



## Psychophysiology of children with autism spectrum disorder

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### Abstract

This study (1) explored the feasibility of using electrodermal activity (EDA) to characterize the arousal and sensory reactivity of children with high functioning autism (HFA) and Asperger's Syndrome (AS), (2) determined the reliability of electrodermal measures and (3) described the variability of EDA in this sample. Forty children with HFA and AS participated. All participants received a diagnostic psychological assessment and a physiological evaluation. Fourteen participated in the retest study on the physiological measures. Results indicated psychophysiological testing was feasible with this sample. Seventy-three percent of the variables had reliability coefficients greater than .33, with a median variable reliability of .45. No significant differences were detected between HFA and AS groups. Visual inspection of skin conductance level (SCL) suggested two patterns: (1) high SCL (high arousal), with higher EDA magnitudes, faster latencies and slower habituation and (2) low SCL (low arousal), with lower EDA magnitudes, slower latencies and faster habituation. The presence of two EDA patterns applied equally when medications were eliminated. The previous inconsistency in studies of EDA in children with HFA and AS may be due to the presence of a high arousal groups and a low arousal group. Hence, this population should not be assumed to be homogeneous.

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The psychophysiological literature on sensory functioning in children with autism spectrum disorder (ASD) is inconsistent. Findings vary among studies although support for atypical psychophysiological reactions to sensory stimuli in ASD using electrodermal activity (EDA) as an outcome exists (Bernal & Miller, 1970; James & Barry, 1980; Lelord, Laffont, Jusseaume, & Stephant, 1973; Lincoln, Courchesne, Harms, & Allen, 1995; Miller, Reisman, McIntosh, & Simon, 2001; Pritchard, Raz, & August, 1987). Interpretation of results is difficult because (1) the samples have broadly defined inclusion criteria thus groups are heterogeneous; (2) the physiology measures do not study the relation between arousal and reactivity; and (3) sensory functioning is studied in only one sensory domain. Thus, not surprisingly, conclusions are contradictory, due to methodological issues and differences in scientific standards (Rogers & Ozonoff, 2005).

Sensory disturbances in children with ASD are often reported by parents and clinicians. Some authors suggest that children with ASD have sensory under-responsivity impeding social and emotional development (DesLauriers & Carlson, 1969; Kanner, 1943; Rutter et al., 1999; van Engeland, 1984). Others hypothesize that children with ASD have sensory over-responsivity, failing to integrate new experiences efficiently (Bergman & Escalona, 1949; Dawson & Lewy, 1989). A third theory is that sensory problems relate to a continual fluctuation of responsiveness, e.g., “perceptual inconstancy” (Ornitz & Ritvo, 1968), or “sensory distortions” (Tanguay & Edwards, 1982) significantly interfering with learning. Although sensory dysfunction is not a core deficit in ASD, the literature suggests that sensory disorders are an associated feature of the disorder (Dunn, 2001; Ermer & Dunn, 1998; Kientz & Dunn, 1997; Liss, Saulnier, Fein, & Kinsbourne, 2006; Pfeiffer, Kinnealey, Reed, & Herzberg, 2005; Rogers, Hepburn, & Wehner, 2003).

The reported incidence of atypical reactions to sensory stimuli in children with ASD ranges from 30 to 80% (Baranek, Foster, & Berkson, 1997a,b; Dahlgren & Gillberg, 1989; Gillberg et al., 1990; Kientz & Dunn, 1997). Abnormal reactions to sensation appear to affect children with high functioning autism (HFA) and Asperger’s Syndrome (AS), with unclear differences between the two diagnostic groups (Baranek et al., 2002; Dunn & Bennett, 2002; Dunn, Myles, & Orr, 2002; Lincoln et al., 1995; Parush, Sohmer, Steinberg, & Kaitz, 1997; Pfeiffer et al., 2005; Rogers et al., 2003). Studies differentiating the sensory responses of individuals with AS versus HFA matched on intelligence quotient (IQ) are needed (Myles et al., 2004). This data would inform the debate about whether AS and HFA are distinct subtypes.

Previous studies measuring electrodermal activity have conflicting results; for example, both non-responding (van Engeland, Roelofs, Verbaten, & Slangen, 1991), and high resting arousal in ASD (Hirstein, Iversen, & Ramachandran, 2001; van Engeland et al., 1991). Only one study conducted in the past 5 years (Hirstein et al., 2001), suggests that individuals with ASD may *have two* different patterns of responding (e.g., either a pattern of high arousal and high reactivity or a pattern of low arousal and low reactivity). Further studies are needed to test this hypothesis.

Also crucial to study is effects of medication on arousal and reactivity which has not been specifically reported in ASD. A review of the related literature suggests that many medications artificially lower BDA (Green, Nuechterlein, & Satz, 1989; Kozak, Rossi, McCarthy, & Foa, 1989; Schlenker et al., 1995; Schneider, 1983; Zahn, Insel, & Murphy, 1984). However, more controlled studies are needed to inform this issue.

This study comprehensively and systematically studies autonomic nervous system functioning in children with ASD. The study improves on previous research by: (a) defining a homogeneous ASD sample with regard to cognitive functioning, (b) measuring both arousal (tonic) and reactivity (phasic) outcomes, and (c) evaluating five sensory domains. The primary

objectives of this study of children with HFA and AS were: (1) to explore the feasibility of using the *Sensory Challenge Protocol* to measure sensory functioning; (2) to evaluate the reliability of electrodermal measures; (3) to describe tonic and phasic electrodermal activity; (4) to analyze differences between HFA and AS on in sensory responsivity; and (5) to note the effect of medication on autonomic function.

## 1. Method

### 1.1. Participants

Forty children (ages 5–15) diagnosed with AS or HFA, by licensed clinical psychologists with expertise in autism at the Autism and Developmental Disabilities Research Group of the University of Colorado Health Sciences Center were recruited. This center houses the Denver site of the Collaborative Programs for Excellence in Autism (NICHD Program Project) and the Centers for Autism and Developmental Disabilities Research in Epidemiology (CADDRE, Centers for Disease Control). Children met stringent diagnostic criteria for ASD based on clinical judgment and scores above the cutoff on the Autism Diagnostic Observation Scale (ADOS, Lord, Rutter, DiLavore, & Risi, 1999) and the Social Communication Questionnaire (Berument, Rutter, Lord, Pickles, & Bailey, 1999). Full-scale IQ scores were above 70. A diagnosis of either AS or HFA was determined by the psychologists on the team, relying upon ADOS, SCQ, and developmental history data. Reliability of specific diagnoses was evaluated using a chart review procedure with clinical psychologists as raters. In 38 of 40 cases, clinicians agreed on the child's specific spectrum diagnosis. In the case of two disagreements, consensus was reached after clarifying documentation of early history.

Of the 40 children referred for the study, two were unable to complete testing due to anticipation anxiety related to the laboratory protocol. Thus, the sample size was 38, with 11 AS and 27 HFA participants. Demographic characteristics of the participants' socioeconomic status (SES; defined as mother's education) indicated >80% were Caucasian and >70% had a high school degree. The average participant age was 8.8 for HFA and 9.5 for AS. Both samples had a majority of males (93 and 73%, respectively). Demographic comparisons of HFA and AS groups were calculated with *t*-tests and chi square statistics. Differences were not significant on any demographic variable except non-verbal intelligence quotient, where the AS group was significantly higher (mean AS = 117, HFA = 93). Seventeen participants were on one or more medications, i.e., seven on two medications, one on three medications. The most common medications were: Ritalin (4) and Zoloft (5). Other medications taken by the 17 participants in the medication group included: Celexa, Clonidine, Depakote, Risperidone, Concerta, Prozac, Adderall, Tegretol, Luvox, Tenex and Effexor.

Twenty-five participants from the original sample of 38 were invited to participate in the retest reliability study within six weeks of their initial testing. Of the 25 contacted, 9 declined and 2 failed to show, leaving a test–retest sample of 14, 71% with HFA and 29% with AS.

### 1.2. Instrumentation

#### 1.2.1. Psychophysiological protocol

The *Sensory Challenge Protocol* is a psychophysiological laboratory paradigm (Hagerman et al., 2002; Mangeot, 1999; McIntosh, Miller, Shyu, & Hagerman, 1999; Miller et al., 1999, 2001) during which tonic and phasic EDA is collected with palmar electrodes using the PSYLAB

System (Contact Precision Instruments, Cambridge, MA). Physiology data were collected by examiners who were blinded to the diagnosis of the participant.

Tonic EDA is collected during baseline and recovery when the child sits quietly and no stimuli are presented. The two tonic variables were: (1) *skin conductance level* (SCL), the mean of the skin conductance amplitudes averaged over 10 s blocks during the rest phases of the experiment (baseline and recovery) of at least .02  $\mu$ S, and (2) *non-specific conductance response* (NSR), responses occurring between 4 and 10 s post-stimulus. NSR was expressed as an average rate per minute.

Phasic EDA measures are based on definitions by Dawson, Schell, and Fillion (2000) and are recorded during the stimulation phase of the experiment for responses that are  $>.02 \mu$ S and occur between 0.8 and 4.0 s after each stimulus. The four phasic variables were: (1) *orienting response* (OR), amplitude of the initial stimulus response; (2) *magnitude of response* (MAG), the average baseline to peak amplitude of all skin conductance responses (SCR) within a sensory domain, including zero responses (e.g., peaks in the tactile domain are labeled MAG\_tactile); (3) *latency* (LAT), the average time from the onset of the skin conductance response to the peak of the response, calculated within a sensory domain (e.g., LAT\_tactile) (note: latency reflects only those trials for which there was a SCR); and (4) *habituation* (HAB) the slope of skin conductance level across trials 1–8.

The laboratory was decorated to look like a spaceship with walls painted to look like three dimensional space ship panels and low light levels. A small console, the “control panel for our space ship”, is centered in front of the child with video monitor and strobe light. The child is seated in a sturdy armchair mounted on a motorized tilting frame. As the experimenter attaches electrodes to the child, the child watches appropriate parts of Apollo 13, depicting astronauts having electrodes attached. EDA was recorded continuously during a 3-min baseline period, followed by the presentation of 48 sensory stimuli (Miller et al., 1999; Miller et al., 2001). Eight trials in each sensory domain were administered in the following order: auditory (tone), visual (flash), auditory (siren), olfactory (wintergreen), tactile (feather) and vestibular (chair tip). Each stimulus was presented in a pseudo-random schedule 12–19 s apart, with 20 s between each sensory modality. The signals were sampled at 1000 Hz, digitized, stored on a computer, and later reduced using PSYLAB (Contact Precision Instruments, Cambridge MA). The experiment ended with a 3-min recovery period during which no stimuli were delivered.

### 1.3. Procedures

Prospective participants/families were first contacted by the staff at the Autism and Developmental Disabilities Research Group, who provided comprehensive information about the risks and benefits of the study. All families contacted lived in the metropolitan Denver area, were recruited through community sources (e.g., parent groups, workshops, regional centers for persons with disabilities), and had given consent to be contacted for future studies. Parents then met with one of the investigators and informed consent was obtained, including consent to review previous assessment materials and to videotape the child. Twenty-five enrolled families were contacted 1 week after the physiologic study to ask if they would participate in the test–retest study. The retest study was conducted 2–6 weeks after the initial session.

Participants attended two sessions conducted within 6 months. Session one was a diagnostic *Psychological Assessment* conducted at the Autism and Developmental Disabilities Research Group at the University of Colorado Health Sciences Center and included cognitive testing, interviews of adaptive functioning, direct assessment of autism symptoms, and parent interviews of symptoms and behaviors associated with autism spectrum disorders. Session two was a

*Physiologic Assessment*, conducted at the Sensory Treatment And Research (STAR) Center at The Children's Hospital of Denver. Families were compensated for their time and transportation to all sessions.

#### 1.4. Statistical analyses

Descriptive statistics summarized data on each variable, using graphic analyses, means and standard deviations. Bivariate Pearson product moment correlation coefficients examined associations among physiological variables. Data were log transformed before analysis (Boucsein, 1992; Dawson et al., 2000) to normalize the distribution of data.

Test–retest scores of the same participant who was administered the laboratory paradigm twice were correlated using intra-class correlation (ICC) coefficients for each variable.

## 2. Results

### 2.1. Feasibility

Psychophysiological testing with a sample of children with AS and HFA individuals was feasible. Ninety-five percent of the sample was able to complete a laboratory protocol involving set-up (10–20 min) and 30 min of stimuli presentation while maintaining palmar electrodes and not engaging in significant movement, which would have created artifacts in the data.

### 2.2. Reliability

To evaluate the consistency of EDA measures of sensory reactivity and arousal, test–retest reliability was calculated on all variables (see Table 1). The tonic variables SCL and NSR had moderate reliability. Orienting response had the lowest reliabilities and therefore were excluded from all further analyses. The phasic variables, MAG, LAT and HAB were also moderately reliable across sensory domains. Among the phasic variables, MAG and LAT variables had higher reliabilities whereas HAB variables tended to have lower reliabilities. Seventy-three percent of the variables studied had reliability coefficients greater than .33, with a median of .45.

### 2.3. Description of tonic and phasic EDA

Participants demonstrated high variability on tonic and phasic variables, reflected in large standard deviations relative to means. The raw data on the tonic variables, SCL and NSR, are reported for baseline and recovery as follows: SCL baseline mean = 1.25 (1.09); SCL recovery mean = 1.99 (.70); NSR baseline mean = 5.3 (4.3); NSR recovery mean = 7.1 (5.1). The raw data on the phasic variables, MAG, LAT and HAB, are reported in Table 2.

### 2.4. Differences between diagnostic groups

Analyses were conducted between the two groups, AS ( $n = 11$ ) and HFA ( $n = 27$ ), by conducting *t*-tests across all variables. No significant differences were detected between diagnostic group for tonic variables (SCL, NSR) or phasic variables (MAG, LAT and HAB), therefore, participants were treated as a single group of individuals with ASD for all subsequent analyses.

Table 1  
Test–retest reliability of tonic and phasic electrodermal activity variables

Tonic variable	Phase	N	M diff (SD diff)	ICC
Skin conductance level	Baseline	14	.37 (.94)	.45
	Recovery	14	.25 (.53)	.51
Non-specific skin responses	Baseline	14	.87 (3.34)	0.54
	Recovery	14	.24 (5.21)	0.55

Phasic variable	Sensory domain	N	M (SD)	ICC
Skin conductance magnitude	Tone	13	−0.05 (.79)	0.83
	Strobe light	14	−0.43 (.68)	0.65
	Siren	13	−0.15 (1.28)	0.16
	Smell	13	−0.95 (1.07)	0.64
	Feather	13	0.23 (1.00)	0.57
	Chair tip	14	−0.47 (1.24)	0.49
Latency	Tone	11	−0.35 (.73)	0.54
	Strobe light	13	−0.02 (.70)	0.39
	Siren	12	−0.21 (.72)	0.52
	Smell	11	−0.32 (.70)	0.11
	Feather	10	0.22 (.73)	0.27
	Chair tip	13	−0.30 (.35)	0.55
Habituation slope of skin conductance level	Tone	13	.01 (.07)	.00
	Strobe light	14	.01 (.03)	.37
	Siren	13	.01 (.08)	.00
	Smell	13	.00 (.04)	.39
	Feather	13	.00 (.06)	.29
	Chair tip	14	.01 (.03)	.33

### 2.5. Analysis of variability by arousal level

Additional analyses were conducted to further examine the variability in the data. A pilot study from our lab suggested two subgroups might emerge based on arousal levels, amongst children with ASD (Brett-Green et al., 2004). Therefore, SCL for each individual was plotted during baseline. Visual analysis suggested a cut point of 6  $\mu$ S (see vertical line in Fig. 1) for categorizing high and low arousal.

These two tonic patterns were used to guide further analyses. SCL was plotted for baseline, across sensory domains and recovery (see Fig. 2). Correlations were also computed between

Table 2  
Phasic variables

Sensory domain	N	Magnitude M (SD)	Latency M (SD)	Habituation M (SD)
Tone	37	−2.24 (1.22)	3.9 (1.03)	.008 (.057)
Strobe light	37	−2.63 (1.00)	4.49 (.54)	−.02 (.034)
Siren	37	−2.27 (1.07)	3.69 (.76)	−.018 (.046)
Smell	36	−2.62 (1.36)	4.49 (.81)	−.02 (.036)
Feather	37	−2.83 (1.09)	4.52 (.69)	−.027 (.032)
Chair tip	38	−1.55 (1.14)	3.78 (.70)	−.025 (.026)

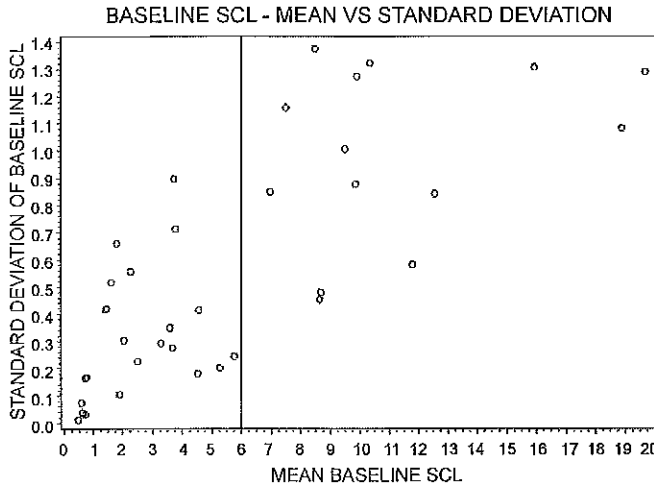


Fig. 1. Cut-point based on mean and standard deviations in baseline SCL.

baseline SCL and recovery SCL and between baseline SCL and SCL throughout all the sensory domains.

Results indicated stability of the tonic patterns across the experiment. One pattern had low amplitude SCL and relatively less variability; the other pattern had higher amplitude SCL and more variability. Baseline SCL and recovery SCL were highly correlated. In addition, correlations were computed between baseline SCL and mean SCL throughout all sensory domains measured during the sensory challenge experiment ( $r = .931-.996$ ;  $p < .001$ ).

2.6. Relation between tonic and phasic EDA variables

Correlations between baseline SCL and the three phasic variables were calculated across all sensory domains. In five of six sensory domains, baseline SCL was positively correlated with

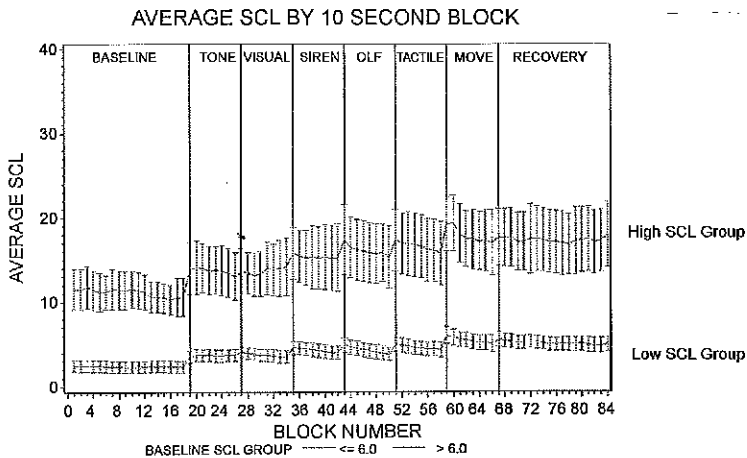


Fig. 2. Means and standard deviations of two patterns of SCL during baseline, Sensory Challenge Protocol, and recovery.

MAG (excluding movement domain). As baseline SCL increased, MAG tended to increase. Correlations between LAT and baseline SCL were also high and statistically significant in four of the six sensory domains (excluding tactile and olfactory domains). In general, as baseline SCL increased, LAT tended to decrease. Across all sensory stimuli the high SCL group had faster latencies and the low SCL group had slower latencies. Analysis of HAB (e.g., slope of SCL) in each domain showed that as SCL increased, HAB tended to be slower (excluding tone stimuli). Overall, the high baseline SCL group tended to habituate more slowly than the low SCL group.

Though statistical significance was not reached for all comparisons, the high SCL group tended to have higher magnitudes, faster latencies and slower habituation. By contrast the low SCL group tended to have lower magnitudes, slower latencies and faster habituation.

### 2.7. Medication effects

To evaluate whether the patterns obtained in the total group was an artifact of a medication effect a secondary analysis was performed. Medication use was evaluated in the total sample.

Results of an analysis comparing the medication and no-medication groups using chi square indicated that 13 of 17 participants in the medication group had low baseline SCL amplitudes (less than 6  $\mu$ S) and 4 participants had high SCL. In the no-medication group an almost equal number of participants had high versus low SCL (11 with low SCL and 10 with high SCL).

To evaluate whether medication had an effect on the results obtained for the total sample, analyses were conducted on the 21 participants in the no-medication group and compared to findings for the total group. In general, the no-medication group had EDA that was similar to the total group. Group differences were smaller and less significant due to smaller sample sizes. However, like the total group, the high SCL no-medication group had higher magnitudes, faster latencies and slower habituation to the stimuli than the no-medication low SCL group (except for the siren stimulus).

### 2.8. Post hoc analyses: non-responding

Related literature also suggests that non-responding has important clinical implications for characterizing some disorders. Utilizing a definition of non-responding on the first trial, we calculated that 29% of our ASD sample were non-responders in at least one sensory domain.

## 3. Discussion

The psychophysiological paradigm, the *Sensory Challenge Protocol*, was successful in measuring arousal and sensory reactivity in children with HFA and AS. Test–retest reliability correlations reported in this study suggest that electrodermal measures are relatively stable in this sample. This is the first objective reliability study of EDA in ASD. However, similar reliability correlations are reported for typically developing samples (Iacono et al., 1984; Schell, Dawson, & Fillion, 1988; Schell, Dawson, Nuechterlein, Subotnik, & Ventura, 2002; Vossel & Zimmer, 1990). Less stability is reported in clinical groups who have deficits in arousal regulation and increased symptomatology (Schell et al., 2002).

Found here was wide variability across a range of EDA measures including arousal, reactivity, latency and habituation. Means and standard deviations are reported to provide general



guidelines of expected values. Needed are studies comparing EDA values for individuals with ASD to typically developing individuals. Patterns of tonic and phasic EDA may be beneficial in differentiating ASD from other groups.

The debate regarding the clinical usefulness of differentiating between HFA and AS is ongoing in the literature. Recent studies controlling for IQ have not produced a clear differentiation between HFA and AS on a wide variety of symptoms including social abilities (Starr, Szatmari, Bryson, & Zwaigenbaum, 2003), language (Howlin, 2003) motor, visuospatial or executive functions (Miller & Ozonoff, 2000) and repetitive behaviors (South, Ozonoff, & McMahon, 2005). Analyses in this study revealed no significant differences between HFA and AS on any EDA variables. Thus, arousal and sensory reactivity were not different in HFA and AS. This study thus provides preliminary evidence that a sympathetic nervous system differentiation between HFA and AS does not exist.

For over four decades ago, theorists have been debating the sensory abnormality in children with ASD. A significant finding of this study was two distinct patterns of arousal and sensory reactivity in ASD. These findings support the hypothesis that some children with ASD are over-aroused and more reactive while others are under-aroused and less reactive. Based on these findings of two patterns it is not surprising that previous studies of EDA in children with ASD have inconsistent results. Likely, one group of children with ASD have high tonic electrodermal arousal and high reactivity, e.g., are quicker to respond to sensory stimuli; the other group of children with ASD have low tonic arousal and lower reactivity, e.g., take longer to respond to sensory stimuli, and tend to habituate faster than the high arousal group. This suggests previous differences may have been due to unique features of the different samples.

The long history of research using electrodermal activity to study individuals with schizophrenia provides a useful model for studying atypical autonomic reactions to sensory stimuli in children with ASD (Bernstein, 1965; Dawson & Nuechterlein, 1984; Dawson, Nuechterlein, Schell, Gitlin, & Ventura, 1994; Gruzelier & Venables, 1972; Hazlett, Dawson, Schell, & Nuechterlein, 1997; Rissling, Schell, Dawson, & Nuechterlein, 2000; Schell et al., 2002; Schell et al., 2005; Spohn & Patterson, 1979; Zahn & Pickar, 2005). In schizophrenia, one psychophysiological pattern reported is abnormally high resting levels of skin conductance, e.g., arousal, with increased non-specific skin conductance reactions, also a measure of arousal. This group tends to habituate quickly. Another finding in the schizophrenia literature is a group of non-responders, e.g., to innocuous tone stimuli. Studies estimate a lack of orienting response in about half of individuals with schizophrenia (Schell et al., 2002), compared to ~10% non-responders in the typically developing population. Schell et al. (2002) suggests that individuals with high resting levels of skin conductance have abnormal arousal while non-responders have an information processing deficit.

Our study suggests that individuals with ASD may resemble individuals with schizophrenia in terms of physiological responsivity. In schizophrenia, the pattern with high arousal (e.g., higher resting skin conductance) was associated with increased symptomatology and poorer functional outcomes (Dawson et al., 1994; Schell et al., 2002). While a similar physiologic pattern was observed in our ASD sample, future research should evaluate whether EDA patterns predict behavioral outcomes in children with ASD.

Another finding in this study similar to studies of schizophrenia is a group of non-responders among children with ASD. Post-hoc analysis revealed that 29% of the sample was non-responders to the first trial. These findings are similar to Hirstein's et al. (2001) finding that 30% of individuals with ASD have flat skin conductance levels with few responses to visual stimuli and vanEngeland's finding that 16% of individuals with ASD do not respond to

the first auditory trial (1984). Hirstein et al. (2001) suggests that some children with ASD might be 'naturally' hypo-responsive, reflecting poor attention to the environment. The lack of response in a subgroup of those with ASD may suggest a defect in information processing of novel stimuli, helping explain some behavioral disturbances in individuals with ASD. Further study of the relation between physiologic non-responding and attention/information processing is needed.

A large percent of our sample was on medication for anxiety or inattention. Post hoc analyses found that the no-medication group ( $n = 21$ ) performed similarly to the total sample. The no-medication group also demonstrated two EDA patterns. Half of the individuals in the no-medication group demonstrated low arousal. Further investigation of the physiological characteristics of individuals with ASD on medication would be fruitful.

This study has important implications for future research of sensory processing in children with ASD. Determining if patterns of high arousal and reactivity compared to non-responding is associated with specific behavioral or cognitive deficits in children with ASD or predictive of outcome, as has been found in studies of individuals with schizophrenia, could be an important initiative.

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