Title: Reduced monitoring of task performance is an effective biomarker of autism

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Short title: Effect of feedback in autism

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Abstract:

People continuously track and adjust their behavior using external and internal signals. Autistic individuals manifest reduced sensorimotor error correction and slower updating of perceptual priors, yielding reduced behavioral flexibility. A potential reason for this reduced flexibility is a lessened sensitivity to both external and internal feedback. A key brain region involved in online monitoring of behavior and updating world models, known to be atypically activated in autism spectrum disorder (ASD), is the anterior cingulate cortex (ACC). During a two-tone pitch discrimination task, we used EEG to track ASD participants' ACC response dynamics, as manifested in their feedback-related negativity (FRN) component produced following "incorrect" feedback. We found that the FRN is missing in more than half of the ASD (17/30) participants, while clearly present in almost all non-autistics (33/35). Moreover, only in non-autistics, feedback affected performance in the following trial. Their responses were slower following an erred trial, their accuracy was improved following hard and correct trials, and they were more affected by the perceptual priors following easy and correct trials. Non-autistics' EEG response to feedback was also sensitive to trial difficulty, while the EEG response of ASD participants was not. Overall, we observed EEG and behavioral correlates of reduced sensitivity to both external and internal feedback in ASD. These observations suggest that increasing the salience of the external feedback could improve ASDs' error monitoring and perhaps increase their behavioral flexibility.

Keywords: decision making; error monitoring; contraction bias; auditory; perceptual learning

Introduction

Two separate and independent lines of evidence suggest that people with autism monitor their behavior less than non-autistics. One is based on computational analyses of behavioral assessments: people with autism spectrum disorder (ASD) are known to be less flexible in updating their motor and perceptual performance to changes in the environment[1,2]. The mechanisms that allow or impede such flexibility are not fully understood. When people perform a perceptual discrimination task they implicitly learn the distribution of the stimuli they experience and form predictions regarding the upcoming stimuli.[3] These predictions are updated on each trial[1,4], but in ASD the rate of this update is reduced.[1,5,6] Similarly, online monitoring of motor (paced finger) synchronization errors is reduced in autism.[7,8] Consistent with these results, there is evidence that ASD participants have difficulty shifting from learned to newly adaptive responses in reversal learning paradigms.[9,10] These results suggest that online tracking of errors with respect to external information is reduced in ASD, and this could relate to the slower rate at which they update their internal representation of the external world.

The second line of evidence suggests atypical online tracking of internal feedback, based on EEG assessments. Specifically, the error-related negativity component, produced when participants detect that they have made an erred motor response in fast motor tasks (such as the flanker task[11–13]) is reduced[14–17] in autism (but see[18,19]). Similarly, an EEG signal is also produced in response to external feedback indicating an error (Feedback-Related Negativity or "FRN"[20]). The FRN was previously studied in the context of success and failure in guessing random rewards[21–24] rather than in evaluating the outcome of reward-maximization strategies. Hence, in those studies, feedback does not indicate a wrong decision process, only bad outcomes. In autism, such studies yielded mixed results[18,24,25] (for review see[15]), leading to the EEG-based hypothesis that responses to external feedback of monetary rewards are typical in autism. Interestingly, EEG responses to external feedback on perceptual success were never assessed.

When tracking one's performance, it is essential to continuously evaluate the validity of the perceptual representations and actions. The Anterior Cingulate Cortex (ACC) is important for such evaluations, integrating internal and external information[26–31], though its specific role is not well understood. Important aspects of its contribution in integrating

information can be tracked by EEG recordings, as it produces a large error-related negativity in response to wrong, fast, and self-paced responses.[12,30,32] ACC was found to be atypical in ASD[6,33], in both connectivity[34–36] and in its pattern of activation[37] particularly when processing perceptual errors.[6,38] This is consistent with recent findings on under-weighting of sensory observation and prediction error in frontal cortices across several mouse models of autism.[5]

Together, the above observations suggest that online monitoring of sensory and motor performance is impaired in ASD. Hypothesizing that the ACC's atypical structure, connectivity, or activity in ASD might also be related to their reduced online sensory monitoring, we measured FRN during a perceptual discrimination (delayed comparison) task. We studied online monitoring of perceptual discriminations with external feedback, both neurophysiologically using EEG and behaviorally with respect to both external (written feedback) and internal (easy versus difficult trials) monitoring of performance. We found that while EEG responses to stimuli and behavioral success rate were similar for ASD participants and non-autistics, responses to both external feedback and internal outcome-evaluations were consistently reduced in ASD. Together, these findings indicate reduced online monitoring of performance in ASD during perceptual decision-making behaviors. Importantly, the effect size of this group difference is very high (Cohen's d = 0.90), implying that the FRN could be used as an indicator of reduced performance monitoring. Clinically, it suggests that increasing feedback's salience may facilitate performance monitoring in autism.

Materials and methods

All experimental methods were approved by the European Research Council and the Ethics Committee of the Hebrew University.

Participants

35 non-autistic and 34 ASD individuals participated in this experiment. One ASD participant was rejected from further analyses because of poor performance in a way suggesting that they did not understand the task, leaving 33 ASD included in our analyses.

Except for one participant in each group, all participants had at most 4 years of musical training. The 35 non-autistics and 33 ASD participants that were included in subsequent analyses were also matched for age, gender, and scaled Block Design score which measures visuospatial reasoning[39] (see Table 1). By contrast, and as expected, there was a significant difference in the scores of the self-reported autism questionnaire, AQ50 (Autism Quotient questionnaire [40]), with an effect size of Cohen's d = 1.10.

Table 1: Demographic information for the subjects included the experiment. For age, block design, and AQ50 the mean is specified for each group, with the standard deviation in parentheses. ** $p < 0.01$ based on a Student's t-test.

	Non-autistics	ASD	t-statistic	p-value
Number of participants	35	33		
Number of females	5	6		
Age (years)	25.9 (3.7)	25.4 (5.3)	0.493	0.624
Block design (scaled score)	12.5 (2.2)	12.9 (3.4)	-0.384	0.702
AQ50**	50.6 (15.8)	74.2 (24.6)	-3.269	0.002

Experiment design

Experiments were coded using PsychToolbox in Matlab (Mathworks). In each trial, participants were sequentially presented with two tones 50 ms in duration with 5 ms onset and offset sine ramps. Tone frequencies ranged from 800 to 1250 Hz and were sampled so that both tones in a pair were either above or below 1000 Hz (as in[41]). The ratio of the two tone frequencies ranged from 1.01 to 1.25 (0.17 to 3.86 semitones). Tone onsets were separated by 2 seconds, resulting in a working memory delay interval of 1.95 seconds. After the second tone, participants had to specify which tone had a higher pitch. Upon response, participants were immediately presented with visual feedback for 400 ms: "Correct" in green if they got the correct answer, or "Incorrect" in red if they were wrong. Participants had 1300 ms to respond after the onset of the second tone, and if they did not respond within this interval, then they saw "Miss" in red on screen for 400 ms (importantly, this "time out" interval is not stressful, as the median response time across all participants

was 646 ms with a range of 398 ms to 923 ms). The next trial was always presented 4 seconds after the first tone onset of the previous trial (see Figure 1A), irrespective of the speed of the participant's response. The inter-trial interval (2 s after the second tone) was set to be equal to the within-trial inter-stimulus interval (2 s) to reduce the effect of within-trial adaptation, which might differ between the groups as it relates to implicit learning[41,42]. The sounds were presented over Sennheiser HD 25 headphones at a comfortable sound level.

Before starting the formal experiment, participants completed a training set of 10-30 trials, presented in blocks of 10 trials with a break in between. To make the task easier, during these training trials the frequency range of the tones was increased to 600 – 1600 Hz, and the tone ratios were randomly selected between 1.35 – 1.4 (equivalent to 5.20 – 5.83 semitones). Participants proceeded to the main experiment upon reaching 8/10 trials correct in a block, or upon completing the three 10-trial blocks, even if this threshold performance was not attained (this was the case for 7/33 ASD participants and 5/35 non-autistics). In the main experiment participants performed 121 trials without breaks.

Behavioral analysis

Performance was assessed by calculating the proportion of trials correct, ignoring misses. To understand the effects of external feedback ("Correct" or "Incorrect" trials) and trial difficulty on subsequent behavior, we further grouped trials based on the participant's accuracy (Correct/Incorrect) and the trial difficulty (Easy/Hard trials). For trial difficulty, easy or hard trials were identified based on a threshold frequency difference of 1.242 semitones, which was equal to the median frequency difference for the distribution of possible frequency differences; specifically, "hard" trials were below this threshold, and "easy" trials were above this threshold. Performance was then calculated for the following trial, conditioned on the accuracy and difficulty of the previous trial.

Additionally, previous work has shown differences between non-autistics and ASD participants in their use of recent history when making decisions in this task.[1] Recent trials bias the decision of participants in subsequent trials by contracting the perceptual memory of the first tone toward what was heard previously, and this effect is stronger for non-autistics than ASD participants. Because of this effect, trials where the tone frequencies

move away from the average frequency of the previous trial are easier than trials where they move towards the average frequency of the previous trial (see Figure 6A for a diagram). To quantify the effect of recent history on choice bias, we split trials into those that should show better performance ("bias+" trials) and those that should show worse performance ("bias-" trials), and then we calculated the difference in performance between these sets of trials. Like our analysis of performance, we also repeated this analysis of bias after conditioning on the accuracy and difficulty of the previous trial.

External feedback also affects behavior by altering reaction times in subsequent trials. Specifically, non-autistics will respond slower after they have made an error, which is an effect not observed in ASD participants in previous studies.[16,43] To quantify this effect in our study, we looked at the response time in correct trials after the participant was correct or incorrect. The difference in response time between incorrect and correct trials is "post-error slowing" and was compared between groups.

EEG recording and preprocessing

Biosemi ActiveTwo with a 32-channel layout was used to record EEG while participants performed the task. Additionally, external channels were recorded bilaterally on the mastoids, to the outside of each eye, above each eye, and on the nose (7 external electrodes total). The EEG data was digitized at 512 Hz.

All preprocessing was done in Matlab (Mathworks). After recording, the EEG data was filtered using a 4th order butterworth filter with -3 dB cutoff frequencies of 1-30 Hz. Eyeblinks and eye motion artifacts were isolated using fastICA[44] applied to the 32 EEG channels as well as the externals and then removed. The data was re-referenced to the average of the mastoid channels. If one of the mastoid channels exceeded three times the median plus interquartile range of the main 32 EEG channels (not including the externals), the mastoid channel was deemed too noisy and only the other mastoid channel was used for reference. In two non-autistic participants, both mastoid channels exceeded this range, and we used the average of neighboring P7 and P8 electrodes instead.

Calculating evoked responses

All analyses of the EEG was done on channel Cz referenced to the mastoids. Event-related potentials (ERP) to the stimuli and the button presses (the participant's response in the task) was first calculated individually for each participant. ERPs to the stimuli were first baselined to the interval of -100 to 0 ms before the stimulus, and ERPs to the button presses were baselined -400 to -200 ms; we chose a different interval before the button press to avoid a negativity that appeared between -200 to 0 ms in some participants (this can be seen in Figure 2B). To avoid the influence of outlier EEG signals on the evoked responses, all ERPs were calculated using a spline-based median of the evoked responses (see[45] for information on the spline transform). First, each EEG segment was transformed into a basis of 173 cubic splines which enforced a smooth representation of the EEG segments with a -3 dB cutoff frequency around 40 Hz. Second, the median of the basis spline weights across trials was calculated. Lastly, the basis spline weights were transformed back into the time domain for interpretation, plotting, and further analysis. These steps were also used when calculating the median ERP across participants. For the shaded regions in the plots of the ERP (e.g. Figure 1B), a bootstrap resampling procedure with replacement was done across participants and repeated 200 times to calculate the 95% confidence interval of the median ERP. Analyses of individual subject ERP magnitudes are based on the minima or maxima of the median ERPs in the time domain.

Follow-up control experiment to understand the feedback-evoked negativity

To rule out other explanations for our EEG results, we invited 3 non-autistics and 3 ASD participants back to do the two-tone discrimination task with three forms of feedback: 1) Correct and immediate feedback, as in the original experiment; 2) Delayed feedback, presented 200 ms after participants' response; 3) Random feedback, where "Correct" and "Incorrect" were presented randomly after the participant's response, independent of their performance, and participants were told in advance that the feedback would be random. All EEG preprocessing and analysis was identical to those of the main experiment.

Results

ERP and behavioral responses to stimuli are similar for people with and without ASD

Participants performed a two-tone discrimination task. On each trial, two tones with different frequencies were presented serially. Participants were asked to determine which of the two tones had a higher pitch (Figure 1A, in the example the second tone frequency is “higher”). Upon responding they were immediately presented with visual feedback: “Correct” in green text or “Incorrect” in red text. Since we were interested in the ability to track performance, which might depend on performance level, we aimed to attain similar success rates for the two groups. We used a simple temporal protocol, with 2 second intervals both between stimuli and between trials, in order to minimize possible effects of neural adaptation that may differ in neurodivergent groups[41]. With these intervals, the peaks of the three main EEG response components (N1 between 70-130 ms, P2 between 150-230 ms, and P3 between 250-350 ms) to the two tones (“1st” and “2nd”) were similar in the two groups (Rank-sum test: 1st N1, $z = -0.52$, $p = 0.606$; 2nd N1, $z = -0.11$, $p = 0.912$; 1st P2, $z = 0.18$, $p = 0.854$; 2nd P2, $z = -0.43$, $p = 0.668$; 1st P3, $z = -1.88$, $p = 0.061$; 2nd P3, $z = -0.87$, $p = 0.384$). We found no significant within trial adaptation of N1 (i.e. the magnitude of N1 was similar in response to the first and second tones in the trial - signed-rank test: Controls, $z = -0.87$, $p = 0.385$; ASD, $z = -0.85$, $p = 0.396$). P2 showed a weak, though significant adaptation in neurotypicals (Controls, $z = -2.23$, $p = 0.023$), which was not significantly different from the absence of adaptation in ASD (ASD, $z = -0.96$, $p = 0.339$; rank-sum test for the difference between groups: $z = -0.92$, $p = 0.357$). As expected, both groups had a P3 component following the second tone (Figure 1B), which characterizes time points where a categorical decision is made ("which tone was higher?"[41,46]). P3 magnitude was similar for the two groups (rank-sum test: $z = 0.209$, $p = 0.835$). Behaviorally, both groups had similar median success rates in the task (Figure 1C; Non-autistics = 71.9%, ASD = 70.0%; rank-sum test: $z = 0.76$, $p = 0.447$), though the cross-participant distribution was broader in the ASD group.

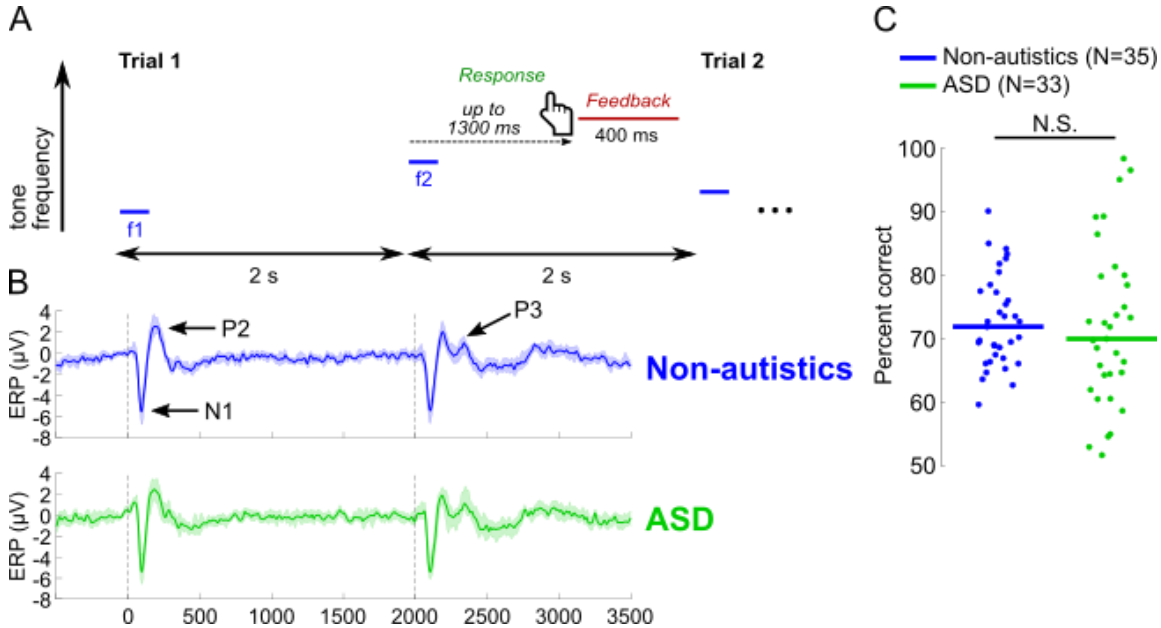

Figure 1: Non-autistics and ASD show similar performance and similar ERP responses to the tones in each trial. (A) A schematic diagram of a trial's temporal structure. Participants performed a two-tone discrimination task where they had to determine if the pitch of the second tone was higher than the that of the first. Visual feedback ("Correct" or "Incorrect") was given every trial immediately upon response. (B) Grand average response (median of participants' median ERPs, see Methods) for electrode Cz. Time zero denotes the onset of the first tone on each trial. Thick lines show the median across participants' median ERPs. Shaded regions show the 95% confidence interval of the median using a bootstrap resampling across participants. Non-autistics are plotted in blue (top) and ASD in green (bottom). Dashed vertical lines mark the onset of each tone. The three main response components, N1, P2 and P3 are marked on the top plot. (C) Distributions of participants' performance accuracy, in each group. Each dot denotes the percent correct of an individual participant, the bars indicate the median in each group. Mean performance was similar in the two groups (no significant difference in a rank-sum test).

The Feedback-Related Negativity (FRN) is significantly weaker in ASD

Under similar task performance and EEG responses to tones in the two groups, we next asked whether the response to external feedback is also similar. The visual feedback was provided immediately (within 20 ms) after the participant's button press. To quantify the EEG response to the feedback, we aligned our analyses to participants' button press. The EEG signal, following "incorrect" feedback, is known as the "feedback-related negativity" or FRN[20], and the topography of activity at the time of the negativity (e.g. inset

in Figure 2B) has been source localized in previous studies to anterior cingulate cortex (ACC).[20,30] We rejected 3 ASD participants who performed very well in the experiment and thus had too few incorrect answers to get a reliable FRN response (< 6 trials incorrect; 3 best performers, see Figure 1C where these participants performed better than all non-autistics). Figure 2A plots samples of 12 single participants, six with ASD (right) and six non-autistics (left), matched for the rank order of their FRN with respect to their group. It illustrates that the majority of ASD non-autistic participants (sampled here by 1st to 26th in FRN magnitude) have a clear and large FRN, whereas most ASD (sampled here by 11th to 26th) do not.

To quantify the magnitude of the FRN, we determined, for each participant, the minimal response between 200-350 ms after the participant's button press (Figure 2C). This time window captured the span of the negativity for all participants (dashed vertical lines in Figure 2A, median ERP in Figure 2B). FRN magnitude was then quantified as the difference between the minimum in incorrect and the minimum in correct trials. Figure 2C indicates that in both groups the minimum for the evoked response to incorrect trials was more negative than to positive trials (signed-rank test: Non-autistic, $z = 5.16$, $p < 0.001$; ASD, $z = 3.16$, $p = 0.002$). However, following "incorrect" feedback most of the non-autistic participants (33/35) showed an FRN. By contrast, only a minority of the ASD participants (13/30) showed an FRN response (Figure 2C, D). This was significantly smaller in ASD participants than non-autistics (Figure 2D, rank-sum test: $z = -3.56$, $p < 0.001$). Importantly, the effect size of this group difference was large (Cohen's $d = 0.90$). For reference, the effect size (Cohen's d) of AQ, a questionnaire specifically designed to detect ASD traits [40], was 1.1 for our participants, comparable to the effect size of the FRN measure. We did not find a correlation within each group between AQ score and FRN magnitude: Non-autistics, $\rho = -0.274$, $p = 0.117$; ASD, $\rho = -0.062$, $p = 0.733$, perhaps because FRN magnitude also depends on performance levels (in non-autistics it was shown that "incorrect" feedback yields a larger error- or feedback-related negativity when the task is easier[20] and when errors are more unexpected[12,23]).

To illustrate the potential of (reduced) FRN as a biomarker of autism, at the single participant level, figure 2D on the right shows a histogram of the differences between incorrect and correct evoked responses in each group. It shows that a discriminant

boundary of -2.5 µV (close to the median magnitude for ASD participants, plotted in Figure 2D) identifies more than half of the ASD participants as ASD (who have non-reliable incorrect responses), while rejecting 33/35 of the non-autistics.

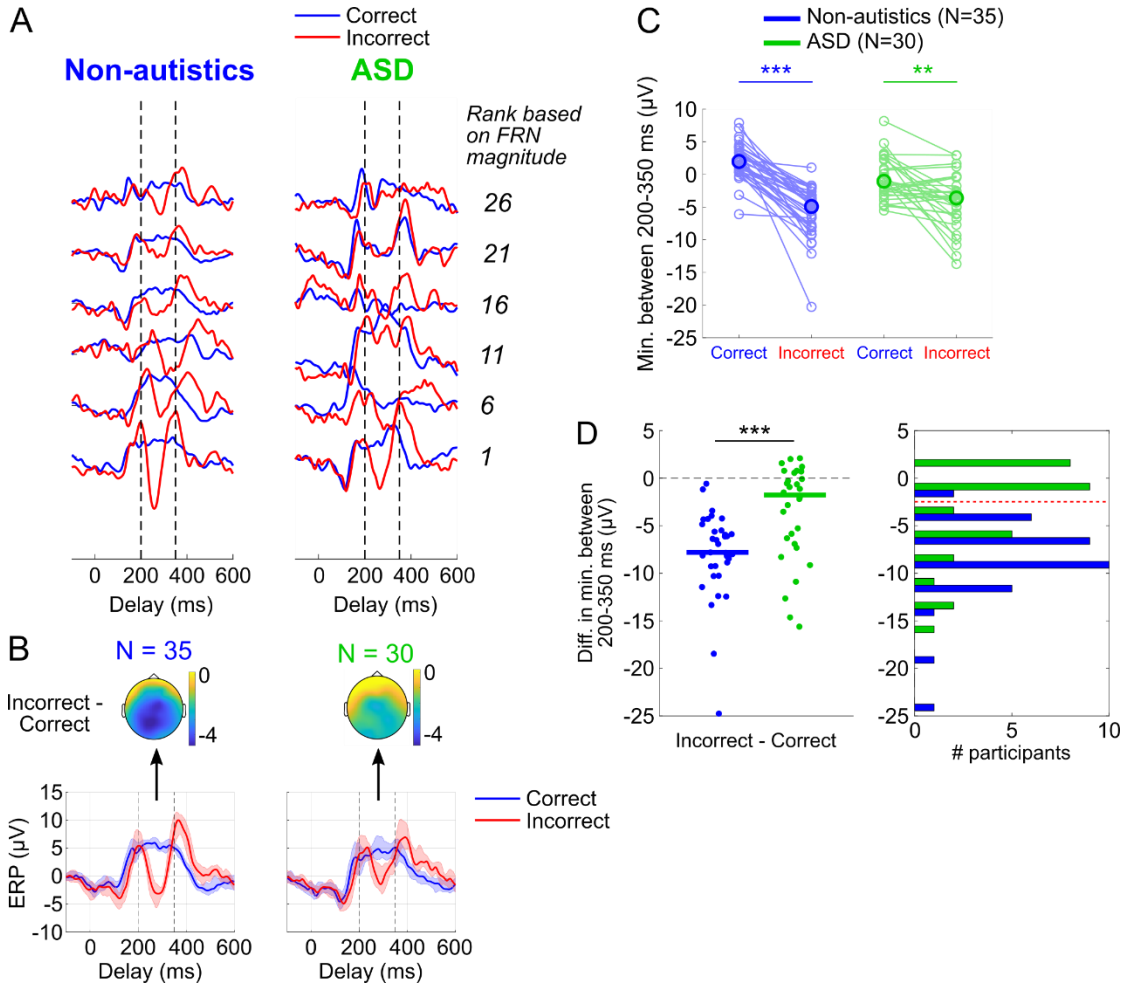

Figure 2: A weaker response to external feedback (Feedback-Related Negativity, FRN) was observed in ASD participants' EEG activity time-aligned to their behavioral response. (A) Single participants' FRN, defined as the difference between (minimal) responses following "Correct" (blue) and "Incorrect" (red) feedback within 200-350 ms after their behavioral response (range marked by the dashed lines). FRNs were ranked according to these differences. We plotted responses of six participants from each group, whose ranked magnitude of FRNs were matched with respect to their group (non-autistics on the left, ASD on the right). These ranks are denoted on the right (1st being largest). While all six non autistics had an FRN, only 2 of the six plotted autistics' responses manifest a clear FRN. (B) Median ERP across participants, shaded error bars indicate the bootstrapped 95% confidence intervals of the median. The topography of the difference in voltage between Incorrect and Correct trials averaged within this time interval is shown above each plot. (C) Quantification of the FRN magnitude as the difference between the ERP minimum for incorrect and correct trials, within the range 200-350 ms as indicated in A and B, plotted separately for each participant.

At the group level both groups show a significant FRN. (D) Individual dots show the difference in magnitude between correct and incorrect, and the horizontal bars denote the median of each group. The group difference in FRN is highly significant (*** = $p < 0.001$). On the left, each symbol denotes a single participant. Note that more than half of the ASD participants (above the green line) are distributed symmetrically around zero difference, meaning that they have no clear FRN. By contrast, only 2/35 neurotypicals do not show a clear FRN (upper-most blue dots in the plot). The histogram on the right also shows the distribution of FRN in each group with 2.5 μV bins. It illustrates that FRN magnitude could be an effective bio marker for ASD; a plausible discrimination boundary of -2.5 μV is shown as a red dashed line.

To better understand the FRN component observed in this study, we administered a small control experiment with 3 non-autistic participants and 3 ASD participants. The non-autistic participants were selected because they had a large FRN in the previous experiment and were available for the follow up, and the ASD participants were selected based on willingness to participate in a follow-up session. Note that because of the reliability of the FRN that we observed in non-autistics in the main study (Figure 2), we were confident that including only 3 participants would be sufficient to observe changes in the FRN across conditions in the follow-up. The follow-up experiment was composed of three blocks in a random order, where each block had a different type of feedback: 1) Immediate-correct feedback was identical to the previous experiment (as in Figure 1A); 2) Delayed-correct feedback, where the feedback was delayed by 200 ms relative to the participant's response, aimed to verify that FRN delay was indeed locked to the external feedback; 3) Immediate-random feedback, which showed "Correct" or "Incorrect" randomly after the participant's response, independent of their choice, to check whether feedback validity was crucial for producing FRN. In the random condition, participants were told that the feedback would be random and uninformative, but they were still instructed to look at the feedback during the task.

Our first test compared (correct) immediate and delayed feedback asking, firstly, for non-autistic participants, who showed a reliable FRN, was the EEG response timed to the button press or the feedback? We found that when the feedback was delayed, both the EEG response to "correct" and "incorrect" feedback were also delayed by 200 ms (Figure 3A, top), indicating that the EEG response was timed to the external feedback rather than to the response itself. Secondly, how does it affect the EEG response for ASD participants? As in the main experiment, we found no FRN here either (Figure 3A, bottom), but there

was an anticipatory response that was especially prominent in the Delayed feedback condition (Figure 3A, bottom right), and it did not differ between "correct" and "incorrect".

Our second test compared the EEG response to true (immediate) and random feedback, asking whether in the absence of informative feedback the FRN response disappears. In non-autistics, randomizing the feedback indeed causes the FRN to disappear (Figure 3B, top). Additionally, non-autistics' positivity 200 ms after correct response also

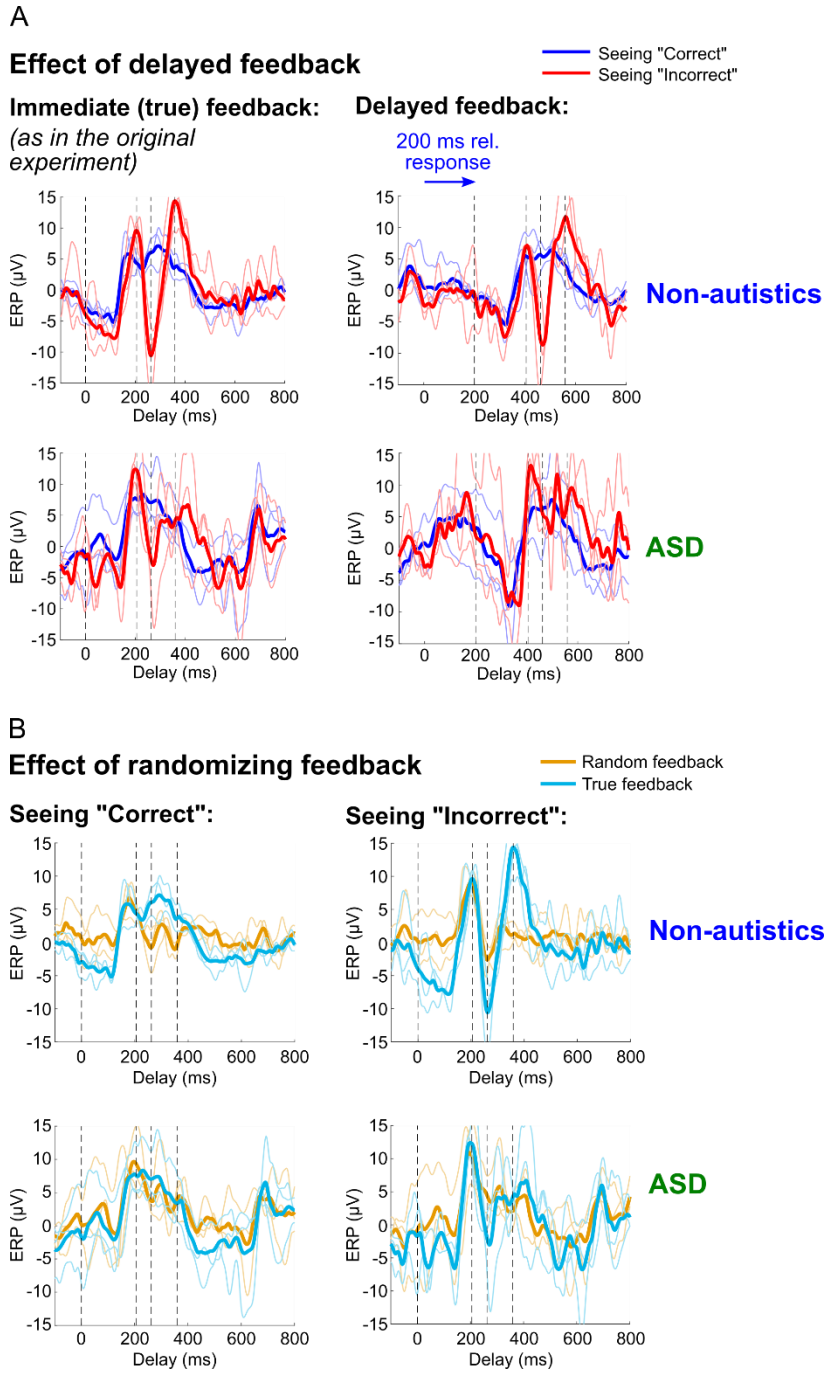

Figure 3: A (mini) follow-up control experiment (3 participants from each group) for feedback timing and validity. (A) Evoked responses to correct (blue) and incorrect (red) feedback presented immediately after the participant's response (left), or delayed by 200ms (right), plotted separately for the non-autistic (top) and ASD participants (bottom). For non-autistic participants the FRN response was delayed by 200ms. For autistic participants there was still no FRN. Interestingly, at a shorter time window than the FRN there is a large negative response, which does not differ between correct and incorrect response, and suggests some enhanced anticipation, whose nature should be further studied. The thin colored lines are the evoked responses for individual participants, and the thick line is the average. Dashed lines label the peaks and troughs of the evoked response to immediate feedback (on the left). The dashed lines on the right mark the same time points in A shifted by 200 ms, to make clear the shift in the response. (B) Evoked responses of non-autistic (upper) and ASD (lower) after seeing "Correct" (left) or "Incorrect" (right) on screen when the feedback was either immediate and true (blue, identical to A) or randomized (orange).

disappeared, indicating that the signal was related to information in the feedback rather than the visual input. Interestingly, the ASD participants had EEG responses to both correct and incorrect feedback that did not appear to differ between the immediate (true) feedback and random feedback conditions, unlike the non-autistics (Figure 3B, bottom). This result suggests that ASD participants may process feedback the same way irrespective of its information content.

Taken together, the follow-up study indicates that the responses we observed in non-autistics were time-locked to the feedback. Importantly, both the positivity in the response to "correct" feedback after 200 ms (Figure 3B) and the FRN reflected the processing of the feedback in the task, rather than visual processing of the written words.

Only non-autistics show slowing of response times following an error

The smaller FRN in ASD participants suggests weaker processing of errors. We therefore asked whether there is also a behavioral group difference in the impact of feedback on subsequent trials. Previous work had shown that in a fast reaching task, neurotypical participants respond more slowly, presumably being more careful, following errors.[12] Given our observation of reduced FRN, we now asked whether behavior also shows reduced sensitivity to feedback, by showing reduced or no slowness (although not "pushed" to be very quick) in ASD. To test this, we quantified the response times of correct

trials comparing those that followed incorrect trials to those that followed correct trials (Figure 4A, see[16]). We found that non-autistics' responses were slower after incorrect trials (known as "post-error slowing"; median = 40.0 ms, inter-quartile range = [71.3 ms]), while ASD participants' did not (-1.4 ms [93.3 ms]) (Figure 3B). Interestingly, non-autistics' slowing had a similar magnitude to that observed in fast responses in flanker tasks (South, 2010[16], Table 2; 37.7 ms), while this effect was completely lacking in ASD. We did not observe post-error slowing on incorrect trials (Supplementary Figure 1). Post-error slowing and the FRN magnitude were significantly (though weakly) correlated in ASD participants (Spearman's correlation: rho = -0.36, p = 0.049).

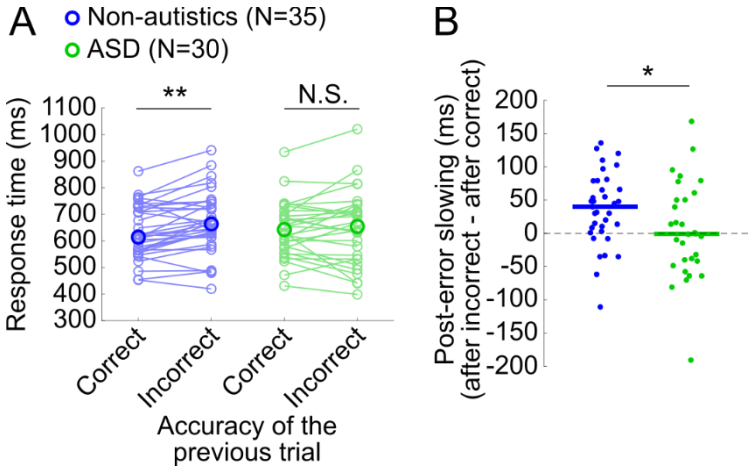

Figure 4: Non-autistic participants slow responses after incorrect trials, while ASD participants do not. (A) Response times following correct and incorrect trials when the subsequent trial was correct, plotted separately for non-autistic (blue) and ASD (green) participants. (B) Post-error slowing, the difference in response time following incorrect versus correct trials, shown for both groups, is significantly longer for non-autistic participants (* $p < 0.05$, ** $p < 0.01$).

Only non-autistics' EEG responses are modulated by task difficulty

External feedback is often processed in tandem with one's confidence in their decision.[47,48] When participants are more confident in their knowledge of task-relevant information they are more likely to use that information.[49] This is a form of internal feedback that can also modulate the participants' behavior in the task, but does it affect their EEG responses to feedback?

As a proxy for participants' assurance of their perceptual decision, we separated the trials into "Hard" and "Easy" trials using a median of the frequency difference in the trial as the threshold (the threshold of 1.242 semitones was the median frequency difference across all trials and participants) (Figure 5A). We reasoned that in easy trials, participants are more confident in their decision. Indeed, non-autistics' EEG responses were modulated by task difficulty (Figure 5B). We used the peak-to-peak difference in the evoked response between the subject's button press (0 ms in Figure 5B) and 350 ms after the button press to quantify the magnitude of this difference (Figure 5C). We found that, in non-autistics, this difference was significantly larger in hard trials compared to easy ones (signed-rank: $z = -2.69$, $p = 0.007$), suggesting that feedback was perhaps weighted more in correct difficult trials. However, ASDs did not show any difference ($z = 1.51$, $p = 0.131$). The group difference was highly significant (rank-sum: $z = -2.98$, $p = 0.003$).

We had also observed that the EEG response was affected by external feedback when the participant saw that they were correct or incorrect (Figure 2). Could the evoked response to feedback reflect joint processing of both external and internal feedback? Specifically, we thought that participants' confidence during the presentation of feedback, and the modulation of the EEG response with task difficulty, is likely affected by whether they were correct. To explore this possibility, we repeated the analysis of task difficulty separately for correct and incorrect trials. The peak-to-peak of the response between 0-350 ms to correct trials was larger when the task was hard than when it was easy for non-autistics (signed-rank: $z = 3.98$, $p < 0.001$), but interestingly not for ASD participants ($z = 0.40$, $p = 0.688$), and this effect was significantly different between groups (rank-sum test: $z = 2.34$, $p = 0.019$) (Figure 5C). The effect was driven particularly by easy trials; there was a significant difference between groups in the peak-to-peak magnitude during easy trials ($z = -1.99$, $p = 0.047$) but not hard ones ($z = -0.76$, $p = 0.447$). During incorrect trials, both groups showed a larger EEG peak-to-peak during easy trials compared to hard ones. But unlike the correct trials, the change in the response magnitude was not significantly different between groups (signed-rank: non-autistics, $z = -2.01$, $p = 0.045$; ASD, $z = -3.12$, $p = 0.002$; rank-sum test of the differences: $z = 0.26$, $p = 0.793$) (Figure 5D). The latter result agrees with past work showing an inverse relationship between the error-related

negativity and task difficulty[12] or the probability of errors.[23] However, group differences are notably absent for incorrect trials, in contrast correct trials.

In theory, the peak-to-peak measure is susceptible to noise[50] and having a mismatch in the number of trials (for example, fewer hard trials that are correct) could artificially create a peak-to-peak difference between conditions. However, the peak-to-peak differences observed during correct trials were not correlated with a difference in the number of trials (Spearman's correlation: Non-autistics, rho = 0.11, p = 0.547; ASD, rho = -0.27, p = 0.137).

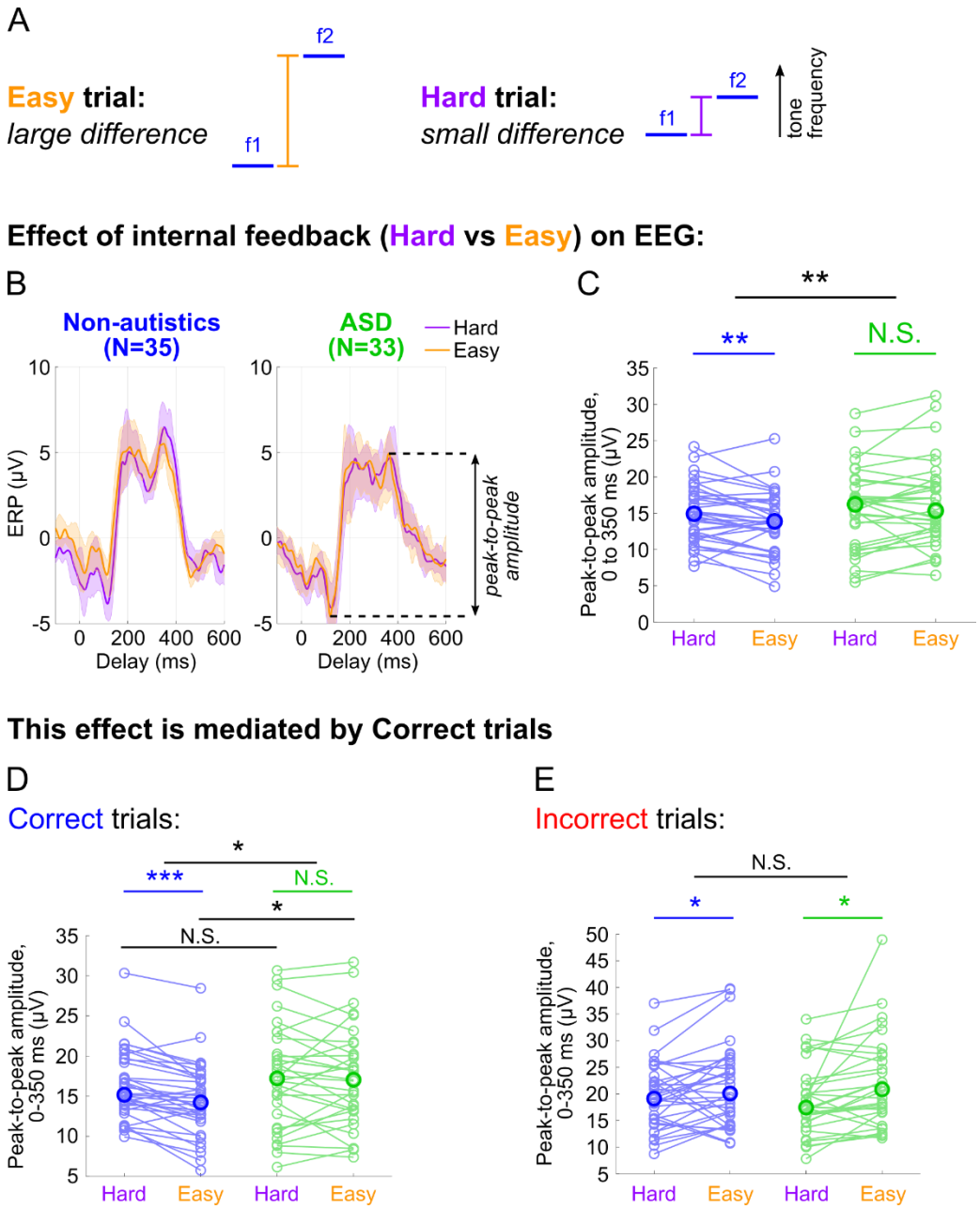

Figure 5: Weaker effects of internal feedback were observed in ASD participants compared to non-autistics. (A) Diagram showing the distinction between easy and hard trials. Easy trials have a larger frequency difference than the median difference across all possible trials (1.242 semitones), while hard trials have frequency differences smaller than 1.242 semitones. (B) Median evoked responses across participants, time-aligned to the behavioral response, plotted separately for hard (purple) and easy (orange) trials. (C) The peak-to-peak difference in the evoked EEG responses to hard compared to easy trials is significantly larger in non-autistic participants. (D) and (E) show that the group difference in C stems from correct recent trials (D) and not from incorrect (E) ones, implying that EEG responses integrate external feedback with internal monitoring of trial difficulty level. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

There was no correlation between the FRN magnitude (after correct vs incorrect feedback) and this peak-to-peak difference (after easy vs hard trials) for either participant group (Spearman's correlation: Non-autistics, rho = -0.07, p = 0.679; ASD, rho = -0.06, p = 0.767). This could be because the measures are noisy across participants, but it implies that internal and external feedback are not necessarily produced by the same neural processes, and that the exact magnitudes of these neural measures do not scale with an increased behavioral effect.

Our results indicate that both internal feedback and external feedback significantly modulate the EEG response in non-autistics but not in ASD, showing that ASD participants have weaker processing of both types of feedback.

Only non-autistics' perceptual bias is modulated by task difficulty

The modulation of non-autistics' ERP response by task difficulty suggests that it may also have a behavioral impact. We therefore examined how external and internal feedback affect participants' response tendencies in subsequent trials. Previous work had shown that participants' behavioral responses in a two-interval forced choice task are substantially biased by previous trials' statistics.[1,3,4,51,52] They interpreted this bias as resulting from an asymmetry in noise level of the first and second tone in each trial at the time of the comparison. Given a Bayesian framework, they proposed that since the first tone is noisier, it is contracted more towards an implicit prior (predicted frequency), which is formed by previous trials' frequencies[3]. When the first tone is closer to this prior,

contraction improves performance (consequently termed bias+ trials), whereas when it is farther, performance deteriorates (bias-)[4,51] (Figure 6A).

We calculated the recency bias, namely the bias of the first tone towards the most recent trial's frequency.[1] The bias is the difference in success rate between bias+ trials (where this contraction produces a performance increase) and bias- trials (where performance decreases)[3,51] (but see[53]). In line with previous work[1], the bias for ASD participants was on average smaller than non-autistics (rank-sum test: z = 2.39, p = 0.017). Next, we then grouped these trials based on the difficulty of the previous trial (less than or

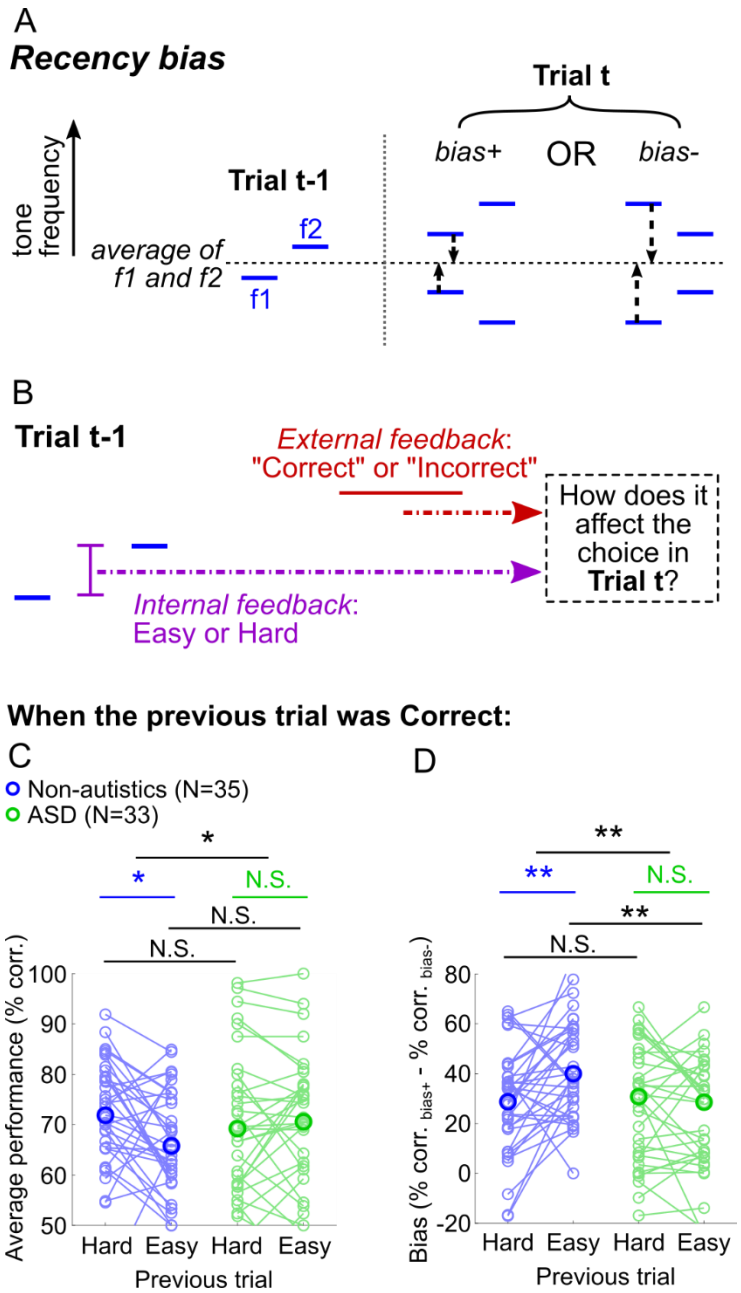

Figure 6: Only non-autistics show a larger recency bias following correct and easy trials. (A) A schematic illustration of a tentative mechanism underlying bias towards recent trial's frequencies in perceptual decisions. We divide trials to bias+ trials where the first tone's frequency is closer to the mean frequency of the previous trial, hence the bias of the perceptual trace towards this mean improves performance, whereas in bias-, the second tone is closer. Better performance occurs in "bias+" trials versus "bias-" trials though they differ only in their preceding trial's frequency. (B) Diagram illustrating the main question of how internal feedback (Hard / Easy) and external feedback (Correct / Incorrect) in previous trial affect the participant's choices in the current trial. (C) When non-autistic participants were previously correct yet less confident (hard trials), there is significantly better performance in the subsequent trial than when they were correct and more confident (easy) in the previous trial. This effect was not found for the ASD group. (D) When the previous trial was both easy (less confident) and was denoted as correct, non-autistics, but not ASD participants, increase the magnitude of their bias. Thus, increased integrated assurance increases the bias. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

greater than 1.242 semitones) and based on participant's accuracy (correct or incorrect) (as in Figure 6B). Participants were rejected if they had no trials in any condition (for example, if they were never incorrect on an easy trial). All participants were included for previously correct trials, and 32 non-autistics and 28 ASD were included for incorrect trials.

When we separated trials into those following easy or hard trials (internal feedback) and correct or incorrect trials (external feedback), we first found a difference in performance depending on the previous trial's difficulty. For non-autistics, performance was better when the previous trial was correct and hard compared to correct and easy (Figure 6C: Correct, $z = 2.41$, $p = 0.016$). (Note that this effect is not due to the participant's response being slower and more cautious because the previous trial was correct.) ASD participants did not show this effect on performance ($z = -0.10$, $p = 0.918$; performance difference between groups: $z = -2.09$, $p = 0.037$). Additionally, the effect of difficulty on subsequent performance was not observed for previously incorrect trials (Non-autistics: $z = 0.59$, $p = 0.557$; ASD: $z = 1.25$, $p = 0.212$; Supplementary Figure 2A).

We also found that non-autistic participants showed a larger perceptual bias when the previous trial was easy compared to when it was hard, but only when they responded correctly in the previous trial (Figure 6D; signed-rank: Correct, $z = -3.01$, $p = 0.003$; Incorrect, $z = -0.55$, $p = 0.583$) (for incorrect trials see Supplementary Figure 2B). In contrast, ASD participants did not show this effect on bias in correct trials ($z = 1.19$, $p = 0.235$) nor incorrect trials ($z = -0.68$, $p = 0.494$), and the bias difference between easy and

hard trials when the previous trial was correct was significantly weaker than non-autistics' ($z = 3.07$, $p = 0.002$). The group difference was particularly driven by a difference in bias when participants were previously correct in an easy trial, where non-autistics showed a larger bias than ASD ($z = 3.15$, $p = 0.002$).

Together, the behavioral group differences suggest that the magnitude of the recency bias and the participant's subsequent decisions are partly affected by a convergence of internal and external feedback. When non-autistic participants are correct and confident in the stimulus information from the previous trial, they update their perceptual priors. ASD participants have weaker processing of both internal and external feedback, and as a result their decisions are unaffected by the accuracy or internal reliability of their response.

Discussion

In this study, non-autistics and ASD individuals performed a two-tone discrimination task. Our results showed that error processing of external feedback in perceptual tasks is reduced in ASD, producing a smaller Feedback-Related Negativity (FRN) than non-autistics. Groups also differed in their evoked responses to "correct" feedback, where non-autistics had a larger response to hard (small frequency difference) compared with easy trials. This interaction indicates that the feedback-related EEG response (the FRN) integrates internal (easy vs difficult) and external feedback (correct vs incorrect). Group differences in EEG were associated with behavioral modifications in the subsequent trials. Following feedback indicating an error, non-autistic responses, but not ASD, were slower, suggesting cautiousness. Non-autistics, but not autistics, showed improved performance after a correct and hard trial, compared to correct and easy trial. Likewise, non-autistics showed larger bias after correct and easy trials, but ASD participants did not show this effect. Taken together, the results show neural and behavioral correlates of internal and external feedback and their effect on perceptual learning, merging two lines of observations in the literature.

As FRN is attributed to ACC[20], our EEG results suggest that error monitoring in ACC, both for external and internal feedback, is reduced in ASD. Importantly, this reduction is not only highly significant, but it also affects the majority of the ASD

participants in our study (17/30 ASD participants had magnitudes above -2.5 µV). The magnitude of this group is large (Cohen's d = 0.90, see Figure 2) and comparable to the AQ[40] (d = 1.10, see Table 1), making it not only scientifically important but also clinically relevant. The effect is strong enough that it could be used to easily assess reduced performance monitoring in more than half of the ASD population.

The results of smaller FRN align with other human studies indicating weaker activation in autistics' ACC.[6] However, previous studies found that ACC encodes the expectation of errors or conflicting information[27,29,54,55] (for review see also[56]) which is subsequently important for identifying changes in task rules and the value of future choices.[57,58] We now show that these ACC signals (as measured with the FRN and peak-to-peak differences in the EEG response) are produced during error monitoring in perceptual discrimination, where subsequent trials are affected by previous trials' level of difficulty and success rate. While there is strong evidence suggesting that our results arise from ACC processing, other neural mechanisms could also be at play. In particular, related work in EEG has demonstrated an effect of confidence on the central parietal positivity, a neural signal related to accumulating information towards a decision.[48] This arises from parietal cortical areas that are involved in decision making, and is unlikely to arise from the ACC. Additionally, some work has shown that feedback may also be processed in the cerebellum[59,60], which could also be involved in generating the behavioral differences observed here.

Previous work had shown that the error-related negativity, in the absence of external feedback (hence reflecting internal feedback), is weaker in ASD participants compared to non-autistics.[14–17] However, evidence for group differences in the FRN were mixed.[15] Importantly, much of this work had focused on the effect of feedback on reward expectations.[18,24,25] Specifically, these studies varied the probability that the participant could get a reward after making a decision. Our results imply that group differences in error monitoring require the formation of perceptual expectations rather than processing of the reward outcome itself. In prior work the actual percept generating the error could be dismissed in subsequent trials because the probability of a reward was randomized[24], while in our study it is well established that participants use previous information to both learn about the task rules[61,62], and update their perceptual bias in subsequent trials.[1,4,51]

We further found that people with ASD have reduced processing of internal feedback, which was observed by looking at EEG and behavioral differences relative to previous trial difficulty. We interpret this effect as a consequence of confidence. Previous work has shown that confidence modulates serial biases[49,61,63], similar to the effects shown here. Correct performance in response to a difficult trial (low confidence), can cause larger learning updates than after an easy trial (high confidence), where learning is already at ceiling.[61] While non-autistics show improved performance after difficult trials compared to easy, this effect is absent in autistic participants (Figure 6C). Behaviorally, there is also a weaker correlation between confidence and performance in ASD participants.[43] Several studies have quantified EEG responses to feedback in the context of reward and error probabilities[21–23], but to our knowledge this has not been measured with respect to trial difficulty or confidence explicitly, especially during a perceptual learning task.

In summary, our findings indicate that both external and internal feedback are incorporated in the behavior of non-autistics and are manifested in their EEG response following the feedback. Non-autistics slow their responses after negative feedback on error trials, perform better after correct response on a difficult trial, and finally increase the use of perceptual priors (internal model of stimulus statistics) after correct response on an easy trial (where confidence is highest). All of these behavioral manifestations could be collectively termed serial effects, namely the impact of recent trials on current performance. ASD participants do not show any of these behavioral or neurophysiological modulations. These results link two findings in the literature. One indicates that ASD participants use recent prior information less than non-autistics in perceptual tasks. This is the case when inter-trial intervals are short and require fast trial-to-trial updating[1,53] or when external statistics are volatile such that optimal inference yields large trial-to-trial prediction updating.[5,64] The second indicates poor online error correction in motor tasks[7,8,65], and perhaps reduced conflict resolution when fast responses are required.[16] Both are suggested to be mediated by ACC pattern of activation.[56,66]

Our findings also add to the growing body of literature that seeks to identify reliable biomarkers for ASD[67], based on atypical brain connectivity[68], altered event-related potentials[69,70], differences in neurochemical[71–73] or immunological profiles.[74,75] Heterogeneity of ASD complicates biomarker development.[67,76] However, a recent study

shows all three tested genetic mouse models of ASD underutilize adaptive statistical regularities during decision-making, and frontal cortices underweight sensory observation and prediction errors.[5] This suggests that despite heterogeneous genetic instantiations, a large fraction of ASD individuals may manifest convergence onto common behavioural and neurophysiological phenotypes related to error monitoring and prior updating. In this regard, our findings on the EEG response following feedback in perceptual decision-making tasks open a promising avenue for developing objective, brain-based criteria for autism diagnosis and monitoring.

Funding

This work was funded by the Israel Research Foundation (grant 1650/17) and by the European Research Council (grant 833694). NZ was also funded by the ELSC-SWC Postdoctoral Fellowship. YW was also funded by the ELSC PhD Fellowship.

Data availability

All EEG and behavioral data from the experiment are available here: <OSF link>. The code used to run the experiment and analyze the data can be found here: <gitlab link>.

Competing interests

The authors report no competing interests.

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