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Relations between caregiver-report of sleep and executive function problems in children with autism spectrum disorder and attention-deficit/hyperactivity disorder

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ABSTRACT

Background: Many children with autism spectrum disorder (ASD) experience comorbid symptoms of attention-deficit/hyperactivity disorder (ADHD). Additionally, children with ASD and ADHD often have sleep disturbances and deficits in executive functioning (EF). In typical development, sleep disturbances are causally linked to EF deficits and exacerbate ADHD-like symptoms.

Aim: The aim of this study was to determine whether caregiver-report sleep and EF difficulties predict ADHD symptoms in children with ASD.

Methods: Caregiver-report of child sleep, EF, and ADHD symptom severity was collected for 101 children with ASD, 7–11 years of age. Hierarchical linear regressions tested the independent and interactive effects of sleep and EF in predicting ADHD symptoms.

Results: Children with ASD were more likely to have symptoms of ADHD if they experienced both sleep and EF difficulties. Children with difficulties in working memory were particularly at risk for clinically significant symptoms of ADHD. Notably, however, sleep did not mediate or moderate the relation between working memory and ADHD symptoms in this sample, suggesting that these variables act through independent mechanisms to increase vulnerability for comorbidity.

Conclusions: These results have clinical significance as sleep and EF deficits may identify an ASD subgroup that is at increased risk for a comorbid ADHD diagnosis.

What this paper adds?

30–80% of children with autism spectrum disorder (ASD) have comorbid attention-deficit/hyperactivity disorder (ADHD). Children with ASD and ADHD often experience sleep disturbances and deficits in executive functioning. Whether sleep and executive functioning difficulties contribute to ADHD symptoms in children with ASD is unknown. The results of this study indicate that symptoms of ADHD were more common among children with ASD who had caregiver-reported difficulties with sleep and executive functioning. Children with difficulties with working memory were particularly at risk for clinically significant symptoms of ADHD.

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Importantly, however, sleep and working memory did not interact to predict ADHD symptoms in this sample, suggesting that these variables act through independent mechanisms to increase vulnerability for comorbidity.

1. Introduction

Attention-deficit/hyperactivity disorder (ADHD) is a common comorbid condition reported among children with autism spectrum disorder (ASD; Simonoff et al., 2008), as approximately 30–80% of children with ASD have concurrent symptoms of ADHD (Rommelse, Franke, Geurts, Hartman, & Buitelaar, 2010). Alongside the hallmark symptoms of these disorders, children with ADHD and ASD often experience sleep disturbances (via caregiver-report: Thomas, Lycett, Papadopoulos, Sciberras, & Rinehart, 2015). Caregivers report sleep problems in 25–50% of children diagnosed with ADHD and in 40–86% of children with ASD (Maski & Kothare, 2013). Across these populations, the types of sleep problems reported are similar: children with ADHD and ASD have high rates of bedtime resistance, delayed sleep onset, and increased night wakings (Thomas et al., 2015).

In addition to sleep difficulties, children with ADHD and ASD have impaired executive function (EF; Barkley, 1997; Gilotty, Kenworthy, Sirian, Black, & Wagner, 2002; Gioia, Isquith, Guy, & Kenworthy, 2000). Executive function refers to a set of higher order cognitive functions, including working memory, inhibition, and set-shifting abilities, which rely upon top-down control over actions and behaviors. A growing body of literature suggests that deficits in EF contribute to developmental psychopathology, namely symptoms of ADHD (Barkley, 1997) and ASD (Russell, 1997). Specifically, performance deficits have been identified in both groups across a variety of working memory (WM) tasks (Goldberg et al., 2005; Martinussen, Hayden, Hogg-Johnson, & Tannock, 2005; Wang et al., 2017), and are associated with symptoms of inattention (Tillman, Eninger, Forssman, & Bohlin, 2011) and impulsivity (Raiker, Rapport, Kofler, & Sarver, 2012). Taken together, this evidence suggests that difficulties in EF, particularly WM, may contribute to comorbidity of ADHD symptoms in children with ASD.

In typical development, insufficient sleep is linked to difficulties in EF measured subjectively and objectively (Short et al., 2018; Vriend & Davidson, 2015). For example, children characterized as “poor sleepers” via actigraphy performed worse on computer-based measures of inhibition when compared to children described as “good sleepers” (Sadeh, Gruber, & Raviv, 2002). Using similar methods, WM deficits were also associated with poor sleep quality (Steenari et al., 2003). Similarly, performance on set-shifting tasks was impaired among a sample of school-aged children that were experimentally sleep restricted (Randazzo, Muehlbach, Schweitzer, & Walsh, 1998). Alongside EF impairments, insufficient sleep also contributes to behavioral difficulties that resemble symptoms of ADHD (Dahl, 1996). Collectively, these findings and others have inspired an emerging area of research focused on relations between sleep and EF deficits in developmental disorders (Deliens, Leproult, Schmitz, & Destrebecqz, 2015; Maski & Kothare, 2013; Weiss, Craig, Davies, Schibuk, & Stein, 2015). Whether sleep and EF interact to contribute to comorbidity of these disorders is of particular interest.

1.1. Current study

The objective of this study was to determine whether sleep and EF predicted comorbid ADHD symptoms in children with ASD. Although sleep and EF deficits are common in children with ASD and ADHD, the independent and interactive contributions of these factors in predicting comorbidity of these developmental disorders is unknown. To test this, the independent effects of sleep and EF were evaluated in relation to ADHD symptoms in this sample. Mediation and moderation models were then tested to evaluate interactive effects of sleep and EF.

Actigraphy and polysomnography are preferred measures of sleep quantity and quality (Lambert et al., 2013). However, these measures are expensive and may be difficult to use in children with ASD due to sensory sensitivities and/or noncompliance (Moore, Evans, Hanvey, & Johnson, 2017). Relatedly, objective measures of EF in children with ASD often yield inconsistent results (Van Eylen, Boets, Steyaert, Wagemans, & Noens, 2015). Thus, as a first step towards understanding relations between sleep and EF in children with comorbidities, subjective measures of sleep and EF were obtained from the caregivers and teachers of a large, well-characterized sample of children with ASD. We hypothesized that symptoms of ADHD would be greater in children with sleep and EF deficits. If this hypothesis is supported, these findings can be used to generate additional hypotheses that can be empirically tested with objective measures in future studies.

2. Methods

2.1. Participants

As part of a larger study, children with an existing diagnosis of ASD were recruited from participant registries and community sources. Caregivers of interested families participated in a phone screening interview to determine their child’s eligibility. Diagnosis of ASD was confirmed via the Autism Diagnostic Interview-Revised (ADI-R; Rutter, LeCouteur, & Lord, 2015) and the Autism Diagnostic Observation Schedule (ADOS-2; Gotham, Risi, Pickles, & Lord, 2007). Exclusion criteria included a Wechsler Abbreviated Scale of Intelligence (WASI-2; Wechsler, 2013) IQ < 80, a history of seizures or serious head injury, or a known neurological or genetic disorder (as reported by caregivers). Of the 102 children with ASD enrolled in the larger study, datasets from 101 children (14 females; $M_{age} = 9.13 \pm 1.38$ years) were used in the current analysis, as one caregiver chose not to respond to questionnaires. The unequal gender distribution in our sample was expected as developmental disorders are more prevalent in males versus females (Baio et al., 2018). Moreover, male predominance is greater among high IQ ASD populations (Banach et al., 2009; Brugha et al., 2011;

Yeargin-Allsopp et al., 2003).

2.2. Standardized questionnaires

Children's Sleep Habits Questionnaire (CSHQ). The CSHQ reliably detects disordered sleep in school-aged children ($\alpha = 0.88$, see Goodlin-Jones, Sitnick, Tang, Lui, & Anders, 2008; Owens, Spirito, & McGuinn, 2000). This measure is also commonly used in samples of children with ASD (Malow et al., 2006). In the current study, the Total CSHQ Score was used to broadly index sleep health. CSHQ subscales (i.e., Bedtime Resistance, Sleep Duration, Night Wakings, Daytime Sleepiness, Sleep Onset Delay, Sleep Anxiety, Parasomnias, Sleep Disordered Breathing, and Daytime Sleepiness) were also evaluated to determine whether specific facets of sleep health were related to ADHD outcomes.

Behavioral Rating Inventory of Executive Function (BRIEF). The BRIEF reliably assesses EF in children 5–18 years of age ($r = 0.76$ to 0.85 across clinical scales, see Gioia et al., 2000) and is commonly used to index EF in children with ASD (Gilotty et al., 2002). The BRIEF Metacognition Index and corresponding subscales (i.e., WM, Initiation, Planning/Organization, Organization of Materials, and Monitoring) were tested to determine if deficits in specific domains of metacognition predicted ADHD outcomes in this group.

Child Behavior Checklist (CBCL). The CBCL reliably assesses psychopathology in children 6–18 years of age (test-retest $\alpha = 0.63$ to 0.97, see Achenbach & Rescorla, 2001) and is also used in ASD research (Malow et al., 2006). The ADHD subscale was used to assess comorbid ADHD symptoms in the current sample.

2.3. Procedure

All procedures were approved by the Institutional Review Boards at the University of Washington (IRB-42650C) and Boston Children's Hospital (IRB-P00014188). Caregivers completed the CSHQ, BRIEF, and CBCL to measure their child's sleep health, EF, and ADHD symptoms, respectively. Teacher-report on the BRIEF was also available for 52 children (8 females; $M_{age} = 9.25 \pm 1.37$ years). On each measure, reporters were instructed to rate the frequency of the child's behavior using 3-point Likert scales. Caregivers also provided information about their child's medication use. Of the 101 children included in our analyses, 33 children (4 females; $M_{age} = 9.64 \pm 1.47$ years) were taking medication(s) for ADHD symptom management.

2.4. Data analyses

Statistical analyses were performed in SPSS Version 23.0 (IBM Corp). Questionnaires were scored following standard procedures, such that responses to individual items were collapsed into Index and subscale scores. In statistical analyses, raw scores were used for the CSHQ whereas T-Scores were used for the BRIEF and CBCL.[1] Higher scores on all measures reflect greater impairment.

In preliminary analyses, we examined partial correlations (controlling for child age and Full Scale IQ from 4 subtests) between the predictor and outcome variables of interest. Primary analyses included a series of hierarchical linear regressions that tested whether sleep problems and EF deficits predicted ADHD symptom severity in children with ASD.

The cumulative Total CSHQ Score was the primary predictor variable used to index sleep. CSHQ subscales (i.e., Bedtime Resistance, Sleep Duration, Night Wakings, Sleep Onset Delay, Sleep Anxiety, Parasomnias, Sleep Disordered Breathing, and Daytime Sleepiness) were then evaluated in a follow-up linear regression to determine whether specific domains of sleep problems predicted ADHD outcomes.

Because WM deficits have been identified in both ASD and ADHD (Goldberg et al., 2005; Martinussen et al., 2005; Wang et al., 2017), the BRIEF Metacognition Index, which includes the WM subscale, was the primary predictor variable used for EF. Subscales within the Metacognition Index (i.e., WM, Initiation, Planning/Organization, Organization of Materials, and Monitoring) were then evaluated separately in a follow-up linear regression to evaluate the impact of specific EF deficits. To address potential confounds related to reporter bias, both caregiver- and teacher-report of WM were evaluated separately in all models.

Next, mediation and moderation models were tested to better understand how sleep problems contributed to the relation between WM and ADHD symptoms in children with ASD. Given that sleep problems contribute to EF deficits in typical development (Randazzo et al., 1998; Sadeh et al., 2002), a mediation model tested whether sleep problems accounted for (i.e., mediated) the relation between EF deficits and ADHD symptoms in children with ASD. Comparably, a moderation model tested whether sleep problems strengthened (i.e., moderated) the relation between EF and ADHD, thereby conferring increased risk for comorbidity.

Finally, exploratory logistic regressions were run to evaluate whether or not sleep and EF predicted clinically significant ADHD problems in our ASD sample. T-Scores from the CBCL were used to dichotomize children with (T-Score > 65) and without ADHD symptoms (T-Score ≤ 65). Because there is little consensus on the best way to quantify ADHD symptoms in children with ASD, a T-Score of 65 was used to identify children within the 'borderline clinical range' of ADHD. Importantly, however, we also re-ran these regressions using the more conservative T-Score of 70 to dichotomize these groups into children with and without clinically

[1] To our knowledge, CSHQ scores have not been standardized for children with ASD or otherwise. As such, raw scores from the CSHQ were used, consistent with previous studies assessing sleep in developmental populations with and without ASD (Malow et al., 2006). T-scores were used for the BRIEF and CBCL. To confirm that the use of raw versus T-score did not alter our results, we re-ran the primary analysis using raw scores for all measures (CSHQ, BRIEF WM, and CBCL ADHD subscale) and results were unchanged. However, to aid in interpretation of findings with the BRIEF and CBCL for other ASD samples, we report results with T-scores in the text.

Table 1
Participant demographics and descriptive statistics.

	Caregiver-Report (n = 101) Mean (SD)	Teacher-Report (n = 52) Mean (SD)
Participant Demographics[a]		
Age (years)	9.13 (1.38)	9.25 (1.37)
Gender (Females: Males)	14:87	8:44
IQ (WASI-2 Full Scale IQ; 4 subtests)	106.12 (14.36)	107.46 (14.74)
ASD Symptom Severity (ADOS-2 Calibrated Severity Score)	8.47 (1.69)	8.25 (1.84)
ADHD Symptom Severity (CBCL ADHD T-Score)	62.82 (7.88)	63.98 (7.68)
EF Measures (BRIEF T-Scores)[a, b]		
Global Executive Composite Index	68.29 (10.78)	68.02 (13.99)
Behavioral Regulation Index	67.02 (12.20)	70.10 (11.77)
Inhibition	64.72 (12.31)	66.22 (11.29)
Shifting	68.16 (12.82)	70.37 (13.41)
Emotional Control	62.25 (12.18)	68.54 (14.66)
Metacognition Index	67.25 (10.57)	67.06 (10.26)
Working Memory	67.02 (10.23)	66.29 (11.27)
Initiation	64.53 (10.36)	66.04 (10.46)
Planning/Organization	65.82 (11.47)	64.56 (10.89)
Organization of Materials	59.26 (9.79)	63.48 (13.79)
Monitoring	64.04 (10.56)	67.67 (11.52)
CSHQ Measures (Raw Scores)[a]		
Total CSHQ Score	45.97 (7.56)	46.38 (7.47)
Bedtime Resistance	8.33 (2.65)	8.46 (2.73)
Sleep Onset Delay	1.59 (0.74)	1.54 (0.70)
Sleep Duration	4.10 (1.51)	4.21 (1.53)
Sleep Anxiety	6.10 (1.96)	6.27 (2.06)
Night Wakings	3.93 (1.37)	4.10 (1.43)
Parasomnias	8.73 (1.82)	8.65 (1.57)
Sleep-Disordered Breathing	3.28 (0.91)	3.19 (0.74)
Daytime Sleepiness	12.95 (3.53)	13.12 (3.65)

Notes: Caregiver-report [a]; Teacher-report [b.]

significant ADHD symptoms and results were consistent with those reported with T-Scores of 65. Group dichotomy was entered as the dependent variable in logistic regressions with the control and predictor variables discussed below.

In all models, CBCL ADHD T-Score was entered as the dependent variable. Child age and Full Scale IQ were entered as control variables in the first block. Predictor variables (e.g., Total CSHQ Score and BRIEF measures) were simultaneously entered in the second block. Continuous control and predictor variables (i.e., child age, IQ, CSHQ and BRIEF measures) were centered at the sample mean to facilitate interpretation of effects. In significant models, unstandardized residuals were plotted against the adjusted predicted values to confirm that assumptions of (1) normality, (2) linearity, and (3) homoscedasticity were not violated.

ADHD medications are associated with both sleep (Konofal, Lecendreux, & Cortese, 2010) and EF (Kempton et al., 1999). To determine whether medication influenced our results, significant models were re-run with ADHD medication use entered as a dichotomous variable (i.e., yes/no) in the first block. Unsurprisingly, ADHD medication significantly predicted ADHD symptoms in most models. Results that were altered by the inclusion of ADHD medication use, and models in which ADHD medication use was *not* a significant predictor, are noted in the text.

3. Results

3.1. Descriptive statistics and correlations

Demographic information and descriptive statistics are outlined in Table 1. Partial correlations between primary predictor and outcome variables are presented in Table 2. Controlling for child age and IQ, sleep problems (Total CSHQ) were positively correlated

Table 2
Partial correlations between primary predictor and outcome variables, controlling for child age and IQ.

	Variables	1	2	3	4	5	6
1.	Total CSHQ Score	–					
2.	BRIEF Metacognition Index (Caregiver-Report)	0.15	–				
3.	BRIEF Metacognition Index (Teacher-Report)	0.06	0.30*	–			
4.	BRIEF Working Memory (Caregiver-Report)	0.14	0.80***	0.24	–		
5.	BRIEF Working Memory (Teacher-Report)	0.15	0.36**	0.83***	0.35**	–	
6.	CBCL ADHD T-Score	0.29**	0.51***	0.31*	0.57***	0.32*	–

$p \leq 0.05$*; $p \leq 0.01$**; $p \leq 0.001$*.

Table 3
Summary of regression analysis with Metacognition and Total CSHQ Score as predictor variables of ADHD symptoms.

	β	B (SE)
Variables		
Age	0.09	0.04 (0.04)
IQ	−0.03	−0.02 (0.05)
BRIEF Metacognition Index	0.48***	0.36 (0.07)
Total CSHQ Score	0.21**	0.22 (0.09)
R^2	0.04	
ΔR^2	0.30***	

Notes: β = standardized regression coefficient; B = unstandardized regression coefficient; SE = standard error; R^2 presented from step 1 mode; ΔR^2 = change in model fit when BRIEF Metacognition Index and Total CSHQ Score are added. All other results presented from the final model.
*p ≤ 0.01**; p ≤ 0.001***.*

Table 4
Summary of regression analysis with caregiver-report of Working Memory and Total CSHQ Score as predictor variables of ADHD symptoms.

	β	B (SE)
Variables		
Age	0.11	0.05 (0.04)
IQ	0.01	< 0.01 (0.04)
BRIEF Working Memory	0.54***	0.42 (0.06)
Total CSHQ Score	0.21**	0.22 (0.08)
R^2	0.04	
ΔR^2	0.36***	

Notes: β = standardized regression coefficient; B = unstandardized regression coefficient; SE = standard error; R^2 presented from step 1 mode; ΔR^2 = change in model fit when BRIEF Working Memory Subscale and Total CSHQ Score are added. All other results presented from the final model.
*p ≤ 0.01**; p ≤ 0.001***.*

with ADHD symptom severity (CBCL ADHD T-Score; $r = 0.29$, $p \leq 0.01$; Table 2). EF deficits, reported by caregivers and teachers, were also positively correlated with ADHD symptom severity ($rs \geq 0.31$, $ps \leq 0.03$). Consistent with our hypothesis, these data indicate that ADHD symptom severity was greater in children with higher rates of sleep problems and EF deficits.

Sleep problems were not correlated with EF deficits as reported by caregivers or teachers ($rs \geq 0.06$, $ps \leq 0.14$; Table 2). Importantly, however, caregiver- and teacher-report of EF were positively correlated ($rs \geq 0.30$, $ps \leq 0.03$).

3.2. Global sleep problems and EF deficits

A hierarchical linear regression was run to determine whether sleep problems (Total CSHQ) and EF deficits (BRIEF Metacognition Index) predicted ADHD symptom severity in children with ASD. In this model, child age and IQ were entered as control variables in the first block. Total CSHQ and the BRIEF Metacognition Index were entered as primary predictor variables simultaneously in the second block. CBCL ADHD T-Score was the dependent variable. Among children with ASD, greater Total CSHQ ($\beta = 0.21$, $p = 0.01$) and Metacognition Index ($\beta = 0.48$, $p \leq 0.01$) scores predicted greater ADHD symptom severity (Table 3), consistent with the results of our correlations (Table 2).

To determine whether specific EF deficits contributed to ADHD outcomes, we performed a follow-up regression with all of the Metacognition Index subscales (i.e., WM, Initiation, Planning/Organization, Organization of Materials, and Monitoring) entered simultaneously in the second block (in place of the BRIEF Metacognition Index). The results of this model indicated that WM accounted for the most variance in ADHD symptoms in this sample (WM: $\beta = 0.39$, $p = 0.001$; other Metacognition index subscales: βs between -0.26 and 0.25). A subsequent regression with only the WM subscale included in the model (in addition to control variables and Total CSHQ) indicated that greater deficits in WM ($\beta = 0.54$, $p \leq 0.01$) and sleep (Total CSHQ Score: $\beta = 0.21$, $p = 0.01$) significantly predicted greater ADHD symptoms (Table 4).

To address potential confounds related to reporter bias, this regression (control variables, Total CSHQ, and WM) was re-run with teacher-report of WM instead of caregiver-report (all other variables remained the same). Consistent with the results from caregiver-report, greater deficits in WM were marginally predictive of greater ADHD symptoms ($\beta = 0.26$, $p = 0.06$; Table 5). Greater sleep

Table 5
Summary of regression analysis with teacher-report of Working Memory and Total CSHQ Score as predictor variables of ADHD symptoms.

	β	B (SE)
Variables		
Age	0.11	0.05 (0.06)
IQ	−0.04	−0.02 (0.07)
BRIEF Working Memory	0.26[t]	0.17 (0.09)
Total CSHQ Score	0.39**	0.40 (0.13)
R^2	0.04	
ΔR^2	0.24**	

Notes: β = standardized regression coefficient; B = unstandardized regression coefficient; SE = standard error; R^2 presented from step 1 mode; ΔR^2 = change in model fit when BRIEF Working Memory Subscale and Total CSHQ Score are added. All other results presented from the final model.
p = 0.06 [t]; *p ≤ 0.01***.

problems significantly predicted ADHD symptoms ($\beta = 0.39$, $p \leq 0.01$).[2] Thus, it is unlikely that relations between WM and ADHD symptoms were a consequence of reporter bias. These results should be interpreted with caution, however, as teacher-report was only available for WM, associations between WM and ADHD symptoms were marginal ($p = 0.06$), and the sample size was notably smaller (approximately 51%).

3.3. Specific sleep problems and WM deficits

To determine whether specific sleep disturbances predicted ADHD symptoms in children with ASD, we performed a follow-up regression with all of the CSHQ subscales (i.e., Bedtime Resistance, Sleep Duration, Night Wakings, Daytime Sleepiness, Sleep Onset Delay, Sleep Anxiety, Parasomnias, Sleep Disordered Breathing, and Daytime Sleepiness) entered simultaneously in the second block (in place of the Total CSHQ Score; all other variables remained the same). In this model, CSHQ subscales did not significantly predict ADHD symptoms (*p*sn ≥ 0.14).

When teacher-report of WM deficits was included in this model (in place of caregiver-report), similar results were obtained such that CSHQ subscales did not predict ADHD symptoms (*p*s ≥ 0.15). Taken together, these findings indicate that global sleep problems contributed to ADHD symptoms in this sample, rather than the specific sleep disturbances indexed by subscales of the CSHQ.

3.4. Mediation and moderation models

Next, a mediation model tested whether relations between WM deficits and ADHD symptoms were a function of sleep problems. In accordance with Baron and Kenny (1986), multiple linear regressions were run to test mediation. In each regression, control variables were entered in the first block and the predictor variable specific to each step in the mediation model was entered in the second block. As expected, WM and sleep independently predicted ADHD symptoms (ADHD symptoms regressed on WM: $\beta = 0.57$, $p \leq 0.01$; ADHD symptoms regressed on sleep: $\beta = 0.28$, $p \leq 0.01$). However, WM was not related to Total CSHQ in this sample (sleep regressed on WM: $\beta = 0.14$, $p = 0.18$), which indicated that the mediation model was not significant.

To evaluate the interactive effect of sleep and EF in predicting risk for ADHD symptoms, we also tested a moderation model. In this model, control variables were entered in the first block, sleep and WM were entered simultaneously in the second block, and the interaction between sleep and memory (Total CSHQ * WM) was entered in the third block. Although sleep and WM each predicted ADHD symptoms (*β*s ≥ 0.21, *p*s ≤ 0.01), the interaction term was not significant ($\beta = 0.04$, $p = 0.65$), suggesting that sleep and WM deficits confer risk for ADHD symptoms independently.

The results of these mediation (sleep regressed on WM: $\beta = 0.12$, $p = 0.40$; ADHD symptoms regressed on WM: $\beta = 0.34$, $p = 0.02$; ADHD symptoms regressed on sleep: $\beta = 0.42$, $p \leq 0.01$) and moderation models (Total CSHQ * WM: $p = 0.40$) were unchanged when teacher-report of WM was used in place of caregiver-report.

3.5. Clinical outcomes

Finally, logistic regression explored whether sleep and WM problems predicted clinically significant comorbid ADHD symptoms in our ASD sample. In accordance with the standard scoring procedure, 39 children had clinically significant ADHD symptoms (CBCL ADHD T-Score > 65) and 62 did not. Group dichotomy was entered as the dependent variable in this model with control variables entered in the first block and sleep and WM entered simultaneously in the second block. Both sleep problems ($p = 0.02$, *Exp (B)* = 1.08) and WM deficits ($p \leq 0.01$, *Exp(B)* = 1.13) predicted clinically significant ADHD problems: for every one-unit increase

[2] Results were unchanged when ADHD medication use was included in this model (Total CSHQ: $\beta = 0.35$, $p \leq 0.01$; teacher-report of WM subscale: $\beta = 0.26$, $p = 0.05$). However, ADHD medication did not significantly predict ADHD symptoms in this model ($\beta = 0.23$, $p = 0.09$).

on the Total CSHQ and WM scales, children with ASD were 1.08 and 1.13 times more likely to have a comorbid ADHD diagnosis, respectively.[3]

Of the 52 children with teacher-report of WM available, 23 children had clinically significant ADHD symptoms whereas 29 did not. Here too, sleep problems ($p \leq 0.01$, $Exp(B) = 1.15$) significantly predicted group outcome: for every one-unit increase on the Total CSHQ scale, children with ASD were 1.15 times more likely to have a comorbid ADHD diagnosis. Unlike the results obtained with caregiver-report, teacher-report of WM did not predict ADHD comorbidity in this subsample ($p = 0.22$, $Exp(B) = 1.04$).[4]

4. Discussion

The current study was the first to examine the effects of sleep and EF in predicting comorbid ADHD symptoms among children with ASD. Results indicated that both caregiver-reported sleep problems and WM deficits predict greater comorbid ADHD symptoms. Provided the high rates of sleep problems and WM difficulties among children with ASD, our findings suggest that having both impairments may mark an ASD subgroup greater risk for comorbid diagnosis. Importantly, these relations were not related to child age or IQ and were observed even after controlling for ADHD medication use, suggesting that sleep and EF contribute to variance in ADHD symptomology among children with ASD beyond the proportion accounted for by other factors.

As expected, sleep problems were prevalent in our sample, as ˜68% of children met criteria for having clinically significant sleep disturbances (CSHQ Total Score > 41, per Owens et al., 2000). Analysis of the CSHQ subscales indicated that the *cumulative* effect of sleep problems (Total CSHQ Score) outweighed the independent effects specific sleep problems had on ADHD outcomes in this sample. It is also possible that we detected a role of sleep only when assessing aggregate sleep problems (Total CSHQ Score rather than subscales) due to the variety sleep problems that affected our sample. Objective measures of sleep, namely actigraphy and polysomnography, are needed to better understand how sleep quantity and quality contributes to comorbidity of developmental disorders.

In typical development, insufficient sleep is related to WM deficits (Steenari et al., 2003) and ADHD-like behaviors (Fallone, Acebo, Seifer, & Carskadon, 2005; Gruber Cassoff, Frenette, Wiebe, & Carrier, 2012; Sadeh, Gruber, & Raviv, 2003). Collectively, these data suggest that sleep difficulties may contribute to, or exacerbate, WM deficits that are common among individuals with ADHD. However, the results of the current study indicate that sleep problems do not mediate or moderate relations between WM and ADHD symptoms in our ASD sample. Instead, our results suggest that sleep and EF independently confer risk for comorbidity with ADHD.

4.1. Limitations and future directions

Although the current study underscores the importance of sleep and EF in developmental disorders, there are notable limitations that should be addressed in future research. First, though our sample size is comparable to previously published papers, the models tested may have been underpowered. Therefore, replication with a larger, more diverse sample is needed. Relatedly, although the higher proportion of males in our sample was expected (Baio et al., 2018), additional studies assessing relations between sleep, EF, and comorbidities in females with ASD are needed.

Second, given that the current study relied upon caregiver-report of child outcomes, our results may have been susceptible to reporter biases. Importantly, similar results were obtained when teacher-report of WM deficits was used in place of caregiver-report, suggesting that this influence is unlikely. Nonetheless, it is possible that both caregivers and teacher-report were influenced by a negative halo effect, and consequently reported higher rates of sleep or WM problems in children with other behavioral concerns. Relatedly, items in the WM subscale of the BRIEF were closely related to items in the ADHD subscale of the CBCL. As such, these results should be interpreted with caution. Additional studies utilizing objective assessments are needed to address these potential confounds. Objective measures may also help differentiate the etiology and severity of sleep and EF deficits in children with ASD, ADHD, and comorbid diagnoses.

Third, we are unable to infer directionality or causality with the current dataset. Intervention studies suggest that improving sleep quality (Hiscock et al., 2015) and EF (Tamm, Nakonezny, & Hughes, 2014) improves symptoms on ADHD. Provided the results of the current study, these findings suggest that similar interventions may mitigate symptoms of ADHD in children with ASD. A recent study by Papadopoulos et al. (2015) supports this hypothesis, as behavioral outcomes were improved in a sample of school-aged children with comorbid ASD and ADHD following a brief, sleep hygiene intervention.

An additional recommendation for future research is the inclusion of an emotional regulation assessment. Sleep and EF are independently associated with emotional regulation. Specifically, sleep problems impair both EF (Short et al., 2018; Vriend & Davidson, 2015) and emotional regulation (Palmer & Alfano, 2017), whereas EF skills promote adaptive emotional regulation (Zelazo & Cunningham, 2007). As children with ASD and ADHD are reported to have difficulty with emotional regulation (Graziano & Garcia, 2015; Mazefsky et al., 2013), it is possible that sleep problems obstruct emotional regulation by reducing the efficacy of EF skills, particularly among children with ASD and comorbid symptoms of ADHD. Unfortunately, emotional regulation was not assessed in the

[3] Results were unchanged when ADHD medication use was included in the model (Total CSHQ: $p = 0.03$, $Exp(B) = 1.08$; caregiver-report of WM: $p \leq 0.01$, $Exp(B) = 1.14$). The effect of ADHD medication use was only marginal ($p = 0.07$, $Exp(B) = 2.61$).

[4] When ADHD medication use was included in the model, only sleep problems significantly predicted clinically significant ADHD problems (Total CSHQ: $p \leq 0.01$, $Exp(B) = 1.15$; teacher-report of WM: $p = 0.18$, $Exp(B) = 1.05$; ADHD medication use: $p = 0.14$, $Exp(B) = 3.19$).

current study, which may be why independent, but not interactive, effects were reported. Future studies should incorporate measures of emotional regulation to better understand the interplay between sleep and EF in this population.

5. Conclusions

To our knowledge, this study was the first to assess relations between sleep, EF, and ADHD symptoms among children with ASD. The results indicated that children with ASD who had both caregiver-reported sleep difficulties and EF deficits were at increased risk for comorbid symptoms of ADHD. These findings have important clinical implications, as sleep and EF deficits may be markers of an ASD subgroup that is at increased risk for a comorbid ADHD diagnosis. If these findings are replicated, interventions that target sleep and EF may be applied to reduce symptom heterogeneity and improve treatment outcomes.

Declaration of Competing Interest

The authors report no conflicts or competing financial interest.

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