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RESEARCH ARTICLE

Parasympathetic functioning and sleep problems in children with autism spectrum disorder

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Abstract

Respiratory sinus arrhythmia (RSA), an index of parasympathetic nervous system activity, has been linked with sleep quality among children with neurotypical development. The current study extended examination of these processes to children with autism spectrum disorder (ASD), a group at considerable risk for sleep problems. Participants included 54 children with ASD (aged 6–10 years, 43% Hispanic). RSA data were collected via a wired MindWare system during a 3-min baseline and a 3-min challenge task. Parents reported on their children's sleep problems and sleep duration using the Children's Sleep Habits Questionnaire, Abbreviated. Although no significant correlations emerged between RSA indices and parent-reported child sleep, baseline RSA and RSA reactivity interacted in the prediction of sleep problems. For children with higher RSA reactivity, higher baseline RSA was associated with fewer sleep problems, but for children with lower RSA reactivity, baseline RSA was not predictive. No main effects or interactions of RSA predicted sleep duration. Findings suggest resilience against sleep problems for children with ASD presenting with higher baseline RSA and higher RSA reactivity. Implications of these results center upon directly targeting psychophysiology (i.e., parasympathetic nervous system regulation) as a possible mechanism to improve sleep in children with ASD, and developing personalized interventions based on physiological markers of risk and resilience.

Lay Summary

Children with autism spectrum disorder (ASD) often have difficulty sleeping, but contributing factors are not well understood. Findings suggest that certain psychophysiological tendencies thought to represent better overall arousal regulation and greater physiological reactivity to challenge may protect against sleep difficulties in children with ASD. Implications highlight the potential for targeting psychophysiological regulation as an avenue for reducing sleep problems.

KEYWORDS
ASD, autism spectrum disorder, parasympathetic nervous system, respiratory sinus arrhythmia, RSA, sleep

INTRODUCTION

High-quality sleep supports children's social, emotional, cognitive, and behavioral well-being (Dewald et al., 2010; Short et al., 2018, 2019). Children with autism spectrum disorder (ASD) are at considerable risk for sleep problems (Díaz-Román et al., 2018; Elrod & Hood, 2015), and in turn, a cascade of related challenging outcomes

(e.g., Cohen et al., 2014). Therefore, elucidating mechanisms that may underlie individual differences in sleep problems and duration in this at-risk population have critical implications. Biopsychosocial models of sleep highlight complex interrelations between sleep and bioregulatory systems among children with and without ASD (Dahl, 1996; Schreck & Richdale, 2020), yet little empirical work has explored physiology and sleep in ASD (e.g., Harder et al., 2016; Pace et al., 2016). Daytime respiratory sinus arrhythmia (RSA), an index of parasympathetic nervous system (PNS) activity, has been linked with sleep quality among children with neurotypical development (Elmore-Staton et al., 2012; El-Sheikh et al., 2013) highlighting an interaction between baseline RSA (RSA-B) and RSA reactivity (RSA-R) to stress (i.e., withdrawal of parasympathetic activity, or RSA withdrawal) in predicting sleep quality (El-Sheikh et al., 2013). As no studies to our knowledge have explored daytime RSA-B and RSA-R to better understand within-group variability in sleep problems and duration in children with ASD, we sought to begin to fill this gap in the literature.

Sleep problems, including insomnia, poor sleep efficiency (i.e., ratio of time asleep/awake while in bed), parasomnias (e.g., nightmares), and advanced or delayed sleep–wake cycles, are ubiquitous in ASD with estimated prevalence rates between 50% and 80% (Richdale & Schreck, 2009; Schreck & Richdale, 2020; Souders et al., 2017). Disrupted sleep in ASD has pervasive short and long-term effects; sleep problems have been linked with increased daytime behavior problems (Mazurek et al., 2019), lower quality of life (Lawson et al., 2020), later unemployment (Baker et al., 2019), and more severe core symptoms (Tudor et al., 2012) in individuals with ASD, and poorer well-being in caregivers (Hodge et al., 2013). As such, researchers have sought to better understand the emergence and maintenance of sleep disturbances in this population. Several contributing factors have been posited, including a combination of related biological (e.g., physiological arousal), psychological (e.g., co-occurring anxiety), and environmental (e.g., stress) factors (Souders et al., 2017). Despite the theoretical support for psychophysiology as a key biological contributor to sleep disruption in ASD (Souders et al., 2017), empirical investigation is sparse.

Research on children with neurotypical development has highlighted links between physiological regulation and sleep, with arousal and sleep existing at opposite ends of a spectrum (Dahl, 1996). Daytime functioning of the PNS, in particular, has emerged as a correlate of sleep quality among children with neurotypical development (Elmore-Staton et al., 2012; El-Sheikh et al., 2007, 2013; El-Sheikh & Buckhalt, 2005). The PNS is a branch of the autonomic nervous system (ANS) that decelerates heart rate, acting as a "brake" to reduce arousal. RSA is a vagally-mediated marker of PNS activity that quantifies the variability in heart rate at the frequency of respiration. RSA is commonly measured during a baseline condition (RSA-B) as well as in response to a challenging or stressful task (RSA-R). Lower values of RSA-B reflect lower vagal tone (i.e., less PNS output) and are conceptualized as a marker of less self-regulatory capacity (Beauchaine et al., 2007). Although several studies have identified lower average RSA-B in people with ASD relative to comparison samples, other investigations have not (for reviews see Arora et al., 2021; Barbier et al., 2022; Benevides & Lane, 2015; Cheng et al., 2020). Among individuals with ASD, there is some evidence linking higher RSA-B with lower levels of anxiety and better social–emotional skills (e.g., Arora et al., 2021; Guy et al., 2014), consistent with within-group patterns among samples with neurotypical development (Beauchaine, 2001). RSA-R quantifies the degree of PNS output withdrawal (i.e., removal of the vagal brake, which increases heart rate and decreases RSA) in response to stress; greater RSA-R is indicative of larger PNS withdrawal (i.e., greater decrease in RSA). Withdrawal of PNS output is often thought to reflect an adaptive response to stress, facilitating increased arousal and task engagement, yet implications of the degree of RSA-R may be specific to the population of interest, measurement, and task (Graziano & Derefinko, 2013). In children with ASD in particular, greater RSA-R in combination with certain other psychophysiological indices (e.g., electrodermal reactivity) may reflect a lack of regulatory control, while lower RSA-R may be indicative of reduced task engagement (Fenning et al., 2019).

Studies of children with neurotypical development reveal direct links between sleep and RSA-B (Elmore-Staton et al., 2012; El-Sheikh & Buckhalt, 2005) and RSA-R (El-Sheikh & Buckhalt, 2005), though results are somewhat mixed. Among preschoolers with neurotypical development, lower RSA-B was related to actigraphy measures indicative of poorer sleep quality (Elmore-Staton et al., 2012), though the opposite association has been found for self-reported sleep problems in school-aged children with neurotypical development (El-Sheikh & Buckhalt, 2005). Additionally, lower RSA-R has been linked with more self-reported sleep problems in school-aged children with neurotypical development, and greater RSA-R has been associated with better actigraphy-measured sleep quality and duration (El-Sheikh & Buckhalt, 2005). A subsequent study helped to further clarify RSA-sleep associations by examining the interaction between RSA-B and RSA-R in predicting sleep among children with neurotypical development (El-Sheikh et al., 2013). Results revealed a significant RSA-B by RSA-R interaction in predicting sleep quality, but not sleep duration. Specifically, associations between RSA-B and sleep quality metrics were only evident in the context of children exhibiting greater RSA-R, such that children with a combination of low RSA-B and high RSA-R demonstrated the most disrupted sleep. El-Sheikh and colleagues interpreted this combination of low RSA-B and high RSA-R as reflecting hyperarousal and/or dysregulation of the ANS

that is incompatible with sleep. Exploring interactions between RSA-B and RSA-R has been highlighted as an important and promising direction for research, especially among clinical samples (Graziano & Derefinko, 2013). To date, studies have only examined related cardiac indices *during* sleep among children with ASD *in comparison* to children without ASD. Findings from these studies are mixed, with evidence for higher (Pace et al., 2016) and lower (Harder et al., 2016) PNS activity during sleep in children with ASD. No known research has examined daytime RSA in relation to *within-group* variability in sleep in children with ASD.

Current study

The current study aimed to better understand links between RSA and sleep in children with ASD, including both main effects and interactive effects of RSA-B and RSA-R in predicting sleep problems and duration. Consistent with research among children with neurotypical development (El-Sheikh et al., 2013), we anticipated that RSA-B and RSA-R would interact in predicting sleep problems such that the association between sleep problems and RSA-B would only be evident under conditions of high RSA-R and that the combination of low RSA-B and high RSA-R would be linked with the greatest sleep problems.

METHOD

Participants

Data were drawn from a larger study exploring interactions between parenting and psychophysiology in children with ASD (Baker et al., 2020; Fenning et al., 2019). Participants were community-recruited via flyers and local service providers. Inclusion criteria required an existing ASD diagnosis by a physician or psychologist confirmed by study-administered assessments. Exclusionary criteria involved presence of a genetic disorder with known etiology and motor impairment that would interfere with task participation. An initial sample of 77 children aged 6–10 years and their primary caregivers were enrolled. Some children were missing RSA data due to common problems with collection, including refusal of lead placement ($n = 11$) and signal quality resulting in artifactual data ($n = 5$; e.g., movement artifacts). Seven additional families did not return the sleep questionnaire, resulting in a final sample of 54 children. Table 1 presents demographic characteristics.

TABLE 1 Demographic and clinical characteristics ($n = 54$)

Variables	
Child age, *M (SD)*	8 (1.5)
Child IQ, *M (SD)*	81.1 (19.9)
Range	47–121
Child gender, *n* (%)	
Male	41 (75.9)
Female	13 (24.1)
Child race/ethnicity, *n* (%)	
Hispanic	23(42.6)
White	19 (35.2)
Multiracial	5 (9.3)
Asian	3 (5.6)
Black/African American	2 (3.7)
Other	5 (3.7)
Mother marital status, *n* (%)	
Married	39 (72.2)
Separated/divorced	9 (16.7)
Never married	6 (11.1)
Income (USD), *n* (%)	
0–15,000	4 (7.5)
15,000–25,000	4 (7.5)
25,000–35,000	6 (11.3)
35,000–50,000	6 (11.3)
50,000–70,000	8 (15.1)
70,000–95,000	6 (11.3)
>95,000	19 (35.8)
Parent-reported co-occurring conditions *n* (%)	
≥1 co-occurring condition	31 (57.4)
ADHD	13 (24.1)
Speech/language conditions	7 (13.0)
Intellectual disability/developmental delay	6 (11.1)
Sensory or auditory processing condition	6 (11.1)
Anxiety, mood, or related disorders	5 (9.3)
Other medical conditions	11 (20.4)

Procedures

The institutional review board approved procedures. Parents and children provided informed consent and verbal assent, respectively. Parent–child dyads participated in a laboratory-based research visit, including direct child testing. Parents completed questionnaires during the laboratory visit or returned them via mail.

RSA data collection

RSA was collected via a wired MindWare system during an established 3-min baseline and a 3-min challenge task (e.g., Baker et al., 2020; El-Sheikh, 2005; Erath et al., 2016; Fenning et al., 2019). The child was seated at a table facing a small television with a video camera positioned above it. A wall and temporary partition were on either side of the child. The parent was seated behind the

temporary partition. Disposable Ag-AgCl electrodes were placed on the child's right clavicle and lower ribs, and the presence of an observable electrocardiogram waveform was confirmed. An initial adjustment period was followed by a baseline involving viewing of nature slides (e.g., scenes of trees, mountains, etc.) on a television (Baker et al., 2020; Erath et al., 2016). The child then engaged in a challenge task designed to elicit physiological arousal related to negative emotion (e.g., El-Sheikh, 2005). The task involved tracing a star with an indirect mirror view of the hand and paper, which is difficult due to image reversal. All participants sufficiently manipulated the pencil and were judged to have understood the request for basic tracing.

Measures

Diagnostic characterization

The Autism Diagnostic Observation Schedule-2 (ADOS-2; Lord et al., 2012) was administered to participants by research-reliable assessors. The ADOS-2 has strong psychometrics (Lord et al., 2012) and is a gold-standard diagnostic instrument involving semi-structured interactions designed to assess social communication, play, repetitive behaviors, and restricted interests. Participants received Module 3 ($n = 35$; 65%), Module 2 ($n = 14$; 26%), and Module 1 ($n = 5$; 9%). The ADOS-2 comparison score was used to quantify level of ASD-related symptoms across modules, with scores ranging from 1 (*minimal to no evidence*) to 10 (*high*). Five children had ADOS-2 scores below the ASD classification threshold; these participants were retained based on a multi-method clinical best estimate performed by a licensed clinical psychologist with research reliability in the ADOS-2 and significant expertise in ASD assessment (see Baker et al., 2020 for more information). All five children met criteria suggestive of ASD on the Social Responsiveness Scale-2 (Constantino & Gruber, 2012), a standardized parent-report questionnaire of ASD symptoms, and four of the five scored at or above the cut-off on the Social Communication Questionnaire, Lifetime Version (Rutter et al., 2003), a commonly used screening tool for ASD.

Intellectual functioning

The Stanford-Binet 5 Abbreviated Battery (SB-5 ABIQ; Roid, 2003) was used to index children's intelligence quotient (IQ). The ABIQ consists of the nonverbal Matrix Reasoning subscale and the verbal Vocabulary subscale. The SB-5 Full Scale IQ and ABIQ are highly correlated for children with and without ASD (Roid, 2003; Twomey et al., 2018). The ABIQ has a mean of 100 and a standard deviation of 15; scores below 76 are consistent with the current IQ criterion for intellectual disability (American Psychiaric Association, 2013).

Sleep problems

The Children's Sleep Habits Questionnaire-Abbreviated (CSHQ-A; NICHD SECCYD-Wisconsin, 2017) is a 22-item parent report questionnaire that measures child sleep problems (night waking, daytime sleepiness, behavior during sleep, and bedtime behavior) and is derived from the original 45-item version (Owens et al., 2000). CSHQ-A response options are on a 5-point scale from Always (*occurs every night*) to Never (*occurs less than once a week*) based on frequency during the past week (or most recent typical week). A sample item is "Child awakens during the night and is sweating, screaming, and inconsolable." The total score of sleep problems on the CSHQ-A was calculated by summing all items; higher scores indicate more sleep problems. Parents also reported upon their child's usual bedtime and the time their child usually wakes up in the morning for both weekdays and weekends. If not indicated, it was assumed wake times were AM and bedtimes were PM. Sleep duration was calculated in minutes. Sleep duration for weekdays and weekends were positively correlated, $r(52) = 0.54$, $p < 0.001$, and were not significantly different, $t(52) = -0.77$, $p = 0.44$. Therefore, a weighted average was calculated for sleep duration across weekdays and weekends. The CSHQ-A and original CSHQ have been used in research on ASD (Irwanto et al., 2016; Mazurek et al., 2019). Previous research with the CSHQ-A demonstrates evidence of validity through associations with child disruptive behavior and parenting factors (Coto et al., 2018). Current study internal consistency was high, $\alpha = 0.81$.

Respiratory sinus arrhythmia

Sampling rate was 500 Hz. RSA scores were calculated using spectral analysis (Berntson et al., 1997) with MindWare HRV analysis software (version 3.0.22) as the natural log of the variance in heart period within age-adjusted respiratory frequency bands (e.g., 0.27–0.50 Hz for 9-year-olds, 0.25–0.50 Hz for 10-year-olds; see Shader et al. (2018), for additional ranges). Standardized and validated respiratory frequency bands were utilized given the demands of direct respiratory measurement and our efforts to include children presenting with a broad range of cognitive and behavioral functioning. Units for RSA are $\ln(ms^2)$. Possible artifacts were identified by MindWare HRV analysis software, which detects and flags R-peaks that reflect improbable interbeat intervals (Berntson et al., 1997). Two trained coders noted and manually corrected unmarked or mismarked R-peaks. Across baseline and challenge periods, respectively, 72% and 80% of participants required no edits, 16% and 13% required 1–2 corrected R-peaks, and 11% and 7% required several corrected R-peaks. RSA reactivity (RSA-R) was calculated as the residual of the regression of RSA during the star-tracing period ($M = 5.61$,

SD = 1.23) on RSA during the baseline period (*M* = 5.89, *SD* = 1.19), as is common practice for calculating reactivity in psychophysiological research (Baker et al., 2020; Beauchaine et al., 2019; Burt & Obradović, 2013). Residualized change scores were multiplied by −1 so that higher RSA reactivity scores indicated greater reductions in RSA (i.e., greater withdrawal) from the baseline to the challenge period.

Data analytic plan

SPSS version 28.0 was employed. Analyses explored whether missing data were related to sociodemographic variables. Descriptive statistics were calculated, and histograms and boxplots were used to examine variable distributions and to screen for extreme values. Bivariate Pearson correlations were performed to examine zero-order associations between sleep (problems and duration), RSA indices, and sociodemographic and child-specific (e.g., IQ) factors. Sociodemographic and child factors found to correlate significantly with variables of interest were controlled in regression models. Two separate stepwise regression analyses were conducted to first test main effects of sociodemographic and child factors, and both RSA indices, and, second, to test the interaction between RSA-B and RSA-R in predicting sleep problems and sleep duration. Predictors were mean centered in the regressions. Significant interactions were probed by examining simple slopes at one standard deviation above and below the mean of the moderator. To further explore the nature of interactions, Regions of Significance (RoS) analyses were conducted via an online program (Roisman et al., 2012) with respect to Z (moderator: RSA-R) and X (predictor: RSA-B). RoS-Z determines the values of the moderator (RSA-R) at which the regression of Y (sleep problems) on X (RSA-B) is significant. RoS-X examine the values of X (RSA-B) at which the regression of Y (sleep problems) on the moderator (RSA-R) is significant.

RESULTS

Preliminary analyses

Participants with missing data (CSHQ-A: $n = 7$, 9%; RSA: $n = 16$, 21%) had significantly higher ASD symptom scores on the ADOS, $t(75) = -2.25$, $p = 0.03$, and were significantly younger, $t(75) = 2.12$, $p = 0.04$. IQ, gender, and household income were unrelated to missingness. Seventeen children were reported to be taking prescription or over-the-counter medications. Medication use was considered dichotomously and was not significantly related to the RSA variables or to reported sleep problems. Examination of variable distributions revealed that all variables were normally distributed, within normal limits of skew and kurtosis, and without extreme values, apart from RSA-R. One extreme value of RSA-R was identified (RSA-R = −2.8) and Winsorized to the next lowest value (RSA-R = −1.35).

Correlations

Correlations between sleep, RSA, and sociodemographic factors are presented in Table 2. A significant inverse association emerged between child age and sleep problems; older children were reported to have fewer sleep problems. Age was also significantly positively related to the (age-adjusted) RSA-B. Therefore, age was included in subsequent regression analyses. IQ, gender, and ASD symptom level were not significantly related to sleep or RSA indices. Sleep problems and duration were not significantly related to either RSA-B or RSA-R.

Regression analyses

Results of regression analyses are presented in Table 3. Step one of the regression models (RSA-B, RSA-R, and child age) explained approximately 8% and 11% of the variance in sleep duration and problems, respectively. No significant main effects of RSA-B or RSA-R were detected, controlling for child age. Older children had fewer parent-reported sleep problems ($B = -2.30$, $t = -2.11$, $p = 0.04$). In step two, the addition of the interaction explained an additional 1% and 7% of the variance in sleep duration and problems, respectively. Controlling for child age, there was a significant interaction between RSA-B and RSA-R in predicting sleep problems, but not sleep duration. Examination of simple slopes revealed a significant inverse association between RSA-B and sleep problems, $B = -4.89$, $t = -2.10$, $p = 0.04$, in the context of high RSA-R (Figure 1). In the context of low RSA-R, the link between sleep problems and RSA-B was non-significant, $B = 2.75$, $t = 1.55$, $p = 0.13$ (Figure 1). RoS-Z analyses indicated that the regression of sleep problems on RSA-B was significant for values of RSA-R lower than −2.25 (outside of observed scores) and higher than 0.44 (within the observed scores; 20% of sample). RoS-X analyses indicated lower and upper bounds of −7.04 and 0.78 (i.e., uncentered values of −1.15 and 6.67), respectively. Given that only the upper bound was plausible and within the region of interest (2 *SD* above and below the mean), higher RSA-R was related to fewer sleep problems when RSA-B was above 6.67 (26% of sample; Figure 1). Probing the interaction with RSA-B as the moderator (low RSA-B: $B = 4.45$, $t = 1.22$, $p = 0.23$; high RSA-B: $B = -9.12$, $t = 2.14$, $p = 0.04$) indicated a highly similar pattern such that the combination of high RSA-B and high RSA-R appeared protective against sleep problems (RoS-X: −2.25, 0.44).

TABLE 2 Descriptive statistics and correlations in the sample with ASD

Variable	*n*	*M*	*SD*	1	2	3	4	5	6	7
1. RSA baseline	54	5.89	1.19	-						
2. RSA reactivity	54	0.03	0.67	−0.06	-					
3. Sleep problems total	54	27.80	11.79	−0.15	−0.08	-				
4. Sleep duration (minutes)	53	589.96	48.05	−0.12	0.26	−0.30*	-			
5. Age (years)	54	8.00	1.50	0.38**	−0.02	−0.32*	−0.10	-		
6. IQ	53	81.08	19.92	−0.25	0.05	−0.16	0.20	−0.24	-	
7. Gender (% female)	54	24.07%	-	−0.11	−0.08	0.13	−0.05	0.15	−0.08	
8. ASD symptom level	54	7.13	2.13	0.004	0.13	−0.04	0.09	−0.06	−0.07	−0.04

Abbreviations: ASD, autism spectrum disorder; IQ, intelligence quotient; RSA, respiratory sinus arrhythmia.
*$p < 0.05$.
**$p < 0.01$.

TABLE 3 Regressions RSA predicting sleep problems and duration

Variable	*B*	*β*	*SE*	*t*	*p*	R^2	ΔR^2
Models predicting sleep problems							
Step 1						0.11	0.11
Constant	27.80		1.56	17.83	<0.001		
Age (years)	−2.67	−0.30	1.13	−2.10	0.04		
RSA baseline	−0.41	−0.04	1.43	−0.28	0.78		
RSA reactivity	−1.54	−0.09	2.36	−0.65	0.52		
Step 2						0.18	0.07*
Constant	27.53**		1.51	18.20	<0.001		
Age (years)	−2.30*	−0.29	1.09	−2.11	0.04		
RSA baseline	−1.07	−0.11	1.42	−0.76	0.45		
RSA reactivity	−2.34	−0.13	2.31	−1.01	0.32		
RSA baseline x RSA reactivity	−5.70*	−0.28	2.71	−2.11	0.04		
Models predicting sleep duration (minutes)							
Step 1						0.08	0.08
Constant	598.84		6.52	91.84	<0.001		
Age (years)	−1.0.75	−0.06	4.71	−0.37	0.71		
RSA baseline	−3.49	−0.09	5.93	−0.59	0.56		
RSA reactivity	17.96	0.25	9.81	1.83	0.07		
Step 2						0.09	0.01
Constant	599.25**		6.57	91.25	<0.001		
Age (years)	−1.85	−0.06	4.73	−0.39	0.70		
RSA baseline	−2.42	−0.06	6.11	−0.40	0.66		
RSA reactivity	19.23	0.27	9.98	1.93	0.06		
RSA baseline x RSA reactivity	9.10	0.11	11.63	0.78	0.44		

Abbreviation: RSA, respiratory sinus arrhythmia.
*$p < 0.05$.
**$p < 0.01$.

DISCUSSION

Existing literature has highlighted sleep disruption as a common and problematic occurrence for children with ASD, yet factors contributing to sleep concerns in this population are not fully understood. With a goal of identifying whether physiological activity and arousal may play a role in sleep problems in this population, we tested associations between RSA, an index of PNS activity, and sleep among children with ASD. Although findings revealed no direct associations between sleep and either RSA-B or RSA-R, sleep problems were predicted

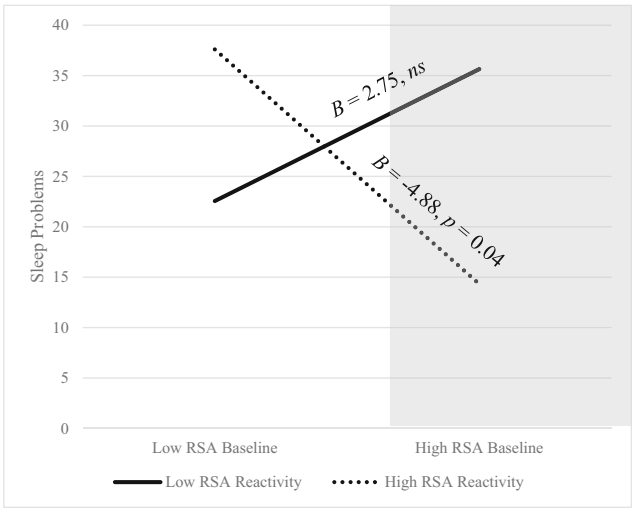

FIGURE 1 RSA-R and RSA-B interact in predicting sleep problems in children with autism spectrum disorder. RSA, respiratory sinus arrhythmia; shaded region reflects RoS-X.

by the interaction of RSA-B and RSA-R. High RSA-B combined with high RSA-R may confer resilience against sleep problems, which are otherwise pervasive in children with ASD.

The present study adds to a growing body of literature in ASD suggesting that the implication of physiological indicators, and particularly RSA-R, may depend on environmental conditions (e.g., level of negative parenting behavior; Baker et al., 2020) or other internal regulatory processes (e.g., electrodermal reactivity; Fenning et al., 2019). Our current examination of sleep revealed that the effect of RSA-B depended on RSA-R to stress such that the association between RSA-B and sleep problems was only evident in the context of high RSA-R, similar to findings from El-Sheikh et al. (2013). That is, in the context of higher levels of parasympathetic withdrawal, greater baseline regulatory capacity may lead to an arousal level compatible with sleep whereas lower baseline regulatory capacity may lead to hyperarousal that is incompatible with sleep. In contrast, baseline regulatory capacity may be less consequential for sleep if parasympathetic reactivity is diminished. As suggested by El-Sheikh et al. (2013), it may be that daytime arousal levels persist throughout the night, and/or that nighttime arousal levels (and its psychological correlates) endure throughout the day; these links, regardless of directionality, may be particularly pronounced in children with ASD given the heightened risk for emotional, behavioral, and physiological dysregulation in this population (Mazefsky & White, 2014; White et al., 2014). These findings further support the utility of exploring *interactions between* psychophysiological baseline and reactivity in clinical samples (Graziano & Derefinko, 2013) to provide clarity regarding dynamic physiological processes that may underlie mixed findings.

Congruent with findings in children with neurotypical development (El-Sheikh et al., 2013), children in our sample with the combination of low RSA-B and high RSA-R demonstrated the greatest sleep difficulties. However, consistent with our focus on a population at elevated risk for sleep problems (Díaz-Román et al., 2018), sleep difficulties were relatively pervasive in our sample, with the exception of children presenting with high levels of both RSA-B and RSA-R. Our findings thus suggest a possible population-specific resilience phenomenon, with high RSA-B and high RSA-R potentially protective against sleep difficulties in children with ASD. This effect appeared highly robust, given the consistency of findings with either RSA-B or RSA-R as the moderator. Additionally, older children in our sample were reported to have fewer overall sleep problems, consistent with evidence that some sleep difficulties may reduce over time for children with ASD (Goldman et al., 2012; Krakowiak et al., 2008; Liu et al., 2006).

Sleep duration was unrelated to RSA, paralleling findings from El-Sheikh et al. (2013). As suggested previously (El-Sheikh et al., 2013), a compensatory mechanism may underlie these null results. Experimental manipulation of sleep extension and restriction suggests that sleep duration may be extended to compensate for poor sleep quality (Sadeh et al., 2003), thereby negating the effect of physiological arousal on sleep duration. Another consideration relates to the metric of sleep duration used in the present study, as the calculated duration from sleep onset to waking does not account for night waking – a frequent problem among children with ASD (Souders et al., 2017); sleep duration may be overestimated in this sample, especially for those with more frequent or extended night waking. Given mixed evidence of associations between sleep duration and daytime functioning in children with ASD (e.g., Shui et al., 2021; Veatch et al., 2017), it will be important for future work to continue to investigate the utility and relevance of different sleep indices for this population.

Implications of the present study center upon targeting psychophysiology as a potential mechanism for decreasing sleep problems in children with ASD. Based on the results of the current study, fostering greater vagal tone and appropriate RSA withdrawal in response to stress, therapeutically and via environmental supports, may be promising avenues for decreasing sleep problems in this population. Wearable physiological technology is becoming more accessible, affordable, and less obtrusive (Guo et al., 2015; Zheng et al., 2014), which may help to readily identify children with ASD who are at psychophysiological risk for, or resilient to, sleep disruption and may especially benefit from related interventions. Accessible biological measurements may also facilitate consideration of psychophysiology as a core treatment target and outcome.

Heart rate variability biofeedback offers one route to therapeutically target physiological cardiac regulation

(Lehrer & Gevirtz, 2014; Nolan et al., 2005). This may be a viable approach for individuals with ASD as well (Brezis et al., 2021; Goodman et al., 2018), but further research is needed to inform biofeedback targets given evidence that certain psychophysiological indices may relate differently to dysregulation in this population (Baker et al., 2020; Fenning et al., 2019). Additionally, cognitive, behavioral, and mindfulness-based interventions have been developed to target self-regulation in youth with ASD (Conner et al., 2019; Reyes et al., 2019; Thomson et al., 2015; Weiss et al., 2018), which may, in turn, support better sleep in this population. In terms of environmental supports, parent co-regulatory support is known to promote development of children's independent regulatory capacity across populations (Cole et al., 1994; Morris et al., 2017; Sameroff, 2010; Thompson & Meyer, 2007). In fact, emotion-related parenting (e.g., parental supportive reactions, scaffolding, etc.) has been related to physiological indicators of regulatory capacity in ASD (Moffitt et al., 2021) and may ameliorate certain physiological risk for maladaptive behavioral outcomes in this population (Baker et al., 2017, 2020). As such, clinicians may work with parents to refine co-regulatory strategies to promote positive daytime behavior and to ameliorate sleep problems in children with ASD. Assessment of sleep problems as an outcome of interventions targeting behavioral and emotion regulation may be a fruitful direction for future practice and research.

Considering the opposite direction of effect, improving sleep could be a conduit for improving psychophysiological regulation in children with ASD. Preliminary evidence suggests a possible beneficial increase in baseline PNS responding in several children with ASD following insomnia intervention (McCrae et al., 2021). Further research is needed, and the growing body of literature on sleep interventions in ASD (Johnson et al., 2013; Loring et al., 2016; Schoen et al., 2017) may benefit from integration of physiological treatment outcomes.

The current study has notable strengths and some limitations. The sample is exceptionally diverse with respect to IQ, race/ethnicity, and income; these strengths bolster the generalizability of our current findings. However, important considerations regarding representation nonetheless remain given evidence that missing data were more common for younger children and those with greater ASD symptoms. Future studies would also benefit from consideration of additional covariates (e.g., height/weight, blood pressure). Although our sample size is sufficient for the analyses conducted, replication in larger samples would further support confidence in our findings and would permit consideration of additional factors in the context of complex three-way interactions. For example, previous findings with this sample suggest the important moderating role of sympathetic activity on sleep-behavior associations in children with ASD (Schiltz et al., 2022) as well as meaningful parasympathetic-sympathetic interactions in prediction of behavioral functioning in this population (Fenning et al., 2019). Therefore, future studies may wish to explore additional physiological indices (e.g., electrodermal activity) in conjunction with indicators of socioemotional functioning and/or other environmental or behavioral factors (e.g., observation of arousal or regulation) that may contribute to sleep disruption in ASD. Additionally, this study relied upon a parent-reported metric of sleep, which is commonly employed and known to correspond with actigraphy measures in ASD (Veatch et al., 2016); future work would benefit from use of a multi-method approach to further enhance understanding. The cross-sectional design of the study limits conclusions regarding causality. Future studies would benefit from examining longitudinal links between RSA and sleep to test directionality of these effects and explore potential bidirectionality. Consistent with the current conceptualization, it may be that physiological arousal underlies sleep problems, but also, as supported by recent research (McCrae et al., 2021), sleep may impact physiological regulation.

The present study is the first to examine and identify interactions between daytime RSA-B and RSA-R in relation to sleep problems and duration in children with ASD. Results suggest the intriguing possibility that the combination of high RSA-B and high RSA-R may confer resilience against sleep problems for children with ASD. As such, fostering optimal physiological regulation may be an important avenue to reduce sleep problems among children with ASD, a group at considerable risk for sleep disruption and its sequelae.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

ETHICS STATEMENT

All study procedures were prospectively reviewed and approved by the institutional review board of the

California State University, Fullerton, and conform to recognized ethical standards (e.g., the Declaration of Helsinki).

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