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Differences in sleep patterns, sleepiness, and physical activity levels between young adults with autism spectrum disorder and typically developing controls

Sarah Benson[a], Amy M Bender[b], Hayley Wickenheiser[a], Alexandra Naylor[a], Margaret Clarke[c], Charles H Samuels[d], and Penny Werthner[a]

[a]Faculty of Kinesiology, University of Calgary; [b]Centre for Sleep, Faculty of Kinesiology, University of Calgary; [c]Faculty of Medicine, University of Calgary; [d]Centre for Sleep & Human Performance, Faculty of Medicine, University of Calgary

ABSTRACT

Objective: To investigate the differences in sleep, sleepiness, and physical activity (PA) between young adults with autism spectrum disorder (ASD) and typically developing controls (TDC).
Method: Actigraphic data and questionnaires on sleep, sleepiness, and PA were compared between fifteen adults with ASD (ADOS range 7–19; ages 22.8 ± 4.5 years) and TDC.
Results: In comparison to the TDC group, the ASD group slept longer on average per night but took longer to fall asleep. In relationship to PA levels, the objective PA levels were lower in the ASD group than the TDC group. Fewer wake minutes during the sleep period in the ASD sample were associated with more PA the following day.
Conclusion: The findings support previous research that demonstrates differences in sleep parameters and PA between ASD and TDC. Interventions aimed at increasing PA in an ASD population may be beneficial for improved sleep.

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Actigraphy; autism spectrum disorder; insomnia; physical activity; Pittsburgh sleep quality index; sleep onset latency

Introduction

Autism spectrum disorder (ASD) affects just over 1% of the population[1] and are a group of multisystem neurodevelopmental disorders characterized by deficits in social communication and interactions with repetitive and stereotypical behaviours and interests[2,3]. Sleep complaints are common in this population[4–10], with 50–80% of children with ASD having sleep problems[4]. The limited existing research suggests sleep problems persist into adolescence and adulthood[11–16], and exacerbate the core stereotypical symptoms of this disorder[17–19], thus further impairing daytime functioning and increasing stress on caregivers for individuals on the spectrum[15,17–21]. Greater levels of daytime sleepiness[10] and fatigue[11] have been reported in the ASD population and may play a role in this exacerbation of core stereotypical symptoms. Further, young adults with high functioning ASD have reported higher scores on sleep disturbances and daytime dysfunction due to sleepiness subscales on the Pittsburgh Sleep Quality Index (PSQI).[22]

Subjective reports of sleep in ASD are typically gathered through parent report. This is problematic as the parent has likely accommodated to their child's disturbed sleep over time[14]. Therefore, using objective measures to support subjective data are beneficial. Studies using objective measurements of sleep generally show poorer sleep parameters in those with ASD compared to typically developing peers. A study using wrist-watch actigraphy in young adults with ASD showed less than 85% of the time in bed (TIB) was spent sleeping (sleep efficiency; SE%) and it took most of the subjects longer than 30 min to fall asleep (sleep onset latency; SOL) which are both features of poor sleep quality[14,23]. In polysomnography (PSG) studies, similar results were found[15,16] including longer SOL, lower SE%, and higher amounts of wake after sleep onset (WASO) for high-functioning young adults with ASD who had no sleep complaints or psychiatric disorders[16].

However, comorbid psychiatric diagnoses are prevalent in the ASD population and have been associated with worsened sleep. When sleep complaints persist despite no significant objective sleep difference, psychiatric disorders have been postulated to be a causative factor in these studies[12,24]. Therefore, comorbid intellectual disability (ID) should be considered when distinguishing between autism specific effects on sleep versus effect of the ID[25]. ID in ASD negatively impacts SOL, SE % and WASO[26,27] and causes greater severity[28] of sleep disorders[27]. Furthermore, individuals with ASD diagnosed and medicated for psychiatric comorbidities (e.g. anxiety/depression), have reduced bedtime sleepiness and increased TST compared to non-medicated ASD controls[22,29] making medication history an important aspect of research in this area.

Along with sleep issues in this population, adolescents and adults with ASD have significantly lower physical activity (PA) levels than their typically developing peers[30,31]. Individuals with ASD tend to have fewer opportunities for participation in PA activities leading to a more sedentary lifestyle than their typically developing peers. Fewer opportunities are due to impairments in motor, social communication, sensory, and behavioural domains which make it more difficult to engage in PA and sport programming with their peers[2,32–34].

It is important to explore the relationship between PA and sleep, especially in because targeting a PA intervention on improving sleep outcomes may consequentially have a positive

CONTACT Sarah Benson benson190@gmail.com University of Calgary, Faculty of Kinesiology

effect on daytime behaviour and quality of life in ASD and their caregivers. PA has been associated with improving sleep more than any other daytime activity and is recommended by the National Sleep Foundation as a non-pharmaceutical modality to improve sleep[35,36]. In epidemiological studies, self-reported PA has consistently been associated with improved sleep quality and reduced daytime sleepiness in the general population[37–40]. In youth with ASD, more physically active children had overall higher sleep quality as measured by actigraphy[41], improved SE%, SOL and WASO as measured by PSG[42], and improved subjective sleep and reduced negative impact on daily functioning[43]. These studies indicate PA may be beneficial on sleep outcomes and daytime functioning in the child and adolescent (7–16 years old) ASD population[41–44], however, not in the young adult ASD population.

Therefore, the present study objectively measured the relationship between daily minutes of PA and sleep in the ASD population and purposely recruited young adults with ASD, a population which has not been previously studied. Therefore, the primary purpose of the present study was to explore objective sleep, PA levels, and the relationship between PA-sleep/daytime sleepiness in young adults between the ages of 18 and 35 with ASD compared to TDC. A secondary analysis was employed to explore daytime sleepiness levels and weekend/weekday sleep parameter differences in young adults with ASD compared to TDC. The present study objectively measures PA and sleep over a consistent period of time and in an older population than has been previously studied.

Method

Participants

Fifteen young adults with a diagnosis of ASD (age, mean± SD (range) = 22.8 ± 4.5 (18–35) years; 4 females) were recruited through the Child Development Centre in Calgary, Canada. The ASD sample completed appointments with a medical doctor and a psychologist prior to the start of the study. The medical doctor took medication history, screened for and excluded individuals with the diagnosis of disorders that may influence brain function in addition to the ASD diagnosis (including Fragile X syndrome, tuberous sclerosis, neurofibromatosis, phenylkenonuria, epilepsy, major depression and gross brain injury), and obtained informed consent from the parent on behalf of the participant. Thirteen of the young adults in the ASD sample (86.7%) were on medication with 66.0% of participants taking medication with a known effect on sleep (Table 1).

All ASD participants had received an ASD diagnosis prior to entering the present study. However, to further characterize the ASD profiles represented in the sample, a registered psychologist performed a psychiatric assessment on 13 of 15 participants based on the Autism Diagnostic Observation Schedule (ADOS) Module 4. Two participants were unable to complete the ADOS due to scheduling issues; however, the medical doctor involved in the study obtained current ASD diagnoses from the parents at the time of the medical appointment. The ADOS assesses social interaction, communication and imagination during a semi-structured interview with the examiner and has shown strong predictive validity against best estimate diagnoses[45,46]. The average (± SD) ADOS score was 13.8 ± 4.2 (median = 15, range: 7–19) (Table 2). All 13 participants assessed were classified as either "autism" ($n = 10$) or "autism spectrum" ($n = 3$)[47]. In total, 7 of 15 (47%) reported part-time employment or volunteer activities. In total, 10 of the 15 individuals required parent report to complete the study questionnaires due to a lower level of intellectual functioning.

The TDC sample included 17 participants (age = 23.7 ± 4.3 years; 5 females) (Table 2). The TDC sample was recruited through posters on the University of Calgary campus. The criteria for TDC participants was: (1) between the age of 18 and 35 years old, and (2) no previous clinically

Table 1. Medication prescribed, use and affect on sleep in ASD participants.

Medication Classification	Typical Use	Affects Sleep?	# Participants using
Tricyclic antidepressant[a]	Anxiety/ Depression	Yes – Drowsiness/ Insomnia	2
CNS Stimulant[b]	Hyperactivity	Yes – Insomnia	2
Antipsychotic[c]	Irritability	Yes – Drowiness/ Insomnia	5
Noradrenergic and specific serotonergic antidepressant[d]	Depression	Yes – Sleepiness	1
Anticonvulsant[e]	Panic attacks	Yes – Drowiness/ Insomnia	1
Hormone[f]	Hypothyroidism	No	1
Anticholinergic[g]	Involuntary movement	No	1
Antidiuretic[h]	Urination	No	1
Antifungal	Athletes foot	No	1
Antibiotic[i]	Bladder infection	No	1
Antibiotic[j]	Acne	No	1

[a]Clompramine. [b]Lisdexamfetamine. [c]Resperidone, Aripiprazole. [d]Mirtazapine. [e]Clonazepam. [f]Levothryoxine sodium. [g]Benztropine. [h]Desmopressin. [i]Nitrofurantoin. [j]Doxycycline.

Table 2. Participant characteristics and questionnaire results.

	ASD	TD	
	Mean ± SD	Mean ± SD	p/x^2
Number of Participants	15	17	
Age (y) (range: 18–35 y)	22.8 ± 4.5	23.7 ± 4.3	0.620
Number of Females (%)	4(26.7)	5(29.4)	
BMI, mean ± SD	24.1 ± 4.6	23.7 ± 3.9	0.760
ADOS, mean ± SD	13.8 ± 4.2	N/A	
PSQI (range 0–21), mean ± SD	5.4 ± 3.8	5.3 ± 2.4	0.930
Poor sleep (PSQI> 5), *N*(%)	6(40)	7(41.2)	>0.999*
GSLTPAQ, mean ± SD	33.0 ± 22.9	46.6 ± 24.6	0.120
MVPA mins, mean ± SD	15.5 ± 6.1	36.8 ± 5.7	0.008
ACSM Categorization, *N*(%)			
Active	6(40.0)	14(82.4)	0.017*
Insufficiently active	9(60.0)	3(17.6)	
Insomnia risk, *N*(%)			>0.999*
Yes	3(20.0)	4(23.5)	
No	12(80.0)	13(76.5)	
Insomnia risk (SOL <30mins)			0.011*
Yes	8(53.0)	2(11.8))	
No	7(47.0)	15(88.2)	
Sleep Apnea risk, *N*(%)			>0.999*
Yes	3(20.0)	3(17.6)	
No	12(80.0)	14(82.4)	

SD: Standard Deviation; N: number of participants; ADOS: Autism Diagnostic Observation Schedule; GSLTPAQ: Godin Shephard Leisure Time Physical Activity Questionnaire; PSQI: Pittsburg Sleep Quality Index; ACSM: American College of Sports Medicine. *Fisher's Exact Test p-value reported.

diagnosed sleep disorder. Only one TDC participant (5.8%) was on medication that affected sleep (i.e. Zopiclone was used by this participant on a single occasion during the collection period). The Conjoint Health Research Ethics Board of the University of Calgary approved the study, and the entire study was performed in accordance to the *Declaration of Helsinki*.

Materials

Body Composition and PA Questionnaire

To categorize subjective PA levels, all participants completed the Godin-Shephard Leisure-Time Physical Activity Questionnaire (GSLTPAQ)[48]. Further, body habitus measures were collected to calculate body mass index (BMI). The GSLTPAQ one-item questionnaire evaluated weekly frequency and intensity of leisure-time PA of "at least 15 min" to estimate their caloric expenditure per week[48,49]. Categorization as "active" based on the results of the GSLTPAQ corresponds to achieving the American College of Sport Medicine (ACSM) PA guidelines of >150 min/week of moderate-vigorous PA (MVPA).[50]

Sleep Questionnaires: Perceived Sleep Quality, Insomnia and Sleep Apnea Risk

All participants completed three sleep questionnaires at the start of the study: the PSQI[51], a modified insomnia screening questionnaire, and the STOP-Bang sleep apnea risk assessment questionnaire[52].

The PSQI assesses perceived sleep quality and disturbances[51]. This 19-item measure makes up seven component scores and when summed gives the PSQI global score (0–21). Higher scores indicate greater sleep disturbance with a score greater than five associated with clinically significant sleep disturbance[51,53]. The seven component scores correspond to: subjective sleep quality, sleep latency, sleep duration, sleep efficiency, sleep disturbance, use of sleep medication and daytime dysfunction.

To identify possible cases of insomnia, the participants completed an insomnia screening questionnaire that was previously developed and validated[54,55]. Consistent with the definition of insomnia according to the International Classification of Sleep Disorders (ICSD-2), this questionnaire identifies insomnia as a combination of a person's perception of sleep difficulty with an assessment of associated impairment of daytime functioning[3,56].

The STOP-Bang, a simple, self-report screening tool, was used to predict risk for obstructive sleep apnea (OSA). The questionnaire includes four subjective measures (STOP: **S**noring, **T**iredness, **O**bserved apnea and high blood **P**ressure) and four demographic items (Bang: **B**MI, **a**ge, **n**eck circumference, **g**ender) and a STOP-Bang score ≥3 predicts high risk for OSA.[52]

Subjective Daytime Sleepiness

The Karolinska Sleepiness Scale (KSS) was employed in this study to evaluate subjective sleepiness and is associated with electroencephalographic and behavioural sleepiness.[57] The KSS is a 9-point Likert scale with 1 = "very alert", 3 = "alert", 5 = "neither alert nor sleepy", 7 = "sleepy (but not fighting sleep)", 9 = "very sleepy (fighting sleep)". Corresponding to the week of actigraphy usage, the participants completed the KSS on the Monday, Wednesday, and Friday at three time points each day: (1) morning, (2) at midday, between 12pm and 2pm, and (3) bedtime.

Actigraphy – Objective Sleep and Physical Activity Measurement

Sleep parameters and PA levels were objectively measured with the *Motionlogger*^{TM} actigraph from Ambulatory Monitoring Inc. (AMI; Ardsley, NY, USA) and supplemented with a sleep diary, to help distinguish between sleep and sedentary periods. The participants wore *Motionlogger*^{TM}, a triaxial piezoelectric accelerometer, on their non-dominant wrist 24-hours a day for 14 consecutive days. Data were digitized in 60-sec epochs and collected using 2 "modes": zero crossing mode (ZCM) for analysis of sleep parameters and proportional integrative mode (PIM) for analysis of PA[58]. The Sadeh algorithm was used for analysis of the sleep parameters[59–61] and University of California, San Diego (UCSD) algorithm was used for PA levels[62]. Analysis was completed using ActionW2 software (AMI)[63].

Given sleep can vary between the weekday and weekend, sleep parameters were averaged to obtain weekday (Mon-Thurs) and weekend (Fri-Sat) scores. The following sleep parameters were evaluated: bedtime (BT), wake time (WT), TIB, TST SOL, WASO, and SE%. TIB was measured as time from lights out, determined by the "lights" marker on the actigraph and the diary, to wake time. SOL was measured as the number of minutes from intended sleep onset (e.g. lights out) to persistent sleep, as determined by the Sadeh algorithm threshold. WASO was measured as the total number of minutes that the individual was awake during the night, after SOL was excluded. TST was calculated from sleep onset to morning wake time, minus WASO. SE% refers to the percentage of time in bed spent asleep (TST/TIB × 100).

PA levels were averaged to obtain overall weekday (Mon-Fri) and weekend (Sat-Sun) scores. Given no studies to date have determined a cut-off point for MVPA with the *Motionlogger*^{TM}, a cut off point of >22,000 cpm was used to distinguish MVPA in this paper. This value was determined in reference to cut-off points gathered through indirect calorimetry and a graded treadmill protocol for a hip-mounted actigraph of similar specifications to the *Motionlogger*^{TM}.[64] Proportions for each level of PA over the range of actigraph scores recorded in the Sasaki et al.[64] study was then applied to our sample to determine the MVPA cut off point. Additionally, a study by Kamada et al.[65] which validated PA levels determined by hip and wrist mounted actigraphs helped us determine MVPA cut-off points. Those who achieved >150 mins of MVPA per week were categorized as meeting the ACSM's PA guidelines[66].

Data from the actigraphs were downloaded to a personal computer where all sleep intervals were manually placed on the actogram based on the sleep diary data and validated by the light measure on the actigraph. Out of the 14 days of data collection, the most continuous seven days of wear were selected as the period for analysis. "Off wrist" periods, identified by the absence of biovibrations and movement, were removed, as inclusion would have overestimated sedentary behaviour. Two participants had actigraphy data that could not be scored on one of the seven nights and an average was calculated based on the days available.

Data Analyses

To compare the ASD and TDC samples, descriptive statistics, *t*-tests, chi-squared tests, and mixed-effects analysis of variance (ANOVA) were used. Independent sample *t*-tests were used to compare group characteristics for variables including age, BMI, perceived sleep quality (global PSQI and seven component scores), and subjective PA levels. Chi-square analysis assessed differences for ACSM categorization, insomnia (questionnaire and SOL > 30min by actigraphy) and sleep apnea risk between ASD and TDC samples. A mixed-effects ANOVA was used to determine group by time interaction effect, group effect and time effect for the subjective daytime sleepiness scores. The comparison of sleep parameters and MVPA minutes was completed using a mixed-effects ANOVA with fixed effects of group (ASD versus TDC), time of week (weekend versus weekday), and their interaction, with testing for and inclusion of appropriate covariates (e.g. age, gender, ACSM categorization, employment/volunteer status, medication).

To begin analysis between sleep and PA in the ASD sample, Pearson and Spearman correlation analyses, as appropriate, were completed to explore the relationship between age, BMI and sex on perceived sleep quality and subjective PA levels. Further, correlation analyses between daytime MVPA minutes and the following night's sleep parameters were completed. As well, to analyse the reciprocal relationship, correlation analyses between sleep parameters and the following day's MVPA minutes were also completed.

The ASD sample was then categorized as either achieving or not achieving the ACSM's PA recommendations based on the GSLTPAQ and the actigraphy MVPA data. A discrepancy between the two measures occurred in 40.0% of the ASD and 23.5% of the TDC sample in which cases objective categorization was used. Once categorized, the comparison between categories and sleep parameters were completed using a mixed-effects ANOVA and effect sizes were reported. These were calculated following Cohen of $0.20 < d > 0.49$ to indicate small (i.e. negligible practical importance), $0.50 < d > 0.79$ to indicate medium (i.e. moderate practical importance, and $d > 0.80$ to indicate large (i.e. crucial practical importance) effect sizes[67]. To further explore the PA-sleep relationship in ASD, correlations were completed to analyse the relationship between daytime sleepiness levels and daily MVPA minutes. Pearson correlations were conducted between MVPA minutes with the evening KSS scores and also between morning KSS scores with the previous day's MVPA minutes. Finally, a spearman correlation was completed to determine the relationship between ACSM categorization and evening KSS scores.

All statistical analyses were performed using SAS 9.2 (SAS Institute, Cary, NC, USA). Our study was sufficiently powered (>80%) to detect a significant difference ($p \leq 0.05$) in SOL between the two samples based off SOL actigraphy data previously published in this population.[11] Statistical significance was set at $p \leq 0.05$.

Results

Table 2 compares the ASD to the TDC groups in terms of demographics and baseline subjective measures of sleep and PA. There were no differences between the groups on BMI, PSQI, PSQI subscales, GSLTPAQ, insomnia risk, or sleep apnea risk. However, significantly more individuals with ASD than TDC had a SOL of greater than 30mins (0.011). No covariates (age, sex, medication use and employment/volunteer status) impacted the results and therefore were not included in the model.

Primary Analysis

Actigraphy: ASD versus TDC samples

SOL was nearly twice as long in the ASD sample (36.5 min) than the TDC sample (19.6 min; $t_{187} = 2.66$, $p = 0.009$) with consistent results when looking separately at the weekday nights ($t_{187} = 2.38$, $p = 0.018$) and weekend nights ($t_{187} = 2.23$, $p = 0.027$) (Figure 2(d)). Eight of the 15 (53%) individuals with ASD took on average >30mins to fall asleep compared to only 2 of 17 (12%) in the TDC sample.

The ASD sample went to bed significantly earlier than the TDC sample on weekday nights by 96.4 min ($t_{187} = -2.61$, $p = 0.010$), on the weekend nights by 126.4 min ($t_{187} = -3.09$, $p = 0.002$), and overall for the week by 109.8 min ($t_{187} = -3.00$, $p = 0.003$) (Figure 2(b)). These earlier bedtimes resulted in the ASD sample spending longer time in bed by 68.9 min on weekday nights ($t_{187} = 3.05$, $p = 0.003$), by 88.7 min ($t_{187} = 3.25$, $p = 0.001$) on the weekend nights and by 77.8 min ($t_{187} = 3.50$, $p = 0.001$) overall for the week (Figure 2(a)). With more opportunity to sleep, TST was also longer in the ASD sample on the weekday nights by 49.0 min ($t_{187} = 2.26$, $p = 0.025$), on the weekend nights by 53.1 min ($t_{187} = 2.05$, $p = 0.042$), and overall for the week by 51.0 min ($t_{187} = 2.41$, $p = 0.017$) (Figure 2(c)). WT, WASO and SE% results did not yield significant differences between samples.

PA Levels

The objective average daily minutes of MVPA (mean ± SEM) was over twice as much in the TD sample (36.8 ± 5.7 mins) than the ASD sample (15.5 ± 6.1 mins; $t_{188} = -2.70$, $p = 0.008$). Significantly fewer individuals with ASD (40%) satisfied the ACSM PA recommendations of >150 mins of MVPA per week compared to the TDC sample (82.4%; $p = 0.017$).

PA-Sleep Relationship in ASD

The individuals with ASD ($N = 6$) who achieved the ACSM PA recommendations of >150 min of MVPA per week went to bed 111 min earlier ($t_{189} = 2.15$, $p = 0.012$) and woke up 97 min earlier ($t_{189} = 2.61$, $p = 0.010$) than those who did not meet the recommendations ($N = 9$) (Table 3). There was a significant correlation between more MVPA minutes achieved and an earlier BT (r = -0.24, $p = 0.016$) and earlier WT (r = −0.33, $p = 0.001$) for the following night (Table 4). MVPA minutes were also significantly correlated to a sleepier KSS score that evening (r = 0.48, $p = 0.002$). Also, there was a significant correlation between an earlier BT (r = −0.27, $p = 0.012$), earlier WT (r = −0.38, $p < 0.001$), less WASO (r = −0.21, $p = 0.046$) and higher SE% (r = 0.23, $p = 0.031$) with more MVPA minutes achieved the following day (Table 4).

Secondary Analysis

Actigraphy: Weekend versus Weekday

The ASD sample showed significantly worse SE% (t_{187} = 2.36, p = 0.019) and WASO (t_{187} = −2.35, p = 0.020) on the weekend compared to the weekday (Figure 2(e–f)). On average, the ASD sample spent 60.2 min awake on the weekend nights, compared to 43.5 min during the week (Figure 2(e)). This pattern was not observed in the TDC sample. However, the TDC went to bed on average 30 min later on the weekend compared to the weekday and this result trended towards significance (t_{187} = −1.85, p = 0.067) (Figure 2(b)). No other differences in sleep parameters between weekend and weekday were observed in either sample.

Daytime Sleepiness

The TDC sample reported significantly more sleepiness at bedtime than the ASD sample (t_{245} = −2.70, p = 0.007). No statistically significant differences were found between samples in the morning (t_{245} = −1.60, p = 0.110) or at midday (t_{245} = −0.09, p = 0.930). The TDC sample reported significantly increased sleepiness levels in the morning than at midday (t_{245} = 2.87, p = 0.005) and increased sleepiness levels at bedtime than at midday (t_{245} = −2.70, p < 0.001) (Figure 1). However, the ASD sample reported consistent levels of sleepiness across time with no significant changes in sleepiness between morning to midday (t_{245} = 0.36, p = 0.720) or midday to bedtime (t_{245} = −1.27, p = 0.110) (Figure 1).

Discussion

When comparing neurotypical young adults to young adults with ASD, the present study found that those with ASD had: (1) a longer SOL and less reported evening sleepiness, (2) extended TIB and TST, (3) lower objective PA levels, and (4) increased sleep disturbance on the weekend nights. In support of PA positively impacting sleep, those with ASD who were physically active had an earlier BT, earlier WT, less WASO, improved SE% and were sleepier in the evening than those who achieved fewer minutes of MVPA.

Table 3. ACSM categorization and sleep parameter relationship in ASD.

	Active (N = 6)	Insufficiently Active (N = 9)			
	mean ± SE	mean ± SE	t_{189}	p	d
TIB (hr:min)	9:26 ± 0:21	9:13 ± 0:27	0.41	0.680	0.060
BT (hh:mm, 24hr)	21:50 ± 0:40	23:41 ± 0:32	2.15	0.033	0.313
WT (hh:mm, 24hr)	7:15 ± 0:29	8:52 ± 0:24	2.61	0.010	0.380
TST (mins)	470.6 ± 23.5	457.6 ± 19.4	0.43	0.670	0.063
SOL (mins)	35.3 ± 7.3	37.2 ± 6.0	0.20	0.840	0.029
WASO (mins)	50.6 ± 13.4	47.0 ± 11.0	0.21	0.830	0.031
SE (%)	90.9 ± 2.6	91.2 ± 2.2	0.09	0.930	0.013

TIB: time in bed, BT: bedtime, WT: waketime, TST: total sleep time, SOL: sleep onset latency, WASO: wake after sleep onset, SE%: sleep efficiency, d: Cohen effect size.

Table 4. Correlation between sleep parameters and preceding and following day's MVPA minutes achieved in ASD.

	Preceding day MVPA		Following day MVPA	
	r	p	r	p
TIB	0.0068	0.95	−0.011	0.920
BT	−0.24	0.016	−0.27	0.012
WT	−0.33	< 0.001	−0.38	< 0.001
TST	0.13	0.20	0.16	0.150
SOL	−0.050	0.61	−0.020	0.850
WASO	−0.11	0.26	−0.21	0.046
SE%	0.14	0.17	0.23	0.031

TIB: time in bed, BT: bedtime, WT: waketime, TST: total sleep time, SOL: sleep onset latency, WASO: wake after sleep onset, SE%: sleep efficiency.

Sleep Parameters

Despite similar subjective sleep results between samples, objectively measurable differences existed, particularly in SOL. The ASD sample took nearly twice as long as the TDC sample to fall asleep, which is consistent with previous findings[11,12]. A potential reason for prolonged sleep latencies in ASD is pre-sleep arousal and the inability to "switch off thoughts"[11]. The KSS findings reported lower levels of sleepiness at BT (i.e. increased arousal) in the ASD sample compared to the TDC. Further, there was not a significant increase in sleepiness from midday to BT scores in ASD which, as expected, occurred in the TDC sample. Without the increase in sleepiness at BT, it is reasonable to expect the ASD sample to have longer SOL due to settling behaviours required prior to sleep initiation. It has been postulated that the reduced BT sleepiness and extended SOL seen in ASD is in part caused by the abnormally low nocturnal melatonin production in this population[68–70]. Recent research indicated endogenous melatonin levels do not differ in children and young adults with ASD not on medication compared to values from typically individuals.[29,71] Interestingly, Baker et al. (2017) also found significantly lower evening melatonin levels in individuals with ASD on medication for symptoms of anxiety and depression. In the present study, 86.7% of the ASD sample was on medication and may have played a role in the reduced bedtime sleepiness levels and extended SOL.

According to current guidelines, sleep latency >30min is indicative of insomnia[56]. This threshold was reached by half of the ASD sample, which is consistent with the literature on prevalence of insomnia in this population[11,22,72]. However, despite the majority having extended sleep latencies, only half of these individuals reported a sleep latency of > 30mins leading to only three out of 15 (20%) identified as having insomnia on the questionnaire. Under reporting is a common pattern found in sleep studies in ASD[10]. Some of the parents assisted with the questionnaires which may have led to the inability to capture the subtleties detected through actigraphy due to reduced involvement in sleeping habits as their child ages into young adulthood. This explanation is supported by a study that noted a significantly higher insomnia symptom prevalence in a sample of high-functioning young adults with ASD who are able to self-report compared to a control group[22].

In contrast to the majority of the literature on ASD children and adolescents, the ASD sample in the present study achieved significantly more sleep on average than the TDC sample. Consistent with young adults with an ASD diagnosis and intellectual disability in Hare et al. (2006)[26], the ASD sample had significantly earlier BT with longer periods of TIB, resulting in longer TST than the TDC sample. Deficits

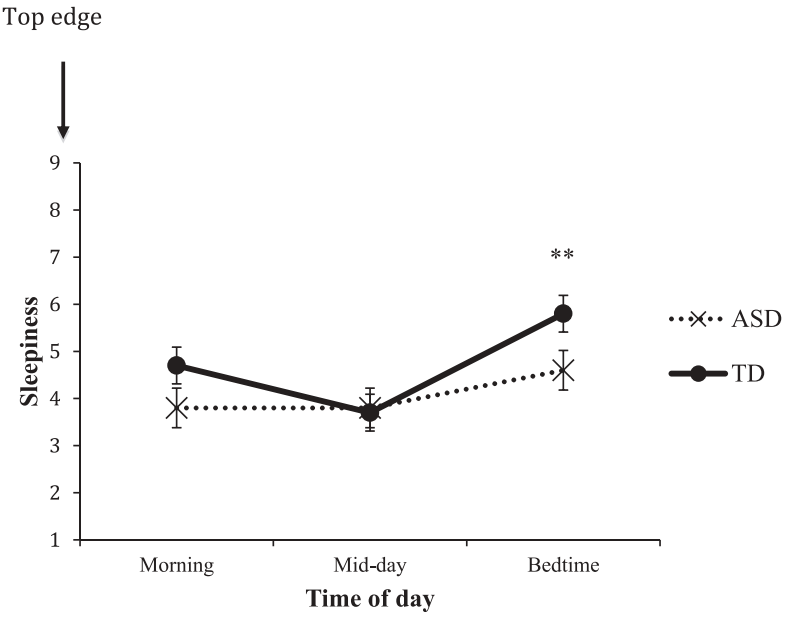

Figure 1. Mean (± SEM) for daytime sleepiness on the Karolinska Sleepiness Scale (KSS). Sleepiness in individuals with Autism Spectrum Disorder (ASD) did not change throughout the day. ASD were not as sleepy at bedtime compared to the typically developing controls (TDC). ** $p < 0.01$.

in social interaction and intellectual disability, common in ASD, may decrease late night socialization participation. Individuals with ASD are less likely to be invited to activities compared to adolescents and young adults with other disabilities and therefore do not encounter the same social pressures facing their TDC peers[73]. It is very plausible that young adults with ASD are able to spend more time in bed and asleep due to these factors[26,73]. The ASD sample had an average TST of 51.0 min more per night than the TDC sample. Although not a confounder in the present study, individuals with ASD with comorbid ID have been observed to have longer TST compared to higher functioning ASD[25,26]. Further, the longer TST finding agrees with Baker and Richdale (2015) study's findings which reported that young adults with ASD medicated for psychopathological diagnoses (i.e. anxiety and/or depression) achieved slightly more TST than a TDC control[22]. Although the present study did not assess psychopathological diagnoses, 66% of the ASD sample were on medications which have been shown to affect sleep.

Significantly longer WASO and lower SE% were found in the ASD sample on the weekend nights compared to the weekday nights despite the opposite being expected and seen in the TDC sample. It is possible that the consistency in the lives of the young adults with ASD is disrupted on the weekends due to changes in things such as parental work schedules, living arrangements or daytime activities. These changes may be enough to cause mental or physiological changes to affect sleep maintenance in this population. MVPA levels measured through actigraphy did not change from weekdays to weekend in the ASD sample and therefore is likely not a potential factor in the SE% differences detected. Further studies evaluating daytime activities and sleep would need to be completed to determine if there is a relationship.

Despite changes across the week, overall only a few individuals from the ASD and TDC sample were identified as having problematic SE% (<85%)[23]. The present results are contradictory to two studies that utilized actigraphy with young adults with ASD, which found problematic SE% across their sample-[14,22]. However, other objective studies on young adults with ASD found similar SE% between ASD and controls[11,12,16].

PA Parameters

The ASD sample objectively achieved fewer minutes of MVPA in daily living activities than the TDC sample which is consistent with accelerometery PA literature in this population[30]. These results are not surprising as individuals with developmental disabilities, including ASD, have a more difficult time engaging in sports and PA likely due to impairments in motor, social communication, sensory and behaviour domains[2]. Only 40% of the ASD sample achieved ACSM's PA guidelines of >150 MVPA per week as measured with actigraphy. This is in line with survey results suggesting 47–51% of older adults with developmental disabilities, including ASD, may be living dangerously sedentary lifestyles[74]. It is worrying that without an intervention, the sedentary habits observed in the ASD sample could lead to significant health problems in adulthood, such as metabolic syndrome[75,76].

PA-Sleep Relationship

Those with ASD who did meet the ACSM PA recommendations had an earlier BT and WT compared to those with ASD who did not met the ACSM PA recommendations. Further, more MVPA minutes during the day were correlated to a significantly earlier BT and WT, with increased levels of bedtime sleepiness. It is therefore possible that those who obtained greater levels of MVPA throughout their day depleted their energy stores with PA causing them to experience more sleepiness in the evening leading to an earlier BT and WT routine.

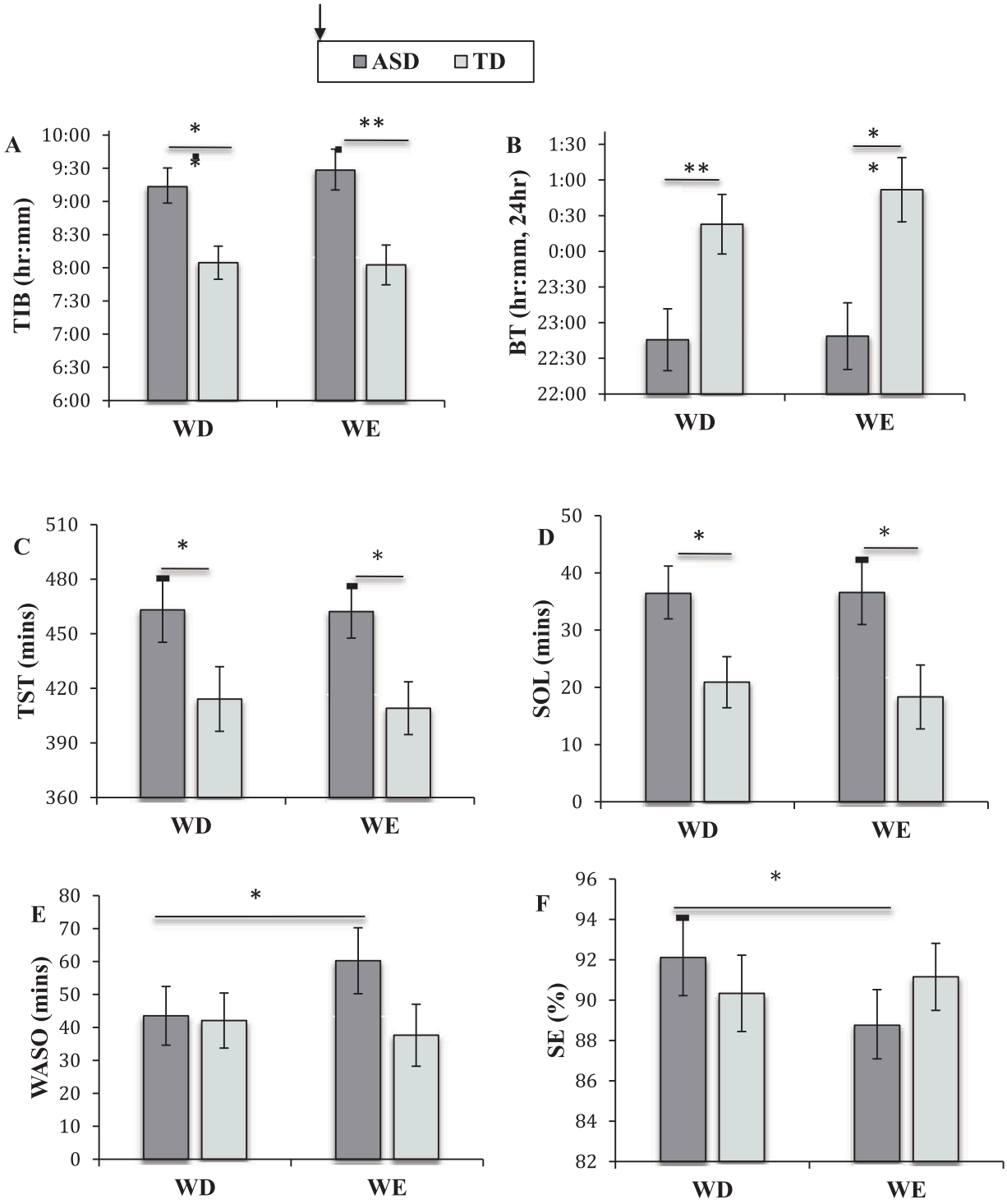

Figure 2. Means (± SEM) for TIB: time in bed (**A**); BT: bedtime (**B**); TST: total sleep time (**C**); SOL: sleep onset latency (**D**); WASO: wake after sleep onset (**E**); and, SE: sleep efficiency (**F**) for the WE: weekend; and, WD: weekday. * $p < 0.05$; ** $p < 0.01$.

Previous literature has found favourable objective sleep parameters following exercise in the ASD population[41,42]. MVPA minutes was not significantly correlated to improved WASO, SOL or SE%, however favourable objective sleep parameters did positively impact the following day's PA; improved sleep maintenance (i.e. improved WASO and SE %) correlated to higher levels of MVPA minutes the following day, supporting the reciprocity of the PA-sleep relationship. Good sleep habits and moderate PA could be mutually beneficial causing a virtuous circle[77]. More powerful and sensitive study designs are required to fully explore this complex, bi-directional relationship; however, these findings suggest promising benefit of the PA-sleep relationship in young adults with ASD[41,42,44].

Methodological Strengths and Limitations

The strengths of the present study were sleep and PA were measured using multiple methods (diary, questionnaires, actigraphy); sleep and PA were objectively measured; actigraphy data was collected at the same time of year in both samples to remove the potential zeitgerber factor of changing daylight hours; the samples were age- and gender-matched; and autism diagnoses were confirmed with completion of the ADOS.

However, there were also several limitations to the present study. Two-thirds (10 of 15) of the individuals with ASD were incapable of self-reporting, so the diary and questionnaire results were confounded by parent report. A second limitation was the TDC participants were not screened for the ASD phenotype or level of intellectual functioning. A third limitation of this study was the PA threshold has not been validated for the specific actigraph tool utilized. To determine the appropriate threshold, raw data and MVPA cut-offs point were compared to a validated actigraph. A fourth limitation was the small sample size, which decreases strength of analyses. Another limitation was the TDC was only screened for sleep medications. The TDC group was not included in the PA-sleep relationship data analysis therefore limiting the conclusion of this relationship in the ASD group. Finally, the high prevalence of medication used in the ASD may have had an impact on the results and future research will require a more robust study design to explore the relationship between medication and sleep.

Conclusion

The findings of the present study support previous research that suggest young adults with ASD have altered sleep patterns and daytime sleepiness levels at bedtime compared to TDC peers. As well, minutes of MVPA were significantly less in those with ASD than TDC. The young adults with ASD who were active, reported significantly greater sleepiness levels upon bedtime, went to bed earlier and woke up earlier the following day compared to their inactive ASD peers. Further, more minutes of MVPA where achieved on days following nights with improved sleep (i.e. decreased WASO, increased SE%) in ASD, exemplifying the reciprocity of the PA-sleep relationship. Results also included longer SOL, longer TST, and changes in sleep maintenance from weekday to weekend in ASD. It will be important in future research to continue the study of the PA-sleep relationship in ASD. Specifically, controlling time of day, intensity, duration and type of exercise, could improve our understanding of the relationship between PA, sleep and daytime sleepiness in the ASD population.

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Ethical Approval

The Conjoint Health Research Ethics Board of the University of Calgary approved the study, and the entire study was performed in accordance to the *Declaration of Helsinki*.

Declaration of Interest

The authors report no declarations of interest. The authors alone are responsible for the content and writing of the paper.

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