Psychiatry Research 187 (2011) 198–203

Contents lists available at ScienceDirect

Psychiatry Research

journal homepage: www.elsevier.com/locate/psychres

Feedback and reward processing in high-functioning autism

Michael J. Larson [a,b,*,1], Mikle South [a,b,1], Erin Krauskopf [a], Ann Clawson [a], Michael J. Crowley [c]

[a] Department of Psychology, Brigham Young University, Provo, UT 84602, United States
[b] Neuroscience Center, Brigham Young University, Provo, UT 84602, United States
[c] Yale Child Study Center, New Haven, CT 06520, United States

ARTICLE INFO

Article history:
Received 5 May 2010
Received in revised form 2 November 2010
Accepted 3 November 2010

Keywords:
Autism
Feedback-related negativity
fERN
Error-related negativity (ERN)
Event-related potential (ERP)
Reward
Feedback

ABSTRACT

Individuals with high-functioning autism often display deficits in social interactions and high-level cognitive functions. Such deficits may be influenced by poor ability to process feedback and rewards. The feedback-related negativity (FRN) is an event-related potential (ERP) that is more negative following losses than gains. We examined FRN amplitude in 25 individuals with Autism Spectrum Disorder (ASD) and 25 age- and IQ-matched typically developing control participants who completed a guessing task with monetary loss/gain feedback. Both groups demonstrated a robust FRN that was more negative to loss trials than gain trials; however, groups did not differ in FRN amplitude as a function of gain or loss trials. N1 and P300 amplitudes did not differentiate groups. FRN amplitude was positively correlated with age in individuals with ASD, but not measures of intelligence, anxiety, behavioral inhibition, or autism severity. Given previous findings of reduced-amplitude error-related negativity (ERN) in ASD, we propose that individuals with ASD may process external, concrete, feedback similar to typically developing individuals, but have difficulty with internal, more abstract, regulation of performance.

1. Introduction

Individuals with Autism Spectrum Disorders (ASD) frequently display social, cognitive, and behavioral deficits that result from the consequences of atypical brain development and altered interactions with the environment. Several studies of the cognitive associations of social dysfunction in individuals diagnosed with ASD report limitations in the ability to process social stimuli, feedback, and reward. For example, Dawson et al. (2001) found that poor performance in individuals with ASD on a delayed non-matching to sample task appeared to arise from a difficulty in forming abstract stimulus–reward associations rather than deficits in visual object recognition. Ingersoll et al. (2003) found that young children diagnosed with autism better imitated the use of toys that were associated with concrete sensory feedback than abstract social feedback. Such difficulties in feedback and reward processing are thought to be the result of dysfunction of the fronto-striatal reward system, which may place greater emphasis on cognitive rather than emotional aspects of feedback (Schmitz et al., 2008).

The neural underpinnings of feedback and reward processing can be measured using the feedback-related negativity (FRN) component of the scalp-recorded event-related potential (ERP). The FRN is a negative deflection in the ERP that occurs approximately 250 ms to 300 ms following the presentation of feedback and is more negative to losses or unexpected outcomes than gains or expected outcomes (e.g., Gehring and Willoughby, 2002; Holroyd et al., 2003; Hajcak et al., 2006). More broadly speaking, the FRN represents an electrophysiological reflection of whether a desired result has been achieved and may represent a mechanism for performance feedback-signaling for adjustments in behaviors when outcomes are not consistent with behaviors or expectations (Hajcak et al., 2006). Source localization studies of the FRN broadly implicate areas of the medial–frontal cortex, including the anterior cingulate cortex (ACC) as the neural generator (Gehring and Willoughby, 2002; Holroyd and Coles, 2002; Ruchsow et al., 2002; Nieuwenhuis et al., 2005), although additional areas such as the posterior cingulate, superior frontal gyrus, fusiform gyrus, and superior temporal gyrus have also been identified (e.g., van Veen et al., 2004; De Pascalis et al., 2010).

The FRN may be an important reflection of discrepancies between predicted and actual reward in the mesencephalic dopamine system. Dopaminergic activity in neurons connecting the basal ganglia and the ACC use reinforcement signals to promote feedback-based learning by processing negative events and determining suitable behavior for the given situation (Holroyd et al., 2003; Fein and Chang, 2008; Crowley et al., 2009). Disruption of the dopaminergic metabolism system involving the ACC, basal ganglia and prefrontal cortex may likewise contribute to behavioral deficits in ASD, by interfering with the ability to respond effectively to reward and punishment (Kendrick, 2004). Dopamine plays an important role in reward, sending error signals through neurons in the mesencephalic dopamine system to the ACC.

* Corresponding author. Department of Psychology, Brigham Young University, 244 TLRB, Provo, UT 84602, United States. Tel.: +1 801 422 6125; fax: +1 801 422 0163.
E-mail address: michael_larson@byu.edu (M.J. Larson).
[1] These authors contributed equally and are considered co-first authors.

0165-1781/$ – see front matter © 2010 Elsevier Ireland Ltd. All rights reserved.
doi:10.1016/j.psychres.2010.11.006

These neurons aid in predicting the discrepancy between predicted and actual reward and are important in systems that code error and determine behavior (Holroyd et al., 2004; Crowley et al., 2009).

There is only one study published to date that relates to ASD and ERP components associated with feedback (Groen et al., 2008). Their study of a small sample ($n=17$) of children diagnosed with subthreshold ASD did not show a typical FRN in individuals with ASD, individuals with attention-deficit hyperactivity disorder (ADHD), or typically developing (TD) children. However, they did find that ERPs associated with feedback anticipation and early feedback processing (e.g., the P2a waveform) did not reliably differ between TD children and those with subthreshold ASD. Groups did differ as a function of feedback valence on late P300 potentials associated with feedback processing. These differences possibly reflect an inability to integrate external error information into performance feedback (Groen et al., 2008). In addition, children with ASD showed greater anticipation for positive feedback throughout the task, in contrast to TD children. Overall, Groen et al. suggest that children with ASD may place greater significance on positive than negative feedback stimuli (see also Wilbarger et al., 2009); however, it is difficult to generalize from the results of this study as the FRN anticipated in their feedback task was not found and the sample consisted of individuals with subthreshold autism.

Several recent studies, including one conducted in our lab with an overlapping sample to those presented here, show reduced response amplitude on the error-related negativity (ERN) component of the ERP in individuals with ASD relative to controls (Vlamings et al., 2008; Sokhadze et al., 2010; South et al., 2010; Santesso et al., in press). The ERN is an internal signal of error commission or conflict detection primarily found following errors on forced-choice reaction time tasks (see van Veen and Carter, 2006, for review). The ERN is reliably associated with emotional traits and processes, such as negative affect (Luu et al., 2000), anxiety and depression (Olvet and Hajcak, 2008), and even satisfaction with life (Larson et al., 2010). The FRN, on the other hand, is a reflection of more concrete processes and is based on external feedback that influences reinforcement-based learning and performance (Nieuwenhuis et al., 2004).

Prominent theories of the ERN and FRN suggest that these components both represent a reinforcement learning response to performance or feedback supported by the same mechanisms in the ACC (Holroyd and Coles, 2002). Studies directly comparing the two components, however, show mixed results. One study shows the ERN and FRN share a neural generator in the ACC, with an additional generator in the prefrontal cortex (Potts et al., 2011) and others showing nearly identical neural processes and generators (Heldmann et al., 2008; Gentsch et al., 2009). Regardless, studies suggest that these components are temporally dissociable, with one representing a response to concrete external feedback and the other representing an internally generated signal of a failure to reach expected outcomes (Gentsch et al., 2009).

The purpose of the present study was to examine the neural response to reward, as represented by the FRN, in individuals with ASD and typically developing controls. Our study adds to the previous study of the FRN in ASD conducted by Groen et al. (2008) by: a) using a task specifically intended to measure the FRN as a function of gains and losses; and, b) using a sample that meets stricter guidelines for ASD. Given performance monitoring and feedback decrements in individuals with autism, we hypothesized that individuals with ASD would show decreased-amplitude FRN values relative to healthy control participants.

2. Method

2.1. Participants

All procedures were approved by the Institutional Review Board at Brigham Young University. Initial study enrollment included 26 individuals with ASD and 25 typically developing control participants. Data from one outlier participant with ASD were excluded due to means more than two standard deviations below the group averages for the FRN. The final sample, therefore, included 25 individuals with ASD between the ages of 9 and 21 years ($M=13.89$ years, S.D. = 2.46; two female) and 25 age- and intelligence -matched typically developing control participants ($M=14.07$ years, S.D. = 2.69; range = 8 to 18 years; one female). Demographic and diagnostic information are presented in Table 1. Exclusion criteria included previous or suspected diagnoses of Attention Deficit Hyperactivity Disorder, learning disability, head injury or concussion, or any other psychological or developmental problems (e.g., diagnosed depression or anxiety disorder).

Diagnosis of an ASD was established by a licensed clinical psychologist (MS) trained to research reliability on the Autism Diagnostic Observation Schedules-Generic (ADOS-G; Lord et al., 2000). Individuals with ASD scored above the recommended cut-off of 7 on the ADOS-G and were above the ASD cut-off score of 12 recommended by Corsello et al. (2007) on the parent-report Social Communication Questionnaire. As shown in Table 1, individuals with ASD were reported to have significantly more anxiety symptoms on the parent-report SCreen for Anxiety and Related Disorders (SCARED; Birmaher et al., 1999), and signs of behavioral inhibition, as reported on the parent-report version of the Behavioral Inhibition Scales/Behavioral Activation Scales (BIS/BAS; Blair et al., 2004). Groups did not differ on the behavioral activation scale of the BIS/BAS.

2.2. Experimental task

We utilized a modified version of the balloon gain context task originally used by Holroyd et al. (2003) in their investigation of the FRN and subsequently adapted by Crowley et al. (2009) for use in children. The task presented individuals with four balloons, each of a different color (red, blue, orange, or green), that randomly appeared in one of four positions centered in a row on the screen. Participants were instructed that they would start the task with no coins, but that one of the balloons on each trial contained a coin that was associated with a gain of 25 cents; however, if they did not choose the correct balloon they would lose 25 cents. Unbeknownst to the participants, the loss and gain feedback was actually presented randomly. That is, there was no pattern as to whether loss or gain feedback would be presented. For each trial, participants had a 50% chance the trial would be a loss and 50% chance the trial would be a gain. Balloons remained on the screen until participant response; feedback appeared 1000 ms following balloon selection and remained on the screen for 800 ms followed by a 700 ms inter-trial interval. Two blocks of 72 trials were presented for a total of 144 trials. At the beginning of each block 10 to 12 consecutive "gain" trials were presented to ensure participants were expecting gains and to avoid frustration at the beginning of each block. After completing the task, participants were debriefed, and all were provided with the same amount of compensation.

2.3. Electrophysiological data recording and reduction

Electroencephalogram (EEG) data were recorded from 128 scalp sites using a geodesic sensor net and Electrical Geodesics, Inc. (EGI; Eugene, OR) amplifier system (20 K nominal gain, bandpass = 0.10–100 Hz). Electroencephalogram was initially referenced to the vertex electrode (Cz) and digitized continuously at 250 Hz with a 24-bit analog-to-digital converter. Impedances were maintained below 50 kΩ. Data were re-referenced to the average reference off-line and digitally low-pass filtered at 30 Hz. Eye movement and blink artifacts were corrected using the Gratton et al. (1983) algorithm.

Following Crowley et al. (2009), individual-subject feedback-locked ERPs were derived separately for gain and loss trials from 100 ms prior to the feedback stimulus and 600 ms following the feedback stimulus and were baseline corrected using the 100 ms pre-feedback window. We generally followed the methodology outlined by Holroyd and Krigolson (2007) by calculating a difference for the most negative peak between gain feedback and loss feedback and estimating polynomial functions that

Table 1
Mean and standard deviation diagnostic and demographic data.

	ASD ($n=25$)		Control ($n=25$)	
	Mean (S.D.)	Range	Mean (S.D.)	Range
ADOS-G total score	12.44 (3.94)	7–20	–	–
Social communication questionnaire	21.45 (5.01)	13–29	–	–
Full scale IQ score	109.64 (12.69)	87–132	110.32 (14.02)	75–138
Verbal IQ score	107.32 (12.70)	88–133	110.28 (16.82)	70–141
Performance IQ score	109.77 (15.50)	89–139	107.80 (10.37)	88–128
SCARED parent-rating*	22.54 (14.12)	1–53	9.24 (5.96)	0–26
BIS parent-rating score*	22.63 (3.73)	13–28	19.83 (2.90)	14–24
BAS parent-rating score	37.58 (7.90)	13–52	40.05 (4.27)	33–49

Note: ADOS-G = Autism Diagnostic Observation Schedules-Generic; IQ = Intelligence Quotient; SCARED = SCreen for Anxiety and Related Disorders; BIS = Behavioral Inhibition Scales; BAS = Behavioral Activation Scales.

* Groups significantly differed at $p<0.01$.

best fit the distribution along the midline (electrodes Fz, FCz, Cz, and Pz). We examined FRN amplitude in two different ways. First, given several studies that indicate FRN amplitude can be confounded by overlap with the P300 (e.g., Holroyd et al., 2003; Holroyd et al., 2004), we calculated difference waves subtracting gain feedback from loss feedback. The FRN was subsequently quantified as the most negative amplitude and latency of the difference wave between 125 ms and 350 ms post-feedback presentation. Second, based on the possibility that differences in waveform morphology between individuals with ASD and controls could spuriously influence the difference waves (e.g., differences in peak latency or overall waveform morphology), we used the peak-to-peak scoring method originally proposed by Holroyd et al. (2003) for FRN analysis. Specifically, FRN peak-to-peak amplitude was calculated as the difference between the maximum amplitude value between 125 ms and 350 ms following feedback onset and the most negative amplitude point between this maximum and 350 ms post-feedback presentation.

To determine if there was a generalized decrement in ERPs for the individuals with ASD, we also examined N1 and P300 amplitudes. N1 amplitude was calculated as the amplitude of the amplitude of the first negative peak between 50 ms and 200 ms at electrode site Oz (location of maximum N1 amplitude). P300 amplitude was calculated as the most positive peak in between 300 ms and 600 ms following the presentation of the feedback stimulus at electrode site Pz.

2.4. Data analysis

Mean response times (RT) for gain and loss trials were first calculated to ensure differences in behavioral performance were not present. Planned contrasts were used to compare RTs, number of ERP trials, and difference wave values between the individuals with ASD and controls. Subsequent ERP data were analyzed using separate repeated measures analyses of variance (ANOVA). The Huynh–Feldt epsilon adjustment was applied to adjust for possible violations of sphericity and *partial-eta*^{2} (η^2) reported as a measure of effect size. Initial 2×2 ANOVAs included the factors group (autism, control) as the between-subjects factor and feedback (gain, loss) as the within-subjects factor. Cohen's-*d* effect sizes are presented for between-subjects comparisons. Zero-order correlations were used to examine the relationship between FRN amplitudes and indices of autism severity, intelligence, anxiety, and behavioral inhibition/activation.

3. Results

3.1. Response times

As expected, mean RTs did not differ between individuals with ASD and controls on loss trials, $t(48)=0.25$, $p=0.80$, $d=0.07$, or gain trials, $t(48)=0.16$, $p=0.88$, $d=0.04$, indicating groups did not respond more impulsively or differentially to loss or gain trials. Participants with ASD had a mean (±S.D.) RT of 1419.40 (604.49) for loss trials and 1503.18 (627.76) for gain trials; controls had a mean RT of 1461.43 (577.92) for loss trials and 1529.81 (575.45) for gain trials.

3.2. Event-related potential data

Similar to the RT data, individuals with ASD and controls did not differ on number of trials retained for ERP averages for either loss, $t(48)=1.46$, $p=0.15$, $d=0.36$, or gain averages, $t(48)=1.29$, $p=0.21$, $d=0.41$. For participants with ASD, loss averages contained 59.12 (12.55; range = 37 to 71) trials and gain averages contained 59.60 (10.61; range = 25 to 72) trials. For control participants, loss averages contained 53.44 (18.25; range = 13 to 72) trials and gain averages contained 53.24 (19.13; range = 13 to 72) trials. Grand average ERP waveforms and scalp voltage maps for gain and loss conditions are presented in Fig. 1.

3.3. N1 analyses

To confirm that findings were not due to sensory differences between conditions or groups, we conducted a Group×Feedback ANOVA on N1 amplitude. The ANOVA showed no significant main effect of feedback, $F(1,48)=1.23$, $p=0.27$, $\eta^2=0.03$, no significant main effect of group, $F(1,48)=1.62$, $p=0.21$, $\eta^2=0.03$, and no significant Group×Feedback interaction, $F(1,48)=0.04$, $p=0.85$, $\eta^2=0.001$.

3.4. FRN analyses

The scalp distribution of the loss minus gain difference wave collapsed across groups showed the most negative amplitude at fronto-central electrode site Fz (−3.62 ± 3.19 μV), followed by FCz (−3.33 ± 2.42 μV), Cz (−1.56 ± 1.83 μV), and Pz (−1.47 ± 3.17 μV). Polynomial contrasts confirmed this distribution with a significant linear trend, $F(1,49)=13.25$, $p<0.001$, and a non-significant quadratic trend, $F(1,49)=0.12$, $p=0.74$. Given the current scalp distribution and previous studies of the FRN (e.g., Holroyd et al., 2003; Holroyd and Krigolson, 2007; Larson et al., 2007) we conducted all analyses of the FRN at electrode site Fz. Loss minus gain difference waves as a function of group are presented in Fig. 2. Mean (±S.D.) amplitudes of the FRN as a function of group and analysis type are presented in Table 2.

3.4.1. FRN difference wave analysis

Feedback-locked loss-minus-gain difference waves showed an FRN occurring at a mean latency of 309.12 ms for participants with ASD and 304.16 ms for typically developing controls. Comparisons of the difference waves revealed no differences in FRN amplitude, $t(48)=0.60$, $p=0.55$, $d=0.13$, or latency, $t(48)=0.44$, $p=0.66$, $d=0.17$, between groups.

3.4.2. FRN peak-to-peak analysis

A Group×Feedback ANOVA on gain and loss peak-to-peak amplitudes yielded a significant main effect of feedback, $F(1,48)=14.37$, $p<0.001$, $\eta^2=0.23$, with loss trials having a greater peak-to-peak difference than gain trials. The main effect of group was also statistically significant, $F(1,48)=4.91$, $p=0.03$, $\eta^2=0.09$, indicating that individuals with ASD had generally lower peak-to-peak values relative to controls. Most important to the current study, the Group×Feedback interaction was not significant, $F(1,48)=1.18$, $p=0.28$, $\eta^2=0.02$, indicating the groups did not differentially respond to gain or loss feedback.

3.4.3. Power to detect differences

The use of post-hoc power calculations on the analyzed dataset is generally thought to be inappropriate (e.g., Hoenig and Heisey, 2001). Thus, we utilized data from two previous studies of the FRN to determine if our statistical power was adequate. Crowley et al. (2009) used the same balloon task employed in this study with children exposed to drugs prenatally and found a statistically significant mean difference of 2.72 μV (Cohen's-*d* effect size = 1.53) on the loss minus gain difference between high and low risk males. In another study (Larson et al., 2007), the mean loss minus gain difference between individuals with traumatic brain injury (TBI) and healthy controls was 1.75 μV (Cohen's-*d* effect size = 1.52). Using the more conservative difference of 1.75 μV and an alpha level of .05, our sample size of 25 individuals per group would have high power (>99%) to detect group differences if present. Thus, our findings cannot be attributed to insufficient statistical power and examination of the difference wave (Fig. 2) shows very little difference between groups.

3.5. P300 analysis

For the scalp distribution of the P300, the loss minus gain difference collapsed across groups was largest at site Pz (4.58 ± 3.75 μV) and smallest at site Fz (2.66 ± 3.38 μV). The polynomial trend analysis showed a significant linear trend, $F(1,49)=6.23$, $p=0.02$, and a non-significant quadratic trend, $F(1,49)=0.67$, $p=0.42$, consistent with the posterior scalp distribution of the P300. Thus, the P300 was measured at site Pz. A Group×Feedback ANOVA on gain and loss P300 amplitudes was consistent with the FRN analyses and revealed a significant main effect of feedback, $F(1,48)=11.96$, $p<0.001$, $\eta^2=0.20$; loss trials had increased P300

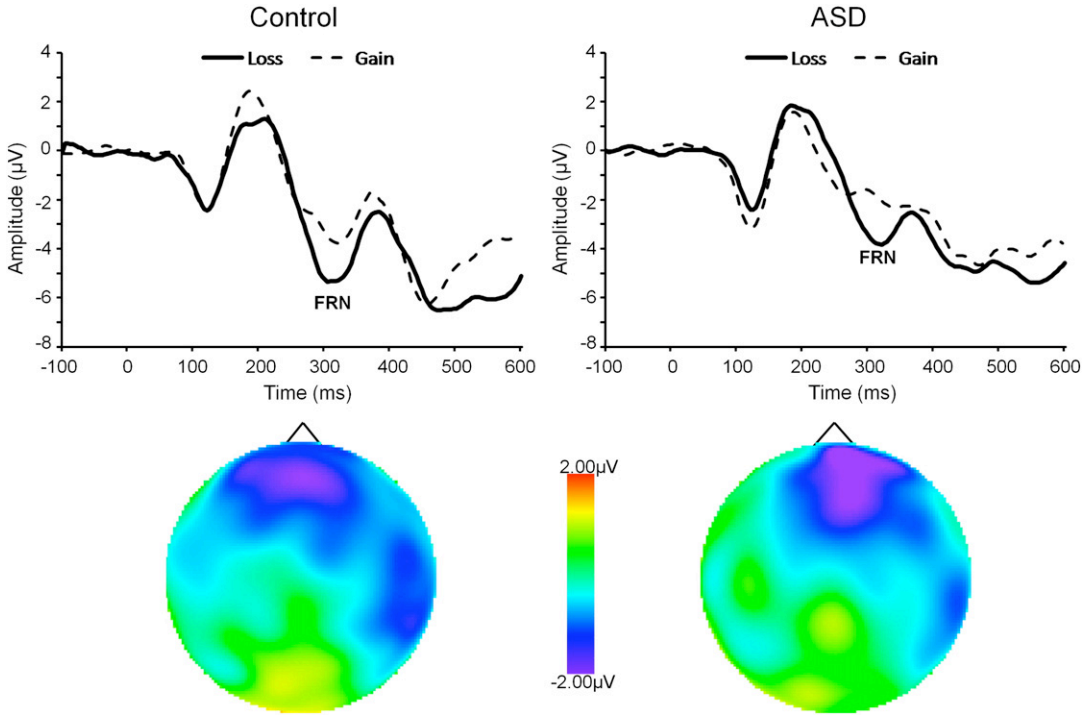

Fig. 1. Grand average ERP waveforms depicting feedback-locked gain and loss activity at recording site Fz and the voltage maps of the loss-minus-gain difference at 304 ms.

amplitude relative to gain trials. The main effect of group was not statistically significant, $F(1,48)=0.02$, $p=0.89$, $\eta^2=0.001$. Similarly, the Group×Feedback interaction was not significant, $F(1,48)=0.95$, $p=0.34$, $\eta^2=0.02$, indicating that P300 amplitude did not differ in response to gain or loss feedback as a function of group.

3.6. Correlations

We used the FRN difference wave to examine potential relationships between FRN amplitude, intelligence, anxiety, BIS/BAS score, autism severity, and age. With the exception of age, there were no significant correlations between FRN difference amplitude and the aforementioned measures for controls, $rs<0.25$, $ps>0.24$, for participants with ASD, $rs<0.31$, $ps>0.14$, or when the groups were combined, $rs<0.19$, $ps>0.19$. There was a significant positive relationship between FRN amplitude and age when data were collapsed across groups, $r=0.40$, $p=0.004$. When broken down by groups, the correlation was significant for the individuals with ASD, $r=0.61$, $p=0.001$, but not the control participants, $r=0.18$, $p=0.39$. That is, the positive correlation in the individuals with ASD indicates that increased age is associated with more positive (i.e., less negative) FRN amplitude.

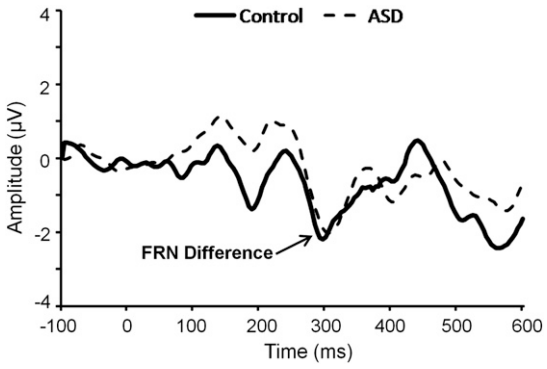

Fig. 2. Grand average ERP loss-minus-gain difference wave for control and ASD participants.

4. Discussion

We did not find significant differences in FRN amplitude between our sample of 25 individuals diagnosed with ASD compared to 25 age- and IQ-matched typically developing controls. Our results generally replicate the only previously published study of the FRN in ASD conducted by Groen et al. (2008), although our sample was diagnostically more severe and our task was utilized more specifically to elicit and analyze the FRN component of the ERP. That is, neither study found consistent group differences on electrophysiological indices of feedback processing. Importantly, N1 and P300 amplitudes also did not differ between groups, indicating that there was a general similarity in the electrophysiological processing of gain and reward feedback between groups.

Based on the current findings and those of several studies indicating that individuals with ASD show reduced ERN amplitudes relative to TD controls (Vlamings et al., 2008; Sokhadze et al., 2010; South et al., 2010; Santesso et al., in press), we propose that the *source* rather than the *valence* of feedback is critical. That is, an important area of cognitive difficulty in autism—across many different types of information processing tasks—is the integration of abstract clues or knowledge (see Ropar and Peebles, 2007; Allen, 2009; Gastgeb et al., 2009), including the formation of internal representations (Kennedy

Table 2
Mean (±S.D.) component amplitude (μV) at electrode Fz.

	ASD	Control
	Amplitude (μV)	
Difference wave	3.41 (3.58)	3.82 (2.81)
Peak-to-peak		
Gain	−6.09 (1.95)	−8.45 (3.09)
Loss	−8.22 (4.35)	−9.63 (3.63)

and Courchesne, 2008). Both children and adults diagnosed with ASD respond better to concrete than abstract stimuli (Garretson et al., 1990; Dawson et al., 2001; Ingersoll et al., 2003). Indeed, many treatment programs for ASD emphasize the provision of concrete feedback mechanisms including the use of schedules and routines and the benefits of explicit, step-by-step instruction and correction, whether the instruction is written, verbal, or picture-based (e.g., Klin and Volkmar, 1995). The amplitude of the FRN, therefore, may provide a neural indication of rapid and generally accurate response to external, concrete feedback in ASD, whereas the reduced-amplitude ERN found in previous studies represents a more abstract response to internally generated errors.

As noted above, the ERN and FRN share underlying neural mechanisms and processes. Thus, the divergence of findings between studies showing decreased ERN amplitudes in ASD and intact FRN amplitudes is interesting. However, as a recent study indicates, the FRN may require additional processes not involved with internal ERN generation (Potts et al., 2011). The sample in the current study overlaps considerably with our recent study showing decreased-amplitude ERN in individuals with ASD (South et al., 2010). Thus, it is not likely that sampling differences led to the current findings. Future studies directly comparing error and feedback processing in ASD, however, are warranted.

Correlational analyses revealed a positive association between age and FRN loss minus gain difference in the individuals with ASD, but not control participants. The finding of increased response to negative feedback in younger individuals is not surprising given previous fMRI and ERP research showing increased neural responses to negative feedback in younger children compared to older children and adults (e.g., van Leijenhorst et al., 2006; Eppinger et al., 2009). This increased neural response has been interpreted to represent an increased sensitivity and reaction to immediate losses (van Leijenhorst et al., 2006; Crone and Van Der Molen, 2007; Carlson et al., 2009). Moreover, a study of individuals with ASD also shows increased neural response, and specifically ACC response, to feedback stimuli (Schmitz et al., 2008). Taken together, it is possible that the younger individuals with ASD showed an increased response to negative feedback relative to older individuals with ASD. Future research directly examining the interaction between age and feedback processing in ASD is warranted.

One of the greatest challenges for research in ASD is the heterogeneity of symptom expression that likely reflects heterogeneity in both etiology and potential response to intervention (Amaral et al., 2008; South et al., 2008a). Rapid improvements in the technology for measuring neural response *in vivo* offer new opportunities to model variation in brain activity across individuals and to link this with behavioral symptoms. We suggest, for example, that characterization of performance on ERN-related versus FRN-related tasks may provide useful profiles that cut across indistinct categorical diagnostic boundaries (such as ASD and ADHD) and also within broad diagnostic entities (such as variation within the autism spectrum) (Glahn et al., 2007; Groen et al., 2008; Hajcak et al., 2010; South et al., 2010). Understanding of the fundamental genetic and neural processes that underlie autism will require ongoing comparison of those abilities that seem to be intact or even enhanced, with those that are not (South et al., 2008b; Soulieres et al., 2009). Thus there is an important foundation for future research on task-specific and sample-specific aspects of the FRN, explicitly in context of similar ongoing research on the ERN and other ERP components that examine the influence and overlap of emotional and cognitive information processing.

Acknowledgements

We gratefully acknowledge the assistance of Oliver Johnston and Peter Clayson in data collection. This study was supported by funds from the Brigham Young University College of Family, Home, and Social Sciences.

References

Allen, M., 2009. Decoding representations: how children with autism understand drawings. Journal of Autism and Developmental Disorders 39, 539–543.
Amaral, D., Schumann, C., Nordahl, C., 2008. Neuroanatomy of autism. Trends in Neurosciences 31, 137–145.
Birmaher, B., Brent, D., Chiapetta, L., Bridge, J., Monga, S., Baugher, M., 1999. Psychometric properties of the Screen for Child Anxiety Related Emotional Disorders (SCARED): a replication study. Journal of the American Academy of Child and Adolescent Psychiatry 38, 1230–1236.
Blair, C., Peters, R., Granger, D., 2004. Physiological and neuropsychological correlates of approach/withdrawal tendencies in preschool: further examination of the behavioral inhibition system/behavioral activation system scales for young children. Developmental Psychobiology 45, 113–124.
Carlson, S.M., Zayas, V., Guthormsen, A., 2009. Neural correlates of decision making on a gambling task. Child Development 80, 1076–1096.
Corsello, C., Hus, V., Pickles, A., Risi, S., Cook, E., Leventhal, B., Lord, C., 2007. Between a ROC and a hard place: decision making and making decisions using the SCQ. Journal of Child Psychology and Psychiatry 48, 932–940.
Crone, E.A., Van Der Molen, M.W., 2007. Development of decision making in school-aged children and adolescents: evidence from heart rate and skin conductance analysis. Child Development 78, 1288–1301.
Crowley, M.J., Wu, J., Crutcher, C., Bailey, C.A., Lejuez, C.W., Mayes, L.C., 2009. Risk-taking and the feedback negativity response to loss among at-risk adolescents. Developmental Neuroscience 31, 137–148.
Dawson, G., Osterling, J., Rinaldi, J., Carver, L., McPartland, J., 2001. Recognition memory and stimulus-reward associations: indirect support for the role of ventromedial prefrontal cortex in autism. Journal of Autism and Developmental Disorders 31, 337–341.
De Pascalis, V., Varriale, V., D'Antuono, L., 2010. Event-related components of the punishment and reward sensitivity. Clinical Neurophysiology 121, 60–76.
Eppinger, B., Mock, B., Kray, J., 2009. Developmental differences in learning and error processing: evidence from ERPs. Psychophysiology 46, 1043–1053.
Fein, G., Chang, M., 2008. Smaller feedback ERN amplitudes during the BART are associated with greater family history density of alcohol problems in treatment naive alcoholics. Drug and Alcohol Dependence 92, 141–148.
Garretson, H., Fein, D., Waterhouse, L., 1990. Sustained attention in children with autism. Journal of Autism and Developmental Disorders 20, 101–114.
Gastgeb, H., Rump, K., Best, C., Minshew, N., Strauss, M., 2009. Prototype formation in autism: can individuals with autism abstract facial prototypes. Autism Research 2, 279–284.
Gehring, W.J., Willoughby, A.R., 2002. The medial frontal cortex and the rapid processing of monetary gains and losses. Science 295, 2279–2282.
Gentsch, A., Ullsperger, P., Ullsperger, M., 2009. Dissociable medial frontal negativities from a common monitoring system for self- and externally caused failure of goal achievement. NeuroImage 47, 2023–2030.
Glahn, D., Thompson, P., Blangero, J., 2007. Neuroimaging endophenotypes: strategies for finding genes influencing brain structure and function. Human Brain Mapping 28, 488–501.
Gratton, G., Coles, M.G., Donchin, E., 1983. A new method for off-line removal of ocular artifact. Electroencephalography and Clinical Neurophysiology 55, 468–484.
Groen, Y., Wijers, A.A., Mulder, L., Waggeveld, B., Minderaa, R.B., Althaus, M., 2008. Error and feedback processing in children with ADHD and children with autistic spectrum disorder: a EEG event-related potential study. Clinical Neurophysiology 119, 2476–2493.
Hajcak, G., Moser, J.S., Holroyd, C.B., Simons, R.F., 2006. The feedback-related negativity reflects the binary evaluation of good versus bad outcomes. Biological Psychology 71, 148–154.
Hajcak, G., MacNamara, A., Olvet, D.M., 2010. Event-related potentials, emotion, and emotion regulation: an integrative review. Developmental Neuropsychology 35, 129–155.
Heldmann, M., Russeler, J., Munte, T.F., 2008. Internal and external information in error processing. BMC Neuroscience 25, 33.
Hoenig, J.M., Heisey, D.M., 2001. The abuse of power: the pervasive fallacy of power calculations for data analysis. The American Statistician 55, 19–24.
Holroyd, C.B., Coles, M.G., 2002. The neural basis of human error processing: reinforcement learning, dopamine, and the error-related negativity. Psychological Reviews 109, 679–709.
Holroyd, C.B., Krigolson, O.E., 2007. Reward prediction error signals associated with a modified time estimation task. Psychophysiology 44, 913–917.
Holroyd, C.B., Nieuwenhuis, S., Yeung, N., Cohen, J.D., 2003. Errors in reward prediction are reflected in the event-related brain potential. Neuroreport 14, 2481–2484.
Holroyd, C.B., Larsen, J.T., Cohen, J.D., 2004. Context dependence of the event-related brain potential associated with reward and punishment. Psychophysiology 41, 245–253.
Ingersoll, B., Schreibman, L., Tran, Q., 2003. Effect of sensory feedback on immediate object imitation in children with autism. Journal of Autism and Developmental Disorders 33, 673–683.
Kendrick, K.M., 2004. The neurobiology of social bonds. Journal of Neuroendocrinology 16, 1007–1008.
Kennedy, D., Courchesne, E., 2008. Functional abnormalities of the default network during self- and other-reflection in autism. Social, Cognitive, and Affective Neuroscience 3, 177–190.
Klin, A., Volkmar, F.R., 1995. Asperger's Syndrome: Guidelines for Treatment and Intervention. Learning Disabilities Association of America, New Haven, Connecticut.

Larson, M.J., Kelly, K.G., Stigge-Kaufman, D.A., Schmalfuss, I.M., Perlstein, W.M., 2007. Reward context sensitivity impairment following severe TBI: an event-related potential investigation. Journal of the International Neuropsychological Society 13, 615–625.

Larson, M.J., Good, D.A., Fair, J.E., 2010. The relationship between performance monitoring, satisfaction with life, and positive personality traits. Biological Psychology 83, 222–228.

Lord, C., Risi, S., Lambrecht, L., Cook, E., Leventhal, B., DiLavore, P.C., Pickles, A., Rutter, M., 2000. The autism diagnostic observation schedule-generic: a standard measure of social and communication deficits associated with the spectrum of autism. Journal of Autism and Developmental Disorders 30, 205–223.

Luu, P., Collins, P., Tucker, D.M., 2000. Mood, personality, and self-monitoring: negative affect and emotionality in relation to frontal lobe mechanisms of error monitoring. Journal of Experimental Psychology: General 129, 43–60.

Nieuwenhuis, S., Holroyd, C.B., Mol, N., Coles, M.G., 2004. Reinforcement-related brain potentials from medial frontal cortex: origins and functional significance. Neuroscience and Biobehavioral Reviews 28, 441–448.

Nieuwenhuis, S., Slagter, H.A., von Geusau, H.J., Heslenfeld, D.J., Holroyd, C.B., 2005. Knowing good from bad: differential activation of human cortical areas by positive and negative outcomes. European Journal of Neuroscience 21, 3161–3168.

Olvet, D.M., Hajcak, G., 2008. The error-related negativity (ERN) and psychopathology: toward an endophenotype. Clinical Psychology Review 28, 1343–1354.

Potts, G.F., Martin, L.E., Kamp, S.M., Donchin, E., 2011. Neural response to action and reward prediction errors: Comparing the error-related negativity to behavioral errors and the feedback-related negativity to reward prediction violations. Psychophysiology 48, 218–228.

Ropar, D., Peebles, D., 2007. Sorting preference in children with autism: the dominance of concrete features. Journal of Autism and Developmental Disorders 37, 270–280.

Ruchsow, M., Grothe, J., Spitzer, M., Kiefer, M., 2002. Human anterior cingulate cortex is activated by negative feedback: evidence from event-related potentials in a guessing task. Neuroscience Letters 14, 203–206.

Santesso, D.L., Drmic, I.E., Jetha, M.K., Bryson, S.E., Goldberg, J.O., Hall, G.B., Mathewson, K.J., Segalowitz, S.J., Schmidt, L.A., 2011. An event-related source localization study of response monitoring and social impairments in autism spectrum disorder. Psychophysiology 48, 241–251.

Schmitz, N., Rubia, K., van Amelsvoort, T., Daly, E., Smith, A., Murphy, D.G., 2008. Neural correlates of reward in autism. British Journal of Psychiatry 192, 19–24.

Sokhadze, E., Baruth, J., El-Baz, A., Horrell, T., Sokhadze, G., Carroll, T., Tasman, A., Sears, L., Casanova, M.F., 2010. Impaired error monitoring and correction function in autism. Journal of Neurotherapy 14, 79–95.

Soulieres, I., Dawon, M., Samson, F., Barbeau, E., Sahyoun, C., Strangman, G., Zeffiro, T.A., Mottron, L., 2009. Enhanced visual processing contributes to matrix reasoning in autism. Human Brain Mapping 30, 4082–4107.

South, M., Ozonoff, S., Suchy, Y., Kesner, R.P., McMahon, W.M., Lainhart, J.E., 2008a. Intact emotion facilitation for nonsocial stimuli in autism: is amygdala impairment in autism specific for social information? Journal of the International Neuropsychological Society 14, 42–54.

South, M., Ozonoff, S., Schultz, R.T., 2008b. Neurocognitive development in autism. In: Nelson, C.A., Luciana, M. (Eds.), Handbook of Developmental Cognitive Neuroscience. MIT Press, Cambridge, MA.

South, M., Larson, M.J., Krauskopf, E., Clawson, A., 2010. Error processing in high-functioning autism spectrum disorders. Biological Psychology 85, 242–251.

van Leijenhorst, L., Crone, E.A., Bunge, S.A., 2006. Neural correlates of developmental differences in risk estimation and feedback processing. Neuropsychologia 44, 2158–2170.

van Veen, V., Carter, C.S., 2006. Error detection, correction, and prevention in the brain: a brief review of data and theories. Clinical EEG and Neuroscience 37, 330–335.

van Veen, V., Holroyd, C.B., Cohen, J.D., Stenger, V.A., Carter, C.S., 2004. Errors without conflict: implications for performance monitoring theories of anterior cingulate cortex. Brain and Cognition 56, 267–276.

Vlamings, P.H., Jonkman, L.M., Hoeksma, M.R., van Engeland, H., Kemner, C., 2008. Reduced error monitoring in children with autism spectrum disorder: an ERP study. European Journal of Neuroscience 28, 399–406.

Wilbarger, J., McIntosh, D., Winkielman, P., 2009. Startle modulation in autism: positive affective stimuli enhance startle response. Neuropsychologia 47, 1323–1331.