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RESEARCH ARTICLE

Increased context adjustment is associated with auditory sensitivities but not with autistic traits

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Abstract

Bayesian models of autism suggest that alterations in context-sensitive prediction error weighting may underpin sensory perceptual alterations, such as hypersensitivities. We used an auditory oddball paradigm with pure tones arising from high or low uncertainty contexts to determine whether autistic individuals display differences in context adjustment relative to neurotypicals. We did not find group differences in early prediction error responses indexed by mismatch negativity. A dimensional approach revealed a positive correlation between context-dependent prediction errors and subjective reports of auditory sensitivities, but not with autistic traits. These findings suggest that autism studies may benefit from accounting for sensory sensitivities in group comparisons.

Lay Summary

We aimed to understand if autistic and non-autistic groups showed differences in their electrical brain activity measured by electroencephalography (EEG) when listening to surprising tones infrequently embedded in a statistical pattern. We found no differences between the autistic and the non-autistic group in their EEG response to the surprising sound even if the pattern switched, indicating their ability to learn a pattern. We did find that, as subjective sensory sensitivities (but not autistic traits) increased, there were increasingly large differences between the EEG responses to surprising tones that were embedded in the different statistical patterns of tones. These findings show that perceptual alterations may be a function of sensory sensitivities, but not necessarily autistic traits. We suggest that future EEG studies in autism may benefit from accounting for sensory sensitivities.

KEYWORDS
atypical perception, autism, Bayesian, EEG, MMN, precision, prediction errors, predictive coding

INTRODUCTION

Atypical sensory experiences are estimated to occur in over 90% of autistic children (Tomchek & Dunn, 2007) as well as in adults (Crane et al., 2009). Sensory disruptions are one of the first characteristics to appear in autism and have also been associated with core diagnostic criteria such as difficulty in social communication (Kern et al., 2007; Thye et al., 2018), which are positively correlated with autistic traits (Tavassoli, Miller, et al., 2014). A decrease in tolerance to sound is highly prevalent in autistic individuals, and most individuals on the spectrum experience it at some point in their lifetime (Williams, He, et al., 2021; Williams, Suzman, & Woynaroski, 2021). Auditory hypersensitivities have been related to language developmental delays in autistic children (Eigsti & Fein, 2013; Jones et al., 2009) and have been shown to increase anxiety and limit participation in social activities (Stiegler & Davis, 2010). Understanding the mechanisms that give rise to such perceptual alterations could be useful for improving diagnostic tools and targeted interventions for improving quality of life for autistic individuals.

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"Bayesian brain" perspectives on autism have given rise to models that attribute sensory perceptual dysfunction in autism to an underlying disorder of precision (Haker et al., 2016; Lawson et al., 2014; Palmer et al., 2017). Specifically, it has been suggested that autistic perception may arise from altered reliability in observations or expectations (measured mathematically as *precision*, which is the inverse of variance of the representative Gaussian distribution). These theories suggest that sensory disruptions may arise either from forming poor models of the environment (the Hypo-priors model; Pellicano & Burr, 2012) or due to sensory observations being too narrowly tuned (the Precise likelihood model; Brock, 2012). Van De Cruys et al. (2014) suggested that the weighting of prediction error (the difference between our expectation of sensory information and the new sensory observation itself) is less flexibly adjusted in individuals on the autism spectrum (AS), particularly across different contexts of uncertainty. This account, termed the "Highly inflexible prediction errors in autism (HIPPEA)" model, is able to explain key characteristics of autism spectrum disorder (ASD) such as altered perceptual processing and resistance to change, as well as difficulty in social communication. Here we aimed to assess this model conjecture that there is reduced context learning and context updating in autism.

Mismatch negativity (MMN) is an ideal physiological marker for investigating sensory prediction errors (Garrido et al., 2009). The auditory MMN reflects preattentive change detection in a pattern of stimuli, and is particularly useful in that it can be studied without participants needing to pay direct attention to the stimuli (Näätänen et al., 2012). There is currently limited literature on predictive processes in autistic adolescents and adults using the MMN, and of the studies that have been conducted the findings are mixed. Studies using classical oddball paradigms with pure-tone frequency or duration deviants have demonstrated either no difference (Chien et al., 2018) or larger MMN (Lepistö et al., 2007) amplitudes in autistic individuals relative to controls. Other studies using more complex stimuli such as speech sounds (e.g., phonemes with or without affect) have also demonstrated either no difference in the mismatch response (Kasai et al., 2005) or reduced responses (Kujala et al., 2005) in ASD compared with neurotypicals (NTs). Two recent meta-analyses of studies employing both complex and pure-tone stimuli revealed no differences in MMN responses between autistic and NT adults (Chen et al., 2020; Schwartz et al., 2018). Schwartz et al. (2018) suggested caution in interpreting their findings, however, because many studies included in their meta-analysis were underpowered. They also noted that there is a tendency for autistic adults to show larger MMN responses than their typically developing peers. Schwartz et al. (2018) also found that autistic individuals show greater differences in the MMN to non-speech sounds versus speech sounds, relative to NT controls. Very few studies, however, have investigated the flexibility of prediction error across different contexts in ASD. Goris et al. (2018), using an oddball paradigm, showed that global context modulated the MMN in both ASD and control groups, but the effect was smaller in the autistic group than in NT controls. Their study suggests that autistic individuals show reduced context updating when the deviant occurs in more frequent versus less frequent contexts.

In the present study our aim was to determine whether autistic individuals demonstrate prediction error adjustments to context, by evoking prediction errors (MMN) in contexts with low or high uncertainty. We used a stochastic oddball paradigm (Garrido et al., 2013) to compare: (a) differential responses to standards and deviants (sensory prediction errors), which reflect an individual's ability to learn about sensory context, their sensitivity to variability, and appropriate attribution of salience to odd but not common events, and (b) MMN responses to the different levels of contextual precision that reflect an individual's sensitivity to contextual uncertainty. The overall goal of the study was to provide evidence for or against models that describe perceptual disruptions in autism as a disorder of prediction error weighting. We also took a dimensional approach in which we investigated both autistic traits and sensory sensitivities, the aim of which was to understand the relative contribution of sensory sensitivities to prediction-error formation in autism.

METHODS

Participants

Participants ($N = 59$) aged 18–35 years were recruited via Asperger's Services Queensland, Autism Queensland and Mind and Hearts, The University of Queensland (UQ) online recruitment system, the UQ newsletter, and online advertisements. Participants were recruited for the NT group if they self-identified as having no diagnosis of neurodevelopmental or psychiatric disorders and no history of medication acting on the nervous system, as well as an autism quotient score of <32. All NT participants had a AQ score less than 32 so we did not need to exclude any participants. Participants with a reported diagnosis of an ASD undertook an Autism Diagnostic Observation Schedule (ADOS; Gotham et al., 2007; Hus & Lord, 2014) interview with a trained clinical psychologist to confirm diagnosis. Participants who received a calibrated severity score below 3 (5 participants) were excluded from the AS group. No participants reported a history of seizures or epilepsy. There were no other exclusion criteria for the AS group. Some of our NT participants also had high anxiety scores, which may indicate undiagnosed psychopathology. Nonetheless, we included them in the sample as this may be a more representative

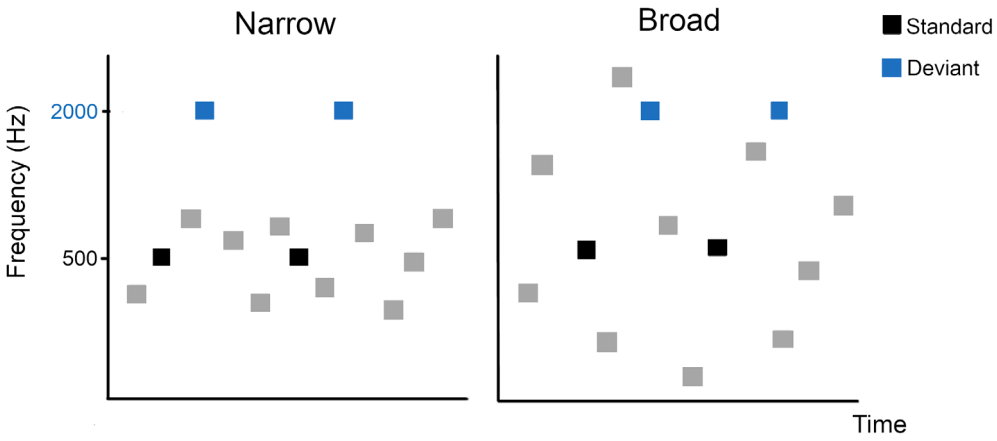


FIGURE 1 Stochastic oddball paradigm. Participants listened to a stream of 500 ms tones drawn from either a narrow (left) or broad (right) distribution of frequencies. Probe tones of 500 Hz (black; standards) and 2000 Hz (blue; deviants) were inserted into the stream each constituting 10% of all tones

non-autistic sample. One participant did not complete the ADOS interview and thus was also excluded from the AS group. Thus, group comparisons were conducted with 23 AS and 23 age- and gender-matched NT participants. All participants (30 NT + 23 AS + 6 other) were included in the dimensional analysis of autistic and sensory sensitivity traits. All participants self-reported normal hearing. The study was approved by the Human Research Ethics Committee of The University of Queensland (Approval No.: 2019000119). Participants were compensated at a rate of $20 per hour for their time.

Procedure

Questionnaires

Self-report questionnaires included the Autism Quotient (AQ) questionnaire (Baron-Cohen et al., 2001) and the Sensory Perception Quotient (SPQ) Questionnaire (Tavassoli, Hoekstra, & Baron-Cohen, 2014), which were used to measure autistic traits and subjective sensory sensitivities, respectively. It is important to note that lower SPQ scores indicate more hypersensitivities. Participants also completed the Beck Anxiety Inventory (Beck et al., 1988) and Beck Depression Inventory (Beck et al., 1961).

Stochastic oddball paradigm

Participants underwent a stochastic frequency oddball paradigm (Garrido et al., 2013) and a simultaneous, visual 2-back task (Sweet, 2011) while their brain activity was measured using EEG. Participants listened to a stream of tones with log-frequencies sampled from two Gaussian distributions with equal means (500 Hz) and different standard deviations (*narrow*: $\sigma_n = 0.5$ octaves; *broad*: $\sigma_b = 1.5$ octaves); see Figure 1. Probe tones of 500 Hz ("standard" tones) or 2000 Hz ("deviant" tones) were embedded in the tone distributions, with each constituting 10% of all tones. Thus, participants listened to a total of 1560 tones in a single condition (i.e., 156 narrow standards, 156 narrow deviant tones). Standard and deviant tones occurred randomly within the stream of tones. All tones had a duration of 50 ms with 10 ms smooth rise and fall periods and inter-stimulus intervals of 500 ms. Participants were instructed to disregard the tones and to focus instead on the visual 2-back task, in which they had to press a button on a keyboard if any letter on the computer screen repeated after 2 letters (visual 2-back task). This experimental component lasted for approximately 30 min and was divided into 4 blocks (2 "narrow" streams and 2 "broad" streams) with short breaks in between each block. The *narrow* and *broad* blocks were counter-balanced across participants. Stimuli were written and delivered using MATLAB version R2018b.

EEG data acquisition and processing

Throughout the auditory oddball experiment, brain activity was recorded using a Bio Semi ActiView EEG system with a 64-channel electrode cap with Ag/AgCl electrodes placed according to the 10-20 international system. Further electrodes were placed on the outer canthi of both eyes, as well as below and above the left eye to measure eye movement. Neural signals were collected at a sampling rate of 1024, 417 Hz bandwidth (3 dB) and 18 dB/octave roll-off. Impedance at each electrode was kept below 20 kΩ. Triggers were marked in the EEG data at the onset of each tone. Raw EEG data were filtered using a band pass filter between 0.5 and 40 Hz. Data were segmented into 500 ms epochs (including 100 ms prestimulus baseline and 400 ms from the onset of the stimulus). Epochs containing artifacts exceeding ±50 V were excluded. Trials were then averaged together by condition (Narrow Standard, Narrow Deviant, Broad Standard, and Broad Deviant) and baseline corrected using a prestimulus interval of 100 ms. Trial rejection rates were similar between the AS group (11.79%) and

NT group (11.86%). Table S2 shows for rejection rates for each condition. We also report standard measurement errors (SME) for event-related potentials (ERPs; Luck et al., 2021). Tables S3 and S4 show SME by participant and group. Data were pre-processed and analyzed using SPM 12 (Wellcome Trust Centre for Neuroimaging, London; http://www.fil.ion.ucl.ac.uk/spm/) in MATLAB version R2018b.

Whole scalp analysis

We undertook a whole scalp analysis using SPM 12. ERPs (i.e., averaged epochs) for each condition and participant were converted into 3D spatiotemporal volumes, by interpolating and dividing the channel data per time point into a 2-dimensional (2D) 32×32 matrix bounded by a scalp map in 10-20 Bio Semi system (which is already within the SPM). Thus, areas outside the scalp map boundaries are not considered. Thus, a scalp map was obtained for each time bin, and the 2D images were stacked according to their prestimulus temporal order. This resulted in a 3D spatiotemporal image volume with scalp $\times$ scalp $\times$ time ($32 \times 32 \times 513$) dimensions per participant. Where one time bin approximated to $\sim$1 ms. More details of ERP conversion to spatiotemporal images and analysis are described in (Litvak et al., 2011).

The spatiotemporal image volumes were modeled using a $2 \times 2 \times 2$ ANOVA, with factors of Group (AS vs. NT), Context (Narrow vs. Broad) and Surprise (Standards vs. Deviants). All statistical comparisons for the whole time–space volume were corrected for multiple-comparisons using a family-wise error rate (FWE) at an alpha level of 0.05.

Single channel analysis (Fz) electrode

To enable comparison with previous literature (Schwartz et al., 2018) we also undertook a single channel analysis by obtaining amplitudes for each condition at the Fz electrode. The MMN was measured as the difference in mean amplitude (μV) between deviants and standards between 125 and 175 ms. This window was defined as 50 ms around the MMN peak at 150 ms, identified in the grand average waveform as in previous studies (Mahajan & McArthur, 2015; Peter et al., 2010). Across the manuscript we use the term delta to indicate the difference in amplitude between Broad and Narrow contexts (e.g., delta MMN = Broad MMN minus Narrow MMN).

We conducted an additional exploratory analysis, given that we observed group differences in a negative deflection in the prediction-error waveforms at 300 ms after stimulus onset. We term this component the N300 as it was a negative deflection at 300 ms. This N300 component was measured as the difference in mean amplitude (μV) between deviants and standards from 300 and 380 ms.

For group analysis we conducted a $2 \times 2 \times 2$ ANOVA with factors of Group (AS vs. NT), Context (Narrow vs. Broad) and Surprise (Deviants vs. Surprise). Bonferroni corrections for tests are applied based on two tests (i.e., MMN and N300). For the trait analysis, we first conducted a regression analysis with delta-MMN or delta-N300 as the outcome variable and AQ scores, SPQ auditory scores and medication use as predictor variables. We included both AQ and SPQ auditory scores in the same model as they were not correlated with each other. Where a significant regression was identified we conducted correlation comparison using R cocor package (Diedenhofen & Musch, 2015) to understand whether the traits relationship with delta-MMN was driven by the standards and deviants on the predictor, we report Pearson and Filon's effect size (z) for these comparisons.

We also conducted Bayesian t-tests (Rouder et al., 2009) and report Bayes Factors with evidence for the Null (BF_{01}). All statistical analyses were run in MATLAB R2018b or R, and figures were created using ggplot2 (Wickham, 2016) in RStudio. We also report post-hoc/observed power calculated using G*Power (Faul et al., 2009).

RESULTS

Participants

Group analysis was done between 23 participants in the AS group (Age $M = 24.35$, $SD = 6.08$; 12 females, 10 males, 1 intersex) and 23 age- and gender-matched NTs (Age $M = 24.04$, $SD = 6.06$; 12 females, 11 males) in the NT group; see Table 1 and Figure 2 for demographic details. Relative to NT controls, the AS group showed more anxiety ($t = 1.697$, Cohen's $d = -0.501$, $p = 0.097$, $BF_{01} = 1.306$) and higher scores for depression ($t = 2.636$, $d = -0.777$, $p = 0.012$, $BF_{01} = 0.248$) and AQ ($t = 6.861$, $d = -2.346$, $p = 1.829 \times 10^{-8}$, $BF_{01} = 2 \times 10^{-6}$). The AS group showed no differences in auditory hypersensitivities (i.e., SPQ auditory subscale score) compared with the NT group ($t = -0.080$, $d = 0.234$, $p = 0.936$, $BF_{01} = 4.560$). One participant reported a co-occurring diagnosis of ADHD, and another participant reported a diagnosis of both depression and anxiety disorder. No other participants had received any other diagnosis. Eleven participants in the AS group reported using antidepressants or anti-anxiety medication (e.g., Escitalopram, Venlafaxine, Setraline, Amitriptyline or similar drugs within the serotonin reuptake inhibitor class), and 6 participants reported ADHD medication (i.e., methylphenidate class). There was no significant difference in the performance between the two groups on the 2-back task, as reflected by reaction times

TABLE 1 Demographic details

	Neurotypical (NT) group ($n = 23$)			Autism spectrum (AS) group ($n = 23$)			Total sample ($n = 59$) (30 NT + 23 AS + 6 other)		
Variable	***M***	***SD***	**Range**	***M***	***SD***	**Range**	***M***	***SD***	**Range**
Age (years)	24.04	6.06	18–35	24.35	6.08	18–35	25.51	6.10	18–35
Sex at birth (F/M/intersex)	12/11/0			12/10/1			33/25/1		
Gender (F/M/other[a])	10/11/2			9/11/3			28/26/5		
Autism quotient (AQ)	17.43	7.51	4–29	34.96	7.43	21–46	26.19	11.05	4–46
SPQ total score	110.30	28.21	49–163	106.04	27.40	58–162	106.36	27.20	49–163
SPQ auditory subscale	26.91	6.25	15–38	26.78	4.67	19–34	26.76	5.46	15–38
Beck anxiety score	11.91	12.55	0–55	18.13	12.29	0–44	14.71	12.24	0–55
Beck depression score	6.83	7.70	0–26	15.48	13.73	0–44	10.86	11.33	0–44
Antidepressant use(Y/N)	0/23			11/12			15/44		
ADHD medication (Y/N)	0/23			6/17			8/51		
ADOS score	N/A			6.56	1.85	4–10	5.86	2.29	2–10

Abbreviations: ADOS, Autism Diagnostic Observation Schedule; SPQ, Sensory Perception Quotient.
[a]Other genders include—trans male.

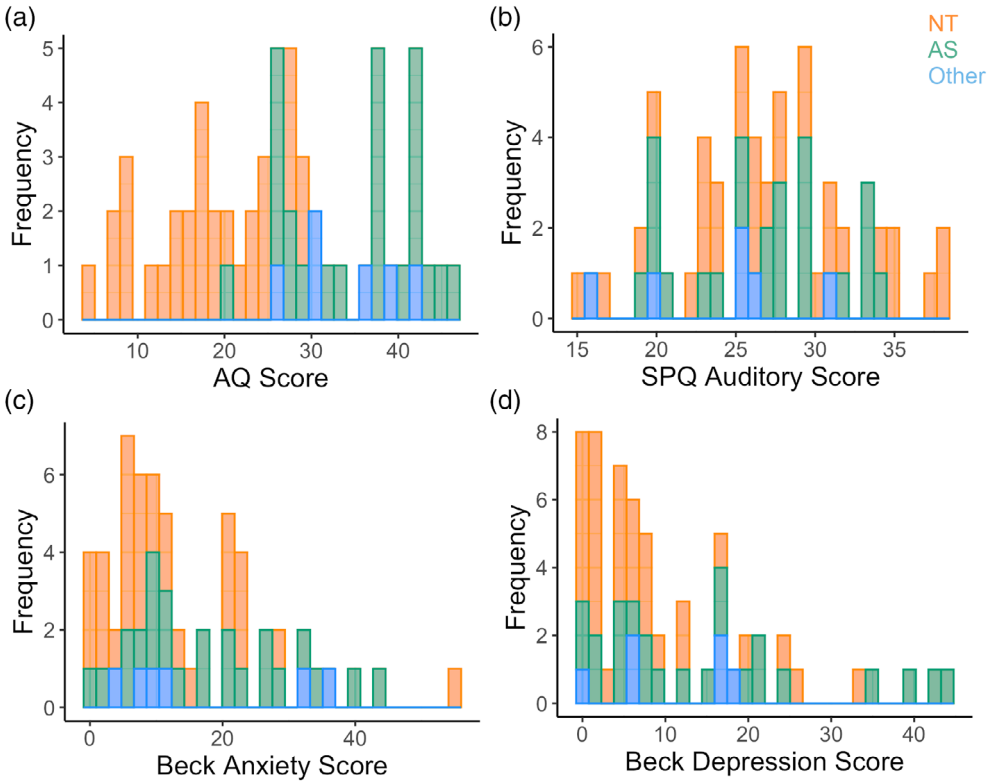


FIGURE 2 Psychometric profile of participants. (a) Autism quotient (AQ), (b) Sensory Perception Quotient-auditory subscale score, (c) Beck Anxiety Inventory Score, (d) Beck Depression Inventory for neurotypicals (NT; orange), confirmed autistic (AS; green) and participants who identified as having received a diagnosis of an autism-spectrum disorder, but which could not be confirmed during interview with a psychologist (other; blue)

($t = -0.036$, $d = -0.011$, $p = 0.971$, $BF_{01} = 4.526$) and accuracy ($t = 0.181$, $d = 0.054$, $p = 0.858$, $BF_{01} = 4.463$). This result implies that attentional level for the N-back task were comparable between the two groups.

In the full sample ($N = 59$; i.e., NT = 30, AS = 23, Other = 6; Figure 2), AQ scores were significantly correlated with anxiety ($r = 0.360$, $p = 0.014$, $BF_{01} = 0.432$) and depression ($r = 0.422$, $p = 0.003$, $BF_{01} = 0.125$), but not with SPQ auditory scores ($r = -0.053$, $p = 0.726$, $BF_{01} = 8.163$). The absence of a significant correlation with SPQ auditory scores is contradictory to literature (Tavassoli, Miller, et al., 2014). Further, it is important to note that SPQ auditory scores were not significantly correlated with anxiety ($r = 0.070$, $p = 0.596$, $BF_{01} = 8.509$) or depression ($r = 0.138$, $p = 0.299$, $BF_{01} = 5.722$) in the full sample.

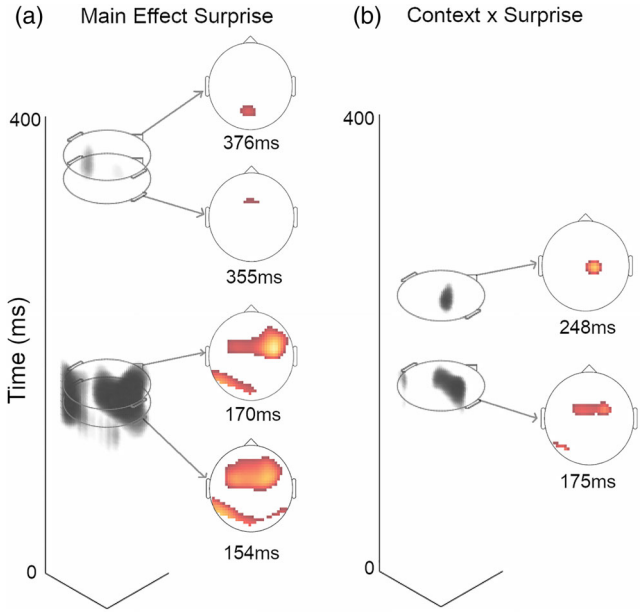


FIGURE 3 Whole scalp results. Spatiotemporal statistical analysis revealed significant clusters for (a) main effect of surprise and (b) context × surprise interaction. 3D *F*-statistic maps demonstrating significant spatiotemporal clusters where spatial dimensions are on the *x*–*y* plane and time is on the *z*-axis. The 2D scalp maps are cross-sections of the 3D maps, denoting time points of interest. Maps are displayed at $p < 0.05$ FWE corrected for the whole space–time volume. FWE, family-wise error rate

EEG: whole scalp analyses

A 2 × 2 × 2 ANOVA of EEG activity revealed significant clusters at the whole-scalp $p_{FWE} < 0.05$ threshold for the main effect of *Surprise* (Figure 3a) arising over fronto-central channels peaking at 165 ms (cluster size $k_E = 10{,}825$, $p_{cluster\text{-}FWE} = <0.001$, $F = 45.38$, $Z = 6.24$), parieto-occipital channels peaking at 149 ms ($k_E = 4006$, $p_{cluster\text{-}FWE} = <0.001$, $F = 40.65$, $Z = 5.93$) and over occipital channels peaking at 366 ms ($k_E = 513$, $p_{cluster\text{-}FWE} = 0.004$, $F = 25.12$, $Z = 4.70$) and at 346 ms ($k_E = 141$, $p_{cluster\text{-}FWE} = 0.017$, $F = 19.95$, $Z = 4.19$). We also observed significant clusters in the *Context* * *Surprise* interaction (Figure 3b) over fronto-central channels peaking at 162 ms ($k_E = 1596$, $p_{cluster\text{-}FWE} = <0.001$, $F = 25.30$, $Z = 4.72$) and 241 ms ($k_E = 463$, $p_{cluster\text{-}FWE} = 0.005$, $F = 23.80$, $Z = 4.58$), and at parieto-occipital channels peaking at 170 ms ($k_E = 137$, $p_{cluster\text{-}FWE} = 0.005$, $F = 21.25$, $Z = 4.32$). Specific channels of significant clusters are listed in Table S5. This confirmed that the paradigm elicited context-specific prediction error responses, as expected.

We found no significant clusters for *Group* * *Surprise* or *Group* * *Context* * *Surprise* interactions at the whole scalp $p_{FWE} < 0.05$ or $p_{uncor} < 0.001$ level, implying no evidence for group differences in the MMN across contexts.

EEG: single channel (Fz) analyses

No group difference in the MMN or N300

A 2 × 2 × 2 ANOVA of mean amplitudes at the Fz channel in the MMN window (see Methods) revealed no significant *Group* * *Context* * *Surprise* (Effect Size $\eta_p^2 = 0.062$, $p = 0.096$, $F = 2.892$, observed power = 0.384), *Group* * *Surprise* ($\eta_p^2 = 0.022$, $p = 0.321$, $F = 1.008$, observed power = 0.166), or *Group* * *Context* ($\eta_p^2 = 0.020$, $p = 0.348$, $F = 0.899$, observed power = 0.153) interactions. Thus, there was no evidence for group differences in MMN, and no differences in context adjustment (delta-MMN). See Figure 4 prediction error waveforms at Fz.

We also investigated amplitudes at a N300 window. We did not observe any significant *Group* * *Surprise* ($\eta_p^2 = 0.098$, $p = 0.034$, $p_{bonf} = 0.068$, $F = 4.776$), *Group* * *Context* ($\eta_p^2 = 0.017$, $p = 0.390$, $F = 0.755$) or *Group* * *Context* * *Surprise* ($\eta_p^2 = 0.018$, $p = 0.369$, $F = 0.823$) interaction.

Auditory sensitivities but not autistic traits are correlated with delta-MMN

A regression analysis with AQ and SPQ scores as predictors of delta-MMN amplitude ($F = 5.823$, $p = 0.005$, $p_{bonf} = 0.010$, observed power = 0.881) revealed that auditory SPQ scores were a significant predictor of delta-MMN amplitudes (*Beta* = 0.419, $p = 0.008$) even when adjusted for AQ Scores, anxiety, depression, and medication use (see Table 2). This indicates that as auditory sensitivities increased (reflected in lower auditory SPQ scores) participants showed a larger difference in MMN between contexts. We also asked whether this delta-MMN relationship with auditory sensitivities could be attributed to the standards or deviants. There was no correlation between the auditory SPQ scores and delta-Standards ($r = -0.192$, $p = 0.145$, $BF_{01} = 3.403$), suggesting that model formation was not modulated by auditory sensitivities. However, there was a weak positive correlation with the delta-deviants ($r = 0.262$, $p = 0.045$, $BF_{01} = 1.335$) (Figure 5) suggesting context-dependent surprise responses increase with increasing sensory sensitivity (i.e., with decreasing SPQ scores). Further, we compared the correlations of SPQ scores with delta-MMN, standards, and deviants. We found a significant difference between the correlation of delta-MMN and SPQ scores and the correlation of delta-standards and SPQ scores ($z = 2.496$, $p = 0.0125$). However, there was no significant difference between the correlation strengths of delta-MMN and SPQ and delta-deviants with SPQ ($z = 0.859$, $p = 0.390$). This suggests that delta-deviants drive the relationship with delta-MMN and SPQ auditory scores.

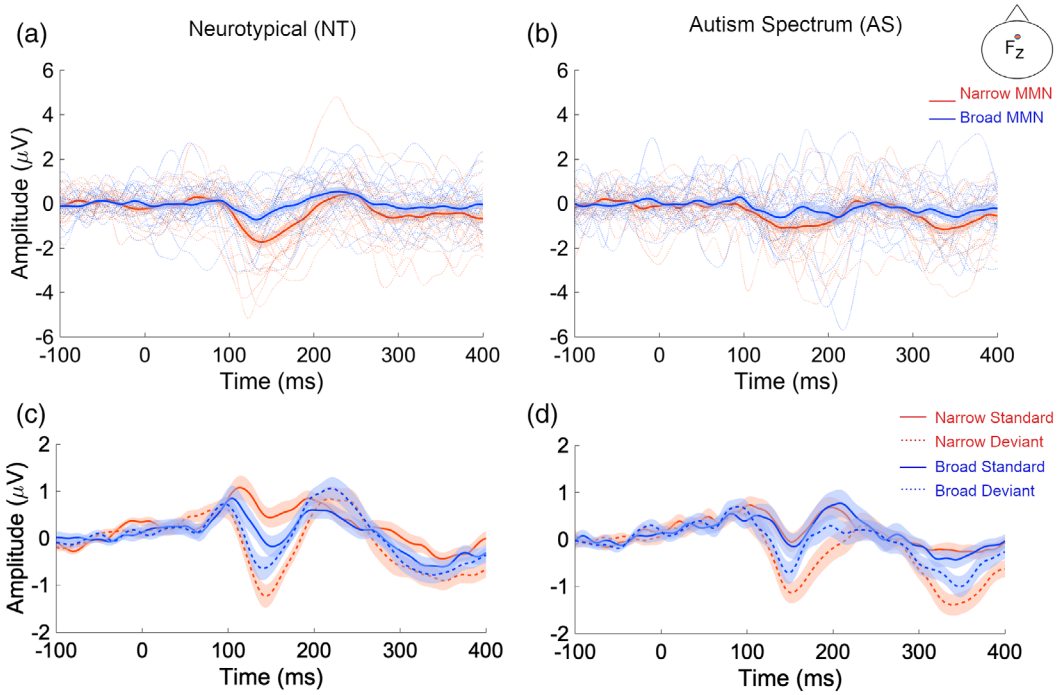


FIGURE 4 Prediction error waveforms at Fz electrode. Mismatch response (difference in deviants > standards) for individual and (a) matched neurotypical (NT) and (b) autistic (AS) groups for narrow (red) and broad (wide) oddball contexts. Dotted lines in lighter colours are waveforms of individual participants and darker colours indicate the average waveform for the group. For each (c) NT and (d) AS group corresponding waveforms for standards (solid line) and deviants (dotted line) are shown for narrow (red) and broad (blue) contexts. Location of Fz electrode on scalp is shown at top right

TABLE 2 Linear regression model predicting delta-MMN amplitude

				95% confidence interval	
Predictor variables	**Beta**	**SE**	***p***	**Lower**	**Upper**
Model 1					
AQ score	0.231	0.018	0.143	−0.010	0.061
SPQ auditory score	0.383	0.030	0.003	0.030	0.150
Model 2					
AQ score	0.042	0.020	0.055	0.002	0.080
SPQ auditory score	0.102	0.033	0.004	0.041	0.169
Beck anxiety	0.009	0.021	0.620	−0.045	0.049
Beck depression	−0.013	0.018	0.463	−0.043	0.030
Anx/Dep medication	−0.339	0.440	0.428	−1.254	0.505
ADHD medication	−0.946	0.698	0.175	−2.373	0.411

Note: Outcome variable: delta-MMN.
Abbreviations: AQ, Autism Quotient; MMN, mismatch negativity; SPQ, Sensory Perception Quotient.

We also found no relationship between ADOS severity scores and delta-MMN ($r = 0.299$, $p = 0.122$, $BF_{01} = 2.093$) or delta-N300 ($r = -0.169$, $p = 0.391$, $BF_{01} = 4.753$).

We also conducted a regression analysis (see Methods) with AQ score and SPQ score as predictors of delta-N300 amplitude (see Table 3). We did not observe any significant predictors of delta-N300 ($F = 1.410$, $p = 0.253$, observed power = 0.533).

No group differences or dimensional associations in variability of amplitude of standards

We also investigated the variability of mean amplitudes of standard trials in the MMN time window across trials. We used the variance (σ^2) of the amplitudes of standards in first 50 trials to determine whether there was variability in standards which might indicate differences in the

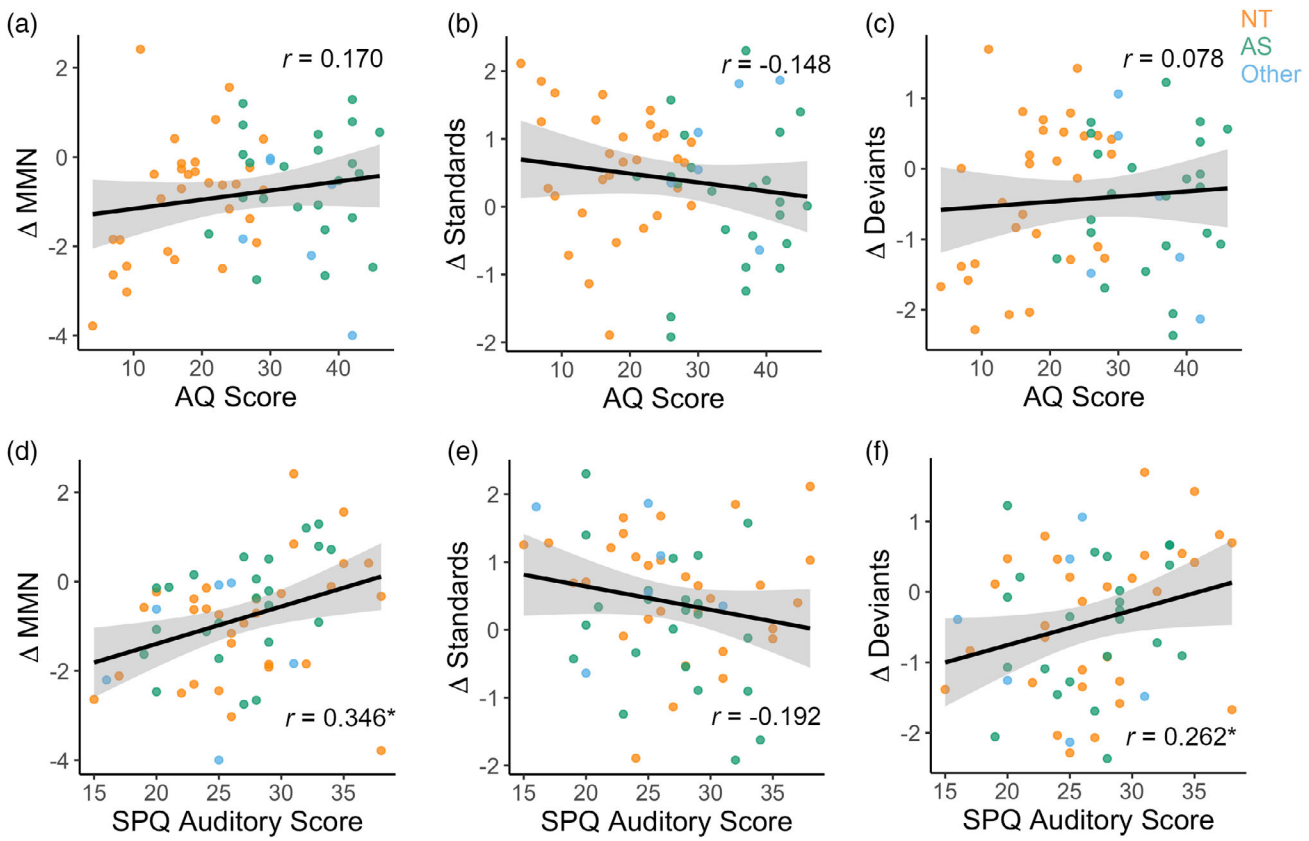


FIGURE 5 Trait analysis. Correlations between autism quotient (top, panels a, b, c) and SPQ auditory subscale scores (bottom, panels d, e, f) with mean amplitude (μV) in the mismatch negativity time window. Differences between broad and narrow conditions are shown for delta-MMN, delta-standards, and delta-deviant amplitudes. Neurotypical (NT; orange), autistic (AS; green) and unconfirmed autistic (other; blue)

TABLE 3 Linear regression model predicting delta-N300 amplitude

				95% confidence interval	
Predictor variables	**Beta**	**SE**	***p***	**Lower**	**Upper**
Model 1					
AQ score	0.170	0.017	0.183	−0.008	0.059
SPQ auditory score	−0.114	0.037	0.409	−0.094	0.052
Model 2					
AQ score	0.039	0.023	0.099	−0.003	0.086
SPQ auditory score	−0.019	0.042	0.645	−0.096	0.068
Beck anxiety	−0.011	0.022	0.555	−0.059	0.032
Beck depression	−0.012	0.017	0.432	−0.047	0.022
Anx/Dep medication	−0.210	0.685	0.757	−1.587	1.052
ADHD medication	−0.320	0.790	0.668	−1.791	1.310

Note: Outcome variable: delta-N300.
Abbreviations: AQ, Autism Quotient; SPQ, Sensory Perception Quotient.

initial learning of a pattern (i.e., the formation of a prior). We found no difference between groups in either the *Narrow Standards* (Mean difference = 15.413, $SD = 17.723$, $t = 0.870$, $d = -0.256$, $p = 0.389$, $BF_{01} = 3.269$) or in the *Broad Standards* (Mean difference = −80.9837, $SD = 104.837$, $t = -0.772$, $d = 0.228$, $p = 0.444$, $BF_{01} = 3.508$) conditions. There was also no significant association between AQ Scores and variability in the *Narrow Standards* ($r = 0.091$, $p = 0.495$, $BF_{01} = 8.768$) and *Broad Standards* ($r = -0.119$, $p = 0.367$, $BF_{01} = 3.768$) conditions; nor did we observe differences between SPQ auditory scores and variability in the *Narrow Standards* ($r = -0.217$, $p = 0.099$, $BF_{01} = 2.533$) and *Broad Standards* ($r = -0.130$, $p = 0.325$, $BF_{01} = 6.049$)

conditions. Finally, there were no differences between ADOS scores and the variance of *Narrow Standards* ($r = 0.033$, $p = 0.867$, BF = 6.759) and *Broad Standards* ($r = 0.092$, $p = 0.647$, $BF_{01} = 6.148$).

DISCUSSION

In this study we aimed to test whether prediction error (i.e., MMN) generation is altered in autism, and whether autistic individuals display anomalies in context adjustment to uncertainty relative to NT controls. We observed no group differences in the classical MMN within conditions. The MMN findings are in line with recent meta-analysis findings (Chen et al., 2020; Schwartz et al., 2018). We note, however, that our findings are not consistent with previous studies, using different tasks to the one employed here, that reported differences in auditory prediction-error signaling in autistic children (Kolesnik et al., 2019), adolescents (Lawson et al., 2015), and young adults (van Laarhoven et al., 2020). Fewer studies have found differences in prediction error generation in adult samples which may correspond to the reduction in reported auditory sensitivities in autistic adults. Further, our findings go against previous studies which have demonstrated that autistic individuals showed increased auditory capacity in that they noticed surprising sounds more than non-autistics (Remington & Fairnie, 2017).

We also investigated the flexibility of prediction errors across contexts with varying uncertainty (low-vs. high-precision contexts). Again, we found no differences between AS and NT groups in adjusting prediction errors between contexts in the MMN window, which is in contradistinction to (Goris et al., 2018), who found a reduction in MMN amplitude. Our findings suggest that both model forming and context adjustment may be intact in autism. Further, while we observed no group differences, we found that autism traits did not predict context adjustment (delta-MMN), but auditory sensitivities did. Our contrasting findings from a group versus trait approach may be due to our AS and NT groups not differing in their SPQ auditory scores. These findings highlight the importance of characterizing sensory symptoms alterations in autistic and NT individuals, and accounting for these differences in group comparisons. Further, the context adjustment association with SPQ scores provides partial evidence for a prediction-error weighting hypothesis as in the HIPPEA model of hypersensitivities specific to autism (Van de Cruys et al., 2014). Importantly as auditory sensitivities increased (reflected in lower auditory SPQ scores) participants showed a larger difference in MMN between contexts. This does not demonstrate an inflexibility to context as would be expected with increased hypersensitivities in the HIPPEA model. Our findings instead maybe explained by an increase in auditory capacity as hypersensitivities increase. Brinkert and Remington (2020) used an auditory load task to assess perceptual capacity and found an increase in perceptual capacity in a group of individuals with hypersensitivity but a decrease in perceptual capacity in a group with hypo-sensitivities as well as no relationship between auditory capacity and autistic traits. Further studies including autistic and non-autistic participants with sensory processing disorders may help to shed light on the relationship with MMN at the intersection of autism and sensory sensitivities.

We also provide moderate evidence in support that autistic adults showed no group differences in the variability of standards. Taking a Bayesian view on sensory learning, this indicates that autistic adults can form models of the environment (form a prior) similar to non-autistics. While this has not been shown previously using MMN specifically, evidence for intact priors in autism has been found for the visual (Croydon et al., 2017; Karvelis et al., 2018; Pell et al., 2016; Van de Cruys et al., 2018) and tactile (Cannon et al., 2021) modalities. Thus we provide moderate evidence against the Hypo-priors model, which take a Bayesian view of sensory learning and suggests that autistic perception is characterized by the formation of less precise models of the world (Pellicano & Burr, 2012). Our findings also provide evidence inconsistent with a non-Bayesian theory, the sensory unreliability hypothesis of autism, which suggests that autistic individuals exhibit greater trial-to-trial variability in behavioral and cortical sensory responses (Haigh, 2018). Our findings support Butler et al. (2017) but are in contrast to previous studies that show greater trial-by-trial variability in evoked-responses in autism (Dinstein et al., 2012; Haigh et al., 2015; Milne, 2011).

Our study results involving auditory sensitivities deviate from prior literature in at least two important ways. First, in contrast to prior studies, we found no correlation between auditory sensitivities and autistic traits (Tavassoli, Hoekstra, & Baron-Cohen, 2014; Taylor et al., 2020). Second, unlike prior literature, we observed no significant differences in auditory sensitivities between our AS and NT groups (Hazen et al., 2014; Kern et al., 2007; Tavassoli, Hoekstra, & Baron-Cohen, 2014). There may be several reasons for these lack of differences in auditory sensitivities in our sample characteristics that could contribute to this. Another consideration may be in the subjective measuring tool we used. The SPQ differs from other self-report tools that assess sensory behaviors in that it is the only tool which focuses on basic sensory discrimination and detection. By aiming to quantify sensory thresholds they theoretically parallel psychophysical assessment approaches (DuBois et al., 2017). Thus, other more widely used self-report tools which measure behavioral symptoms such as the Adult Sensory Profile (Brown et al., 2001) and the Glasgow Sensory Questionnaire (Robertson & Simmons, 2013) might have yielded different results. Finally, in addition to the SPQ, our study would have benefitted from the use of psychophysics tasks such as auditory pitch discrimination tasks, aimed at obtaining more objective measures of sensory thresholds.

There are a number of caveats to consider in relating our findings to hypotheses arising from Bayesian views of sensory learning in autism. First, MMN amplitudes have been shown to be influenced by factors such as medication. More than 50% of our AS participants reported antidepressant use. Selective serotonin reuptake inhibitor (SSRI) class drugs, such as Escitalopram, have been shown to increase MMN amplitudes in healthy individuals (Oranje et al., 2008; Wienberg et al., 2010). A smaller number of our AS participants also reported use of attention modulating drugs of the Methylphenidate class, such as Ritalin. Methylphenidate has been shown to reduce group differences in ERP indices in children with ADHD due to increased MMN amplitudes (Lawrence et al., 2005; Ozdag et al., 2004). Similarly, our ASD group may have displayed larger MMN amplitudes as an effect of attention-modulating medication, which may account for the absence of group differences in the present study. Further study of prediction errors using MMN in larger samples of drug-naïve autistic participants will be important for understanding the relative contributions of medication to sensory processing. Further, MMN amplitudes have been shown to decline with age (Cheng, Baillet, et al., 2013; Cheng, Hsu, & Lin, 2013). This limits comparability of our findings in an adult sample with MMN findings in children. A global theory of prediction error adjustment relevant to autism would ideally be validated across age groups. We also did not objectively assess the hearing profiles of participants. Sensorineural hearing loss has been associated with reduced MMN amplitudes (Oates et al., 2002), and so future work should assess hearing profiles in conjunction with MMN to assess the relationship with sensitivities. It is important to note that the AQ also has its limitations in quantifying autistic traits as it has also been shown to be closely associated with anxiety, not only autistic traits (Ashwood et al., 2016). Finally, we also note that the group analysis specifically may be underpowered to detect differences between groups. We recommend future studies in this area to include larger samples encompassing different degrees of sensory sensitivity and autistic trait scores.

In summary, our study demonstrates the importance of undertaking a dimensional approach, specifically taking sensory sensitivities into account when investigating uncertainty under different contexts. Schwartz et al. (2018) pointed out that within-group variability in MMN anomalies may serve as a better avenue for study than between-group analysis. Our findings thus provide evidence for reduced context updating with sensory sensitivities and underpin the importance of studying sensory learning in autism under varying contexts.

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CONFLICT OF INTEREST

The authors have declared that there are no conflicts of interest in relation to the subject of this study.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available at UQ eSpace: Randeniya, R., Mattingley, J. B., and Garrido, M. I. (2022). Dataset: Increased context adjustment is associated with auditory sensitivities but not with autistic traits. The University of Queensland. Data Collection. https://doi.org/10.48610/3e62869

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SUPPORTING INFORMATION

Additional supporting information may be found in the online version of the article at the publisher's website.

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