JAMA Psychiatry | Original Investigation

Association Between Failures in Perceptual Updating and the Severity of Psychosis in Schizophrenia

Sonia Bansal, PhD; Gi-Yeul Bae, PhD; Benjamin M. Robinson, BA; Britta Hahn, PhD; James Waltz, PhD; Molly Erickson, PhD; Pantelis Leptourgos, PhD; Phillip Corlett, PhD; Steven J. Luck, PhD; James M. Gold, PhD

IMPORTANCE Recent accounts suggest that delusions and hallucinations may result from alterations in how prior knowledge is integrated with new information, but experimental evidence supporting this idea has been complex and inconsistent. Evidence from a simpler perceptual task would make clear whether psychotic symptoms are associated with overreliance on prior information and impaired updating.

OBJECTIVE To investigate whether individuals with schizophrenia or schizoaffective disorder (PSZ) and healthy control individuals (HCs) differ in the ability to update their beliefs based on evidence in a relatively simple perceptual paradigm.

DESIGN, SETTING, AND PARTICIPANTS This case-control study included individuals who met *DSM-IV* criteria for PSZ and matched HC participants in 2 independent samples. The PSZ group was recruited from the Maryland Psychiatric Research Center, Yale University, and community clinics, and the HC group was recruited from the community. To test perceptual updating, a random dot kinematogram paradigm was implemented in which dots moving coherently in a single direction were mixed with randomly moving dots. On 50% of trials, the direction of coherent motion changed by 90° midway through the trial. Participants were asked to report the direction perceived at the end of the trial. The Peters Delusions Inventory and Brief Psychiatric Rating Scale (BPRS) were used to quantify the severity of positive symptoms. Data were collected from September 2018 to March 2020 and were analyzed from approximately March 2020 to March 2021.

MAIN OUTCOMES AND MEASURES Critical measures included the proportion of responses centered around the initial direction vs the subsequent changed direction and the overall precision of motion perception and reaction times.

RESULTS A total of 48 participants were included in the PSZ group (31 [65%] male; mean [SD] age, 36.56 [9.76] years) and 36 in the HC group (22 [61%] male; mean [SD] age, 35.67 [10.74] years) in the original sample. An independent replication sample included 42 participants in the PSZ group (29 [69%] male; mean [SD] age, 33.98 [11.03] years) and 34 in the HC group (20 [59%] male; mean [SD] age, 34.29 [10.44] years). In line with previous research, patients with PSZ were less precise and had slower reaction times overall. The key finding was that patients with PSZ were significantly more likely (original sample: mean, 27.88 [95% CI, 24.19-31.57]; replication sample: mean, 26.70 [95% CI, 23.53-29.87]) than HC participants (original sample: mean, 18.86 [95% CI, 16.56-21.16]; replication sample: mean, 15.67 [95% CI, 12.61-18.73]) to report the initial motion direction rather than the final one. Moreover, the tendency to report the direction of initial motion correlated with the degree of conviction on the Peters Delusions Inventory (original sample: $r = 0.32$ [$P = .05$]; replication sample: $r = 0.30$ [$P = .05$]) and the Brief Psychiatric Rating Scale Reality Distortion score (original sample: $r = 0.55$ [$P = .001$]; replication sample: $r = 0.35$ [$P = .03$]) and severity of hallucinations (original sample: $r = 0.39$ [$P = .02$]; replication sample: $r = 0.30$ [$P = .05$]).

CONCLUSIONS AND RELEVANCE The findings of this case-control study suggest that the severity of psychotic symptoms is associated with a tendency to overweight initial information over incoming sensory evidence. These results are consistent with predictive coding accounts of the origins of positive symptoms and suggest that deficits in very elementary perceptual updating may be a critical mechanism in psychosis.

JAMA Psychiatry. doi:10.1001/jamapsychiatry.2021.3482
Published online December 1, 2021.

Supplemental content

Author Affiliations: Maryland Psychiatric Research Center, Department of Psychiatry, University of Maryland School of Medicine, Baltimore (Bansal, Robinson, Hahn, Waltz, Gold); Department of Psychology, Arizona State University, Tempe (Bae); Department of Psychiatry, University of Chicago, Chicago, Illinois (Erickson); Department of Psychiatry, Connecticut Mental Health Center, Yale University, New Haven, Connecticut (Leptourgos, Corlett); Center for Mind and Brain and Department of Psychology, University of California, Davis (Luck).

Corresponding Author: Sonia Bansal, PhD, Maryland Psychiatric Research Center, Department of Psychiatry, University of Maryland School of Medicine, 55 Wade Ave, Baltimore, MD 21228 (sbansal@som.umaryland.edu).

Delusions are tenaciously maintained, even in the face of clear disconfirmatory evidence. Similarly, hallucinations are perceptual experiences that occur despite a lack of corresponding external stimuli. Recent accounts suggest that alterations in the integration of prior expectations and new sensory inputs play a role in delusions and hallucinations. At issue is whether these abnormalities arise at the level of perceptual processes or at the level of higher-order reasoning processes. The notion that perception involves inferential processes akin to deliberative reasoning dates to seminal writings and observations of von Helmholtz,[1] who argued that, similar to reasoning, perception involved drawing conclusions about what is likely to exist in the world given sensory evidence. This perspective assumes no sharp boundaries among perception, reasoning, and belief.[2-4]

However, many researchers have emphasized the role of higher-level reasoning processes in the genesis of delusional beliefs.[5,6] This work has highlighted abnormalities in evidence gathering, belief evaluation, the impact of confirmation biases, conflict detection, and the engagement and weighting of intuitive vs analytic reasoning.[7-10] For example, Woodward et al[11] suggested that a bias against integrating disconfirmatory evidence underlies the inflexibility of delusional beliefs. Multiple reports suggest that people with more severe delusions are less likely to modify their initial beliefs when presented with subsequent information.[12-14] However, these evidence integration paradigms involve multiple complex cognitive processes (language comprehension, reasoning, and working memory) that could affect task performance, which complicates their interpretation.

Paradoxically, there is evidence that patients show excessive belief updating. For example, in the beads task, participants are asked to decide which of 2 hidden jars is the source for a series of beads.[15] Here, patients jump to conclusions, sometimes deciding after seeing only a single bead. In such tasks in which disconfirmatory evidence is presented, patients rapidly shift their judgments, updating based on very limited evidence.[16-19] Therefore, the tendency to jump to conclusions is plausibly implicated in the genesis of unusual beliefs. However, performance on the beads task does not correlate consistently with severity of delusions.[9,20,21] Thus, belief updating appears to be altered in psychosis, but the relevant paradigms involve multiple cognitive processes, and the direction of the alteration varies across methods.[22-24] These tasks involve combinations of multiple cognitive and motivational processes that are altered in schizophrenia or schizoaffective disorder (PSZ), making it difficult to isolate specific deficits in belief updating.

The present study assessed whether aberrant belief updating can be observed in a relatively simple motion perception task that poses minimal demands on conscious reasoning and is well understood at the neural level in primate and rodent models.[25] The task was a motion direction estimation paradigm (**Figure 1**) with random dot kinematogram (RDK) stimuli. In the key trials, 35% of dots move in the same coherent direction for 500 milliseconds and then move in a direction shifted by 90° relative to the initial direction for 500 milliseconds. Participants are instructed to report the direction of motion at the end of the trial. We hypothesized that patients with PSZ would favor initial evidence relative to subsequent sensory evidence, leading them to incorrectly report the initial motion direction on motion-change trials, and that this tendency would be associated with more severe positive symptoms. Such a finding would provide evidence that impaired perceptual updating plays a role in psychotic symptoms.

Key Points

Question Are psychotic symptoms related to failures in perceptual updating?

Findings In this case-control study of 160 participants in 2 independent samples, perceptual updating was assessed via a random dot motion perception task in which motion direction changes midway through a trial, and participants were asked to report the direction at the end of the trial. Individuals with schizophrenia tended to overreport the direction of the initial motion rather than the changed motion direction as instructed, which was associated with the severity of delusions and hallucinations.

Meaning The findings of this study suggest that failure to integrate new sensory evidence with prior knowledge may be associated with psychotic symptoms in schizophrenia.

Methods

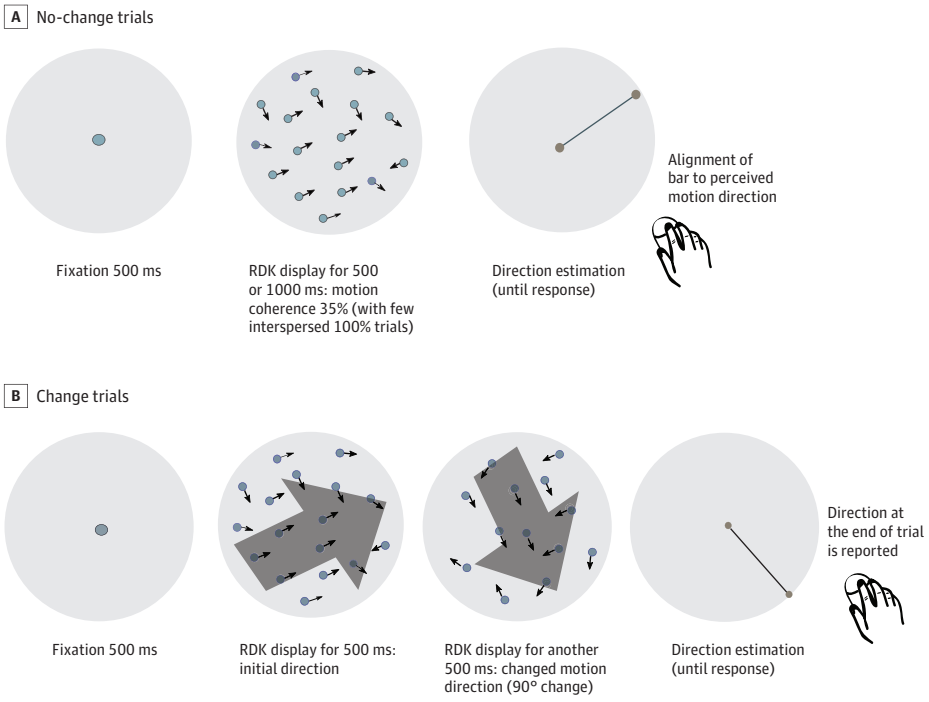
Participants

In this case-control study, we tested 48 clinically stable individuals meeting *DSM-IV-TR* criteria for PSZ[26] and 36 matched healthy control (HC) participants recruited from the outpatient clinics of the Maryland Psychiatric Research Center, Baltimore, and via internet and print advertisements. In a second independent replication sample, we tested 42 patients with PSZ and 34 HC participants, most of whom were recruited at Maryland Psychiatric Research Center, with others recruited at Yale University, New Haven, Connecticut, and community clinics. This protocol was approved by the institutional review board at the University of Maryland, Baltimore, and Yale University, and all participants gave written informed consent before taking part in the study. Participant characteristics are summarized in the **Table**. This study followed the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) reporting guideline.

Neuropsychological and Symptom Measures

Data for this study were collected from September 2018 to March 2020. We used the Measurement and Treatment Research to Improve Cognition in Schizophrenia Consensus Cognitive Battery[27] and the Wide Range Achievement Test 4[28] to assess current and premorbid cognitive functioning. We administered the Brief Psychiatric Rating Scale (BPRS)[29] to quantify symptom severity in PSZ and the Peters Delusions Inventory (PDI)[30] to quantify delusional beliefs in both the PSZ and HC groups.

Figure 1. Results of No-Change and Change Trials



A, On each no-change trial, the observer fixated the central dot for 500 milliseconds and then saw a random dot kinematogram (RDK) for 500 or 1000 milliseconds. Motion coherence was either 35% (most trials) or 100% (few interspersed trials), and the coherent motion could be in any direction from the 360° space. At the end of the RDK, the central dot changed color, indicating that the observer should report the motion direction by adjusting a line to match the perceived motion direction. B, On each change trial, the observer fixated the central dot for 500 milliseconds and then saw an RDK for an initial 500 milliseconds in which coherent motion was initially in any direction from the 360° space, and the subsequent motion of dots was presented ±90° from the initial motion direction for another 500 milliseconds. The central dot then changed color, indicating that the observer should report the motion direction observed at the end of the trial.

Stimuli and Procedure

As illustrated in Figure 1, participants were asked to report the direction of a coherent motion signal in an RDK.[31] Stimuli were 35% coherent (ie, proportion of dots moving coherently relative to those replotted randomly) on 85% of trials, with the remaining 15% of trials being "catch trials." All directions were equiprobable, and observers were instructed to report the direction of motion at the end of the trial by using a mouse to adjust a bar to match the perceived motion direction. There were 2 trial types: (1) no-change trials, in which motion direction stayed constant through 500 or 1000 milliseconds of stimulus presentation, and (2) motion change trials (always 1000 milliseconds) wherein the direction of motion changed by −90° or +90°, resulting in 500 milliseconds of initial direction, followed by 500 milliseconds of changed direction. The motion was continuous during the period of a given trial, with no break when the direction changed.

At 35% coherence, we administered 192 change trials and 192 no-change trials (half at each duration). Participants also completed 24 catch trials (100% coherence) for each of the change/no-change 500-millisecond and no-change 1000-millisecond trial types, allowing us to verify that participants understood the instruction. Trial types were presented in random order. Further details of the stimuli, procedure, and apparatus are described in eMethods 1 in the Supplement.

Statistical Analysis

Data were analyzed from approximately March 2020 to March 2021. All statistical tests were 2 tailed (α = .05). Key measures included (1) proportion of responses centered (±15°) around the target direction angle (the direction of motion at the end of the trial), (2) proportion of responses centered (±15°) around the initial motion direction, and (3) lapse rate (responses reflecting random guesses or attentional lapses). Details on how these measures were quantified are provided in the Data Analysis subsection of eMethods 1 in the Supplement.

For the no-change trials, each measure was analyzed in a 2-way analysis of variance with factors of group (PSZ vs HC) and duration of RDK presentation (500 or 1000 milliseconds). For the change trials, which were always 1000 milliseconds in duration, between-group differences were assessed using 3 independent groups *t* tests with a Bonferroni-adjusted α level of .016 per test. We used Cohen *d* as a measure of effect size and Pearson correlations to examine relationships among measures. We conducted separate analyses of the 2 samples of participants.

Results

Change Trials (35% Coherence) Response Proportions

A total of 48 participants were included in the PSZ group (31 [65%] male and 17 [35%] female; mean [SD] age, 36.56 [9.76] years) and 36 participants in the HC group (22 [61%] male and 14 [39%] female; mean [SD] age, 35.67 [10.74] years) in the original sample. The independent replication sample included 42 participants in the PSZ group (29 [69%] male and 13 [31%] female; mean [SD] age, 33.98 [11.03] years) and 34 participants in the HC group (20 [59%] male and 14 [41%] female; mean [SD] age, 34.29 [10.44] years). **Figure 2** shows the results from the change trials. The PSZ group had a lower proportion of target responses in the original sample (t = 5.34; P < .001; Cohen

Table. Participant Characteristics

Characteristic	Participant group[a]							
	Original sample				Replication sample[b]			
	PSZ (n = 48)	HC (n = 36)	Statistic	*P* value	PSZ (n = 42)	HC (n = 34)	Statistic	*P* value
Demographics								
Age, y	36.56 (9.76)	35.67 (10.74)	t = 0.40	.69	33.98 (11.03)	34.29 (10.44)	t = 0.20	.85
No. female/male	17/31	14/22	χ^2 = 0.10	.74	13/29	14/20	χ^2 = 0.86	.35
Race and ethnicity, No. Black/White/other[c]	18/24/6	16/16/4	χ^2 = 0.41	.81	14/23/5	5/20/9	χ^2 = 4.83	.09
Educational attainment, y								
Participant	13.54 (2.27)	15.54 (2.32)	t = 3.93	<.001	13.18 (3.16)	15.38 (3.64)	t = 2.82	.006
Maternal	14.24 (2.76)	14.40 (3.58)	t = 0.95	.35	16.24 (1.99)	14.10 (2.25)	t = 1.52	.13
Paternal	13.66 (4.16)	12.89 (5.84)	t = 0.94	.35	15.44 (3.58)	14.83 (2.75)	t = 1.01	.32
Neurocognitive test results								
WRAT-4	97.77 (14.2)	110.37 (14.17)	t = 3.98	.001	103.22 (23.33)	117.11 (9.39)	t = 2.95	<.001
MD								
Processing speed	38.40 (14.64)	54.03 (8.49)	t = 5.58	<.001	42.25 (15.60)	53.86 (16.76)	t = 2.78	.01
Working memory	38.23 (11.09)	51.94 (8.88)	t = 5.96	<.001	42.38 (16.30)	52.79 (13.68)	t = 2.66	.01
Verbal learning	38.34 (9.33)	49.97 (10.25)	t = 5.31	<.001	40.28 (15.41)	48.32 (12.49)	t = 2.20	.03
Visual learning	37.11 (14.35)	47.76 (7.83)	t = 3.92	<.001	NA	NA	NA	NA
Reasoning	43.45 (11.06)	51.85 (8.58)	t = 3.70	<.001	NA	NA	NA	NA
Social cognition	40.17 (12.26)	52.38 (8.58)	t = 4.99	<.001	NA	NA	NA	NA
Attention vigilance	40.04 (12.09)	49.09 (11.74)	t = 4.37	<.001	NA	NA	NA	NA
MCT overall	32.77 (14.4)	50.24 (11.81)	t = 6.87	<.001	NA	NA	NA	NA
Ratings								
PDI								
Conviction	3.25 (0.19)	1.91 (0.30)	t = 24.99	<.001	3.67 (0.87)	1.82 (1.87)	t = 5.26	<.001
Distress	3.00 (0.16)	0.93 (0.14)	t = 61.85	<.001	2.39 (1.10)	1.10 (1.17)	t = 4.49	<.001
Preoccupation	3.01 (0.18)	1.41 (0.20)	t = 38.44	<.001	3.22 (0.74)	1.14 (1.18)	t = 8.67	<.001
Total	11.16 (1.35)	2.15 (0.42)	t = 38.61	<.001	9.47 (6.69)	2.17 (2.63)	t = 5.54	<.001
Clinical ratings								
BPRS								
Positive symptoms	2.3 (1.21)	NA	NA	NA	2.50 (1.24)	1.01 (0.05)	t = 6.33	<.001[d]
Negative symptoms	1.61 (0.52)	NA	NA	NA	1.78 (0.89)	1.02 (0.10)	t = 4.53	<.001[d]
Disorganized symptoms	1.25 (0.27)	NA	NA	NA	1.30 (0.42)	1.01 (0.04)	t = 3.69	<.001[d]
Total score	34.83 (9.38)	NA	NA	NA	35.12 (9.59)	21.29 (1.61)	t = 7.54	<.001[d]
Antipsychotic medication								
CPZ dose equivalent, mg/d	486.53 (403.93)	NA	NA	NA	418.33 (362.12)	NA	NA	NA
Duration of illness, y[e]	14.04 (9.86)	NA	NA	NA	10.15 (10.65)	NA	NA	NA

Abbreviations: BPRS, Brief Psychiatric Rating Scale; CPZ, chlorpromazine equivalent; HC, healthy control; MCT, Measurement and Treatment Research to Improve Cognition in Schizophrenia Consensus Cognitive Battery (MCCB) Composite Total; MD, MCCB cognitive domain; NA, not applicable; PDI, Peters Delusions Inventory; PSZ, schizoaffective disorder; WRAT-4, Wide Range Achievement Test 4.

[a] Unless otherwise indicated, data are expressed as mean (SD).

[b] Fifteen participants in sample 2 (10 HCs and 5 patients with PSZ) were recruited at Yale University, New Haven, Connecticut.

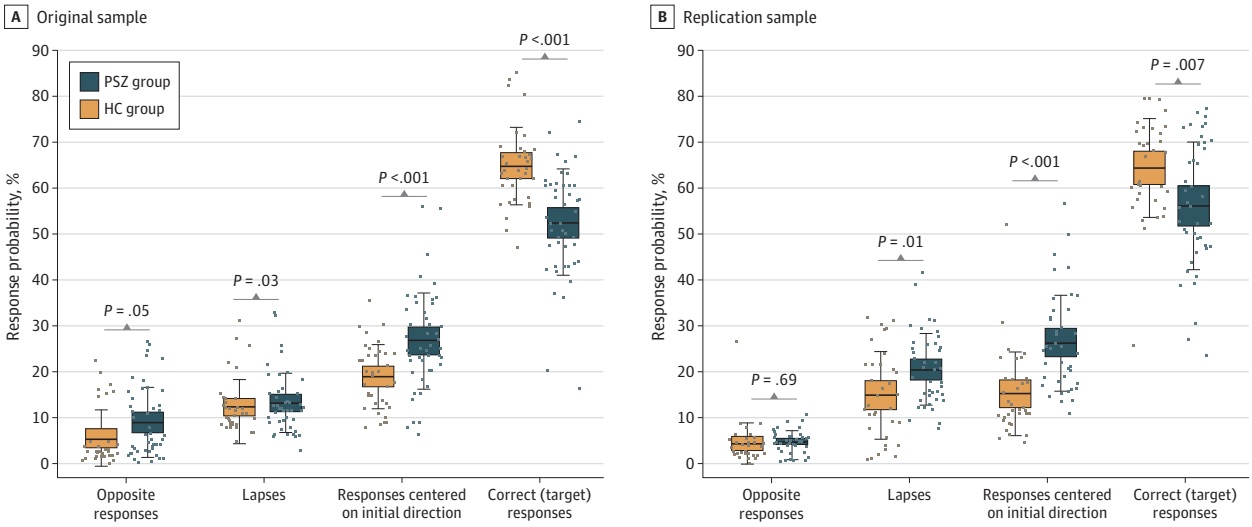
[c] Data were self-reported. Other category includes Asian, Native Hawaiian or Other Pacific Islander, or 1 or more (mixed).

[d] Ratings were available for 39 patients with PSZ and 30 HCs.

[e] This information was available for 30 patients with PSZ in the original sample and 40 patients with PSZ in the replication sample.

d = 1.18) and in the replication sample (t = 2.79; P = .007; Cohen d = 0.64). The PSZ group also had a higher proportion of lapses than the HC group in the replication sample (t = −2.78; P = .01; Cohen d = −0.64), but this difference was not significant at the corrected α level in the original sample (t = −2.16; P = .03; Cohen d = −0.46). The key result was that patients with PSZ often failed to update to the changed direction: they had a higher proportion of responses that were centered on the initial motion direction relative to HC participants in the original sample (PSZ mean, 27.88 [95% CI, 24.19-31.57]; HC mean, 18.86 [95% CI, 16.56-21.16]; t = −3.90; P < .001; Cohen d = −0.83) and in the replication sample (PSZ mean, 26.70 [95% CI, 23.53-29.87]; HC mean, 15.67 [95% CI, 12.61-18.73]; t = −4.84; P < .001; Cohen d = −1.12). Patients with PSZ were also less precise than HC participants (**Figure 3**C and D), quantified as greater mean response error in the original sample

Figure 2. Response Probabilities for Change Trials



To quantify responses for change trials, we derived the proportions of responses centered around the opposite direction from the target (changed) motion direction, lapse rates, proportion of responses centered around initial motion direction, and proportion of responses centered around the target direction (the direction of motion at the end of the trial). Each filled circle represents the proportion for a single participant. These points are layered over a 1.96 SEM (95% CI) in the boxplot and over a 1-SD vertical line, with mean group proportion indicated as a horizontal line within each boxplot. The group with schizophrenia and schizoaffective disorder (PSZ) had a lower proportion of target responses and a higher proportion of lapses than did healthy controls (HCs). Importantly, patients with PSZ often failed to update to the changed direction; they had a higher proportion of responses that were centered on the initial motion direction relative to HCs.

(t = −3.69; P < .001; Cohen d = −0.81) and in the replication sample (t = −2.14; P = .04; Cohen d = −0.49).

We also examined response times separately in trials for which the initial and final motion direction was reported (eMethods 7 in the Supplement). The difference between response times for initial motion reports vs changed motion reports was significantly larger in the PSZ group (t = 4.87; P < .001; Cohen d = 1.11) than in the HC group (t = 3.16; P = .002; Cohen d = 0.67). Specifically, patients with PSZ had faster response times when reporting the direction of initial motion than when reporting the direction of the changed motion, suggesting that the decision was reached more rapidly in patients with PSZ than in HC participants, consistent with greater weighting of initial evidence in PSZ.

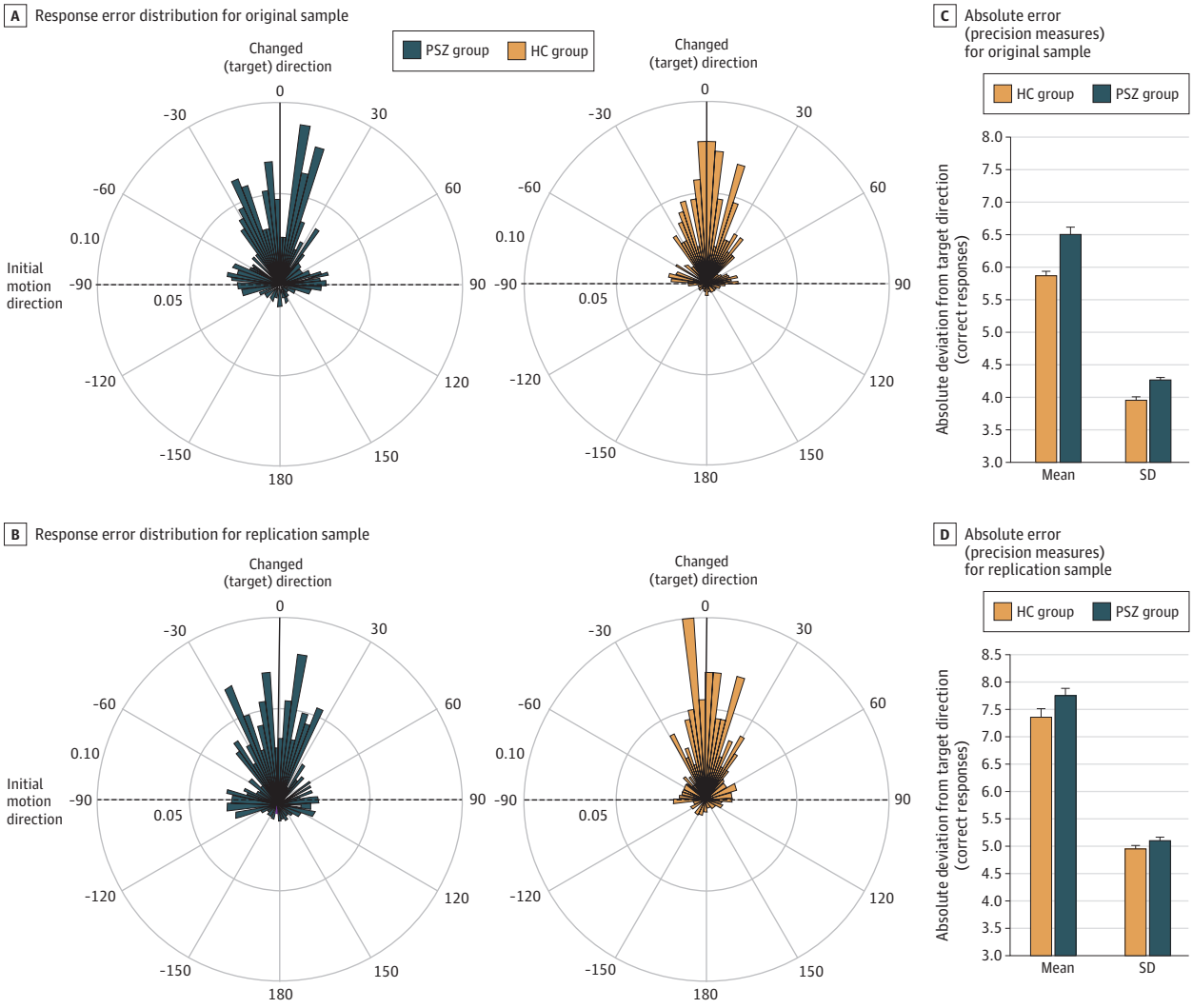
No-Change Trials (35% Coherence) Response Proportions

Statistical analyses of the no-change trials are provided in eMethods 6 in the Supplement, along with reaction time results. Compared with HC participants, patients with PSZ had (1) longer reaction times, (2) a lower proportion of responses centered on the target direction, (3) more lapses, and (4) less precision in their motion reports (eMethods 5 in the Supplement). The group by target duration interaction was not significant in either sample, suggesting that poorer performance in the PSZ group was not attributable to slowing in perceptual processing or impaired working memory. Although patients with PSZ were impaired in their motion processing relative to HC participants in the no-change condition, their deficit relative to HC participants was similar with both the 500- and 1000-millisecond stimulus presentations.

Correlation Analyses

To examine the correlation between updating and symptoms, we used regression models with backward elimination to assess the association between the proportion of initial motion reports and aspects of psychosis, namely the PDI total score, PDI conviction score, BPRS Reality Distortion,[32] disorganization, negative symptom factors, and BPRS total score. In the combined patient sample, BPRS Reality Distortion scores were significantly associated with the accuracy of motion direction reports (t =3.79; P < .001), whereas the associations between mean PDI conviction ratings and the accuracy of motion direction reports were not (t = 1.93; P = .06) (see **Figure 4** for illustration of the univariate correlations for the BPRS Hallucination rating and eTables 1 and 2 in the Supplement for the full set of correlations). In the original sample, both PDI conviction (t = 2.52; P = .02) and BPRS Reality Distortion (t = 4.42; P < .001) scores were significantly associated with the accuracy of motion direction reports. In the replication sample, only the BPRS Reality Distortion score factor remained significant (t = 2.44; P = .02). These analyses all suggest that psychosis severity is associated with failures of perceptual updating. Further, these associations were not simply a consequence of poor performance per se: we did not observe a significant correlation between lapse rate and psychosis severity or between lapse rate and increased initial motion reports, nor did we observe any negative correlation between accuracy (proportion of responses centered around the target) on the no-change trials and psychosis severity. There were no associations between task measures and duration of illness or medication (see eMethods 8 in the Supplement).

Figure 3. Response Error Distributions and Absolute Error Precision Measures

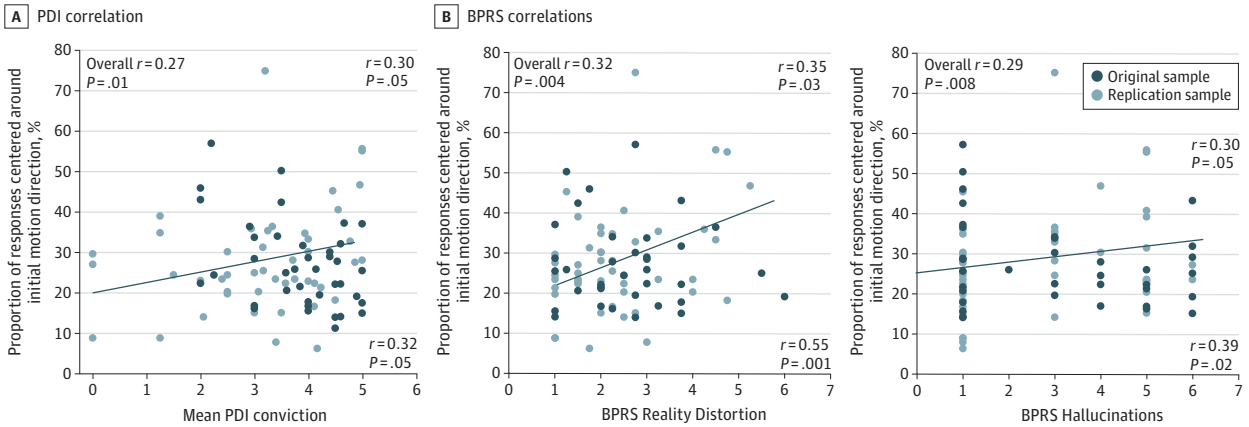


A and B, Behavioral performance was quantified as the response error (the angular difference between the true motion direction and the reported motion direction) for each trial. The rose plots show the distributions of response errors for each group for the motion change trials. The rose plots have been rotated so the target (changed) motion direction appears at the top (12 o'clock) position, whereas the initial motion position appears 90° to the left or right (9 o'clock and 3 o'clock positions, respectively). Most response errors were clustered around 0°, with smaller clusters around the initial motion direction (±90°). C and D, To quantify precision of responses, the absolute mean error around accurate trials (ie, trials that were marked to be centered around changed/target motion direction) was derived. The bar plots represent mean response errors and SDs. For responses that were centered around the correct (changed) motion direction, in both samples, the absolute magnitude of response error was larger in patients with schizophrenia or schizoaffective disorder (PSZ) than healthy control (HC) participants. The variability (SD) of this response error was not significantly different between groups.

100% Coherence Trials

Results from 100% coherence trials demonstrate that all participants in both groups understood the instruction to report the final direction of motion: 70% to 80% of responses in the original sample (PSZ group mean, 70.07 [95% CI, 65.4-74.7]; HC group mean, 76.71 [95% CI, 71.3-82.1]) and in the replication sample (PSZ group mean, 73.66 [95% CI, 69.8-77.5]; HC group mean, 79.82 [95% CI, 76.5-83.1]) were centered around the changed direction in the HC and PSZ groups, respectively (see eMethods 4 in the Supplement; above chance at statistically significant levels, $P < .01$ for original and replication samples in the PSZ and HC groups). Across both samples, the proportion of responses centered around initial motion direction was zero in most participants (59 of 70 HCs and 72 of 90 patients with PSZ). To ensure that the results in the 35% condition did not reflect a failure to understand the task or a general impairment in reporting the second of 2 stimulus intervals, we performed an additional analysis in which we only included this subset of participants who made 0 errors in the 100% change condition. Critically, as with the full set of participants, patients with PSZ had a higher proportion of responses centered on the initial motion direction relative to HC participants in the original sample ($t = -4.32$; $P < .001$; Cohen $d = -1.03$) and the replication sample ($t = -4.99$; $P < .001$; Cohen $d = -1.29$).

Figure 4. Correlation of Responses



A, Scatterplot for Peters Delusions Inventory (PDI) illustrates the correlation between the proportion of responses around initial motion direction and the PDI conviction measure (which captures strength of delusional beliefs) in patients with schizophrenia or schizoaffective disorder (PSZ) from both samples. B, Scatterplots for the Brief Psychiatric Rating Scale (BPRS) illustrate the correlation between the proportion of responses around initial motion direction and the BPRS Reality Distortion scores in patients with PSZ from both samples. The BPRS Reality Distortion measure (left) captures individual symptoms such as grandiosity, suspiciousness, hallucinations, and unusual thought content. This scatterplot demonstrates that the severity of psychotic symptoms is associated with a tendency to overweight initial information. We also observed that the univariate correlation between the proportion of responses around initial motion direction and the BPRS Hallucinations score (right) was significant in both PSZ samples.

Discussion

In this case-control study, patients with PSZ exhibited failures of perceptual updating, even in a low-level task, and these failures were correlated with psychotic symptoms. These findings were replicated in a second sample including participants tested by different researchers at a different site, enhancing confidence in the generalizability of our results. Critically, patients with PSZ were more likely than HCs to report the direction of initial motion on the motion change trials, failing to update their perceptual representations in light of new evidence. Further, this failure to update was associated with the severity of positive symptoms. Moreover, PSZ performance on 100% coherence trials indicated that this effect was unlikely to be due to poor comprehension of instructions. Overall, consistent with prior literature on motion processing, patients with PSZ exhibited poorer integration of spatially distributed motion signals than did HC participants,[33] suggesting impairments in early visual processing. Previous studies have found that cortical processing in the early visual system, especially the magnocellular pathway, is deficient in schizophrenia.[34] However, there is no reason to expect that these deficits would produce a bias to report the initial motion direction.

These results demonstrate that even in a relatively simple perceptual paradigm, patients with PSZ fail to update their perceptual beliefs when faced with new information, echoing previous observations.[35] The fact that the degree of updating failure in this simple perceptual task was correlated with positive symptoms and degree of delusional conviction suggests that the severity of psychosis may reflect a fundamental alteration of basic perceptual and cognitive processes.

Moreover, the RDK paradigm is widely used in animal models, providing a potential tool to be used in translational treatment development research guided by a mechanistic model of symptom formation. We know of no animal studies using motion-change trials examined in the present study, but future work could include this manipulation. Methodological advances in human neurophysiological investigations[36,37] and computational work[38] have enabled progress in understanding of the neural underpinnings of decision-making that both complement and expand the knowledge gained through invasive recordings in animals. The possibility of integrating findings from noninvasive electroencephalographic and/or magnetoencephalographic recordings with preclinical research could potentially lead to the identification of pathophysiological mechanisms and novel treatment targets.

The present findings are consistent with hierarchical bayesian predictive coding accounts of psychosis.[4,39,40] These accounts propose that perception is a form of inference, combining prior beliefs and sensory evidence weighted by their reliability or inverse variance. Further, these accounts have emphasized that prior knowledge at low and high hierarchical levels may be differentially affected in psychotic illness. Previous studies have examined perceptual inference deficits in patients with PSZ through complex cognitive tasks that tap higher-level reasoning. Multiple experiments suggest that prior beliefs may be overweighted, particularly in individuals with severe psychotic symptoms.[35,41-44] Such prior overweighting manifests in our data as an overreliance on the initial motion direction and the failure to update during some of the motion change trials. To our knowledge, our study is the first to observe behavior resembling analytic reasoning in a low-level task that does not require extensive learning, overt strategizing or deliberation, or integration of multiple cues or sources of information.

Our findings are also consistent with an "all-or-none" style of updating. Nassar et al,[45] using a probabilistic reinforcement learning task, reported that patients with PSZ showed an all-or-none style of updating. That is, patients with PSZ failed to update on many trials when they experienced a prediction error, whereas on other trials, they fully updated based on a single instance of an unexpected outcome. Although our task did not involve learning, as did that of Nassar et al,[45] the tendency toward all-or-none updating appears to describe our results as well: in some change trials, patients with PSZ reported the final direction (all), but in others they reported the initial direction (none) (Figure 2A).

In another study, Schmack et al[44] reported a failure of perceptual maintenance across a short delay (all updating) and an inordinate influence of instructed beliefs on perceptual inference (no updating). Both phenomena correlated with PDI conviction scores, as did the updating failures in our data set. Taken together, the findings across studies are not consistent with a general tendency to overweight or underweight prior knowledge. Instead, we suggest that the illness compromises bayesian inference more generally, relating perhaps to perturbed weighting of data streams by their reliability and compensatory reliance on other sources of information when drawing conclusions.[46] Our results stand in contrast to those of Valton et al,[47] who used a motion direction detection paradigm with stimuli presented at a near-individualized contrast threshold. In their study, 1 motion direction was presented more frequently, which created a reporting bias, but this bias was similar in patients and HC participants, indicating equivalent use of prior evidence. We suspect that the use of near-threshold stimuli by Valton et al may have resulted in their patients adopting a more conservative response bias, which would explain why they did not observe the same use of prior knowledge that we observed in our study.

An alternate explanation in our study might be that patients with PSZ are more prone to report the initial motion direction because they have slowed processing speed and simply did not have enough time to process the second motion direction. This explanation is unlikely, however, because there was no group by duration interaction effect on motion accuracy in the no-change trials. Furthermore, slowing would favor reporting the second motion direction, because the first motion direction was masked by the second motion direction. Similarly, increased reports of initial motion direction are unlikely to be attributable to lapses of attention. Our modeling approach provided an estimate of lapse rate (eMethods 2 and eMethods 3 in the Supplement) wherein the lapse parameter was indeed higher in patients. However, it is difficult to consider an accurate report of the initial direction of motion as reflecting a random response that might arise from an attentional lapse. However, lapses would lead to random responses, not an accurate report of the initial direction of motion. Furthermore, our task did not require participants to remember the initial direction or to detect changes in direction per se, so the task did not put any more demand on white matter than more typical perceptual tasks. It is also noteworthy that the direction of the effect is the opposite of what would be expected based on reduced white matter capacity in PSZ: although patients with PSZ have reduced white matter capacity, they were more likely than HCs to retain the motion direction from the first interval.

Another possibility that has emerged from perceptual decision-making studies[48-51] is that patients manifested temporal integration problems, with an exaggeration of the normal tendency to make responses biased toward information presented earlier in time, as shown by Kalisvaart et al[52] in an experiment using very briefly presented sequential stimuli. Given the large differences in experimental design, it seems unlikely that the information integration observed by Kalisvaart et al[52] can explain increased reports of initial motion direction seen herein. Further study is needed to investigate the association between temporal integration and bayesian models of perception.

Limitations

A major limitation of this study is that we were only able to account for a limited amount of variance in psychosis severity. This limitation is shared with nearly all other behavioral studies reporting on correlates of psychosis.

Conclusions

The results of this case-control study—which were fully replicated in a second sample—suggest that the RDK paradigm taps an important mechanism for understanding psychotic symptoms. Although our ability to account for variance in psychosis severity was limited, these data suggest that the processes involved in accumulating, evaluating, and updating evidence are reliably implicated in psychosis and that these processes can be assessed using simple perceptual judgment paradigms.

ARTICLE INFORMATION

Accepted for Publication: October 13, 2021.

Published Online: December 1, 2021. doi:10.1001/jamapsychiatry.2021.3482

Author Contributions: Drs Bansal and Gold had full access to all the data in the study and take responsibility for the integrity of the data and the accuracy of the data analysis.
Concept and design: Bansal, Bae, Corlett, Luck, Gold.
Acquisition, analysis, or interpretation of data: All authors.
Drafting of the manuscript: Bansal, Corlett, Gold.
Critical revision of the manuscript for important intellectual content: All authors.
Statistical analysis: Bansal, Bae, Corlett.
Obtained funding: Waltz, Erickson, Corlett, Luck, Gold.
Administrative, technical, or material support: Bae, Robinson, Waltz, Erickson, Corlett, Gold.
Supervision: Gold.

Conflict of Interest Disclosures: Dr Corlett reported receiving grants from the National Institutes of Health (NIH) during the conduct of the study. Dr Luck reported receiving grants from the NIH during the conduct of the study. Dr Gold reported receiving grants from the NIH during the conduct of the study; personal fees from Acadia Pharmaceuticals Inc outside the submitted work; and royalty payments from the Brief Assessment of Cognition in Schizophrenia. No other disclosures were reported.

Funding/Support: This work was supported by grants R01MH065034 and R01MH112887 from the NIH and a Brain and Behavior Research Foundation Young Investigator Award.

Role of the Funder/Sponsor: The funding organizations had no role in the design and conduct of the study; collection, management, analysis, and interpretation of the data; preparation, review, or approval of the manuscript; and decision to submit the manuscript for publication.

Additional Contributions: Jenna Dutterer, BS, Sharon August, MA, and Leeka Hubzin, MA, University of Maryland, Baltimore, assisted with the study; they did not receive any additional compensation beyond their university pay for this assistance.

REFERENCES

1. von Helmholtz H. *Hanbuch der physiologischen Optik*. Leopold Voss; 1867.

2. Fletcher PC, Frith CD. Perceiving is believing: a bayesian approach to explaining the positive symptoms of schizophrenia. *Nat Rev Neurosci*. 2009;10(1):48-58. doi:10.1038/nrn2536

3. Powers AR III, Kelley M, Corlett PR. Hallucinations as top-down effects on perception. *Biol Psychiatry Cogn Neurosci Neuroimaging*. 2016;1(5):393-400. doi:10.1016/j.bpsc.2016.04.003

4. Adams RA, Stephan KE, Brown HR, Frith CD, Friston KJ. The computational anatomy of psychosis. *Front Psychiatry*. 2013;4:47. doi:10.3389/fpsyt.2013.00047

5. Moritz S, Woodward TS, Hausmann D. Incautious reasoning as a pathogenetic factor for the development of psychotic symptoms in schizophrenia. *Schizophr Bull*. 2006;32(2):327-331. doi:10.1093/schbul/sbj034

6. Lawrence E, Peters E. Reasoning in believers in the paranormal. *J Nerv Ment Dis*. 2004;192(11):727-733. doi:10.1097/01.nmd.0000144691.22135.d0

7. Balzan R, Delfabbro P, Galletly C, Woodward T. Confirmation biases across the psychosis continuum: the contribution of hypersalient evidence-hypothesis matches. *Br J Clin Psychol*. 2013;52(1):53-69. doi:10.1111/bjc.12000

8. Bronstein MV, Pennycook G, Joormann J, Corlett PR, Cannon TD. Dual-process theory, conflict processing, and delusional belief. *Clin Psychol Rev*. 2019;72:101748. doi:10.1016/j.cpr.2019.101748

9. Ashinoff BK, Singletary NM, Baker SC, Horga G. Rethinking delusions: a selective review of delusion research through a computational lens. Published online March 3, 2021. *Schizophr Res*. doi:10.1016/j.schres.2021.01.023

10. Ward T, Garety PA. Fast and slow thinking in distressing delusions: a review of the literature and implications for targeted therapy. *Schizophr Res*. 2019;203:80-87. doi:10.1016/j.schres.2017.08.045

11. Woodward TS, Moritz S, Cuttler C, Whitman JC. The contribution of a cognitive bias against disconfirmatory evidence (BADE) to delusions in schizophrenia. *J Clin Exp Neuropsychol*. 2006;28(4):605-617. doi:10.1080/13803390590949511

12. Sanford N, Veckenstedt R, Moritz S, Balzan RP, Woodward TS. Impaired integration of disambiguating evidence in delusional schizophrenia patients. *Psychol Med*. 2014;44(13):2729-2738. doi:10.1017/S0033291714000397

13. Eisenacher S, Zink M. Holding on to false beliefs: the bias against disconfirmatory evidence over the course of psychosis. *J Behav Ther Exp Psychiatry*. 2017;56:79-89. doi:10.1016/j.jbtep.2016.08.015

14. Woodward TS, Buchy L, Moritz S, Liotti M. A bias against disconfirmatory evidence is associated with delusion proneness in a nonclinical sample. *Schizophr Bull*. 2007;33(4):1023-1028. doi:10.1093/schbul/sbm013

15. Huq SF, Garety PA, Hemsley DR. Probabilistic judgements in deluded and non-deluded subjects. *Q J Exp Psychol A*. 1988;40(4):801-812. doi:10.1080/14640748808402300

16. Moritz S, Woodward TS. Jumping to conclusions in delusional and non-delusional schizophrenic patients. *Br J Clin Psychol*. 2005;44(pt 2):193-207. doi:10.1348/014466505X35678

17. So SH, Siu NY, Wong HL, Chan W, Garety PA. "Jumping to conclusions" data-gathering bias in psychosis and other psychiatric disorders: two meta-analyses of comparisons between patients and healthy individuals. *Clin Psychol Rev*. 2016;46:151-167. doi:10.1016/j.cpr.2016.05.001

18. Speechley WJ, Whitman JC, Woodward TS. The contribution of hypersalience to the "jumping to conclusions" bias associated with delusions in schizophrenia. *J Psychiatry Neurosci*. 2010;35(1):7-17. doi:10.1503/jpn.090025

19. Young HF, Bentall RP. Probabilistic reasoning in deluded, depressed and normal subjects: effects of task difficulty and meaningful versus non-meaningful material. *Psychol Med*. 1997;27(2):455-465. doi:10.1017/S0033291796004540

20. Dudley R, Taylor P, Wickham S, Hutton P. Psychosis, delusions, and the "jumping to conclusions" reasoning bias: a systematic review and meta-analysis. *Schizophr Bull*. 2016;42(3):652-665. doi:10.1093/schbul/sbv150

21. Fine C, Gardner M, Craigie J, Gold I. Hopping, skipping or jumping to conclusions? clarifying the role of the JTC bias in delusions. *Cogn Neuropsychiatry*. 2007;12(1):46-77. doi:10.1080/13546800600750597

22. Tripoli G, Quattrone D, Ferraro L, et al; EU-GEI WP2 Group. Jumping to conclusions, general intelligence, and psychosis liability: findings from the multi-centre EU-GEI case-control study. *Psychol Med*. 2021;51(4):623-633. doi:10.1017/S003329171900357X

23. Falcone MA, Murray RM, Wiffen BDR, et al. Jumping to conclusions, neuropsychological functioning, and delusional beliefs in first episode psychosis. *Schizophr Bull*. 2015;41(2):411-418. doi:10.1093/schbul/sbu104

24. Baker SC, Konova AB, Daw ND, Horga G. A distinct inferential mechanism for delusions in schizophrenia. *Brain*. 2019;142(6):1797-1812. doi:10.1093/brain/awz051

25. Hanks TD, Summerfield C. Perceptual decision making in rodents, monkeys, and humans. *Neuron*. 2017;93(1):15-31. doi:10.1016/j.neuron.2016.12.003

26. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders.* 4th ed, text revision. American Psychiatric Association; 2000.

27. Neuchterlein KH, Green MF. *Measurement and Treatment Research to Improve Cognition in Schizophrenia (MATRICS) Consensus Cognitive Battery.* Psychological Assessment Resources; 2008.

28. Robertson GJ, Wilkinson GS. *Wide Range Achievement Test 4 Professional Manual*. Psychological Assessment Resources; 2006.

29. Overall JE, Gorham DR. The Brief Psychiatric Rating Scale. *Psychol Rep*. 1962;10:799-812. doi:10.2466/pr0.1962.10.3.799

30. Peters E, Joseph S, Day S, Garety P. Measuring delusional ideation: the 21-item Peters et al Delusions Inventory (PDI). *Schizophr Bull*. 2004;30(4):1005-1022. doi:10.1093/oxfordjournals.schbul.a007116

31. Roitman JD, Shadlen MN. Response of neurons in the lateral intraparietal area during a combined visual discrimination reaction time task. *J Neurosci*. 2002;22(21):9475-9489. doi:10.1523/JNEUROSCI.22-21-09475.2002

32. McMahon RP, Kelly DL, Kreyenbuhl J, Kirkpatrick B, Love RC, Conley RR. Novel factor-based symptom scores in treatment resistant schizophrenia: implications for clinical trials. *Neuropsychopharmacology*. 2002;26(4):537-545. doi:10.1016/S0893-133X(01)00387-6

33. Chen Y. Abnormal visual motion processing in schizophrenia: a review of research progress. *Schizophr Bull*. 2011;37(4):709-715. doi:10.1093/schbul/sbr020

34. Butler PD, Martinez A, Foxe JJ, et al. Subcortical visual dysfunction in schizophrenia drives secondary cortical impairments. *Brain*. 2007;130(pt 2):417-430. doi:10.1093/brain/awl233

35. Powers AR III, Mathys C, Corlett PR. Pavlovian conditioning-induced hallucinations result from overweighting of perceptual priors. *Science*. 2017;357(6351):596-600. doi:10.1126/science.aan3458

36. O'Connell RG, Kelly SP. Neurophysiology of human perceptual decision-making. *Annu Rev Neurosci*. 2021;44:495-516. doi:10.1146/annurev-neuro-092019-100200

37. O'Connell RG, Shadlen MN, Wong-Lin K, Kelly SP. Bridging neural and computational viewpoints on perceptual decision-making. *Trends Neurosci*. 2018;41(11):838-852. doi:10.1016/j.tins.2018.06.005

38. Huys QJ, Maia TV, Frank MJ. Computational psychiatry as a bridge from neuroscience to clinical applications. *Nat Neurosci*. 2016;19(3):404-413. doi:10.1038/nn.4238

39. Sterzer P, Adams RA, Fletcher P, et al. The predictive coding account of psychosis. *Biol Psychiatry*. 2018;84(9):634-643. doi:10.1016/j.biopsych.2018.05.015

40. Corlett PR, Horga G, Fletcher PC, Alderson-Day B, Schmack K, Powers AR III. Hallucinations and strong priors. *Trends Cogn Sci*. 2019;23(2):114-127. doi:10.1016/j.tics.2018.12.001

41. Teufel C, Subramaniam N, Dobler V, et al. Shift toward prior knowledge confers a perceptual advantage in early psychosis and psychosis-prone healthy individuals. *Proc Natl Acad Sci U S A*. 2015;112(43):13401-13406. doi:10.1073/pnas.1503916112

42. Haarsma J, Knolle F, Griffin JD, et al; NSPN Consortium. Influence of prior beliefs on perception in early psychosis: effects of illness stage and hierarchical level of belief. *J Abnorm Psychol*. 2020;129(6):581-598. doi:10.1037/abn0000494

43. Davies DJ, Teufel C, Fletcher PC. Anomalous perceptions and beliefs are associated with shifts toward different types of prior knowledge in perceptual inference. *Schizophr Bull*. 2018;44(6):1245-1253. doi:10.1093/schbul/sbx177

44. Schmack K, Gòmez-Carrillo de Castro A, Rothkirch M, et al. Delusions and the role of beliefs

in perceptual inference. *J Neurosci*. 2013;33(34):13701-13712. doi:10.1523/JNEUROSCI.1778-13.2013

45. Nassar MR, Waltz JA, Albrecht MA, Gold JM, Frank MJ. All or nothing belief updating in patients with schizophrenia reduces precision and flexibility of beliefs. *Brain*. 2021;144(3):1013-1029. doi:10.1093/brain/awaa453

46. Leptourgos P, Corlett PR. Embodied predictions, agency, and psychosis. *Front Big Data*. 2020;3:27. doi:10.3389/fdata.2020.00027

47. Valton V, Karvelis P, Richards KL, Seitz AR, Lawrie SM, Seriès P. Acquisition of visual priors and induced hallucinations in chronic schizophrenia. *Brain*. 2019;142(8):2523-2537. doi:10.1093/brain/awz171

48. Ludwig CJ, Gilchrist ID, McSorley E, Baddeley RJ. The temporal impulse response underlying saccadic decisions. *J Neurosci*. 2005;25(43):9907-9912. doi:10.1523/JNEUROSCI.2197-05.2005

49. Huk AC, Shadlen MN. Neural activity in macaque parietal cortex reflects temporal integration of visual motion signals during perceptual decision making. *J Neurosci*. 2005;25(45):10420-10436. doi:10.1523/JNEUROSCI.4684-04.2005

50. Kiani R, Hanks TD, Shadlen MN. Bounded integration in parietal cortex underlies decisions even when viewing duration is dictated by the environment. *J Neurosci*. 2008;28(12):3017-3029. doi:10.1523/JNEUROSCI.4761-07.2008

51. Tsetsos K, Chater N, Usher M. Salience driven value integration explains decision biases and preference reversal. *Proc Natl Acad Sci U S A*. 2012;109(24):9659-9664. doi:10.1073/pnas.1119569109

52. Kalisvaart JP, Noest AJ, van den Berg AV, Goossens J. Saccade target selection relies on feedback competitive signal integration. *J Neurosci*. 2013;33(29):12077-12089. doi:10.1523/JNEUROSCI.2613-12.2013