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Sensory Prediction Errors Are Less Modulated by Global Context in Autism Spectrum Disorder

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ABSTRACT

BACKGROUND: Recent predictive coding accounts of autism spectrum disorder (ASD) suggest that a key deficit in ASD concerns the inflexibility in modulating local prediction errors as a function of global top-down expectations. As a direct test of this central hypothesis, we used electroencephalography to investigate whether local prediction error processing was less modulated by global context (i.e., global stimulus frequency) in ASD.

METHODS: A group of 18 adults with ASD was compared with a group of 24 typically developed adults on a well-validated hierarchical auditory oddball task in which participants listened to short sequences of either five identical sounds (local standard) or four identical sounds and a fifth deviant sound (local deviant). The latter condition is known to generate the mismatch negativity (MMN) component, believed to reflect early sensory prediction error processing. Crucially, previous studies have shown that in blocks with a higher frequency of local deviant sequences, top-down expectations seem to attenuate the MMN. We predicted that this modulation by global context would be less pronounced in the ASD group.

RESULTS: Both groups showed an MMN that was modulated by global context. However, this effect was smaller in the ASD group as compared with the typically developed group. In contrast, the P3b, as an electroencephalographic marker of conscious expectation processes, did not differ across groups.

CONCLUSIONS: Our results demonstrate that people with ASD are less flexible in modulating their local predictions (reflected in MMN), thereby confirming the central hypothesis of contemporary predictive coding accounts of ASD.

Keywords: ASD, Autism, EEG, Mismatch negativity (MMN), Prediction error, Predictive coding

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Autism spectrum disorder (ASD) is characterized by severe difficulties in social interaction and communication as well as nonsocial symptoms such as repetitive patterns of behavior and hyper- or hyposensitivities to sensory stimuli (1). Ever since autism was first described, researchers have tried to identify a single cognitive deficit that can account for this heterogeneous set of symptoms. However, most theories have provided an explanation for either the social or sensory symptoms but failed to explain both. A recent evolution in ASD research attempts to fill this gap by using predictive coding to explain both social and sensory symptoms in ASD [(2–5); for a review, see (6)]. Central to many of these accounts [e.g., (2,4)] is the hypothesis that people with ASD show an inflexible precision in regulating their low-level sensory expectations. Here, we put this hypothesis to the test.

The predictive coding framework states that the brain constantly makes predictions about the world and processes incoming sensory information in light of those predictions (7,8). When incoming information is different than expected, the brain generates what is referred to as a prediction error (i.e., a surprise), and this signal can then be used to adapt future predictions. However, an adaptive use of prediction errors to guide behavior requires distinguishing between behaviorally relevant and irrelevant prediction errors based on contextual information. Sometimes prediction errors signify that there are learnable regularities in the environment and predictions should be adapted, while in other contexts prediction errors are just noise in the environment that can be ignored. For example, the unexpected noise of someone clearing her throat could mean something important when in a company meeting (i.e., a social signal) but is more likely to be irrelevant to us when sitting in the doctor's waiting room. Central to our everyday behavior is our ability to dissociate these more informative prediction errors from less informative ones. The mechanism through which this relative weight of bottom-up prediction errors and top-down predictions can be adjusted is often referred to as precision (7). Importantly, it has been proposed that the weighting of prediction error signals is less flexibly adjusted across contexts in ASD (2,4). In other words, ASD is hypothesized to be characterized by an inflexible precision of prediction errors. This hypothesized deficit naturally explains key symptoms of ASD such as difficulties in cognitive flexibility, altered perceptual processing, repetitive behavior, and resistance to change. In fact, according to these authors, a broad range of ASD symptoms can be understood in light of

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this deficit, including both sensory and social problems [see (2,4)]. There are differences in the specific hypotheses of these different theories. For example, Van de Cruys and colleagues (4,5,9) emphasized not only inflexible but also consistently high precision of prediction errors. Lawson and colleagues (2,10) similarly hypothesized that there is an inflexible precision of prediction errors due to a failure of attenuating sensory precision. However, while different proposals [e.g., (3,11); for a review, see (6)] have different accents, all of them are based on the idea that there is an inflexible precision of predictions and prediction errors in ASD.

Recent studies already provided preliminary support for the assumption that in individuals with ASD there is an inflexible weighing of the relative importance of sensory prediction errors [e.g., (12–18)]. However, these studies focused mostly on higher-level mechanisms by testing the effects of social manipulations [e.g., (12,17)], expectancy instructions (13), or volatility manipulations in decision making (14). Some behavioral studies focused on more low-level processes by studying movement adaptation (15) or Gabor orientation discrimination (18), but no study has investigated this hypothesis focusing on early sensory processing, which we believe requires the use of neural measurements rather than behavioral paradigms. Therefore, we used a well-validated hierarchical predictive coding task as first introduced by Bekinschtein et al. [(19); see also (20,21)] to dissociate the relative weighting of sensory prediction errors in an ASD group and a matched typically developed (TD) control group. The paradigm is an auditory oddball task where participants are presented with sequences of five tones, which consist of either local standards (five identical tones) or local deviants (four identical tones followed by a deviant tone). To determine whether the processing of local deviants can be modulated by global context, the relative frequency of local deviants versus local standards is manipulated across blocks, thereby creating different levels of global expectancy; in some blocks local deviants are rare, while in other blocks they occur frequently. Following the reasoning that less frequent events are often more relevant to us (i.e., they do not match with our global predictions), this context manipulation should induce a larger surprise reaction in blocks where the surprising event is more rare.

Indeed, using this manipulation, a well-replicated finding in TD adults is that event-related potential components of local prediction error processing can be modulated by this global context. To show this, Bekinschtein et al. (19) and others dissociated two event-related potential components. First, the onset of the fifth tone in local deviant sequences elicits a very early component called the mismatch negativity (MMN) (22), believed to reflect early sensory prediction error processing in a preattentive and nonconscious manner (21,23,24). Because the MMN is also elicited by unexpected tone omissions, it is thought to represent predictive activity rather than just adaptation to repeated stimuli (21). Second, a later positive deflection with a parietal distribution, the P3b, is elicited, and this reflects top-down attention directed toward a stimulus (25). Consistent with this dissociation, Bekinschtein et al. (19) demonstrated that the P3b is sensitive to global expectancies, while the MMN is sensitive to local deviances. However, as shown by Wacongne et al. (21), the MMN is also influenced by the global context, showing a smaller amplitude when local deviances are more frequent. This effect shows that processing of local prediction errors is modulated by global context in TD individuals. If individuals with ASD indeed show an impeded ability to modify their local predictions, we expect to see that the amplitude of the MMN should be less modulated by global context in the ASD group compared with the TD group. For the P3b following global deviances, we did not have specific hypotheses.

METHODS AND MATERIALS

Participants

In total, 25 adults with ASD (17 men) and 30 TD adults (22 men) participated in the study. All participants were right-handed and free of hearing problems. Participants in the TD group were screened to have no reported history of neurological or psychiatric disorders. Because the focus was on high-functioning ASD, all participants had a full-scale IQ above 80. A score above the cutoff on either the Autism Spectrum Quotient (32 or higher) (26) or the Social Responsiveness Scale–Adult version (T-score of 61 or higher) (27) was used as an exclusion criterion in the TD group (see also Figure 1). These are the cutoffs as described in the original questionnaires and are meant to screen for autistic traits in an adult population. Therefore, 6 participants were excluded, resulting in 24 remaining TD adults. All participants gave written informed consent before participation and were financially compensated. The study was approved by the local Ghent University ethics committee.

All adults with ASD had received a clinical diagnosis of ASD (n = 19), autistic disorder (n = 2), or Asperger's syndrome (n = 4), prior to the experiment, by an independent clinician or multidisciplinary team. For 1 subject, the clinical diagnosis of ASD was withdrawn by an independent clinician during the study; therefore, this participant was excluded, leaving a total of 24 participants in the ASD group. Within this group, the diagnosis was verified with the Autism Diagnostic Observational Schedule (ADOS) (28) Module 4 by a trained researcher using the revised scoring algorithm (29). In line with earlier ASD studies (30–33), we included only participants with ASD who scored 1 point below the cutoff for ADOS total score or higher (see also Figure 1). As a result, 6 participants were removed from the ASD group. Thus, final data analysis was carried out on 18 subjects in the ASD group (13 men) and 24 subjects in the TD group (16 men). There was no significant difference in age between the two groups, $t_{39.88} = 1.02$, $p = .31$.

Importantly, the results did not change in a statistically significant way when we used the ADOS total score cutoff as the exclusion criterion, resulting in 17 participants in the ASD group. Similarly, our main finding reached the same level of significance when keeping all 24 adults with ASD in the analysis (see Results).

Intelligence was assessed by using the Kaufman 2 short form Wechsler Adult Intelligence Scale–third edition (WAIS-III) as a reliable measure of IQ in ASD (34). For 3 participants with ASD, we used a full WAIS-III that was available and completed within the past 5 years. There was no significant difference in IQ between the two groups, $t_{31.14} = 2\ 0.83$, $p = .41$.

Task and Stimuli

We used the paradigm by Bekinschtein et al. (19) that dissociates two types of local deviants: local deviants that appear

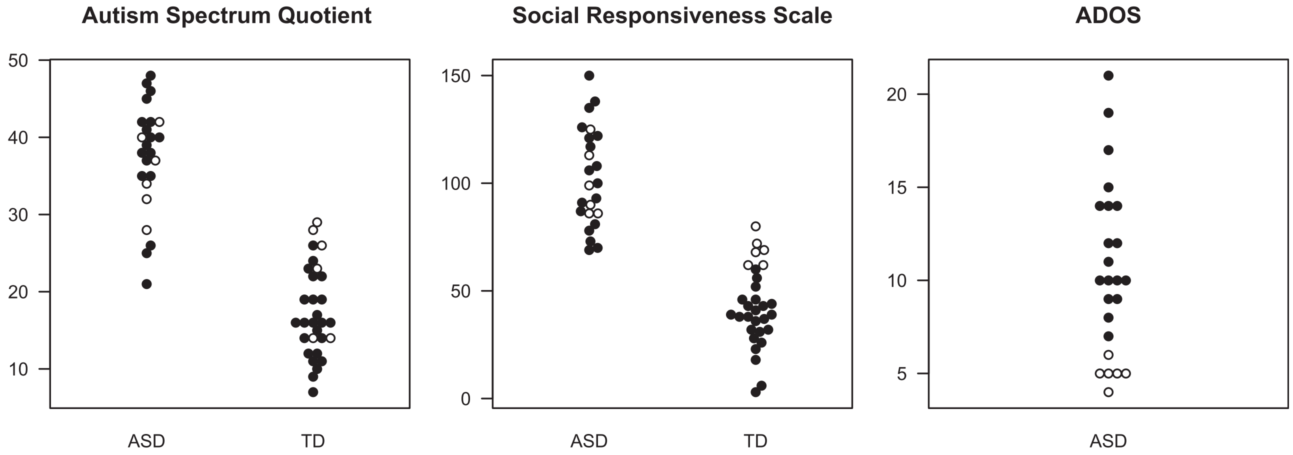

Figure 1. Total scores on the Autism Spectrum Quotient, Social Responsiveness Scale–Adult version, and Autism Diagnostic Observation Scale–Module 4 (ADOS). The unfilled dots represent the individuals who were excluded. ASD, autism spectrum disorder group; TD, typically developed group.

frequently in the global context and local deviants that are rare in the global context (see Figure 2). Stimuli consisted of two sequences of five sounds: local standards (five identical tones: xxxxx) and local deviants (four identical tones followed by a different tone: xxxxY). These sequences were presented in two types of blocks. In the low-frequency deviant blocks, local standards (xxxxx) were presented with a high frequency (initially 100% and then 80%; see below) and local deviants (xxxxY) were rare (initially 0% and then 20%). The opposite applied for the high-frequency deviant blocks (see Figure 2). This way, sequences could also be dissociated by being either a global standard (the sequence that appears most frequently in a block: xxxxx in low-frequency deviant blocks and xxxxY in high-frequency deviant blocks) or a global deviant (the sequence that is rare in a block: xxxxY in low-frequency deviant blocks and xxxxx in high-frequency deviant blocks). In this design, thus, the local versus global distinction is operationalized temporally: a smaller (e.g., more local) versus larger (e.g., more global) time window [see also (19–21,35,36)].

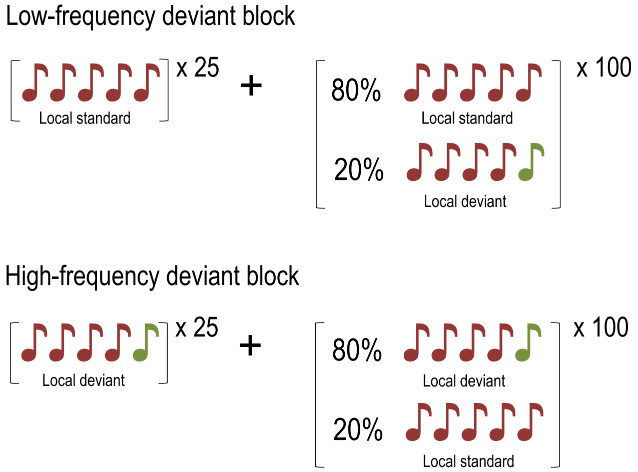

Figure 2. Task and procedure. The experiment consisted of two block types: the low-frequency deviant blocks and the high-frequency deviant blocks. In the low-frequency deviant blocks, the local standard sound sequences (five identical tones: xxxxx) were most frequent, while the local deviant sound sequences (four identical tones and a different tone: xxxxY) were rare. In the high-frequency deviant blocks, the local deviant sound sequences were frequent, while the local standard sound sequences were rare. This way, local and global deviances were created. A local deviance refers to a deviant tone after four identical tones (xxxxY). A global deviance refers to the sequence that is rare within the context of a block.

Participants were presented with six low-frequency deviant blocks and six high-frequency deviant blocks, resulting in a total of 12 blocks (each with an approximate duration of 3 minutes). Each block consisted of 125 trials, where the first 25 trials consisted of the frequent sequence in order to establish the global rule. The following 100 trials consisted of 80 frequent sequences and 20 rare sequences in random order. Between blocks, there were self-paced breaks, resulting in a total duration of approximately 40 minutes.

The sequences were composed using two tones, each consisting of three superimposed sine waves (350, 700, and 1400 Hz for tone A or 500, 1000, and 2000 Hz for tone B). Each tone was 50 ms long and stimulus onset asynchrony was 150 ms, resulting in sequences of 650 ms. Intertrial intervals were jittered between 700 and 1000 ms. Each participant received six replications of each block type (low-frequency deviant or high-frequency deviant) in which x = A and Y = B in three blocks and x = B and Y = A in the other three blocks. Block order was completely randomized. All stimuli were presented binaurally with an intensity of 70 dB through electroencephalogram (EEG)-compatible insert earphones (ER-3C, MedCaT, Klazienaveen, The Netherlands) using Tscope5.

Procedure

During the first test session, participants first completed the EEG experiment. Then, they took part in a second EEG experiment of approximately 10 minutes that was conducted in the context of another study for which the results will be presented elsewhere. Next, they filled in three questionnaires measuring autistic traits: the Autism Spectrum Quotient [(26); Dutch version: (37)], the Social Responsiveness Scale–Adult version [(27); Dutch version: (38)], and the Adolescent/Adult

Sensory Profile [(39); Dutch version: (40)]. For the TD participants, the WAIS-III was completed at the end of this session. For the ASD participants for whom the ADOS and WAIS-III were not present in our database (14 of 24), the ADOS and WAIS-III were completed during a second session on a later day.

EEG Recordings and Analyses

EEG activity was recorded at a sample rate of 1024 Hz using an ActiveTwo EEG amp (BioSemi) from 64 active Ag/AgCl scalp electrodes placed according to the international 10–20 system. Additional electrodes were applied at the mastoids, near the canthi and above and below the left eye. The data were referenced online to the common mode sense electrode. Data were recorded in an electrically shielded chamber. Electrode offsets were kept between 2 20 and 20 mV at all electrodes.

EEG data analysis was performed using EEGlab 13.6.5b in MATLAB R2014b (The MathWorks, Inc., Natick, MA) and using R 3.3.1 in RStudio 0.99.903 (RStudio, Boston, MA). Bad electrodes were determined visually in the beginning of the preprocessing pipeline. We excluded an electrode only when it had a very obviously distorted signal (because it was broken or not well connected). Data were first rereferenced to the average of all scalp electrodes (with the exclusion of malfunctioning channels), downsampled to 500 Hz, and filtered with a notch filter at 48 to 52 Hz, a high-pass filter at 0.5 Hz, and a low-pass filter at 30 Hz. Trials were then epoched from 200 ms before onset to 1350 ms after onset of the first tone. Independent component analysis was run on the epoched data on scalp electrodes (with the exclusion of bad channels) to remove eye movement components. Subsequently, bad electrodes were interpolated using spherical interpolation. Epochs exceeding an amplitude of 6 100 mV were rejected. When more than 10% of epochs were rejected due to amplitudes exceeding 6 100 mV in a specific electrode, we started preprocessing again excluding this electrode. Finally, a baseline correction was applied from 2 200 to 0 ms relative to onset of the fifth tone (13,20). There was no significant difference in the final number of trials between the TD and ASD groups for all four conditions (all ps . .45).

MMN was defined as the mean amplitude (6 20 ms) surrounding the peak negativity between 80 and 220 ms after the fifth stimulus onset, in the difference wave of the local deviants minus local standards, for each participant, electrode, and block type separately [see also (41)]. The later and more sustained P3b component was calculated to study global deviance and defined as the mean amplitude in the fixed interval 400 to 650 ms after fifth stimulus onset (interval based on visual inspection of topographies).

Repeated-measures multivariate analyses of variance (MANOVAs) were conducted on the components' mean amplitude within their respective time frames. As outlined above, and similar to Bekinschtein et al. (19), we analyzed how block type modulated the effects of local deviance on the MMN, and we analyzed the effects of global deviance on the P3b. Specifically, the MMN analyses included group (ASD or TD) as a between-subject factor and block type (low-frequency deviant or high-frequency deviant) as a within-subject factor. Additional within-subject factors were included to account for electrode selection. Based on earlier studies and topographic maps in our study, we included data of nine frontocentral electrodes (F3, Fz, F4, FC3, FCz, FC4, C3, Cz, and C4) for the MMN, using row (3 levels: F, Fc, or C) and laterality (3 levels: left-3, central-z, or right-4) as additional factors in the MANOVA (42,43). For the P3b, the MANOVA included group (ASD or TD) as a between-subject factor and stimulus type (global deviant or global standard) as a within-subject factor. To be consistent with the MMN analysis, data of nine posterior electrodes (CP3, CPz, CP4, P3, Pz, P4, PO3, POz, and PO4) were included for the P3b, with row (3 levels: CP, P, or PO) and laterality (3 levels: left-3, central-z, or right-4) as within-subject factors.

RESULTS

Mismatch Negativity

First, we investigated whether there were differences between TD and ASD participants in the context-dependent modulation of local prediction error processing, as measured with the MMN. The repeated-measures MANOVA on nine frontocentral

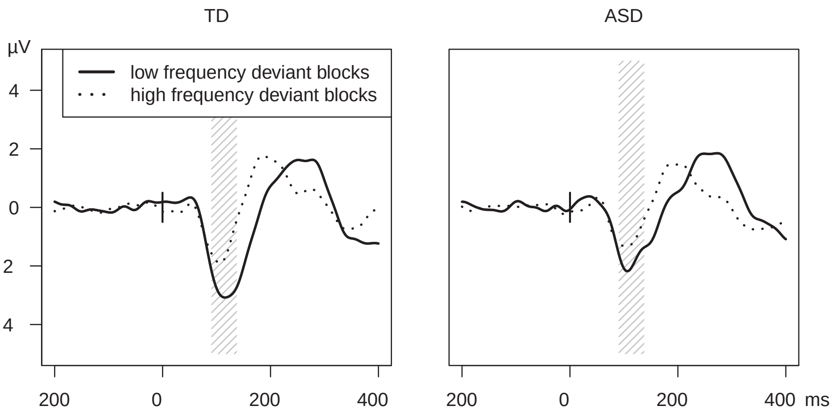

Figure 3. Event-related potential time course of the local effects at Fz. The time course of the local effect (deviant minus standard) on electrode Fz is shown for each block type. The vertical line at 0 ms indicates the onset of the fifth sound. The gray zone indicates 20 ms before the mismatch negativity peaks until 20 ms after these peaks. The difference in mismatch negativity mean amplitude between low-frequency deviant blocks and high-frequency deviant blocks is significantly smaller for the autism spectrum disorder (ASD) group than for the typically developed (TD) group. Plots for other electrodes can be found in Supplemental Figures S1 and S2.

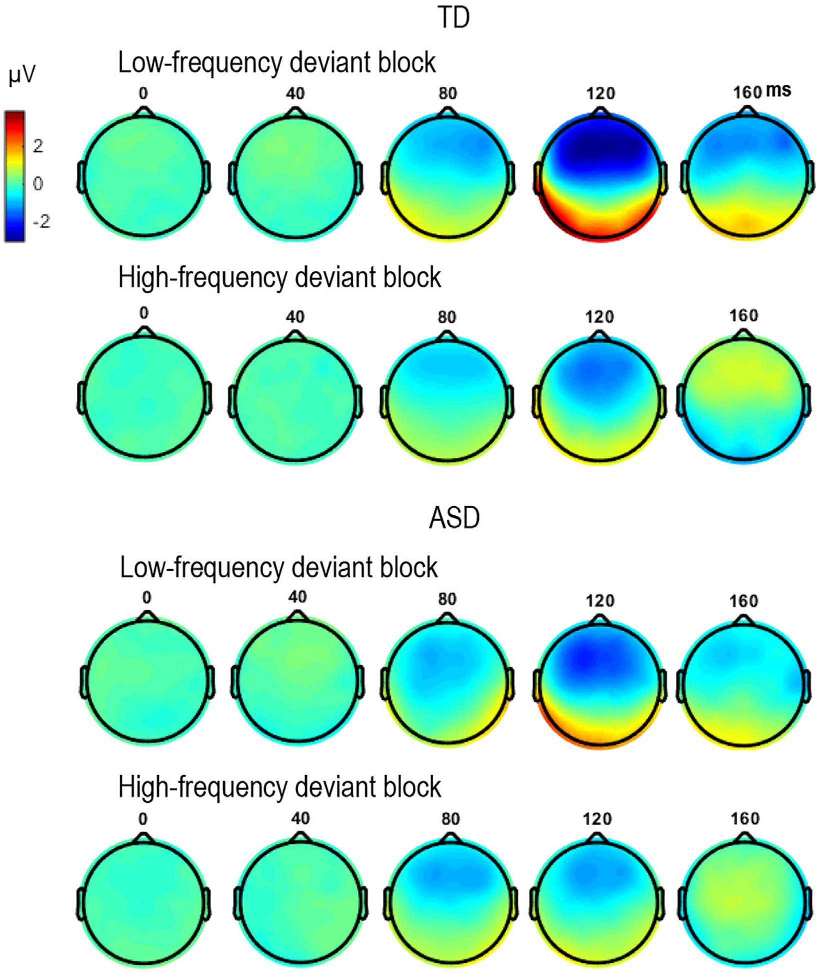

Figure 4. Topographies of the brain responses following local deviances. The topographies show the difference between local deviances and local standards. On the left side, 0 ms indicates the onset of the fifth sound. The difference in mismatch negativity mean amplitude between low-frequency deviant and high-frequency deviant blocks is significantly smaller for the autism spectrum disorder (ASD) group than for the typically developed (TD) group.

electrodes revealed a significant main effect of block type, $F_{1,40} = 99.23$, $p < .001$, indicating a larger MMN amplitude in low-frequency deviant blocks (mean = -2.16, SD = 1.33) compared with high-frequency deviant blocks (mean = -1.21, SD = 0.98), which replicates the earlier observation that the MMN, as a measure of sensory prediction errors, is modulated by the global context (21). Crucially, however, this effect of block type further interacted with group, $F_{1,40} = 6.34$, $p = .02$. The Bayes factor for this interaction was 6.84, which is considered substantial to strong evidence for the alternative hypothesis (44). Follow-up analyses showed that while block type significantly modulated the MMN in both groups (TD: $F_{1,23} = 79.64$, $p < .001$; ASD: $F_{1,17} = 29.94$, $p < .001$), this effect was larger for the TD group than for the ASD group (difference between conditions: TD: mean = -1.14, SD = 0.99; ASD: mean= -0.68, SD = 0.82) (see Figures 3 and 4). Follow-up MANOVAs on each block type separately indicated that there was a significant effect of group in the low-frequency deviant block, $F_{1,40} = 5.43$, $p = .02$, indicating a smaller MMN for the ASD group (mean = -1.76, SD = 1.02) compared with the TD group (mean = -2.45, SD = 1.45). In the high-frequency deviant block, however, there was no effect of group, $F_{1,40} = 0.96$, $p = .33$.

Furthermore, a main effect of group trended to significance, $F_{1,40} = 3.34$, $p = .08$, suggesting a lower overall MMN in the ASD group (mean = -1.42, SD = 0.97) than in the TD group (mean = -1.88, SD = 1.41). Last, there was a significant main effect of row, $F_{2,39} = 31.94$, $p < .001$, and laterality, $F_{2,39} = 5.35$, $p = .009$, and a significant interaction between block type and row, $F_{2,39} = 7.01$, $p = .003$. However, the above-mentioned effects of group or group by block type did not interact significantly with row and/or laterality (see also Supplemental Figures S1 and S2).

When all 24 ASD participants were included in the analysis, we again found an interaction between block type and group, $F_{1,46} = 6.15$, $p = .02$. The other effects were also highly similar to the above-mentioned analysis; there was a significant main effect of block type, $F_{1,46} = 131.38$, $p < .001$, group, $F_{1,46} = 5.63$, $p = .02$, laterality, $F_{2,45} = 6.69$, $p < .01$, and row, $F_{2,45} = 38.48$, $p < .001$, and an interaction between block type and row, $F_{2,45} = 11.39$, $p < .001$.

P3b

Having established that the MMN, as a measure of local prediction error processing, is less modulated by global context in ASD, we also wanted to investigate whether the later P3b component, as a measure of more conscious top-down attention, showed differences between the ASD and TD groups. The MANOVA revealed a significant main effect of stimulus type, $F_{1,40} = 48.04$, $p < .001$, indicating a larger P3b for global deviants (mean = 0.15, SD = 0.68) than for global standards (mean = -0.35, SD = 0.60). Crucially, this effect of stimulus type did not interact with group, $F_{1,\,40} = 1.53$, $p = .22$, nor was there a main effect of group, $F_{1,40} = 1.29$, $p = .26$ (see Supplemental Figure S3). Furthermore, there was a significant effect of row, $F_{2,39} = 38.00$, $p < .001$, a significant interaction between row and stimulus type, $F_{2,39} = 7.77$, $p = .001$, and a significant interaction between row and laterality $F_{4,37} = 3.97$, $p = .009$.

DISCUSSION

Recent accounts of ASD [e.g., (2,4)] attempt to explain both social and sensory symptoms in ASD by using the predictive coding framework. Central to these accounts is the hypothesis that the weighting of different prediction errors is less flexibly adjusted to the context in ASD. This study investigated this central hypothesis by using a well-validated hierarchical predictive coding paradigm that investigates the context-dependent modulation of the MMN, an event-related potential component reflecting early sensory prediction errors. While both the adult ASD and control groups showed a context-dependent modulation of MMN amplitude (i.e., larger MMN amplitudes for local deviants in the low-frequency deviant blocks compared with the high-frequency deviant blocks), this modulation was less pronounced in the ASD group. These results confirm the hypothesis of less flexible context-dependent weighting of prediction errors in ASD. In contrast, the P3b in response to global deviants did not differ between the ASD and TD groups, suggesting comparable top-down expectations between the ASD and TD group. However, this should be confirmed in future studies investigating the effect of unannounced changes in global frequency in more detail. In sum, these findings provide much-needed support for the key hypothesis of recent ambitious accounts of ASD (2,4).

Our results are concordant with earlier findings of studies investigating contextual modulations of sensory processing in ASD [e.g., (12,15–18)]. For example, a recent study by Lawson et al. (14) showed that individuals with ASD have a tendency to overestimate the volatility of the environment instead of developing correct contextually guided expectations. This fits well with our results, showing that participants with ASD were less influenced by contextual manipulations in stimulus frequency. Different from these previous studies, we are the first to study the context-dependent weighting of prediction errors at the (neural) level of early sensory prediction error processing, using the MMN as a preattentive and nonconscious measure of low-level sensory prediction errors. Importantly, our study shows that people with ASD still showed a modulation of the MMN to a certain degree. This suggests that people with ASD show an impairment, rather than total incapacity, in the context-dependent modulation of prediction errors in ASD.

By using different global contexts, we tested the flexibility with which people modulate the MMN. In contrast, previous studies on ASD focused on overall differences in the MMN, often showing mixed results. Specifically, these studies showed a reduced (45–49), comparable (50), and even larger (51–54) auditory MMN in people with ASD. If anything, in line with this first group of studies, our study also seems to hint at an overall reduced MMN in people with ASD. However, these results need to be interpreted with great caution given the inconsistent findings in earlier studies [e.g., (55)]. Because it is difficult to infer what the baseline MMN in ASD is, we cannot distinguish whether our between-group comparisons per block type indicate attenuated prediction errors in low-frequency deviant blocks in ASD or a failure to attenuate prediction errors in high-frequency deviant blocks [see (2)]. Importantly, none of the previous studies on the MMN in ASD investigated the impact of global context on the MMN. Therefore, by taking into account this important factor, the current study might offer an important step toward better understanding the nature of these mixed findings in previous studies.

Finally, we derived our predictions from recent predictive coding theories on autism. However, other theories, such as weak central coherence theory and enhanced perceptual functioning that proposed a bias for local processing (56,57), can also be considered consistent with an observed diminished modulation by the global context. Behavioral studies inspired by these theories have also shown how local processing in an auditory task was less influenced by the global structure in ASD (58,59), while global processing was intact. These findings are consistent with our results, although they do not allow drawing conclusions on the level of early sensory processing.

In sum, we found that the MMN, an EEG component reflecting low-level sensory prediction errors, was less modulated by global context in the ASD group compared with the TD group. These findings confirm a key hypothesis of recent predictive coding accounts of ASD, indicating that individuals with ASD are less flexible in modulating their low-level prediction errors according to more global contexts.

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