Children With Autism: Sleep Problems and Symptom Severity

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Abstract

Relationships between the specific sleep problems and specific behavioral problems of children with autism were evaluated. Mothers' reports of sleep habits and autism symptoms were collected for 109 children with autism. Unlike previous research in this area, only children diagnosed with autism without any commonly comorbid diagnoses (e.g., intellectual disability, epilepsy) were included in the analysis. Consistent with prior work, a positive correlation between the severity of sleep problems and the severity of autism symptoms was obtained. Sleep onset delay and sleep duration were positively correlated with autism symptoms and autism severity. Sleep onset delay was the strongest predictor of communication deficit, stereotyped behavior, and autism severity. These results provide support for specific sleep problem and symptom relationships that are unique to autism and suggest the importance of including the treatment of sleep problems as part of a comprehensive behavioral intervention for children with autism.

Keywords

autism, sleep problems, stereotyped behaviors, communication, social interaction

Children with autism are reported to have a greater prevalence of sleep problems than typically developing children (e.g., Hoffman, Sweeney, Gilliam, & Lopez-Wagner, 2006; Schreck & Mulick, 2000). Parents' reports suggest that 15% to 35% of typically developing children experience sleep problems (Mindell, 1993), with the respective rate for children with autism ranging from 44% to 83% (Richdale, 1999). In a study of young children aged 3 to 5 years, Krakowiak, Goodlin-Jones, Hertz-Picciotto, Croen, and Hansen (2008) suggested that serious sleep problems exist in 53% of children with autism, compared with 46% of children with other developmental disabilities (DDs), and only 32% of typically developing children. These authors also found that children with autism were reported to experience more difficulties with sleep onset and night wakings than children in the comparison groups. Previously, researchers found that children with autism of various ages experience a variety of sleep problems, such as prolonged sleep onset, decreased time asleep, and frequent night wakings (e.g., Allik, Larsson, & Smedje, 2006; Honomichl, Goodlin-Jones, Burnham, Gaylor, & Anders, 2002; Wiggs & Stores, 2004; Williams, Sears, & Allard, 2004).

Maladaptive sleep patterns also are evidenced as a detriment to daytime behavior and cognition in typically developing children (National Institutes of Health, 2003). Disturbed sleep is associated with diminished performance in typically developing children's functioning in areas such as memory, vigilance, and affect (Kheirandish & Gozal, 2006; Sadeh, Gruber, & Raviv, 2002). Significant cognitive delays in higher mental functions, such as verbal fluency and abstract thinking, are displayed in typically developing children following just one night of sleep deprivation (Randazzo, Meuhlbach, Schweitzer, & Walsh, 1998). Furthermore, children's sleep-disordered breathing is suggested to be associated with higher rates of inattention, hyperactivity, and aggression (Chervin et al., 2002; Gottlieb, Vezina, & Chase, 2003).

Symptoms for a variety of disorders may be intensified when sleep problems are present. For example, intensified symptoms of attention-deficit/hyperactivity disorder (ADHD) are associated with the sleep problems of these children, with symptoms appearing to intensify when sleep problems were reported (Corkum, Tannock, Moldofsky, Hogg-Johnson, & Humphries, 2001). A sample of children with the general diagnosis of intellectual disability (ID) demonstrated worsened cognitive dysfunction when severe sleep problems were present (Didden, Korzilius, van Aperlo, van

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Overloop, & de Vries, 2002). Evidence also suggests that intensified problem behavior is associated with sleep problems in children with pervasive developmental disorders (PDD; Patzold, Richdale, & Tonge, 1998). DeVincent, Gadow, Delosh, and Geller (2007) documented differential sleep–behavior relationships for young children with PDD and for typically developing children. Both groups of children were reported to have higher rates of problem behavior when sleep problems were concurrently reported. Specifically, all children with sleep problems reportedly showed more aggression, hyperactivity, distraction, and defiance than children who did not experience sleep problems. However, children with PDD were found to exhibit more overall severe sleep disturbance and inappropriate behavior than typically developing children. These findings collectively suggest a strong relationship between sleep problems and behavioral symptoms seen in children with a variety of DDs. The notion that sleep problems exacerbate daytime symptoms is possible but unclear based on the reported correlations. Notably, the sleep–symptom relationships described above represent behavioral or medical diagnoses that are either highly comorbid or diagnostically inherent to a diagnosis of autism.

Research has been conducted with regard to the relationship between sleep problems and children's autism symptoms. In 2004, Schreck, Mulick, and Smith used the Behavioral Evaluation of Disorders of Sleep (Schreck & Mulick, 2000) and the *Gilliam Autism Rating Scale* (GARS; Gilliam, 1995) to examine the potential relationship between parents' reports of their children's sleep problems and autism symptoms. The GARS subscales represented the triad of symptom severity for stereotyped behavior, communication, and social interaction, as well as developmental disturbance. Overall, autism severity was represented by the autism index (AI), derived from the cumulative scores on the aforementioned GARS subscales. Parents' reports of sleep problem severity were found to be positively correlated with their reports of their children's autism symptom severity. In addition, short sleep duration was found to be the best sleep problem predictor for overall autism severity and also predicted stereotyped behavior and social skills deficits subscales scores. Both the Expressive Awakening and High Sensitivity to Environmental Stimuli subscales were related to children's communication deficit. These findings are interpreted to suggest that sleep problems may exacerbate autism symptoms and that there are specific relationships found among these sleep problems and autism symptoms.

Hoffman et al. (2005) also used parent reports on the GARS (Gilliam, 1995) to assess severity of autism symptoms in a larger sample of children with autism. To measure sleep problems, the researchers used the widely used Children's Sleep Habits Questionnaire (CSHQ; Owens, Spirito, & McGuinn, 2000). In line with the previous research,

significant relationships between mothers' reports of their children's sleep problems and their report of their children's autism severity were found. Results indicated that sleep-disordered breathing was positively correlated with overall severity of autism scores as well as GARS subscales assessing stereotyped behavior and social interaction deficits. In addition, parasomnias (such as night terrors) were found to predict developmental disturbance. The authors suggested that future research in this area should endeavor to control for comorbid DD diagnoses, which may confound the sleep–autism symptom relationships. Similarly, in a meta-analysis, Richdale and Schreck (2009) recommended that future studies use controlled sampling that excludes the various DD diagnoses that are related to autism.

The aim for the current study was to examine the relationships between sleep problems and autism symptoms in a sample of children with a diagnosis of autism but without any comorbid DD diagnoses. Given that a variety of DDs, such as ID (Richdale, Francis, Gavidia-Payne, & Cotton, 2000), ADHD (Corkum et al., 2001), and epilepsy (Cortesi, Giannotti, & Ottaviano, 2005), are commonly comorbid with autism and that these disorders present their own repertoire of related sleep problems (Matson & Nebel-Schwalm, 2005), controlling for comorbid conditions was expected to derive a more accurate assessment of the relationship between sleep problems and symptom severity in children with autism. Given the exploratory design and novel sampling method of the current study, it was expected that a relationship between sleep problem severity and symptom severity would be replicated and that relationships between other sleep problems and autism symptoms would be identified.

Method

Participants

Participants consisted of mother volunteers who were recruited through a treatment and research program. Mothers reported on their children who were independently diagnosed with autism by independent professionals, such as pediatricians and licensed clinical psychologists, which qualified them for state-provided behavioral services. The independent diagnoses were not conducted by any person involved in the current study. All of the participants received treatment at a center-based behavior modification and parental education program located on the campus of an inland southern California university. As consumers of the program, families attended 2.5 hr weekly sessions, with parents attending a counselor-led support group while their children with autism received one-on-one behavioral treatment. The local California State Regional Center provides funding for families to receive such services (California Department of Developmental Services, 2008). The protocol

for the current investigation was approved by the Institutional Review Board of the university campus where the study was conducted.

Mothers' reports on the GARS–Second Edition (GARS-2; Gilliam, 2006) and the CSHQ were extracted from a larger archival data set consisting of data collected from families who volunteered to participate in the center's research program. A total of 109 children (18 females and 91 males) out of 402 met the criteria of having an independent diagnosis of autism with no other comorbid DDs or epilepsy. Notably, the sample did not include children with PDD-not otherwise specified (PDD-NOS) or Asperger syndrome. The ages for the children in the present study ranged from 3 to 18 years ($M = 7.06$, $SD = 2.67$). The children's ethnicities were reported as follows: African American/Black, $n = 10$ (9.3%); Asian/Pacific Islander, $n = 10$ (9.3%); Hispanic, $n = 25$ (23.1%); Middle Eastern, $n = 1$ (0.9%); Native American, $n = 2$ (1.9%); White/Caucasian, $n = 42$ (38.9%); and Mixed or Other, $n = 16$ (14.8%).

Measures

CSHQ. The CSHQ is a parent-report survey used to identify behavioral and medical sleep habits and problems in school-aged children (Owens et al., 2000). The measure includes 45 items that represent prominent sleep difficulties and prevalent diagnostic symptom severity for pediatric sleep disorders (American Sleep Disorders Association, 1990). The CSHQ consists of eight subscales of sleep problems frequently experienced by children (Owens et al., 2000): Bedtime Resistance, Sleep Onset Delay, Sleep Duration (e.g., not getting enough hours of sleep per night), Sleep Anxiety, Night Wakings, Parasomnias (e.g., sleep apnea, night terrors, restless leg syndrome), Sleep-Disordered Breathing, and Daytime Sleepiness. These subscales generate a total sleep disturbance score. Mothers were asked to respond to questions while focusing on their children's most recent and typical week of sleep. Frequencies of certain sleep behaviors displayed by the child are indicated using a 3-point scale: *usually* (5–7 nights per week), *sometimes* (2–4 nights per week), or *rarely* (0–1 night per week). Other items, such as usual bedtime and time of waking, require free response by the parent. The CSHQ has proven useful in numerous studies related to children and their sleep problems, including the study of children with autism spectrum disorders (e.g., Hoffman et al., 2005; Honomichl et al., 2002).

GARS-2. The GARS-2 is a parent-report survey used to evaluate autism based on the diagnostic criteria of the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM-IV*; American Psychiatric Association, 2000) and the Autism Society of America's (1994) definition of the disorder. The three subscales included in the GARS-2 are based on the main domains of symptomology of the disorder: Stereotyped Behaviors, Social Interaction, and Communication. The

summation of subscale scores yield the AI, which has a mean of 100 and a standard deviation of 15. The AI score represents an overall estimate of autism severity. According to the GARS-2 test manual (Gilliam, 2006), in a sample of children and adolescents diagnosed with autism ($N = 1,107$), 90% yielded an AI score higher than 85, which indicates a high likelihood of meeting criteria for autism diagnosis. The original version of this measure, the GARS, has been commended as a valid and reliable tool for diagnosis of autism (Filipek et al., 2000; National Research Council, 2001). The GARS-2, used in the current study, includes improvements that were the focus of critique in the earlier form of the measure (Lecavalier, 2005; South et al., 2002). The GARS-2 has been used in several previous studies related to autism symptom severity (e.g., Hoffman et al., 2005; O'Connor & Healy, 2010; Stuart & McGrew, 2009)

For the current study, overall GARS-2 scores for the child participants had a range of 59 to 130 *(M = 93.86, SD = 16.47)*. In this sample, 69% of the children had an AI at or above 85. A total of 30 children who do not use spoken language to communicate did not have a Communication subscale score, as the measure is not sensitive to children who use alternative forms of communication. Therefore, the Communication subscale-related analyses were not representative of children who use sign language, Picture Exchange Communication System (PECS) boards, or other forms of communication beyond verbal speech. Nonetheless, the GARS-2 provides transformation guidelines so that children who communicate nonverbally were still represented in regard to stereotyped behavior, communication, and AI. Limited communication abilities are commonly associated with a lower intelligence quotient and, therefore, ID (e.g., Waterhouse et al., 1996); however, none of the participants in the current study had an independent professional diagnosis of ID.

Procedure

A two-part assessment process preceded enrollment in regular weekly center sessions as part of the behavior and research program described above. This process consisted of behavioral center staff visiting the family's home for a period of approximately 1.5 hr. A trained staff member assisted mothers in their completion of the GARS-2 by explaining the measure and providing clarification of items as requested. Staff did not participate in the actual rating process beyond this level of support to ensure that parents provided an unbiased estimate of their children's behavior. One or two additional staff members observed and interacted with the child to collect baseline behavioral data. Mothers also were presented with information regarding the program and its integrated ongoing research program during the initial session. After being provided with appropriate informed consent and agreeing to participate in the research program, mothers were given a packet of research surveys

Table 1. Pearson Bivariate Correlations for CSHQ and GARS-2 Factors

CSHQ	GARS-2			
	Stereotyped behavior	Communication	Social interaction	AI
Bedtime Resistance	.11	.11	.11	.13
Sleep Onset Delay	.31**	.28*	.40**	.42**
Sleep Duration	.32**	.22*	.33**	.38**
Sleep Anxiety	.08	−.05	.11	.09
Night Wakings	.11	.16	.26**	.15
Parasomnias	.17	.28*	.21*	.26**
Sleep-Disordered Breathing	.15	−.04	.19*	.14
Daytime Sleepiness	.10	.19	.11	.14
Total sleep disturbance score	.24*	.25*	.22*	.32**

Note: CSHQ = Children's Sleep Habits Questionnaire (Owens, Spirito, & McGuinn, 2000); GARS-2 = *Gilliam Autism Rating Scale–Second Edition* (Gilliam, 2006). *N* = 109.
*$p < .05$. **$p < .01$.

and questionnaires, including the CSHQ, to complete and return to the center at their earliest convenience. The anonymity (and confidentiality) of these measures was assured for participants, as response packets were identifiable only by a predetermined code number.

Data Analysis

All data were entered separately by two research assistants for error control. Specialized syntax from manuals for the utilized measures were used to compute all scores (Gilliam, 2006; Owens et al., 2000). All data collection and analyses were performed using the Statistical Package for the Social Sciences (2006).

Pearson correlations were performed on scores for the CSHQ and the GARS-2 to examine relationships between sleep problems and autism symptomology. Further analysis was performed using a simultaneous multiple regression model to examine CSHQ sleep problem variables as predictors for each of the GARS-2 subscales as well as for AI scores (the criterion measures). Sleep duration and daytime sleepiness were not used as predictors in the analyses because of their likelihood of being outcomes of actual sleep problems, and not necessarily the sleep problem itself (see Hoffman et al., 2005). Total sleep disturbance was excluded from the analyses as this score comes from and is, therefore, highly correlated with all the other CSHQ subscales. These regressions were further analyzed for partial correlations to reveal which specific sleep problems showed the strongest predictive value for the criterion variables while controlling for the variance of all other predictor variables.

Results

To ensure the integrity of the statistical analyses, the data were screened for missing values, skewness, and kurtosis.

The series mean was imputed for all missing values on the CSHQ subscales; the number or missing values ranged from 3 participants (2%, Sleep Onset Delay) and 21 participants (19%, Bedtime Resistance). Mean imputation is a debated statistical method, yet is appropriate for the current study given that the missing data appear to be nonsystematic and the total number of missing values of any given subscale is relatively low (Acock, 2005). Mean imputation was not performed for the Communication subscale of the GARS-2 given that the absence of a value for this variable indicates that the child possesses no oral communication and, therefore, no transformation could provide a meaningful estimation of their communication abilities. Only one variable (the Sleep-Disordered Breathing subscale of the CSHQ) yielded kurtosis and skewness estimates that slightly exceeded normality assumptions. However, transforming this variable did not result in a change among the correlation and regression values reported below, so the originally reported values were left intact.

Correlations between the eight CSHQ subscales and the three GARS-2 subscales and AI score were calculated. The results of the Pearson bivariate correlations are displayed in Table 1. Sleep Onset Delay, Sleep Duration, and Total Sleep Disturbance were significantly correlated with all autism symptom subscales and overall AI. The Parasomnias subscale was significantly correlated with Communication, Social Interaction, and AI, but not the Stereotyped Behaviors subscale. Finally, Night Wakings and sleep-disordered breathing were significantly correlated with social interaction scores alone but none of the other GARS-2 subscales.

The significant results of the regression model are displayed in Table 2. Separate multiple regression analyses were performed to evaluate CSHQ subscales as predictors and each of the GARS-2 subscales and AI score as the criterion variables. Statistically significant overall regression models were evidenced for each of the four analyses. The

Table 2. Significant Regression and Partial Correlation Values for CSHQ Predictors of GARS-2 Criterion Measures

GARS-2 criterion measures	Overall regression		Significant CSHQ predictors	Partial correlation
	F	R²		
Stereotyped behavior	2.23*	.12	Sleep Onset Delay	.27**
Social interaction	5.05***	.23	Sleep Onset Delay	.38***
			Night wakings	.22*
Communication	2.79*	.19	Sleep Anxiety	.26*
			Parasomnias	.27*
Autism index	4.42***	.210	Sleep Onset Delay	.37***

Note: CSHQ = Children's Sleep Habits Questionnaire (Owens, Spirito, & McGuinn, 2000); GARS-2 = *Gilliam Autism Rating Scale–Second Edition* (Gilliam, 2006). *N* = 109.
*$p < .05$. **$p < .01$. ***$p < .001$.

included group of CSHQ subscales was shown to significantly predict the magnitude of each of the criterion variables: Stereotyped Behaviors, $F(6, 108) = 2.23$, $p < .05$, $R^2 = .12$; Social Interaction, $F(6, 108) = 5.05$, $p < .001$, $R^2 = .23$; Communication, $F(6, 78) = 2.79$, $p = .05$, $R^2 = .19$; and AI, $F(6, 108) = 4.42$, $p < .001$, $R^2 = .21$.

Subsequent partial correlation analyses were conducted to assess the value of each discrete CSHQ subscale on the criterion GARS-2 subscales. When controlling for all of the variance contributed by other sleep problems, Sleep onset delay ($pr = .27$, $p < .01$) attained the strongest predictive value of the Stereotyped Behaviors subscale. Similarly, sleep onset delay ($pr = .38$, $p < .001$) was shown to be the strongest predictor of the Social Interaction subscale, although night wakings ($pr = .22$, $p < .05$) also emerged as a significantly strong predictor of this criterion variable. Furthermore, sleep anxiety ($pr = -.23$, $p < .05$) and parasomnias ($pr = .27$, $p < .05$) were evidenced as the strongest predictors of the Communication subscale when all other sleep problems were controlled for. Finally, and consistent with the regression models with the Stereotyped Behaviors and Social Interaction subscales as criterion variables, sleep onset delay ($pr = .37$, $p < .001$) was shown as the strongest predictor of AI score or overall autism severity.

Discussion

As anticipated, the current findings are consistent with previous research in that relationships between children's sleep difficulties and their behavioral symptoms were obtained. The results also present new information on precisely how sleep symptoms relate to the severity of autism symptoms. Mothers' reports in the current study, unlike previous work, reflected the sleep problems and symptom severity of children with autism and no comorbid DD diagnoses. The results of the current study are interpreted to corroborate findings of previous researchers to conclude that mothers'

reports of the severity of their children's sleep problems were related to their reports of their children's autism symptom severity. Unprecedented findings were revealed in regard to the specific relationships between sleep problems and symptoms, which was not unexpected given that the current methodological framework was designed to suggest relationships that are unique to autism without comorbid DD diagnoses.

Overall sleep disturbance, problems with sleep onset delay, and short sleep duration were associated with all autism symptoms as well as overall autism severity. These findings suggest that overall lack of sleep is associated with a more severe repertoire of autism symptoms during the day, a finding that is consistent with results obtained with broader samples of children with other DDs (Schreck et al., 2004). Much as the functioning of typically developing children suffers from lack of sleep, children with autism suffer from an increase in stereotyped behavior, social deficits, and communication deficits when sleep is limited.

Sleep onset delay was evidenced as a predictor of stereotyped behavior, social interaction deficit, and overall autism severity, but not as a predictor of communication deficit. Similar to the aforementioned findings regarding restricted sleep duration, the prevalence of sleep onset delay surely also denotes that the children with autism were not getting enough sleep because they were unable to fall asleep for several hours. Therefore, these findings suggest that overall lack of sleep in children with autism, which may be commonly caused by sleep onset delay, may contribute to the intensification of most autism symptoms. It is problematic to draw implications from sleep onset delay's failure to predict communication deficits, given that children without verbal abilities are excluded from the autism severity measure used in the study. It is possible that this methodological concern as well as the truncated range of communication data complicated these results. Nevertheless, the significance of sleep onset delay in relation to the other symptoms further supports this particular sleep problem as a possible intensifier of daytime autism symptoms. The prominence of sleep onset delay as a problem experienced by children with autism has been found in a study that included children with a variety of ASD (Krakowiak et al., 2008) and another study that focused on children with ID and autism (Cotton & Richdale, 2006), but not in previous work that included children with autism and other comorbid DDs (Hoffman et al., 2005).

Parasomnias were significantly related to Stereotyped Behaviors, Communication, and overall autism severity. These associations are consistent with findings for populations with autism and other comorbidities, as is the finding that parasomnias were a significant predictor of communication symptoms; however, previous researchers also found parasomnias to be a predictor of overall autism severity (Hoffman et al., 2005). It has been questioned

whether parasomnias are indeed a sleep disorder particular to autism or whether their presence in children with autism is a problem associated with comorbid disorders (Hoffman et al., 2005). According to the current results, parasomnias are present in children with a sole diagnosis of autism and are related to communication deficits. Further study of specific parasomnias that children with autism experience, such as restless leg syndrome and night terrors, is warranted.

Other specific sleep problem–autism symptom relationships were found. Night wakings were associated with communication symptoms. In regard to prediction value, night wakings were demonstrated as a predictor of social interaction symptoms. Night wakings have not been previously noted in significant associations with or as a significant predictor of autism symptoms. Night wakings result in overall less sleep throughout the night, and, as stated above, these results point to a general lack of sleep as an important factor in symptom severity.

A similar pattern of results was shown with sleep-disordered breathing; this sleep problem was associated with communication symptoms. These findings were different from previous findings, where sleep-disordered breathing was indicated as the most prominent associative and predictive factor of all autism symptoms (Hoffman et al., 2005). This suggests that sleep-disordered breathing is not as relevant to populations with autism as it is to populations with other DDs. Sleep-disordered breathing has been shown as a major sleep problem for children with epilepsy and children with ID, both conditions that are often comorbid with autism (Maganti et al., 2006; Wiggs, 2003). Consequently, we believe that the sampling methods of the current study avoided the confounds that may result from a diagnostically diverse sample of children with autism.

Finally, sleep anxiety was a significant predictor of communication symptoms. Specifically, less severe communication symptoms were associated with higher sleep anxiety. This relationship may well be attributable to children with better communication abilities; children with verbal abilities can adequately communicate their fears or anxiety about sleep to their parents. Conversely, children with nonverbal, limited verbal, or otherwise impaired communication abilities were unlikely able to alert their parents to sleep anxiety problems. Parents may interpret sleep anxiety as noncompliance or other forms of problem behavior. Unlike the obvious and observable problem of their children not sleeping, sleep anxiety may be an internalized sleep problem that is reported only by parents who have children who are capable of communicating with them. Relationships between anxiety and sleep problems have been noted in a previous sample of children with various autism spectrum disorders (Wiggs & Stores, 2004) and a sample of children with Asperger syndrome (Paavonen et al., 2008). Past work, in combination with the current findings, cannot help clarify whether sleep anxiety is a sleep problem that is unique to children with

verbal abilities, who also are more likely to have higher intellectual abilities (Ritvo & Freeman, 1978), and further study is warranted.

According to the findings of this study, the prolonged time needed for children with autism to fall asleep and their overall lack of sleep may intensify their symptoms in most symptom areas. Therefore, it is important to consider how to treat these sleep problems in this population. Sleep hygiene has been suggested as a behavioral method of improving sleep for typically developing children and children with DDs (Hoban, 2000). These methods include parental involvement in establishing bedtime routines, a sleep-conducive environment, and being wary of giving the child any unintentional reinforcement for staying awake. Indeed, positive behavioral strategies have been shown to decrease night waking and overall problematic sleep experienced by children with autism (Christodulu & Durand, 2004). The necessity of empirically based sleep intervention is especially important given that parent accommodation of maladaptive sleep patterns has been cited as strong predictors of sleep problems for children with autism (Liu, Hubbard, Fabes, & Adam, 2006), which may exacerbate mothers' stress and personal sleep problems (Hoffman et al., 2008). Furthermore, pharmacological treatments, such as melatonin, are supported as effective sleep treatment for many children with autism (for a review, see Guénolé et al., 2011).

These options for alleviating and assessing sleep problems should be considered as part of an overall treatment plan, given the evidence that sleep problems exacerbate daytime behavioral symptoms of children with autism, which may in turn affect response to treatment and various other activities of daily living. Sleep problems, like physical pain or emotional distress, could be considered a "setting event" that can trigger problem behavior for children with autism (Carr, 1992). If sleep problems are weighted as a setting event for autism symptoms, a more comprehensive and effective individually tailored treatment program can be made for each child with autism.

Furthermore, future research in this field requires more in-depth inspection of the actual sleep problems and symptoms of children with autism. The current and many related studies have relied on parent reports; less subjective measures could enhance our knowledge in this field. Use of actigraphy to physiologically measure the children's sleep might be helpful in identifying sleep habits in a nonsubjective and noninvasive manner (Allik et al., 2006; Oyane & Bjorvatn, 2005). Wearing an actigraphic wristband has proven an effective measure of sleep problems in samples of children with autism (Goodlin-Jones et al., 2009; Schwichtenberg, Iosif, Goodlin-Jones, Tang, & Anders, 2011; Wiggs & Stores, 2004) and is an appealing approach to sleep measurement with this population, as children with autism often have heightened sensitivity to sensory stimuli that makes invasive measurements inoperable. Furthermore, polysomnographic techniques

combined with video monitoring have been an effective means of measuring sleep problems for children with autism (Elia et al., 2000; Malow et al., 2006). In addition to these types of sleep measurements, a more objective and observational means of measuring children's symptoms would greatly benefit this area of research. In addition to improvements on sleep measurement in children with autism, the need for multiple indicators of specific diagnoses should be addressed in future research. Here, professional independent diagnoses were used to define our sample, which is more objective than the typical parent-report approach to diagnosis. Nevertheless, multiple professional and standardized sources that corroborate on specific autism and comorbid diagnoses, as well as medication regimens, are keys for understanding the differential relationships between sleep and psychopathology.

In conclusion, we suggest that children with autism experience specific sleep problem–autism symptom relationships that have not been evidenced in previous research and may not exist in populations with other diagnoses that are commonly comorbid with or inherent to autism, which is of great import to the clinical issues and future research related to this field. Further research examining these relationships, using multiple indicators of the disorder as well as more objective indices of children's sleep difficulties, are suggested. The results of the current study evidence the importance of treatments that aid in the research that focuses on the sleep health of children with autism, with direct consideration of the pattern of individual sleep problems and unique symptom severity of each child. Such information is invaluable, as any means that assist children with autism to sleep better could, in turn, help reduce symptom severity. Therefore, targeting sleep problems as part of intervention plans may increase interaction and response to intervention, and improve the quality of life for children with autism and their family members.

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Declaration of Conflicting Interests

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