

Reward anticipation and processing of social versus nonsocial stimuli in children with and without autism spectrum disorders

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Background: How children respond to social and nonsocial rewards has important implications for both typical and atypical social-cognitive development. Individuals with autism spectrum disorders (ASD) are thought to process rewards differently than typically developing (TD) individuals. However, there is little direct evidence to support this claim. **Methods:** Two event-related potentials were measured. The stimulus preceding negativity (SPN) was utilized to measure reward anticipation, and the feedback related negativity (FRN) was utilized to measure reward processing. Participants were 6- to 8-year-olds with ($N = 20$) and without ($N = 23$) ASD. Children were presented with rewards accompanied by incidental face or nonface stimuli. Nonface stimuli were composed of scrambled face elements in the shape of arrows, controlling for low-level visual properties. **Results:** Children with ASD showed smaller responses while anticipating and processing rewards accompanied by social stimuli than TD children. Anticipation and processing of rewards accompanied by nonsocial stimuli was intact in children with ASD. **Conclusions:** This is the first study to measure both reward anticipation and processing in ASD while controlling for reward properties. The findings provide evidence that children with autism have reward anticipation and processing deficits for social stimuli only. Our results suggest that while typically developing children find social stimuli more salient than nonsocial stimuli, children with ASD may have the opposite preference. **Keywords:** Autism spectrum disorder, social motivation, event-related potentials, social stimuli.

Introduction

Children's learning is strongly motivated by social signals such as eye contact, smiling, speech sounds, and contingent interaction. For example, language learning requires a socially interactive context rather than auditory exposure alone (Kuhl, Tsao, & Liu, 2003). In typically developing individuals, at least one kind of social stimulation, eye contact, activates the brain's reward system (Kampe, Frith, Dolan, & Frith, 2001). Children with autism spectrum disorders (ASD) have profound social deficits that may be linked to a neural reward system that differs from typically developing (TD) individuals. Here, we empirically compare social motivation and reward processing using electrophysiology in TD and ASD children.

Children with ASD appear to lack enjoyment in social activities, and the *social motivation hypothesis* (SMH; Chevallier, Kohls, Troiani, Brodtkin, & Schultz, 2012; Dawson, 2008; Dawson et al., 2002, 2005; Grelotti, Gauthier, & Schultz, 2002; Schultz, 2005) suggests that this leads to downstream autism symptomatology including: abnormal brain responses to faces (e.g., McPartland, Dawson, Webb, Panagiotides, & Carver, 2004), language impairments (e.g., Charman et al., 1998), and joint attention deficits (e.g., Mundy, Sigman, Ungerer, & Sherman, 1986). In ASD interventions, the lack of enjoyment in social interaction is often referred to as lack of

intrinsic motivation. Behavioral interventionists often utilize extrinsic means to motivate children with ASD to socially engage, for example using candy to reward children for making eye contact (Jones & Carr, 2004). This is problematic because when the extrinsic motivator is no longer presented, social behaviors can regress (Whalen & Schreibman, 2003). Increasing social motivation in ASD is a critical step in improving the efficacy of behavioral interventions (Stavropoulos & Carver, 2013a).

A small number of neuroscience studies have evaluated social motivation in adolescents and adults with ASD (Delmonte et al., 2012; Dichter, Richey, Rittenberg, Sabatino, & Bodfish, 2012; Kohls et al., 2011, 2013; Richey et al., 2014; Scott-Van Zeeland, Dapretto, Ghahremani, Poldrack, & Bookheimer, 2010). Results suggest that individuals with ASD anticipate and process rewards differently than TD individuals. However, studies differ with regard to whether reward anticipation, reward processing, or both were tested and also varied regarding whether monetary rewards, social rewards, or both were employed.

One potential issue in previous studies is that the rewards for social and nonsocial conditions were not equated. Tangible rewards, such as money, were contrasted with intangible incentives (e.g., pictures of faces). It is not clear, then, whether differences between the responses of individuals with ASD and typical development are due to differences in reward processing, or differences in responses to tangible versus intangible rewards.

Conflict of interest statement: No conflicts declared.

Anticipation phase: stimulus preceding negativity

One effective way to investigate neural anticipation of rewards is by using electrophysiology, specifically event-related-potentials (ERPs). The SPN is a component of the ERP that reflects brain activity occurring before expected feedback about one's performance (Brunia, Hackley, van Boxtel, Kotani, & Ohgami, 2011). SPN reflects the *expectation* of reward, and related activity of the dopaminergic reward system (van Boxtel & Böcker, 2004). fMRI studies provide evidence that tasks typically used to elicit the SPN lead to activity in the insular cortex (Tsukamoto et al., 2006) and caudate nucleus (Delgado, Nystrom, Fissell, Noll, & Fiez, 2000), brain areas involved in reward processing. A spatiotemporal dipole model of the SPN (Böcker, Brunia, & van den Berg-Lenssen, 1994) suggested the SPN is generated in the insular cortex, which is innervated with dopamine neurons (Gaspar, Berger, Febvret, Vigny, & Henry, 1989). Further evidence that the SPN involves the dopamine reward system comes from studies showing a reduced SPN in individuals with Parkinson's disease (who have a degradation of structures responsible for dopamine production) compared to control individuals (Mattox, Valle-Inclán, & Hackley, 2006).

Two studies have compared reward anticipation between TD individuals and those with ASD (Groen et al., 2008; Kohls et al., 2011). One study used a probabilistic learning task with monetary rewards. Children with ASD and ADHD demonstrated larger SPN amplitudes than TD children when anticipating positive outcomes, but equivalent SPN amplitudes anticipating negative outcomes (Groen et al., 2008). A second study measured the P300 in response to cues triggering trials with social versus nonsocial reward anticipation in adolescents with and without ASD. As a control, a condition without rewards was used. TD children exhibited larger P300s during reward versus nonreward conditions, but children with autism did not. In addition, children with autism exhibited smaller P300s after cues initiating social reward anticipation trials (Kohls et al., 2011).

Response phase: feedback related negativity component

It is also informative to investigate the neural underpinnings of reward processing *after* feedback. The feedback related negativity (FRN) is an ERP component occurring 200–300 ms after feedback, and characterized by a negativity in response to 'loss' versus 'gain' trials (Hajcak, Moser, Holroyd, & Simons, 2006). Source localization studies suggest that the FRN reflects activity in the dopamine reward system (Holroyd & Coles, 2002), and is generated by the striatum, medial-frontal cortex and anterior cingulate cortex – areas related to

reward processing (Foti, Weinberg, Dien, & Hajcak, 2011; Nieuwenhuis, Slagter, von Geusau, Heslenfeld, & Holroyd, 2005).

Previous studies compared the FRN in adolescents and young adults with and without ASD during a guessing game in which participants won money for correct answers, and lost money for incorrect answers (Larson, South, Krauskopf, Clawson, & Crowley, 2011), or won money for correct answers, and did not lose or win anything for incorrect answers (McPartland et al., 2012). Both studies found similar activation patterns in individuals with and without ASD, suggesting that individuals with ASD do not demonstrate deficits in feedback processing when the rewards involve money. No previous studies have measured the FRN in response to social versus nonsocial rewards in TD, or in ASD compared with TD.

Design of the study

Previous studies provide mixed results about whether reward anticipation and reward processing after feedback are dampened in individuals with ASD for monetary rewards, social rewards, or both. Although the social motivation hypothesis suggests that children lack motivation for social interaction, no evidence exists to clarify whether differences in motivation in children with ASD are due to a *lack* of social motivation or an *increase* in nonsocial motivation. Social deficits could occur because children with ASD are impaired in social motivation, because they are more motivated by nonsocial rewards than typically developing children, or a combination of the two. Previous authors have raised this possibility (Kohls, Chevallier, Troiani, & Schultz, 2012; Richey et al., 2014), but it has not been explored directly.

This study expands upon previous investigations and seeks to add additional information about the reward system in ASD. We have developed an ERP paradigm, in which the reward for correct answers is controlled between social and nonsocial conditions, and the low-level physical properties of social versus nonsocial stimuli are matched (Stavropoulos & Carver, 2013b). Previous studies have not combined the SPN and FRN components in investigations of responses to social stimuli. Here, we contrast performance on this task between individuals with ASD and TD, and measure both the anticipation and outcome phases of reward processing. We hypothesize that children with ASD will demonstrate attenuated ERP responses while anticipating feedback accompanied by social stimuli (reflected in a reduced amplitude of the SPN), and attenuated response to feedback accompanied by social stimuli (via the FRN). Examining both the SPN and FRN in the same children has the potential to reveal the time course of reward anticipation and processing in children with ASD.

Methods

Participants

We tested TD children ($N = 23$) and children with ASD ($N = 20$). Exclusionary criteria for participants with ASD included history of seizures, brain injury, neurological disorders, or any concurrent psychiatric condition (other than ASD), based on parent report. Exclusionary criteria for TD participants included all of the above criteria, plus an immediate family history of ASD. None of the children in the TD group were taking psychoactive medications. Three children in the ASD group were taking medication in order to improve concentration, but one of the three did not take his medication on the day he came in for this study. Participants were recruited from a UC San Diego subject pool and through postings on websites for parents of children on the autism spectrum. All participants had normal hearing and normal or corrected to normal vision. Procedures were approved by the institutional review board, and written consent was obtained from caregivers. All children over 7 years of age signed an assent form. Data from 17 children in the TD group were reported previously (Stavropoulos & Carver, 2013b), and were used to match children tested in the ASD group on gender and full-scale IQ.

Table 1 provides detailed participant information. IQ scores (Wechsler Abbreviated Scale of Intelligence, Wechsler, 1999) were available for all 20 children with ASD, and 22 of 23 TD children (one TD child was unable to complete the WASI due to time constraints). Of the final sample of 43 children, no significant differences were found between groups on full-scale IQ scores $F(1,40) = .36$. There were differences between the TD and ASD groups in chronological age, $F(1,41) = 5.86$, $p = .02$. In order to confirm that age did not affect SPN or FRN amplitude in our sample, we examined correlations between age and ERP amplitude for all conditions for the SPN and FRN. These analyses revealed no correlation between age and ERP amplitude (all $r_s < .13$). Children in the ASD group had been previously diagnosed with ASD through various sources (e.g. formal evaluations through an autism center, or school diagnosis). Diagnosis was confirmed for this study with Module 3 of the ADOS-2 (Lord et al., 2012). The ADOS-2 was administered by an individual trained to research reliability on administration, scoring, and interpretation of the measure.

Behavioral measures

Participants' caregivers completed the Social Responsiveness Scales (SRS-2; Constantino & Gruber, 2012), which measure social responsiveness and behavior. We also tested for overt motivation or affective differences between groups for each condition. To accomplish this, children ($N = 21$ TD, 19 ASD) completed a 1–7 Likert rating scale of how much they enjoyed the game (1 = 'I do not like this game', and 7 = 'I love this game') after each block. This was used in order to gather more information about whether one group felt more or less motivated to engage in the task. Previous research suggests that

the presence of reward versus no-reward affects SPN amplitude – with greater SPN amplitude in reward versus no-reward conditions (Kotani et al., 2003) – and we wished to assess whether both groups felt equally invested in the game. Participants also completed a 1–7 Likert scale about their perception of getting correct answers (1 = 'I never got correct answers', and 7 = 'I always got correct answers'). In reality, the correct versus incorrect answers was predetermined and controlled by experimental design, and the rating was used to verify that the groups did not differ in their perception that they were obtaining correct answers.

Stimuli and task

The stimuli and task are described in detail in Stavropoulos and Carver (2013b). Briefly, the task was a guessing game that presented blocks of trials that used left and right visual stimuli (question marks). Participants were asked to indicate their guess via button press whether the left or right stimulus was 'correct.' After this choice, the left and right question marks were replaced with an arrow in the middle pointing toward whichever question mark the participant chose. This was done to reinforce the idea that participants had control over the task and their responses were being recorded.

There were two blocked feedback conditions: *social* versus *nonsocial*. Incidental stimuli in the social condition were faces obtained from the NimStim database (Tottenham et al., 2009) that were smiling for 'correct' answers and frowning for 'incorrect' answers. In order to avoid confounds resulting from use of a single face or gender, 33 faces (18 female, 15 male) from the database were utilized. Incidental stimuli in the nonsocial condition were composed of scrambled face elements from the social condition formed into an arrow that pointed upwards for 'correct' answers and downwards for 'incorrect' answers. The use of scrambled faces to construct the arrow controlled for low-level visual features of the stimuli. Both faces and arrows were presented in pseudorandom order, with no image repeating on consecutive trials. Presented stimuli subtended a horizontal visual angle of 14.5°, and a vertical visual angle of 10.67°. Each participant viewed identical stimuli in the same order for each condition (e.g. the social feedback block was the same for each participant), but whether individuals viewed the social versus nonsocial block first was counterbalanced between participants.

Participants were told that the reward for each correct answer was a goldfish cracker, or if they preferred, fruit snacks. Participants were told there was no penalty for incorrect answers. Participants were told that if they guessed correctly, they would see a ring of intact goldfish crackers, and the goldfish would be crossed out for incorrect answers. Importantly, in both the social and nonsocial feedback trials, the face/arrow information was incidental. Figure 1 depicts the stimuli and timeline in the social and nonsocial conditions. A computer program predetermined correct versus incorrect answers in pseudorandom order such that children got 50% 'correct' and 50% 'incorrect', with no more than three of the same answer in a row.

Table 1 Participant characteristics including: IQ (WASI), age, gender, SRS-2 T-score, and ADOS-2 severity scores for the ASD group

Group	Participants	WASI (full-scale)	Age	Gender	SRS-2 SCI T-Score	SRS-2 RBB T-Score	ADOS-2 Severity score
ASD	20	M = 107.35 SE = 3.54	M = 8.28* SE = .23	19 M 1 F	M = 71.26** SE = 2.14	M = 69.63** SE = 2.26	M = 6.88 SE = .48
TD	23	M = 111.60 SE = 3.30	M = 7.47* SE = .21	22 M 1 F	M = 48.52** SE = 1.95	M = 50.69** SE = 2.07	N/A

* $p = .02$.

** $p \leq .0001$.

The two feedback conditions (face/'social' trials and arrow/'nonsocial' trials) were tested in separate blocks, each composed of 80 trials. Within each block of 80 trials, there were 30-s breaks every 15 trials. During breaks, participants were asked to relax, or move if they felt restless. Between blocks, a longer break (5–10 min) was taken. To control attentional effects, children were observed via webcam, and trials in which they were not attending to the stimulus were marked and discarded during analysis. Of the final sample, three children had trials excluded for this reason, and of those three, none had more than 10 trials excluded in this way.

EEG recording

Participants wore a standard, fitted cap (Electrocap International) with 33 silver/silver-chloride (Ag/AgCl) electrodes placed according to the extended international 10–20 system. Continuous EEG was recorded with a NeuroScan 4.5 System with a reference electrode at Cz and re-referenced offline to the average activity at left and right mastoids. Electrode resistance was kept under 10 kOhms. Continuous EEG was amplified with a low pass filter (70 Hz), a directly coupled high pass filter (DC), and a notch filter (60 Hz). The signal was digitized at a

rate of 250 samples per second via an Analog-to-Digital converter. Eye movement artifacts and blinks were monitored via horizontal electrooculogram (EOG) placed at the outer canthi of each eye and vertical EOG placed above and below the left eye. ERP trials were time locked to the onset of the feedback stimulus. For the SPN component, the baseline period was –2,200 to –2,000 ms, and the data were epoched from –2200 to 100 ms. For the FRN component, the baseline period was –200 to 0 ms, and the data were epoched from –200 to 800 ms. The interval between trials was varied between 1,800–2,000 ms. Trials with no behavioral response, or containing electrophysiological artifacts, were excluded from the averages.

Artifacts were removed via a four-step process. Data were visually inspected for drift exceeding ± 200 mV in all electrodes, high frequency noise visible in all electrodes larger than 100 mV, and flatlined data. Following inspection, data were epoched and eyeblink artifacts were identified using independent component analysis (ICA). Individual components were inspected alongside epoched data, and blink components were removed. To remove additional artifacts, we utilized a moving window peak-to-peak procedure in ERPLab (Lopez-Calderon & Luck, 2014), with a 200 ms moving window, a 100 ms window step, and a 150 mV voltage threshold. We excluded FRN data from one subject because

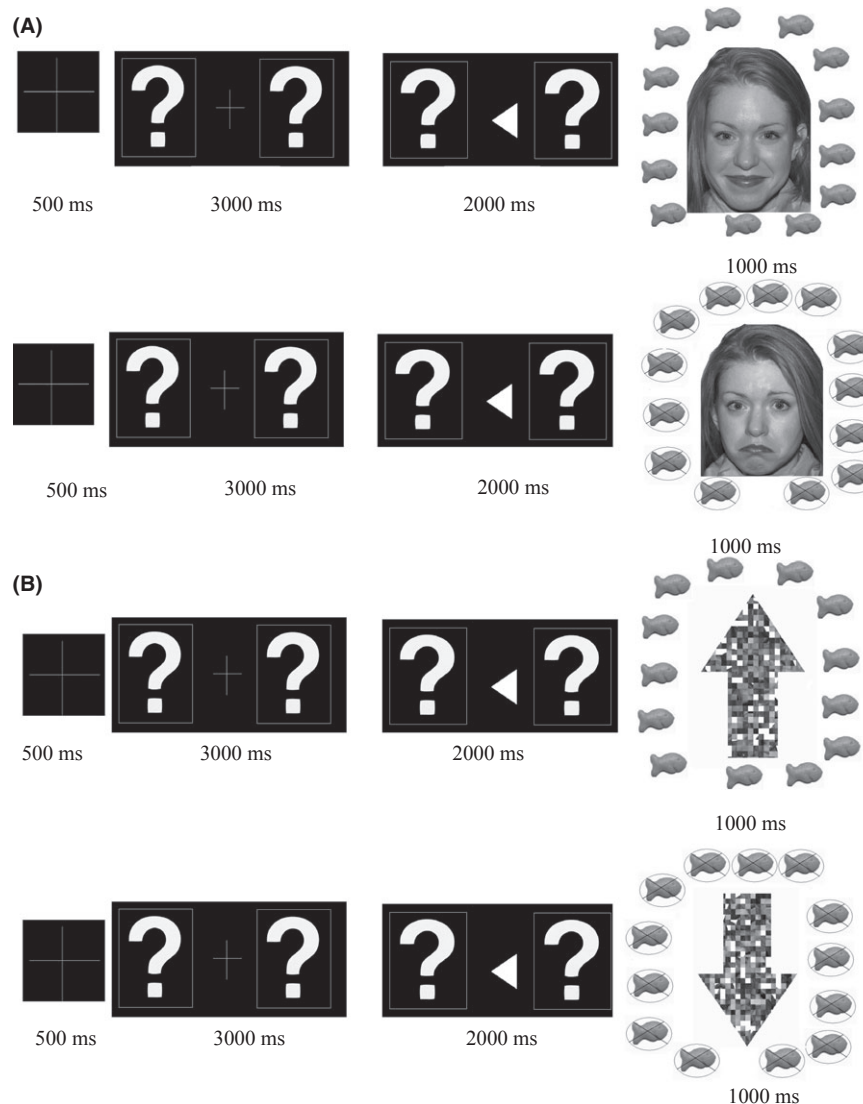


Figure 1 Stimulus presentation. (A) Schematic of the stimuli and timing for the social condition. (B) Schematic of the stimuli and timing for the nonsocial condition. Feedback for 'correct' answers is shown on top, and feedback for 'incorrect' answers is shown below. [Correction added online on 27th June 2014 after first publication on 30th May 2014. The caption is correct but the labels were placed in the wrong order. This error has been corrected in this version of the article and in the future printed issue]

they had fewer than 10 trials in their final average. Our final analyses for the SPN included 20 children with ASD, and 23 TD children, and our final analyses for the FRN includes 19 children with ASD and 23 TD children.

Results

Data were analyzed using JMP (version 10.0). We used repeated-measures analysis of variance (ANOVA) to test for differences between groups, conditions, hemisphere, and caudality (anterior-posterior scalp locations). Greenhouse-Geisser corrected degrees of freedom are reported to account for violations of sphericity.

Behavior

No significant differences were found between groups on children's Likert ratings of liking the game, $F(1,39) = .72$ ns, or perception of generating correct answers, $F(1,39) = .95$, ns. As expected, significant differences were found between groups on the SRS-2 social subscale, $F(1,41) = 64.27$, $p < .001$, and the repetitive behavior subscale, $F(1,41) = 38.23$, $p < .001$, with children with ASD scoring significantly higher on both subscales compared to TD children. Means and standard deviations for both groups on the SRS-2 are shown in Table 1.

Event-related-potential

Stimulus preceding negativity. The mean amplitude of the SPN was measured between -210 and -10 ms, prior to feedback onset, as defined in previous research (Kotani, Hiraku, Suda, & Aihara, 2001). Electrode sites F3/F4, C3/C4, P3/P4, and T5/T6, which are typically maximum amplitude sites for SPN (Kotani et al., 2003), were analyzed. Grand average waveforms for the face and arrow conditions for TD children and those with ASD are plotted in Figure 2.

A $2(\text{Group}) \times 2(\text{Conditions}) \times 2(\text{Hemisphere}) \times 4(\text{Electrode location})$ was conducted. No effects of hemisphere were found in either group or condition. We then conducted a $2(\text{Group}) \times 2(\text{Condition}) \times 4(\text{Electrode location})$ that was collapsed across hemispheres. This ANOVA showed a significant group \times condition interaction, $F(1, 41.05) = 7.19$, $p = .01$. Pair-wise comparisons revealed a significant group difference for social stimuli, 95% CI $[-13.18$ to $-.48]$ $F(1, 78.97) = 4.4$, $p = .038$. SPN amplitude was greater in the social condition for TD participants versus participants with ASD. There was a significant difference between the social versus nonsocial conditions for the TD group, 95% CI $[-11.42$ to $-.275]$ $F(1,41.52) = 4.19$, $p = .046$, with TD participants showing a larger SPN to the social versus nonsocial conditions. Children in the ASD group demonstrated the opposite pattern – a larger SPN response to arrows versus faces – however, this difference within the ASD group did not reach

significance ($p = .09$). There was no significant group difference for nonsocial stimuli ($p > .05$). There was a significant main effect of electrode position, $F(3, 123.1) = 3.15$, $p = .027$, with parietal and central electrodes eliciting larger SPNs than frontal or temporal electrodes, but Tukey's HSD test revealed no significant differences between individual electrode positions. Figure 3 shows topographic maps of mean ERP amplitude between -210 and -10 ms in the face and arrow conditions for TD and ASD children.

Trial numbers for each group in both the face and arrow conditions are shown in Table 2. No significant differences for trial numbers between groups were found in the social condition, $p > .1$. Significant differences in trial numbers between groups were found in the nonsocial condition $F(1,41) = 7.44$, $p < .01$. Due to this difference, we analyzed data from a subset of participants who were matched on number of trials (criteria for matching was within 4 trials). Thirteen children in each group were successfully matched. Comparisons of numbers of trials for each condition between groups were nonsignificant (all $ps > .5$). The group by condition interaction remained significant, $F(1,24.17) = 4.45$, $p = .045$ such that TD children had a larger SPN to social versus nonsocial stimuli, and children with ASD showed the opposite pattern.

Feedback related negativity. Previous literature has examined the FRN between 275–375 ms (Bress, Smith, Foti, Klein, & Hajcak, 2012). However, visual inspection of our waveforms revealed that our FRN occurred between 300–450 ms. Therefore, we used this time window for analysis. The FRN was measured separately for correct and incorrect trials as mean amplitude between 300–450 ms after feedback onset in frontal electrodes Fz, FCz, and Cz. Figure 4 shows grand averaged waveforms for electrodes Fz, Cz, and Cz for the TD and ASD groups.

A $2(\text{Group}) \times 2(\text{Condition}) \times 2(\text{Correct/incorrect}) \times 3(\text{Electrode})$ ANOVA was conducted. An interaction that approached statistical significance occurred between group, condition, and correctness, $F(1, 33.36) = 3.94$, $p = .055$ such that TD children had a larger FRN to correct versus incorrect answers in the face condition, but in the arrows condition their incorrect answers elicited a larger FRN compared to correct answers. For children with ASD, the pattern was reversed. That is, children with ASD had a larger FRN to correct versus incorrect answers in the arrow condition, but in the face condition their incorrect answers elicited a larger FRN. Pairwise comparisons revealed only marginal effects of specific contrasts by group or condition. These effects reached traditional significance (at the .05 level), but correction for multiple comparisons yielded a critical p -value of .0083, and by this criterion, none of the pairwise comparisons were significant. Figure 3 shows topographic maps of mean amplitude of ERP amplitude

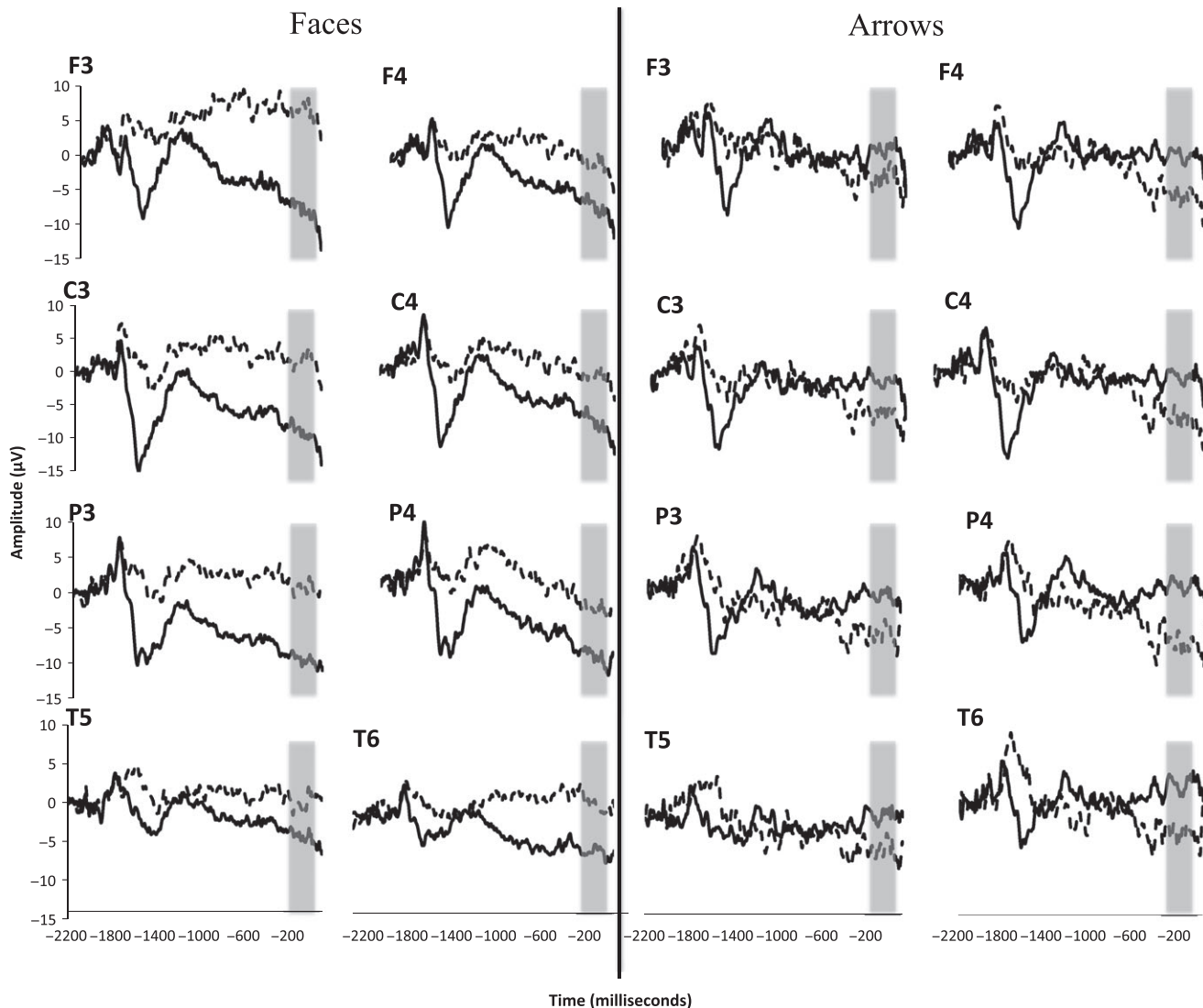


Figure 2 Grand averaged waveforms for TD children and those with ASD from the Stimulus Preceding Negativity (SPN) in response to social/faces (left) and nonsocial/arrows (right). TD children are represented by a solid line, and children with ASD with a dashed line. The area between -210 and -10 ms, used for statistical analysis, is highlighted with a gray box

between 300–450 ms for the face and arrow conditions in both the TD and ASD groups.

Previous literature has also investigated incorrect minus correct difference waves in the FRN (Bress et al., 2012; McPartland et al., 2012). Therefore, we conducted a $2(\text{Group}) \times 2(\text{Condition}) \times 3(\text{Electrode})$ analysis of the mean amplitude between 300 and 450 ms of the incorrect minus correct difference wave for each participant using a repeated-measures ANOVA. Consistent with our results when correct and incorrect answers were analyzed separately, there was a group by condition interaction for the difference wave. Children with ASD had a larger FRN difference wave than TD children to social stimuli, and TD children had a larger FRN difference wave than those with ASD to nonsocial stimuli, $F(1, 33.36) = 3.94$, $p = .055$. No pairwise comparisons were significant (all $ps > .05$). Note that we calculated an incorrect minus correct difference wave, and TD children demonstrated *larger* FRNs to correct

versus incorrect responses in the social condition while children with ASD had the opposite pattern.

Trial numbers for both groups are displayed in Table 2. Due to differences in trial numbers between groups in the nonsocial condition – $F(1, 40) = 7.42$, $p < .01$ for nonsocial correct, $F(1, 40) = 7.64$, $p < .01$ for nonsocial incorrect – we analyzed data from a subset of participants who were matched on number of trials (criteria for matching was within four trials). Ten children per group were successfully matched. Analysis of number of trials for each condition between groups of matched participants were all nonsignificant (all $ps > .1$). The previous condition by group by correct interaction was highly significant $F(1, 17.68) = 9.15$, $p = .007$.

To examine latency differences between groups and conditions, we used a $2(\text{Group}) \times 2(\text{Condition}) \times 3(\text{Electrode}) \times 2(\text{Correct})$ ANOVA to examine fractional peak latency. Fractional peak latency, defined as the point in the waveform where the

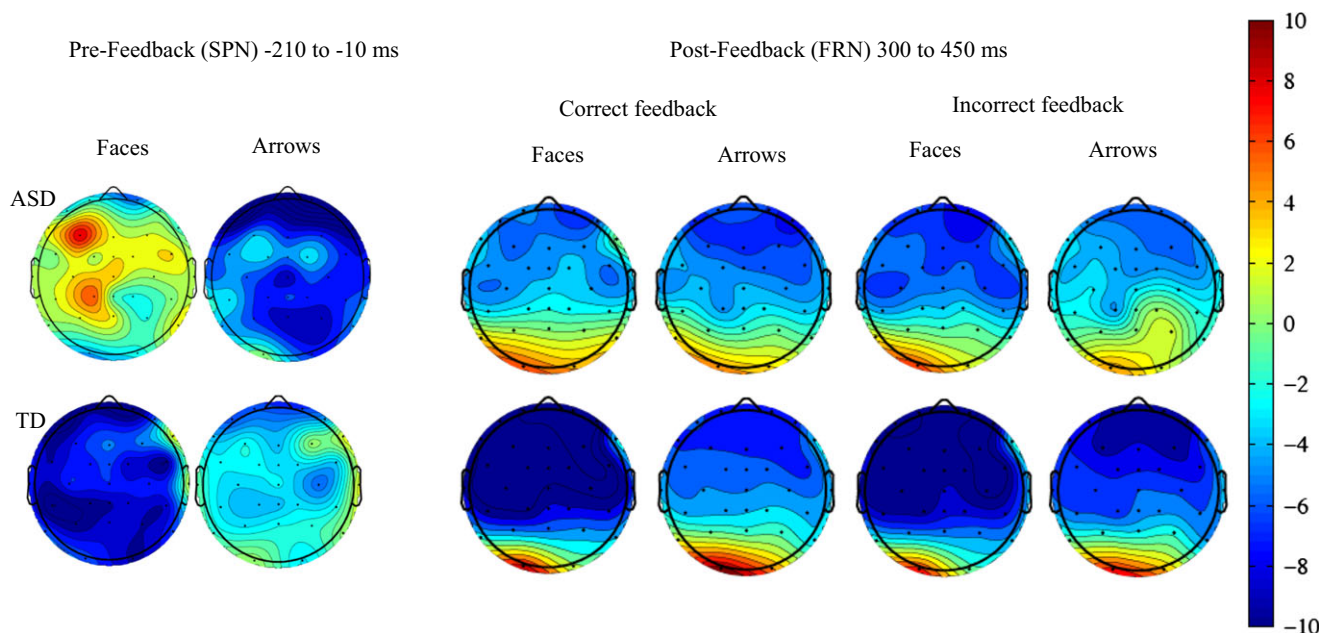


Figure 3 Topographic maps of mean amplitude between -210 and -10 ms (SPN), and 300 and 450 ms (FRN) for children with ASD (top), and TD children (bottom). Both the SPN and FRN are negative waveforms, thus darker (blue) areas indicate greater activation

area under the curve is 50% of the total, is thought to be the most rigorous measure of the timing of ERP activity, because it is less influenced by noise than latency to the peak (Luck, 2005; Woodman, 2010). Latency was measured between 300 – 450 ms for correct and incorrect trials separately. An interaction between condition and correct answers that approached significance was found, $F(1, 41.11) = 3.89$, $p = .055$, such that for both groups, the FRN was faster for faces versus arrows during incorrect feedback, but faster for arrows versus faces during correct feedback.

Discussion

This study investigated brain correlates of social versus nonsocial feedback on both reward anticipation and processing in young children with TD and ASD using a paradigm that controlled for both rewards and physical stimulus properties. The paradigm has general applicability in studies of TD children and the development of the reward system and its role in children's social-cognitive functioning. It also has applicability in other atypically developing populations, such as in children with Williams Syndrome, who may have abnormally high social motivation, and in whom learning is also affected. These results add significantly to our understanding of reward systems, in that previous investigations of social motivation in ASD have not controlled for tangibility of rewards between conditions.

SPN: Differences between social stimuli in TD children versus children with ASD

The current results extend our previous finding that TD children exhibit larger SPNs when anticipating

social versus nonsocial stimuli (Stavropoulos & Carver, 2013a) by showing that TD children have larger SPNs when anticipating social stimuli compared to children with ASD. Importantly, the results also suggest that children with ASD have anticipatory reward deficits for social stimuli, as opposed to global deficits in reward anticipation. No differences were observed between TD individuals and those with ASD in the *nonsocial* condition, suggesting that reward anticipation is blunted in ASD for social stimuli alone – anticipation for nonsocial stimuli is intact.

Our results are largely consistent with previous studies examining reward anticipation in this population (Groen et al., 2008; Kohls et al., 2011). One previous study utilized a probabilistic learning task with nonsocial stimuli, and found that children with ASD and ADHD showed equivalent SPN activations when anticipating negative feedback, but enhanced SPN when anticipating positive feedback (Groen et al., 2008). While it is important to note that our task differed from this previous investigation (because participants could not predict whether upcoming feedback would be positive or negative), we also found that TD children and those with ASD elicited a statistically equivalent SPN response to *nonsocial* feedback. Our results are consistent with findings by Kohls et al. (2011), who reported that children with ASD have an attenuated anticipatory P300 response to trials indicative of social rewards. Our results differ with regards to TD children, however, because we found that TD children elicited a larger SPN response to social versus nonsocial stimuli, whereas Kohls et al. (2011) found the opposite pattern. Our use of a tangible reward (goldfish crackers) for both social and nonsocial blocks may explain these differences. It is possible that both TD

Table 2 Descriptive statistics of trial numbers and amplitude for typically developing (TD) individuals in the top row, and those with autism spectrum disorder (ASD) on the bottom row for each condition separately

Group	FRN: Correct faces	FRN: Incorrect faces	FRN: Correct arrows	FRN: Incorrect arrows	SPN: Faces	SPN: Arrows
TD	Trials: $M = 27.56$ $SD = 6.65$ Amplitude: $M = -8.72$ $SD = 7.80$	Trials: $M = 26.26$ $SD = 6.60$ Amplitude: $M = -6.86$ $SD = 9.26$	Trials: $M = 27.56$ $SD = 7.20$ Amplitude: $M = -4.80$ $SD = 7.62$	Trials: $M = 28.56$ $SD = 6.42$ Amplitude: $M = -5.02$ $SD = 7.05$	Trials: $M = 47.54$ $SD = 12.90$ Amplitude: $M = -7.15$ $SD = 12.53$	Trials: $M = 45.26$ $SD = 16.57$ Amplitude: $M = -1.31$ $SD = 8.93$
ASD	Trials: $M = 27.36$ $SD = 4.98$ Amplitude: $M = -2.60$ $SD = 9.49$	Trials: $M = 25.21$ $SD = 6.43$ Amplitude: $M = -4.51$ $SD = 6.44$	Trials: $M = 21.68$ $SD = 6.65$ Amplitude: $M = -4.56$ $SD = 8.05$	Trials: $M = 22.68$ $SD = 7.35$ Amplitude: $M = -3.42$ $SD = 7.08$	Trials: $M = 41.55$ $SD = 14.73$ Amplitude: $M = -0.33$ $SD = 11.10$	Trials: $M = 31.75$ $SD = 15.75$ Amplitude: $M = -5.63$ $SD = 8.93$

children and those with ASD show more reward anticipation when rewards are tangible, such as in the monetary condition of Kohls et al. (2011). Previous research suggests that neural activity when viewing faces is changed under different motivational conditions (Skelly & Decety, 2012). It is therefore possible that our results differed from previous investigations because we utilized a design in which the faces and arrows were incidental, rather than the central focus of the task. Future studies should attempt to clarify this point by directly comparing tangible versus intangible rewards within both social and nonsocial conditions.

FRN: Social stimuli and feedback in TD children versus children with ASD

Our results show that TD children and those with ASD are differentially affected by correct versus incorrect feedback and that this interacts with social versus nonsocial stimulus type. In our study, TD children have a larger FRN response to correct feedback when viewing social stimuli, but a larger FRN response to incorrect feedback when viewing nonsocial stimuli. Children with ASD show the opposite pattern (i.e., larger FRN to correct feedback during the nonsocial condition, and larger FRN to incorrect feedback during the social condition). In contrast to previous research using the FRN in children with ASD versus TD children (Larson et al., 2011; McPartland et al., 2012), we did not find a main effect of feedback type such that incorrect feedback elicited a larger FRN versus correct feedback. We found that TD participants' responses were larger to correct feedback in the social condition, but larger to incorrect feedback in the nonsocial condition. Thus, in the nonsocial condition, our results with TD children are consistent with previous findings. Previous FRN literature did not utilize social versus nonsocial stimuli, and it is possible that our results in the social condition may be due to the highly salient nature of viewing faces for TD children.

In the ASD group, results during the social condition were consistent with previous investigations (i.e., larger FRN to incorrect vs. correct feedback), but results in the nonsocial condition differ from previous studies. If we use analogous logic as with TD children, results from children with ASD point to nonsocial stimuli (arrows in this study) being highly salient, because the FRN to *nonsocial* stimuli in the ASD group was largely analogous to the FRN to *social* stimuli in the TD group. The current results suggest that future research on the FRN in both TD children and those with ASD should investigate how social and nonsocial rewards affect reward outcome processing.

Our results suggest that both TD children and those with ASD elicit a faster FRN response to faces versus arrows during incorrect feedback, and faster FRN response to arrows versus faces during

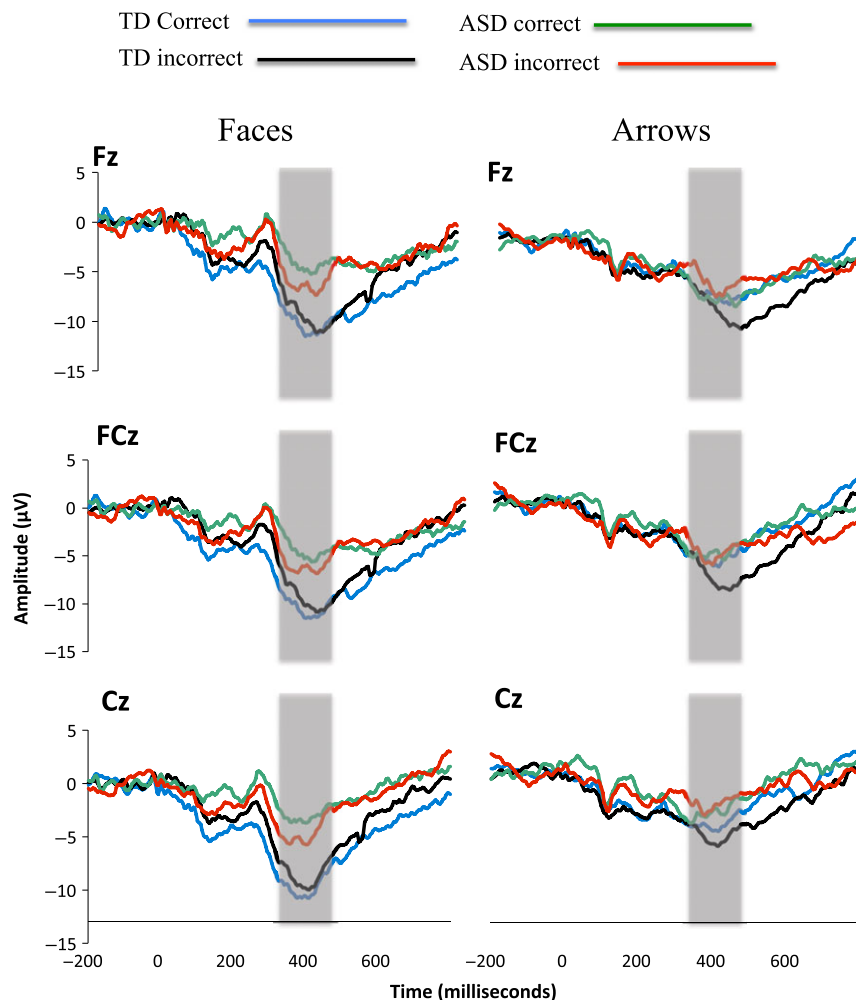


Figure 4 Grand averaged waveforms for TD children and those with ASD from the feedback related negativity (FRN) in response to social/faces (right) and nonsocial/arrows (left). The area between 300 and 450 ms, used for statistical analyses, is highlighted with a gray box

correct feedback. Our latency data indicate a later response than previous FRN studies (Larson et al., 2011; McPartland et al., 2012). This is likely explained by the fact that the current study utilized a younger population than previous studies, and younger children typically have longer latency ERP responses (Courchesne, 1978). In summary, the FRN results demonstrate that TD children are affected by correct versus incorrect feedback while viewing social versus nonsocial stimuli differently than those with ASD, which could point to higher salience of social stimuli for TD children (vs. nonsocial stimuli), and the opposite pattern obtains for children on the autism spectrum. Further research using the FRN may benefit by utilizing principle component analysis (PCA) in order to help tease apart the effects of viewing social stimuli versus nonsocial stimuli between groups.

Testing only high functioning children with ASD allowed us to match groups on IQ scores, however this means that the results cannot be immediately extrapolated to all individuals with ASD independent of severity, and because ASD is a developmental disorder, the current results cannot be extrapolated

to younger individuals on the spectrum. Adaptation of the current paradigm would allow us to test both lower functioning children with ASD and younger children.

Conclusions

We examined reward processing of social and nonsocial stimuli in children using a paradigm that can be widely employed to study both typical and atypical populations. Our results comparing typically developing children and children with autism provide evidence of a social reward anticipation impairment in children with ASD. Reward processing evidence suggests that TD children may find social stimuli more salient than nonsocial stimuli, whereas children with ASD demonstrate the opposite pattern. It is interesting to consider, then, whether children with ASD may have increased motivation for nonsocial stimuli at the expense of social stimuli, rather than only a deficit in social motivation. While our study was not designed to examine this directly, future studies should investigate this further. Using two components of the ERP, we showed differences

between typically developing children and children with autism in reward anticipation (via the SPN component of the ERP), and reward processing (via the FRN component of the ERP), finding that both reward anticipation and reward processing are impaired in ASD in response to social stimuli. These findings increase our understanding of the nature of the reward system's response to social stimuli in typically developing children and the nature of deficits seen in children with autism.

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Key points

- Children with autism spectrum disorder demonstrate selective deficits in reward anticipation and processing for social stimuli.
- Children with autism spectrum disorder process reward feedback differently than typically developing children for social versus nonsocial stimuli.

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