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Examining the impact of physical activity on sleep quality and executive functions in children with autism spectrum disorder: A randomized controlled trial

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Abstract

Sleep disturbance and executive dysfunction have been widely reported in children with autism spectrum disorder. While the positive impacts of physical activity on sleep quality and cognition are documented in children with typical development, similar studies in children with autism spectrum disorder are scarce. The objective of this study was to examine the impact of physical activity on sleep quality and cognition in children with autism spectrum disorder. A total of 40 children diagnosed with autism spectrum disorder (mean age = 9.95 years) were randomly assigned into two groups: physical activity intervention and control. Four sleep parameters (sleep efficiency, sleep onset latency, sleep duration, and wake after sleep onset) and two executive functions (inhibition control and working memory) were assessed. Results revealed a significant improvement in sleep efficiency, sleep onset latency, and sleep duration in the intervention group but not in the control group during weekdays. Moreover, a significant improvement in inhibitory control was shown in the intervention group but not in the control group. No significant improvement in working memory capacity was documented in either group ($ps > 0.05$). Our findings highlight the value of physical activity in improving sleep quality and cognition among children with autism spectrum disorder, but specific physical activity may be required to benefit individual executive functions.

Keywords

autism spectrum disorder, children, inhibition control, physical activity, sleep, working memory

Introduction

Children with autism spectrum disorder (ASD) are characterized by deficits in social ability, abnormalities in communication, and repetitive stereotypic behavior (World Health Organization, 2016). In addition to these core symptoms, sleep disturbance is also commonly observed among children with ASD (Cortesi, Giannotti, Ivanenko, & Johnson, 2010; Liu, Hubbard, Fabes, & Adam, 2006). Several studies have reported that the prevalence of sleep disturbance is higher in children with ASD (40% to 80%) than in children with typical development (TD; 25% to 40%) (Calhoun, Fernandez-Mendoza, Vgontzas, Liao, & Bixler, 2014; Goldman, Richdale, Clemons, & Malow, 2012). The most frequently reported types of sleep disturbance include delayed sleep onset, difficulty in sleep maintenance, and insufficient sleep duration (SD; Cortesi et al., 2010; Richdale & Schreck, 2009; Souders et al., 2009). Research shows that poor sleep can exacerbate various symptoms of autism, resulting in increased stereotypic behavior (Mazurek & Sohl, 2016), more severe

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communication impairment (Taylor, Schreck, & Mulick, 2012), and intensified emotional control problems (Gregory & Sadeh, 2012). Meanwhile, given the importance of sleep on cognitive development, memory processes, and school performance in TD children (Curcio, Ferrara, & De Gennaro, 2006), it is reasonable to believe that consequences of sleep disturbance on cognitive functioning in children with ASD are also taxing. In a recent case-control study, Maski and colleagues (2016) indicated poor sleep could aggravate the impaired memory consolidation in children with ASD.

In fact, cognitive impairments are commonly found among children with ASD (Russo et al., 2007; Sachse et al., 2012). Executive functions generally refer to several interacting but potentially dissociable mental operations, including planning, working memory, inhibition, and flexibility (Ozonoff, 1995). Children with ASD are often reported to have poor working memory capacity (Barendse et al., 2013; Russell, Jarrold, & Henry, 1996), difficulties in switching attention between tasks (Reed, Watts, & Truzoli, 2013; Wallace et al., 2016), and inhibition response problems (Hopkins, Yuill, & Branigan, 2017), which can seriously affect their school performance and everyday functioning (Gilotty, Kenworthy, Sirian, Black, & Wagner, 2002). Meanwhile, it is well-documented that sleep disturbance and poor cognitive functioning are closely related to each other (see Dewald, Meijer, Oort, Kerkhof, & Bogels, 2010, for review). With the long-term negative interaction effects posed by sleep disturbance and executive dysfunction, developing an effective intervention that can directly address both cognitive deficits and sleep disturbance is critical. Physical activity is one type of intervention that may be useful in this context.

Over decades, a number of studies have reported compelling evidence for the benefits of physical activity on sleep quality (see Kredlow, Capozzoli, Hearon, Calkins, & Otto, 2015 for review) and executive functions (Álvarez-Bueno et al., 2017). For example, Kredlow and colleagues (2015) examined the effects of acute and regular exercise on sleep. The results revealed that both acute and regular exercise could increase total sleep time, shorten sleep onset latency (SOL), reduce rapid eye movement, and promote slow wave sleep (Kredlow et al., 2015). Álvarez-Bueno and colleagues recently conducted a meta-analysis of 36 interventional studies examining the effect of physical activity on executive functions and metacognition. The design of these studies included randomized controlled trials (RCTs), non-RCTs, and controlled ore-post studies. The physical activity intervention aimed to increase the physical activity participation time or to increase the cognitive demands of physical activity tasks for healthy children aged 4 to 18 years. The outcomes included cognitive performance assessments of nonexecutive cognitive functions, core executive functions, and/or metacognition (Álvarez-Bueno et al., 2017). Overall, the findings suggested that physical activity benefits multiple facets of executive function (e.g. inhibition control and working memory capacity; Álvarez-Bueno et al., 2017). Based on these findings in children with TD, the use of physical activity as an alternative intervention to improve sleep quality and executive functions in children with ASD appears to be promising.

In the present study, we conducted an RCT to investigate the impact of physical activity intervention (12-week basketball skill learning) on sleep quality and two executive functions (inhibition and working memory) in children with ASD. Basketball skill learning intervention was chosen because it was a natural cognitive-engaging physical activity requiring learner's attention and working memory on motor coordination, which is shown to be beneficial to both physical and cognitive developments in children with TD (Diamond & Lee, 2011). We hypothesized that the intervention would positively impact both sleep quality and executive functions in children with ASD.

Method

Participants

In previous related studies (Anderson-Hanley, Tureck, & Schneiderman, 2011; Brand, Jossen, Holsboer-Trachsler, Pühse, & Gerber, 2015; Pan et al., 2017; Wachob & Lorenzi, 2015), effect sizes on sleep quality ranged from $d=0.13$ (Brand et al., 2015) to $d=1.69$ (Anderson-Hanley et al., 2011). The mean value of the effect size ($d=0.95$) was used to calculate the sample size required in the present study. With a 5% level of significance, a sample size of 36 (18 per group) was needed to achieve a power of 90% for interaction effect using G*Power 3.1.9.2 software (Faul, Erdfelder, Lang, & Buchner, 2007). The study was approved by the ethics committee of the appropriate university. The inclusion criteria were as follows: (1) aged 8–12 years; (2) ASD diagnosis from a physician based on the Diagnostic and Statistical Manual of Mental Disorders, 5th edition (*DSM*-5, American Psychiatric Association, 2016); (3) non-verbal IQ above 40 as assessed by the Wechsler Intelligence Scale for Children (Chinese revised; C-WISC; see Gong & Cai, 1993 for more information); (4) ability to follow instructions; (5) ability to perform the requested physical intervention and executive function measures; (6) no prior experience in formal basketball skill training (no formal knowledge about how to perform basketball shooting); and (7) no history of reading disabilities according to parents. The exclusion criteria were as follows: (1) having other medical conditions that limited their physical activity capacities (e.g. asthma, seizure, cardiac disease) and (2) having a complex neurologic disorder (e.g. epilepsy, phenylketonuria, fragile X syndrome,

Table 1. Demographic statistics of participants of each group.

	Intervention group ($n = 19$)	Control group ($n = 21$)	p
Gender	14 boys and 5 girls	18 boys and 3 girls	0.36
Age (years)	10.11 ± 1.20	9.81 ± 1.17	0.43
Weight (kg)	36.48 ± 4.02	35.14 ± 4.52	0.34
Height (m)	1.35 ± 0.08	1.37 ± 0.08	0.67
BMI (kg/m^2)	19.86 ± 2.71	18.76 ± 2.27	0.17
Non-verbal IQ	64.72 ± 19.00	67.51 ± 15.01	0.09
SRS-2 Row-scores	75.94 ± 10.92	77.19 ± 11.05	0.72
Medication (n)			
Yes	2	4	
No	17	17	

BMI: body mass index; SRS-2: Social Responsiveness Scale (Second Edition).

tuberous sclerosis). All the screenings were conducted by trained research assistants at different schools before the first class. After screening, a total of 50 participants (40 boys and 10 girls) from four local special schools were successfully recruited. Participants were randomly assigned to one of two groups: intervention ($n = 25$) and control ($n = 25$). To ensure equal allocation ratios for both groups, block randomization (Efird, 2011) was used. A block size of five was used in the study (i.e. five participants in one group and five in the other group for every 10 consecutively entered participants). The block randomization process was performed by a trained research assistant. Of the 50 participants, 10 were excluded (six from the intervention group and four from the control group) because of incomplete information in the go/no-go (GNG) task (see the Measures section), resulting in 19 participants in the intervention group (14 boys and five girls; mean age $= 10.11 \pm 1.20$) and 21 participants in the control group (18 boys and three girls; mean age $= 9.81 \pm 1.17$). We collected information about participants' autism symptoms by asking their parents to complete the Social Responsiveness Scale (Second Edition; SRS-2; Constantino & Gruber, 2012). We also collected information about current treatments (e.g. medications, speech therapy, occupational therapy) from the participants' parents. Six participants were reported to take regular medication treatment during the screening. The medications were hydrocortisone ($n = 2$), fusidic acid cream ($n = 3$), and risperidone ($n = 1$). The side effects of these medications were non-significant according to the parents; therefore, the participants were allowed to participate in the present study. Written consent was obtained from participants' parents/guardians. The study was approved by the university ethics committee. Demographic data for the two groups are shown in Table 1.

Intervention group. A 12-week basketball skill learning intervention consisting of 24 sessions (two sessions per week; 45 min per session) was implemented in a hall/gymnasium of each participating school in the morning. The staff-to-participant ratio for both groups ranged from 1:3 to 1:2 depending on the attendance. The overall attendance rate was 97.79%. Each intervention session was conducted in an identical format, comprising three activities: warm-up (10 min), basketball (30 min), and cool-down (5 min). In the basketball activity, participants were partnered with a staff member to learn different basketball skills (see Supplemental material for more information). To motivate participants, they were positively reinforced verbally with compliments for their efforts in learning and practicing the skill.

Control group. Participants in the control group received no basketball intervention and were asked to follow their normal daily routine without participating in any additional physical activity/exercise program between the two measurements (T1–T2).

Procedure

Each participant attended two 1-week assessments, in which their habitual sleeping patterns and executive functions (i.e. inhibition control and working memory capacities) were assessed. The two 1-week assessments were conducted before the intervention (T1: baseline) and immediately after the 12 weeks of physical activity intervention or regular treatment (T2: post-intervention). Presentations of the two cognitive assessments were counterbalanced across participants. The procedure of the present study is shown in Figure 1.

Measures

Sleep quality. Four sleep parameters including sleep efficiency (SE, actual sleep time divided by time in bed, expressed as a percentage), SOL (length of time to fall asleep, expressed in minutes), SD (expressed in hours), and wake after sleep onset (WASO, length of wake time after falling to sleep, expressed in minutes) were measured with an actigraphy accelerometer (Model no: ActiGraph

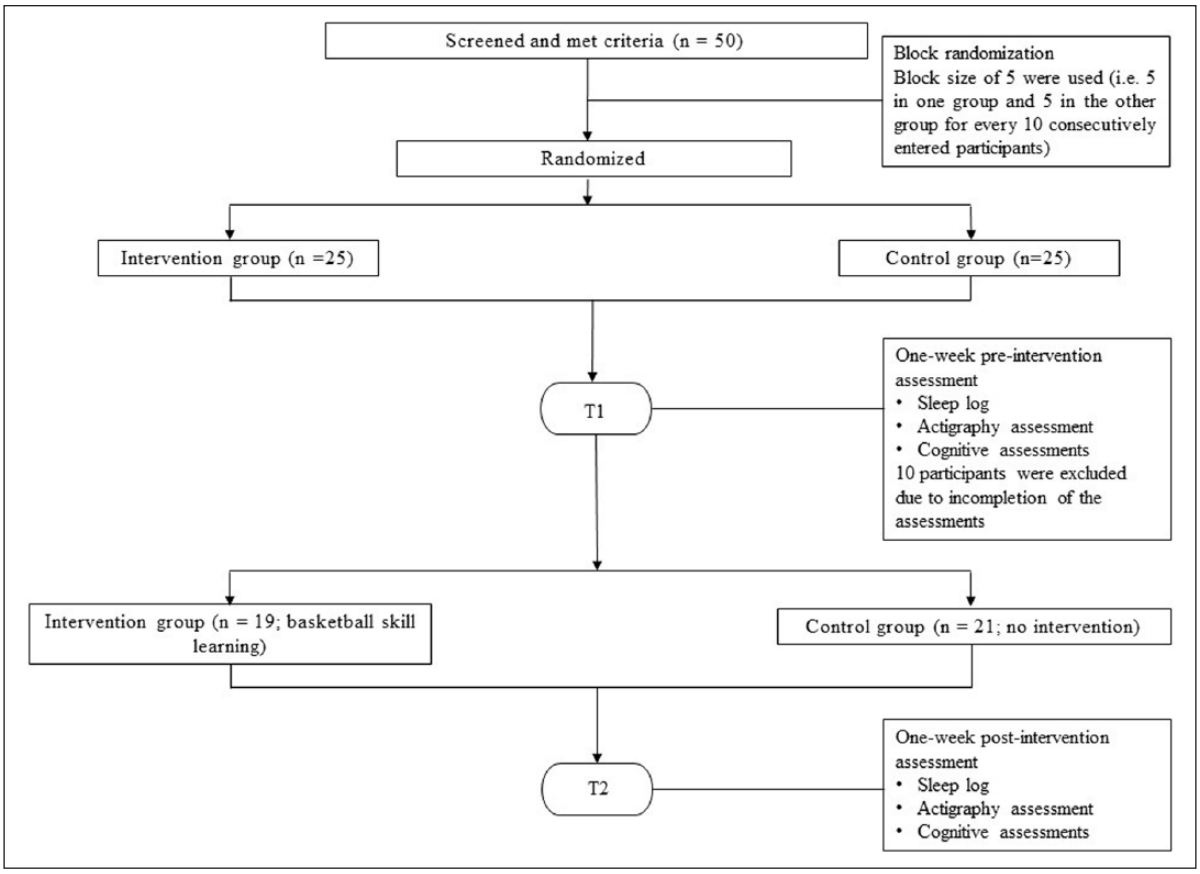

Figure 1. Flowchart.

GT3X; Wachob & Lorenzi, 2015) and a sleep log (Morgan, David, & Gasoigne, 2007). These parameters were chosen because they were previously found to be the most representative measures of sleep behaviors and sleep quality in children with ASD (Malow et al., 2006; Souders et al., 2017). For the actigraphy assessment, participants were asked to wear the device on the non-dominant wrist for seven consecutive days (Monday to Sunday) and were instructed to only take it off when they were taking baths. Non-wear time was defined as 60 min of consecutive 0 scores with a 2 min spike tolerance (Wachob & Lorenzi, 2015). The night (2200–0700) was considered invalid if the wear time was less than 8 h and was excluded from data analysis. The Sadeh algorithm (Sadeh, Sharkey, & Carskadon, 1994) was implemented to identify sleep onset and sleep offset. For the sleep log assessment, participants were assisted by their parents to recall specific sleep patterns (e.g. bedtime, sleep start, sleep end, wake-up time, and sleep length) and reported them in a sleep log for the whole assessment duration (T1 and T2).

Executive functions. Two executive functions, inhibition control and working memory, were examined. These two functions were selected because they are widely considered to constitute the core components of executive functions (Barendse et al., 2013; Verté, Geurts, Roeyers, Oosterlaan, & Sergeant, 2006). Importantly, impairments of these two executive functions are relatively common in children with ASD (Andersen, Hovik, Skogli, Egeland, & Øie, 2013; Xiao et al., 2012).

For inhibition control, participants' ability to inhibit unwanted responses to changing stimuli were measured using a computer-based version of the GNG task. The GNG task was identical to that used by Uzefovsky, Allison, Smith, and Baron-Cohen (2016). Participants were seated in front of a computer monitor with a keyboard. They were asked to press left or right arrow key as quickly as possible when the corresponding arrow appeared on the center of the screen (Go response), and to not press any key whenever the *up arrow* appeared on the screen (No-go response). Following 20 practice trials, participants completed 300 trials: 220 trials requiring a Go response (110 left and 110 right) and 80 trials (26.7%) requiring a No-go response (not pressing any key) (Uzefovsky et al., 2016). The stimuli were randomly presented, one at a time, for 500 ms followed by 1000 ms of blank interval using E-Prime 3.0 software (Psychology Software Tools, Inc, 2012). At an interval of 60 trials, children were offered a break of 2 min. No feedback was given upon response and the response time was recorded but not analyzed because of the

unreliability of the recording procedure (Uzefovsky et al., 2016). Moreover, participants were excluded if they failed to respond correctly to at least 50% of the Go trials (10 children were excluded, as described above). As in Uzefovsky et al.'s (2016) study, a Go response in a No-go trial was coded as a false alarm (FA). FA errors are considered an indicator of inhibition control (Wright, Lipszyc, Dupuis, Thayapararajah, & Schachar, 2014).

For working memory, participants were asked to perform the Corsi block tapping task (CBTT, Corsi, 1972), forward digit span test (FDS), and backward digit span test (BDS) (Thorndike, Hagen, & Sattler, 1986) respectively to measure the capacities of visual-spatial working memory and auditory working memory. In the CBTT, participants were required to observe the sequence of blocks being "tapped," then repeat the sequence back in order. The initial sequence length was three blocks, and after every four trials the sequence length increased by one. The task ended after four incorrectly repeated sequences of the same length, and the longest correctly repeated sequence length was recorded (de Vries, Prins, Schmand, & Geurts, 2015). For the digit span tests, digits were presented at a rate of one digit per second, and participants were required to repeat the sequence verbally. The initial sequence length was two digits, and the sequence length increased by one after every two trials. The test ended after two incorrectly repeated sequences of the same length and the maximum digit span was established. All the cognitive assessments were conducted in the morning before the school commencement by two research assistants who were blinded to the grouping.

Data analysis

All statistical analyses were conducted using SPSS (version 23.0) for Windows (SPSS Inc., Chicago, IL, USA). All data were entered into SPSS by a research assistant, who was blinded to the grouping. To assess the effects of the intervention on sleep, linear mixed model (LMM) was performed for each sleep parameter measured by sleep log and actigraph accelerometer. Actigraphy-assessing parameters were averaged across weekday nights (Monday to Friday) and weekend nights (Saturday and Sunday) for each participant. Spearman's correlations were used to determine the relationship between the actigraphy assessment and sleep log on two sleep parameters (SE and SD) to check the validity of the sleep log. SOL and WASO were not used for validity check because these two sleep parameters could not be measured simultaneously by actigraphy assessment and sleep log. Meanwhile, to assess the effects of the intervention on executive functions, a series of 2 (block: pre vs post) × 2 (group: intervention vs control) analyses of covariance (ANCOVAs) with repeated measures was performed for each executive function outcome. Considering the potential confounding effects of developmental factors, age was controlled as a covariate for the raw scores of the executive function outcomes (i.e. CBTT score, FDS and BDS test scores, and GNG task accuracy) to compare the changes between and within groups over different time points. Finally, Spearman's correlations were used to determine the relationship between changes in sleep parameters and changes in executive functions. A p value < 0.05 was considered statistically significant. Results are presented as mean (standard deviation). Preliminary tests of the assumptions of the statistical tests, including data normality using Shapiro–Wilk tests (all p values > 0.05), homogeneity of variance (Levene's tests: all p values > 0.05), and homogeneity of regression slopes (all p values > 0.05) for the ANCOVAs were met.

Results

Sleep parameters

At T1, all the sleep parameters measured by actigraphy assessment and sleep logs were comparable between groups during weekday and weekend (see Tables 2 and 3).

Actigraphy assessments. As shown in Table 2, the LMM showed that there was significant group by time interaction effects for both SE and WASO (all ps < 0.001) during weekday and weekend. The intervention group showed significant improvements for both sleep parameters (all ps < 0.001) with moderate effect sizes (d ranged from 0.66 to 0.77) from T1 to T2. In contrast, the control group showed significant drop for SE and increase in WASO (all ps < 0.001) with moderate effect sizes (d ranged from 0.59 to 0.68) from T1 to T2. Meanwhile, the LMM indicated that there was a significant interaction effect for SD ($p < 0.01$) during weekday but not during weekend ($p > 0.05$). Both intervention group and control group showed significant improvements for SD (all ps < 0.001) with small effect sizes (d ranged 0.16 to 0.29) from T1 to T2 during weekday. However, only the intervention group showed a significant improvement for SD ($p < 0.01$) with small effect size ($d = 0.003$) from T1 to T2 while there was not significant change in control group ($p > 0.05$).

Sleep log. As shown in Table 3, the LMM showed that there was significant group by time interaction effects for both SE and SOL (all ps < 0.001) during weekday and weekend. The intervention group showed significant improvements for both sleep parameters (all ps < 0.001) with moderate effect sizes (d ranged from 0.61 to 0.62) from T1 to T2. In contrast, the control group showed no significant change for both parameters (all $p > 0.05$) with small effect sizes (d ranged from 0.002 to 0.04) from T1 to T2. Meanwhile, the LMM indicated no significant interaction effects for SD (ps > 0.05) during weekday and weekend. Both intervention group and control group showed significant improvements for SD (all ps < 0.05) with small to moderate effect sizes (d ranged 0.11 to 0.38) from T1 to

Table 2. Sleep parameters during weekday and weekend sleeps measured by actigraphy assessment.

Sleep parameters	Intervention group (*SE*)	Control group (*SE*)	*p* value (group effect)	*p* value (interaction effect)
Weekday				
Sleep efficiency (%)				<0.001
T1	87.17 (1.69)	90.73 (1.67)	0.14	
T2	96.33 (0.71)	83.90 (2.38)	<0.001	
p value (time effect)	<0.001	<0.001		
Cohen's *d* effect size (95% CI)	0.77 (0.46, 1.08)	0.59 (0.28, 0.90)		
Wake after sleep onset (min)				<0.001
T1	57.97 (8.60)	43.22 (8.19)	0.21	
T2	17.00 (3.52)	81.62 (13.54)	<0.001	
p value (time effect)	<0.001	<0.001		
Cohen's *d* effect size (95% CI)	0.66 (0.35, 0.97)	0.68 (0.37, 0.99)		
Sleep duration (hour)				0.008
T1	6.79 (0.78)	6.82 (0.22)	0.94	
T2	7.21 (0.16)	6.54 (0.19)	0.008	
p value (time effect)	0.04	0.03		
Cohen's *d* effect size (95% CI)	0.29 (–0.02, 0.60)	0.16 (–0.15, 0.48)		
Weekend				
Sleep efficiency (%)				<0.001
T1	80.90 (1.93)	86.09 (2.27)	0.05	
T2	94.46 (0.95)	87.20 (1.80)	<0.001	
p value (time effect)	0.002	0.64		
Cohen's *d* effect size (95% CI)	0.16 (–0.16, 0.47)	0.07 (–0.23, 0.38)		
Wake after sleep onset (min)				0.002
T1	99.84 (12.51)	99.41 (5.41)	0.06	
T2	64.23 (13.48)	63.95 (8.17)	0.001	
p value (time effect)	<0.001	0.99		
Cohen's *d* effect size (95% CI)	1.12 (0.81, 1.43)	0.84 (0.53, 1.15)		
Sleep duration (hour)				0.18
T1	7.38 (0.27)	7.71 (0.33)	0.43	
T2	8.59 (0.25)	7.48 (0.28)	0.03	
p value (time effect)	0.002	0.63		
Cohen's *d* effect size (95% CI)	0.003 (–0.31, 0.31)	0.008 (–0.30, 0.31)		

CI: confidence interval.

T2 during weekday. No significant change of SD was shown in the intervention group ($p > 0.05$) from T1 to T2 during weekend. The control group, interestingly, showed a significant decrease in SD ($p < 0.01$) with small effect size ($d = 0.005$) from T1 to T2 during weekend. Comparisons of sleep parameters between groups and within groups at different timeslots by different measurements are shown in Tables 2 and 3.

Inhibition control and working memory capacity

At T1, inhibition control and working memory capacities were comparable between groups, as reflected by the GNG computer task and the three working memory tasks (i.e. the CBTT, FDS test, and BDS tests; see Table 4). Repeated-measures ANCOVAs were performed separately for each neuropsychological test to examine the effects of the physical activity intervention on executive functions after controlling for age. After adjusting for the effect of age, we found a significant interaction effect ($F(1, 37) = 6.58$, $p = 0.02$) and a significant group effect ($F(1, 37) = 4.15$, $p = 0.04$) in FA error. However, no significant interaction effects were revealed in CBTT score ($F(1, 37) = 1.00$, $p = 0.32$), FDS score ($F(1, 37) = 2.21$, $p = 0.15$), or BDS score ($F(1, 37) = 0.77$, $p = 0.39$). Subsequent tests revealed that the FA errors of the intervention group ($M = 10.89$, $SD = 5.17$) were significantly smaller than those of the control group ($M = 18.38$, $SD = 8.15$) at T2 ($t(38) = -3.43$, $p = 0.001$), and there was a significant reduction of FA errors between T1 and T2 in the intervention group ($t(18) = 2.55$, $p = 0.02$) but not in the control group ($t(20) = -0.37$, $p = 0.72$). Comparisons of neuropsychological measures between groups and within groups at different timeslots are shown in Table 4.

Table 3. Sleep parameters of weekday and weekend sleep patterns measured by sleep log.

Sleep parameters	Intervention group (*SE*)	Control group (*SE*)	*p* value (group effect)	*p* value (interaction effect)
Weekday				
Sleep efficiency (%)				0.001
T1	93.34 (0.60)	92.78 (0.54)	0.48	
T2	95.77 (0.42)	92.79 (0.54)	<0.001	
p value (time effect)	<0.001	0.97		
Cohen's *d* effect size (95% CI)	0.61 (0.30, 0.92)	0.002 (–0.3, 0.31)		
Sleep onset latency (min)				0.001
T1	33.42 (3.14)	36.24 (2.61)	0.50	
T2	21.57 (2.23)	37.19 (2.3)	<0.001	
p value (time effect)	<0.001	0.57		
Cohen's *d* effect size (95% CI)	0.62 (0.31, 0.93)	0.04 (–0.27, 0.35)		
Sleep duration (hour)				0.30
T1	7.78 (0.19)	7.81 (0.18)	0.93	
T2	8.19 (0.22)	8.00 (0.18)	0.54	
p value (time effect)	0.02	0.04		
Cohen's *d* effect size (95% CI)	0.38 (0.07, 0.69)	0.11 (–0.20,0.42)		
Weekend				
Sleep efficiency (%)				0.001
T1	94.63 (0.56)	94.00 (0.43)	0.37	
T2	96.51 (0.37)	92.91 (0.46)	<0.001	
p value (time effect)	<0.001	0.003		
Cohen's *d* effect size (95% CI)	0.53 (0.22, 0.84)	0.30 (–0.01, 0.61)		
Sleep onset latency (min)				0.001
T1	33.63 (3.40)	35.62 (2.91)	0.50	
T2	20.55 (2.34)	38.82 (2.27)	<0.001	
p value (time effect)	0.001	0.1		
Cohen's *d* effect size (95% CI)	0.55 (0.24, 0.86)	0.14 (–0.17, 0.45)		
Sleep duration (hour)				0.18
T1	9.67 (0.20)	9.46 (0.24)	0.17	
T2	9.27 (0.22)	8.06 (0.22)	0.008	
p value (time effect)	0.39	0.004		
Cohen's *d* effect size (95% CI)	0.006 (–0.30, 0.32)	0.005 (–0.31, 0.31)		

CI: confidence interval.

Correlations between actigraphy assessment and sleep log

To check the validity of the sleep log, the average values of the sleep parameters (i.e. SE and SD) across the whole week were taken. At T1, the correlation between the two assessments on SE and SD were 0.18 ($p=0.04$) and 0.33 ($p=0.03$), respectively. At T2, the correlations between the two assessments on SE and SD were 0.27 ($p=0.03$) and 0.22 ($p=0.04$), respectively.

Correlations between changes in sleep parameters and changes in executive functions

Changes in SE during weekend as measured by both assessments (i.e. actigraph and sleep log) were significantly correlated with changes in FA error. Moderate correlations were revealed between the changes in these two parameters (i.e. SE and FA error) during weekday by actigraphy assessment ($r=–0.43$, $p=0.006$) and sleep log ($r=–0.34$, $p=0.03$). Moreover, moderate correlations were found between changes in SOL and FA error during weekday ($r=–0.33$, $p=0.04$) and during weekend ($r=–0.47$, $p=0.002$). No significant correlation was found among changes in other sleep parameters and changes in other executive functions during weekday and weekend (all $ps>0.05$).

Discussion

The present study examined the impact of physical activity intervention (a 12-week basketball skill learning) on sleep quality and executive functions in children with ASD. We hypothesized that the intervention would benefit both sleep quality and executive functions among the children. In accordance with this hypothesis, the intervention was

Table 4. Comparisons of neuropsychological measures.

Neuropsychological assessment	Intervention group (*SD*)	Control group (*SD*)	*p* value (group effect)	*p* value (interaction effect)
Inhibition control (false alarm (FA) error)				0.02
T1	16.16 (8.31)	18.05 (7.80)	0.46	
T2	10.89 (5.17)	18.38 (8.15)	<0.001	
p value (time effect)	0.02	0.72		
Corsi block tapping task (CBTT)				0.80
T1	4.32 (1.38)	3.86 (1.15)	0.26	
T2	3.95 (1.27)	3.57 (0.93)	0.29	
p value (time effect)	0.09	0.25		
Forward digit span (FDS)				0.54
T1	6.05 (1.47)	5.33 (1.32)	0.11	
T2	5.58 (1.30)	5.14 (1.39)	0.31	
p value (time effect)	0.17	0.56		
Backward digit span (BDS)				0.57
T1	4.32 (1.25)	3.67 (0.86)	0.06	
T2	3.79 (0.98)	3.95 (0.92)	0.59	
p value (time effect)	0.06	0.38		

found to be effective for improving SE, shortening SOL, increasing SD, reducing WASO, and enhancing inhibition control. However, it did not have any effect on working memory, and participants in the intervention and control groups did not differ in their performance on working memory assessments.

The sleep-related findings of our study are consistent with a recent small-scale study conducted by Brand and colleagues (2015), in which a physical activity intervention (30 min sessions of bicycle workout plus 30 min balance and coordination training) was shown to be effective for increasing SE and shortening SOL in 10 children with ASD (Brand et al., 2015). In the present study, participants in the intervention group exhibited greater SE, shorter SOL, longer SD, and reduced WASO than those in the control group during both weekdays and weekend (see Tables 2 and 3 for more information). One possible explanation for these findings is related to the ability of physical activity to alter melatonin levels (Lee, Kim, & Kim, 2014). Melatonin is a natural hormone serving as a key regulator of the circadian rhythm and promotes sleep onset and sleep maintenance (Wirojanan et al., 2009). Secretion of melatonin normally increases shortly after dark, peaks in the middle of the night, and falls slowly during early morning hours (Rossignol & Frye, 2011). This hormonal response allows for maintaining a normal circadian rhythm and sleeping through the night. Compared with children with TD, melatonin levels were found to be lower in some children with ASD (Krakowiak, Goodlin-Jones, Hertz-Picciotto, Croen, & Hansen, 2008; Liu et al., 2006). To counter this melatonin deficit, supplemental melatonin is commonly used to treat insomnia in children with ASD (Arendt, 2003). Previously, researchers suggested that melatonin levels could also be moderated by physical activity (Marrin et al., 2011). In one experiment, Marrin et al. (2011) asked seven healthy participants to complete a moderately intense cycling exercise in the morning, measuring their salivary melatonin concentration at baseline, during exercise, after exercise, and upon exercise recovery. The results revealed that participants' melatonin levels were significantly increased during and after exercise compared with those at baseline and recovery. In the current study, the intervention might have increased participants' melatonin levels, resulting in better sleep quality among them. Apart from melatonin-mediated factor, other factors such as reduced anxiety (DiLorenzo et al., 1999), improved mood (Hartescu, Morgan, & Stevinson, 2015) and increased brain-derived neurotrophic factor level (Szuhany, Bugatti, & Otto, 2015) may also mediate the mechanism of exercise–sleep relation. As a result, future studies examining the underlying mechanism of how physical activity affects sleep quality in children with ASD are therefore warranted. Meanwhile, it should be noted that we found moderate correlations for most of the sleep parameters measured by actigraphy assessment and sleep logs, suggesting that both assessment methods could be used interchangeably to measure the habitual sleep patterns of children with ASD.

For cognitive measurements, the improvement in inhibition control shown by the intervention group further strengthens the existing evidence for the cognitive benefits of physical activity reported in previous studies (e.g. Anderson-Hanley et al., 2011; Pan et al., 2017). For example, Pan and colleagues (2017) recently used a 12-week table tennis intervention combined with executive function training for 22 children with ASD to examine the impact of such an intervention on motor skill proficiency and executive functions. The results revealed

significant improvements on both motor skill proficiency and executive functions in the participants after the intervention (Pan et al., 2017). A possible explanation for the cognitive benefit is related to the neurotrophic hypothesis (Khan & Hillman, 2014). According to this hypothesis, physical activity increases metabolic demands and triggers a cascade of biochemical changes, such as enhancing cerebral blood flow and increasing the availability of brain-derived neurotrophic factor (Khan & Hillman, 2014), which in turn strengthens brain plasticity for higher level cognitive activities such as those involved in executive functions (Gomez-Pinilla & Hillman, 2013). However, this hypothesis cannot explain the present findings regarding working memory, which was unaffected by the physical activity intervention. Therefore, rather than focusing purely on physiological factors, a psychological mediating mechanism should also be considered. In this context, a self-regulation theory has been proposed (Audiffren & André, 2015).

Self-regulation or self-control refers to any effort by a human to alter one's own affective or behavioral responses according to environmental demands (Audiffren & André, 2015; McEwan, Ginis, & Bray, 2013). Under this theoretical framework, physical activity can enhance self-regulation if it is mentally effortful and attention-demanding (Lakes & Hoyt, 2004; Newell, 1991; Pesce et al., 2016; Zimmerman & Kitsantas, 1997). As such, the process of motor skill acquisition is thought to enhance self-regulation. According to the three stages of the learning model proposed by Fitts and Posner (1967), learning any new motor skill typically progresses through cognitive, associative, and autonomous stages (Fitts and Posner, 1967). The cognitive stage is characterized by the peak demand on cognitive resources, where high levels of consciousness and inhibition control are required to produce a specific sequence of actions to achieve the desired goal (see Anderson et al., 2013, for a review). In the present study, the basketball skill learning intervention might have enhanced participants' self-regulation by requiring them to be highly attentive to regulating their body movements during exercise. Throughout this process, participants' inhibition control was highly engaged as they had to inhibit any impulses to motor movements other than the movements they were asked to perform. As a result, the inhibition control ability of the participants in the intervention group may have been enhanced. In contrast, participants' working memory may not have been strongly engaged because of the simplicity of the instructions and the physical guidance from the staff (for practical reasons), resulting in a lack of improvement in participants' working memory capacity. Since the present study did not incorporate any of the measurements of self-regulation or self-control applied in other studies (Lakes, 2013; Lakes & Hoyt, 2004), we are unable to verify the explanation of our cognition-related findings by self-regulatory mechanisms. Future studies should incorporate such measurements to further elucidate this issue.

The sleep-cognition relation was further analyzed with specific correlations between changes in sleep parameters and changes in executive functions from T1 to T2. Our data revealed significant correlations of the changes in two sleep parameters: SE and SOL with the changes in inhibition control, whereas no significant correlation was found between changes in any sleep parameters and changes in any working memory measurements. This may suggest that the improvement of SE and sleep onset may play a role on improving the inhibition control. However, due to the limitation of our data (that we only have measured at two time points), the causal pathway between exercise, sleep, and executive function could not be examined. Future studies should incorporate the third time point (e.g. mid-progress) to further examine this causal relationship.

Despite the strengths of this study and its findings, there is an important limitation that requires attention The researchers did not examine the baseline physical activity level (e.g. number of hours of daily physical exercise within a 1-week period) and the basketball skill level from each participant prior to randomization and/or as exclusion/inclusion criteria to ensure that children in both groups maintained similar physical activity and basketball skill level per day. Without this control, it is difficult for the researchers to determine treatment effects. For example, children in the treatment condition may naturally engage in physical activity at a higher daily frequency as compared with children in the waitlist condition prior to the basketball intervention, or vice versa. Future study should include such assessment in baseline to address this question. Finally, this RCT protocol was not published in advance, future similar study should consider publishing the protocol first in order to strengthen the validity and reliability of the study.

Conclusion

The positive influences of physical activity on sleep quality and inhibition control revealed in our study are in accordance with the accumulating evidence of the cognitive benefits of physical activity in children with ASD. Practically, the current findings shed light on ways to improve sleep and cognitive outcomes among children. Physicians and educators may consider prescribing physical activity to children with ASD to alleviate their symptoms of sleep disturbance and executive dysfunction. Theoretically, our findings are important for guiding future researchers to further examine the exercise–sleep and exercise–cognition relationships in children with ASD through melatonin-mediated mechanisms and self-regulatory mechanisms.

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Supplemental material

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