Predictive coding in ASD: reduced adaptability of priors to environmental changes

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Abstract

Individuals with autism spectrum disorder (ASD) have been widely reported to show atypicalities in predictive coding, though there remains a controversy regarding what causes such atypical processing. Suggestions range from overestimation of volatility to rigidity in the reaction to environmental changes. Here, we tested two accounts directly using duration reproduction of volatile and non-volatile interval sequences, which were generated from the same set of intervals (i.e., the same ensemble prior). We found that both individuals with ASD and matched controls were able to adjust the weight of the ensemble prior for the reproduction according to the volatility of the sequence. However, the ASD group, as compared to the control group, relied generally less on the prior while also exhibiting marked carry-over of the weight of the prior when environmental volatility changes. Of note, though, four extremes among the 32 ASD individuals showed a reproduction pattern on the opposite end of the spectrum: heavy reliance on the prior in the volatile environment. Overall, our findings suggest that it is not the learning of the prior per se that is compromised in ASD. Rather, a less adaptive response to a change of the volatility regimen or to a volatile environment causes a highly inflexible weighting of prediction errors and the prior.

keywords: autism, predictive coding, prediction errors, adaptability, volatility

1. Introduction

Autism Spectrum Disorder (ASD) is characterized by symptoms in social interaction and communication, and concerning repetitive and stereotypical behaviour [1]. Compared to typically developed individuals (TD), individuals with ASD often find it more difficult to adapt to situations with overwhelming sensory stimulation [2–4]. There is a growing body of evidence of atypical sensory processing in ASD [5,6]. Thus, for instance, several studies have reported correlations between abnormal visual sensory processing and symptom severity [7–10]. The sensory processing abnormalities also extend to time processing [9,11,12], though atypical performance is rather mixed [9,11].

Over the past decade, several accounts based on the predictive coding theory have been formulated to explain sensory atypicalities in autism [13–19]. While it is commonly agreed certain predictive differences occur in ASD, the various accounts differ with respect to the component of predictive processing that is compromised in ASD. To better elaborate the theoretical differences, we should first consider the key idea of predictive coding, namely, that the goal of our perception and action is to update our predictions and minimize prediction errors based on Bayesian inference. In a simple form of Bayesian inference, the perceptual estimate ($D_{percept}$) is an optimal integration of the prediction based on the internal prior (D_{prior}) with the sensory measurement (D_{sense}):

$$D_{percept} = (1 - w)D_{prior} + wD_{sense}, \quad (1)$$

where w is the weight of the sensory measurement based on its precision. With a simple mathematical transformation, the same integration can also be expressed as updating the posterior belief ($D_{posterior}$) based on the prediction error:

$$D_{posterior} = D_{prior} + w\Delta, \quad (2)$$

where the prediction error $\Delta = D_{sense} - D_{prior}$. According to the predictive-coding framework [20], the posterior is adjusted by each prediction error with learning rate w, such that it minimizes future prediction errors. This form is also known as delta learning rule [21]. Incorporating cross-trial dynamic updating within the Bayesian inference framework renders an iterative Bayesian model [22–24] which takes a similar form to Eq. (2). One important implication of Eq. 2 is that, rather than being fixed, the prior is dynamically adjusted trial by trial according to the delta rule to minimize future prediction errors. Accordingly, perceptual estimation can be influenced by recent trial history, known as sequential-dependence effect [25,26].

Pellicano and Burr's attenuated-prior account [13] advocates chronic differences in precision weighting in ASD: individuals with ASD, in general, place less trust on the prior, because their prior beliefs are compromised. Van de Cruys and colleagues [15], on the other hand, have argued that it is the 'High and Inflexible Precision of Prediction Errors in Autism' (HIPPEA) that underlies the observed atypicalities. In a similar vein, Lawson et al. [14] surmised that a failure to attenuate sensory precision may lead to overweighting of sensory inputs in ASD. Although conceptually distinct, these theories agree that individuals with ASD place greater trust on sensory inputs (Eq. 1) or prediction errors (Eq. 2). Supportive evidence has been provided by recent studies, including findings of reduced utilization of predictable information [27,28], needing more time to perform goal-directed anticipations [29], and greatly reduced usage of the prior in duration reproduction [30]. Karaminis and colleagues [30] used

the central-tendency effect as their main tool to disassociate the weights of the sensory measurement and, respectively, the internal prior. The central-tendency effect describes a classical perceptual bias: in a set of duration estimations, short durations tend to be overestimated and long durations underestimated. This can be seen from Eq. 1: if the weight (w) of the sensory input is low, the perceptual estimate ($D_{percept}$) regresses toward the prior (D_{prior}) [17,31]. Thus, a high central tendency means that the prior information is weighted highly (i.e., the learning rate w is low), and observers are less sensitive to the prediction errors generated by new sensory information. Conversely, a low central tendency means that the inference is primarily driven by the sensory inputs (i.e., the learning rate w is high), and observers trust the sensory information over the prior. Using this Bayesian framework, Karaminis et al. [30] demonstrated that, even though children with ASD exhibited a much stronger central-tendency effect compared to matched controls, their observed central tendency was far less than the theoretical model prediction on the basis of their time discrimination – a tool measuring sensory precision. In other words, although children with ASD exhibit a stronger central-tendency effect and their priors are of poorer precision, they place less trust on the prior than predicted by the model – consistent with the 'attenuated-prior' [13] and 'aberrant-precision' [14] accounts.

It should be noted, though, that not all types of prior, or learning of priors, are compromised in ASD. In fact, priors based on experience or top-down knowledge are often preserved in ASD, such as in one-shot learning (e.g., perception of a Dalmatian dog hidden in an image composed of black patches) [32], the influence of gaze cues from previous trials [33,34], and reliance on external-world coordinates in tactile spatial processing [35]. Those mixed findings of usage of priors led Palmer et al. [17] argue that the simple Baysian model has a crucial limitation in assuming an unchanging world; instead, they speculated that the atypicalities in ASD may lie in the differential expectation about the uncertainty of changes in hidden states in a hierarchical inference. To examine how individuals with ASD learn uncertainty about environmental change (in their term 'metavolatility'), Lawson et al. [19] manipulated the cue-outcome association in a discrimination task, in which a probabilistic cue (a high or low tone) predicted the upcoming stimulus (a house or face picture) to which participants had to produce a speeded two-alternative ('house' vs. 'face') response. The cue-outcome probabilistic association could be either stable within a block of trials or randomly switched (i.e., volatile). Compared to matched TD individuals, participants with ASD showed a smaller difference in response times (RTs) and pupil-size changes between the expected and unexpected cue-outcome association, and Lawson et al.'s computational model suggested that individuals with ASD show reduced behavioral surprise and larger metavolatility. Lawson et al. [19] took this as evidence that individuals with ASD have a larger "gain (precision) on cortical responses (prediction errors) under conditions of uncertainty" (p. 1298); as a result, they tend to overestimate volatility, thus rendering unexpected events less surprising.

At the same time, Manning et al. [36] directly compared reward-probability learning between children with ASD and matched TD controls employing a task they adapted for children from an earlier study by Behrens et al. [37]: On each trial, the children had to choose between two different treasure chests, of which only one actually contained a reward. The potential reward in each chest was indicated in advance, but not which of the chests contained

a reward. In some blocks of trials, there was a fixed probability distribution of each chest containing a reward (stable condition), whereas in other blocks the distribution changed regularly (volatile condition). In contrast to Lawson et al. [19], Manning et al. [36] found both groups to display a higher learning rate in the volatile relative to stable condition, without any difference between the two groups (i.e., there were no effects involving the factor Group). Manning et al. [36] concluded that, while "atypical predictive mechanisms account for perception in autism, [this] … may not extend to learning tasks" (p. 10). This echoes a recent finding that at the root of the problem is not a nonspecific learning deficit, but rather that learning other people's intention is compromised in high-functioning adults with ASD [38]. Recent evidence also suggests that, while individuals with ASD are able to extract environmental statistics appropriately, the rate at which their internal priors are updated is greatly reduced compared to neurotypical individuals [39,40].

The ability of individuals with ASD to learn prior information has also been confirmed in our recent study of distractor-location probability cueing in a visual-search paradigm [41]. In this paradigm, unbeknown to participants, a salient – that is, potentially attention-capturing – singleton distractor (which was task-irrelevant and so to be ignored for optimal performance) appeared more likely in one display region or one particular location [42,43]. Learning this spatial distribution would be beneficial for reducing attentional capture by distractors occurring at high- (vs. low-) probability locations [43–46]. Similar to Manning et al. [34], Allenmark et al. [41] observed that individuals with ASD learned the high- vs. low-probability distractor locations equally well to matched TD controls, and they successfully used this prior information to proactively prevent attentional capture. However, compared to the controls, individuals with ASD showed an atypically strong reaction to a prediction error when the distractor appeared at an unlikely location: they strongly marked that location as being a distractor position, setting up a bias that carried over to the next few trials. Thus, when the task-relevant target appeared at that location, this stimulus was often mis-interpreted as a distractor when the eye first landed on that location. Consequently, oculomotor scanning proceeded to other, non-target items before eventually returning to the target and identifying it as the response-relevant item. Assuming that a distractor appearing at an unexpected location results in a prediction error, this pattern reflects overweighting of prediction errors in individuals with ASD, as proposed by Van de Cruys et al. [15].

Thus, while there is a consensus that individuals with ASD display atypical sensory processing, the underlying causes remain controversial: does it arise from overlearning of environmental volatility [14,19] or reduced reliance on priors [13]? Or, alternatively, is learning intact [36], but the learning rate is reduced [39,40]? Of note in this context, while predictive-coding models of ASD [14,15,19] predict differences in predictive error handling in individuals with ASD relative to neurotypical individuals, the extant studies have focused primarily on differences in global priors and the consequent influences on sensory estimates – thus largely neglecting effects of sequential uncertainty (i.e., volatility) and session order (i.e., the direction of volatility changes). In particular, examining how individuals with ASD (compared to TD individuals) handle short-term trial-to-trial changes and longer-term environmental volatility changes may provide crucial evidence for deciding between two promising accounts of abnormal predictive coding in ASD, namely: (a) do individuals with

ASD form atypical priors regarding volatility; or, rather, (b) do they show atypical handling of prediction errors, such as slow updating, in response to volatility changes?

Accordingly, the present study was designed to examine how individuals respond to sequential uncertainty, employing a duration-reproduction paradigm [23,30,47]. Specifically, we compared the handling of (and shifting between) two types of duration sequences that were generated from the same (duration-) sample distribution, but differed in terms of trial-to-trial volatility (see Figure 1). We hypothesized that if the prior is chronically compromised (i.e., weaker) in individuals with ASD, they would display a reduced central-tendency effect in duration reproduction compared to TD individuals. In addition, if (a) was the case and individuals with ASD overestimate the environment volatility [19] and place an overly high weight on sensory inputs, their central-tendency and serial-dependence effects should be affected less by changes in the environmental volatility regimen (from low to high, or vice versa), compared to TD individuals. In contrast, if both groups learn the volatility in a similar manner but (b) differ in their handling of prediction errors [41] induced by volatility changes, individuals with ASD and matched TD controls would be expected to show comparable changes in the central tendency, but differ in terms of the carry-over of the previously learnt prior following a change in the volatility regimen.

2. Methods

(a) Participants

32 individuals (13 females, 19 males, aged between 18 and 67 years, M = 32.0; SD = 12.3) with confirmed ICD-10 ASD diagnosis [48] of F84.0 or F84.5 were recruited from the database and network partners of the Outpatient Clinic for Autism Spectrum Disorders at the Department of Psychiatry, LMU Munich. 32 TD controls (13 females, 19 males, aged between 18 and 70 years, M = 31.6, SD = 13.6) with no reported history of mental illnesses or neurological deficits were recruited via local advertising. The groups were matched pairwise using the 'Wortschatztest' [WST, 49], a measure of crystalline intelligence. Both groups completed the Autism-Spectrum Quotient [AQ, 50], Empathy Quotient [EQ, 51], Systemizing Quotient [SQ, 52], and Beck's Depression Inventory [BDI, 53]. The groups did not differ significantly in terms of IQ (p=.28), age (p=.9). As expected, the groups differed significantly on AQ (p < .001), EQ (p < .001), SQ (p = .005), and BDI (p=.018) (see Appendix Table A1).

All participants gave written informed consent prior to the experiment, and they were compensated for their time and participation at a rate of 10 Euros per hour. The study was approved by the Ethics Board of the Faculty of Pedagogics and Psychology at LMU Munich, Germany.

(b) Design and procedure

The experiment was carried out in a sound-reduced and moderately lit experimental cabin. The visual stimulus was a yellow disk patch (diameter: 4.7° of visual angle; luminance; 21.7 cd/m^2), which was presented on a 21-inch LACIE CRT monitor with a refresh rate of 85

Hz. The experimental code was developed using the Matlab PsychoToolbox (Kleiner et al., 2007).

We adopted the duration production-reproduction paradigm [54] (Figure 1a). A typical trial started with a fixation cross (size: 0.75° of visual angle) in the center of the screen for 500 ms, which was followed by a white dot (diameter: 0.2°), prompting the participant to press and hold the mouse button (either left or right) to start the production phase. Immediately after pressing the mouse button, a yellow circle was shown on the screen for a given duration, randomly selected from 400 ms to 1800 ms (see next subsection for details), and then disappeared, upon which the participant had to release the key immediately. The reproduction phase was separated from the production phase by a 500-ms blank screen. Again, a white dot appeared, prompting participants to reproduce the duration that they had just experienced by pressing the mouse button for as long as the yellow circle had been displayed earlier on, and then release it. Immediately after the participant pressed the mouse button, a visual display with a yellow disk appeared on the screen, which disappeared again immediately after the participant released the button. Following the reproduction, a feedback display was shown for 500 ms to indicate the reproduction accuracy, using the ratio of the reproduction error relative to the respective physical duration. The relative reproduction accuracy consisted of the highlighting, in green or red, of one among five horizontally arranged disks which, from the left to the right, were mapped to the relative error ranges: less than -30%; between -30% and -5%, between -5% and 5%, between 5% and 30%, and greater than 30%, respectively. The three circles in the middle were highlighted in green, and the outer left and right circles in red, the latter indicating a large error which should be avoided.

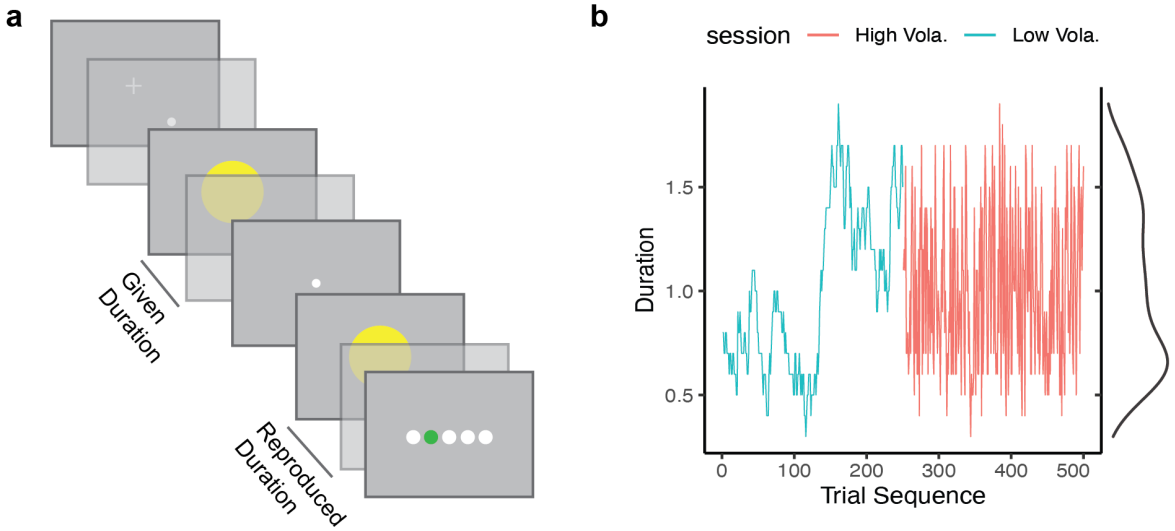

Figure 1. (a) Schematic illustration of a trial sequence used in the production-reproduction task. (**b**) Example duration (trial) sequences in two consecutive 'volatility' sessions. The first session (depicted in cyan) consists of a low-volatility sequence (Low Vola.), and the second session (red) of a high-volatility sequence (High Vola.). Both sessions comprised exactly the same durations (the same density function depicted on the right), differing only in their orders (right panel: the same density profile).

The experiment consisted of two sessions. Both sessions comprised the same set of stimulus durations and the same number of duration repetitions, but differed in their presentation order. Each session consisted of 10 mini-blocks of 25 trials each. First, we generated a sequence of durations employing a random-walk process, that is, the duration on trial *n* was calculated based on the duration on trial *n–1* with a small random fluctuation. Given that random fluctuation over the trials may exceed the probe range of 400 to 1600 ms, we scaled the durations in the sequence back to this range and rounded them up or down to the nearest 100-ms value, making it possible to present multiple repetitions of the tested durations. As the resulting fluctuation of durations across trials was relatively modest in this sequence, we will refer to it as the 'low volatile' sequence. Using the durations of this sequence, we randomly shuffled their positions to generate a new sequence. Due to the randomization, this sequence was characterized by high variation from trial to trial, compared to the 'low volatile' sequence (see Figure 1b); accordingly, we will refer to the session with this sequence as the high-volatility session. Figure 1b illustrates typical sequences for a low- and, respectively, a high-volatility sequence in two successive sessions. The two sequences were generated prior to the experiment and the order of the two, low- and high-volatility, sessions was counterbalanced across participants. Of note, we administered the same sequences to (age- and IQ-) matched (pairs of) participants in the ASD and TD groups, ensuring that any differences we observed would truly reflect differences between the two groups.

Prior to the experiment, participants were given detailed written and verbal instructions. In addition, all participants underwent a pre-experimental training session with an individualized number of trials in order to make sure they understood the instructions. Once this was confirmed by the experimenter, the formal experiment started, which took about 60 minutes to complete. Following the formal experiment, the participants filled out the various questionnaires (see above).

(c) Data analysis

The individual, raw reproduction data were first pre-processed and screened for outliers, that is: reproduced durations exceeding the range [Duration/3, 3×Duration], which were omitted from further analysis. Such extreme trials were very rare: only 0.58% of the trials in total.

Given that the central-tendency effect approximates linearly (see Eq. 1), we applied linear regression to estimate the central-tendency effect and the weight of the prior (*1-w*, in Eq. 1, equivalent to the *1-slope*) as the *central-tendency index* (CTI). A CTI close to 0 indicates less influence of the prior, whereas a CTI near 1 indicates a strong dependence on the prior. In addition, the short-term trial-to-trial updating could be different between the ASD and TD groups. To measure the trial-to-trial sequential dependence, we conducted linear regression with the duration of the previous trial (trial *n–1*) as the predictor for the reproduction error on the current trial (*n*), and used the estimated slope as the *sequential- dependence index* (SDI). If the current estimate is independent of the previous sensory input (i.e., the regression slope is close to zero), one should expect zero sequential dependence. Note, both assimilation and repulsion effects have been observed in sequential dependence [26,55], thus the regression slope of the sequential dependence could be ranged between -1 to 1.

The estimated CTIs and SDIs were then submitted to mixed ANOVAs with the within-subject factor Volatility and the between-subject factors Group and Session Order. We also conducted statistical analyses separately for the ASD and TD groups to further investigate influences of Volatility and Session Order within each group.

2. Results

Using regression analysis, we estimated the CTIs and SDIs for individual participants (Appendix Figure A1 shows the histograms). Based on the three-sigma rule (< 0.3% according to a Gaussian population), we identified four extreme outliers among the 64 participants, who all showed a markedly different pattern of performance from the other participants. In the next two subsections, we report the results without outliers (28 pairs after outlier exclusion) and the outliers (four pairs) separately.

2.1 Results excluding outliers

Fig. 2 depicts the average reproduction as a function of the probe durations for the two groups, plus representative individuals in the insets, separately for the low- and high-volatility sessions. By visual inspection of the linear slopes (less than 1.0), both groups show central-tendency biases in the duration reproduction, which were more marked in the high- (red) vs. the low-volatility session (cyan). Applying linear regression, for each participant, we estimated the slopes for each experimental condition. The slope is a good approximation for the weight (w) of the sensory input in Eq. 1. Accordingly, we calculated *1-slope* (approximation of the prior's weight) as the CTI. A CTI of 0% means no central-tendency bias, while a CTI of 100% would indicate a strong bias.

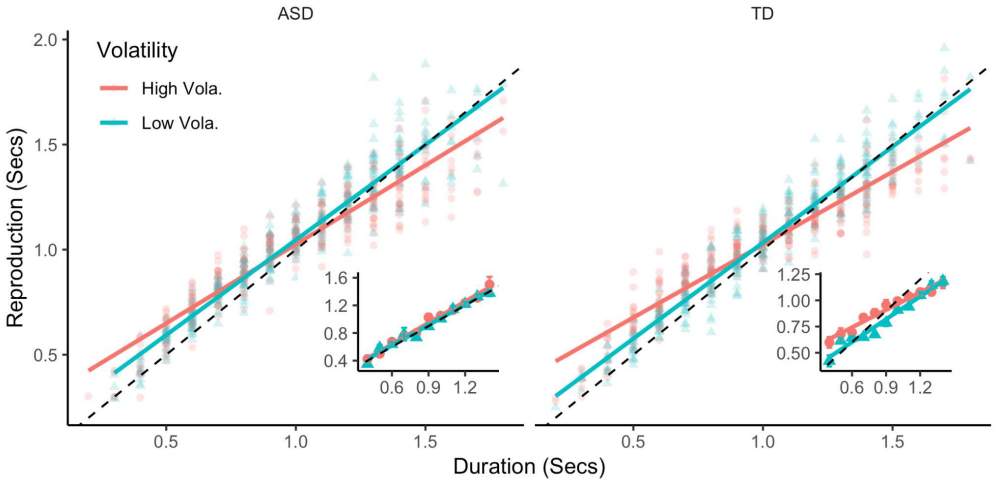

Figure 2. Mean duration reproduction for the ASD and TD groups. Insets: duration reproduction from a representative individual with ASD (left inset) and a representative, matched TD individual (right inset), separately for the low- (cyan) and high-volatility (red) sessions. Both participants received the same duration sequences. The dashed lines represent veridical reproduction. Dots above the dashed line indicate overestimates and dots below the dashed line underestimates. Solid lines show the fitted trend of the reproduction. The slopes of

the fitted trend were shallower than the dashed line (i.e., 1), indicative of central-tendency biases.

Since participants performed the high- and low-volatility in close succession, expectations about the statistical properties of the stimulus sequence acquired during the first session may be carried over to the second session. Thus, we included Session Order as a between-participants factor in the further analyses. Figure 3a shows the mean CTI for the ASD and TD groups, for the two session orders. Visually, the main difference between the two groups concerns the Session Order: the two lines are clearly separated in the ASD group but overlapping in the TD group. A mixed ANOVA of the CTIs, with the within-subject factor Volatility and the between-subject factors Group and Session Order revealed all main effects to be significant [Group: $F_{1,52} = 5.26$, $p = .026$, $\eta_g^2 = .057$; Volatility: $F_{1,52} = 88.38$, $p < .001$, $\eta_g^2 = .41$; Session Order: $F_{1,52} = 4.51$, $p = .039$, $\eta_g^2 = .05$]. On average, the ASD group exhibited less central tendency (CTI of 17.3%) than the TD group (CTI of 22.9%), suggesting that individuals with ASD trusted the ensemble prior less in their duration judgments. Further, the high-volatility session yielded a stronger central tendency (CTI of 30.5%) than the low-volatility session (CTI of 9.8%). Moreover, performing the high-volatility session first, relative to the low-volatility session first, gave rise to a stronger central tendency (22.7% vs. 17%). The two-way interactions were significant or marginal significant, with similar effect sizes: Volatility × Session Order, $F_{1,52} = 4.36$, $p = .042$, $\eta_g^2 = .032$; Group × Session-Order, $F_{1,52} = 2.93$, $p = .09$, $\eta_g^2 = .033$; and Group × Volatility, $F_{1,52} = 3.08$, $p = .085$, $\eta_g^2 = .023$. The three-way interaction, however, was not significant, $F_{1,52} = 0.813$, $p = .37$, $\eta_g^2 = .006$.

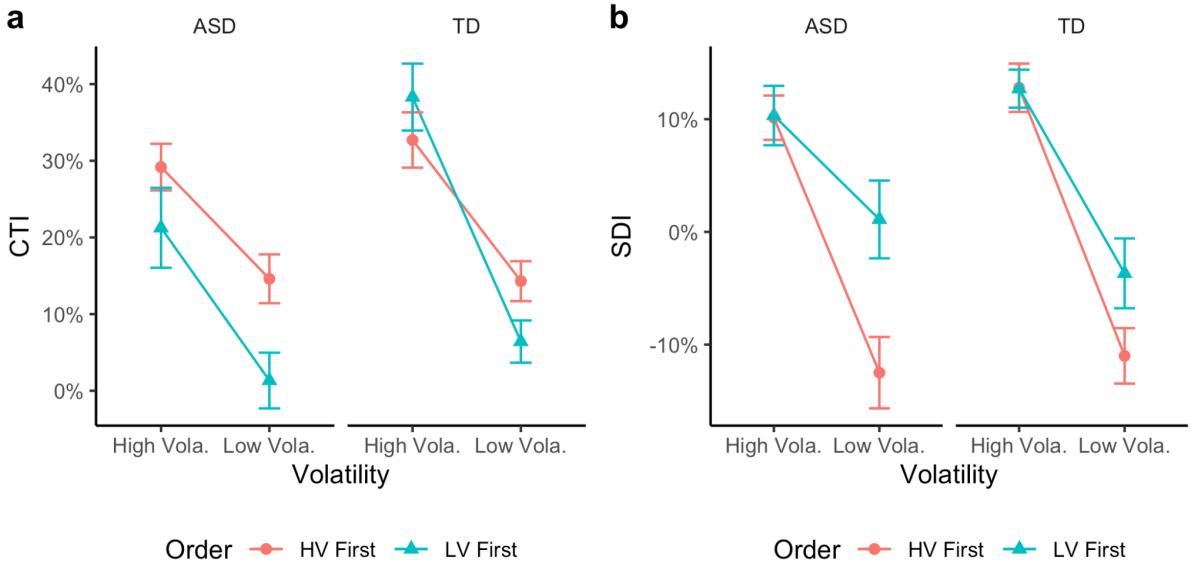

Figure 3. (**a**) The mean central tendency indices (CTIs) and (**b**) the mean sequential dependence indices (SDIs) and their associated standard errors plotted for the high/low volatility sessions, separated for the session order and the ASD/TD groups.

Given that the two-way interactions yielded similar small to medium effect sizes (0.023 to 0.033) and the focus of our study was to look for differential reactions, between the ASD and TD groups, to the change in Volatility modulated by the Session Order, we went on to

analyze the two groups separately. For the ASD group, a mixed ANOVA of the CTIs revealed both main effects to be significant: Volatility, $F_{1,26} = 32.67$, $p < .001$, $\eta_g^2 = .29$, and Session Order, $F_{1,26} = 5.94$, $p = .022$, $\eta_g^2 = .134$, but not the (Volatility × Session Order) interaction, $F_{1,26} = 0.787$, $p = .383$, $\eta_g^2 = .01$. The significant Volatility main effect indicates that individuals with ASD actually learned the volatility and changed the weight of the prior according to the uncertainty of the sequence, with a similar pattern as for the ASD group (see the next analysis). However, their adjustment of the weight of the prior also depended on the session order: individuals with ASD who started with the high-volatility session elevated the weight of the prior compared to those starting with the low-volatility session. For the TD group, by contrast, only the main effect of Volatility was significant, $F_{1,26} = 56.39$, $p < .001$, $\eta_g^2 = .52$ (main effect of Session Order, $F_{1,26} = 0.111$, $p = .74$, $\eta_g^2 = .002$; Volatility × Session Order interaction, $F_{1,26} = 4.05$, $p = .055$, $\eta_g^2 = .07$). Thus, the separate analyses revealed that the main effect of Session Order in the combined analysis was contributed mainly from the ASD group (the CTI was elevated by 10.6% when the high-volatility session was performed first), rather than their matched controls (the change of the CTI was only 1.1%), suggesting that individuals with ASD were significantly influenced by the order of the environment changes: starting with a low-volatile environment reduced the central tendency (indicative of reduced trust in the prior) in the subsequent, high-volatile environment, whereas encountering the high-volatile environment first led to a carry-over of the (high) the central-tendency bias (indicative of greater trust in the prior) to the following, low-volatile environment. The latter evidences the characteristic 'behavioral rigidity' despite changes in the environmental condition (see Fig. 3a).

Another short-term 'local' bias that we examined, in addition to the more 'global' central tendency, is the sequential (inter-trial) dependence. Specifically, we estimated the linear relation of the reproduction error on a given trial *n* as a function of the probe duration on the previous trial *n–1*. Fig. 3b shows the sequential dependence effect for the ASD and TD groups, for the two session orders. A mixed ANOVA of the sequential-dependence indices (SDIs) revealed significant main effects of Volatility and Session Order [Volatility: $F_{1,52} = 98.81$, $p < .001$, $\eta_g^2 = .49$, Session Order: $F_{1,26} = 8.08$, $p = .006$, $\eta_g^2 = .07$], as well as a significant Session-Order × Volatility interaction, $F_{1,26} = 7.59$, $p = .008$, $\eta_g^2 = .07$. The interaction was due to the session order influencing the SDI only in the low-volatility condition (see Figure 3b). However, the critical main effect of Group was not significant, $F_{1,52} = 0.11$, $p = .74$, $\eta_g^2 = .001$, that is, both (the ASD and TD) groups showed comparable sequential dependence. In other words, the patterns of short-term 'local' biases were similar for both groups across the different volatility environments and session orders.

Further, we calculated the mean reproduction errors (i.e., reproduced duration – probe duration) and the reproduction precision (measured by the standard deviation of the reproduced errors) for each condition. On average, the ASD group over-reproduced durations by 45.2 ± 9.1 ms (mean ± SE), and the control group by 33.9 ± 8.5 ms, with the estimates being significantly positive for both groups (*p*s < .001). However, there were no significant differences between Groups or among conditions ($Fs_{1,52} < 3.3$, *p*s > .07, $\eta_g^2 < .006$).

The reproduction precision was influenced by Volatility, $F_{1,52} = 32.9$, $p < .001$, $\eta_g^2 = .04$: the reproduction variability was higher in the high- relative to the low-volatility session. But there were no main effects of Session Order, $F_{1,52} = 0.26$, $p = .6$, $\eta_g^2 = .004$, or Group, $F_{1,52} = 0.80$, $p = .37$, $\eta_g^2 = .014$. Among the interactions, only the Volatility × Session Order interaction was significant, $F_{1,52} = 18.5$, $p < .001$, $\eta_g^2 = .024$, mainly attributable to a variability spike in the high-volatility session when tested first (SD: 182 ms as compared to 163, 153, and 160 ms in the other three, high-volatility second, low-volatility first, low-volatility second, conditions).

Finally, we conducted correlation analysis to examine whether there was any relation between the AQ, EQ, BDI scores, as a proxy of symptom severity, and the reproduction biases shown by CTI and SDI, separately for Group, Volatility, and Session Order. We found no significant correlations (all *p*s > .1 after correction for multiple comparisons), which suggests that neither CTI nor SDI was modulated by symptom severity (Appendix C, Fig. A2).

2.2 Outliers

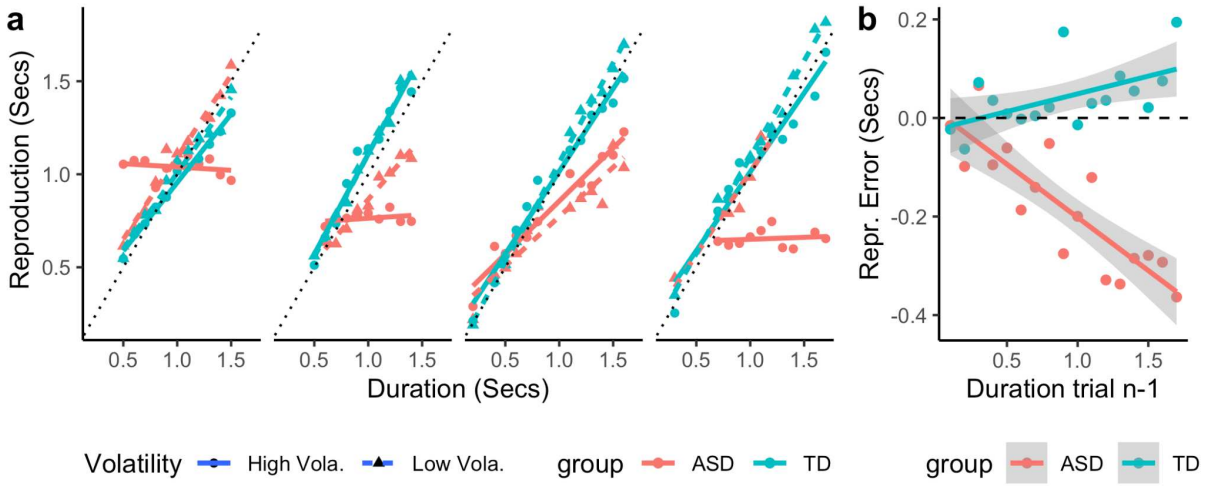

Figure 4. (**a**) Duration reproduction by 'extreme' ASD individuals (red) and their matched TD individuals (cyan), separately for the high- (dots) and low-volatility (triangles) sessions. For three of the four 'extreme' ASD individuals, the reproduced durations were similarly 'flat' (red solid lines) across the range of durations in the high-volatility session, while being in line with other data sets from both groups in the low-volatility session (dashed lines). The diagonal dashed line denotes veridical reproduction. (**b**) One of the four 'extreme' ASD individuals (the third in Fig. 4a) produced strong negative sequential dependence (red) relative to its matched control participant (cyan). The dashed line indicates no sequential dependence.

Four outlier participants, all in the ASD group, were identified according to the three-sigma rules applied for the CTI and SDI scores across all participants (Appendix B). Three of the four outliers reproduced the durations similarly across the probe range in the high-volatility session (see the flat solid red lines in Fig. 4a; the CTIs were 0.9, 0.99, 0.45, and 0.9, respectively, for the four outliers). A mixed ANOVA of the CTIs from the outliers and their matched controls with Volatility as within-subject factor and Group (ASD vs TD) as between-

subject factor revealed both main effects to be significant: Group, $F_{1,6} = 58.37$, $p < .001$, $\eta_g{}^2 = .73$, and Volatility, $F_{1,6} = 11.48$, $p = .014$, $\eta_g{}^2 = .57$; the Group × Volatility interaction was not significant, $F_{1,6} = 3.11$, $p = .13$, $\eta_g{}^2 = .26$. The outliers significantly relied significantly more on the prior (they exhibited a stronger central tendency) than their matched controls, more so in the high- than in the low-volatility session – in fact showing performance at the opposite end of the spectrum (in the high-volatility session) where the duration reproduction was little influenced by the variation of actual durations. One of the outliers, identified by the SDI, also showed an extreme negative sequential dependence, indicative of heavy usage of the short-term prior experience (from the previous trial).

Thus, in brief, a minority of individuals in the ASD group (4/32) heavily relied on prior knowledge, particularly in the high-volatile environment, exhibiting extreme rigidity in responding.

3. Discussion

The aim of the current study was to investigate two promising avenues of explaining atypical predictive coding in ASD, namely: (a) atypical prior formation regarding volatility [17], and (b) atypical handling of prediction errors in response to volatility changes [39–41]. To this end, the present study compared duration reproduction in individuals with ASD with matched TD controls in a paradigm allowing for variation of volatility using the same set of presented durations. In one session, the order of presented durations was randomized, rendering it highly volatile and unpredictable; in the other session, the order of durations was created by a random-walk process, producing a more predictable sequence (see Figure 1b). We found both groups were influenced by the volatility manipulation, showing a larger central-tendency effect in the high- relative to the low-volatility session. However, the majority of high-functioning individuals (excluding outliers) with ASD relied less on the prior overall (i.e., they exhibited less central tendency) compared to matched TD participants, which is consistent with the 'reduced-prior' account [13,30]. On the other hand, we also found the weights of the sensory inputs and prior carried over partially from the first to the second session, and this was mainly driven by the ASD group, rather than the TD group. In other words, updating of the prior was lagging and sticky across sessions for individuals with ASD, consistent with slow updating of the prior [39]. However, the short-term trial-by-trial bias, measured by the sequential-dependence index, was comparable for both groups. In contrast to the majority of individuals with ASD, four out of 32 revealed rigidity behavior on the opposite end of the spectrum, that is, they reproduced an average duration across all probed durations in the high-volatility session.

The overall comparable duration-reproduction accuracy for the TD and ASD groups and variability between the two groups suggest intact sensitivity for visual interval timing in individuals with ASD. This is in line with previous studies [9,56,57], though some studies have reported reduced sensitivity in ASD, albeit specific to certain temporal intervals and involving auditory stimuli [9,12,58,59]. Interestingly, though, we found the majority of adults with ASD (excluding the extreme minority) to show greater reliance on sensory input and less on prior knowledge, compared to TD individuals, as evidenced by their reduced central tendency in the current task. At face level, this finding is opposite to the previous study [30] on children

(whereas we tested adults) with ASD: their younger participants exhibited a stronger central tendency and worse temporal resolution than matched TD children. However, using Bayesian modeling, Karaminis et al. [30] determined the central tendency in children with ASD to be far weaker than the theoretical model prediction on the basis of their performance, indicating that their priors were poorer compared to matched controls. In the present study, the finding of a weaker central tendency in (adult) participants with ASD suggests that they placed less trust on priors than matched controls. In this respect, the interpretation offered by [30] is in line with the current findings with adult participants. Of note, though, while individuals with ASD in the present study showed comparable precision to their matched controls in the interval-timing task, Karaminis et al.'s children with ASD performed overall rather poorly – pointing to a developmental delay in interval timing in the latter sample. Thus, while individuals with ASD improve their temporal resolution from child- to adulthood, likely in a slow updating mode [39,40], their internal prior seems to remain poorer compared to matched controls – which would be in line with a chronically attenuated prior [13]. However, when taking the differential responses to volatility into account, the picture becomes more multifaceted – not in keeping with the notion of a generally attenuated prior, as we will argue below.

The focus of the present study was on the volatility of the tested duration sequences. In two sessions, the tested durations were drawn from the same sample distribution (ensemble prior), but differed in the trial-to-trial volatility (Figure 1b). The results revealed volatility to matter greatly for the central-tendency and serial-dependence effects. Interestingly, though, both groups equally showed a greater central tendency and a stronger serial dependence in the high-, relative to the low-, volatility session, indicating that both groups were able to adjust their decision making according to the volatility of the respective environment. Thus, our results provide no clear support for a general difference in the learning of volatility between the two groups. However, the central tendency was impacted differentially between the ASD and TD groups by the order in which the volatility conditions were encountered. While the TD group was not sensitive to the order change, the ASD group showed a 'sticky' carry-over from the first to the second session (Fig. 3a). In other words, the weight of the prior in the second session was influenced not only by the volatility itself, but also partially by weight of the prior acquired in the first session. This finding is somewhat different from Lawson et al. [19] who reported that, compared to matched controls, individuals with ASD tended to overestimate volatility, rendering them less surprised by volatility changes. Our findings suggest that, while both groups updated and used their priors according to the volatility prevailing in the respective session, the ASD group tended to persist with the decision-making strategy they developed previously, evidencing stickiness in reaction to environmental changes.

Of note, this difference was only seen in the 'global' central tendency, but not in the short-term trial-to-trial sequential dependence. The latter reflects the 'local' integration and updating strategy [25], which turned out to be comparable between the two groups in the current study. Thus, it suggests that it is likely *not* the *updating* of the prior[1] that is compromised in the low-volatility-first order; rather, the usage of the prior is not 'optimal' according to standard Bayesian inference. The majority of individuals with ASD (excluding

[1] Note, the updating of the prior should be distinguished from the chronic attenuated prior. As shown in the result section, individuals with ASD showed a general attenuated prior as compared to the matched controls.

outliers) persist using prior information from the previous environment, even though the environmental volatility has changed. Similar evidence of cognitive rigidity and slow updating has been reported in several recent studies [39,40,60–62]. For example, in a voluntary task-switching test, high-functioning individuals with ASD often stay with the same task longer compared to matched controls [61]. Thus, the present finding is in line with the 'slow-updating' account [39,40], given that individuals with ASD showed strong carry-over of the central tendency across volatility regimes.

Our finding that individuals with ASD show intact short-term updating but slow updating of the longer-term prior may well be consistent with the mixed reports of predictive coding in autism. Individuals with ASD can acquire appropriate priors from one-shot learning [32], prior trials [33], or statistically global settings [34,35,41]. In those studies, however, the volatility of the environment and the prior were often fixed throughout the test. Thus, probing prior acquisition alone may not reveal any atypicalities. Indeed, the present study revealed that only when the uncertainty regimen changed did high-functioning individuals with ASD show atypical inflexibility in dealing with prediction errors. In fact, studies that reported atypicalities often also included changes in the volatility regimen, pushing individuals with ASD out from a certain into an uncertain zone [19,27,28,63].

While the effect pattern described above was quite consistent among individuals with ASD, there were four individuals, out of our sample of 32 participants (12.5%), who showed a marked deviation from the others in the ASD group while exhibiting striking similarities among themselves (even though they actually performed the two volatility conditions in different orders): for the relatively stable and predictable (i.e., the low-volatility) sequence, extreme individuals produced time intervals proportional to the to-be-reproduced durations (albeit with some general over- or underestimation); for the volatile, random sequences, by contrast, they kept reproducing the same duration across all sampled intervals. That is, they appeared to completely disregard the (external) sensory inputs and solely base their reproduction performance on an overly strong (internal) prior duration under highly volatile, unpredictable task conditions, whereas their performance accorded with that of the other 28 participants with ASD in the low-volatility, predictable environment. The latter effectively rules out that their deviant behavior is simply attributable to a misunderstanding of the instruction. Importantly, those participants, when specifically asked during debriefing, stated that they had not noticed any difference in the randomization (i.e., sequential duration volatility) regimens between the two sessions, which thus influenced their performance only implicitly. One possible, if speculative, explanation is that the extreme participants reacted by 'shutting out' the sensory input when being confronted with a highly volatile sequence. In the present study, the unpredictability of the sequence may have engendered a sensory hyposensitivity. In principle, this explanation would be in keeping with an interpretation of compromised adaptability in ASD [4] as advanced above. Further investigation would be required to corroborate the deviant pattern of performance in these four individuals.

Conclusion

In summary, while our results confirm that high-functioning adults with ASD have a chronically attenuated prior [13], they actually are able to learn the prevailing (task-) environmental volatility and adapt to environmental changes to a certain degree. However,

high-functioning adults with ASD show strong carry-over of the weight of the acquired prior when environmental volatility changes. The rigid sticking to prior information from the past environment evidences inflexible or slow updating of prediction errors [15]. Accordingly, we propose to interpret the current, and possibly previous, findings of atypical predictive coding in ASD in terms of a reduced adaptability to environmental changes.

Ethic. The study was approved by the Ethics Board of the Faculty of Pedagogics and Psychology at LMU Munich, Germany (29.05.2018).

Data accessibility

Experimental codes, data, and analyses are published at g-node: 10.12751/g-node.d47f53

Author's contributions. Z.S., F.A., and C.F. study conceptualization. L.T., C. F. funding acquisition, C.F., L.T., R.P., and F.A. participant recruitment, Z.S., F.A., and C.F. supervision, L.T., R.P., F.A. data collection, L.T., R.P., F.A., and Z.S. data analysis and manuscript draft; Z.S., L.F., F.A., R.P., H.M., and C.F. critical revision and editing.

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References

1. APA, American Psychiatric Association. 2013 Diagnostic and statistical manual of mental disorders: DSM-5. *Arlington, VA*

2. Leekam SR, Nieto C, Libby SJ, Wing L, Gould J. 2007 Describing the sensory abnormalities of children and adults with autism. *J. Autism Dev. Disord.* **37**, 894–910.

3. Tomchek SD, Dunn W. 2007 Sensory processing in children with and without autism: a comparative study using the short sensory profile. *Am. J. Occup. Ther.* **61**, 190–200.

4. Gernert C, Falkai P, Falter-Wagner CM. 2020 The Generalized Adaptation Account of Autism. *Front. Neurosci.* **14**, 534218.

5. Mottron L, Dawson M, Soulières I, Hubert B, Burack J. 2006 Enhanced perceptual functioning in autism: an update, and eight principles of autistic perception. *J. Autism Dev. Disord.* **36**, 27–43.

6. Simmons DR, Robertson AE, McKay LS, Toal E, McAleer P, Pollick FE. 2009 Vision in autism spectrum disorders. *Vision Res.* **49**, 2705–2739.

7. Falter CM, Noreika V. 2011 Interval timing deficits and abnormal cognitive development. *Front. Integr. Neurosci.* **5**, 26.

8. Falter CM, Elliott MA, Bailey AJ. 2012 Enhanced visual temporal resolution in autism spectrum disorders. *PLoS One* **7**, e32774.

9. Isaksson S, Salomäki S, Tuominen J, Arstila V, Falter-Wagner CM, Noreika V. 2018 Is there a generalized timing impairment in Autism Spectrum Disorders across time scales and paradigms? *J. Psychiatr. Res.* **99**, 111–121.

10. Robertson CE, Baron-Cohen S. 2017 Sensory perception in autism. *Nat. Rev. Neurosci.* **18**, 671–684.

11. Casassus M, Poliakoff E, Gowen E, Poole D, Jones LA. 2019 Time perception and autistic spectrum condition: A systematic review. *Autism Res.* **12**, 1440–1462.

12. Falter CM, Noreika V, Wearden JH, Bailey AJ. 2012 More consistent, yet less sensitive: interval timing in autism spectrum disorders. *Q. J. Exp. Psychol.* **65**, 2093–2107.

13. Pellicano E, Burr D. 2012 When the world becomes 'too real': a Bayesian explanation of autistic perception. *Trends Cogn. Sci.*

14. Lawson RP, Rees G, Friston KJ. 2014 An aberrant precision account of autism. *Front. Hum. Neurosci.* **8**, 1–10.

15. Van de Cruys S, Evers K, Van der Hallen R, Van Eylen L, Boets B, de-Wit L, Wagemans J. 2014 Precise minds in uncertain worlds: predictive coding in autism. *Psychol. Rev.* **121**, 649–675.

16. Sinha P, Kjelgaard MM, Gandhi TK, Tsourides K, Cardinaux AL, Pantazis D, Diamond SP, Held RM. 2014 Autism as a disorder of prediction. *Proceedings of the National Academy of Sciences* **111**, 15220–15225.

17. Palmer CJ, Lawson RP, Hohwy J. 2017 Bayesian approaches to autism: Towards volatility, action, and behavior. *Psychol. Bull.* **143**, 521–542.

18. Cannon J, O'Brien AM, Bungert L, Sinha P. 2021 Prediction in Autism Spectrum Disorder: A Systematic Review of Empirical Evidence. *Autism Res.* **14**, 604–630.

19. Lawson RP, Mathys C, Rees G. 2017 Adults with autism overestimate the volatility of the sensory environment. *Nat. Neurosci.* **20**, 1293–1299.

20. Friston K, Kiebel S. 2009 Predictive coding under the free-energy principle. *Philos. Trans. R. Soc. Lond. B Biol. Sci.* **364**, 1211–1221.

21. Sutton RS, Barto AG. 2018 *Reinforcement Learning, second edition: An Introduction*. MIT Press.

22. Petzschner FH, Glasauer S, Stephan KE. 2015 A Bayesian perspective on magnitude estimation. *Trends Cogn. Sci.* , 1–9.

23. Glasauer S, Shi Z. 2021 The origin of Vierordt's law: The experimental protocol matters. *PsyCh J.* (doi:10.1002/pchj.464)

24. Glasauer S, Shi Z. 2019 Central Tendency as Consequence of Experimental Protocol. *2019 Conference on Cognitive Computational Neuroscience*. (doi:10.32470/ccn.2019.1148-0)

25. Cicchini GM, Mikellidou K, Burr DC. 2018 The functional role of serial dependence. *Proc. Biol. Sci.* **285**. (doi:10.1098/rspb.2018.1722)

26. Glasauer S, Shi Z. 2022 Individual beliefs about temporal continuity explain variation of perceptual biases. *Sci. Rep.* **12**, 1–15.

27. Thillay A, Lemaire M, Roux S, Houy-Durand E, Barthélémy C, Knight RT, Bidet-Caulet A, Bonnet-Brilhault F. 2016 Atypical Brain Mechanisms of Prediction According to Uncertainty in Autism. *Front. Neurosci.* **10**, 317.

28. Fogelson N, Li L, Diaz-Brage P, Amatriain-Fernandez S, Valle-Inclan F. 2019 Altered predictive contextual processing of emotional faces versus abstract stimuli in adults with Autism Spectrum Disorder. *Clin. Neurophysiol.* **130**, 963–975.

29. Ganglmayer K, Schuwerk T, Sodian B, Paulus M. 2020 Do Children and Adults with Autism Spectrum Condition Anticipate Others' Actions as Goal-Directed? A Predictive Coding Perspective. *J. Autism Dev. Disord.* **50**, 2077–2089.

30. Karaminis T, Cicchini GM, Neil L, Cappagli G, Aagten-Murphy D, Burr D, Pellicano E. 2016 Central tendency effects in time interval reproduction in autism. *Sci. Rep.* **6**, 1–13.

31. Shi Z, Church RM, Meck WH. 2013 Bayesian optimization of time perception. *Trends Cogn. Sci.* **17**, 556–564.

32. Van de Cruys S, Vanmarcke S, Van de Put I, Wagemans J. 2018 The Use of Prior Knowledge for Perceptual Inference Is Preserved in ASD. *Clin. Psychol. Sci.* **6**, 382–393.

33. Pell PJ, Mareschal I, Calder AJ, von dem Hagen EAH, Clifford CWG, Baron-Cohen S, Ewbank MP. 2016 Intact priors for gaze direction in adults with high-functioning autism spectrum conditions. *Mol. Autism* **7**, 25.

34. Pantelis PC, Kennedy DP. 2017 Deconstructing atypical eye gaze perception in autism spectrum disorder. *Sci. Rep.* **7**, 14990.

35. Hense M, Badde S, Köhne S, Dziobek I, Röder B. 2019 Visual and Proprioceptive Influences on Tactile Spatial Processing in Adults with Autism Spectrum Disorders. *Autism Res.* **12**, 1745–1757.

36. Manning C, Kilner J, Neil L, Karaminis T, Pellicano E. 2017 Children on the autism spectrum update their behaviour in response to a volatile environment. *Dev. Sci.* **20**, e12435.

37. Behrens TEJ, Woolrich MW, Walton ME, Rushworth MFS. 2007 Learning the value of information in an uncertain world. *Nat. Neurosci.* **10**, 1214–1221.

38. Rosenthal IA, Hutcherson CA, Adolphs R, Stanley DA. 2019 Deconstructing Theory-of-Mind Impairment in High-Functioning Adults with Autism. *Curr. Biol.* **29**, 513–519.e6.

39. Lieder I, Adam V, Frenkel O, Jaffe-Dax S, Sahani M, Ahissar M. 2019 Perceptual bias reveals slow-updating in autism and fast-forgetting in dyslexia. *Nat. Neurosci.* **22**, 256–264.

40. Vishne G, Jacoby N, Malinovitch T, Epstein T, Frenkel O, Ahissar M. 2021 Slow update of internal representations impedes synchronization in autism. *Nat. Commun.* **12**, 5439.

41. Allenmark F, Shi Z, Pistorius RL, Theisinger LA, Koutsouleris N, Falkai P, Müller HJ, Falter-Wagner CM. 2020 Acquisition and Use of 'Priors' in Autism: Typical in Deciding Where to Look, Atypical in Deciding What Is There. *J. Autism Dev. Disord.* (doi:10.1007/s10803-020-04828-2)

42. Goschy H, Bakos S, Müller HJ, Zehetleitner M. 2014 Probability cueing of distractor locations: both intertrial facilitation and statistical learning mediate interference reduction. *Front. Psychol.* **5**, 1195.

43. Zhang B, Allenmark F, Liesefeld HR, Shi Z, Müller HJ. 2019 Probability cueing of singleton-

distractor locations in visual search: priority-map-or dimension-based inhibition? *J. Exp. Psychol: Hum. Percept. Perform.* (doi:10.1037/xhp0000652)

44. Sauter M, Liesefeld HR, Zehetleitner M, Müller HJ. 2018 Region-based shielding of visual search from salient distractors: Target detection is impaired with same- but not different-dimension distractors. *Atten. Percept. Psychophys.* **80**, 622–642.

45. Allenmark F, Zhang B, Shi Z, Müller HJ. 2022 Learning to suppress likely distractor locations in visual search is driven by the local distractor frequency. *Journal of Experimental Psychology: Human Perception and Performance*

46. Allenmark F, Zhang B, Liesefeld HR, Shi Z, Müller HJ. 2019 Probability cueing of singleton-distractor regions in visual search: the locus of spatial distractor suppression is determined by colour swapping. *Vis. cogn.* , 1–19.

47. Shi Z, Ganzenmüller S, Müller HJ. 2013 Reducing Bias in Auditory Duration Reproduction by Integrating the Reproduced Signal. *PLoS One* **8**, e62065.

48. World Health Organization. 1992 *The ICD-10 classification of mental and behavioural disorders : clinical descriptions and diagnostic guidelines*. World Health Organization.

49. Schmidt KH, Metzler P. 1992 WST-Wortschatztest. *Göttingen: Beltz Test*

50. Baron-Cohen S, Wheelwright S, Skinner R, Martin J, Clubley E. 2001 The Autism-Spectrum Quotient (AQ): Evidence from Asperger Syndrome/High-Functioning Autism, Males and Females, Scientists and Mathematicians. *J. Autism Dev. Disord.* **31**, 5–17.

51. Baron-Cohen S, Wheelwright S. 2004 The empathy quotient: an investigation of adults with Asperger syndrome or high functioning autism, and normal sex differences. *J. Autism Dev. Disord.* **34**, 163–175.

52. Baron-Cohen S, Richler J, Bisarya D, Gurunathan N, Wheelwright S. 2003 The systemizing quotient: an investigation of adults with Asperger syndrome or high-functioning autism, and normal sex differences. *Philos. Trans. R. Soc. Lond. B Biol. Sci.* **358**, 361–374.

53. Beck AT, Steer RA, Brown G. 2011 Beck Depression Inventory–II. *PsycTESTS Dataset*. (doi:10.1037/t00742-000)

54. Ren Y, Allenmark F, Müller HJ, Shi Z. 2021 Variation in the 'coefficient of variation': Rethinking the violation of the scalar property in time-duration judgments. *Acta Psychol.* **214**, 103263.

55. Czoschke S, Fischer C, Beitner J, Kaiser J, Bledowski C. 2018 Two types of serial dependence in visual working memory. *Br. J. Psychol.* (doi:10.1111/bjop.12349)

56. Jones CRG *et al.* 2009 Auditory discrimination and auditory sensory behaviours in autism spectrum disorders. *Neuropsychologia* **47**, 2850–2858.

57. Mostofsky SH, Goldberg MC, Landa RJ, Denckla MB. 2000 Evidence for a deficit in procedural learning in children and adolescents with autism: implications for cerebellar contribution. *J. Int. Neuropsychol. Soc.* **6**, 752–759.

58. Martin JS, Poirier M, Bowler DM. 2010 Brief report: Impaired temporal reproduction performance in adults with autism spectrum disorder. *J. Autism Dev. Disord.* **40**, 640–646.

59. Szelag E, Kowalska J, Galkowski T, Pöppel E. 2004 Temporal processing deficits in high-functioning children with autism. *Br. J. Psychol.* **95**, 269–282.

60. Poljac E, Poljac E, Yeung N. 2012 Cognitive control of intentions for voluntary actions in individuals with a high level of autistic traits. *J. Autism Dev. Disord.* **42**, 2523–2533.

61. Watanabe T, Lawson RP, Walldén YSE, Rees G. 2019 A Neuroanatomical Substrate Linking Perceptual Stability to Cognitive Rigidity in Autism. *J. Neurosci.* **39**, 6540–6554.

62. Robertson CE, Kravitz DJ, Freyberg J, Baron-Cohen S, Baker CI. 2013 Slower rate of binocular rivalry in autism. *J. Neurosci.* **33**, 16983–16991.

63. van Laarhoven T, Stekelenburg JJ, Eussen ML, Vroomen J. 2020 Atypical visual-auditory predictive coding in autism spectrum disorder: Electrophysiological evidence from stimulus omissions. *Autism* **24**, 1849–1859.

Electronic supplementary material

Appendix A: Descriptive characteristics of the ASD and TD groups

The groups were matched pairwise using the 'Wortschatztest'[WST, 49], a measure of crystalline intelligence. Both groups completed the Autism-Spectrum Quotient[AQ, 50], Empathy Quotient[EQ, 51], Systemizing Quotient[SQ, 52], and Beck's Depression Inventory[BDI, 53].

Table A1. *Descriptive characteristics (Means and SDs) for ASD and TD group.*

Measures	***ASD (n=32)***	***TD (n=32)***	***Group comparison***
Age	32.03 (12.3)	31.6 (13.6)	$t31 = 0.13, p = .898$
IQ score	107.4 (9.7)	109.1 (13.4)	$t31 = 0.573, p = .28$
Autism-Spectrum Quotient score	36.7 (7.4)	15.8 (6.0)	$t31 = 18.06, p < .001^{**}$
Empathy Quotient score	26.97 (12.32)	50.38 (14.46)	$t31 = -7.85, p < .001^{**}$
Systemizing Quotient score	35.25 (14.93)	26.63 (10.1)	$t31 = 3.05, p < .01^{**}$
Beck's Depression Inventory score	10.69 (8.37)	5.25 (7.46)	$t31 = 2.49, p = .018^{*}$

Note: ** denotes p < .05 and ** p < .001.

Appendix B: Outlier detection and outliers

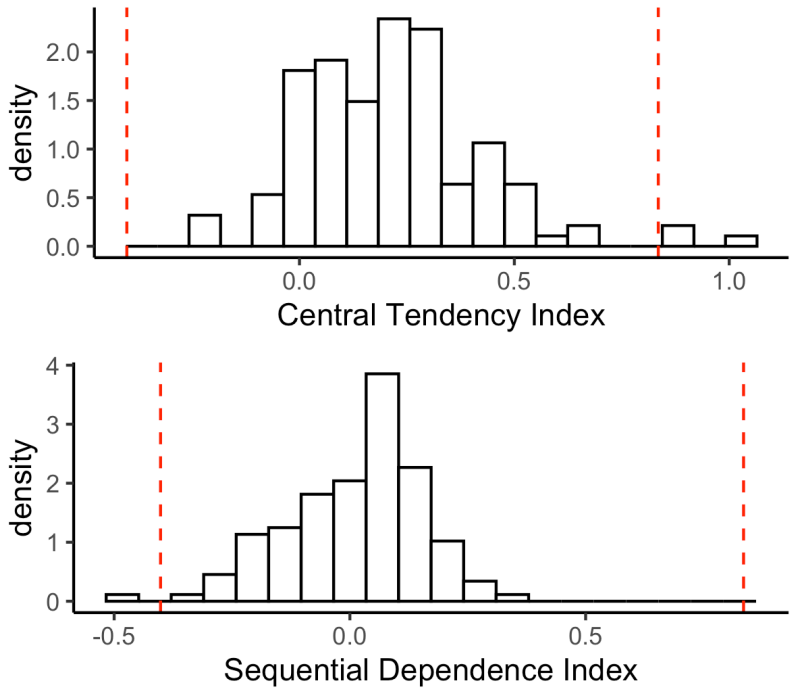

Figure A1. Histogram (normalized as density) plots of the central tendency and sequential dependence. The left and right red dashed lines indicate the three-sigma range deviation from the mean. The data points outside the three-sigma range were treated as outliers.

We first calculated the central-tendency and sequential-dependence indices for individual participants. Figure A1 presents the respective histogram plots for all participants. The red vertical lines indicate the locations of the three-sigma deviation from the mean. Using the standard three-sigma rule, we defined the data points outside this three-sigma range as outliers. Based on this criterion, four matched pairs were excluded from further analysis in the main Results text.

Appendix C: Correlations between symptom severity and reproduction biases

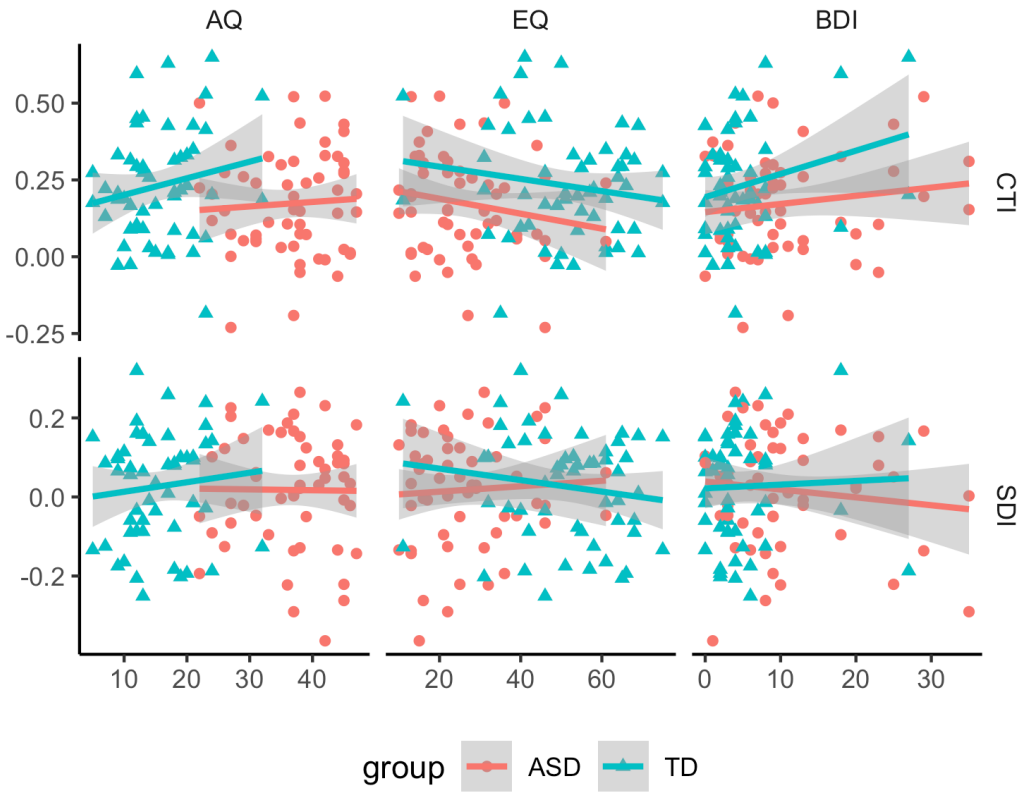

Figure A2. Scatterplots between survey scores (AQ, EQ, and, respectively, BDI) and reproduction-bias (CTI and, respectively, SDI) scores. The lines are the fitted linear regressions, and the gray areas the 95% confidence intervals. There were no significant correlations between survey scores and reproduction biases (all *p*s > .1).