

# Electrodermal Response to Reward and Non-Reward Among Children With Autism

Emily Neuhaus, Raphael A. Bernier, and Theodore P. Beauchaine

Pervasive social difficulties among individuals with autism spectrum disorder (ASD) are often construed as deriving from reduced sensitivity to social stimuli. Behavioral and neurobiological evidence suggests that typical individuals show preferential processing of social (e.g., voices, faces) over nonsocial (e.g., nonvocal sounds, images of objects) information, whereas individuals with ASD may not. This reduction in sensitivity may reflect disrupted reward processing [Dawson & Bernier, 2007], with significant developmental consequences for affected individuals. In this study, we explore effects of social and monetary reward on behavioral and electrodermal responses (EDRs) among 8- to 12-year-old boys with ( $n = 18$ ) and without ( $n = 18$ ) ASD, with attention to the potential moderating effects of stimulus familiarity. During a simple matching task, participants with and without ASD had marginally slower reactions during social vs. nonsocial reward, and boys with ASD had less accurate responses than controls. Compared to baseline, reward and non-reward conditions elicited more frequent and larger EDRs for participants as a whole, and both groups showed similar patterns of EDR change within reward blocks. However, boys with and without ASD differed in their EDRs to non-reward, and response amplitude was correlated with social and emotional functioning. These findings provide some support for altered reward responding in ASD at the autonomic level, and highlight the discontinuation of reward as an important component of reward-based learning that may play a role in shaping behavior and guiding specialized brain development to subserve social behavior and cognition across the lifespan. *Autism Res* 2015, 8: 357–370. © 2015 International Society for Autism Research, Wiley Periodicals, Inc.

**Keywords:** autism; ASD; reward; electrodermal responding; skin conductance; social motivation

## Introduction

Among impairments that characterize autism spectrum disorder (ASD), social deficits are central and early emerging, typically appearing in domains such as eye contact, pointing, imitation, joint attention, and face processing [Dawson & Bernier, 2007; Mundy, Sigman, & Kasari, 1990; Osterling & Dawson, 1994]. Many of these behavioral features can be construed as reflecting reduced sensitivity to stimuli that are social in nature [Dawson & Bernier, 2007; Klin, Jones, Schultz, Volkmar, & Cohen, 2002]. Whereas typically developing individuals show preferential responding to social stimuli such as human voices, images of faces, and biological motion over nonsocial stimuli such as nonvocal sounds, images of objects, and nonbiological motion, those with ASD are less likely to show such distinctions, an effect that is apparent at both behavioral and neurobiological levels of analysis, in response to a wide range of stimuli, and across a variety of brain regions [e.g., Bernier, Dawson, Webb, & Murias, 2007; Gervais et al., 2004; Ober-

man, Hubbard, McCleery, Altschuler, Ramachandran, & Pineda, 2005; Pierce, Conant, Hazin, Stoner, & Desmond, 2011].

These observations have prompted suggestions that altered reward processing in the brain might account for reduced sensitivity to social stimuli in ASD [Chevalier, Kohls, Troiani, Brodtkin, & Schultz, 2012; Dawson & Bernier, 2007; Dichter, Felder, Green, Rittenberg, Sasson, & Bodfish, 2012; Schmitz, Rubia, van Amelsvoort, Daly, Smith, & Murphy, 2008]. According to the social motivation hypothesis, social information is poorly integrated into central nervous system reward processing networks very early in development, resulting in diminished attention to social stimuli. Over the course of development, this alteration amplifies, yielding dramatic and pervasive effects in social functioning, including compromised conditioned preferences for social affiliation, impaired orienting to social cues, and further failure to integrate social information within neurobiological systems subserving reward [Dawson et al., 2005; Kohls, Chevalier, Troiani, & Schultz, 2012].

From the Seattle Children's Research Institute, Center for Child Health, Behavior, and Development, M/S CW8-6, PO Box 5371, Seattle, Washington, 98121 (E.N.); Dept of Psychiatry and Behavioral Sciences, University of Washington, Box 357920, Seattle, Washington, 98195 (R.A.B.); Department of Psychology, The Ohio State University, 1835 Neil Avenue, Columbus, Ohio, 43210 (T.P.B.)

Received May 18, 2014; accepted for publication November 25, 2014

Address for correspondence and reprints: Emily Neuhaus, Seattle Children's Research Institute, M/S CW8-6, PO Box 5371, Seattle, WA 98121. E-mail: eneuhaus@uw.edu

Published online 20 January 2015 in Wiley Online Library (wileyonlinelibrary.com)

DOI: 10.1002/aur.1451

© 2015 International Society for Autism Research, Wiley Periodicals, Inc.

Although research on behavioral and neurobiological correlates of reward processing in autism has been quite limited historically, emerging findings indicate that children with ASD differ from those without ASD in both behavioral and neurobiological responding to reward. Early studies suggested impaired ability to form stimulus-reward associations [Dawson, Osterling, Rinaldi, Carver, & McPartland, 2001], and more recent findings reveal reduced awareness of reward contingencies among children with ASD [Faja, Murias, Beauchaine, & Dawson, 2013]. In studies of social reward, which is often operationalized as smiling human faces or verbal praise following correct task performance, individuals with ASD often differ from controls in the effect of social rewards on their behavior. For example, children with ASD show faster reaction times to monetary vs. social reward, whereas typically developing children display equal reaction times across tasks [Demurie, Roeyers, Baeyens, & Sonuga-Barke, 2011]. Similarly, children with ASD show less improvement in cognitive performance during social motivation conditions (e.g., competition with peers) than children with other psychiatric diagnoses [Geurts, Luman, & van Meel, 2008]. In some instances, imaging and electrophysiology data reveal differential responses between groups with and without ASD, despite comparable behavioral performance [see Kohls et al., 2012]. Taken together, this body of research suggests reward processing deficiencies in ASD, but also highlights the complexity of any such deficits.

Within the autonomic nervous system, motivated, appetitive behaviors associated with reward are effected peripherally through the sympathetic nervous system (SNS). The SNS originates in the spinal cord and promotes increases in alertness, arousal, and heart rate in response to environmental demand. Consequently, this system is often conceptualized as “fight or flight” responding. However, the SNS can also be conceptualized as supporting approach toward potentially rewarding stimuli [Beauchaine, 2001; Fowles, 1980]. Both Gray [1987] and Fowles [1980] describe a motivational system now termed the “behavioral activation system” that works to maximize rewards through active approach and avoid punishment although active avoidance. Gray describes this system as heavily mediated by mesolimbic dopaminergic mechanisms central to reward processing, and several lines of reasoning link it with sympathetic function as well. Primary among these is the observation that behavioral activation depends on an increase in energy and cardiac output, which is traditionally attributed to the SNS [Beauchaine, 2001]. Furthermore, heart rate acceleration necessary for approach is elicited largely through sympathetic mechanisms [Sherwood, Allen, Obrist, & Langer, 1986]. Finally, injections of dopamine agonists

directly into the ventral tegmental area (a component of the mesolimbic reward system) produce SNS-mediated increases in blood pressure and heart rate [van den Buuse, 1998]. Thus, this model links sympathetic and reward-related dopaminergic mechanisms with behavioral approach.

One index of SNS activity is electrodermal responding (EDR), a cholinergically mediated function of the eccrine sweat glands [Uno, 1977]. EDR to psychological stimuli are modulated in the brain by regions including the ventromedial prefrontal cortex, the orbitofrontal cortex, the anterior cingulate cortex, the amygdala, and the insula [Dawson, Schell, & Fillion, 2007], regions critical to both reward processing and social function [see Dichter, Richey, Rittenberg, Sabatino, & Bodfish, 2012; Neuhaus, Beauchaine, & Bernier, 2010]. These regions and EDR are particularly responsive to stimuli of emotional significance [Dawson et al., 2007], making EDR a valuable measure for understanding reward responding.

Early investigations found comparable levels of baseline EDR between participants with and without ASD when controlling for cognitive ability [Van Engeland, 1984; Zahn, Rumsey, & van Kammen, 1987]. However, larger responses and slower rates of habituation to visual and auditory stimuli are observed among those with ASD. In response to social stimuli, patterns of EDR among individuals with ASD differ from controls across multiple paradigms assessing various facets of social cognition. In one of the first reports, Hirstein, Iversen, and Ramachandran [2001] found that, whereas typically developing children display larger EDRs to images of their mother’s face than to images of an inanimate object, children with ASD showed comparable EDRs to both, suggesting a failure to differentiate between social and nonsocial stimuli. Adults with ASD also appear to exhibit fewer and smaller EDRs than controls during a task requiring judgement of emotion [Hubert, Wicker, Monfardini, & Deruelle, 2009], implying blunted EDR. However, conflicting evidence suggests that adults with ASD fail to habituate (i.e., fail to decrease EDRs) to a series of neutral faces, consistent with heightened arousal to faces [Mathersul, McDonald, & Rushby, 2013]. Consistent with this, direct eyegaze from others also elicits heightened EDRs in participants with ASD, relative to both averted eyegaze and to those without ASD [Kylliainen & Hietanen, 2006; Kylliainen et al., 2012]. Thus, the current literature presents a picture of altered electrodermal activity in response to social stimuli among individuals with ASD, but the precise nature of those alterations is unclear.

Evidence related to EDR and reward among individuals with ASD is just beginning to emerge. Initial findings suggested dampened EDR amplitude during a gambling task among adolescents and young adults with ASD [Johnson, Yechiam, Murphy, Queller, &

Stout, 2006]. In a similar task, however, Faja et al. [2013] found that children with ASD had typical electrodermal responding during anticipation of win/loss feedback and during feedback of losses, but they appeared to increase EDR amplitude to feedback of winnings over the course of the task. This was in contrast to typically developing peers who maintained consistent EDR to wins throughout the task. Furthermore, change in EDRs over the course of the game was correlated with social, emotional, and executive function [Faja et al., 2013]. Although limited, this early body of research suggests that reward-related EDR may be altered in ASD for some aspects of reward processing (e.g., receipt of incentive) while relatively preserved for others (e.g., incentive loss).

Thus, there is growing evidence of (1) atypical reward processing in ASD at multiple levels of analysis, and (2) links between disrupted reward processing and impaired social functioning. However, the degree to which general (e.g., monetary) vs. social reward processing is altered remains unclear, and no studies have explored the potential moderating role of familiarity of social reward. Our goals were to examine behavioral responses and EDR under conditions of nonsocial and social reward. We chose to include both unfamiliar and familiar social reward conditions, as several recent studies indicate that sensitivity to social stimuli among those with ASD may be moderated by stimulus familiarity, with more typical patterns of neural responding observed for familiar social stimuli. For example, in contrast to findings of reduced neural activation to faces in general, activation in the fusiform gyrus and amygdala is near normal when participants with ASD view faces of well-known individuals [Hadjikhani, Joseph, Snyder, & Tager-Flusberg, 2007; Pierce, Haist, Sedaghat, & Courchesne, 2004]. Similarly, those with ASD typically display altered patterns of activity in regions comprising the mirror neuron system while watching videos of human action [Bernier et al., 2007], but activation more closely resembles that of controls when participants watch videos of their own actions, or those of people they know [Oberman, Ramachandran, & Pineda, 2008].

In addition to these reward conditions, we sought to examine response to non-reward among children with and without ASD. Often overlooked in discussions of reward processing, response to non-reward (defined here as the discontinuation or withholding of an expected reward) may have implications for etiological theories as well as learning, social behavior, and development of clinical interventions. Furthermore, studies of EDR under non-reward have yielded important differences among other clinical groups [e.g., those with externalizing disorders; Beauchaine, Katkin, Strassberg, & Snarr, 2001], and thus, EDR may be well suited for finding subtle group differences between those with ASD and controls.

With this discussion in mind, we predicted that children with ASD would differ from controls in their behavioral response to reward, and that this divergence would be strongest for social reward. However, we also anticipated an effect of familiarity, such that responding would be more similar under familiar social reward than unfamiliar social reward. We also predicted that EDR would differ between the groups and that differences would be most apparent during conditions of non-reward. Finally, we predicted that EDR would be associated with parent-reported indices of social and emotional functioning.

## Methods and Materials

### *Participants*

Families were recruited through a registry at a university Autism Center, and through fliers and advertisements placed in the larger community. Eligibility was established through a phone screen in which parents completed: (1) a demographic questionnaire; (2) a brief developmental history; (3) the Social Communication Questionnaire (SCQ; [Rutter, Bailey, & Lord, 2003]); and (4) the Child Behavioral Checklist (CBCL; [Achenbach, 1991]). All participants were required to be free of stimulant medications for at least 36 hr prior to participation to avoid potential effects on the neural substrates of autonomic reactivity to reward [Swanson & Volkow, 2002]. All families provided informed consent and assent prior to participation, and the University Institutional Review Board prospectively reviewed and approved all study procedures. In total, 36 male children participated (18 ASD, 18 control).

Eligibility for the ASD group was established with the Autism Diagnostic Interview—Revised (ADI-R) [Lord, Rutter, & Le Couteur, 1994] and Autism Diagnostic Observation Schedule (ADOS; [Lord, Rutter, DiLavore, & Risi, 2003]), revised algorithm [Gotham, Risi, Pickles, & Lord, 2007]. Participants were required to meet diagnostic cut-offs for autism (ADI-R) and autism spectrum (ADOS) on these measures, as well as meet DSM-IV-TR [APA, 2000] criteria for autism, Asperger's disorder, or PDDNOS according to expert clinician judgment. In addition, participants were required to have functional communicative speech (defined as the ability to talk using short phrases or sentences according to parent report) to ensure they could understand instructions. The mean age of children with ASD was 119.8 months (SD = 13.2; range = 97.8 to 141.1 months). The racial/ethnic composition of this group was 72.2% Caucasian, 5.6% Asian/Pacific Islander, 5.6% Latino, and 16.7% who identified as more than one race. Participant characteristics are reported by group in Table 1.

Children in the control group were screened for sub-threshold features of ASD using the lifetime version of

**Table 1. Psychopathology Means and Standard Deviations by Group**

	Controls	ASD	F	$\eta_p^2$
Age in months	120.1 (11.1)	119.8 (13.2)	0.01	0.00
Full scale IQ	114.8 (13.5)	108.3 (21.4)	1.17	0.03
Verbal IQ	115.3 (9.9)	102.4 (23.0)	4.78**	0.12
Performance IQ	110.7 (15.7)	113.3 (19.6)	0.20	0.01
SCQ score <sup>a</sup>	3.0 (2.3)	19.7 (5.0)	172.65****	0.84
SSIS Social Skills standard score <sup>a</sup>	101.3 (10.8)	79.8 (10.7)	36.13****	0.52
SSIS Problem Behaviors standard score <sup>a</sup>	101.3 (7.2)	120.6 (17.6)	18.51****	0.35
CBCL Internalizing T-Score <sup>a</sup>	52.0 (9.3)	61.4 (10.3)	8.21***	0.20
CBCL Externalizing T-Score <sup>a</sup>	48.6 (8.0)	56.9 (10.4)	7.30**	0.18

Notes. <sup>a</sup>SCQ = Social Communication Questionnaire [Rutter et al., 2003]; SSIS = Social Skills Improvement System [Gresham & Elliot, 2008]; CBCL = Child Behavior Checklist [Achenbach, 1991].

\* $P < 0.10$ ; \*\* $P < 0.05$ ; \*\*\* $P < 0.01$ ; \*\*\*\* $P < 0.001$ .

the SCQ [which has high agreement with the ADI-R; Rutter et al., 2003] and the thought problems subscale of the CBCL (which includes several items related to possible repetitive behaviors). Exclusion criteria were an SCQ score above 9; a T-score above 65 on the thought problems subscale of the CBCL; a history of significant head injury, seizures, or a diagnosis related to their development (e.g., dyslexia, ADHD); and presence of a first-degree relative with ASD. The mean age of controls was 120.1 months (SD = 11.1; range = 96.4 to 136.0 months), which did not differ significantly from the ASD group,  $F(1, 34) = 0.01$ ,  $P = 0.94$ ,  $\eta_p^2 = 0.00$ . The racial/ethnic composition of the control group was 61.1% Caucasian, 5.6% Asian/Pacific Islander, 11.1% African American, and 22.2% who identified as more than one race.

#### Measures

**Autism diagnostic measures.** The presence of an ASD was determined through clinician judgment on the basis of the ADOS, ADI-R, and DSM-IV-TR diagnostic criteria.

**Social Communication Questionnaire—lifetime form.** The lifetime form of the SCQ is a 40-item parent-report questionnaire that assesses social behaviors and communication skills among children above age 4 years and yields a total score that can be interpreted in relation to a clinical cut-off of 15. The lifetime form has high agreement with the ADI-R, and internal consistencies with children of this age range from 0.89 to 0.93 [Rutter et al., 2003]. Typically developing school-age children have a mean score of 3.89 (SD = 2.77) on the SCQ [Mulligan, Richardson, Anney, & Gill, 2009].

**Wechsler Abbreviated Scale of Intelligence [Wechsler, 1999].** The Wechsler Abbreviated Scale of Intelligence was included to assess cognitive abilities of participants in both groups. It yields scores for verbal

and performance ability, and an IQ composite. Test-retest reliabilities are high, ranging from 0.83 to 0.92 for the composite scores.

**Child Behavior Checklist.** The CBCL is a 113-item questionnaire on which parents rate their child's behaviors using a 3-point scale ranging from 0 (not true) to 2 (very true or often true). The measure yields continuous T-scores on eight subscales, including aggressive behavior, anxiety/depression, attention problems, rule-breaking behavior, social problems, somatic complaints, thought problems, and withdrawal/depression. The CBCL has high internal consistency (range = 0.66 to 0.92 across subscales) and test-retest reliability (range = 0.63 to 0.97; [Achenbach & Rescorla, 2001]).

**Social Skills Improvement System [Gresham & Elliott, 2008].** The Social Skills Improvement System (SSIS) is a multi-rater assessment of social behaviors. The parent-report version contains 55 statements relevant to social skills and problem behaviors. Parents rate the frequency with which their child displays each behavior on a 3-point scale ranging from 0 (never) to 2 (very often), and the importance of each behavior to the child's development on a 3-point scale ranging from 0 (not important) to 2 (critical). The parent-report form yields standard scores for social skills and problem behaviors, and is appropriate for use with children between 3- and 18-year old. For the elementary age group, internal consistency across subscales ranges from 0.74 to 0.95 and test-retest reliability ranges from 0.70 to 0.92.

**Electrodermal responding.** Electrodermal data were collected via two 0.8 cm<sup>2</sup> Ag–AgCl electrodes attached to the thenar eminence of the nondominant hand. Data were sampled at 1 kHz, and recorded using a Grass 15LT Physiodata Amplifier System and a 15A12 DC

Amplifier (West Warwick, RI). EDR were hand scored with Grass PolyVIEW software. EDR were defined as fluctuations exceeding 0.05  $\mu$ S in amplitude. Epochs in which artifacts due to movement or other factors were present were excluded from analysis and considered as missing data. With this procedure, approximately 90% of epochs were retained for the sample as a whole, and diagnostic groups did not differ in the percentage of epochs for which data were retained,  $t(34) = 1.37$ ,  $P = 0.19$ . Because current guidelines recommend assessment of both the frequency of responses and the size of EDR [Dawson et al., 2007], we counted the number of EDRs within each epoch as a measure of EDR frequency (NED), and computed the mean amplitude of responses (AED) within each epoch as a measure of EDR size.

### *Procedure*

**Baseline.** After electrodes were adhered, children were asked to sit quietly and rest. They sat alone in a child-sized armchair in a sound attenuated room for 5 min and were observed via video camera to ensure cooperation. The final 2 min of this resting period were recorded as baseline electrodermal activity. This strategy was chosen to minimize any fidgeting due to the novelty of the room or physiological sensors, and yielded 4 epochs (each lasting 32 sec) of electrodermal data in all.

**Reward task.** Following the baseline period, participants played a simple computer game under three different reward conditions (described below), and during non-reward. Reward and non-reward conditions occurred in distinct blocks of 192 sec (6 epochs of 32 sec) each, with short break periods between blocks for a total task length of approximately 24 min. This yielded 6 epochs of data for each reward condition. Throughout the task, participants viewed a large screen on which a single digit was displayed for 3 sec, followed by a period of 3 sec in which they pressed the corresponding key on a 10-key pad. During reward conditions, correct responses were followed by 3 sec of either monetary reward, unfamiliar social reward, or familiar social reward, and incorrect responses were followed by a 3-sec blank screen and an audio tone. During non-reward conditions, the behavior-reward contingency was altered such that no feedback was provided following responses.

In the monetary reward condition, participants received \$0.05 for each correct response. Following a correct response, the \$0.05 value was displayed on the video screen for 3 sec with an auditory tone, and a running total of money earned was displayed in the upper right corner of the screen. Because each reward block lasted for 32 trials, the maximum amount that could be earned was \$1.60. The amount of reward per correct response was determined on the basis of previous work

examining reward sensitivity among autonomic biomarkers with children in similar age ranges [e.g., Beauchaine et al., 2001].

In the unfamiliar social reward condition, participants were shown a 3-sec video following each correct response. The video depicted an unfamiliar woman providing a praise statement (e.g., Great job!) with appropriate vocal affect and facial expression. To minimize habituation over the course of the block, three videos were created with varying praise statements (e.g., Nice work!) and were shown with equal frequency following each correct response. All participants viewed the same set of videos during the unfamiliar social reward condition.

In the familiar social reward condition, participants were shown a 3-sec video in which their parent provided a praise statement following each correct response. For the majority of participants (30), mothers participated in the familiar reward videos; for the remaining six participants, fathers participated in the videos. To ensure that unfamiliar and familiar social reward videos were matched closely on content, length, and affect, parents viewed the three unfamiliar social reward videos and then reenacted the actor's statements, affect, and expression with coaching and feedback from a research assistant, who then recorded their reenactments digitally and prepared the familiar social reward videos for the reward task. Parents recreated all three videos, which were shown with equal frequency following correct responses.

The order of reward blocks (monetary, unfamiliar social, familiar social) was counter-balanced across the sample to minimize carry-over effects of reward type, and non-reward blocks (two total) occurred following the second and third reward blocks for all participants. Two behavioral indices were recorded during the reward task: reaction time (time between appearance of the number and the participant's button press) and accuracy (percent correct).

## **Results**

Both behavioral outcomes and EDR were analyzed. For all repeated measures analyses, sphericity was assessed, and Greenhouse-Geisser-corrected  $P$ -values are reported whenever Mauchly's  $W$  indicated that sphericity was violated ( $P < 0.05$ ). Associated epsilons are reported.

### *Behavioral Outcomes During Reward and Non-Reward*

To test for effects of diagnostic group and reward condition on reaction times, a repeated measures analysis of variance (ANOVA) was conducted with reward type as a within-subjects factor and diagnostic group as a between-subjects factor (see Table 2 and Fig. 1). The effect of condition on reaction time approached

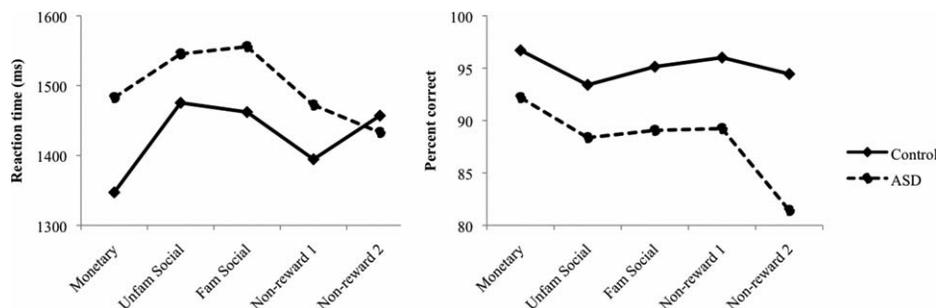
significance,  $F(4, 136) = 2.80$ ,  $P = 0.07$ ,  $\eta_p^2 = 0.08$ ,  $\varepsilon = 0.45$ . Within condition, simple contrasts were used to compare reaction times during monetary reward to those during all other conditions. Compared to the monetary reward condition, participants responded more slowly during the unfamiliar,  $F(1, 34) = 11.75$ ,  $P = 0.002$ ,  $\eta_p^2 = 0.26$ , and familiar,  $F(1, 34) = 11.34$ ,

**Table 2. Effects of Condition and Diagnostic Group on Behavioral and Electrodermal Outcomes Across Reward and Non-Reward Blocks**

Outcome	Effect	$F$	$\eta_p^2$
Reaction time	Condition	2.80*	0.08
	Monetary vs. US	11.75***	0.26
	Monetary vs. FS	11.34***	0.25
	Group	1.24	0.04
	Condition $\times$ Group	1.23	0.04
Accuracy	Condition	1.97	0.06
	Group	6.01**	0.15
	Condition $\times$ Group	0.99	0.03
Number of responses	Condition	23.18****	0.43
	BL vs. Monetary	65.84****	0.68
	BL vs. US	33.36****	0.52
	BL vs. FS	52.36****	0.63
	BL vs. NR1	25.04****	0.45
	BL vs. NR2	45.97****	0.60
	Group	0.08	0.00
	Condition $\times$ Group	0.21	0.01
Amplitude of responses	Condition	10.61****	0.26
	BL vs. Monetary	13.15***	0.30
	BL vs. US	14.08***	0.31
	BL vs. FS	22.44****	0.42
	BL vs. NR1	30.61****	0.50
	BL vs. NR2	37.42****	0.55
	Group	1.15	0.04
	Condition $\times$ Group	1.19	0.04

Notes. BL = baseline condition; Monetary = monetary reward condition; US = unfamiliar social reward condition; FS = familiar social reward condition; NR1 = first non-reward condition; NR2 = second non-reward condition.

\* $P < 0.10$ ; \*\* $P < 0.05$ ; \*\*\* $P < 0.01$ ; \*\*\*\* $P < 0.001$ .



**Figure 1.** Reaction time and accuracy across conditions.

$P = 0.002$ ,  $\eta_p^2 = 0.25$ , social reward conditions, but there were no differences between monetary reward and non-reward conditions, all  $F$ -values  $< 0.63$ , all  $P$ -values  $> 0.40$ . In addition, there was not a significant effect of diagnostic group on reaction time,  $F(1, 34) = 1.24$ ,  $P = 0.27$ ,  $\eta_p^2 = 0.04$ , nor was there a significant Condition  $\times$  Group interaction,  $F(4, 136) = 1.23$ ,  $P = 0.30$ ,  $\eta_p^2 = 0.04$ . Taken together, these analyses suggest a possible difference between reward types in which monetary incentives elicited faster task performance compared with social reward—an effect that was common to participants with and without ASD.

Next, participants' accuracy (assessed as the percentage of trials on which they responded correctly) during the task was examined (see Fig. 1). The ASD group responded less accurately during the task than the control group,  $F(1, 34) = 6.01$ ,  $P = 0.02$ ,  $\eta_p^2 = 0.15$ . However, there was not a significant main effect of condition,  $F(4, 136) = 1.97$ ,  $P = 0.14$ ,  $\eta_p^2 = 0.06$ ,  $\varepsilon = 0.62$ , and there was no significant Condition  $\times$  Group interaction,  $F(4, 136) = 0.99$ ,  $P = 0.39$ ,  $\eta_p^2 = 0.03$ .

#### Electrodermal Outcomes During Reward And Non-Reward

As described above, EDR was collected during a 2-min baseline period, followed by the reward task, yielding six conditions in all (baseline, monetary reward, unfamiliar social reward, familiar social reward, first non-reward, second non-reward). Across these conditions, both NED per epoch and mean AED within each epoch were examined. Analyses followed a two-step approach. First, mean NED and AED values for each condition were compared using repeated measures ANOVA to determine whether EDR differed by diagnostic group or reward type. Next, data were analyzed with repeated measures ANOVAs with diagnostic Group as a between-subjects factor and time (repeated epochs) as a within-subjects factor to determine whether there was an effect of group or epochs on participants' patterns of EDR within each type of reward. In the interest of space, significant effects are described in detail here, whereas the comprehensive set of results is displayed in Tables 2 and 3.

**Table 3. Effects of Time and Diagnostic Group on Electrodermal Responding Within Conditions**

Effect	Number of responses		Amplitude of responses		
	<i>F</i>	$\eta_p^2$	Effect	<i>F</i>	$\eta_p^2$
Baseline					
Time	2.31*	0.07	Time	2.92*	0.09
Group	0.17	0.01	Group	2.40	0.07
Time × Group	4.87***	0.13	Time × Group	0.31	0.01
Linear	3.72*	0.10			
Cubic	9.82***	0.24			
Monetary					
Time	2.36**	0.07	Time	2.54*	0.08
Linear	8.56***	0.22	Linear	4.07*	0.12
Group	0.00	0.00	Group	0.39	0.01
Time × Group	0.45	0.01	Time × Group	0.89	0.03
Unfamiliar social					
Time	2.46**	0.08	Time	3.45***	0.11
Linear	4.12*	0.13	Linear	10.26***	0.27
4th order	4.63**	0.14			
Group	0.00	0.00	Group	0.10	0.00
Time × Group	0.82	0.03	Time × Group	0.27	0.01
Familiar social					
Time	1.93*	0.06	Time	0.96	0.03
Group	0.03	0.00	Group	0.12	0.00
Time × Group	0.77	0.03	Time × Group	0.72	0.02
Non-reward 1					
Time	0.55	0.02	Time	1.33	0.05
Group	0.00	0.00	Group	0.02	0.00
Time × Group	1.97*	0.07	Time × Group	1.69	0.06
Non-reward 2					
Time	1.94	0.07	Time	2.95**	0.10
Group	0.03	0.00	Linear	5.72**	0.18
Time × Group	3.05**	0.11	Group	0.02	0.00
Quadratic	11.70***	0.31	Time × Group	0.38	0.01

\* $P < 0.10$ ; \*\*  $P < 0.05$ ; \*\*\*  $P < 0.01$ ; \*\*\*\*  $P < 0.001$ .

**Number of EDR across conditions.** The number of responses differed significantly across the conditions,  $F(5, 155) = 23.18$ ,  $P < 0.001$ ,  $\eta_p^2 = 0.43$ ,  $\varepsilon = 0.72$ . See Figure 2. Simple contrasts comparing each condition to baseline indicated that participants exhibited EDRs more frequently during periods of reward and non-reward than during baseline, monetary  $F(1, 31) = 65.84$ ,  $P < 0.001$ ,  $\eta_p^2 = 0.68$ ; unfamiliar social  $F(1, 31) = 33.36$ ,  $P < 0.001$ ,  $\eta_p^2 = 0.52$ ; familiar social  $F(1, 31) = 52.36$ ,  $P < 0.001$ ,  $\eta_p^2 = 0.63$ ; first non-reward  $F(1, 31) = 25.04$ ,  $P < 0.001$ ,  $\eta_p^2 = 0.45$ ; second non-reward  $F(1, 31) = 45.97$ ,  $P < 0.001$ ,  $\eta_p^2 = 0.60$ . The effect of group was not significant,  $F(1, 31) = 0.08$ ,  $P = 0.78$ ,  $\eta_p^2 < 0.00$ , nor was there a Condition × Group interaction,  $F(5, 155) = 0.21$ ,  $P = 0.92$ ,  $\eta_p^2 < 0.01$ .

**Number of EDRs within conditions.** Next, we examined the number of responses within each condition (see Fig. 3) for effects of time and diagnostic group. Within the baseline condition, there was a significant Group × Time interaction,  $F(3, 96) = 4.87$ ,  $P = 0.003$ ,

$\eta_p^2 = 0.13$ , indicating that the pattern of change in number of EDRs per epoch differed between the control and ASD groups during the resting baseline period. Tests of simple effects indicated that this interaction was such that the control group showed significant variability in the number of EDRs per epoch across the baseline period, with each epoch differing significantly from its neighboring epochs ( $P_s < 0.04$ ). In contrast, the number of EDRs per epoch did not vary significantly across the epochs of the baseline period for the ASD group (all  $P_s > 0.05$ ). During the monetary block, the effect of time was significant,  $F(5, 155) = 2.36$ ,  $P = 0.04$ ,  $\eta_p^2 = 0.07$ , and linear in nature,  $F(1, 31) = 8.56$ ,  $P = 0.006$ ,  $\eta_p^2 = 0.22$ , with the number of EDRs decreasing over the course of each block. A similar effect of time was observed within the unfamiliar social reward condition as well,  $F(5, 140) = 2.46$ ,  $P = 0.04$ ,  $\eta_p^2 = 0.08$ , linear effect,  $F(1, 28) = 4.12$ ,  $P = 0.05$ ,  $\eta_p^2 = 0.13$ , where responses became less frequent over the course of the block. With regard to non-reward, the second non-reward block was characterized by a Group × Time interaction,  $F(5, 130) = 3.05$ ,  $P = 0.023$ ,  $\eta_p^2 = 0.11$ ,  $\varepsilon = 0.75$ . Simple effects tests revealed that the control group decreased the number of EDRs from the first to the second epochs ( $P = 0.005$ ), and maintained that decrease over the remaining epochs. The ASD group, in contrast, initially decreased in number of EDRs ( $P = 0.01$ ) but then significantly increased in EDRs for a series of epochs ( $P = 0.01$ ) before decreasing toward the end of the non-reward block ( $P = 0.008$ ).

To better understand this interaction during the second non-reward period, we then conducted a repeated measures ANOVA to determine whether the number of EDRs during non-reward differed according to whether the non-reward condition followed a social (unfamiliar and familiar combined) or nonsocial (monetary) reward block.<sup>1</sup> Diagnostic group and reward type (nonsocial vs. social) immediately preceding the second non-reward block were entered as between-subjects factors, with repeated epochs as a within-subjects factor. This analysis revealed a main effect of time,  $F(5, 120) = 2.95$ ,  $P = 0.03$ ,  $\eta_p^2 = 0.11$ , with a cubic component such that the number of EDRs per epoch initially decreased before increasing and then decreasing again,  $F(1, 24) = 9.32$ ,  $P = 0.005$ ,  $\eta_p^2 = 0.28$ . Reward type was significant as well,  $F(1, 24) = 4.91$ ,  $P = 0.04$ ,  $\eta_p^2 = 0.17$ , such that more EDRs per epoch were observed during non-reward when it followed monetary reward vs. social reward. The Time × Reward type interaction was significant as well,  $F(5, 120) = 2.58$ ,  $P = 0.048$ ,  $\eta_p^2 = 0.10$ , and the difference in number of EDRs between the non-social and social reward types decreased over the course of the non-reward block; Epoch 1,  $P = 0.003$ ; Epoch 6,  $P = 0.702$ .

<sup>1</sup>We thank an anonymous reviewer for this analytic suggestion.

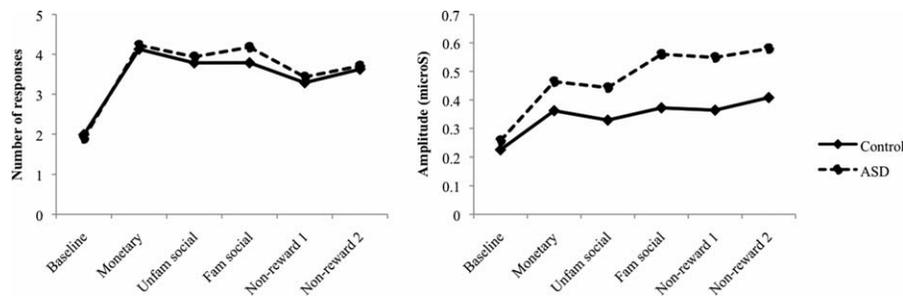


Figure 2. Electrodermal responses across all conditions.

Finally, the Group  $\times$  Time interaction identified earlier remained significant,  $F(5,120) = 3.18$ ,  $P = 0.02$ ,  $\eta_p^2 = 0.12$ . Of note, there was not a significant interaction between time, reward type, and diagnostic group,  $F(5,120) = 0.43$ ,  $P = 0.83$ ,  $\eta_p^2 = 0.02$ .

Together, these analyses suggest that the two groups showed generally similar patterns of electrodermal activity to reward, with decreases in the number of EDRs observed per epoch over the course of the reward blocks. In contrast, the groups were marked by subtle differences in patterns of responding under conditions of non-reward. Follow up analyses revealed that ASD-

related differences in non-reward EDR did not appear to be due to the social vs. nonsocial nature of the most recent reward type, as the sample as a whole exhibited more frequent EDRs following discontinuation of non-social reward, with no effect of diagnosis.

**Amplitude of EDRs across conditions.** Analyses of EDR amplitudes revealed an effect of condition,  $F(5, 155) = 10.61$ ,  $P < 0.001$ ,  $\eta_p^2 = 0.26$ ,  $\epsilon = 0.73$  (see Fig. 2). Mean amplitudes during all of the reward and non-reward conditions were significantly higher than those

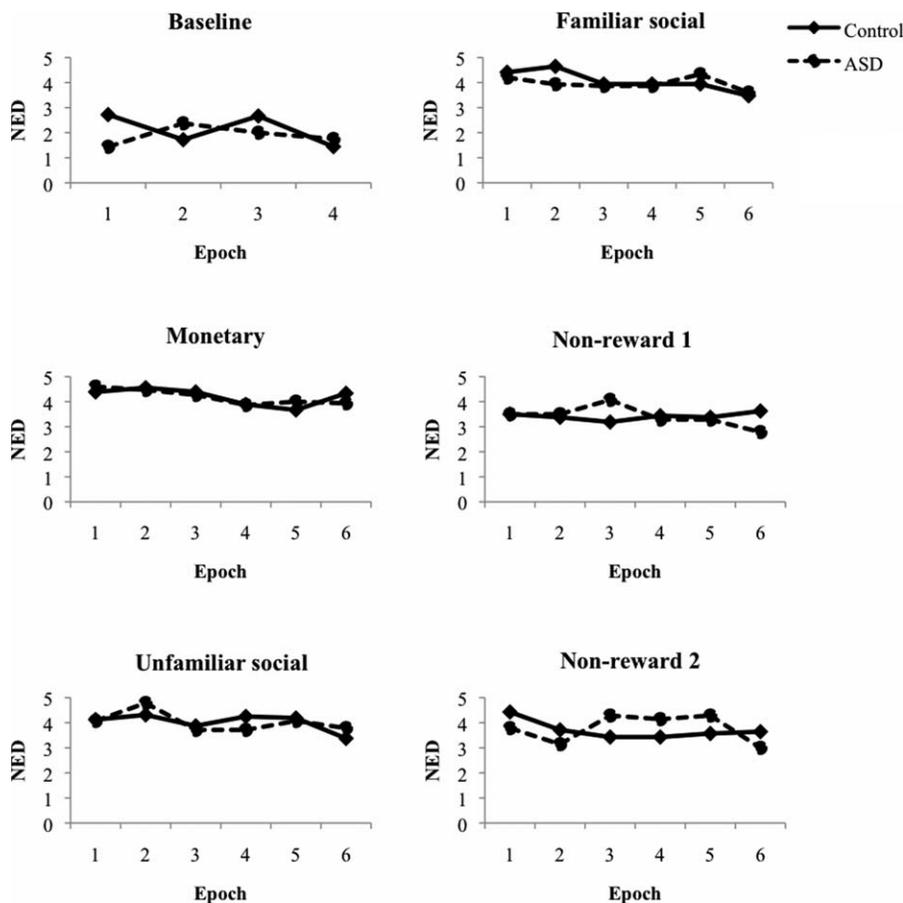


Figure 3. Number of electrodermal responses by group within reward type.

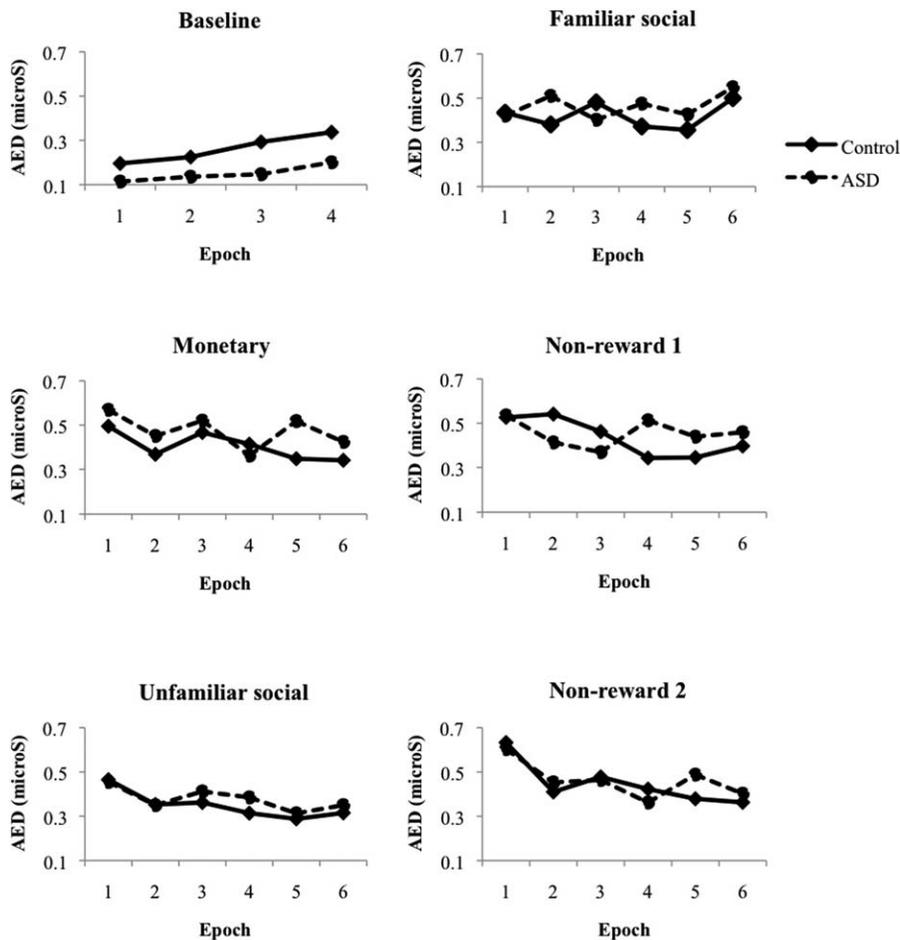
observed during baseline, all  $F_s > 13.1$ , all  $P_s < 0.002$ . The groups did not differ overall,  $F(1, 31) = 1.15$ ,  $P = 0.29$ ,  $\eta_p^2 = 0.04$ , nor was there an interaction between Group and Time,  $F(5, 155) = 1.19$ ,  $P = 0.32$ ,  $\eta_p^2 = 0.04$ .

**Amplitude of EDRs within conditions.** Next, changes in response amplitudes were investigated within each reward and non-reward block (see Fig. 4). In response to unfamiliar social reward, EDR amplitudes decreased significantly over the course of the block,  $F(5, 140) = 3.45$ ,  $P = 0.006$ ,  $\eta_p^2 = 0.11$ , in a linear fashion,  $F(1, 28) = 10.26$ ,  $P = 0.003$ ,  $\eta_p^2 = 0.27$ . Interestingly, there was not a comparable decrease in amplitude during familiar social reward,  $F(5, 150) = 0.96$ ,  $P = 0.45$ ,  $\eta_p^2 = 0.03$ . The second non-reward block also elicited decreasing amplitudes,  $F(5, 130) = 2.95$ ,  $P = 0.03$ ,  $\eta_p^2 = 0.10$ ,  $\varepsilon = 0.67$ , that followed a linear pattern,  $F(1, 26) = 5.72$ ,  $P = 0.02$ ,  $\eta_p^2 = 0.18$ . None of the conditions examined were characterized by differences between diagnostic groups, nor by Group  $\times$  Time interactions, all  $F_s < 2.4$ , all  $P_s > 0.13$ . Thus, participants exhibited similar patterns of EDR amplitude change, regardless of

diagnostic group. Amplitudes tended to decrease over the course of some reward and non-reward blocks within the task, and this pattern was shared by participants with and without ASD.

*Correlations Between EDR and Social-Emotional Functioning*

Relations between EDR and parent-report variables were next examined for both diagnostic groups (see Table 4). As a first step, reliability analyses were conducted for outcomes of interest (SSIS Social Skills, SSIS Problem Behaviors, CBCL Internalizing, CBCL Externalizing) in our sample. Internal consistency as assessed with Cronbach's alpha ranged from 0.88 to 0.94 for these four measures, all falling within the good to excellent range. With regard to EDR-behavior links, the most consistent patterns emerged with respect to problem behaviors. A consistent pattern of negative correlations between problem behavior and the amplitude of EDRs was observed across multiple reward and non-reward conditions. Children with larger responses to reward and non-reward tended to have fewer problem behaviors over all. There was a similar pattern of negative correlations between the AED to reward and non-reward and



**Figure 4.** Amplitude of electrodermal responses by group within reward type.

**Table 4. Correlations Between Electrodermal Responding and Parent-Reported Social and Emotional Functioning**

	SSIS social skills	SSIS problem behavior	CBCL internalizing	CBCL externalizing
Control Group				
Baseline				
NED	0.44*	0.18	0.31	0.40
AED	0.44*	0.00	0.19	0.13
Monetary				
NED	0.37	-0.19	-0.05	0.11
AED	0.40	-0.57**	-0.20	-0.12
Unfamiliar social				
NED	-0.09	-0.18	0.32	-0.07
AED	-0.04	-0.49**	-0.12	-0.30
Familiar social				
NED	0.44*	-0.07	0.27	0.23
AED	0.21	-0.49**	-0.18	-0.25
Non-reward 1				
NED	0.28	0.04	0.23	0.13
AED	0.48**	-0.17	-0.07	0.21
Non-reward 2				
NED	0.32	0.00	0.28	0.09
AED	0.12	-0.64***	-0.22	-0.32
ASD Group				
Baseline				
NED	0.33	-0.46*	-0.47 <sup>a</sup>	-0.41
AED	-0.26	-0.46*	-0.37	-0.23
Monetary				
NED	0.16	-0.23	-0.09	-0.09
AED	-0.23	-0.55**	-0.55**	-0.14
Unfamiliar social				
NED	0.04	-0.13	-0.13	-0.03
AED	-0.36	-0.48*	-0.49 <sup>a</sup>	-0.09
Familiar social				
NED	-0.02	-0.49*	-0.35	-0.14
AED	-0.33	-0.43*	-0.49*	-0.12
Non-reward 1				
NED	-0.07	0.04	-0.17	0.13
AED	-0.36	-0.43	-0.49 <sup>a</sup>	-0.02
Non-reward 2				
NED	0.00	0.11	0.07	0.15
AED	-0.45*	-0.41	-0.52**	-0.16

Notes. SSIS = Social Skills Improvement System [Gresham & Elliot, 2008]; CBCL = Child Behavior Checklist [Achenbach, 1991]; NED = Number of electrodermal responses; AED = Amplitude of electrodermal responses.

<sup>a</sup>\* $P < 0.10$ ; \*\* $P < 0.05$ ; \*\*\* $P < 0.01$ .

scores on the Internalizing subscale of the CBCL. Again, children with higher amplitude responses tended to have fewer internalizing symptoms. Results also suggested links between both the number and AEDs and children's social skills. In general, more frequent and larger EDRs were associated with better social skills.

## Discussion

Our primary goal was to characterize behavioral and electrodermal responses to both reward (monetary,

unfamiliar social, familiar social) and non-reward among children with ASD, including links between reward responding and social and emotional functioning. Our data yielded both expected and unexpected findings with regard to these goals. Behaviorally, performance on the reward task was influenced by diagnostic status, such that the group with ASD was less accurate than controls. This difference may reflect deficits in neurocognitive functioning (e.g., set-shifting, sustained attention, perseveration), but the fact that accuracy in all conditions exceeded 88% suggests that participants with ASD were able to perform with a high degree of success. The effect of reward condition on reaction time approached significance, suggesting that participants were slightly faster to respond under conditions of monetary reward than social reward. From a functional perspective, this could suggest that they experienced money as more reinforcing than videos of adults giving praise, which would be consistent with some findings among children with typical development, in which monetary reward elicited better performance on a go/no-go task than did social reward [Kohls, Peltzer, Herpertz-Dahlmann, & Konrad, 2009]. Alternatively, children in our study may have experienced a novelty effect during the social reward conditions, such that viewing the social reward video for a given trial captured their attention and slowed their processing of the subsequent trial. Unfortunately, this possibility cannot be ruled out based on the current data. Regardless, the absence of any Group  $\times$  Condition interactions on behavioral outcomes was contrary to hypotheses, and suggests that behavioral response to reward was largely similar across the two groups.

With regard to electrodermal data, participants as a whole showed more frequent and larger EDRs during reward and non-reward conditions than during resting baseline, and patterns of change within the various reward conditions were generally similar between the groups. That is, both the number and AEDs tended to decrease across monetary and unfamiliar social reward conditions, suggesting habituation, consistent with previous studies. During non-reward, however, subtle differences were evident between the groups. The second non-reward block was marked by an interaction effect, in which the control group responded to the absence of expected reward with a rapid decrease in the number of EDRs. In contrast, those with ASD showed increased EDRs initially before decreasing. A similar pattern was observed during the first non-reward block, but in that case only approached significance. Together, this pattern might suggest that whereas controls adapted their physiological responding quickly and efficiently to changing reward contingencies, those with ASD were less flexible and adaptive in their physiological responses when environmental cues and contingencies

changed. To the extent that this effect carries over into real-life social interactions, it would suggest that children with ASD struggle to adapt underlying physiological processes to meet changing environmental demands. Such difficulties may appear as a tendency to perseverate on a behavior that has previously elicited reward but is no longer appropriate or actually elicits negative feedback from others.

EDRs to reward correlated with parent-reported behavior in interesting patterns within our sample. The most consistent correlations were between response amplitude and problem behaviors as assessed by the SSIS, such that higher amplitudes in response to reward and non-reward were associated with fewer problem behaviors. Finer-grained behavioral assessment through the CBCL revealed a series of correlations between EDR amplitude and Internalizing *T*-scores within the group with ASD. Again, higher amplitude was associated with better functioning—that is, fewer internalizing symptoms. This subscale is a composite of items assessing anxiety (e.g., fearfulness, shyness), depression (e.g., guilt, sadness), and somatic complaints (e.g., headaches). To the extent that its correlations with EDR amplitude were driven by anxiety features, our finding is in keeping with previous literature describing lower tonic and phasic skin conductance amplitudes among individuals high in anxiety [Naveteur & Baque, 1987; Naveteur & Roy, 1989]. In terms of social behavior, our analyses suggested that EDRs were related to social skills, such that better social skills corresponded to larger responses. This is particularly interesting in light of recent evidence that skin conductance amplitude may be sensitive to the effects of oxytocin [Lin, Kashino, Ohta, Yamada, Tani, & Kato, 2014], which underlies a host of social cognitive skills and behaviors including the use of eye gaze [Guastella, Mitchell, & Dadds, 2008], perspective-taking [Domes, Heinrichs, Michel, Berger, & Herpertz, 2007], and social affiliation [Feldman, 2012], and has been suggested to contribute to alterations in reward-related brain processes [Chevallier et al., 2012; Dawson & Bernier, 2007; Stavropoulos & Carver, 2013]. Although replication in a larger sample will be valuable, our findings indicate that EDR in response to potentially rewarding situations may be an important marker of social and emotional well-being.

A number of limitations should be considered in interpreting our findings. First, inclusion of an additional non-reward block at the start of the reward task would have allowed assessment of behavioral and EDR based solely on task performance (with no knowledge or expectation of reward), and thus could have disentangled more clearly effects of task activity (e.g., attention, button-pressing) vs. reward-oriented activity. Second, several participants reported regular use of medications. Among controls, one participant used an

antihypertensive (guanfacine). Within the ASD group, three participants used selective serotonin reuptake inhibitors (citalopram, sertraline, fluoxetine), two used atypical antipsychotics (aripiprazole, risperidone), one used an anticonvulsant (topiramate), and one used an antihypertensive (guanfacine). Although none of our participants had used stimulants (which likely carry the largest effects on the neural substrates of autonomic reactivity to reward; [Swanson & Volkow, 2002]) within the 36 hr prior to data collection, our sample was not large enough to permit analysis of medication effects and future research may benefit from such analyses. Finally, the absence of group differences in the number and amplitude of EDRs during reward conditions may be due in part to the size of our sample (36 boys in total). Replication in a larger sample will be important to more conclusively understand the effects of monetary and social reward on electrodermal activity in ASD.

These issues notwithstanding, the findings presented here may have implications for etiological models of ASD, and intervention strategies. The social motivation hypothesis [Chevallier et al., 2012; Dawson & Bernier, 2007] suggests that deficits in reward processing among individuals with ASD underlie a range of social impairments across development, including disruptions in social orienting, speech and face perception, joint attention, communication, and imitation. Our study provides some support for alterations to reward processing mechanisms in ASD. Despite similar patterns of responding to monetary and social reward, participants with ASD differed in subtle ways from controls during periods of non-reward, and EDRs to reward and non-reward correlated with social and emotional functioning. Although not always included within investigations of reward processing, non-reward is a critical component of reward-based learning and likely plays a critical role both in shaping behavior and in guiding the specialized brain development that subserves social behavior and cognition across the lifespan [Dawson & Bernier, 2007; Dichter, Damiano, & Allen, 2012; Kohls et al., 2012].

Our findings suggest a number of avenues that may be fruitful for future research. First, comparison of EDR among individuals with differing symptom presentations across the full range of ASD could be quite valuable. Heterogeneity of ASD is well documented [Faja & Dawson, 2013], and reward responding and EDR may differ according to characteristics or subgroups within the ASD spectrum. For example, Wing and Gould [1979] described three profiles of social behavior among children with ASD: aloof, characterized by low apparent interest in social interactions; passive, characterized by acceptance of social interaction but few efforts to approach others; and active-but-odd, characterized by social approach that is repetitive or idiosyncratic and

received poorly by others. Based on our findings, one might anticipate that these subgroups would exhibit different profiles of reward responding and EDR. Whereas individuals in the passive or aloof subgroups might display reduced response to social reward (thus failing to motivate social approach), those in the active-but-odd subgroup might exhibit intact response to reward (thus motivating social approach) but altered response to non-reward such that social approach is not appropriately dampened or discontinued when social or environmental cues would suggest it should be. Thus, EDR may map more finely onto social strengths and difficulties within the larger ASD category.

Similarly, electrodermal and other indices of reward responding may vary according to individual differences in intellectual ability, severity of ASD symptoms, or intervention history. Indeed, conflicting findings among early investigations of EDR in ASD were revealed to be the result of differing IQ levels among study samples [Van Engeland, 1984; Zahn et al, 1987]. Our sample was selected such that participant groups were generally similar with regard to IQ, and our procedures required that participants understand verbal instructions, tolerate collection of physiological data, and complete a lengthy study protocol. Consequently, our results are most applicable to individuals with relatively high-functioning ASD, and the exploration of EDR and reward among individuals with lower IQ, greater sensory sensitivity, or more severe ASD symptoms could yield a different set of insights. To the extent that altered EDR to non-reward is a core etiological feature of ASD, we would expect consistent findings across these diverse populations. However, the generalizability of links between EDR and non-reward remains an open question and will be an important avenue of future research.

Turning to the conceptualization of reward itself, our understanding of reward-related processes would be enriched by increased efforts to empirically equate non-social and social reward within study tasks. Nonsocial reward has often been operationalized as receipt of money [e.g., Scott-Van Zeeland, Dapretto, Ghahremani, Poldrack, & Bookheimer, 2010] or food [e.g., Stavropoulos & Carver, 2014], with social reward operationalized as smiling faces [e.g., Kohls, Peltzer, Schulte-Ruther, Kamp-Becker, Remschmidt, & Konrad, 2011] or spoken praise [e.g., Kohls, Perino, Taylor, Madva, Cayless, & Schultz, 2013]. Inherent within this approach are differences in the tangibility of the reward and the degree to which the reward accumulates over time. Consequently, findings comparing nonsocial vs. social reward are confounded by these factors, as differences between reward conditions cannot be attributed with certainty to the type of reward rather than these factors. Future efforts might attempt to calibrate reward types at the

individual level to maximize comparability between conditions before exploring differences in behavioral or biological markers across diagnostic groups.

Finally, an intriguing strategy would be to use an event-locked design with mobile monitoring to allow assessment of electrodermal and behavioral responding during real-life social interactions. Such an approach would more finely characterize responses to social interactions theorized to be rewarding (e.g., a peer's positive response to a social bid), as well as those theorized to be extinguishing (e.g., a peer's ignoring of a social bid), and also better describe the degree to which these experiences predict or modify future behavior. Although laboratory-based designs allow control of extraneous variables, questions related to social reward are valuable because of both their theoretical importance and their direct relevance for the everyday experiences of individuals with ASD. Thus, improving the ecological validity in this area of research could yield meaningful benefits with significant implications for intervention.

### Acknowledgments

Preparation of this manuscript was provided in part by an Autism Speaks Meixner Translational Postdoctoral Fellowship to Emily Neuhaus.

### References

- Achenbach, T.M. (1991). *Manual for the Child Behavior Checklist/4-18 and 1991 profile*. Burlington, VT: University of Vermont Department of Psychiatry.
- Achenbach, T.M., & Rescorla, L.A. (2001). *Manual for ASEBA School-Age Forms and Profiles*. Burlington, VT: University of Vermont, Research Center for Children, Youth, and Families.
- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders (4th ed., text rev.)* Washington, DC: Author.
- Beauchaine, T.P. (2001). Vagal tone, development, and Gray's motivational theory: Toward an integrated model of autonomic nervous system functioning in psychopathology. *Development and Psychopathology*, 13, 183–214.
- Beauchaine, T.P., Katkin, E.S., Strassberg, Z., & Snarr, J. (2001). Disinhibitory psychopathology in male adolescents: Discriminating conduct disorder from attention-deficit/hyperactivity disorder through concurrent assessment of multiple autonomic states. *Journal of Abnormal Psychology*, 110, 610–624.
- Bernier, R., Dawson, G., Webb, S., & Murias, M. (2007). EEG mu rhythm and imitation impairments in individuals with autism spectrum disorder. *Brain and Cognition*, 64, 228–237.
- Chevallier, C., Kohls, G., Troiani, V., Brodtkin, E.S., & Schultz, R.T. (2012). The social motivation theory of autism. *Trends in Cognitive Science*, 16, 231–239.
- Dawson, G., & Bernier, R. (2007). Development of social brain circuitry in autism. In Coch, D., Dawson, G., & Fischer, K. (Eds.),

- Human behavior and the developing brain: Atypical development (2nd ed., pp. 28–55). New York: Guilford Press.
- Dawson, G., Osterling, J., Rinaldi, J., Carver, L., & McPartland, J. (2001). Brief report: Recognition memory and stimulus-reward associations: indirect support for the role of ventromedial prefrontal dysfunction in autism. *Journal of Autism and Developmental Disorders*, 31, 337–341.
- Dawson, G., Webb, S.J., Wijsman, E., Schellenberg, G., Estes, A., Munson, J., et al. (2005). Neurocognitive and electrophysiological evidence of altered face processing in parents of children with autism: Implications for a model of abnormal development of social brain circuitry in autism. *Development and Psychopathology*, 17, 679–697.
- Dawson, M.E., Schell, A.M., & Filion, D.L. (2007). The electrodermal system. In Cacioppo, J.T., Tassinari, L.G., & Berntson, G.G. (Eds.), *Handbook of Psychophysiology* (3rd ed., pp. 159–181). New York: Cambridge University Press.
- Demurie, E., Roeyers, H., Baeyens, D., & Sonuga-Barke, E. (2011). Common alterations in sensitivity to type but not amount of reward in ADHD and autism spectrum disorders. *Journal of Child Psychology and Psychiatry*, 52, 1164–1173.
- Dichter, G.S., Damiano, C.A., & Allen, J.A. (2012). Reward circuitry dysfunction in psychiatric and neurodevelopmental disorders and genetic syndromes: Animal models and clinical findings. *Journal of Neurodevelopmental Disorders*, 4, 19.
- Dichter, G.S., Felder, J.N., Green, S.R., Rittenberg, A.M., Sasson, N.J., & Bodfish, J.W. (2012). Reward circuitry function in autism spectrum disorders. *Social Cognitive and Affective Neuroscience*, 7, 160–172.
- Dichter, G.S., Richey, J.A., Rittenberg, A.M., Sabatino, A., & Bodfish, J.W. (2012). Reward circuitry function in autism during face anticipation and outcomes. *Journal of Autism and Developmental Disorders*, 42, 147–160.
- Domes, G., Heinrichs, M., Michel, A., Berger, C., & Herpertz, S.C. (2007). Oxytocin improves "mind-reading" in humans. *Biological Psychiatry*, 61, 731–733.
- Faja, S., & Dawson, G. (2013). Autism spectrum disorders. In Beauchaine, T.P., & Hinshaw, S.P. (Eds.), *Child and Adolescent Psychopathology* (2nd ed., pp. 649–684). Hoboken, NJ: Wiley.
- Faja, S., Murias, M., Beauchaine, T.P., & Dawson, G. (2013). Reward-based decision making and electrodermal responding by young children with autism spectrum disorders during a gambling task. *Autism Research*, 6, 494–505.
- Feldman, R. (2012). Oxytocin and social affiliation in humans. *Hormones and Behavior*, 61, 380–391.
- Fowles, D.C. (1980). The three arousal model: Implications of Gray's two-factor learning theory for heart rate, electrodermal activity, and psychopathy. *Psychophysiology*, 17, 87–104.
- Gervais, H., Belin, P., Boddaert, N., Leboyer, M., Coez, A., Sfaello, I., et al. (2004). Abnormal cortical voice processing in autism. *Nature Neuroscience*, 7, 801–802.
- Geurts, H.M., Luman, M., & van Meel, C.S. (2008). What's in a game: The effect of social motivation on interference control in boys with ADHD and autism spectrum disorders. *Journal of Child Psychology and Psychiatry*, 49, 848–857.
- Gotham, K., Risi, S., Pickles, A., & Lord, C. (2007). The Autism Diagnostic Observation Schedule: Revised algorithms for improved diagnostic validity. *Journal of Autism and Developmental Disorders*, 37, 613–627.
- Gray, J. (1987). Perspectives on anxiety and impulsivity: A commentary. *Journal of Research in Personality*, 21, 493–509.
- Gresham, F.M., & Elliott, S.N. (2008). *Social Skills Improvement System*. Minneapolis, MN: Pearson.
- Guastella, A.J., Mitchell, P.B., & Dadds, M.R. (2008). Oxytocin increases gaze to the eye region of human faces. *Biological Psychiatry*, 63, 3–5.
- Hadjikhani, N., Joseph, R.M., Snyder, J., & Tager-Flusberg, H. (2007). Abnormal activation of the social brain during face perception in autism. *Human Brain Mapping*, 28, 441–449.
- Hirstein, W., Iversen, P., & Ramachandran, V.S. (2001). Autonomic responses of autistic children to people and objects. *Proceedings in Biological Science*, 268, 1883–1888.
- Hubert, B.E., Wicker, B., Monfardini, E., & Deruelle, C. (2009). Electrodermal reactivity to emotion processing in adults with autistic spectrum disorders. *Autism*, 13, 9–19.
- Johnson, S.A., Yechiam, E., Murphy, R.R., Queller, S., & Stout, J.C. (2006). Motivational processes and autonomic responsiveness in Asperger's disorder: Evidence from the Iowa Gambling Task. *Journal of the International Neuropsychological Society*, 12, 668–676.
- Klin, A., Jones, W., Schultz, R., Volkmar, F., & Cohen, D. (2002). Visual fixation patterns during viewing of naturalistic social situations as predictors of social competence in individuals with autism. *Archives of General Psychiatry*, 59, 809–816.
- Kohls, G., Chevallier, C., Troiani, V., & Schultz, R.T. (2012). Social 'wanting' dysfunction in autism: Neurobiological underpinnings and treatment implications. *Journal of Neurodevelopmental Disorders*, 4, 10.
- Kohls, G., Peltzer, J., Schulte-Ruther, M., Kamp-Becker, I., Remschmidt, H., & Konrad, K. (2011). Atypical brain responses to reward cues in autism as revealed by event-related potentials. *Journal of Autism and Developmental Disorders*, 41, 1523–1533.
- Kohls, G., Peltzer, J., Herpertz-Dahlmann, B., & Konrad, K. (2009). Differential effects of social and non-social reward on response inhibition in children and adolescents. *Developmental Science*, 12, 614–625.
- Kohls, G., Perino, M.T., Taylor, J.M., Madva, E.N., Cayless, S.J., & Schultz, R.T. (2013). The nucleus accumbens is involved in both the pursuit of social reward and the avoidance of social punishment. *Neuropsychologia*, 51, 2062–2069.
- Kylliäinen, A., & Hietanen, J.K. (2006). Skin conductance responses to another person's gaze in children with autism. *Journal of Autism and Developmental Disorders*, 36, 517–525.
- Kylliäinen, A., Wallace, S., Coutanche, M.N., Leppänen, J.M., Cusack, J., Bailey, A.J., et al. (2012). Affective-motivational brain responses to direct gaze in children with autism spectrum disorder. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, 53, 790–797.
- Lin, I.-F., Kashino, M., Ohta, H., Yamada, T., Tani, M., & Kato, N. (2014). The effect of intranasal oxytocin versus placebo treatment on the autonomic responses to human sounds in autism: A single-blind, randomized, placebo-controlled, crossover design study. *Molecular Autism*, 5, 20.
- Lord, C., Rutter, M., DiLavore, P.C., & Risi, S. (2003). *Autism Diagnostic Observation Schedule Manual*. Los Angeles, CA: Western Psychological Services.

- Lord, C., Rutter, M., & Le Couteur, A. (1994). Autism Diagnostic Interview-Revised: A revised version of a diagnostic interview for caregivers of individuals with possible pervasive developmental disorders. *Journal of Autism and Developmental Disorders*, 24, 659–685.
- Mathersul, D., McDonald, S., & Rushby, J.A. (2013). Psychophysiological correlates of social judgement in high-functioning adults with autism spectrum disorder. *International Journal of Psychophysiology*, 87, 88–94.
- Mulligan, A., Richardson, T., Anney, R.J.L., & Gill, M. (2009). The Social Communication Questionnaire in a sample of the general population of school-going children. *Irish Journal of Medical Science*, 178, 193–199.
- Mundy, P., Sigman, M., & Kasari, C. (1990). A longitudinal study of joint attention and language development in autistic children. *Journal of Autism and Developmental Disorders*, 20, 115–128.
- Naveteur, J., & Freixa I Baque, E. (1987). Individual differences in electrodermal activity as a function of subjects' anxiety. *Personality and Individual Differences*, 8, 615–626.
- Naveteur, J., & Roy, J.-C. (1989). Inhibition of the electrodermal activity in anxious subjects during a frustrative non reward situation. *International Journal of Psychophysiology*, 7, 330–332.
- Neuhaus, E., Beauchaine, T.P., & Bernier, R. (2010). Neurobiological correlates of social functioning in autism. *Clinical Psychology Review*, 30, 733–748.
- Oberman, L.M., Hubbard, E.M., McCleery, J.P., Altschuler, E.L., Ramachandran, V.S., & Pineda, J.A. (2005). EEG evidence for mirror neuron dysfunction in autism spectrum disorders. *Cognitive Brain Research*, 24, 190–198.
- Oberman, L.M., Ramachandran, V.S., & Pineda, J.A. (2008). Modulation of mu suppression in children with autism spectrum disorders in response to familiar or unfamiliar stimuli: The mirror neuron hypothesis. *Neuropsychologia*, 46, 1558–1565.
- Osterling, J., & Dawson, G. (1994). Early recognition of children with autism: A study of first birthday home videotapes. *Journal of Autism and Developmental Disorders*, 24, 247–257.
- Pierce, K., Conant, D., Hazin, R., Stoner, R., & Desmond, J. (2011). Preference for geometric patterns early in life as a risk factor for autism. *Archives of General Psychiatry*, 68, 101–109.
- Pierce, K., Haist, F., Sedaghat, F., & Courchesne, E. (2004). The brain response to personally familiar faces in autism: Findings of fusiform activity and beyond. *Brain*, 127, 2703–2716.
- Rutter, M., Bailey, A., & Lord, C. (2003). *Social Communication Questionnaire (SCQ) manual*. Los Angeles: Western Psychological Services.
- Schmitz, N., Rubia, K., van Amelsvoort, T., Daly, E., Smith, A., & Murphy, D.G.M. (2008). Neural correlates of reward in autism. *British Journal of Psychiatry*, 192, 19–24.
- Scott-Van Zeeland, A.A., Dapretto, M., Ghahremani, D.G., Poldrack, R.A., & Bookheimer, S.Y. (2010). Reward processing in autism. *Autism Research*, 3, 53–67.
- Sherwood, A., Allen, M.T., Obrist, P.A., & Langer, A.W. (1986). Evaluation of beta-adrenergic influences on cardiovascular and metabolic adjustments to physical and psychological stress. *Psychophysiology*, 23, 89–104.
- Stavropoulos, K.K.M., & Carver, L.J. (2013). Social motivation and oxytocin in autism—Implications for joint attention development and intervention. *Journal of Child Psychology and Psychiatry*, 54, 603–618.
- Stavropoulos, K.K.M., & Carver, L.J. (2014). Reward anticipation and processing of social versus nonsocial stimuli in children with and without autism spectrum disorders. *Journal of Child Psychology and Psychiatry*, 55, 1398–1408.
- Swanson, J.M., & Volkow, N.D. (2002). Pharmacokinetic and pharmacodynamics properties of stimulants: Implications for the design of new treatments for ADHD. *Behavioural Brain Research*, 130, 73–78.
- Uno, H. (1977). Sympathetic innervation of the sweat glands and pilorrector muscles of macaques and human beings. *Journal of Investigative Dermatology*, 69, 112–120.
- van den Buuse, M. (1998). Role of the mesolimbic dopamine system in cardiovascular homeostasis. Stimulation of the ventral tegmental area modulates the effect of vasopressin on blood pressure in conscious rats. *Clinical and Experimental Pharmacology and Physiology*, 25, 661–668.
- Van Engeland, H. (1984). The electrodermal orienting response to auditive stimuli in autistic children, normal children, mentally retarded children, and child psychiatric patients. *Journal of Autism and Developmental Disorders*, 14, 261–279.
- Wechsler, D. (1999). *Wechsler Abbreviated Scale of Intelligence*. San Antonio, TX: The Psychological Corporation.
- Wing, L., & Gould, J. (1979). Severe impairments of social interaction and associated abnormalities in children: Epidemiology and classification. *Journal of Autism and Developmental Disorders*, 9, 11–29.
- Zahn, T.P., Rumsey, J.M., & Van Kammen, D.P. (1987). Autonomic nervous system activity in autistic, schizophrenic, and normal men: Effects of stimulus significance. *Journal of Abnormal Psychology*, 96, 135–144.