Title: Sleep-Wake Cycle and Circadian Misalignment in people with Autism Across the Lifespan with an emphasis on living conditions

Short running title: Sleep-wake cycle in people with autism.

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ACKNOWLEDGEMENTS

This project was funded by the Alicia Koplowitz grant for Research in Neurosciences (Madrid, Spain) code: UGP14-011, and Dr. Pura Ballester Navarro received a predoctoral grant from La Caixa Foundation (Barcelona, Spain) code: PT13/0001. We would like to thank to all participants, their families and the staff from the Spanish autism associations (San Rafael, EDUCATEA, APNAV, and Infanta Leonor) for their time and interest.

CONFLICT OF INTERESTS

Authors declare no conflict of interest.

Abstract

Introduction: Sleep problems among individuals with autism spectrum disorder (ASD) are a persistent issue that spans from early childhood to adulthood. The present study aimed to objectively investigate sleep continuity and alignment using ambulatory circadian monitoring (ACM) in a group of autistic individuals, with and without intellectual disabilities. **Methods:** We studied 214 participants with a diagnosis of ASD. Apart from autism, co-occurring health conditions and ongoing medications were either obtained from electronic health records (EHR; adults) or provided by parents/legal representants (children and adolescents). Sleep was assessed using a minimum of 3-days of ACM. ACM-measured sleep continuity: Total Sleep Time (TST), Latency, Number of Awakenings, duration (WASO), and alignment with SleepM5 and L10. **Results:** Participants were divided into four groups: (1) age <10 years (*n*=40, 87.5% males, M = 6.78 ± 1.40 years), (2) age 10-17 years (*n*=53, 90.6% males, M = 12.62 ± 2.04 years), (3) age 18-27 years (*n*=59, 74.6% males, M = 23.50 ± 2.60 years), and (4) age 28-65 years (*n*=62, 74.2% males, M = 39.04 ±9.49 years). Participants in groups 1 and 2, were living at home, had no ongoing medications, and 17% had intellectual disability (ID). In groups 3 and 4, all were living in residential facilities, took a median (IQR) of 4 (3–6) medications, and all had ID. All groups had significantly impaired sleep outcomes, except for TST. Adults had longer SOL and WASO duration, than children and adolescents. However, those differences were attenuated if participants 'percentages of sleep parameters within normal range' were compared. When evaluating circadian misalignment, sleep M5 is delayed in children and adolescents (2:56 am and 3:00 am, respectively), and strongly advanced in the older adults (group 4). **Conclusion:** Sleep problems that manifest in autism during childhood can endure throughout adulthood. To cope with these difficulties, adults may increase their bedtime, leading to a normal TST. However, evaluating sleep continuity should not be limited to average sleep duration alone. It is crucial to also examine the percentage of participants whose sleep falls within the normal range for age. Furthermore, there is a necessity to investigate how living conditions, such as enforced schedules in residential facilities, can influence the timing of the sleep midpoint.

Keywords: Autism spectrum disorder; Intellectual disability; Sleep problems; Age; ambulatory circadian monitoring

Introduction

Autism spectrum disorder (autism) is a neurodevelopmental condition involving persistent deficits in social communication and interaction, repetitive patterns of behavior, and restricted interests or activities (American Psychiatric Association, 2013). The prevalence of autism has been recently updated and affects as many as 1 in 36 newborns (Maenner, 2023) with an estimation of 1.97% to 2.42% adult prevalence, taking account of those who are undiagnosed (Dietz et al., 2020). People with autism[1] have frequent co-occurring conditions including sleep problems, such as insomnia-related symptoms or sleep phase misalignments, which are lifelong challenges (Carmassi et al., 2019; Jovevska et al., 2020). These demand a more comprehensive investigation from the scientific community, using combined evidence-based medical (Cortese et al., 2020), psychosocial and environmental frameworks (Schreck & Richdale, 2020).

An appropriate sleep-wake cycle across the lifespan involves both sufficient sleep (i.e., duration) and proper sleep alignment (i.e., circadian phase of the sleep-wake cycle) both of which have been reported as problematic in autism (Baker & Richdale, 2017; Elrod & Hood, 2015; Martínez-Cayuelas et al., 2022). Adequate sleep duration ranges from 10-12 hours in children, around 9-10 hours in adolescence, to at least 7 hours of sleep during adulthood (M. M. Ohayon et al., 2004), with reduced or excessive sleep associated with physical and mental health conditions (Wang et al., 2024). Sleep alignment refers to its synchronization with environmental factors such as the 24-hour light/dark cycle (LeGates et al., 2014). The sleep phase drifts to later sleep onset and wake during adolescence associated with pubertal changes; this is reverted in adulthood (Crowley et al., 2007; Roenneberg et al., 2004). A significant portion of children in the general population, ranging from 25% to 40%, experiences challenges with insomnia-related issues, such as settling and bedtime routines, and night awakenings (Ophoff et al., 2018); these struggles can persist or recur in adulthood with 33-45% of adults reporting poor sleep (Adams et al., 2017). Sleep problems reported by people with autism align with those reported by the general population, as they commonly experience

[1] While there has been a preference for identity first language in English speaking countries, a recent study reported a preference for person-first language in adults with an autism diagnosis living in Belgium and The Netherlands(De Laet et al., 2023). Thus, in this manuscript we use person-first language (i.e., person with autism)

difficulty in falling asleep, poor sleep efficiency, and struggles with sleep maintenance (Díaz-Román et al., 2018; McGovney et al., 2020; Morgan et al., 2020; Schreck & Richdale, 2020). These insomnia symptoms in individuals with autism are well-documented from early childhood (Elrod et al., 2016), persist into adolescence (E. Baker et al., 2013; S. Goldman et al., 2017; Martínez-Cayuelas et al., 2022; Øyane & Bjorvatn, 2005), and are also reported as a common complaint in adulthood (Hohn et al., 2019; Jovevska et al., 2020; Leader et al., 2021), irrespective of cognitive functioning (E. K. Baker & Richdale, 2017; Ballester et al., 2019).

Among insomnia symptoms, children with autism present with greater bedtime resistance, parasomnia and night awakenings (S. E. Goldman et al., 2012). Adolescents with autism have shorter sleep duration and more daytime sleepiness (Deliens et al., 2015; Díaz-Román et al., 2018; S. E. Goldman et al., 2011), while a meta-analytic systematic review of sleep in adults with autism described difficulties in sleep continuity (e.g., longer sleep onset latency, frequent awakenings) and lower self-perceived sleep quality, sleep efficiency, and more napping compared to non-autistic adults (Morgan et al., 2020). However, sleep phase misalignments in autism have received limited attention. Children and adolescents with autism may experience a sleep phase delay (Hodge et al., 2014; Souders et al., 2009; Wasdell et al., 2008; Wiggs & Stores, 2004), which is consistent with puberty hormonal shifts that occur in adolescence (Carskadon, 2011). Contradictory findings have emerged in adults, as those with unimpaired cognitive ability generally exhibit a delayed phase (E. K. Baker et al., 2017), whereas individuals with autism and intellectual disability living in residential facilities demonstrated a notable phase advancement in comparison to the general population (Ballester et al., 2019). The latter may be due to fixed daily living schedules in residential facilities. However, especially in adults, several other factors contribute to sleep alignment including employment, sleep routine regularity, life-style (e.g., living conditions), and light intensity and timing within the 24-hour cycle (Deserno et al., 2019; Lawson et al., 2020; Martinez-Cayuelas et al., 2022). Despite their capacity and desire to work, adults with autism have high levels of unemployment or underemployment (Frank et al., 2018). For example, only 37% of adults with autism reported being employed for more than a year in the four years following finishing school (Fong et al., 2021). Most adults with autism, regardless of their cognitive ability, struggle with independent living (Ghanouni

et al., 2021), and therefore the ability to self-select their sleep schedules according to their preferences (Duncan et al., 2021). Thus, there is a need to better understand sleep and sleep-wake circadian differences in people with autism from childhood through to adulthood.

Sleep variables are usually evaluated using questionnaires, sleep logs, or actigraphy (Moore et al., 2017). To date, there is no work that objectively investigates insomnia-related issues and sleep circadian alignment in autistic people through childhood, adolescence and adulthood. In this work we used ambulatory circadian monitoring (ACM) which measures not only sleep, but also motion, peripheral temperature and TAP (see Method), which are useful variables to ascertain sleep phase alignment (Ortiz-Tudela et al., 2010, 2016). Using ACM, which gives insomnia symptom estimations close to polysomnography (Ortiz-Tudela et al., 2014) can not only examine the sleep-wake rhythm, but also other rhythms that can affect sleep. Together with acknowledging risk factors and living conditions, ACM allows examination of the potential multifaceted causes of sleep problems in autism, as raised in biopsychosocial models of sleep (Charlton et al., 2023; Gustemps et al., 2021; Henderson et al., 2023; Richdale et al., 2023; Richdale & Schreck, 2009; Schreck & Richdale, 2020).

Therefore, the purpose of this work was to explore, cross-sectionally, sleep dimensions (e.g., duration, regularity) using ACM in children and adolescents with autism living with their parents or legal guardians, and adults with autism living in supported residential accommodation. This study is important, as to the best of our knowledge it is the first that focusses on these crucial aspects of sleep using reliable and objective metrics in individuals with autism across ages.

Methods

Ethics

The present study is a sub-analysis of two prospective observational studies UGP14-011, and PIC018_18FJD examining sleep in children, adolescents and adults with autism. The studies were conducted at Health Alicante Department- General Hospital (Alicante, Spain) and Fundación Jiménez Díaz Hospital (Madrid, Spain) from February 2015 to March 2022, excluding summer (June to – August) as weather could affect the accuracy of the temperature sensor. Both studies had Hospital Ethics Review Board approval at both sites and were performed in accordance with the principles of

the Helsinki Declaration. All participants, and/or their legal guardians received information about the design and purpose of the study. Given that this study included children, adolescents and adults with intellectual disability, we developed an information sheet that contained the study process in simplified language with images and photos which were handed, when possible, to participants. Depending on level of cognitive functioning, participants' or legal guardians' informed consent was obtained, and when possible, according to cognitive function, participants assented.

Participant recruitment and inclusion criteria.

Two hundred and fourteen individuals with an autism spectrum disorder (ASD) diagnosis were included in the study. Participants were included during clinical visits. The adult participants were residents across three institutions for adults with autism, whereas children and adolescents lived at home. For inclusion participants were aged from 4 to 65 years; gave agreement to an initial clinic visit; and had a previous autism spectrum disorder diagnosis substantiated using the Diagnostic and Statistical Manual of Mental Disorders 5th edition (DSM-5) criteria (American Psychiatric Association, 2013). Individuals taking medications for sleep including melatonin, and those with any medical condition that was incompatible with the study conditions were excluded from participation. Diagnosis based on DSM-5 criteria was confirmed by a clinician who was an experienced psychiatrist from our research team, skilled in diagnosing ASD. All children completed an ADOS assessment. Intellectual disability (ID) defined by an IQ <70 was confirmed from professional's medical records from the Spanish social services. All participants continued their regular medications, if any, during the study. Existing sleep problems were not required to participate in the study. Participants could withdraw from the study at any time. No participant was receiving a psychological therapy for their sleep.

Measurements

Ambulatory circadian monitoring

The ambulatory circadian monitoring (ACM) device measured sleep parameters and sleep alignment combining information from three sensors. The first measures peripheral temperature (Thermochron iButton DS1921H, ±1 °C accuracy, sampling every 10 min) (Carrier & Monk, 1997; Sarabia et al., 2008). The second is an actimeter that estimates physical activity using body triaxial changes of angles between the Y/Z/X-axes (90° verticality to 0° maximum horizontality) (HOBO Pendant G

Acceleration Data Logger UA- 004-64, programmed to record data every minute (Bonmati-Carrion et al., 2015; Carvalho Bos et al., 2003; Mormont et al., 2000). The third sensor is a luxometer capturing the intensity of light received (HOBO Data Logger UA-002 64, measured in 30-s intervals (Martinez-Nicolas et al., 2011)).

Van Someren and Witting non-parametric indexes and sleep parameters were calculated from the ACM loadings. The information stored in the ACM device was transferred via USB to a personal computer using the software provided by the manufacturer, through circadianware software implemented in the Kronowizard platform (https://kronowizard.um.es/). It calculates a single integrated variable, TAP, integrating peripheral temperature (inverted), motor activity and body position. TAP maximum values should occur at the same time of the day indicating a high level of activation (values near 1) or complete rest and sleep (around 0). Sleep was reversely inferred from TAP and converted into a binary code, with 1 corresponding to a resting period and 0 to an active period (Ortiz-Tudela et al., 2014).

Ambulatory circadian monitoring: Sleep parameters determination

Sleep continuity parameters were calculated as (a) total sleep time (TST), defined as sleep minutes between sleep onset and sleep offset; (b) time in bed (TIB), total minutes in bed from bedtime until rising time; (c) sleep onset latency (SoL) minutes from bedtime until sleep onset; (d) number of awakenings after sleep onset; (e) awake period duration after sleep onset duration (WASO), the awake period length in minutes during the TST interval; and (f) sleep efficiency (SE), calculated as the ratio of TST/TIB multiplied by 100. The results of sleep parameters from all participants were classified as normal or abnormal values according to age, and ranges previously described for the general population (Natale et al., 2009; M. Ohayon et al., 2017).

Ambulatory circadian monitoring: Circadian sleep–wake rhythm disorders determination

Non-parametric circadian rhythm analysis is a method for extracting circadian characteristics from the rest-activity cycle (Ortiz-Tudela et al., 2014; Van Someren & Riemersma-Van Der Lek, 2007; Weitzman et al., 1981; Witting et al., 1990). Of major interest is the relative amplitude (RA), as it shows how activity is distributed throughout the day compared with night: the higher the RA, the better the consolidation of daytime activity and nighttime sleep. The RA is calculated from the ratio of the most active 10-hour period (M10) to the least active 5-hour period (L5) across the averaged 24-

hour profile. A second characteristic is the inter-daily stability (IS), which quantifies invariability day by day, that is, how well the sleep–wake cycle is synchronized to supposedly stable environmental cues. Third, intra-day variability (IV) gives an indication of the fragmentation of the rhythm. Timing and value (V) information comes from determining the onset of the 5 hours with least activity (L5 onset) and onset of the 10 hours with most activity (M10 onset), sleep and peripheral temperature rhythms are reverse, measuring M5 and L10. Finally, the circadian function index (CFI), which assesses circadian rhythmicity status, was calculated as (IS+(2-IV) + RA)/3. CFI has proved to be very sensitive to changes in circadian robustness (Ortiz-Tudela et al., 2010).

Procedure

Demographic information (age, biological sex) was obtained from EHRs. Since we wanted to study the evolution of sleep-wake cycle across the lifespan, groups were established according to age and living conditions. As Tanner stage was unknown, children were classified as those <10 years; and adolescents were 10-17 years. Adults were split into two age groups according to our previous research results (Ballester et al., 2019), as the present study aimed to explore when the previously reported phase advancement in sleep-wake cycle appears in adults with autism living in supported accommodation.

Sleep parameters and alignment were obtained using ACM (Kronowise). All participants were asked to follow their usual routines and wore the ACM device on the wrist of their non-dominant arm for 7 days. The device was removed during showering or any other activity where it could get wet; data were collated to remove erroneous measurements produced by its temporary removal. All participants completed a 7-day, sleep–wake diary (mornings and evenings). A parent or caregiver was responsible for completing each participant's sleep diary. Sleep diaries were used as a backup for the device recordings if needed.

Statistical analyses

The Shapiro–Wilks normality test was used as the basis for selection of parametric or nonparametric statistical tests. Continuous variables are presented as mean ± standard deviation (SD), mean ± standard error (SEM) or median and interquartile range (IQR, P25, P75). Categorical variables are expressed as percentages. A Kruskal-Wallis test was used to assess group differences. Frequencies were compared using the Chi-Square test, with Yate's continuity correction as

appropriate and X^2 were reported. Despite the unequal sex distribution observed between the autism age groups, differences in sleep parameters were analyzed by sex to ensure a lack of bias. All statistical analyses were carried out with IBM SPSS Statistics 29.0.0 and R 4.3.3 software; p-values < 0.05 were considered to indicate significance for all analyses.

Results

A total of 214 participants, aged 4 to 58 years were included in the study. The sample was predominantly male (80.8%), however, no significant differences among sleep parameter values were found when compared by sex intra-group, or inter-groups (all p-values > 0.05). Thus, given the small number of females in each group we did not use sex as a covariant. All participants were recruited after researchers met with their parents at institutions for those diagnosed with autism (56.5%) or during clinical visits to one of the participating hospitals (43.5%). Participants were placed into one of four age groups: (1) < 10 years (n = 40), (2) 10-17 years (n = 53), (3) 18-27 years (n = 59), and (4) 28-65 years (n = 62) (Table 1). According to their EHRs, children and adolescents did not have any co-occurring conditions apart from ID (17%) and were medication naïve. According to EHRs, all adults had ID and 33% had a diagnosis of epilepsy. Adult participants had a median (IQR) of 4 (3–6) medications (50% antipsychotics, 47% with > 1 antipsychotic simultaneously; 22% anticonvulsants, 16% anxiolytics, and 12% antidepressants).

Ambulatory circadian monitoring: Sleep parameters

Comparison of ACM sleep parameter data across the four age groups is presented in Table 2. From all sleep parameters in both adult groups, only the number of awakenings reduced with an ongoing anxiolytic medication ($p < 0.05$); other medicines that produce sedation as a side-effect did not affect sleep parameters in this study. However, as can be seen in Table 2, the average number of awakenings is high in all groups regardless of medication status. Significant differences among the four age groups were found for TIB, SoL, number of awakenings, WASO and SE. Sleep duration was not significantly different across the groups. However, that may be due to the greater opportunity for sleep that the two adult groups in residential care have, based on their TIB. Sleep onset latency and WASO were significantly impaired in the adults, and consequently SE. When calculating the percentage of participants exhibiting sleep variable parameters aligned with age recommendations for

each parameter within each group, it becomes clear that symptoms of insomnia persist consistently throughout the lifespan. Intra-group ranges were set for TST according to participants' age in the youngest age group (Group 1).

Ambulatory circadian monitoring: Circadian sleep–wake rhythm disorders

Analysis of the circadian rhythms (wrist temperature, motor activity, TAP, sleep, and light intensity) showed significant differences between groups (Table 3). The two younger groups with autism (children and adolescents) displayed significantly higher IS values, for all rhythms except for light intensity where no differences were found. In the adult age groups, motor activity was more fragmented according to IV values. The adult groups also presented a wider RA of light intensity, and narrower sleep amplitude. The lack of amplitude in the sleep rhythm at night, may be compensated by a significantly greater value of sleep VL10 that indicates either napping or daytime somnolence.

The children and adolescents exhibited a phase delay according to phase markers for wrist temperature, motor activity, sleep, light intensity, and TAP. A phase advancement in the adults with ID living in residential facilities, especially in the oldest age group (see figure 1) was also found. When examining how living conditions may cause a shift towards an advanced phase, despite all groups receiving the same light intensity (light intensity VM10), there were timing differences. On average, the oldest group was exposed to either light or darkness 40 minutes earlier than other age groups (Figure 2).

Discussion

To the best of our knowledge, the present study gathers the largest sample of participants with autism whose sleep has been assessed using an objective measure such as ACM. This study represents a cross-sectional, natural life trajectory for people with an ASD diagnosis, including those under 18 years and living at home, young adults transitioning into adulthood who were living in residential facilities for adults with autism, and older adults who had been in residential care for at least 5 years. Regardless of age, all groups had significantly impaired sleep outcomes. When evaluating circadian misalignment, children and adolescents showed a sleep phase delay. However, adults had a progressive phase advancement of the sleep midpoint, indicating a phase advance and suggesting that their living conditions are responsible for this sleep phase shift in adulthood, as these are largely determined by their residential institution. Children and adolescents were medication-free with no

other reported co-occurring conditions except ID which was present for 17% of children. Conversely, the adults with autism all had co-occurring ID and took multiple ongoing medications, which is dissimilar to studies of adults with autism and no co-occurring ID (E. K. Baker & Richdale, 2015; Cvejic et al., 2018; Esler et al., 2019). Nevertheless, medication side-effects only impacted the average number of nights awakenings; there were no significant associations with other sleep or circadian parameters.

Regarding sleep durability variables, there were no significant age-group differences for TST which was unexpected. While many individuals slept significantly fewer hours than recommended for their age, particularly in the youngest age group where the group average TST was lower than expected for age, the adult group averages were longer than might be expected (Hirshkowitz et al., 2015). The lack of differentiation it total sleep with age is a is key finding, as developmentally, total sleep decreases across childhood and into adulthood, decreasing again in old age (Buysse et al., 1992; Hirshkowitz et al., 2015; Yoon et al., 2003). A recent meta-analysis found that autistic adults, most of whom did not have ID, slept less than comparisons samples (Morgan et al., 2020) and reviews report that autistic children also sleep less than typically developing children (e.g., (Hodge et al., 2014)). Adolescents and adults with autism and no ID are reported to sleep about seven hours on average (E. K. Baker & Richdale, 2015; Jovevska et al., 2020), which is less than the current sample, with adults in middle age have the least sleep of about six hours (Jovevska et al., 2020). While the adults in the current study had an ID, they spent a longer time in bed and therefore had more opportunities to achieve more sleep at night which most likely accounts for their tendency to increased TST. Other factors that may contribute to differences in adults' TST across studies are employment status, environmental factors associated with residential care including timing of meals, recreation, bedtime and rising time (Cohen et al., 2018; Lawson et al., 2020).

Adults with autism had longer SoL and WASO than the younger age groups. However, the percentage of individuals with values within the normal range was extremely low across all age groups, which is consistent with previous research in children with autism (Elrod & Hood, 2015) and adults (Morgan et al., 2020). Increased SoL and WASO may be associated with the higher rates of anxiety reported for individuals with autism from childhood to adolescence (Haugland et al., 2021), with abnormal values of insomnia related issues that oscillate from 28% to 77% in childhood (Vasa &

Mazurek, 2015) and persist into adulthood (Lever & Geurts, 2016; Tani et al., 2012). Researchers have speculated that high rates of anxiety may indicate increased cognitive and/or somatic hyperarousal which impacts sleep (E. K. Baker, Richdale, Hazi, et al., 2019; Mazurek et al., 2019; Mazurek & Petroski, 2015). Indeed, Baker found that higher subjective somatic arousal was associated with increased SoL and poorer sleep efficiency among participants with autism, those unmedicated participants had greater reductions in evening cortisol levels, suggesting a hyperarousal hypothesis of insomnia in adults with autism (E. K. Baker, Richdale, Hazi, et al., 2019).

According to the phase markers, the 24-hour temperature rhythm was advanced in both adult groups, and in general, higher values of peripheral temperature were found in the adult groups during both day and night. This higher peripheral temperature can be explained by an impaired noradrenergic signaling that interferes with the body's capacity to cool (Wallengren, 1997), and it can be altered by selected medications that some adults in our study took (Fazekas et al., 2021). Also, another factor that influences heat loss is the light intensity experienced, as this has the potential to promote an earlier melatonin onset followed by the peripheral heat loss prior bedtime (Benedetti et al., 2022). This peripheral temperature advancement could be a *by proxy* measure of an aberrant function in the melatonin pathway as reported previously in this population (Tordjman et al., 2018).

The motor activity phase markers represent, in general, a high level of sedentariness across the sample that increases with age. Adolescents had a level of activity which was consistent with a previous report (Liang et al., 2020). The adolescents' rates of movement were like those in the oldest age group. This could indicate that adolescents are more involved in educational/therapeutical activities, or screen time than in sport or active leisure activities during breaks and after school hours, compared to typically developing peers (Berard et al., 2022).

Sleep worsened with age in our population, with decreased stability, and increased fragmentation, a narrower relative amplitude of the rhythm, and a progressively more advanced sleep midpoint. Circadian sleep-wake disorders have been described in and related with unemployment in adults with autism without cognitive impairment (E. K. Baker et al., 2017; E. K. Baker, Richdale, & Hazi, 2019). The circadian rhythms and sleep-wake disorders in our sample of adults are more like those reported in aged populations with mild cognitive impairment (Gao et al., 2022), or the severely ill (J Madrid-Navarro et al., 2015). In our young adult group this phenomenon may be explained by

factors such as alterations in the melatonin pathway (Díaz-Román et al., 2018), or environmental factors such as reduced light exposure (Siraji et al., 2023). While all age groups were regularly exposed to light and receiving the same amount of light intensity during the day, adults were receiving light 40 minutes earlier in the day than children and adolescents. A shift towards morning outdoors time with natural light exposure can result in a phase advancement (Siraji et al., 2023).

The main strength of this study is that it uses large sample of people with autism across the lifespan who have all had their sleep, as well as temperature and motor movements recorded objectively using ACM. The sample size for both children and adults exceed previous published work using actimetry for children and adolescents (Miner et al., 2023; Wiggs & Stores, 2004) and adults (E. K. Baker & Richdale, 2015; Benson et al., 2019) with autism, giving us confidence in our results. The sex distribution in our sample is like other publications (e.g., (Yavuz-Kodat et al., 2019)). However, our small number of females did not allow us to replicate a recent finding that women with autism have poorer sleep outcomes (Jovevska et al., 2020). Other limitations include that our age groups differed in terms of medication and co-occurring conditions, however, this was examined in relation to sleep parameters and few differences were found. Also, only 17% of children and adolescents had ID, but all adults had ID, but recent publications suggest that ID is more strongly associated with sleep satisfaction, a dimension of sleep not assessed in the present study, rather than functional impairments (Gilbertson et al., 2021). Differences in our participants' living conditions potentially played a role in their sleep outcomes, as children and adolescents lived at home while our adults were in a supported residential setting. Thus, it will be important for future research to examine sleep in adults with autism in different living conditions to begin to tease out the effects of environment on their sleep. Further studies designed as clinical trials also need to explore the effects of shifting light exposure and reducing TIB or sleep opportunities have for sleep and circadian parameters for adults with autism, with and without ID.

In conclusion, the present work indicates that sleep problems that appear during childhood in autism persist into adulthood, including abnormal sleep onset latency and wake after sleep onset, and reduced total sleep among children. Variation in total sleep reported across adults may be due to compensation mechanisms such as increased opportunity to sleep, due to a greater time in bed. Sleep parameters should not only be assessed by average times, but by also examining the proportion of

participants who fall within normal sleep values adjusted by age, and by examining individual differences (e.g., ongoing medication). Sedentarism and light exposure in people with autism need to receive further attention, together with how living conditions, such as imposed schedules in residential facilities, shift the sleep midpoint. The consistent and increasing reports of poor sleep among individuals with autism over the last forty years (e.g., see (Hoshino et al., 1984)) indicates a need for further studies of sleep trajectories longitudinally in this population to improve our understanding of causal and maintenance factors, and to target prevention and intervention strategies.

Table 1. *Participant Demographics for each age group: 1 (between 0-9 years old), 2 (10-17 years old), 3 (18-27 years old) and 4 (28-65 years old).*

Age groups	**0-9 years old** (*n* = 40)	**10-17 years old** (*n* = 53)	**18-27 years old** (*n* = 59)	**28-65 years old** (*n* = 62)
Sex (n, % male)	35 (87.5%)	48 (90.6%)	44 (74.6%)	46 (74.2%)
Age (mean ± SD)	6.90 ± 1.40	12.77 ± 2.04	23.64 ± 2.60	40.11 ± 9.49

Table 2. *Sleep parameters comparison obtained from ambulatory circadian monitoring (ACM) recordings between the four participants groups 1 (between 0-9 years old), 2 (10-17 years old), 3 (18-27 years old) and 4 (28-65 years old).*

Sleep parameters	**0-9 years old** (*n* = 40)	**10-17 years old** (*n* = 53)	**18-27 years old** (*n* = 59)	**28-65 years old** (*n* = 62)	***p*-value**
Total sleep time (TST, minutes)	520 ± 49	510 ± 52	526 ± 138	525 ± 108	.427
% (*n*) Normal range	42.5 (17)	75.5 (40)	35.8 (19)	36.0 (50)	**< .001**
Time in bed (TIB, minutes)	576 ± 72	567 ± 50	683 ± 119	685 ± 91	**< .001**
% Normal range, *n*/*N*	2.5 (1)	0	0	0	.566
Sleep onset latency (SOL, minutes)	29 ± 20	32 ± 19	52 ± 34	43 ± 23	**< .001**
% (*n*) Normal range	25 (10)	54.7 (29)	26.41 (14)	32.0 (16)	**< .001**
Number of awakenings (n)	3.7 ± 0.9	3.8 ± 1.3	3.3 ± 1.9	3.2 ± 1.9	**.002**
% (*n*) Normal range	0	1.9 (1)	17.0 (9)	12.0 (6)	**< .001**
Wakes after sleep onset (WASO, minutes)	28 ± 18	24 ± 12	81 ± 56	93 ± 76	**< .001**
% (*n*) Normal range	2.5 (1)	5.7 (3)	9.4 (5)	6.0 (3)	.363
Sleep efficiency (SE %)	89 ± 5	86 ± 6	76 ± 13	76 ± 13	**< .001**

% (*n*) Normal range	12.5 (5)	66.0 (35)	26.4 (14)	28 (14)	**< .001**

Sleep parameters derived from ACM recordings in participants expressed as mean ± SD. Kruskal Wallis (sleep parameter values) or Chi Square (percentages of normality) tests $p < 0.05$ are highlighted in bold. The *p*-value is the overall comparison between the four groups.

Table 3. *Comparison among all non-parametric circadian rhythms along 24-hour period between the four participants groups 1 (between 0-9 years old), 2 (10-17 years old), 3 (18-27 years old) and 4 (28-65 years old).*

Groups	**0-9 years old** (n = 40)	**10-17 years old** (n = 53)	**18-27 years old** (n = 59)	**28-65 years old** (n = 62)	***p*-value**
			Wrist Temperature		
IS	0.49 ± 0.19	0.50 ± 0.18	0.39 ± 0.24	0.47 ± 0.22	.101
IV	0.01 ± 0.04	0.02 ± 0.05	0.13 ± 0.08	0.14 ± 0.08	**< .001**
RA	0.41 ± 0.14	0.44 ± 0.19	0.30 ± 0.17	0.33 ± 0.18	**< .001**
M5	2:42 ± 3:22	3:01 ± 2:47	2:08 ± 2:10	1:54 ± 3:41	**.024**
VM5	33.65 ± 0.89	33.55 ± 0.77	34.32 ± 1.24	34.78 ± 1.22	**< .001**
L10	16:06 ± 2:44	16:45 ± 4:39	14:01:49 ± 2:27	14:18 ± 1:53	**< .001**
VL10	31.53 ± 0.89	31.30 ± 1.14	32.43 ± 1.17	32.75 ± 1.19	**< .001**
CFI	0.61 ± 0.11	0.63 ± 0.12	0.53 ± 0.16	0.52 ± 0.13	**< .001**

			Motor Activity		
IS	0.36 ± 0.14	0.30 ± 0.11	0.31 ± 0.15	0.38 ± 0.20	**.009**
IV	0.45 ± 0.18	0.45 ± 0.24	0.98 ± 0.16	1.01 ± 0.20	**< .001**
RA	0.71 ± 0.18	0.54 ± 0.19	0.79 ± 0.15	0.74 ± 0.19	**< .001**
M10	15:11 ± 1:15	14:58 ± 1:57	15:00 ± 2:10	15:06 ± 2:28	.883
VM10	31.71 ± 8.53	23.91 ± 9.35	29.67 ± 13.30	24.16 ± 9.40	**< .001**
L5	3:07 ± 1:23	3:27 ± 1:27	2:31 ± 1:56	2:19 ± 2:15	**.005**
VL5	2.31 ± 1.29	2.18 ± 0.61	4.20 ± 4.53	4.06 ± 4.27	.102
CFI	0.67 ± 0.05	0.63 ± 0.06	0.54 ± 0.09	0.54 ± 0.12	**< .001**
			TAP		
IS	0.74 ± 0.10	0.64 ± 0.15	0.49 ± 0.22	0.60 ± 0.21	**< .001**
IV	0.16 ± 0.09	0.18 ± 0.07	0.36 ± 0.24	0.38 ± 0.23	**< .001**
RA	0.70 ± 0.12	0.63 ± 0.13	0.53 ± 0.19	0.55 ± 0.18	**< .001**

M10	14:33 ± 1:20	15:28 ± 3:24	14:59 ± 1:24	14:35 ± 1:48	.268
VM10	0.53 ± 0.07	0.49 ± 0.08	0.57 ± 0.11	0.57 ± 0.12	**< .001**
L5	2:49 ± 1:40	3:26 ± 1:41	2:34 ± 1:38	1:42 ± 1:39	**< .001**
VL5	0.10 ± 0.04	0.11 ± 0.03	0.19 ± 0.12	0.17 ± 0.09	**< .001**
CFI	0.54 ± 0.06	0.51 ± 0.09	0.61 ± 0.13	0.65 ± 0.12	**< .001**
			Sleep		
IS	0.80 ± 0.08	0.73 ± 0.15	0.57 ± 0.23	0.64 ± 0.24	**< .001**
IV	0.13 ± 0.09	0.12 ± 0.07	0.33 ± 0.14	0.36 ± 0.17	**< .001**
RA	0.94 ± 0.06	0.90 ± 0.13	0.78 ± 0.26	0.82 ± 0.23	**.004**
M5	2:58 ± 1:25	3:02 ± 1:20	2:27 ± 1:50	1:58 ± 1:39	**< .001**
VM5	0.93 ± 0.07	0.91 ± 0.09	0.83 ± 0.19	0.82 ± 0.21	**.012**
L10	14:42 ± 1:34	14:59 ± 1:32	14:18 ± 2:50	13:38 ± 1:16	**.005**
VL10	0.01 ± 0.02	0.03 ± 0.12	0.12 ± 0.18	0.12 ± 0.21	**< .001**

CFI	0.91 ± 0.04	0.89 ± 0.08	0.73 ± 0.14	0.75 ± 0.17	**< .001**
			Light Intensity		
IS	0.62 ± 0.12	0.57 ± 0.15	0.60 ± 0.19	0.59 ± 0.34	.214
IV	0.08 ± 0.05	0.07 ± 0.04	0.17 ± 0.10	0.18 ± 0.13	**< .001**
RA	0.57 ± 0.16	0.53 ± 0.21	0.99 ± 0.01	0.95 ± 0.19	**< .001**
M10	14:25 ± 00:45	14:33 ± 1:14	14:05 ± 00:50	13:33 ± 00:57	**< .001**
VM10	1.61 ± 0.42	1.56 ± 0.61	1.61 ± 0.64	1.34 ± 0.62	.057
L5	2:21 ± 1:18	2:49 ± 1:16	1:39 ± 1:35	1:48 ± 3:18	**< .001**
VL5	0.01 ± 0.04	0.003 ± 0.004	0.003 ± 0.01	0.03 ± 0.09	**< .001**
CFI	0.85 ± 0.05	0.83 ± 0.07	0.84 ± 0.07	0.79 ± 0.17	**.037**

IS:inter-daily stability; IV: intra-daily variability; RA: relative amplitude. Phase markers: maximum (M5 and M10) and minimum (L5 and L10) values indicate consecutive 10- and 5- hour period; CFI: circadian function index. Non-parametric circadian function indices derived from ACM recordings in participants expressed as mean ± SD. Kruskal Wallis tests were performed, when $p < 0.05$ are highlighted in bold. The p-value is the overall comparison between the four groups.

Figure 1. Averaged 24h wave of the sleep-wake rhythm at the four groups 1 (between 0-9 years old), 2 (10-17 years old), 3 (18-27 years old), and 4 (28-65 years old).

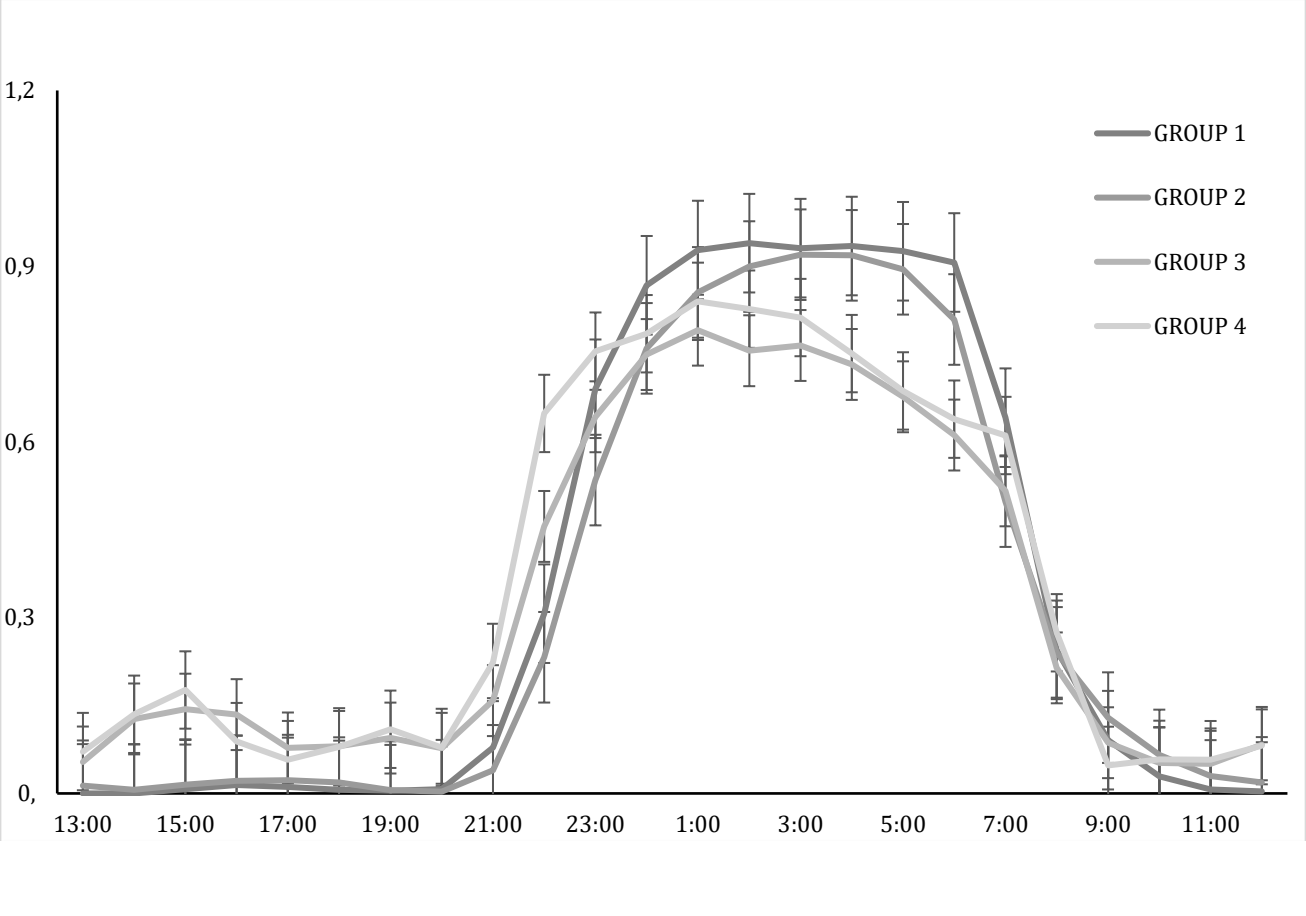
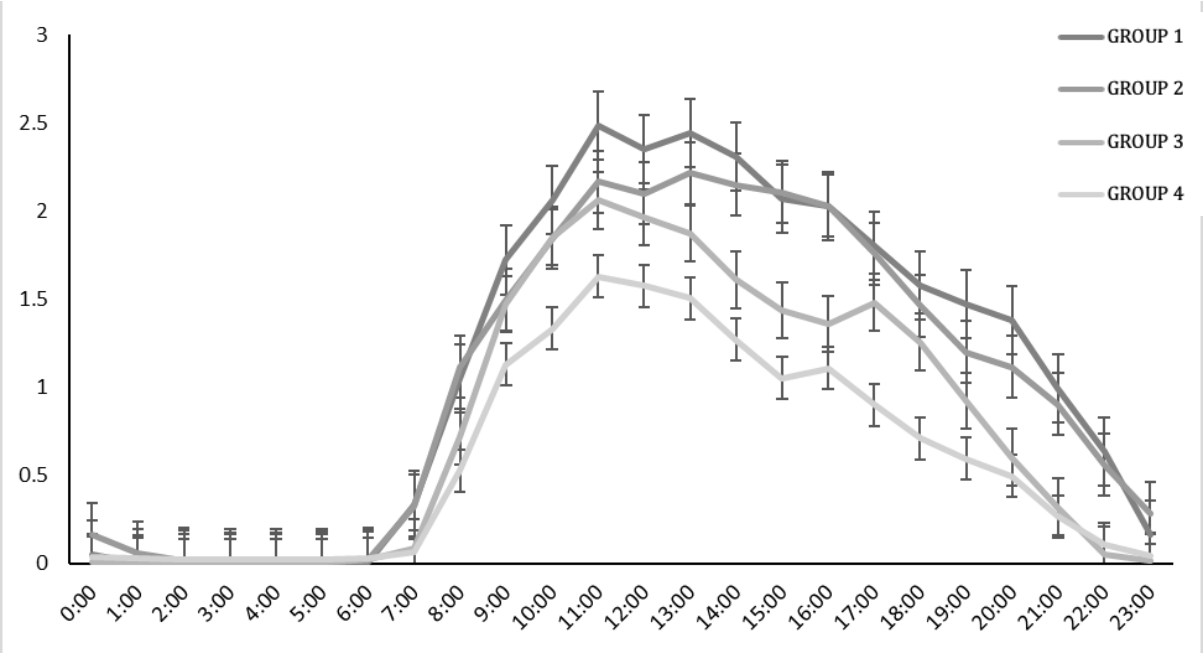


Figure 2. Averaged 24h wave of the light intensity exposure rhythm at the four groups 1 (between 0-9 years old), 2 (10-17 years old), 3 (18-27 years old) and 4 (28-65 years old).



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