Psychotic Experiences in Schizophrenia and Sensitivity to Sensory Evidence

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Perceptual inference depends on an optimal integration of current sensory evidence with prior beliefs about the environment. Alterations of this process have been related to the emergence of positive symptoms in schizophrenia. However, it has remained unclear whether delusions and hallucinations arise from an increased or decreased weighting of prior beliefs relative to sensory evidence. To investigate the relation of this prior-to-likelihood ratio to positive symptoms in schizophrenia, we devised a novel experimental paradigm which gradually manipulates perceptually ambiguous visual stimuli by disambiguating stimulus information. As a proxy for likelihood precision, we assessed the sensitivity of individual participants to sensory evidence. As a surrogate for the precision of prior beliefs in perceptual stability, we measured phase duration in ambiguity. Relative to healthy controls, patients with schizophrenia showed a stronger increment in congruent perceptual states for increasing levels of disambiguating stimulus evidence. Sensitivity to sensory evidence correlated positively with the individual patients' severity of perceptual anomalies and hallucinations. Moreover, the severity of such experiences correlated negatively with phase duration. Our results indicate that perceptual anomalies and hallucinations are associated with a shift of perceptual inference toward sensory evidence and away from prior beliefs. This reduced prior-to-likelihood ratio in sensory processing may contribute to the phenomenon of aberrant salience, which has been suggested to give rise to the false inferences underlying psychotic experiences.

*Key words:* psychosis/Bayesian perceptual inference/predictive coding/bistable perception

Introduction

When perceiving our surroundings, we are confined to inherently noisy and ambiguous sensory representations of the environment. However, conscious experience usually provides us with an unequivocal impression of our world. According to Bayesian theories,[1–3] our brain bridges this gap by actively employing beliefs to interpret sensory information and forms a hypothesis (or *posterior* probability distribution, figure 1A) about the cause of current sensory data.[4] Along this line of thought, conscious experience represents a *controlled hallucination*, that is concurrently being shaped by internally generated beliefs (*prior* distributions) and constrained by external sensory information (the *likelihood* distribution).[5]

Alterations in the relative weighting (or *precision*[6]) of prior and likelihood may lead to false (or dysfunctional) inferences[7–9]: If prior precision is overestimated relative to the likelihood (increased prior-to-likelihood ratio, figure 1B), inference will be driven too strongly by prior beliefs and violations of prior beliefs by sensory data (ie, *prediction errors*) will be overly attenuated. In contrast, a decreased prior-to-likelihood ratio (figure 2C) will lead to a stronger weighting of the sensory data, thus instigating aberrant prediction errors.

Previous work has discussed both increases and decreases of the prior-to-likelihood ratio in relation to cognitive and perceptual anomalies in psychosis-prone individuals and patients with schizophrenia (Scz, for review, see[10] and[11]). Interestingly, delusions have often been related to a decreased prior-to-likelihood ratio,[8,12–16] whereas studies on hallucinations have pointed to an increased prior-to-likelihood ratio.[17–22] As it seems unlikely that delusions and hallucinations, 2 frequently co-occurring symptom domains, should be due to opposing alterations in inference, it was recently proposed that these apparently contradictory findings may be reconciled within the framework of hierarchical predictive coding[1,2,23]: The prior-to-likelihood ratio may indeed be generally reduced at low levels, eg, in early sensory areas, leading to aberrant salience of sensory stimuli and the emergence of delusions.[24,25]
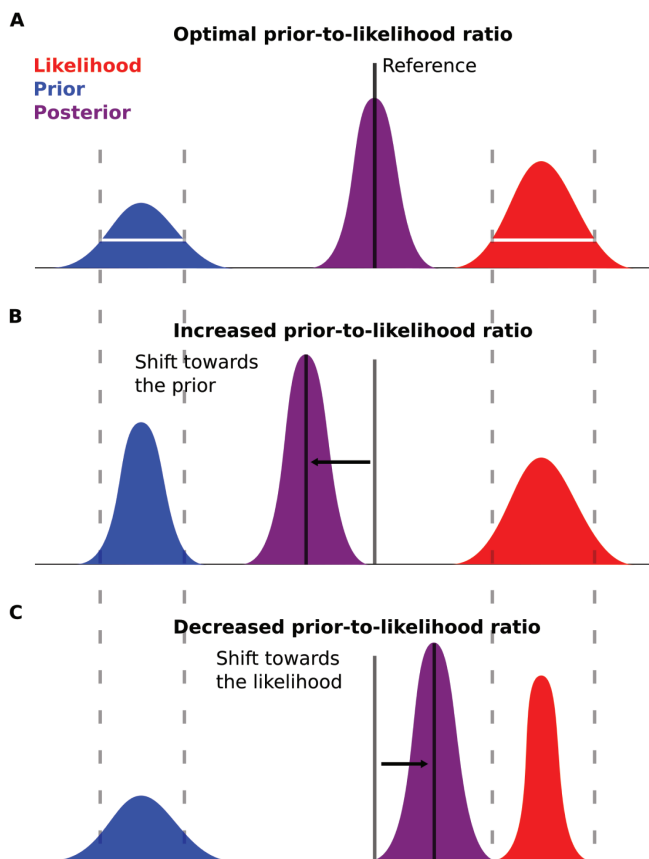
Fig. 1. The prior-to-likelihood ratio in Bayesian perceptual inference. Perceptual inference depends on the ratio of prior and likelihood precision. (**A**) Here, we depict a reference scenario with optimal precision estimates (Gaussian distributions, variance in white, mean of the posterior in black). (**B**) Changes in these estimates of precision may lead to alterations in perception. In case of an overestimation of prior precision and/ or underestimation of likelihood precision, the posterior is shifted toward the prior. (**C**) By analogy, an overestimation of likelihood precision and/or underestimation of prior precision is associated with a shift of the posterior toward the likelihood.

In contrast, higher-level priors may become overly precise in an attempt to compensate for aberrant salience and contribute to the emergence of hallucinations.[10,11,26]

In the present study, we tested the hypothesis that psychotic experiences in Scz are related to a decreased prior-to-likelihood ratio at low hierarchical levels. We asked whether the precision of the likelihood mapping between the causes of sensations and the sensory consequences was elevated in Scz relative to healthy controls. This precision is often referred to as sensory precision, where an elevated precision is sometimes attributed to a failure of sensory attenuation. Moreover, we tested whether such a stronger weighting of sensory evidence is associated with the experience of delusions, hallucinations, or both.

We developed a novel experimental paradigm based on bistable perception, ie, the spontaneous alternation between 2 perceptual states that occurs when sensory information is ambiguous.[27] Predictive coding posits that the dynamics

of bistability reflect the 2 components of the prior-to-likelihood ratio[28,29]: The current perceptual state represents the best hypothesis (ie, the prior) about the cause of sensory information (ie, the likelihood). Due to ambiguity, neither of the 2 mutually exclusive perceptual hypotheses can fully account for the sensory data. Hence, a prediction error accumulates and eventually leads to a perceptual transition.

Here, we induced the phenomenon of *graded ambiguity* by parametrically manipulating the available sensory evidence for the 2 alternative perceptual hypotheses of an ambiguous Lissajous figure (see figure 2A and Supplementary Video 1). When a perceptual hypothesis is congruent to disambiguating stimulus evidence, prediction errors should be reduced and perceptual transitions to the incongruent perceptual states less likely. Incongruence, in turn, should lead to enhanced prediction errors and increased probability of a transition to the congruent perceptual state. In sum, the probability of perceptual states congruent with disambiguating stimulus evidence should vary with the individual participants' sensitivity to sensory evidence. Thus, it serves as a proxy for the prior-to-likelihood ratio.

We studied the sensitivity to disambiguating stimulus evidence in patients with paranoid Scz and a matched control group. Under the assumption of a decreased prior-to-likelihood ratio in psychosis, we expected an increased sensitivity to disambiguating stimulus evidence in patients with Scz. We furthermore hypothesized a positive correlation of sensitivity to disambiguating stimulus evidence with the severity of delusions and hallucinations.

Methods

Participants

We excluded 1 control due to impaired stereovision, 3 controls due to elevated scores for Cardiff Anomalous Perception Scale (CAPS) and Peters Delusion Inventory (PDI) (threshold/scores ≥ 3 SDs above the group's mean), 1 control due to reduced frequency of congruent perceptual states (frequency ≤ 3 SDs below the mean computed across groups in any of the conditions D1–D7), and 1 patient who did not complete the experiment. The final sample was matched for gender, age, and handedness (see table 1) and consisted of 23 patients (International Classification of Diseases 10: F20.0, 18 male, age = 37.13 ± 2.42) recruited from in- and out-patient services at Charité Universitétsmedizin Berlin and 23 control participants (17 male, age = 33.57 ± 1.74 y). All participants had (corrected-to-)normal vision, were naive to the purpose of the study, and gave informed, written consent prior to the experiment authorized by the Charité Ethics Committee.

Questionnaires and Clinical Rating

Participants completed the 40-item PDI[30] to quantify delusional ideation[13,14,17,31–33] and the 32-item CAPS[34] to
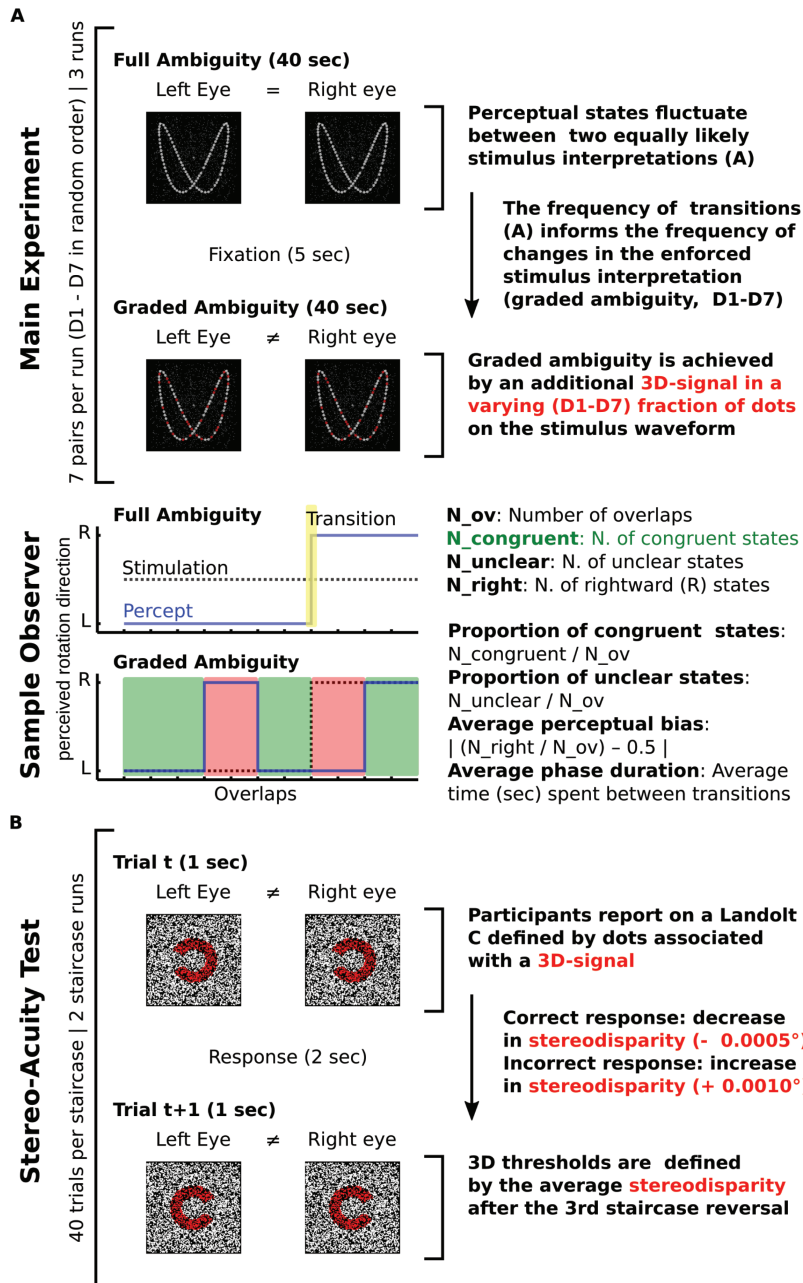
Fig. 2. Behavioral experiment. **(A)** In the main experiment, we measured the individual participants' sensitivity to disambiguating stimulus evidence as a proxy for the prior-to-likelihood ratio. To visualize relevant variables, the lower panel displays typical perceptual responses in an ambiguous block and the corresponding partially disambiguated block. (B) To probe potential differences in stereovision, we determined individual stereo-disparity thresholds in an independent stereoacuity test.

Table 1. Sample Characteristics

Group	N	Female	Smoking	Stat	Age	ED	CAPS	PDI	PANSS: P	N	G	DOI	CPZe
Controls	23	6	10	Mean	33.6	77	6.7	22	NA	NA	NA	NA	NA
				SD	8.4	40	9.2	28	NA	NA	NA	NA	NA
Patients	23	5	15	Mean	37.1	75	65.0	139	18.4	19.4	33	15	190
				SD	11.6	44	50.1	80	6.3	8.2	10	12	172

Note: Patients with Scz scored higher than controls on the PDI (patients: 138.83 ± 16.64 SEM, controls: 21.87 ± 5.75, Welch 2-sample t-test: $T(27) = 6.64$, $P = 3.81 \times 10^{-7}$) and CAPS (patients: 64.96 ± 10.45, controls: CAPS of 6.65 ± 1.91, $T(23) = 5.49$, $P = 1.32 \times 10^{-5}$). One patient received a typical antipsychotic, 18 patients were prescribed an atypical antipsychotic, and 4 were without medication.

measure perceptual anomalies. Reported scores reflect sums over questionnaire subscales. We assessed clinical symptom severity using the Positive and Negative Syndrome Scale (PANSS).[35]

Behavioral Experiments

Apparatus. We presented all stimuli using a mirror stereoscope placed in front of a 98PDF-CRT-Monitor (60 Hz, 1042×768 pixels, 59.50 cm viewing distance, 30.28 pixels per degree visual angle; °) using Psychtoolbox 3[36] and Matlab R2007b (MathWorks).

Main Experiment. The main experiment (figure 2A) assessed the modulation of perceptual states by levels of disambiguating stimulus evidence. In 3 runs (10.52 min each), participants viewed 7 pairs of ambiguous and partially disambiguated versions of a rotating discontinuous Lissajous figure (see Supplementary Video 1) presented in blocks of 40.08 s each, separated by 5 s of fixation. We randomly placed 300 dots (0.05°) on the stimulus waveform ($2.05° \times 2.05°$) defined by the perpendicular intersection of 2 sinusoids [$x(t) = sin(A * t)$ and $y(t) = cos(B * t + \delta)$ with $A = 3$, $B = 6$, and δ increasing from 0 to 2π at 6.80 s per revolution and 6 revolutions per block]. We relocated the dots at a probability of 0.02 per frame. Stimuli were surrounded by rectangular fusion frames and presented on the background of random-dot noise (700 dots of 0.05°, 1.98°/s speed, changes in motion direction at 1 Hz). We displayed a fixation cross in the center of the visible screen (0.10°).

During ambiguous blocks, we presented identical Lissajous figures to the 2 eyes. Participants indicated changes in the perceived direction of rotation by pressing the left (rotation of the front surface to the left, right index finger), right (rotation to the right, right ring finger), or down (unclear direction of rotation, right middle finger) arrow key on a standard USB keyboard.

The indicated direction of rotation in an ambiguous block determined the time-points of changes in sensory evidence in the upcoming disambiguated block. To add additional sensory evidence (graded disambiguation) to the Lissajous figure, we shifted a proportion of the stimulus dots by a δ of 0.02π in the corresponding direction between monocular channels. Crucially, we varied the amount of disambiguating stimulus evidence across 7 conditions (D1: 1.25%, D2: 3.75%, D3: 8.75%, D4: 16.25%, D5: 26.25%, D6: 50.00%, and D7: 100.00% of dots disambiguated). Each condition appeared once per run and in random order. Participants reported changes in the perceived direction of rotation as well as unclear perceptual states.

Stereoacuity. We assessed stereo-disparity thresholds in an independent stereoacuity test (similar to[37], figure 2B). To this end, we presented a number of 5000 dots (each at

0.15°) within a square of $11 \times 11°$. We attached a stereo-disparity signal to dots lying on a Landolt C, ie, a circle (1.37° radius, 2.06° width) with a 90° gap located at the left, top, right, or bottom. Following 5 s of fixation and 1 s of stimulus presentation, participants reported the location of the gap in the Landolt C by pressing the up-, down-, left-, or right-arrow key (response interval = 2 s). Fixation crosses (0.10°) were presented in the center of visible screen.

Participants performed 2 runs of 40 trials each. At each trial, we determined the amount of presented stereo disparity based on the response from the previous trial by a 2-up-1-down staircase procedure (correct response: decrease in the available stereo disparity by 1 step; incorrect response: increase by 2 steps, initial step size: 0.001°, reduction to 0.0005° after first reversal). The initial stereo disparity was 0.0045° in run 1 and 0.0005° in run 2.

Analyses

Main Experiment. For the main experiment, we based our analyses on perceptual transitions reported by the participants. Because perceptual transitions occur at overlapping configurations of the Lissajous figure,[29,38–41] we corrected the timing of each perceptual transition to the time of the overlap preceding the corresponding button press. This decomposed the perceptual time course into a sequence of discrete perceptual states (leftward, rightward, and unclear rotation of the front surface, 3.40 s inter-overlap interval).

As variable-of-interest (see figure 2A), we computed the proportion of congruent perceptual states (ie, perceptual states perceived in congruence with the disambiguating stimulus evidence) for all parametric levels of disambiguation (D1–D7). This variable served as a proxy for the prior-to-likelihood balance during graded ambiguity. In addition, we determined individual perceptual stability in terms of average phase duration (ie, time spent between 2 perceptual transitions). As potential confounds, we computed the probability of unclear perceptual states for all conditions (ambiguity and D1–D7) separately and absolute perceptual bias[42] (ie, the absolute difference between the probability of both perceptual states and chance level) in ambiguous blocks. Within participants, we averaged all dependent variables across runs.

We performed group-level statistics using mixed ANOVA (within-subject factor: levels of disambiguating stimulus evidence D1–D7; between-subject factor: diagnostic group). Given heteroscedasticity between groups for congruent perceptual states (Levene test: $P = .043$), we used a linear mixed-effects (nlme R-package) model. The diagnostic group and disambiguating stimulus evidence defined fixed effects. Individual participants defined random effects. Weights were adjusted to account for unequal variance between groups.

We further fitted a set of functions [linear: $y = a + b * x$; exponential: $y = c * exp(g * x)$; sigmoid: $y = 0.5 + (0.5 - l)/(1 + exp(-(x - m)/n))$] to the proportion of congruent perceptual states across conditions D1–D7. After identifying the exponential fit by means of the highest adjusted R^2, we compared individual growth rates as surrogates for the sensitivity to sensory evidence between groups. Because the number of free parameters (ie, complexity) in these models was fixed, the measure of accuracy can be treated as model evidence (ie, we performed a simple form of model comparison). Due to non-normality (Kolmogorov-Smirnov test: $P < .0001$), we used bootstrapping (R-dabestr[43]) to estimate confidence intervals (CI) for between-group differences in growth rates (see Supplementary Materials 1 for analyses of the linear fit) and perceptual bias.

In Supplementary Materials 2, we provide post hoc simulation analyses to illustrate the relation of our psychophysical approach to the predictive coding model of bistable perception.[29]

Stereo Disparity. We determined stereo-disparity thresholds by computing the average of presented stereo disparity at trials following the third reversal of each run and averaged across runs. Due to non-normality (Kolmogorov-Smirnov test: $P < .0001$), we probed a potential between-group difference by bootstrapping CIs.

Correlative Analyses. Finally, we asked whether individual questionnaire scores (PDI and CAPS; Bonferroni-corrected) correlated with the sensitivity to sensory evidence and average phase duration. In addition, we tested correlations with the PANSS subitems P1 (delusions) and P3 (hallucinations). Control analyses probed potential correlations to perceptual bias, unclear perceptual states, stereoacuity, as well as negative and general PANSS subscales (see Supplementary Materials 1 for median split analyses of CAPS/P3 and complete correlograms). Due to non-normality (Kolmogorov-Smirnov tests $P < .0001$ for all variables), we computed standard Spearman correlations. To correct for potential confounds that may influence performance in the Lissajous task and/or the severity of psychotic experiences, we assessed partial correlation coefficients. Such factors comprised stereoacuity (due to its potential influence on graded ambiguity, see above), the participants' age (due to its impact on bistable perception[44]), as well as the duration of illness and chlorpromazine equivalents as measures of disease severity. To ascertain specificity for the dimensions of psychotic experience, we also included scores on the alternative questionnaire (for correlations with PDI/CAPS), the respective alternative PANSS subitems (for correlations with P1/P3) and PANSS subscales (general and negative).

Results

Main Experiment

The nlme R-package model indicated a main effect of disambiguating stimulus evidence on the fraction of congruent perceptual states [$F(6) = 15.16$, $P = 6.44 \times 10^{-15}$], but no main effect of group [$F(1) = 0.02$, $P = .88$]. Importantly, we observed a significant interaction between diagnostic group and disambiguating stimulus evidence [$F(6) = 2.52$, $P = .02$, see figure 3A]. Mixed ANOVA yielded qualitatively identical results.

The change in the fraction of congruent perceptual states across D1–D7 was best fit by an exponential function (adjusted $R^2 = 0.39 \pm 0.10$, best fit in 70% of Scz patients and 65% of controls) as compared with linear (adjusted $R^2 = 0.38 \pm 0.10$) and sigmoid (adjusted $R^2 = 0.10 \pm 0.10$) functions. Sensitivity to additional sensory evidence as expressed by the growth rate of the exponential function was equal to 0.06 ± 0.01 in patients and 0.02 ± 0.02 in controls. Bootstrapping revealed a borderline significant difference between patients and controls (95% CI = 0.004 to -0.08, see figure 3B). Analysis of the linear fit yielded qualitatively identical results (see Supplementary Materials 1).

Mixed ANOVA did not yield a main effect of group or disambiguating stimulus evidence nor a between-factor interaction for the proportion of unclear perceptual states (patients: 0.01 ± 0.001; controls: 0.004 ± 0.001) or phase duration (patients: 21.25 ± 0.35 s; controls: 21.56 ± 0.36 s; see Supplementary Materials 1). Furthermore, we did not observe a significant between-group difference with regard to perceptual biases in ambiguity (patients: 0.09 ± 0.02, controls: 0.10 ± 0.02, 95% CI = -0.06 to 0.04).

Stereoacuity

Stereo-disparity thresholds amounted to $0.003 \pm 0.001°$ in patients and $0.003 \pm 0.001°$ in controls with no significant between-group difference (95% CI = -0.002 to 0.001).

Correlative Analyses

Within patients, sensitivity to disambiguating stimulus evidence correlated positively with the CAPS ($R = 0.51$, $P = .02$; figure 4). This was corroborated by the respective partial correlation ($R = 0.55$, $P = .03$, see above). Similarly, there was a significant correlation of the sensitivity parameter to PANSS subitem P3 (standard correlation: $R = 0.52$, $P = .01$; partial correlation: $R = 0.52$, $P = .04$). We did not observe a significant association between sensitivity to disambiguating stimulus evidence and PDI (standard correlation: $R = 0.36$, $P = .19$; partial correlation: $R = -0.35$, $P = .19$) or P1 (standard correlation: $R = 0.35$, $P = .11$; partial correlation: $R = 0.07$, $P = .78$). Analyses of the linear fit yielded qualitatively identical results.
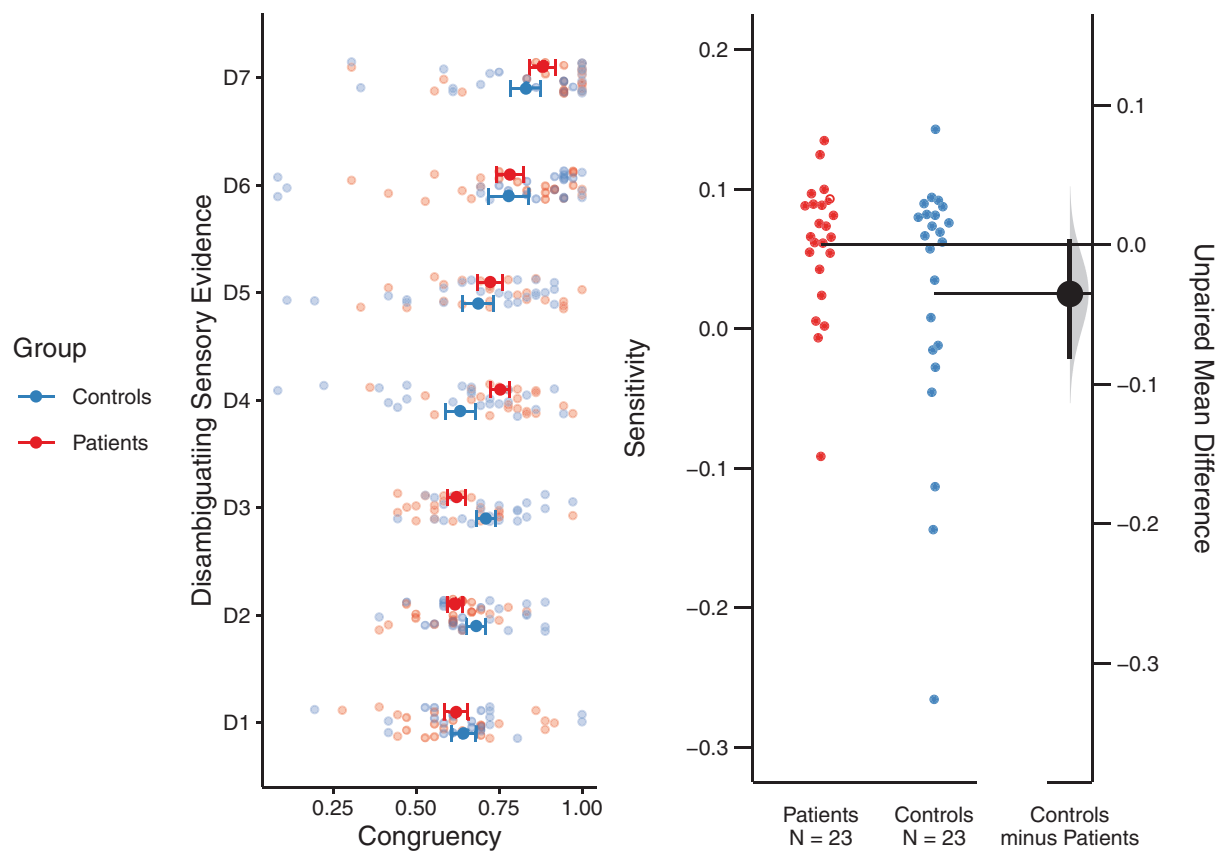
Fig. 3. Sensitivity to disambiguating stimulus evidence. We depict the fraction of congruency between perceptual states and sensory evidence across the levels of disambiguating stimulus evidence (D1–D7, left panel). Error bars represent the respective standard error of the mean. The nlme model yielded a main effect of disambiguating stimulus evidence [$F(6) = 15.16$, $P = 6.44 \times 10^{-15}$], and a significant interaction between the diagnostic group and the disambiguating stimulus evidence [$F(6) = 2.52$, $P = .02$]. The left panel shows the implicit interaction between levels of disambiguating stimulus evidence and diagnostic group: At low levels of disambiguation (D1–D3), controls exhibit a marginally higher proportion of congruent perceptual states. This is reversed for higher levels of disambiguating stimulus evidence (D4–D7), where patients show a greater proportion of congruency. We used the growth rate of individual exponential fits to the fraction of congruent perceptual states to express the individual sensitivities to disambiguating stimulus evidence during graded ambiguity (right panel; horizontal lines point to sample means; vertical line spans over the 95% CI). Bootstrapping revealed a borderline-significant between-group difference (estimated 95% CI = 0.004 to −0.08).

Furthermore, we observed a significant negative correlation of average perceptual phase duration with the CAPS (standard correlation: $R = -0.54$, $P = .01$; partial correlation: $R = -0.64$, $P = .01$) and a trendwise correlation to P3 (standard correlation: $R = -0.39$, $P = .07$; partial correlation: $R = -0.46$, $P = .07$). We did not find a significant association of phase duration to PDI or P1 in standard (PDI: $R = -0.21$, $P = .68$; P1: $R = -0.26$, $P = .23$) or partial correlations (PDI: $R = -0.35$, $P = .19$; P1: $R = -0.21$, $P = .44$).

Confirmatory analyses indicated a significant positive correlation of the sensitivity parameter to the positive and general PANSS subscale ("Positive": $R = 0.5$, $P = .02$; "General": $R = 0.52$, $P = .01$; "Negative": $R = 0.11$, $P = .61$). Interestingly, there were no significant correlations between sensory precision and negative symptoms or signs. CAPS and PDI were highly correlated in patients ($R = 0.76$, $P = 2.81 \times 10^{-5}$) and showed a trend for controls ($R = 0.35$, $P = .1$).

Neither of the 2 questionnaire scores (PDI/CAPS) and PANSS subitems (P1/P3) correlated with perceptual biases, fraction of unclear perceptual states, stereo-disparity thresholds, duration of illness, or chlorpromazine equivalents. Within controls, we did not find any significant correlation between questionnaire scores and the aforementioned variables (see Supplementary Materials 1 for additional correlation analyses and correlograms).

Discussion

In this study, we asked whether the experience of psychotic symptoms is associated with an increased impact of sensory evidence on perceptual inference relative to prior predictions (ie, a reduced prior-to-likelihood ratio at sensory processing levels).

Firstly, Scz patients showed an increased proportion of disambiguation-congruent perceptual states at high levels of stimulus information (D4–D7). At low levels (D1–D3),
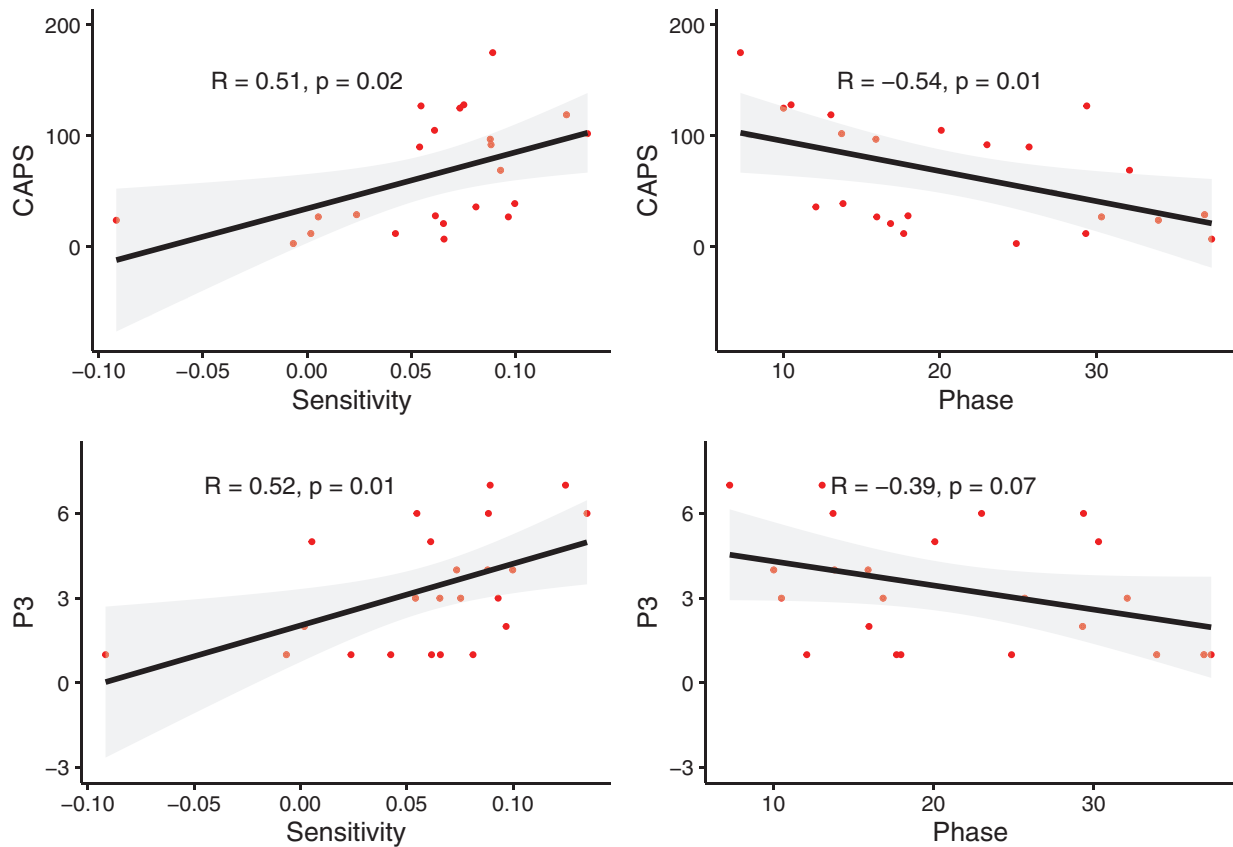
Fig. 4. Individual symptom severity. Here, we depict the individual patients' symptom severity with regard to perceptual anomalies (CAPS, top) and hallucination (P3, bottom) against the sensitivity to stimulus evidence (left) and phase duration (right) alongside regression lines (black) and 95% CI (light gray).

this proportion was similar between groups or even appeared to be reduced in patients (D3). This interaction thus speaks against a global increase in sensitivity to sensory evidence in Scz. Rather, it may suggest that patients show a greater benefit (or *gain*) at increasing levels of stimulus information. Indeed, due to this nonlinearity, these findings defy a simple explanation. Supplementary Materials 2 provides post hoc simulations of this interaction from a predictive coding model of bistable perception.[28,29]

Secondly, we found that the severity of perceptual anomalies and hallucinations correlated *positively* with the sensitivity to disambiguating stimulus evidence and *negatively* with average phase duration in Scz. Predictive coding models of bistable perception[28,29] relate enhanced sensory sensitivity to a shift of precision estimates *toward stimulus representations* (ie, the likelihood). In turn, such models assume that shorter phase durations signal a shift of precision estimates *away from implicit predictions* about perceptual stability (see[29] and Supplementary Materials 2). Through this lens, the two behavioral results, therefore, suggest that hallucinations are related to a decreased prior-to-likelihood ratio at sensory processing levels. At the same time, they contradict the hypothesis that a global shift toward prior precision (ie, an increased prior-to-likelihood ratio) underlies the experience of hallucinations.

These findings align with the "canonical" predictive coding account of Scz,[10] which assumes that psychotic symptoms arise due to a relative shift of inference away from priors and toward sensory evidence.[8] Along these lines, our results reverberate with the association of Scz to a reduced susceptibility to visual illusions,[16] impaired smooth pursuit,[45] and reduced sensory attenuation during force matching.[15,46] While our findings speak for a decrease as opposed to an increase in the prior-to-likelihood ratio, they cannot distinguish between a decrease in prior precision alone, an increase in likelihood precision alone or a combination of the two. Moreover, our results are compatible with alternative algorithms of dynamic belief updating such as circular inference[47,60] and alternative implementational frameworks of bistable perception such as mutual inhibition and adaption models.[48] In this context, differences in the excitation-inhibition balance[49] may lead to weaker inhibition between competing neuronal populations, which could explain why hallucinations correlated with individual characteristics of bistable perception.

Importantly, our results seem to contradict the association of hallucinations to overly precise priors.[19,21,22] However, this apparent discrepancy may be resolved by a differential modulation of the prior-to-likelihood ratio

across levels of the predictive coding hierarchy: Our paradigm targeted the interaction of prior and likelihood at sensory levels. A reduced prior-to-likelihood ratio may elicit the aberrant salience of sensory events.[24,25] This may drive higher levels into an overly strong weighting of priors and entail enhanced top-down influences on perception.[11] Finally, such a compensatory mechanism may trigger hallucinations,[21] thereby *explaining away*[5] aberrant salience at sensory levels.

Albeit strongly correlated with perceptual anomalies and hallucinations, our current findings did not reveal an association of delusional ideation to either sensitivity to sensory evidence or perceptual stability. This discrepancy to previous work[14] may result from differences between the experimental paradigms (Schmack et al.[14] stabilized perceptual states through intermittent presentation,[50] while we used a continuous stimulus). Speculatively, intermittent paradigms may boost perceptual priors and thus be more sensitive toward the relation of perceptual stability and delusions. In turn, manipulating sensory evidence through graded ambiguity may be more apt to detect associations to perceptual abnormalities. To resolve this discrepancy, future work should combine the novel paradigm of graded ambiguity with both intermittent presentation of bistable stimuli[13,14] and manipulations of higher-level beliefs.[33,51–53]

In contrast to our findings, previous research has revealed deficits in binocular depth perception in Scz.[54–57] Our stereoacuity assessment was analogous to the established *Random-Dot test*,[37,55] but estimated perceptual thresholds in a psychophysical staircase. This yielded values in the range commonly reported for stereoacuity.[55] In addition, our study did not show a global reduction in perceptual performance in Scz patients relative to controls. It thus seems less likely that low-level deficits (eg, reduced stereoacuity, contrast sensitivity,[55] or motion intergration[58]) can account for the current findings. Finally, perceptual biases (eg, when perceiving facial expressions[59]) are frequently reported in Scz. In the context of bistable perception, global differences in the probabilities of perceptual alternatives are a common phenomenon.[42] Importantly, this study did not reveal any significant effect of bias, which is thus unlikely to contribute to our results.

In sum, this study associates the experience of psychotic symptoms with an altered integration of prior beliefs and sensory evidence. Our results relate perceptual anomalies and hallucinations to a reduction of the prior-to-likelihood ratio in perception. This provides empirical evidence for the view that predictive processing deficits contribute to the emergence of psychotic symptoms and will enable novel approaches to the pathophysiological mechanisms of psychosis.

Supplementary Material

Supplementary data are available at *Schizophrenia Bulletin* online.

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