

No increased circular inference in autism or autistic traits

Nikitas Angeletos Chrysaitis¹, Renaud Jardri²³, Sophie Denève², and Peggy Seriès^{1*}

¹Institute for Adaptive and Neural Computation, School of Informatics, University of Edinburgh, Edinburgh, United Kingdom

²École Normale Supérieure, Institut de Sciences Cognitives, LNC (INSERM U960), Paris, F-75005, France

³Université de Lille, INSERM U1172, Lille Neuroscience & Cognition Research Centre, Plasticity & Subjectivity (PSY) team, CHU Lille, 59037 Lille, France

*** Corresponding author: peries@inf.ed.ac.uk**

Short title: No increased circular inference in autism or autistic traits

Keywords: Circular Inference; Bayesian Brain; Autism; Schizophrenia; Computational Psychiatry; Excitation-to-inhibition ratio

Abstract

Autism spectrum disorders have been proposed to arise from impairments in the probabilistic integration of prior knowledge with sensory inputs. Circular inference is one such possible impairment, in which excitation-to-inhibition imbalances in the cerebral cortex cause the reverberation and amplification of prior beliefs and sensory information. Recent empirical work has associated circular inference with the clinical dimensions of schizophrenia. Inhibition impairments have also been observed in autism, suggesting that signal reverberation might be present in that condition as well. In this study, we collected data from 21 participants with diagnosed autism spectrum disorders and 155 participants with a broad range of autistic traits in an online probabilistic decision-making task (the fisher task). We used previously established Bayesian models to investigate possible associations between autism or autistic traits and circular inference. No differences in prior or likelihood reverberation were found between autistic participants and those with no diagnosis. Similarly, there was no correlation between any of the circular inference model parameters and autistic traits across the whole sample. Furthermore, participants incorporated information from both priors and likelihoods in their decisions, with no relationship between their weights and psychiatric traits, contrary to what common theories for both autism and schizophrenia would suggest. These findings suggest that there is no increased signal reverberation in autism, despite the known presence of excitation-to-inhibition imbalances. They can be used to further contrast and refine the Bayesian theories of schizophrenia and autism, revealing a divergence in the computational mechanisms underlying the two conditions.

Author Summary

Perception results from the combination of our sensory inputs with our brain’s previous knowledge of the environment. This is usually described as a process of *Bayesian inference* or *predictive coding* and is thought to underly a multitude of cognitive modalities. Impairments in this process are thought to explain various psychiatric disorders, in particular autism and schizophrenia, for which similar

Bayesian theories have been proposed despite important differences in their symptoms. Recently, a new model of Bayesian impairment in schizophrenia has been proposed and validated using behavioural experiments, called the “circular inference” model. In the current study, we used the same task and computational modelling to explore whether circular inference could also account for autism spectrum disorder. We find that participants with autistic traits or diagnoses of autism do not present increased levels of circularity. This is the first study to investigate circular inference in autism, and one of the very few to explore possible autism and schizophrenia impairments with the same task and identical analytical methods. Our findings indicate one potential way in which the explanations of the two conditions might differ.

Introduction

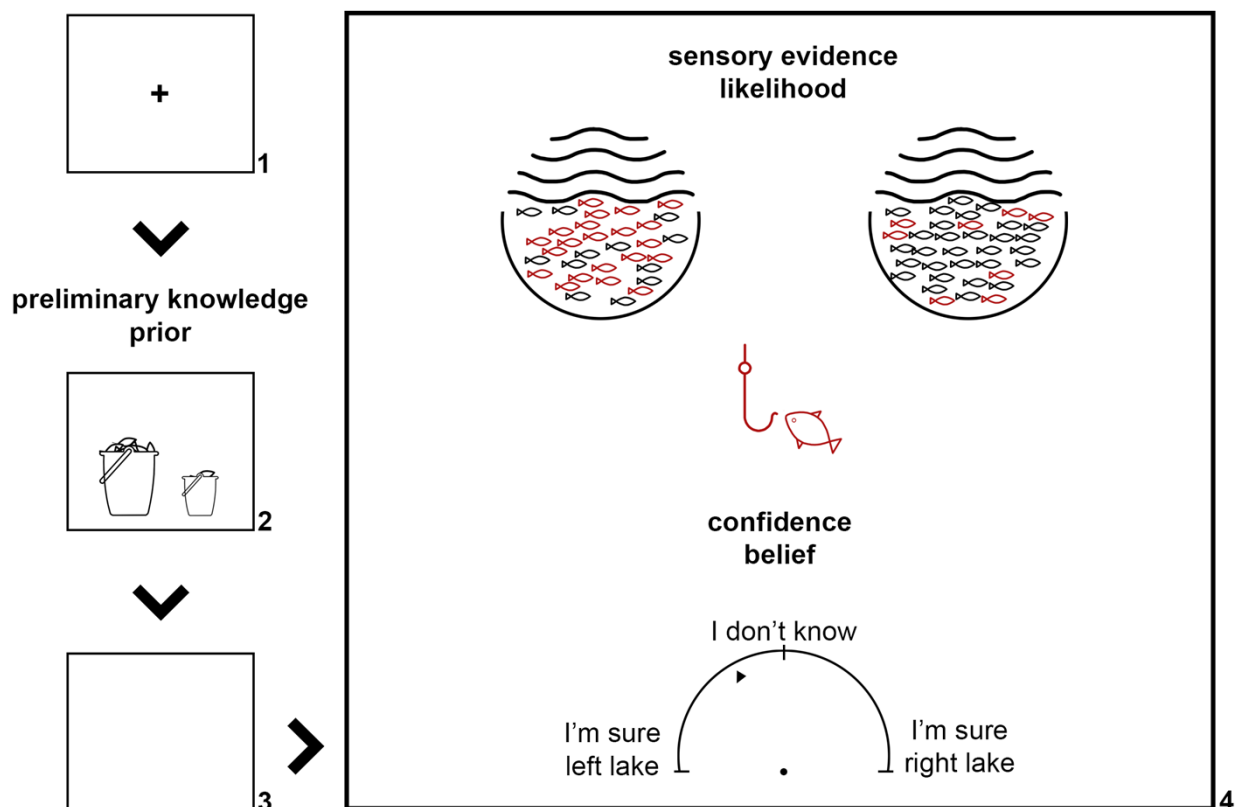
Autism spectrum disorder (ASD) and schizophrenia (SCZ) are two heterogeneous mental disorders with a complicated relationship [1,2]. While the term “autism” was initially used to refer to one of schizophrenia’s symptoms [3], the two disorders have since been considered as separate conditions and have been studied as such by most researchers. Despite that, numerous links have been observed between them, from behavioral and neurophysiological similarities in social cognition impairments [4,5], to immune [6] or intestinal [7] dysregulation and genetic overlap [8], among others. Such findings suggest that the relationship between schizophrenia and ASD should be more thoroughly explored, within a framework that is able to handle and explain their differences [9,10].

In Bayesian theories of perception and cognition, the brain is viewed as constantly making probabilistic calculations in order to infer the true state of the environment. The information coming from sensory inputs is captured by the likelihood function and is combined with prior beliefs about the environment, in a process akin to Bayesian inference [11]. This framework has been widely adopted in both ASD and SCZ research, with a frequently proposed hypothesis for both disorders being that sensory inputs are overweighted relative to prior beliefs [12–16] (see [17–19] for an alternative SCZ hypothesis). In schizophrenia, this theory attempts to explain the tendency of patients to jump to conclusions [20] and their partial immunity to perceptual illusions [21], with hallucinations and delusions being interpreted as the formation of bizarre beliefs to account for strange, hypersalient sensory data [22]. Intriguingly, the hypothesis of overweighted sensory information is also suggested to account for most of ASD’s symptoms, such as sociocognitive impairments, attention to detail, sensory hypersensitivity, and decreased susceptibility to illusions [15]. The similarity of the proposed theories for autism and schizophrenia is surprising given their distinct symptomatology. However, very few Bayesian studies have examined both conditions using the same experimental or computational paradigm, which would be crucial for understanding their relationship and mechanisms of action.

In 2013, Jardri and Denève proposed a new computational explanation for schizophrenia, called *Circular Inference* [23], motivated by an attempt to understand the potential consequences of the increased excitation-to-inhibition (E/I) ratio that is associated with the condition [24,25]. Using hierarchical network simulations, they showed that inhibitory impairments in the cortex might lead to sensory evidence or prior beliefs being reverberated throughout the network that the brain uses to represent the environment, overwhelming the inferential process. Sensory input reverberation could cause the reported “jumping to conclusions” bias in schizophrenia, where patients get overconfident in their beliefs based on relatively little evidence [26]. The positive symptoms, then, can be seen as an extension of the same process, where hallucinations and delusions are produced by misplaced certainty in noisy perceptual and other non-sensory information, respectively.

Jardri et al. supported this hypothesis with behavioral evidence from 25 SCZ patients and 25 controls [27], using a probabilistic variant of the beads task [28], called the *fisher task*. In the fisher task, subjects are asked to estimate the chance that a red fish caught by a fisherman came from one of two lakes, while being presented with the lake preferences of the fisherman and the proportions of red fish in each lake (Fig 1). The researchers interpreted the preferences (which were presented first) as a Bayesian prior and the fish proportions as the sensory evidence. They showed that all participants exhibited signs of signal reverberation. Importantly, they found that sensory evidence was reverberated more in patients, with the magnitude of reverberation being correlated with their positive symptoms. A following study confirmed these findings, utilizing a social version of the beads task in a sample of 35 patients with schizophrenia or schizoaffective disorder and 40 controls [29]. The researchers found that the circular inference model fitted best the participants’ behavior, with increased sensory reverberation in patients. They also presented strong evidence for an association between that reverberation and various clinical features in patients (e.g., delusions, anhedonia-asociality).

112



113 **Fig 1. An outline of the four stages of the fisher task.**

114 1) The fixation cross is presented; 2) participants are shown the preference of the fisherman,
115 visualized as two baskets of varying sizes, one for each lake; 3) a blank screen is presented; 4)
116 participants are shown the fish proportions and are asked to make a confidence estimate about the
117 lake of origin of the fish (Adapted from [27]).

118
119 Impaired inhibition has been strongly associated with autism [30–34]. A question that arises naturally
120 is, therefore, whether circular inferences are present in ASD, and whether they would then be of the
121 same nature as in schizophrenia (e.g., sensory vs prior reverberation). In the present study, we aimed
122 to assess cue integration in both autistic participants [“autistic” is the preferred term by people on
123 the autism spectrum [35]] and participants with no ASD diagnosis but a broad range of autistic traits.
124 This allowed us to investigate signal reverberation within a dimensional as well as a more traditional,
125 categorical view of autism [36–39]. To achieve that, we utilized an online version of the fisher task,

126 and both circular inference and more traditional Bayesian models. This provided us with an
127 opportunity to explore the influences of ASD in probabilistic decision-making and directly compare
128 them with past SCZ findings.

Methods and Materials

Sample

We recruited 204 naive participants; 61 voluntarily via our social media networks and 143 with fixed monetary compensation via the Prolific recruiting platform [40]. All participants had normal or corrected-to-normal vision and were not taking any psychotropic medication. 28 subjects were excluded for providing low quality data (Section A3 in S1 Supplementary Information). The final sample included 102 male and 71 female participants, with a median age of 26.6 years. Our study was approved by the University of Edinburgh, School of Informatics Ethics Committee (RT number 29368).

Half of the Prolific subsample was selected to have a diagnosis of ASD or to identify as part of the autism spectrum (Section A1 in S1 Supplementary Information), with 21 subjects having a diagnosis in the final sample. All participants filled in the Autism Spectrum Quotient (AQ) questionnaire [41] and the 21-item Peters et al. Delusions Inventory (PDI) [42]. The final sample showed indeed stronger autistic traits ($M=22.9$, $SD=6.5$) than what is usually found in the general population ($M=16.9$, $SD=5.6$) [43], but no difference in delusional ideation ($M=6.1$, $SD=3.1$ vs $M=6.7$, $SD=4.4$) [42]. Interestingly, the participants with the ASD diagnoses had AQ scores on the low-end ($M=28.0$, $SD=8.0$) compared to those reported in the literature for autistic individuals ($M=35.2$, $SD=6.3$) [43]. Statistical power for our tests could not be calculated, as model parameters were not verifiably following any known distribution. However, the strength of Jardri et al.'s findings [27] suggests that comparable effects would reach statistical significance in our larger sample, according to an exploratory analysis (Section A4 in S1 Supplementary Information).

Procedure

The task was kept as similar to the original fisher task [27] as possible. The participants were shown a fisherman having caught a red fish and were asked which of two lakes the fish was caught from.

To make this decision, they were presented with two kinds of information in each trial: 1) the preferences of the fisherman for each of the lakes, visualized as two baskets of varying sizes (prior); 2) the proportions of red versus black fish in each lake, visualized as 100 fish in two lake drawings (sensory evidence or likelihood). Subjects were instructed to gauge their confidence and respond using a continuous semi-circular scale, ranging from “I’m sure LEFT LAKE” to “I’m sure RIGHT LAKE”, with “I don’t know” in the middle. Confidence estimates were interpreted probabilistically, in a continuous manner, with a click on the left edge of the scale corresponding to a probability of 1 for the fish originating in the left lake (0 for the right) and vice versa.

Trials were structured as follows (Fig 1): Initially, a fixation cross was shown for 800ms, followed by the two baskets for 1000ms, and a blank screen lasting 50ms. Then, the lake drawings, the fisherman with the red fish, and the scale appeared until the subject gave a response. Participants were presented with detailed instructions telling them to respond “as fast and as accurately as possible”, which they could repeat many times before proceeding to the task. After the instructions, subjects completed 11 training trials with easy stimulus combinations to acclimate themselves with the task.

Due to concerns about participants' potential distractibility in an online environment if the task was too long, we reduced the number of trials to 130 (Section A2 in S1 Supplementary Information). The trials appeared in a random order, with lake drawings being different for every trial. Every 22 trials, the participants were prompted to take a break, which they could end with the press of a button. Lakes had 9 possible ratios of red to black fish, while baskets appeared in 9 possible sizes, both corresponding to the probabilities 0.1 to 0.9. In all trials, likelihoods and priors were complimentary (e.g., if the left fish proportions were 0.3, the right would be 0.7). Therefore, probabilities mentioned in the text refer to the left lake, as the probabilities for the right can be immediately inferred.

Model-free analysis

A linear mixed-effects model was used to verify that the task components contributed to the participant responses in the expected way and as an initial exploration of the possible effects of autistic traits. We chose the absolute confidence of the participants as the response variable ($|c - 0.5|$, with c being the participant confidence estimate). We modelled the following as fixed effects with repeated measures across the subjects: i) the absolute likelihood ($|likelihood - 0.5|$); ii) the prior congruency, that is how much the prior agreed with the likelihood ($|prior - 0.5| \cdot \text{sgn}[(prior - 0.5)(likelihood - 0.5)]$); iii) the total AQ scores; iv) the PDI Y/N scores; v) the reaction times. The latter one was used to investigate the possibility of a speed-accuracy trade-off. The participants were modelled as a random effect.

Bayesian models

Data were fitted with four models: Simple Bayes (SB), Weighted Bayes (WB), and two variants of the circular inference model: Circular Inference – Interference (CII) and Circular Inference – No Interference (CINI). Originally [23], the inferential processes expressed by these models were simulated in a hierarchical network, where priors corresponded to top-down signals and likelihoods to bottom-up ones. In the current study, we followed Jardri et al. in fitting simplified models, that capture the network effects with significantly fewer free parameters [27].

SB combines the two sources of information using Bayes' theorem. This is expressed in logits as

$$L_c = L_p + L_s ,$$

with subscript p corresponding to trial prior, s to sensory evidence, and c to the confidence estimate, while L denotes the respective logit.

WB expands upon SB:

$$L_c = F(L_p, w_p) + F(L_s, w_s) ,$$

where F is the sigmoid function

$$F(L, w) = \ln \left(\frac{we^L + 1 - w}{(1 - w)e^L + w} \right),$$

allowing for the underweighting of priors or likelihoods. Each weight w expresses the reliability of the corresponding signal.

CII has the form:

$$L_c = F(L_p + I, w_p) + F(L_s + I, w_s),$$

$$I = F(a_p L_p, w_p) + F(a_s L_s, w_s),$$

where top-down and bottom-up signals get reverberated, interfering with one another, and end up corrupting prior beliefs and sensory evidence by the same amount, I . Parameters a_p and a_s affect the number of times the respective information is overcounted, expressing the signals' reverberation.

CINI is similar to CII, but it assumes that both signals get reverberated or overcounted separately and are only combined at the end of the process:

$$L_c = F(L_p + F(a_p L_p, w_p), w_p) + F(L_s + F(a_s L_s, w_s), w_s).$$

SB has 0 free parameters, WB 2, and both CII and CINI have the same 4. All parameters take values within $[0, 1]$ (for details see Section A5 in S1 Supplementary Information). A weight value of $w=0$ shows no influence of the corresponding signal, while both $w=1$ make WB equivalent to SB and both $a=0$ make CII and CINI equivalent to WB. The difference between CII and CINI is subtle, but important. In CINI, the sensory and prior signals are combined linearly, while in CII, one signal's influence on the model estimate depends on the strength of the other, due to the interference between them. Figure 2 illustrates the behavioral patterns predicted by the different models.

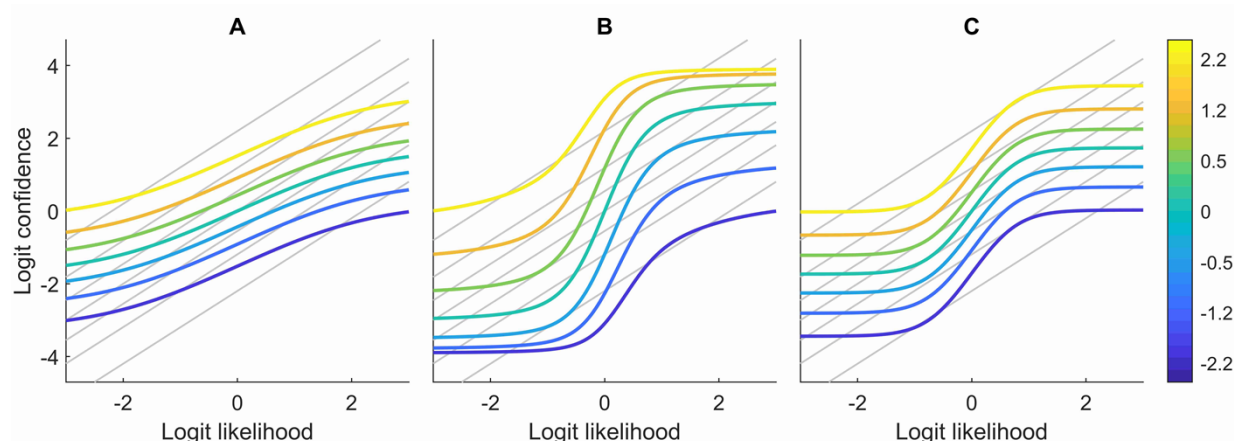


Fig 2. Illustration of WB (A), CII (B), and CINI(C) behavior.

The graphs show how logit model confidence estimates change as a function of logit likelihood (fish proportions). Different colors represent different prior values (basket size) and grey lines represent the SB model predictions. The SB model simply combines the information of the two signals by adding their logits. WB can underweight either or both signals, while, in addition to that, the circular inference models allow for signal overcounting. In CII, the contribution of the likelihood on the confidence estimate depends on the prior value and vice versa. In contrast with that, in the CINI model, each source of information affects the confidence independently, and therefore the graph lines are completely parallel to each other. Parameter values were the same for all models ($\alpha_p = 0.02$, $\alpha_s = 0.05$, $w_p = 0.8$, $w_s = 0.06$).

We followed Jardri et al., assuming Gaussian noise in the logit model estimates (L_c), and therefore fitted models via least squares, which is equivalent to maximum likelihood estimation in that case. Model comparison was performed using an approximation of the Bayesian information criterion (BIC) for normally distributed errors,

$$\text{BIC} = n \ln(\sigma^2) + k \ln(n) ,$$

where n is the number of datapoints, k the number of free parameters, and σ the model's mean squared error. To choose a model across all subjects, we followed the random-effects Bayesian model selection [44], implemented in the SPM12 [45]. Group-level BIC [46], a fixed-effects approach,

produced similar results. Experiment code, models, and anonymized data can be found at <https://osf.io/yqug2/>.

Statistical analysis and validity of results

We investigated the hypothesis of an association between autism and circular inference (H_1) in three ways: 1) correlations between model parameters and total AQ scores; 2) differences between the low- and high-AQ groups, defined as participants in the top and bottom 15% of the sample ($AQ \geq 30$, $n=29$ vs $AQ \leq 16$, $n=30$); 3) differences between subjects with an ASD diagnosis and those without, who also did not identify as part of the autism spectrum (ASD, $n=21$ vs ND, $n=61$; answers 1, 2 vs 5 in Section A1 in S1 Supplementary Information). The nonparametric measures of Kendall rank correlation coefficient and Mann-Whitney U test were chosen, as model parameters were not normally distributed (*Shapiro-Wilk test*; $p \leq 0.0068$) and there is no reason to expect a linear relationship between them and psychiatric traits. The common language effect size statistic (f) was reported for the Mann-Whitney U [47]. All analyses were performed in MATLAB R2020a.

To quantify the evidence for the null hypothesis (H_0) in favor of the alternative one (H_1), we calculated the Bayes factors BF_{01} for each of our tests. $1 < BF_{01} \leq 3$ constitutes weak evidence in favor of H_0 , $3 < BF_{01} \leq 20$ positive evidence, and $BF_{01} > 20$ strong [48]. Note that $BF_{10} = 1/BF_{01}$. Bayes factors were calculated in JASP 0.14, using the default priors [49]. To verify the fitting and model selection processes, we performed parameter and model recovery on CINI and CII (Section A6 in S1 Supplementary Information), as SB and WB scored very poorly in model comparisons.

Both models showed moderate recovery for the reverberation parameters (CINI a_p , $r=0.54$; a_s , $r=0.58$; CII a_p , $r=0.54$; a_s , $r=0.71$), although this was partly due to Pearson's correlation sensitivity to outliers [50] (Fig A2 and A3 in S1 Supplementary Information). The models exhibited excellent recovery for the weight parameters (CINI w_p , $r=0.96$; w_s , $r=0.91$; CII w_p , $r=0.94$; w_s , $r=0.93$). They

also showed no correlation between different parameters (Table A3 and A4 in S1 Supplementary Information). Model recovery was good for both models, with approximately 80% of the simulated participants being better fitted by their generating model (Table 1).

Table 1. Confusion matrix for model recovery.

		Recovered	
		CINI	CII
Simulated	CINI	799	201
	CII	185	815

Perfect model recovery would result in 1000 participants in the (CINI, CINI) and (CII, CII) cells, and 0 in the rest.

Results

Model-free findings

Participant responses adapted to changes in both priors and likelihoods, showing that they took both sources of information into account to make their confidence estimate (Fig 3). Despite that, their behavior was not strictly Bayesian. A change from 0.5 to 0.4 or 0.6 in either prior or likelihood corresponded to a disproportionately large shift in the average response, indicative of signal reverberation.

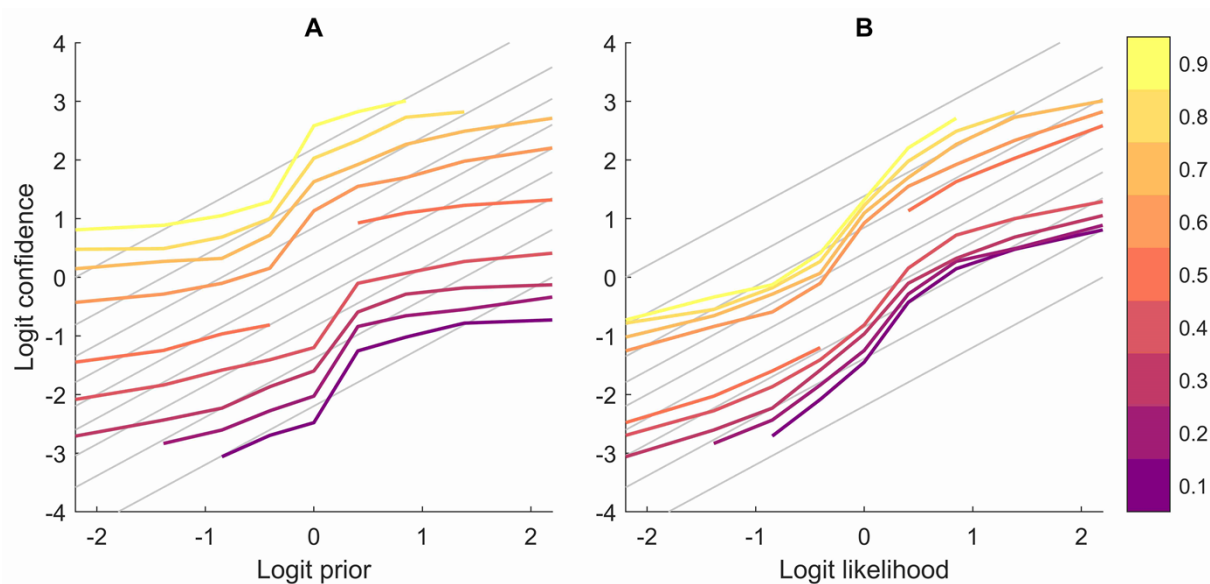


Fig 3. Average logit confidence estimates for all participants as a function of priors (A) and likelihoods (B).

Logit confidence estimates for the left lake increase following an increase in either prior probability for the left lake (baskets) or likelihood (fish ratios), showing that participants incorporate both information sources in their decision-making. However, their behavior is far from strictly Bayesian, as evidenced by the differences between colored and grey lines (SB confidence estimates). Different colors correspond to different likelihood (probability) values in the left graph and different prior (probability) values in the right.

The fitting of a linear mixed-effects model, predicting the absolute confidence estimate, confirmed the influence of both absolute likelihood ($t=40.30$, $p<10^{-323}$) and prior congruency ($t=103.02$, $p<10^{-323}$). It also showed a slight positive association with non-clinical delusional beliefs (PDI) ($t=2.99$, $p=0.003$) and a strong association with reaction times, with higher values corresponding to clicking towards the center of the scale ($t=-18.49$, $p=10^{-75}$). The influence of autistic traits (AQ) on absolute confidence was weak and non-significant ($t=-1.75$, $p=0.08$). Neither AQ nor PDI were correlated with mean reaction times (AQ $\tau=-0.06$, $p=0.29$; PDI, $\tau=0.05$, $p=0.33$). The relationship between reaction times and absolute confidence might be a result of participants taking more time to respond when they are uncertain [51].

Model-based findings

Both random- and fixed-effects model comparisons showed that Circular Inference – No Interference was the best fitting model, followed by Circular Inference – Interference (Fig 4). Since model fit plots showed that both CINI and CII fit the data relatively well (Fig C1 in S1 Supplementary Information), for the sake of completeness, we conducted the same analysis with parameters from both models. Results from CINI are reported below, while those from CII can be found in S1 Supplementary Information (Section C2).

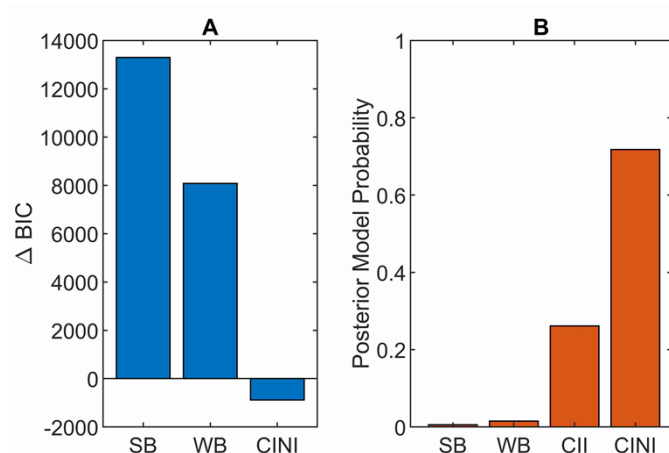
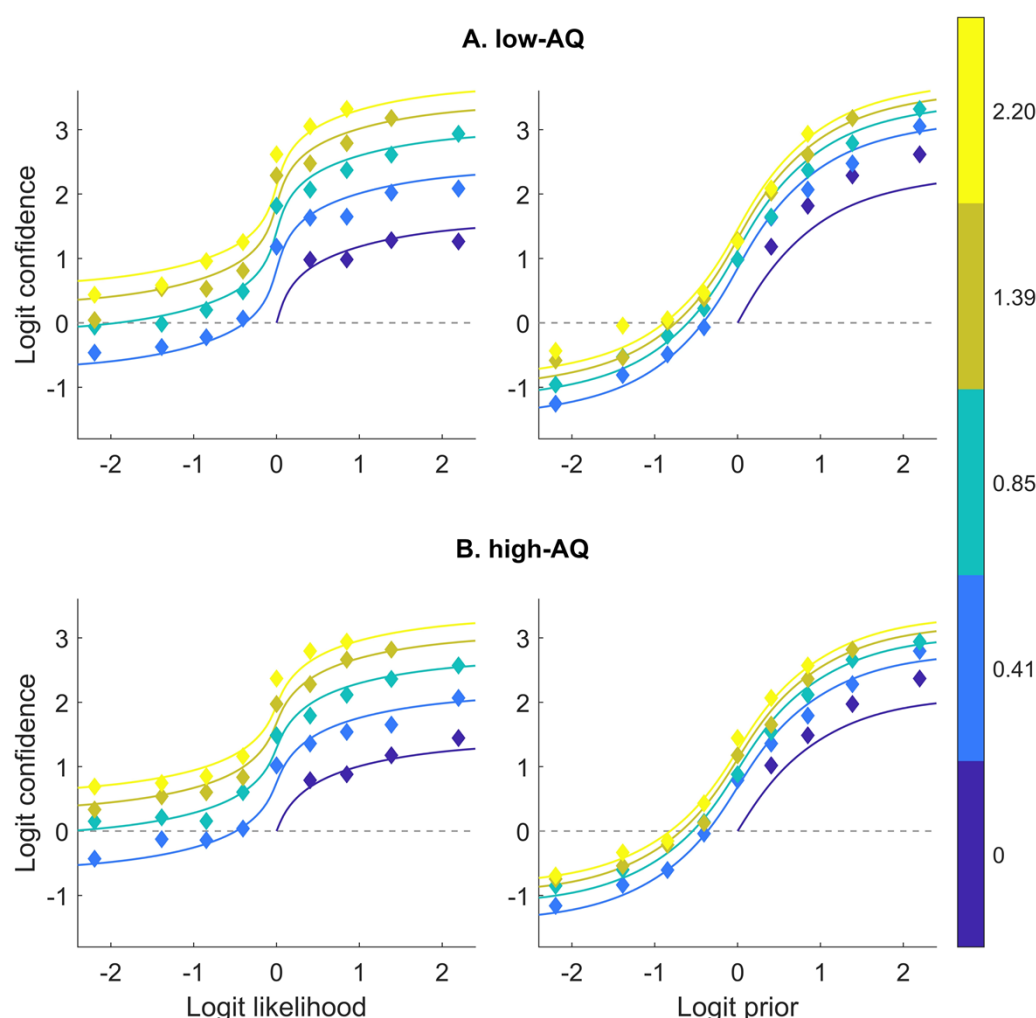


Fig 4. Results of fixed (A) and random (B) model comparisons.

(A) Group-level ΔBIC is defined as the sum of individual participant BIC scores for each model minus the sum for CII, used as a baseline, as it was the winning model in the Jardri et al. study [27]. The lower the BIC the better the model, with differences of more than 20 between BIC scores considered very strong evidence [48]. ΔBIC for CII is by definition 0. (B) Posterior model probabilities calculated using Bayesian model selection [44]. Both measures clearly show that Circular Inference models better account for the data, with CINI being a slightly better fit than CII.

There was no evidence of a relationship between prior or likelihood reverberation and total AQ scores (Table 2). The only correlation that reached an (uncorrected) p -value of lower than 0.05 was a negative correlation between AQ and the CINI prior weight ($\tau = -0.12$, $p = 0.02$, $BF_{10} = 1.57$), but this did not survive controlling for multiple comparisons [52]. Furthermore, the low- and the high-AQ groups behaved in a similar way (Fig 5), and the comparison between the parameters of high- and low-AQ groups did not reveal any difference, neither did the comparison between the ASD participants and those with no diagnosis (ND) (Fig 6, Table 3). Since it is possible that ND subjects with high autistic traits have an undiagnosed autism spectrum disorder, we performed an additional comparison between the ASD group and the subgroup of ND participants with weak autistic traits ($AQ \leq 17$, $n = 21$). No difference between these groups was found (Section C1 in S1 Supplementary Information). A weak positive correlation was found between PDI and the likelihood weight ($\tau = 0.13$, $p = 0.02$, $BF_{10} = 2.08$; Table 2), that again is not significant when corrected. No relationship was present between psychiatric traits or diagnoses and CII parameters (Section C2 in S1 Supplementary Information).

341



342 **Fig 5. Participant confidence estimates and CINI model fits for the low-AQ (A) and the high-**
 343 **AQ (B) groups.**

344 *Model and participant logit confidence as function of logit likelihoods and priors. Colored lines*
 345 *represent model predictions and rhombuses the participant confidence estimates. Different colors*
 346 *represent logit likelihood in A and logit prior values in B, and are equivalent to probabilities of 0.5*
 347 *to 0.9. Since both the task and the CINI model structure are symmetrical around 0 logit confidence*
 348 *(0.5 probability), participant estimates have been averaged between symmetric trials to reduce noise*
 349 *(e.g., a trial with a logit prior of -1 and a logit likelihood of 2 is symmetrical to one with a logit prior*
 350 *of 1 and a logit likelihood of -2).*

351

352 **Table 2. Kendall rank correlations between CINI parameters and psychiatric traits.**

	AQ			PDI		
CINI params	τ	p	BF ₀₁	T	p	BF ₀₁
a_p	0.04	0.5	7.98	-0.04	0.48	7.76
a_s	-0.02	0.65	9.11	0.01	0.85	9.94
w_p	-0.12	0.02	0.64	0.07	0.20	4.14
w_s	-0.02	0.69	9.35	0.13	0.02	0.48

Total AQ scores and Y/N PDI scores were used for the correlations. τ signifies the correlation coefficient. p -values are uncorrected for multiple comparisons. BF₀₁ stands for the Bayes factor 01, with higher values corresponding to stronger evidence for the null hypothesis.

Table 3. Mann-Whitney U test results between the CINI parameters of the ASD and ND groups and the low-AQ and high-AQ groups.

	ND vs ASD			low-AQ vs high-AQ		
CINI params	f	p	BF ₀₁	F	p	BF ₀₁
a_p	0.55	0.50	3.82	0.54	0.63	3.60
a_s	0.45	0.46	3.15	0.47	0.70	3.88
w_p	0.47	0.64	3.85	0.40	0.19	1.96
w_s	0.53	0.71	3.71	0.42	0.31	2.17

Total AQ scores were used for the comparisons. f signifies the common language effect size, with larger f values corresponding to larger parameter values for the ASD and the high-AQ groups. An f of 0.5 corresponds to no differences. p -values are uncorrected for multiple comparisons. BF₀₁ stands for the Bayes factor 01, with higher values corresponding to stronger evidence for the null hypothesis.

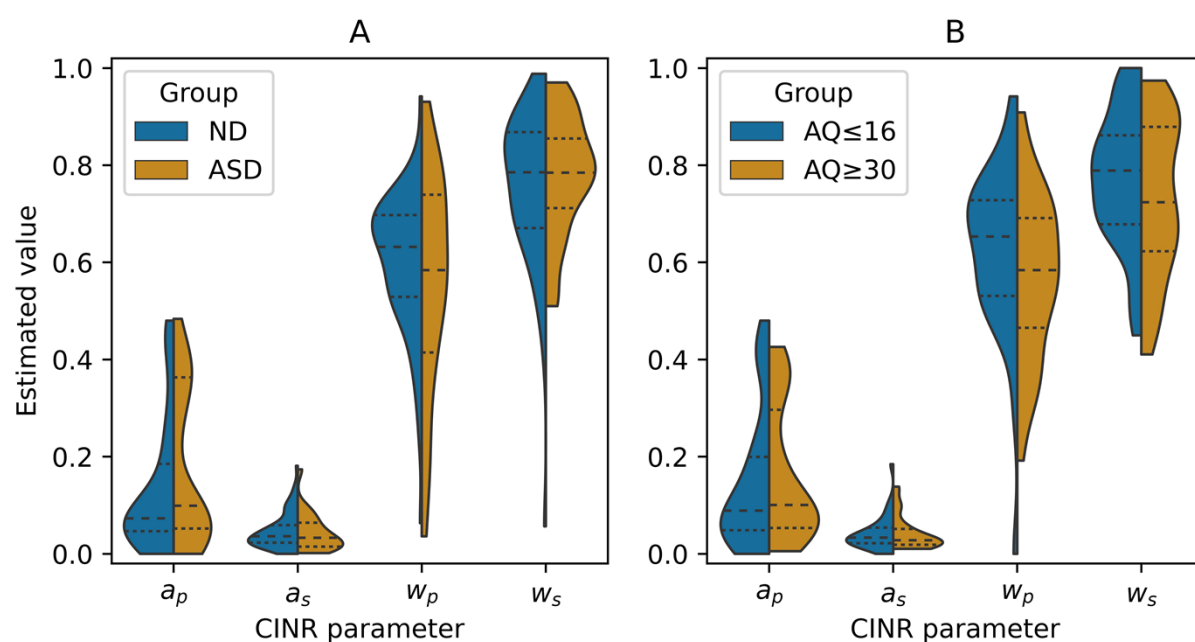


Fig 6. CINI parameter values of ND vs ASD groups (A) and low-AQ vs high-AQ groups (B).

Violin plots show the density of estimated parameters over the possible values, relative to the subgroup size. Dashed lines in the middle represent the median, while dotted ones represent the top and bottom quartiles in each group. No differences are observed between groups.

Discussion

In the present study, we investigated the relationship between circular inference and autistic traits or autism. Circular inference is an impairment in Bayesian hierarchical networks where top-down or bottom-up signals get reverberated throughout the network, becoming significantly amplified [23]. We hypothesized that stronger autistic traits and ASD diagnoses would be associated with stronger reverberation of priors or sensory evidence. We used the fisher task, a probabilistic decision-making task that had been used previously with patients with schizophrenia [27]. To our knowledge, this is the first study to explore signal reverberation in ASD. Our analysis showed that the circular inference models perform best across the whole sample, similarly to previous results [27,29]. However, our hypothesis was refuted. Specifically, no correlation was found between autistic traits and either reverberation parameter. Similarly, there were no differences in these parameters between the groups with the strongest and weakest autistic traits, and no differences between the autistic subjects and those with no diagnosis.

Circular inference attempts to model the effects of increased excitation-to-inhibition ratio, a phenomenon which has been strongly associated with schizophrenia [24,25]. Indeed, Jardri et al. found experimental evidence for stronger likelihood reverberation in patients with schizophrenia, using the fisher task [27]. On that account, our results are surprising, given the observed inhibitory impairments in ASD [33,34] and the commonalities between autism and schizophrenia regarding E/I imbalances [53,54]. Our findings reveal a divergence in the Bayesian mechanisms of the two conditions. This has also been observed by Karvelis et al., which showed an association between autistic traits and increased sensory precision, but no discernible imbalance in schizotypy, in a statistical learning task [55]. No other studies are known to us that compare ASD and SCZ using the same tasks and Bayesian models, despite the commonalities between their computational explanations (for reviews see [14,15]).

In agreement with the findings of Jardri et al. [27], we found compelling evidence for signal reverberation across our sample. Interestingly though, one variant of the model, Circular Inference – No Interference (CINI), was a better fit for our data compared to the other variant, Circular Inference – Interference (CII), contrary to the Jardri et al. study. Additional analysis of the Jardri et al. dataset revealed that this is partially because we used fewer trials than in the original study (Section D2 in S1 Supplementary Information). Furthermore, even in the original dataset, CII was dominant mostly in the SCZ subsample, while it performed equally well with CINI in controls. The dominance of CINI across our sample (Section D2 in S1 Supplementary Information) could be an indication that prior beliefs and sensory evidence are usually reverberated and amplified separately in the brain. In schizophrenia, however, inhibition impairments in certain brain areas could lead these signals to get reverberated in a way that they interfere with each other, making CII a better fit for those individuals.

Surprisingly, we found no evidence for an association between prior or likelihood weights and ASD diagnoses or AQ scores. This result is seemingly in contrast with previous studies showing an overweighting of likelihoods relative to priors in autistic individuals or those with stronger autistic traits (e.g., [55–58]). However, these effects have been demonstrated exclusively in perceptual tasks, with the rare study of Bayesian decision-making in ASD showing no such imbalance [59]. Our analysis revealed a slight increase of absolute confidence with stronger non-clinical delusional beliefs (PDI), but no association between PDI and any model parameter. This confirms the Jardri et al. findings of no such relationship in healthy subjects, although only 8 participants had scores above the clinical PDI mean ($M=11.9$, [42]). This result would deserve further investigation with a more thorough assessment of schizotypy, so as to assess how it can fit with the dimensional view of schizophrenia [60].

Limitations and future work

Through our recruitment methods, we had aimed to recruit participants with a broad range of autistic traits. However, the resulting variance of AQ in our sample ($SD=6.5$) was only marginally higher than what is found in the general population ($SD=5.6$, [43]). Moreover, only 4 participants had an AQ score of more than 1 SD below the neurotypical mean ($M=16.9$, [43]) and only 5 participants had an AQ above the clinical mean ($M=35.2$, [43]). A wider range of autistic traits would be useful in investigating Bayesian impairments that might be associated with the extremes of the AQ distribution. Moreover, the diagnoses of our participants in the Prolific subsample were self-reported. Our findings will need to be confirmed in a sample verified by a mental health professional, possibly differentiating between Autistic disorder, Asperger's syndrome, and other related conditions [61]. Another limitation that also nuances the comparison with previous investigations in schizophrenia [27] concerns the fact that our experiment took place online. The lack of a lab-controlled environment could have substantially affected the quality of the collected data. Adding to that is the absence of in-person communication between participants and researchers, so the instructions of the task could have been clearly conveyed and possible questions answered. Such effects were visible in our dataset by the large portion of subjects that were excluded ($\approx 14\%$).

As with other findings relating behavior to Bayesian inference impairments, it will be important to assess how our findings can be generalized to other tasks or modalities. Circular inference is formalized within a hierarchical Bayesian framework of cognitive processing. This framework assumes that priors express the (top-down) influences of the more abstract representations of the environment to the less abstract ones, while likelihoods encode the reverse (bottom-up) influences [62]. It is difficult to verify that the information presented in the current task (baskets and fish proportions) is encoded by subjects in the expected way – that is, that the preferences of the fisherman correspond to more abstract or contextual information and the fish proportions to more sensory. If these stimuli were processed by the participants as being in the same conceptual level, the task structure would be more akin to a delayed cue integration task [63]. Additionally, it is possible that

the basket size is treated by participants as a qualitative variable, leading them to disregard the exact difference in size, something that would appear as prior overcounting in the models. We believe that these concerns do not invalidate our results, but further research would be needed to understand how delayed cue integration tasks or qualitative information fit within the circular inference framework.

Future research should replicate both ASD and SCZ findings in other tasks, involving different cognitive modalities. The social beads task of Simonsen et al. [29], for example, might be well suited for the investigation of signal reverberation in ASD, given the condition's impairments. Perceptual tasks, on the other hand, would avoid conscious strategies that are especially prevalent in decision-making, focusing instead on more fundamental computations in the brain and connecting circular inference with the rest of the Bayesian literature. Equally important is clarifying the connection between reverberation and neurophysiological measures, with a focus on the spatial patterns of E/I imbalances across brain areas. Differences in such patterns could explain why computational [14,15] and neurobiological [4,9] theories of ASD and SCZ partially overlap, while their phenotypic expressions differ [53].

Acknowledgements

References

1. Joyce EM, Roiser JP. Cognitive heterogeneity in schizophrenia: Curr Opin Psychiatry. 2007 May;20(3):268–72.
2. Masi A, DeMayo MM, Glozier N, Guastella AJ. An Overview of Autism Spectrum Disorder, Heterogeneity and Treatment Options. Neurosci Bull. 2017 Apr;33(2):183–93.
3. Kuhn R, Cahn CH. Eugen Bleuler’s Concepts of Psychopathology. Hist Psychiatry. 2004 Sep;15(3):361–6.
4. Couture SM, Penn DL, Losh M, Adolphs R, Hurley R, Piven J. Comparison of social cognitive functioning in schizophrenia and high functioning autism: more convergence than divergence. Psychol Med. 2010 Apr;40(4):569–79.
5. Pinkham AE, Hopfinger JB, Pelphrey KA, Piven J, Penn DL. Neural bases for impaired social cognition in schizophrenia and autism spectrum disorders. Schizophr Res. 2008 Feb;99(1–3):164–75.
6. Patterson PH. Immune involvement in schizophrenia and autism: Etiology, pathology and animal models. Behav Brain Res. 2009;9.
7. Cade R, Privette M, Fregly M, Rowland N, Sun Z, Zele V, et al. Autism and Schizophrenia: Intestinal Disorders. Nutr Neurosci. 2000 Jan;3(1):57–72.
8. Carroll LS, Owen MJ. Genetic overlap between autism, schizophrenia and bipolar disorder. Genome Med. 2009;1(10):102.
9. King BH, Lord C. Is schizophrenia on the autism spectrum? Brain Res. 2011 Mar;1380:34–41.
10. Rapoport J, Chavez A, Greenstein D, Addington A, Gogtay N. Autism Spectrum Disorders and Childhood-Onset Schizophrenia: Clinical and Biological Contributions to a Relation Revisited. J Am Acad Child Adolesc Psychiatry. 2009 Jan;48(1):10–8.
11. Knill DC, Pouget A. The Bayesian brain: the role of uncertainty in neural coding and computation. Trends Neurosci. 2004 Dec;27(12):712–9.
12. Pellicano E, Burr D. When the world becomes ‘too real’: a Bayesian explanation of autistic perception. Trends Cogn Sci. 2012 Oct;16(10):504–10.
13. Lawson RP, Rees G, Friston KJ. An aberrant precision account of autism. Front Hum Neurosci [Internet]. 2014 May 14 [cited 2020 Nov 27];8. Available from: <http://journal.frontiersin.org/article/10.3389/fnhum.2014.00302/abstract>

- 496 14. Valton V, Romaniuk L, Douglas Steele J, Lawrie S, Seriès P. Comprehensive review:
497 Computational modelling of schizophrenia. *Neurosci Biobehav Rev*. 2017
498 Dec;83:631–46.
- 499 15. Palmer CJ, Lawson RP, Hohwy J. Bayesian approaches to autism: Towards volatility,
500 action, and behavior. *Psychol Bull*. 2017;143(5):521–42.
- 501 16. Sterzer P, Adams RA, Fletcher P, Frith C, Lawrie SM, Muckli L, et al. The Predictive
502 Coding Account of Psychosis. *Biol Psychiatry*. 2018 Nov;84(9):634–43.
- 503 17. Chambon V, Pacherie E, Barbalat G, Jacquet P, Franck N, Farrer C. Mentalizing under
504 influence: abnormal dependence on prior expectations in patients with
505 schizophrenia. *Brain*. 2011 Dec;134(12):3728–41.
- 506 18. Powers AR, Mathys C, Corlett PR. Pavlovian conditioning–induced hallucinations
507 result from overweighting of perceptual priors. *Science*. 2017 Aug
508 11;357(6351):596–600.
- 509 19. Cassidy CM, Balsam PD, Weinstein JJ, Rosengard RJ, Slifstein M, Daw ND, et al. A
510 Perceptual Inference Mechanism for Hallucinations Linked to Striatal Dopamine.
511 *Curr Biol*. 2018 Feb;28(4):503–514.e4.
- 512 20. Speechley W. The contribution of hypersalience to the “jumping to conclusions” bias
513 associated with delusions in schizophrenia. *J Psychiatry Neurosci*. 2010 Jan
514 1;35(1):7–17.
- 515 21. King DJ, Hodgekins J, Chouinard PA, Chouinard V-A, Sperandio I. A review of
516 abnormalities in the perception of visual illusions in schizophrenia. *Psychon Bull*
517 *Rev*. 2017 Jun;24(3):734–51.
- 518 22. Fletcher PC, Frith CD. Perceiving is believing: a Bayesian approach to explaining the
519 positive symptoms of schizophrenia. *Nat Rev Neurosci*. 2009 Jan;10(1):48–58.
- 520 23. Jardri R, Denève S. Circular inferences in schizophrenia. *Brain*. 2013
521 Nov;136(11):3227–41.
- 522 24. O'Donnell P. Adolescent Onset of Cortical Disinhibition in Schizophrenia: Insights
523 From Animal Models. *Schizophr Bull*. 2011 May 1;37(3):484–92.
- 524 25. Grent-’t-Jong T, Gross J, Goense J, Wibrals M, Gajwani R, Gumley AI, et al. Resting-state
525 gamma-band power alterations in schizophrenia reveal E/I-balance abnormalities
526 across illness-stages. *eLife*. 2018 Sep 27;7:e37799.
- 527 26. Evans S, Averbeck B, Furl N. Jumping to conclusions in schizophrenia.
528 *Neuropsychiatr Dis Treat*. 2015 Jul;1615.
- 529 27. Jardri R, Duverne S, Litvinova AS, Denève S. Experimental evidence for circular
530 inference in schizophrenia. *Nat Commun*. 2017 Apr;8(1):14218.

- 531 28. Ross RM, McKay R, Coltheart M, Langdon R. Jumping to Conclusions About the Beads
532 Task? A Meta-analysis of Delusional Ideation and Data-Gathering. *Schizophr Bull.*
533 2015 Sep;41(5):1183–91.
- 534 29. Simonsen A, Fusaroli R, Petersen ML, Vermillet A-Q, Bliksted V, Mors O, et al. Taking
535 others into account: combining directly experienced and indirect information in
536 schizophrenia. *Press.* 2021;
- 537 30. Casanova MF, Buxhoeveden DP, Switala AE, Roy E. Minicolumnar pathology in
538 autism. *Neurology.* 2002 Feb 12;58(3):428–32.
- 539 31. Rubenstein JLR, Merzenich MM. Model of autism: increased ratio of
540 excitation/inhibition in key neural systems. *Genes Brain Behav.* 2003 Oct;2(5):255–
541 67.
- 542 32. Brown C, Gruber T, Boucher J, Rippon G, Brock J. Gamma Abnormalities During
543 Perception of Illusory Figures in Autism. *Cortex.* 2005 Jan;41(3):364–76.
- 544 33. Kana RK, Keller TA, Minshew NJ, Just MA. Inhibitory Control in High-Functioning
545 Autism: Decreased Activation and Underconnectivity in Inhibition Networks. *Biol*
546 *Psychiatry.* 2007 Aug;62(3):198–206.
- 547 34. Gogolla N, LeBlanc JJ, Quast KB, Südhof TC, Fagiolini M, Hensch TK. Common circuit
548 defect of excitatory-inhibitory balance in mouse models of autism. *J Neurodev*
549 *Disord.* 2009 Jun;1(2):172–81.
- 550 35. Kenny L, Hattersley C, Molins B, Buckley C, Povey C, Pellicano E. Which terms should
551 be used to describe autism? Perspectives from the UK autism community. *Autism.*
552 2016 May;20(4):442–62.
- 553 36. Robinson EB, Munir K, Munafò MR, Hughes M, McCormick MC, Koenen KC. Stability
554 of Autistic Traits in the General Population: Further Evidence for a Continuum of
555 Impairment. *J Am Acad Child Adolesc Psychiatry.* 2011 Apr;50(4):376–84.
- 556 37. Wiggins LD, Robins DL, Adamson LB, Bakeman R, Henrich CC. Support for a
557 Dimensional View of Autism Spectrum Disorders in Toddlers. *J Autism Dev Disord.*
558 2012 Feb;42(2):191–200.
- 559 38. Kim H, Keifer C, Rodriguez-Seijas C, Eaton N, Lerner M, Gadow K. Quantifying the
560 Optimal Structure of the Autism Phenotype: A Comprehensive Comparison of
561 Dimensional, Categorical, and Hybrid Models. *J Am Acad Child Adolesc Psychiatry.*
562 2019 Sep;58(9):876–886.e2.
- 563 39. American Psychiatric Association, editor. *Diagnostic and Statistical Manual of Mental*
564 *Disorders* [Internet]. Fifth Edition. American Psychiatric Association; 2013 [cited
565 2021 Mar 2]. Available from:
566 <https://psychiatryonline.org/doi/book/10.1176/appi.books.9780890425596>
- 567 40. Palan S, Schitter C. Prolific.ac—A subject pool for online experiments. *J Behav Exp*
568 *Finance.* 2018 Mar;17:22–7.

- 569 41. Baron-Cohen S, Wheelwright S, Skinner R, Martin J, Clubley E. The Autism-Spectrum
570 Quotient (AQ): Evidence from Asperger Syndrome/High-Functioning Autism,
571 Males and Females, Scientists and Mathematicians. *J Autism Dev Disord*.
572 2001;31(1):5–17.
- 573 42. Peters E, Joseph S, Day S, Qarety P. Measuring Delusional Ideation: The 21-Item
574 Peters et al Delusions Inventory (PDI). *Schizophr Bull*. 2004;30(4):18.
- 575 43. Ruzich E, Allison C, Smith P, Watson P, Auyeung B, Ring H, et al. Measuring autistic
576 traits in the general population: a systematic review of the Autism-Spectrum
577 Quotient (AQ) in a nonclinical population sample of 6,900 typical adult males and
578 females. *Mol Autism*. 2015;6(1):2.
- 579 44. Stephan KE, Penny WD, Daunizeau J, Moran RJ, Friston KJ. Bayesian model selection
580 for group studies. 2009;14.
- 581 45. SPM12 Toolbox - Statistical Parametric Mapping [Internet]. Wellcome Centre for
582 Human Neuroimaging; 2020 [cited 2020 Dec 4]. Available from:
583 <https://www.fil.ion.ucl.ac.uk/spm/software/spm12/>
- 584 46. Li J, Wang ZJ, Palmer SJ, McKeown MJ. Dynamic Bayesian network modeling of fMRI:
585 A comparison of group-analysis methods. *NeuroImage*. 2008 Jun;41(2):398–407.
- 586 47. McGraw KO, Wong SP. A common language effect size statistic. *Psychol Bull*.
587 1992;111(2):361–5.
- 588 48. Raftery AE. Bayesian Model Selection in Social Research. *Sociol Methodol*.
589 1995;25:111.
- 590 49. JASP [Internet]. JASP Team; 2020 [cited 2020 Dec 14]. Available from: [https://jasp-](https://jasp-stats.org/)
591 [stats.org/](https://jasp-stats.org/)
- 592 50. Kim Y, Kim T-H, Ergün T. The instability of the Pearson correlation coefficient in the
593 presence of coincidental outliers. *Finance Res Lett*. 2015 May;13:243–57.
- 594 51. Bonnet C, Ars JF. Reaction times as a measure of uncertainty. :7.
- 595 52. Benjamini Y, Hochberg Y. Controlling the False Discovery Rate: A Practical and
596 Powerful Approach to Multiple Testing. *J R Stat Soc Ser B Methodol*. 1995
597 Jan;57(1):289–300.
- 598 53. Foss-Feig JH, Adkinson BD, Ji JL, Yang G, Srihari VH, McPartland JC, et al. Searching
599 for Cross-Diagnostic Convergence: Neural Mechanisms Governing Excitation and
600 Inhibition Balance in Schizophrenia and Autism Spectrum Disorders. *Biol Psychiatry*.
601 2017 May;81(10):848–61.
- 602 54. Davenport EC, Szulc BR, Drew J, Taylor J, Morgan T, Higgs NF, et al. Autism and
603 Schizophrenia-Associated CYFIP1 Regulates the Balance of Synaptic Excitation and
604 Inhibition. *Cell Rep*. 2019 Feb;26(8):2037–2051.e6.

55. Karvelis P, Seitz AR, Lawrie SM, Seriès P. Autistic traits, but not schizotypy, predict increased weighting of sensory information in Bayesian visual integration. *eLife*. 2018 May 14;7:e34115.
56. Karaminis T, Cicchini GM, Neil L, Cappagli G, Aagten-Murphy D, Burr D, et al. Central tendency effects in time interval reproduction in autism. *Sci Rep*. 2016 Sep;6(1):28570.
57. Powell G, Meredith Z, McMillin R, Freeman TCA. Bayesian Models of Individual Differences: Combining Autistic Traits and Sensory Thresholds to Predict Motion Perception. *Psychol Sci*. 2016 Dec;27(12):1562–72.
58. Król M, Król M. The world as we know it and the world as it is: Eye-movement patterns reveal decreased use of prior knowledge in individuals with autism. *Autism Res*. 2019 Sep;12(9):1386–98.
59. Manning C, Kilner J, Neil L, Karaminis T, Pellicano E. Children on the autism spectrum update their behaviour in response to a volatile environment. *Dev Sci*. 2017 Sep;20(5):e12435.
60. Nelson MT, Seal ML, Pantelis C, Phillips LJ. Evidence of a dimensional relationship between schizotypy and schizophrenia: A systematic review. *Neurosci Biobehav Rev*. 2013 Mar;37(3):317–27.
61. Young RL, Rodi ML. Redefining Autism Spectrum Disorder Using DSM-5: The Implications of the Proposed DSM-5 Criteria for Autism Spectrum Disorders. *J Autism Dev Disord*. 2014 Apr;44(4):758–65.
62. Denève S, Jardri R. Circular inference: mistaken belief, misplaced trust. *Curr Opin Behav Sci*. 2016 Oct;11:40–8.
63. Ernst MO, Banks MS. Humans integrate visual and haptic information in a statistically optimal fashion. *Nature*. 2002 Jan;415(6870):429–33.

632 **S1 Supplementary Information. Additional analyses and visualizations.**