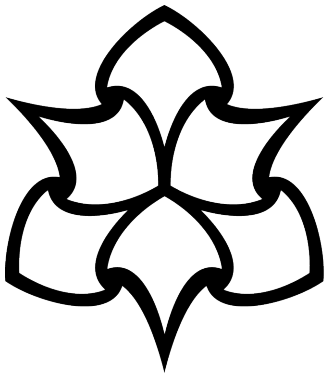

# 'Investigating longitudinal associations between parent reported sleep in early childhood and teacher reported executive functioning in school-aged children with autism'

Rackeb Tesfaye[1], Nicola Wright[2], Anat Zaidman-Zait[4], Rachael Bedford[3,5], Lonnie Zwaigenbaum[6], Connor M. Kerns[7], Eric Duku[8], Pat Mirenda[9], Teresa Bennett[8], Stelios Georgiades[8], Isabel M. Smith[10], Tracy Vaillancourt[11], Andrew Pickles[2], Peter Szatmari[12,13,14], Mayada Elsabbagh[1] and Pathways Team.

[1]Montreal Neurological Institute, Azrieli Centre for Autism Research, McGill University, Montreal, Canada

[2] King's College London, Biostatistics and Health Informatics Department, Institute of Psychiatry, Psychology & Neuroscience, King's College London, London, UK

[3] King's College London, Department of Child and Adolescent Psychiatry, Institute of Psychiatry, Psychology & Neuroscience, King's College London, London, UK

[4] Tel Aviv University, Department of Educational Sciences, Tel Aviv, Israel

[5]University of Bath, Department of Psychology, Bath, UK

[6]University of Alberta, Department of Pediatrics, Edmonton, Canada

[7] University of British Columbia, Department of Psychology, Vancouver, Canada

[8] McMaster University, Psychiatry and Behavioural Neurosciences, Hamilton, Canada,

[9]University of British Columbia, Education and Counseling Psychology, Vancouver, Canada

[10]Dalhousie University, IWK Health Centre, Pediatrics, Halifax, Canada

[11]University of Ottawa, Department of Education Ottawa, Canada

[12]University of Toronto, Department of Psychiatry, Toronto, Canada

[13]Centre for Addiction and Mental Health, Child and Youth Mental Health, Toronto, Canada

[14]SickKids Department of Psychiatry, child and adolescent psychiatry, Toronto, Canada

*Correspondence to:*
Dr. Mayada Elsabbagh
Associate Professor, Montreal Neurological Institute, Azrieli Centre for Autism Research, McGill University, 3775 University Street, Room C18, Montreal, Quebec H3A 2B4
mayada.elsabbagh@mcgill.ca

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=3.5years). 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 (7.8-11.8years). We applied latent growth curve models to examine associations between sleep and EF, accounting for relevant covariates, including school-age sleep (Mean=6.7years). 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.

*Keywords*: sleep, autism, executive functioning, development, children

## Statement of impact

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 80% of children with autism experiencing sleep problems, the impact of sleep disturbances on EF development in autism are 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.

## 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 5-18 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., 2013[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 excutive 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[69].

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[43,44]. 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[45]. 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[46]., 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[47]. 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[48]. 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[49]. 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[50–53], as well as differential impacts on cognitive outcomes[43].

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: a) were 2-4 years of age at the time of diagnosis; (b) met diagnostic criteria for ASD on the *Autism Diagnostic Observation Schedule* (ADOS)[54] and in the social and one other domain of the *Autism* Diagnostic Interview-Revised[55]; and (c) met criteria for ASD at each phase of assessment on the DSM-IV-TR[56] 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%, $p$=.009), had significantly higher cognitive standard scores (mean=56.06, SD=17.95 versus mean=50.56, SD=17.58; $p$=.031), and were more likely to be from the Montreal site ($p$=.003) and less likely to be from Edmonton ($p$=.009) or Vancouver ($p$=.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)*[57]*,* 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[58], 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[59] 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[60] (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.[61], 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 7.7-11.8 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[62–65]. 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 [66–69]. Childhood usage of psychotic drugs in other ASD cohorts are higher than the usage in the Pathways cohort[62,64,65,70]. Estimates of psychoactive drug usage in school-age children with ASD are reported to be as high as 70%[65], 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. ~~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) $\geq$ .90 was considered an adequate model fit[71]. 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 hours (SD = 1.35) at 3.5 years (ranging 2-4 years of age), consistent with National Sleep Foundation (NSF) recommendations[72] for toddlers (11–14 hours)/ preschoolers (10-13 hours). 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 hours (SD = 0.92), also within NSF recommendations (9 to 11 hours). 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, $p$ =.001) and early cognitive ability and ASD severity in Model 4 *(*$b$ = -8.51, $p$ = .001). The Montreal site was significantly associated with lower BRI difficulties at the intercept in Model 1 *(*$b$ = -6.84, $p$ = .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, $p$ < .041), and this remained significant in Model 4 ($b$ = 2.37, $p$ = .025*).* Delayed sleep onset at 6.7 years also significantly predicted higher BRI difficulties at the intercept in Model 3 ($b$ = 7.78, $p$ = .002) and in Model 4 ($b$ = 7.85, $p$ = .001). In Model 4, shorter sleep duration at 3.5 years was found to predict higher BRI difficulties at the intercept ($b$ = -2.05, $p$ = .046). Conversely, a shorter sleep duration at 6.7 years predicted lower BRI difficulties at the intercept in Model 3 ($b$ = 3.15, $p$ = .010), and this remained significant in Model 4 ($b$ = 3.64, $p$ = .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). 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, $p$ = .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 $p$ = .017.

A delayed sleep onset in Model 1 was associated with lower emotional control difficulties at the intercept ($b$ = -2.08, $p$ = .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, $p$ = .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, $p$ = .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, $p$ = .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* = .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* = .004), cognitive shifting (*b* = 2.20, *p* = 0.01) and emotional control (*b* = 2.77, *p* = .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* = .228, *p* = .007) and emotional control difficulties (*b* = 1.07, *p* = .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* = .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* = .123).

## 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[7,73,74]. 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 minutes rather than the 20 minutes on the CSHQ[7,74]. Difficulties initiating sleep for more than 20 to 30-minutes is generally accepted as clinically problematic in pediatric populations[75], 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 minutes to get to sleep, five to seven nights per week. Again, this is compared to the 30-minute 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[76]. 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[72]. 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[77]. However, our findings do parallel preliminary evidence from two cross-sectional studies reporting no significant relationship between sleep and metacognition in children with ASD[46,48]. 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[43,44]. 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[25,78]. 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[79,80], 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[81]. Sleep problems like delayed onset are thought to arise from a de-synchrony between one's endogenous circadian rhythm and preferred sleep-wake schedule[82]. 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[83]. Moreover, delayed sleep phase syndrome, a circadian rhythm disorder characterized by sleep that is delayed by at least two hours[58], is found in adults with ASD[84,85]. 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[10,84] 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[86,87], while decreasing sleep problems across childhood have been correlated with better inhibitory control but not shifting[86]. 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[25,88], 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[89,90]. It is posited that poor sleep impairs the connectivity between the prefrontal cortex and amygdala, a central neural pathway for affective processing[90]. A lack of prefrontal-amygdala connectivity during emotional responses is found in children with ASD[91]: 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[92,93].

**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 hours, 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[6,94]. Negative outcomes for both extremes of under- or over-sleeping 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[95]. 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[43].

**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 modelling 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[96]. 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[97]. 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[98,99]. 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[100]. 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[101]. 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[102,103]. 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[104], 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[105]. 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.

## Disclosures

**a) Financial Disclosure.** Authors acknowledge funding by: The Azrieli Centre for Autism Research (ACAR), The Canadian Institutes of Health Research, Fonds de Recherche du Québec, Kids Brain Health Network (formerly NeuroDevNet), Autism Speaks (US), Government of British Columbia, Alberta Innovates Health Solutions, and the Sinneave Family Foundation. R. Bedford is supported by a King's Prize Fellowship (204823/Z/16/Z). A.Pickles was partially supported by the NIHR NF-SI-0617-10120, the Biomedical Research Centre at South London, the Maudsley NHS Foundation Trust and King's College London. R.Tesfaye is supported by a doctoral fellowship from the Transforming Autism Care Consortium (TACC) and Healthy Brains, Healthy Lives (HBHL).

**b) Non-financial disclosure.** We have no conflicts of interest to disclose.

**Acknowledgment**

We'd like to thank our Pathways families who participated. The authors also acknowledge the past and present members of the Pathways in ASD study team, who made equal contributions to the study.

**References**

1. Richdale AL, Schreck KA. Sleep problems in autism spectrum disorders: Prevalence, nature, & possible biopsychosocial aetiologies. *Sleep Medicine Reviews*. 2009;13(6):403-411. doi:10.1016/j.smrv.2009.02.003

2. Díaz-Román A, Zhang J, Delorme R, Beggiato A, Cortese S. Sleep in youth with autism spectrum disorders: systematic review and meta-analysis of subjective and objective studies. *Evidence-Based Mental Health*. 2018;21(4):146-154. doi:10.1136/ebmental-2018-300037

3. Verhoeff ME, Blanken LME, Kocevska D, et al. The bidirectional association between sleep problems and autism spectrum disorder: a population-based cohort study. *Molecular Autism*. 2018;9(1). doi:10.1186/s13229-018-0194-8

4. Morgan B, Nageye F, Masi G, Cortese S. Sleep in adults with Autism Spectrum Disorder: a systematic review and meta-analysis of subjective and objective studies. *Sleep Medicine*. 2020;65:113-120. doi:10.1016/j.sleep.2019.07.019

5. Williamson AA, Mindell JA, Hiscock H, Quach J. Child sleep behaviors and sleep problems from infancy to school-age. *Sleep Medicine*. 2019;63:5-8. doi:10.1016/j.sleep.2019.05.003

6. Kocevska D, Rijlaarsdam J, Ghassabian A, et al. Early Childhood Sleep Patterns and Cognitive Development at Age 6 Years: The Generation R Study. *J Pediatr Psychol*. 2017;42(3):260-268. doi:10.1093/jpepsy/jsv168

7. Petit D, Touchette É, Tremblay RE, Boivin M, Montplaisir J. Dyssomnias and Parasomnias in Early Childhood. *Pediatrics*. 2007;119(5):e1016-e1025. doi:10.1542/peds.2006-2132

8. Gregory AM, O'Connor TG. Sleep problems in childhood: a longitudinal study of developmental change and association with behavioral problems. *J Am Acad Child Adolesc Psychiatry*. 2002;41(8):964-971. doi:10.1097/00004583-200208000-00015

9. Nguyen AKD, Murphy LE, Kocak M, Tylavsky FA, Pagani LS. Prospective Associations Between Infant Sleep at 12 Months and Autism Spectrum Disorder Screening Scores at 24 Months in a Community-Based Birth Cohort. *J Clin Psychiatry*. 2018;79(1). doi:10.4088/JCP.16m11127

10. MacDuffie KE, Shen MD, Dager SR, et al. Sleep Onset Problems and Subcortical Development in Infants Later Diagnosed With Autism Spectrum Disorder. *AJP*. 2020;177(6):518-525. doi:10.1176/appi.ajp.2019.19060666

11. Sivertsen B, Posserud M-B, Gillberg C, Lundervold AJ, Hysing M. Sleep problems in children with autism spectrum problems: a longitudinal population-based study. *Autism*. 2012;16(2):139-150. doi:10.1177/1362361311404255

12. Humphreys JS, Gringras P, Blair PS, et al. Sleep patterns in children with autistic spectrum disorders: a prospective cohort study. *Arch Dis Child*. 2014;99(2):114-118. doi:10.1136/archdischild-2013-304083

13. Turnbull K, Reid GJ, Morton JB. Behavioral Sleep Problems and their Potential Impact on Developing Executive Function in Children. *Sleep*. 2013;36(7):1077-1084. doi:10.5665/sleep.2814

14. Demetriou EA, Lampit A, Quintana DS, et al. Autism spectrum disorders: a meta-analysis of executive function. *Molecular Psychiatry*. 2018;23(5):1198-1204. doi:10.1038/mp.2017.75

15. Diamond A. Executive Functions. *Annual Review of Psychology*. 2013;64(1):135-168. doi:10.1146/annurev-psych-113011-143750

16. Gioia GA, Isquith PK, Guy SC, Kenworthy L. TEST REVIEW Behavior Rating Inventory of Executive Function. *Child Neuropsychology*. 2000;6(3):235-238. doi:10.1076/chin.6.3.235.3152

17. Zelazo PD, Carlson SM. Hot and Cool Executive Function in Childhood and Adolescence: Development and Plasticity. *Child Development Perspectives*. June 2012:n/a-n/a. doi:10.1111/j.1750-8606.2012.00246.x

18. Best JR, Miller PH, Jones LL. Executive Functions after Age 5: Changes and Correlates. *Developmental review : DR*. 2009;29(3):180. doi:10.1016/j.dr.2009.05.002

19. Prencipe A, Zelazo PD. Development of affective decision making for self and other: evidence for the integration of first- and third-person perspectives. *Psychol Sci*. 2005;16(7):501-505. doi:10.1111/j.0956-7976.2005.01564.x

20. Carlson SM, Davis AC, Leach JG. Less is more: executive function and symbolic representation in preschool children. *Psychol Sci*. 2005;16(8):609-616. doi:10.1111/j.1467-9280.2005.01583.x

21. Willoughby M, Kupersmidt J, Voegler-Lee M, Bryant D. Contributions of Hot and Cool Self-Regulation to Preschool Disruptive Behavior and Academic Achievement. *Developmental Neuropsychology*. 2011;36(2):162-180. doi:10.1080/87565641.2010.549980

22. Prencipe A, Kesek A, Cohen J, Lamm C, Lewis MD, Zelazo PD. Development of hot and cool executive function during the transition to adolescence. *Journal of Experimental Child Psychology*. 2011;108(3):621-637. doi:10.1016/j.jecp.2010.09.008

23. Hooper CJ, Luciana M, Conklin HM, Yarger RS. Adolescents' performance on the Iowa Gambling Task: implications for the development of decision making and ventromedial prefrontal cortex. *Dev Psychol*. 2004;40(6):1148-1158. doi:10.1037/0012-1649.40.6.1148

24. Huizinga M, Smidts DP. Age-Related Changes in Executive Function: A Normative Study with the Dutch Version of the Behavior Rating Inventory of Executive Function (BRIEF). *Child Neuropsychology*. 2010;17(1):51-66. doi:10.1080/09297049.2010.509715

25. Rosenthal M, Wallace GL, Lawson R, et al. Impairments in real-world executive function increase from childhood to adolescence in autism spectrum disorders. *Neuropsychology*. 2013;27(1):13-18. doi:10.1037/a0031299

26. Vogan VM, Leung RC, Safar K, Martinussen R, Smith ML, Taylor MJ. Longitudinal Examination of Everyday Executive Functioning in Children With ASD: Relations With Social, Emotional, and Behavioral Functioning Over Time. *Front Psychol*. 2018;9. doi:10.3389/fpsyg.2018.01774

27. Ozonoff S, McEvoy RE. A longitudinal study of executive function and theory of mind development in autism. *Development and Psychopathology*. 1994;6(3):415-431. doi:10.1017/S0954579400006027

28. Pellicano E. The Development of Core Cognitive Skills in Autism: A 3-Year Prospective Study. *Child Development*. 2010;81(5):1400-1416. doi:https://doi.org/10.1111/j.1467-8624.2010.01481.x

29. O'Hearn K, Asato M, Ordaz S, Luna B. Neurodevelopment and executive function in autism. *Dev Psychopathol*. 2008;20(4):1103-1132. doi:10.1017/S0954579408000527

30. de Vries M, Geurts H. Influence of Autism Traits and Executive Functioning on Quality of Life in Children with an Autism Spectrum Disorder. *J Autism Dev Disord*. 2015;45(9):2734-2743. doi:10.1007/s10803-015-2438-1

31. Lawson RA, Papadakis AA, Higginson CI, et al. Everyday executive function impairments predict comorbid psychopathology in autism spectrum and attention deficit hyperactivity disorders. *Neuropsychology*. 2015;29(3):445-453. doi:10.1037/neu0000145

32. Wallace GL, Kenworthy L, Pugliese CE, et al. Real-World Executive Functions in Adults with Autism Spectrum Disorder: Profiles of Impairment and Associations with Adaptive Functioning and Co-morbid Anxiety and Depression. *J Autism Dev Disord*. 2016;46(3):1071-1083. doi:10.1007/s10803-015-2655-7

33. Tesfaye R, Gruber R. The Association between Sleep and Theory of Mind in School Aged Children with ADHD. *Medical Sciences*. 2017;5(3):18. doi:10.3390/medsci5030018

34. Astill RG, Van der Heijden KB, Van IJzendoorn MH, Van Someren EJW. Sleep, cognition, and behavioral problems in school-age children: A century of research meta-analyzed. *Psychological Bulletin*. 2012;138(6):1109-1138. doi:10.1037/a0028204

35. Courchesne E, Pierce K, Schumann CM, et al. Mapping Early Brain Development in Autism. *Neuron*. 2007;56(2):399-413. doi:10.1016/j.neuron.2007.10.016

36. Horne JA. Human Sleep, Sleep Loss and Behaviour: Implications for the Prefrontal Cortex and Psychiatric Disorder. *The British Journal of Psychiatry*. 1993;162(3):413-419. doi:10.1192/bjp.162.3.413

37. Verweij IM, Romeijn N, Smit DJ, Piantoni G, Van Someren EJ, van der Werf YD. Sleep deprivation leads to a loss of functional connectivity in frontal brain regions. *BMC Neuroscience*. 2014;15(1):88. doi:10.1186/1471-2202-15-88

38. Krause AJ, Simon EB, Mander BA, et al. The sleep-deprived human brain. *Nature Reviews Neuroscience*. 2017;18(7):404-418. doi:10.1038/nrn.2017.55

39. Funahashi S, Andreau JM. Prefrontal cortex and neural mechanisms of executive function. *Journal of Physiology-Paris*. 2013;107(6):471-482. doi:10.1016/j.jphysparis.2013.05.001

40. Stuss DT. Traumatic brain injury: relation to executive dysfunction and the frontal lobes. *Current Opinion in Neurology*. 2011;24(6):584. doi:10.1097/WCO.0b013e32834c7eb9

41. Shay N, Yeates KO, Walz NC, et al. Sleep Problems and Their Relationship to Cognitive and Behavioral Outcomes in Young Children with Traumatic Brain Injury. *Journal of Neurotrauma*. 2014;31(14):1305-1312. doi:10.1089/neu.2013.3275

42. Tham SW, Palermo TM, Vavilala MS, et al. The longitudinal course, risk factors, and impact of sleep disturbances in children with traumatic brain injury. *J Neurotrauma*. 2012;29(1):154-161. doi:10.1089/neu.2011.2126

43. Bernier A, Carlson SM, Bordeleau S, Carrier J. Relations between physiological and cognitive regulatory systems: infant sleep regulation and subsequent executive functioning. *Child Dev*. 2010;81(6):1739-1752. doi:10.1111/j.1467-8624.2010.01507.x

44. Bernier A, Beauchamp MH, Bouvette-Turcot A-A, Carlson SM, Carrier J. Sleep and Cognition in Preschool Years: Specific Links to Executive Functioning. *Child Development*. 2013;84(5):1542-1553. doi:10.1111/cdev.12063

45. Taveras EM, Rifas-Shiman SL, Bub KL, Gillman MW, Oken E. Prospective Study of Insufficient Sleep and Neurobehavioral Functioning Among School-Age Children. *Acad Pediatr*. 2017;17(6):625-632. doi:10.1016/j.acap.2017.02.001

46. Reynolds KC, Patriquin M, Alfano CA, Loveland KA, Pearson DA. Parent-Reported Problematic Sleep Behaviors in Children with Comorbid Autism Spectrum Disorder and Attention-Deficit/Hyperactivity Disorder. *Res Autism Spectr Disord*. 2017;39:20-32. doi:10.1016/j.rasd.2017.04.003

47. Cremone-Caira A, Buirkle J, Gilbert R, Nayudu N, Faja S. Relations between caregiver-report of sleep and executive function problems in children with autism spectrum disorder and attention-deficit/hyperactivity disorder. *Research in Developmental Disabilities*. 2019;94:103464. doi:10.1016/j.ridd.2019.103464

48. Mayes SD, Calhoun SL. Variables related to sleep problems in children with autism. *Research in Autism Spectrum Disorders*. 2009;3(4):931-941. doi:10.1016/j.rasd.2009.04.002

49. Tsai T-H, Chen Y-L, Gau SS-F. Relationships between autistic traits, insufficient sleep, and real-world executive functions in children: a mediation analysis of a national epidemiological survey. *Psychological Medicine*. undefined/ed:1-8. doi:10.1017/S0033291719003271

50. Sletten TL, Rajaratnam SMW, Wright MJ, et al. Genetic and Environmental Contributions to Sleep-Wake Behavior in 12-Year-Old Twins. *Sleep*. 2013;36(11):1715-1722. doi:10.5665/sleep.3136

51. Jansen PR, Watanabe K, Stringer S, et al. Genome-wide analysis of insomnia in 1,331,010 individuals identifies new risk loci and functional pathways. *Nature Genetics*. 2019;51(3):394. doi:10.1038/s41588-018-0333-3

52. Jones SE, Tyrrell J, Wood AR, et al. Genome-Wide Association Analyses in 128,266 Individuals Identifies New Morningness and Sleep Duration Loci. *PLOS Genetics*. 2016;12(8):e1006125. doi:10.1371/journal.pgen.1006125

53. Jones SE, Lane JM, Wood AR, et al. Genome-wide association analyses of chronotype in 697,828 individuals provides insights into circadian rhythms. *Nature Communications*. 2019;10(1):343. doi:10.1038/s41467-018-08259-7

54. Lord C, Risi S, Lambrecht L, et al. The autism diagnostic observation schedule-generic: a standard measure of social and communication deficits associated with the spectrum of autism. *J Autism Dev Disord*. 2000;30(3):205-223.

55. Rutter, M., Le Couteur, A., & Lord, C. Autism diagnostic interview-revised. *Los Angeles, CA: Western Psychological Services*. 2003;29(30).

56. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders (4th Ed., Text Revision).* Washington, DC: Author.; 2000.

57. Owens JA, Spirito A, McGuinn M. The Children's Sleep Habits Questionnaire (CSHQ): Psychometric Properties of A Survey Instrument for School-Aged Children. *Sleep*. 2000;23(8):1-9. doi:10.1093/sleep/23.8.1d

58. Association AP. *Diagnostic and Statistical Manual of Mental Disorders (DSM-5®)*. American Psychiatric Pub; 2013.

59. Roid, G. H., & Sampers, J. L. *Merrill-Palmer-Revised: Scales of Development.* Wood Dale, IL: Stoelting Co; 2004.

60. Gotham K, Pickles A, Lord C. Standardizing ADOS Scores for a Measure of Severity in Autism Spectrum Disorders. *J Autism Dev Disord*. 2009;39(5):693-705. doi:10.1007/s10803-008-0674-3

61. Bennett, T. A., Szatmari P, Georgiades K, et al. Language Impairment and Early Social Competence in Preschoolers with Autism Spectrum Disorders: A Comparison of DSM-5 Profiles. *J Autism Dev Disord*. 2014;44(11):2797-2808. doi:10.1007/s10803-014-2138-2

62. Madden JM, Lakoma MD, Lynch FL, et al. Psychotropic Medication Use among Insured Children with Autism Spectrum Disorder. *J Autism Dev Disord*. 2017;47(1):144-154. doi:10.1007/s10803-016-2946-7

63. Murray ML, Hsia Y, Glaser K, et al. Pharmacological treatments prescribed to people with autism spectrum disorder (ASD) in primary health care. *Psychopharmacology (Berl)*. 2014;231(6):1011-1021. doi:10.1007/s00213-013-3140-7

64. Mandell DS, Morales KH, Marcus SC, Stahmer AC, Doshi J, Polsky DE. Psychotropic Medication Use Among Medicaid-Enrolled Children With Autism Spectrum Disorders. *Pediatrics*. 2008;121(3):e441-e448. doi:10.1542/peds.2007-0984

65. Oswald D, Sonekla N. Medication Use Among Children with Autism-Spectrum Disorders. *Journal of child and adolescent psychopharmcology*. 2007;17(3):348-355.

66. Aman MG, Lam KSL, Collier-Crespin A. Prevalence and Patterns of Use of Psychoactive Medicines Among Individuals with Autism in the Autism Society of Ohio. *J Autism Dev Disord*. 2003;33(5):527-534. doi:10.1023/A:1025883612879

67. Polimeni MA, Richdale AL, Francis AJP. A survey of sleep problems in autism, Asperger's disorder and typically developing children. *Journal of Intellectual Disability Research*. 2005;49(4):260-268. doi:https://doi.org/10.1111/j.1365-2788.2005.00642.x

68. Malow BA, Katz T, Reynolds AM, et al. Sleep Difficulties and Medications in Children With Autism Spectrum Disorders: A Registry Study. *Pediatrics*. 2016;137(Supplement 2):S98-S104. doi:10.1542/peds.2015-2851H

69. Green VA, Pituch KA, Itchon J, Choi A, O'Reilly M, Sigafoos J. Internet survey of treatments used by parents of children with autism. *Research in Developmental Disabilities*. 2006;27(1):70-84. doi:10.1016/j.ridd.2004.12.002

70. Ziskind D, Bennett A, Jawad A, Blum N. Therapy and Psychotropic Medication Use in Young Children With Autism Spectrum Disorder. *Pediatrics*. 2020;145(Supplement 1):S99-S107. doi:10.1542/peds.2019-1895M

71. Hu L, Bentler PM. Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. *Structural Equation Modeling: A Multidisciplinary Journal*. 1999;6(1):1-55. doi:10.1080/10705519909540118

72. Hirshkowitz M, Whiton K, Albert SM, et al. National Sleep Foundation's updated sleep duration recommendations: final report. *Sleep Health*. 2015;1(4):233-243. doi:10.1016/j.sleh.2015.10.004

73. Byars KC, Yolton K, Rausch J, Lanphear B, Beebe DW. Prevalence, Patterns, and Persistence of Sleep Problems in the First 3 Years of Life. *Pediatrics*. 2012;129(2):e276-e284. doi:10.1542/peds.2011-0372

74. Ottaviano S, Giannotti F, Cortesi F, Bruni O, Ottaviano C. Sleep Characteristics in Healthy Children From Birth to 6 Years of Age in the Urban Area of Rome. *Sleep*. January 1996. doi:10.1093/sleep/19.1.1

75. Mindell JA, Owens JA. *A Clinical Guide to Pediatric Sleep: Diagnosis and Management of Sleep Problems*. Lippincott Williams & Wilkins; 2015.

76. Liu X, Liu L, Owens JA, Kaplan DL. Sleep patterns and sleep problems among schoolchildren in the United States and China. *Pediatrics*. 2005;115(1 Suppl):241-249. doi:10.1542/peds.2004-0815F

77. Touchette É, Petit D, Séguin JR, Boivin M, Tremblay RE, Montplaisir JY. Associations Between Sleep Duration Patterns and Behavioral/Cognitive Functioning at School Entry. *Sleep*. 2007;30(9):1213-1219.

78. Luna B, Doll SK, Hegedus SJ, Minshew NJ, Sweeney JA. Maturation of executive function in autism. *Biol Psychiatry*. 2007;61(4):474-481. doi:10.1016/j.biopsych.2006.02.030

79. Cohen S, Conduit R, Lockley SW, Rajaratnam SM, Cornish KM. The relationship between sleep and behavior in autism spectrum disorder (ASD): a review. *Journal of Neurodevelopmental Disorders*. 2014;6(1):44. doi:10.1186/1866-1955-6-44

80. Mazurek MO, Sohl K. Sleep and Behavioral Problems in Children with Autism Spectrum Disorder. *J Autism Dev Disord*. 2016;46(6):1906-1915. doi:10.1007/s10803-016-2723-7

81. LeBourgeois MK, Wright KP, LeBourgeois HB, Jenni OG. Dissonance Between Parent-Selected Bedtimes and Young Children's Circadian Physiology Influences Nighttime Settling Difficulties. *Mind Brain Educ*. 2013;7(4):234-242. doi:10.1111/mbe.12032

82. Lack LC, Lovato N, Micic G. Circadian rhythms and insomnia. *Sleep Biol Rhythms*. 2017;15(1):3-10. doi:10.1007/s41105-016-0072-8

83. Carmassi C, Palagini L, Caruso D, et al. Systematic Review of Sleep Disturbances and Circadian Sleep Desynchronization in Autism Spectrum Disorder: Toward an Integrative Model of a Self-Reinforcing Loop. *Front Psychiatry*. 2019;10. doi:10.3389/fpsyt.2019.00366

84. Baker EK, Richdale AL. Examining the Behavioural Sleep-Wake Rhythm in Adults with Autism Spectrum Disorder and No Comorbid Intellectual Disability. *Journal of Autism and Developmental Disorders; New York*. 2017;47(4):1207-1222. doi:http://dx.doi.org/10.1007/s10803-017-3042-3

85. Hare DJ, Jones S, Evershed K. A comparative study of circadian rhythm functioning and sleep in people with Asperger syndrome. *Autism*. 2006;10(6):565-575. doi:10.1177/1362361306068509

86. Friedman NP, Corley RP, Hewitt JK, Wright KP. Individual differences in childhood sleep problems predict later cognitive executive control. *Sleep*. 2009;32(3):323-333. doi:10.1093/sleep/32.3.323

87. Gregory AM, Caspi A, Moffitt TE, Poulton R. Sleep Problems in Childhood Predict Neuropsychological Functioning in Adolescence. *Pediatrics*. 2009;123(4). doi:10.1542/peds.2008-0825

88. Gioia GA, Isquith PK, Kenworthy L, Barton RM. Profiles of Everyday Executive Function in Acquired and Developmental Disorders. *Child Neuropsychology*. 2002;8(2):121-137. doi:10.1076/chin.8.2.121.8727

89. Baglioni C, Nanovska S, Regen W, et al. Sleep and mental disorders: A meta-analysis of polysomnographic research. *Psychol Bull*. 2016;142(9):969-990. doi:10.1037/bul0000053

90. Gruber R, Cassoff J. The interplay between sleep and emotion regulation: conceptual framework empirical evidence and future directions. *Curr Psychiatry Rep*. 2014;16(11):500. doi:10.1007/s11920-014-0500-x

91. Pitskel NB, Bolling DZ, Kaiser MD, Pelphrey KA, Crowley MJ. Neural systems for cognitive reappraisal in children and adolescents with autism spectrum disorder. *Dev Cogn Neurosci*. 2014;10:117-128. doi:10.1016/j.dcn.2014.08.007

92. Gruber R, Wiebe S, Montecalvo L, Brunetti B, Amsel R, Carrier J. Impact of Sleep Restriction on Neurobehavioral Functioning of Children with Attention Deficit Hyperactivity Disorder. *Sleep*. 2011;34(3):315-323. doi:10.1093/sleep/34.3.315

93. Kuula L, Pesonen A-K, Martikainen S, et al. Poor sleep and neurocognitive function in early adolescence. *Sleep Med*. 2015;16(10):1207-1212. doi:10.1016/j.sleep.2015.06.017

94. van Oostrom SH, Nooyens ACJ, van Boxtel MPJ, Verschuren WMM. Long sleep duration is associated with lower cognitive function among middle-age adults - the Doetinchem Cohort Study. *Sleep Med*. 2018;41:78-85. doi:10.1016/j.sleep.2017.07.029

95. Wing YK, Li SX, Li AM, Zhang J, Kong APS. The Effect of Weekend and Holiday Sleep Compensation on Childhood Overweight and Obesity. *Pediatrics*. 2009;124(5):e994-e1000. doi:10.1542/peds.2008-3602

96. Hall WA, Liva S, Moynihan M, Saunders R. A Comparison of Actigraphy and Sleep Diaries for Infants' Sleep Behavior. *Front Psychiatry*. 2015;6. doi:10.3389/fpsyt.2015.00019

97. Lambert A, Tessier S, Rochette A-C, Scherzer P, Mottron L, Godbout R. Poor sleep affects daytime functioning in typically developing and autistic children not complaining of sleep problems: A questionnaire-based and polysomnographic study. *Research in Autism Spectrum Disorders*. 2016;23:94-106. doi:10.1016/j.rasd.2015.11.010

98. Richdale AL, Schreck KA. Examining sleep hygiene factors and sleep in young children with and without autism spectrum disorder. *Research in Autism Spectrum Disorders*. 2019;57:154-162. doi:10.1016/j.rasd.2018.10.008

99. Reynolds AM, Soke GN, Sabourin KR, et al. Sleep Problems in 2- to 5-Year-Olds With Autism Spectrum Disorder and Other Developmental Delays. *Pediatrics*. 2019;143(3):e20180492. doi:10.1542/peds.2018-0492

100. Sneddon P, Peacock GG, Crowley SL. Assessment of Sleep Problems in Preschool Aged Children: An Adaptation of the Children's Sleep Habits Questionnaire. *Behavioral Sleep Medicine*. 2013;11(4):283-296. doi:10.1080/15402002.2012.707158

101. Zaidman-Zait A, Zwaigenbaum L, Duku E, et al. Factor analysis of the children's sleep habits questionnaire among preschool children with autism spectrum disorder. *Research in Developmental Disabilities*. 2020;97:103548. doi:10.1016/j.ridd.2019.103548

102. Nelson TD, Kidwell KM, Hankey M, Nelson JM, Espy KA. Preschool executive control and sleep problems in early adolescence. *Behavioral Sleep Medicine*. 2018;16(5):494-503. doi:10.1080/15402002.2016.1228650

103. Tomaso CC, Nelson JM, Espy KA, Nelson TD. Associations between different components of executive control in childhood and sleep problems in early adolescence: A longitudinal study. *J Health Psychol*. September 2018:1359105318801065. doi:10.1177/1359105318801065

104. Holingue C, Volk H, Crocetti D, Gottlieb B, Spira AP, Mostofsky SH. Links between parent-reported measures of poor sleep and executive function in childhood autism and attention deficit hyperactivity disorder. *Sleep Health*. January 2021. doi:10.1016/j.sleh.2020.12.006

105. de Bruin EJ, van Run C, Staaks J, Meijer AM. Effects of sleep manipulation on cognitive functioning of adolescents: A systematic review. *Sleep Med Rev*. 2017;32:45-57. doi:10.1016/j.smrv.2016.02.006

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[a] | | 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 [b] | | 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 |

[a] *Age at T1 was used to account for cognitive raw scores not standardized by age.*
[b] 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.

Table 2. *CSHQ Sleep duration frequencies*

| | ***2yrs** (T1) | | **3-4yrs** (T1) | | **6-7yrs** (T4) | |
|---|---|---|---|---|---|---|
| Duration (hours) | 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 20mins'* | | | | |
| *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 |
| $\chi^2$ (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 (.082 -.287) |
| CFI | 0.98 | 0.98 | 0.96 | 0.922 |

Table 5. *Parameter estimates for behavioral regulation difficulties Model 1-4*

| Predictors | Intercept | | | | Slope | | | |
|---|---|---|---|---|---|---|---|---|
| | 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 | .834 | -0.05 (-0.14) | -0.14 - 0.05 | 0.06 | .410 |
| Halifax site | -5.49 (-0.47) | -11.45 - 0.47 | 3.62 | .130 | 1.13 (0.36) | -1.27 - 3.54 | 1.46 | .438 |
| Montreal site | **-6.84 (-0.58)** | **-11.78 - -1.92** | **3.00** | **.022** | 0.17 (0.05) | -1.98 - 2.33 | 1.31 | .895 |
| Edmonton site | 2.29 (0.19) | -3.27 - 7.85 | 3.38 | .498 | -0.72 (-0.23) | -3.85 - -2.42 | 1.90 | .707 |
| Vancouver site | -2.39 (-0.20) | -7.27 - -2.39 | 2.97 | .420 | 1.29 (0.41) | -1.05 - 3.64 | 1.43 | .364 |
| Income | -4.01 (-0.34) | -7.39 - -0.63 | 2.05 | .051 | 0.97 (0.31) | -0.61 - 2.55 | 0.96 | .313 |
| T1 Sleep onset | **-4.14 (-0.35)** | **-7.54 - -0.75** | **2.07** | **.045** | 1.01 (0.32) | -0.55 - 2.57 | 0.95 | .287 |
| T1 Night awakening | 1.53 (0.13) | -1.92 - 4.99 | 2.10 | .466 | 0.01 (0.01) | -1.46 - 1.46 | 0.89 | .999 |
| T1 Sleep duration | -0.80 (0.09) | -2.11 - 0.52 | 0.80 | .317 | 0.31 (0.14) | -0.35 - 0.97 | 0.40 | .437 |
| **Model 2** | | | | | | | | |
| Age | 0.12 (0.09) | -0.12 - 0.35 | 0.14 | .410 | -0.09 (-0.25) | -0.20 - 0.02 | 0.07 | .177 |
| Halifax site | -3.46 (-0.29) | -9.53 - 2.60 | 3.68 | .347 | 1.15 (0.34) | -1.29 - 3.59 | 1.48 | .438 |
| Montreal site | -4.21 (-0.35) | -9.13 - 0.70 | 2.99 | .159 | -0.65 (-0.19) | -2.89 - 1.59 | 1.36 | .633 |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Edmonton site | 5.66 (0.47) | 0.03 - 11.29 | 3.42 | .098 | -0.99 (-0.29) | -4.30 - 2.33 | 2.02 | .624 |
| Vancouver site | -0.95 (-0.08) | -5.93 - 4.02 | 3.02 | .753 | 1.19 (0.36) | -1.22 - 3.61 | 1.47 | .415 |
| Income | -3.16 (-0.26) | -6.80 - 0.48 | 2.21 | .153 | 0.82 (0.25) | -0.92 - 2.56 | 1.06 | .436 |
| ASD Severity | **1.45 (0.20)** | **0.43 - 2.47** | **0.62** | **.020** | -0.53 (-0.26) | -1.03 - -0.03 | 0.31 | .084 |
| Early Cognition | -0.08 (-0.17) | -0.16 – 0.01 | 0.05 | .082 | 0.03 (0.21) | -0.01 - 0.07 | 0.02 | .207 |
| T1 Sleep onset | **-5.44 (-0.45)** | **-8.99 - -1.89** | **2.16** | **.012** | 1.26 (0.37) | -0.42 - 2.93 | 1.02 | .217 |
| T1 Night awakening | 1.553 (0.13) | -2.04 - 5.15 | 2.19 | .477 | 0.32 (-0.10) | -1.89 - 1.25 | 0.95 | .736 |
| T1 Sleep duration | -1.48 (-0.17) | -2.96 - -0.01 | 0.90 | .099 | 0.41 (0.16) | -0.33 - 1.14 | 0.45 | .362 |
| **Model 3** | | | | | | | | |
| Age | -0.01 (1.03) | -0.23 - 0.21 | 0.13 | .926 | -0.01 (-0.02) | -0.12 - 0.10 | -0.11 | .913 |
| Halifax site | -5.36 (0.46) | 12.73 - 2.01 | 4.48 | .231 | 0.93 (0.29) | -2.16 - 4.09 | 1.88 | .622 |
| Montreal site | -5.10 (-0.44) | -10.13 - -0.08 | 3.05 | .094 | -0.03 (-0.01) | -2.32 - 2.25 | 1.39 | .980 |
| Edmonton site | 0.30 (0.02) | -5.62 - 6.23 | 3.60 | .933 | 0.21 (0.07) | -3.54 - 3.97 | 2.27 | .925 |
| Vancouver site | -0.69 (-0.06) | -5.36 - 3.98 | 2.84 | .808 | 1.01 (0.32) | -1.52 - 3.52 | 1.53 | .513 |
| Income | -3.57 (-0.31) | -7.27 - 0.13 | 2.25 | .113 | 1.16 (0.37) | -0.53 - 2.86 | 1.03 | .259 |
| T1 Sleep onset | **-7.19(-0.62)** | **-10.85 - -3.53** | **2.22** | **.001** | **2.07 (0.66)** | **0.40 - 3.73** | **1.01** | **.041** |
| T1 Night awakening | 2.37 (0.20) | -1.69 - 6.44 | 2.47 | .337 | -0.38 (-0.12) | -2.07 - 1.30 | 1.02 | .707 |
| T1 Sleep duration | -1.39 (-0.16) | -2.96 - 0.18 | 0.96 | .146 | 0.22 (0.09) | -0.50 - 0.93 | 0.44 | .620 |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| T4 Sleep onset | **7.78 (0.67)** | **3.69 - 11.87** | **2.49** | **.002** | -1.47 (-0.47) | -3.22 - 0.28 | 1.06 | .166 |
| T4 Night awakening | 3.74 (0.32) | -0.14 - 7.61 | 2.36 | .113 | -0.76 (-0.24) | -2.52 - 1.01 | 1.07 | .479 |
| T4 Sleep duration | **3.15 (0.26)** | **1.14- 5.17** | **1.23** | **.010** | -0.92 (-0.28) | -1.93 - 0.09 | 0.61 | .133 |
| **Model 4** | | | | | | | | |
| Age | 0.02 (0.02) | -0.23 - 0.28 | 0.16 | .883 | -0.025 (-0.06) | -0.16 - 0.11 | 0.08 | .753 |
| Halifax site | -5.54 (-0.47) | 13.29 - 2.20 | 4.71 | .239 | 1.654 (0.46) | -1.56 - 4.87 | 1.95 | .398 |
| Montreal site | -3.53 (-0.30) | -8.13 - 1.06 | 2.79 | .206 | -0.636 (-0.18) | -3.01 - 1.74 | 1.44 | .660 |
| Edmonton site | 2.95 (0.25) | -2.92 - 8.82 | 3.57 | .409 | 0.297 (0.08) | -3.78 - 4.37 | 2.48 | .905 |
| Vancouver site | 0.70 (0.06) | -3.67 - 5.09 | 2.67 | .792 | 0.85 (0.24) | -1.68 - 3.38 | 1.54 | .581 |
| Income | -1.97 (-0.17) | -5.88 - 1.93 | 2.374 | .406 | 0.76 (0.21) | -1.14 - 2.66 | 1.155 | .508 |
| ASD Severity | **1.43 (0.20)** | **0.44 - 2.43** | **0.61** | **.018** | -0.52 (-0.24) | -1.02 - -0.01 | 0.31 | .092 |
| Early Cognition | **-0.10 (-0.20)** | **-0.17 - -0.02** | **0.05** | **.045** | 0.04 (0.25) | -0.01 - 0.08 | 0.03 | .165 |
| T1 Sleep onset | **-8.51 (-0.72)** | **-12.15 - -4.86** | **2.22** | **.000** | **2.37 (0.66)** | **0.63 - 4.10** | **1.05** | **.025** |
| T1 Night awakening | 3.05 (0.26) | -1.01 - 7.11 | 2.47 | .216 | -1.02 (-0.28) | -2.78 - 0.73 | 1.07 | .337 |
| T1 Sleep duration | **-2.05 (-0.24)** | **-3.74- -0.36** | **1.03** | **.046** | 0.27 (0.10) | -0.51 - 1.05 | 0.47 | .565 |
| T4 Sleep onset | **7.85 (0.66)** | **3.86 - 11.84** | **2.43** | **.001** | -1.58 (-0.44) | -3.41 - 0.24 | 1.11 | .154 |
| T4 Night awakening | 4.47 (0.38) | 0.35 - 8.60 | 2.51 | .075 | -0.816 (-0.23) | -2.76 - 1.13 | 1.18 | .490 |
| T4 Sleep duration | **3.64 (0.30)** | **1.54 - 5.74** | **1.28** | **.004** | -1.16 (-0.31) | -2.24 - -0.07 | 0.66 | .079 |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| **Model 4** ***(without medication)*** | | | | | | | | |
| Age | -0.07(-0.05) | -0.40-0.25 | -0.44 | .659 | -0.02(-0.05) | -0.18-0.14 | -0.23 | .815 |
| Halifax site | -7.78(-0.19) | -17.05-1.48 | -1.65 | .099 | 3.34(0.28) | -0.74-7.43 | 1.60 | .109 |
| Montreal site | -5.36(-0.21) | -11.08-0.35 | -1.84 | .066 | 0.68(0.09) | -2.30-3.66 | 0.45 | .654 |
| Edmonton site | 3.05(0.08) | -5.37-11.47 | 0.71 | .478 | 0.07(0.01) | -5.91-6.06 | 0.02 | .981 |
| Vancouver site | 0.328(0.01) | -5.15-5.81 | 0.12 | .907 | 1.47(0.18) | -1.64-4.59 | 0.92 | .354 |
| Income | -1.58(-0.06) | -6.48-3.31 | -0.63 | .526 | 0.45(0.06) | -1.89-2.79 | 0.37 | .707 |
| ASD Severity | **1.55(0.21)** | **0.30-2.81** | **2.42** | **.015** | **-0.68(-0.31)** | **-1.31--0.06** | **-2.15** | **.032** |
| Early Cognition | -0.10(-0.20) | -0.20-0.01 | -1.85 | .065 | 0.04(0.25) | -0.02-0.09 | 1.285 | .199 |
| T1 Sleep onset | **-7.30(-0.29)** | **-12.20--2.41** | **-2.93** | **.003** | 1.88(0.26) | -0.39-4.16 | 1.62 | .104 |
| T1 Night awakening | 0.13(0.01) | -5.33-5.60 | 0.05 | .961 | 0.03(0.004) | -2.31-2.37 | 0.02 | .980 |
| T1 Sleep duration | -2.50(-0.26) | -4.71--0.29 | -2.22 | .026 | 0.38(0.14) | -0.60-1.37 | 0.76 | .449 |
| T4 Sleep onset | **8.44(0.33)** | **3.28-13.59** | **3.21** | **.001** | -1.64(-0.22) | -3.91-0.64 | -1.41 | .158 |
| T4 Night awakening | 4.96(0.20) | -0.45-10.37 | 1.80 | .072 | -0.14(-0.02) | -2.60-2.32 | -0.11 | .909 |
| T4 Sleep duration | **4.14(0.33)** | **1.44-6.84** | **3.01** | **.003** | -0.98(-0.26) | -2.33-0.37 | -1.42 | .155 |