Physiological Responses to Social and Non-social Reward Among Children with Autism

Emily Neuhaus

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Reading Committee:

Wendy L. Stone, Chair

Raphael A. Bernier

Theodore P. Beauchaine

Program Authorized to Offer Degree:

Department of Psychology

University of Washington

Abstract

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Emily Neuhaus

Chair of the Supervisory Committee:
Professor Wendy L. Stone
Psychology

Well-documented and widespread functional impairments among those with autism spectrum disorders (ASDs) are often construed as stemming from either reduced sensitivity to or aversion to stimuli that are social in nature. In fact, individuals with ASDs display altered patterns of neural responding to a variety of social stimuli, although familiarity may moderate such effects. Dawson and colleagues (Dawson et al., 2005; Dawson & Bernier, 2007) argue that reduced sensitivity to social stimuli reflects disrupted reward processing, reflected in behavioral and neurological impairments. In this study, I explored the effects of social and nonsocial reward on autonomic functioning among 8- to 12-year old boys with and without ASDs, with attention to the potential moderating effects of familiarity. During a simple matching task, participants with and without ASDs had slower reactions during social versus nonsocial reward, and boys with ASDs were less accurate than controls in their responses. Physiologically, the groups differed on baseline parasympathetic (respiratory sinus arrhythmia; RSA) functioning, but did not differ on baseline sympathetic (cardiac pre-ejection period; PEP) functioning, or on RSA or PEP

reactivity. In addition, baseline RSA was correlated with parent-reported social behavior but not observational measures of social functioning. Finally, child RSA and PEP reactivity were not correlated with interaction partners' social behavior during a structured task. These findings may suggest a need to integrate relationship-based and reinforcement-based strategies in intervention, and to further explore relations between parasympathetic function and social behavior among individuals with ASDs.

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INTRODUCTION

Although current diagnostic criteria for autism include three broad areas of impairment (social, communicative, and behavioral; American Psychiatric Association, 2000), research indicates that social impairments may be foundational, as they are among the earliest symptoms to emerge (Dawson & Bernier, 2007). Parents of children later diagnosed with autism spectrum disorders (ASDs) often report early concerns regarding poor eye contact, lack of shared enjoyment, and discomfort with physical touch (Young, Brewer, & Pattison, 2003). Osterling and Dawson (1994) found that reductions in pointing, showing objects, looking at others, and orienting to one's name at one year of age provided near perfect classification of infants who were and were not later diagnosed with ASDs. Within the next few years of life, the former group was differentiated from controls by higher ratings in preference for aloneness, lack of eye contact, and gaze aversion, as well as deficits in social interaction, peer interest, and imitation (Adrien et al., 1993; Clifford, Young, & Williamson, 2007; Receveur et al., 2005). By early childhood, children with ASDs show impairments relative to typically developing children in a number of important areas, including social orienting, joint attention, face processing, imitation, and responses to emotional cues (Dawson & Bernier, 2007; Dawson et al., 2004; Rosset et al., 2008). Consequently, many of the behavioral characteristics of ASDs can be construed as reduced sensitivity to stimuli that are social in nature.

A corresponding reduction in sensitivity to social stimuli is apparent at the neurobiological level. For example, among individuals without ASDs, vocal sounds elicit greater responding in the superior temporal sulcus (STS) than do nonvocal sounds (Gervais et al., 2004), suggesting that the region differentiates between social and nonsocial auditory stimuli. In contrast, among individuals with ASDs, activation patterns of the STS are similar across stimulus types. Moreover, in contrast to findings from normal controls, no areas of auditory cortex display

greater activation to vocal stimuli over non-vocal stimuli among those with ASDs, suggesting an absence of differentiation between stimulus types. Similarly, typically-developing participants display increased STS activation to direct versus averted gaze (Pelphrey, Morris, & McCarthy, 2005; Senju, Tojo, Yaguchi, & Hasegawa, 2005) and biological versus nonbiological motion (Carter & Pelphrey, 2006), whereas participants with ASDs do not.

Individuals with ASDs also fail to display selective sensitivity to social stimuli across regions important to face processing and emotion recognition. Among controls, activation in the so-called fusiform face area (FFA) is greater in response to faces than objects, a pattern not observed among participants with ASDs (Schultz et al., 2000). Indeed, among samples with ASDs, the FFA is less responsive to human faces across a variety of tasks (Hubl et al., 2003; McPartland, Dawson, Webb, Panagiotides, & Carver, 2004; Pierce, Muller, Ambrose, Allen, & Courchesne, 2001). Similar findings extend to the amygdala, where reduced activation among those with ASDs relative to controls is observed during emotion processing tasks (Ashwin, Baron-Cohen, Wheelwright, O'Riordan, & Bullmore, 2007), emotion discrimination (Critchley et al., 2000), and mental state judgment (Baron-Cohen et al., 1999). These findings are bolstered by reports of altered ERPs across a network of neural regions important to imitation while those with ASDs observe intentional action (Bernier, Dawson, Webb, & Murias, 2007; Oberman et al., 2005). Taken together, this body of literature indicates that reduced sensitivity to social stimuli extends beyond behavioral indices and is also evident neurobiologically.

Several recent studies indicate that sensitivity to social stimuli among those with ASDs may be moderated by familiarity with the stimuli in question. In these studies, more familiar stimuli elicit neural responding that more closely resembles that of participants without ASDs. For example, in contrast to findings of reduced activation to faces in general, activation in the fusiform gyrus and amygdala is near normal when participants with ASDs view the faces of well

known individuals (Hadjikhani et al., 2007; Pierce, Haist, Sedaghat, & Courchesne, 2004). Similarly, those with ASDs typically display reduced activity in regions comprising the mirror neuron system while watching videos of human action (Bernier, Dawson, Webb, & Murias, 2007). However, such activity is increased when participants watch videos of their own actions or those of people they know (Oberman, Ramachandran, & Pineda, 2008). Thus, neurobiological sensitivity to stimuli among those with ASDs appears to be influenced by both the social nature of and familiarity with the stimuli in question.

Reward Deficits in ASDs

Dawson and colleagues (e.g., Dawson, 2008; Dawson & Bernier, 2007; Dawson & Faja, 2008) propose that reduced sensitivity to social stimuli might result from impaired reward processing. According to this *social motivation* hypothesis, individuals with ASDs may have difficulty anticipating the reward value of social stimuli. An early deficit in reward processing—either general or specific to social stimuli—would likely have dramatic and pervasive implications for social functioning across development, including compromised conditioned preferences for social affiliation, impaired orienting to social cues, and a failure to integrate social information with neurobiological systems underlying reward (Dawson et al., 2005). Consistent with this notion, Dawson and colleagues found that the ability to learn stimulus-reward associations during neuropsychological tasks was associated with concurrent joint attention, and predicted growth in social behavior during early childhood (Dawson et al., 2002; Munson, Faja, Meltzoff, Abbott, & Dawson, 2008).

Despite the potential importance of this work, researchers have only begun to examine the behavioral and neurobiological correlates of altered reward processing among those with ASDs. However, the neural substrates of reward processing in general are well characterized. A large

literature implicates the mesolimbic dopamine (DA) system, the mesocortical DA system, and functional connections between systems in the processing of reward, and in individual differences in the propensity to seek rewards (Gatzke-Kopp & Beauchaine, 2007; Gatzle-Kopp et al., 2009). Together, these networks comprise what is often referred to as the mesocorticolimbic DA system. Although few studies have examined mesocorticolimbic functioning among those with ASDs, those that have suggest impairment in both structure and function. Relative to controls, medication-naïve individuals with ASDs display increased caudate volumes, a mesolimbic structure (Langen, Durston, Staal, Palmen, & van Engeland, 2007). In a functional imaging study, Dichter et al. (2010) found reduced nucleus accumbens activation to monetary incentives among those with ASDs, but more typical activation patterns to visual images hypothesized to be intrinsically rewarding (e.g., vehicles, electronic devices).

Aberrant mesocortical reactivity to reward among those ASDs has also been reported. During a monetary incentive task, Schmitz et al. (2008) found increased activation in the left anterior cingulate gyrus and left middle frontal gyrus, mesocortical regions implicated in motivation and arousal (Bush, Luu, & Posner, 2000). In addition, activation of the left anterior cingulate cortex (ACC) during reward was correlated significantly with scores on the reciprocal social interaction domain of the Autism Diagnostic Interview-Revised (ADI-R; Lord, Rutter, & Le Couteur, 1994).

Emerging evidence suggests there may be group differences in response to social reward as well. When exposed to smiling faces following correct responses during an implicit learning task, children with ASDs show reduced activation in the ventral striatum relative to controls, as assessed by fMRI (Scott-Van Zeeland, Dapretto, Ghahremani, Poldrack, & Bookheimer, 2010). Thus, although limited, there is growing evidence of atypical reward processing among those with ASDs. Moreover, there is evidence of links between disrupted reward processing and impaired social functioning. However, the degree to which general versus social reward

processing is altered remains unclear, and no studies to date have explored the potential moderating role of familiarity of social reward.

Assessment of Reward Mechanisms

These few studies notwithstanding, the relative lack of research exploring reward processing among those with ASDs may be attributed in part to methodological challenges, particularly when recruiting child samples. Ethical and practical considerations restrict the use of imaging techniques such as positron emission tomography (PET), which can localize brain activation in specific neural systems, yet requires ingestion of radioactive isotopes (Downie & Marshall, 2007; Munson, Eshel, & Ernst, 2006). Non-invasive imaging techniques such as fMRI have eased this challenge, but are subject to other difficulties, such as movement artifacts, claustrophobia, behavioral compliance, and limited temporal resolution (Byars et al., 2002). In fact, successful completion rates for fMRI protocols may be even lower among children with ASDs than for children with epilepsy or unmedicated ADHD (Yerys et al., 2009). In contrast, peripheral (autonomic) measures provide information regarding reward processing with fewer methodological limitations. Among autonomic markers, cardiac pre-ejection period (PEP) is of particular interest as a biomarker of reward responding.

Cardiac PEP is a systolic time interval that begins at the onset of left ventricular depolarization and ends when blood is ejected into the aorta. Changes in PEP are effected through the sympathetic nervous system via β-adrenergic mechanisms (Sherwood, Allen, Obrist, & Langer, 1986). PEP reactivity to incentives likely serves as a peripheral marker of mesolimbic DA reactivity (Beauchaine, in press; Beauchaine, Gatzke-Kopp, & Mead, 2007; Brenner, Beauchaine, & Sylvers, 2005). This argument is based on both theoretical and empirical considerations. First, injections of DA agonists directly into the ventral tegmental area, a

mesolimbic neural region that is critical to reward processing, produce sympathetically-mediated increases in blood pressure and heart rate (van den Buuse, 1998), linking central DA concentrations directly to cardiac output via SNS mechanisms. In addition, pharmacologic blockade studies indicate that changes in cardiac PEP are determined solely by SNS effects (Sherwood et al., 1986). Increases in cardiac output are also necessary for engagement in appetitive behaviors, which are usually mediated by CNS reward processes. Finally, PEP shortening is specific to conditions of reward, and is not observed during extinction or negative mood induction among typically developing individuals (Brenner et al., 2005).

To date, PEP has been used as a marker of CNS reward sensitivity among children, adolescents, and adults, including many with behavioral disorders and challenges (e.g., Beauchaine, Hong, & Marsh, 2008; Beauchaine, Katkin, Strassberg, & Snarr, 2001; Crowell et al., 2006; Richter & Gendolla, 2009). Because it provides for non-invasive assessment of reward responding without the challenges, expense, and safety concerns of imaging techniques such as PET and fMRI, assessing PEP during reward tasks—both social and nonsocial in nature—may inform our understanding of reward processing among individuals with ASDs[1].

Though not specific to reward, the effects of parasympathetic nervous system (PNS) reactivity on cardiac functioning are also relevant—particularly with regard to social reward. Porges (2001, 2003, 2004) describes a parasympathetically-mediated *social engagement system* (SES), in which social behavior emerges from three hierarchically-organized response systems within the autonomic nervous system. The third and most phylogenetically advanced response system is mediated by the myelinated vagus (Porges, 2001), which originates in the nucleus ambiguous and innervates a number of organs above the diaphragm, including the larynx, pharynx, bronchi, esophagus, and heart. Control of these organs, together with enhanced neural control of facial muscles, allows the myelinated vagus substantial influence over a number of

socially-oriented behaviors—including facial gestures and vocalizations—and increases the flexibility and sophistication of an individual's repertoire of available social and communicative behaviors. For example, muscles of the middle ear enhance the ability to detect a human voice and extract it from background noise, facial muscles increase the range and specificity of expressions of emotion, the larynx and pharynx control prosody and intonation of speech, and muscles within the neck and head support head turning for social gesture.

In addition, in threatening situations rapid temporary withdrawal of inhibitory parasympathetic efference provides for almost immediate metabolic bursts of energy without the delay required for a complete activation of the sympathetic-adrenal system (typically 20 to 30 s; Berntson et al., 1997). Thus, evolution of the myelinated vagus has enhanced mammalian behavioral repertoires and facilitated quicker, more sensitive, and more finely tuned responses to environmental challenges. Extensive connections link these structures and processes to many other systems in the body, including the HPA axis, neuropeptides systems including oxytocin and vasopressin, and the immune system (Porges, 2001).

According to Porges's framework, PNS contributions to heart rate variability (HRV) mark an individual's ability to respond to social challenges (Porges, 1995, 2001, 2004). Heart rate variability describes cyclic increases and decreases in heart rate, usually indexed by beat-to-beat intervals. The parasympathetic contribution to HRV—often referred to as vagal tone when measured at rest—can be characterized by respiratory sinus arrhythmia (RSA; see Berntson et al., 1997), a quantification of these cyclic increases and decreases of heart rate over the respiratory cycle. RSA reflects cholinergic activity of the parasympathetic nervous system. Among typically developing samples, tonic PNS-mediated cardiac activity during a variety of tasks is associated with a number of social behaviors, including social competence, engagement with peers, adaptive and support-seeking coping strategies, sympathy for characters in a short

film, and the number of spontaneous eye gazes produced (Eisenberg et al., 1995; Fabes et al., 1993; Henderson et al., 2004). Similarly, vagal reactivity is correlated with soothing talk to a distressed infant (Fabes et al., 1994). Among children with ASD, baseline RSA is linked to parent report of social skills, as well as measures of emotion recognition (Bal et al., 2010; Van Hecke et al., 2009). Across studies, higher RSA, reflecting more PNS-mediated heart rate variability, is associated with higher social functioning. However, RSA has yet to be explored in the context of social reward, despite its theoretical and empirical links with social functioning.

Goals

With this background in mind, I had three goals in conducting the current study. The first was to examine autonomic correlates of general and social reward processing among children with ASDs, with attention to potential moderating effects of familiar social stimuli. I predicted that children with ASDs would show deficits in physiological responding to reward relative to typically developing children, including less PEP reactivity to monetary incentives and less RSA reactivity to social reward. I also predicted a Group × Reward Type interaction, with more typical patterns of autonomic responding to familiar social rewards among those with ASDs.

My second goal was to examine relations between physiological responses to social reward and behavior during social interactions. Because sensitivity to the reward properties of social stimuli is thought to underlie a wide range of social behaviors across development, I predicted that greater autonomic responses to social rewards would be associated with higher scores on measures of social functioning, and with more contingent and reciprocal social behavior during a naturalistic social interaction task.

My third goal was to examine time-linked patterns between parental behaviors and children's

autonomic responses. If children with ASDs differ from typically developing children in the degree to which they experience others' social behavior as rewarding, they may display different levels of correspondence between parental behaviors and cardiac PEP. Whereas typically developing children may show a high degree of correspondence between parental behaviors and PEP, children with ASDs may show a lower degree of correspondence.

METHOD

Participants

Eighteen children participated in the ASDs group, and 18 participated as community controls (total $N = 36$). The sample was limited to male participants for two related reasons. First, ASDs are diagnosed more frequently in males, by a ratio of about 4:1 (Fombonne, 2009). Second, recent findings suggest that the molecular genetic bases of ASDs may differ among males vs. females (Schellenberg et al., 2006). Thus, the inclusion of female participants might increase etiological heterogeneity in a sample that was not large enough to explore sex effects with adequate statistical power.

Participants were recruited from a registry of families at the University of Washington Autism Center, and from the general community through fliers and advertisements. Interested parents completed a preliminary phone screening interview that included (1) demographic information; (2) a brief developmental history to assess previous developmental diagnoses, head injuries, seizure history, family history of ASD, and current medication status; (3) the Social Communication Questionnaire (SCQ; Rutter, Bailey, & Lord, 2003); and (4) the Child Behavioral Checklist (CBCL; Achenbach, 1991). The final racial/ethnic composition of the sample was 66.7% Caucasian, 5.6% African American, 2.8% Asian, 2.8% Latino, 2.8% Pacific Islander, and 19.4% who identified as more than one race. Because of potential effects on autonomic reactivity to reward, all participants were required to be free of stimulant medications for at least 36 hours prior to participation. However, one control and five ASDs participants were using at least one non-stimulant psychiatric medications at the time of data collection. Specifically, the control participant used an antihypertensive. Within the ASDs group, three participants took selective serotonin reuptake inhibitors (SSRIs), two took atypical antipsychotics, one used an anticonvulsant, and one used an antihypertensive.

Of the 18 participants with ASDs, 14 were recruited from a previous study at the University of Washington Autism Center. The additional 4 participants were recruited from online and community advertisements. Diagnostic eligibility for the ASDs group was established either by reviewing previous diagnostic records from the Autism Center, or by administering the ADI-R (Lord et al., 1994) and Autism Diagnostic Observation Schedule (ADOS; Lord, Rutter, DiLavore, & Risi, 2003) for participants who had not completed these measures previously. Participants were included if they (1) met or exceeded diagnostic thresholds on the ADI-R and the ADOS, and (2) had a diagnosis of autism, Asperger's Syndrome, or pervasive developmental disorder, not otherwise specified (PDD-NOS) given by an experienced clinician on the basis of the ADI-R, ADOS, and clinical judgment. In addition, participants were required to have functional communicative speech in order to ensure that they would be able to participate in the study tasks. The mean age of ASDs children was 119.9 months (*SD* = 13.2).

Eligibility for the control group was established through the preliminary phone screening interview. Potential participants were screened for subthreshold features of ASDs using the lifetime version of the SCQ, and for behavior problems using the CBCL. Participants were excluded if their score on the SCQ exceeded 9, or if their symptom *T*-score on the thought problems subscale of CBCL exceeded 65. Participants were also excluded if they had ever received a diagnosis related to their development, had a history of significant head injury or seizure activity, or had a first-degree relative with a diagnosis of an ASD. The mean age of children in this group was 120.2 months (*SD* = 11.1). Psychopathology and IQ scores are reported by group in Table 1.

Measures

Autism Diagnostic Observation Schedule (ADOS). The ADOS is a semi-structured assessment of communication, social interaction, play skills, and restricted and repetitive

behavior. The session contains a series of prompts, activities, and scenarios designed to elicit social and communicative behaviors, which are then coded and compiled according to diagnostic algorithms (Gotham, Risi, Pickles, & Lord, 2007). Individuals participate in 1 of 4 modules on the basis of language ability. Module 3, the module appropriate for children and adolescents with fluent language, has high sensitivity (80%) and specificity (94%) in discriminating those with ASDs from controls. Internal consistency for the subdomains of behavior assessed ranges from .51 to .92 (Gotham et al., 2007) and test-retest reliability for the subdomains ranges from .82 to .93 (Lord et al., 2003).

Autism Diagnostic Interview—Revised (ADI-R). The ADI-R is a semi-structured interview conducted with parents or caregivers of individuals suspected of having an ASD. It assesses skills in the three domains of functioning specified by the DSM-IV-TR (APA, 2000), including language/communication, social interaction, and restricted or repetitive behavior and interests. The ADI-R is most appropriate for individuals three years or older, with a mental age of at least 2 years. Test-retest reliabilities are uniformly high (range = .93 to .97) across the symptom and domain scores of the ADI-R (Rutter et al., 2003), which also has high sensitivity (86%) and specificity (93%) in discriminating ASDs from other psychopathology (Lord et al., 1997). The combination of the ADI-R and the ADOS is considered the gold standard for diagnosis of ASDs among children and adults (Ozonoff, Goodlin-Jones, & Solomon, 2005).

Child Behavior Checklist (CBCL). 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 scores on eight scales, including aggressive behavior, anxiety/depression, attention problems, rule-breaking behavior, social problems, somatic complaints, thought problems, and withdrawal/depression. The CBCL has been normed among children ages 18 months and older (Achenbach & Rescorla, 2001), and has high internal

consistencies (range = .66 to .92) and test-retest reliabilities (range = .63 to .97).

Social Communication Questionnaire (SCQ) – 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 four years of age. The lifetime form of the SCQ assesses the child's developmental history on 40 yes-or-no items, and yields a total score that can be interpreted either in relation to a clinical cut-off of 15, or as a continuous measure of symptom severity. The lifetime form has high agreement with the ADI-R, and internal consistencies with children in this age group range from .89 to .93 (Rutter et al., 2003). However, test-retest reliabilities have not been published. Typically-developing school-age children have a mean score of 3.89 (*SD*=2.77) on the SCQ (Mulligan, Richardson, Anney, & Gill, 2009). An exclusion cutoff score of 9 was chosen for the control group of the current sample because it allowed for normal variation in scores among typically-developing children, while also falling well under the clinical cutoff of 15.

Social Skills Improvement System (*SSIS*; Gresham & Elliott, 2008). The SSIS is a multi-rater assessment of social behaviors in childhood. The parent-report version contains 55 statements relevant to social skills and problem behaviors. For each item, 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 a standard social skills score, and is appropriate for use with children between 3 and 18 years. For the elementary age group, internal consistencies for the subscales range from .74 to .95 and test-retest reliabilities range from .70 to .92.

Vineland Adaptive Behavior Scales, Second Edition (*VABS-II*; Sparrow, Cicchetti, & Balla, 2005). The VABS-II is a measure of daily skills necessary for personal and social independence.

The survey interview form is a semi-structured parent interview that yields both composite adaptive behavior scores and standard scores across three domains (communication, daily living skills, socialization). Internal consistencies for the domain scores of the survey interview form are high (range = .88 to .94), as are test-retest reliabilities (range = .75 to .91).

Wechsler Abbreviated Scale of Intelligence (*WASI*; Wechsler, 1999). The WASI was included in order to ascertain the cognitive abilities of participants in both groups. The WASI is a brief measure of cognitive ability that comprises four subtests (vocabulary, block design, similarities, matrices). It yields standard scores for verbal and performance ability as well as an IQ composite. The WASI has been normed with participants above six years of age, and correlates highly with comprehensive cognitive assessment instruments such as the Wechsler Intelligence Scale for Children—third edition (WISC-III; Wechsler, 1991). Test-retest reliabilities of the WASI are also high, ranging from .83 to .92 for the composite scores.

Cardiac pre-ejection period (PEP). Cardiac PEP was derived by collecting both impedance cardiographic (ICG) and electrocardiographic (EKG) signals. The ICG was obtained using a HIC2000 Impedance Cardiograph (Bio-Impedance Technologies, Chapel Hill, NC), with a sampling rate of 1kHz. An external ECG signal was obtained externally using a Grass Model 15LT Physiodata Amplifier System (West Warwick, RI), which also sampled at 1 kHz. External ECGs provided a better signal-to-noise ratio than can be obtained though impedance electrodes. The ICG and ECG data were collected and digitized using COP-WIN software, version 6.10 (Bio-Impedance Technologies, Chapel Hill, NC). A spot electrode configuration allowed some physical movement with minimal signal disruption, as recommended by Qu, Zhang, Webster, and Tompkins (1986).

Pre-ejection period was quantified by the QB interval. This requires identification of the ECG Q-wave, which signifies the onset of ventricular depolarization, and the dZ/dt B-wave,

which marks the opening of the aortic valve. The Q-wave is an inflection immediately preceding the onset of the R-wave (see Sherwood et al., 1990). The B-wave is marked by the first upstroke of the dZ/dt signal (see Lozano et al., 2007). Cardiac PEP was computed as the time interval in ms between the ICG Q-wave and the dZ/dt B-wave. Data were ensemble-averaged (Kelsey & Guethlein, 1990) in 32 s epochs. Because β-adrenergic (sympathetic) influences on cardiac activity operate through a second messenger system and are therefore not immediate, changes in PEP lag behind changes in stimulus conditions by about 20 s (Berntson et al., 1997). Accordingly, the first epoch within each 6-epoch reward period was discarded. During conditions of reward, typically-developing children, adolescents, and adults exhibit shortened PEPs (Beauchaine et al., 2007; Brenner at al., 2005). In contrast, those with disrupted reward processing exhibit either attenuated or no changes in PEP during incentive conditions (e.g., Beauchaine et al., 2001, 2008; Brenner & Beauchaine, in press; Crowell et al., 2006).

Respiratory sinus arrhythmia (RSA). Parasympathetic nervous system-linked cardiac activity was quantified by spectral-analyzing the R-wave time series of the ECG using software developed by Richard Sloan and colleagues at Columbia University. High frequency spectral densities (>0.15 Hz) were computed in 30-s epochs. As is standard, RSA values were natural log-transformed prior to analyses. Cholinergic blockade studies have supported the validity of RSA as a marker of PNS-linked cardiac activity (see Berntson et al., 1997).

Procedure

Parents of children who met eligibility criteria for either group were invited to the lab for a session of approximately 2 hours. During their visit, children completed the WASI, the task designed to assess psychophysiological responses to general, unfamiliar social, and social rewards, and a naturalistic social interaction task. Parents of participating children completed the

SSIS and VABS-2, helped to create a set of social reward videos for their child, and participated in the social interaction task.

Reward task. Following a baseline period of 320 s (10 epochs of 32 s each) during which resting psychophysiological signals were collected, participants played a simple computer game. Because the purpose of the computer game was to elicit success and provide a continuous experience of reward, the game was designed to be simple enough to be mastered easily by participants of this age range and ability level. The game required participants to view a series of numbers on a screen and press the corresponding numbers on a 10-key pad in response (see e.g., Beauchaine et al., 2001).

Participants played the computer game under each of three conditions of reward: *general*, in which they received money for correct responses; *unfamiliar social*, in which they received praise from a research assistant for correct responses; and *familiar social*, in which they received praise from their parent for correct responses. Each reward condition occurred in a distinct block of 192 s (6 epochs of 32 s). In order to minimize carry-over effects from one type of reward condition to the next, the order of reward types was counterbalanced across participants. The task ended with a final reward block that was not included in the physiological data and was intended only to provide a positive end to the task.

In the general 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 s with an accompanying tone. Because each reward block lasted for 32 trials, the maximum amount that could be earned was $1.60. In the unfamiliar social reward condition, participants were shown a 3-s video following each correct response. The video depicted an unfamiliar research assistant providing a praise statement (e.g., “Great job!”) with appropriate vocal affect and facial expression. In order 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-s video in which their parent provided a praise statement following each correct response. In order to ensure that unfamiliar and familiar social reward videos were matched closely on content, length, and affect, parents were asked to view the three unfamiliar social reward videos and then reenact the research assistant’s statements, affect, and expression as closely as possible. They received coaching and feedback from a different research assistant, who then videotaped 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.

Social interaction task. Each participant completed a social interaction task with his parent to assess social behavior with a familiar partner, and a parallel task with a research assistant to assess social behavior with an unfamiliar partner. To avoid confounding practice effects with interaction partner, half of the participants interacted first with their parent, and half interacted first with the research assistant. Following a brief warm-up period with each adult, participants were asked to work cooperatively on a challenging block-building activity for 320 s (10 epochs of 32 s). The activity consisted of a set of geometric patterns that were to be constructed out of blocks and assembled in layers of increasing difficulty. The task was designed to provide an opportunity for goal-directed interaction, collaboration, and negotiation. Throughout the interaction task, the child and adult were seated at a table to minimize movement and facilitate cooperation. Psychophysiological data were collected during this task in order to assess reward responding during naturalistic social interactions.

Participants and their interaction partners were videorecorded during the social interaction

task, and the resulting recordings were coded for social behavior along a number of dimensions using an interval coding strategy. For each 32 s epoch of interaction, coders naïve to the diagnostic status of the child rated both child and adult behavior across four dimensions of behavior adapted from those described in the Social Interaction Rating Scale (SIRS; Ruble, McDuffie, King, & Lorenz, 2008). This scale was created originally for use with young children with ASDs and their caregivers, and captures aspects of adult and child behavior that are likely highly relevant to intervention approaches and outcomes. Because this scale was developed as a global measure of behavior over the course lengthy interactions, it was necessary to modify the behaviors included within each dimension in order to assign a rating for each 32-s epoch.

The final dimensions on which each participant and adult were rated were:

1) *Initiation*, defined as efforts to begin interactions with the other.
2) *Contingency*, defined as frequency and valence of reactions to the other's initiations and tendency to follow the other's lead in activity or conversation.
3) *Maintenance and structuring of interaction*, defined as efforts to build on the other's initiations, assist the other's actions, and provide appropriate structure and direction.
4) *Level of affect*, defined as interest, attention, and affect displayed toward the other.

Each of the four dimensions were rated on a 5-point scale, with the lowest values of each scale corresponding to an absence of a given behavior and the highest values corresponding to frequent performance of a given behavior. Adult (parent and research assistant) and child behavior during the social interaction task was coded independently by two coders naïve to participants' diagnostic status. One fourth of the sessions were coded by both coders to allow analyses of interrater reliability. Intraclass correlation coefficients (ICCs) were computed for each of the four behavioral dimensions of interest (initiation, contingency, maintenance, and affect) and ranged from .38 to .71. In order to produce a more reliable measure, the four

dimensions were collapsed into a single behavioral dimension reflecting social competence, yielding an ICC of .75 for adult behavior and .59 for child behavior. From this, a mean social competence score was computed for each child with each interaction partner. With unfamiliar partners, the ICC for this score was .75; with familiar partners, the ICC was .65.

RESULTS

Effects of Diagnostic Group and Reward Type on Behavioral and Physiological Responses

Behavioral outcomes during reward. Because SNS reactivity has been linked to effort and level of engagement among typically developing samples (e.g., Iani, Gopher, & Lavie, 2004; Kelsey & Guethlein, 1990; Peters et al., 1998), the reaction time for each reward condition was recorded by the computer program for possible inclusion as a covariate in analyses of PEP data. See Table 2 for task performance by group and reward condition. In order to test for effects of diagnostic group and reward type on reaction times, a repeated measures analysis of variance (ANOVA) was constructed with reward type as a within-subjects factor and diagnostic group as a between-subjects factor. Because the assumption of sphericity was not violated, Mauchly's W=.96, $\chi^2(2, N=36)=1.51$, $p=.47$, uncorrected *F*-values are reported. The effect of reward type on reaction times was significant, $F(2, 68)=8.51$, $p=.001$, $d=1.0$, see Figure 1. Within reward type, a Helmert contrast indicated that reaction times in the monetary reward condition were significantly faster than in the combined social reward conditions, $F(1, 34)=14.04$, $p=.001$, $d=1.28$. In contrast, reaction times during the unfamiliar and familiar social reward conditions did not differ, $F(1, 34)=.004$, $p=.95$, $d=.00$. There was not a significant effect of diagnostic group on reaction times, $F(1, 34)=2.19$, $p=.15$, $d=.51$, nor was there a significant Group × Reward type interaction, $F(2, 68)=.79$, $p=.46$, $d=.31$. Because reaction times did not differ between groups, reaction time was not entered as a covariate in further analyses.

Next, participants' accuracy during the task was examined by conducting a repeated measures ANOVA with reward type as a within-subject factor and diagnostic group as a between-subjects factor. Once again, the sphericity assumption was not violated, Mauchly's W=.89, $\chi^2(2, N=36)=3.9$, $p=.14$, so uncorrected *F*-values are interpreted. As shown in Figure 2, the ASDs group had a lower accuracy rate during the task than the control group, $F(1, 34)=4.81$,

p =.035, d=.75. There was not a significant main effect of reward type, $F(2, 68)=1.66$, p=.20, d=.44, nor was there a significant Group × Reward type interaction, $F(2, 68)=.08$, p=.92, d=.09.

Physiological outcomes during reward. For both PEP and RSA, baseline and reactivity scores were extracted. Of the 10 epochs of cardiac data collected during the resting baseline, the final 4 (2 min, 8 s total) were averaged to produce mean baseline PEP and RSA scores for each participant. Because this technique allowed participants the first 6 epochs (3 min, 12 s total) to adjust to the study room and equipment before collecting baseline data, it maximizes the likelihood of capturing a true baseline reading. For both PEP and RSA, reactivity scores were then computed by subtracting the respective baseline values from the score for each epoch during reward conditions. Doing so yielded a series of change scores for PEP (ΔPEP) and RSA (ΔRSA) that reflected reactivity relative to baseline during each reward condition. Negative ΔPEP scores indicated shortened PEP during reward (greater SNS reactivity), whereas positive ΔPEP scores indicated lengthened PEP. Similarly, negative ΔRSA scores indicated lower RSA during reward (more vagal withdrawal), whereas positive ΔRSA scores indicated greater RSA.

A series of multilevel models (MLMs) was constructed using Hierarchical Linear Modeling (HLM)—version 6.08 to model the effects of diagnostic group and reward type on physiological reactivity. For each of the physiological variables (ΔPEP and ΔRSA), analyses proceeded in two steps. First, separate MLMs were created for each reward type (general, unfamiliar social, and familiar social) with repeated epochs entered at Level 1 and diagnostic group entered as a dummy-coded fixed effect at Level 2. Age in months was entered as a Level 2 covariate to control for well-documented developmental increases in both PEP and RSA. In step two, individual intercepts and slopes in physiological functioning from each of the three MLMs (one for each reward type) were extracted and compared using repeated measures ANOVAs to test for the effects of reward type. For the ANOVAs, assumptions of sphericity and compound symmetry

were tested by inspecting Mauchly's *W*. For analyses in which violations were found, both Greenhouse-Geisser corrected *F*-statistics and their associated epsilon values (ε) are reported. See Table 3 for means of physiological variables by group and reward condition.

Levels of baseline PEP did not differ between the ASDs and control groups, $t(34)=-.52$, $p=.61$, $d=.18$. As shown in Figure 3, analyses revealed no effects of diagnostic group on ΔPEP intercepts, all ts$(33) \leq 1.48$, all ps $\geq .15$ or slopes, all ts$(33) \leq .63$, all ps $\geq .53$, within any of the three reward conditions. The groups did not differ significantly in mean ΔPEP within each reward condition, nor did they differ in the rate at which ΔPEP changed within each block. The effect of reward type on ΔPEP was also nonsignificant for intercepts, $F(2, 70)=1.52$, $p=.23$, $\varepsilon = .79$, $d = .41$, and slopes, $F(2, 70)=.06$, $p=.91$, $\varepsilon = .79$, $d = .09$. Thus, there was no evidence of any effects of group or reward type on ΔPEP scores.

Next, parasympathetic functioning as indexed by RSA was analyzed for effects of diagnostic group and reward type. At baseline, the control group displayed higher RSA than the group with ASD, $t(34)=2.18$, $p=.036$, $d=.75$. However, as shown in Figure 4, analyses revealed no effects of diagnostic group on ΔRSA intercepts, all ts$(33) \leq .7$, all ps $\geq .45$ or slopes, all ts$(33) \leq .80$, all ps $\geq .43$, within any of the three reward conditions. The groups did not differ in mean ΔRSA within any reward condition, nor did they differ in the rate at which ΔRSA changed within each block. The effect of reward type on ΔRSA was also nonsignificant for intercepts, $F(2, 70)=.26$, $p=.77$, $\varepsilon = .92$, $d = .17$, and slopes, $F(2, 70)=.07$, $p=.93$, $\varepsilon = .96$, $d = .09$. These analyses indicate that although baseline RSA values differed between groups, there were no effects of group or reward type on vagal reactivity to reward.

To further explore group differences in baseline RSA, regression analyses were conducted to test for effects of diagnostic group over and above the effects of internalizing symptoms. Reductions in RSA have been documented in a number of clinical groups other than those with

ASDs, including those with anxiety, panic, depression, ADHD, and conduct problems, and vagal activity and reactivity are often been interpreted as biomarkers of emotion regulation (Beauchaine, 2001; Crowell et al., 2005; Lyonfields, Borkovec, & Thayer, 1995). As shown in Table 1, parents reported significantly higher levels of internalizing symptoms for the ASD group than for controls, $F(1,34)=8.21$, $p=.01$, $d=.98$. To test whether the group difference in baseline RSA was due to differential internalizing symptoms, diagnostic group and CBCL Internalizing T-scores were entered simultaneously into a regression model. The resulting model accounted for 25.5% of the variance in baseline RSA scores, $R^2=.255$, $F(2,33)=5.65$, $p=.008$. Within the model, the effect of internalizing scores was significant, $\beta=-.41$, $p=.02$, but the effect of diagnostic group was not significant over and above internalizing symptoms, $\beta=-.17$, $p=.31$. Thus, diagnostic status did not make a unique contribution to baseline RSA after accounting for internalizing symptoms.

Correlations Between Physiological Variables and Social Functioning

Parent-reported social functioning. The second hypothesis was that psychophysiological functioning during the reward task would be correlated with social functioning, as indexed by parent-report and observational measures. Recall that parents provided ratings of social functioning on three separate instruments, the Social Skills standard score of the SSIS, the Socialization domain standardized score of the VABS-2, and the Social Problems subscale *T*-score of the CBCL. Baseline scores and intercept values from the MLMs described above were entered into the correlational analysis as indices of psychophysiological functioning. As shown in Table 4, there were no significant correlations between social functioning and either baseline PEP or intercepts for ΔPEP, all rs<.24, all ps>.18. However, baseline RSA was correlated with social functioning across all three parent-report measures. In particular, higher RSA was associated with higher social functioning as measured by the SSIS and VABS-2, and with fewer

social problems as measured by the CBCL. Intercept values for ΔRSA were not correlated significantly with social functioning.

Observations of social functioning. As shown in Table 5, mean social competence scores for children and adults did not differ by group, except during the unfamiliar partner interaction. In that case, research assistant behavior was rated as more socially competent when interacting with children in the ASDs group than in the control group, $F(1,34)=8.47$, $p=.006$, $d=1.0$. Table 6 provides correlations between children's mean social competence scores and their psychophysiological functioning during reward. As with the parent-report measures of social functioning, there were no significant correlations between social behavior and baseline PEP, or intercepts or slopes for ΔPEP, all rs<.23, all ps>.14. Baseline RSA was not correlated with observed social competence. However, the RSA intercept during familiar social reward was correlated negatively for the unfamiliar partner, $r(34)=-.36$, $p=.03$. Greater ΔRSA during familiar social reward was associated with lower social functioning with an unfamiliar adult.

Correspondence between Adult Behavior and Child Physiological Functioning

To assess for time-varying correspondences between children's physiological functioning and the concurrent social behavior displayed by their interaction partner, a series of MLMs were constructed. Separate models were created for each interaction partner (parent or research assistant) and each physiological variable (ΔPEP and ΔRSA). In each model, repeated epochs were entered at Level 1. The adult social competence score was also entered at Level 1 as a time-varying covariate to test the correspondence between adult behavior and child autonomic reactivity. Diagnostic group was entered at Level 2 as a dummy-coded fixed effect. Age in months was again included at Level 2 to control for developmental effects on the physiological variables.

Analyses of correspondence between adult social behavior and child physiology revealed non-significant effects for both ΔPEP, $t(33)=1.67$, $p=.104$, and ΔRSA, $t(33)=1.25$, $p=.22$, during the interaction task with the unfamiliar partner. During the interaction with the familiar partner, the effects of parent behavior were non-significant for both ΔPEP, $t(33)=-.86$, $p=.40$, and ΔRSA, $t(33)=.12$, $p=.92$, as well. There were no effects of diagnostic group on the association between partner's behavior and children's ΔPEP or ΔRSA, all $ts(33) \leq 1.23$, all $ps \geq .22$, for either the unfamiliar or familiar adult. Overall, child autonomic functioning was largely independent of adult social behavior. Furthermore, there was no evidence that participants with and without ASDs differed in the correspondence between their physiological functioning and the behavior their interaction partners displayed.

DISCUSSION

My goal in conducting this study was to characterize autonomic responses to non-social, unfamiliar social, and familiar social reward among children with ASDs, including links between physiological functioning and social behavior. The inclusion of both sympathetic and parasympathetic indices of cardiac reactivity follows from the integrative nature of this goal. Cardiac PEP is an established index of reward sensitivity among children and adults, and provides a peripheral marker of well-characterized mesolimbic dopaminergic activity during reward (Beauchaine et al., 2001; Brenner et al., 2005). In contrast, although RSA has little association with general reward processes, it indexes regulatory capabilities that are highly relevant to social functioning and affiliation (Porges, 2001, 2004). It may therefore inform our understanding of more specific conditions of social reward. Thus, PEP and RSA were included for both theoretical and empirical reasons, bearing in mind the importance of stimulus conditions (reward, social interaction) when including particular psychophysiological variables in a study, and when interpreting their outcomes (see Beauchaine, in press).

This study yielded both expected and unexpected findings with regard to the effects of reward among children with and without ASDs. Behaviorally, performance on the reward task was influenced by both diagnostic status and reward type. Participants were significantly faster to respond under conditions of general reward in which they earned money for correct responses. From a functional perspective, this suggests that they experienced money as more reinforcing than videos of adults giving praise. In contrast, participants responded with approximately equal speed in the unfamiliar and familiar social reward conditions, suggesting that they experienced those as similarly reinforcing. This finding is consistent with recent findings among children with typical development, for whom monetary incentives increased performance on a go/no-go task more than social incentives (photographs of positive facial expressions) did (Kohls, Peltzer,

Herpertz-Dahlmann, & Konrad, 2009). However, the absence of a Group × Reward type interaction was contrary to the hypothesis that children with typical development would experience social reward as more reinforcing than children with ASDs. There was no effect of reward type on accuracy, and the groups did not differ from one another in reaction time across the reward conditions, although the presence of medium effect sizes suggest that this may be due to the small sample size and a consequent lack of statistical power. The only group difference in behavioral performance was in accuracy, where the ASD group scored lower. This difference may reflect deficits in neurocognitive functioning (e.g., set-shifting, sustained attention, perseveration), but given that accuracy in all conditions exceeded 88%, participants with ASDs were able to perform with a high degree of success.

In contrast to behavioral performance, there were no effects of diagnostic group or reward type on baseline PEP. This is among the first studies to explore SNS regulation among individuals with ASDs, particularly as assessed via cardiac PEP. Although the sample size is small, this suggests that baseline SNS functioning may not be altered in ASDs, at least with regard to high-functioning individuals. Similarly, participants did not differ in PEP reactivity, a marker of dopaminergic sensitivity to reward among typically-developing and clinical samples (e.g., Beauchaine, in press; Beauchaine et al., 2001; Brenner et al., 2005), on the basis of ASD diagnosis. With regard to reward type, participants did not show differential degrees of SNS responding to the different reward conditions, although effect sizes suggest this is likely due in part to limited power. The absence of a significant reward type effect is surprising, because typically-developing children and adolescents reliably show an attenuation of PEP (evidenced by negative scores for ΔPEP) under conditions of monetary reward, yet this effect was absent in the current sample. One interpretation of this discrepancy is that participants did not experience any of the incentive conditions as rewarding. However, this interpretation conflicts with the

behavioral findings of faster reaction times during the monetary reward condition. An alternative interpretation might suggest that reaction times were slower in the social reward conditions due to a factor other than reward value – for example, due to difficulties shifting attention from processing the reward videos to responding to the next task trial. However, such processes were not assessed during the task, so they are highly speculative. Additionally, the magnitude of reward offered during the nonsocial condition may have contributed to this null effect, as discussed in more detail below.

With respect to PNS functioning, controls showed higher RSA than participants with ASDs at baseline. These findings fit closely with hypotheses, as higher RSA reflects greater PNS efference, superior emotion regulation, and better functioning of the social engagement system proposed by Porges (2001). Thus, children with higher RSA should display more flexible and sophisticated social behaviors. Consistent with this expectation, higher baseline RSA was associated with better social functioning and fewer social problems according to parent report. However, group differences in baseline RSA were no longer significant after accounting for internalizing symptoms, indicating that the reduced RSA observed in the ASD group may be more closely related to comorbid anxiety and depression than to ASD per se. Individuals with ASD show high rates of comorbidity with both internalizing and externalizing disorders (Bauminger, Solomon, & Rogers, 2010; Kim, Szatmari, Bryson, Streiner, & Wilson, 2000; Solomon, Miller, Taylor, Hinshaw, & Carter, 2011), and it is possible that apparent RSA reductions in ASD in the broader literature are due in part to concurrent symptoms of anxiety or depression.

Parasympathetic reactivity and social functioning were also related, as higher ΔRSA during familiar social reward was associated with lower social competence during the interaction with an unfamiliar adult. Thus, participants who demonstrated greater social competence with the

unfamiliar adult had the least vagal reactivity during social reward. In previous studies of RSA reactivity, optimum socio-emotional functioning was associated with moderate vagal withdrawal (i.e., a moderate decrease in RSA) during challenge (e.g., Crowell et al., 2005). However, mean ΔRSA values in the current sample were close to zero for both groups. Of course, positive values of ΔRSA reflect increases in vagal influence, whereas negative values of ΔRSA reflect vagal withdrawal. Consequently, participants with low ΔRSA values were the most likely within this sample to evidence the moderate vagal withdrawal linked with adaptive functioning, as indicated by the observed correlation between lower values of ΔRSA and higher social competence. In this interpretation, it is important to recall that studies documenting greater degrees of vagal withdrawal typically induced reactivity through emotion induction tasks (e.g., Crowell et al., 2005), whereas the structured reward task in the current study likely elicited very little emotion and therefore very little vagal reactivity.

Theoretically, interpretations of RSA as reflecting both emotion regulation and social engagement raise questions as to its specificity. From the polyvagal perspective, emotional experience represents an epiphenomenon of evolution of the vagal system (Porges, 1999). According to this view, as the vagal system (and thus the social engagement system) evolved, its emerging structure and function allowed the experience of emotions, particularly those associated with social affiliation. As discussed earlier, the vagus innervates muscles of the head, neck, face, and larynx. Porges cites these structures as facilitating both social interaction and expressing emotion (Porges, 1999, 2001). As a result of their overlapping physiology, emotional experience is bounded by the functional status of the vagal system, which promotes behavior conducive to social interactions. Emotions in turn place boundaries on the social behaviors available to an individual and "determine proximity, social contact, and the quality of communication" in which that individual may engage (p. 66, Porges, 1999). As a result,

measures of vagal functioning reflect both emotional experience (lability and trait affectivity) and the functioning of the social engagement system. Thus, adequate vagal influence results both in flexible, adaptive emotional experience and regulation, and flexible, adaptive social behavior.

Finally, the participant groups did not differ in the degree to which adult social behavior correlated with physiological functioning. Although it was expected that children with ASDs might show less correspondence between their physiological state and the social behavior they experienced from their partner, this was not supported. In fact, there was no evidence of significant effects of adult social behavior on children's physiological functioning. This was contrary to hypotheses, in which I expected to find associations between children's autonomic functioning and adult behavior. In light of reduced sensitivity to social cues among those with ASDs (Dawson & Bernier, 2007), I expected that control participants would display greater levels of correspondence with adult behavior, but child physiology was unrelated to adult behavior for both groups. The absence of association may reflect a restricted range of adult social behavior due to the structured nature of the social interaction task, and a less structured or more interpersonally oriented task may have elicited a greater range of social behavior from adult interaction partners.

In interpreting the findings of this study, a number of shortcomings must be considered. First, the absence of an effect of reward on ΔPEP values for participants in the control group is unexpected, and so raises questions as to whether the task activated the same reward mechanisms in this sample as in previous samples. Ideally, PEP shortening in the control sample would have provided some measure of assurance that the task induced an experience of reward for typically developing children. This did not occur, which may suggest that the reward value in the monetary condition ($.05 per correct response) was not sufficiently high or that the periods of reward were too brief, consisting of single reward blocks lasting 192 s each. Previous studies

with children of the same age have demonstrated reward-induced PEP shortening during an analogous task awarding a similar amount ($.06) per trial, but participants completed far more trials and therefore earned much more money than those in the current study (Beauchaine, Hong, & Marsh, 2008).

An additional shortcoming of the current study is that potential autonomic effects of psychiatric medication were not assessed. In a recent study, Mathewson and colleagues (2011) found reduced RSA in adults with ASDs relative to controls, but group differences diminished after dividing the ASDs group on the basis of medication status. Although medication status was likely confounded with ASD symptom severity, the authors highlight the importance of considering medication effects on cardiac functioning. As described above, participants in the current study were using a number of psychiatric medications at the time of participation, most commonly SSRIs (citalopram, fluoxetine, and sertraline) and atypical antipsychotics (aripiprazole, risperidone, and quetiapine). Early reports (e.g., Cohen, Kotler, Matar, & Kaplan, 2000; Davidson et al., 2005) suggested that antidepressant medications specifically targeting serotonin reuptake had either no effect or increased PNS-mediated cardiac activity, but recent longitudinal findings suggest long-term use of SSRIs may contribute to decreased RSA (Licht, de Geus, van Dyck, & Penninx, 2010). In contrast, risperidone, the most thoroughly studied of the antipsychotics in this sample, affects sympathetically- but not parasympathetically-mediated heart rate variability (Hempel, Tulen, van Beveren, Roder, & Hengeveld, 2008; Silke, Campbell, & King, 2002). Consequently, although medication effects cannot be ruled out definitively, the bulk of the literature suggests they likely do not account entirely for the group differences in RSA observed here. Future studies assessing RSA prior to and following administration of SSRIs and atypical antipsychotics would help to clarify this issue.

These issues notwithstanding, the findings presented here have several important

implications, both for theories of the etiology of ASDs and for intervention strategies. The social motivation hypothesis proposed by Dawson and Bernier (2007) suggests that deficits in reward processing among individuals with ASDs could underlie a range of social impairments across development, including disruptions in social orienting, speech and face perception, joint attention, communication, and imitation. The current study did not find evidence of reward deficits among participants with ASDs during the general or social reward conditions, nor was ΔPEP correlated significantly with any of measures social functioning. However, as noted above, the absence of a reward-induced PEP attenuation among the control group raises doubts about the degree to which the reward task successfully activated the reward mechanisms typically associated with PEP. Thus, it may be that individuals with ASDs have deficits in reward responding that were not captured in the current findings.

Reductions in baseline RSA are consistent with hypothesized impairments to the social engagement system suggested by Porges (2004). This study is among the first to evaluate RSA within the context of social stimuli among individuals with ASDs, and provides evidence for altered parasympathetic functioning. Given the role of the social engagement system in facilitating appropriate social behavior across a range of contexts, it is interesting to note that ΔRSA did not differ significantly according to whether participants viewed videos of their parent or an unfamiliar adult. In everyday life, individuals are expected to respond differently to familiar versus unfamiliar interaction partners; whereas some (e.g., family members, friends) should elicit positive, intimate interaction, others (e.g., strangers) should elicit behavior characterized by reticence and evaluation for threat. To the degree that the familiarity (and lack thereof) of the adults in the reward videos mirrored the familiarity of individuals encountered in daily life outside of the laboratory, one might expect differences in RSA since they should activate different behavioral responses. However, if ΔRSA, and vagal functioning in general,

reflects an individual's ability to flexibly adapt their social behavior for a given social encounter, then RSA reactivity to familiar and unfamiliar social partners should not differ, consistent with the current findings.

Implications for Intervention

Within the current sample, evidence of ASD-related deficits in autonomic functioning was limited to parasympathetic mechanisms, with lower baseline RSA corresponding to poorer social functioning. Given Porges's (2001, 2003, 2004) suggestion that higher RSA supports the development and flexible implementation of a repertoire of social behaviors, interventions targeting vagus nerve efference may be effective at promoting social behavior among this group. Early treatment approaches using vagus nerve stimulation among adults with epilepsy and comorbid symptoms of ASDs have provided intriguing suggestions of improved social functioning, but have consisted only of uncontrolled case studies and archival reports to this point (Rychlicki et al., 2006; Warwick, Griffith, Reyes, Legesse, & Evans, 2007). Vagus nerve stimulation also has a therapeutic on symptoms of depression (see Daban, Martinez-Aran, Cruz, & Vieta, 2008 for a review), so improvements in behavior among those with ASDs may reflect improvements in emotional functioning or seizure activity generally, rather than benefits specific to social behavior.

Perhaps the most promising intervention approaches are those that build social engagement through intense interaction, increasing the salience and frequency of social stimuli. Recent findings with toddlers who received long-term treatment through the Early Start Denver Model, which incorporates "interpersonal exchange and positive affect, shared engagement with real-life materials and activities, [and] adult responsivity and sensitivity to child cues" within a developmental context improves a range of outcomes related to social functioning (p. e20,

Dawson et al., 2010). No research to date has explored the effects of these interventions on parasympathetic function, but psychophysiological changes in PNS activity may mediate positive outcomes, contributing to the development of more flexible and appropriate social behavior. Indeed, behavioral genetics studies indicate both heritable and environmental effects on RSA, with approximately 50% of the variance in RSA due to each (Kupper et al., 2005; Sneider, Boomsma, van Doornen, & DeGeus, 1997). Thus, improvement in social behavior elicited by behavioral interventions may be due in part to effective shaping of parasympathetic function. The degree to which improvements in social behavior are apparent above and beyond improvements in emotion regulation is also an important question, in light of Porges's (1999) theory outlining their evolutionary and physiological overlap.

The current findings shed little light on the long tradition of reinforcement-based ASD interventions, such as applied behavior analysis (ABA) and discrete trial training (DTT). Despite their importance, a disconnect exists between the heavy reliance on reward processing in such approaches and suggestions of possible impairments in SNS-mediated reward-processing mechanisms among children with ASDs (Dawson & Bernier, 2007). Characterizing these processes among children with ASDs remains an important goal, as treatment efficacy is likely tied to decisions about the type(s) of reinforcement (tangible versus social) offered and the relationship (unfamiliar therapists versus parents) of the primary treatment providers to the child. Although these choices can be addressed in part through functional behavioral analyses, an understanding of their neurobiological substrates would further enrich our understanding. Nonetheless, given deficits in PNS-mediated social engagement, the incorporation of intensive social approaches that remediate deficits in RSA may enrich the benefits of reinforcement-oriented approaches by increasing the salience of social stimuli and potentially increasing the reward value of social interaction. Such an approach is similar to those advocated for behavior

management of externalizing disorders, which suggest establishing a strong foundation of positive social interaction prior to introducing positive/negative reinforcement strategies (e.g., Webster-Stratton, 2005). Indeed, the Early Start Denver Model, which is the first rigorous evidence of the effect of early intervention with toddlers with ASD, includes parent training on the use of operant principles to shape behavior, reflecting a synthesis of relationship- and reward-based approaches (Dawson et al., 2010).

Taking a broader view of intervention throughout development, RSA is often viewed as a physiological marker of psychological vulnerability or resilience (e.g., Shannon, Beauchaine, Brenner, Neuhaus, & Gatzke-Kopp, 2007). Among children with familial and psychosocial risk factors such as marital conflict, parental psychopathology, and hostile parenting, RSA moderates outcomes such that higher baseline RSA predicts more positive social and emotional functioning, whereas children with lower RSA are at greater risk for internalizing and externalizing symptoms (El-Sheikh, Hinnant, & Erath, 2011; Leary & Katz, 2004; Shannon et al., 2007). In the current findings, reductions in RSA relative to controls may suggest that individuals with ASDs represent a group at biological risk for negative outcomes following stressors, and emphasize the need for increased supportive, preventive, and intervention services. Whereas typically-developing children and adolescents may require minimal support to negotiate transitions and normative social challenges (e.g., school changes, peer conflict, parental divorce, loss of attachment figures), individuals with ASDs likely require additional support to prevent maladaptive social and emotional outcomes.

Directions for Future Research

Moving forward, this research leaves unanswered a number of issues worthy of further investigation. Although the concept of "social behavior" is often used as though it represented a

single construct, the term likely reflects interrelated but distinct abilities such as social awareness, social cognition, social motivation, social communication, and social anxiety. Neuroimaging research has begun to differentiate among the neural substrates of various facets of social behavior, documenting specific relations between neural processes and social abilities. For example, the superior temporal sulcus is activated preferentially by vocal sounds and biological motion (Belin, Zatorre, Lafaille, Ahad, & Pike, 2003). Similarly, a region of the fusiform gyrus is uniquely sensitive to visual presentation of human faces (Kanwisher, McDermott, & Chun, 1997). A similar approach might prove fruitful with regard to autonomic functioning among individuals with ASDs, correlating sympathetic and parasympathetic function with more specific facets of social behavior. For example, social motivation might be tied more tightly to sympathetic than parasympathetic function, given links between PEP and approach behavior in general. By the same token, social anxiety in ASDs might be more closely related to RSA, given its correlations with internalizing symptoms. Although the behavioral coding system used during the social interaction task of the present study was not sufficiently reliable to explore autonomic correlates of individual dimensions of social behavior, this study represents a first step toward mapping more precise social characteristics onto specific autonomic processes.

Additionally, continued investigation of RSA and vagal function among individuals with ASDs will be important to placing reductions in RSA within a larger etiological framework. Future studies should include measures of ASD symptom severity (e.g., Gotham, Pickles, & Lord, 2009) and level of functioning, as PNS deficits likely fall along a spectrum that parallels the heterogeneity characteristic of the behavioral features of ASDs. Further, neurobiological substrates upstream from RSA deficits will be of interest as well. The social engagement system described by Porges (2001) links vagal functioning to the HPA axis, neuropeptides oxytocin and vasopressin, and limbic structures including the amygdala. Individuals with ASDs show well-

documented alterations to both the amygdala and the oxytocin system (Green et al., 2001; Schultz, 2005), which may contribute to physiological deficits downstream. If so, RSA may mediate the relation between amygdala or oxytocin function and social behavior. Because autonomic activity is intermediate to neural and behavioral measures, investigation of these possibilities would provide another link in our etiological understanding of ASD, and another potential marker of treatment response.

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FOOTNOTES

The use of PEP attenuation as a marker of dopaminergic functioning highlights the need to consider stimulus conditions when interpreting psychophysiological markers (Beauchaine, in press). Under conditions other than reward, such as effortful concentration during mental arithmetic tasks (e.g., Bernston, Cacioppo, & Fieldstone, 1996), PEP shortening can be elicited through non-dopaminergic mechanisms associated with other motivational states. Consequently, PEP reactivity does not always reflect central DA responding and must be interpreted with respect to the stimulus conditions under which it is elicited, and the neural systems associated empirically and theoretically with those stimuli.

Table 1

Psychopathology Means and Standard Deviations by Group

	Controls	ASD	*F*-value	*p*-value	Effect size (*d*)
Age in months	120.2 (11.1)	119.9 (13.2)	.01	.94	.0
Full scale IQ	114.8 (13.5)	108.3 (21.4)	1.17	.29	.37
Verbal IQ	115.3 (9.9)	102.4 (23.0)	4.78*	.04	.75
Performance IQ	110.7 (15.7)	113.3 (19.6)	.20	.66	.16
SCQ score	3.0 (2.3)	19.7 (5.0)	172.65***	.00	4.50
SSIS Social Skills standard score	101.3 (10.8)	79.8 (10.7)	36.13***	.00	2.06
VABS-2 Socialization standard score	119.3 (6.7)	77.6 (17.7)	87.18***	.00	3.20
CBCL anxious/depressed T-Score	55.2 (6.8)	60.2 (9.7)	3.22	.08	.62
CBCL withdrawn T-Score	55.6 (6.5)	65.6 (8.4)	16.24***	.00	1.38
CBCL somatic complaints T-Score	53.3 (5.1)	57.6 (8.7)	3.18	.08	.61
CBCL social problems T-Score	53.8 (4.5)	62.6 (7.6)	17.92***	.00	1.45
CBCL thought problems T-Score	53.8 (3.8)	64.9 (8.1)	28.12***	.00	1.82
CBCL attention problems T-Score	52.7 (3.1)	64.7 (8.0)	35.05***	.00	2.03
CBCL rule breaking T-Score	54.3 (5.3)	57.0 (6.7)	1.75	.20	.45
CBCL aggressive behavior T-Score	53.3 (4.4)	59.7 (9.5)	6.74*	.01	.89

CBCL internalizing broadband T-Score	52.0	(9.3)	61.4 (10.4)	8.21**	.01	.98
CBCL externalizing broadband T-Score	48.6	(8.0)	56.9 (10.4)	7.30**	.01	.93

Notes. SCQ=Social Communication Questionnaire (Rutter, Bailey, & Lord, 2003); SSIS=Social Skills Improvement System (Gresham & Elliot, 2008); VABS-2=Vineland Adaptive Behavior Scales, 2nd Edition (Sparrow, Cicchetti, & Balla, 2005); CBCL=Child Behavior Checklist (Achenbach, 1991).

*p<.05, **p<.01, ***p<.001

Table 2

Means for Reward Task Performance by Group and Reward Condition

	Control	ASD
Reaction time (ms)		
General	1347.3 (152.2)	1482.8 (247.5)
Unfamiliar social	1475.4 (194.9)	1545.4 (225.6)
Familiar social	1462.1 (203.2)	1555.7 (285.7)
Accuracy (% correct)		
General	96.7 (3.5)	92.2 (9.9)
Unfamiliar social	93.4 (.84)	88.4 (13.6)
Familiar social	95.1 (5.2)	89.1 (14.1)

Table 3

Means of Psychophysiological Variables by Group and Reward Condition

Physiological variable	Control	ASD
PEP		
Baseline	100.35 (11.78)	102.29 (10.47)
ΔPEP		
General	1.13 (4.07)	0.47 (3.28)
Unfamiliar social	0.09 (2.93)	-0.85 (5.56)
Familiar social	1.39 (3.58)	-0.60 (4.31)
RSA		
Baseline	7.66 (0.87)	6.94 (1.09)
ΔRSA		
General	.00 (.61)	-.16 (.84)
Unfamiliar social	-.04 (.81)	-.03 (.98)
Familiar social	-.06 (.78)	.06 (.76)

Notes. PEP=cardiac pre-ejection period; RSA=respiratory sinus arrhythmia.

Table 4

Correlations Between Psychophysiological Variables and Parent-reported Social Functioning

Physiological variable		SSIS Social Skills	VABS-2 Socialization	CBCL Social Prob
PEP Baseline		-.16	-.08	-.16
ΔPEP General	Intercept	-.00	.06	-.05
	Slope	-.16	-.19	.00
ΔPEP Unf. Social	Intercept	-.03	-.09	.13
	Slope	.02	.08	-.07
ΔPEP Fam. Social	Intercept	.21	.17	-.06
	Slope	.23	.12	-.17
RSA Baseline		.41*	.45**	-.35*
ΔRSA General	Intercept	-.06	.08	-.28
	Slope	.03	.08	.13
ΔRSA Unf. Social	Intercept	-.10	.02	-.06
	Slope	-.06	-.08	.06
ΔRSA Fam. Social	Intercept	-.13	-.08	.03
	Slope	-.20	-.09	-.07

Notes. SSIS=Social Skills Improvement System; VABS-2=Vineland Adaptive Behavior Scales, 2nd Edition; CBCL=Child Behavior Checklist; PEP=cardiac pre-ejection period; RSA=respiratory sinus arrhythmia.

*p<.05, **p<.01, ***p<.001

Table 5

Mean Social Behavior Competence Scores by Group and Interaction Partner

	Control	ASD	*F*-value	*p*-value	Effect size (*d)*
Unfamiliar partner					
Child	11.0 (2.7)	11.4 (2.4)	.25	.62	.17
Adult	12.4 (1.7)	14.1 (1.8)	8.47**	.00	1.0
Familiar partner					
Child	12.4 (2.9)	11.8 (2.4)	.57	.46	.26
Adult	13.0 (2.4)	13.0 (1.9)	.00	.99	.00

Notes. *p<.05, **p<.01, ***p<.001

Table 6

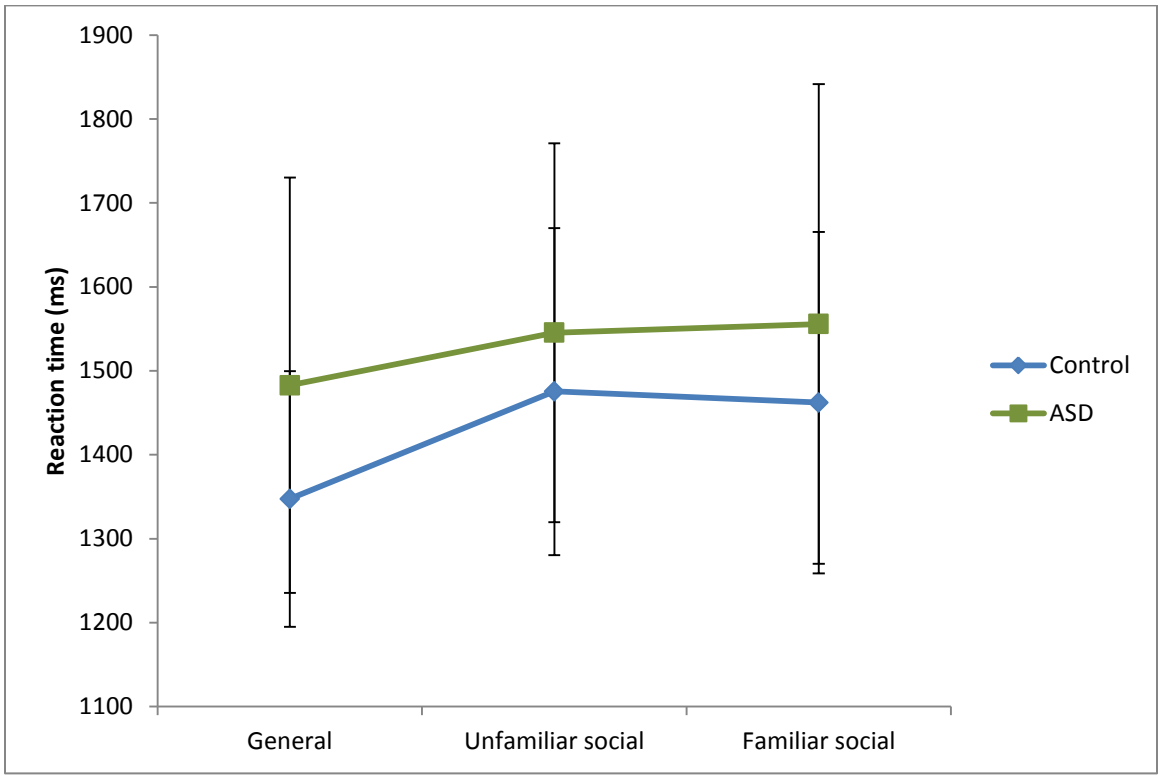
Correlations Between Psychophysiological Variables and Child's Observed Social Functioning

Physiological variable		Unfamiliar partner	Familiar partner
PEP			
Baseline		-.15	-.08
ΔPEP			
General	Intercept	-.06	-.16
	Slope	-.13	.04
Unf. Social	Intercept	-.22	-.14
	Slope	-.05	.00
Fam. Social	Intercept	-.12	-.12
	Slope	-.06	.05
RSA			
Baseline		-.05	.07
ΔRSA			
General	Intercept	-.20	-.04
	Slope	-.03	.05
Unf. Social	Intercept	-.30	-.06
	Slope	.08	-.08
Fam. Social	Intercept	-.36*	-.25
	Slope	.20	.19

Notes. PEP=cardiac pre-ejection period; RSA=respiratory sinus arrhythmia.

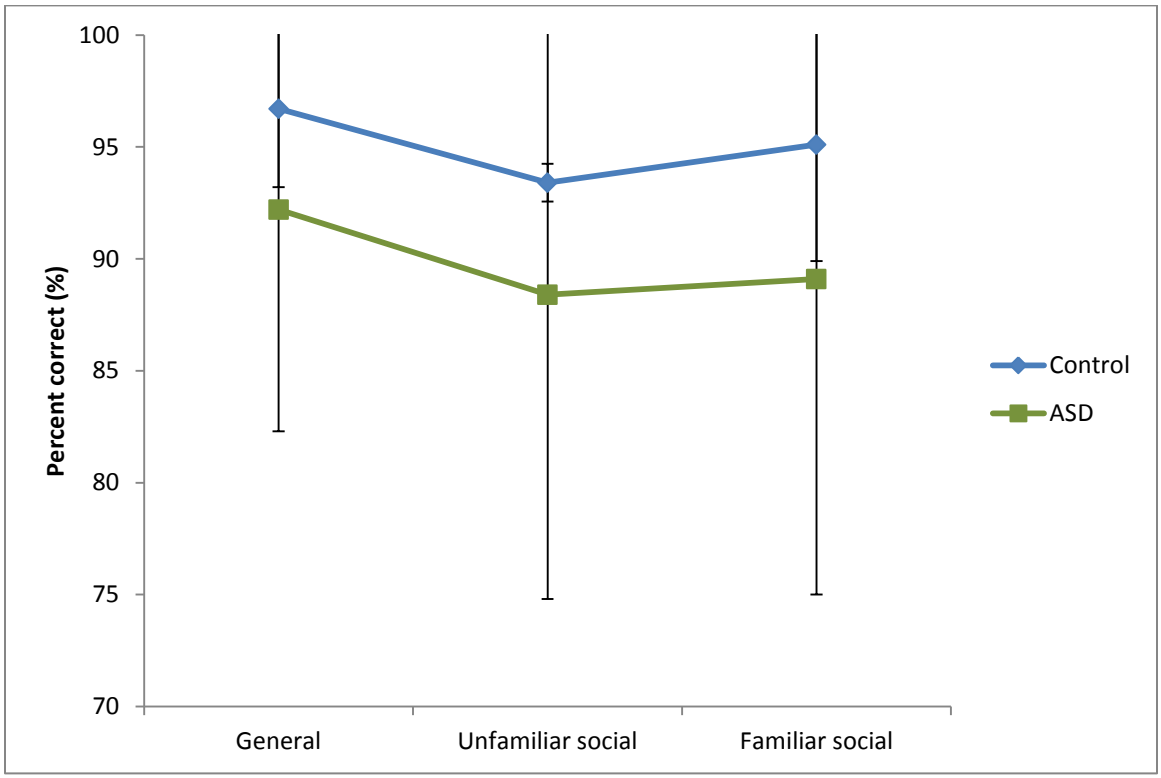
*p<.05, **p<.01, ***p<.001.

Figure 1.



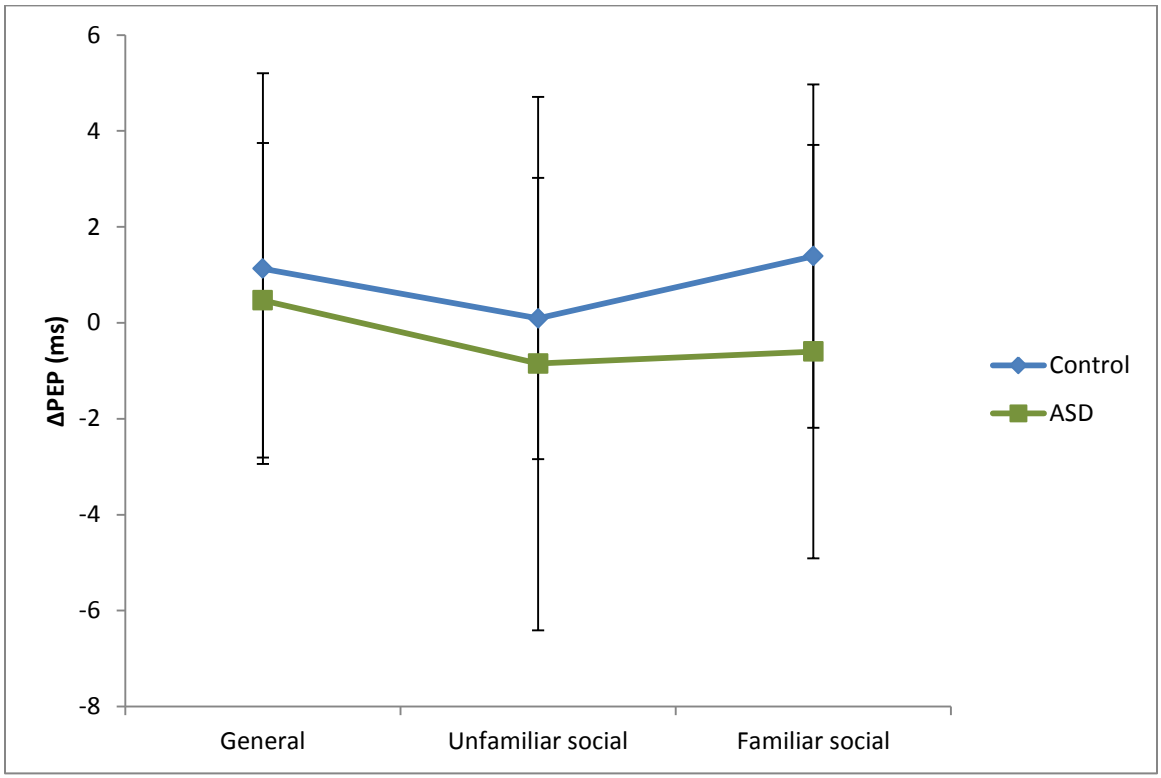
Reaction times in milliseconds by diagnostic group and reward condition. Error bars represent standard deviation from the mean.

Figure 2.



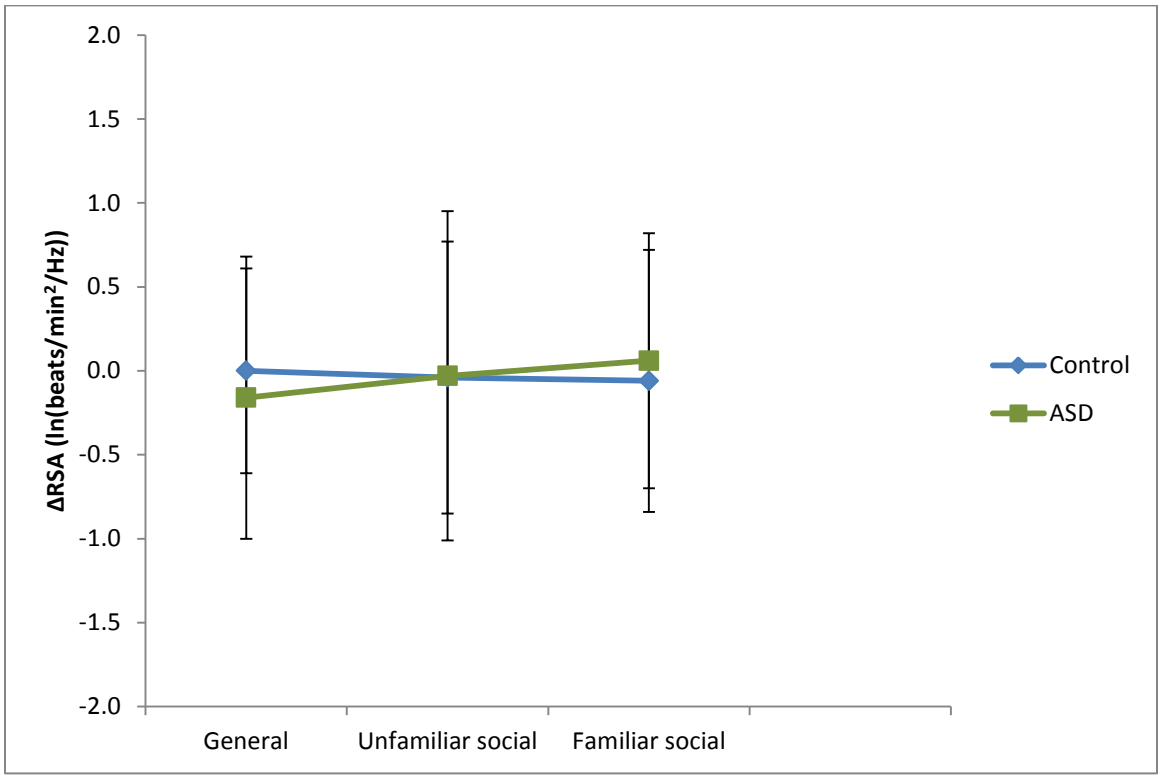
Percentage of correct responses during reward task by diagnostic group and reward condition. Error bars represent standard deviation from the mean.

Figure 3.



PEP reactivity (ΔPEP) in milliseconds to general, unfamiliar social, and familiar social reward conditions by diagnostic group. Error bars represent standard deviation from the mean.

Figure 4.



RSA reactivity (ΔRSA) to general, unfamiliar social, and familiar social reward conditions by diagnostic group. Error bars represent standard deviation from the mean.

CURRICULUM VITAE

Educational Background

Graduate: University of Washington
Ph.D. in Clinical Psychology, 2012
Dissertation: Physiological Responses to Social and Non-social Reward in Autism

University of Oregon
M.A. in Psychology, 2004

Undergraduate: University of Oregon, Clark Honors College
B.A. with Departmental Honors in Psychology, 2002
Summa Cum Laude
Major Area of Study: Psychology
Minor Area of Study: Spanish Literature

Clinical Experience

2011 – 2012	Predoctoral Intern University of Colorado Denver School of Medicine
2010 – 2011	Therapist, Child and Adolescent Coping Study University of Washington, Department of Education
2010	Practicum Student, Autism Center Seattle Children's Hospital
2006 – 2010	Student Therapist, Psychological Services and Training Clinic University of Washington
2008 – 2010	Clinical Interviewer, Behavioral Activation Research Study Seattle Children's Hospital
2008 – 2009	Practicum Student, Outpatient & Residential Programs Ryther Child Center
2007 – 2008	Practicum Student, Neuropsychology Consultation Service Inpatient Psychiatric Unit, Seattle Children's Hospital

Specialized Clinical Training

2010 Autism Diagnostic Interview, Revised. Workshop provided by Dr. Natacha Akshoomoff. Certified "research reliable" by Dr. Akshoomoff.

2009 Autism Diagnostic Observation Schedule, Clinician and Researcher Workshops. Provided by the University of Michigan Autism and Communication Disorder Center.

Professional & Research Positions

2009 – 2011	Graduate Research Assistant, Adult Brain Imaging Study University of Washington Autism Center
2005 – 2009	Graduate Research Assistant, Child and Adolescent Adjustment Project University of Washington
2003 – 2005	Study Coordinator, Birth to Three Evaluation Oregon Research Institute & University of Oregon
2002 – 2005	Lab/Study Coordinator, Baldwin Infant Cognition Lab University of Oregon
2002 – 2005	Family and Peer Process Coder, IGS Cognitive Coder Oregon Social Learning Center

Publications

Beauchaine, T.P., Gatzke-Kopp, L., Chipman, J., Neuhaus, E., Reid, M.J., & Webster-Stratton, C. (under review). Sympathetic- and parasympathetic-linked cardiac function prospectively predict differential aspects of treatment response among preschoolers with ADHD.

Kleinhans, N.M., Pauley, G., Richards, T., Neuhaus, E., Martin, N., Corrigan, N. M., Shaw, D. W., Estes, A., & Dager, S. R. (under review). Age-related abnormalities in white matter microstructure in autism spectrum disorders.

Neuhaus, E., Beauchaine, T.P., & Bernier, R.A. (under revision). Physiological responses to social and non-social reward among children with autism. *Development and Psychopathology*.

Neuhaus, E., & Beauchaine, T. P. (in press). Behavioral disinhibition and vulnerability to psychopathology. In T. P. Beauchaine & S. P. Hinshaw (Eds.), *Child and adolescent psychopathology, Second Ed.* Hoboken, NJ: Wiley.

Beauchaine, T. P., Neuhaus, E., Zalewski, M., Crowell, S. E., & Potapova, N. (2011). Allostatic load and psychopathology. *Development and Psychopathology, 23*, 975-999.

Neuhaus, E., Beauchaine, T. P., & Bernier, R. (2010). Neurobiological correlates of social functioning in autism. *Clinical Psychology Review, 30,* 733-748.

Neuhaus, E., Beauchaine, T. P., Reid, M. J., & Webster-Stratton, C. (2009). Coercive processes and child vagal tone in families of preschoolers with attention-deficit/hyperactivity disorder. In S. M. Gordon & A. E. Mitchell (Eds.), *Attention-Deficit/Hyperactivity Disorder* (pp. 145-165). New York: Nova Science Publishers.

Beauchaine, T. P., Neuhaus, E., Brenner, S. L., & Gatzke-Kopp, L. (2008). Ten good reasons to consider biological processes in prevention and intervention research. *Development and Psychopathology, 20*, 745-774.

Beauchaine, T. P., & Neuhaus, E. (2008). Behavioral disinhibition and vulnerability to psychopathology. In T. P. Beauchaine & S. P. Hinshaw (Eds.), *Child and adolescent psychopathology* (pp. 129-156). Hoboken, NJ: Wiley.

Shannon, K. E., Beauchaine, T. P., Brenner, S. L., Neuhaus, E., & Gatzke-Kopp, L (2007). Familial and temperamental predictors of resilience in children at risk for conduct disorder and depression. *Development and Psychopathology, 19*, 701-727.

Unpublished Manuscripts

Neuhaus, E. E. (2004). *Weak central coherence affects social inferences in individuals with autism.* Unpublished master's thesis, University of Oregon, Eugene, Oregon.

Neuhaus, E. E. (2002). *Forest for the trees: The weak central coherence theory of autism.* Unpublished undergraduate thesis, University of Oregon, Eugene, Oregon.

Published Abstracts

Crowell, S. E., Beauchaine, T. P., Marsh, P., Shannon, K. E., Neuhaus, E., & Chipman-Chacon, J. (2006). Correspondence between psychophysiological and self-report measures of emotion regulation in a pre-adolescent sample. *Psychophysiology, 43*, S32.

Presentations

Neuhaus, E. (2012). Autism spectrum disorders: Red flags for referral and treatment recommendations. Presentation to Developmental Neuropsychology Clinic at the University of Denver.

Neuhaus, E., Beauchaine, T.P., Reid, M.J., & Webster-Stratton, C. (2010). The role of father characteristics in treatment outcome among children with conduct problems. Poster presented at the annual meeting of the Society for Research in Psychopathology, Seattle, Washington.

Neuhaus, E. (2009). Dysthymia, social anxiety, and cognitive therapy. Paper presented at the University of Washington Science Informed Case Presentations, Seattle, Washington.

Neuhaus, E., Beauchaine, T. P., Brenner, S. L., Crowell, S. E., Mead, H. K., Shannon, K. E., & Derbidge, C. (2008). Longitudinal relations between parental discipline and adolescent conduct problems. Poster presented at the annual meeting of the Society for Research in Psychopathology, Pittsburgh, Pennsylvania.

Neuhaus, E., Beauchaine, T. P., Kopp, L., & Chipman, J. (2007). Coercive processes in the families of preschoolers with ADHD. Poster presented at the annual meeting of the Society for Research in Psychopathology, Iowa City, Iowa.

Neuhaus, E. (2007). Coercive processes in the families of preschoolers with ADHD. Paper presented at the 36th Annual University of Washington Psychology Research Festival, Seattle, Washington.

Neuhaus, E. & Baldwin, D. (2006). Temperament differences in children with and without autism spectrum disorders. Poster presented at the annual meeting of the Society for Research in Psychopathology, San Diego, California.

Crowell, S. E., Beauchaine, T. P., Marsh, P., Shannon, K. E., Neuhaus, E., & Chipman-Chacon, J. (2006). Correspondences between psychophysiological and self-report measures of emotion regulation in a pre-adolescent sample. Poster presented at the annual meeting of the Society for Psychophysiological Research, Vancouver, British Columbia.

Neuhaus, E. E. (2002). Forest for the trees: The weak central coherence theory of autism. Poster presented at the annual Psychology Undergraduate Research Conference, Eugene, Oregon.

Teaching Experience

Approaches to Psychological Assessment (Teaching assistant)

University of Washington

Introduction to Psychology (Teaching assistant)
University of Washington

Honors and Awards

2008 Travel Award, Graduate School Fund for Excellence and Innovation, University of Washington
2007 Smadar Levin Award for Graduate Student Poster, Society for Research in Psychopathology
2006 Travel Award, Nathaniel Wagner Memorial Endowment Fund, University of Washington
Phi Beta Kappa
Presidential Scholarship, University of Oregon
General University Scholarship, University of Oregon
Dean's List, University of Oregon
Summa Cum Laude, University of Oregon
Psychology Departmental Honors, University of Oregon
Passed Undergraduate Oral Thesis Defense "with Distinction," University of Oregon
President's Award for Outstanding Scholastic Record and Distinguished Thesis, University of Oregon

Professional Organizations

Student member, International Society for Autism Research
Student member, American Psychological Association
Student member, American Psychological Association Division 53 – Society of Clinical Child and Adolescent Psychology
Member, Phi Beta Kappa National Honor Society
Member, Psi Chi National Honor Society

Editorial Experience

Ad Hoc Reviewer:
Archives of General Psychiatry
International Journal of Psychophysiology