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Modulation of reward in a live social context as revealed through interactive social neuroscience

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Abstract

Social neuroscience research investigating autism spectrum disorder (ASD) has yielded inconsistent findings, despite ASD being well-characterized by difficulties in social interaction and communication through behavioral observation. In particular, specific etiologies and functional and structural assays of the brain in autism have not been consistently identified. To date, most social neuroscience research has focused on a single person viewing static images. Research utilizing interactive social neuroscience featuring dual-brain recording offers great promise for the study of neurodevelopmental disabilities. Reward processing has been implicated in the pathology of ASD, yet mixed findings have brought uncertainty about the role reward processing deficits may play in ASD. The current study employed dual-brain EEG recording to examine reward processing during live interaction and its relation to autistic traits. 16 typically-developing (TD) adults played a competitive treasure-hunt game against a computer and against a human partner. EEG results revealed enhanced neural sensitivity to reward outcome during live interaction with a human competitor. Further, individuals with higher levels of autistic traits demonstrated reduced sensitivity to reward outcome during live interaction. These findings provide novel insight into reward processing mechanisms associated with autistic traits, as well as support the necessary utility of interactive social neuroscience techniques to study developmental disorders.

Keywords

autism spectrum disorder; EEG/ERP; reward; interactive social neuroscience

Introduction

Vulnerabilities in social communication and interpersonal interactions are hallmark characteristics of ASD. Atypicalities in social behavior are robustly demonstrated in the clinical and behavioral assessments used to diagnose and measure symptom severity in ASD. Nevertheless, extant social neuroscience research has produced inconsistent findings regarding the integrity of neural systems underpinning social behavior (McPartland, 2016). These inconsistencies may reflect heterogeneity in neural pathology converging in common clinical and behavioral phenotypes; however, they may also reflect the reduced ecological

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validity of experimental investigations. Clinical and behavioral data are typically collected during naturalistic interpersonal interactions (Volkmar et al., 1987). In contrast, most social neuroscience research acquires data during passive viewing of static social images on a computer monitor in relative social isolation. Such acquisition contexts present a challenge to ecological validity in that these experiments do not mirror the complexity, contingency, and dynamism of real-life social interactions, all particularly relevant for understanding ASD (Rolison, Naples, & McPartland, 2015). This failure to emulate contexts most likely to elicit social impairment in ASD may explain inconsistent neuroscientific findings (Hadjikhani et al., 2004; Kuhn et al., 2010; McPartland et al., 2012; Webb et al., 2010) and poor alignment of research findings and clinical characteristics (Klin, 2000). For these reasons, measurement of neural responses in actual social contexts, also called *interactive social neuroscience* or *second-person neuroscience*, represents a vital next step for ASD research (Rolison et al., 2015; Schilbach et al., 2013). In particular, investigation of the neural mechanisms of reward processing represents a domain of research ripe for advancement via interactive social neuroscience.

Atypical reward circuitry is implicated in the pathology of ASD. However, it remains unclear whether atypical reward processing is generalized across domains or is specific to social information. Children with ASD pay less attention to social stimuli relative to typically developing peers (Dawson, Meltzoff, Osterling, Rinaldi, & Brown, 1998; Klin, Jones, Schultz, Volkmar, & Cohen, 2002), putatively reflecting disruption of social reward systems (Dawson et al., 2004; Dawson, Webb, & McPartland, 2005). Stavropoulos and Carver (2014) identified attenuated neural response in social reward processing in children with ASD and McPartland et al. (2012) have found intact non-social reward processing in children with ASD. However, some studies have revealed dysfunctional reward processing in individuals with ASD in response to both social and non-social rewards (Dichter, Richey, Rittenberg, Sabatino, & Bodfish, 2012; Kohls et al., 2011; Kohls et al., 2013; Scott-Van Zeeland, Dapretto, Ghahremani, Poldrack, & Bookheimer, 2010). As described above and as is the case for most social neuroscience research, studies of social reward processing have tended to focus on a single person in isolation, e.g., recording brain activity while a participant performs a task and receives social feedback via photographs or videos of smiling faces (Cox et al., 2015; Kohls et al., 2011; Kohls et al., 2013; McPartland et al., 2012; Scott-Van Zeeland et al., 2010). Because these experimental paradigms differ significantly from naturalistic contexts of social reward, they may limit investigative power to understand subtle differences in reward processing particularly salient for understanding ASD. Consistent with this notion, our prior work, simulating real-time social praise from a video-conferenced observer, revealed a significant and specific correlation between social behavior and neural response to social reward (Cox et al., 2015). This work also demonstrated a correspondence among neural response to social reward and autistic traits, even among those falling short of clinical thresholds (i.e., the broader autism phenotype; Hurley, Losh, Parlier, Reznick, & Piven, 2007).

Since the ability to discriminate among the salience of environmental stimuli is critical for successful information and reward processing, interactive social neuroscience methods might provide a more accurate experimental approximation of reward processing due to the shifting topography of saliency associated with the presence of another person (Klin, Jones,

Schultz, & Volkmar, 2003). From birth, human's attention is drawn preferentially to conspecifics when they are present due to intrinsic social reward (Dawson et al., 2004), thereby changing the meaning and importance of other environmental cues, including those that may be rewarding. Consequently, the value of rewarding stimuli changes when humans are present, akin to most real-life settings. Further, since it is hypothesized that reduced sensitivity to social reward results in the observed social difficulty characteristic of ASD, it is critical to study reward processing in an ecologically valid social context through interactive social neuroscience methods.

Several studies have applied interactive social neuroscience methods during social game play. In studies based on the "Prisoner's Dilemma", in which each participant must decide whether to cooperate with an opponent or defect to earn the highest score, functional magnetic resonance imaging (fMRI) data has evidenced differential activation of the left amygdala based on whether participants believed they were either playing against a computer or a human opponent (Sakaiya et al., 2013). Electroencephalography (EEG) studies have revealed greater inter-brain correlations across participants during cooperation (Astolfi et al., 2011; De Vico Fallani et al., 2010) and higher beta and theta power during defect (Astolfi et al., 2010; Astolfi et al., 2011) conditions. In this context, Babiloni et al. (2007) hypothesized that activity of the anterior cingulate cortex (ACC) when "defecting" demonstrates accurate predicting of others behavior. Additional studies have used simultaneous EEG recording to examine inter-personal synchrony in other social contexts, such as card games (Babiloni et al., 2006) and musicians playing ensemble (Babiloni et al., 2011)¹. In contrast, electrophysiology has suggested that individuals with ASD have difficulty differentiating social exclusion and discerning among social contexts compared to their typically-developing peers when playing the Cyberball social exclusion paradigm (McPartland et al., 2011). While prior studies have not directly examined reward processing in an interactive context, the differential neural sensitivity toward others demonstrated by individuals with ASD in these non-interactive contexts implicates differences in reward processing systems. In TD individuals playing a betting game, social context during decision making modulated activation in the amygdala and right dorsolateral prefrontal cortex (Nawa, Nelson, Pine, & Ernst, 2008). These findings suggest that social context changes the saliency of environmental stimuli and modulates reward system circuitry. Thus, interactive social neuroscience holds promise to clarify diverse results from prior studies investigating social reward processing in ASD.

Due to the rapid, fluid nature of social interaction, brain imaging techniques with high temporal resolution can be applied to support parsing of distinct, sequential cognitive processes occurring in short periods of time. The millisecond resolution of event-related potentials (ERPs), electrophysiological brain responses time-locked to a stimulus, have been effective in revealing such temporal dynamics in reward processing (Holroyd, Coles, & Nieuwenhuis, 2002). ERP components reflective of both generic, low-level sensory processing (P1, N2) and higher-order function of the reward system (feedback-related negativity (FRN), P3) have been established as relevant to understanding reward processing.

¹See Rolison et al. (2015) for review of interactive neuroscience experiments.

The P1 is a positive-going deflection over occipitotemporal sites approximately 90–110 ms following the presentation of a visual stimulus that is associated with thalamocortical inhibitory input to visual cortex (Pratt, 2012). The N2 is a negative-going deflection over occipitotemporal cortex approximately 180–325 ms following presentation of an auditory or visual stimulus that is associated with deviation in form or context of a prevailing stimulus (Patel & Azzam, 2005). The FRN is a negative-going deflection observed over frontocentral scalp approximately 250 ms after receiving feedback indicating suboptimal outcome (Gehring & Willoughby, 2002; Hajcak, Moser, Holroyd, & Simons, 2006). The P3 is a positive component peaking between 300 to 600 ms post-stimulus, with greatest amplitude at centro-parietal scalp sites (Goldstein et al., 2006). The P3 has been implicated in reward processing (Sutton, Braren, Zubin, & John, 1965) and is influenced by stimulus probability, task relevance, and emotional value of stimuli (Goldstein et al., 2006).

The current study applied the temporal sensitivity of the EEG in an interactive social neuroscience paradigm to examine the relationship between reward processing and autistic traits. Using simultaneous EEG recording of two people directly interacting in the context of a competitive game, we examined ERP indices of reward processing and the broader autism phenotype. We hypothesized that individuals would demonstrate stronger response to reward as indexed by the FRN and P3 when interacting with another person instead of playing alone. Further, we hypothesized that severity of autistic traits would correlate with magnitude of neural response to reward, with individuals with higher levels of autistic traits demonstrating attenuated FRN and P3 response relative to those with lower levels of autistic traits. Consistent with the notion that social behavior, both on and off the autism spectrum, reflects function in specific neural circuitry subserving social interaction, we did not predict relationships in earlier sensory components (P1 or N2) and reward processing, given that these ERP components are associated with lower-level visual perception and deviations in visual stimuli, respectively (Patel & Azzam, 2005; Pratt, 2012). This study was designed to investigate individual differences in reward processing in a social context to reveal the effects of live social interaction on neural response relative to non-social interaction.

Methods

Participants

20 college-aged students ($M=21.7$ years, $SD=0.45$, 6 male) were recruited and voluntarily participated in this study in 10 same-sex dyads (recruited independently and paired arbitrarily). Participants were recruited from the Yale University and broader New Haven community. Exclusionary criteria included participants taking prescription medications that may affect cognitive processes (including benzodiazepines, barbiturates, antiepileptics, carbamazepine, and valproic acid), reporting significant head trauma or serious brain or psychiatric illness, or history of current learning or intellectual disability. All procedures were conducted with the understanding and written consent of participants and with approval of the Human Investigations Committee at the Yale School of Medicine. Participants were compensated \$25 for their participation in the study.

Behavioral Measures

Participants completed a series of self-report questionnaires that assessed social and communicative behavior and sub-clinical autistic behaviors including the Social Responsiveness Scale, 2nd edition – Adult Self-Report (SRS-2-A-SR; Constantino & Gruber, 2012), the Autism-Spectrum Quotient (AQ; Baron-Cohen, Wheelwright, Skinner, Martin, & Clubley, 2001), and the Broad Autism Phenotype Questionnaire (BAPQ; Hurley et al., 2007). To control for any pre-existing social relationships between dyad partners, the Relationship Closeness Inventory (RCI; Berscheid, Snyder, & Omoto, 1989) was administered to quantify variation and strength of interpersonal relationships. It was found that relationship closeness did not impact outcomes and will therefore not be discussed further. All participants fell below the clinical threshold for ASD based on the SRS-2-A-SR.

ERP Procedures

Task—For the first condition of this experiment, one participant left the room while the other participant played a game against the computer. Participants playing the game were seated in front of a computer screen with a wireless mouse. Stimuli were presented to each participant on a 43.5-cm LCD monitor (60-Hz, 1,280×1,024 resolution) with the Psychophysics Toolbox extensions of MATLAB 2012b (Brainard, 1997; Kleiner, Brainard, & Pelli, 2007; MathWorks, 2012b; Pelli, 1997) at a viewing distance of approximately 60 centimeters in a room with low ambient illumination. A 10×10 square grid was displayed on the screen, with the player's and computer's score at the bottom of the screen (Figure 1a). Participants were told that they were competing against the computer in a search for buried treasure on a common grid. They were instructed to use the mouse to click on one space at a time (alternating turns with the computer) to determine whether it hid buried treasure. When a player clicked to select a grid square revealing treasure, the grid flashed off the screen and, following a 700 ms delay, a white circle was displayed centrally on the screen for 700 ms. If the grid square selected did not contain buried treasure, a white square was displayed centrally on the screen for 700 ms, after a 700 ms delay following the mouse click. When the grid reappeared, the circle or square filled the corresponding space, indicating that the grid square had previously been selected (Figure 1b). A circle was always displayed when treasure was uncovered and a square was always displayed when no treasure was found. Players earned a point, which was reflected on the bottom of the screen, when they found treasure. Circle and square feedback had context-dependent meaning for the player depending on whether it was their turn or the computer's turn. For example, on a player's turn a circle was a positive outcome, but on the computer's turn, the circle was a negative outcome because the opponent won a point. In this way, reward value was independent of low-level visual stimulus properties. The paradigm was designed such that the baseline likelihood of “finding” or “missing” treasure on any given turn was 50%. To maintain close scores (to sustain participant engagement), if there was a score difference of 3 or more points between the two players, the chances of finding treasure became 75% for the player with the lower score and 25% for the player with the higher score. Additionally, the task was designed so the ultimate outcome of the game could not be determined until the final turn. If one player gained a lead such that they would automatically win before the last turn, they would “miss” all but the last turn so that the outcome would remain undetermined until the

end of the game; in this way, all turns mattered to each player, and a winner or loser would not be apparent until the conclusion of the game. This design ensured that the score was always within one point difference on the final round so that motivation remained constant and all trials could be used to calculate ERPs. The game was finished when all the grid squares had been selected. The two participants then switched places so that the second participant had a chance to play the game against the computer. After both participants had played, they were brought into the same room and played the game against each other, instead of against the computer. All participants played against the computer before playing against each other. Since “playing against computer” is akin to traditional ERP experiments, this design allowed for more direct comparison to prior studies (Cox et al., 2015; Dichter et al., 2012; Kohls et al., 2011; Kohls et al., 2013; McPartland et al., 2012; Scott-Van Zeeland et al., 2010), and avoided potential artifacts that might derive from playing the novel interactive condition first. Each game-play session took approximately 15 minutes to complete, resulting in 45 minutes of total recording.

EEG was recorded using the B-Alert X-24 EEG net (Advanced Brain Monitoring Inc., Carlsbad CA). The X-24 system is a wireless EEG sensor net with 20 electrodes placed per the standard international 10–20 system. Continuous EEG data was recorded at 256 Hz using the B-Alert acquisition software (Version 2.05.05; Advanced Brain Monitoring, 2013) with joint mastoid reference. Electrode resistance was kept under 10 kOhms with Synapse Conductive Electrode Cream. Continuous EEG data was synchronized using an ABM External Sync Unit (ESU) connected to the stimulus presentation computer via parallel port, the acquisition computer via USB, and the X-24 headset via Bluetooth. EEG was recorded from each participant separately when playing against the computer as well as when participants played against each other.

EEG data were processed using the ERPLAB 4.0.3.1 toolbox of EEGLAB (Delorme & Makeig, 2004; Lopez-Calderon & Luck, 2014). Data was filtered using an IIR Butterworth 30 Hz low-pass filter. Data were hand-edited to remove artifactual trials, and eye-blink artifacts were identified using independent component analysis (ICA). Independent components were inspected alongside epoched data, and blink components were removed. Continuous data was epoched to the onset of feedback (hit or miss as indicated by circles or squares, respectively). Participants with more than 75% bad trials were excluded from analysis. Final analyses included 16 participants (6 male) in 8 dyads.

Electrodes of interest for the P1, N2, FRN, and P3 were selected based on maximal observed amplitude of each component and on scalp sites reported in the literature (Gehring & Willoughby, 2002; Goldstein et al., 2006; Hajcak et al., 2006; Hauser et al., 2014). Temporal windows for EEG components were based on inspection of the grand averaged waveforms and confirmed in individual averages. The P1 was measured as maximum amplitude within 100–200 ms from feedback onset across O1 and O2 and the N2 was measured as minimum amplitude within 100–250 ms from feedback onset across O1 and O2. The FRN was measured as mean amplitude for electrodes Fz and Cz within 250–365 ms following feedback (Yeung, Holroyd, & Cohen, 2005). The P3 was measured as maximum amplitude for electrodes Cz, Pz, and POz within 310–465 ms following feedback. ERP amplitudes for

each component was exported to IBM SPSS Statistics for Macintosh (Version 22.0) for analysis.

Data Analysis

Peak amplitudes for P1, N2, and P3 and mean amplitude for FRN were analyzed separately using repeated measures ANOVAs, with outcome (win/miss), player (self/opponent), and competitor (partner/computer) as within-subjects factors. Planned comparisons (paired samples t-tests) were used to compare FRN and P3 amplitudes between outcomes, players, and competitors, and Pearson product-moment correlations were used for assessing the relationship between ERP components and behavioral measures. For all analyses, the statistical significance level was set at $\alpha < 0.05$. Effect size estimates for analyses of variance were calculated with partial eta-squared (η^2_{partial} ; $\eta^2 = 0.01$ is a small effect size, 0.06 is a medium effect size, and 0.14 is large effect size; (Kittler, Menard, & Phillips, 2007)); for t-tests with Cohen's d (d ; $d = 0.2$ is a small effect size, 0.5 is a medium effect size, and 0.8 is a large effect size; (Kittler et al., 2007)); and for behavioral correlations with Pearson's correlation coefficient (r).

Results

ERP measures

P1 and N2 amplitudes—Grand average waveforms for the P1 and N2 are presented in Figure 2. There were no significant effects of outcome, player, competitor, or interactions for P1 amplitude, all F s < 2.6 , all p s > 0.13 , or N2 amplitude. all F s < 1.8 , all p s > 0.10 .

FRN amplitude—Grand average waveforms for the FRN are presented in Figure 3. There was a main effect of outcome, $F(1, 15) = 15.62$, $MSE = 8.97$, $p = 0.001$, $\eta^2_{\text{partial}} = 0.510$, indicating a more negative FRN amplitude in response to miss outcomes than win outcomes. Additionally, there was a main effect of player, $F(1, 15) = 6.73$, $MSE = 43.85$, $p = 0.02$, $\eta^2_{\text{partial}} = 0.31$, indicating more negative FRN amplitude in response to opponent action as compared to self action. There was no main effect of competitor, $F(1, 15) = 2.10$, $MSE = 42.69$, $p = 0.17$, $\eta^2_{\text{partial}} = 0.12$, but there was an interaction effect between competitor and player, $F(1, 15) = 49.66$, $MSE = 30.87$, $p = 0.007$, $\eta^2_{\text{partial}} = 0.39$. There were no significant interactions between competitor and outcome, $F(1, 15) = 0.11$, $MSE = 13.31$, $p = 0.74$, $\eta^2_{\text{partial}} = 0.007$, player and outcome $F(1, 15) = 1.87$, $MSE = 10.62$, $p = 0.19$, $\eta^2_{\text{partial}} = 0.11$, or between player, outcome, and competitor, $F(1, 15) = 1.10$, $MSE = 34.53$, $p = 0.33$, $\eta^2_{\text{partial}} = 0.06$. In summary, miss outcome and opponent action elicited a more negative FRN amplitude, with an additional interaction effect between competitor and player. Comparisons of means investigating the directionality of these effects revealed more negative FRN amplitude in response to self-win, $t(15) = 2.26$, $p = 0.04$, $d = 0.83$, and self-miss, $t(15) = 3.31$, $p = 0.01$, $d = 0.69$, when playing against a computer than when playing against another person. Additionally, when playing against another person, self-win had a less negative FRN amplitude compared to opponent-win, $t(15) = 3.70$, $p < 0.002$, $d = 1.30$, self-miss, $t(15) = 2.40$, $p = 0.03$, $d = 0.70$, and opponent-miss, $t(15) = 6.17$, $p < 0.001$, $d = 1.62$. Further, when playing against another person self-miss had less negative FRN

amplitude than opponent-win, $t(15) = -2.42$, $p = 0.03$, $d = -0.64$, and opponent-miss had more negative FRN amplitude than self-miss, $t(15) = 2.62$, $p = 0.02$, $d = 0.85$.

P3 amplitude—Grand average waveforms for the P3 are presented in Figure 4. There was a main effect of player, $F(1,15) = 8.04$, $MSE = 30.94$, $p = 0.013$, $\eta^2_{\text{partial}} = 0.35$, indicating greater P3 amplitude in response to self rather than opponent action. There were no main effects of competitor or outcome, $F_s < 1$. There was an interaction effect between player and competitor, $F(1,15) = 14.10$, $MSE = 47.25$, $p = 0.002$, $\eta^2_{\text{partial}} = 0.49$. There were no significant interaction effects of competitor and outcome, player and outcome, or competitor, player, and outcome, $F_s < 1$. Comparisons of means investigating the directionality of these effects revealed greater P3 amplitude for self-win, $t(15) = 2.36$, $p = 0.03$, $d = 1.01$, and self-miss, $t(15) = 2.40$, $p = 0.03$, $d = 0.63$, when playing against another person instead of a computer. Additionally, when playing against another person P3 amplitude was greater for self-win than opponent-win, $t(15) = 3.77$, $p = 0.002$, $d = 1.35$, and opponent-miss, $t(15) = 3.64$, $p = 0.002$, $d = 1.20$. Further, the P3 amplitude for opponent-win was greater than for self-miss, $t(15) = -4.70$, $p < 0.001$, $d = -1.16$, and the amplitude for self-miss was greater than for opponent-miss, $t(15) = 3.77$, $p = 0.002$, $d = 0.99$.

Behavioral measures

To examine the relationship between reward system activity and autistic traits, we computed difference scores between self-win, opponent-win, self-miss, and opponent-miss while playing against another person and a computer for the FRN and P3 and explored their relationships with measures of autistic phenotype (AQ, BAPQ, and SRS).

Associations among autistic traits and FRN amplitude are depicted in Figure 5. Difference in FRN amplitude between self-win and opponent-win conditions when playing against another person correlated with both AQ score, $r = -0.51$, $p = 0.05$, and BAPQ score, $r = -0.56$, $p = 0.03$, with individuals with higher levels of autistic traits demonstrating smaller differences between self-win and opponent-win conditions. Individuals with higher levels of autistic traits, as indexed by the BAPQ, demonstrated smaller differences in FRN amplitude between self-win and opponent-miss when playing against another human, $r = -0.54$, $p = 0.03$. Additionally, they showed greater difference in FRN amplitude for opponent-win and self-miss when playing against another human, $r = 0.53$, $p = 0.04$.

Associations among autistic traits and P3 amplitude are depicted in Figure 6. With respect to the P3, individuals with higher levels of autistic traits, as indexed by the SRS, demonstrated less difference in P3 amplitude in response to self-miss when playing against another person versus a computer, $r = -0.50$, $p = 0.05$. Additionally, when autistic traits were measured using the BAPQ, individuals with higher levels of autistic traits demonstrated a smaller difference in P3 amplitude between self-win and opponent-win conditions when playing against another person, $r = -0.52$, $p = 0.04$. They also showed a greater difference in P3 amplitude between opponent-win and self-miss conditions when playing against another person, $r = 0.52$, $p = 0.04$.

Discussion

Studies of social neuroscience have largely focused on studying the brain in isolation, providing limited insight into neural processes underlying actual social behavior. Interactive social neuroscience holds promise to investigate neural mechanisms underlying social information processing with greater ecological validity, a goal of paramount importance in investigations of social disabilities, such as ASD. The current study examined the neural bases of reward processing in the context of a competitive game played against both a computer and live partner. Dual brain EEG recording explored the influence of live social interaction on reward processing as indexed by early (P1 and N2) and late (FRN and P3) ERP components implicated in stimulus processing. Effects of interpersonal versus computer interaction were then examined in relation to social characteristics, i.e., level of autistic traits.

Participants demonstrated no significant differences in early sensory ERP components, suggesting that early perceptual processing was not influenced by our experimental manipulation. In contrast, effects of social interaction were observed at components specifically associated with reward processing. Differential effects of player at the FRN were greatest when playing against a human competitor. As expected, the least negative FRN was observed in response to self-win; this attenuation of FRN to this optimal outcome is consistent with its interpretation as an index of neural response to sub-optimal outcomes (Hajcak et al., 2006). Correspondingly, self-miss, a suboptimal outcome, elicited a greater amplitude FRN than self-win. However, when playing against another person, self-miss, in turn, elicited a less negative FRN than opponent-win. These results indicating varied FRN response to these distinct outcomes may be interpreted in several ways. Since participants are competing for maximum points, opponent-win results in the competitor gaining a point, which is a more negative outcome for the current player, whereas self-miss results in no net change in points. In this respect, our results may be interpreted as a graded FRN response in proportion to the magnitude of “badness” of the outcome. Such an interpretation contradicts a robust literature suggesting that, rather than representing such a relative index of suboptimality, FRN amplitude reflects binary evaluation of good versus suboptimal outcomes (Holroyd, Hajcak, & Larsen, 2006). Given the incongruence of this interpretation of our findings with prior data, we speculate that the tiered differences we observe in FRN amplitude suggest a distinct social contribution. Overlaying the established distinction between good and bad outcomes, we observe further enhancement of FRN indication of suboptimality in social context. The P3 also demonstrated a similar tiered response when playing against a human competitor – with self-win demonstrating the greatest amplitude response, followed by self-miss, opponent-miss, and opponent-win. These results demonstrate a bias towards self-relevant feedback at the P3, which is consistent with previous findings (Zhou, Yu, & Zhou, 2010). Though our study and our methods, with limited spatial resolution, are not designed to address questions of regional connectivity, we speculate that this unprecedented pattern of results reflects the interplay of two distinct brain system, the dopaminergic midbrain-anterior cingulate cortex (ACC) circuit presumed to generate the FRN (Holroyd & Coles, 2002) and the network of regions composing social brain circuitry (Allman, Hakeem, Erwin, Nimchinsky, & Hof, 2006; Pelphrey, Yang, &

McPartland, 2014). This question can be more effectively investigated in future studies applying brain imaging tools with greater spatial resolution. Our interpretation suggests specific modulation of reward systems by social brain circuitry; a second, and related, possibility is that social interaction elicits a non-specific increase in salience or attention. Although this cannot be ruled out, we find the absence of effects at early sensory components as most consistent with an interpretation of specific social influence on reward circuitry.

Further, differences in neural response to reward outcome were modulated by level of autistic traits. Individuals with higher levels of autistic traits demonstrated reduced differentiation in neural response between self-relevant and opponent-relevant outcomes when playing against a human competitor. Importantly, relationship among autistic traits and neural response were only evident in the context of interactions with another person, with individuals higher in autistic traits demonstrating reduced sensitivity in differences between reward outcomes. Prior research has found intact neural response to non-social reward in children with ASD (McPartland et al., 2012; Stavropoulos & Carver, 2014), however these studies have been limited to interaction with a computer. The current interactive social neuroscience paradigm, however, revealed differences in FRN amplitude across outcome as a function of level of autistic traits when playing against a human competitor. These differences reveal that autistic trait severity modulates the response to reward in social context. Of note, the rewards employed here are not conventional social rewards (e.g., smiling faces). Given that points are not tied to monetary reward or other incentive, they represent a reward that may be social by virtue of context. The current findings suggest that when individuals are in a social context (e.g. in the presence of another person), reward processing is modulated—even when the rewards themselves are nonsocial (squares, circles representing points). These findings have particular relevance for the study of ASD since challenges faced by individuals with ASD are more evident in complex social interactions rather than in highly structured or simulated social scenarios.

The present study should be considered in light of several limitations. Due to the relatively small sample size, the findings have limited statistical power. However, these results have large effect sizes, demonstrating the value of continuing this line of research. Additionally, each participant played against the computer before playing against another participant, which may have influenced the results. Though our results are consistent with prior ERP research indicating modulations of the FRN and P300, future studies with larger sample sizes can counterbalance these conditions and confirm these results. Further, because no individuals had ASD, all analyses were based on sub-clinical autistic traits in individuals in the range of typical functioning. Thus, these results may provide limited insight into reward processing in individuals who have met diagnostic criteria for ASD and therefore replication of the current methods in an ASD sample is warranted. Additionally, due to sex differences in ASD (Anderson et al., 2013; Coffman, Anderson, Naples, & McPartland, 2015), it is important to extend the research in samples that will support analysis of sex differences.

This study aimed to investigate the effectiveness of interactive social neuroscience methods to elucidate neural processes underlying social information processing. Although reward processing has been implicated in the pathology of ASD, existing findings have been

inconsistent and provided a mixed perspective regarding the role reward processing deficits may play in ASD. Through the use of a presumably more ecologically valid methodological approach, this study identified differences in reward processing, observed in FRN and P3 amplitudes, in a live social context, which were also modulated by autistic traits. These findings have potential clinical implications for the diagnosis and treatment of individuals with ASD. If it is possible to pinpoint ways that the brain responds differently in ASD simply by having another person in the room, there could be potential for measuring treatment change in response to social intervention. Additionally, if these indices exist early in life, it might be possible to detect atypical brain activity in social contexts early developmentally, which could improve early detection and diagnosis of ASD.

Conclusions

The current study used interactive social neuroscience to investigate reward processing during live interaction in relation to levels of autistic traits. This was the first study to examine reward-feedback monitoring during live interaction between two individuals, as opposed to previous studies that have examined reward processing during interaction with a computer. Results of the current study reveal modulation of brain activity during live interaction that is not present during interaction with a computer. Individuals with higher levels of autistic traits demonstrated reduced sensitivity to differences between reward outcomes during live interaction. Our findings provide new insight into differences in reward processing mechanisms associated with autistic traits and further emphasize the utility of interactive social neuroscience approaches for the study of neurodevelopmental disabilities.

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Figure 1.

(a) Game board seen by both players. Each player was randomly assigned to be Player 0 or Player 1. Players selected a blank space on the 10×10 square grid when it was their turn. Once selected, spaces were filled with a circle or square if treasure was won or missed, respectively. Each player's score was displayed at the bottom of the screen. (b) Trial sequence for win and miss conditions. Participants were shown a treasure map for unlimited duration. Once they selected a box, a blank screen appeared for 700 ms, followed by feedback whether they found (circle, “win”) or did not find (square, “miss”) treasure for 700 ms, followed by a blank screen for 700 ms. Participants alternated each trial with one person acting and one person observing.

Figure 2.
Scalp plots for P1, N2, FRN, and P3 components when playing against a computer.

Figure 3.
Scalp plots for P1, N2, FRN, and P3 components when playing against a human.

Figure 4. Grand averaged waveform depicting P1 and N2 amplitudes (a) when playing against a computer and (b) when playing against a human. P1 and N2 components were extracted from electrodes O1 and O2.

Figure 5. Grand averaged waveform depicting FRN amplitude (a) when playing against a computer and (b) when playing against a human. FRN was extracted from electrodes Fz and Cz.

Figure 6. Grand averaged waveform depicting P3 amplitude (a) when playing against a computer and (b) when playing against a human. P3 was extracted from electrodes Pz, Oz, and POz.

Figure 7. Scatterplots depicting association of autistic traits and difference in FRN amplitude between outcomes when playing against another person: (a) difference between self-win and opponent-win and AQ score; (b) difference between self-win and opponent-win and BAPQ score; (c) difference between self-win and opponent-miss and BAPQ score; (d) difference between opponent-win and self-miss and BAPQ score.

Figure 8.

Scatterplots depicting association of autistic traits and differences in P3 amplitude: (a) difference between self-miss when playing against another person and a computer and SRS score; (b) difference between self-win and opponent-win when playing against another person and BAPQ score; (c) difference between opponent-win and self-miss when playing against another person and BAPQ score.