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ORIGINAL ARTICLE

Investigating longitudinal associations between parent reported sleep in early childhood and teacher reported executive functioning in school-aged children with autism

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Abstract

Up to 80% of children with autism spectrum disorder (ASD) experience sleep disturbance. Poor sleep impairs executive functioning (EF), a lifelong difficulty in ASD. Evidence suggests EF difficulties in ASD are exacerbated by poor sleep. We examine whether early childhood sleep disturbances are associated with worsening EF trajectories in school-aged children with ASD. A subsample (n = 217) from the *Pathways in ASD* longitudinal study was analyzed. The *Children's Sleep Habits Questionnaire* captured sleep duration, onset, and night awakenings before age 5 (mean = 3.5 years). Metacognition (MI) and Behavioral Regulation (BRI) indices, on the *Teacher Behavior Rating Inventory of Executive Functioning*, were used to measure cognitive and affective components of EF respectively at four time-points (7.8–11.8 years). We applied latent growth curve models to examine associations between sleep and EF, accounting for relevant covariates, including school-age sleep (mean = 6.7 years). Sleep traits had different age-related impacts on behavioral regulation, but not metacognition. Longer sleep onset at 3.5 years was associated with a worsening BRI difficulties slope (b = 2.07, p < 0.04), but conversely associated with lower BRI difficulties at 7.7 years (b = –4.14, p = 0.04). A longer sleep onset at 6.7 years was related to higher BRI difficulties at 7.7 years (b = 7.78, p < 0.01). Longer sleep duration at 6.7 years was associated with higher BRI difficulties at age 7.7 (b = 3.15, p = 0.01), but subscale analyses revealed shorter sleep duration at age 6.7 was linked to a worsening inhibition slope (b = –0.60, p = 0.01). Sleep onset is a robust early correlate of behavior regulation in children with ASD, whereas sleep duration is a later childhood correlate.

Statement of Significance

Executive functioning (EF) is a lifelong difficulty in autism, negatively impacting quality of life. Poor sleep in typically developing youth and youth with other neurodevelopmental disorders impairs EF. Despite up to 80% of children with autism experiencing sleep problems, the impact of sleep disturbances on EF development in autism is unknown. This is the first longitudinal study to examine if early childhood sleep disturbances commonly found in autism are related to worsening executive functioning later in school-age. Delayed sleep onset in toddlerhood and shorter school-age sleep duration reported by parents was linked to worsening EF development underlying behavior regulation, but not pure cognitive processes. We discuss methodological limitations and future directions needed to inform sleep-based interventions for atypical EF in autism.

Key words: sleep; autism; executive functioning; development; children

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Introduction

Sleep is disturbed in up to 80% of children with Autism Spectrum Disorder (ASD) [1]. Shorter sleep duration and traits related to insomnia, including difficulty falling asleep and frequent night awakenings, are most commonly reported [2]. Problematic sleep in ASD is found early in infancy and persists into adulthood [3, 4]. In typical development, sleep problems, particularly night awakenings, are commonly reported in infancy, but then decline throughout childhood [5–8]. Although longitudinal investigations of sleep patterns in ASD are sparse, evidence suggests that compared to typically developing (TD) infants, infants diagnosed with ASD or with increased ASD symptomology are more likely to experience sleep problems (e.g. night awakenings, sleep onset, "general sleep problems") [3, 9, 10], and contrary to observations in typical development, these sleep problems are documented to increase during childhood in ASD [3]. This observation extends to individuals with increased ASD symptomatology, who are documented to develop more sleep problems from school-age to adolescence [11]. Further, one prospective cohort study found from 30 months to 11 years of age, children with ASD have consistently shorter sleep durations compared to TD children [12].

Poor sleep in children impairs executive functioning (EF) [13], a well-documented lifelong difficulty in individuals with ASD [14]. EF is an umbrella term that describes higher order cognitive processes that enable individuals to engage in deliberate, goal-directed thoughts and actions and is known to play a pivotal role in early social-cognitive development [15]. These processes are commonly condensed into three core components: working memory, inhibitory control and cognitive flexibility [15]. Behavioral regulation and metacognition are evaluated on frequently used EF questionnaires [16], to capture "hot" and "cold" EF respectively. Cold EF is suggested to encompass purely cognitive processes (e.g. planning and organizing), whereas hot EF refers to cognitive processes underlying the management of affective behavior (e.g. emotional control) [17].

In TD children, the first 5 years are marked by rapid attainment of EF skills, while more complex EF develops during middle childhood and adolescence [18]. There is evidence to support the distinct development of core EF components (for comprehensive review, see Best and Miller, 2010). Rudimentary inhibition, like self-control and delaying gratification, emerges early in development. By age 4, children successfully master simple response inhibition tasks and begin completing more complex tasks that require inhibition and a subsequent response (e.g. Luria's Hand Game, Go/No-Go, The Dimension Change Card Sort). Dramatic gains of inhibitory control occurring in toddler and preschool years, are then proceeded by slower improvements during school-age and adolescence. Advancements of complex inhibitory control also depends on integrating working memory abilities (e.g. remembering and executing rules). Unlike inhibitory control, temporarily maintaining and deploying memory develops linearly and gradually from preschool to adolescence. It is suggested that with age individuals improve on complex tasks that require processing more information, rather than the type of information processed (e.g. visual or verbal). Similar to working memory, improvements in shifting between tasks (cognitive flexibility), follows a protracted developmental course from preschool to adolescence. By 3 to 4 years of age, preschoolers can shift between two simple rules. Later in childhood, children are able to complete tasks requiring them to act and refine their responses based on multiple different rules. Like inhibitory control, complex cognitive flexibility tasks place greater demands on working memory, while performance accuracy and speed improve with age.

Our understanding of EF trajectories outlined above, have mainly derived from experimental and cold EF tasks. Hot and cold EF are dissociable early in childhood [19, 20] and are independently associated with academic and behavioral outcomes [21]. After preschool, hot and cold EF are found to mature independently into adolescence, with hot EF developing more slowly [22, 23]. In a normative sample from 5 to 18 years of age, general EF in everyday settings reported by parents on the commonly used Behavior Rating Inventory of Executive Function (BRIEF) [16], was shown to improve with age. However, when hot and cool abilities were looked at separately by Huizinga and Smidts, 2010 [24], behavioral regulation components of EF (hot) were the main contributors of overall EF improvement, as problems decreased in each age group. Contrastingly, metacognitive EF components (cool) remained stable across childhood, with the exception of working memory improvements from ages 5–8 to 9–11 years. As Rosenthal et al. [25] discuss, although these results indicating stable metacognitive ability may appear to contradict experimental studies finding age-dependent improvements of cold EF, the BRIEF captures problems in utilizing EF in everyday contexts. Hence, these BRIEF findings more likely reflect TD children adapting to increasing metacognitive demands, like planning, organizing and working memory, which occur during schooling (e.g. testing, multiple classrooms and teachers, etc).

Despite consistent documentation of everyday EF impairments in ASD across a wide age range [14], to our knowledge, only one longitudinal study on everyday EF abilities has been conducted. Parent-reported behavioral regulation and metacognition abilities in children with ASD aged 7–14 years did not significantly change when reported 2 years later, but at both time-points, children with ASD had poorer behavioral regulation and metacognition abilities compared to TD controls [26]. This study provides evidence that executive dysfunction appears to be stable in middle childhood and adolescence in ASD, periods when complex EF skills are refining. However, due to the wide age range of the sample, the study does not allow for any conclusions to be made about the developmental trajectory of EF in childhood and adolescence. Limited longitudinal investigations using experimental tasks provide mixed results, finding no cold EF gains from childhood to adolescence in ASD [27], while one study reported improvements of cold EF from early to middle childhood [28]. Reviews of cross-sectional studies of EF in ASD generally suggest that, whilst EF is consistently impaired compared to normative samples, there is linear development in EF throughout childhood and adolescence, with EF performance differences becoming less evident in adulthood [14, 29]. Currently, there are no investigations of EF trajectories and factors that may contribute to successful EF development across childhood in ASD. This is problematic because executive dysfunction in ASD is known to contribute to a poorer quality of life, increased psychiatric comorbidities, and adaptive functioning difficulties [30–32]. Hence, greater research effort should focus on characterizing EF trajectories in ASD and factors that contribute to EF difficulties early in development.

One possible factor underlying executive dysfunction in ASD is poor sleep. Numerous observational and experimental

studies with TD children as well as children with attention-deficit/hyperactivity disorder (ADHD), a highly co-morbid neurodevelopmental disorder in ASD, have associated poor sleep with deficits in hot and cold EF performance [13, 33, 34]. Developmental changes in typical EF occur in parallel to significant structural and functional changes in the prefrontal cortex, a core region subserving EF [17] and characterized by abnormalities in ASD [35]. Sleep deprivation negatively disrupts prefrontal cortex connectivity and it has long been hypothesized that the prefrontal cortex benefits from the regenerative role of sleep [36, 37]. It is well established that poor sleep and EF performance are both related to atypical prefrontal cortex activity [38, 39].

Arguably, developmental accounts of the interplay between sleep and EF from youth with traumatic brain injury (TBI) provide the most insightful evidence to draw from in the context of the frontal cortex. Incurring a TBI commonly affects frontal brain regions impairing prefrontal cortical functioning [40]. Both EF impairments and sleep disturbance are commonly reported in youth following mild or moderate/severe forms of TBI and seen to persist during long-term follow-ups [41]. Recent accounts suggest early brain damage to such frontal regions, compounded by sleep disturbances, results in poorer EF [42]. EF deficits in ASD may be caused by disruptions to the prefrontal cortex, which are in turn exacerbated by elevated sleep disturbance affecting the same neural pathways. If sleep disturbances occur at time-points associated with rapid age-related changes in neural circuitry responsible for the development of EF, this can be even more problematic, as it may inhibit the acquisition of basic EF skills. This is concerning because early attainment of basic EF skills is foundational to and predictive of successful EF performance later in life [15, 43].

Preliminary longitudinal evidence indicates shorter nocturnal sleep in infancy leads to poorer impulse control later in infancy and is associated with poorer complex EF performance later in preschool aged children [44, 45]. Further, it has been shown that insufficient sleep duration early in childhood predicts worse EF in later TD childhood, including behavioral regulation and metacognition impairments [46]. To date, there has been no longitudinal investigations into the relationship between sleep and EF in children with ASD, while only a few cross-sectional studies examining the impact of sleep on EF performance have been conducted. Reynolds et al [47]., found that elevated total sleep problems were correlated with worse behavioral regulation and metacognition reported by parents in 6- to 11-year-old children with comorbid ASD and ADHD. However, a subsequent multivariate analysis including IQ, ADHD and ASD severity, and other confounders, found no association between sleep and EF. A later study using similar measures with 101 children showed that general sleep problems reported by parents was not correlated with teacher and parent reported metacognition, however, in this ASD cohort having both a sleep problem and metacognition difficulties increased children's risk of developing ADHD symptoms [48]. Another study examining 477 children with ASD aged 1–15 years, found parent-reported total sleep problems were not associated with working memory on an experimental task [49]. More recently, an analyses of 6832 primary and secondary students reported sleep deficits, the difference between parents perceived sleep duration need for their child and children's nocturnal sleep duration, mediated the relationship between higher autistic traits and greater metacognition and behavioral regulation problems [50]. However, cross-sectional studies that cannot capture inter-individual variations across time, do not allow us to ascertain whether certain developmental periods when sleep problems manifest have a greater impact on various aspects of EF development. Further, many of these investigations have been limited to "total sleep problem" scores from parent-reported questionnaires, collapsing multiple sleep phenotypes. Investigations into specific sleep phenotypes (i.e. sleep onset, sleep duration, chronotype) are needed, as they have been shown to have variations in genetic underpinnings [51–54], as well as differential impacts on cognitive outcomes [44].

The aim of the current study was to examine whether early-reported sleep problems in toddlers/preschoolers with ASD are associated with later EF trajectories. Specifically, we hypothesized that shorter sleep duration, frequent night awakenings, and longer sleep onset at 3.5 years of age would be associated with worsening behavioral regulation and metacognition trajectories in 7.7- to 11.8-year-olds with ASD, as reported by teachers at four time-points.

Methods

Design and participants

Participants were enrolled in *Pathways in ASD*, a Canadian multisite longitudinal study examining the developmental trajectories in an inception cohort of children with ASD recruited across five provinces. The study was approved by the Research Ethics Boards at participating sites. Children in the study met the following inclusion criteria: (1) were 2–4 years of age at the time of diagnosis; (2) met diagnostic criteria for ASD on the *Autism Diagnostic Observation Schedule* (ADOS) [55] and in the social and one other domain of the *Autism* Diagnostic Interview-Revised [56]; and (3) met criteria for ASD at each phase of assessment on the DSM-IV-TR [57] according to clinicians with diagnostic expertise. Exclusion criteria included known genetic or chromosomal abnormalities, neuromotor disorders, and/or severe hearing and/or vision impairment.

From a total of 421 Pathways participants, 348 had complete sleep data at their time one (T1) visit. Three participants were excluded due to data input errors. EF data from at least one out of four time-points were available for 217 children, and thus comprise the sample reported in this study (mean age at T1 = 3.5 years; *SD* = 0.77) at T1. We compared the 217 participants included in the analysis to the 204 who did not have follow-up teacher data, presented in the Table S1. The 217 participants were significantly more likely to belong to the high income category (50.1% versus 37.3%, $p = 0.009$), had significantly higher cognitive standard scores (mean = 56.06, *SD* = 17.95 versus mean = 50.56, *SD* = 17.58; $p = 0.031$), and were more likely to be from the Montreal site ($p = 0.003$) and less likely to be from Edmonton ($p = 0.009$) or Vancouver ($p = 0.026$) (Hamilton site used as reference category). However, there were no significant differences on any of the T1 sleep variables, age of T1 assessment, or ADOS symptom severity between included and excluded participants. Descriptive data for the included sample can be found in Table 1.

Measures

Sleep.

Parent-reported sleep concerns were obtained using the *Children Sleep Habits Questionnaire (CSHQ)* [58], comprising 33 items and eight subscales. The frequency of each sleep behavior corresponds to responses of: 1= *never/rarely (0–1 nights per week)*, 2= *sometimes (2–4 nights per week)*, 3= *usually (5–7 nights per week)*. The CSHQ was used to assess sleep at T1 and at T4 when children were on average 6.7 years old (*SD* = 0.31). We selected items corresponding to commonly identified sleep problems in ASD from both parent and objective reports in the literature, such as insomnia traits (i.e. night awakenings, sleep onset) and sleep duration [2]. ***Sleep onset*** was assessed based on the one-item subscale of the CSHQ; "*child falls asleep within 20 minutes,*" and was scored from 1 to 3 (reverse-coded). ***Night awakenings*** was assessed based on two items, "*awake once*" and "*awakes more than once*." Both items were scored from 1–3; hence, our overall night awakenings score ranged from 2 to 6. The third item of the original CSHQ night awakenings subscale, "*moving to other's beds during the night*," was not included. Items were selected to investigate the frequency of awakenings, rather than reasons for their occurrence. Insomnia traits are generally required to be present for three or more nights a week [59], which spans the "sometimes" and "always" response categories. In this sample, the "always" response was endorsed infrequently, which could lead to problems with empty cells in the growth curve models. We therefore transformed night awakenings and reverse coded sleep onset into binary variables for the growth curve analysis, responses of "*never/rarely*" were coded as 0 and "*sometimes*" to "*always*" were coded as 1. ***Sleep duration*** was reported within an hour range, then averaged.

Executive functions.

The Behavior Rating Inventory of Executive Function (BRIEF) [16], was used to measure EF reported by teachers at four time-points (T5-T8) with an approximate one-year interval from 7.7 to 11.8 years. The BRIEF consists of 86 items assessing EF in real world settings over the past 6 months. It is composed of eight subscales collapsed into two indices. Metacognition difficulties capturing cold EF, are measured on the Metacognition Index (MI), which comprises five subscales (Initiate, Organize/Plan, Organization of Materials, Working Memory, and Monitor). Behavioral regulation difficulties capturing hot EF, are measured with the Behavioral Regulation Index (BRI), which comprises three subscales (Inhibit, Emotional Control and Shifting).

Cognition.

The Merrill-Palmer-Revised Developmental Index Standard Score [60] is a standardized measure of intellectual ability for children 2 to 78 months of age. Higher cognitive raw scores correspond to stronger early cognitive skills. The Cognitive Index raw score was used to measure early cognitive ability as a proxy for IQ at 3.5 years.

ASD severity.

The ADOS calibrated severity score [61] (CSS), a 10-point scale derived from raw ADOS scores, was used to measure ASD symptom severity at 3.5 years. The ADOS CSS captures core ASD symptom severity independent of age and language level. Higher scores indicate greater symptom severity.

Income.

Family income was reported by parents on the Family Background Information Questionnaire developed for the Pathways study. Caregivers reported their yearly family income (Canadian currency; CAD) on a scale ranging from 1 (< CAD$5,000) to 11 (> CAD$80,000). Similar to Bennett et al. [62], a binary variable for income was created based on the median of $70,000.

Data analysis

To analyze the association between sleep at 3.5 years (T1) and the BRI and MI assessed four times between 7.7 and 11.8 years, two separate growth curve models were estimated in Mplus v8.4. The use of latent growth curves allows for testing of the contribution of poor sleep to the initial starting point of the trajectory (intercept) and to change in EF over time (slope). EF time-points for the slope growth factor were fixed at 0, 1, 2, and 3 to define a linear growth model with equidistant time-points, and fixed at 0, 1, 4, 9 to define a quadratic growth model. The zero-time score for the slope growth factor at T1 defined the intercept factor. To handle missing data, Full Information Maximum Likelihood estimation was used.

We first ran unconditional growth curve models (without predictors) for both the MI and BRI, followed by conditional models which included predictors. To determine the best model fit, unconditional growth curve models were run with both a linear and quadratic growth factor. In the first conditional model, covariates from T1 (age, family income, and testing site) and T1 sleep variables were entered as predictors of the intercept and slope. In the second model, early cognition and ASD severity were added to Model 1 as confounding variables to test the specificity of results to EF. In the third model, sleep at ages 3.5 and 6.7 years were added to test whether sleep at 3.5 years of age was still associated with later EF after accounting for more proximal sleep problems. Covariates from Model 1 were also included. Finally, in Model 4, early cognition and ASD severity were added to Model 3, to test the specificity of results to EF. Usage of medications known to disrupt or augment sleep at each time-point is documented in Table 1. Parents reported 1.8% of toddlers at 3.5 years and 5.5% of school children at 6.7 years took melatonin. The melatonin usage in our Pathway's sample is lower than the prevalence of melatonin use previously reported in ASD, from 3.4% and up to 36% in children with sleep problems [63–66]. Other psychotic and epileptic medication that could interfere with sleep were taken by 3.2% and 7.8% of participants at 3.5 and 6.7 years respectively. Stimulants, anti-psychotics (e.g. Risperdone), and anti-depressants, were among the most common medications used. For a breakdown of medications used see supplementary materials (Table S1). Previous reports of psychotic medication use in toddlers and preschoolers have been similar to (2.9%) or higher than (11%–32%) the use reported in this cohort [67–70]. Childhood usage of psychotic drugs in other ASD cohorts are higher than the usage in the Pathways cohort [64, 66, 67, 71]. Estimates of psychoactive drug usage in school-age children with ASD are reported to be

Table 1. Pathways participant characteristics

	Time-points	*N* (%)	Mean	*SD*
Age (years)	T1		3.46	0.77
	T4		6.66	0.31
	T5		7.74	0.22
	T6		8.73	0.21
	T7		9.71	0.22
	T8		10.77	0.25
Sex	T1			
Male		183 (84.3)	–	–
Female		34 (15.7)	–	–
Site	T1			
Halifax		28 (12.9)	–	–
Montreal		83 (38.2)	–	–
Hamilton		23 (10.6)	–	–
Vancouver		57 (26.3)	–	–
Edmonton		26 (12.0)	–	–
Income	T1			
Less than $5000		2 (0.9)	–	–
Less than $10,000		4 (1.8)	–	–
Less than $15,000		4 (1.8)	–	–
Less than $20,000		7(3.2)	–	–
Less than $30,000		13(6.0)	–	–
Less than $40,000		10(4.6)	–	–
Less than $50,000		12(5.5)	–	–
Less than $60,000		34(15.7)	–	–
Less than $70,000		21(9.7)	–	–
Less than $80,000		19(8.8)	–	–
More than $80,000		91(41.8)	–	–
Medication usage				
Melatonin	T1	4 (1.8)		
	T4	12 (5.5)		
	T5	13 (6.0)		
	T6	15 (6.9%)		
	T7	11 (5.1%)		
	T8	16 (7.4%)		
Other psychotropic/anti-epileptic drugs*	T1	7 (3.2%)		
	T4	17 (7.8%)		
	T5	26 (12%)		
	T6	38 (17.5%)		
	T7	29 (13.4%)		
	T8	44 (20.3%)		
Merrill-Palmer-Revised:	T1			
Cognitive raw score†		210	56.06	17.95
Cognitive standard score		201	61.40	24.15
CSS	T1	216	7.70	1.67
BRI raw scores	T5	89	59.47	13.45
	T6	98	58.44	14.74
	T7	81	57.12	13.71
	T8	81	52.84	14.42
MI raw scores	T5	87	91.15	17.03
	T6	93	89.15	20.79
	T7	77	91.71	18.89
	T8	79	83.39	19.55

*Age at T1 was used to account for cognitive raw scores not standardized by age.
† Other psychotropic/epileptic drugs are medication that can have an impact on regular sleep patterns. Further breakdown of these drugs can be found in the supplementary materials.

as high as 70% [66], while in our cohort the most reported usage of medication was at T8 with 20.3% of participants. We re-ran Model 4 removing participants that were reported to be taking melatonin or anti-psychotic/epileptic medication to check that this did not change the pattern of results. Post hoc analyses applying Bonferroni corrections for multiple comparisons were conducted to further investigate subscales of significant BRI or MI models.

All continuous predictors were mean centered to aid interpretation. A Root Mean Square Error (RMSEA) below 0.60 and Comparative Fit Index (CFI) ≥ 0.90 was considered an adequate model fit [72]. Descriptive analyses were conducted using SPSS

25. Linear and logistic regressions were used to test for differences in characteristics between participants who provided T1 data and BRIEF data from at least one time-point (T5-T8) and participants who had missing data. Bivariate, point-biserial and tetrachoric correlations were estimated for associations between binary and continuous variables. A log transformation of MI raw scores at T8 was applied to generate an approximately normal distribution for bivariate analysis.

Results

See Table 1 for participant characteristics. Participants had a mean sleep duration of 11.0 h (*SD* = 1.35) at 3.5 years (ranging 2–4 years of age), consistent with National Sleep Foundation (NSF) recommendations [73] for toddlers (11–14 h)/preschoolers (10–13 h). However, 32.4% of toddlers and 21.4% of preschoolers in the Pathways cohort, slept for a shorter duration than recommended for their age group. Less than 3% of toddlers and 4.7% of preschoolers slept longer than their recommended duration. The average sleep duration reported at 6.7 years when participants were school-aged was 10.2 h (*SD* = 0.92), also within NSF recommendations (9–11 h). Twelve percent of participants at 6.7 years slept more than the recommended sleep duration, while 6% slept less than recommended. See Table 2.

At 3.5 years, 57% of participants were found to "sometimes or usually" have a problem with night awakenings and 50% had delayed sleep onset issues documented more than twice per week. At 6.7 years, 50.7% and 46.5% of participants were reported as "sometimes or usually" having night awakenings and sleep onset delay per week, respectively. See Table 3. Correlations among sleep, EF, and covariates can be found in the supplementary materials (Tables S2-S10).

Latent growth curve models

BRI difficulties.

The unconditional growth model with a linear growth factor fit the data best (fit indices are presented in Table 4). The intercept mean was 59.5 (*SE* = 0.96) and the slope mean was –1.42 (*SE* = 0.42), indicating that BRI difficulties decreased over time. The mean and variance of both the intercept and slope were significant, the latter indicating significant decrease in the BRI from T5 to T8 (reflecting improvement). Models 1–4 are found in Table S9. Results from Model 1 (see Table 5) showed that a delayed sleep onset significantly predicted lower BRI difficulties at the intercept (b = –4.14, p = 0.04). A delayed sleep onset remained a significant predictor of lower difficulties after the inclusion of early cognitive ability and ASD severity in Model 2 and after the addition of sleep variables at 6.7 years in Model 3 (b = –7.18, p = 0.001) and early cognitive ability and ASD severity in Model 4 (b = –8.51, p = 0.001). The Montreal site was significantly associated with lower BRI difficulties at the intercept in Model 1 (b = –6.84, p = 0.022) but this became non-significant after the inclusion of early cognitive ability and ASD severity in Model 2.

When sleep variables at 6.7 years were entered in Model 3, a delayed sleep onset at 3.5 years significantly predicted a worsening slope of BRI difficulties (b = 2.07, p < 0.041), and this remained significant in Model 4 (b = 2.37, p = 0.025). Delayed sleep onset at 6.7 years also significantly predicted higher BRI difficulties at the intercept in Model 3 (b = 7.78, p = 0.002) and in Model 4 (b = 7.85, p = 0.001). In Model 4, shorter sleep duration at 3.5 years was found to predict higher BRI difficulties at the intercept (b = –2.05, p = 0.046). Conversely, a shorter sleep duration at 6.7 years predicted lower BRI difficulties at the intercept in Model 3 (b = 3.15, p = 0.010), and this remained significant in Model 4 (b = 3.64, p = 0.004). Night awakenings at 3.5 or 6.7 years showed no significant associations with the intercept or slope. For full parameter estimates see Table 5. Model 4 was re-run removing 30 children who were taking sleep or psychoactive medication when sleep information was captured at 3.5 or 6.7 years of age. The pattern and significance of the BRI model results were unchanged when accounting for medication usage (Table 5).

MI difficulties.

The unconditional latent growth curve model with a linear growth factor only fit the data best (see Table 4). The intercept mean was 91.2 (*SE* = 1.29) and the slope mean was = –1.3 (*SE* = 0.06), indicating MI difficulties decreased over time. The mean and variance of both the intercept and slope were significant, the latter indicating significant growth in the MI from 7.7 to 11.8 years of age. Models 1–4 also had an adequate fit (Table S11). No sleep variables were significantly associated with MI difficulties at the intercept or slope. See Table S12 for full parameter estimates. Model 4 was re-run removing 30 children who were taking sleep or psychoactive medication when sleep information was captured at 3.5 or 6.7 years of age (Table S13).

Table 2. CSHQ Sleep duration frequencies

	*2 years (T1)		3–4 years (T1)		6–7 years (T4)	
Duration (h)	*N*	%	*N*	%	*N*	%
6.50	–	n/a	n/a	n/a	3	1.4
7.50	1	1.5	n/a	n/a	n/a	n/a
8.50	6	8.8	9	6.0	10	4.6
9.50	5	7.4	23	15.4	60	27.6
10.50	10	14.7	55	36.9	91	41.9
11.50	23	33.8	41	27.5	23	10.6
12.50	17	25.0	14	9.4	3	1.4
13.50	4	5.9	3	2.0	n/a	n/a
14.50	3	2.9	4	2.7	n/a	n/a

*Ages based on National Sleep Foundation developmental categories of infants, toddlers and school-age.

Table 3. CSHQ night awakenings and sleep onset item scores

	T1		T4	
	N	%	*N*	%
Night awakenings				
0*	94	43.3	107	49.3
1*	123	56.7	110	50.7
"awakes once"				
Never/rarely	101	46.5	113	58.2
Sometimes	78	35.9	55	28.3
Usually	38	17.5	26	13.4
"awakes more than once"				
Never/rarely	162	74.6	165	85.4
Sometimes	43	19.8	22	11.4
Usually	12	5.5	6	3.10
Sleep onset				
0*	109	50.2	116	53.5
1*	108	49.8	101	46.5
"child falls asleep after 20 min"				
Never/rarely	109	50.2	116	53.5
Sometimes	71	31.7	51	23.5
Usually	37	17.1	27	12.4

*Transformed binary variables

Table 4. Unconditional BRI and MI models

	BRI		MI	
	Linear model	Quadratic model	Linear model	Quadratic Model
χ^2 (*df*)	8.572(5), $p = 0.12$	5.084(1), $p = 0.02$	9.40 (5), $p = 0.09$	8.744(1), $p = 0.003$
RMSEA (95%CI)	0.053 (0.00– 0.11)	0.126 (0.037–0.243)	0.059 (0.00–0.12)	0.174 (0.082–0.287)
CFI	0.98	0.98	0.96	0.922

The pattern and significance of the MI model results were unchanged, with the exception of shorter sleep duration at time 1, which became significantly associated with increased MI difficulties at the intercept ($b = -3.19$, $p = 0.038$).

Subscale analysis.

Post hoc analyses were conducted to further investigate the three subscales of the BRI: *inhibition*, *shifting*, and *emotional control*. Fit indices and parameter estimates of unconditional growth curves and Models 1–4 for each subscale are reported in supplementary materials (Table S13-S18). Multiple comparisons for our three exploratory models were corrected for, with significance set at $p = 0.017$.

A delayed sleep onset in Model 1 was associated with lower emotional control difficulties at the intercept ($b = -2.08$, $p = 0.004$). This was not found for inhibition or shifting. Upon entering sleep at 6.7 years in Model 3, delayed sleep onset at 3.5 years became significantly associated with lower inhibition difficulties at the intercept ($b = -2.28$, $p = 0.008$). The addition of ASD severity and early cognitive ability in Model 4 did not significantly alter these associations. In Model 3, a delayed sleep onset at 3.5 years was significantly associated with a worsening emotional control slope ($b = 0.95$, $p = 0.008$), which was not found for shifting or inhibition. Subsequently, with the addition of ASD severity and early cognitive ability in Model 4, delayed sleep onset at 3.5 years significantly predicted a worsening inhibition slope ($b = 1.01$, $p = 0.009$), but this was not found for shifting. The Montreal site was significantly associated with lower shifting difficulties at the intercept in Model 1 ($b = -2.60$, $p = 0.012$) which became non-significant after the inclusion of early cognitive ability and ASD severity in Model 2.

Associations were found between a delayed sleep onset at 6.7 years and higher difficulties at the intercept for all three BRI subscales in Model 3: inhibition ($b = 2.82$, $p = 0.004$), cognitive shifting ($b = 2.20$, $p = 0.01$) and emotional control ($b = 2.77$, $p = 0.001$). These results remained significant in Model 4.

No significant associations were found between sleep duration at 3.5 years and BRI subscales. In model 3, similarly to the overall BRI, longer sleep duration at 6.7 years was associated with lower inhibition ($b = 0.228$, $p = 0.007$) and emotional control difficulties ($b = 1.07$, $p = 0.015$) at the intercept, but not cognitive shifting. These results remained in Model 4, but with the addition of early cognitive ability and ASD severity, shorter sleep duration at 6.7 years predicted a worsening inhibition slope ($b = -0.60$, $p = 0.011$).

Finally, neither night awakenings at 3.5 or 6.7 years were significantly associated with BRI subscales. Model 4 was re-run removing 30 children who were taking sleep or psychoactive medication when sleep information was captured at 3.5 or 6.7 years of age. The pattern and significance of BRI subscale model results were unchanged when accounting for medication use, with the exception of the association between delayed sleep onset at 3.5 years and shifting difficulties at the intercept, which whilst not substantially reduced in size became non-significant ($b = -1.59$, $p = 0.123$).

Table 5. Parameter estimates for behavioral regulation difficulties models 1–4

Intercept					Slope			
Predictors	Estimate (standardized)	95% CI	*SE*	*p*	Estimate (standardized)	95% CI	*SE*	*p*
Model 1								
Age	0.03 (0.02)	−0.17–0.22	0.12	0.834	−0.05 (−0.14)	−0.14–0.05	0.06	0.410
Halifax site	−5.49 (−0.47)	−11.45–0.47	3.62	0.130	1.13 (0.36)	−1.27–3.54	1.46	0.438
Montreal site	**−6.84 (−0.58)**	**−11.78 to −1.92**	**3.00**	**0.022**	0.17 (0.05)	−1.98–2.33	1.31	0.895
Edmonton site	2.29 (0.19)	−3.27–7.85	3.38	0.498	−0.72 (−0.23)	−3.85 to −2.42	1.90	0.707
Vancouver site	−2.39 (−0.20)	−7.27 to −2.39	2.97	.0420	1.29 (0.41)	−1.05–3.64	1.43	0.364
Income	−4.01 (−0.34)	−7.39 to −0.63	2.05	0.051	0.97 (0.31)	−0.61–2.55	0.96	0.313
T1 sleep onset	**−4.14 (−0.35)**	**−7.54 to −0.75**	**2.07**	**0.045**	1.01 (0.32)	−0.55–2.57	0.95	0.287
T1 night awakening	1.53 (0.13)	−1.92–4.99	2.10	0.466	0.01 (0.01)	−1.46–1.46	0.89	0.999
T1 sleep duration	−0.80 (0.09)	−2.11–0.52	0.80	0.317	0.31 (0.14)	−0.35–0.97	0.40	0.437
Model 2								
Age	0.12 (0.09)	−0.12–0.35	0.14	0.410	−0.09 (−0.25)	−0.20–0.02	0.07	0.177
Halifax site	−3.46 (−0.29)	−9.53–2.60	3.68	0.347	1.15 (0.34)	−1.29–3.59	1.48	0.438
Montreal site	−4.21 (−0.35)	−9.13–0.70	2.99	0.159	−0.65 (−0.19)	−2.89–1.59	1.36	0.633
Edmonton site	5.66 (0.47)	0.03–11.29	3.42	0.098	−0.99 (−0.29)	−4.30–2.33	2.02	0.624
Vancouver site	−0.95 (−0.08)	−5.93–4.02	3.02	0.753	1.19 (0.36)	−1.22–3.61	1.47	0.415
Income	−3.16 (−0.26)	−6.80–0.48	2.21	0.153	0.82 (0.25)	−0.92–2.56	1.06	0.436
ASD severity	**1.45 (0.20)**	**0.43–2.47**	**0.62**	**0.020**	−0.53 (−0.26)	−1.03 to −0.03	0.31	0.084
Early cognition	−0.08 (−0.17)	−0.16 – 0.01	0.05	0.082	0.03 (0.21)	−0.01–0.07	0.02	0.207
T1 sleep onset	**−5.44 (−0.45)**	**−8.99 to−1.89**	**2.16**	**0.012**	1.26 (0.37)	−0.42–2.93	1.02	0.217
T1 night awakening	1.553 (0.13)	−2.04–5.15	2.19	0.477	0.32 (−0.10)	−1.89–1.25	0.95	0.736
T1 sleep duration	−1.48 (−0.17)	−2.96 to−0.01	0.90	0.099	0.41 (0.16)	−0.33–1.14	0.45	0.362
Model 3								
Age	−0.01 (1.03)	−0.23–0.21	0.13	0.926	−0.01 (−0.02)	−0.12–0.10	−0.11	0.913
Halifax site	−5.36 (0.46)	12.73–2.01	4.48	0.231	0.93 (0.29)	−2.16–4.09	1.88	0.622
Montreal site	−5.10 (−0.44)	−10.13 to −0.08	3.05	0.094	−0.03 (−0.01)	−2.32–2.25	1.39	0.980
Edmonton site	0.30 (0.02)	−5.62–6.23	3.60	0.933	0.21 (0.07)	−3.54–3.97	2.27	.925
Vancouver site	−0.69 (−0.06)	−5.36–3.98	2.84	0.808	1.01 (0.32)	−1.52–3.52	1.53	0.513
Income	−3.57 (−0.31)	−7.27–0.13	2.25	0.113	1.16 (0.37)	−0.53–2.86	1.03	0.259
T1 Sleep onset	**−7.19(−0.62)**	**−10.85 to −3.53**	**2.22**	**0.001**	**2.07 (0.66)**	**0.40–3.73**	**1.01**	**0.041**
T1 Night awakening	2.37 (0.20)	−1.69–6.44	2.47	0.337	−0.38 (−0.12)	−2.07–1.30	1.02	0.707
T1 Sleep duration	−1.39 (−0.16)	−2.96–0.18	0.96	0.146	0.22 (0.09)	−0.50–0.93	0.44	0.620
T4 Sleep onset	**7.78 (0.67)**	**3.69–11.87**	**2.49**	**0.002**	−1.47 (−0.47)	−3.22–0.28	1.06	0.166
T4 Night awakening	3.74 (0.32)	−0.14–7.61	2.36	0.113	−0.76 (−0.24)	−2.52–1.01	1.07	0.479
T4 Sleep duration	**3.15 (0.26)**	**1.14– 5.17**	**1.23**	**0.010**	−0.92 (−0.28)	−1.93–0.09	0.61	0.133
Model 4								
Age	0.02 (0.02)	−0.23–0.28	0.16	0.883	−0.025 (−0.06)	−0.16–0.11	0.08	0.753
Halifax site	−5.54 (−0.47)	13.29–2.20	4.71	0.239	1.654 (0.46)	−1.56–4.87	1.95	0.398
Montreal site	−3.53 (−0.30)	−8.13–1.06	2.79	0.206	−0.636 (−0.18)	−3.01–1.74	1.44	0.660
Edmonton site	2.95 (0.25)	−2.92–8.82	3.57	0.409	0.297 (0.08)	−3.78–4.37	2.48	0.905
Vancouver site	0.70 (0.06)	−3.67–5.09	2.67	0.792	0.85 (0.24)	−1.68–3.38	1.54	0.581
Income	−1.97 (−0.17)	−5.88–1.93	2.374	0.406	0.76 (0.21)	−1.14–2.66	1.155	0.508
ASD severity	**1.43 (0.20)**	**0.44–2.43**	**0.61**	**0.018**	−0.52 (−0.24)	−1.02 to −0.01	0.31	0.092
Early cognition	**−0.10 (−0.20)**	**−0.17 to −0.02**	**0.05**	**0.045**	0.04 (0.25)	−0.01–0.08	0.03	0.165
T1 sleep onset	**−8.51 (−0.72)**	**−12.15 to −4.86**	**2.22**	**0.000**	**2.37 (0.66)**	**0.63–4.10**	**1.05**	**0.025**
T1 night awakening	3.05 (0.26)	−1.01–7.11	2.47	0.216	−1.02 (−0.28)	−2.78–0.73	1.07	0.337
T1 sleep duration	**−2.05 (−0.24)**	**−3.74– −0.36**	**1.03**	**0.046**	0.27 (0.10)	−0.51–1.05	0.47	0.565
T4 sleep onset	**7.85 (0.66)**	**3.86–11.84**	**2.43**	**0.001**	−1.58 (−0.44)	−3.41–0.24	1.11	0.154
T4 night awakening	4.47 (0.38)	0.35–8.60	2.51	0.075	−0.816 (−0.23)	−2.76–1.13	1.18	0.490
T4 Sleep duration	**3.64 (0.30)**	**1.54–5.74**	**1.28**	**0.004**	−1.16 (−0.31)	−2.24 to −0.07	0.66	0.079
Model 4								
(without medication)								
Age	−0.07(−0.05)	−0.40–0.25	−0.44	0.659	−0.02(−0.05)	−0.18–0.14	−0.23	0.815
Halifax site	−7.78(−0.19)	−17.05–1.48	−1.65	0.099	3.34(0.28)	−0.74–7.43	1.60	0.109
Montreal site	−5.36(−0.21)	−11.08–0.35	−1.84	0.066	0.68(0.09)	−2.30–3.66	0.45	0.654
Edmonton site	3.05(0.08)	−5.37–11.47	0.71	0.478	0.07(0.01)	−5.91–6.06	0.02	0.981
Vancouver site	0.328(0.01)	−5.15–5.81	0.12	0.907	1.47(0.18)	−1.64–4.59	0.92	0.354
Income	−1.58(−0.06)	−6.48–3.31	−0.63	0.526	0.45(0.06)	−1.89–2.79	0.37	0.707
ASD severity	**1.55(0.21)**	**0.30–2.81**	**2.42**	**0.015**	**−0.68(−0.31)**	**−1.31−−0.06**	**−2.15**	**0.032**
Early cognition	−0.10(−0.20)	−0.20–0.01	−1.85	0.065	0.04(0.25)	−0.02–0.09	1.285	0.199
T1 sleep onset	**−7.30(−0.29)**	**−12.20−−2.41**	**−2.93**	**0.003**	1.88(0.26)	−0.39–4.16	1.62	0.104
T1 night awakening	0.13(0.01)	−5.33–5.60	0.05	0.961	0.03(0.004)	−2.31–2.37	0.02	0.980
T1 sleep duration	−2.50(−0.26)	−4.71−−0.29	−2.22	0.026	0.38(0.14)	−0.60–1.37	0.76	0.449
T4 sleep onset	**8.44(0.33)**	**3.28–13.59**	**3.21**	**0.001**	−1.64(−0.22)	−3.91–0.64	−1.41	0.158
T4 night awakening	4.96(0.20)	−0.45–10.37	1.80	0.072	−0.14(−0.02)	−2.60–2.32	−0.11	0.909
T4 sleep duration	**4.14(0.33)**	**1.44–6.84**	**3.01**	**0.003**	−0.98(−0.26)	−2.33–0.37	−1.42	0.155

Discussion

In the current study, the association between early childhood sleep and later EF at school-age was examined in children with ASD. Our results partially confirm our hypothesis, indicating that sleep quantity and quality are exclusively related to "hot" (behavioral regulation) rather than "cold" (metacognition) EF in school-aged children with ASD. Contrary to our prediction, participants with delayed sleep onset at 3.5 years had better behavioral regulation, as first reported by teachers on the BRIEF at 7.7 years. Examination of the three BRI subscales showed that this association was only significant for emotional and inhibitory control. However, as hypothesized, delayed sleep onset at 3.5 years exclusively predicted a worsening behavioral regulation trajectory after the more proximal school-age sleep was accounted for, and this same pattern was seen for two behavioral regulation subscales, emotional and inhibitory control. Of the school-age sleep variables (assessed at age 6.7), a delayed sleep onset was associated with greater behavioral regulation difficulties at 7.7 years. Longer sleep duration at age 6.7 was associated with greater behavioral regulation difficulties at 7.7 years, and this was also seen for the three behavioral regulation subscales. In contrast, a shorter sleep duration at 6.7 years predicted a worsening inhibition trajectory, but this was not seen for overall behavioral regulation difficulties or the other two subscales. Neither night awakenings nor shorter sleep duration at 3.5 years of age was related to EF as expected.

Given previous documentation of sleep problems in 40%–80% of children with ASD and evidence suggesting early poor sleep impairs EF development, we hypothesized that poor sleep may serve as a mechanism to exacerbate EF difficulties found in children with ASD. It is difficult to ascertain whether early sleep problems are more prevalent in our ASD cohort compared to normative samples due to measurement differences [7, 74, 75]. The CSHQ was not developed for children under age 6; hence, questions and scoring differed from what we have documented. For example, previous studies captured the number of awakenings per night rather than per week as in the CSHQ, whereas sleep onset difficulty was usually set at 30 min rather than the 20 min on the CSHQ [7, 75]. Difficulties initiating sleep for more than 20 to 30 min is generally accepted as clinically problematic in pediatric populations [76], however, neither time cutoff is recommended over the other in the current ICD-10 or DSM-5 criteria. Up to 28% of TD preschoolers at 3.5 years of age have at least one awakening per night [7], whereas 56.7% of our same-aged Pathways cohort had one or more night awakenings between two to seven nights per week. At first glance it may seem as though there are more awakenings in our cohort, but upon examination of the item "*awakes once during the night*" only 17% of Pathways parents responded their child woke up once five to seven nights per week (closer to one night per week). Hence, night awakenings may actually be lower in our cohort, but this cannot be conclusive given the variability of parents' reports. Similarly, 16% of TD 3.5-year-olds have had sleep onset difficulties documented per night [7], whereas 49.8% of our same-aged preschool cohort had sleep onset difficulties two to seven nights per week. More comparably, in our cohort, 17% of preschoolers took more than 20 min to get to sleep, five to seven nights per week. Again, this is compared to the 30-min cutoff of the TD sample. When school-aged, greater sleep problems on the CSHQ were found in our ASD cohort compared to a normative North American sample with a similar mean age of 7 years [77]. In the TD cohort 21% "sometimes" or "usually" had sleep onset difficulties, compared to 46.5% in our cohort. 25.1% of typically developing children had one-night awakening "sometimes" or "usually" per week, compared to 41.7% of Pathways children. Although the age range of the normative sample is larger, we can say with more certainty that Pathways youth had more disturbed sleep reported by their parents at school-age, in line with estimates from the literature. Interestingly, during both age assessments, our Pathways cohort slept comparably to normative samples and within the recommended sleep duration for their age [73]. Hence, links between EF difficulties and low sleep quantity may be more pronounced in a clinical cohort with greater sleep duration disturbances.

This was the first study to examine growth in EF across multiple time-points in middle childhood in ASD. We found significant but small improvement over time in both behavioral regulation and metacognition. We did not replicate previous longitudinal associations between sleep and metacognition documented in TD children [78]. However, our findings do parallel preliminary evidence from two cross-sectional studies reporting no significant relationship between sleep and metacognition in children with ASD [47, 49]. In TD literature, a greater proportion of nighttime sleep early in infancy has been related to better hot and cold EF, but not to general cognitive ability, later in infancy and in preschool [44, 45]. The authors suggest that sleep impacts components of EF that are distinct from general cognitive abilities. Indeed, when we examine Model 4 (Table S11) controlling for early cognition along with other covariates like ASD severity and school-age sleep, we find shorter sleep duration and greater night awakenings become marginally associated with metacognitive abilities with a moderate to large effect size. In the model run without any children taking sleep or psychoactive medication this association between shorter sleep duration and metacognitive abilities became significant. Similar to Bernier et al. (2010, 2013) we find that sleep traits and early general cognition are not correlated in this cohort. Hence, this may indicate that isolating metacognitive ability from general cognition may allow for a sleep link to emerge. However, unlike the previous studies in TD children, we did not use experimental measures to tap into "pure" metacognition, which compared to behavioral regulation is more related to cognitive abilities as it corresponds to cool EF. Since we examine the use of metacognition observed in behaviors it may be less distinguished from general cognitive ability than when assessed using lab procedures. Another consideration is that cold EF is found to develop more quickly than hot EF, and it is reported that older children and adolescents with ASD experience greater lab and real-world impairments of cold EF compared to earlier ages or to hot components like inhibitory control [25, 79]. It may be that sleep problems are more impactful during the earlier development of cold EF rather than later in childhood, however, more research with concurrent sleep and EF measures are needed to understand this association.

We also substantiated previous links found between poor sleep and behavioral problems in ASD children [80, 81], as the BRI subscale shows some overlap with behavioral problems. Overall, our novel findings highlight a non-uniform relationship, whereby age, type of behavioral regulation, and sleep traits need to be taken into account.

Links between sleep onset and behavioral regulation

The finding that delayed sleep onset predicted lower behavioral regulation difficulties at the first EF time-point when participants were 7.7 years was unexpected. It is important to take into account that bedtime is more externally determined earlier in childhood; therefore, delayed sleep onset may reflect both internal sleep processes and external parental regulation. One possible explanation for the association may be that toddlers and preschoolers who tolerate being put to bed earlier than their natural circadian rhythm bedtime show improved behavioral regulation around the start of school-age. It has been previously documented that preschoolers whose parentally determined bedtime does not match their internal circadian rhythm have greater bedtime resistance [82]. Sleep problems like delayed onset are thought to arise from a de-synchrony between one's endogenous circadian rhythm and preferred sleep-wake schedule [83]. The proposed interpretation is in line with previous documentation of circadian rhythm alterations in ASD. This includes atypical levels and synthesis of melatonin, involved in resetting circadian rhythms, and disturbances of circadian genes [84]. Moreover, delayed sleep phase syndrome, a circadian rhythm disorder characterized by sleep that is delayed by at least 2 h [59], is found in adults with ASD [85, 86]. Suggesting that a mismatch of external bedtimes and internal sleep preferences is an issue in ASD. Hence, young children with delayed sleep onset may be regulating their behaviors based on parental instructions, despite not being ready to sleep. These children may be required to control their emotions (e.g. crying) and inhibit resistant behavior (e.g. getting out of bed) during bedtime; indeed, improved inhibitory and emotional control were the only two subscales at 7.7 years linked to early delayed onset. However, delayed sleep onset measured more proximally to the EF assessments (age 6.7 years) showed the expected association of delayed onset with increased behavioral regulation difficulties. The present findings do suggest a specific role for delayed sleep onset in later behavioral regulation difficulties, but the pattern of findings is complex and warrants replication and further investigation to understand the mechanisms involved. Parental report of sleep is likely confounded with both parent and child characteristics, so future studies using objective measures of sleep will be important. Delayed sleep onset is a particularly salient sleep problem in individuals with ASD throughout their lifespan [10, 85] and may be a significant source of stress for parents, underscoring the need for a better understanding of the role of sleep onset delay in the development of later difficulties in individuals with ASD.

In contrast to emotional and inhibitory control, cognitive shifting was not significantly linked to sleep onset. Previous findings have found total sleep problems (including sleep onset) from preschool and school-age were not longitudinally associated with experimental or neuropsychological measures of shifting [87, 88], while decreasing sleep problems across childhood have been correlated with better inhibitory control but not shifting [87]. The latter provides evidence that sleep may better predict inhibitory control outcomes over shifting. As shifting difficulties are consistently documented to be the most impaired EF component in ASD [25, 89], it consequently may be less malleable to significant developmental changes.

This is the first finding linking delayed sleep onset in early childhood to later emotional control in children with ASD. The finding is unsurprising given extensive documentation that sleep quantity and quality (including insomnia traits) affects mood, affective information processing and the capacity to regulate emotions [90, 91]. It is posited that poor sleep impairs the connectivity between the prefrontal cortex and amygdala, a central neural pathway for affective processing [91]. A lack of prefrontal-amygdala connectivity during emotional responses is found in children with ASD [92]: hence, this may be one possible mechanism by which sleep disturbance further exacerbates emotional regulation impairments in ASD. Finally, inhibitory control was most consistently impacted negatively by all sleep disturbances in the current cohort, not only by sleep onset, mirroring previous findings with restricted sleep duration and night awakenings in TD and children with ADHD [93, 94].

Links between sleep duration and behavioral regulation

Interestingly, longer sleep duration at school-age was associated with worse behavioral regulation difficulties during early school-age. Twelve percent of these participants slept more than 11 h, which according to the NSF falls into the *may be appropriate* or *not recommended* range. Oversleeping is known to have negative consequences for cognitive processes such as EF [6, 95]. Negative outcomes for both extremes of under- or oversleeping reflect an inverted-U shaped relationship [6]. Weekday oversleeping can also be compounded by school-aged children who tend to sleep more during the weekends [96]. Further investigations of subscales revealed that shorter sleep duration at 6.7 years predicted a worsening inhibition but not overall behavioral regulation trajectory during school-age. As both preschool and school-aged sleep durations are positively correlated in our sample, children might have accumulated a sleep debt, known to have negative consequences for behavioral modulation.

Links between night awakenings and behavioral regulation

Frequent night awakenings did not predict EF difficulties. It is possible that earlier night awakenings at age 3.5 years were not significant predictors as awakenings are commonly found during toddlerhood and may have more impact on EF when they are less developmentally appropriate. This is also supported by previous findings showing night awakenings in infants was not associated with later impulse control at 6-months and 1-year follow-ups [44].

Strengths and limitations

A major strength of the current study is the large sample of systematically diagnosed and phenotyped children with ASD. Pathways is a unique and advantageous cohort as the participants had a narrow age range at study entry and have been regularly assessed by multiple respondents. This includes sleep repeatedly reported by parents and EF assessments by various teachers at four time-points, limiting responder bias. Multiple assessment points allowed for longitudinal modeling techniques, another major strength, providing the first growth curve analysis of EF in ASD. Controlling for factors including early cognitive ability and ASD severity also provides some confidence

that our significant findings are unique to the relationship between sleep and EF.

We did not use psychometric or experimental tasks that may evaluate "pure" EF components; however, performance on these tasks varies based on presentation modality and response type and do not reflect natural everyday demands [14]. Questionnaires like the BRIEF capture EF in multiple everyday scenarios, adding to the generalizability of our results.

Parent reports of sleep have been flagged as a limitation due to their susceptibility to biases and discrepancies with physiological sleep reports [97]. For instance, night awakenings might only be documented if they are significant enough to impair parental sleep, whereas sleep onset concerns may be missed if children do not present with disruptive behavior. This reporting obstacle may be more pronounced in school-age children, when parents might be less involved in monitoring sleep. In fact, previous evidence indicates that electroencephalographic markers of sleep are atypical in ASD even when parents do not report sleep problems [98]. Reporting biases may also underlie the lack of sleep problems documented in our cohort. Further, the CSHQ was not developed for documenting sleep during early childhood periods, yet still remains used [99, 100]. Evidence for the validity of the items in a toddler and preschool aged sample using actigraphy and sleep diaries has been provided, with the best correspondence between measures reported for sleep onset [101]. In the present study the individual items were used rather than the proposed subscales, as a revised and a recent review of its psychometric properties suggests that factor structure modifications are needed for children with ASD [102]. Although for our study purposes we used specific sleep traits of interest that correspond to DSM-5 criteria of insomnia, bypassing psychometric issues of the current 8 factor subscales, using the CSHQ for a young age group limited our ability to assess sleep differences between our cohort and normative samples. The lack of suitability for capturing early sleep problems also raises methodological concerns about how well these questionnaire items are isolating actual sleep problems, as opposed to varying parent preconceptions of appropriate sleep. Moreover, we did not have access to information indicating if our participants received a clinical diagnosis of sleep problems. The use of objective sleep monitoring is crucial to remove these report biases and may help delineate current sleep onset findings that are difficult to disentangle.

A further limitation of our study is that we did not have access to a control group. Finally, there was attrition from the first assessment point, from which our key sleep measures were taken, to the final four assessment points conducted at school-age, at which the EF outcome was measured. Attrition is common with longitudinal studies. In this sample non-response at school-age was associated with lower income and lower child cognitive scores, but not with sleep problems or ASD severity at 3.5 years. Family income and child cognitive scores were both included as covariates in the analysis, which helps to account for any effects of attrition on the analysis. There was some variability in response during the four EF assessment points from 7.7 to 11.8 years; thus, the main analysis used maximum likelihood estimation to include participants with teacher-reported data at any school-age time-point in the analysis.

Another study limitation is that concurrent sleep and EF were not documented. We only tested a unidirectional relationship (i.e. sleep predicting EF); nevertheless, this does not rule out a bidirectional association. Previous studies have reported that EF problems during preschool and school-age are longitudinally associated with later sleep disturbances in TD children [103, 104]. Finally, as previously flagged [13], the presence of co-morbid psychiatric conditions (e.g. ADHD or anxiety) or environmental stressors (e.g. parental stress), may also modify the interplay between sleep and EF. This was documented in a recent study by Holingue et al [105], which showed the link between poor sleep and behavioral regulation difficulties was no longer significant after correcting for anxiety, while increased sleep disturbance was no longer associated with greater metacognition problems when controlling for hyperactivity/impulsivity.

Conclusion

Current findings suggest that poorer sleep in childhood may serve as a risk factor for developing atypical EF outcomes in ASD. The novelty of our findings suggest greater nuance is needed to decipher the relationship between sleep and EF, as independent sleep phenotypes have different age related impacts on selective components of EF. We are the first to report poor sleep specifically impacts behavioral regulation and more specifically, inhibitory control development, above cold EF in ASD. This information is beneficial for informing possible EF interventions in ASD, by targeting sleep in children. In fact, a randomized controlled trial found that TD adolescents who received treatment for their insomnia had improved EF after six sessions, compared to waiting list controls [106]. Parents and clinicians should carefully monitor and consider treatment if children with ASD are having early sleep problems. Current interpretations of our results suggest the need to promote better matching bedtimes, which may require later bedtimes and wake-times for some children. More widespread awareness and dialogue is needed to help parents navigate enforcing bedtimes that are appropriate for their child's internal clock. Further investigations on the interplay between sleep and EF in ASD are needed with both objective and parent report measures to validate current findings.

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