# Predictive coding in ASD: inflexible weighting of prediction errors when switching from stable to volatile environments

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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. Critically, both sequences had the same set of intervals but differed in their stimulus presentation orders. Comparing individuals with ASD vs. their matched controls, we found both groups to respond to the volatility in a similar manner, albeit with a generally reduced prior in the ASD group. Interestingly, though, relative to the control group, the ASD group exhibited a markedly reduced trust in the prior in the volatile trial session when this was performed after the non-volatile session, while both groups performed comparably in the reverse session order. Our findings suggest that it is not the learning of environmental volatility that is compromised in ASD. Rather, it is their response to a change of the volatility regimen from stable to volatile, which causes a highly inflexible weighting of prediction errors.

*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 [7–9]. The sensory processing abnormalities also extend to time processing [9–11], though atypical performance is rather mixed [9,10].

Over the past decade, several accounts based on the predictive coding theory have been formulated to explain sensory atypicalities in autism [12–18]. 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 ($D_p$) is an optimal integration of the prediction based on the internal prior ($D_{prior}$) with the sensory measurement ( $D_{sensory}$):

$$D_p = (1 - w)D_{prior} + wD_{sensory} \quad (1)$$

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

$$D_{posterior} = D_{prior} + w(D_{sensory} - D_{prior}) \quad (2)$$

According to the predictive-coding framework [19], the posterior is adjusted by each prediction error ($D_{cur} - D_{pr}$) with learning rate $w$, such that it minimizes any future prediction errors. Incorporating cross-trial dynamic updating within the Bayesian inference framework renders an iterative Bayesian model [20–22], which takes a similar form to Eq. (2). If we further consider that prediction is based primarily on recent trial history, such as the previous trial ($D_{prev}$), rather than the distant prior ($D_{prior}$), the predictive updating mechanism illustrated in Eq. (2) captures the sequential-dependence effect [see similar approach in 23]:

$$D_p - D_{sensory} = (1 - w)(D_{prev} - D_{sensory}), \quad (3)$$

which highlights the influence of local context, such as the previous stimulus, on the current perceptual judgment [22,23].

Pellicano and Burr's attenuated-prior account [12] 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 [14], 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. [13] 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 [24,25], needing more time to perform goal-directed anticipations [26], and greatly reduced usage of the prior in duration reproduction [27]. Karaminis and colleagues [27] 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 we vary the sensory inputs ($D_{sensory}$), the perceptual estimate ($D_p$) regresses toward the prior [16,28]. A low central tendency means that the inference is driven mainly by the sensory inputs (i.e., the learning rate $w$ is high), and observers trust the sensory

information over the prior. Conversely, 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 of the new sensory information. Using this Bayesian framework, Karaminis et al. [27] 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 performance. 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' [12] and 'aberrant-precision' [13] accounts.

It should be noted, though, that not all types of prior 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) [29], the influence of gaze cues from previous trials [30,31], and reliance on external-world coordinates in tactile spatial processing [32]. Those mixed findings of usage of priors led Palmer et al. [16] argue that the simple Baysian model has a crucial limitation in assuming an unchanging world; instead, they speculate that the atypicalities in ASD may lie in the differential expectation about the precision of changes (i.e., volatility) in hidden states in a hierarchical inference. To directly examine how individuals with ASD learn about volatility changes, Lawson et al. [18] 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 or volatile (i.e., the cue-outcome association switched three times) within a block of trials. Compared to matched TD individuals, participants with ASD showed a smaller difference between stable and volatile blocks in response time (RT) and pupil-size changes, where the latter are thought to reflect neural (noradrenaline, activity in locus coeruleus) regulation processes [33]. In particular, compared to the control group, the ASD group exhibited a smaller increase in pupil size to violations of the cue-outcome contingency, but a larger increase in response to changes in the volatility of the stimulus sequence. Lawson et al. [18] 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. [34] 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. [35]: 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 (stable condition), there was a fixed probability distribution of each chest containing a reward, whereas in other blocks (volatile condition) the distribution changed regularly. In contrast to Lawson et al. [18], Manning et al. [34] found both groups to display a higher learning rate in the volatile vs. the stable condition, without any difference between the two groups (i.e., there were no effects involving the factor Group). Manning et al. [34] concluded that, while "atypical predictive mechanisms account for perception in autism, [this] … may not extend to learning tasks" (p. 10).

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 [36]. 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 [37,38]. Learning this spatial distribution would be beneficial for reducing attentional capture by distractors occurring at high- (vs. low-) probability locations [38–40]. Similar to Manning et al. [34], Allenmark et al. [36] 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. [14].

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 volatility [13,18] or reduced reliance on priors [12]? Or, alternatively, is learning intact [34], but the response to prediction errors is altered [36]? Of note in this context, while predictive-coding models of ASD [13,14,18] predict differences in predictive error handling in individuals with ASD (compared to TD individuals), the extant studies have focused primarily on differences in global priors and the consequent influences on sensory estimates – thus largely neglecting inter-trial effects (see [36] for an exception). In particular, examining how individuals with ASD (compared to TD individuals) handle trial-to-trial changes within high- and, respectively, low-volatility environments and switches between the two environments might 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 in response to volatility changes?

Accordingly, the present study was designed to examine how individuals respond to environmental volatility, employing a duration-reproduction paradigm [21,27,41]. Specifically, we compared the handling of (and switching between) two types of duration sequences that were generated from the same (duration-) sample distribution, but differed in terms of trial-to-trial volatility. 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 [18] 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 [36] 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 the carry-over of the previously learnt prior following a change in the volatility regimen.

## 2. Methods

### (a) Participants

26 individuals (12 females, 14 males, aged between 18 and 67 years, $M = 30.1$; $SD = 13.1$) with confirmed ICD-10 ASD diagnosis [42] 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. 26 TD controls (11 females, 15 males, aged between 18 and 70 years, $M = 31.2$, $SD = 14.5$) with no reported history of mental illnesses or neurological deficits were recruited via local advertising. The groups were matched pairwise using the 'Wortschatztest', a measure of crystalline intelligence.

Both groups completed the Autism-Spectrum Quotient (AQ), Empathy Quotient (EQ), Systemizing Quotient (SQ), and Beck's Depression Inventory (BDI). The groups did not differ significantly in terms of IQ ($p$=.32), age ($p$=.45), or BDI ($p$=.09). As expected, the groups differed significantly on AQ, EQ, and SQ ($p$s < .05, see Table S1).

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 [43] (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.

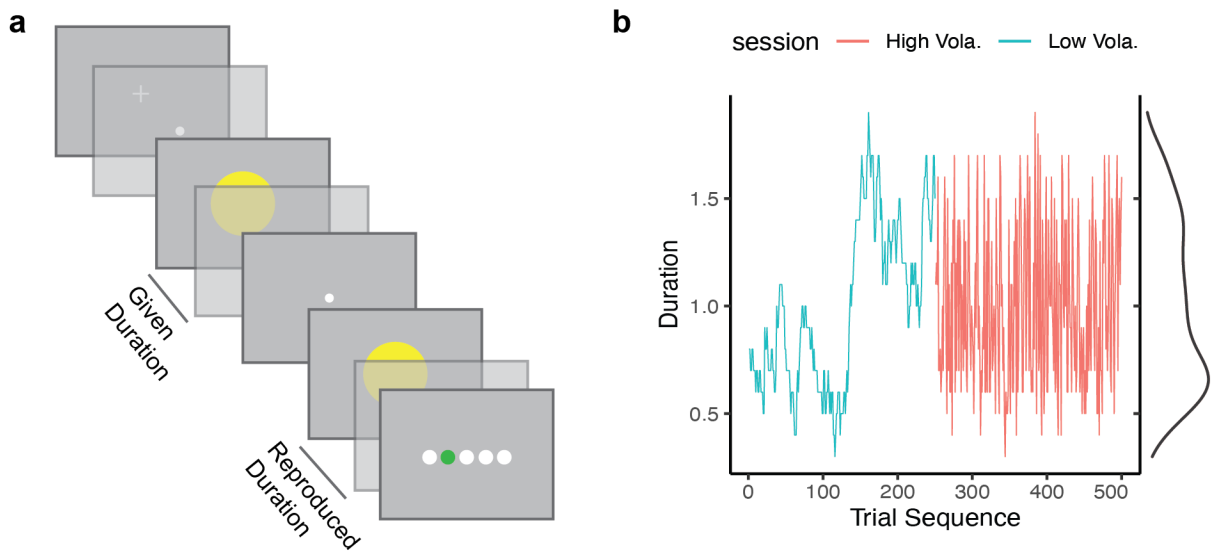

**Figure 1. (a)** Schematic illustration of a trial sequence used in the production-reproduction task. (**b**) Example duration (trial) sequences in two consecutive 'volatility' sessions. The first session (depicted in cyan) consists of a low-volatility sequence (Low Vola.), and the second session (red) of a high-volatility sequence (High Vola.). Both sessions comprised exactly the same durations, 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 Wiener random-walk process (i.e., a random fluctuating series of duration values with the trial-to-trial fluctuation following the normalized Gaussian distribution) for a given participant (or, more precisely a pair of matched participants; see below). We then scaled the durations to the range of 400 to 1600 ms, and rounded the durations to 100 ms, making it possible to present multiple repetitions of the tested durations. As the fluctuation of durations across trials in this sequence was relatively modest compared to the randomized condition (see Figure 1b), we will refer to the session with this sequence as the low-volatility session. Next, we randomly shuffled the durations from the low-volatility session to generate a new sequence, which, due to the randomization, was characterized by high variation from trial to trial. 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 session. 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 processing and outlier screening

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. Screening the mean reproductions for any extreme behaviors, we found two ASD individuals who showed a markedly different performance pattern from the rest of the group (see Appendix B): their reproduced durations were 'flat' (i.e., similar) across the probe range in the high-volatility session, while showing a comparable pattern to other ASD and TD individuals in the low-volatility session. Given that their behavioral patterns clearly deviated from the other participants, we left the two data sets (including those of the matched controls) out of the main (between-group) analysis, but discuss them separately. Consequently, the analyses reported next included data from 24 matched pairs.

## 3. Results

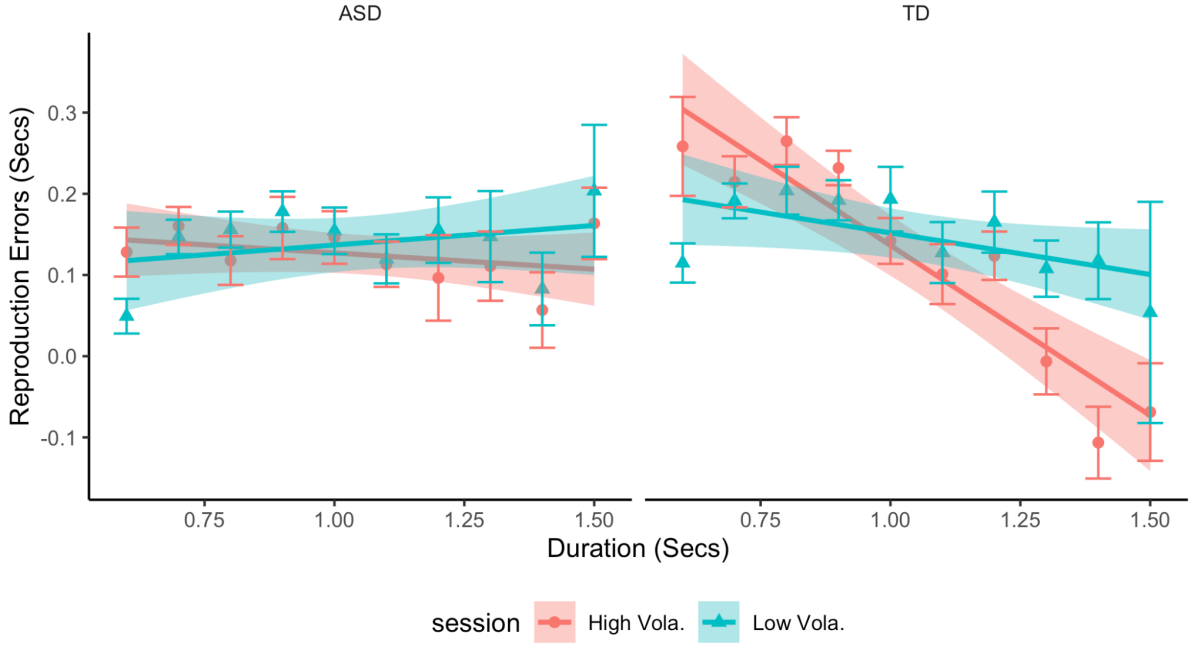


**Figure 2**. Reproduction errors from a representative individual with ASD (left panel) and a representative, matched TD individual (right panel), separately for the low- (cyan) and high-volatility (red) sessions. Both participants received the same duration sequences. The linear fits show the trends of the reproduction errors. A negative slope indicates a central-tendency effect.

Figure 2 depicts the reproduction errors (across the probe durations) for two typical participants (left panel from a participant in the ASD group and the right panel from the matched participant in the TD group), separately for the low- and high-volatility sessions. By visual inspection, there are two types of errors. First, both participants showed a general overestimation of durations, that is, errors above zero for the majority of the probe durations. On average, the ASD group over-reproduced durations by 48.1 ± 10.3 ms (mean ± standard error, *SE*), and the control group by 29.8 ± 8.1 ms, with the estimates being significantly positive in both groups ($ts_{47} > 3.7$, $ps < .001$, $BFs > 48$). However, a further mixed ANOVA, with the within-subject factor Session and between-subject factors Group and (Session) Order failed to reveal any significant (main or interaction) effects ($Fs_{1,144} < 2.02$, $p > .16$) – indicative of the positive reproduction biases being comparable between the two groups and among the various conditions. The second type of error is the central-tendency bias. The participant from the TD group (right panel) exhibits 'steeper' negative slopes than the participant from the ASD group (the left panel). We estimated the central-tendency effect using the negative value of the regression slope from the reproduction errors. An index of 0% means no central tendency, while an index of 100% would indicate a strong central-tendency bias.

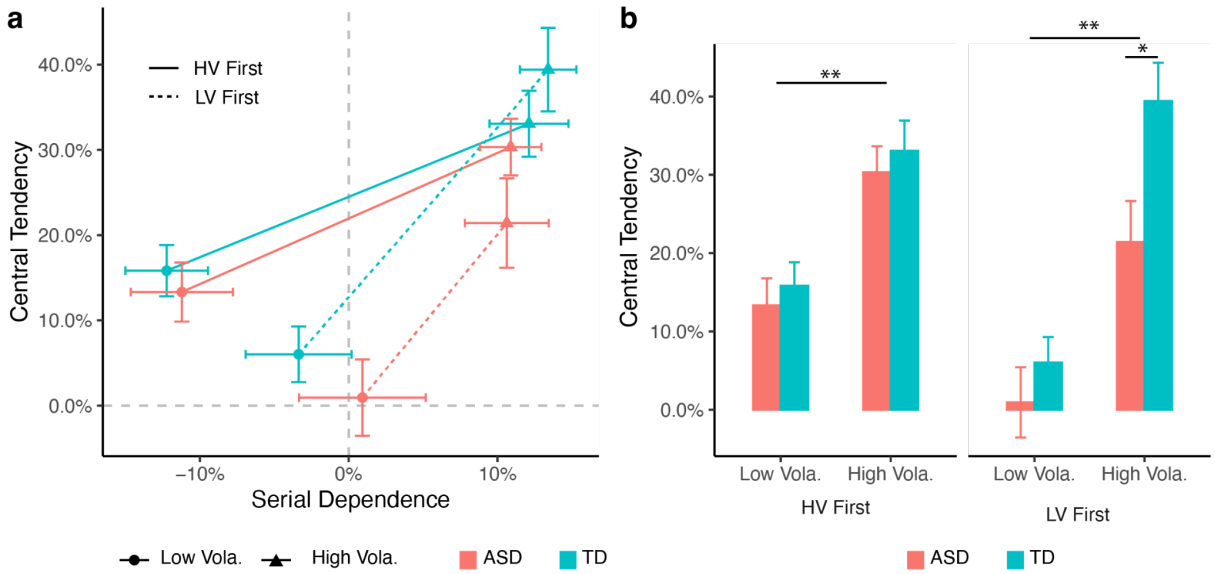


**Figure 3**. (**a**) The central-tendency index plotted against the serial-dependence effect for the duration reproduction, separately for the ASD (red) and TD (cyan) groups. The biases from two sessions (circles: the high volatility session, triangles: the low volatility session) are connected by the lines (solid lines: high-volatility session performed first; dashed lines: low-volatility session performed first). The horizontal and vertical error bars indicate one standard error (SE). The horizontal and vertical gray dashed lines indicate zero central tendency and zero serial dependence, respectively. (**b**) The mean central tendency effects and their associated standard errors plotted for the high/low volatility sessions, separated for the ASD and TD groups, and session order.

Another 'local' bias that we examined, in addition to the more 'global' central tendency, is the serial (inter-trial) dependence. Specifically, we estimated the 'absolute sequential dependence'[1] using linear approximation, that is, the reproduction error on a given trial *n* as a function of the probe duration on the previous trial *n–1*. A positive slope indicates an assimilation effect by the previous duration. Since participants performed the high- and low-volatility in close succession, the expectations about the statistical properties of the stimulus sequence acquired during the first session may be carried over to the second session. Given this, we also included Session Order as a between-participants factor in the further analyses. Figure 3 shows both the central-tendency and serial-dependence effects for the ASD and TD groups, linked by the session orders.

By visual inspection, individuals with ASD exhibited overall less central tendency than their matched TD controls (witness the red lines falling below the cyan lines in Figure 3a), while the local serial dependence was relatively comparable between the two groups (apart from the low-volatility condition when this was performed first). Interestingly, the central tendency and serial dependence were similar for both groups when the high-volatility session was performed first, but they differed when the low-volatility session performed first (see the solid and dashed lines in Figure 3a respectively).

[1] There are two types of sequential dependence, depending on their relation definitions. The relative sequential dependence quantifies the relation between the measurement error and the inter-trial stimulus difference [23,44]. This relation is captured by Eq. 2. However, this measure is partially coupled with the central-tendency effect [22] for the open scaled range (see also the relation between Eqs. 1 and 2). An alternative measure is the absolute sequential dependence, which quantifies the sequential effect as the dependence of the current measurement error on the stimulus on the previous trial. This measure is independent of the central tendency. Given this, we estimated the latter, absolute sequential dependence here.

To confirm these observations, a mixed ANOVA of the the central-tendency indices, with the within-subject factor Volatility and between-subject factors of Group and Session Order, revealed all main effects to be significant [Group: $F_{1,44} = 4.75$, $p = .03$, $\eta_g^2 = .06$; Volatility: $F_{1,44} = 74.36$, $p < .001$, $\eta_g^2 = .41$; Session Order: $F_{1,44} = 4.15$, $p = .047$, $\eta_g^2 = .05$]. The Volatility × Session Order interaction was marginally significant, $F_{1,44} = 3.84$, $p = .056$, $\eta_g^2 = .03$; the other interactions were non-significant, $F$s$_{1,44} < 2.19$, $p$s $> .14$, $\eta_g^2 < .028$. To obtain a better picture for each group, we ran mixed ANOVAs for the two session orders separately. The main effects of Volatility and Group remained both significant for participants who performed the low-volatility session first [Group: $F_{1,19} = 5.22$, $p = .034$, $\eta_g^2 = .15$; Volatility: $F_{1,19} = 49.15$, $p < .001$, $\eta_g^2 = .49$]; in contrast, only the Volatility effect was significant for participants who performed the high-volatility session first [Group: $F_{1,25} = 0.53$, $p = .47$, $\eta_g^2 = .012$; Volatility: $F_{1,25} = 27.6$, $p < .001$, $\eta_g^2 = .33$]. The Group × Session-Order interactions were non-significant in both conditions, $F$s $< 2.76$, $p > .11$, $\eta_g^2 < .05$. This effect pattern indicates that individuals with ASD were sensitive to the session order. In particular, when starting with the low-volatility session ('low-volatility first'), participants reproduced the sampled durations accurately without any significant central-tendency bias, irrespective of whether they belonged to the ASD or the TD group (central-tendency mean ± SE, ASD group: 0.9 ± 4.5%, *BF* =0.314; *TD* group: 6 ± 3.3%, *BF* = 1.06, *t*-test between groups: $t_{16.8} = 0.92$, $p = .37$, *BF* = 0.53). However, the ASD and TD groups differed significantly in the way they performed in the subsequent high-volatility session: while both groups showed significant central-tendency effect (ASD group: 21.4 ± 5.2%, BF = 17.8; TD group: 39 ± 4.9%, BF > 1000), the bias was significantly less pronounced for the ASD vs. the TD group ($t_{18.7} = 2.51$, $p = .02$, *BF* = 3.02). In other words, relative to their matched controls, individuals with ASD showed a much reduced central-tendency effect in the second-performed high-volatility session – that is, they placed greater trust on the sensory input (rather than the prior), evidencing the characteristic 'behavioral rigidity' when the environment changed from a standard stable to a 'volatile' one. Importantly, no such group difference was evident when participants performed the high-volatility session first (Figure 3b).

An analogous mixed ANOVA of the serial-dependence indices revealed significant main effects of Volatility and Session Order [Volatility: $F(1,44) = 85.42$, $p < .001$, $\eta_g^2 = .47$, Session Order: $F(1,44) = 6.36$, $p = .015$, $\eta_g^2 = .07$], and the Session-Order × Volatility interaction also turned out significant, $F(1,44) = 5.83$, $p = .02$, $\eta_g^2 = .059$, due to session order influencing performance mainly in the low-volatility condition (see Figure 3a). However, the critical main effect of Group was not significant, $F(1,44) = 0.016$, $p = .9$, $\eta_g^2 < .001$, that is, both (the ASD and TD) groups showed comparable sequential dependence. In other words, the patterns of 'local' biases were comparable for both groups across different volatility environments and session orders.

Further, the mean standard deviations (and associated standard errors) of the reproduction for the ASD and TD groups were 170 ± 7.9 and 171 ± 7.6 ms, respectively, in the high-volatility session, and 159 ± 7.57 and 149 ± 7.53 ms, respectively, in the low-volatility session. Both groups showed comparable variability in their duration estimations, ($F_{1,46} = 0.05$, $p = .83$, $\eta_g^2 = .001$, albeit with higher variability in the high- relative to the low-volatility session, $F_{1,46} = 22.0$, $p < .01$, $\eta_g^2 = .047$. And the Volatility × Group interaction was not significant, $F_{1,46} = 0.81$, $p = .37$, $\eta_g^2 = .002$.

Finally, we conducted correlation analysis to examine whether there was any relation between the AQ score, as a proxy of symptom severity, and the central-tendency effect, separately for the two

groups. We found no significant correlations (both *p*s > .15), which suggests that the central-tendency effect is not modulated by symptom severity as measured with the AQ.

## 4. Discussion

The aim of the current study was to decide between two promising avenues of explaining atypical predictive coding in ASD, namely (a) atypical prior formation regarding volatility [16] vs. (b) atypical handling of prediction errors in response to volatility changes [36]. 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 Wiener process, producing a more predictable sequence (see Figure 1b). We found the central-tendency effect to be larger in the high- relative to the low-volatility session, with individuals with ASD showing an overall less marked central tendency compared to TD participants. The Volatility × Group interaction was, however, not significant, that is, both groups were comparably affected by the volatility manipulation. Interestingly, we also found the session order to influence the central tendency, which was generally lower when participants performed the low- (rather than the high-) volatility condition first. This session-order effect was particularly marked for individuals with ASD. In contrast, the serial dependence, which reflected the inter-trial influence, was comparable for both groups.

The overall comparable duration-reproduction accuracy (average overestimation of 29.8 ms and 48.1 ms for the TD and ASD groups, respectively; not significantly different) and precision between the two groups suggest intact sensitivity for visual interval timing in individuals with ASD. This is in line with previous studies [9,45,46], though some studies have reported reduced sensitivity in ASD, albeit specific to certain temporal intervals and involving auditory stimuli [9,11,47,48]. Interestingly, though, we found individuals with ASD 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 [27] 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. [27] 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 Karaminis et al. [27] 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 [49,50], their internal prior seems to remain poorer compared to matched controls – which would be in line with a chronically attenuated prior [12]. 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 (global 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 –

without these effects being modulated by Group (non-significant Group × Volatility interaction). This finding is somewhat different from Lawson et al. [18] who reported that, compared to matched controls, individuals with ASD tended to overestimate volatility, rendering unexpected (volatile) events less surprising than expected (non-volatile) events. Thus, our results provide no clear support for a general difference in volatility estimation between the two groups. However, the central tendency was impacted differentially between the ASD and TD groups by the order of the volatility conditions. When being first exposed to a high-volatility environment and then to a low-volatility environment, both groups showed comparable central-tendency effects: an elevated central tendency in the high-volatility session and a reduced central tendency in the low volatility session – suggesting that both groups updated and used their priors according to the volatility prevailing in the respective session. However, there was a marked difference between the groups when the session order was reversed: encountering the low-volatility environment first and the high-volatility environment second. While both groups exhibited no central tendency in the first, low-volatility session, the ASD group placed greater trust on the sensory inputs (indicated by the low central tendency) than the TD group in the second, high-volatility session. Taken together, this pattern suggests that individuals with ASD make less use of the prior only when the environment shifts from a normal predictable (low-volatility) to an unpredictable (high-volatility) one, a shift direction that, arguably, poses higher demands on adaptation.

Of note, this difference was only seen in the 'global' central tendency, but not in the trial-to-trial sequential dependence. The latter reflects the 'local' integration and updating strategy [23], which turned out to be comparable between the two groups, as was the case in the current study. In a recent study of probability learning in a stable vs. volatile environment, Manning et al. [34] similarly showed that the order in which the two conditions were encountered did not significantly impact the learning rate. Thus, convergent evidence (intact learning rate and local updating, as well as comparable central-tendency effects in the high-volatility-first order) suggests that it is likely *not* the *updating* of the prior[2] that is compromised in the low-volatility-first order; rather, the usage of the prior is not 'optimal' according to standard Bayesian inference. Individuals with ASD appear to set the weight of the prior lower (and that of the sensory inputs higher) than optimal when the environment shifts from a normal to an unpredictable, volatile regimen. A similar inability to adapt to process unexpected sensory stimulation has been reported in several recent studies [4,24,36,51]. For example, using audiovisual recordings of hand-clapping with unexpected omissions of sound [51] found an early negative omission response (the oN1 component of the event-related potential) – a key signature of processing unexpected sensory stimulation – to be significantly more pronounced in their ASD relative to the TD group. In our previous study on probability learning of distractor locations in visual search [36], individuals with ASD showed comparable learning of the spatial distractor distribution, but an atypically strong reaction to a prediction error only when the distractor appeared at an unlikely (i.e., surprising) location.

One might argue that the present findings can be explained by the slow-updating account [49,50], given that individuals with ASD showed a strong carry-over effect in the central tendency, that is: the lower central tendency they exhibited in the second-performed high-volatility session (i.e., after they had performed the low-volatility session) might reflect slow prior updating as a by-product of the 'carry-over' effect. However, again, the slow-updating hypothesis alone cannot explain the differential session-order effects. The change in central tendency from high to low volatility was largely the same for the ASD as that for the TD group, though individuals with ASD exhibited a general, 'chronically' compromised prior. The two groups only differed when the low-volatility session preceded the high-volatility session – indicating that inflexible weighting of prediction errors [29] only occurs when the environment changes from stable to volatile, but not vice versa.

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[2] 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.

Thus, our findings highlight that individuals with ASD display atypicalities only in specific situations, when they encounter environmental changes from relatively certain to highly uncertain conditions. This may well explain the mixed reports of predictive coding in autism. Individuals with ASD can acquire appropriate priors from one-shot learning [29], prior trials [30], or statistically global settings [31,32,34,36]. That the priors are typically intact is also evidenced by the present finding that the ASD group of participants did not differ in their local prior updating from the TD group. Only when uncertainty increased from a predictable to an unpredictable regimen did 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 [18,24,25,51].

While the effect pattern described above was quite consistent among individuals with ASD, there were two individuals (<10% of the sample) who showed a marked deviation from the others in the ASD group while exhibiting a striking similarity between themselves (even though they actually performed the two volatility conditions in different orders) (see Figure S1): for the relatively stable and predictable (i.e., the low-volatility) sequence, both 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 the highly volatile, unpredictable task conditions, whereas their performance accorded with that of the other 24 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, both 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 two 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 as advanced above. Further investigation would be required to corroborate the deviant pattern of performance in these two individuals.

*Conclusion*

While our results confirm that individuals with ASD have a chronically attenuated prior [12], they actually are able to learn the prevailing (task-) environmental volatility and adapt to high- as well as low-volatility conditions. A difference between the ASD and TD groups emerged only when the high-volatility environment was encountered second (subsequent to the low-volatility environment) in the session order. In this case, individuals with ASD trusted the sensory inputs more than TD individuals in the second, high-volatility session (despite intact local updating) – evidencing inflexible weighting of prediction errors [14] or compromised adaptability when the environment becomes more unstable. In contrast, there was no evidence of such a 'cognitive rigidity' [52] when the environment changed from volatile to stable. Arguably, changing from a more stable and predictable to a highly unstable and unpredictable environment poses much greater demands on adaptability than vice versa. Accordingly, we argue for interpreting the current, and possibly previous, findings of atypical predictive coding in ASD in terms of a reduced ability to adapt to demanding environmental conditions.

**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 available in the following github repository. https://github.com/msenselab/predictive_coding_in_asd

**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.

**Competing interests.** The authors declare no competing financial interests.

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