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Guided by Expectations: Overweighted Semantic Priors in Schizotypy and Their Links to Glutamate

Franziska Knolle, Elisabeth F. Sterner, Verena F. Demler, Lucy J. MacGregor, and Christoph Mathys

ABSTRACT

BACKGROUND: An imbalance in the weighting of prior beliefs and sensory evidence is thought to contribute to the development of psychotic symptoms, such as hallucinations and delusions. We investigated 1) how much individuals with schizotypal traits, a subclinical expression of psychosis proneness, use high-level semantic priors and sensory evidence to understand noise-degraded language; 2) whether an imbalance would potentially result in task-based hallucinations—perceptions that match expectations but not the input; and finally, 3) whether a potential imbalance was linked to altered levels of cortical glutamate.

METHODS: In a language comprehension task, we simultaneously manipulated semantic predictability, sensory degradation, and surprisal to estimate the prior weight using a Bayesian belief updating model. We conducted 2 studies. Study 1 (N = 109) tested the language comprehension task behaviorally; study 2 (N = 55) was used to replicate the findings of study 1, but was also combined with proton magnetic resonance spectroscopy to assess cortical levels of glutamate.

RESULTS: Study 1 showed that high-level priors were overweighted, with increasing schizotypy providing a potential explanation for the increased number of task-based hallucinations observed in the same individuals. Importantly, replicating the results of study 1, study 2 revealed that an overweighting of priors was associated with increased cingulate glutamate, providing a neurobiological basis for overreliance on top-down predictions.

CONCLUSIONS: These results offer a mechanistic and neurobiological understanding of how predictive coding alterations contribute to symptom development along the psychosis spectrum.

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Predictive coding models propose that hallucinations and delusions arise from an imbalance in how individuals with psychosis integrate information to form perceptions and beliefs (1–5). In predictive coding, inference is portrayed as the end product of a processing hierarchy in which higher levels encode core beliefs, and lower levels process concrete sensations. Using Bayesian inference, at each level and based on the beliefs, the brain generates a prediction about what is expected next. In a top-down process, this prediction is fed to lower levels and compared with perceptual inputs. Discrepancies, or prediction errors, are propagated in a bottom-up process to higher levels to update beliefs and minimize future errors.

Research on psychosis has revealed altered weighting of prior beliefs and sensory evidence as well as prediction errors (2,6–8), but the findings have been contradictory. Some studies found an underweighting of prior beliefs (9–12), consistent with an overweighting of sensory evidence (8,13), while others showed an overweighting of prior beliefs (7,14–17). A proposed resolution (4) suggests differential aberrations along the processing hierarchy: At lower levels, increased dopamine activity leads to heightened salience of irrelevant inputs, strengthening prediction errors and contributing to delusions (3,18,19). At higher levels, overweighted priors enhance sensory cortex signaling, generating hallucinations without external stimuli (20), potentially due to NMDA receptor (NMDAR) dysregulation (21–23).

However, defining processing hierarchies remains difficult, as prior beliefs are variably described (7,11,24), even in structured domains such as language, where levels form a continuum (25,26). This may explain inconsistencies in prior weighting findings (11,27,28). Therefore, systematic experimental manipulation of prior beliefs and sensory evidence, combined with computational modeling, is needed (29). To address these issues, we designed a language comprehension task (Figure 1A) modeled within a Bayesian inference framework that simultaneously manipulates the precision of high-level prior beliefs and sensory evidence (Figure 1B).

The relative weighting of priors and sensory evidence may also depend on symptom profiles (e.g., hallucinations vs. delusions) and illness stage (e.g., early vs. chronic psychosis) (7,15,30), potentially mediated by dopaminergic, serotonergic, and glutamatergic neurotransmission (31–34). Given the complexity of the disorder, psychosis is increasingly viewed dimensionally (35), suggesting that altered predictive coding mechanisms are present in individuals with subclinical

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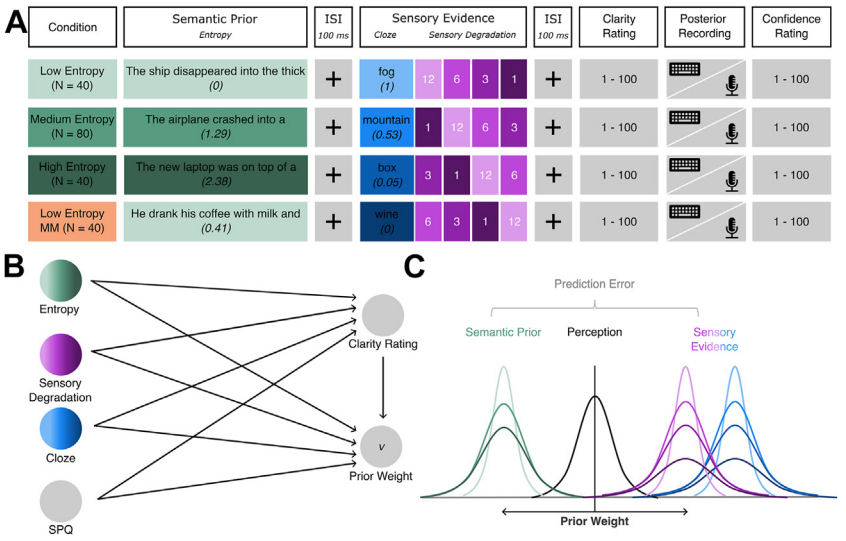

Figure 1. **(A)** The language comprehension task. Participants listened to sentences via headphones. Each sentence consisted of a sentence beginning up to the sentence final word inducing a high-level semantic prior with varying precision, measured in entropy (i.e., continuous measure of predictability; sentences were categorized as low [light green], medium [green], and high [dark green] entropy). Values in brackets represent the entropy for the respective German sentence equivalent. The sensory evidence that was represented by the noise vocoded target word was quantified using the word's cloze probability (shades of blue, the darker the color the lower the cloze probability; values in brackets represent the cloze probability for the respective German target word) and the level of sensory degradation (i.e., numbers of channels for noise vocoding, 1 channel = unintelligible [dark purple], 12 channels = highly intelligible [light pink]). After the participants heard the target word, they rated the clarity of the target word (Likert scale: 0–100). Participants were then asked to write down (study 1) or say (study 2) the word that they heard and rate how confident they were with their response (Likert scale: 0–100). Each participant completed 200 trials; each trial contained one sentence. **(B)** Directed acyclic graph and linear model of the causal relationships between the trial- and population-level parameters and the prior weight. The graph depicts a direct, population effects model, which shows the linear contributions of the parameters to the estimation of the prior weight ν. The participant effect (i.e., Schizotypal Personality Questionnaire [SPQ]) directly affects the prior weight ν, and all trial parameters (i.e., entropy, channel number, cloze probability) are stratified by the individual clarity rating. **(C)** Display of the predictive coding model. Predictive coding postulates that a posterior (i.e., the perception, black) is formed through the integration of prior (green) and sensory likelihood (pink/blue). The posterior moves toward the more reliable or more precise source of information (i.e., lower variance of the distribution), which is quantified in the computational model as the prior weight. ISI,interstimulus interval; MM, mismatch.

psychotic-like experiences or schizotypal traits. Studies in these populations have revealed similar alterations in prior weighting (7,16,30,36) and sensory likelihood (11,37,38) as seen in clinical groups, making them a well-controlled cohort for investigating the mechanisms that underlie psychosis, free of the confounding impact of antipsychotic medication, illness duration, or comorbidities.

Therefore, in this study, we tested the language comprehension task in healthy individuals assessed for schizotypy (39). We chose a language paradigm for 2 reasons. Firstly, language processing is intrinsically predictive (40–42), with predictive coding models providing a key theoretical framework (43–46). In noisy environments, for example at a party, prior expectations help listeners interpret speech (47,48). However, overly precise priors may lead to us hear what we expect rather than what is said, resulting in misperception, or so-called nonclinical hallucinations. Thus, this language paradigm represents a natural Bayesian inferential process and can be modeled accordingly. Secondly, language deficits are core to schizophrenia (49–53) and may contribute to auditory hallucinations and delusions via altered predictive coding.

This study fills a gap in current research by independently manipulating prior precision and sensory evidence while applying mechanistic Bayesian modeling. Study 1 (N = 109) established the model and examined whether increased schizotypy shifted the prior weight. Study 2 (N = 55) aimed to replicate these findings and explore the role of cortical glutamate in the anterior cingulate cortex (ACC) and bilateral dorsolateral prefrontal cortex (DLPFC), measured via proton magnetic resonance spectroscopy (^{1}H-MRS), in shifting the prior weight. The ACC and DLPFC play a crucial role in the integration of prediction errors (54–56), including weighted prediction errors (57–60), and therefore are potential neural correlates of the prior weight. Supporting this, alterations in (weighted) prediction error processing have been reliably detected across different stages of psychosis in these brain regions (6,61,62). Alterations in the level of glutamate have also been consistently reported in the ACC (33,63–65) and the DLPFC (63,66–68). Furthermore, we assessed the impact of autistic traits on the prior weight, as predictive coding models propose distinct, potentially opposite prior weighting in autism (69–71), expecting that autistic traits would have either no effect or an effect in the opposite direction on the shift in the prior weight. Additionally, we used the Verbal Comprehension Index (VCI) to rule out a potential effect of verbal intelligence on the weighting of prior information.

METHODS AND MATERIALS

Participants

Study 1 included 109 German native speakers (mean age = 32.91 years; 79 female) recruited via online advertisements and internal databases. Participants completed an online language comprehension task and the Schizotypal Personality Questionnaire (SPQ) (72). Study 2 included 55 new participants (mean age = 23.73 years; 28 female), who also completed the Autism Spectrum Quotient (AQ) (73) and the VCI of the Wechsler Adult Intelligence Scale, Fourth Edition (74). ^{1}H-MRS data were collected for 53 individuals; 2 did not complete the scan. Sample size was estimated using a power analysis (see the Supplement). All participants reported no history of psychiatric or neurological illnesses or brain injury, provided written informed consent, and received financial compensation or course credit. The study was approved by the Ethics Commission of the Technical University of Munich (Approval Nos. 786/20 S-SR, 2023/270 S-KH).

Subclinical and Psychological Questionnaires

The SPQ (74-item, yes/no format) assesses schizotypal traits. Study 1 participants had a mean SPQ score of 13.41 (SD = 11.06, range: 0–49), while study 2 participants scored lower (mean = 4.73, SD = 5.80, range: 0–26; Wilcoxon rank-sum test: $W = 1387$, $p < .001$). Study 2 participants had a mean AQ of 21.09 (SD = 7.27, range: 7–36; 50-item, 4-point Likert scale converted to a binary score, max = 50) and a mean VCI of 110.56 (SD = 9.30, range: 83–136; derived from Vocabulary, Information, and Similarities subtests; range: 50–150).

Task Specifics

Participants completed a language comprehension task (Figure 1A), designed to manipulate key elements of a predictive coding model. Participants listened to 200 sentences inducing a semantic prior of varying predictability (quantified by entropy) (75) and a sentence final target word, which constituted the sensory evidence. Sensory evidence was manipulated along 2 axes: 1) surprisal (measured via the cloze probability of a word) (75) and 2) acoustic clarity [similar to previous tasks (47,76,77)] using 4 levels of noise vocoding (78,79). After hearing the full sentence including the semantic prior and target word, participants rated target word clarity (100-point scale), reported their perceived word (study 1: written response; study 2: spoken response), and rated their confidence (100-point scale). Clarity ratings were expected to reflect sensory degradation (lower clarity for lower channel numbers) and prior predictability (higher clarity for expected words in low-entropy sentences).

Entropy and cloze probability were pre-estimated using a separate sample (n = 40–80 per sentence) and confirmed using a large language model (Figure S1 and Supplement). Entropy was estimated using Shannon entropy for the observed counts of all word predictions of each sentence. Cloze probability, a concept originally developed to measure readability by assessing the tendency of individuals to complete (close) language patterns with familiar items using the Cloze Test (80), was calculated as the proportion of the number of times a given word was predicted out of the total number of predictions. The entropy of the sentences varied from low entropy (i.e., highly predictable) to high entropy (i.e., highly unpredictable). The target word always fit the semantic context of the sentence prior but varied in surprisal [i.e., cloze probability (75)]. Sentences fell into 4 categories: low-entropy match (high cloze, n = 40), low-entropy mismatch (low cloze, n = 40), medium entropy (mixed cloze, n = 80), and high entropy (low cloze, n = 40). Sentence and target word characteristics were matched across conditions. The target words of each sentence were vocoded to reduced sensory detail (https://github.com/egaudrain/vocoder) (78) with 1, 3, 6, or 12 channels [1-channel highly unintelligible, 12-channel >80% intelligible (77)]. Each sentence's target word was presented at all 4 degradation levels, counterbalanced across participants.

Analyses

Modeling. To examine whether schizotypal traits are associated with an overweighting of high-level priors (i.e., entropy) relative to sensory evidence (i.e., noise degradation/channel number and surprisal/cloze probability), we fitted a Bayesian belief updating model to the data from study 1 (Figure 1B, C). This model provides a mechanistic estimate of the relative weight of the prior during sentence comprehension. The starting point of this approach is the assumption that participants maximize the probability of their answer being correct, which undergoes an update during each trial. Initially, this probability takes a prior value after the presentation of the prior-inducing phrase and is then updated to a posterior value upon hearing the degraded target word (sensory evidence). This process follows a Bayesian update in a beta-Bernoulli model, where the observation is binary (Bernoulli distributed) because a participant's response either corresponds (1) or does not correspond (0) to the presented stimulus. The prior and posterior distributions are beta-distributed, representing uncertain probabilities of correctness.

According to Bayes' rule, the update to the expected correctness probability takes the form of a precision-weighted prediction error:

$$P_{m_i|n_i} = P_{m_i} + \frac{1}{\nu_i+1}\left(\delta_{m_i,n_i} - P_{m_i}\right), \quad (1)$$

where P_{m_i} and $P_{m_i|n_i}$ are the prior and posterior cloze probabilities, respectively; m_i is the answer given at the ith trial; and n_i is the stimulus word presented at that trial. δ_{m_i,n_i} is a Kronecker delta indicating whether $m_i = n_i$ while $\nu_i > 0$ represents the implied number of previous Bernoulli observations (81), which can be directly interpreted as the weight of the prior relative to sensory evidence. The prediction can be formalized as the cloze probability of the answer given, which is all that participants could know before hearing the final stimulus. The prediction error then is the correspondence of the stimulus to the answer (1 or 0) minus the prediction and is used to update the expected correctness probability by adding it to the prediction after weighting by a factor of $1/(1+\nu)$, where ν is an implied number of previous observations that can be interpreted as the strength of the prior (i.e., its weight relative to the sensory evidence) (81).

We modeled ν_i on a trial-by-trial basis using a hierarchical linear model, incorporating task parameters—entropy, cloze probability of the stimulus (distinct from the cloze probability of the answer used in Bayesian updating), channel number, and clarity rating—as predictors. We further hypothesized that these effects would be modulated by participants' schizotypal traits, measured via the SPQ score. Accordingly, we used the following hierarchical model for $\log \nu_i$, with participant-level coefficients varying with SPQ score (Figure 1B):

$$\begin{aligned}\log(\nu_i) = {} & \beta_{1j[i]} + \beta_{2j[i]} \times \text{channels}_i + \beta_{3j[i]} \times \text{entropy}_i \\ & + \beta_{4j[i]} \times \text{cloze}_i + \beta_{5j[i]} \times \text{clarity}_i\end{aligned}$$

$$\beta_{kj} = \gamma_{1k} + \gamma_{2k} \times spq_j$$

$$\gamma_{lk} \sim N(0, 0.2), \quad (2)$$

where i indexes the trial, $j[i]$ is the participant involved in trial i, β_{kj} are participant-level coefficients, and γ_{lk} are population-level coefficients. We estimated ν_i in logarithmic space because it is bounded at 0 in native space, whereas in logarithmic space, it is unbounded. To assess the direct effects, we stratified our estimates by clarity rating.

The posterior distribution of all parameters (γ_{lk}) were estimated using Hamiltonian Monte Carlo sampling with the rstan software package (82). Sampling took place with 4 chains, each with 2000 iterations including 1000 warmup draws, which resulted in 4000 samples to generate the posterior distribution for each parameter.

In study 2, we replicated the model exploring the effect of SPQ on the prior weight and also repeated the procedure using autistic traits or VCI, allowing us to compare their impact. Using a distribution analysis, we compared the impact of the SPQ score, autistic traits, and VCI.

Posterior Predictive Simulations of Impact of Schizotypy on Prior Weight. Counterfactual simulations were made by constructing a typical trial, setting all task condition predictors to task-average values. Samples of participant-level coefficient values β_{kj} for varying SPQ levels were constructed by taking the fitted posterior samples of the population-level coefficient values γ_{lk} and varying the SPQ predictor systematically over its full range from 1 to 70. This yielded a posterior predictive distribution of v_i for each SPQ level in the typical trial. The same approach was applied across entropy conditions (Figure 2C).

Spectroscopy Data Acquisition and Analysis

Participants in study 2 underwent ^{1}H-MRS to assess glutamate in the ACC and the bilateral DLPFC. Details are provided in the Supplement and elsewhere (83,84).

Associations Between Cortical Glutamate and the Schizotypy-Informed Prior Weight

To investigate whether levels of cortical glutamate were associated with how strongly participants relied on prior knowledge, we fitted a linear model (ordinary least squares) to predict the simulated schizotypy-informed prior weight with levels of glutamate in the left DLPFC, right DLPFC, and ACC and with biological sex as a covariate. Sex was included as a covariate to control for consistently reported sex-related differences in levels of glutamate (85–87), especially in psychosis (88,89). All scores were normalized.

Impact of Predictability and Sensory Evidence on Clarity and Confidence Ratings and Word Perception and Its Interaction With Schizotypy

To examine the effects of predictability (i.e., entropy) and sensory evidence (i.e., acoustic degradation, number of channels) on clarity and confidence ratings, 2-factor repeated-measures analyses of variance (ANOVAs) were conducted separately for mean clarity and confidence ratings. These ANOVAs included the factors entropy level (low, medium, high, low-entropy mismatch) and channel degradation (1,3,6,12). To investigate the impact of sentence predictability on the distribution of behavioral response types, separate repeated-measures ANOVAs with entropy level as a factor were performed for correct responses and no responses.

For task-based hallucinations, a 2-factor repeated-measures ANOVA was carried out, including entropy level and channel degradation as factors. For all ANOVAs, Mauchly's test was applied assessing sphericity; if violated, degrees of freedom were adjusted using Greenhouse-Geisser estimates. Significant effects were followed by Tukey post hoc tests. Lastly, Pearson correlations were used to examine associations with schizotypy.

RESULTS

Impact of Schizotypy on Prior Weight During Auditory Sentence Processing

The belief updating model estimation converged and met all quality checks (Figures S2 and S3). The results revealed an overall significant positive impact of schizotypy (Table 1 and Figure 2A) on the prior weight ν, indicating that higher schizotypy shifts weight toward prior belief and away from sensory evidence (coefficient gamma[2,1]). Modeling results revealed that increasing schizotypy (SPQ) by 1 point increased prior weight by 3.03% relative to sensory evidence (i.e., exp(−0.10 + 0.33/11.06)/exp(−0.10) = 1.0303, where 11.06 is the SPQ SD, −0.10 is the gamma[1,1] intercept, and 0.33 is gamma[2,1], reflecting SPQ impact). This effect was replicated in study 2 with an independent sample of 55 individuals (Figure 2A). Study 2 also examined autistic traits and VCI to test SPQ specificity on prior weight. Neither autistic traits (Figure S4A) nor VCI (Figure S4B) significantly influenced the prior weight. Distribution

Table 1. Parameter Estimates for Gammas for Each Task Parameter Indicating the Population Effect of SPQ (*N* = 109)

	Median	MAD	2.5% CI	25% CI	50% CI	75% CI	97.5% CI	Effective *n*	Rhat
Gamma[1,1], Intercept	−0.10	0.062	−0.23	−0.14	−0.10	−0.06	0.02	4903	1
Gamma[1,2], Channel Number	−1.52	0.075	−1.67	−1.57	−1.52	−1.47	−1.38	6111	1
Gamma[1,3], Entropy	−0.03	0.060	−0.15	−0.07	−0.03	0.01	0.08	7763	1
Gamma[1,4], Cloze Probability	−1.34	0.137	−1.61	−1.44	−1.34	−1.25	−1.08	5301	1
Gamma[2,1], SPQ→Intercept	0.33	0.056	0.22	0.29	0.33	0.37	0.45	5416	1
Gamma[2,2], SPQ→Channel Number	0.05	0.056	−0.06	0.01	0.05	0.09	0.16	8590	1
Gamma[2,3], SPQ→Entropy	−0.03	0.053	−0.13	−0.06	−0.03	0.01	0.08	7132	1
Gamma[2,4], SPQ→Cloze Probability	0.12	0.126	−0.13	0.04	0.12	0.21	0.36	6038	1

In this model the prior weight was stratified by the effect of the clarity rating. As the posterior gamma distribution of clarity is uninterpretable, it is not presented in the table. Effective sample size of 10,000 samples have been drawn. Rhat indicates the potential scale reduction factor on split chains. Rhat < 1.04 indicates convergence.

MAD, median absolute deviation; SPQ, Schizotypal Personality Questionnaire.

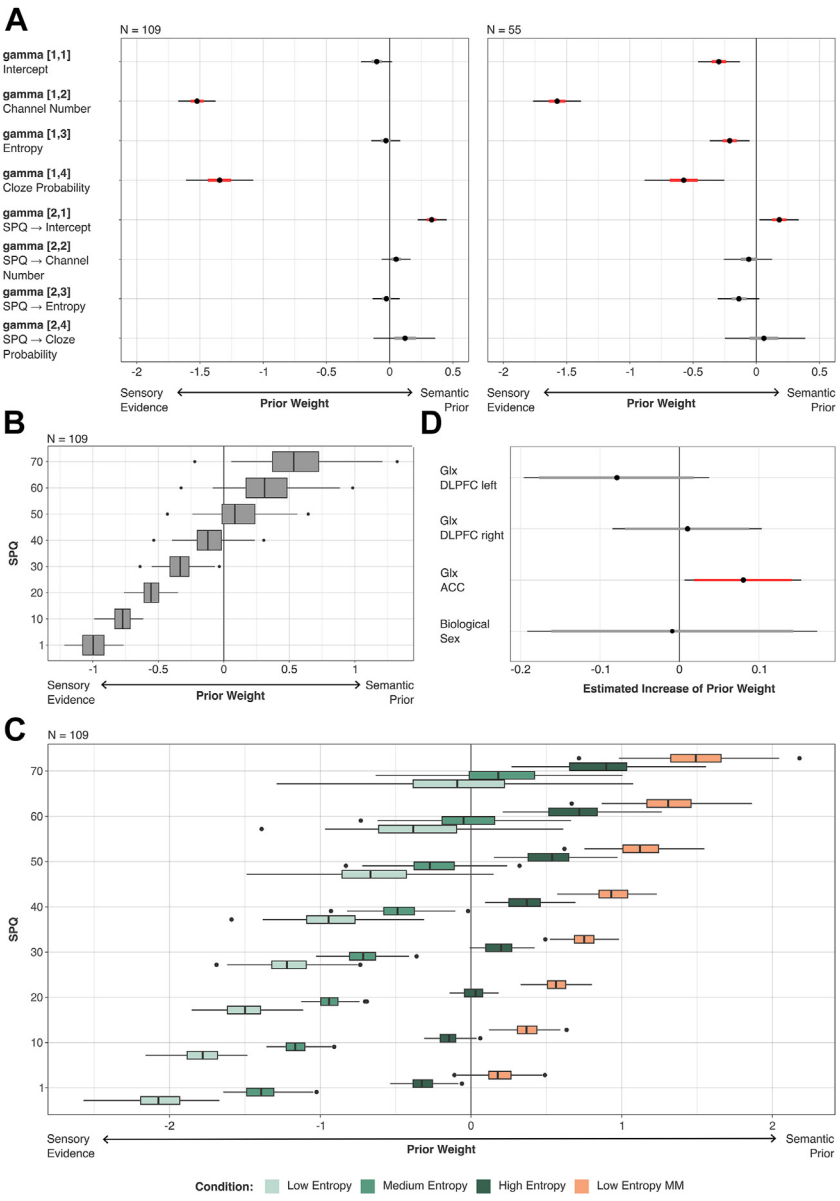

Figure 2. Impact of schizotypy on the prior weight and its associations with cortical glutamate. **(A)** For each trial of our task in study 1 (N = 109, left), we estimated the weight ν of the prior relative to the sensory evidence using the normative intratrial Bayesian belief update. Simultaneously, to explain trial-level values of ν, we used a regularized hierarchical linear model with task conditions as predictors and coefficients modulated by the participant's Schizotypal Personality Questionnaire (SPQ) score. Parameters with 95% CI excluding 0 indicate a substantial contribution. For an intuition, coefficient gamma[1,2] quantifies the effect of acoustic degradation on the prior weight, meaning that when the input becomes clearer (channel number going up/acoustic degradation going down) individuals rely more on the sensory input, which would be expected. Importantly, the SPQ-dependent modulation of the intercept (coefficient gamma[2,1]) quantifies the effect of schizotypy in increasing the relative weight of the prior. This effect was replicated in study 2 (N = 55, right). **(B)** Posterior predictive simulation of the relative prior weight ν_i for an average task trial when systematically increasing SPQ of study 1. The simulation illustrates the overweighting of prior beliefs relative to sensory evidence with increasing schizotypy. **(C)** Posterior predictive simulations of the impact of increasing SPQ on the prior weight ν_i for an average sentence from all 4 categories of entropy (study 1). **(D)** Associations between cortical glutamate and the prior weight were assessed in study 2. Levels of glutamate (Glu) measured in the anterior cingulate cortex (ACC) significantly predicted increasing prior weight. Associations with Glu levels in the left and right dorsolateral prefrontal cortex (DLPFC) could not conclusively be quantified with our data. MM, mismatch.

analyses confirmed that the SPQ's effect was stronger than the effect of autistic traits (Tukey's honestly significant difference [HSD]: mean difference = −0.139; 95% CI, −0.143 to −0.135; $p <$ .001) (Figure S5) and VCI (Tukey's HSD: mean difference = −0.325; 95% CI, −0.329 to −0.321; $p <$ .001) (Figure S5).

Posterior predictive simulations illustrated how schizotypy affected prior weight in typical trials (Figure 2B) and specific sentence scenarios (Figure 2C). Individuals with higher schizotypal traits consistently overweighted priors, particularly in uncertain conditions when priors were imprecise or the final word was unexpected. Noise degradation simulations further confirmed this effect, showing greater reliance on priors under increased degradation, particularly in individuals with higher levels of schizotypy (Figure S6).

Associations Between Cortical Glutamate and the Schizotypy-Informed Prior Weight

A linear regression model, controlling for sex (85), was used to examine associations between glutamate levels and prior weight (R^2 = 0.13; adjusted R^2 = 0.05; beta = −0.51; 95% CI, −0.77 to −0.26). Glutamate levels in the ACC significantly predicted prior weight in posterior predictive simulations (beta = 0.08; 95% CI, 1.83×10^{-3} to 0.16; t_{45} = 2.06, p = .045, standardized beta = 0.31), indicating that increasing ACC glutamate led to stronger prior weighting in schizotypy, making task-based hallucinations more likely. The effects of glutamate in the left/right DLPFC or sex were inconclusive (Figure 2D and Table S8). Standardized parameters, 95% CIs, and p values were computed using a Wald t-distribution approximation. The

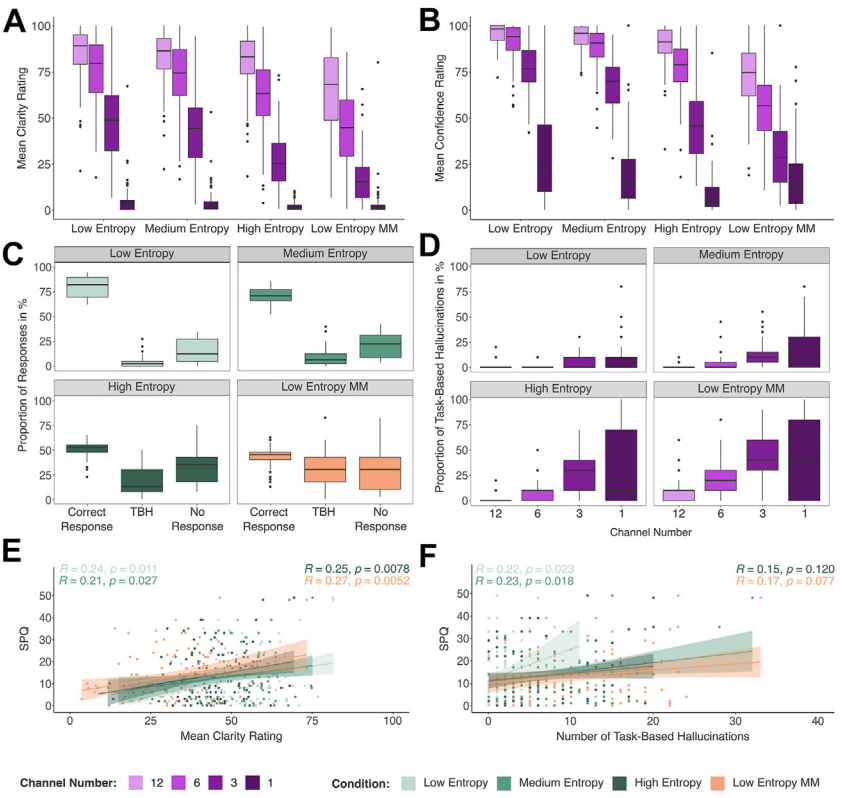

Figure 3. Behavioral effects. **(A)** The clarity rating and **(B)** confidence rating significantly decreased with increasing degradation (decreasing channel numbers) similarly in all conditions. **(C)** Correct responses decreased and false responses increased with increasing entropy (i.e., predictability) and mismatch (MM) manipulations, while no responses stayed relatively stable. **(D)** Nonclinical task-based hallucinations (TBHs) increased across all conditions with increasing acoustic degradation (decreasing channel numbers), with strongest effects in the high-entropy and low-entropy MM conditions. **(E)** Higher levels of schizotypy (measured with the Schizotypal Personality Questionnaire [SPQ]) were linked to higher clarity ratings across all conditions of entropy. **(F)** Higher levels of schizotypy (SPQ) were linked to more false posteriors, or task-based hallucinations, especially in the low- and medium-entropy conditions but marginally also in the low-entropy MM condition.

observed effect size was notably smaller (R^2 = 0.13, f^2 = 0.14) than expected, which highlights the need for replication in a larger sample.

Impact of Predictability and Sensory Evidence on Word Perception and Clarity and Confidence Ratings and Its Interaction With Schizotypy

To validate the paradigm, we used ANOVAs to test manipulations. Clarity (Figure 3A) and confidence (Figure 3B) ratings significantly decreased with increased degradation (fewer channels) and higher entropy (clarity: $F_{5.50,593.53}$ = 90.87, p < .001, general effect size = 0.97; confidence: $F_{6.00,378.07}$ = 51.16, p < .001, effect size = 0.178). Words with fewer channels and higher entropy were perceived as less clear, with lower confidence (Tables S1 and S4). These findings support the clarity rating as an indirect measure of predictability (precision of the prior), as words that followed precise priors (lower entropy) were rated as clearer regardless of degradation level (Tables S1 and S2).

Misperceptions (task-based hallucinations) increased with higher entropy and mismatch manipulations ($F_{1.51,163.45}$ = 274.17, p < .001, η = 0.433), particularly in high/low-entropy mismatch conditions (Figure 3C and Table S7). Correct responses decreased ($F_{1.89,204.06}$ = 922.08, p < .001, η = 0.77), and nonresponses increased ($F_{2.08,225.07}$ = 170.50, p < .001, η = 0.165) (Figure 3C; Tables S5 and S6). Task-based hallucinations increased across all conditions with greater acoustic degradation ($F_{4.10,442.3}$ = 41.38, p < .001, general effect size = 0.77) (Figure 3D and Table S7), suggesting that increasing uncertainty, driven by semantic prior and sensory evidence manipulations, contributes to task-based hallucinations.

Importantly, schizotypy (SPQ) positively correlated with clarity ratings across low (r = 0.24, p = .011), medium (r = 0.21, p = .027), and high (r = 0.25, p = .0078) entropy and low-entropy mismatch (r = 0.27, p = .0052) (Figure 3E). Individuals with higher levels of schizotypy perceived words following predictable contexts more clearly, providing direct evidence for prior overweighting. SPQ scores also positively correlated with task-based hallucinations in low (r = 0.22, p = .023) and medium (r = 0.23, p = .018) entropy. A marginal effect was found for low-entropy mismatch (r = 0.17, p = .077), with no effect in high entropy (r = 0.15, p = .12) (Figure 3F), suggesting that prior overweighting may induce more task-based hallucinations.

DISCUSSION

In this study, we manipulated the precision of high-level semantic priors and sensory evidence simultaneously in a language comprehension task to examine whether individuals with increased schizotypal traits rely more on prior beliefs or sensory input when processing noisy speech. Using a Bayesian belief updating model informed by schizotypy scores, we quantified the relative weight of priors on a trial-by-trial basis. Additionally, we explored whether altered cortical glutamate levels contribute to imbalanced prior weighting via ^{1}H-MRS. Our results indicate that individuals with increased

schizotypy overweighted semantic priors relative to sensory evidence (Figure 3A, B). This effect was specific to schizotypy, as neither autistic traits nor VCI affected the prior weight significantly. Furthermore, we provide preliminary evidence that prior overweighting was associated with increased cortical glutamate in the ACC, potentially suggesting a neurobiological basis for impaired semantic prior weighting (Figure 3D). Our finding demonstrates how the observed overreliance on semantic prior beliefs could result in a biased interpretation of noisy speech input, manifested as false percepts or nonclinical task-based hallucinations, in subclinical stages of psychosis. This is consistent with predictive coding models of psychosis (2,4,5), suggesting a direct link between impaired language processing and the emergence of positive symptoms over time.

Recent empirical research has highlighted the critical role of prior weighting in psychosis, although findings regarding whether priors or sensory evidence are overweighted remain inconsistent. Some argue that the imbalances in weighting may depend on the processing hierarchy and/or disease stage (4,7,13). Studying subclinical psychosis provides valuable insights into early mechanisms, potentially enabling early intervention. Various tasks have been used to explore prior weighting in schizotypal individuals, with probabilistic learning and perceptual decision-making tasks suggesting sensory overweighting (11,37,38,90,91), while visual and auditory disambiguation tasks indicate stronger prior reliance (16,30,36,92–95), although some results also remain inconclusive (24,96,97).

Unlike previous studies, we simultaneously manipulated the precision of high-level semantic priors and sensory evidence in a language comprehension task. This allowed us to analyze the specific contributions of each component to the prior weighting, which was evident behaviorally in the objective task performance and in subjective clarity ratings, as reflected in the significant interaction analyses. Individuals with higher levels of schizotypy generally perceived words more clearly, showing an indirect effect of overly strong priors. Notably, in our task, prior refers to a prediction induced trial-by-trial, potentially generating an overly precise local prior. However, other work has shown overly strong global and local priors in psychosis, particularly in disambiguation tasks (30,36).

Our Bayesian belief updating model provides a mechanistic, trial-specific quantification of prior weight, moving beyond statistical estimations. Conditional simulations confirmed that schizotypy-related prior overweighting varied with uncertainty: When language input was predictable (low/medium entropy), and sound quality was good (12 and 6 channels) (Figure S6), sensory input was weighted more strongly. From a brain processing perspective, this enables fast detection of mismatching information that may convey novel and important information (98,99). As soon as uncertainty increased through less predictable context or noisier input, the simulations indicated that all individuals weighted the prior more strongly than the sensory input. Also, this behavior is ecologically sensible, as highly ambiguous situations may be resolved faster when prior knowledge is used (17,30). Crucially, individuals with higher levels of schizotypy weighted prior beliefs more regardless of context predictability or clarity. This may provide a mechanistic explanation for the propensity of individuals on the psychosis spectrum to perceive speech in noise (100–104). An effect that was observed by Alderson-Day *et al.* (36), who found that individuals prone to auditory hallucinations identified sine-wave speech more frequently without prior exposure, suggests a general tendency to perceive speech in noise—a pattern that has been observed in visual processing as well (16,17,30).

Phenomenologically, it is interesting that an overreliance on prior information increased the susceptibility of individuals with higher levels of schizotypal traits to nonclinical task-based hallucinations that confirmed their expectations. This highlights how high-level semantic priors shape speech perception and suggests that imbalanced prior weighting may contribute to misperceptions already at subclinical stages. Relatedly, Powers *et al.* (15) demonstrated that hallucination-prone individuals more often perceived absent sounds when predicted, an effect that has been replicated in other studies (105,106). Our model extends this work by jointly manipulating prior and sensory precision, identifying misperceptions as psychosis markers.

Beyond computational insights, our study provides preliminary evidence for a neurobiological correlate of prior overweighting. However, the effect size was smaller than expected, underlining the need for replication in larger samples. These preliminary findings indicate that increased ACC glutamate may explain how neurobiological alterations drive cognitive biases through heightened prior reliance (14,33). Within hierarchical predictive coding, forward connections convey prediction errors via AMPA receptors, while backward connections transmit priors via NMDARs (107,108). NMDAR blockade (e.g., ketamine) induces psychosis-like symptoms (109,110), supporting the NMDA hypofunction model of schizophrenia (111). NMDAR deficits on GABAergic (gamma-aminobutyric acidergic) interneurons may cause pyramidal neuron disinhibition, thereby increasing excitability and disrupting excitatory/inhibitory balance (112,113), a key factor in cortical neural computations. This disruption enhances glutamate release through AMPA receptors and is consistent with the hyperglutamatergic accounts of schizophrenia (33,114–116).

The ACC may integrate high-level abstract predictions (potentially semantic priors, as in the current study) with lower-level sensory evidence (107,117), as well as sensory multimodal integration (118,119), prediction error processing (120), or processing of uncertainty (121). Structural and functional ACC alterations are common in psychosis (122), including increased glutamatergic input, abnormal glutamate-dopamine circuit connectivity, and reduced synaptic density (123–125). ^{1}H-MRS studies have consistently found altered ACC glutamate levels across psychosis stages (32,33,64), with decreased levels at later stages (126) and increased levels at the earliest stages, especially in individuals at high risk for psychosis (33) or individuals with increased schizotypy (65).

Our results suggest that ACC hyperactivation (elevated glutamate) underlies excessive prior weighting and impaired sensory integration, consistent with prior studies. Scholl *et al.* (127) reported that higher ACC glutamate levels correlated with increased reliance on task-related information. Similarly, Cai *et al.* (118) observed a positive association between ACC glutamate and altered sensory integration in schizophrenia. Our

findings suggest that ACC glutamate dysregulation may contribute to the overreliance on priors, potentially driving positive symptom development in at-risk psychosis stages. By contrast, Leptourgos *et al.* (106) found no ACC correlation, possibly due to task type (auditory oddball) and sample (patients with schizophrenia rather than at-risk individuals) differences.

Despite novel insights, patient studies are required to understand whether these can be translated to clinical stages. The cross-sectional design precludes causal inference; thus, longitudinal studies are needed to clarify perceptual abnormality trajectories in psychosis. Future work should include psychosis-proneness measures [e.g., Cardiff Anomalous Perceptions Scale (128), Peters et al. Delusions Inventory (129)] to assess symptom-specific effects. Larger studies are needed to replicate the computational and neurobiological effects.

Conclusions

Our findings demonstrate that increased schizotypy is linked to an overreliance on semantic priors over sensory evidence, which is associated with elevated ACC glutamate. Importantly, the prior weight was estimated using a mechanistic belief updating model. This supports predictive coding models of psychosis and offers a neurobiological and neurocomputational explanation for biased perception in schizotypy. Over time, this imbalance may contribute to the emergence of psychotic symptoms, such as hallucinations and delusions.

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ARTICLE INFORMATION

From the School of Medicine and Health, Department of Diagnostic and Interventional Neuroradiology, Technical University of Munich, Munich, Germany (FK, EFS, VFD); School of Medicine and Health, the Technische Universität München Neuroimaging Center, Technical University of Munich, Munich, Germany (FK, EFS, VFD); Medical Research Council Cognition and Brain Sciences Unit, University of Cambridge, Cambridge, United Kingdom (LJM); and Interacting Minds Centre, Aarhus University, Aarhus, Denmark (CM).

Address correspondence to Franziska Knolle, Ph.D., at franziska.knolle@tum.de.

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