Schizophrenia Bulletin
https://doi.org/10.1093/schbul/sbae014

Clarifying Cognitive Control Deficits in Psychosis via Drift Diffusion Modeling and Attractor Dynamics

Chen Shen[1,†], Olivia L. Calvin[2,†], Eric Rawls[3], A. David Redish[2], and Scott R. Sponheim[*,1,3,4]

[1]Department of Psychology, University of Minnesota, Minneapolis, MN, USA; [2]Department of Neuroscience, University of Minnesota, Minneapolis, MN, USA; [3]Department of Psychiatry and Behavioral Sciences, University of Minnesota, Minneapolis, MN, USA; [4]Mental Health, Minneapolis Veterans Affairs Health Care System, Veterans Affairs Medical Center, Minneapolis, MN, USA

[*]To whom correspondence should be addressed: tel: 612-467-3916, fax: 612-273-9779, e-mail: sponh001@umn.edu

[†]Co-first authors.

***Background and Hypothesis*: Cognitive control deficits are prominent in individuals with psychotic psychopathology. Studies providing evidence for deficits in proactive control generally examine average performance and not variation across trials for individuals—potentially obscuring detection of essential contributors to cognitive control. Here, we leverage intertrial variability through drift-diffusion models (DDMs) aiming to identify key contributors to cognitive control deficits in psychosis. *Study Design*: People with psychosis (PwP; $N = 122$), their first-degree biological relatives ($N = 78$), and controls ($N = 50$) each completed 120 trials of the dot pattern expectancy (DPX) cognitive control task. We fit full hierarchical DDMs to response and reaction time (RT) data for individual trials and then used classification models to compare the DDM parameters with conventional measures of proactive and reactive control. *Study Results*: PwP demonstrated slower drift rates on proactive control trials suggesting less efficient use of cue information. Both PwP and relatives showed protracted nondecision times to infrequent trial sequences suggesting slowed perceptual processing. Classification analyses indicated that DDM parameters differentiated between the groups better than conventional measures and identified drift rates during proactive control, nondecision time during reactive control, and cue bias as most important. DDM parameters were associated with real-world functioning and schizotypal traits. *Conclusions*: Modeling of trial-level data revealed that slow evidence accumulation and longer preparatory periods are the strongest contributors to cognitive control deficits in psychotic psychopathology. This pattern of atypical responding during the DPX is consistent with shallow basins in attractor dynamic models that reflect difficulties in maintaining state representations, possibly mediated by excess neural excitation or poor connectivity.**

Key words: dot pattern expectancy task/schizophrenia/drift rate/nondecision time/first-degree relatives/explainable machine learning

Introduction

Cognitive control is the ability to regulate, coordinate, and sequence thoughts and actions in accordance with internally maintained behavioral goals.[1] Deficits of cognitive control in psychotic psychopathology are often characterized by difficulties incorporating information about the context to determine a correct response (ie, proactive control).[2,3] The AX-Continuous Performance Test (AX-CPT)[4] and its non-letter variant the dot pattern expectancy task (DPX)[5] were developed to capture cognitive control. These tasks sequentially present the within-stimulus conflicts in cognitive control tasks with "cue" (context) and "probe" periods (figure 1A). The stimulus sequences allow participants to engage in proactive or reactive control by anticipating the correct response from the cue or probe.

Behavior on the AX-CPT and DPX tasks has typically been characterized using accuracy, average reaction times (RT) on correct trials, and indices based on these features. A popular index, d'-context, assesses context processing via accuracy.[6] Deficits in cognitive control among people with psychosis (PwP) are characterized by lower BX accuracy,[7,8] prolonged RT,[8,9] and lower d′-context.[10,11] Relatives of PwP often demonstrate attenuated deficits in these measures.[11–15] While these approaches are adequate for describing trends in the central tendencies of RT and accuracy, they omit within-subject variation in responding which contains valuable information about underlying cognitive processes. Accuracy and RT have long been shown to be intertwined behavioral outcomes with

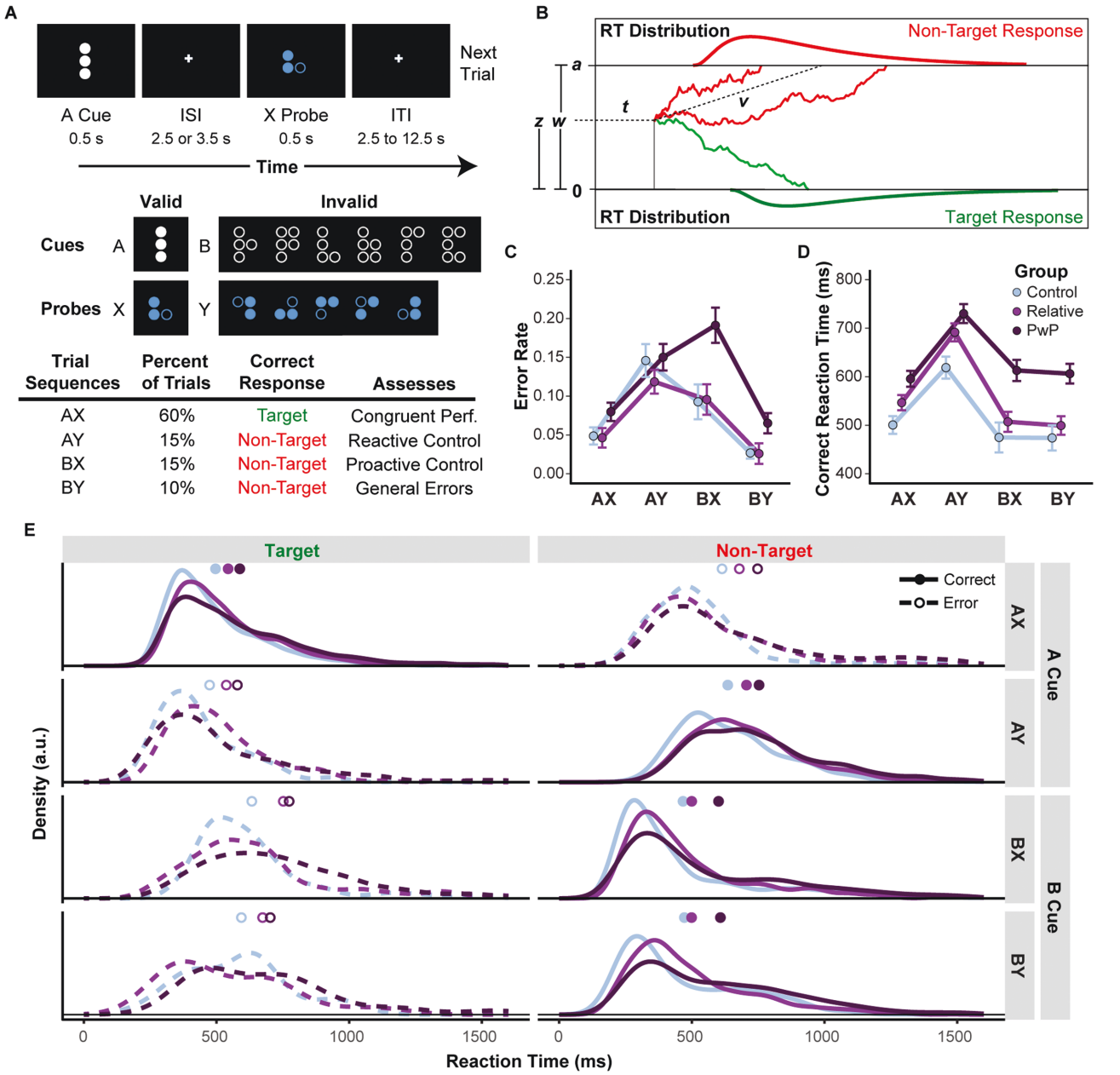

Fig. 1. (**A**) The dot pattern expectancy (DPX) task. Each trial consists of a "cue" stimulus presentation followed by a "probe" stimulus after a brief interstimulus interval (ISI). The stimuli consist of dot patterns with their only being 1 valid cue (A) and 1 valid probe (X) among similar invalid stimuli (B and Y). The most frequent cue-probe sequence is AX, which promotes a response prepotency on A cues for the target response. After each trial, there is a variable intertrial interval (ITI). (**B**) The drift-diffusion model (DDM). Four parameters are used by DDMs to fit the reaction time and error performance of the subject. These parameters are the bias (z), the decision threshold (a), the drift rate (v), and the nondecision time (t). These 4 parameters produce reaction time and response probabilities for the subjects' target and nontarget responses. The ratio of the bias and decision threshold (z/a) is typically fit via hierarchical DDM, which is denoted here as w. (**C**) Error rate means and standard errors of participants in each group across trial types. (**D**) Reaction time means and standard errors of participants in each group across trial types. (**E**) The normalized reaction time probability density distributions for each group's target and nontarget responses. The dots above each of the panels indicate the participant mean reaction times from panel B.

informative variation across trials. Assessing accuracy and RT independently via central tendencies underappreciates the speed-accuracy tradeoff in AX-CPT and DPX data.[16]

Drift-diffusion modeling (DDM) addresses accuracy and RT in a singular analysis that encompasses within-subject variability across trials (figure 1B). In DDMs, an underlying cognitive process noisily accumulates evidence to produce a response. DDM characterizes decision-making processes with 4 parameters, representing the rate of evidence accumulation (drift

rate, *v*), the amount of evidence required to act (decision threshold, *a*), prestimulus bias toward a particular response (bias, *w*), and response delay from non-decision-making related processes (nondecision time, *t*). Given that PwP showed both longer RT and higher error rates in the AX-CPT and DPX tasks,[8,10,17–19] we suspected that they could markedly differ from controls in how they accumulate evidence to produce responses.

DDM of task performance in people with schizophrenia has revealed deficits in drift rates and nondecision times on N-back and coding tasks.[20–22] On a reward-punishment task, Moustafa et al.[23] observed longer nondecision times, but with greater decision thresholds. Recent analyses by Smucny et al. suggested a slower drift rate in individuals with recent-onset schizophrenia during reward anticipation,[24] and cognitive control on the AX-CPT,[25] but their analytic approach was limited and could not resolve differences in nondecision times or bias. One study demonstrated a heightened bias in schizophrenia with a temporal prediction task.[26] The single study of relatives identified slower drift rates and longer nondecision time in siblings of individuals with schizophrenia than controls on a sustained attention task.[21] Recent simulations[27,28] of the effects of excitation-inhibition imbalance suggest that longer nondecision times and slower accumulation rates observed in schizophrenia could be attributed to deviations in the efficacy of N-methyl-D-aspartate receptor (NMDAR)-mediated glutamate (excitation) and gamma-aminobutyric acid (GABA; inhibition). These deviations lead to weak neural representations of stimuli within circuits required for appropriate responding. Magnetic resonance spectroscopy suggests that this weakening of neural representations is mediated by glutamatergic hypofunction.[29] The potential effects of excitation-inhibition balance on responses and RT suggested that analyzing cognitive control through a full DDM of trial-level data would provide insights into psychosis.

We applied hierarchical DDMs to DPX RT and response data obtained from PwP, their first-degree biological relatives, and controls. Our goal was to examine how subprocesses of cognitive control differ across these groups during proactive and reactive control. We hypothesized that PwP would have longer nondecision times and slower drift rates, with similar but less severe anomalies in relatives. We expected a reduced bias in PwP given past findings of deficits in proactive control. We predicted no differences in the decision threshold among groups.

Methods

Participants and Clinical Measures

Two hundred and eighty-six participants were recruited as part of the Psychosis Human Connectome Project. All participants underwent an informed consent procedure, gave written consent, and their capacity to provide informed consent was evaluated using the University of California Brief Assessment of Capacity to Consent.[30] All procedures were approved by the Institutional Review Board at the University of Minnesota, and were in accordance with the guidelines for human subjects research set by the Declaration of Helsinki. For a detailed description of the Psychosis Human Connectome Project, please see Demro et al.[31] Among 259 participants who completed the DPX task, we excluded participants based on reported recent substance use that may affect performance, or if psychotic symptoms could be partially attributed to medical conditions. In addition, 7 participants did not meet inclusion criteria for the DPX task based on their responses (see details in the DPX section below). In summary, 122 PwP, 78 of their first-degree biological relatives, and 50 controls were included in the analysis. Participants completed a clinical interview and self-report questionnaires, as well as cognitive and motor assessments. Trained research assistants conducted the Structured Clinical Interview for DSM-IV-TR disorders,[32] and the Psychosis Module of the Diagnostic Interview for Genetic Studies[33] with each of the participants to obtain diagnostic information. Diagnostic consensus was completed by a team of at least 2 qualified assessors (clinical psychology graduate students, postdoctoral associates, or licensed psychologists) to determine which diagnostic criteria were met, and reached consensus on the most appropriate DSM diagnoses. Among the 122 PwP, 72 were individuals with schizophrenia or schizophreniform, 14 were individuals with schizoaffective disorder, and 36 were individuals with bipolar disorder with psychosis. In addition to making diagnostic determinations, we also collected symptomatology measures. The Brief Psychiatric Rating Scale-24 Item Version (BPRS)[34,35] and the Scales for the Assessment of Negative/Positive Symptoms[36,37] were used to assess the participants' psychotic, depressive, and manic symptoms for the 30 days leading up to the evaluation based on the participants recollection. The Schizotypal Personality Questionnaire was used to assess schizotypal traits in participants as possible indicators of manifestations of genetic liability for psychosis. Table 1 presents the demographics information of the participants.

The DPX Task and Conventional Measures

We used the dot pattern version of the AX-CPT cognitive control task, the DPX task, where stimuli were braille-based arrangements of dots.[5] On each trial, a cue stimulus and a probe stimulus were sequentially presented with an interstimulus interval of 2500 or 3500 ms (figure 1A). Cue and probe stimuli were differentiable by being colored white and light blue, respectively. Stimuli were grouped into "valid" and "invalid" categories and only when the cue and probe were both valid was the participant

Table 1. Demographics and Clinical Information of the Participants.

	Controls	Relatives	PwP	
	(*N* = 50)	(*N* = 78)	(*N* = 122)	Tests
Diagnoses/Diagnosis of Relative (*N*)	N/A	Bipolar w/psychosis (24) Schizoaffective disorder (9) Schizophrenia (45)	Bipolar w/psychosis (36) Schizoaffective disorder (14) Schizophrenia (72)	N/A
Age (*SD*)	38.7 (13.0)	44.2 (15.2)	37.1 (12.2)	$F_{(2,247)} = 6.8$, $P = .001$[a]
Female *N* (%)	25 (50%)	50 (64%)	54 (44%)	$\chi^2_{(2)} = 7.56$, $P = .02$ *$P = .15$
Race (*N*)				
American Indian or Alaskan Native	0	0	1	
Asian or Pacific Islander	1	1	6	
Black, not of Hispanic Origin	3	4	19	
Hispanic	0	3	5	
White, not of Hispanic Origin	45	68	87	
Other	1	2	4	
Education (Years)	16.2 (2.5)	15.1 (2.2)	14.1 (2.1)	$F_{(2,246)} = 17.43$, $P < .001$[b]
Parent Education	6	6	6	**$\chi^2_{(2)} = 4.60$, $P = .10$
Estimated IQ (*SD*)	106.9 (11.0)	102.5 (11.0)	98.3 (11.2)	$F_{(2,247)} = 11.28$, $P < .001$[c]
BPRS total (*SD*)	27.7 (4.1)	32.2 (6.5)	45.2 (12.7)	$F_{(2,247)} = 73.95$, $P < .001$[b]
SPQ total (*SD*)	7.4 (7.8)	14.2 (12.5)	29.5 (15.7)	$F_{(2,247)} = 59.66$, $P < .001$[b]

Note: PwP, people with a history of psychosis; BPRS, The Brief Psychiatric Rating Scale-24 Item Version; SPQ, Schizotypal Personality Questionnaire. *Fisher's exact test. Parent education: median of max of parents' education; (1: 7th grade or less; 2: between 7th and 9th grade; 3: between 10th and 12th grade; 4: high school graduate/GED; 5: partial college; 6: college graduate; 7: graduate degree) **Kruskal-Wallis rank sum test. Significant post hoc group differences: a = relative vs PwP; b = all 3 groups differed from one another, c = PwP vs both controls and relatives.

supposed to provide a "target" response with the index finger of their right hand. The only target sequence was the valid cue ("A") followed by the valid probe ("X"), and all other cue-probe combinations were considered nontarget (figure 1A). As such, there were 4 cue-probe permutations: AX, AY, BX, and BY. The participant was to provide a nontarget response with the middle finger of the same hand for any nontarget sequence (ie, AY, BX, or BY). An expectation bias was induced by having 60% of the cue-probe trials being the target sequence (AX). The remaining trials were distributed such that 15% of trials were AY, 15% BX, and 10% BY. Each participant responded to 120 trials that were equally distributed across 3 blocks. While not analyzed in this manuscript, the participants were undergoing functional magnetic resonance imaging (fMRI) during this task.

We evaluated the RT and participant response patterns to restrict the analysis to valid data. Participants were allowed to respond in a 2000 ms response window and we excluded individual responses that were faster than 100 ms (too fast to have recognized the probe stimulus). Any block of trials that was unusable was excluded. An unusable block was defined as the participant providing only 1 type of response regardless of the trial sequence (eg, responding with either key for more than 95% of responded trials in a block) or the participant not responding for more than 50% of trials (see supplementary figure 1 for distribution details). We required that participants have at least 2 of the 3 usable trial blocks to be included in the data set. If a participant's responses were nearly perfectly the opposite of what the instructions indicated during a block, we reversed the participants' responding (ie, nontarget to target and target to nontarget), because participants did not receive feedback while performing the task and, thus, could not correct their understanding of the task.

Based on these criteria, we excluded some of the participants' responses. About 2% of all responses were too fast. Seven participants were excluded due to having less than 2 usable blocks, and we removed an additional 3 unusable blocks from the remaining participants. One participant was identified as having flipped the response buttons given their response patterns, which we corrected before including their behavior in the analysis. In conclusion, data from 250 participants were included in the analysis.

d′-context and Proactive Behavioral Index Calculation

A number of conventional measures have been used in the DPX literature to assess cognitive control. We calculated *d′*-context scores, a measure of context processing, as the difference between correct responses to the AX trial sequence and incorrect responses to the BX trial sequence: $z(AX_{Hit}) - z(BX_{FalseAlarm})$.[6] A correction was applied so that the index could be calculated as suggested by Servan-Shreiber et al. (see supplementary methods).[6] Higher *d′*-context scores are suggestive of better context processing. We calculated proactive behavioral index (PBI) for DPX with accuracy and RT by $(AY - BX)/(AY + BX)$.[3] This measure evaluates

proactiveness by directly comparing error rates or RT of proactive vs reactive trial types. Higher PBIs suggest a preference for proactive control over reactive control. Due to poor psychometric properties (excessively kurtotic with large numbers of outliers), we did not relate the PBI accuracy with the DDM parameters.

Hierarchical DDM

Hierarchical DDMs were applied to the DPX task data across groups using the HDDM 0.9.1 Python package.[38] Since we had a priori reasons to believe that the evidence accumulation process would vary across groups, we fitted the hierarchical DDMs to each group separately. To determine the best description of the participant behaviors, we fitted several models that varied whether the parameter values of a, t, and v parameters were consistent across trial sequences. We decided to only permit w to vary by the cue stimulus because it theoretically should only influence the start of the evidence accumulation process and should, thus, not be modified by the probe stimulus. We identified the best-fit models by comparing the deviance information criteria and by evaluating how well the model converged. Model convergence was assessed via the Gelman-Rubin $\hat{R}$ statistic.[39]

Conventional and DDM Statistical Analyses

All statistical analyses were conducted in R (version 4.3.1)[40] and corrected for age, biological sex, and family-level dependencies. Conventional variables from DPX, including error rates and correct trial RT, and DDM parameters were examined by linear mixed-effect regression models (LMER) using the lme4 package (version 1.1-33).[41] Partial eta squared (η_p^2) were calculated for effect sizes. Post hoc analyses examining interaction and group effects were performed via the emmeans package (version 1.8.7)[42] with multivariate t-distribution adjustments for multiple comparisons. d'-context scores and PBIs were analyzed with one-way analyses of variance (ANOVAs), including post hoc group comparisons corrected by multivariate t-distributions. Since most of the conventional measures were non-Gaussian, correlational analyses between conventional variables and DDM parameters were performed with the robust, nonparametric Kendall's Rank Correlation Tests.[43] We controlled for the false discovery rate (FDR) when making multiple family-wise correlational tests.[44]

eXtreme Gradient Boosting Classifier and Shapley Value Model Explanation

To determine the multivariate predictive utility of DDM parameters, we used a nonparametric machine learning classification approach called eXtreme Gradient Boosting (XGBoost),[45] implemented in the R package xgboost version 1.7.5. This classifier is more stable with a smaller sample size than competing methods like support vector machines.[46,47] Additionally, this classifier is robust to class imbalance,[48] which is ideal because our groups varied in sizes. We fit 3 classifiers to classify PwP from controls, PwP from first-degree relatives, and first-degree relatives from controls. We fit another 3 classifiers to classify across the same groups but with conventional behavioral indices from the DPX: accuracy and RT of all 4 trial types and the PBI-RT, PBI-accuracy, and d'-context. All XGBoost analyses used the area under the receiver operating characteristic curve (AUC) as the objective function. The AUC can range from 0 to 1, where 1 is perfect classification and 0.5 indicates completely random performance for a binary outcome. XGBoost requires several hyperparameters, which were tuned using Bayesian optimization in 25% of the sample. Classification models were then fitted to the remaining 75% of sample data using 5-fold cross-validation to avoid overfitting. Further details are included in supplementary methods.

XGBoost provides a powerful tool for machine learning classification, but multivariate machine learning models like XGBoost can suffer from reduced interpretability due to the black-box nature of these models and the complicated model fitting process. We addressed this problem by applying a recently developed model explanation tool to the output of each XGBoost classifier, using the R package SHAPforxgboost version 0.1.1.[49] SHapley Additive exPlanations (SHAP),[50,51] is an information theoretic approach that explains the output of a machine learning model by ranking the input variables according to which variables had the greatest independent contribution to the output of the classifier. Details of SHAP are included in supplementary methods.

Association Analysis

We performed regression analyses to examine the relationships between DDM parameters and clinical measures across all participants and within the PwP group only. To focus on the most important parameters and minimize multicollinearity, we identified parameters that were ranked more than once within the top 5 for group classification utility and included them as independent variables in the regression models. Similarly, conventional measures of d'-context scores, and PBI-RT were included as independent variables in a separate set of regression models. Factor scores from the BPRS for positive symptoms, negative symptoms, disorganization, mania, and depressive symptoms,[35] and factor scores from the Schizotypal Personality Questionnaire (SPQ) for interpersonal functioning, cognitive-perceptual anomalies, and disorganization were included as dependent variables.[52] Finally, social

and role functioning were examined as dependent variables to evaluate the relationship of cognitive control indices to real-world functioning. Age, sex, and random effects of family were included as covariates when applicable. All DDM parameters, conventional measures, and symptom measures were standardized to enable simple comparisons. FDR corrections within regressions were performed to control for multiple comparisons.

Results

Conventional Measures of DPX

Error Rates. Groups differed in their error rates (figure 1C). A LMER model of error rates revealed a main effect of group ($F_{(2,234.63)} = 4.32$, $P = .01$, $\eta_p^2 = 0.04$), a main effect of trial sequences ($F_{(3,741)} = 33.61$, $P < .001$, $\eta_p^2 = 0.12$), and an interaction between group and trial sequence ($F_{(6,741)} = 2.46$, $P = .02$, $\eta_p^2 = 0.02$). Consistent with past literature, PwP made more errors on BX trials than controls ($t_{(685)} = 3.65$, $P < .001$, $\eta_p^2 = 0.02$) and relatives ($t_{(652)} = -3.86$, $P < .001$, $\eta_p^2 = 0.02$), while controls and relatives did not differ on BX error rates ($t_{(613)} = 0.20$, $P = .98$, $\eta_p^2 < 0.01$). The groups did not differ significantly on the other trial sequences ($|t|$s< 1.49, Ps > .30).

RT. Groups also differed in their RT (figure 2B). A LMER model of the by-individual mean correct response RT showed a main effect of group ($F_{(2,170.39)} = 11.83$, $P < .001$, $\eta_p^2 = 0.12$), a main effect of trial sequence ($F_{(3,733.36)} = 180.59$, $P < .001$, $\eta_p^2 = 0.42$), and an interaction between group and trial sequence ($F_{(6,733.40)} = 4.14$, $P < .001$, $\eta_p^2 = 0.03$). PwP were slower on BX and BY trials compared to controls ($t_{BX(343)} = -4.46$, $P < .001$, $\eta_p^2 = 0.05$; $t_{BY(341)} = -4.36$, $P < .001$, $\eta_p^2 = 0.05$) and relatives ($t_{BX(300)} = 4.27$, $P < .001$, $\eta_p^2 = 0.06$; $t_{BY(299)} = 4.37$, $P < .001$, $\eta_p^2 = 0.06$). PwP were significantly slower on AY trials ($t_{(341)} = -3.69$, $P < .001$, $\eta_p^2 = 0.04$) and AX trials ($t_{(341)} = -2.95$, $P = .009$, $\eta_p^2 = 0.02$) than controls.

d′-context and PBI

One-way ANOVAs revealed group differences in *d′*-context scores ($F_{(2,231.46)} = 8.73$, $P < .001$, $\eta_p^2 = 0.07$) and PBI-RT ($F_{(2,196.84)} = 5.83$, $P = .003$, $\eta_p^2 = 0.06$), and in PBI-accuracy ($F_{(2,144.93)} = 3.21$, $P = .043$, $\eta_p^2 = 0.04$). PwP demonstrated lower *d′*-context scores and PBI-RT than both relatives ($t_{d'\text{-}context(243)} = -3.70$, $P_{d'\text{-}context} < .001$, $\eta_p^2 = 0.05$; $t_{PBI\text{-}RT(205)} = -2.78$, $P_{PBI\text{-}RT} = .02$, $\eta_p^2 = 0.04$) and controls ($t_{d'\text{-}context(245)} = 3.02$, $P_{d'\text{-}context} = .008$, $\eta_p^2 = 0.04$; $t_{PBI\text{-}RT(238)} = 2.68$, $P_{PBI\text{-}RT} = .02$, $\eta_p^2 = 0.03$), suggesting less efficient context processing and less utilization of proactive control. The control and relative groups did not differ from each other on *d′*-context scores or PBI-RT ($t_{d'\text{-}context(2187)} = -0.31$, $P_{d'\text{-}context} = .95$, $\eta_p^2 < 0.01$; $t_{PBI\text{-}RT(230)} = 0.21$, $P_{PBI\text{-}RT} = .98$, $\eta_p^2 < 0.01$). The post hoc group comparisons for PBI accuracy did not survive multiple comparison corrections ($|t|$s < 2.01, Ps > .09).

DDM Parameters

The group RT distributions indicated that the participant's RT were poorly described by their mean RT, and suggested that there were significant group differences in the underlying evidence accumulation process (figure 1D). Thus, we hierarchically fit DDMs to the participant behavior to get individual estimates of the drift rate (v), decision threshold (a), nondecision time (t), and the response bias (w) (figure 1B). We found that the maximally flexible model (figure 2A), which allowed a, t, and v parameters to vary by trial sequence and w by the cue type, was the best description of participant RT and responses (supplementary table 1). All of the best models showed good convergence via Gelman-Rubin with $\hat{R}$s < 1.031 on all individual parameters (supplementary table 2).

Drift Rate (v)

For the sake of comparing across all trial sequences, we made the direction of AX trial drift rates positive (the only target trial; figure 2B). A LMER model examining drift rate across group and trial sequences revealed main effects of group ($F_{(2,245)} = 28.66$, $P < .001$, $\eta_p^2 = 0.19$) and trial sequence ($F_{(3,741)} = 53.02$, $P < .001$, $\eta_p^2 = 0.18$), as well as an interaction between group and trial sequence ($F_{(6,741)} = 5.67$, $P = .001$, $\eta_p^2 = 0.04$). PwP showed lower rates of evidence accumulation than controls (AX: $t_{(708)} = 3.55$, $P = .001$, $\eta_p^2 = 0.02$; BX: $t_{(708)} = 6.45$, $P < .001$, $\eta_p^2 = 0.06$; BY: $t_{(708)} = 5.73$, $P < .001$, $\eta_p^2 = 0.04$) and relatives (AX: $t_{(674)} = -4.19$, $P < .001$, $\eta_p^2 = 0.03$; BX: $t_{(674)} = -5.49$, $P < .001$, $\eta_p^2 = 0.04$; BY: $t_{(674)} = -6.86$, $P < .001$, $\eta_p^2 = 0.07$) across all trials, except for AY trials where PwP showed comparable drift rates to relatives ($t_{(674)} = -2.20$, $P = .07$, $\eta_p^2 < 0.01$) and controls ($t_{(708)} = 1.45$, $P = .32$, $\eta_p^2 < 0.01$). This pattern of findings indicates a specific deficit in evidence accumulation when PwP can utilize proactive control, but not when they are utilizing reactive control. This difference is particularly exacerbated when the cue stimulus provides *definitive* evidence about the correct response (ie, B cue trials), rather than *suggestive* evidence (ie, A cue trials). Relatives did not significantly differ from controls on drift rates across all trials ($|t|$s < 1.44, Ps > .32).

Decision Threshold (a)

There were interactions between group and trial sequences in decision thresholds (figure 2C). An LMER model of decision threshold revealed a main effect of trial sequence ($F_{(3,741.00)} = 92.71$, $P < .001$, $\eta_p^2 = 0.27$) and an interaction between group and trial sequences ($F_{(6,741.00)} = 4.13$, $P < .001$, $\eta_p^2 = 0.03$), but no main effect of group ($F_{(2,177.20)} = 1.96$, $P = .14$, $\eta_p^2 = 0.02$). Relatives had a higher decision threshold than PwP on AX ($t_{(415)} = -2.44$, $P = .040$, $\eta_p^2 = 0.01$) and BY ($t_{(415)} = -3.47$,

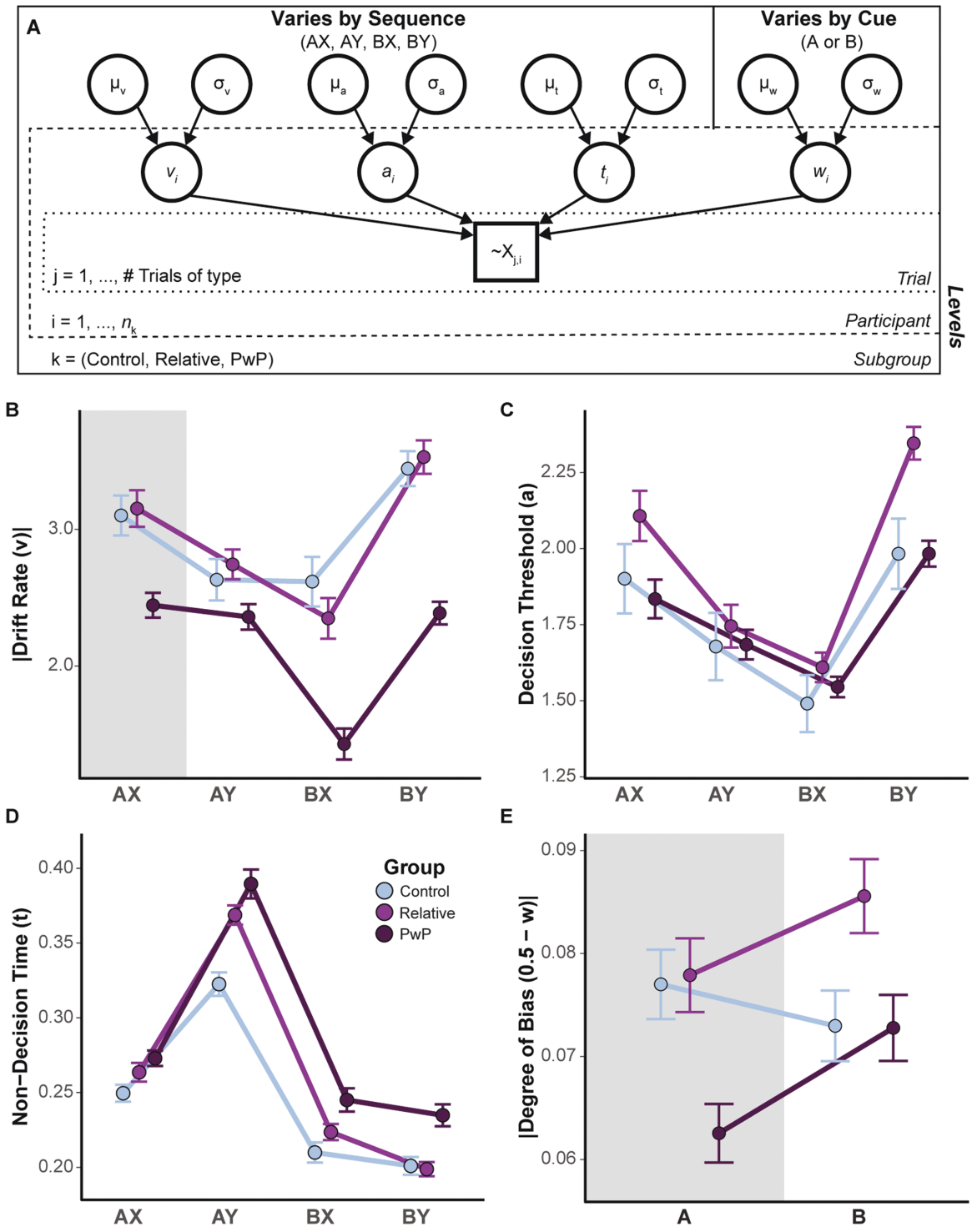

Fig. 2. (**A**) Diagram of the best fitting hierarchical drift diffusion model (DDM) to the distribution of participant responses across trials ($\sim X_{i,j}$). The best fitting hierarchical DDM permitted the rate of evidence accumulation, decision threshold, and nondecision time to vary by trial type (ie, 1 of each parameter for AX, AY, BX, and BY) and the bias to vary by the cue (ie, 1 parameter for A cues and 1 for B cues). μ and σ are the subgroup means and standard deviations of the parameters that are denoted in their subscripts. Each subgroup was independently fitted. (**B**) The means and standard errors of the drift rate parameter by group. The shaded section indicates that the sign of the parameter was negative (ie, toward a target response) prior to taking its absolute value. (**C**) The means and standard errors of the decision threshold by group. (**D**) The means and standard errors of the nondecision time threshold by group. (**E**) The means and standard errors of the degree of bias. The shaded section indicates that the sign of the calculated parameter was negative (ie, toward a target response) prior to taking its absolute value. The absolute value was taken after calculating the mean and standard errors.

$P = .02$, $\eta_p^2 = 0.03$) trials. Relatives had a higher decision threshold than controls on BY trials ($t_{(441)} = -2.95$, $P = .009$, $\eta_p^2 = 0.02$).

Nondecision Time (t)

There were interesting group differences in the nondecision time (figure 2D). An LMER model revealed a main effect of trial sequence ($F_{(3,741.00)} = 397.67$, $P < .001$, $\eta_p^2 = 0.62$), a main effect of group ($F_{(2,245)} = 15.34$, $P < .001$, $\eta_p^2 = 0.11$), and an interaction between group and trial sequence ($F_{(6,741)} = 3.97$, $P < .001$, $\eta_p^2 = 0.03$). The groups did not differ significantly on AX trials in their nondecision times ($|t|$s < 2.29, Ps > .058). However, PwP showed longer nondecision times than controls on AY ($t_{(611)} = -6.16$, $P < .001$, $\eta_p^2 = 0.06$), BX ($t_{(611)} = -3.33$, $P = .003$, $\eta_p^2 = 0.02$), and BY ($t_{(611)} = -3.22$, $P = .004$, $\eta_p^2 = 0.02$) trials. This effect was most pronounced on AY trials, which require reactive control. The longer nondecision times on trials with a B cue suggests that there may be less proactive response planning when there is definitive evidence on the correct response compared to control participants. Relatives showed longer nondecision times than control participants on AY trials ($t_{(543)} = -3.14$, $P = .005$, $\eta_p^2 = 0.02$). Relatives also showed shorter nondecision time on AY ($t_{(580)} = 3.07$, $P = .006$, $\eta_p^2 = 0.02$), BX ($t_{(580)} = 3.13$, $P = .005$, $\eta_p^2 = 0.02$) and BY ($t_{(580)} = 4.59$, $P < .001$, $\eta_p^2 = 0.02$) trials than PwP.

Degree of Bias (w)

To better equate differences in bias we adjusted the value such that it was an estimate of the deviation from indifference (ie, 0.5) (figure 2E). An LMER model on degree of bias revealed a main effect of group ($F_{(2,214.52)} = 4.31$, $P = .01$, $\eta_p^2 = 0.04$), no main effect of the cue ($F_{(1,247)} = 1.52$, $P = .22$, $\eta_p^2 < 0.01$), and no interaction between group and trial sequence ($F_{(2,247)} = 1.19$, $P = .31$, $\eta_p^2 = 0.01$). Post hoc pairwise comparisons showed that PwP showed a lower degree of bias than their relatives ($t_{(223)} = -2.87$, $P = .01$, $\eta_p^2 = 0.04$). PwP did not differ from controls on degree of bias ($t_{(242)} = 1.30$, $P = .39$, $\eta_p^2 < 0.01$).

Classification Models to Determine Utility

Controls vs PwP. An XGBoost model classifying PwP from control participants had high discriminative performance, indicated by a cross-validated area under the curve (AUC) of 0.89. The SHAP explanation of this model (figure 3A) found that the most important variable in this model was BY drift rates, where lower drift rate on BY trials predicted a higher likelihood of being classified as a member of the PwP group. Other variables that were especially important to categorizing participants were *v*-BX, where lower drift rate on BX trials predicted higher likelihood of being labeled PwP, *w*-B, where increased bias predicted a higher likelihood of being classified as a member of the PwP group, *a*-BY, where decreased BY decision thresholds predicted greater likelihood of being labeled PwP, and *t*-AY, where longer AY nondecision times predicted greater likelihood of being labeled PwP.

A comparison model attempting to classify PwP from controls using conventional measures from the DPX produced a cross-validated AUC of 0.69, indicating worse performance than the DDM-parameter-based classification (figure 3E). The majority of the parameters that had the most utility in differentiating PwP from controls were RT indices (AX, AY, and BX trial RT and PBI-RT), with the exception of AX trial accuracy which also showed utility. Given its ubiquity in the literature, it was surprising that d′-context was only the seventh most important parameter for differentiating between controls and PwP. Overall, this comparison supports the utility of RT and the DDM.

Relatives vs PwP. An XGBoost model classifying PwP from their first-degree relatives produced a cross-validated AUC of 0.90, indicating high discrimination performance. The SHAP analysis (figure 3B) found that the most important variable in this model was the drift rate on BY trials where slower drift rates predicted a greater likelihood of PwP membership. Other important variables included *a*-BY, where decreased BY thresholds predicted greater likelihood of being labeled PwP, *v*-BX, where lower drift rate predicted greater likelihood of being labeled PwP, *w*-A, where decreased A-cue bias predicted higher likelihoods of being labeled PwP, and *t*-AY, where longer AY nondecision time predicted higher likelihood of being labeled PwP. A comparison model attempting to classify PwP from their first-degree relatives using conventional measures from the DPX produced a cross-validated AUC of 0.69, indicating a substantial decrease in performance by comparison to the DDM parameters.

Controls vs Relatives. An XGBoost model classifying controls from relatives produced a cross-validated AUC of 0.85, indicating high discrimination performance. The SHAP analysis (figure 3C) found that the most important variable in this model was *a*-BY, where increased BY thresholds predicted higher likelihood of relative membership. Other important variables included *w*-B, where increased B-cue bias predicted higher likelihoods of being labeled relatives, *t*-AY, where longer AY nondecision time predicted higher likelihood of being labeled relatives, *a*-BX, where increased BX thresholds predicted higher likelihood of being labeled relatives, and *w*-A, where decreased A-cue bias predicted higher likelihoods of being labeled relatives. A comparison model attempting to classify relatives from control using traditional behavioral

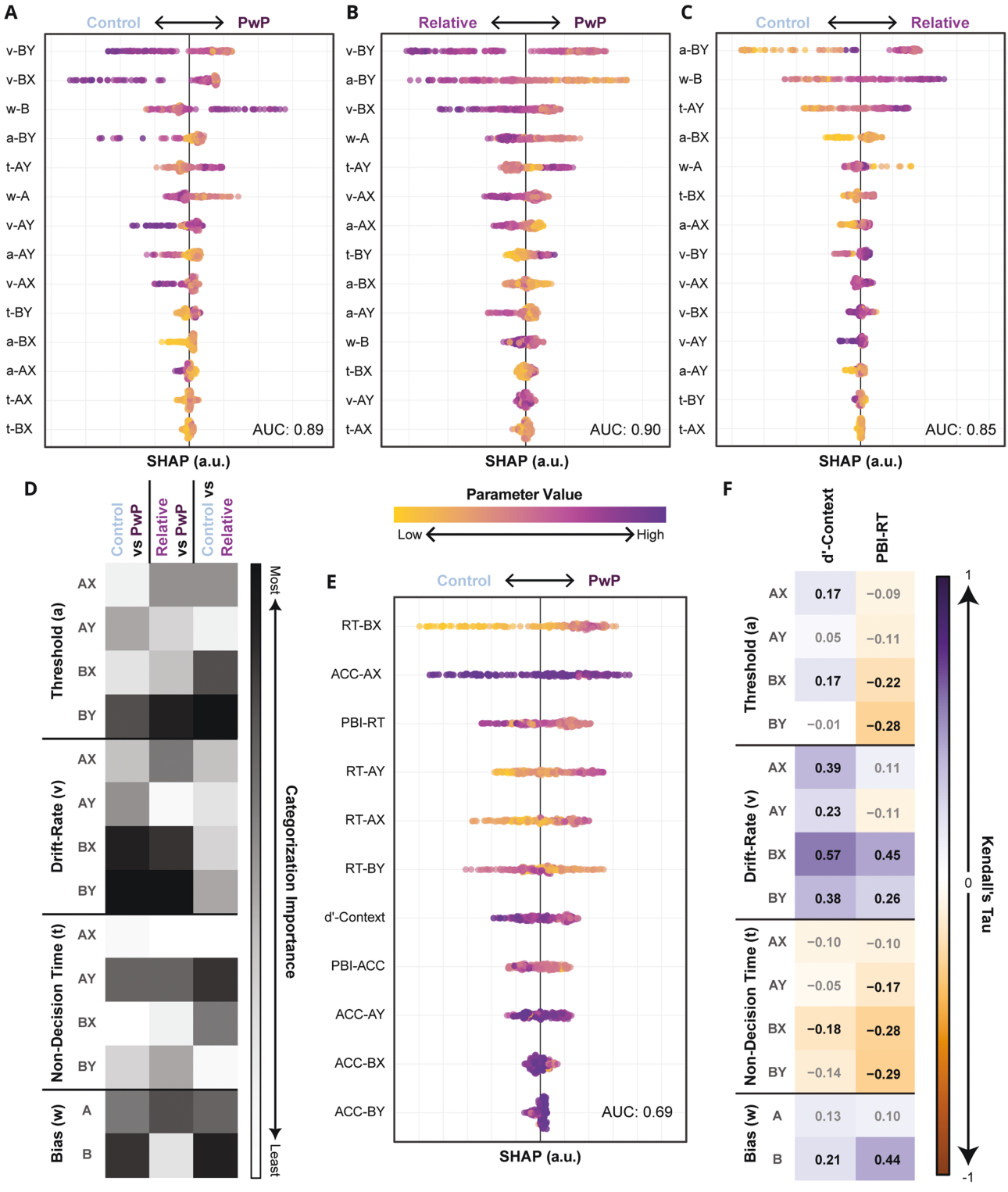

Fig. 3. (**A–C**) SHAP explanations of XGBoost model classifications. Each dot represents a single participant. Dots are arranged along the *X*-axis according to the impact each variable had on the model classification for each individual with the black line in the center indicating zero value. Dots to the left of the zero line indicate that the variable predicted membership in the class label to the left, and dots to the right similarly indicate that the variable predicted membership in the class label to the right. Parameters are listed in the order of classification importance. (**D**) Summary of how important each parameter was for differentiation between the groups. (**E**) SHAP explanation of XGBoost model classification using typical DPX parameters. (**F**) Correlations of the DDM parameters with conventional indices.

indices from the DPX produced a cross-validated AUC of 0.62, indicating a substantial decrease in performance by comparison to the DDM parameters.

Summary of Comparisons

While the exact ordering of the most useful to least useful parameters found by the SHAP analysis for differentiating between groups varied, there were some commonalities across them (figure 3D). The most useful parameters for categorizing group membership were the decision threshold and drift rate on BX and BY trials, the nondecision time on AY trials, and the bias on trials with B cues.

Relationship of DDM Parameters With Conventional Indices

When examining the correlations between conventional DPX measures and DDM parameters, we found that they were highly associated with each other (figure 3F). *d′*-context was positively correlated with drift rate (Kendall's $\tau_{vAX} = 0.39, P_{adj} < .001; \tau_{vAY} = 0.23, P_{adj} < .001; \tau_{vBX} = 0.57, P_{adj} < .001$; $\tau_{vBY} = 0.38, P_{adj} < .001$) and degree of bias ($\tau_{zA} = 0.13, P_{adj} = .047$; $\tau_{zB} = 0.21, P_{adj} = .002$). It was also positively correlated with decision threshold in AX and BX trials ($\tau_{aAX} = 0.17, P_{adj} = .01$; $\tau_{aBX} = 0.17, P_{adj} = .01$), and negatively correlated with nondecision time in BX ($\tau_{tBX} = -0.18, P_{adj} = .007$) and BY trials ($\tau_{tBY} = -0.14, P_{adj} = .046$). PBI-RT was significantly correlated with drift rate, nondecision time, bias, and decision threshold parameters extracted from trials with B cues which assess proactive control ($\tau_{aBX} = -0.22, P_{adj} < .001$; $\tau_{aBY} = -0.28, P_{adj} < .001; \tau_{vBX} = 0.46, P_{adj} < .001; \tau_{vBY} = 0.26, P_{adj} < .001; \tau_{tBX} = -0.28, P_{adj} < .001; \tau_{tBY} = -0.29, P_{adj} < .001; \tau_{zB} = -0.44, P_{adj} < .001$). PBI-RT was also negatively correlated with nondecision time in AY trials ($\tau_{tAY} = -0.17, P_{adj} = .01$).

Relationships With Clinical Symptoms and Community Functioning

We examined clinical symptomatology for associations with DDM parameters selected as informative (ie, *w*-B, *w*-A, *v*-BX, *v*-BY, *t*-AY, and *a*-BY). Across all participants, greater negative symptomatology was associated with longer nondecision time on AY trials ($\beta = 0.27, P_{adj} = .004$). No other symptom factors were associated with the selected DDM parameters. For conventional indices, *d′*-context, but not PBI-RT, was associated with disorganization symptoms ($\beta = -0.16, P_{adj} = .02$).

We also examined schizotypal traits for associations with DDM parameters as possible indicators of genetic liability for psychosis. Greater difficulties with social-interpersonal functioning were associated with longer nondecision time on AY trials ($\beta = 0.27, P_{adj} = .001$), slower drift rates on BX trials ($\beta = -0.31, P_{adj} = .001$) and lower decision boundary on BY trials ($\beta = -0.18, P_{adj} = .04$). More anomalous cognitive-perceptual functioning was also associated with longer nondecision time on AY trials ($\beta = 0.23, P_{adj} = .01$) and slower drift rates on BX trials ($\beta = -0.23, P_{adj} = .02$). Greater difficulties with social interpersonal functioning were associated with lower *d′*-context scores ($\beta = -0.19, P_{adj} = .009$). Greater difficulties with cognitive-perceptual functioning were also associated with lower *d′*-context scores ($\beta = -0.14, P_{adj} = .03$), as well as lower PBI-RT ($\beta = -0.19, P_{adj} = .007$).

To better understand how well laboratory-based assessment of cognitive control predicted real-world functioning we investigated whether DDM parameters were associated with measures of social and role functioning. Higher social functioning was predicted by faster drift rates on BX trials ($\beta = 0.27, P_{adj} = .004$), shorter nondecision time on AY trials ($\beta = -0.25, P_{adj} = .004$), and higher thresholds on BY trials ($\beta = 0.23, P_{adj} = .004$). Higher role functioning was predicted by faster drift rates on BY trials ($\beta = 0.25, P_{adj} = .007$) and shorter nondecision times on AY trials ($\beta = -0.26, P_{adj} = .003$). Our examination of conventional cognitive control indices revealed that higher global social functioning was associated with higher *d′*-context scores ($\beta = 0.18, P_{adj} = .006$) and higher PBI-RT ($\beta = -0.23, P_{adj} = .02$). Higher global role functioning was associated with higher *d′*-context scores ($\beta = 0.28, P_{adj} < .001$). Within PwP association results were included in supplementary results.

Discussion

We applied hierarchical DDMs to behavior on the DPX task and found that it provided novel insights into cognitive control deficits in psychotic psychopathology. The DDM revealed that cognitive control deficits in psychosis are mainly due to two differences in the underlying decision process. First, slower drift rates across AX, BX, and BY trials suggest a deficit in accumulating evidence to respond when the cue stimulus is informative. This is further emphasized by the absence of a similar deficit on AY trials, which are typically responded to correctly and efficiently by refraining from a prepotent response. Second, we observed longer nondecision times for PwP during all but AX trials. The slowed nondecision time on proactive control trials (ie, BX and BY trials) suggests that PwP take longer to represent the probe stimulus or access working memory of the B cues. It is important to note that the nondecision time parameters estimated by DDM may not map directly onto sensory and motor delays.[52] On the reactive AY trials, the DDM identified protracted nondecision time and intact rate of evidence accumulation in PwP. Poorer proactive control in individuals with psychosis would theoretically improve performance on AY trials due to lower prepotency to override. It could be that we do not observe any difference in error rates on

AY trials but observe longer response times, because a deficit in evidence accumulation on AY trials is obscured by less response anticipation for the target response on A cue trials. This is partially supported by the tendency toward lower A bias in PwP (figure 2E).

While we hypothesized a reduced bias in PwP due to previously observed deficits in proactive control and simulation of the underlying process,[27] findings were mixed. PwP showed lower bias than their relatives after controlling for age and sex differences, but their bias was not significantly different from controls. It could be that we failed to detect an overall difference in degree of bias due to the heterogeneity of our PwP sample which included patients with bipolar disorder. Evidence for this comes from the XGBoost classification which revealed that *increased* B-cue bias differentiated PwP from controls, but only for a subset of participants (figure 3A). Additionally, XGBoost classification highlighted the possibility that degree of bias on B trials is reflective of genetic liability, given that it is similarly important for differentiating relatives from controls. Drift rates, on the other hand, for BX and BY trials may reflect the actual occurrence of the disease, as these indices played an important role in separating PwP from relatives and controls, but not relatives from controls.

To our knowledge, this is the first study to investigate cognitive control in psychotic psychopathology using full-parameter DDMs. DDM findings of decreased drift rates and longer nondecision time in PwP are consistent with previous findings on sustained attention, coding, punishment, and reward anticipation tasks.[20,21,23,24] The current results suggest that the typically observed inefficiencies in information processing extend to cognitive control, especially on those trials where responses could be proactively planned. The DDM was also able to identify atypical response patterns in relatives that went undetected with conventional measures. In general, conventional indices of cognitive control on the DPX were less useful for differentiating between groups than we expected and had interesting relationships with the DDM parameters. d'-context was most correlated with DDM parameters on BX trials, but there were also significant correlations with the drift rate on trials with other trial sequences. Since the drift rate parameter captures error rates more than the other parameters, this suggests that d'-context is measuring general error rates and may be less specific to contextual processing. PBI-RT was more informative for differentiating between groups than d'-context. Given its basis in RT, PBI-RT captured the nondecision time differences and was primarily related to DDM parameters of B cue trials, consistent with a measure of proactive control. Nevertheless, conventional indices seem to lack an index that specifically taps into the nondecision time on AY trials which showed utility in the group membership classification.

In investigating the clinical relevance of the most fruitful DDM and conventional parameters, we identified associations between DDM parameters and genetic liability for psychotic psychopathology as reflected in schizotypal traits. Consistent with past literature,[53] proactive control was associated with schizotypal traits as assessed by questionnaire (SPQ). Interpersonal-social difficulties and cognitive-perceptual anomalies were related to slowed evidence accumulation on proactive control (BX) trials. Similar associations with protracted nondecision times for reactive control (AY) trials suggest that both cognitive and sensory processes are manifestations of genetic liability for psychosis. This also suggests that genetic liability of psychotic psychopathology is associated with aspects of reactive control. Conventional measures (d'-context and PBI-RT) showed associations with schizotypal traits consistent with them capturing proactive control tied to genetic liability. Additionally, we found worse real-world functioning to be related to protracted nondecision times on reactive control trials and slowed evidence accumulation on proactive control trials. Conventional measures of d'-context and PBI-RT showed similar relationships with measures of real-world functioning. The clinical implication is that the modeling of DPX could be particularly useful in capturing the slowed decision-making that impairs cognitive control and functioning outside research settings, even in individuals who do not manifest a clinical disorder.

Relation of the DDM Model to Attractor Network and State Processes, Implications for Psychosis

Models of psychosis suggest that symptoms arise from representations of information becoming "noisy."[27,28,54,55] This hypothetical mechanism can be understood via attractor state dynamics, to which DDMs have been found to be mathematically similar.[56–59] Attractor state models can be envisioned as an energy landscape (figure 4A) with the final pattern as the low-energy state (a valley, called the "basin of attraction"). The non-decision-time parameter in the DDM maps onto (figure 4B) the period of time before neural activity starts to systematically move toward a specific basin of attraction. The rate of evidence accumulation is how quickly the neural activity moves toward the basin of attraction once the decision process is initiated. The decision threshold parameter is the gap between the basins of attraction, and the bias parameter is when the starting point favors 1 basin over another. Importantly, the depths of these basins depend on the strength of the synaptic interconnections between the neurons within the pattern.[27,57,60–63] Psychoses may arise from a flattening of the basin, so that the patterns do not as easily fall into the right option.[27,28,54,55] In the DDM mathematics, this shows up as a decrease in the drift rate (v) with lower drift rates

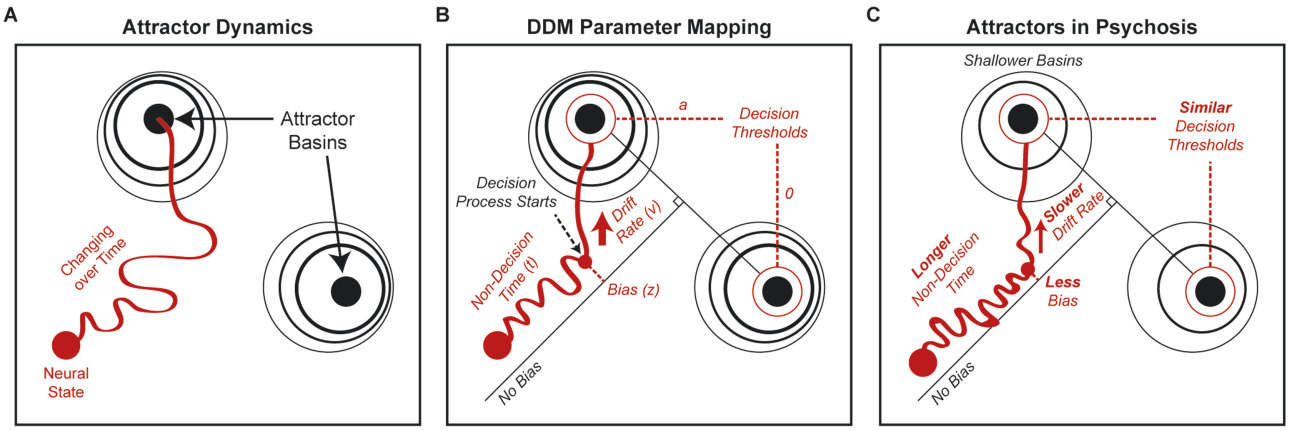

Fig. 4. Mapping between attractor dynamics and DDM parameters. (**A**) During tasks, the neural state (ie, the pattern of neural activity that reflects a representation of information) changes over time until it falls into an attractor basin, which leads to a typical response to a stimulus. (**B**) DDMs can be thought to reflect this same dynamic. The parameters from the DDM can be mapped to the changes in neural state over time. (**C**) The changes in attractor dynamics in psychosis suggested by DDM.

implying shallower basins (figure 4C). As shown in figure 2, the largest difference between PwP and both relatives and controls is the drift rate, with PwP having a significantly lower drift rate consistent with shallower basins of attraction and less stable neural states. Additionally, PwP show longer nondecision times (t), implying that it takes longer for them to start moving toward an attractor basin. The fact that decision threshold (a) is mostly unaffected suggests that the downstream systems are able to interpret the neural patterns correctly—once that pattern arrives.

Limitations

One limitation of this study is that we had to use hierarchical DDMs to estimate individual parameters, which uses information from the group level to influence parameters at the participant level. This approach could have caused our XGBoost method and association studies to overestimate the area under the curve and the strengths of the associations compared to other grouping approaches. Attempting to fit all participants as a single group is also problematic, however, because it would obscure group differences by assuming a homogeneity across groups. It would be beneficial to conduct an experiment with an order of magnitude more trials to estimate the parameters of individual subjects without the influence of other participants. Within this current study, it is best to interpret the XGBoost and SHAP results as the order of the most useful parameters for categorization and to be somewhat cautious of the AUC estimates.

Overall, our results provide unique and useful information about underlying cognitive control mechanisms that are not captured by conventional measures. By analyzing accuracy and RT from a DDM perspective, we learned that slowed motor/perceptual time as well as inefficiencies in proactive evidence accumulation likely contribute to deficits in cognitive control in psychosis. DDM also revealed atypical trial-level patterns in relatives that went undetected through conventional analyses. Our findings also provide additional support for a deficit in proactive control in psychotic psychopathology, and further highlight the importance of perceptual and motor functions in understanding compromised cognitive control in people with a history of psychosis.

Supplementary Material

Supplementary material is available at https://academic.oup.com/schizophreniabulletin.

Acknowledgments

The authors would like to thank Angus W. MacDonald III who provided helpful feedback on an earlier version of this manuscript. The authors have declared that there are no conflicts of interest in relation to the subject of this study.

Funding

This work was supported by the National Institutes of Health (T32-DA037183, U01MH108150, and P50MH119569).

References

1. Braver TS. The variable nature of cognitive control: a dual mechanisms framework. *Trends Cogn Sci.* 2012;16(2):106–113. doi:10.1016/j.tics.2011.12.010
2. Braver TS, Barch DM, Keys BA, et al. Context processing in older adults: evidence for a theory relating cognitive control to neurobiology in healthy aging. *J Exp Psychol Gen.* 2001;130(4):746–763. doi:10.1037/0096-3445.130.4.746

3. Braver TS, Paxton JL, Locke HS, Barch DM. Flexible neural mechanisms of cognitive control within human prefrontal cortex. *Proc Natl Acad Sci USA.* 2009;106(18):7351–7356. doi:10.1073/pnas.0808187106
4. Cohen JD, Barch DM, Carter C, Servan-Schreiber D. Context-processing deficits in schizophrenia: converging evidence from three theoretically motivated cognitive tasks. *J Abnorm Psychol.* 1999;108(1):120–133. doi:10.1037/0021-843X.108.1.120
5. MacDonald AW, Goghari VM, Hicks BM, Carter CS, Flory JD, Manuck SB. A convergent–divergent approach to context processing, general intellectual functioning, and the genetic liability to schizophrenia. *Neuropsychology.* 2005;19(6):8.
6. Servan-Schreiber D, Cohen JD, Steingard S. Schizophrenic deficits in the processing of context: a test of a theoretical model. *Arch Gen Psychiatry.* 1996;53(12):1105–1112. doi:10.1001/archpsyc.1996.01830120037008
7. Poppe AB, Barch DM, Carter CS, et al. Reduced frontoparietal activity in schizophrenia is linked to a specific deficit in goal maintenance: a multisite functional imaging study. *Schizophr Bull.* 2016;42(5):1149–1157. doi:10.1093/schbul/sbw036
8. Stephenson DD, El Shaikh AA, Shaff NA, et al. Differing functional mechanisms underlie cognitive control deficits in psychotic spectrum disorders. *J Psychiatry Neurosci.* 2020;45(6):430–440. doi:10.1503/jpn.190212
9. Lesh TA, Westphal AJ, Niendam TA, et al. Proactive and reactive cognitive control and dorsolateral prefrontal cortex dysfunction in first episode schizophrenia. *NeuroImage Clin.* 2013;2:590–599. doi:10.1016/j.nicl.2013.04.010
10. Jones JAH, Sponheim SR, MacDonald AW. The dot pattern expectancy task: reliability and replication of deficits in schizophrenia. *Psychol Assess.* 2010;22(1):131–141. doi:10.1037/a0017828
11. Poppe AB, Carter CS, Minzenberg MJ, MacDonald AW. Task-based functional connectivity as an indicator of genetic liability to schizophrenia. *Schizophr Res.* 2015;162(1–3):118–123. doi:10.1016/j.schres.2014.11.022
12. Delawalla Z, Csernansky JG, Barch DM. Prefrontal cortex function in nonpsychotic siblings of individuals with schizophrenia. *Biol Psychiatry.* 2008;63(5):490–497. doi:10.1016/j.biopsych.2007.05.007
13. MacDonald AW, Pogue-Geile MF, Johnson MK, Carter CS. A specific deficit in context processing in the unaffected siblings of patients with schizophrenia. *Arch Gen Psychiatry.* 2003;60(1):57–65. doi:10.1001/archpsyc.60.1.57
14. Reilly JL, Hill SK, Gold JM, et al. Impaired context processing is attributable to global neuropsychological impairment in schizophrenia and psychotic bipolar disorder. *Schizophr Bull.* 2016;43(2):397–406. doi:10.1093/schbul/sbw081
15. Richard AE, Carter CS, Cohen JD, Cho RY. Persistence, diagnostic specificity and genetic liability for context-processing deficits in schizophrenia. *Schizophr Res.* 2013;147(1):75–80. doi:10.1016/j.schres.2013.02.020
16. Lee J, Park S. The role of stimulus salience in CPT-AX performance of schizophrenia patients. *Schizophr Res.* 2006;81(2–3):191–197. doi:10.1016/j.schres.2005.08.015
17. Lopez-Garcia P, Young Espinoza L, Molero Santos P, Marin J, Ortuño Sanchez-Pedreño F. Impact of COMT genotype on cognition in schizophrenia spectrum patients and their relatives. *Psychiatry Res.* 2013;208(2):118–124. doi:10.1016/j.psychres.2012.09.043
18. MacDonald AW, Carter CS. Event-Related fMRI study of context processing in dorsolateral prefrontal cortex of patients with schizophrenia. *J Abnorm Psychol.* 2003;112(4):689–697. doi:10.1037/0021-843X.112.4.689
19. Smucny J, Barch DM, Gold JM, et al. Cross-diagnostic analysis of cognitive control in mental illness: insights from the CNTRACS consortium. *Schizophr Res.* 2019;208:377–383. doi:10.1016/j.schres.2019.01.018
20. Mathias SR, Knowles EEM, Barrett J, et al. The processing-speed impairment in psychosis is more than just accelerated aging. *Schizophr Bull.* 2017;43(4):814–823. doi:10.1093/schbul/sbw168
21. Fish S, Toumaian M, Pappa E, et al. Modelling reaction time distribution of fast decision tasks in schizophrenia: evidence for novel candidate endophenotypes. *Psychiatry Res.* 2018;269:212–220. doi:10.1016/j.psychres.2018.08.067
22. Gupta A, Bansal R, Alashwal H, Kacar AS, Balci F, Moustafa AA. Neural substrates of the drift-diffusion model in brain disorders. *Front Comput Neurosci.* 2022;15:678232. doi:10.3389/fncom.2021.678232
23. Moustafa AA, Kéri S, Somlai Z, et al. Drift diffusion model of reward and punishment learning in schizophrenia: modeling and experimental data. *Behav Brain Res.* 2015;291:147–154. doi:10.1016/j.bbr.2015.05.024
24. Smucny J, Hanks TD, Lesh TA, O'Reilly RC, Carter CS. Altered associations between motivated performance and frontostriatal functional connectivity during reward anticipation in schizophrenia. *Schizophr Bull.* 2023;49(3):717–725. doi:10.1093/schbul/sbac204
25. Smucny J, Hanks TD, Lesh TA, Carter CS. Altered associations between task performance and dorsolateral prefrontal cortex activation during cognitive control in schizophrenia. *Biol Psychiatry Cogn Neurosci Neuroimaging.* 2023;8:S2451902223001301. doi:10.1016/j.bpsc.2023.05.010
26. Limongi R, Bohaterewicz B, Nowicka M, Plewka A, Friston KJ. Knowing when to stop: aberrant precision and evidence accumulation in schizophrenia. *Schizophr Res.* 2018;197:386–391. doi:10.1016/j.schres.2017.12.018
27. Calvin OL, Redish AD. Global disruption in excitation-inhibition balance can cause localized network dysfunction and Schizophrenia-like context-integration deficits. *PLoS Comput Biol.* 2021;17(5):e1008985. doi:10.1371/journal.pcbi.1008985
28. Lam NH, Borduqui T, Hallak J, et al. Effects of altered excitation-inhibition balance on decision making in a cortical circuit model. *J Neurosci.* 2022;42(6):1035–1053. doi:10.1523/JNEUROSCI.1371-20.2021
29. Limongi R, Jeon P, Mackinley M, et al. Glutamate and dysconnection in the salience network: neurochemical, effective connectivity, and computational evidence in schizophrenia. *Biol Psychiatry.* 2020;88(3):273–281. doi:10.1016/j.biopsych.2020.01.021
30. Jeste DV, Palmer BW, Appelbaum PS, et al. A new brief instrument for assessing decisional capacity for clinical research. *Arch Gen Psychiatry.* 2007;64(8):966–974. doi:10.1001/archpsyc.64.8.966
31. Demro C, Mueller BA, Kent JS, et al. The psychosis human connectome project: an overview. *Neuroimage.* 2021;241:118439. doi:10.1016/j.neuroimage.2021.118439
32. First MB, Spitzer RL, Gibbon M, Williams JBW. *Structured Clinical Interview for DSM-IV-TR Axis I Disorders, Patient Edition (SCID-I/NP, 11/2002 Revision).* New York, NY: Biometrics Research Department, New York State Psychiatric Institute; 2002.
33. Nurnberger JI, Blehar MC, Kaufmann CA, et al. Diagnostic interview for genetic studies: rationale, unique features, and

training. *Arch Gen Psychiatry.* 1994;51(11):849–59; discussion 863. doi:10.1001/archpsyc.1994.03950110009002

34. Lukoff D, Liberman RP, Nuechterlein KH. Symptom monitoring in the rehabilitation of schizophrenic patients. *Schizophr Bull.* 1986;12(4):578–602. doi:10.1093/schbul/12.4.578
35. Wilson S, Sponheim SR. Dimensions underlying psychotic and manic symptomatology: extending normal-range personality traits to schizophrenia and bipolar spectra. *Compr Psychiatry.* 2014;55(8):1809–1819. doi:10.1016/j.comppsych.2014.07.008
36. Andreasen NC. *The Scale for the Assessment of Negative Symptoms (SANS)*. Cambridge, United Kingdom: Cambridge University Press; 1981.
37. Andreasen NC. *The Scale for the Assessment of Positive Symptoms (SAPS)*. Cambridge, United Kingdom: Cambridge University Press; 1983.
38. Wiecki TV, Sofer I, Frank MJ. HDDM: hierarchical Bayesian estimation of the drift-diffusion model in python. *Front Neuroinform*. 2013;7:14. doi:10.3389/fninf.2013.00014
39. Gelman A, Carlin JB, Stern HS, Dunson DB, Vehtari A, Rubin DB. *Bayesian Data Analysis*. Boca Raton, FL: Chapman and Hall/CRC; 2013. doi:10.1201/b16018
40. R Core Team. *R: A Language and Environment for Statistical Computing*. Published online 2023. https://www.R-project.org/. Accessed November 21, 2023.
41. Bates D, Mächler M, Bolker B, Walker S. Fitting linear mixed-effects models using lme4. *J Stat Softw*. 2015;67(1):1–48. doi:10.18637/jss.v067.i01
42. Lenth R. *emmeans: Estimated Marginal Means, aka Least-Squares Means*. Published online 2022. https://CRAN.R-project.org/package=emmeans. Accessed November 21, 2023.
43. Kendall MG. A new measure of rank correlation. *Biometrika*. 1938;30:81–93.
44. Benjamini Y, Hochberg Y. Controlling the false discovery rate: a practical and powerful approach to multiple testing. *J R Stat Soc Ser B Methodol.* 1995;57(1):289–300. doi:10.1111/j.2517-6161.1995.tb02031.x
45. Chen T, Guestrin C. XGBoost: a scalable tree boosting system. In: Proceedings of the 22nd ACM SIGKDD International Conference on Knowledge Discovery and Data Mining. KDD'16. Association for Computing Machinery; 2016:785–794. doi:10.1145/2939672.2939785
46. Floares AG, Ferisgan M, Onita D. The smallest sample size for the desired diagnosis accuracy. *International Journal of Oncology and Cancer Therapy.* Vol 2. 2017.
47. Mukherjee S, Tamayo P, Rogers S, et al. Estimating dataset size requirements for classifying DNA microarray data. *J Comput Biol.* 2003;10(2):119–142. doi:10.1089/106652703321825928
48. Wang C, Deng C, Wang S. Imbalance-XGBoost: leveraging weighted and focal losses for binary label-imbalanced classification with XGBoost. *Pattern Recognit Lett.* 2020;136:190–197. doi:10.1016/j.patrec.2020.05.035
49. Just AC, Liu Y, Sorek-Hamer M, et al. Gradient boosting machine learning to improve satellite-derived column water vapor measurement error. *Atmospheric Meas Tech*. 2020;13(9):4669–4681. doi:10.5194/amt-13-4669-2020
50. Lundberg S, Lee SI. *A Unified Approach to Interpreting Model Predictions*. Published online November 24, 2017. http://arxiv.org/abs/1705.07874. Accessed August 14, 2023.
51. Lundberg SM, Erion GG, Lee SI. *Consistent Individualized Feature Attribution for Tree Ensembles*. Published online March 6, 2019. http://arxiv.org/abs/1802.03888. Accessed August 14, 2023.
52. Calkins ME, Curtis CE, Grove WM, Iacono WG. Multiple dimensions of schizotypy in first degree biological relatives of schizophrenia patients. *Schizophr Bull.* 2004;30(2):317–325. doi:10.1093/oxfordjournals.schbul.a007081
53. Steffens M, Meyhöfer I, Fassbender K, Ettinger U, Kambeitz J. Association of schizotypy with dimensions of cognitive control: a meta-analysis. *Schizophr Bull.* 2018;44(suppl_2):S512–S524. doi:10.1093/schbul/sby030
54. Compte A, Brunel N, Goldman-Rakic PS, Wang XJ. Synaptic mechanisms and network dynamics underlying spatial working memory in a cortical network model. *Cereb Cortex.* 2000;10(9):910–923. doi:10.1093/cercor/10.9.910
55. Durstewitz D, Seamans JK. The dual-state theory of prefrontal cortex dopamine function with relevance to catechol-o-methyltransferase genotypes and schizophrenia. *Biol Psychiatry.* 2008;64(9):739–749. doi:10.1016/j.biopsych.2008.05.015
56. Wang XJ. Probabilistic decision making by slow reverberation in cortical circuits. *Neuron.* 2002;36(5):955–968. doi:10.1016/S0896-6273(02)01092-9
57. Hertz J, Krogh A, Palmer RG. *Introduction to the Theory of Neural Computation*. Boston, MA: Addison-Wesley Pub. Co; 1991.
58. Wang S, Falcone R, Richmond B, Averbeck BB. Attractor dynamics reflect decision confidence in macaque prefrontal cortex. *Nat Neurosci.* 2023;26(11):1970–1980. doi:10.1038/s41593-023-01445-x
59. Wong KF, Wang XJ. A recurrent network mechanism of time integration in perceptual decisions. *J Neurosci.* 2006;26(4):1314–1328. doi:10.1523/JNEUROSCI.3733-05.2006
60. Kurth-Nelson Z, Bickel W, Redish AD. A theoretical account of cognitive effects in delay discounting. *Eur J Neurosci.* 2012;35(7):1052–1064. doi:10.1111/j.1460-9568.2012.08058.x
61. Kahana MJ. Computational models of memory search. *Annu Rev Psychol.* 2020;71(1):107–138. doi:10.1146/annurev-psych-010418-103358
62. Runge CF, Johnson JA, Nelson E, Redish AD. A neuroscience-based analysis of impacts of disaster memory on economic valuation. *J Neurosci Psychol Econ*. 2023;16(1):24–49. doi:10.1037/npe0000168
63. Eliasmith C, Anderson CH. *Neural Engineering: Computational, Representation, and Dynamics in Neurobiological Systems*, Vol 1. Cambridge, MA: MIT Press; 2004.