Research Paper

Confidence in visual motion discrimination is preserved in individuals with schizophrenia

Nathan Faivre,* PhD; Matthieu Roger,* MD; Michael Pereira, PhD; Vincent de Gardelle, PhD; Jean-Christophe Vergnaud, PhD; Christine Passerieux, MD, PhD; Paul Roux, MD, PhD

Background: Metacognition is the set of reflexive processes that allows humans to evaluate the accuracy of their mental operations. Metacognitive deficits have been described in people with schizophrenia using mostly narrative assessment, and they have been linked to several key symptoms. **Methods:** We assessed metacognitive performance objectively by asking people with schizophrenia or schizoaffective disorder (n = 20) and matched healthy participants (n = 21) to perform a visual discrimination task and report their 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 2 groups, despite a weaker association between confidence and trajectory tracking during task execution among people with schizophrenia. We reproduced these results using an evidence accumulation model, which showed similar decisional processes in the 2 groups. **Limitations:** These results from a relatively small study sample cannot be generalized to other perceptual and nonperceptual tasks. To meet this purpose, ecological tasks are needed. As well, the role of antipsychotic medication and design deserves greater attention in the future. **Conclusion:** We found similar decisional and metacognitive capabilities between people 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 person's assumption of thoughts, sensations, intentions or links between events to form more complete and lasting representations.[1,2] Regarding the latter, people 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, such as positive and negative symptoms,[5] disorganization,[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 a first-order task) and then assess how well they performed (i.e., a second-order task consisting of a confidence judgment, error detection or postdecisional 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 have reported deficits in metacognitive performance in people with schizophrenia across several domains, including perception,[10] agency[11] and memory.[12] Although these studies have provided valuable insights into putative deficits in discrete metacognition, several biases might interfere with the assessment of metacognitive performance in people with schizophrenia. First, metacognitive performance depends on first-order performance: it is easier to provide confidence judgments or

Correspondence to: N. Faivre, Laboratoire de Psychologie et Neurocognition, CNRS UMR 5105, UGA BSHM, 1251 Avenue Centrale, 38058 Grenoble Cedex 9, France; nathan.faivre@univ-grenoble-alpes.fr.

*These authors contributed equally to this paper.

Submitted Jan. 31, 2020; Revised Jun. 5, 2020; Accepted Jun. 9, 2020; Early-released Sept. 24, 2020

DOI: 10.1503/jpn.200022

detect errors for easy tasks than for difficult ones. Thus, it is crucial to control for first-order task performance, which is usually lower in people with schizophrenia than in controls. Other biases might influence metacognition in people with schizophrenia, such as depression, which has been associated with better metacognition[13] and cognitive deficits that are 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 the result of deficits at any of these levels.

Here, we first sought to test the existence of a metacognitive deficit in people with schizophrenia while accounting for possible differences in first-order performance. Then, we aimed at pinpointing the putative origins of metacognitive deficits in people with schizophrenia and describing how first- and second-order cognitive processes unfold over time by analyzing behavioural responses together with trajectory tracking and by reproducing them using an evidence accumulation model of decision-making. That is, 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] We also modelled first- and second-order responses as derived from an evidence accumulation process, starting when participants initiated a mouse movement.[17–20] Together, these 2 approaches following a preregistered plan allowed us to finely characterize decision-making and metacognitive monitoring in people with schizophrenia in relation to clinical traits while avoiding the typical confounds that may have contaminated previous results in the field.

Methods

This was a transversal monocentric study. The experimental paradigm and analysis plan detailed below were registered before data collection (NCT03140475) and are available together with anonymized data and analysis scripts (https://osf.io/84wqp/).

Participants

Twenty-three healthy volunteers (15 males, 8 females) from the general population and 20 patients with a schizophrenia spectrum disorder (16 males, 4 females) took part in this study. Two healthy volunteers were excluded from the analysis, 1 because of a convergence failure during the staircase procedure and 1 because of an estimated IQ of less than 70. Patients with schizophrenia spectrum disorders (schizophrenia or schizoaffective disorder) were recruited from community mental health centres and outpatient clinics in the Versailles area. Control participants were recruited from the volunteers' panel at the Centre d'Economie de la Sorbonne and Versailles Hospital (for details, see Appendix 1, available at jpn.ca/200022-a1). Exclusion criteria for both groups were as follows: a moderate to severe substance use disorder (according to DSM-5 criteria[21]) within the 12 months preceding the study; a current or previous untreated medical illness, including neurologic illness; an IQ of less than 70 based on 3 subtests of the Wechsler Adult Intelligence Scale[22] (see Appendix 1); and age older than 60 years. Schizophrenia and schizoaffective disorders were diagnosed by a psychiatrist investigator based on the Structured Clinical Interview for assessing DSM-5 criteria.[23] Another licensed psychiatrist (the participant's treating psychiatrist) confirmed the diagnosis for each participant according to DSM-5 criteria.[21] All participants were right-handed, had normal hearing, and had normal or corrected-to-normal vision. They were naive to the purpose of the study and provided informed consent. The investigators checked whether patients were capable of providing 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 after the proposal to participate in the research by the patient's referring psychiatrist. Information was provided orally and adapted to the patient's verbal comprehension skills, but also provided in written form. The investigators answered any questions before the participant signed the consent form and then obtained written informed consent from each participant.

The study was approved by the ethical committee Sud Méditérannée II (217 R01). Our plan at preregistration was to collect data until we reached a Bayes factor of either 0.3 or 3 with respect to the difference in metacognitive performance between groups. We halted data collection when we obtained evidence for the null hypothesis for a group difference in M-ratio (see analyses below).

Neuropsychological and clinical evaluation

We evaluated participants with schizophrenia spectrum disorders and healthy controls using several clinical and neuropsychological continuous measures, described in Appendix 1.

Experimental design

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

Statistical analysis

All analyses were performed using R (2018).

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

The second objective was to test for differences in metacognitive performance between patients and controls, using

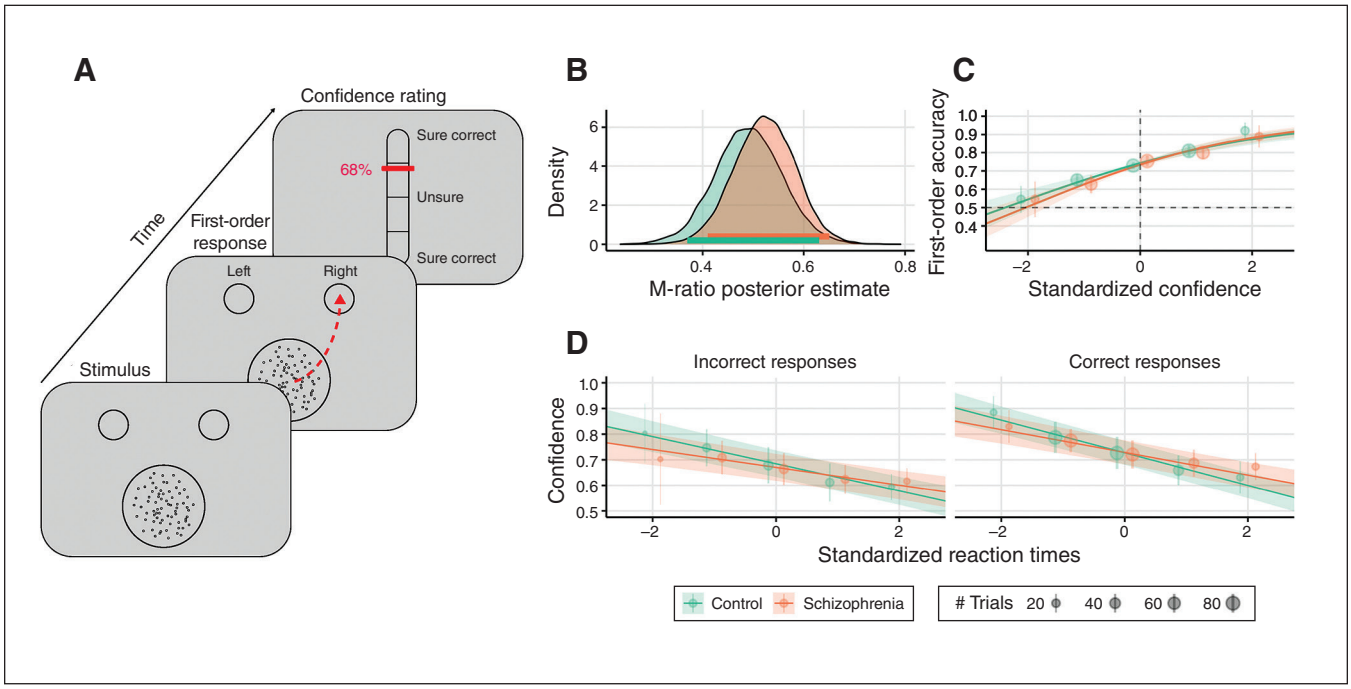

Fig. 1: Experimental paradigm and behavioural performance. (A) Experimental paradigm. Participants were presented with a random-dot kinetogram stimulus moving rightward or leftward and asked to report motion direction by moving the mouse cursor toward a circle presented at the top left or top right of the screen (first-order response). Then, participants reported the confidence they had in their response by moving a cursor on a visual analogue scale (second-order response). Exemplar mouse trajectory and confidence ratings are shown in red. (B) Posterior distribution density of the M-Ratio for the control (green) and schizophrenia groups (orange). The coloured 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 and D, regression lines and 95% confidence intervals around them represent the model fit. Although the model took continuous variables as input, for illustrative purposes we plotted dots and error bars that represent mean ± 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.

2 complementary measures. We quantified metacognitive sensitivity using 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 v. patients), random intercepts by participants and a full random-effects structure. We quantified metacognitive efficiency in a Bayesian framework as the ratio between meta-d′ (continuous variable) and d′ (M-ratio, continuous variable).[24,25]

The third objective was to compare bias 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[26]).

The fourth objective was to compare the strength of the relationships between confidence and response motion kinematics in patients and controls. Mouse spatial trajectories (x, y; continuous variables) were preprocessed (see Appendix 1) and fitted using a model II linear regression with the major axis method.[27] Kinematics (velocity, acceleration; continuous variables) were standardized across participants (z score) and analyzed as a function of confidence using mixed-effects linear regressions.

Results

Cognitive and clinical variables

The 2 groups did not differ in terms of sex ($\chi^2 = 0.45$, $p = 0.50$), age, education, premorbid intelligence level or neuropsychological performance, except for total score in the Six Elements Test,[28] which was marginally lower for patients (mean ± 95% confidence interval [CI] 801.0 ± 101.3) than for controls (920.8 ± 86.4; $t_{37.07} = 1.76$, $p = 0.09$; Table 1).

Two other variables differed between patients and controls: depressive symptoms, which were higher in patients (mean ± 95% CI 4.5 ± 1.8) than in controls (0.5 ± 0.4; $t_{21.21} = -4.3$, $p = 0.001$); and cognitive insight scores, which were also higher in patients (mean ± 95% CI 5.8 ± 3.2) than in controls (−0.6 ± 1.7; $t_{28.74} = -3.4$, $p = 0.002$). Of note, the latter difference was no longer significant 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 ($\beta = 0.75 \pm 0.30$, $t_{37} = 2.52$, $p = 0.02$, Bayes factor [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. Depression scores

Table 1: Clinical, neuropsychological and behavioural characteristics of patients and controls

	Control, mean ± 95% CI (n = 21)	Schizophrenia, mean ± 95% CI (n = 20)	t statistic	p value	Bayes factor
Age, yr	42.6 ± 4.8	38.8 ± 5.1	$t_{37.87} = 1.09$	0.29	0.50
Beck Cognitive Insight Scale, score	−0.6 ± 1.7	5.8 ± 3.2	$t_{28.74} = -3.39$	0.002	20.96
Calgary Depression Scale, score	0.5 ± 0.4	4.5 ± 1.8	$t_{21.21} = -4.26$	0.001	171.20
Education level, yr	12.5 ± 0.4	13.6 ± 1.3	$t_{22.70} = -1.56$	0.13	0.80
Premorbid IQ, score	104.0 ± 3.6	102.3 ± 3.9	$t_{37.74} = 0.65$	0.52	0.37
Six Elements Test, errors	9.2 ± 1.4	8.0 ± 2.1	$t_{32.94} = 0.93$	0.36	0.44
Six Elements Test, points	920.8 ± 86.4	801.0 ± 101.3	$t_{37.07} = 1.76$	0.09	1.04
WAIS matrix subtest, score	10.2 ± 1.1	9.0 ± 1.3	$t_{37.13} = 1.33$	0.19	0.62
WAIS letter–number sequencing subtest, score	9.1 ± 1.2	7.7 ± 1.1	$t_{37.98} = 1.69$	0.10	0.94
WAIS vocabulary subtest, score	10.0 ± 1.2	11.1 ± 1.5	$t_{36.54} = -1.06$	0.30	0.48
Criterion	0.00 ± 0.21	−0.32 ± 0.15	$t_{37.7} = 2.50$	0.020	3.24
Sensitivity, d'	1.38 ± 0.11	1.29 ± 0.08	$t_{36.7} = 0.85$	0.40	0.41
Motion variance	2.01 ± 0.20	1.59 ± 0.17	$t_{38.9} = 3.09$	0.004	10.60

CI = confidence interval; IQ = intelligence quotient; WAIS = Wechsler Adult Intelligence Scale.

were 6 or greater for 45% of patients, indicating possible major depressive disorder in these participants.[29] The Calgary Depression Scale[30] total score was less than 6 for all participants in the control group. In the patients' sample, we measured the intensity of schizophrenia using the Positive and Negative Syndrome Scale:[31] the mean total score was 78.5 ± 6.8; the mean positive symptom score was 17.2 ± 2.2; the mean negative symptom score was 20.5 ± 2.3; and the mean general psychopathology score was 40.9 ± 3.8. The mean illness duration was 14.7 ± 3.7 years, and the mean chlorpromazine equivalent was 439.7 ± 118.4 mg/24 h. The mean score on the Personal and Social Performance Scale[32] was 55.7 ± 5.4, and the mean total score on the Birchwood Insight Scale[33] was 10.8 ± 1.1. The group of patients included 13 participants with schizophrenia and 7 with schizoaffective disorders.

First- and second-order performance

Participants indicated the direction of a random-dot kinetogram (first-order task) and then reported their confidence in their decision (second-order task, see Methods and Appendix 1). Analysis of participant performance on the first-order task is reported in Table 1 and Appendix 1.

At the second-order level, average confidence ratings were similar between groups (patients: 0.71 ± 0.05, controls: 0.70 ± 0.06; $t_{38.4} = 0.12$, $p = 0.91$, BF = 0.31), as was 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). Behavioural results (confidence, B-ROC) remained unchanged when the Calgary Depression Scale total score was entered as a covariate. We then estimated metacognitive efficiency (i.e., the 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 ± standard deviation = 0.2 ± 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 colleagues.[34] Results showed that the 2 groups had similar metacognitive efficiency (patients: 0.52, 95% highest posterior density interval = 0.40 to 0.65; controls: 0.49, highest posterior density interval = 0.37 to 0.64), with a BF of 0.18 supporting the absence of difference between groups (Fig. 2B). We also computed another metric of metacognitive performance — metacognitive sensitivity — which corresponds to the slope of the logistic regression between first-order accuracy and confidence. We made a similar prior assumption for higher metacognitive efficiency in the control group, represented by a steeper slope (i.e., Gaussian distribution with mean ± standard deviation = 1 ± 5). We chose a weakly informative prior in the absence of published evidence. We found no interaction between group and confidence (estimate = 0.06, highest posterior density interval = −0.15 to 0.27; BF = 0.02; Fig. 2C). Importantly, BFs less than 0.3 for both metacognitive efficiency and sensitivity supported the null hypothesis: that people with schizophrenia would have no impairment while adjusting confidence to their performance.

Following our preregistered plan, we then sought to assess how motor behaviour related to first-order responses modulated confidence ratings. We quantified the relationship between confidence, first-order accuracy and standardized reaction times for both groups using 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 to −0.04]; evidence ratio > 4000), indicating 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 to −0.01]; evidence ratio = 221.22) and group (interaction group × reaction times: estimate = 0.02 [0.00 to 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

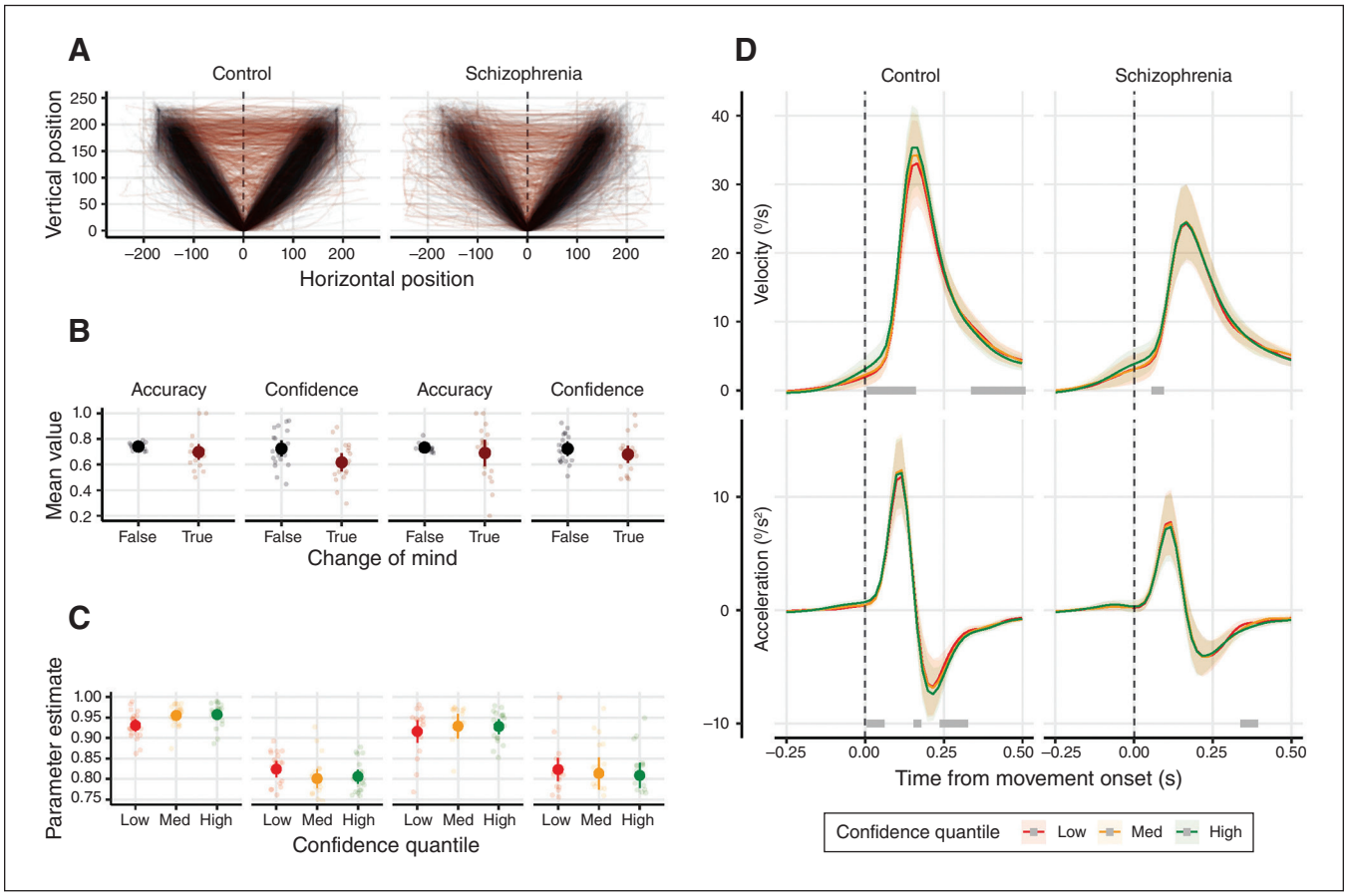

Fig. 2: Trajectory tracking. (A) Single-trial mouse trajectories leading to the first-order response in case of a change of mind (red) or no change of mind (black) in the control (left panel) and patient groups (right panel). (B) Average first-order accuracy and confidence in the presence (red) and absence (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 before movement onset as it was defined as a function of the maximal velocity in a given trial (see Appendix 1). Shaded areas represent the 95% confidence intervals. Grey bars represent samples for which confidence covaried significantly with kinematics ($p < 0.05$, corrected for false discovery rate).

results indicate that reaction times covary with confidence to a lesser extent in people with schizophrenia, suggesting that this group may have relied less on this input to form confidence estimates.

Trajectory tracking

Beyond reaction times, we quantified how mouse trajectories leading to first-order responses predicted subsequent confidence judgments (see Fig. 2A for raw trajectories). First, we isolated trials in which a change of mind occurred; that is, when participants started moving toward one response circle and later changed direction toward the other (see Appendix 1). Changes of mind corresponded to 7.9 ± 2.7% and 7.3 ± 2.9% of total trials in the patient and control groups, respectively ($t_{37.9}$ = 0.32, p = 0.75, BF = 0.32). Interestingly, 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 = 0.040; see Fig. 2B), without significant interaction between group and accuracy (estimate = 0.18, z = 0.84, p = 0.40). Conversely, 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 in confidence was more pronounced in controls than in patients (interaction term: $F_{1,30.3}$ = 5.03, p = 0.03), indicating that patients revised their confidence level less 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 group and first-order accuracy (Fig. 2C). This finding suggests that both patients and controls moved the mouse more laterally for responses associated with high confidence, whether correct or not. We also fitted a linear model to individual trajectories and found a positive relationship between

goodness of fit (R^2) and confidence ($F_{1,40.1} = 17.5$, $p < 0.001$) that was independent of group and first-order accuracy, revealing that confidence ratings were higher after responses following more linear trajectories. Of note, a trend suggested a 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 centred data to 0 to account for potential motor impairment in patients with schizophrenia.[35] 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 2 groups, but stronger in the control group than in the patient group ($p < 0.05$, corrected for false discovery rate). We explored this interaction 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 (Fig. 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 ($p < 0.05$, corrected for false discovery rate). As for velocity, we explored this interaction by fitting acceleration models for each group, which showed that the correlation between confidence and acceleration was significant at both movement onset and offset in the control group (Fig. 2D). Among patients, we found only a weaker correlation following movement offset. 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 controls compared with patients with schizophrenia.

Finally, we examined the relationship between second-order behavioural measures (confidence, M-ratio) and these cognitive and clinical variables using Bayesian robust regressions (see Appendix 1). Regarding cognitive variables, we found that M-ratio covaried positively with the Wechsler Adult Intelligence Scale matrix subtest[22] ($r = 0.46$, highest posterior density interval = 0.20 to 0.70, BF = 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 to be significant (see Appendix 1, Table S1). No significant correlation was found between second-order behavioural measures and clinical variables specific to the patient population (see Appendix 1, Tables S2).

Discussion

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

Metacognitive performance

We found no significant difference in metacognitive performance between groups, and Bayesian analyses favoured 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 were compatible with a recent account suggesting that people with schizophrenia may not be affected to form confidence estimates per se, but rather to interpret their saliency.[29] 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. The absence of a difference between groups was unlikely to be a result of abnormally poor metacognitive performance in our control population, because healthy controls performed similarly to participants from previous studies involving a coherent motion discrimination paradigm (e.g., mean M-ratio = 0.66 in a recent study[36]). As well, the unexpected better cognitive insight found in patients compared with controls could not explain the lack of difference in metacognitive performance between groups, because cognitive insight was related to neither metacognitive efficiency nor metacognitive sensitivity. (One could argue that including patients with schizoaffective disorders might have compensated for a potential deficit in metacognition in patients with schizophrenia by increasing depressive symptomatology, which has been associated with better metacognitive efficiency.[37] However, the prevalence of possible depression [based on the Calgary Depression Scale cut-off] in this sample of patients with schizophrenia spectrum disorders was close to the 40% value reported in another study investigating outpatients with schizophrenia only.[38]) In addition, a post hoc comparison of M-ratio with cognitive insight as a 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 2 groups was that the patients with schizophrenia were medicated with antipsychotics acting as dopaminergic antagonists. Although there is evidence that confidence may be modulated by dopamine,[39,40] we found evidence for the null hypothesis using Bayesian correlations between confidence and chlorpromazine equivalents in our sample.

Recent studies have reported lower visual metacognitive performance in people with a first episode of psychosis relative to age-matched controls.[34,41] In the present study, we tested people with a chronic disorder and found no deterioration of metacognition relative to controls, but we did find a negative relationship between metacognitive efficiency and illness duration (Appendix 1, Table S2). This finding suggests that metacognitive performance in patients may evolve nonlinearly 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[42] and perceptual categorization task[43] compared with psychiatric

control groups with similar type 1 accuracy. However, our results were in line with those of previous studies investigating metacognitive performance controlling for first-order accuracy in chronic schizophrenia, which reported equivalent metacognitive sensitivity (area under the type 2 receiver operating characteristic curve) between patients and controls for facial emotion recognition,[44] comparable metacognitive sensitivity (strength of the association between first-order accuracy and confidence) for episodic memory[45] and comparable metacognitive efficiency (M-ratio) during a detection task.[46] In contrast, many studies have reported a metacognitive deficit in chronic schizophrenia without controlling for concomitant lower first-order performance.[10,12,47,48] Therefore, an important aspect of future studies quantifying confidence and metacognitive performance in people 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 patients with schizophrenia spectrum disorders, nor were mouse movement onsets, possibly as a consequence of the artifactual matching of task difficulty and accuracy between the 2 groups. Both groups featured a negative relationship between reaction times and confidence, but this was significantly stronger in the control group. This result was in line with the findings of a previous report showing a lack of correlation between reaction times and confidence in emotion recognition for people with schizophrenia, whereas reaction times were negatively associated with confidence in controls.[49] Together, these results suggest that decisional parameters such as reaction times have less influence on subsequent confidence ratings in people with schizophrenia spectrum disorders.

The analysis of reaction times with an evidence accumulation model revealed no difference in parameters between groups (see Appendix 1). 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 group than in the patient 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 electromyographics[50] and α power over somatosensory scalp regions[51] covary with confidence, and that altering sensorimotor signalling by increasing movement speed[52] or by inducing sensorimotor conflicts[53] disrupt metacognitive accuracy. The weaker link between trajectories and confidence in patients with schizophrenia may be related to slower and noisier motor behaviour, or to the tendency of people with schizophrenia to neglect relevant internal cues to control motor actions.[54] The fact that metacognitive performance was preserved in patients with schizophrenia despite a decreased link between confidence and trajectories suggests that sensorimotor signals may globally up- or downregulate confidence estimates, with no influence on the calibration between confidence and first-order performance as reported recently.[54]

Relationships between behavioural and neuropsychological outcomes

We found no difference between patients and controls according to premorbid IQ, perceptual and verbal reasoning, and working memory. Executive functions were marginally lower in the patient group. In contrast, coherent motion discrimination was significantly worse in patients compared with controls, in line with previous studies,[55] suggesting a deficient integration of spatially distributed motion signals in patients. In the patient group, schizophrenic symptomatology was moderate[56] and the level of depression was slightly higher than what is usually reported in a sample of stabilized outpatients.[37,38] Depressive symptomatology was also higher in patients than in controls in the current study. Patients reported mean clinical[57] and cognitive[58–60] insights that 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 with previous studies.[61–63] These studies included much younger and more educated nonclinical participants than those included in the current sample. Because age was reported to be negatively correlated with composite index scores and education was negatively correlated with self-reflectiveness in a nonclinical sample,[64] we believe the low cognitive insight found in the control group in the present study was explained by demographic characteristics, which were deliberately matched with the demographic characteristics of the patient group. The higher level of depression found in patients compared with controls and the low cognitive insight found in controls related to the level usually reported both converged to explain that cognitive insight was unexpectedly better in the patient group than in the control group. This difference was no longer significant when depression was entered as a covariate. The absence of metacognitive deficit found in patients with schizophrenia recruited for this study may be explained by their preserved cognitive insight.

We found a significant correlation between metacognitive efficiency and visual reasoning for the whole group of participants, which suggests that metacognition and reasoning abilities depend on partially overlapping cognitive mechanisms.[65] Previous studies have reported that metamemory is correlated with executive function, visual recognition memory[66] and working memory.[47] Contrary to a previous study reporting a significant association between poor insight and metacognitive deficits in people with schizophrenia,[67] we found no correlation between illness insight and metacognitive performance or confidence bias. As well, metacognitive performance did not correlate with psychosocial functioning in our patient group. Therefore, our study did 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[68]) and functioning previously reported.[8]

Limitations

We note that these results should be interpreted with caution, considering the relatively small sample size on which they are based.

Conclusion

This study emphasizes the importance of running future studies that control for first-order accuracy and reasoning before concluding that people with schizophrenia have a specific metacognitive deficit. Although our results indicate that performance monitoring during a visual discrimination task may not be impaired, future work is 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 people with schizophrenia.

Acknowledgments: The authors thank Eric Brunet for his pertinent comments about the interpretation of our results, and Elisa Filevich and Lucien Gyger for helpful comments on our manuscript. We are also grateful to Laure Morisset at Versailles Hospital for her institutional support. We thank three anonymous reviewers for their helpful comments.

Affiliations: From the Université Grenoble Alpes, Grenoble, France (Faivre, Pereira); the Centre d'Economie de la Sorbonne, Paris, France (Faivre, de Gardelle, Vergnaud); the Université Paris-Saclay, Université Versailles Saint-Quentin-En-Yvelines, INSERM UMR1018, Centre de recherche en Épidémiologie et Santé des Populations, Equipe DevPsy, Villejuif, France (Roger, Passerieux, Roux); the Centre Hospitalier de Versailles, Service Hospitalo-Universitaire de psychiatrie d'adultes et d'addictologie, Le Chesnay, France (Roger, Passerieux, Roux); and the Fondation Fondamental, Créteil, France (Passerieux, Roux).

Competing interests: None declared.

Funding: This project has received funding from the European Research Council (ERC) under the European Union's Horizon 2020 research and innovation programme (grant agreement No 803122)

Contributors: N. Faivre, M. Roger, J.-C. Vergnaud, and P. Roux designed the study. N. Faivre, M. Roger and P. Roux acquired the data, which N. Faivre, M. Roger, M. Pereira, V. de Gardelle, C. Passerieux and P. Roux analyzed. N. Faivre, M. Roger and P. Roux wrote the article, which all authors reviewed. All authors approved the final version to be published and can certify that no other individuals not listed as authors have made substantial contributions to the paper.

Data sharing: Experimental paradigm, anonymized data and analysis scripts are available online: https://osf.io/84wqp.

References

1. Lysaker PH, Vohs JL, Ballard R, et al. Metacognition, self-reflection and recovery in schizophrenia. *Future Neurol* 2013;8:103-15.
2. Bob P, Pec O, Mishara AL, et al. Conscious brain, metacognition and schizophrenia. *Int J Psychophysiol* 2016;105:1-8.
3. Hasson-Ohayon I, Avidan-Msika M, Mashiach-Eizenberg M, et al. Metacognitive and social cognition approaches to understanding the impact of schizophrenia on social quality of life. *Schizophr Res* 2015;161:386-91.
4. Lysaker PH, Dimaggio G. Metacognitive capacities for reflection in schizophrenia: implications for developing treatments. *Schizophr Bull* 2014;40:487-91.
5. McLeod HJ, Gumley AI, MacBeth A, et al. Metacognitive functioning predicts positive and negative symptoms over 12 months in first episode psychosis. *J Psychiatr Res* 2014;54:109-15.
6. Vohs JL, Lysaker PH, Francis MM, et al. Metacognition, social cognition, and symptoms in patients with first episode and prolonged psychoses. *Schizophr Res* 2014;153:54-9.
7. Lysaker PH, Dimaggio G, Carcione A, et al. Metacognition and schizophrenia: the capacity for self-reflectivity as a predictor for prospective assessments of work performance over six months. *Schizophr Res* 2010;122:124-30.
8. Arnon-Ribenfeld N, Hasson-Ohayon I, Lavidor M, et al. The association between metacognitive abilities and outcome measures among people with schizophrenia: a meta-analysis. *Eur Psychiatry* 2017;46:33-41.
9. Fleming SM, Lau HC. How to measure metacognition. *Front Hum Neurosci* 2014;8:443.
10. Moritz S, Woznica A, Andreou C, et al. Response confidence for emotion perception in schizophrenia using a continuous facial sequence task. *Psychiatry Res* 2012;200:202-7.
11. Metcalfe J, Van Snellenberg JX, DeRosse P, et al. Judgements of agency in schizophrenia: an impairment in autonoetic metacognition. *Philos Trans R Soc Lond B Biol Sci* 2012;367:1391-400.
12. Moritz S, Woodward T, Jelinek L, et al. Memory and metamemory in schizophrenia: a liberal acceptance account of psychosis. *Psychol Med* 2008;38:825-32.
13. Lysaker PH, Carcione A, Dimaggio G, et al. Metacognition amidst narratives of self and illness in schizophrenia: associations with neurocognition, symptoms, insight and quality of life. *Acta Psychiatr Scand* 2005;112:64-71.
14. Davies G, Greenwood K. A meta-analytic review of the relationship between neurocognition, metacognition and functional outcome in schizophrenia. *J Ment Health* 2018:1-11.
15. Dotan D, Meyniel F, Dehaene S. On-line confidence monitoring during decision making. *Cognition* 2018;171:112-21.
16. Dotan D, Pinheiro-Chagas P, Al Roumi F, et al. Track it to crack it: dissecting processing stages with finger tracking. *Trends Cogn Sci* 2019;23:1058-70.
17. Pereira M, Faivre N, Iturrate I, et al. Disentangling the origins of confidence in speeded perceptual judgments through multimodal imaging. *Proc Natl Acad Sci U S A* 2020;117:8382-90.
18. Pleskac TJ, Busemeyer JR. others. Two-stage dynamic signal detection: a theory of choice, decision time, and confidence. *Psychol Rev* 2010;117:864.
19. Resulaj A, Kiani R, Wolpert DM, et al. Changing your mind: a computational mechanism of vacillation. *Nature* 2009;461:263-6.
20. Van Den Berg R, Anandalingam K, Zylberberg A, et al. A common mechanism underlies changes of mind about decisions and confidence. *eLife* 2016;5:e12192.
21. *Diagnostic and statistical manual of mental disorders.* 5th ed. Arlington (VA): American Psychiatric Association; 2013.
22. Wechsler D, Coalson DL, Raiford SE. *WAIS-IV: Wechsler adult intelligence scale.* San Antonio (TX): Pearson; 2008.
23. First MB, Williams JB, Karg RS, et al. *User's guide for the SCID-5-CV: structured clinical interview for DSM-5 disorders, clinician version.* Arlington (VA): American Psychiatric Association; 2016.
24. Fleming SM. HMeta-d: hierarchical Bayesian estimation of metacognitive efficiency from confidence ratings. *Neurosci Conscious* 2017;nix007.
25. Maniscalco B, Lau H. A signal detection theoretic approach for estimating metacognitive sensitivity from confidence ratings. *Conscious Cogn* 2012;21:422-30.
26. Kornbrot DE. Signal detection theory, the approach of choice: model-based and distribution-free measures and evaluation. *Percept Psychophys* 2006;68:393-414.

27. Legendre P. *Lmodel2: model II regression.* Montreal (QC): Université de Montreal; 2018. Available: CRAN.R-project.org/package=lmodel2 (accessed 2020 Sep. 10).
28. Wilson BA, Evans JJ, Alderman N, et al. Behavioural assessment of the dysexecutive syndrome. *Methodol Front Exec Funct* 1997;239:250.
29. Moritz S, Pfuhl G, Lüdtke T, et al. A two-stage cognitive theory of the positive symptoms of psychosis. Highlighting the role of lowered decision thresholds. *J Behav Ther Exp Psychiatry* 2017;56:12-20.
30. Addington D, Addington J, Maticka-Tyndale E, et al. Reliability and validity of a depression rating scale for schizophrenics. *Schizophr Res* 1992;6(3):201-8.
31. Kay SR, Fiszbein A, Opler LA. The positive and negative syndrome scale (PANSS) for schizophrenia. *Schizophr Bull* 1987;13:261-76.
32. Morosini P, Magliano L, Brambilla L, et al. Development, reliability and acceptability of a new version of the DSM-IV Social and Occupational Functioning Assessment Scale (SOFAS) to assess routine social funtioning. *Acta Psychiatr Scand* 2000;101:323-9.
33. Birchwood M, Smith J, Drury V, et al. A self-report insight scale for psychosis: reliability, validity and sensitivity to change. *Acta Psychiatr Scand* 1994;89:62-7.
34. Davies G, Rae CL, Garfinkel SN, et al. Impairment of perceptual metacognitive accuracy and reduced prefrontal grey matter volume in first-episode psychosis. *Cogn Neuropsychiatry* 2018;23:165-79.
35. Manschreck TC, Maher BA, Rucklos ME, et al. Disturbed voluntary motor activity in schizophrenic disorder. *Psychol Med* 1982;12:73-84.
36. Konishi M, Compain C, Berberian B, et al. Resilience of metacognition in a dual-task visual paradigm. *PsyArXiv* 2019. doi:10.31234/osf.io/xcseh
37. Roux P, Misdrahi D, Capdevielle D, et al. Mediation analyses of insight, quality of life, depression, and suicidality: results from the FACE-SZ Cohort. *J Clin Psychiatry* 2018;79:17m11638.
38. Tanaka T, Tomotake M, Ueoka Y, et al. Clinical correlates associated with cognitive dysfunction in people with schizophrenia. *Psychiatry Clin Neurosci* 2012;66:491-8.
39. Andreou C, Moritz S, Veith K, et al. Dopaminergic modulation of probabilistic reasoning and overconfidence in errors: a double-blind study. *Schizophr Bull* 2014;40:558-65.
40. Lou HC, Skewes JC, Thomsen KR, et al. Dopaminergic stimulation enhances confidence and accuracy in seeing rapidly presented words. *J Vis* 2011;11:15.
41. Alkan E, Davies G, Greenwood K, et al. Brain structural correlates of metacognition in first-episode psychosis. *Schizophr Bull* 2020;46:552-61.
42. Moritz S, Woodward TS. Metacognitive control over false memories: a key determinant of delusional thinking. *Curr Psychiatry Rep* 2006;8:184-90.
43. Moritz S, Ramdani N, Klass H, et al. Overconfidence in incorrect perceptual judgments in patients with schizophrenia. *Schizophr Res Cogn* 2014;1:165-70.
44. Pinkham AE, Klein HS, Hardaway GB, et al. Neural correlates of social cognitive introspective accuracy in schizophrenia. *Schizophr Res* 2018;202:166-72.
45. Bacon E, Izaute M. Metacognition in schizophrenia: processes underlying patients' reflections on their own episodic memory. *Biol Psychiatry* 2009;66:1031-7.
46. Powers AR, Mathys C, Corlett PR. Pavlovian conditioning induced hallucinations result from overweighting of perceptual priors. *Science* 2017;357:596-600.
47. Eifler S, Rausch F, Schirmbeck F, et al. Metamemory in schizophrenia: retrospective confidence ratings interact with neurocognitive deficits. *Psychiatry Res* 2015;225:596-603.
48. Köther U, Veckenstedt R, Vitzthum F, et al. "Don't give me that look"—overconfidence in false mental state perception in schizophrenia. *Psychiatry Res* 2012;196:1-8.
49. Jones MT, Deckler E, Laurrari C, et al. Confidence, performance, and accuracy of self-assessment of social cognition: a comparison of schizophrenia patients and healthy controls. *Schizophr Res Cogn* 2019;19:002.
50. Gajdos T, Fleming SM, Saez Garcia M, et al. Revealing subthreshold motor contributions to perceptual confidence. *Neurosci Conscious* 2019;2019:niz001.
51. Faivre N, Filevich E, Solovey G, et al. Behavioral, modeling, and electrophysiological evidence for supramodality in human metacognition. *J Neurosci* 2018;38:263-77.
52. Palser ER, Fotopoulou A, Kilner J. Altering movement parameters disrupts metacognitive accuracy. *Conscious Cogn* 2018;57:33-40.
53. Faivre N, Vuillaume L, Bernasconi F, et al. Sensorimotor conflicts alter metacognitive and action monitoring. *Cortex* 2020;124:224-34.
54. Filevich E, Koß C, Faivre N. Response-related signals increase confidence but not metacognitive performances. *Eneuro* 2020;7:0326-19.
55. Chen Y. Abnormal visual motion processing in schizophrenia: a review of research progress. *Schizophr Bull* 2011;37:709-15.
56. Leucht S, Kane JM, Kissling W, et al. What does the PANSS mean? *Schizophr Res* 2005;79:231-8.
57. Ehrminger M, Urbach M, Passerieux C, et al. Modeling the longitudinal effects of insight on depression, quality of life and suicidality in schizophrenia spectrum disorders: results from the FACE-SZ Cohort. *J Clin Med* 2019;8:1196.
58. Kim JH, Lee S, Han A-Y, et al. Relationship between cognitive insight and subjective quality of life in outpatients with schizophrenia. *Neuropsychiatr Dis Treat* 2015;11:2041.
59. Misdrahi D, Denard S, Swendsen J, et al. Depression in schizophrenia: the influence of the different dimensions of insight. *Psychiatry Res* 2014;216:12-6.
60. Phalen PL, Viswanadhan K, Lysaker PH, et al. The relationship between cognitive insight and quality of life in schizophrenia spectrum disorders: symptom severity as potential moderator. *Psychiatry Res* 2015;230:839-45.
61. Kao Y-C, Liu Y-P. The Beck Cognitive Insight Scale (BCIS): translation and validation of the Taiwanese version. *BMC Psychiatry* 2010;10:27.
62. Martin JM, Warman DM, Lysaker PH. Cognitive insight in non-psychiatric individuals and individuals with psychosis: an examination using the Beck Cognitive Insight Scale. *Schizophr Res* 2010;121:39-45.
63. Uchida T, Matsumoto K, Kikuchi A, et al. Psychometric properties of the Japanese version of the Beck Cognitive Insight Scale: relation of cognitive insight to clinical insight. *Psychiatry Clin Neurosci* 2009;63:291-7.
64. Buchy L, Brodeur MB, Lepage M. The Beck Cognitive Insight Scale: psychometric properties in a Canadian community sample. *Schizophr Res* 2012;137:254-5.
65. Mäntylä T, Rönnlund M, Kliegel M. Components of executive functioning in metamemory. *Appl Neuropsychol* 2010;17:289-98.
66. Chiu C-Y, Liu C-C, Hwang T-J, et al. Metamemory in patients with schizophrenia measured by the feeling of knowing. *Psychiatry Res* 2015;230:511-6.
67. Koren D, Seidman LJ, Poyurovsky M, et al. The neuropsychological basis of insight in first-episode schizophrenia: a pilot metacognitive study. *Schizophr Res* 2004;70:195-202.
68. Semerari A, Carcione A, Dimaggio G, et al. How to evaluate metacognitive functioning in psychotherapy? The Metacognition Assessment Scale and its applications. *Clin Psychol Psychother* 2003;10:238-61.