Sensory Processing, Physiological Stress, and Sleep Behaviors in Children With and Without Autism Spectrum Disorders

Stacey Reynolds, Shelly J. Lane, Leroy Thacker

key words: hypersensitivity, neuroendocrine, electrophysiology

ABSTRACT

Sleep problems have been frequently identified in children with autism spectrum disorders (ASD). It has been proposed that some sleep problems are due to sensory sensitivity. The purpose of this study was to examine the relationship between physiologic responses to sensation and sleep in children with and without ASD. Fifty-five children participated in the study (ASD, n = *27; typical,* n = *28). All children participated in a sensory challenge laboratory protocol. Electrodermal reactivity and salivary cortisol were used as physiological indicators of sensory responsivity. Behavioral data were collected using the Sensory Profile and the Child Behavior Checklist. Results confirmed that children with ASD have a higher prevalence of atypical sensory behaviors and sleep disturbances than typical children. Behavioral and physiological measures were able to predict good sleepers versus poor sleepers with 85.7% accuracy, suggesting that atypical sensory behaviors are important to consider in relation to sleep deficits in children.*

Children with autism spectrum disorders (ASD) are known to demonstrate both physiological and behavioral responses to sensory stimulation that are different from typically developing peers (Ben-Sasson et al., 2009; Reynolds & Lane, 2008; Schoen, Miller, Brett-Green, & Nielsen, 2009). In addition, a high percentage of young children with ASD experience sleep disorders in the form of frequent night-wakening or difficulty getting to sleep (Krakowiak, Goodlin-Jones, Hertz-Picciotto, Croen, & Hansen, 2008). Although the interaction between sensory modulation and sleep has not been studied in children with ASD, sensitivity to sensory stimuli has been suggested as a contributing factor to sleep problems (Milner, Cuthbert, Kertesz, & Cote, 2009; Shani-Adir, Rozenman, Kessel, & Engel-Yeger, 2009). Both sleep and sensory modulation have been linked to overall arousal and release of the stress hormone cortisol (Dahl, 2007; Dahl & Lewin, 2002; Reynolds, Lane, & Gennings, 2010; Scher, Hall, Zaidman-Zait, & Weinberg, 2010; Schoen, Miller, Brett-Green & Nielsen, 2009). This study investigated the relationship between responses to sensory stimuli and sleep quality in children with ASD, and explored variables predictive of good sleepers versus poor sleepers. We posited that higher levels of sensory sensitivity, higher physiological arousal, and higher nighttime levels of salivary cortisol

Stacey Reynolds, PhD, OTR/L, is Assistant Professor, Department of Occupational Therapy, School of Allied Health Professions, Virginia Commonwealth University, Richmond, Virginia, and Assistant Professor and K12 Scholar, Department of Occupational Therapy, School of Public Health and Health Professions, University of Florida, Gainesville, Florida. Shelly J. Lane, PhD, OTR/L, FAOTA, is Professor, Department of Occupational Therapy, and Assistant Dean for Research, School of Allied Health Professions, Virginia Commonwealth University, Richmond, Virginia. Leroy Thacker, PhD, is Assistant Professor, Department of Biostatistics, School of Medicine, Virginia Commonwealth University, Richmond, Virginia.

Originally submitted October 18, 2010. Accepted for publication March 14, 2011. Posted online May 20, 2011.

The authors have no financial or proprietary interest in the materials presented herein.

Address correspondence to Stacey Reynolds at reynoldsse3@vcu.edu.

10.3928/15394492-20110513-02

would strongly predict sleep problems identified in children with and without ASD.

Sleep and Development

Sleep is a vital function for all living creatures; not merely a state of shut-down, but as a state of activity that serves to facilitate maturation, reorganization, and restoration (Breedlove, Rosenzweig, & Watson, 2007). From an ontogenic perspective, sleep patterns vary throughout development with more sleep needed during critical periods of brain growth. During the first 2 years of life, children spend more hours asleep than awake (7:5 ratio) (Dahl, 2007) and it is during this period of time in early childhood that neurological connections are formed in the central nervous system, providing the foundation for all future cognitive, sensory-motor, and social emotional development. As such, from an evolutionary perspective, sleep has not only retained its status as a vital occupation but has proven to be critical in the establishment of all other areas of development (Dahl, 2007).

It has been recognized that sleep has a bidirectional or cyclical relationship with behaviors and events that occur while the individual is awake. Sleep quality can influence observable behaviors in children such as attention, cognitive performance, motor coordination, and certain aspects of executive functioning (Malow et al., 2006; Moran, Carvalho, Prado, & Prado, 2005; Paavonen et al., 2010; Tucker, Whitney, Belenky, Hinson, & VanDogen, 2010), and children with behavioral and psychological disorders have higher rates of sleep problems (for review see Alfano & Gamble, 2009). Further events, particularly emotionally salient events that occur during the day, may influence the ability to fall asleep and the overall quality of sleep (for review see Vandekerckhove & Cluydts, 2010). Therefore, sleep is an important daily life task that should be examined in the context of the individual's overall behavioral and physiological functioning.

Sleep in Autism

Although sleep disturbances occur in 20% to 40% of the general pediatric population, sleep disturbances in ASD have been estimated at 40% to 80%, indicating that this is a significant area of concern for this population (Krakowiak et al., 2008; Richdale, 1999). Some of the most common sleep difficulties reported by parents include the child waking often during the night or waking too early in the morning, restlessness during sleep, and difficulty falling asleep (Krakowiak et al., 2008; Mayes & Calhoun, 2009). Children with ASD who are poor sleepers also demonstrate more significant affective problems and greater difficulty with reciprocal social interaction (Malow et al., 2006). Mayes and Calhoun (2009) found that sleep problems increased with severity of autism symptoms, and suggested that sleep disturbance is part of the overall autism symptom complex.

Sensory Modulation in Autism

Although not currently identified as a core deficit in the diagnosis of ASD, atypical responses to sensory stimulation have been widely reported in this population. According to Dunn (1999), atypical responses to sensory stimulation can be classified according to neurological threshold and corresponding behavioral responses to stimuli. Based on this model, individuals with a low neuronal threshold will be more sensitive to sensation and will either have an exaggerated behavioral response when faced with unpleasant stimuli (sensory sensitive) or attempt to avoid sensations or environments deemed noxious (sensory avoiding). Conversely, individuals with a high neuronal threshold may either require a higher intensity or frequency of input to register the sensation (low registration), or seek out additional sensory input to maintain optimal levels of arousal (sensory seekers). According to parent report, children with ASD demonstrate behaviors associated with both high and low sensory thresholds, sometimes in combination (Baranek, David, Poe, Stone, & Watson, 2006; Leekam, Nieto, Libby, Wing, & Gould, 2007).

The term sensory modulation disorder (SMD) is currently being used to encompass the range of atypical behavioral responses to sensory stimulation (Miller, Anzalone, Lane, Cermak, & Osten, 2007). Although SMD and ASD are separate conditions (Reynolds & Lane, 2008; Schoen et al., 2009), 60% to 90% of children with ASD have been shown to have co-occurring SMD (Baranek et al., 2006; Leekam et al., 2007). It has been suggested that under-arousal is the more common pattern of sensory behavior observed in ASD (Ben-Sasson et al., 2009). Schoen et al. (2009) used electrodermal reactivity (EDR) as a measure of sympathetic nervous system functioning to examine baseline levels of arousal and responses to sensory stimulation in a laboratory setting. Their results showed that children with ASD had lower arousal and lower physiological stress reactivity to sensory stimuli compared to children with SMD only and typical children, providing preliminary support for the behavioral characterization of underresponsiveness in ASD.

An alternate means of measuring central nervous system response to stimuli examines neuroendocrine functioning of the hypothalamic pituitary adrenal

axis reflected in salivary cortisol. Children with ASD, but not typical children, have been shown to have elevations in the salivary cortisol in response to novel stimuli (Corbett, Mendoza, Abdullah, Wegelin, & Levine, 2006) and sensory sensitivity has been associated with higher afternoon levels of salivary cortisol (Corbett, Schupp, Levine, & Mendoza, 2009). Taken together, these results suggest that (1) sympathetic and neuroendocrine functioning may be disrupted in children with ASD and (2) that these disturbances may be related to sensory modulation. A better understanding of the mechanisms is warranted.

Sensory Modulation and Sleep

It has been proposed that some sleep problems may be related to difficulties with sensory modulation, particularly when children tend to be easily over-aroused by sensory stimuli. Shochat, Tzischinsky, and Engel-Yeger (2009) found that, in typically developing school children, tactile sensitivity was a significant predictor for sleep difficulties and that both tactile sensitivity and sensation seeking were significant predictors for behavioral problems. Similarly, Shani-Adir et al. (2009) found that sensory hypersensitivity was correlated with lower sleeping quality in children with atopic dermatitis. Supporting both of these behavioral studies, electroencephalographic research has shown that sensory gating impairments (P50 sensory gating) are present in poor sleepers during pre-sleep wakefulness (Milner et al., 2009). These authors note that "good sleepers can initiate and maintain sleep by disengaging from the environment in an automatic and effortless manner and can successfully gate irrelevant stimuli" (p. 335). This suggests that for individuals with SMD, sleep may be a more effortful process, possibly stemming from difficulty disengaging from the sensory environment, and that intervention may be needed to develop regular sleep patterns that may prevent secondary behavioral problems from occurring.

Given the high rate of co-occurrence of SMD in the ASD population, we assert that the higher rate of sleep difficulties seen in this population may be related to difficulties in sensory modulation. Specifically, those children with ASD who behaviorally exhibit sensory overresponsiveness may be more prone to sleep difficulties compared to children with ASD who do not have SMD or who are primarily underresponsive. Because sleep is an occupational task in which all children are expected to participate, it was of interest to determine whether we could distinguish poor sleepers from good sleepers within the group as a whole, based on behavioral, physiological, and neuroendocrine responses to sensory stimuli.

The purpose of the current study was to examine the relationship between physiologic responses to sensory stimuli and sleep quality in children with and without ASD, and to determine which variables best distinguish good sleepers from poor sleepers, irrespective of diagnosis. The following hypotheses were established: (1) children with ASD would demonstrate a higher prevalence of both sleep problems and SMD; (2) there would be a high and significant correlation between low threshold areas of SMD (Sensory Avoiding, Sensory Sensitivity) and number of sleep problems; (3) poor sleepers (with and without ASD) would demonstrate higher levels of physiological arousal and physiological reactivity compared to good sleepers; and (4) poor sleepers (with and without ASD) would demonstrate a higher diurnal pattern of salivary cortisol and higher cortisol reactivity levels compared to good sleepers.

Methods

This study was approved by the sponsoring university's Institutional Review Board prior to beginning participant recruitment. A descriptive cross-sectional design was used to assess the relationship between sensory modulation, sleep, and physiological stress in children with and without ASD. Diurnal patterns of salivary cortisol were collected to account for normal daily fluctuations in stress hormones while physiological stress was elicited through a Sensory Challenge Protocol (SCP) in a laboratory setting. Report of sensory and sleep behaviors was acquired through parent questionnaires.

Sample

Participants were recruited through flyers, word-of-mouth, and the Interactive Autism Network. Parents of potential participants were interviewed by telephone prior to enrolling in the study to ensure that children met inclusion criteria. All children in the ASD group (n = 27) were between the ages of 6 and 12 years and were required to have documentation of an ASD diagnosis given by a licensed psychologist or psychiatrist using standardized tools (i.e., the Autism Diagnostic Interview or the Autism Diagnostic Observation Schedule) (Lord, Rutter, & LeCouteur, 1994; Lord, Rutter, DiLavore, & Risi, 2002). Children ages 6 to 12 years old without either ASD or SMD (n = 28) constituted the control (TYP) group. Siblings of children with ASD were excluded from the control group, as were children with identified psychological disorders (e.g., attention-deficit hyperactivity disorder, bipolar disorder, or anxiety disorder). For both groups, children with significant

motor impairments such as cerebral palsy, history of seizures, intellectual disability, or any known endocrine or metabolic dysfunctions were excluded.

All children passing the telephone screening were screened by the examiners for normal intelligence using the Leiter-R non-verbal scale of intelligence; data from children with a non-verbal intelligence quotient (IQ) below 70 were not included in this study. This exclusion criterion was in place to enhance the validity and reliability of physiological data interpretation because children with lower IQs have been shown to have reduced sympathetic activity (Fernhall & Otterstetter, 2003; Nomura, Kimura, Arai, & Segawa, 1997).

Procedures

Following determination of eligibility, parents were mailed a Sensory Profile (Dunn, 1999), the Child Behavior Checklist (CBCL) (Achenbach & Rescorla, 2001), the informed consent and assent, a short demographic questionnaire, and six pre-coded saliva collection tubes with instructions for collecting and storing saliva samples at home. Saliva collection and storage were also discussed with families during the initial telephone conversation. If children were taking psychostimulant mediation, parents were asked to withhold this medication for 24 hours prior to their scheduled laboratory visit.

On the day of testing, parents brought their children, the completed test forms, and the frozen saliva samples to the laboratory. Both parental consent and child assent were obtained prior to proceeding with the session. The SCP was administered following application of electrodes and collection of baseline saliva samples. A detailed description of the entire SCP is published in Miller, Reisman, McIntosh, and Simon (2001). A series of eight sensory stimuli in each of six sensory domains were presented to the child in the following order: Classic Tone, a recorded steady tone played at 78 decibels; Visual, a strobe light set at 10 flashes per second; Auditory, a recorded fire engine played at 84 decibels; Smell, wintergreen oil moved back and forth 1 inch below the child's nose; Touch, a feather manually stroked from the child's right ear along his or her chin to left ear; and Movement, the child's chair electronically tipped back and returned to an upright position. Each stimulus was presented eight times with a variable 10- to 15-second inter-stimulus interval. Timing for the stimuli was monitored through a customized computer program (PsyLab) developed by and purchased from Contact Precision Instruments (Cambridge, MA). Following administration of the SCP, which took approximately 20 minutes, seven additional samples of saliva were collected at 5-minute intervals. During the post-challenge sampling, the children were told to rest while watching a 30-minute cartoon.

Measures

Sensory Profile. The Sensory Profile (Dunn, 1999) establishes scores based on standard deviation (*SD*) from the norm in four quadrants: Low Registration, Sensory Seeking, Sensory Sensitivity, and Sensory Avoiding (Dunn, 2006). Cut scores for quadrants reflect a continuum of sensory modulation abilities: Much Less Than Others (more than 2 *SD* above the mean), Less Than Others (between 1 and 2 *SD* above the mean), Similar to Others (within 1 *SD* of the mean), More Than Others (between 1 and 2 *SD* below the mean), and Much More Than Others (more than 2 *SD* below the mean).

CBCL. The school-age CBCL is part of the Achenbach System of Empirically Based Assessments and was designed for children ages 6 to 18 years (Achenbach & Rescorla, 2001). The CBCL is completed by parents or caregivers who observe the children in their natural environments. Parents are asked to answer questions related to the child's behavior by responding 0 = not true, 1 = somewhat or sometimes true, or 2 = very true or often true. Overall, the CBCL has been shown to significantly discriminate between referred and non-referred children ($p < .01$), and has been deemed acceptable to use across groups of different race, ethnicity, and socioeconomic status (Achenbach & Rescorla, 2001). For this study, we wished to explore parental concerns about sleep. As such, questions from the CBCL that related to sleep quality, duration, or behavior were examined. These questions were: Nightmares, Overtired, Sleeps less than most kids, Sleeps more than most kids, Talks or walks in sleep, and Trouble sleeping. We chose not to include the item Wets the bed because enuresis can have medical etiologies unrelated to general sleep dysfunction.

Salivary Cortisol. Activity of the hypothalamic pituitary adrenal axis has been widely studied through measurement of cortisol. Salivary cortisol has been strongly correlated with serum cortisol (Kos-Kudla, Bunter, Marek, Ostrowska, & Swietochowska, 1996) and presents as an easily administered, non-invasive way to measure stress in children (Hanrahan, McCarthy, Kleiber, Lutgendorf, & Tsalikian, 2006; Schmidt, 1998). Cortisol levels fluctuate throughout the day, indicating a steady diurnal pattern by 1 year of age evidenced by high cortisol on wakening, a steady decrease throughout the day, and a plateau in the late afternoon and evening (Hiramatsu, 1981; Kiess et al., 1995). Elevations in cortisol at any point during the day can be observed in response to stress,

novelty, or perceived threats (Luecken & Appelhans, 2006; Tyrka et al., 2007).

We examined both diurnal and reactive measures of cortisol. Parents collected cortisol at home during two separate days, within 2 weeks prior to coming to the laboratory. Samples were collected in the morning (within 30 minutes of the child waking up), afternoon (between 3 and 4 p.m.), and at night (30 minutes prior to bedtime) on both days. Parents were instructed not to allow their children to eat, drink, chew gum, or brush their teeth for 30 minutes before each home collection time, and to have the children rinse their mouths thoroughly with cold water 5 minutes prior to beginning sample collection. Parents were asked to withhold their child's psychostimulant medication (if applicable) for 24 hours before sample collection to eliminate any potential neurochemical interactions and inter-subject variation. A minimum of 0.5 mL of liquid was collected for each sample by placing a plain (non-citric acid) cotton Salivette (Sarstedt, Newton, NC) under the child's tongue for 60 seconds. Home samples were frozen and then brought in a cooler to the laboratory site, where they were transferred to the laboratory freezer. Parents were asked during the laboratory visit to validate procedures related to data collection; any alterations in collection methods were recorded and reviewed at the time of analysis.

For the laboratory procedures, all children were tested on non-school days and between the hours of 1 and 5 p.m. (van de Wiel, van Goozen, Matthys, Snoek, & van Engeland, 2004). Parents were asked to not have children eat, drink, chew gum, or brush their teeth for 30 minutes before their appointment. The first baseline (B1) cortisol measure was taken on entering the laboratory; a second baseline sample (B2) was taken after the electrodes were applied, the rules explained, and a movie clip (*Apollo 13*) played. Changes in cortisol levels take approximately 5 minutes to register and approximately 18 to 30 minutes to peak in human saliva (Hiramatsu, 1981; Schmidt, 1998). The entire SCP takes approximately 20 minutes to complete. This timeframe permitted the collection of post-challenge data points throughout the period when peak responses would be expected. The first sample was collected immediately following completion of the challenge (0P), and then at 5 (5P), 10 (10P), 15 (15P), 20 (20P), 25 (25P), and 30 (30P) minutes post-challenge. All samples were taken using plain (non-citric acid) cotton Salivette, maintained at room temperature until the child's session was complete, and stored in the laboratory freezer at -20°C until analysis.

Samples were analyzed in duplicate at the General Clinical Research Center at Virginia Commonwealth University using a high sensitivity salivary cortisol enzyme immunoassay kit (HS-Cortisol kit) produced by Salimetrics LLC (State College, PA).

Electrodermal Activity/Reactivity. A measure of eccrine sweat gland activity, EDR reflects sympathetic nervous system responses to sensory stimuli. EDR was assessed during a 3-minute baseline, throughout the SCP, and during a 3-minute recovery. The general method of collection followed the procedures recommended by Fowles et al. (1981) and previously reported by our laboratory (Lane, Reynolds, & Thacker, 2010). Two 5-mm electrodes were applied to the thenar and hypothenar surface of the right hand, secured with double-sided sticky collars, and wrapped with Coban™ (3M™, St. Paul, MN) to ensure that the electrodes remained in place. The electrodes were attached to a PsyLab SC5 coupler, which digitizes skin conductance at the point where electrodes plug into the pre-amplifier, using a 24-bit A-D converter. A constant 0.5-volt potential was applied across the electrode pair. A low-cut filter set to 0.2 Hz was used; signals greater than 0.2 Hz are passed without distortion with respect to amplitude. The signals were sampled at 80 Hz, then digitized and stored on a computer. For each subject, the electrodermal record was visually checked for movement artifact and questionable responses removed. PsyLab measurement software was used for wave detection; specific responses were taken as occurring between 0.8 and 3.999 seconds after the stimulus; responses before and after this window were considered non-specific responses. The amplitude of the peaks was measured from the point at which the skin conductance increases sharply (i.e., baseline) to the point at which the conductance begins to fall (i.e., peak). Only peaks greater than 0.05 micro ohms (Dawson, Schell, & Filion, 1990) were considered valid.

A variety of measurement parameters of EDR have been used to assess tonic and phasic sympathetic activity and response to sensation. In this study, tonic electrodermal activity or skin conductance level was averaged across a 3-minute baseline and during a 3-minute recovery. The phasic, or reactive, variables used included mean response magnitude within each domain (e.g., tactile or olfactory). As is typical for studies evaluating magnitude of skin conductance responses, our magnitude data required logarithmic transformation before analysis (Boucsein, 1992; Dawson et al., 1990).

Statistical Analysis

For all analyses, an alpha level of 0.05 significance level was considered to be significant; no attempt was made to correct for multiple comparisons. The as-

sumption of normality was assessed for all outcome variables and when the assumption was rejected the appropriate non-parametric tests were used. Statistical analyses were conducted using SPSS Version 17.0 (SPSS, Inc., Chicago, IL). Spearman correlation statistics were used to examine the association between variables. When examining group differences for multiple dependent variables, MANCOVA models were used. Because differences in IQs were found between our ASD and typical (TYP) groups, cognition was entered as a covariate in these models as an attempt to control for potential effects on the dependent variables.

Results

Demographic Data

Of 60 children recruited for this study, two were excluded due to low IQ and three due to inability to provide proof of formal autism diagnosis, resulting in a final number of 55. There was no significant difference in age between the ASD and TYP groups, but a significant group difference in non-verbal IQ was identified ($p < .001$) and a higher percentage of children in the ASD group were males compared to the TYP group (Table 1).

Table 1
Demographic Data for Sample

Characteristic	TYP (*n* = 28)	ASD (*n* = 27)
Mean age (mo) (SD)	105.04 (22.9)	104.85 (21.9)
Mean IQ (SD)	112.36 (12.8)	95.88 (17.8)
Gender		
Male	14	23
Female	14	4
Race		
White	21	19
Asian	0	3
African American	3	1
Native American/ Alaska Native	0	1
Mixed	3	3
Ethnicity		
Hispanic/Latino	1	4
Non-Hispanic/ Latino	26	23
Other	1	0

TYP = typical; ASD = autism spectrum disorders; SD = standard deviation; IQ = intelligence quotient.

Frequency of Sleep Problems

Clinical assessment of childhood sleep difficulties generally involves a parent report questionnaire, with queries regarding getting to sleep and staying asleep. In the current study, we used the CBCL, which contains questions similar to those found on most sleep questionnaires. Parents reported whether sleep behaviors were not true, sometimes true, or very true for their child. Overall, children in the ASD group were reported to have a higher frequency of problem sleep behaviors with more very true responses on all questions except for the item related to having nightmares (Table 2).

To enable examination of sleep as an occupational task, ratings were assigned a numeric value (not true = 0, sometimes = 1, very true = 2) and a total sleep index was calculated for each subject by adding scores for each of the six sleep behavior questions. Total sleep index scores ranged from 0 to 6. A Kruskal–Wallis one-way analysis of variance test was performed to examine group differences on the total sleep index. Based on this analysis, children with ASD had significantly higher sleep index scores compared to typical children ($p = .012$)

Frequency of SMD

Quadrant scores on the Sensory Profile were used to examine the frequency of SMD in children

Table 2
Item Level Scores on the CBCL Sleep Questions

	TYP			ASD		
Item	Not True	Sometimes	Very True	Not True	Sometimes	Very True
Nightmares	60.7%	25%	3.6%	65.4%	33.3%	–
Overtired	85.7%	3.6%	–	66.7%	25.9%	3.7%
Sleep less than other children	82.1%	7.1%	–	66.7%	14.8%	11.1%
Sleep more than other children	89.3%	–	–	77.8%	11.1%	3.7%
Talks or walks in sleep	78.6%	10.7%	–	77.8%	11.1%	3.7%
Trouble sleeping	75%	7.1%	7.1%	70.4%	3.7%	18.5%

CBCL = Child Behavior Checklist; TYP = typical; ASD = autism spectrum disorders.

Table 3
Percentage of Scores in Definite Difference (More Than) on the Sensory Profile

Item	TYP	ASD
Low registration	3.6%	55.6%
Sensory seeking	3.6%	33.3%
Sensory sensitivity	–	40.7%
Sensory avoiding	–	51.9%

TYP = typical; ASD = autism spectrum disorders.

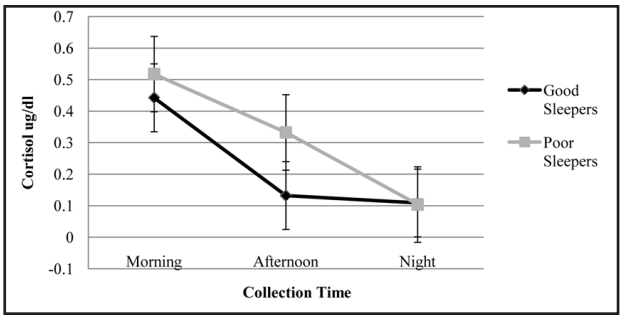

Figure 1. Diurnal patterns of salivary cortisol.

with and without ASD. Percentage of children who scored in the definite difference range (presents this behavior more than other children) for each quadrant were calculated (Table 3). Children with ASD had a higher percentage of definite difference scores in each quadrant of the Sensory Profile and 81% of children in the ASD group scored in the definite difference range in at least one quadrant of the Sensory Profile. A multivariate analysis of covariance (MANCOVA) was used to determine whether children with ASD scored significantly differently on the Sensory Profile compared to typical children. IQ was entered as a covariate in the model to adjust for preexisting group differences in cognition. Results indicated that children with ASD had significantly lower scores (greater dysfunction) across all four quadrants than typical peers ($p < .001$). Cognition did not have a significant effect on the overall model ($p = .610$).

Relationship Between Sleep and Sensory Modulation Behaviors

The relationship between sensory processing and sleep behaviors was examined for the TYP and ASD groups separately and the sample as a whole, using Spearman correlation statistics. Quadrant scores on the Sensory Profile were used as an indicator of the child's pattern(s) of sensory modulation. The sleep index score was used as an indicator of sleep problems. For the TYP group, a relationship between sensory modulation and the total sleep index was found to be significant across all four sensory quadrants: Low Registration ($r = .528$, $p = .008$), Sensory Seeking ($r = .552$, $p = .005$), Sensory Sensitivity ($r = .486$, $p = .016$), and Sensation Avoiding ($r = .537$, $p = .007$). For children with ASD, the relationship between SMD and sleep problems was only significant in the domain of Sensation Avoiding ($r = .502$, $p = .011$). When both groups were examined together, the total sleep index was moderately and significantly correlated to all four quadrants on the Sensory Profile: Low Registration ($r = .466$, $p = .001$), Sensory Seeking ($r = .547$, $p < .001$), Sensory Sensitivity ($r = .504$, $p < .001$), and Sensory Avoiding ($r = .573$, $p < .001$).

Sleep and Physiological Responses to Sensation

Diurnal Patterns of Salivary Cortisol. Although there was a higher incidence of poor sleepers in the ASD group, both groups had children who were classified as poor sleepers. We were interested in examining all poor sleepers in this study to obtain a clearer picture of sleep relative to our measurement parameters. Because both the ASD and TYP groups had children with problem sleep behaviors, we collapsed across diagnostic group and identified "good sleepers" and "poor sleepers." Children whose sleep index score was above the 75th percentile (a score of 3 or higher) were classified as poor sleepers ($n = 10$), whereas children with a sleep index score of 2 or lower were classified as good sleepers ($n = 41$). This grouping included 19 children with ASD and 22 typical children in the good sleeper group, and 7 children with ASD and 3 typical children in the poor sleeper group. Four subjects had incomplete CBCL questionnaires; because a total sleep index could not be calculated, these children were therefore excluded from further analysis.

Diurnal cortisol (morning, afternoon, and nighttime) differences between poor sleepers and good sleepers were examined using MANCOVA, with IQ as the covariate. Only day 2 diurnal data were included in this analysis because several parents in the ASD group reported that they had difficulty getting their children to suck on the cotton swab on the first day of collection, resulting in more missing data and outlier values on collection day 1 versus day 2. The overall MANCOVA model approached significance ($p = .07$); cognition did not have a significant effect on the overall model ($p = .265$). When the variables of morning, afternoon, and nighttime cortisol were examined separately, only afternoon salivary cortisol was significantly different between good sleepers and poor sleepers ($p = .03$). A visual examination of these data (Fig. 1) suggests that higher afternoon

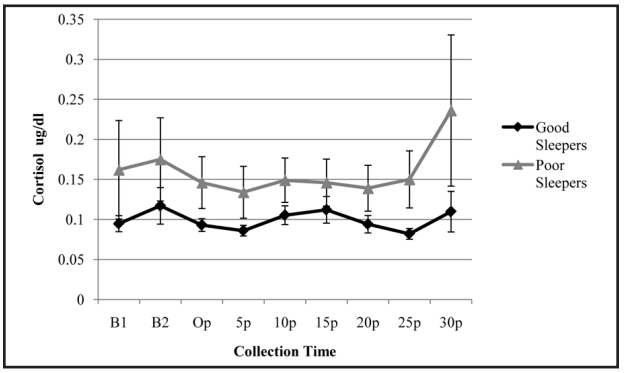

Figure 2. Cortisol levels at baseline and post-Sensory Challenge Protocol. B1 = baseline 1; B2 = baseline 2; 0p to 30p = cortisol measures taken at 0 to 30 minutes post-challenge.

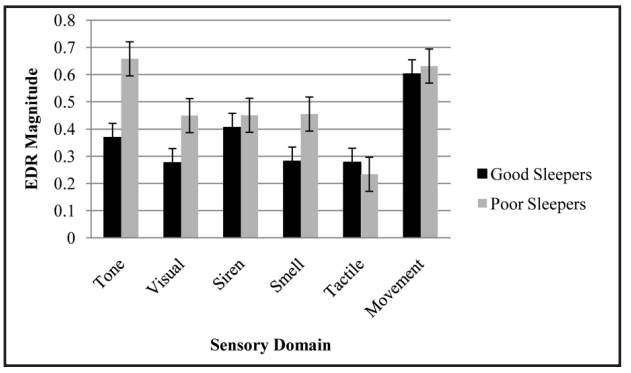

Figure 3. Electrodermal reactivity (EDR) magnitude during the Sensory Challenge Protocol.

levels in the poor sleeper group accounted for relative differences in cortisol pattern; the morning slope was less steep and the afternoon steeper. By nighttime, cortisol had returned to normal.

Cortisol Reactivity. There were no significant differences at baseline (B1, B2) between the good sleeper and poor sleeper groups ($p = .246$). Post-challenge salivary cortisol differences, examined using a MANCOVA with average baseline cortisol and IQ used as covariates, indicated no significant differences between groups ($p = .411$). Cognition did have a significant effect on the overall model ($p = .036$). Interestingly, visual examination of the data (Fig. 2) showed poor sleepers have higher levels at each time point, both prior to and following completion of the SCP, with greater differences apparent at 25 and 30 minutes post-challenge.

EDR. We hypothesized that children in the poor sleeper group would demonstrate heightened skin conductance level at baseline and greater magnitude of EDR during the SCP. The log value of skin conductance level at baseline and the number of non-specific responses during the baseline period were examined using a MANCOVA model (IQ as covariate). No group differences were found at baseline ($p = .304$).

A similar MANCOVA model was applied to examine log values for magnitude of EDR to the different stimuli during the SCP and recovery following the protocol. The model was significant ($p = .007$), which indicated differences between the mean vectors for the two groups; no effects for cognition were found ($p = .385$). When each sensory domain magnitude variable was examined separately, significance between subjects' effects was found for Tone ($p = .039$); significance levels for Visual stimulus approached significance ($p = .068$) (Fig. 3). The groups did not significantly differ during the recovery period following the protocol.

Prediction of Good Sleepers Versus Poor Sleepers

Our final aim was to determine whether behavioral and physiological variables examined in this study would be useful in predicting good sleepers and poor sleepers. Variables that were found to be meaningful in prior analysis were entered into a logistic regression model. These variables included afternoon salivary cortisol, raw scores on the four quadrant scores of the sensory profile, the log value of magnitude of EDR to Tone, and the average score of the two post-SCP salivary cortisol samples taken at 25 and 30 minutes post-challenge. Although none of these variables, once adjusted for others in the model, were statistically significant predictors of sleeping status (Table 4), the overall model did provide a good fit to the data (Hosmer and Lemeshow test, chi-square = 1.649, 7 *df*, $p = .977$). This model enabled us to correctly classify 92.3% of good sleepers and 66.7% of poor sleepers accurately with an overall correct classification rate of 85.7% (Table 5).

Discussion

The results of our study confirm that children with ASD have a higher prevalence of both sensory modulation disorders and sleep disturbances compared to a population of typical children. Interestingly, the patterns of linkage between sensory modulation disorder and sleep differed between children with ASD and typical children. Items on the Sensory Profile indicating Sensory Avoiding behaviors were highly correlated with sleep problems in children with ASD. Sensory Sensitivity and Sensory Avoiding are considered indicative of a low neuronal threshold according to Dunn's model of sensory processing, and high arousal according to other investigators (Liss, Saulnier, Fein, & Kinsbourne, 2006). Our findings of a correlation between sleep problems in

Table 4
Logistic Regression Model Parameters

Parameter	B	SE	Wald	*df*	*p*	Exp(B)
Cort_D2_A	9.776	6.519	2.249	1	.134	17601.325
SPQ_LR_raw	.032	.108	.087	1	.768	1.032
SPQ_SK_raw	-.060	.056	1.127	1	.288	.942
SPQ_SS_raw	.104	.081	1.642	1	.200	1.110
SPQ_SA_raw	-.190	.136	1.961	1	.161	.827
magtone	3.165	2.141	2.185	1	.139	23.693
CortAvgPost	5.661	4.707	1.447	1	.229	287.568
Constant	11.421	10.534	1.175	1	.278	91198.943

Cort_D2_A = afternoon cortisol collected at home on day 2; SPQ_LR_raw = sensory processing quadrant raw score for low registration; SPQ_SK_raw = sensory processing quadrant raw score for sensory seeking; SPQ_SS_raw = sensory processing quadrant raw score for sensory sensitivity; SPQ_SA_raw = sensory processing quadrant raw score for sensation avoiding; magtone = electrodermal magnitude of response to auditory tone; CortAvgPost = average of salivary cortisol levels taken at 25 and 30 minutes post-sensory challenge.

Table 5
Prediction of Good Sleepers Versus Poor Sleepers

Observed Group	Predicted No. of Good Sleepers	Predicted No. of Poor Sleepers	% Correct
Good sleepers	24	2	92.3
Poor sleepers	3	6	66.7
Overall %			85.7

children with ASD and a low sensory threshold/high arousal is commensurate with previous assertions that children who have difficulty filtering sensory stimuli may have difficulty lowering arousal or tuning out necessary environmental stimuli to fall asleep or stay asleep. Further, these results suggest that children with ASD deal with low neuronal threshold actively, using behaviors designed to remove either the stimuli or themselves from the situation.

These findings contrast with the sensory modulation strategies used by typical children. In this group, sleep difficulties correlated strongly across all four sensory processing quadrants. Thus, typical children showed both low and high threshold responses to sensation and matched their threshold with both passive and active behaviors to sensory stimuli. It should be no surprise that typical children showed a full range of sensory modulation strategies. Importantly, no single strategy emerged to characterize the relationship of sensory modulation to sleep. Poor sleep behaviors in otherwise typical children may be more complex relative to sensory modulation.

Sleep is an important occupation that is crucial for maturation, reorganization, and restoration (Breedlove et al., 2007). Because we found poor sleepers in both groups of children, it became important to reconfigure the data, allowing us to look at the occupation of sleep relative to neurophysiologic parameters. Examining good sleepers (sleep index $\geq$ 2) and poor sleepers (sleep index $\leq$ 3) irrespective of diagnosis indicated that poor sleepers demonstrated higher afternoon levels of salivary cortisol, a greater magnitude of EDR to sensory stimuli, and a tendency toward higher cortisol levels 25 to 30 minutes following a sensory challenge. Because electrodermal response reflects sympathetic nervous system activity, these findings suggest differences in both autonomic nervous system and neuroendocrine function in children with poor sleep behavior. The finding that auditory tone was the most salient sensory stimuli to distinguish between good sleepers and poor sleepers was also interesting. Admittedly, because the stimuli in the SCP are not presented in a random order, the observed response to tone may be somewhat confounded by the fact that Tone is the first stimulus presented in our protocol. Regardless, children who are identified as poor sleepers had a significantly higher response to this novel stimulus than they did to other stimuli, including a familiar auditory stimulus (siren). Unfamiliar auditory tones that come from either inside or outside of the house are less predictable and less controllable than familiar sensory stimuli and may be more likely to prevent or disrupt sleep in children who are unable to properly filter auditory stimuli. Another explanation takes into account the development of the nervous system related to sleep behaviors. Children who were identified as poor sleepers demonstrated higher average magnitudes of response to Tone (novel

sound), Smell, and Visual Stimuli. These senses may be interpreted as more distant senses compared to those of movement and touch. From an evolutionary perspective, distant senses would have needed to be keener during sleep to detect potential threats; if the predator was close enough to elicit touch sensations, the individual would not likely have survived the encounter. Therefore, children identified as poor sleepers may demonstrate more primitive mechanisms of high arousal designed to protect and defend that are less conducive to rest and restoration.

In contrast to our original hypothesis, good sleepers when compared to poor sleepers showed lower salivary cortisol in the afternoon and no differences were found between groups for morning or nighttime levels. Corbett et al. (2009) found similar results when conducting a general comparison of children with ASD to a neurotypical population. Interestingly, these authors found, as did we, that greater sensory sensitivity was associated with higher afternoon cortisol levels. Therefore, heightened levels of afternoon salivary cortisol may be a neuroendocrine response to sensory stresses that occur throughout the day and subsequently relate to poor sleep patterns indirectly through a common sensory modulation mechanism. Alternatively, poor sleep may be a result of an overall dysregulation of circadian rhythms, and sensory sensitivity may play some role in this process.

The identified tendency toward significant group differences in cortisol at 25 to 30 minutes post-challenge resembles our prior studies subclassifying children with attention-deficit hyperactivity disorder, with and without sensory overresponsivity, using the SCP (Lane et al., 2010; Reynolds et al., 2010). Both of these prior studies indicated that group differences based on behavioral hypersensitivity were not observed until approximately 30 minutes after the challenge itself was completed. This pattern is likely indicative of the time it takes salivary cortisol to peak in saliva (18 to 30 minutes) and the summative effects of the sensory stressors across the entire sensory challenge. Future investigations will need to extend the collection of post-challenge measures beyond the 30-minute point to examine stability of group differences over time and return-to-baseline patterns.

Overall, sensory modulation behaviors, neuroendocrine and physiological response to sensation, and afternoon levels of salivary cortisol were able to distinguish good sleepers from poor sleepers with 85.7% accuracy. This suggests that atypical sensory behaviors are an important characteristic to consider in relation to sleep deficits in both diagnostic and non-diagnostic groups of children. Potential mechanisms for these relationships have been discussed, but further investigation into these causal relationships and neural circuitry is warranted.

Limitations

This study is limited in its use of parent report measures to identify and quantify sleep disturbances in children. Certainly, more advanced measures of identifying these problems can be employed with use of methods such as actigraphy or electroencephalography. Nonetheless, parent report of sleep problems has been commonly used in the literature to distinguish good sleepers from poor sleepers and use of parent report measures has been validated through use of polysomnography and actigraphy in children with ASD (Allik, Larsson, & Smedje, 2006; Goldman et al., 2009). Therefore, although future studies should consider more objective measures of sleep in SMD research, we believe that these preliminary parent report data are valid for identifying sleep disturbances in this sample of children. Further, we examined differences between good and poor sleepers in the group as a whole, despite a greater incidence of poor sleepers in the ASD group. Future investigations will need to include larger samples of children to guide a more nuanced understanding of the relationship of poor sleep behaviors and physiologic measures of sensory response within individual diagnostic groups.

Implications

Interest in sensory modulation differences in children with ASD is increasing; however, the possible link between sensory modulation disorder and disordered sleep in this population has not been investigated. Because sleep has been identified as an area of occupation, understanding what both promotes and interferes with it becomes crucial to occupational therapists. The data presented here suggest a link between sleep and sensory modulation deficits; further evidence linking sensory modulation disorders with sleep deficits may have implications for the treatment of sleep dysfunction.

Occupational therapists frequently use sensory integration treatment to address functional deficits in children with ASD (Baranek, 2002; Dawson & Watling, 2000). Miller et al. (2007) documented improvement in children with SMD following sensory integration treatment. Further, the use of sensory-based interventions with children with autism has been shown to be effective in reducing anxiety and sensory sensitivity, and improving social interaction and play (Ayres & Tickle, 1980; Case-Smith & Bryan, 1999; Edelson, Edelson, Kerr, & Grandin, 1999; Lin-

derman & Stewart, 1999). No studies have been conducted to examine the influence of sensory-based occupational therapy, such as sensory integration intervention, on sleep behaviors. The positive effects of sensory-based intervention on such things as sensory sensitivity and anxiety strongly suggest that this line of research is warranted.

Acknowledgments

This research was supported by the National Institutes of Health General Clinical Research Center (Grant M01 RR00065, NCRR, NIH), the Virginia Commonwealth University A.D. Williams research development fund, and the National Center for Medical Rehabilitation Research and the National Institute of Neurological Disorders and Stroke (K12 HD055929) Rehabilitation Research Career Development Program Award. The authors thank all of the children and families who participated in this study and the research assistants who helped in the project: Jennifer Elswick, Carolyn Queck, Nicole Wray, Kelli Bloom, and Jessica Lynn. They also recognize the Pediatric Therapy Network for their assistance in data collection and subject recruitment.

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