Psychological Medicine

cambridge.org/psm

Original Article

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Cite this article: Tsai T-H, Chen Y-L, Gau SS-F (2021). Relationships between autistic traits, insufficient sleep, and real-world executive functions in children: a mediation analysis of a national epidemiological survey. *Psychological Medicine* **51**, 579–586. https://doi.org/10.1017/S0033291719003271

Received: 12 February 2019
Revised: 16 October 2019
Accepted: 24 October 2019
First published online: 26 November 2019

Key words:
Autism spectrum disorder; autistic traits; executive function; insufficient sleep; mediation

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Relationships between autistic traits, insufficient sleep, and real-world executive functions in children: a mediation analysis of a national epidemiological survey

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Abstract

Background. Although the literature documents low executive functions and sleep deficits in individuals with autism spectrum disorder or subclinical autistic traits, no study has simultaneously examined their relationships in the general child population. This study aimed to examine whether autistic traits impacted real-world executive functions through insufficient sleep in a nationally representative sample of children.

Methods. This was a national survey of 6832 primary and secondary school students, aged 8–14 years old, with equal sex distribution (3479 boys, 50.8%). Parents reported their child's nocturnal sleep duration and the need for sleep to maintain their daytime function and the Social Responsiveness Scale and the Behavior Rating Inventory of Executive Function (BRIEF) for their children's autistic traits and real-world executive functions, respectively.

Results. We found that autistic traits exerted indirect effects on real-world executive functions through sleep deficits, independent of sex, and age. Moreover, such an indirect effect was observed only from restricted and repetitive behaviors to executive functions through sleep deficits, but not in the other components of autistic traits (i.e. social communication and interaction).

Conclusions. Our novel findings underscore the importance of sleep and autistic traits in executive functions and suggest potential mechanisms that may underlie the observed correlational structure among autistic behaviors, sleep deficits, and low executive function performance.

Introduction

The executive function deficit theory is among the neuropsychological accounts of autism spectrum disorder (ASD) (Hill, 2004). Convergent evidence supports impaired executive functions in individuals with ASD, regardless of performance-based or real-world rating measures of executive functions (Baron, 2000; Chen et al., 2016; Chien et al., 2015; Demetriou et al., 2018). With emerging evidence that better executive functions predict better adaptive functioning and quality of life in patients with ASD (de Vries & Geurts, 2015; Pugliese et al., 2015), it is important to identify modifiable factors that influence executive functions in individuals with ASD. Moreover, a few studies have reported an inverse association between autistic traits and executive functions in the community-based child population (Dai, Lin, Liang, Wang, & Jing, 2018; Hyseni et al., 2018; Leung, Vogan, Powell, Anagnostou, & Taylor, 2016). However, the nature of this association remained unclear. More studies are warranted to examine executive function and autistic traits and their modifiable factors in the general child population to understand ASD better and improve the mental health in children.

Executive functions encompass a collection of cognitive processes that regulate cognitive, emotional, and behavioral functions and enable individuals to demonstrate self-regulation, perform goal-directed behaviors, and find solutions to novel problems that cannot be solved with habitual behaviors (Baron, 2000; Diamond, 2013). Deficits in these processes are proposed to underlie restricted and repetitive patterns of behaviors (Mostert-Kerckhoffs, Staal, Houben, & de Jonge, 2015) and social impairment (Leung et al., 2016) in ASD. Alternatively, it is posited that high executive functions may protect children with genetic risks from ASD by compensating atypical functioning in other brain systems in their early life (Johnson, 2012) and could mask symptoms of ASD despite persisting core deficits at cognitive and/or neurobiological levels in individuals with ASD (Livingston, Colvert, Social Relationships Study, Bolton, & Happé, 2019; Livingston & Happe, 2017). Although current evidence does not find impairment in a single subdomain of executive functions to be a universal feature of individuals with ASD (Demetriou et al., 2018), it is generally accepted that

https://doi.org/10.1017/S0033291719003271 Published online by Cambridge University Press

executive function deficits are widespread in individuals with ASD and individual variation of executive functions may contribute to the heterogeneity of other area of difficulty in ASD, language, (Weismer, Kaushanskaya, Larson, Mathée, & Bolt, 2018) and adaptive functioning (Kenny, Cribb, & Pellicano, 2018) for instance.

In contrast to the view that primary deficits in executive functions relate to ASD diagnosis and symptoms, some researchers have hypothesized that core features or comorbidities of ASD serve as limiting factors on the development of executive functions (Pellicano, 2012). For typically developing children, executive functions are expected to improve with age from childhood through adolescence to adulthood due to brain maturation (Best & Miller, 2010; Happe, Booth, Charlton, & Hughes, 2006). With improving executive functions, children are able to learn new knowledge and adaptive behavior skills (Sulik et al., 2015). Children's behavioral problems or inadequate cognitive functions related to immature executive ability observed in their early life are assumed to decline with age (Sulik et al., 2015). However, children with some neurodevelopmental difficulties may encounter difficulties in brain maturation with time. For example, executive functions in children with ASD do mature over time. Yet their maturation is slower than typically developing children (Chen et al., 2016; Happe et al., 2006) and often unable to compensate for the increasing demands of executive functions to behave age-appropriately, which may result in increased real-world executive function deficits (Pugliese et al., 2015; Rosenthal et al., 2013). Children with or without ASD can learn executive function skills and improve their performance via social interaction and participation of cognitively challenging activities (Diamond, 2013; Kenworthy et al., 2014). Thus, it is possible that autistic traits continuously contribute to low executive function performance through impairing children's behavior patterns, or through comorbidities during their childhood and adolescence. Understanding these possible pathways may lead to developing effective interventions to improve patients' adaptive functioning and quality of life.

The literature documents that insufficient sleep could impair working memory and other executive functions in typically developing child and adolescent populations (Astill, Van der Heijden, Van Ijzendoorn, & Van Someren, 2012; de Bruin, van Run, Staaks, & Meijer, 2017). In one cohort study of 1046 children, children who slept less than the sleep duration recommended from the National Sleep Foundation in their preschool or early school years had poorer executive functions in their later life (Taveras, Rifas-Shiman, Bub, Gillman, & Oken, 2017). Moreover, some researchers posited that chronic insufficient sleep has cumulative effects on children's executive functions (Beebe, 2011). However, the effect size of sleep duration on executive functions seems to increase with age (Lowe, Safati, & Hall, 2017) and is small (Astill et al., 2012) or not significant (Short et al., 2018) in school-aged children. It is under debate whether executive functions of school-aged children are truly less vulnerable of sleep restriction than young adults (Astill et al., 2012) or methodological difficulties may contribute to the current evidence of small or no effects (Short et al., 2018).

There is a growing concern about the effect of insufficient sleep on children with ASD due to the high prevalence of sleep initiation and maintenance problems in these children (Cortesi, Giannotti, Ivanenko, & Johnson, 2010). Evidence showed that children with ASD had comparatively short nocturnal sleep duration (Humphreys et al., 2014; Souders et al., 2017) and a greater difference between actual nocturnal sleep duration and sleep need (Chou et al., 2012) as compared to typically developing children. Similarly, the inverse association between levels of autistic traits and sleep duration was noted in typically developing adolescents even after comorbid psychiatric symptoms were controlled (Salmela, Kuula, Merikanto, Raikkonen, & Pesonen, 2018). Moreover, a recent study showed that the diagnosis of ASD predicted more severe and chronic sleep problems in children's later life, but not reversely (Verhoeff et al., 2018). Therefore, it is possible that ASD and autistic traits impaired children's daytime functioning partially through insufficient sleep (Cohen, Conduit, Lockley, Rajaratnam, & Cornish, 2014). However, few studies have documented the relationships between subclinical autistic traits and sleep duration, and the effects of insufficient sleep on executive functions in children with ASD or children with autistic traits are not well-established. Only one related study reports the sleep quality is inversely correlated with some components of executive functions (selective attention) but not others (working memory and sustained attention) in patients with ASD (Limoges, Bolduc, Berthiaume, Mottron, & Godbout, 2013).

In this study, as an attempt to characterize the relationships among autistic traits, insufficient sleep, and real-world executive functions, we examined the possible mediation relationships using a nationally representative sample from Taiwan's National Epidemiological Study of Child Mental Disorders (Chen, Chen, Lin, Shen, & Gau, 2019). Based on the current literature, we hypothesized that insufficient sleep mediates the links between autistic traits and low executive function performance. Moreover, we examined the degree to which subcomponents of autistic traits uniquely contributed to the mediation. Our study would provide an opportunity to examine the possible relationship for how autistic traits and insufficient sleep interacted to bring a negative impact on the real-world executive functions in children.

Methods

Participants

Participants in this study were from Taiwan's National Epidemiological Study of Child Mental Disorders, which is a school-based epidemiological study of DSM-5 childhood mental disorders using a nationally representative sample of third, fifth, and seventh graders ($n = 10\,118$) from 69 schools in Taiwan during June 2015 to January 2017 (Chen et al., 2019). The eligible students were selected with the stratified (urbanicity levels and geographic strata) clustering sampling method. Three informants completed questionnaires in the study, i.e. 9560 student participants (94.5%), 6846 parents (67.7%), and 9758 teachers (96.5%) of student participants. We only selected children whose parents completed parent-reported questionnaires in the current study ($n = 6846$). These children were at the mean age 11.3 (S.D. = 1.74) year old with age ranging from 8 to 14 years old, and 50.8% ($n = 3479$) of them were boys. We further excluded 14 children because of missing data on their sex, yielding a final sample of 6832 children to be included in the current study. The characteristics of these 6832 children are presented in Table 1. The study was approved by the Research Ethics Committee of National Taiwan University Hospital, Taipei, Taiwan (approval number: 201411056RINA) and all the student participants, their parents, and their teachers provided their written informed consent to the study before study implementation.

Table 1. Demographics, autistic traits, sleep parameters, and real-world executive functions of participants

	Total sample ($n = 6832$)
Variables	*N* (%)
Boy	3479 (50.9%)
Grade	
3 (reference)	1941 (28.4%)
5	1932 (28.3%)
7	2959 (43.3%)
	Mean (s.d.)
Age	11.25 (1.78)
SRS total score	31.84 (17.39)
Restricted and repetitive behavior	15.66 (3.88)
Social communication and interaction	90.45 (14.29)
Sleep duration (h)	8.09 (0.88)
Sleep need (h)	8.45 (0.91)
Sleep deficits (h)	0.56 (0.76)
BRIEF	
Global executive composite	138.51 (39.26)
Behavioral regulation index	58.95 (17.10)
Metacognition index	79.57 (22.32)
Inhibit	15.35 (4.68)
Shift	12.01 (3.49)
Emotional control	20.28 (5.84)
Self-monitor	11.32 (3.60)
Initiate	15.15 (4.48)
Working memory	16.20 (4.68)
Plan/Organize	20.03 (5.76)
Task monitor	12.14 (3.55)
Organization of materials	16.06 (4.52)

SRS, Social Responsiveness Scale; BRIEF, Behavior Rating Inventory of Executive Function-Parent Form.

Measures

The Chinese version of the Social Responsiveness Scale (SRS)

The SRS is a parent-reported 4-point Likert-type scale for quantifying autistic traits based on the frequency of each commonly observed behavior (Constantino et al., 2003). The SRS is a 65-item scale using a 4-point scale from 0 (never true) to 3 (almost always true), and it has been reported that the Chinese version of SRS has excellent reliability and validity (Gau, Liu, Wu, Chiu, and Tsai, 2013). The SRS scores demonstrate a continuous distribution in the general population (Hsiao, Tseng, Huang, & Gau, 2013), which is not affected by intelligence, age, race, or the education level of respondents (Constantino et al., 2003). We further applied the two-factor model (i.e. 'restricted and repetitive behavior' and 'social communication and interaction'), which was derived a priori based on current views of autism symptom structure represented in support of the DSM-5 and validated for the SRS by Frazier et al. (2012). In the current study, we used the SRS to measure autistic traits and access two subcomponents of autistic traits, 'restricted and repetitive behavior' and 'social communication and interaction.' A confirmatory factor analysis was conducted to examine the differences in the model fit between the unidimensional factor model and the two-factor model of SRS. The two-factor model had a better model fit than the unidimensional factor model based on the chi-square change test $\Delta\chi^2(1) = 145.4$, $p < 0.001$.

Sleep deficits

The Sleep Habit Questionnaire (SHQ) is designed to investigate sleep habits in children and adolescents aged 4–18 years old (Gau & Soong, 2003). For the sleep need and nocturnal sleep duration about the children, the parents were asked 'how many hours of sleep does your child need every night to maintain his/her daytime functions?' and 'how many hours does your child sleep every night on average?' in the past 6 months. Space for hours and minutes are provided following the question for the student participants and parents to fill in. The data of these two questions were double-checked with the reports by the student participants. The child–parent agreement for sleep duration and sleep need were 0.36 and 0.17, respectively. Furthermore, the child–parent agreement increased across grade 3, grade 5, and grade 7 from 0.18 to 0.41 for sleep duration and from 0.10 to 0.22 for sleep need with all $ps < 0.001$ (online Supplementary Table S1). However, because sleep needs are variable in children and adolescents, it has been suggested sleep duration alone not be an adequate indicator of insufficient sleep in this age group (Anderson, Storfer-Isser, Taylor, Rosen, & Redline, 2009). Following the suggestion of our early work (Gau & Merikangas, 2004; Gau & Soong, 2003) and Hysing, Pallesen, Stormark, Lundervold, & Sivertsen (2013), we used the difference between perceived sleep need and nocturnal sleep duration (i.e. sleep need minor sleep duration hereafter referred to as 'sleep deficits') as an indicator of insufficient sleep in this study (Gau & Merikangas, 2004; Hysing et al., 2013).

The Chinese version of the Behavior Rating Inventory of Executive Function (BRIEF)

We used the Chinese version of the BRIEF Parent form, an 86-item questionnaire for assessing daily, real-world executive function abilities in children aged 5–18 (Baron, 2000). The BRIEF encompasses two broader indices with eight clinical subscales: (1) Behavioral Regulation Index (BRI: Inhibit, Shift and Emotional Control) and (2) Metacognition Index (MI: Initiate, Working Memory, Planning and Organizing, Organization of Materials and Monitor), and an overall index, the Global Executive Composite (GEC). The GEC represents how a child performs in solving problems in unstructured, uncontrolled environments. It has been suggested that the BRIEF has good psychometric properties, including high interrater and retest reliability and face and predictive validity (Baron, 2000).

Statistical analysis

Analyses in this study were performed using SAS 9.4 (SAS Institute Inc., Cary, NC). For the mediation analyses, we used the PROCESS macro (version 2.16) developed by Hayes in the SAS (Hayes, 2013).

https://doi.org/10.1017/S0033291719003271 Published online by Cambridge University Press

Missing data

Proportions of participants with missing data on the SRS score, nocturnal sleep duration, and sleep need were 5.6%, 8.9%, and 10.2%, respectively; while the percentage of missing data on all BRIEF subscales and indices ranged from 6.2% to 6.4%. We used the Expected-Maximization algorithm to impute these missing variables based on variables that were part of the planned analysis.

Data binning

In addition to analysis using the original raw data of SRS, to reduce the effects of minor observation errors, we conducted a data binning for autistic traits (i.e. the total score of SRS). We used 20 bins with each bin including 5% of participants as the predictor variable for autistic traits. However, we used 10 bins for two subcomponents of autistic traits, restricted and repetitive behavior, and social communication and interaction because there was a narrower score range in subscales than in the total score in the SRS.

Models

Two models were examined in the current study: (1) mediation model with covariates (Fig. 1*a*) and (2) mediation model with multiple predictors (Fig. 1*b*). In the mediation model (Fig. 1*a*), 'sleep deficits' are considered the potential mediators of the link between autistic traits and various real-world executive functions while controlling for sex and age. Following suggestion from Hayes (2013), we calculated the indirect effects, the products of a (the pathway from autistic traits to sleep deficits) and b (the pathway from sleep deficits to real-world executive functions), that represent how autistic traits influence sleep deficits, and in turn influence real-world executive functions. For a mediation model with multiple predictors (Fig. 1*b*), the total score of SRS was replaced by 'social communication and interaction' and 'restricted and repetitive behavior.' The 95% bias-corrected (BCa) bootstrap confidence intervals (CIs) with 10 000 bootstrap samples were used to test the statistical significance of the indirect effects. The proportion of the total effect explained by the mediation variables was provided to assess the effect size of the indirect effects (Hafeman, 2009). The PROCESS (version 2.16) developed by Hayes (2013), in which different numbers are used to specify different preset complex models. The PROCESS (version 2.16) Model 4 was used for mediation models in this study. A sensitivity analysis was conducted to determine the differences between the analyses using the SRS scores with and without data binning.

Results

Table 1 presents the descriptive characteristics, means, and S.D. of the subscales of the BRIEF, the subcomponents of the SRS, sleep duration, sleep need, and sleep deficits. The two components of the SRS, 'social communication and interaction' and ' restricted and repetitive behavior,' were moderately correlated with each other (Pearson's $r = 0.53$, $ps < 0.01$).

Sleep deficits as a mediator in the link between autistic traits and executive functions

Table 2 presents the unstandardized coefficient estimates, indirect effects, and 95% BCa bootstrap CIs for the mediation model with the indices of the BRIEF as dependent variables. Generally, we found that children with higher autistic traits had more sleep deficits and increased sleep deficits were found associated with more real-world executive function deficits. Furthermore, we found that sleep deficits mediate the link of autistic traits and real-world executive function deficits. The mediating role of sleep deficits were found for autistic traits on all subscales of the BRIEF (GEC, behavioral regulation index, and metacognition index) in Table 2 and the BRIEF index (inhibition, shift, emotional control, self-monitor, initiate, working memory, plan/organize, task monitor, and organization of materials) in online Supplementary Table S2 after adjusting for sex and age.

Contribution of subcomponents of autistic traits

To examine whether the mediation relationship differed between two specific subcomponents of autistic traits ('restricted and repetitive behavior' and 'social communication and interaction'), we simultaneously analyzed the two subcomponents of the SRS. We found that only 'restricted and repetitive behavior' had independent prediction on sleep deficits (unstandardized coefficient estimates = 0.063, $p < 0.001$), but not social communication and interaction (unstandardized coefficient estimates = 0.032, $p > 0.05$). As a result, the significant indirect effect was only found in restricted and repetitive behavior on executive function through sleep deficits, but not in social communication and interaction although both restricted and repetitive behavior and social communication and interaction had significant direct effects on executive function. The detailed unstandardized coefficient estimates, indirect effects, and 95% BCa bootstrap CIs for the mediation model with multiple predictors are shown in Table 3. The results of the sensitivity analysis indicated that statistical significance remained the same, regardless of the approaches with or without data binning.

Discussion

To the best of our knowledge, the current work is the first study to examine sleep deficits as a mediator between the link of autistic traits and real-world executive functions and to explore whether subcomponents of autistic traits differentially influence executive functions through sleep deficits in a nationally representative sample of children. The results support our hypothesis that autistic traits exert detrimental indirect effects on children's executive functions through sleep deficits independent of sex and age. Moreover, we found that when two subcomponents corresponding to the DSM-5 defining ASD were considered simultaneously, 'restricted and repetitive behavior,' instead of 'social communication and interaction,' continued to uniquely exert indirect effects on executive functions through sleep deficits. Some speculations of potential mechanisms that might underlie the observed correlational structure are provided in the following discussion.

Our finding of the association between autistic traits and impaired real-world executive functions is consistent with previous studies (Chen et al., 2016; Chien et al., 2015; Demetriou et al., 2018), suggesting that executive dysfunctions might be one of the common psychopathologies in the diagnosis of ASD and dimensions of autistic features as well (Leung et al., 2016). Furthermore, our work replicated and extended the result of a prior study in adolescents (Salmela et al., 2018) that sleep deficits are inversely associated with the level of autistic traits in a sample of over 6000 typically developing school-aged children, suggesting the correlation between autistic traits and sleep deficits is not

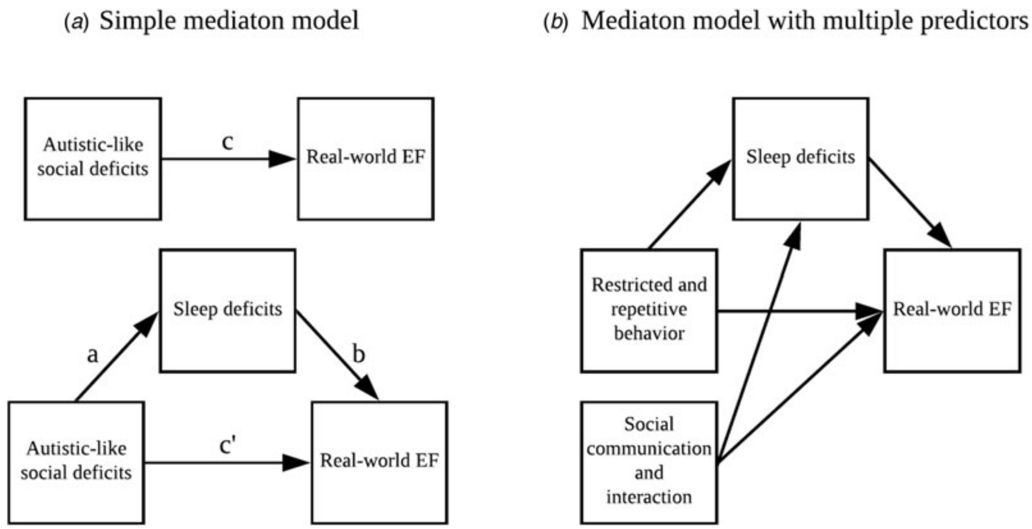

Fig. 1. Visualization of mediation models of autistic traits, insufficient sleep and real-world executive functions. (*a*) A simple mediation model of autistic traits on real-world executive functions through insufficient sleep. (*b*) A mediation model of social communication and interaction and restricted and repetitive behavior on real-world executive functions through insufficient sleep.

Table 2. Results of the mediation models of autistic traits, sleep deficits, and real-world executive functions

	Unstandardized estimates (s.e.)				Indirect effects (s.e.)		
BRIEF	a	b	c	c′	ab	95% BCa CI	Effect size (ab/c)
GEC	0.02 (0.002)***	3.82 (0.59)***	2.25 (0.071)***	2.19 (0.072)***	0.06 (0.01)	0.0427–0.0851	0.03
BRI	0.02 (0.002)***	1.61 (0.26) ***	0.97 (0.03) ***	0.95 (0.03) ***	0.03 (0.03)	0.0172–0.0359	0.03
MI	0.02 (0.002)***	2.21 (0.033)***	1.27 (0.04) ***	1.24 (0.04) ***	0.04 (0.006)	0.0244–0.0493	0.03

BCa CI, bias-corrected bootstrap confidence interval; BRIEF, Behavior Rating Inventory of Executive Function; GEC, Global Executive Composite; BRI, Behavioral Regulation Index; MI, Metacognition Index; s.e., standard error.
Note: Indirect effects (ab), direct effect (c′), total effect (c). If the 95% BCa CI of ab does not include zero, it is suggested that autistic traits exerted indirect effects on executive functions through sleep deficits.
In this mediation model, the predictor is the total score of the social responsiveness scale, the mediator is sleep deficits, and outcomes are the subscales of BRIEF.
Sex and age were covariates.
*$p < 0.05$, **$p < 0.001$.

Table 3. Results of the mediation model of restricted and repetitive behavior and social communication and interaction of Social Responsiveness Scale (SRS), Sleep Deficit, and GEC of BRIEF

	Unstandardized estimates (s.e.)				Indirect effects (s.e.)		
							Effect size
SRS	a	b	c	c′	ab	95% BCa CI	(ab/c)
Social communication and interaction	0.063 (0.015)	3.52 (0.58)***	0.62 (0.07)***	0.62 (0.07)***	0.05 (0.01)	−0.0172 to 0.0286	0.08
Restricted and repetitive behavior	0.032 (0.003)***	3.52 (0.58) ***	4.39 (0.15) ***	4.28 (0.15) ***	0.11 (0.02)	0.0746–0.1580	0.03

BCa CI, bias-corrected bootstrap confidence interval; BRIEF, Behavior Rating Inventory of Executive Function; GEC, Global Executive Composite; s.e., standard error.
Note: Indirect effects (ab), direct effect (c′), total effect (c). If the 95% BCa CI of ab does not include zero, it is suggested that autistic traits exerted indirect effects on executive functions through sleep deficits.
In this mediation model with multiple predictors, predictors are two subscales of the SRS (i.e. social communication and interaction and restricted and repetitive behavior) mediator is sleep deficits, and outcomes are the GEC of BRIEF.
Sex and age were covariates.
**$p < 0.01$, ***$p < 0.001$.

exclusively in children with clinical diagnosis of ASD but also general child population. Moreover, our mediation analyses of overall autistic traits (assessed by the total score of the SRS) are consistent with previous studies reporting that autistic traits and insufficient sleep were inversely associated with executive functions in children (Astill et al., 2012; Hyseni et al., 2018).

https://doi.org/10.1017/S0033291719003271 Published online by Cambridge University Press

We assessed two subcomponents of autistic traits, 'social communication and interaction' and 'restricted and repetitive behavior,' and found that both subcomponents were associated with sleep deficits, which was consistent with previous studies in children with ASD (Hollway, Aman, & Butter, 2013). However, the results of our analyses further point out that the mediation relationship of sleep problems between core symptoms and executive functions was only observed in restricted and repetitive behavior, but not in social communication and interaction. A recent review study suggested that restricted and repetitive behavior may be a precipitating factor of behavioral insomnia (Mazzone, Postorino, Siracusano, Riccioni, & Curatolo, 2018). Cognitive inflexibility, significant distress, and time-consuming rituals from restricted and repetitive behavior may delay sleep onset or determine physiological hyperarousal and emotional overreaction (Harvey, Tang, & Browning, 2005). In addition, hyperreactivity of sensory input may also contribute to difficulty in initializing and maintaining sleep (Mazurek & Petroski, 2015). By contrast, social communication and interaction act more like a perpetuating factor for sleep problems (Ibañez et al., 2018). These social deficits can interfere with the child to follow parents' sleep instruction or to seek help for insomnia (Mazzone et al., 2018). Our findings support the different roles of restricted and repetitive behavior and social communication and interaction for sleep problems and suggest the mediation relationship of autistic traits and impaired real-world executive functions through sleep deficits is specifically related to restricted and repetitive behavior. However, it is worth noting that some researchers suggested that social deficits may have a more profound effect on sleep. Frustration in social situations may result in anxiety, which was found to be associated with the severity of sleep problems in individuals with ASD (Hollway et al., 2013). Further studies are warranted to distinguish the differential roles of restricted and repetitive behavior and social communication and interaction on sleep and executive functions.

Limitations and strengths

Some methodological limitations of this study need to be mentioned when interpreting our findings. First, although we adopted some theories and hypotheses to frame these causal relationships between autistic traits, sleep deficits, and real-world executive functions, the causality was still limited due to the cross-sectional study design in this study. Second, the generalizability of this study to the clinical ASD population and other age groups is limited because we used the SRS rather than the clinical diagnosis in a general child population with the age range from 8 to 14 years old. Third, the child–parent agreement on sleep variables was not high in the current study. However, such low child–parent agreement has also observed in a previous study (Liu, Wang, Ji, Cui, & Liu, 2018). It has been reported that greater child–parent agreement would be more easily to be found in children with more severe mental health problems because of the presence of significant symptoms (Bajeux et al., 2018). Because the study sample is a nationally representative school-based sample, we might not observe high child–parent agreement on their reports. Finally, although the SRS is a widely used screening questionnaire to measure autistic traits and ASD severity, some researchers suggest that the score of SRS may be more appropriately interpreted as an indicator of general levels of social impairment in the non-clinical population (Hus, Bishop, Gotham, Huerta, & Lord, 2013).

By contrast, several features of this study constitute its strengths. First, the current study was conducted in a large-scale nationally representative sample, providing high statistical power to examine the possible patterns of statistical associations. Second, unlike previous ASD-related studies usually used only the categorical approach, we used the dimensional approach regarding autistic traits and found consistent results. Furthermore, among studies of subclinical autistic traits, our study is the first to access the relationship between sleep and different components of autistic traits, instead of considering autistic traits as one dimension. We found that the mediation relationship from autistic traits to real-world executive functions through sleep deficits was more profoundly in the dimension of restricted and repetitive behaviors than in the dimension of social communication and interaction. Third, instead of sleep duration, we used sleep deficits as an indicator to better capture insufficient sleep in an age group with relatively variable sleep need. Finally, we measured real-world executive functions using the BRIEF, which assessed more components of executive functions than most laboratory-based tests assessing executive functions. Furthermore, BRIEF was a sensitive and reliable tool for identifying ASD-associated cognitive deficits (Demetriou et al., 2018). Future studies should extend our findings using laboratory-based or performance-based executive function tests in the clinical ASD group.

Supplementary material. The supplementary material for this article can be found at https://doi.org/10.1017/S0033291719003271

Acknowledgements. We express our thanks to all the students, parents, and teachers who participated in the study.

Financial support. This work was supported by grants from the Ministry of Health and Welfare (M03B3374), National Health Research Institute (NHRI-EX104-10404PI, NHRI-EX105-10404PI, and NHRI-EX106-10404PI), and the Ministry of Science and Technology (MOST 103-2314-B-002-021-MY3).

Conflict of interest. None declared.

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