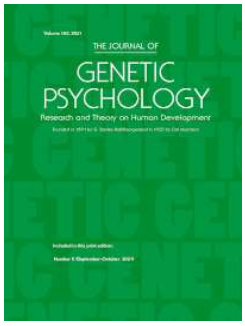

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Sleep Dysregulation and Daytime Electrodermal Patterns in Children With Autism: A Descriptive Study

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ABSTRACT

Sleep deficiency influences emotion and behavior regulation but the mechanisms of influence are poorly understood. Emotion, behavioral, and sleep theories highlight differences in autonomic function as a potential pathway of influence and research in typical populations draw links between sleep deficiency and autonomic dysregulation (e.g., elevated reactivity within the sympathetic nervous system). In populations at elevated risk for sleep deficiency/problems (i.e., individuals with autism), greater variability in sleep and autonomic/arousal profiles may be particularly informative. Using electrodermal activity (EDA) as an indicator of sympathetic nervous system activation, this descriptive pilot study aimed to document daytime EDA patterns in children with autism and to explore their relations with sleep dysregulation/deficiency. EDA and sleep were measured using ankle and wrist worn sensors in 13 children ($Mean_{age}$ 6.11 years). EDA indices included nonspecific skin conductance responses (NSSCR) and tonic skin conductance levels (SCL). Descriptively, children in the dysregulated sleep group had fewer NSSCRs and lower SCL in the afternoon. This blunted physiological arousal profile/pattern is consistent with previous research, but this is the first study to explore how sleep may be linked. Notably, this pattern may not reflect sleep but an overall dysregulation profile which in this sample included: dysregulated sleep, a blunted afternoon arousal profile, and elevated ASD symptom severity. Replication with larger, more diverse samples is needed to disentangle the complex relations among sleep, arousal, and ASD behavioral features. However, this study represents an important first step in documenting extended daytime arousal patterns.

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Autism; electrodermal activity; sleep; treatment

Introduction

Up to 80% of children with an autism spectrum disorder (ASD) experience sleep dysregulation (Carmassi et al., 2019; Reynolds & Malow, 2011), compared to 25 – 40% of their typically developing peers (Mindell & Owens, 2003). Although sleep problems in children with ASD are associated with a cascade of emotional and behavioral challenges, the mechanisms of how sleep may influence daytime behavior is not well understood. Behavioral theories (e.g., Pressman & Fry, 1989; Shinar, Akselrod, Dagan, & Baharav, 2006), sleep theories (e.g., Trinder et al., 2001), and previous studies (e.g., Zhong et al., 2005) highlight autonomic dysfunction (i.e., increased sympathetic drive and physiological arousal as indexed by heart rate, cortisol, or skin conductance) as a potential pathway of influence. Specifically, the two-process model of sleep (Borbély, Daan, Wirz-Justice, & Deboer, 2016) draws attention to the circadian elements of activity and physiological

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arousal when considering sleep regulation and similarly theories of circadian rhythms (e.g., Carpenter & Grossberg, 1985) and highlight how cycles are present in our daytime behaviors and physiological arousal patterns. Building on these theories, this exploratory, descriptive study provides a critical step in understanding how sleep/circadian patterns and autonomic functions (e.g., physiological arousal) in ASD may be linked.

Sleep and EDA

The restorative theory of sleep posits that sleep is a period of recovery for the body and serves to restore resources (e.g., energy) depleted during the day (Adam & Oswald, 1977; Shapiro, 1982). This includes restoration of the body tissues (i.e., muscle repair), protein synthesis, and nervous system. Restoration of the nervous system is reflected by the increased activation of the parasympathetic nervous system (i.e., rest and digest) and the moderation of our sympathetic nervous system (i.e., fight or flight response). Specifically, sleep provides a relief to an active and sustained sympathetic branch of the autonomic nervous system, decreasing sympathetic tone while increasing parasympathetic vagal tone to maintain internal homeostasis (Trinder et al., 2001; Zoccoli & Amici, 2020). Studies on individuals with sleep problems such as chronic sleep deprivation, sleep apnea, insomnia, and cardiovascular-related sleep problems have demonstrated elevated sympathetic activity, suggesting impaired autonomic functioning and a failure to recover (e.g., Zhong et al., 2005). Relations between sleep problems and autonomic functioning have been documented using common and reliable physiological measures such as heart rate variability (Stein & Pu, 2012; Trinder et al., 2001) and electrodermal activity (EDA; Oliver, Baldwin, & Datta, 2020).

EDA is a continuous measure of the changes in electrical conductance of the skin due to fluctuations in eccrine sweat glands which are controlled by the sympathetic nervous system (SNS; Boucsein, 2012). Henceforth, we use the broader term 'physiological arousal' to describe general autonomic arousal reflective of physiological responses (as opposed to stimulus-induced or behavioral arousal); specifically, we use the term to describe changes of the SNS as indexed by EDA (Boucsein, 2012). Using signal processing, EDA is often interpreted using tonic and phasic indices (Dawson, Schell, Filion, & Berntson, 2007). Tonic skin conductance level (SCL) measures gradual and continuous changes in EDA over time. Nonspecific skin conductance responses (NSSCRs) are a phasic index of EDA marked by discrete increases of more than .01-.05 microsiemens (μS) in skin conductance in the absence of a known stimuli. NSSCRs can be quantified in several ways including frequency over a period of time or by NSSCR average amplitude. EDA is measured non-invasively by placing sensors on the surface of the skin and can be influenced by factors other than the SNS such as environmental temperature, breathing, and movement (Boucsein, 2012).

Specific to sleep/circadian behaviors, altered tonic SCL levels are associated with poor sleep quality, increased sleep latency, or short sleep durations in undergraduate students (Oliver et al., 2020) and typically developing children (El-Sheikh & Arsiwalla, 2011). Likewise, Liu, Verhulst, Massar, and Chee (2015) and Miro, Cano-Lozano, and Buela-Casal (2002) reported elevated tonic SCL following sleep deprivation. Additionally, Kim, Ku, Bae, Han, and Kim (2018) reported a circadian rhythm in tonic SCL of healthy adults; wherein, the highest average SCLs were reported at roughly 7:00 followed by a gradual drop throughout the day until about 17:00 and then a gradual climb throughout the night and early morning hours. When considering phasic elements of EDA, McCarthy and Waters (1997) reported a reduced electrodermal-orienting response (a phasic EDA index of attention) in sleep-deprived adults. However, there are no studies to date that have examined daytime EDA (tonic or phasic) in relation to sleep patterns in children with ASD, a population at elevated risk for sleep dysregulation.

EDA in ASD

To study physiological arousal in individuals with and without ASD, both tonic and phasic elements of EDA have been utilized (e.g., O'Haire, McKenzie, Beck, & Slaughter, 2015; Schoen, Miller, Brett-Green, & Hepburn, 2008). However, comparisons between children with ASD and typically developing peers on measures of EDA have produced mixed results (Eilam-Stock et al., 2014; Kushki et al., 2013; McCormick et al., 2014; Prince et al., 2017). These variations could reflect differences in age, gender, sensor placement/characteristics, paradigm specifics, and data post-processing protocols (Boucsein, 2012; Kleckner et al., 2018; Venables & Mitchell, 1996). In the context of ASD specifically, the variability in EDA measurements may reflect different physiological arousal patterns/profiles, which can go undetected in large group comparisons (Panju, Brian, Dupuis, Anagnostou, & Kushki, 2015). Most studies measuring EDA have examined responses to tightly-controlled stimuli within lab settings across short assessments. Until recently, few studies have assessed patterns of physiological arousal across the day or within naturalistic contexts.

In children with autism, we could only identify one study that has previously assessed the associations between sleep and elements of EDA. Reynolds, Lane, and Thacker (2012) assessed parental concerns on their child's sleep using the Child Behavioral Checklist and reported no association between poor sleep and tonic SCL, but did note that poor sleep was associated with elevated phasic EDA responses to a specific sensory stimuli. Taken together, research on sleep and EDA in typically developing people and people with disordered sleep or ASD, highlights the potential connections between sleep and daytime autonomic function. The present study aims to move the field forward by providing the first 'in the wild' extended recordings of EDA and sleep for children with ASD. Within the context of sleep, understanding these extended patterns could be particularly informative given that sleep is a malleable biosocial process and could be a therapy target to improve daytime arousal profiles (if clear patterns/associations can be identified).

Method

Participants and procedure

All procedures were reviewed and approved by the Institutional Review Board at Purdue University. Participants included 13 children ($Mean_{age} = 6.11$, $SD = 2.04$) drawn from a larger study examining sleep and challenging behaviors in children with ASD ($N = 39$) (Abel et al., 2018). Children were recruited from five behavioral treatment centers and study personnel completed an enrollment visit at either the child's home or behavioral treatment center. During the enrollment visit, parents/caregivers completed a series of questionnaires on family demographics, medical history, the child's ASD symptoms, and finally, his/her adaptive functioning.

As part of enrollment criteria, all children had an ASD diagnosis (i.e., a medical and/or an educational diagnosis) and had a score on the Social Communication Questionnaire (SCQ; Rutter, Bailey, & Lord, 2003) above the autism threshold (> 11). Each child was attending a center-based applied behavior analysis (ABA) program for at least 20 hours per week and received one-on-one intervention services with a trained ABA therapist (programs were written and supervised by a board certified behavior analyst) between 08:00 and 17:00, for up to 9 hours per treatment day, five days per week. In the subsample utilized for this study, five children had co-occurring conditions such as Attention-Deficit/Hyperactivity Disorder and anxiety. Additionally, six children were taking medications to help them sleep (e.g., melatonin; details in Table 1). None of the children were asked to stop taking their medication during the study, as the primary goal of this study was to assess the child's sleep patterns in the context of their typical weekly routine and treatment program.

Table 1. Demographic information on regulated and dysregulated sleep groups.

	Range	Regulated sleep group ($n = 6$) *M(SD)*	Dysregulated sleep group ($n = 7$) *M(SD)*	F^b
Maternal education (years)	10.00 – 18.00	13.5 (2.25)	13.14 (1.95)	0.09
Age of child (years)	3.08 – 9.58	4.46 (1.28)	7.53 (1.35)	17.57**
Gender (% male)		80%	63%	0.96
SCQ Scores	11.00 – 31.00	17.6 (4.28)	22.5 (6.35)	2.57
Use of sleep medications		15.4%	30.8%	.611
Use of SSRIs/ antipsychotics		15.4%	30.8%	.611
Presence of co-occurring conditions		15.4%	23.1%	.106
Sleep measures (hours)				
Duration of sleep	5.05 – 9.57	8.63 (0.73)	6.84 (1.27)	9.20**
Wake after sleep onset	0.33 – 3.35	0.84 (0.42)	2.15 (0.56)	21.80***
Duration of longest waking	0.27 – 2.17	0.54 (0.29)	1.33 (0.46)	14.66**
Variation in onset	–	1.57 (1.92)	4.16 (4.54)	1.67
Variation in offset	–	1.60 (1.63)	3.10 (1.56)	2.84
Number of nights with wakings of more than 1 hour	–	1.67(1.21)	3.43(0.96)	8.46*
Number of wake episodes	1.4 – 5.8	2.43(0.95)	3.43(1.40)	2.17
VABS-II				
Standard scores[a]				
Communication	43 – 95	71.33 (14.90)	64.00 (18.66)	0.48
Daily living	50 – 79	70.50 (7.34)	63.50 (10.34)	2.08
Socialization	53 – 81	71.33 (6.50)	63.50 (11.00)	2.05
Motor	54 – 91	70.17 (13.99)	65.50 (9.40)	0.34

Note. SCQ = Social Communication Questionnaire. SSRIs = Selective serotonin reuptake inhibitors. VABS-II = Vineland Adaptive Behavior Scale – Second Edition.
[a]Only $n = 6$ and $n = 4$ were included for the regulated and dysregulated sleep groups respectively.
[b]Degrees of freedom (between groups)= 1.
*$p < .05$.
**$p < .01$.
***$p < .001$.

Sleep and EDA data were collected during the same treatment week, where EDA was collected in the context of the child's typical treatment day. A sensor-trained graduate research assistant provided training to each ABA therapist on sensor placement and was present whenever possible during placement and removal. Each night, study personnel visited each center to download the EDA data and charge each device for the following day. Child sensor usage was based on sensor availability at the time of their sleep recording week. Sleep was measured using actigraphy and collected for five full 24-hour periods in conjunction with the EDA measurement week. Caregivers were taught how to remove and replace their child's actigraph ankle band at home. Using actigraphy data children were categorized into two groups, those with regulated and dysregulated sleep.

From the original sample, 13 children had at least 180 consecutive minutes of EDA data, 17 children had partial data or fewer than 180 consecutive minutes, and 9 children were not enrolled in the EDA portion of the study based on equipment availability. Given the questions of interest, only children ($n = 13$) with the highest caliber data and EDA recordings of at least 180 minutes were included in the present study. The use of segments of at least 180 minutes was a functional one, reflecting our desires to maximize data use while still reflecting EDA over an extended period of time. If the child was unwell during the period of observation or if there was equipment malfunctions, data were recorded again when it was convenient for the family.

Measures

Sleep

To measure sleep, each child wore a micromini-motionlogger® actigraph (Ambulatory Monitoring, Incorporated) in a neoprene ankle band to index their wake-sleep patterns for five consecutive 24-hour periods. Based on current guidelines, a minimum of 72 hours of valid actigraphy data was required for inclusion in the current study (Ancoli-Israel et al., 2003). Sleep-wake patterns and timing (i.e., sleep duration (total sleep time in minutes), frequency of wakings (number of wake episodes after sleep onset), and wake after sleep onset (WASO) duration (total minutes scored as awake after sleep onset) were analyzed based on the Sadeh algorithm. See Sadeh, Sharkey, and Carskadon (1994) for more details on the discriminant function used to estimate sleep/wake activity. In accordance with pediatric actigraphy recommendations (Meltzer, Montgomery-Downs, Insana, & Walsh, 2012; Schoch, Kurth, & Werner, 2020), parent-report sleep diaries were also used to aid in scoring data. For example, sensor removals and time placed in bed (i.e., the start of a down interval) were verified through parent reports. Parents also noted whether the child was sick or taking any medications during each day of the recording week.

Currently, there are no distinct criteria used to classify whether children have good or poor sleep health—especially when measuring sleep using an actigraph. However, sleep parameters are typically measured in a multi-dimensional way that can fall within broader categories of sleep health (Meltzer, Williamson, & Mindell, 2021), such as timing (e.g., placement of sleep within the day/night), efficiency (ease of falling asleep, continuity of sleep, and returning to sleep), and duration (total amount of sleep).

Therefore, we applied a combination of three common indices of sleep health (that fall within the broader domains of timing, efficiency, and duration; Meltzer et al., 2021) to classify sleep groups. Each child's sleep was classified as either regulated (RegS group, $n = 6$) or dysregulated (DysS group, $n = 7$). Children were specifically categorized as dysregulated sleepers if they met at least one of the following criteria: (1) slept less than American Academy of Sleep Medicine recommendations for their age (i.e., total amount of sleep), (2) woke for more than an hour on at least two nights (i.e., WASO), and/or (3) had morning rise or bedtimes that varied by more than two hours across nights (intra-individual variability in the timing or placement of sleep within the 24-hour day).

Electrodermal activity and accelerometer data

EDA, temperature, heart rate, and movement were recorded using an E4 multi-sensor (Empatica Inc., Boston, MA). The E4 multi-sensor is a crossover device designed for mass-market dissemination using research-grade development. Previous studies have successfully used it (or its predecessor) in adult (Kikhia et al., 2016; Sano & Picard, 2012) and child populations (Hoyniak et al., in press) and in children with autism (Cabibihan, Javed, Aldosari, Frazier, & Elbashir, 2016; Sano et al., 2012; Song, Liu, & Kong, 2016).

EDA was the target signal for this study and temperature, heart rate, and movement were utilized to assess the quality of the EDA signal and to explore patterns between daytime EDA and movement. Phasic and tonic indices of EDA across the collection period were split into one-minute intervals. For each minute, phasic indices included the number of nonspecific skin conductance responses (NSSCRs), peak NSSCR amplitude, and average NSSCR amplitude. Tonic indices were measured by skin conductance levels in the absence of phasic NSSCRs. Temperature was assessed using an infrared thermopile. Heart-rate was estimated using a photoplethysmography sensor to estimate blood volume pulse and movement was assessed with an accelerometer with gravitational force on three spatial dimensions. The EDA, temperature, heart rate, and accelerometer are integrated components in one E4 sensor.

EDA data were processed using Ledalab, an open source Matlab software used to analyze skin conductance data. Each second of processed data was examined for usability following guidelines

Table 2. Total number of days and minutes of recorded data and usable data for each child.

Child	Total number of days recorded	Total actively recorded minutes[a]	Usable minutes	Percentage of usable data
Child 1	3	1070	793	74.11%
Child 2	2	845	782	92.54%
Child 3	4	1636	1437	87.84%
Child 4	2	710	549	77.32%
Child 5	4	2159	1251	57.94%
Child 6	3	1048	975	93.03%
Child 7	2	750	655	87.33%
Child 8	2	795	496	62.39%
Child 9	3	1095	929	84.84%
Child 10	3	1175	686	58.38%
Child 11	2	795	551	69.31%
Child 12	2	815	534	65.52%
Child 13	2	800	464	58.00%

Note. [a]Actively recorded minutes refer to blocks that indicated start and end points.

provided by Kleckner and colleagues (2018). A data point was excluded if it failed to meet any of the following criteria: (1) EDA within the range of 0.05 and 60 microsiemens; (2) change in EDA per second within 10 microsiemens of the previous second; (3) heart rate within the range of 60 – 120 beats per minute; and (4) temperature within the range of 30 – 40 degrees Celsius. According to guidelines, data within 5 seconds of an invalid data point were also considered invalid and excluded from analyses.

The percentage of valid seconds within a minute epoch was used to index the validity of that 1 minute epoch. For a one-minute epoch to be considered valid, it contained 70% valid data, or 42 seconds per minute. Sessions with at least 180 consecutive minutes of valid data were used for this study. This resulted in a final usable data set of 10,102 minutes across 44 sessions in 13 participants. There was insufficient data for analysis between 08:00 – 09:00 and 16:00 – 17:00, corresponding to the start and end times of treatment hours. Since treatment hours ranged from 4 – 8 hours, data from 2 children were not available between 15:00 – 16:00. An average of each index – tonic: SCL and phasic: NSSCR, peak NSSCR amplitude, and average NSSCR amplitude – was calculated across each treatment hour (up to 7 hours). For a detailed breakdown of each child's usable data see Table 2.

ASD symptom scores

Child ASD symptoms were indexed with the Social Communication Questionnaire (SCQ; Rutter et al., 2003). The SCQ is a 40-item screener for ASD for children ages 30-months and above. Parents indicated the presence of ASD symptomatology for their child, specifically communication skills and social functioning. The SCQ total score ranges from 0 to 39. All children in this study were above the ASD cutoff (Moody et al., 2017), see Table 1.

Adaptive behavior

Child adaptive behavior was measured using the Vineland Adaptive Behavior Scale-Second Edition (VABS-II). VABS-II is a parent interview that assesses four domains – *communication, daily living skills, socialization, and motor skills* – with an optional domain on *maladaptive behaviors* (Sparrow, Cicchetti, & Balla, 2005). Standardized scores were used to describe the sample for each domain (Table 1).

Results

Overall, children in the RegS group slept more, woke less, and had less sleep variability (Table 1) when compared to children in the DysS group. Both RegS and DysS groups of children had

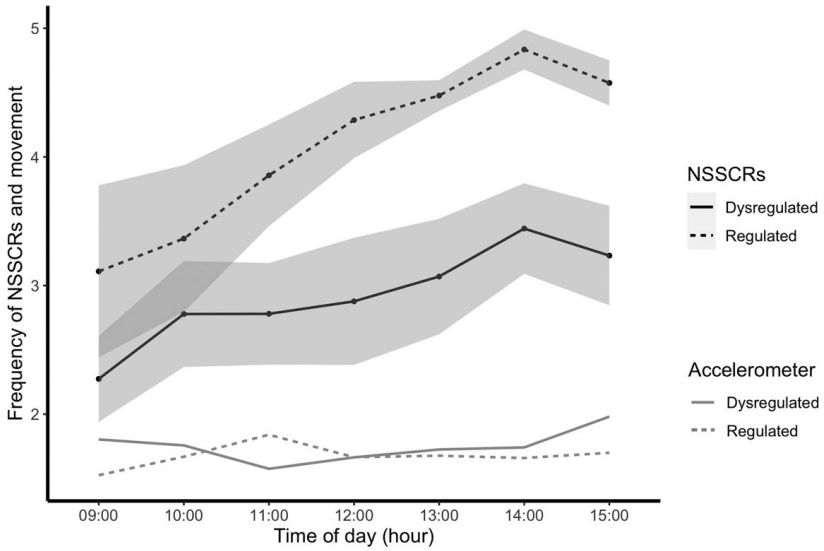

Figure 1. Frequency of nonspecific skin conductance responses (NSSCRs; black lines), standard errors of NSSCRs (gray band), and average standardized accelerometer movement (gray lines) at each hour from 09:00 to 15:00 stratified by child sleep group. Accelerometer/movement data were first converted into z scores and then reduced by 1.25 units to fit into the same reference figure.

similar VABS domain scores and daytime accelerometer data. Since all children had an ASD diagnosis and were receiving intensive behavioral intervention services, their SCQ scores were all at or above the cutoff of 11 (range = 11.0 – 31.0, $M = 20.28$, $SD = 5.86$). However, compared to the RegS group, children in the DysS group had higher SCQ scores (mean = 22.5, $SD = 6.35$) and were older (Table 1).

Initially, parametric comparisons of EDA across RegS and DysS groups were explored for this study but reviews of our analyses drew into question if such comparisons could (or should) be interpreted. Therefore, we have now adopted a descriptive approach and provide detailed information on daytime EDA patterns with several visual depictions of the data (Figures 1–3) to help guide future studies.

The number of NSSCRs increased over the course of the day for both groups of children with ASD (Table 3; Figure 1). For the RegS group, scores grew from an average 3.11 to 4.58 NSSCRs per minute. Within the DysS group, the scores followed a more modest growth pattern from 2.27 to 3.23 NSSCRs per minute. The average NSSCR differential grew between the two groups over the course of the day with the largest NSSCR differences occurring between 12:00 and 15:00. Given the known association between skin conductance responses and movement artifact, we also plotted the average activity for each group to visually inspect if activity also followed a similar pattern throughout the day. Overall, the patterns between average NSSCR and average activity do not appear coupled (Figure 1).

Throughout the day, skin conductance levels or tonic EDA gradually grew for the RegS group from 2.49 to 11.37 average microsiemens per minute (Table 3, Figure 3). For the DysS group the pattern was similar but attenuated, with scores from 1.28 to 2.58 average microsiemens per minute. Overall, children in the DysS group displayed a pattern of less physiological arousal and this pattern was most pronounced in the afternoon when children in the RegS group had higher EDA indices.

Discussion

The purpose of this exploratory, descriptive study was to share the first 'in the wild' extended recordings of EDA and sleep for children with ASD. The described patterns allude to a circadian

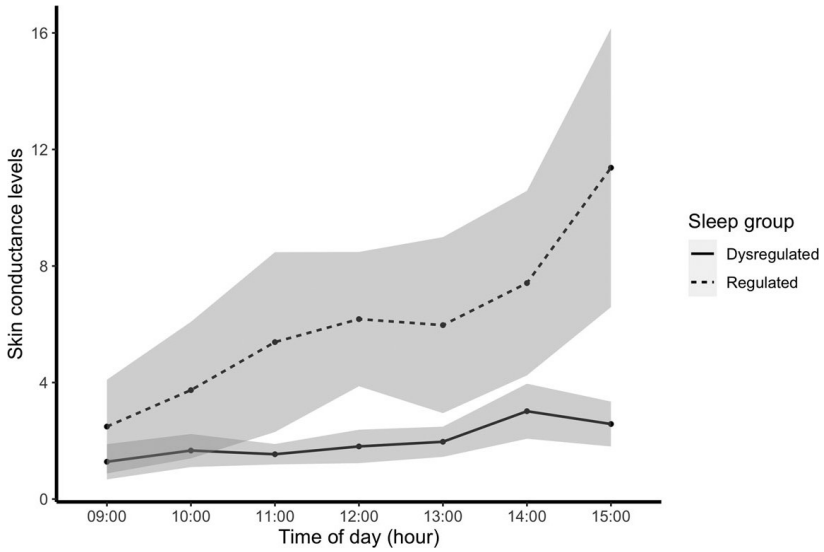

Figure 2. Average skin conductance levels (e.g., tonic EDA) at each hour from 09:00 to 15:00 stratified by child sleep group. Black lines represent average and gray bands reflect the standard errors.

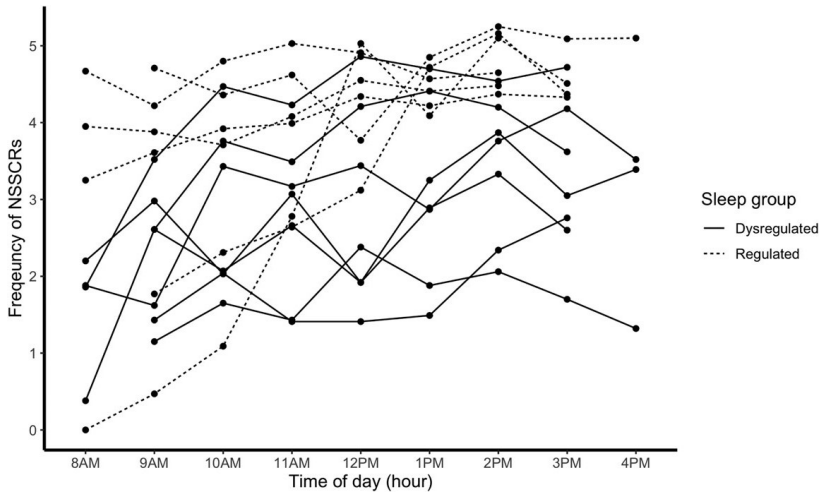

Figure 3. Frequency of nonspecific skin conductance responses (NSSCRs) for each individual from 09:00 to 15:00 stratified by child sleep group.

pattern or daily cycle in EDA that may be related to sleep or an overall regulatory pattern, but further research is needed to build on these preliminary results.

According to the restorative theory of sleep, decreased daytime EDA due to poor sleep may signal a failure to fully recover/restore during the night. For instance, Michael, Passmann, and Becker (2012) reported a decrease in NSSCRs following circadian oscillations in adults during a 24-hour sleep deprivation protocol, indicating main effects and an interaction of circadian patterns and sleep duration on NSSCRs in adults. Similarly, in this study, children classified in the DysS group had reduced sleep duration with more (and longer) night wakings and relatively fewer NSSCRs in the afternoon.

Although our findings on decreased NSSCRs run contrary to extant literature on hyperarousal in children with ASD (e.g., Kushki, Brian, Dupuis, & Anagnostou, 2014), Hirstein, Iversen, and Ramachandran (2001) hypothesized that there may be two subtypes of autonomic status in individuals with ASD. One subtype includes children with elevated EDA, thereby showing persistently elevated SCRs in response to most stimuli. The other subtype refers to children with ASD that have a flat or blunted response and tend to engage in self injurious or extreme behavior to

Table 3. Average number of NSSCRS and SCL per minute for each hour of the day between sleep groups.

	Range	Regulated sleep group ($n = 6$) *M(SD)*	Dysregulated sleep group ($n = 7$) *M(SD)*
Number of NSSCRs			
Between 09:00 and 10:00	0.47 – 4.71	3.11(1.64)	2.27 (0.88)
Between 10:00 and 11:00	1.09 – 4.80	3.37(1.40)	2.78 (1.09)
Between 11:00 and 12:00	1.41 – 5.03	3.86 (0.97)	2.78 (1.04)
Between 12:00 and 13:00	1.41 – 5.03	4.29 (0.73)	2.88 (1.31)
Between 13:00 and 14:00	1.49 – 4.85	4.48 (0.30)	3.07 (1.19)
Between 14:00 and 15:00	2.06 – 5.25	4.84 (0.38)	3.44 (0.93)
Between 15:00 and 16:00[a]	1.70 – 5.09	4.58 (0.32)	3.23 (1.02)
SCL			
Between 09:00 and 10:00	0.20 – 10.40	2.49 (3.94)	1.28 (1.60)
Between 10:00 and 11:00	0.32 – 15.33	3.74 (5.74)	1.67 (1.50)
Between 11:00 and 12:00	0.39 – 20.64	5.39 (7.56)	1.54 (0.93)
Between 12:00 and 13:00	0.30 – 15.16	6.18 (5.64)	1.81 (1.52)
Between 13:00 and 14:00	0.34 – 20.67	5.97 (7.40)	1.97 (1.37)
Between 14:00 and 15:00	0.40 – 21.98	7.41 (7.76)	3.02 (2.50)
Between 15:00 and 16:00[a]	0.56 – 25.43	11.37 (9.57)	2.58 (2.04)
Accelerometer data			
Between 09:00 and 10:00	20316.08 – 43418.41	29513.91 (8499.87)	36610.42 (33772.11)
Between 10:00 and 11:00	25352.03 – 54738.20	33182.39 (11134.61)	35425.00 (25401.42)
Between 11:00 and 12:00	26390.45 – 66685.31	37535.09 (15103.53)	30785.47 (23588.62)
Between 12:00 and 13:00	170.60 – 47032.36	33081.77 (16961.71)	33053.11 (28815.74)
Between 13:00 and 14:00	21189.93 – 54390.40	33386.38 (12148.41)	34612.78 (27862.06)
Between 14:00 and 15:00	17479.35 – 44781.95	32917.12 (11688.11)	35028.01 (23646.36)
Between 15:00 and 16:00[a]	25977.82 – 44339.79	33968.35 (8154.30)	41127.44 (35896.21)

Note. NSSCRs = nonspecific skin conductance responses, SCLs = skin conductance levels.
[a]Only $n = 4$ and $n = 7$ were included for the regulated and dysregulated sleep groups, respectively, due to shorter treatment days for some children.

activate their autonomic nervous system (e.g., Song et al., 2016). In the current study, the unbalanced group composition (i.e., older and higher SCQ scores in the DysS group) did not allow us to disentangle sleep, elevated ASD features, and age but the overall patterns described by Hirstein et al. (2001) do appear in our data. Additionally, the group with a more blunted response in this study is arguably the more impacted group with more parent-reported autism features (as indexed by SCQ scores) and more sleep problems.

Previous studies of circadian EDA patterns in healthy adults describe a pattern that was not present in this study. Kim et al. (2018) reported a circadian rhythm in tonic SCL; wherein, the highest average SCLs were reported at roughly 7:00 followed by a gradual drop throughout the day until about 17:00 and then a gradual climb throughout the night and early morning hours. We anticipated that the RegS group would follow a similar pattern but instead both groups seem to follow an increasing pattern throughout the day. These discrepancies highlight how further chronobiology research is needed in the area of EDA. We could not identify a study that documented daytime circadian patterns in EDA for children; hence, it is unclear if the pattern differences reflect the younger age of this sample or another unconsidered variable. Previous studies document EDA differences with age (e.g., Bari, Yacoob Aldosky, & Martinsen, 2020) therefore, further exploration of potential age effects is warranted.

From a physiological perspective, our findings emphasize the importance of considering child characteristics such as sleep patterns when recommending treatment hours and activity. NSSCRs can reflect sympathetic arousal to environmental stimuli, and in the context of intervention may be associated with attention and emotional responses (Betancourt, Dethorne, Karahalios, & Kim, 2017). Lower NSSCRs may reflect lower emotional arousal and attention, as evidenced by an MRI study where the ventromedial prefrontal cortex, a brain region responsible for social and emotional behavior, is also the site of NSSCR generation (Critchley, Elliott, Mathias, & Dolan, 2000).

Therefore, fewer NSSCRs in the afternoon may indicate less engagement with the environment, which in turn may influence learning and daytime behavior presentations.

Notably, this is a pilot study and replication is needed before clinical recommendations can be made. Recognizing that sleep-wake cycles are influenced by circadian rhythms, future studies should consider the effects of time of day, sleep regulation, stimuli (e.g., type of behavioral intervention), and current emotional state when examining physiological arousal in children (Dawson et al., 2007). The inclusion of other EDA indices (e.g., skin conductance responses to social or contextual stressors) could also inform physiological mechanisms of sleep dysregulation as these indicators are associated with different concepts such as stimulus-induced arousal or current attention.

Limitations

One of the limitations of this study is the small and heterogeneous sample. It is probable that child co-occurring conditions (e.g., ADHD, anxiety) and medication use could influence both EDA estimates and sleep within this sample. Although the rates of co-occurring conditions and medication use were not vastly different across the sleep groups, future studies should aim to account for these potential confounding factors. Additionally, the small heterogeneous sample size did not allow us to disentangle the effects of age, ASD symptoms, and sleep on EDA.

Sleep is a complex biosocial process that can be influenced by several biological and contextual elements (e.g., Liu, Hubbard, Fabes, & Adam, 2006). Although our study used actigraphy measures to record objective sleep parameters, such as total sleep time and duration of night awakenings, there was insufficient information on environmental or psychosocial factors (e.g., children's sleeping arrangement, light exposure) that may influence their sleep behaviors in this study.

Our use of the E4 multi-sensor may have fit the practical needs of this project but may have also resulted in less-usable data and potentially lower quality data (i.e., a higher noise to signal ratio). Relatedly, the use of EDA as a measure of the sympathetic branch of the autonomic nervous system reflects only one element of a large complex arousal system. This incomplete appraisal of the arousal system limits how this study can inform the larger field. Future research can build on this study by including other arousal indices such as cortisol (neuroendocrine system) or heart rate variability (parasympathetic nervous system). For instance, examining stress-related cortisol responses may inform cycles of stress, sleep, and EDA reactivity.

Finally, since each child had an individualized program for their behavioral intervention, we did not have information on the specific activities each child engaged in during the periods of observation. The variation in activities may contribute to differences in EDA across the day and it is unclear if data for individuals that were excluded were missing during specific activities (i.e., not missing at random).

Conclusions

Overall, findings from this study indicate that physiological arousal levels may follow a circadian pattern for children with ASD and may be influenced by sleep dysregulation or a dysregulation profile. Poor sleep can impede a full recovery during the night and may lead to altered physiological response the following day. However, given the characteristics of our sample, these findings may reflect an overall dysregulation profile of dysregulated sleep, blunted afternoon physiological arousal profile, and elevated ASD symptom presentation. The richness of this physiological study serves as a valuable starting point for future work examining clinical populations in naturalistic settings.

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Data availability statement

Upon publication, data will be made publicly available on the Open Science Framework (DOI: 10.17605/OSF.IO/XRY87).

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