattentive symptoms (7.83 ± 1.49 in ADHD-SOI versus 7.33 ± 1.50 in ADHD-noSOI) ($P = 0.15$) or hyperactive/impulsive symptoms (6.52 ± 2.19 in ADHD-SOI versus 7.17 ± 2.15 in ADHD-noSOI) ($P = 0.14$). Scores on the insomnia scale of the SDQ were 2.65 ± 0.53 in ADHD-SOI and 2.06 ± 0.45 in ADHD-noSOI ($P = 0.0082$). Between-group differences in mean PLMS/RLS scores (1.61 ± 0.66 in ADHD-SOI versus 1.41 ± 0.90 in ADHD-noSOI) or OSAS scores (1.25 ± 0.33 in ADHD-SOI versus 1.27 ± 0.35 in ADHD-noSOI) were not significant ($P = 0.082$ resp. $P = 0.82$).

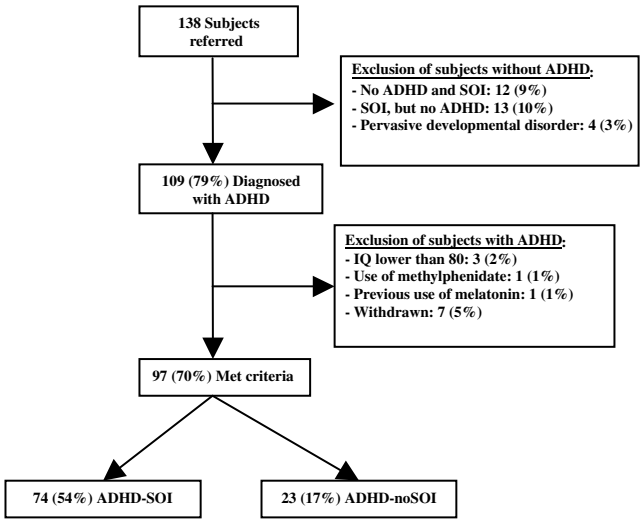

Figure 1. Trial profile. Attention-deficit/hyperactivity disorder (ADHD) children with sleep onset insomnia (ADHD-SOI) and ADHD children without insomnia (ADHD-noSOI).

Table 1 Patient characteristics for ADHD-SOI and ADHD-noSOI

	ADHD-SOI (n = *74*)	*ADHD-noSOI* (n = *23*)	*Pooled* (n = *97*)
Age, years (mean ± SD)	9.1 ± 2.1	7.9 ± 1.8	8.9 ± 2.0
Male, no. (%)	56 (76)	20 (87)	76 (78)
Subtype, no. (%)			
ADHD-C	56 (75)	19 (83)	75 (76)
ADHD-I	15 (20)	3 (13)	18 (19)
ADHD-HI	3 (4)	1 (4)	4 (4)
Comorbidity, no. (%)			
Disruptive behavioural disorder	38 (52)	13 (57)	51 (54)
Anxiety disorder	9 (12)	2 (10)	11 (11)
Affective disorder	1 (1)	1 (3)	2 (2)

ADHD, attention-deficit/hyperactivity disorder; ADHD-SOI, children with ADHD and chronic sleep onset insomnia; ADHD-noSOI, children with ADHD without insomnia; ADHD-C, ADHD combined subtype; ADHD-I, ADHD inattentive subtype; ADHD-HI: ADHD hyperactive/impulsive subtype.

Table 2 Sleep characteristics in ADHD children with chronic sleep onset insomnia and ADHD children without insomnia

	ADHD-SOI (n = *65*)	*ADHD-noSOI* (n = *18*)	P-*value*
Lights out, h:min (SD)	20:57 ± 0:52	20:21 ± 0:47	0.08
Sleep onset, h:min (SD)	21:49 ± 0:56	20:41 ± 0:45	< 0.001*
Sleep latency, h:min (SD)	0:53 ± 0:25	0:26 ± 0:25	< 0.001*
Total sleep duration, h:min (SD)	9:42 ± 0:44	10:09 ± 0:43	0.18
Actual sleep time, h:min (SD)	8:43 ± 0:52	9:13 ± 1:16	0.40

Group mean ± SD. ADHD, attention-deficit/hyperactivity disorder; ADHD-SOI, children with ADHD and chronic sleep onset insomnia; ADHD-noSOI, children with ADHD without insomnia.
*Significant difference between ADHD-SOI and ADHD-noSOI after correction for age ($P \leq 0.05$, two-sided).

In ADHD-SOI there were four (5.4%) children and in ADHD-noSOI one (4.3%) with signs of PLMS/RLS as reported during the medical history taking. Signs of sleep apnea were present in one (4.3%) child of the ADHD-noSOI group. Due to low occurrence and equal distribution over the two patient groups, these children were not excluded.

Actigraphy

Mean (±SD) SO in the ADHD-SOI group was 21:49 ± 0:56 h and in the ADHD-noSOI group 20:41 ± 0:45 h ($P < 0.001$) (Table 2). Lights out was 20:57 ± 0:52 h in ADHD-SOI and 20:21 ± 0:47 h in ADHD-noSOI ($P = 0.08$). There was a significant longer SL in ADHD-SOI (0:53 ± 0:25 h) as compared to ADHD-noSOI (0:26 ± 0:25 h) ($P < 0.001$). The difference in TSD between ADHD-SOI (9:42 ± 0:44 h) and ADHD-noSOI (10:09 ± 0:43 h) was not significant ($P = 0.18$). The difference in AST between ADHD-SOI (8:43 ± 0:52 h) and ADHD-noSOI (9:13 ± 1:16 h) was also not significant ($P = 0.40$).

Sleep hygiene

Mean (±SD) total sleep hygiene score in the ADHD-SOI group (56.4 ± 10.5) and ADHD-noSOI group (53.0 ± 10.6) did not differ significantly ($P = 0.17$) from each other (Table 3). The ADHD-SOI group showed a significantly lower score on item 1 (i.e. taking naps during the 4 h before bedtime more often) ($P = 0.011$) and a significantly higher score on item 7 (i.e. going to bed at about the same time every evening less often) ($P = 0.002$), as compared to the ADHD-noSOI group.

There was a significant relationship between total sleep hygiene score and lights out ($R = 0.23$; $P = 0.04$) in the pooled group, however, not in the separate groups (Table 4). Total sleep hygiene score was not significantly related to any other sleep parameter (i.e. SO, SL, TSD or AST) or to gender ($R = 0.015$; $P = 0.88$), age ($R = 0.13$; $P = 0.20$), comorbid DBD ($R = 0.081$; $P = 0.43$), ADHD subtypes ADHD-C ($R = 0.16$; $P = 0.11$), ADHD-I ($R = 0.097$; $P = 0.34$), or ADHD-HI ($R = 0.080$; $P = 0.43$), or to severity of inattentive ADHD symptoms ($R = -0.007$; $P = 0.95$) or hyperactive/impulsive ADHD symptoms ($R = 0.19$; $P = 0.092$).

DISCUSSION

The results of the present study showed that ADHD children with chronic SOI did not differ in sleep hygiene from their counterparts without insomnia, although there were marked between-group differences in SO and SL. Furthermore, sleep hygiene was not related to SO, SL, TSD, or AST, while we did find a relationship with lights out. Therefore, the present results do not support our initial hypothesis that sleep hygiene could play a role in the etiology of insomnia in ADHD children. This lack of association of insomnia and sleep hygiene level was also found in adult insomniacs (without ADHD) (Cheek *et al.*, 2004; Harvey, 2000b), but disagree with more recent data (Jefferson *et al.*, 2005).

We found that ADHD children with chronic SOI napped less frequently within 4 h before bedtime than those without insomnia. Two explanations can be proposed: (1) the insomniacs were less tired in the evening due to an elevated cognitive and physiological arousal level in the evening, as has been found in adult insomniacs (Harvey, 2000a; Vgontzas *et al.*, 2001) and (2) the ADHD children with chronic SOI were more tired than their counterparts without SOI but, as a consequence of their sleepiness, expressed more hyperactive behaviour (Ali *et al.*, 1996) in the evening, which hampered their ability to fall asleep.

Possibly, a disturbance of the circadian pacemaker plays a role in the late SO as suggested by our recent finding of a

Table 3 Sleep hygiene practices in ADHD children with chronic sleep onset insomnia and ADHD children without insomnia

Item	*Mean ADHD-SOI (n = 74)**	*Mean ADHD-noSOI (n = 23)**	P-*value*
1 Naps within 4 h before bedtime	1.00	1.09	0.011†
2 Caffeine within 4 h before bedtime	2.22	1.83	0.13
3 Does things that are relaxing before bedtime	3.23	3.17	0.92
4 Drinks a lot of liquids before bedtime	2.45	2.30	0.49
5 Plays rough before bedtime	3.11	3.39	0.50
6 Does things that are alerting before bedtime	3.64	3.35	0.47
7 Goes to bed at about the same time every day	2.19	1.73	0.002†
8 Complains about being hungry at bedtime	2.27	2.26	0.89
9 Does things in bed that keeps him/her awake	3.35	3.09	0.43
10 Goes to bed in the same place	1.53	1.64	0.55
11 Goes to bed feeling upset	1.78	1.61	0.44
12 Goes to bed with worries	2.21	2.00	0.35
13 Sleeps in a darkened room	3.07	1.83	0.029
14 Sleeps in a room that is too hot or too cold	1.49	1.78	0.14
15 Sleeps in a room where there are loud noises	1.32	1.26	0.76
16 Sleeps in alone (in his/her own bed)	1.72	1.43	0.60
17 Sleeps in a room that is 'stuffy'	1.47	1.35	0.23
18 Sleeps all or part of the night with someone else	2.24	1.91	0.50
19 Sleeps in a bed that is comfortable	1.12	1.09	0.75
20 Sleeps in a home where someone smokes	2.62	3.13	0.24
21 Has a calming bedtime routine	3.63	3.48	0.69
22 Uses bed for things other than sleep	2.68	2.57	0.62
23 Put to bed after falling asleep	1.18	1.52	0.19
24 Stays up past usual bedtime	2.93	2.26	0.027
25 Gets out of bed about same time in morning	2.70	2.04	0.023
Total (SD)	56.4 (10.5)	53.0 (10.6)	0.17

ADHD, attention-deficit/hyperactivity disorder; ADHD-SOI, children with ADHD and chronic sleep onset insomnia; ADHD-noSOI, children with ADHD without chronic sleep onset insomnia.
*Higher score represents worse sleep hygiene.
†Significant difference between ADHD-SOI and ADHD-noSOI ($P \leq 0.01$, two-sided).

Table 4 Relationship between sleep hygiene and sleep parameters

	ADHD-SOI		*ADHD-noSOI*		*Pooled groups*	
	R	P-*value*	*R*	P-*value*	*R*	P-*value*
Lights out	0.16	0.95	0.24	0.57	0.23	0.04*
Sleep onset	0.076	0.77	0.18	0.14	0.19	0.092
Sleep latency	0.11	0.66	0.09	0.48	0.031	0.78
Total sleep duration	0.14	0.57	0.009	0.94	0.055	0.62
Actual sleep time	0.14	0.28	0.063	0.80	0.054	0.63

ADHD, attention-deficit/hyperactivity disorder; ADHD-SOI, children with ADHD and chronic sleep onset insomnia; ADHD-noSOI, children with ADHD without chronic sleep onset insomnia.
*Significant relationship ($P < 0.05$, two-sided).

markedly delayed onset of endogenous melatonin in ADHD children with chronic SOI (van der Heijden *et al.*, 2005c). The ADHD children with chronic SOI in the present study showed a significantly later SO of more than 1 h and a longer SL of approximately half an hour as compared to the children with insomnia, while sleep duration was not significantly different. These are characteristics of a delayed sleep phase syndrome and corroborate our previous finding that chronic SOI in ADHD is a circadian rhythm sleep disorder (van der Heijden *et al.*, 2005c).

Recent data showed that a polymorphism of the *Per3* gene of the circadian pacemaker, which has previously been associated with delayed sleep phase syndrome in adults (Archer *et al.*, 2003; Ebisawa *et al.*, 2001), is not likely to be involved (van der Heijden *et al.*, 2005e). Yet, undiscovered clock genes (Takahashi, 2004) or other still unknown factors might also play a role. It was recently shown that the most commonly precipitating factor of insomnia in adolescents and adults was stress at school or work (Bastien *et al.*, 2004) which we did not measure. Further studies on the influence of school-related stress in ADHD-related chronic SOI are needed.

We investigated ADHD children without psychotropic medication, thus, a possible influence of this on SO can be excluded. Furthermore, we selected a group of consecutively referred subjects who were rigorously diagnosed for ADHD according to well-established criteria (American Psychiatric Association, 1994) and clinical practice guidelines (American Academy of Pediatrics, 2000; Dulcan and Benson, 1997) and for chronic SOI with strict and extensively discussed diagnostic criteria (van der Heijden *et al.*, 2005d). In none of the children, sleep hygiene-, or actigraphy measurements were preceded by behavioural sleep interventions to avoid possible manipulation of these factors in the insomnia group. Furthermore, a confounding influence of ADHD severity is unlikely since it was not related to sleep hygiene, nor were there differences in ADHD severity between the insomniacs and children without insomnia.

Actigraphy is a well-validated instrument to measure sleep in children (Littner *et al*., 2003b). Furthermore, the present scores on the CSHS (56.4 versus 53.0) agree satisfactorily with previous data in a general population of 246 US children (total score = 57.2; see Harsh *et al*., 2002), which suggests that the sleep hygiene level in our ADHD children was normal. We could not demonstrate a relationship between sleep hygiene and ADHD subtype or disruptive behavioural disorder, which makes a confounding involvement of these factors unlikely.

There were several limitations to this study that need to be acknowledged. First, we evaluated sleep with actigraphy and not with gold standard polysomnography (psg). Therefore, we could not objectively evaluate for sleep disorders such as sleep-disordered breathing, periodic limb movement disorder, restless legs syndrome, and sleep architecture disorders that may relate to chronic SOI and might, therefore, have confounded our data. Nonetheless, we have limited the chance that such sleep disorders were present by careful history taking (Allen *et al*., 2003; Farber, 2002; Stiasny *et al*., 2002; Stoohs *et al*., 2001; Walters, 1995). Furthermore, we found low scores and no group differences on various scales of the sleep disorders questionnaire which reduce the chance that these disorders might have confounded our data. Of note, the SDQ has not been validated for use in parents. However, the SDQ insomnia scale was significantly higher in the insomnia group as compared to the group without insomnia, which suggests at least a reasonable sensitivity on some scales. Our main reason to estimate sleep actigraphically and not polysomnographically is that actigraphy provides the possibility to assess sleep–wake rhythm over several consecutive days, in contrast to psg. A single night psg measure may fail to properly characterize the full extent of the sleep problem since insomnia typically varies in severity across nights (Littner *et al*., 2003a).

A second methodological issue is that a selection bias might have occurred due to the fact that a subset of the participants was recruited for participation in an ongoing study on melatonin efficacy. Consequently, the occurrence of chronic SOI of 76% in our ADHD sample was markedly higher than in earlier studies (Corkum *et al*., 1999; Stein, 1999). Notwithstanding the high proportion of insomniacs in the present study, the composition of gender, ADHD subtypes, and comorbid oppositional defiant disorder (DBD) resembles previous findings in ADHD (The MTA Cooperative Group, 1999). Furthermore, these variables were not related to sleep hygiene in the present study. In none of the children behavioural sleep interventions were employed by health care professionals before participating in the present study. However, a number of parents that participated in the present study and in the ongoing study on melatonin efficacy reported that, before enrolment, they had (vainly) tried several approaches to let their child fall asleep more easily. This might have improved sleep practices and, therefore, have influenced the present findings.

A further limitation in the present study is that the CSHS was not validated in the Dutch child population, nor were there any Dutch normative data available. However, we do not expect significant intercultural differences in sleep hygiene within western countries since definitions of sleep hygiene across studies are very similar in their core factors (Stepanski and Wyatt, 2003).

To conclude, the present results show that sleep hygiene is not a likely etiological factor in ADHD-related chronic SOI. Nevertheless, for individual patients in clinical practice, an evaluation of sleep hygiene can still be valuable to identify possible poor sleep hygiene practices that could affect SO. Furthermore, we underline that further research is needed to confirm and strengthen the present results.

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