Running head: ***Sleep, attention and ADHD behaviours***

Poor sleep has negative implications for children with and without ADHD, but in different ways.

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SUBMITTED TO: *Behavioural Sleep Medicine*

WORD COUNT: 3843

Abstract

Background: Sleep problems are commonly reported in attention deficit/hyperactivity disorder (ADHD), and are also a familiar characteristic of typical development (TD). We sought to elucidate the relationship between sleep, ADHD-trait behaviours and cognitive inattention, and how it manifests between ADHD and TD children.

Participants: 18 children diagnosed with ADHD and 20 age-matched TD controls aged 5-11-year-old participated in the study.

Methods: Sleep profiles were assessed using Children's Sleep Habits Questionnaire and actigraphy measures. Behavioural functioning was examined using Conners' Parent Report Scale and attention using the computerised Conners' Continuous Performance Task.

Results: We found evidence of 1) poorer sleep quality in the ADHD group, despite no difference in actual sleep time; 2) poor sleep quality in TD children predicted increased ADHD-trait behaviours, despite no association with attention; 3) a consistent trend for poor sleep quality predicting reduced attentional control in ADHD children, despite no association with behaviour.

Conclusions: Poor sleep quality affects developmental sub-groups in different ways. For ADHD children poor sleep worsens their predisposed attentional deficit, whilst for TD children it mimics ADHD behaviours. These findings have important implications for the debate on over-diagnosis of childhood ADHD, and the use of sleep-based interventions. Above all, they highlight the importance of promoting good sleep hygiene in all children.

Keywords: Sleep; Attention deficit/hyperactivity disorder; ADHD; Development; Attention; Inattention

Abbreviations: ADHD; TD; CSHQ; CPRS; CPT

1 Introduction

Attention Deficit/Hyperactivity Disorder (ADHD) is a common neurodevelopmental disorder, which affects between 1.4% and 7.1% of children and adolescents worldwide (Polanczyk, de Lima, Horta, Biederman & Rohde, 2007; Russell, Rodgers, Ukoumunne & Ford, 2014). It is diagnosed using core behavioural characteristics centring on developmentally inappropriate levels of inattention, hyperactivity and impulsivity. In presentation, the disorder can appear far more complex than its diagnostic checklist might suggest. Additional behavioural traits associated with ADHD include defiance, restless motor activity, shyness and withdrawal, and emotional problems (Classi et al., 2012). ADHD has also been linked with atypical executive function (for review see Frazier, Demaree & Youngstrom, 2004). Additionally, high rates of comorbidity with other childhood psychiatric disorders and medical problems are reported (Jensen, Martin & Cantwell, 1997). One such comorbidity, suggested to affect behaviour and cognition, is the presence of sleep problems and disorders. Leading researchers call for a better understanding of the association between sleep problems, attention and behavioural regulation between ADHD and typical developing (TD) children (Owens et al., 2013; Spruyt & Gozal, 2011).

The association between ADHD and sleep disturbance is well documented (Owens, 2005; Yoon, Jain & Shapiro, 2012). Estimates of the prevalence of sleep disturbances in ADHD vary widely; parents frequently report high prevalence rates (between 25-70%), which are rarely corroborated by objective measures (such as actigraphy and polysomnography; for review see Kirov & Brand, 2014). Despite questions surrounding prevalence rates, mounting evidence substantiates the claim that sleep disturbances

worsen ADHD symptomatology. For instance, poor sleep in ADHD has been associated with increased distractibility (Sawyer et al., 2009); increased prevalence of conduct problems, hyperactivity, and restlessness (Stephens et al., 2013); as well as deficits in the overnight consolidation of declarative and emotional memories (Prehn-Kristensen et al., 2011, 2013). Probing the direction of this effect, Gruber and colleagues (2011) report that the experimental restriction of sleep in ADHD children serves to move sub-clinical levels of sustained attention into the clinical range. Importantly, the attentional deterioration observed in ADHD participants was far greater than in their TD peers. Owing to the ethical issues surrounding sleep restriction in children, studies such as this are few. An alternative approach to examining the directional effect of sleep problems in ADHD is to improve sleep whilst monitoring variations in daytime functioning. From this perspective, Hiscock and colleagues (Hiscock et al., 2015; Papadopoulos et al., 2015) reported wide-ranging benefits of a brief behavioural sleep therapy. In a large sample of ADHD children with moderate-to-severe sleep problems, the intervention was successful in reducing sleep problems, alongside improving ADHD behaviour severity, classroom behaviour and working memory task performance.

A parallel issue is that of how sleep problems affect behaviour and cognition in the TD child. TD children are often used as the control group for ADHD studies, despite many reports suggesting that sleep problems are not only normal in healthy child development (Mindell & Owens, 2003), but affect TD children in a way which mimics ADHD. Poor sleep in school-aged children is often associated with inattention and challenging daytime behaviours (Paavonen et al., 2002); and ADHD-like cognitive problems including difficulties with attention, memory and learning (Gruber, Cassoff, Frenette, Wiebe & Carrier, 2012). In accordance, TD children experimentally restricted of sleep display

ADHD-like inattention (Fallone, Acebo, Arnedt, Seifer & Carskadon, 2001), and increased problematic behaviours (Fallone, Acebo, Seifer & Carskadon, 2005). The prevalence of sleep problems in TD children, and their effect on behaviour begs the questions: Could some diagnosed cases of ADHD in fact represent the behavioural presentation of underlying paediatric sleep disturbance?

In the current study, we set out to investigate the relationship between sleep, ADHD-behaviours and attention in school age children with and without a diagnosis of ADHD. We were interested in examining differences in sleep profiles, and how they manifest in terms of behaviour and attention between the two groups. We used the Child Sleep Habits Questionnaire (CSHQ) and actigraphy as complimentary measures of sleep. We selected the Conners Parent Rating Scale (CPRS) for its detail on ADHD trait-behaviours commonly associated with ADHD. Finally, Conners Continuous Performance Task (CPT) was used as a measure of attention across a number of domains.

2 Methods

2.1 Participants

Eighteen children with ADHD aged 5-11 years, and 20 age-matched TD children participated. Four ADHD and one TD participants were excluded from analysis due to inability to complete testing procedures or incomplete data provided by parents. ADHD participants were recruited through local schools, ADHD support groups, and online forums. TD participants were recruited through four schools in Greater London, with an attempt at matching SES between groups as closely as possible.

Children were excluded from the study if (1) their parents reported co-morbid medical or psychiatric disorders with the exception of co-morbid Opposition Defiance Disorder (ODD), (2) including a diagnosed sleep disorder (1 & 2 elucidated in the Background questionnaire), (3) had an IQ ≤ 75 according to their performance on the IQ tests below in relation to standardised norms. Given the potential to disrupt sleep, children were also screened for poorly controlled asthma or eczema however no participants were sufferers. Finally, children were required to have functional hearing and vision.

2.1.1 ADHD diagnosis and medical status

Children in the ADHD group received formal diagnosis from a registered Paediatrician, Child Psychiatrist or Child Psychologist. In the UK practitioners use the DSM-V diagnostic criteria, supported by a series of interviews with parent and child, and reports from teachers. In all but one case ADHD subtype was not reported as part of the child's diagnosis. We assessed ADHD subtype using the DSM-IV criteria using Conners' CPRS as recommended in the Conners' rating Scales Manual. The Predominantly Inattentive (ADHD-PI) subtype was met if the participant received six or more scores of two or above on the DSM-IV Inattentive symptom checklist, likewise for the Predominantly Hyperactive-Impulsive (ADHD-PH) subtype and symptom checklist. Combined subtype was met given six or more scores of two or above from each subtype. One child met the ADHD-PI criteria, two children met the ADHD-PH criteria; the rest met the Combined subtype. These scores were checked against the norms-based criteria across the three DSM-IV subtypes provided within the CPRS (Barkley, 1997). All children within the ADHD group scored 'moderate'-to-'markedly atypical' on one or more of the three axes; one TD child met this criterion and was removed from analysis. Two children with ADHD were taking Methylphenidate (5mg twice a day).

2.2 Measures

2.2.1 Background questionnaire & IQ testing

Demographic information was collected using a general background questionnaire developed by the LiLAS laboratory (Lifespan Learning and Sleep laboratory). The questionnaire probes socioeconomic status, medical history, family structure and parental stress. We used two established standardised tests to assess children's IQ. Raven's Coloured Progressive Matrices (Raven, Raven & Court, 1998) was used to evaluate children's non-verbal reasoning. From the British Ability Scale (BAS-III, Elliot, Smith & McCulloch, 1996), we used the recall of digits forward to test short-term verbal memory.

2.2.2 ADHD measure: Conners Parent Report Scale (CPRS)

ADHD behaviours were measured using the CPRS (long-version), a validated scale with high internal consistency (0.86-0.95) and test-retest reliability (Conners, Sitarenios, Parker & Epstein, 1998). The scale includes the DSM-IV diagnostic profile, as well as evaluating key ADHD trait-behaviours including hyperactivity/impulsivity, inattentiveness, executive function, and learning, social, and behavioural problems.

2.2.3 Sleep measures

Child Sleep Habits Questionnaire (Owens, Spirito & McGuire, 2000): The CSHQ, which shows good test-retest reliability, was used to profile habitual sleep. The CSHQ returns scores on nine scales: Bedtime resistance (BR), Sleep onset delay, Sleep duration, Sleep anxiety, Night awakenings, Parasomnias, Sleep disordered breathing, Daytime sleepiness and Total Sleep disturbance score.

Actigraphy and sleep diary: Parents and children were provided with a simple sleep diary to record bed and rise times, and night awakenings. MotionWatch 8 (CamNTech, Cambridge, UK) actigraphy devices were used for one week as an objective measure of sleep in the child's home environment. Watchstraps were replaced with plastic wristbands, which were not easily removable or adjustable. Data were analysed using MotionWare 1.1.3 software, employing the validated 'High' sensitivity threshold, meaning activity levels for every epoch within the set period of time must be less than 20 to be categorised as sleep. The programme algorithm scores each one-minute epoch as either sleep or wake based on movement levels during that minute, relative to the two minutes either side (± 2 mins). 'Lights out' and 'got up' times were inputted from the sleep diary to support the actigraphy data. Six measures were identified as variables of interest: time in bed (total time spent in bed); assumed sleep time (time from onset to offset); actual sleep time (assumed sleep time minus any periods of wake); sleep latency (time from 'lights out' to sleep start); sleep efficiency (percentage of time spend asleep from sleep onset to offset), and the fragmentation Index (an indication of sleep quality, or lack thereof, where 1 indicates fragmented sleep and 0 indicates stable sleep). The fragmentation index represents the sum of mobile time (the percentage of mobile time during the 'assumed sleep' block) and the immobile bouts lasting less than or equal to one minute (expressed as a percentage of the total number of immobile bouts). Four days worth of weekday data were successfully recorded from each participant; the average was used for analysis.

2.2.4 Experimental task: Conners' Continuous Performance Task (CPT)

Conners' CPT provided a measure of attention across a number of domains. Conners' CPT requires participants to press a button in response to every stimulus *except from* the target. Visually displayed stimuli consisted of colourful cartoon animals presented sequentially for 300 ms in the centre of a white screen, with an inter-stimulus interval of 2000 ms, in a pseudo-randomised order (random without replacement). Participants were presented with 200 trials across five blocks; target stimuli (which required response inhibition) occurred on 20% of trials, all other trials were classified as standard trials.

Task instructions were provided both written and verbally before beginning. Children were asked to rest the index finger of their dominant hand on the response key. Each participant was given 20 practice trials. During testing, children were given prompts if they failed to inhibit a response to a target. Children took short breaks between blocks. In total, the task lasted approximately 7 minutes.

Six CPT variables were identified for analysis: *Accuracy*, proportion of correct responses overall; *Commission*, incorrect responses to target trials, typically taken as a measure of behavioural inhibition; *Omission*, incorrect non-responses to standard trials, typically taken as a measure of sustained attention; *RT hits*, reaction times to correct non-target responses; *RT commissions*, incorrect reaction times to target responses (requiring inhibition of response); *RT variability*, standard deviation in RT to hits for each participant. Button presses before 150ms and after 2000ms were treated as incorrect responses.

2.3 Procedure

Testing was performed in the child's own homes, in a quiet room, free from distractions. The child performed the Conners' CPT first; the Raven's CPM and the BAS-III digits forward tests were counterbalanced between participants within experimental group. Average testing lasted 40 minutes per session including breaks. The primary-caregiver was given the Background questionnaire, the CPRS and the CSHQ to complete during this time. There was no monetary incentive for participation; parents' received a profile of their child's behaviours and sleep patterns post-testing.

Written informed consent was gained from parents or legal guardians, and written assent from the child. Methodological procedures were approved by the Institute of Education's Research Ethics Committee prior to testing.

2.4 Data analysis

Data analysis was performed in three phases. After confirming diagnostic and basic IQ differences between the groups using planned t-tests, we explored group differences in sleep characteristics using 2 MANCOVAs controlling for age. The first used the full set of subcategories of the CSHQ as the dependent variables, with group as the independent factor and age as the covariate. The second had the same independent and covariate factors, the dependent variables were made up of the six identified actigraphy measures. In the second phase of analysis, regressions of sleep variables onto ADHD-behaviour traits were performed between groups. Finally, group differences in CPT task performance were explored using MANCOVAs controlling for age. Subsequent regressions were performed in order to scrutinise the predictive nature of sleep problems on attention task performance.

3 Results

3.1 Participant demographic information

Table 1. presents participant characteristics between groups, along with relevant between group test statistics. This table excludes the four ADHD and two TD participants who did not complete the testing protocol. The groups differed only on the DSM ADHD total score.

			t-test		
	ADHD	**TD**	***t***	***p***	***d***
N	14 (3 female)	18 (9 female)			
Age in years (range)	8.54 (5.58-11.33)	8.96 (5.10-11.83)	*ns (p = .53)*		
DSM total score (SD)	**66.57 (18.92)**	**50.83 (16.79)**	**2.49**	**.009**	**.88**
Ravens (SD)	29.54 (8.83)	30.83 (4.88)	*ns (p = .61)*		
BAS digits forward (SD)	21.33 (5.55)	22.53 (4.23)	*ns (p = .48)*		

3.2 Group differences in sleep

Three separate MANCOVAs were performed on CSHQ and actigraphy data. For each, *Group* (ADHD, TD) represented the independent variable, and *Chronological Age (CA)* as a covariate. For the first MANCOVA, the dependent variables were the 9 subcategories of the CSHQ. Using Wilk's lambda, there were significant effects of *Group* and *CA* on measures of the CSHQ [λ = .48, F (8, 22) = 2.94, p = .02, np^2 = .52; and λ = .54, F (8, 22) = 2.37, p = .05, np^2 = .46 respectively]. We followed up the significant main effect of Parasomnias (and interaction with Age) with a further MANCOVA exploring each of the seven parasomnias included in the CSHQ (bedwetting, sleep talking, restlessness during

sleep, sleep walking, bruxism, distressed awakenings, and nightmares), with CA as a covariate. Using Wilk's lambda, we found a significant effect of Group, but not CA [λ = .44, F (7, 20) = 3.68, p = .01, np^2 = .56; and λ = .85, F (7, 20) = .518, p = .81, np^2 = .15 respectively]. Table 2 displays group means, standard deviations and MANCOVA test statistics for individual dependent variables showing significant effects. For the second MANCOVA, with the six actigraphy variables as dependent variables, no effect of *CA* was found. Although the main effect of *Group* was not significant, it did reveal a trend with a strong effect size, λ = .63, F (6, 24) = 2.34, p = .06, np^2 = .37. No significant effects were returned from isolated dependent variables. Owing to the non-significant effect of *CA* and the trend in the *Group* variable with a strong effect size (Cohen, 1988; Miles & Shelvin, 2001), t-tests between groups on the six actigraphy variables were performed. See Table 3 for details.

Table 2 and 3

	GROUP					AGE			GROUP * AGE		
	ADHD	TD	Main effect			Main effect			Interaction		
CSHQ	*M (SD)*		*F*	*p*	np^2	*F*	*p*	np^2	*F*	*p*	np^2
Sleep Duration	5.86 (1.56)	4.11 (1.68)	9.35	.005	.25	*NS p = .51*			5.82	.02	.17
Parasomnias	10.07 (2.43)	7.94 (1.11)	10.99	.003	.28	*NS p = .09*			6.93	.01	.20
- Distressed awakenings	1.08 (.29)	1.00 (.00)	5.05	.03	.16	4.12	.05	.14	4.12	.05	.14
- Nightmares	1.25 (.45)	1.17 (.38)	5.98	.02	.19	*NS p = .13*			5.70	.03	.18
Daytime Sleepiness	13.50 (3.86)	12.00 (2.74)	*NS p = .44*			6.20	.02	.18	*NS p = .25*		
CSHQ Total	51.00 (7.90)	43.22 (5.99)	4.92	.04	.15	*NS p = .27*			*NS p = .14*		

Actigraphy subscales	**ADHD** Mean (SD)	**TD** Mean (SD)	**t-test** *t*	*p*	*d*
Time in bed	**11:01:58 (00:40:12)**	**10:07:02 (00:37:55)**	**3.70**	**.001**	**1.45**
Assumed sleep	**9:45:19 (00:32:57)**	**9:05:46 (00:39:01)**	**2.83**	**.005**	**1.11**
Actual sleep	7:53:16 (00:32:56)	7:42:50 (00:44:05)	*ns (p = .50)*		
Sleep efficiency (%)	71.99 (7.96)	75.71 (7.78)	*ns (p = .23)*		

Sleep latency	**00:50:22 (00:32:43)**	**00:30:27 (00:20:44)**	**2.00**	**.03**	**.78**
Fragmentation index	**33.17 (8.12)**	**28.42 (7.33)**	***ns trend***		
			1.62	***.059***	***.64***

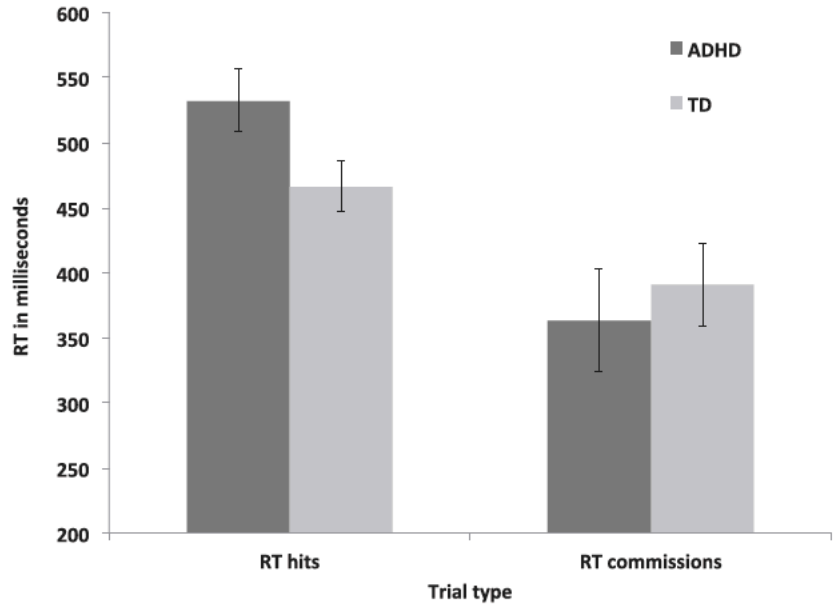
3.2.1 The relationship between sleep & behaviour

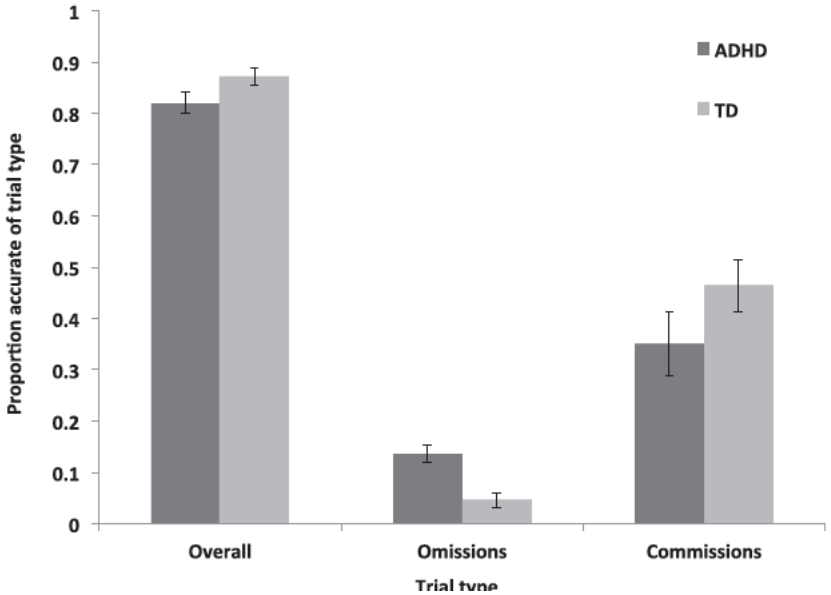
Linear regressions of CSHQ total score on DSM total score were performed for ADHD and TD participants separately. For ADHD participants no significant effect was observed. For TD participants the CSHQ total score strongly predicted the DSM total score, such that increased CSHQ score (indicating more sleep problems) predicted a higher ADHD rating, F (1, 17) = 10.53, p = .005, R^2 = .40. A hierarchical regression was performed for TD participants only, to elucidate the predictive nature of the CSHQ variables, *Parasomnias* and *Sleep Duration (SD),* on DSM total score. *Parasomnias* and *SD* were selected from the CSHQ given their showing greatest departure from the norm between ADHD and TD children. CSHQ total was not included in these models as it is a composite score of other subscales. The model explained 45% of the variance in the DSM total score for TD children, F (2, 17) = 6.03, p = .01, R^2 = .45. *SD* and *Parasomnias* both represented significant predictors of DSM total score, *Beta* = .52, t (17) = 2.70, p = .02, and *Beta* = .42, t (17) = 2.19, p = .05 respectively.

3.3 Group performance on the CPT task

In order to examine differences in performance the six CPT measures were analysed. A MANCOVA was performed with Group as the independent variable, *CA* as the covariate. Overall, *Group* represented a significant main effect, λ = .51, F (6, 21) = 3.39, p = .017, np^2

= .49, as did *Age*, λ = .34, F (6, 21) = 6.67, p = .000, np^2 = .66. The interaction between *Group* and *CA* approached significance, λ = .60, F (6, 21) = 2.36, p = .067, np^2 = .40. Table 4 displays group means, standard deviations and MANCOVA test statistics for dependent variables showing significant effects. Figures 1 and 2 displays between group means of six of the CPT measures, corresponding to the MANCOVA results.





3.4 The relationship between sleep & attention

Two sets of hierarchical regressions were performed to determine the predictive nature of sleep on attentional task performance between groups, the first using CSHQ variables and the second using actigraphy variables. Regressions were performed for *Accuracy, Omissions* and *RT variability* for each group separately. As above, *Parasomnias* and *SD* were selected as predictors, whilst CSHQ total score was excluded. For the actigraphy measures, *Time in Bed* and *Sleep Latency* were selected as predictors owing to large group differences. Table 5 displays regression statistics. *CA* was not included in these models in order to reduce predictors given low sample numbers; this was deemed appropriate given successful age matching between groups. From these analyses, *Parasomnias, Time in Bed* and *Sleep Latency* consistently returned trends with moderate-to-strong effect sizes for the ADHD group. Results from these regression analyses should be treated as preliminary owing to low sample size following attrition. However given the strong trends shown in the data for ADHD participants, we suggest that these effects would likely be significant given a larger sample size.

CPT		GROUP					AGE			GROUP * AGE		
		ADHD	TD	Main effect			Main effect			Interaction		
		m (sd)		*F*	*p*	np^2	*F*	*p*	np^2	*F*	*p*	np^2
Means	**Accuracy**	.82 (.12)	.87 (.04)	10.08	.004	.28	*NS p = .083*			8.07	.009	.24
	Omissions	.14 (.12)	.05 (.03)	18.30	.000	.41	18.95	.000	.42	11.85	.002	.31
	RT hits	535.31 (117.91)	462.83 (97.98)	*NS p = .486*			17.34	.000	.40	*NS p = .249*		
SDs	**RT variability**	137.37 (40.47)	108.21 (23.43)	4.28	.049	.14	13.79	.001	.35	11.85	.002	.31

GROUP	SLEEP MEASURE	MODEL - *Predictor*	R^2	*Beta*	*F*	*t*	*p*
ADHD	**CSHQ**	**Omissions**	**.31**		**4.40**		**.062**
		- Parasomnias		*.55*		*2.10*	*.062*

		RT variability	**.29**		**4.17**	**.068**
		- Parasomnias		*-.54*	*2.04*	*.068*
		Accuracy	**.26**		**3.59**	**.088**
		- Parasomnias		*-.51*	*1.89*	*.088*
TD	**CSHQ**	**Omissions**	**.18**		**3.55**	**.078**
		- Parasomnias		*.43*	*1.88*	*.078*
ADHD	**ACTIGRAPHY**	**Accuracy**	**.46**		**3.85**	**.062**
		- Time in Bed		*2.31*	*2.49*	*.035*
		- Sleep Latency		*2.53*	*2.72*	*.023*
		Omissions	**.45**		**3.62**	**.070**
		- Time in Bed		*2.28*	*2.43*	*.038*
		- Sleep Latency		*2.49*	*2.65*	*.027*
		RT variability	**.41**		**3.11**	**.094**
		- Time in Bed		*1.93*	*1.98*	*.079*
		- Sleep Latency		*2.25*	*2.31*	*.046*

4 Discussion

Three key findings came from this study. Firstly, in line with previous research, we found evidence of more sleep problems in our ADHD sample than our TD sample. Secondly, we found that sleep problems were predictive of increased ADHD-trait behaviours in our TD sample, but were not associated with impaired attentional capacity. Finally, we found consistent trends suggesting that poor sleep is predictive of reduced attentional capacity in the ADHD sample, with no predictive nature towards ADHD behaviours. The latter two findings demonstrate that problems with sleep impact on both childhood populations, but in distinct ways.

4.1 Sleep in children with ADHD

Actigraphy data indicated that children with ADHD spend more time in bed, and more time in assumed sleep, with no significant group difference in actual sleep time. Children with ADHD were also found to have longer sleep latencies (50 vs. 30 min), suggesting that settling into sleep is an issue for this population. Difficulties with sleep onset delay in the

ADHD population are commonly reported by parents (Schneider, Lam, & Mahone, 2016); however, this is not always corroborated using actigraphy data (e.g., Corkum, Tannock, Moldofsky, Hogg-Johnson, & Humphries, 2001). In a meta-analytic review of 24 actigraphy sleep studies comprising data from 631 ADHD and 692 TD children, De Crescenzo et al. (2016) found sleep-onset delay to be a stable feature of the sleep problems associated with ADHD, despite high heterogeneity (see also Cortese, Faraone, Konofal, & Lecendreux, 2009). Our actigraphy data showed that both groups spent just under 8 hr asleep during the night (7:53 and 7:42 for ADHD and TD respectively), despite parents of the ADHD group reporting lower sleep duration. Similar discrepancies have previously been reported in other developmental disorders (Ashworth, Hill, Karmiloff-Smith, & Dimitriou, 2014).Actigraphy data indicated that ADHD children spent significantly longer in bed, and more time in assumed sleep, with no significant group difference in actual sleep time. ADHD children were also found to have longer sleep latencies (50 vs. 30 minutes), suggesting that children with ADHD find it harder to settle into sleep. Both groups spent just under eight hours asleep during the night (7:53 and 7:42 for ADHD and TD respectively), despite parents of the ADHD group reporting lower sleep duration. This phenomenon has previously been reported in other developmental disorders (Ashworth, Hill, Karmiloff-Smith, & Dimitriou, 2014). Parent reports suggested that sleep problems centred on parasomnias and sleep duration. Similar discrepancies have previously been reported in other developmental disorders (Ashworth, Hill, Karmiloff-Smith, & Dimitriou, 2014). Parent reports suggested that sleep problems centered on parasomnias as well as sleep duration. Parasomnias are disruptive sleep behaviors such as nightmares and sleepwalking, which are common in childhood and typically reduce with age (Mason & Pack, 2007). In the current study, parents reported

problems specifically with nightmares and distressed awakening in the ADHD group. Our finding corroborates previous literature, finding an increased presence of parasomnias in ADHD children, including nightmares and bedwetting as measured by the CSHQ or similar measures (Gomes, Parchão, Almeida, Clemente, & De Azevedo, 2014; Rodopman-Arman, Perdehli, Eking & Berkem, 2011), as well as other nighttime behaviors associated with parasomnias such as increased nighttime restlessness (Rodopman-Arman, Perdehli, Eking, & Berkem, 2011), and overactivity (Sung, Hiscock, Sciberras, & Efron, 2008). It should be noted that the CPRS does not diagnose the presence of clinically certified parasomnias, but rather screens for symptoms suggestive of parasomnias. These findings point to parents of children with ADHD harbouring more concerns over their child's night-time behaviours.

4.2 Poor sleep and behaviour in TD children

Our data indicate that sleep disturbances affect both ADHD and TD children, but in different ways. In our TD sample poor sleep was associated with increased ADHD-like behaviours. This finding has implications for the discussion on the over-diagnosis of ADHD in the modern world. If poor sleep produces ADHD-like symptoms, it is possible that some diagnosed cases of ADHD are in fact the behavioural consequence of underlying paediatric sleep disturbance. Indeed, there is evidence to support this theory. For example, Chervin et al. (2006) found that corrective surgery for sleep-disordered breathing alleviates ADHD symptoms. Despite many patients meeting the behavioural criteria for ADHD prior to surgery, 50% no longer met those criteria post-surgery. It is wholly possible that, for these children, their ADHD profile was not a stable and enduring characteristic of an existing disorder, but a consequence of their disturbed sleep. For the 50% whose ADHD symptoms did not reduce, their ADHD diagnosis predominates the

sleep problems they suffer, and may be largely unaffected by them. Although sleep problems are apparent in TD children, persistent sleep problems can have a notable negative effect on behaviour and cognitive functioning. Quach, Hiscock, Ukoumunne and Wake (2011) report that a brief behavioural sleep intervention had a sustained positive effect on sleep habits and prosocial behaviours for TD children (5-6-year-old) experiencing moderate-to-severe sleep problems.

4.3 Poor sleep and sustained attention in children with ADHD

Our findings indicate that poor sleep is predictive of poorer attentional task performance in ADHD, specifically sustained attention. Whilst regression models returned trends, we go on to discuss them as preliminary evidence. We do so owing to: 1) the strength of the effect; 2) despite the low sample size; 3) that Sleep latency and Time in Bed represented significant predictors of reduced attentional performance; and 4) its consistency with previous findings, specifically related to Omission errors and RT variability (Gruber et al., 2011; Hansen et al., 2014).

The association between ADHD and domains of attention as an executive function is longstanding (Barkley, 1997). However a new discussion is emerging about its association with dysfunction of the HPA axis as a common regulatory system (for review see Corominas et al., 2012). The HPA axis is a regulatory system, vitally important for the control of attention, movement, and the sleep/wake cycle. As such, deficits in this system may underlie difficulties in attentional regulation, behavioural and movement regulation, as well as sleep/wake regulation (Gruber & Sadeh, 2004). Atypical cortisol levels are reported in conjunction with ADHD and disrupted sleep patterns (Imeraj et al., 2012), including disruptive movement during sleep (Castelo et al., 2012); behavioural inhibition

(Hong, Shin, Lee, Oh & Noh. 2003); and inappropriate stress responses (Blomqvist et al., 2007; Ma et al., 2011). Here, our ADHD sample has worse sleep, which centres on longer sleep latencies, the presence of parasomnias, as well as poorer attentional control. Given the responsibilities of HPA activity this pattern of findings fits inline with a theory of ADHD, sleep and attention regulation based on disregulation of those biological systems. We, amongst others, propose that function of the HPA axis, as a common underlying biological substrate, seems an appropriate focus for future research.

4.4 Limitations

There are a number of limitations associated with the current study. Firstly, the low sample size meant that we were unable to probe meaningful differences in gender, ADHD subtype, and medical status, all of which may influence the relationship between sleep, attention & daytime function (for review see Kirov & Brand, 2014). Using parental responses from the CPRS, our ADHD group was made up of one ADHD-PI, two ADHD-PH, and eleven Combined. However it should be noted that in most cases the diagnosis of ADHD from a clinical professional did not come with a report of subtype. The authors have discussed this with clinical professionals, who suggest that this is common practice in the UK. Two participants within the ADHD group were taking medication at time of testing. It has previously been noted that methylphenidate can positively affect task performance on CPT tasks (e.g. Pollack, de Lima, Horta, Biederman & Rohde, 2010). Given a larger sample it would have been interesting to probe differences in relation to subtypes and medical status. Secondly, obtaining more nights' actigraphy data from each participant would strengthen this measure. Recommendations vary from three to seven consecutive nights recording (Acebo, Sadeh, Siefer, et al., 1999; Littner, Kushida & Anderson, 2003; Martin & Hakim, 2011), however in general there is agreement that

the more nights recorded the greater the reliability of sleep patterns. It would also be interesting to observe differences in weekday and weekend sleep behaviours, because without the imposition of school schedules, weekends represent a period of more natural sleep habits. Finally, the attentional task is limited in its application to real world context. As researchers we aim to provide a well-controlled test environment, free from distractors. However in ridding the environment of distractors we remove the very thing with which ADHD children struggle. For example, no classroom is free from distraction; it is therefore important that we investigate attentional ability in the presence of distractors (e.g. Areces, Rodríguez, García, Cueli & González-Castro, 2016) and how sleep modulates this capacity.

4.5 Future directions

Here we propose that using TD children as a control group for investigating poor sleep in the ADHD population poses certain difficulties. Firstly, modern sleep habits in the TD population are frequently suggested to be insufficient (e.g. Smaldon, Honig, & Byrne, 2007; however this position is hotly contested; see Matricciani, Olds, Blunden, Rigney, & Williams, 2012). Secondly, poor sleep in the TD population has negative implications for cognition and behaviour (for review see Beebe, 2012), but these manifest in a different way to those observed in ADHD children as found here. This begs the question; is it appropriate to model ADHD sleep on modern TD sleep? Future research should further explicate these inherent differences, and remain mindful of this when using the TD population as a comparison group. Furthermore, given the finding of decreased attentional control in ADHD children with poor sleep reported here, and in order to gain more meaningful insight into how sleep may modulate attentional capacities in ADHD children, we propose that future research should consider attentional capacity within a

context that more accurately reflects the demands of a typical childhood environment, such as a Virtual Reality (VR) classroom (Díaz-Orueta et al., 2014).

4.6 Conclusion

Poor sleep has adverse effects for children with and without ADHD, but in different ways. For TD children, problems with sleep increase the cardinal behavioural symptoms of ADHD. Importantly, our findings reinforce the suggestion that for children with ADHD poor sleep has a negative effect on cognitive functioning that outweighs that of TD children. This is particularly meaningful for many children with ADHD who are at an attentional disadvantage from the outset. Given the central importance of attentional capacities in healthy cognitive development and classroom learning, focussing interventions on improving sleep in childhood ADHD may prove an effective and relatively inexpensive means of improving attentional capacities and academic success in this vulnerable group (Hiscock et al., 2015). Reducing the prevalence of disruptive ADHD traits is of societal significance owing to their established link with heavier reliance on school input, healthcare utilisation, and later prison sentencing (Classi, Milton, Ward, Sarsour & Johnston, 2012).

5 Acknowledgements

We would like to warmly thank all the ADHD and TD children who took part, as well as their parents. It was a pleasure to work with you all. This research was supported by a grant from the Waterloo Foundation under their Child Development Fund (Waterloo Grant agreement no. 1282/1981).

References

Areces, D., Rodríguez, C., García, T., Cueli, M., & González-Castro, P. (2016). Efficacy of a continuous performance test based on virtual reality in the diagnosis of ADHD and its clinical presentations. *Journal of attention disorders*, 1087054716629711.

Ashworth, A., Hill, C. M., Karmiloff-Smith, A., & Dimitriou, D. (2014). Sleep enhances memory consolidation in children. *Journal of Sleep Research*, *23*(3), 304-310.

Barkley, R. A. (1997). Behavioral inhibition, sustained attention, and executive functions: constructing a unifying theory of ADHD. *Psychological Bulletin*, *121*(1), 65.

Blomqvist, M., Holmberg, K., Lindblad, F., Fernell, E., Ek, U., & Dahllöf, G. (2007). Salivary cortisol levels and dental anxiety in children with attention deficit hyperactivity disorder. *European Journal of Oral Sciences*, *115*(1), 1-6.

Castelo, P. M., de Souza Barbosa, T., Pereira, L. J., Fonseca, F. L. A., & Gavião, M. B. D. (2012). Awakening salivary cortisol levels of children with sleep bruxism. *Clinical Biochemistry*, *45*(9), 651-654.

Chervin, R. D., Ruzicka, D. L., Giordani, B. J., Weatherly, R. A., Dillon, J. E., Hodges, E. K., ... & Guire, K. E. (2006). Sleep-disordered breathing, behavior, and cognition in children before and after adenotonsillectomy. *Pediatrics*, *117*(4), e769-e778.

Classi, P., Milton, D., Ward, S., Sarsour, K., & Johnston, J. (2012). Social and emotional difficulties in children with ADHD and the impact on school attendance and healthcare utilization. *Child & Adolescent Psychiatry & Mental Health*, *6*(1), 1-8.

Conners, C, Sitarenios, G, Parker, JD, Epstein, JN. (1998). The revised Conners' Parent Rating Scale (CPRS-R): factor structure, reliability, and criterion validity. *Journal of Abnormal Child Psycholo*gy, *26*, 257–268.

Corominas, M., Ramos-Quiroga, J. A., Ferrer, M., Sáez-Francàs, N., Palomar, G., Bosch, R., & Casas, M. (2012). Cortisol responses in children and adults with attention deficit hyperactivity disorder (ADHD): a possible marker of inhibition deficits. *ADHD Attention Deficit and Hyperactivity Disorders*, *4*(2), 63-75.

Díaz-Orueta, U., Garcia-López, C., Crespo-Eguílaz, N., Sánchez-Carpintero, R., Climent, G., & Narbona, J. (2014). AULA virtual reality test as an attention measure: Convergent validity with Conners' Continuous Performance Test. *Child Neuropsychology*, *20*(3), 328-342.

Elliott, C. D., Smith, P. & McCulloch, K. (1996). *British Ability Scales Second Edition (BAS II). Administration and Scoring Manual*. London: Nelson.

Fallone, G., Acebo, C., Arnedt, J. T., Seifer, R., & Carskadon, M. A. (2001). Effects of acute sleep restriction on behavior, sustained attention, and response inhibition in children. *Perceptual & Motor Skills*, *93*(1), 213-229.

Fallone, G., Acebo, C., Seifer, R., & Carskadon, M. A. (2005). Experimental restriction of sleep opportunity in children: effects on teacher ratings. *Sleep-New York then Westchester-*, *28*(12), 1561.

Frazier, T. W., Demaree, H. A., & Youngstrom, E. A. (2004). Meta-analysis of intellectual and neuropsychological test performance in attention-deficit/hyperactivity disorder. *Neuropsychology*, *18*(3), 543.

Gomes, A. A., Parchão, C., Almeida, A., Clemente, V., & De Azevedo, M. H. P. (2014). Sleep–wake patterns reported by parents in hyperactive children diagnosed according to ICD-10, as compared to paired controls. *Child Psychiatry & Human Development*, *45*(5), 533-543.

Gruber, R., & Sadeh, A. (2004). Sleep and neurobehavioral functioning in children with ADHD. *SLEEP, 27,* 267-73.

Gruber, R., Wiebe, S., Montecalvo, L., Brunetti, B., Amsel, R., & Carrier, J. (2011). Impact of sleep restriction on neurobehavioral functioning of children with attention deficit hyperactivity disorder. *Sleep*, *34*(3), 315-323.

Gruber, R., Cassoff, J., Frenette, S., Wiebe, S., & Carrier, J. (2012). Impact of sleep extension and restriction on children's emotional lability and impulsivity. *Pediatrics*, *130*(5), e1155-e1161.

Hiscock, H., Sciberras, E., Mensah, F., Gerner, B., Efron, D., Khano, S., & Oberklaid, F. (2015). Impact of a behavioural sleep intervention on symptoms and sleep in children with attention deficit hyperactivity disorder, and parental mental health: randomised controlled trial. *BMJ*, *350*, h68.

Hong, H. J., Shin, D. W., Lee, E. H., Oh, Y. H., & Noh, K. S. (2003). Hypothalamic-pituitary-drenal reactivity in boys with attention deficit hyperactivity disorder. *Yonsei Medical Journal*, *44*(6087), 614.

Howell, M. J. (2012). Parasomnias: an updated review. *Neurotherapeutics*, **9,** 753–75.

Imeraj, L., Antrop, I., Roeyers, H., Swanson, J., Deschepper, E., Bal, S., & Deboutte, D. (2012). Time-of-day effects in arousal: disrupted diurnal cortisol profiles in children with ADHD. *Journal of Child Psychology and Psychiatry*,*53*(7), 782-789.

Isaksson, J., Nilsson, K. W., Nyberg, F., Hogmark, Å., & Lindblad, F. (2012). Cortisol levels in children with attention-deficit/hyperactivity disorder. *Journal of psychiatric research*, *46*(11), 1398-1405.

Jensen, P. S., Martin, D., & Cantwell, D. P. (1997). Comorbidity in ADHD: implications for research, practice, and DSM-V. *Journal of the American Academy of Child & Adolescent Psychiatry*, *36*(8), 1065-1079.

Kaneko, M., Hoshino, Y., Hashimoto, S., Okano, T., & Kumashiro, H. (1993). Hypothalamic-pituitary-adrenal axis function in children with attention-deficit hyperactivity disorder. *Journal of Autism & Developmental Disorders*, *23*(1), 59-65.

Kirov, R., & Brand, S. (2014). Sleep problems and their effect in ADHD. *Expert review of neurotherapeutics*, *14*(3), 287-299.

Ma, L., Chen, Y. H., Chen, H., Liu, Y. Y., & Wang, Y. X. (2011). The function of hypothalamus–pituitary–adrenal axis in children with ADHD. *Brain Research*, *1368*, 159-162.

Mason, T. B. A., & Pack, A. I. (2007). Pediatric parasomnias. *Sleep-New York then Westchester-*, *30*(2), 141.

Matricciani, L. A., Olds, T. S., Blunden, S., Rigney, G., & Williams, M. T. (2012). Never enough sleep: a brief history of sleep recommendations for children. *Pediatrics*, *129*(3), 548-556.

Mindell, J. A., & Owens, J. A. (2003). Sleep problems in pediatric practice: clinical issues for the pediatric nurse practitioner. *Journal of Pediatric Health Care*, *17*(6), 324-331.

Owens, J. A., Spirito, A., & McGuinn, M. (2000). The Children's Sleep Habits Questionnaire (CSHQ): psychometric properties of a survey instrument for school-aged children. *Sleep-New York-*, *23*(8), 1043-1052.

Owens, J. A. (2005). The ADHD and sleep conundrum: a review. *Journal of Developmental & Behavioral Pediatrics*, *26*(4), 312-322.

Owens, J., Gruber, R., Brown, T., Corkum, P., Cortese, S., O'Brien, L., ... & Weiss, M. (2013). Future research directions in sleep and ADHD report of a consensus working group. *Journal of attention disorders*, *17*(7), 550-564.

Paavonen, E. J., Almqvist, F., Tamminen, T., Moilanen, I., Piha, J., Räsänen, E., & Aronen, E. T. (2002). Poor sleep and psychiatric symptoms at school: an epidemiological study. *European Child & Adolescent Psychiatry*, *11*(1), 10-17.

Papadopoulos, N., Sciberras, E., Hiscock, H., Mulraney, M., McGillivray, J., & Rinehart, N. (2015). The efficacy of a brief behavioral sleep intervention in school-aged children with ADHD and comorbid autism spectrum disorder. *Journal of Attention Disorders*, 1087054714568565.

Polanczyk, G., de Lima, M. S., Horta, B. L., Biederman, J., & Rohde, L. A. (2007). The worldwide prevalence of ADHD: a systematic review and meta-regression analysis. *American Journal of Psychiatry*, *164*(6), 942-948.

Pollak, Y., Shomaly, H. B., Weiss, P. L., Rizzo, A. A., & Gross-Tsur, V. (2010). Methylphenidate effect in children with ADHD can be measured by an ecologically valid continuous performance test embedded in virtual reality. *CNS spectrums*, *15*(02), 125-130.

Prehn-Kristensen, A., Göder, R., Fischer, J., et al. (2011). Reduced sleep-associated consolidation of declarative memory in attention-deficit/ hyperactivity disorder. *Sleep Med, 12(7),* 672-9

Prehn-Kristensen, A., Munz, M., Molzow, I., Wilhelm, I., Wiesner, C. D. & Baving, L. (2013). Sleep promotes consolidation of emotional memory in healthy children but not in children with attention-deficit hyperactivity disorder. *PLoS One*, **8,** e65098.

Quach, J., Hiscock, H., Ukoumunne, O. C., & Wake, M. (2011). A brief sleep intervention improves outcomes in the school entry year: a randomized controlled trial. *Pediatrics*, *128*(4), 692-701.

Raven, J., Raven, J. C., & Court, J. H. (1998). *Manual for Raven's Progressive Matrices and Vocabulary Scales*. Section 2: The Coloured Progressive Matrices. San Antonio, TX: Harcourt Assessment.

Rodopman-Arman, A., Perdahli-Fis, N,. Ekinci, O. & Berkem, M. (2011). Sleep habits, parasomnias and associated behaviors in school children with attention deficit hyperactivity disorder (ADHD). *Turk J Pediatr*, **53,** 397–403.

Russell, G., Rodgers, L. R., Ukoumunne, O. C., & Ford, T. (2014). Prevalence of parent-reported ASD and ADHD in the UK: findings from the Millennium Cohort Study. *Journal of Autism and Developmental Disorders*, *44*(1), 31-40.

Sawyer, A. C., Clark, C. R., Keage, H. A., et al. (2009). Cognitive and electroencephalographic disturbances in children with attention- deficit/hyperactivity disorder and sleep problems: new insights. *Psychiatry Research,170(2-3),*183-91.

Smaldone, A., Honig, J. C., & Byrne, M. W. (2007). Sleepless in America: inadequate sleep and relationships to health and well-being of our nation's children. *Pediatrics*, *119*(Supplement 1), S29-S37.

Spruyt, K., & Gozal, D. (2011). Sleep disturbances in children with attention-deficit/hyperactivity disorder. *Expert review of neurotherapeutics*, *11*(4), 565-577.

Stephens, R. J., Chung, S. A., Jovanovic, D., et al. (2013). Relationship between polysomnographic sleep architecture and behavior in medication-free children with TS, ADHD, TS and ADHD, and controls. *J Dev Behav Pediatr, 34(9),* 688-96

Sung, V., Hiscock, H., Sciberras, E. & Efron, D. (2008). Sleep problems in children with attention- deficit/hyperactivity disorder: prevalence and the effect on the child and family. *Arch Pediatr Adolesc Med;162(4*), 336-42.

Yoon, S. Y., Jain, U., & Shapiro, C. (2012). Sleep in attention-deficit/hyperactivity disorder in children and adults: past, present, and future. *Sleep Med Rev, 16*, 371–88.

Highlights

1. Poor sleep affects developmental subgroups in different ways.
2. Poor sleep in TD children predicted increased ADHD-like behaviours, with no relation to performance on an attentional task.
3. Poor sleep in ADHD children returned persuasive trends in predicting poorer attentional control, even in a low sample.
4. Given concerns over the modern child's sleep profile, both findings make specific contributions towards a) the debate on the over-diagnosis of ADHD, b) promoting sleep-based interventions as a route to mitigating symptoms of ADHD fundamental to optimal child development and academic success.