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Confidence in visual motion discrimination is preserved in individuals with schizophrenia

Running title: Preserved confidence in schizophrenia

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Anonymized data, analysis and modeling scripts will be made freely available upon publication.

Abstract

Background: Metacognition is the set of reflexive processes allowing humans to evaluate the accuracy of their mental operations. Metacognitive deficits have been described in individuals with schizophrenia using mostly narrative assessment and linked to several key symptoms.

Methods: Here, we assessed metacognitive performance objectively by asking individuals with schizophrenia or schizoaffective disorder (N=20) and matched healthy participants (N = 21) to perform a visual discrimination task and subsequently report confidence in their performance. Metacognitive performance was defined as the adequacy between visual discrimination performance and confidence.

Results: Bayesian analyses revealed equivalent metacognitive performance in the two groups despite a weaker association between confidence and trajectory-tracking during task execution among patients. These results were reproduced using an evidence accumulation model which showed similar decisional processes in the two groups.

Limitations: These results from a relatively small study sample should be generalized to other perceptual and non-perceptual tasks which are more ecological. The link between metacognitive performance in such tasks and clinical or cognitive insight remains to be explored.

Conclusions: We found similar decisional and metacognitive capabilities between individuals with schizophrenia and healthy controls in a visual discrimination task.

Introduction

Metacognition refers to a spectrum of mental activities whose objects are one's own thoughts. Some of these mental activities can be described as discrete (recognition and monitoring of ongoing thoughts or percepts), others as more transversal and synthetic, integrating a subject's assumption of thoughts, sensations, intentions or links between events to form more complete and lasting representations ^{1,2}. Regarding the latter, individuals with schizophrenia have persistent difficulties in considering thoughts as essentially subjective, in recognizing complex mental states in others, in viewing events from perspectives other than their own, and in using their metacognitive knowledge to manage their distress ^{3,4}. These deficits have been linked to core features of the illness like positive and negative symptoms ⁵, disorganisation ⁶, functioning ⁷, and quality of life ⁸. Synthetic metacognition is usually measured through structured interviews and self-reported questionnaires that evaluate multiple processes such as emotion recognition, theory of mind and verbal abilities. In contrast, discrete metacognition is measured by focusing on a specific cognitive domain: participants are asked to perform a cognitive or perceptual task (sometimes referred to as the first-order task), and subsequently assess how well they performed (i.e., a second-order task consisting of a confidence judgment, error detection, or post-decisional wagering). In this context, metacognitive performance is defined as the capacity to adapt second-order judgments to first-order performance ⁹.

Studies relying on such combinations of first and second-order tasks reported deficits in metacognitive performance in individuals with schizophrenia across several domains, such as perception ¹⁰, agency ¹¹ and memory ¹². Although these studies have provided valuable insights regarding putative deficits in discrete metacognition, several biases might interfere with the assessment of metacognitive performance in individuals with schizophrenia. First, it is important to consider that metacognitive performance depends on first-order performance: it is easier to provide confidence judgments or detect errors for easy than for difficult tasks. Thus, it is crucial to control for first-order task performance, which is usually lower in individuals with schizophrenia compared to controls. Other biases might influence metacognition in individuals with schizophrenia such as depression which has been associated with better metacognition ¹³ and cognitive deficits that have been associated with metacognitive impairments with a small-to-moderate effect size ¹⁴. Considering the many stages of processing leading from first to second-order decisions, poor metacognitive performance in a given task may be due to deficits at any of these levels.

Here, we first sought to test the existence of a metacognitive deficit in individuals with schizophrenia while accounting for possible differences regarding first-order performance. Second, we aimed at pinpointing the putative origins of metacognitive deficits in individuals with schizophrenia and describe how first and second-order cognitive processes unfold over time by analyzing behavioral responses together with trajectory-tracking, and by reproducing them using an evidence accumulation model of decision-making. Namely, we continuously tracked the position and kinematics of the mouse that participants used to indicate their first-order response during a motion discrimination task ^{15,16}. In addition, we modeled first and second-order responses as derived from an evidence accumulation process starting when participants initiated a mouse movement ¹⁷⁻²⁰. Together, these two approaches following a pre-registered plan allowed us to finely characterize decision-making and metacognitive monitoring in individuals with schizophrenia in relation to clinical traits while avoiding the typical confounds that may have contaminated previous results in the field.

Methods

This is a transversal monocentric study. The experimental paradigm and analysis plan detailed below were registered prior to data collection ([NCT03140475](https://www.clinicaltrials.gov/ct2/show/study/NCT03140475)) and are available together with anonymized data and analyses scripts (<https://osf.io/84wqp/>).

Participants

Twenty-three healthy volunteers (15 males, 8 females) from the general population and twenty individuals with a schizophrenia spectrum disorder (16 males, 4 females) took part in this study. Two healthy volunteers were excluded from the analysis, respectively due to a convergence failure during the staircase procedure, and an estimated IQ < 70. Individuals with schizophrenia spectrum disorders (schizophrenia or schizoaffective disorder) were recruited from community mental health centers and outpatient clinics in the Versailles area. The control participants were recruited from the volunteers' panel at the *Centre d'Economie de la Sorbonne* and Versailles Hospital (see SI for details). Exclusion criteria for both groups of participants were a moderate-to-severe substance use disorder (DSM-5 criteria) within the 12 months preceding the study, and a current or prior untreated medical illness, including neurologic illness, an IQ < 70 based on three subtests of the Wechsler Adult Intelligence Scale (see SI), and an age > 60 years. Schizophrenia and schizoaffective disorders were diagnosed by a psychiatrist investigator based on the Structured Clinical Interview for assessing the DSM-5 criteria 1. Another licensed psychiatrist (patient's treating psychiatrist) confirmed the diagnosis for each patient according to the DSM-5 criteria. All participants were right-handed, had normal hearing and normal or corrected-to-normal vision. They were naive to the purpose of the study and gave informed consent. The investigators checked whether patients were capable of giving a fully informed consent through a specific interview (focused on the ability to comprehend and retain information about the research and to use and weigh this information to make an appropriate decision). This interview was done at the first appointment scheduled after the proposal to participate in the research by the patient's referring psychiatrist. The information was oral and adapted to the patient's verbal comprehension skills, but also in written form. The investigators answered any questions the patient may have had before he or she signed the consent form". Written informed consent was then obtained from each participant.

The study was approved by the ethical committee *Sud Méditerranée II* (217 R01). Our plan at pre-registration was to collect data until we reach a Bayes Factor of either 1/3 or 3 regarding the difference in metacognitive performance between groups. We halted data collection when

evidence for the null hypothesis for a group difference of M-Ratio was obtained, see analyses below).

Neuropsychological and clinical evaluation

Both individuals with schizophrenia spectrum disorders and healthy controls were evaluated on several clinical and neuropsychological continuous measures, described in SI.

Experimental Design

On each trial, participants were asked to indicate the mean motion direction of a random-dot kinetogram (RDK) by clicking within a circular frame located on the top, to the right or to the left of the stimulus (first-order task), and report how confident they were in their response (second-order task) by moving the mouse cursor on a visual analog scale with marks between 0 % (certainty that the first-order response was erroneous) and 100 % (certainty that the first-order response was correct) with 5% steps (Figure 1). All details are provided in SI.

Statistical analysis

All analyses were performed with R (2018).

The first objective was to establish the comparability of the two groups in terms of demographic and cognitive characteristics. We thus compared groups' characteristics using the Welch t-test or χ^2 test when appropriate.

The second objective was to test for differences in metacognitive performance between patients and controls, using two complementary measures. Metacognitive sensitivity was quantified using a mixed-effects logistic regression between first-order accuracy (binary categorical variable) and confidence (continuous variable), including a fixed effect of group (binary categorical variable: controls vs. patients), random intercepts by participants and full random effects structure. Metacognitive efficiency was quantified in a Bayesian framework as the ratio between meta-d' (continuous variable) and d' (M-Ratio, continuous variable) ^{21,22}.

The third objective was to compare biases in metacognition between patients and controls. Confidence bias quantified differences in the tendency to use high or low confidence ratings, based on the second-order receiver operating characteristic curve (B-ROC ²³).

The fourth objective was to compare the strength of relationships between confidence and response motion kinematics in patients and controls. Mouse spatial trajectories (X, Y, continuous variables) were preprocessed (see SI), and fitted using a model II linear regression with the major axis method ²⁴. Kinematics (velocity, acceleration, continuous variables) were

standardized across participants (z-score) and analyzed as a function of confidence using mixed-effects linear regressions.

Results

1. Cognitive and clinical variables

The two groups did not differ in terms of gender ($\chi^2 = 0.45$, $p = 0.50$), age, education, premorbid intelligence levels, and neuropsychological performance, except for the total score in the Six Elements Test which was marginally lower for patients (mean 801.0 ± 101.4) than in controls (mean 920.8 ± 86.4 , $t(37.1) = 1.76$, $p = 0.086$, see Table 1). Two other variables differed between patients and controls: depressive symptoms which were higher in patients (mean 4.5 ± 1.8) compared with controls (mean 0.5 ± 0.4 , $t(21.2) = -4.3$, $p < 0.001$), and cognitive insight scores which were higher in patients (mean 5.8 ± 3.2) than in controls (mean -0.6 ± 1.7 , $t(28.7) = -3.4$, $p = 0.002$). Of note, the latter difference was not significant anymore when taking into account depression as a covariate: a linear model of insight as a function of group and depression scores revealed a main effect of depression (beta = 0.75 ± 0.30 , $t(37) = 2.52$, $p = 0.02$, BF = 102.86), but no effect of group ($t(37) = 1.55$, $p = 0.13$, BF = 1.21), suggesting that the difference in insight between groups was explained by depression. Depressions scores were ≥ 6 for 45% of patients indicating a possible major depressive disorder in these participants²⁷. The CDS total score was < 6 for all participants in the control group. In the patients' sample, the intensity of schizophrenia was measured with the Positive And Negative Syndrome Scale (PANSS). The mean PANSS total score was 78.5 ± 6.8 , the mean positive symptoms score was 17.2 ± 2.2 , the mean negative symptoms score was 20.5 ± 2.3 and the mean general psychopathology score was 40.9 ± 3.8 . The mean illness duration was 14.7 years ± 3.7 and the mean chlorpromazine equivalent was $439.7 \text{ mg}/24\text{h} \pm 118.4$. The mean score on PSP was 55.7 ± 5.4 and the mean total score on BIS was 10.8 ± 1.1 . The group of patients included 13 participants with schizophrenia and 7 with schizo-affective disorders.

	Control (N = 21) (mean \pm 95%CI)	Schizophrenia (N = 20) (mean \pm 95%CI)	t-statistic	Degrees of freedom	p-value	Bayes factor

Age (years)	42.6	4.8	38.8	5.1	1.09	37.87	0.285	0.50
Beck Cognitive Insight Scale	-0.6	1.7	5.8	3.2	-3.39	28.74	0.002	20.96
Calgary Depression Scale	0.5	0.4	4.5	1.8	-4.26	21.21	0.001	171.20
Education level (years)	12.5	0.4	13.6	1.3	-1.56	22.70	0.133	0.80
Premorbid IQ	104.0	3.6	102.3	3.9	0.65	37.74	0.521	0.37
Six Elements Test (errors)	9.2	1.4	8.0	2.1	0.93	32.94	0.359	0.44
Six Elements Test (points)	920.8	86.4	801.0	101.3	1.76	37.07	0.086	1.04
WAIS matrix subtest	10.2	1.1	9.0	1.3	1.33	37.13	0.192	0.62
WAIS letter-number sequencing subtest	9.1	1.2	7.7	1.1	1.69	37.98	0.100	0.94
WAIS vocabulary subtest	10.0	1.2	11.1	1.5	-1.06	36.54	0.297	0.48

criterion	0.00	0.21	-0.32	0.15	2.50	37.7	0.02	3.24
sensitivity (d')	1.38	0.11	1.29	0.08	0.85	36.7	0.40	0.41
Motion variance	2.01	0.20	1.59	0.17	3.09	38.9	0.004	10.60

Table 1. Clinical, neuropsychological, and behavioral characteristics of patients and controls.

2. First and second-order performance

Participants indicated the direction of a RDK (first-order task) and subsequently reported the confidence in their decision (second-order task, see methods and SI). The analysis of the first-order task is reported in SI and Table 1. At the second-order level, average confidence ratings were similar between groups (schizophrenia: 0.71 ± 0.05 ; controls: 0.70 ± 0.06 , $t(38.4) = 0.12$, $p = 0.91$, $BF = 0.31$), as well as confidence bias defined as B-ROC (patients: -1.93 ± 0.26 ; controls: -2.06 ± 0.21 , $t(36.5) = 0.54$, $p = 0.59$, $BF = 0.35$). Behavioral results (confidence, B-ROC) remained unchanged when CDS total score was entered as a covariate. We then estimated metacognitive efficiency (i.e., ratio between meta- d' and d') to capture the amount of perceptual evidence used by participants when computing confidence estimates. We made the prior assumption that controls had higher metacognitive efficiency (i.e., prior with Gaussian distribution of mean = 0.2 and SD = 1 for the difference in metacognitive efficiency between groups), based on the difference in metacognitive accuracy between first-episode psychosis and healthy controls recently reported by Davies and colleagues²⁵. Results showed that the two groups had similar metacognitive efficiency (schizophrenia: 0.52, highest posterior density interval = [0.40 0.65], controls: 0.49, highest posterior density interval = [0.37 0.64]), with a Bayes factor of 0.18 supporting the absence of difference between groups (Figure 2B). Another metric of metacognitive performance was computed, namely metacognitive sensitivity which corresponds to the slope of the logistic regression between first-order accuracy and confidence. A similar prior assumption for higher metacognitive efficiency in the control group was made, represented by a steeper slope (i.e., Gaussian distribution with mean = 1, SD = 5). We chose a weakly informative prior in the absence of published evidence. No interaction between group and confidence was found (estimate = 0.06, highest posterior density interval = [-0.15 0.27], Bayes factor = 0.02) (Figure 2C). Importantly, Bayes factors smaller than 0.3 both for metacognitive efficiency and sensitivity support the null hypothesis, according to which individuals with schizophrenia have no impairment while adjusting confidence to their performance.

Following our pre-registered plan, we then sought to assess how motor behavior related to first-order responses modulated confidence ratings. We quantified the relationship between confidence, first-order accuracy and standardized reaction times between groups using a mixed-effects linear regression including perceptual evidence as a regressor of no interest. We found a negative relationship between confidence and standardized reaction times (estimate = -0.05 [-0.07 -0.04], evidence ratio > 4000), which indicates that confidence was high following fast first-order responses. This relationship was modulated by first-order accuracy (interaction

accuracy * reaction times: estimate = -0.01 [-0.02 -0.01], evidence ratio = 221.22) and group (interaction group * reaction times: estimate = 0.02 [0.00 0.04], evidence ratio = 22.26) indicating that the slope between confidence and standardized reaction times was steeper for correct responses and for the control group. Together, these results indicate that reaction times covary with confidence to a lesser extent in individuals with schizophrenia, suggesting they may rely less on this input to form confidence estimates.

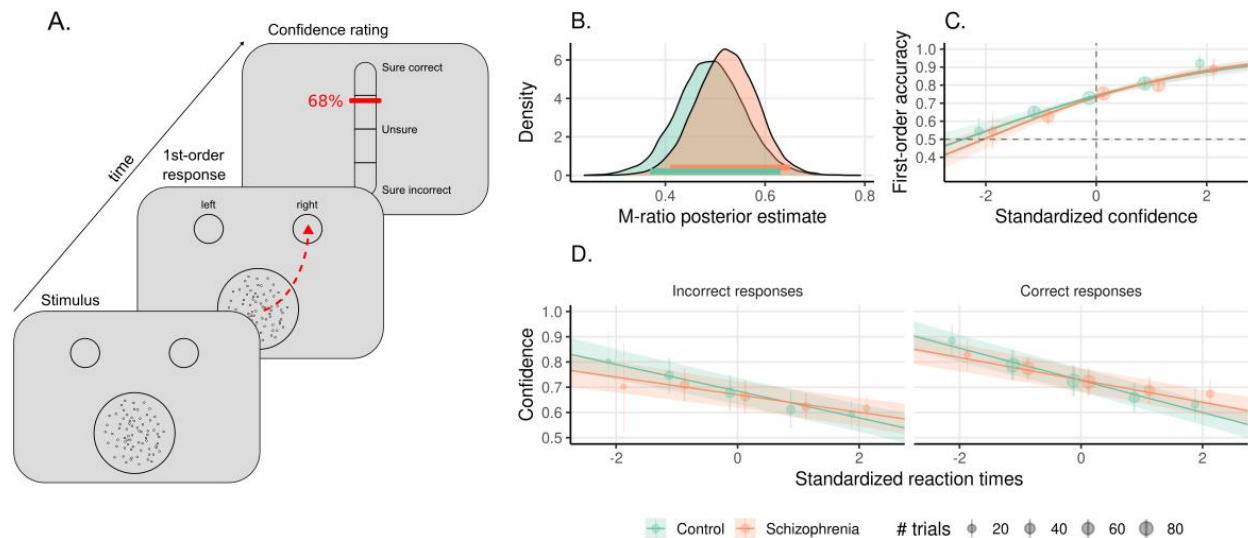


Figure 1: Experimental paradigm and behavioral performance. A. Experimental paradigm. Participants were presented with a random dot kinetogram stimulus moving rightward or leftward and were asked to report motion direction by moving the mouse cursor towards a circle presented at the top-left or top-right of the screen (first-order response). Subsequently, participants reported the confidence they had in their response by moving a cursor on a visual analog scale (second-order response). Exemplar mouse trajectory and confidence ratings are shown in red. B. posterior distribution density of M-Ratio for the control (green) and schizophrenia groups (orange). The colored lines at the bottom of the plot represent the 95% highest posterior density intervals. C. Mixed-effects logistic regression between first-order accuracy and standardized confidence. D. Mixed-effects linear regression between standardized reaction times and confidence. In panels C-D, regression lines and 95 % confidence intervals around them represent the model fit. Although the model took continuous variables as input, we plot for illustrative purposes dots and error bars that represent mean \pm 95% confidence interval over participants after rounding standardized confidence (C) and reaction times (D). The size of each dot is proportional to the number of represented trials.

3. Trajectory-tracking

Beyond reaction times, we quantified how mouse trajectories leading to first-order responses predicted subsequent confidence judgments (see Figure 2A for raw trajectories). First, we isolated trials in which a change of mind occurred, that is when participants started moving towards one response circle and later changed direction towards the other (see SI). Changes of

mind corresponded respectively to 7.9 ± 2.7 % and 7.3 ± 2.9 % of total trials in the patient and control groups ($t(37.9) = 0.32$, $p = 0.75$, $BF = 0.32$). Interestingly, a mixed-effects logistic regression revealed that changes of mind were associated with lower first-order accuracy in both groups (main effect: estimate = -0.32, $z = -2.07$, $p = 0.04$, see Figure 2B), without significant interaction between group and accuracy (estimate = 0.18, $z = 0.84$, $p = 0.40$). Conversely, a mixed-effects linear regression revealed that changes of mind were associated with lower confidence ($F(1,30.3) = 32.05$, $p < 0.001$), and that this decrease was more pronounced in controls vs. patients (interaction term: $F(1,30.33) = 5.03$, $p = 0.03$) indicating that patients revised less their confidence following changes of mind. Next, we assessed how the slopes of individual trajectories covaried with confidence. We found a negative relationship between slopes and confidence ($F(1,50.5) = 5.7$, $p = 0.02$), independent of groups and first-order accuracy (Figure 2C). This suggests that both patients and controls moved the mouse more laterally for responses associated with high confidence, whether correct or not. In addition, we fitted a linear model to individual trajectories and found a positive relationship between the goodness of fit (R^2) and confidence ($F(1,40.1) = 17.5$, $p < 0.001$) independent of group and first-order accuracy, revealing that confidence ratings were higher after responses following more linear trajectories. Of note, a trend suggested lower R^2 in the patient group ($F(1,37.1) = 3.73$, $p = 0.06$). Besides spatial trajectories, we quantified how velocity and acceleration profiles related to confidence, by fitting mixed-effects linear regressions for each time sample across individual trials, with confidence and group as fixed effects. Of note, we centered data to zero to account for potential motor impairment in individuals with schizophrenia²⁶. For velocity, we found a main effect of confidence indicating that velocity reached higher peaks in high confidence trials, and an interaction between confidence and groups indicating that the positive correlation between velocity and confidence was significant in the two groups, but stronger in the control than in the schizophrenia group ($p < 0.05$ *fdr*-corrected). This interaction was explored by fitting velocity models for each group, which showed a sustained correlation between confidence and velocity at movement onset and offset among the control group, and a short-lived correlation at movement onset in the patient group (Figure 2D). For acceleration, we found a main effect of confidence, by which acceleration at movement onset reached higher values in high confidence trials, and an interaction between confidence and group close to movement offset, by which movement acceleration reached more negative values for healthy controls in high confidence trials ($p < 0.05$ *fdr*-corrected). As for velocity, this interaction was explored by fitting acceleration models for each group, which showed that the correlation between confidence and acceleration was significant both at movement onset and offset among the control group (Figure 2D). Among

patients, only a weaker correlation following movement offset was found. Together, these results confirm the existence of kinematics correlates of confidence at the motor execution stage, and suggest that they may be stronger predictors of confidence in healthy individuals compared to schizophrenia patients.

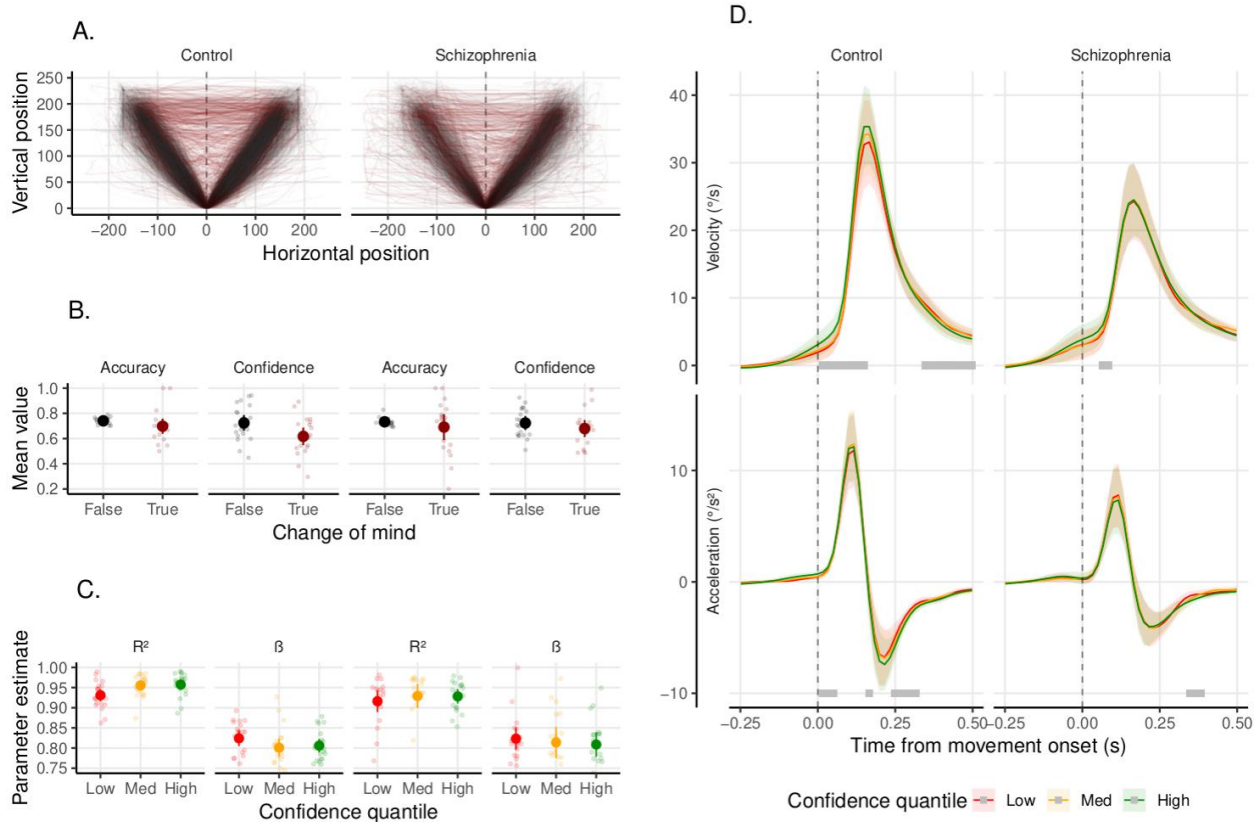


Figure 2: trajectory-tracking. A. Single-trial mouse trajectories leading to the first-order response in case of a change of mind (red) or no change of mind (black) in the control (left panel) and patient groups (right panel). B. Average first-order accuracy and confidence in the presence (red) and absence (black) of a change of mind in the control (left panels) and patient groups (right panels). C. Goodness of fit (R^2) and slope (β) of the linear fit between vertical and horizontal mouse positions as a function of confidence quantile (low: red, medium: orange, high: green). Large dots represent average estimates, error bars represent the 95 % confidence intervals. Small dots represent individual estimates. D. Average velocity (upper panel) and acceleration (lower panel) from first mouse movement onset as a function of confidence quantile (low: red, medium: orange, high: green). Of note, velocity may be non-null prior to movement onset as it was defined as a function of the maximal velocity in a given trial (SI). Shaded areas represent the 95 % confidence intervals. Gray bars represent samples for which confidence covaried significantly with kinematics ($p < 0.05$, *fdr*-corrected).

Finally, we examined the relationship between second-order behavioral measures (confidence, M-Ratio) and these cognitive and clinical variables using Bayesian robust regressions (see SI). Regarding cognitive variables, we found that M-Ratio covaried positively with the WAIS matrix subtest ($r = 0.46$, HDP = [0.20 0.70], Bayes Factor = 13.88) on the whole group of participants, indicating that participants with good perceptual reasoning also had high metacognitive performance. No other correlation was found significant (see Table S1). No significant correlation was found between second-order behavioral measures and clinical variables specific to the patient population (see Tables S2).

Discussion

The current study assessed the quality of metacognitive monitoring in perceptual decision-making in individuals with schizophrenia using bias-free measures of metacognitive performance combined with trajectory-tracking and evidence accumulation models.

Metacognitive performance

No significant difference in metacognitive performance was found between groups, with Bayesian analyses favoring the null hypothesis rather than inconclusive results. In addition, an evidence accumulation model suggested that both groups relied on equivalent first and second-order decisional mechanisms when compensating for first-order perceptual deficits in patients. These results are compatible with a recent account putting forward that individuals with schizophrenia may not be impacted to form confidence estimates per se but rather to interpret their saliency²⁷. In this perspective, the lack of differences found in metacognition between patients and controls in our study might be explained by the use of a continuous scale rather than a binary choice. We note that these results should be taken with caution, considering the relatively small sample size on which they are based. The absence of a difference between groups is unlikely due to abnormally poor metacognitive performance in our control population, as healthy participants performed similarly to participants from previous studies involving a coherent motion discrimination paradigm (e.g., mean M-Ratio = 0.66 in a recent study²⁸). Plus, the unexpected better cognitive insight found in patients compared to controls can not explain the lack of difference in metacognitive performance between groups since cognitive insight was related neither with metacognitive efficiency nor metacognitive sensitivity¹. In addition, a post-hoc comparison of M-Ratio with cognitive insight as covariate confirmed the absence of difference between groups ($t(37) = 0.61$, $p = 0.54$). It is worth noting that a critical difference between our two groups was that individuals with schizophrenia were medicated with antipsychotics acting as dopaminergic antagonists. Although there is evidence that confidence may be modulated by dopamine^{29,30}, we found evidence for the null hypothesis using bayesian correlations between confidence and chlorpromazine equivalent in our sample.

¹ One could argue that including individuals with schizo-affective disorders might have compensated for a potential deficit in metacognition in individuals with schizophrenia, by increasing depressive symptomatology, which has been associated with better metacognitive efficiency⁴⁸. However, the prevalence of possible depression (based on the CDS cut-off) in this sample of individuals with schizophrenia spectrum disorders was close to the 40% value reported in another study investigating outpatients with schizophrenia only⁴⁹.

Recent studies reported lower visual metacognitive performance in individuals with a first episode of psychosis relative to age-matched controls ^{25,31}. Here, we tested individuals with a chronic disorder and found no deterioration of metacognition relative to controls, but a negative relationship between metacognitive efficiency and illness duration (Table S2). This suggests that metacognitive performance in patients may evolve non-linearly over time, with prevalent deficits at the early and late stages of schizophrenia. Longitudinal studies with large sample sizes and varying ages of onset will be needed to assess this possibility.

Regarding chronic schizophrenia, a study reported lower metacognitive performance in a source memory task ³² and perceptual categorization task ³³ compared to psychiatric control groups with similar type-1 accuracy. However, our results are in line with previous studies investigating metacognitive performance controlling for first-order accuracy in chronic schizophrenia, which reported equivalent metacognitive sensitivity (area under the type 2 ROC curve) between patients and controls for facial emotion recognition ³⁴, comparable metacognitive sensitivity (strength of the association between first-order accuracy and confidence) for episodic memory ³⁵, and comparable metacognitive efficiency (M-Ratio) during a detection task ³⁶. In contrast, many studies reported a metacognitive deficit in chronic schizophrenia without controlling for concomitant lower first-order performance ^{10,12,37,38}. Therefore, an important aspect of future studies quantifying confidence and metacognitive performance in individuals with schizophrenia will be to systematically control for potential confounds in terms of first-order performance.

Relationships between action execution and metacognitive performance

Reaction times were not longer in individuals with schizophrenia spectrum disorders, nor were mouse movement onsets, maybe as a consequence of the artefactual matching of task difficulty and accuracy between the two groups. Both groups featured a negative relationship between reaction times and confidence, but significantly stronger in the control group. This result is in line with a previous report showing a lack of correlation between reaction times and confidence in emotion recognition for individuals with schizophrenia whereas reaction times were negatively associated with confidence in controls ³⁹. Together, these results suggest that decisional parameters such as reaction times have less influence on subsequent confidence ratings in individuals with schizophrenia spectrum disorders.

The analysis of reaction times with an evidence accumulation model revealed no difference in parameters between groups (see SI). Beyond mere reaction times, we also analyzed the mouse trajectories leading to first-order responses, considered as a relevant time-resolved proxy to parse the processing steps underlying confidence judgments^{15,16}. We found that velocity and acceleration during the decision movement were more closely linked to confidence in the control than in the schizophrenia group. The link between confidence and trajectories in the control group corroborates the view that sensorimotor signals shape confidence estimates. Indeed, previous studies showed that electromyographic⁴⁰ and alpha power over somatosensory scalp regions⁴¹ covary with confidence, and that altering sensorimotor signaling by increasing movement speed⁴² or by inducing sensorimotor conflicts⁴³ disrupt metacognitive accuracy. The weaker link between trajectories and confidence in individuals with schizophrenia may be related to slower and noisier motor behavior, or to the tendency of patients to neglect relevant internal cues to control motor actions⁴⁴. The fact that metacognitive performance was preserved in individuals with schizophrenia despite a decreased link between confidence and trajectories suggest that sensorimotor signals may globally up or down-regulate confidence estimates, with no influence on the calibration between confidence and first-order performance as reported recently⁴⁵.

Relationships between behavioral and neuropsychological outcomes

No difference was found between patients and controls according to premorbid IQ, perceptual and verbal reasoning and working memory. Executive functions were marginally lower in patients. In contrast, coherent motion discrimination was significantly worse in patients compared to controls in line with previous studies⁴⁶ thus suggesting a deficient integration of spatially distributed motion signals in patients. In the group of patients, schizophrenic symptomatology was moderate⁴⁷ and the level of depression slightly higher than what is usually reported in stabilized outpatients sample^{48,49}. Depressive symptomatology was also higher in patients than in controls for the current study. Patients reported mean clinical⁵⁰ and cognitive⁵¹⁻⁵³ insights which were comparable to those reported in previous studies including stabilized outpatients. In contrast, cognitive insight, measured as the difference between self-reflectiveness and self-certainty, was markedly lower in the controls we had recruited compared to previous studies⁵⁴⁻⁵⁶. These studies included much younger and more educated non-clinical participants than the ones included in the current sample. As age was reported to be negatively correlated with composite index scores while education was found to be negatively correlated with self-reflectiveness in a non-clinical sample⁵⁷, we believe the low cognitive insight found in

the present control group was explained by its demographics characteristics, which were matched on purpose with the demographic characteristics of the patients sample. The higher level of depression found in patients compared to controls and the low cognitive insight found in controls regarding the level usually reported both converged to explain that cognitive insight was unexpectedly better in patients than in controls. This difference was indeed not significant anymore when depression was entered as a covariate. The absence of metacognitive deficit found in participants with schizophrenia recruited in this study may be explained by their preserved cognitive insight.

We found a significant correlation between metacognitive efficiency and visual reasoning on the whole group of participants, which suggests that metacognition and reasoning abilities depend on partially overlapping cognitive mechanisms ⁵⁸. Previous studies reported that metamemory correlated with executive function, visual recognition memory ⁵⁹ and working memory ³⁷. Contrary to a previous study reporting a significant association between poor insight and metacognitive deficits in individuals with schizophrenia ⁶⁰, we found no correlation between illness insight and metacognitive performance or confidence bias. Plus, metacognitive performance did not correlate with psychosocial functioning in patients. Our study thus does not confirm the significant association between synthetic metacognition (drawing upon a broad range of social, executive, linguistic, and metacognitive processes, such as the Metacognitive Assessment Scale) and functioning previously reported ⁸.

Conclusions

This study emphasizes the importance to run future studies controlling for first-order accuracy and reasoning before concluding that individuals with schizophrenia have a specific metacognitive deficit. While our results indicate that performance monitoring during a visual discrimination task may not be impaired, future work will be needed to assess how such monitoring generalizes to the capacity to evaluate the veridicality of complex representations such as hallucinations, delusional ideation or the emotional states and intentions of others in individuals with schizophrenia.

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