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Visuo-motor updating in autism symptomatology.

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SUMMARY

Autism Spectrum Disorder (ASD) presents a range of challenges, including heightened sensory sensitivities. Here, we examine the idea that sensory overload in ASD may be linked to issues with efference copy mechanisms, which predict the sensory outcomes of self-generated actions, such as eye movements. Efference copies play a vital role in maintaining visual and motor stability. Disrupted efference copies hinder precise predictions, leading to increased reliance on actual feedback and potential distortions in perceptions across eye movements. In our first experiment, we tested how well individuals with varying levels of autistic traits updated their mental map after making eye movements. We found that those with more autistic traits had difficulty using information from their eye movements to update the spatial representation of their mental map, resulting in significant errors in object localization. In the second experiment, we looked at how participants perceived an object displacement after making eye movements. Using a trans-saccadic spatial updating task, we found that those with higher autism scores exhibited a greater bias, indicating under-compensation of eye movements and a failure to maintain spatial stability during saccades. Overall, our study underscores efference copy's vital role in visuo-motor stability, aligning with Bayesian theories of autism, potentially informing interventions for improved action-perception integration in autism.

Keywords: perceptual stability, visual updating, motor updating, Autism, autistic traits, efference copy

INTRODUCTION

Autism spectrum disorders (ASD) present a complex array of challenges, with sensory sensitivities, a recent addition to diagnostic criteria[1] , standing out as a dominant aspect of this condition. Individuals with ASD often exhibit a general inflexibility in the motor domain and struggle to learn from motor errors, usually linked to restricted and repetitive behaviors[2-4]. This difficulty in understanding motor errors can hinder their ability to adapt to new sensory information, further exacerbating sensory overload[4-6].

Recent research has increasingly emphasized the role of predictive abilities in ASD[7-10]. In their daily lives within an unpredictable and often chaotic world, humans are consistently engaged in anticipating what might happen next and preparing adaptively for potential threats in their surroundings[1]. Within this framework, recent theories propose that individuals with ASD may demonstrate a reduced reliance on prior information when making perceptual judgments. The Bayesian perspective suggests that the predictive system deficits in autism stem from a diminished Bayesian prior[10,11]. From this perspective, the brain unconsciously infers information about the world by applying Bayes' rule: it assesses the probability of a prediction given sensory data, considering the likelihood of sensory data given that prediction and the Bayesian prior. The Bayesian prior is essentially a probabilistic distribution representing the expectation of the environment being in a particular state before any observations are made[10]. These predictive abilities include the role of perceptual priors, which play a crucial role in guiding individuals to make accurate predictions, especially when sensory data is limited. However, in cases of autism, the influence of prior knowledge is believed to be weakened, resulting in predictions that heavily rely on sensory input.

But how do these priors interact with the sensory and motor systems in individuals with ASD, and how does this influence their perceptual stability? Predicting the consequences of one's behavior is of vital importance when our movements produce changes on our sensory receptors. When we move our eyes, head, or body, we displace external stimuli on the retina. We must know about this self-produced displacement to avoid confusion with external stimulus motion. In other words, if we would not be able to predict the sensory consequences of our behavior, we would experience that the world is moving.

The most frequent movements we perform are saccades. To bring the region of highest resolution, i.e. the fovea, to regions of interest as fast as possible, we shift the eye ball with high speed about 3 times per second. Internal predictions about an imminent saccade ensure the smooth and stable continuity of our perception. This anticipation involves a signal incorporating information about the motor vector to predict the sensory consequences of the upcoming saccade. Such a signal is known as an efference copy, i.e. a copy of the motor command (for a review see [12]). Imagine a scenario in which you're watching a video of a fast-moving car on a bumpy road. As the car hits a pothole, your eyes naturally make quick, involuntary saccades to keep the car in your visual field. Without a functional efference copy system, your brain would have difficulty accurately determining the current position of your eye in space, which in turn affects its ability to anticipate where the car should appear after each eye movement. With accurate efference copy signals, your brain predicts the car's new location with remarkable precision. This allows for smooth and stable visual perception, as if the car never jolted over that pothole. Consequently, individuals who may have challenges with their efference copy system, might experience a similar scenario differently. They may misestimate their own eye movements, leading to a jumpy perception, where they perceive the car as moving unpredictably in their vision. A key pathway responsible for transmitting efference copy signals involves the superior colliculus[13], a saccade command area, to the frontal eye field via the medio-dorsal nucleus of the thalamus. Lesions affecting this pathway can impair both motor and visual updating[12]. We recently reported that in patients with lesions in thalamic nuclei, evidence for two pathways conveying efference copies exists. We found that

patients with lesions in the medio-dorsal nucleus showed impairments in visual updating, while those with lesions in both the medio-dorsal and ventrolateral (VL) nuclei exhibited issues with motor updating[14-19].

Here, we hypothesized that the impairment of efference copy signals would result in an enduring lack of complete confirmation for pre-saccadic predictions through post-saccadic feedback. A failure in processing efference copy information might have severe consequences for perception around the execution of saccades. Their reliance on post-saccadic sensory feedback, rather than accurate efference copy predictions, results in a distorted perception of the visual world following eye movements (see **Figure 1**).
First, intra-saccadic vision might be affected. We never perceive the self-produced motion stimulation on the retina produced by our own saccades. According to theories of saccadic suppression, an efference copy is necessary to predict the occurrence of a saccade. Suppression strength scales as a function of the saccade amplitude[20]. If predictive information about the saccade amplitude cannot be generated due to failures in efference copy processing, suppression might organize only inadequately the omission of the self-produced, intra-saccadic motion stimulus.
Second, the internal updating of the spatial representation for the saccade vector would be affected. Since intra-saccadic vision is omitted, visual perception is confronted with a retinal image before the saccade and a different one after the performance of the saccade. Predicting the appearance of the post-saccadic image leads to a remapping of internal space and thereby to a seamless perception across the saccade. If the remapping process is not accurate, the appearance of the post-saccadic image would come as a surprise. Surprising stimuli appear much more salient than predicted ones. Failures in trans-saccadic remapping in individuals with autistic symptomatology could be the reason or at least contribute to the sensory overload they experience.
Third, in addition to problems in visual updating, a lack of proper efference copy transfer should produce problems in motor updating. In cases in which visual information is not available or reliable, the sensorimotor system must rely on internal estimates about its own movements.
In the laboratory, trans-saccadic updating can be investigated in motor behavior and in perceptual localization. In the double-step paradigm, two consecutive saccades are made to briefly displayed targets[21,22]. The first saccade occurs without visual references, relying on internal updating to determine the eye's position. The accuracy of the second saccade quantifies the first saccade's updating process. Perceptual updating is tested similarly, with subjects remembering the position of a target and then indicating it relative to a second target after a single saccade. The perceived displacement path reveals how well the eye movement integrates into vision; perfect compensation results in a vertical trajectory, while small biases indicate stabilization issues[23-25]. In cases where efference copies are absent, as in a double-step task's second saccade localization, significant saccade under-compensation occurs[26].

In the present study, we directly investigated whether the precision of efference copy signals influences the accumulation of sensory predictions over time and explored potential variations in this mechanism among individuals with a heightened autistic phenotype. In two experiments, we focused on the role of efference copy in the pathophysiology of autistic symptomatology, particularly in maintaining perceptual continuity during saccades. We hypothesized that individuals with a high autistic phenotype would exhibit decreased accuracy in second saccades during double-step tasks and under-compensation when localizing a stimulus based on efference copy signals. These findings could shed light on the visual stability and predictive challenges faced by individuals with ASD.

RESULTS

Motor updating

We asked participants to perform a classical double-step task in order to measure the accuracy of motor updating. In the double-step task, the measure of interest is spatial accuracy of the second saccade. For statistical analyses, we divided our group of participants into quartiles based on the autistic symptom severity. **Figure 2C** shows two example participants at the extremes of the lower (Autistic quotient = 3, in orange) and upper quartile (Autistic quotient = 31, in magenta) of the autistic spectrum distribution: participants were instructed to make saccades to the locations indicated by two brief target appearances (T1 and T2), as quickly and as accurately as possible and in the same order as that indicated by the targets. To shift gaze to the second target accurately, one must account for the first saccade from the fixation point without spatial references or visual feedback.

To control for general impairments in saccade generation, we first checked for the accuracy of the first saccade (from FP to T1). **Figure 3A** shows the average saccade landing positions of the two subgroups of participants (lower quartile and upper quartile of the AQ distribution in orange and magenta respectively) in in the double-step task. For the analysis we collapsed data from trials with second saccades into the lower and upper visual field. For statistical analyses, we first compared horizontal landing positions of the first saccade for the two groups (**Figure 3B**). Saccade landing positions of participants in the lower quartile (mean degree ± SEM: 10.17± 0.50) did not deviate significantly from those in the upper quartile (mean degree ± SEM: 9.65 ± 0.77). This result was also confirmed by a paired sample t-test ($t(7) = 0.66$; $p = 0.66$, BF10 = 0.40) and by a non-significant correlation between the landing position of the first saccade and the autistic symptoms of our participants ($r = -0.09$; $p = 0.55$, BF10 = 0.14, not shown as a figure).

We then calculated the angle between the participants' second saccade vector and the optimal vector that would have led to the physical target position T2 from the second saccades' starting position. The analysis of the second saccade provided the means to determine the effect of efference copies signals in underlying extraretinal information. If the participant fails to compensate for the first saccade due to disrupted efference copy, the second saccade vector deviates from the location of the second target (Fig. 1B). A correct remapping of the previously seen target position would lead to smaller angle of deviations from the optimal saccade vector; however, a failure in using efference copy-based updating of visual space would results in larger deviations. **Figure 3C** plots the motor bias (expressed as angle of deviations) as a function of autism severity. We found a significant correlation between the autistic symptomatology of our group of participants and the amount of motor bias ($r = 0.52$; $p < 0.001$; BF10 = 57.37). This result is also confirmed by a strong group difference, between the lower and upper quartile of AQ distribution ($t(7) = -4.68$; $p = 0.002$; BF10 = 21.45), with a mean bias of 6.26±0.80 for the lower quartile and of 20.61±3.40 for the upper quartile.

Results on the double step task suggest that although participants did not show an impairment in the first saccade execution, those with higher autistic traits reported difficulties in using extra-retinal information about the amplitude and direction of the motor vector of the first saccade, in order to update the spatial representation of the second target. Thus, they were less able to use the efference copy of the first saccade to construct a spatial representation of the second target location in generating an accurate saccade towards it.

Visual updating

With a trans-saccadic localization task, we tested whether saccades modulate visual stability in a similar fashion, between low and high autistic traits. Participants were instructed to report the position of a second dot relative to a first dot, after saccade execution (see **Figure 4A**). The two dot stimuli, presented below and above the saccade path, were horizontally displaced from each other to a variable degree. One dot was always presented before saccade onset, the other after saccade completion, so that the saccade dissociates retinal from screen coordinates. To perceive vertical displacement in screen coordinates, the saccade vector must be compensated for – likely through efference copy information. At saccade completion, on the retina the first probe falls to the right side of the fovea while the second falls to the left side (see **Figure 4B**, retinal coordinates). To compensate for the effects of the saccade, the visual system corrects the expected location of T2 in the opposite direction to the saccade (black arrow, remapped target position). If this correction is accurate, the displacement of the dots is perceived relatively vertical (Figure 4B, screen coordinates), rather than in retinotopic direction (relatively oblique, gray dashed line) and space constancy is maintained. Every shift from the vertical displacement indicates an under or over-compensation of the saccade vector. Previous studies have shown that the dots trajectory is indeed seen as approximately vertical, although a systematic slant of trajectories implies a tendency to overcompensate for the saccade vector[24,25]. However, if the retinotopic displacement is uncompensated, a failure in spatial stability is encountered. **Figure 5A** plots two example psychometric functions for one participant at the extremes of the lower (AQ = 3, in orange) and upper quartile of the distribution (AQ = 31, in magenta). The mean of the psychometric function (PSE) represented the location at which the references and the probe appeared to be vertically aligned. The PSE for the two example participants resulted very different: while ~0° bias represents accurate, spatiotopic correction, a negative bias (PSE < 0) reflects an under compensation of the saccade vector.

We first checked that all participants were able to execute accurate saccades. The saccade amplitude did not correlate with the autistic symptomatology of our participants ($r = -0.18$; $p = 0.33$; BF10 = 0.22, not shown as a figure). Moreover, when checking on the mean amplitude across trials (**Figure 5B**), we did not find a significant difference between the lower (mean degree ± SEM: 8.66 ± 0.27) and upper (mean degree ± SEM: 7.96 ± 0.46) quartile of participants AQ distribution (two sample t-test, $t(13) = 1.42$; $p = 0.17$; BF10 = 0.62). We then checked that all participants were able to complete the task. A measure of precision (JND) was extracted from each participant's psychometric function. **Figure 5C** shows the averaged JND for lower and upper quartile of the distribution. We report a non-significant difference between the upper and lower quartile of the autistic score's distribution ($t(13) = -0.46$; $p = 0.64$; BF10 = 0.29) nor a significant correlation between JND and the autistic quotient of all our participants ($r = 0.17$; $p = 0.36$; BF10 = 0.21, not shown as a figure). Indeed, the averaged precision in reporting the position of the second dot was very similar between the upper (mean ± SEM: 0.72 ± 0.04) and lower (mean ± SEM: 0.65 ± 0.14) quartile of the distribution.

By contrast, the averaged biases (expressed as the PSE) varied as a function of the autistic symptom severity (**Figure 5D**): the higher the autistic scores the stronger the bias in reporting the displacement in retinotopic coordinates, hence correcting less for the saccade eye movement ($r = -0.58$; $p < 0.001$; BF10 = 47.43). Indeed, while we report a slightly slant of the dots trajectory for the lower quartile of the distribution (mean ± SEM: 0.12 ± 0.13), the reported bias was slanted in the opposite direction for participants falling in the upper quartile (mean ± SEM: –0.64 ± 0.19), implying an under-compensation of the saccade vector and a partial failure in visual stability (see **Figure 5E** for a schematic illustration of the results).

This result is also confirmed by a significant difference between the two groups ($t(13) = 3.63$; $p = 0.003$; BF10 = 15.23; Fig4E, bar plot).

We finally examined the relationship between the motor and visual biases in the participants that completed both tasks. Figure 6 plots the angles of deviations (motor bias) as a function of the PSE of our participants (visual bias). We found that motor and visual biases very strongly correlated ($r = -0.57$; $p < 0.001$; BF10 = 37.4), suggesting that a similar mechanism – controlled by the same efference copy signal – might be responsible for both motor and visual updating.

DISCUSSION

In the current study we examined oculomotor efference copy signals in a sample of healthy adults with variable autistic traits using both, a motor and visual updating task. We show that a disrupted efference copy mechanism can lead to inaccurate predictions about impending eye movements, forcing the visuomotor system to rely more heavily on actual sensory input rather than the anticipated sensory consequences of self-generated eye movements.

In our first experiment, the double step saccade task, we found an impairment in the capacity of participants exhibiting high levels of autistic symptoms to program double-step - saccades which require efference copy information about the size of the previous saccade. Conversely, participants with relatively low autistic symptoms perform accurately in the double-step task. In the high-autism-symptom subsample, the final eye position after the second saccade markedly deviates from the position of the second target, and the magnitude of this error far surpasses that observed in the low-autism group. This specific impairment in the accuracy of second saccades lends support to the hypothesis that heightened autistic symptomatology correlates with a deficit in constructing a spatial representation of the second target's location based on forward models of efference copy. Notably, the mislocalization of the second saccade target cannot be explained by differences in the dynamics of the saccade to the first target: The observation that the accuracy of the initial saccade in the double step task remains consistent regardless of the degree of autism severity implies that individuals with more pronounced symptoms do not exhibit inherent challenges in the encoding and processing of spatial information for intended saccades. Instead, when preparing for a saccade that must compensate for a preceding eye movement that altered the retinotopic representation of the target, those with more severe symptoms encounter difficulties. Likewise, the bias in localizing the target cannot be attributed to working memory deficits within our high-autism-symptom subsample; it is improbable that the mislocalization of the second target results solely from documented working memory deficits in ASD[27]. Furthermore, in contrast to the symmetrically greater variability in saccade endpoints around the second target predicted by impaired working memory, we observed a systematic bias in the mislocalization of T2, consistent with the direction predicted by an impaired efference copy.

Individuals with ASD show remarkable similarities to the symptoms of schizophrenia patients[28-31]. Both ASD and schizophrenia patients are considered to have impairments at both extremes, involving either an excessive or inadequate reliance on sensory signals, resulting in inflexible behavior and issues in recurrent neural network representation[32,33]. Using a double-step saccade task, previous research has reported multiple lines of evidence demonstrating disrupted efference copy signals in schizophrenia. Importantly, similar to our findings, this disruption has implications for perceptual and motor precision, highlighting the interconnectedness of corollary discharge deficits and motor inflexibility[34,35].

A dramatic consequence of altered efference copy signaling, is the inability to account for own eye movements when judging the location of a target presented intra-saccadically. The trans-saccadic localization task yielded results that provide further insights into the role of efference copy in individuals with varying levels of autistic traits. In this task, all participants, regardless of their autistic trait levels, successfully executed accurate saccades to peripheral targets. However, the key factor emerged in the subsequent perceptual judgments made by these individuals. Individuals with high autistic traits exhibited a significant mislocalization of a remembered stimulus position across saccades. In essence, they perceived the visual stimulus as having shifted in space more than it actually did during the eye movement. This indicates that

their brain is relying more on the actual sensory feedback than the efference copy prediction. The consequence is an inaccurate perception of the stimulus's location in space.
The results of both experiments shed light on the relationship between efference copy failure and Bayesian theories of autism[9,10,36]. In the double step task, individuals with higher autistic traits exhibited difficulties in utilizing efference copy signals to construct a spatial representation of the second target location. This impairment suggests that in high autistic symptomatology pre-saccadic predictions will never be completely confirmed by the post-saccadic feedback. This constant lack of a successful match between predicted sensory consequences of self-generated eye movements and post-saccadic evidence can lead over time to the build-up of weak priors with high variance.
Similarly, in the trans-saccadic localization task, high autistic traits were associated with failures in spatial updating across saccade execution. These findings align with the Bayesian framework, where the brain continuously updates its internal model by combining sensory information with prior knowledge. In the context of autism, a failure in efference copy can further contribute to the unique sensory integration processes observed in individuals on the autism spectrum.

We have recently suggested a disruption in causal inference in the high autistic phenotype, as these individuals tend to attribute post-saccadic errors to external factors—a dynamic and unstable world—rather than accurately linking them to their own motor actions[4]. Consequently, a bias in causal inference emerges, stemming from the efference copy disruption, where the brain struggles to correctly attribute the cause of motor errors, leading to a heightened reliance on sensory feedback instead of motor predictions. As previously postulated this skewed causal inference also interconnect with motor inflexibility. Inflexibility in the motor domain might also have consequences for visual stability. Here, participants with high autistic traits faced challenges in executing saccades with precision, particularly when compensating for previous eye movements that had altered target representations. In the trans-saccadic localization task, we observed that these individuals leaned more heavily on post-saccadic sensory feedback than efference copy predictions. This not only led to an inaccurate perception of stimuli location but also underscored their limited ability to update and adapt motor actions based on sensory information.

In conclusion, our study underscores the critical role of efference copy in maintaining perceptual stability during eye movements and highlights its potential relevance to the sensory processing differences observed in individuals with heightened autistic traits. These findings contribute to our understanding of the complex interplay between prior knowledge, sensory integration, and motor control in the context of ASD, opening avenues for future research and potential interventions aimed at enhancing perceptual stability in this population.

METHODS

Participants

Forty-two participants (31 female, mean age 23, SD = 4.19) took part to Experiment 1 (motor updating). Thirty-one of them (21 females, mean age 22.81, SD = 3.11) participated also in Experiment 2 (visual updating). Subjects were either German native speakers or English speakers with no neurological or psychiatric diseases. Participants either reported to have normal vision or they wore lenses during their acquisition. All participants were recruited through the Heinrich-Heine University Düsseldorf and received either course credit or payment of 10 euros per hour. The experimental procedure was approved by the local ethics committee of the Faculty of Mathematics and Natural Sciences of Heinrich-Heine-University, Düsseldorf (ethics approval number: PO01_2022_01).

Experimental materials and procedures

Stimuli were displayed on a 27-inch Acer XB272 LCD monitor driven by a Alienware pc (Aurora R7) with a 240-Hz refresh rate (frame duration 4.16 ms) and a resolution of 1920 × 1080 pixels. The experimental program was implemented in MATLAB 2016b (Mathworks, Natick, MA, USA) using Psychtoolbox[37]. Eye movements and pupil diameters were recorded with the EyeLink 1000 system (SR Research Ltd., Mississauga, Ontario, Canada), which sampled eye positions at a rate of 1000 Hz. The head was sustained with a chin- and forehead-rest. For all participants the left eye was recorded. Viewing was binocular. At the beginning of each session, the Eyelink was calibrated with the standard nine-point Eyelink procedure. The system detected the start and the end of a saccade when eye velocity exceeded or fell below 30°/s.

Experiment 1: motor updating

A trial started with the presentation of a black fixation point (0.55 × 0.55°, FP) at the screen center. After 1000 ms plus a randomly chosen duration between 0–500 ms, the first saccade target ST1 (0.55 × 0.55°, black) appeared 10° to the right of the screen center (see **Figure 2A**), which remained visible for 64 ms. Upon extinction of ST1, ST2 appeared 10° upwards or downwards from ST1, and remained visible for another 64 ms. The position of ST2 was randomized across trials. ST2 and the fixation point were extinguished together, and this cued participants to start the saccade sequence: from the fixation point to ST1, and from ST1 to ST2. Participants completed 3 sessions of 100 trials each.

Experiment 2: visual updating

The trial sequence is shown in **Figure 4A and 3B**. A trial started with the presentation of a fixation point (black square FP, 0.55° × 0.55°) 5° to the left of the screen center. The fixation point stayed on for 1000 ms plus a randomly chosen duration between 0–500 ms. At a random time within the fixation point presentation time, the first stimulus was presented (Bottom Dot, BD). After disappearance of the first fixation point, participants were instructed to saccade to the second fixation point (black square FP, 0.55° × 0.55°) 5° to the right of the screen center (total saccade size 10°). At saccade completion, a second stimulus was delivered (Top Dot, TD). The stimuli consisted of two red dots (1.5° diameter), each flashed for one monitor frame with at least a temporal separation of 300 ms on average (see **Figure 4 C** for distribution of saccade offset and second stimulus onset). The first dot could appear randomly above or below gaze level at a fixed horizontal location, halfway between the two fixations (x = 0, y = –5° or +5° depending on the trial). The second dot was then shown orthogonal to the first one at a variable horizontal location (x = 5° ± 2.5°). The stimulus was perceived as a single dot moving downward or upward (depending on the position of the first dot) with a near-vertical trajectory, i.e. orthogonal to the direction of saccades. Participants completed 3 sessions of 110 trials each.

QUANTIFICATION AND STATISTICAL ANALYSIS

Experiment 1

In the analysis of eye movement data in Experiment 1 we excluded trials if: i) participants did not perform a saccade or they blinked during the execution of the saccade; ii) participants started the saccade sequence before the extinction of the fixation point; iii) the amplitude of the first saccade was smaller than half of the required distance, i.e. < 5°, iv) ,the vertical amplitude of the second saccade was smaller than 5°; v) participants deviated more than 2.5° on the horizontal or vertical dimension when fixating; vi) the latency of the first saccade was <100 ms. Saccade amplitudes were averaged into 10 separate bins of ~30 trials each for first, and second saccades that passed the selection criteria. These criteria were applied to ensure that both saccades were large enough to reveal a putative deviation of the second saccade. If, for instance, the executed first saccade is much smaller than the required distance, the efference copy should signal a smaller amplitude, thus leading to smaller influences on the direction of the second saccade. We tested our hypothesis concerning impaired use of efference copy information in high autism phenotype by analyzing the direction of the second saccade. Specifically, we determined how far the second saccade deviated from the optimal vector that would have directed the gaze onto the target. We calculated the angle between the second saccade vector and the optimal vector connecting the starting position of the second saccade and the second saccade target (see Figure 2B). These angles were computed separately for upward and downward saccades, but since they resulted in very similar values, we collapsed the data into upward angles only.

Experiment 2

We only analyzed trials where the saccade was performed between the presentation of the two dots (first dot at least 60 ms before the saccade, second dot after its completion), therefore, in retinotopic coordinates, the two dots were always displaced horizontally by about 10°. That is when the dots were presented on the screen, they appeared at a different location on the retina, horizontally separated by an angle of approximatively 10° (see also Figure 4B). In other words, each dot was perceived on the retina at a slightly different position in the visual field, due to horizontal displacement. That subjects perceived the dots displaced along a nearly vertical trajectory, indicates that the retinotopic displacement is largely compensated, ensuring spatial stability. However, small biases of reported direction can indicate relative failures in this stabilization process. To estimate biases in trans-saccadic updating, we varied the location of the second dot with the method of constant stimuli and asked subjects to report in 2AFC whether the second dot was more slanted to the right or to the left compared to the first one. Data were analyzed as psychometric functions, plotting the proportion of “correct” judgments as function of the position of the second dot relative to the first one. Distributions were fitted with cumulative Gaussian functions; the median of the curve estimated the PSE, or the position of the second dot that led to vertical stimulus displacement. A negative bias (PSE < 0) implies a bias towards seeing rightward displacement and a positive bias (PSE > 0) implies a bias towards seeing leftward displacement (as in Fig. 3B, dashed gray line). The former negative bias can be interpreted as an under-compensation of the saccade vector. Moreover, a trial was discarded if: i) participants did not perform a saccade or they blinked during the execution of the saccade; ii) participants started the saccade before the extinction of the fixation point; iii) the amplitude was smaller than half of the required distance, iv) participants deviated more than 2.5° on the horizontal or vertical dimension when fixating.

Autistic Quotient (AQ)

All participants completed the self-administered Autistic Quotient questionnaire, in the German or English validated version[38,39]. This contains 50 items, grouped in five subscales: attention switching, attention to detail,

imagination, communication, and social skills. For each question, participants read a statement and selected the degree to which the statement best described them: “strongly agree,” “slightly agree,” “slightly disagree,” and “strongly disagree”. The standard scoring described in the original paper was used: 1 when the participant’s response was characteristic of ASD (slightly or strongly), 0 otherwise. Total scores ranged between 0 and 50, with higher scores indicating higher degrees of autistic traits. All participants scored below 32, the threshold above which a clinical assessment is recommended[40].

The median of the scores for the first experiment was 16, with lower and upper quartiles of 13 and 19. Scores were normally distributed, as measured by the Jarque–Bera goodness-of-fit test of composite normality ($JB = 0.98$, $p = 0.5$). The median score of the subsample participants for experiment 2 was 17, with lower and upper quartiles of 14 and 20. Scores were normally distributed, as measured by the Jarque–Bera goodness-of-fit test of composite normality ($JB = 1.39$, $p = 0.31$).

As we were interested in the effect of autistic personality traits on the results, correlation analyses were complemented with standard t-tests comparing the upper and the lower quartile of the AQ distribution’s scores for the two experiments separately. Both measures were accompanied with Bayes Factors estimation. Bayes Factors[41] quantify the evidence for or against the null hypothesis as the ratio of the likelihoods for the experimental and the null hypothesis. We express it as the ratio, where negative numbers indicate that the null hypothesis is likely to be true, positive that it is more likely false. By convention, absolute Bayes factors > 3 are considered substantial evidence for either the alternate or null hypothesis, > 10 strong evidence, and > 100 decisive.

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AUTHOR CONTRIBUTIONS

All authors contributed to the study concept and to the design. Stimuli were designed by A.P. and A.P. performed the data analysis. All authors contributed to the interpretation of results. A.P. drafted the manuscript, and E.Z. provided critical revisions. All authors approved the final version of the manuscript for submission.

COMPETING INTERESTS

The authors declare no competing interests.

FIGURES AND CAPTIONS

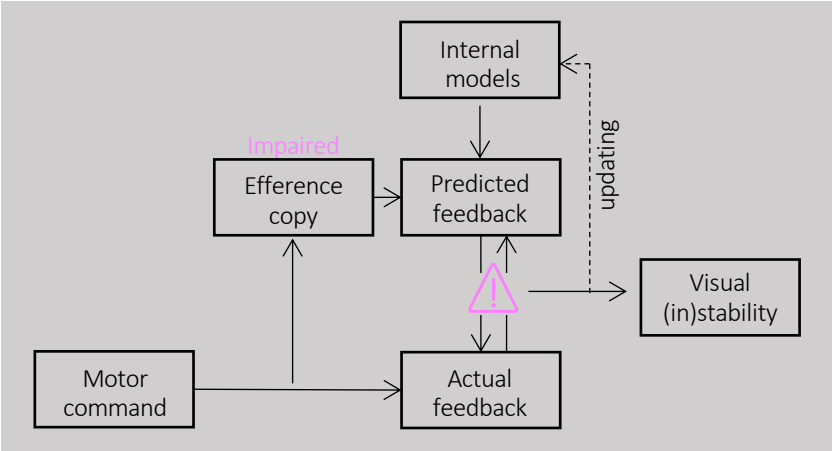

Figure 1. Schematic illustration of the proposed model. For saccade generation, a motor command is sent to the motor plan which produces the desired eye movement. A correct plan of the movement depends on a comparison between the position of the eye before and after a saccade and therefore on the accuracy of the efference copy signal. If the efference copy is impaired, pre saccadic prediction will be never completely confirmed by the post saccadic feedback. This constant lack of successful match between prediction and sensory evidence could influence the accumulation of sensory predictions over time, leading to a less stable perception.

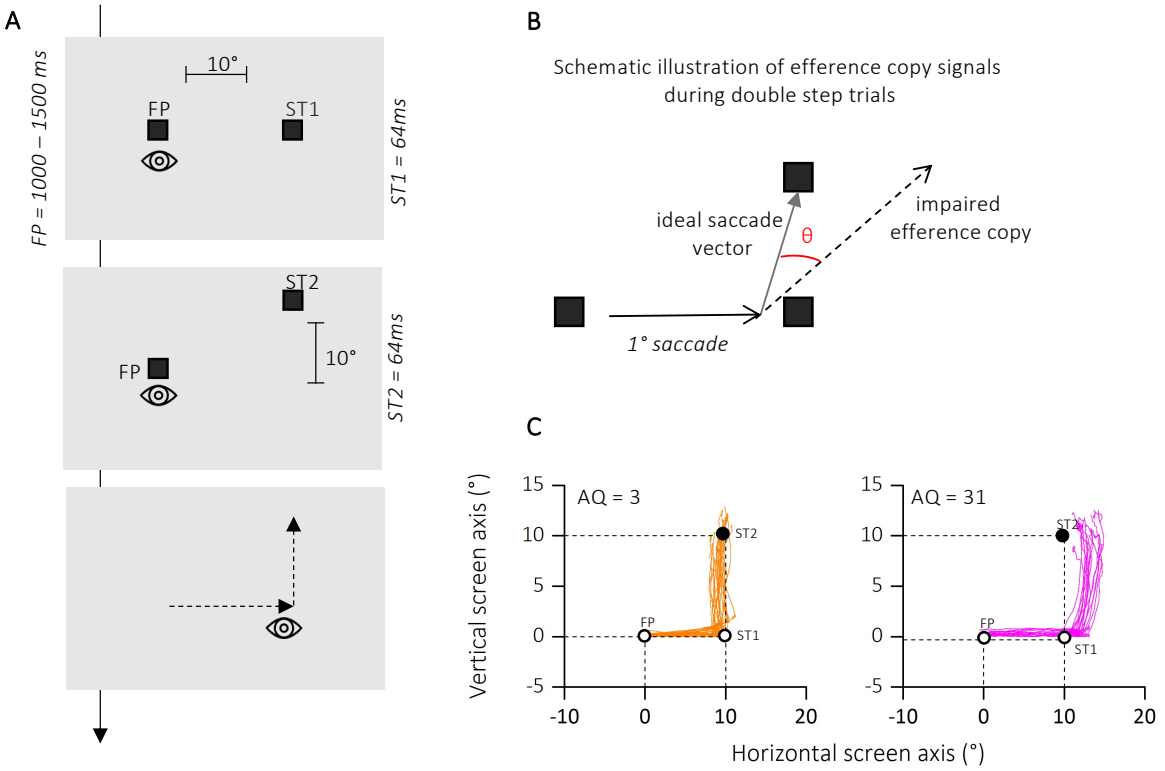

Figure 2. A) Time course of presentations and spatial arrangement of stimuli during double step task. Participants made double-step saccades from a fixation point (FP) to the first saccadic target (ST1), and from there to a second saccade target (ST2). Both FP and saccade targets were separated by 10°. Participants were instructed to initiate the saccade only after all stimuli had disappeared, so to avoid any visual references. B) Hypothetical pattern of first and second saccades on a particular trial and illustration of the variables angle shift and horizontal endpoint shift of second saccades. We determined how far the second saccade deviated from the optimal vector that would have directed the gaze onto the target. We calculated the angle between the second saccade vector and the optimal vector connecting the starting position of the second saccade and the second saccade target. These angles were computed separately for upward and downward saccades, but since they resulted in very similar values, we collapsed the data into upward angles only. C) Eye position traces for two representative participants. The eye position between the initiation of the first saccade from FP toward ST1 (empty circles) and the termination of the second saccade toward ST2 (filled circle) are depicted for each trial in which successive saccades to ST1 and ST2 were produced. Plots are separated for low autistic traits (AQ = 3, in orange) and high autistic traits (AQ = 31, in magenta) example participants.

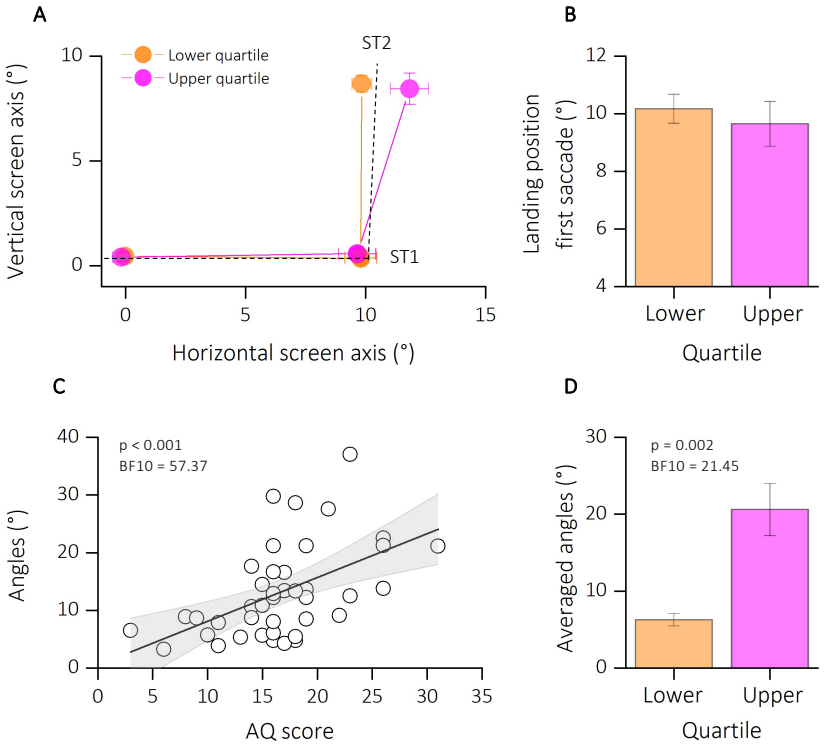

Figure 3. **A)** Average saccade vectors from FP (0, 0) to ST1 (10, 0) and from ST1 to ST2 (10, 10). Data are shows for a subsample of participants, falling in the lower (orange) and upper (magenta) quartile of the autistic quotient distribution. Data from upward and downward second saccades are collapsed. Dashed lines indicate the required saccade path from FP to ST1 and from ST1 to ST2. **B)** Average first saccade landing position for lower and upper quartile of the AQ distribution. Error bars are SEM. **C)** Linear regression between angles (distance the actual second saccade vector and the optimal vector that would have led to the physical target position ST2 from the second saccades' starting position) and the Autistic Score of participants. Text insets report *p*-values and associated Bayes Factors of Pearson's Rho. Thick black line shows the linear fit through the data. The shaded area represents the standard error for each observation mean as predicted by the regression line. **D)** Average angles (°) for lower (AQ < 13, orange) and upper (AQ > 19, magenta) quartile of the AQ distribution. Error bars are SEM. Text insets report *p*-values and associated Bayes Factors of paired sample T-test.

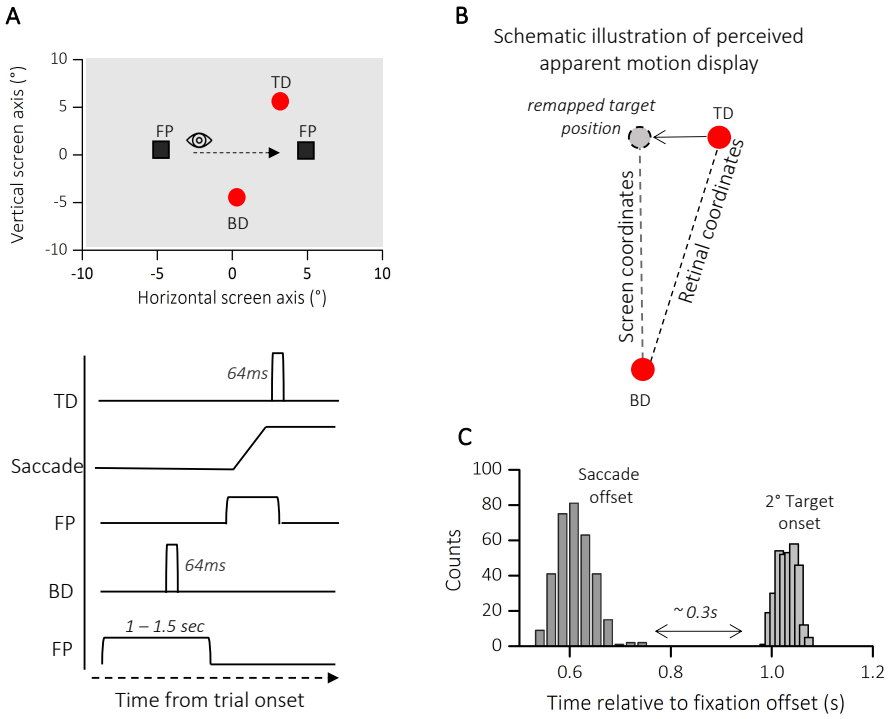

Figure 4. A) Spatial arrangement of stimuli and time course of presentations during trans-saccadic localization task. Participants made a saccade from a fixation point (FP, left) to a second fixation point (FP, right). At a random time before the first FP disappearance, a red circle (Bottom Dot, BD) was presented for 64ms, at saccade completion a second red dot (Top Dot, TD) was delivered for another 64ms. Participants were instructed to initiate the saccade only after the first FP had disappeared. B) Schematic illustration of perceived apparent motion display. On the retina, the first probe falls to the right side of the fovea while the second falls to the left side. To compensate for these effects of the saccade, the visual system corrects the expected location of the second dot in the opposite direction to the saccade (remapped target position, gray dot). If this correction is accurate, the displacement is perceived in its spatiotopic (vertical dashed line: from remapped target position to BD) rather than retinotopic arrangement (oblique dashed line: from TD to BD) and space constancy is maintained. C) Average frequency distribution of saccade offsets and second target onset. We analyzed only the trials in which the second target was delivered after saccade completion (on average around 0.3 sec), so that the visual system could dissociate the retinal from the screen coordinates.

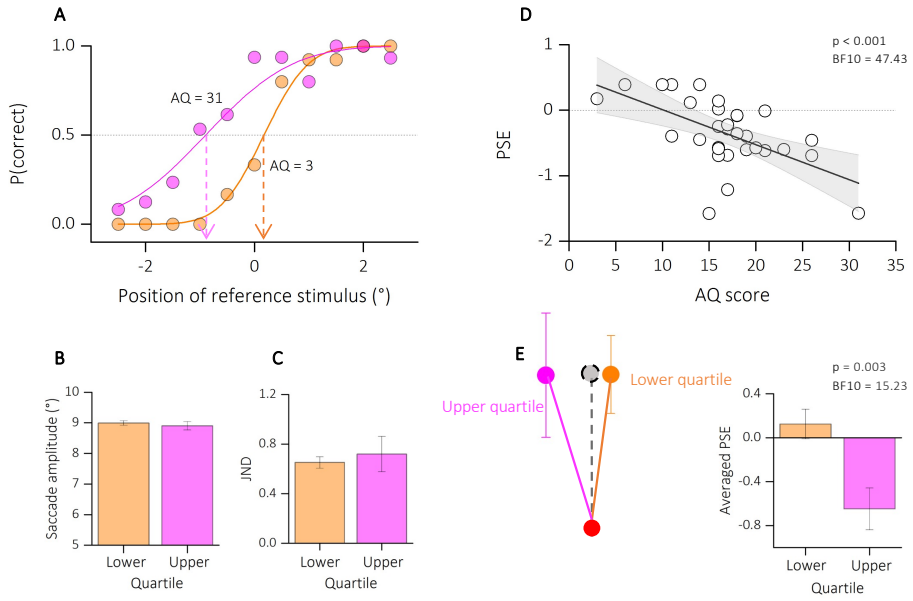

Figure 5. A) Psychometric functions for two example participants at the very extremes of the autistic quotient distribution (AQ = 3, in orange; AQ = 31, in magenta). The functions plot the proportion of correct responses, as function of the position of the second dot relative to the first one (shown in the abscissa). The vertical-colored lines show the estimates of the PSE, given by the median of the fitted cumulative Gaussian functions. **B)** Average saccade amplitude (°) for lower (AQ < 14, in orange) and upper (AQ > 20, in magenta) quartile of the autism distribution. Saccade amplitude did not vary with autistic traits. **C)** Precision thresholds (JND) for lower and upper quartile of the AQ distribution, same convention as in B. Here, the precision in reporting the dot displacement was similar between sub-samples. **D)** Linear regression between PSE and the Autistic score of participants. Text insets report *p*-values and associated Bayes Factors of Pearson's Rho. Thick black line shows the linear fit through the data. The shaded area represents the standard error for each observation mean as predicted by the regression line. **E)** Schematic illustration of perceived dots displacement for upper and lower quartile of the autistic quotient. A localization compensating fully for the saccadic eye movement would lead to a perfect vertical localization (gray dot, dashed line). However, deviations from the vertical indicate and under or overcompensation of the saccade vector. We found a large under compensation of the saccade vector for the participants with higher autistic symptoms, compared to participants with lower autistic symptoms. See also bar plot, average PSE, on the right. Text insets report *p*-values and associated Bayes Factors of two sample T-test.

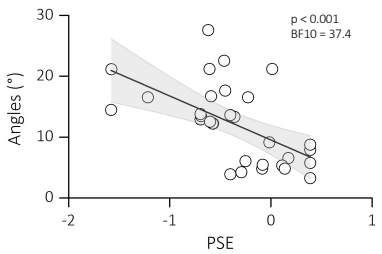

Figure 6. Linear regression between PSEs of the trans-saccadic localization task and the angles of deviation from the double step saccade task. Text insets report *p*-values and associated Bayes Factors of Pearson's Rho. Thick black line shows the linear fit through the data. The shaded area represents the standard error for each observation mean as predicted by the regression line.