Dynamic Evidence Integration Patterns Distinguish Schizophrenia From Healthy and Psychiatric Controls

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Methodological Issue Being Addressed Psychiatry lacks objective biomarkers for psychotic disorders, creating critical challenges in treatment selection, monitoring therapeutic response, and clinical trial design. Among these is the ability to evaluate reasoning, critical for assessing delusional thinking and overall functional capacity. We have created a brief computerized assessment that probes evidence integration in the context of scenarios that vary working memory load, credit assignment across multiple agents/contexts, and parallel hypothesis evaluation. This approach achieves robust diagnostic discrimination and tracks treatment response in a real-world clinical setting.

Introduction Objective assessment of reasoning deficits in schizophrenia patients remains a fundamental barrier to improving clinical care and trial design. Unlike other medical specialties that rely on imaging or laboratory tests to guide treatment decisions and measure outcomes, psychiatry depends on subjective symptom ratings that are vulnerable to bias and lack precision. This absence of quantitative biomarkers impedes treatment selection, makes it difficult to objectively track therapeutic response, and creates challenges for clinical trials in demonstrating efficacy. Schizophrenia patients demonstrate specific deficits in reasoning, rendering them susceptible to delusional thinking and lowering their overall functional capacity. Bias against disconfirmatory evidence (BADE) has been a research framework to study such deficits, but have been reserved for research settings due to their relatively low signal-to-noise and impracticality in a clinical setting. Here we modify classical BADE paradigms and develop a brief task that achieves a high discriminative signal-to-noise with as few as 10 scenarios.

Methods We assessed N=36 participants: healthy controls (HC, n=14), schizophrenia patients with delusions (SCZ, n=15), psychiatric controls without delusions (PC, n=4), and post-treatment schizophrenia patients (SCZ_POST, n=3). Participants completed 10 computerized scenarios via interactive graphical interface: 5 scenarios using traditional sequential evidence integration and 5 scenarios varying working memory load, credit assignment across multiple agents/contexts, and parallel hypothesis evaluation requirements.
Each scenario presented three sequential evidence statements (S1→S2→S3). After each statement, participants rated multiple interpretation plausibility using continuous visual analog scales. The system captured all rating adjustments with precise timing. We extracted quantitative features characterizing how ratings changed across the evidence sequence, including measures of belief

updating dynamics, response consistency within and across trials, and performance differences between traditional versus cognitively demanding task types.
Data analysis employed Principal Component Analysis (PCA) for dimensionality reduction to identify primary sources of variance distinguishing groups. We calculated effect sizes (Cohen's d) for group comparisons and assessed treatment effects by comparing pre- and post-treatment patient performance. Statistical significance was evaluated using appropriate non-parametric tests given sample size constraints.

Results The first principal component (PC1) provided robust discrimination between groups, accounting for 21.7% of total variance. Healthy controls consistently showed positive PC1 scores reflecting systematic evidence integration patterns, while schizophrenia patients showed negative PC1 scores reflecting high trial-to-trial variability in reasoning. Effect sizes for HC versus SCZ comparisons exceeded Cohen's d = 3.0, indicating very large group separation.
Scenarios requiring higher working memory and parallel hypothesis evaluation demonstrated substantially stronger discriminative power than traditional evidence integration tasks. Group differences on these cognitively demanding scenarios showed effect sizes exceeding d = 2.0, compared to smaller effects for traditional sequential updating scenarios. This suggests tasks imposing higher cognitive load are more sensitive to the reasoning deficits present in schizophrenia.
All three post-treatment patients demonstrated positive PC1 scores, similar to healthy controls and Patient Controls exhibited intermediate values.

Conclusion Brief computerized reasoning assessment provides objective, quantitative biomarkers addressing psychiatry's critical need for measurement tools analogous to imaging or laboratory tests in other specialties. Scenarios imposing higher cognitive demands reveal treatment-responsive mechanisms with exceptional discriminative power (d > 3.0). Principal component analysis achieves robust patient-control separation while capturing treatment response, enabling objective monitoring that could improve clinical trial design through quantitative outcome measures. This methodology demonstrates that cognitively demanding reasoning tasks provide sensitive, clinically feasible biomarkers for psychotic disorders, potentially transforming assessment and monitoring of reasoning deficits in routine psychiatric care and clinical trials design.

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