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ORIGINAL PAPER

Examining the Behavioural Sleep-Wake Rhythm in Adults with Autism Spectrum Disorder and No Comorbid Intellectual Disability

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Abstract This study aimed to examine the behavioural sleep-wake rhythm in 36 adults with autism spectrum disorder (ASD) and to determine the prevalence of circadian sleep-wake rhythm disorders compared to age- and sex-matched controls. Participants completed an online questionnaire battery, a 14-day sleep-wake diary and 14-day actigraphy assessment. The results indicated that a higher proportion of adults with ASD met criteria for a circadian rhythm sleep-wake disorder compared to control adults. In particular, delayed sleep-wake phase disorder was particularly common in adults with ASD. Overall the findings suggest that individuals with ASD have sleep patterns that may be associated with circadian rhythm disturbance; however factors such as employment status and co-morbid anxiety and depression appear to influence their sleep patterns.

Keywords Autism · Adults · Sleep-wake rhythm · Circadian · DSWPD · ASWPD

Introduction

Sleep problems are one of the most common comorbid conditions experienced by individuals with autism spectrum disorder (ASD), with insomnia symptoms including increased sleep onset latency (SoL), increased wake after sleep onset (WASO) episodes, early morning waking, and reduced total sleep time (TST) being most frequently reported (Mannion and Leader 2014; Richdale and Schreck 2009). Irregularities in the timing of sleep have also been reported for individuals with ASD, suggesting a disturbance of underlying circadian rhythms and thus a circadian rhythm sleep-wake disorder (CRSWD; Glickman 2010; Patzold et al. 1998; Stores and Wiggs 1998). Further support for the presence of CRSWDs in individuals with ASD comes from the body of literature demonstrating that mutations in CLOCK genes have been found in individuals with ASD (see Bourgeron 2007 for a review) and specific mutations have recently been noted in ASD individuals with insomnia symptoms (Yang et al. 2016). Moreover, variants in genes involved in the melatonin pathway (e.g., ASMT) have also been reported (Jonsson et al. 2010) with a dysregulation in the melatonin rhythm being reported by some researchers (Kulman et al. 2000; Melke et al. 2008; Nir et al. 1995; Tordjman et al. 2005, 2012); however a typical melatonin rhythm has also been reported in a small sample of children with ASD (Goldman et al. 2014).

Despite this growing body of literature, CRSWDs are rarely considered in the interpretation of sleep difficulties present in ASD, with the majority of research primarily focusing on insomnia and its symptoms. Nevertheless, there is some limited evidence for circadian sleep-wake rhythm disturbance and an increased prevalence of CRSWDs, particularly delayed sleep-wake phase disorder (DSWD), in individuals with ASD. However, most research conducted to date is limited by inadequate control groups (Hare et al. 2006; Wiggs and Stores 2004); DSM diagnostic criteria have not always been used to characterize the participants with ASD (Segawa 1985); and data primarily rely on subjective reports (Segawa 1985) or when objective measures are used, the ASD sample size is small (Hare et al. 2006; Limoges et al. 2005). Further, only one study (Hare et al. 2006) specifically addressed the question of the circadian

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sleep-wake rhythm in ASD. Nevertheless, these studies provide some preliminary insights concerning the presence of CRSWDs.

In an early Japanese study (Segawa 1985) mothers of 63 children with autism, aged 1–12 years, recorded their child's sleep-wake rhythm for at least 6-months using daily plots. Irregular sleep-wake patterns, with later sleep onset and wake times, and reduced night and increased day sleep were reported compared to siblings; one child had an apparent free-running rhythm. It was suggested that an abnormality in the "modulation of the sleep-wakefulness cycle" was present (p. 152). More recently, Wiggs and Stores (2004) used actigraphy to assess sleep in 62 children and adolescents (5–16 years) with ASD, finding objectively abnormal sleep patterns regardless of a reported sleep problem. Four sleep disorder categories (behavioural sleep problems, circadian sleep-wake problems, anxiety-associated sleep problems and other/unclassified sleeplessness) were identified. Eight children had a circadian sleep-wake problem; six children had a phase delay, that is later bedtimes and wake times and two of the children displayed an irregular pattern of sleep with at least two sleep episodes within 24 h. In addition, nine children had unclassified sleeplessness, where their sleep problems did not meet "conventional diagnostic criteria" (p. 377); symptoms included sleeplessness when going to sleep, during the night or in the early morning, during which time they did not seek attention or engage in specific activities. Comparison of 46 of the children (6–12 years) with 17 children from an age-matched, normative database indicated that the children with autism generally differed on the timing of sleep onset and waking (early or late), had increased SoL and night waking, lower sleep efficiency (SE%), and an abnormal activity pattern. Thus, both studies indicate the presence of circadian rhythm sleep disturbances.

Souders and colleagues (2009), also used actigraphy to compare sleep in 57 children with ASD and 37 controls aged 4 to 10 years, and classified their sleep problems according to the International Classification of Sleep Disorders—Second Edition (ICSD-2; American Academy of Sleep Medicine [AASM] 2005) criteria. Again poor sleep was more common in ASD, with the ASD group showing longer SoL, more awakening overall, and increased activity levels during sleep. Like Wiggs and Stores (2004), they found that insomnia with no apparent cause was present in 11 children who they classified as having insomnia due to their pervasive developmental disorder. They proposed that abnormalities in the melatonin rhythm or clock genes may underlie the children's sleep problems, indicating that a CRSWD might be the most likely explanation for the sleep difficulties experienced by this sub-group of children. Thus there is some evidence for circadian sleep-wake disturbances in children with ASD that may be related to other circadian abnormalities, and CRSWDs appear more common than in typical populations.

Two adult studies also support circadian sleep-wake disturbances in ASD (Hare et al. 2006; Limoges et al. 2005). In a sample of high functioning adults with ASD ($n=27$), earlier sleep timing (bed and wake times) on a sleep habits questionnaire was shown, indicating a mild phase advance compared to 78 age- and sex-matched controls (Limoges et al. 2005). Similar to the child research, there was more variability in the ASD group for sleep timing variables. Despite the mild phase advance reported for sleep timing, no significant differences were found between the two groups on Horne and Östberg's (1976) chronotype questionnaire; four individuals with ASD and five controls reported a moderate evening or morning type and no participants in either group reported an extreme chronotype.

Using actigraphy, Hare and colleagues (2006) investigated the circadian sleep-wake cycle and sleep difficulties in 10 adults with ASD (M_{age} = 30.8 years, $SD=6.91$) as compared with a convenience sample of 18 healthy controls. A phase delay with mean periodicity of 24.5 h and lower rhythm amplitude was found for the ASD group. Individuals with ASD had lower overall activity levels with less differentiation between their most and least active phases, and lower inter-daily stability, suggesting that their circadian rhythms "were less strongly linked to external zeitgebers" (p. 570). In addition, there was much more variability in the ASD group for the onset of the least active 5 h (range 22:00–04:00) and most active 10 h period (range 06:00–14:00). There was one instance of significant phase advancement and one instance of phase delay in the ASD group, which were not present in the control sample. Nevertheless, the mean acrophase did not differ between the groups, suggesting that the circadian rhythms of the ASD group were not desynchronised.

Some of the core behaviours and comorbid conditions experienced by individuals with ASD may make them more susceptible to the development of a CRSWD, due to reduced exposure to external zeitgebers. Individuals with ASD are reported to have hyper- or hypo-reactivity to sensory input (American Psychiatric Association [APA] 2013) which can include artificial lighting. Both light exposure and strategic avoidance or reduction in light exposure can shift circadian timing (Burgess and Molina 2014). Short wavelength blue light has been reported to have the most significant impact on human circadian timing; however Smith and Eastman (2009) reported that at bright intensities (e.g., 4000–5000 lux) both white and blue light have similar phase shifting effects. As a result, if individuals with ASD significantly alter their light exposure, this may impact on the entrainment of their sleep-wake schedules.

Circadian rhythms are also entrained via social zeitgebers (Aschoff et al. 1971). The social deficits that

individuals with ASD experience (APA 2013), in conjunction with the poor employment outcomes that are reported for adults with ASD (Levy and Perry 2011), may lead to a reduction or lack of exposure to these external zeitgebers, impacting on the entrainment of sleep/wake schedules. While the International Classification of Sleep Disorders—3rd Edition (ICSD-3; AASM 2014) indicates that a CRSWD puts one at risk of significant impairment in a range of areas, including occupational functioning, the relationships between employment status and CRSWDs are tenuous in the general population, with very little research directly addressing this association. In a large population study in the US (Grandner et al. 2010), unemployment was associated with more self-reported sleep complaints (trouble falling asleep and/or staying asleep, and sleeping too much). Although these symptoms are typically associated with insomnia, there is a large degree of overlap with CRSWD criteria. Similarly, in a large questionnaire survey in New Zealand (Paine et al. 2014), only night-work was associated with the presence ASWPD and DSWPD, not unemployment, while in a Japanese study of euythmic bipolar disorder patients (Takaesu et al. 2016), the ratio of employed to unemployed individuals was similar in those with and without a CRSWD. Lastly, individuals with ASD have an increased rate of comorbid psychopathology (APA 2013) including anxiety and depression (Buck et al. 2014). Psychopathology symptoms, particularly depression, and associated psychotropic medication use have been associated with circadian rhythm disturbances (Germain and Kupfer 2008).

Thus, the limited pool of current research and the inherent characteristics and accompanying comorbidities associated with ASD suggest that CRSWDs may be highly prevalent in individuals with ASD. This indicates a need for further investigation using larger, well-characterized samples of individuals with ASD. Classification of the specific sleep disorders underlying common reports of sleeplessness and insomnia symptoms in ASD, using well-defined samples and objective sleep measures will assist in determining whether or not CRSWDs are highly prevalent. This has important implications for the development of appropriate prevention and treatment techniques for poor sleep in ASD.

Therefore, the first aim of the current study was to investigate the sleep schedules and chronotype of adults with ASD and no intellectual impairment compared to control adults matched on age and sex. The second aim was to assess the presence of specific CRSWDs in both samples of adults and to compare the proportion of individuals in each group who met criteria for such disorders. Lastly, we explored the impact external zeitgebers may have on the presence of CRSWDs in both groups.

Method

Participants

The participants included here are the same as those previously described in Baker and Richdale (2015). Thirty-six adults with a clinical diagnosis of ASD (47.2% male; M_{age} = 34.41 years, SD = 6.52, range = 21:10–44:2) and 36 control adults (47.2% male; M_{age} = 32.66 years, SD = 5.49, range = 22:1–43:3) participated in this study; the groups did not differ on age, t (70) = −1.24, p = .221. The participants with ASD were recruited through the Olga Tennison Autism Research Centre (OTARC) participant registry (15), various Australian Autism Associations (11), ASD adult support groups (6), advertisements placed in clinics specialising in adults with ASD (1), flyers placed around the university campus (1), social media (1), and word of mouth (1). The control participants were recruited through flyers placed at the university and in the general public (10), the La Trobe University School of Psychological Science (7) and the OTARC participant registries (3), word of mouth (10), and social media (6). The flyer stated individuals were being sought for a 'sleep study' which also specifically stated in bold text "You DO NOT need to have a sleep problem to participate".

All participants completed the Autism Quotient (AQ; Baron-Cohen et al. 2001) and as expected ASD participants had significantly higher scores, t (70) = 15.35, p < .001; all control participants scored below the clinical cut-off of 26 (Woodbury-Smith et al. 2005). Thirty-four ASD participants provided a copy of their diagnostic report or a statement from their diagnosing clinician (n = 1) confirming their diagnosis and 32 of them completed Module 4 of the Autism Diagnostic Observation Schedule-2 (ADOS-2; Lord et al. 2012). The two participants, who did not provide a copy of their diagnostic report, completed the ADOS-2 and met criteria for ASD.

Thirty-two ASD and 33 control participants completed the Wechsler Abbreviated Scale of Intelligence (WASI; Wechsler 1999) or had their IQ scores obtained from their diagnostic report; the groups did not differ on FSIQ, t (63) = −0.736, p = .465, with all FSIQ scores > 80. The remaining participants had completed or were currently pursuing higher education qualifications including a diploma (post-secondary school; 1 ASD, 1 Control), a Bachelor's degree (3 ASD, 1 Control), or a post-graduate degree (1 Control). See Table 1 for information regarding demographic variables.

Forty-two percent of the participants with ASD were medicated for a comorbid diagnosis of an anxiety or mood disorder. Specifically, six individuals reported having both anxiety and depression, five had depression only, and four participants had a diagnosis of anxiety. Of these

Table 1 Demographic information for the ASD and control groups

	Control	ASD
ADOS score (M ± SD)	–	11.59 ± 4.13
AQ score (M ± SD)	11.31 ± 5.17	36.11 ± 8.20
FSIQ (M ± SD)	117.88 ± 8.80	119.81 ± 12.17
Employment status		
Full time	55.6%	25.0%
Part time	22.2%	30.6%
Student	19.4%	13.9%
Home keeper	0.0%	5.6%
Unemployed	2.8%	25%

participants seven had a morning prescription, five had an evening prescription, one had both morning and evening prescriptions, and the remaining two participants did not report this information in their sleep/wake diary.

The majority of participants in both groups were either employed on a full-time or part-time basis (Table 1). However, there was a significant difference between the two groups regarding employment status with significantly more control adults being employed full-time, $\chi^2 = 5.774$, $p = .016$, $\Phi = 0.312$ and significantly more adults with ASD being unemployed, $\chi^2 = 5.690$, $p = .017$, $\Phi = -0.321$. One participant with ASD working part-time undertook night-shift work on Fridays and Saturdays, finishing at 01:00, while another ASD participant working full-time commenced work at 04:00 (Mon-Fri).

The majority of participants in the ASD group lived with their parents (30.6%) or with their Spouse/Partner with or without children (30.6%). The remaining ASD participants lived alone (19.4%), in a share house (13.9%) or as a single parent (5.6%). The majority of control participants lived with their Spouse/Partner with or without children (47.2%). The remaining control participants lived with their parents (25%), in a share house (19.4%), alone (5.6%) or as a single parent (2.8%). The two groups did not differ on the proportion of those participants who lived with others versus living alone, Fisher's Exact Test $p = .151$.

Materials

Sleep Measures

Participants completed a 14-day sleep/wake diary (mornings and evenings). Sleep diary variables were averaged and the following sleep timing parameters were determined: Desired Bedtime, Bedtime (BT), Lights Out (LO), and Wake Time (WT). Participants were asked to respond to the following question to determine their desired bedtime "If you were free to plan your day today, what time would you prefer to go to bed tonight?" To determine the difference between actual bedtime and desired bedtimes a Bedtime Difference score was calculated for each night of the diary, and was subsequently averaged across all nights of the diary. As alarm clocks are a strong external synchronizer of circadian rhythms, participants also indicated their method of awakening each day by selecting from the following options: Alarm clock/radio, someone whom I asked to wake me (e.g., partner, parent), noises, or just woke up.

Participants wore an actigraph monitor (Actiwatch 2, Respironics Inc., Murraysville, PA) on their non-dominant wrist for the 14-day diary period. Data were digitized in 1-minute epochs with a sensitivity of 0.025 g (output voltage) and a bandwidth of 0.35–7.5 Hz (acceleration signal), and analyzed with Respironics Actiware 6 software (Respironics Inc., Murraysville, PA). Lights Out and Wake Time were extracted from the Actigraphy data. Sleep onset was defined as the first of five consecutive epochs of actigraphic sleep at the beginning of the scoring interval. Sleep offset was defined as the last of at least five consecutive epochs of actigraphic sleep at the end of the scoring interval. The actigraph monitor also assessed the amount of white light (lux) participants were exposed to during the 14-day data collection period and the average amount of white light each participant was exposed to during the 14 days was extracted from actigraphy data.

Chronotype

The Composite Scale of Morningness questionnaire (Smith et al. 1989) was used to assess each participant's chronotype that is, whether they were a morning- or evening-type person. The scale comprises 13 questions relating to the respondents' timing of waking and going to bed, preferred times for physical and mental activity, and subjective alertness. Higher scores are associated with a morning chronotype, while lower scores are associated with an evening chronotype. This can give an indication of the participant's circadian phase.

Classification of Circadian Rhythm Disorders

According to the ICSD-3 (AASM 2014), CRSWDs are classified as six distinct disorders; however only Delayed Sleep-Wake Phase Disorder (DSWPD), Advanced Sleep-Wake Phase Disorder (ASWPD) and Non-24-Hour Sleep-Wake Rhythm Disorder (N24SWD; formerly Free-Running Rhythm Disorder) were present in the participants in this study. According to the ICSD-3 criteria for CRSWDs, sleep diaries and actigraphy for a minimum of 7-days (preferably 14 days) is essential in evaluating and diagnosing CRSWDs. Assessment of endogenous circadian rhythms (e.g., melatonin levels) is desirable, but not essential in diagnosing such disturbances. The

classifications of each relevant CRSWD, as described below, are based on these criteria.

DSWPD is characterized by habitual sleep and wake times that are delayed, generally by two or more hours, relative to conventional or socially acceptable timing (AASM 2014). ICSD-3 reports that sleep onset is typically delayed until between 01:00 and 06:00. Individuals with DSWPD also have difficulty waking at a socially acceptable time required to prepare for work or education, with wake times typically occurring in the late morning/early afternoon. In particular on free days such as weekends delayed sleep and wake times are almost always seen (AASM 2014). Thus, the participants were classified as meeting criteria for DSWPD if they met one or more of the following criteria: (a) average bedtime was later than 01:00; (b) on average there was an approximate 2-hr bedtime delay from their diary reported desired bedtime; or (c) a 2-hr bedtime delay from their desired bedtime reported in the chronotype questionnaire.

ASWPD is characterized by an advance of the major sleep episode with habitual sleep and wake times typically occurring two or more hours prior to the individual's desired or required timing as evidenced by difficulties remaining awake until the required or desired bedtime in conjunction with an inability to remain asleep until the desired wake time (AASM 2014). Consequently, those with ASWPD typically report early morning waking or sleep maintenance insomnia. Individuals were classified as meeting criteria for ASWPD if they met each of the following criteria: (a) their average bedtime was prior to 22:00; (b) they had actigraphic WASO duration >30 min; and (c) they reported experiencing early morning waking or sleep maintenance issues three or more times per week on the Pittsburgh Sleep Quality Index (PSQI; Buysse et al. 1989), which was also completed as part of the larger study (Baker and Richdale 2015).

Lastly, N24SWD is typically characterized by a delay in sleep and wake times each day with a circadian period that is longer than 24 h (AASM 2014). Individuals with N24SWD typically have a history of insomnia and daytime sleepiness. A gradual increase in sleep latency resulting in delayed sleep onset occurs. As the sleep propensity rhythm shifts into daytime, individuals will have difficulties falling asleep at night time and difficulties staying awake during the day. As the propensity rhythm drifts further, individuals will experience late afternoon and evening sleepiness and reduced sleep latency resulting in an early sleep onset time (AASM 2014). Each participant's actogram was visually inspected to determine the presence of N24SWD according to these criteria.

Procedure

Institutional ethics approval was granted (Approval #HEC12-018). Participants responding to an advertisement were contacted and completed a screening questionnaire. Individuals were excluded if they had a diagnosis of Schizophrenia, or were taking sleeping medications, or beta-blockers. Control adults with a current diagnosis of anxiety or depression and/or who had a first degree relative with a diagnosis of ASD were also excluded. It was the intention to also exclude those adults with ASD who were medicated for a diagnosis of anxiety and/or depression given the impact such medications may have on sleep patterns. However, a significant number of individuals with such diagnoses were interested in participating in the study and given the heightened prevalence of comorbid anxiety and depression reported for adults with ASD (Buck et al. 2014) they were included in the current study. Further details regarding exclusionary criteria can be found elsewhere (Baker and Richdale 2015).

At the initial appointment participants provided written consent, completed the WASI and ADOS-2 (ASD only) and received their actigraphy monitor. Prior to this appointment, each participant was sent a link to complete their questionnaires, and 14-day sleep/wake diary online. The investigator (EB) logged in daily to check participants' diaries for completion and accuracy. Actigraph monitors were retrieved following the 14-day data collection period and data were downloaded via the Respironics Actiware 6 software. All data were entered into the IBM Statistics Software Package (IBM Corp., Armonk, NY, 2013) for analysis.

Data Analysis

All sleep timing variables were normally distributed in both groups. Scores on the chronotype questionnaire were normally distributed in the ASD group, while they were not in the control group. Average white light exposure was not normally distributed in either group. One participant with ASD only completed 3-days of the sleep-wake diary and thus their diary data were excluded from the analyses. Preliminary analyses revealed no differences on any sleep scheduling variables between the ASD adults medicated for psychopathology diagnoses (ASD-Med) and those who were not medicated for a psychopathology diagnosis (ASD-Only; Table 2). Re-analysis comparing those ASD adults medicated for anxiety and/or depression with an evening prescription to those not taking any psychotropic medications showed significantly later wake times (Diary: t (24) = 2.586, p = .016, ES = 0.22; Actigraphy: t (25) = 2.410, p = .024, 19); there were no other significant

Table 2 Comparison of diary and actigraphy variables between the two ASD groups using independent samples *t*-tests

Sleep variable	ASD-Only		ASD-Med		*p*	95% CI	η²
	M	*SD*	*M*	*SD*			
Diary							
Desired bedtime	23:00	1:11	22:18	1:16	0.104	−1:33, 0:09	0.08
Bedtime	23:41	1:32	22:47	1:43	0.115	−2:01, 0:13	0.07
Lights out	23:50	1:23	23:16	1:42	0.290	−1:37, 0:30	0.03
Wake time	7:02	1:17	7:34	1:57	0.340	−0:35, 1:39	0.03
Bedtime difference	37.50	41.60	28.33	61.11	0.601	−44.47, 26.13	0.01
Actigraphy							
Lights out	24:00	1:37	23:18	1:51	0.235	−1:53, 0:28	0.04
Wake time	7:27	1:29	8:14	1:59	0.182	−0:23, 1:57	0.05

differences between ASD-Only and ASD adults taking an evening prescription.

Therefore, the ASD group as a whole was compared to the control group. Independent Samples *t* tests were used to compare the two groups on these variables (or Mann–Whitney *U* tests when the assumption of normality was violated). We also report 95% confidence intervals (95% CI) and effect sizes. The Chi square test for independence (or Fisher's exact test when the assumptions of Chi square were violated) was used to compare chronotype data and proportions of individuals in each group who met criteria for a CRSWD as well as both ASWPD and DSWPD. Lastly, for both groups of adults we examined the impact external zeitgebers may have on chronotype and meeting criteria for a CRSWD, using Mann–Whitney *U* tests and Chi square tests for independence.

Results

We have previously shown that this group of adults with ASD have more general sleep disturbances as reported on the PSQI, significantly longer SoL (actigraphy), poorer sleep efficiency (SE%; diary) and they report poorer refreshment upon waking compared to the control group (Baker and Richdale 2015).

Sleep Timing Variables (Table 3)

The two groups did not differ significantly on any of the diary or actigraphy sleep timing variables and effect sizes were small; however there was significantly more variability in the ASD group on all sleep timing variables (Levene's test for equality of variance $p < .01$), except desired bedtime. Although no significant differences were found for desired bedtimes, difference scores between desired and actual bedtimes (Bedtime Difference) were greater in adults with ASD, with moderate effect size. ASD participants' actual bedtimes were on average 33 min later than their desired bedtime compared to a difference of only 11 min in the control group. The number of days that participants woke to an alarm clock did not significantly differ between the two groups (Baker and Richdale 2015).

Strong correlations were found between sleep-wake diary and actigraphy estimates of Lights Out Time in both the ASD group, $r = .982$, $p < .001$ and the control

Table 3 Comparison of sleep timing variables between the two groups using independent sample's t-tests

Sleep variable	ASD		Control		*p*	95% CI	η²
	M	*SD*	*M*	*SD*			
Diary[a]							
Desired bedtime	22:42	1:15	22:56	0:50	0.363	−0:16, 0:44	0.01
Bedtime	23:18	1:39	23:04	0:41	0.442	−0:50, 0:21	0.01
Lights out	23:35	1:32	23:31	0:42	0.786	−0:38, 0:29	0.00
Wake time	7:16	1:36	6:51	0:42	0.168	−1:00, 0:10	0.03
Bedtime difference	33.29	49.87	10.53	43.90	**0.045**	−44.99, −0.53	0.06
Actigraphy							
Lights out	23:43	1:44	23:31	0:45	0.537	−0:49, 0:26	0.01
Wake time	7:46	1:43	7:22	0:53	0.222	−1:07, 0:16	0.02

Bolding indicates significance

[a] $n = 35$ for the ASD group

group, $r = .970$, $p < .001$. Sleep-wake diary and actigraphy estimates of wake time were also strongly correlated in the ASD group, $r = .902$, $p < .001$ and the control group $r = .581$, $p < .001$.

Chronotype

The majority of participants in each group identified themselves as being an intermediate chronotype (28 Control, 26 ASD). Of the remaining participants, eight control participants and four ASD participants reported a morning chronotype and six ASD participants and no control participants reported an evening chronotype. The differences on chronotype were significant, Likelihood ratio = 9.751, $p = .008$, $\Phi = 0.321$. Of the six individuals with ASD who reported an evening chronotype, two were medicated for a diagnosis of anxiety and one for depression. Likewise, one participant with ASD who reported a morning chronotype was medicated for a diagnosis of anxiety and another for depression and anxiety.

Age was not significantly correlated with chronotype scores in either group; controls $r_s = 0.225$, $p = .187$, ASD $r = .118$, $p = .492$. Scores on the chronotype questionnaire were not significantly correlated with diary SoL in either group, controls $r_s = -0.328$, $p = .051$, ASD $r = -.205$, $p = .238$. However, scores on the chronotype questionnaire were significantly correlated with actigraphic SoL in the ASD group, $r_s = -0.425$, $p = .010$, but not in the control group, $r_s = -0.158$, $p = .357$.

Classification of Circadian Rhythm Sleep Disorders

Overall adults with ASD (44.4%) were more likely to meet criteria for a circadian rhythm disorder compared to control adults (5.6%), $\chi^2 = 12.519$, $p < .001$, $\Phi = -0.513$. Despite there being a higher proportion of ASD-Med adults meeting criteria for a CRSWD (60.0%) compared to ASD-Only participants (33.3%), the difference was not significant, $\chi^2 = 1.556$, $p = .212$, $\Phi = 0.112$. Both groups of ASD participants had a significantly higher proportion of participants meeting criteria for a CRSWD compared to controls; ASD-Only, Fisher's exact test $p = .009$ and ASD-Med Fisher's exact test $p < .001$. In addition, comparing those ASD-Med adults with an evening prescription, to ASD-Only participants, the prevalence of those meeting criteria for a CRSWD did not differ, Likelihood ratio = 2.127, $p = .145$, $\Phi = 0.282$.

Delayed Sleep Wake Phase Disorder

A significantly higher proportion of adults with ASD (30.6%) met criteria for DSWPD compared to control adults (2.8%), $\chi^2 = 8.100$, $p = .004$, $\Phi = -0.373$. Similar to the chronotype data, 54.5% of the ASD participants meeting criteria for DSWPD were medicated for a diagnosis of depression and/or anxiety. In comparing chronotype data with the classification data, five of the participants with ASD meeting criteria for DSWPD reported an evening chronotype. The only control participant meeting criteria for DSWPD reported being an intermediate chronotype. Demographic and sleep-wake information for each individual meeting our criteria for DSWPD is presented in Table 4.

Figure 1 provides an example actogram of an individual with ASD who met criteria for DSWPD.

Advanced Sleep Wake Rhythm Disorder

Although a higher proportion of adults with ASD (8.3%) met criteria for ASWPD compared to control adults (2.8%), the difference was not significant, Fisher's exact test $p = .239$. Another two ASD participants had early morning waking (before 05:00). One of these participants was a shift-worker, required to wake early for a 04:00 work start. Given it is not clear whether this individual's sleep patterns are associated with her employment status or whether she has selected her employment based on her sleep schedule (i.e., she has an ASWPD), we did not classify her as ASWPD; her sleep profile is presented in in the last row of Table 5. The other participant had an average bedtime of 22:49 thus, not fulfilling criteria for ASWPD. Those participants who met criteria also reported having sleep maintenance and/or early morning waking *three or more times per week* on the PSQI. Two out of three of these participants reported a morning chronotype and one had diagnoses of anxiety and depression. Although eight control participants reported a morning chronotype only one of these participants met criteria for ASWPD. For the remainder of those control participants, the average bedtimes and wake times were 23:24 and 06:44, respectively. Demographic and sleep-wake information for each individual meeting criteria for ASWPD are presented in Table 5.

Figure 2 provides an example actogram for an individual with ASD who met criteria for ASWPD.

Non-24-H-Sleep-Wake Rhythm Disorder

One adult with ASD met criteria for N24SWD, which was not observed in any of the control participants. This individual was medicated for a diagnosis of depression and was unemployed. Figure 3 displays the actogram for this participant.

Table 4 Demographic and sleep-wake information for individuals classified with delayed sleep-wake phase disorder

Group	Sex	Employment status	Psychotropic use (prescription time)	Average weekday LO	Average weekday WT	Average weekend LO	Average weekend WT	Chronotype desired BT	BT difference	Classification criteria
ASD	F	Part-time[a]	None	1:17 22:00–3:01	10:25 7:53–12:56	2:19 0:00–3:30	11:31 10:19–12:17	0:30–1:45	120.0	A, B
ASD	M	Unemployed	None	1:49 0:20–2:45	7:09 5:54–08:19	1:39 12:49–2:38	7:23 6:08–8:49	22:15–0:30	94.00	A
ASD	M	Part-time	Tricyclic (NR)	0:51 23:31–02:41	9:33 8:48–10:48	1:20 0:10–4:10	8:04 6:41–9:07	21:00–22:15	86.00	A, C
ASD	F	Part-time	SSRI (M)	0:44 22:36–2:41	8:27 6:33–9:50	0:40 0:10–1:29	7:23 7:09–7:33	1:45–3:00	111.00	B
ASD	F	Student	SSRI (M)	0:47 23:30–3:02	9:32 8:08–11:35	1:49 1:07–2:09	9:04 7:27–9:53	1:45–3:00	4.00	A
ASD	F	Part-time	Norepinephrine uptake inhibitor (E)	1:28 23:35–2:18	7:48 6:53–9:13	1:19 0:44–1:53	8:11 7:49–8:28	0:30–1:45	87.00	A
ASD	M	Unemployed	SSRI (E)	1:26 0:22–2:01	9:20 7:06–10:36	1:27 12:51–2:12	10:11 9:00–11:28	20:00–21:00	48.00	A, C
ASD	M	Unemployed	None	2:55 1:13–4:38	8:33 7:25–9:36	2:37 1:13–4:38	8:23 6:10–9:42	0:30–1:45	36.00	A
ASD	F	Unemployed	Anti-psychotic (NR)	0:11 23:04–1:10	9:20 6:50–10:09	1:13[b] 12:55–1:30	9:13[b] 7:53–10:34	20:00–21:00	65.00	C
ASD	M	Unemployed	None	1:19 0:04–3:12	8:13 6:14–9:23	1:36 12:38–3:17	8:19 7:24–9:28	0:30–1:45	77.00	A
ASD	M	Student	None	3:43 22:31–7:20	11:10 8:11–15:32	4:07 3:36–5:21	11:31 9:24–13:47	0:30-1.45	–[b]	A, C
Control	M	Full-time	NA	0:55 0:24–1:30	8:00 7:41–9:26	1:27 0:33–3:57	9:26 7:44–11:13	10:15–12:30	39.00	A

Medication prescription type—(NR) = not reported; (E) evening; (M) morning

Classification Criterion—A = LO > 1:00am; B = Approximate 2-hr BT delay from desired BT; C = 2-hr BT delay from desired chronotype BT

[a]Undertakes shift-work on weekends

[b]Based on only two days of data; only 3 days of diary data provided therefore no BT difference was calculated

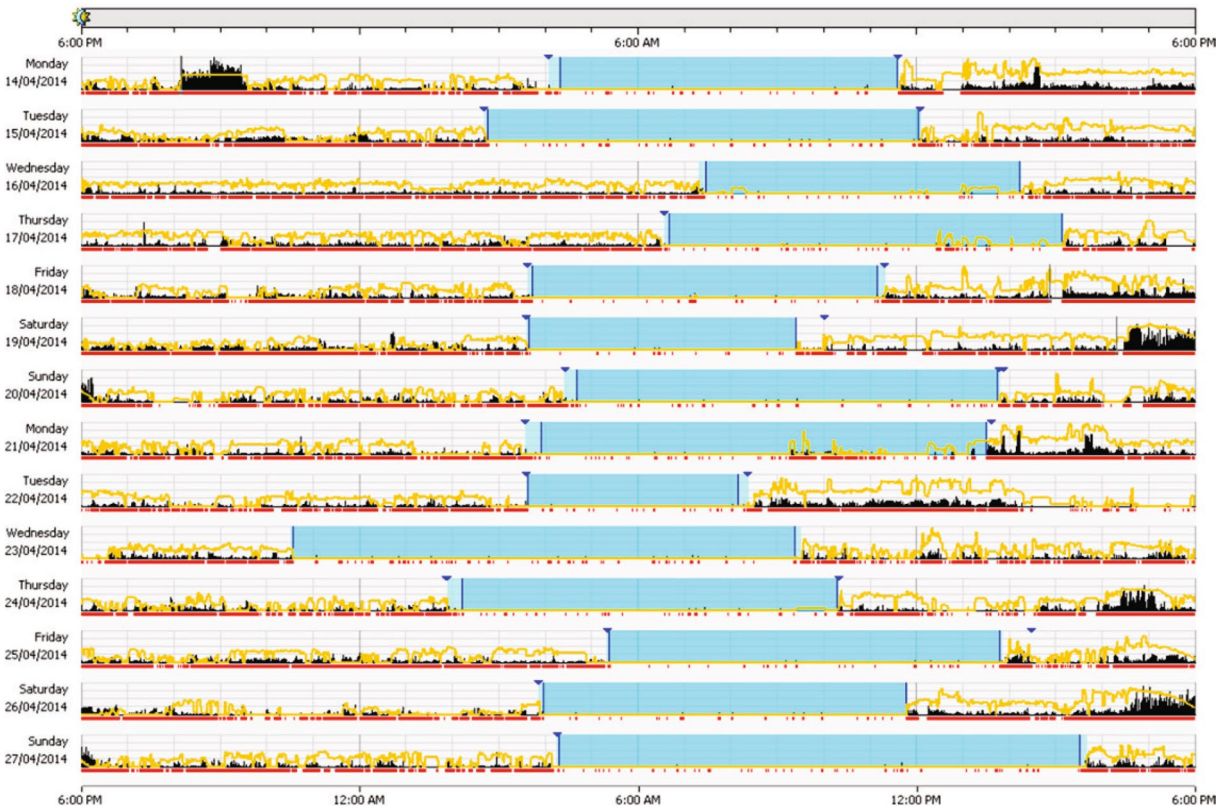

Fig. 1 Delayed sleep-wake rhythm example actogram

Unclassifiable Sleep-Wake Rhythm Disorder

Figure 4 displays the actogram of a participant with ASD who had very irregular sleep-wake patterns but did not meet the ICSD-3 criteria for Irregular Sleep-Wake Rhythm Disorder (ISWRD). In particular ICSD-3 reports that at least three sleep episodes should be observed across a 24-hour period and that sleep episodes are typically brief (AASM 2014). As can be seen in Fig. 4 this individual never had more than two sleep episodes during a 24-hour period and sleep episodes were variable in length with some short episodes (e.g., 142 min) and some very long episodes (e.g., 788 min). Similar to the individual classified with N24SWD, this individual was medicated for comorbid diagnoses of anxiety and depression and was also unemployed.

Relationship between Chronotype and CRSWDs with External Zeitgebers

Light

While the ASD participants were exposed to lower levels of white light ($M = 98.95$ lux, $SD = 103.92$, $Md = 58.22$ lux) on average they did not differ significantly from control participants ($M = 118.01$ lux, $SD = 117.23$, $Md = 80.78$ lux), $U = 553.00$, $p = .285$, $r = .12$. White light levels were significantly correlated with chronotype scores in the ASD group, $r = .333$, $p = .047$, but not in the controls $r = -.163$, $p = .342$. Inspecting the light levels of participants, the ASD participant classified with N24SWD and the ASD participant with irregular sleep-wake patterns both had very low average light exposure across the 14-day data collection period; specifically 10.13 lux and 5.86 lux of white light, respectively.

Social Cues (Employment and Living Arrangements)

In assessing the relationship between CRSWDs and living arrangements in the adults with ASD, the proportion of those who met criteria for a CRSWD who lived alone (25.0%) did not differ significantly from those who did not meet criteria for a CRSWD (15.0%), Fisher's Exact test $p = .369$.

A significantly higher proportion of adults with ASD meeting criteria for a CRSWD were unemployed or a student (62.5%) compared to adults who did not meet criteria for a CRSWD (20.0%), $\chi^2 = 5.086$, $p = .024$, $\Phi = 0.433$. Inspecting the specific CRSWDs five participants meeting criteria for DSWPD were unemployed, two were students

Table 5 Demographic and sleep-wake information for individuals classified with advanced sleep-wake phase disorder

Group	Sex	Employment status	Average weekday LO	Average weekday WT	Average weekend LO	Average weekend WT	Average WASO (min)	Chronotype desired BT	PSQI early morning waking or WASO	Classification criteria
ASD	F	Full-time	20:49 19:57–22:04	6:21 5:07–7:44	21:47 21:14–22:35	6:55 5:50–8:26	75.00	8:00–9:00	Yes	A, B, C
ASD	F	Part-time	21:33 20:37–23:13	5:25 4:19–6:59	21:58 21:43–22:06	7:20 5:29–8:34	92.38	9:00–10:15	Yes	A, B, C
ASD[a]	M	Unemployed	20:14 19:00–22:00	5:22 3:57–6:19	19:29 19:21–19:37	5:49 5:34–6:03	65.15	8:00–9:00	Yes	A, B, C
Control	F	Part-time	21:48 21:00–22:59	6:29 6:05–7:15	22:15 22:00–22:33	6:31 5:31–7:03	51.43	9:00–10:15	Yes	A, B, C
ASD[a]	F	Full-time	20:42 19:50–21:16	3:53 2:49–6:26	21:13[b] 20:42–21:44	7:38[b] 6:41–8:36	65.33	9:00–10:15	Yes	A, B, C

Classification criterions–A = Average BT < 10:00 p.m.; B = difficulties maintaining sleep (actigraphy); C = Report early morning waking/WASO issues three or more times per week on the PSQI

[a]Taking an SSRI, morning prescriptions

[b]Based on only 2 days of data

and four worked part-time; one of whom worked shift-work on weekends. Of those meeting criteria for ASWPD, one worked full-time, one worked part-time and the other was unemployed, as was the individual with N24SWD.

Discussion

A key finding of this study was that a higher proportion of individuals with ASD met criteria for a CRSWD compared to control adults matched on age and sex, with a large effect size. More specifically adults with ASD were more likely to meet ICSD-3 criteria for DSWPD compared to control adults. While ASWPD was also more common in the ASD group, the groups did not significantly differ on this CRSWD. Moreover, one individual with ASD met criteria for N24SWD and another had very irregular sleep-wake patterns that did not meet full ICSD-3 criteria for ISWRD. Neither of these latter sleep patterns were observed in the control group. These findings are similar to those of Hare and colleagues (2006) who reported an overall phase delay in their sample of 10 adults with ASD who were of a similar age to our participants. Additionally, Hare et al. (2006) also noted one case (10%) of significant phase advancement in their ASD group, which is similar to the proportion found in our ASD adults. Our findings also indicate some degree of overlap between insomnia symptoms and DSWPD, as actigraphic SoL was positively associated with eveningness on the chronotype questionnaire in the ASD group.

The prevalence of CRSWDs in our sample of adults is higher than that reported in children and adolescents with ASD (Wiggs and Stores 2004). Wiggs and Stores (2004) observed DSWPD and irregular sleep wake patterns in 11.6% of their group; similar to our adults, DSWPD was more prominent than irregular sleep patterns. In children with ASD, it is possible that familial, environmental, and social factors help to maintain acceptable entrainment and sleep-wake timing, making them less vulnerable to the development of a CRSWD than adults, who are more able to self-select sleep timing and other activities. However, the diagnostic criteria for DSWPD do not include reference to stalling or refusal to go to sleep (Glickman 2010), which are also commonly reported in children with ASD (Wiggs and Stores 2004; Souders et al. 2009) and being generally "more energetic and less sleepy at bedtime" (Patzold et al. 1998, p. 532); such disturbances may be associated with DSWPD. Children may not be in the sleep propensity phase of their rhythm and thus may not be tired at the time their parents are putting them to bed. Therefore, those children with ASD who stall or refuse to go to sleep may meet criteria for DSWPD or be susceptible to developing it later in life.

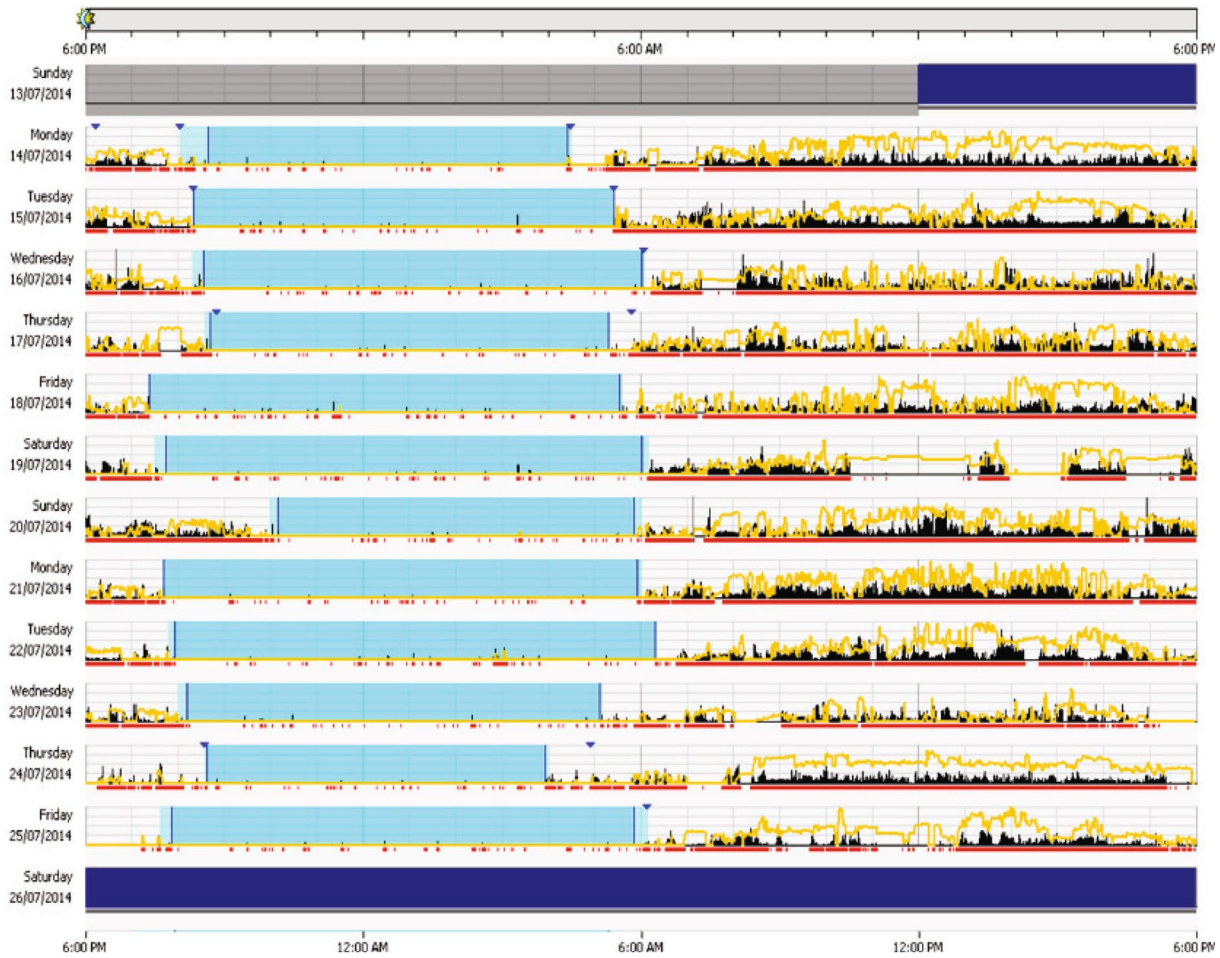

Fig. 2 Advanced sleep-wake rhythm example actogram

Three of our adults also met criteria for ASWPD, which has not been reported in the ASD child literature. ASWPD is typically associated with aging and is more frequently observed in older adults (AASM 2014), while a delayed chronotype tends to peak around the age of 20 years in the general population (Roenneberg et al. 2004), and then declines during young adulthood. This shift in chronotype with age may explain the difference in prevalence of ASWPD between children and adults with ASD. All the adults in our sample who met criteria for ASWPD were aged over 30 years (ASD: 33:1, 39:2, 43:11 years; Control: 38:7); Limoges et al. (2013) also found a mild phase advance in their adults with ASD. Thus it appears that while delayed sleep timing, evening chronotype, and the presence of DSWPD are more frequent in ASD samples in general, as adults with ASD move into their thirties they may be vulnerable to the development of advanced sleep timing.

Despite the higher proportion of CRSWDs in the ASD group, the two groups did not differ on sleep-wake timing variables. This may be due to 44% of the ASD participants meeting criteria for one of several CRSWD diagnoses, resulting in extreme bed and wake times at both ends of the sleep timing spectrum, with the remainder of the ASD sample having typical sleep timing patterns. Thus variance was high and significantly different from the controls, but group average values were consistent with a typical sleep-wake pattern. Supporting the higher proportion of adults meeting criteria for DSWPD, the difference between desired and actual bedtimes (bedtime difference) scores were also significantly larger in the ASD group. Thus the high variability in sleep parameters noted in some ASD child sleep literature (Allik et al. 2006; Inanuma 1984) may mask individual children with ASD and a CRSWD.

Employment status was associated with the presence of a CRSWD. Those adults meeting criteria for a CRSWD were more likely to be unemployed or a student. The two individuals who were homemakers had young children who attended school and the regular routines associated with caring for children may be protective against developing a CRSWD. The lack of social cues and more flexibility in timing of daily routines for those who are unemployed or

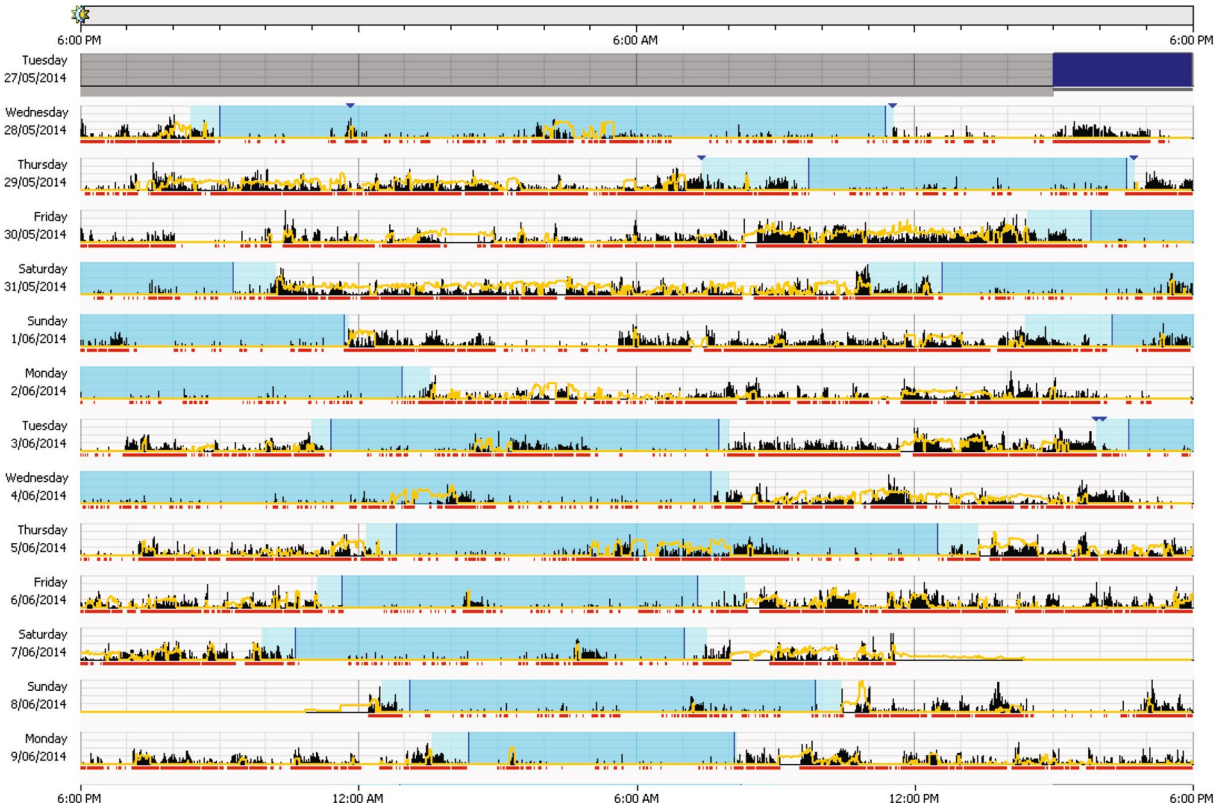

Fig. 3 Non-24-h sleep-wake rhythm example actogram

are students may increase their susceptibility to the development of a CRSWD, further exacerbating difficulties in gaining employment and opportunities for socialisation. This relationship is also likely bi-directional, with the presence of a CRSWD resulting in reduced structure and stability, affecting employment opportunities. Employment outcomes are frequently poor in individuals with ASD (Levy and Perry 2011) and the late rise times associated with DSWPD are likely to further impact on their potential for regular employment.

Given the cross-sectional nature of this study it is difficult to determine if these individuals with ASD had a CRSWD first, leading to difficulties with employment, or it is the nature of ASD that has made it difficult to obtain employment with the consequent development of problematic sleep patterns. Difficulties in understanding typical social relationships and emotional reciprocity may make it difficult for an adult on the autism spectrum to obtain and maintain employment. Nonetheless, if a CRSWD is present the individual may adopt a lifestyle, including employment, that fits with their sleep pattern. One of our individuals with ASD, DSWPD and a reported evening chronotype undertook shift-work on weekends. Thus, it may be that her weekend employment had some influence on her sleep patterns, her choice of employment may have been dictated by her chronotype and preferred sleep patterns. But it may not always be possible to match employment with sleep preferences. This participant still had significantly delayed bedtimes on non-work nights and there were large discrepancies between her actual and desired bedtimes, thus fulfilling our criteria for DSWPD. Although the participant who worked early morning shifts met all our criteria for ASWPD, and she may have also chosen her employment based on her sleep patterns, we did not have enough evidence to warrant a diagnosis of ASWPD. It is possible that once this individual ceases such shift-work her sleep patterns may revert to a typical sleep schedule.

According to ICSD-3 (AASM 2014) social and behavioural factors play an important role in the development and maintenance of delayed sleep patterns. Personal, social and occupational activities that continue into the late evening are likely to perpetuate and exacerbate a sleep phase delay. For individuals with ASD, later than desired bedtimes and consequent delayed sleep onset may be a result of the inherent social difficulties and repetitive, stereotyped and restricted range of interests that are the hallmark of ASD. Individuals with ASD may become fixated on their 'special interest' or feel the need to complete certain activities or tasks before retiring to bed, resulting in a later than desired bedtime. Individuals with ASD are

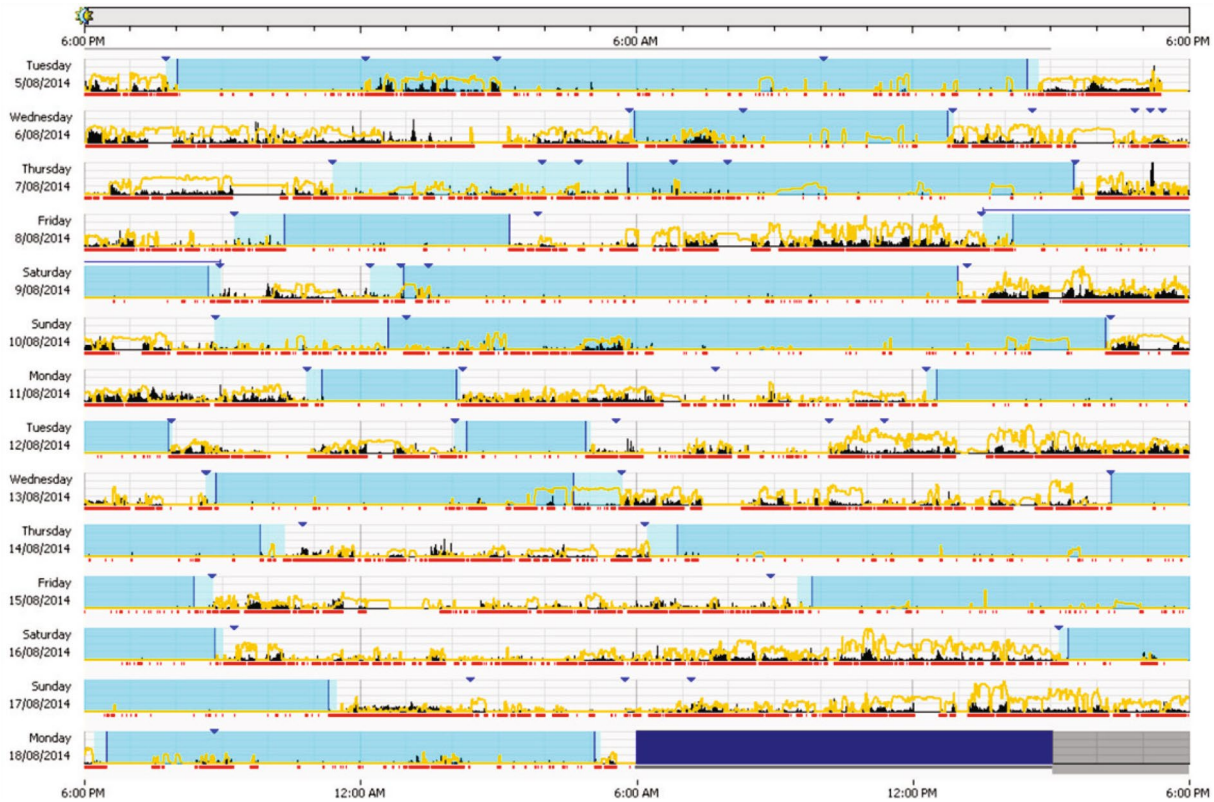

Fig. 4 Non-classifiable sleep-wake rhythm example actogram

also reported to spend more time using non-social media (e.g., television and computer games) than the general population (Mazurek et al. 2012; Mazurek and Wenstrup 2013). The blue light emitted from such devices has been reported to delay sleep onset, delay circadian timing and suppress melatonin levels (Chang et al. 2015). Thus, late night use of electronic devices may delay bedtime as well as sleep onset in adults with ASD. Supporting this notion, one participant with ASD reported playing computer games until 07:30 on one occasion during the data collection period, while another reported during her ADOS-2 assessment that video games were her 'special interest'.

Individuals with ASD frequently report sensory issues including hypo- or hyper-sensitivity to light (APA, 2013). Therefore, some individuals may reduce their light exposure leading to a lack of entrainment of the circadian rhythm. Although the ASD group on average were exposed to less white light than control participants, the difference was not significant; however the ASD participant classified with N24SWD and the ASD participant with irregular sleep-wake patterns both had extremely low average white light exposure, which may play a crucial role in their very irregular sleep patterns. Moreover, adults with ASD did not significantly differ on their exposure to other key external zeitgebers including waking to an alarm clock and their living arrangements.

While ASWPD is not likely to impact on the ability to gain employment it may affect an individual's ability to form and maintain social relationships. Individuals with ASWPD often feel distressed as their increased sleepiness and fatigue in the early evening prevents them from participating in evening social or family activities (Gamaldo et al. 2014). One individual with ASD who met criteria for ASWPD, expressed difficulties in making and keeping friends during the ADOS-2 assessment. Early sleep times may be preventing this individual from finding opportunities to socialise with others. As adults with ASD already have difficulties with social relationships problematic sleep patterns may further exacerbate their social difficulties.

Although more individuals with ASD who were medicated for a comorbid diagnosis of anxiety or depression met criteria for a CRSWD than adults with no comorbid psychopathology, the proportion was not significantly different. Depression is often associated with DSWPD (AASM 2014) while anxiety has been associated with a morning chronotype (Tsaousis 2010). Tsaousis (2010) found that morning types were more neurotic compared to evening types and the overlap between neuroticism and anxiety is

high (Hettema et al. 2004). While individuals in Limoges et al.'s (2005) study had no comorbid psychiatric diagnosis, they had higher trait anxiety scores on the Speilberger State Trait Anxiety Inventory (STAI; Spielberger et al. 1983) compared to control participants. Thus the authors postulated that anxiety may be involved in advanced sleep timing in their adults with ASD. The current study also supports this notion. One of the participants meeting criteria for ASWPD and the shift-worker with an ASWPD presentation were both medicated for diagnoses of anxiety. Moreover, the other two participants with ASD and ASWPD and the participant with very early wake times all obtained a maximum score (2) for the ADOS-2 anxiety code, which requires the individual to demonstrate marked anxiety either intermittently or continuously throughout the assessment. Thus as has been reported in the general population, individuals with ASD who have high levels of anxiety may prefer early bed times. However further research in a larger sample of adults with comorbid psychopathology diagnoses is required.

Clinical Implications

CRSWDs in adults with ASD have the potential to exacerbate difficulties associated with ASD such as gaining employment and social activities, but there is also growing evidence that CRSWDs are associated with poor health outcomes in the general population. Circadian disruption and/or misalignment can result in "physiological aberrations, alterations and dysfunctions that are relevant for the maintenance of health and development of disease" (Summa and Turek 2014, p. 78). More specifically circadian misalignment has been associated with obesity, cardiovascular disease, gastrointestinal disease, and some forms of cancer (Summa and Turek 2014) and there is emerging evidence that adults with ASD have elevated rates of diabetes, gastrointestinal disorders, hypertension, and obesity (Croen et al. 2015). Thus given individuals with ASD are at a higher risk for the development of sleep problems including CRSWDs as well as physical health problems that have been associated with circadian misalignment in the general population, further research is vital to understand these relationships and their potential impact on individuals with ASD.

Strengths and Limitations

This is one of the first studies to examine and classify CRSWDs in adults with ASD. The findings are generally consistent with a small body of research that has suggested abnormalities in sleep timing in ASD with a possible disruption of the underlying circadian rhythms. Our prevalence rates of CRSWDs are higher than those previously reported; however, children were assessed in earlier research (Souders et al. 2009; Wiggs and Stores 2004) and the sample size in the one adult study was smaller than ours (Hare et al. 2006). Nevertheless, it is important to note that ICSD-3 (AASM 2014) requires symptoms to be present for at least 3 months for a diagnosis of a CRSWD, but participants in this study were classified based on 14 days of actigraphy and a 14-day sleep/wake diary. Thus we may have overestimated the prevalence of CRSWDs in the current sample, as this may not have been a typical sleep period or symptoms may have been of recent origin. Nonetheless, two ASD participants reported in the screening questionnaire that they had previously been diagnosed with DSWPD. One of these participants was then classified with DSWPD based on our study criteria, while the second participant had the irregular sleep-wake pattern. Additionally, since participating in the study a third participant with ASD who had met study criteria for DSWPD wrote to the first author stating that he had recently been diagnosed with DSWPD. Moreover, with the exception of two Japanese studies that assessed sleep continuously in children with ASD for 1 month (Inanuma 1984) and 6 months (Segawa 1985) by parent report, the assessment period in this study is similar to Wiggs and Stores (2004; 14-days) and longer than that of Hare and colleagues (2006; 7-days).

Lastly, our ASD and control participants were not matched on employment status and controls were excluded if they had an anxiety and/or depression diagnosis, which may account for the significantly higher proportion of adults with ASD meeting criteria for a CRSWD. Nonetheless, in the study conducted by Paine et al. (2014), only night work was associated with the presence of ASWPD or DSWPD, and only a minority of our ASD participants engaged in such employment. Given, the significant associations between depression and CRSWDs in the general population (Germain and Kupfer 2008) research comparing adults with ASD (medicated and non-medicated) to non-ASD adults with anxiety and depression diagnoses, would be beneficial in elucidating the relationship between psychopathology symptoms and CRSWDs in adults with ASD. Future research aiming to delineate factors that contribute to the presence of CRSWDs in individuals with ASD (e.g., employment and psychopathology status) or whether it is indeed the core features of ASD that result in problematic sleep timing, will assist in furthering our understanding in this area.

Conclusion

Overall our findings indicate that there is a significant sub-group of adults with ASD who meet behavioural sleep criteria for a CRSWD. Several factors are likely to

be associated with the development of these problems including the core symptoms of ASD, employment status, and comorbid psychopathology. However, future research should aim to include larger samples and longitudinal designs that track sleep timing and patterns in large samples from childhood through to adulthood. This is likely to further highlight factors, including those described here, which may be associated with the development of CRSWDs. With this knowledge targeted interventions and treatments can be developed to improve health outcomes and well-being for individuals with ASD. Lastly, studies investigating melatonin profiles, including the dim light melatonin onset (DLMO), would be beneficial to further confirm the presence of CRSWDs in individuals with ASD.

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Author Contributions EB participated in the design of the study, conducted the analyses and drafted the manuscript. AR participated in the design of the study, advised on the interpretation of data and reviewed the manuscript. Both authors read and approved the final manuscript.

Compliance with Ethical Standards

Conflict of interest This study was partially supported by a PhD grant awarded to the first author from the APEX Trust for Autism.

Ethical Standards All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed Consent Informed consent was obtained from all individual participants included in the study.

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