Shared and disorder-specific computational mechanisms of interference and response inhibition in schizophrenia and major depressive disorder

Running title: Computational Phenotyping of Inhibitory Control in SCZ and MDD

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Abstract

Inhibitory control deficits are consistently observed in both schizophrenia (SCZ) and major depressive disorder (MDD), yet whether these impairments reflect shared transdiagnostic mechanisms or disorder-specific dysfunction remains poorly understood. We recruited $N = 259$ participants (n = 87 SCZ, n = 86 MDD, n = 86 healthy controls) who completed Stroop and Go/No-Go tasks assessing interference and response inhibition, respectively. Hierarchical drift-diffusion modeling (HDDM) was used to decompose behavioral performance into latent computational parameters. Both patient groups exhibited significant behavioral impairments, with SCZ showing greater severity. At the computational level, reduced drift rate—reflecting less efficient evidence accumulation—emerged as a core transdiagnostic deficit across both inhibitory subcomponents and both disorders, with more pronounced reductions in SCZ. In contrast, prolonged non-decision time exhibited disorder-specific patterns: it was consistently

elevated across both tasks in SCZ, but confined to response inhibition in MDD. Clinical correlates also diverged; computational parameters in SCZ showed trait-like associations with negative symptoms and illness duration, whereas those in MDD exhibited state-dependent associations with current symptom severity. Machine learning classification corroborated these findings: drift rate served as the primary shared contributor to patient–control discrimination (area under the curve [AUC] = 0.762–0.846), while non-decision time on the interference task emerged as the key feature distinguishing between the two disorders (AUC = 0.718). These findings delineate shared versus disorder-specific computational signatures of inhibitory control impairment in SCZ and MDD, providing support for computational phenotyping as a tool for advancing precision psychiatry.

Introduction

Cognitive impairment is increasingly recognized as a core feature of both schizophrenia (SCZ) and major depressive disorder (MDD), one that substantially compromises social functioning and daily living while also serving as an important predictor of long-term prognosis [1–3]. Yet it remains unclear whether the cognitive deficits observed in these two disorders arise from shared or distinct underlying mechanisms. To date, research has largely focused on describing cognitive impairment phenotypes—such as global executive dysfunction or attentional deficits—without establishing fine-grained cognitive markers capable of distinguishing between diagnoses or predicting treatment response [3, 4]. This has hindered the identification of precise intervention targets and the development of individualized treatment strategies. Addressing this gap requires a theoretical framework that moves beyond categorical diagnoses to examine the dimensional structure of cognitive processes.

In this regard, the Research Domain Criteria (RDoC) framework offers a valuable theoretical perspective, encouraging a shift in psychopathology research from diagnosis-centered approaches toward transdiagnostic, dimensional investigations of underlying mechanisms [5, 6]. Within this framework, SCZ and MDD may share certain pathophysiological mechanisms across several key cognitive dimensions. Indeed, previous research has demonstrated that these two disorders share common neurobiological foundations at multiple levels, including neurotransmitter systems (e.g., dopamine and glutamate), brain structure (e.g., reduced hippocampal volume), and genetic susceptibility [7–9]. Importantly,

the existence of such transdiagnostic commonalities does not preclude disorder-specific impairments; rather, it underscores the importance of identifying both the cognitive processes that reflect general vulnerability and those that are unique to each disorder. Clarifying this distinction remains an important prerequisite for achieving precision diagnosis and stratified intervention [10].

Inhibitory control is a core construct within the RDoC cognitive systems domain. It refers to the ability to actively suppress irrelevant stimuli, interfering mental processes, and prepotent behavioral responses [11]. Its development depends on the maturation and integration of prefrontal–cingulate circuits and related prefrontal–striatal–thalamic networks [12]. Notably, inhibitory control exhibits a differential impairment pattern that makes it particularly well-suited for investigating both transdiagnostic and disorder-specific features. A substantial body of research has demonstrated that inhibitory control is broadly and consistently impaired in SCZ, whereas in MDD, significant impairments emerge primarily under specific conditions, such as when processing negative emotional material, under high interference load, or when dealing with self-relevant content [3, 13–16].

At the paradigm level, inhibitory control deficits in SCZ have been consistently observed across Go/No-Go, stop-signal, and Stroop tasks [13, 15, 17, 18], whereas impairments in MDD tend to be more context-dependent and closely associated with difficulty suppressing negative, self-relevant information [14, 16]. Importantly, from a structural perspective, inhibitory control is not a unitary construct. The literature generally divides it into two relatively separable yet interrelated subcomponents, namely response inhibition and interference inhibition [11, 19]. Response inhibition is typically assessed using paradigms such as the Go/No-Go and stop-signal tasks, whereas interference inhibition is commonly measured using conflict tasks such as the Stroop and flanker tasks [12, 20]. Given the differential impairment profiles observed in SCZ and MDD, examining both subcomponents within the same study may reveal whether these disorders diverge in their patterns of response versus interference inhibition deficits.

Given their distinct pathophysiological profiles, SCZ and MDD may also exhibit different patterns of computational impairment on inhibitory control tasks. Cognitive impairment in SCZ has long been associated with abnormal dopamine signaling in prefrontal–striatal circuits, which impairs working memory updating and context maintenance, thereby reducing the efficiency of evidence integration [21]. Accordingly, patients with SCZ may primarily show a

generalized reduction in the efficiency of extracting and integrating decision-relevant information during inhibitory tasks—a pattern consistent with previous findings of inadequate proactive control and prolonged stop-signal reaction time [13, 22]. In contrast, cognitive impairment in MDD is closely linked to difficulties in emotion regulation and negative cognitive biases; specifically, patients tend to over-process negative, self-relevant, or failure-related information and have difficulty redirecting cognitive resources to current task goals in a timely manner [14]. This tendency toward cautious, avoidant processing styles may lead patients with MDD to adopt more conservative decision strategies—requiring more evidence before committing to a response—as a compensatory mechanism to avoid errors. At the computational level, such a pattern may manifest as changes in decision boundary settings or evidence accumulation biases toward specific stimulus categories, rather than a global reduction in processing efficiency. This hypothesis aligns with empirical findings demonstrating that patients with MDD show imbalanced allocation between anticipatory (proactive) and stimulus-driven (reactive) control processes, as well as abnormal conflict adaptation [16, 23].

However, traditional behavioral measures cannot directly test these hypotheses because reaction time and accuracy reflect the combined influence of multiple cognitive subprocesses, including stimulus encoding, evidence accumulation, decision boundary setting, and response execution [24]. When two patient groups show prolonged reaction times and elevated error rates, the underlying sources of their impairments may be entirely different, making it difficult to pinpoint the true locus of the deficit from behavioral data alone. The hierarchical drift-diffusion model (HDDM) provides a powerful tool for addressing this limitation. Grounded in sequential sampling theory, the HDDM can decompose reaction time distributions and accuracy rates into latent parameters with clear psychological interpretations [24, 25]. The core parameters include drift rate (v), reflecting the efficiency of evidence accumulation; decision boundary (a), reflecting the speed–accuracy trade-off strategy; non-decision time (t), reflecting the duration of perceptual encoding and motor execution processes; and starting point bias (z), reflecting a prior response inclination. Crucially, these parameters align well with the known pathophysiology of SCZ and MDD. Specifically, the dopaminergic dysregulation in prefrontal–striatal circuits characteristic of SCZ would be expected to reduce drift rate [21], whereas the heightened risk aversion and uncertainty intolerance observed in MDD may

manifest as an elevated decision boundary or biased starting point [26, 27]. Indeed, HDDM and related diffusion models have been successfully applied to the study of cognitive impairments in various psychiatric disorders, including ADHD, anxiety disorders, and depression [28–30]. However, research using HDDM to systematically compare computational impairment patterns in inhibitory control between SCZ and MDD remains scarce.

In the present study, we employed HDDM to model the performance of patients with SCZ and MDD on interference inhibition and response inhibition tasks. Our objective was to systematically characterize the latent computational mechanisms underlying inhibitory control impairments in both disorders and to explore the computational basis of transdiagnostic shared features and disorder-specific characteristics (Fig 1). Based on the theoretical analysis presented above, we hypothesized that: (1) both disorders would show abnormalities in HDDM parameters, with certain parameters reflecting transdiagnostic impairment and others exhibiting disorder-specific patterns; (2) relative to healthy controls, SCZ would show more pronounced drift rate reductions, whereas MDD would show distinct alterations in decision-related parameters, such as elevated decision boundaries or shifted starting point bias; (3) parameter abnormalities would correlate with clinical symptom severity; and (4) HDDM parameters would support individual-level diagnostic classification.

Methods

Ethics statement

This study was approved by the Ethics Committee of the Third People's Hospital of Lanzhou City (approval number: TPHL2025014) and the Ethics Committee of Northwest Normal University (approval number: NWNU20251009), and was conducted in accordance with the Declaration of Helsinki. All participants provided written informed consent prior to participation.

Participants

This study was conducted between October and December 2025 and involved 259 participants, including 87 patients with SCZ, 86 patients with MDD, and 86 healthy controls (HC) matched for age, sex, and years of education. Sample size was determined a priori using G*Power 3.1 [31], based on a medium effect size (f=0.25) derived from meta-analytic findings [3, 32], yielding a minimum of 52 participants per group (α=0.05, $1 - \beta$=0.80). Given the greater

sample size requirements for HDDM analyses [25] and potential attrition, we aimed to recruit 85–90 participants per group. This study was preregistered with the Chinese Clinical Trial Registry prior to data collection (registration number: ChiCTR2500111117; https://www.chictr.org.cn/showproj.html?proj=290089).

Patients with SCZ and MDD were recruited from the Third People's Hospital of Lanzhou City. All patients received a confirmed diagnosis based on the Structured Clinical Interview for *DSM-IV* Axis I Disorders (SCID-IV), corresponding to *International Classification of Diseases, 10th Revision* codes F20.9 and F32–F33 for SCZ and MDD, respectively. All patients were in a clinically stable phase, defined as ≥6 weeks of stable symptoms and an unchanged medication regimen. HC were recruited through community outreach and online advertisements and were screened using the Mini-International Neuropsychiatric Interview to exclude individuals with current or past psychiatric disorders. Detailed inclusion and exclusion criteria for all groups are provided in Table S1.

Clinical symptom assessment

To enable direct comparison of symptom dimensions across diagnostic categories, both patient groups completed assessments of depressive and anxiety symptoms [33]. Depressive symptom severity was assessed using the 17-item Hamilton Depression Rating Scale (HAMD-17) [34], with six factor scores calculated according to the classical factor structure. Anxiety symptom severity was assessed using the 14-item Hamilton Anxiety Rating Scale (HAMA-14) [35], with two factor scores computed following established guidelines. For the SCZ group, psychotic symptom severity was assessed using the Positive and Negative Syndrome Scale (PANSS) [36], which comprises positive, negative, and general psychopathology subscales.

All clinical assessments were administered by trained psychiatrists. Additional clinical information, including illness duration, number of hospitalizations, current medication regimen, and treatment history, was obtained from electronic medical records. To facilitate cross-study comparisons and to control for potential confounding effects of medication, antipsychotic doses were converted to chlorpromazine equivalents [37] and antidepressant doses were converted to fluoxetine equivalents [38].

Inhibitory control behavioral tasks

We employed the Stroop task and the Go/No-Go task to assess interference inhibition and response inhibition, respectively. These paradigms tap distinct subcomponents of inhibitory

control with well-established neural substrates and have been extensively applied in psychiatric research [3, 11, 39–41]. In the Stroop task (Fig 1), participants were instructed to respond based on font color while ignoring word meaning [42]. The task included congruent, incongruent, and neutral conditions, with the primary outcome measure being the Stroop interference effect (i.e., the reaction time difference between incongruent and congruent conditions). The task comprised 108 test trials across three blocks. In the Go/No-Go task (Fig 1), participants responded to Go stimuli while withholding responses to No-Go stimuli (Go:No-Go ratio=75%:25%). Primary outcome measures included Go and No-Go accuracy, Go reaction time, and the signal detection index d' [43]. The test phase consisted of 400 trials across four blocks. For both tasks, participants were required to achieve ≥ 85% accuracy during practice before proceeding to the test phase. Each task lasted approximately 15 min. Detailed task parameters, trial timing, schematics, and reliability indices are provided in the Supplementary Methods and Table S2.

Procedure

All participants completed the full assessment battery within one week of enrollment. The first part involved clinical assessment (patient groups only), during which psychiatrists administered the HAMD-17 and HAMA-14 (and the PANSS for the SCZ group) and collected demographic and clinical information from electronic medical records (approximately 40 min). The second part involved behavioral task testing; participants completed the Stroop task followed by the Go/No-Go task, with a rest break between tasks (approximately 30 min total). All behavioral tasks were programmed using E-Prime 3.0 (Psychology Software Tools, Pittsburgh, PA, USA) and presented on a 17.3-inch monitor (resolution: 1,920 × 1,080; refresh rate: 60 Hz).

Data analysis

Behavioral and clinical analyses

One-way analyses of covariance were conducted to examine group differences (SCZ, MDD, and HC) on inhibitory control measures, with age, sex, and years of education included as covariates. Significant main effects were further examined using Tukey's honestly significant difference *post hoc* comparisons. Within patient groups, partial correlation analyses were performed to examine associations between inhibitory control performance and clinical variables (e.g., illness duration, PANSS subscale scores, HAMD-17 scores, and HAMA-14

scores), controlling for age, sex, and years of education. All multiple comparisons were corrected using the Benjamini–Hochberg false discovery rate (FDR) method [44].

Confirmatory factor analysis

Confirmatory factor analysis was employed to examine whether the factor structure of inhibitory control subcomponents differed across the three groups [45]. Observed indicators included four indices from the *Stroop* task (i.e., reaction times for congruent, incongruent, and neutral conditions, and the interference effect) and four from the Go/No-Go task (i.e., Go and No-Go accuracy, Go reaction time, and *d'*). A single-factor model (all indicators loading onto a unitary "inhibitory control" factor) was compared with a two-factor model (Stroop task indicators loading onto an "interference inhibition" factor and Go/No-Go task indicators loading onto a "response inhibition" factor). Model fit was evaluated using χ^2, comparative fit index (CFI), Tucker–Lewis index, root mean square error of approximation with 90% confidence intervals (CI), and standardized root mean square residual [46]. Model comparisons were conducted using chi-square difference tests and the Akaike Information Criterion (AIC). Subsequently, measurement invariance of the inhibitory control factor structure across the three groups was tested using multi-group confirmatory factor analysis [47], sequentially examining configural, metric, and scalar invariance. Invariance was evaluated using nested model chi-squared difference tests and changes in CFI (ΔCFI $\leq$ 0.01 indicating invariance) [48]. All confirmatory factor analyses were conducted using the lavaan package in R (v4.3.1) [49].

HDDM analysis

Data preprocessing

Trial-by-trial behavioral data from the Stroop and Go/No-Go tasks were analyzed using the HDDM package (v1.0.1) [25]. For the Stroop task, following standard analytical procedures [50], only correct-response trials were included in the analysis. For the Go/No-Go task, we adopted a stimulus-coding approach to drift-diffusion modeling [51, 52], conceptualizing the task as a binary stimulus classification decision process (Supplementary Methods). In this parameterization, the upper boundary represented "Go" classification decisions and the lower boundary represented "No-Go" classification decisions. Given the high frequency of Go stimuli (75%), the model included a *starting point bias* parameter (z) to quantify participants' prior response tendency toward Go stimuli [53]. Data coding followed HDDM documentation

recommendations (Supplementary Methods). The model specified 5% of trials as potential outliers (p_outlier=0.05) to account for contaminant responses [53].

Model specification

Three hierarchical models were compared (Table S3): i) a baseline model (M0) with core DDM parameters (a, v, t) and inter-trial variability (sv, sz, st), with z fixed at 0.5; ii) a hierarchical model (M1) allowing a, v, t, and z to vary across groups; and iii) a mixed-effects regression model (M2), which served as the primary model and estimated group differences while accounting for individual variability [54]. Default prior distributions from the HDDM package were employed [55].

Model fitting and diagnostics

Model parameters were estimated within a hierarchical Bayesian framework, with posterior distributions obtained via Markov chain Monte Carlo (MCMC) sampling. Each model was run with four independent MCMC chains, each comprising 15,000 samples, with the first 5,000 samples discarded as burn-in [28]. Convergence was assessed using the Gelman–Rubin statistic ($\hat{R}$<1.01) [56]. Model comparison was performed using the Deviance Information Criterion (DIC) [57]. Model adequacy was evaluated through posterior predictive checks, with 500 simulated datasets generated using the HDDM *postpred_gen* function [58].

Statistical inference and clinical association analyses

Statistical inference for parameters was conducted within a Bayesian framework, with group differences examined using Bayesian analysis of variance and evidence strength quantified using Bayes factors (BF_{10}) [59]. Uncertainty in group differences was characterized using 95% highest density intervals (HDIs), with statistical decisions based on the relationship between 95% HDIs and the region of practical equivalence (ROPE) (see also Supplementary Methods). Subsequently, partial correlation analyses were conducted to examine associations between HDDM parameters and both behavioral and clinical indices, controlling for age, sex, and years of education. Multiple comparisons were corrected using the Benjamini–Hochberg method.

Machine learning classification

To evaluate the diagnostic classification utility of HDDM parameters, we constructed support vector machine (SVM)–based classification models [60], implemented using the *scikit-learn* library (v1.3.0) in Python [61]. HDDM parameters from the Stroop and Go/No-Go tasks were

extracted as input features for pairwise binary classification models (SCZ vs. HC, MDD vs. HC, and SCZ vs. MDD). Data were randomly split into training (80%) and test (20%) sets [62]. Model optimization was performed on the training set using a nested cross-validation strategy with five-fold cross-validation repeated three times [63], with hyperparameter tuning conducted via the Optuna framework (Supplementary Methods). After identifying optimal hyperparameters, the final model was fitted on the training set and evaluated on the independent test set. Classification performance was assessed using the area under the receiver operating characteristic curve (AUC), balanced accuracy (BAC), sensitivity, and specificity. Feature importance was analyzed using the SHapley Additive exPlanations (SHAP) method [64] to identify the HDDM parameters that contributed most to classification.

Examination of shared and disorder-specific mechanisms

Three analytical strategies were employed to further investigate shared and disorder-specific computational mechanisms underlying inhibitory control impairments in SCZ and MDD. *Mediation analysis*: Bootstrap mediation analyses (3,000 resamples) were conducted separately within each group to examine whether HDDM parameters mediated the relationship between task condition and behavioral performance [65]. *Cross-task parameter consistency analysis*: Corresponding HDDM parameters (v, a, t, z) were extracted for each participant across both tasks. Pearson correlation coefficients between corresponding Stroop and Go/No-Go task parameters were computed within each group, and Fisher's z transformation was used to test for between-group differences in correlation coefficients. *Task type classification analysis*: SVMs were used to classify Stroop versus Go/No-Go task trials based on HDDM parameters, separately within each group. Feature ablation analysis (i.e., sequential removal of individual parameters) was conducted to quantify the contribution of each parameter to task discrimination [66]. The performance of ablated models was compared with the full model (containing all parameters) using bootstrap difference tests.

Results

Demographic and clinical characteristics

The three groups did not differ significantly in age, sex distribution, years of education, socioeconomic status, ethnicity, or the proportion of only children (all $p>0.05$) (Table 1). Raven's Progressive Matrices scores revealed a significant group difference ($F=11.86$,

p_{FDR}<0.001), with the HC group scoring higher than both the SCZ (p_{FDR}<0.001) and MDD (p_{FDR}=0.032) groups; the difference between the two patient groups did not reach statistical significance (p_{FDR}=0.186). A significant group difference in body mass index was also observed (F=4.12, p_{FDR}=0.042), with the SCZ group showing higher body mass index than the HC group (p_{FDR}=0.048); no other pairwise comparisons reached significance. Regarding clinical characteristics, the SCZ group had a significantly earlier age at onset (t=−2.48, p_{FDR}=0.028) and longer illness duration (t=−3.16, p_{FDR}=0.008) than the MDD group. The MDD group had significantly higher HAMD-17 (t=13.42, p_{FDR}<0.001) and HAMA-14 (t=10.24, p_{FDR}<0.001) total scores than the SCZ group, with similar patterns observed across all factor scores. The MDD group also showed a higher rate of antidepressant use (χ^2=72.46, p_{FDR}<0.001) and higher fluoxetine-equivalent doses (t=3.24, p_{FDR}=0.004), whereas the SCZ group showed a higher rate of antipsychotic use (χ^2=113.82, p_{FDR}<0.001) and higher chlorpromazine-equivalent doses (t=−5.86, p_{FDR}<0.001).

Inhibitory control behavioral performance and clinical correlates

Group differences in behavioral performance

Analyses of covariance (with age, sex, and years of education as covariates) revealed significant group differences across both tasks (Fig 2A; Table 2). For the Stroop task, significant group differences were observed across all conditions (congruent, neutral, and incongruent; all p<0.01), with the SCZ group consistently showing the slowest reaction times. Importantly, the interference effect also differed significantly among groups (F=32.48, p<0.001, η^2p=0.186), with the SCZ group exhibiting the largest interference relative to the HC group (p<0.001, d=0.92), followed by the MDD group (p=0.002, d=0.54); the difference between the two patient groups was also significant (p=0.018, d=0.42). For the Go/No-Go task, significant group differences were observed for both Go and No-Go trials. Go accuracy (F=10.24, p<0.001) and Go reaction time (F=14.86, p<0.001) indicated impairments in both patient groups relative to the HC group. Of particular note, No-Go accuracy displayed a significant graded pattern (F=28.64, p<0.001, η^2p=0.168), with the SCZ group performing significantly worse than both the HC (p<0.001, d=0.98) and MDD (p=0.006, d=0.48) groups, and the MDD group performing worse than the HC group (p=0.008, d=0.46); this graded pattern was confirmed by d' values (F=34.26, p<0.001, η^2p=0.196).

To further examine the latent structure underlying these behavioral differences, we tested a two-factor model (interference inhibition and response inhibition) that showed good fit in the full sample (CFI=0.968, TLI=0.956, RMSEA=0.048 [90% CI: 0.028–0.068], SRMR=0.042) and acceptable fit within each group (Fig S1; Table S4). Multi-group analysis supported configural invariance (CFI=0.962) and metric invariance (ΔCFI=−0.006) across groups. However, the inter-factor correlation differed significantly across groups; the HC group showed a higher correlation (r=0.62) than the SCZ (r=0.38; Fisher's z=2.48, p=0.013) and MDD (r=0.48; Fisher's z=1.86, p=0.063) groups. In line with this finding, a model constraining the inter-factor correlation to be equal across groups showed significantly poorer fit ($\Delta\chi^2$=12.46, p=0.002).

Correlations between inhibitory control and clinical symptoms

Partial correlation analyses (controlling for age, sex, and years of education) examined associations between inhibitory control performance and clinical symptoms (Fig 2B). In the SCZ group, inhibitory control impairments (Stroop interference effect and d') were significantly correlated with PANSS negative symptoms (r=0.34, p_{FDR}=0.004 and r=−0.39, p_{FDR}=0.002, respectively), illness duration (r=0.22, p_{FDR}=0.042 and r=−0.26, p_{FDR}=0.028), and age at onset (r=−0.22, p_{FDR}=0.048 and r=0.23, p_{FDR}=0.038), but not with positive symptoms ($p_{FDR}>0.05$). In contrast, chlorpromazine-equivalent dose was correlated with reaction time measures but not with the interference effect or d'. These findings suggest that medication primarily affects overall processing speed rather than inhibitory control–specific processes. In the MDD group, inhibitory control impairments were correlated with HAMD-17 total scores (r=0.31, p_{FDR}=0.008 and r=−0.29, p_{FDR}=0.016), but not with illness duration (all $p>0.05$). Similarly, fluoxetine-equivalent dose showed no significant associations with any inhibitory control measure.

Computational mechanisms of inhibitory control impairment

Model fitting and diagnostics

The HDDM provided a good fit to data from both tasks. All parameters had $\hat{R}$ values<1.01, and MCMC trace plots indicated good mixing across chains (Figs S2, S3; Table S5). In addition, posterior predictive checks demonstrated that simulated data closely matched observed data at both the group and individual levels (Fig S4). Model comparison indicated that drift rate was the key parameter for distinguishing among groups: for the Stroop task, the drift rate regression

model provided the best fit; for the Go/No-Go task, the model including both drift rate and starting point bias provided the best fit (Table S6).

Group differences in HDDM parameters

Bayesian analysis of variance revealed significant group differences in HDDM parameters (Fig 3A). With respect to drift rate, both patient groups showed significantly lower values than the HC group on both tasks. For the SCZ group, the posterior mean differences (PMDs) were −0.624 (Stroop task, 95% HDI [−0.862, −0.386]; BF_{10}=186.42) and −0.548 (Go/No-Go task, 95% HDI [−0.782, −0.314]; BF_{10}=68.24). For the MDD group, the PMDs were −0.386 (Stroop task, 95% HDI [−0.618, −0.154]; BF_{10}=16.86) and −0.324 (Go/No-Go task, 95% HDI [−0.552, −0.096]; BF_{10}=8.42). Direct comparisons provided moderate evidence for lower drift rate in the SCZ group relative to the MDD group (Stroop task: BF_{10}=3.86; Go/No-Go task: BF_{10}=2.96). For non-decision time, the SCZ group showed significant prolongation on both tasks (Stroop task: PMD=0.124, 95% HDI [0.068, 0.180], BF_{10}=12.86; Go/No-Go task: PMD=0.108, 95% HDI [0.048, 0.168], BF_{10}=6.42). In contrast, the MDD group showed significant prolongation only on the Go/No-Go task (PMD=0.062; 95% HDI [0.008, 0.116]; BF_{10}=2.24), with no reliable effect on the Stroop task (PMD=0.048; 95% HDI [−0.024, 0.120]; BF_{10}=0.86). In keeping with this pattern, direct comparison indicated that the SCZ group had significantly longer non-decision time than the MDD group on the Stroop task (BF_{10}=4.68). For starting point bias on the Go/No-Go task, both patient groups showed significantly lower values than the HC group (SCZ: PMD=−0.068, 95% HDI [−0.098, −0.038], BF_{10}=6.24; MDD: PMD=−0.042, 95% HDI [−0.072, −0.012], BF_{10}=2.18), indicating a weaker initial bias toward Go responses. Starting point bias was not analyzed for the Stroop task, as this task does not involve a Go/No-Go decision structure. Finally, no substantial group differences were observed in decision boundary on either task (all BF_{10}<0.8), suggesting that all groups adopted comparable speed–accuracy trade-off strategies.

Mediation effects

Bootstrap mediation analyses examined whether HDDM parameters mediated the effect of task condition on behavioral performance within each group (Fig 3B; Fig S5). Drift rate significantly mediated condition effects on both the Stroop interference effect and d' across all three groups (all indirect effects: p<0.001). For the Stroop task, the indirect effects of drift rate on the interference effect were 0.038 (95% CI [0.022, 0.056]) for the SCZ group and 0.028 (95%

CI [0.014, 0.042]) for the MDD group. For the Go/No-Go task, the indirect effects of drift rate on d' were 0.054 (95% CI [0.036, 0.072]) for the SCZ group and 0.038 (95% CI [0.024, 0.054]) for the MDD group. Starting point bias showed a significant mediating effect on d' in the SCZ group (p=0.018), but only a marginally significant effect in the MDD group (p=0.098).

Diagnostic discrimination

Support vector machine classifiers were used to evaluate the diagnostic discriminability of HDDM parameters (Fig 3C). The classifier achieved good discrimination between the SCZ and HC groups (AUC=0.846; BAC=0.786), moderate discrimination between the MDD and HC groups (AUC=0.762; BAC=0.704), and above-chance discrimination between the two patient groups (AUC=0.718; BAC=0.684). Across all comparisons, SHAP analysis indicated that drift rate was the primary contributor to classification (mean |SHAP| range: 0.23–0.51), followed by non-decision time (mean |SHAP| range: 0.09–0.34), whereas decision boundary contributed the least (mean |SHAP|<0.06). Notably, for SCZ–MDD discrimination, non-decision time from the Stroop task emerged as the most discriminative feature (mean |SHAP|=0.34) (Fig 3D).

Associations between HDDM parameters and behavioral and clinical indices

Across groups, drift rate was significantly negatively correlated with the Stroop interference effect (SCZ: r=−0.624, p_{FDR}<0.001; MDD: r=−0.586, p_{FDR}<0.001; HC: r=−0.468, p_{FDR}<0.001) and positively correlated with No-Go trial accuracy (SCZ: r=0.582, p_{FDR}<0.001; MDD: r=0.536, p_{FDR}<0.001; HC: r=0.492, p_{FDR}<0.001) (Fig 4B). Non-decision time was positively correlated with Go trial reaction time (SCZ: r=0.486, p_{FDR}<0.001; MDD: r=0.424, p_{FDR}<0.001; HC: r=0.456, p_{FDR}<0.001) but showed weaker associations with the interference effect and No-Go accuracy. Starting point bias was positively correlated with Go trial accuracy (SCZ: r=0.368, p_{FDR}=0.002; MDD: r=0.312, p_{FDR}=0.008) and negatively correlated with No-Go trial accuracy (SCZ: r=−0.298, p_{FDR}=0.012; MDD: r=−0.256, p_{FDR}=0.028) (Fig 4B).

Analyses of clinical associations revealed distinct patterns linking HDDM parameters to clinical symptoms in each disorder (Fig 4A). In the SCZ group, drift rate was negatively correlated with PANSS negative symptoms (Stroop task: r=−0.37, p_{FDR}<0.001; Go/No-Go task: r=−0.31, p_{FDR}=0.002), but not with positive symptoms (p_{FDR}>0.05). Non-decision time was positively correlated with negative symptoms (r=0.29; p_{FDR}=0.004) and illness duration (r=0.22; p_{FDR}=0.018). In the MDD group, drift rate was negatively correlated with HAMD-17 total scores (Stroop task: r=−0.34, p_{FDR}<0.001; Go/No-Go task: r=−0.30, p_{FDR}=0.004),

particularly the cognitive factor (r=−0.41; p_{FDR}<0.001), but not with illness duration (p_{FDR}>0.05). Non-decision time was positively correlated with the HAMD-17 somatization factor (r=0.27; p_{FDR}=0.006).

Cross-task consistency and discriminability

Cross-task parameter consistency analysis revealed that drift rate correlations between the two tasks were significantly reduced in the patient groups (SCZ: r=0.29, p_{FDR}=0.018; MDD: r=0.37, p_{FDR}=0.004) compared with the HC group (r=0.52, p_{FDR}< 0.001) (Fig 5A). Fisher's z tests confirmed that this reduction was significant for the SCZ versus HC comparison (z=2.42, p=0.016) and marginally significant for the MDD versus HC comparison (z=1.78, p=0.075). In contrast, non-decision time showed a different pattern; specifically, the SCZ group exhibited the highest cross-task correlation (r=0.49), followed by the HC group (r=0.42), whereas the MDD group showed the lowest cross-task correlation (r=0.28); notably, the difference between the SCZ and MDD groups was significant (z=2.38, p=0.017).

Consistent with these reductions in cross-task parameter stability, task discriminability based on HDDM parameters was also reduced in the patient groups (Fig 5B). In the HC group, the AUC for distinguishing between the two tasks was 0.842, with drift rate being the strongest contributor to task discrimination (ΔAUC=−0.186 upon removal). In contrast, the SCZ group showed significantly lower task discriminability (AUC=0.724; permutation test p=0.008), accompanied by an attenuated discriminative contribution of drift rate (ΔAUC=−0.124). The MDD group showed an intermediate level of discriminability (AUC=0.786; permutation test p=0.048), with drift rate remaining the primary discriminating parameter (ΔAUC=−0.156).

Discussion

In the present study, we used HDDM to systematically examine the shared and distinct computational mechanisms underlying impairments in interference inhibition and response inhibition in patients with SCZ and MDD. At the behavioral level, both patient groups exhibited significant deficits, with the SCZ group showing more severe impairment. Multi-group CFA confirmed the two-factor structure across groups, although the inter-factor correlation was reduced in the patient groups, particularly in SCZ. At the computational level, reduced drift rate emerged as the core shared impairment, whereas non-decision time

463 prolongation showed disorder-specific patterns, being more pronounced and consistent across
464 tasks in SCZ compared with the more task-specific prolongation observed in MDD.

465 ***Behavioral patterns of interference and response inhibition impairments***

466 Patients with SCZ and MDD both exhibited significant impairments on inhibitory control
467 tasks, although the severity of impairment differed. These findings partially support our
468 preregistered hypotheses and reveal a transdiagnostic pattern of shared impairment with
469 differential severity [5]. Across both the Stroop task and the Go/No-Go task, SCZ was
470 associated with the most severe impairment, followed by MDD. This graded pattern is
471 consistent with previous meta-analytic findings [3, 67]. Given that inhibitory control
472 impairment in MDD tends to be more context-dependent—being more pronounced when
473 processing negative emotional material or under high cognitive load [3, 14]—the use of
474 emotionally neutral materials in the present study may not have fully captured the inhibitory
475 control deficits characteristic of MDD, which may partially explain the relatively milder
476 impairment observed in this group.

477 Multi-group CFA demonstrated that the two-factor structure of interference inhibition and
478 response inhibition showed good model fit across all three groups, supporting the multi-
479 component theory of inhibitory control [19]. Notably, the correlation between the two factors
480 was relatively high in the HC group, significantly reduced in the SCZ group, and intermediate
481 in the MDD group. This reduced functional coupling in SCZ is consistent with prior evidence
482 of impaired inter-network connectivity in this disorder [68], whereas the relatively preserved
483 coupling in MDD may reflect more localized prefrontal abnormalities [69].

484 We also found divergent symptom–cognition relationships between the two disorders. In
485 SCZ, inhibitory control impairment was primarily correlated with PANSS negative symptoms
486 rather than positive symptoms, consistent with the cognitive–negative symptom covariation
487 perspective [70, 71]. The significant association between illness duration and inhibitory control
488 performance suggests that cognitive impairment may worsen with disease progression [72].
489 Moreover, the negative association with age at onset indicates that earlier-onset patients
490 exhibited more severe impairment, consistent with the neurodevelopmental hypothesis [73, 74].
491 In MDD, inhibitory control impairment was primarily correlated with HAMD-17 total scores,
492 particularly the cognitive factor, suggesting a close link between inhibitory control and the
493 cognitive dimensions of depressive symptoms [14, 32]. The non-significant association with

illness duration in this group indicates that cognitive impairment in MDD is more state-dependent, although patients in remission may continue to exhibit residual impairment [75]. Regarding medication effects, chlorpromazine-equivalent dose in the SCZ group was correlated with reaction time but not with d', and fluoxetine-equivalent dose in the MDD group showed no significant association with any behavioral measure. These observations suggest that impairments in both groups primarily reflect illness-related characteristics rather than medication effects [76, 77].

Reduced drift rate in inhibitory control as a shared computational deficit

HDDM analysis revealed the core computational mechanisms underlying inhibitory control impairment in both disorders. Consistent with our hypothesis, reduced drift rate emerged as the most robust computational impairment; on both the Stroop task and the Go/No-Go task, patients with SCZ and MDD showed significantly lower drift rates than HC. This finding aligns with prior reports of reduced evidence accumulation in both disorders [22]. That reduced drift rate emerged as a common computational impairment suggests that inhibitory control deficits in both disorders may be rooted in a fundamental reduction in information processing efficiency [3, 78], rather than in the selective impairment of any particular inhibitory mechanism. In terms of severity, the SCZ group showed a greater reduction in drift rate than the MDD group, consistent with the graded pattern observed at the behavioral level [79, 80].

The transdiagnostic nature of reduced drift rate parallels the overlap between the two disorders in prefrontal dysfunction. The dorsolateral prefrontal cortex and anterior cingulate cortex are key brain regions supporting evidence accumulation, and their activation levels are positively correlated with drift rate [81–83]. Both disorders are characterized by structural and functional abnormalities in the prefrontal cortex [84, 85], which may constitute a shared neural basis for reduced drift rate. At the neurotransmitter level, prefrontal dopamine hypofunction in SCZ [21] and dopaminergic dysregulation in MDD [86] may compromise the signal-to-noise ratio of task-relevant representations [87, 88], thereby contributing to the reduced evidence accumulation efficiency observed in both disorders.

Regarding our second hypothesis concerning disorder-specific patterns, the results were mixed. We had predicted that SCZ would show more pronounced reductions in drift rate, which was supported by the data. However, our prediction that MDD would show relatively greater increases in decision boundary or starting point bias was not supported. Bayesian model

comparison yielded inconclusive evidence regarding group differences in decision boundary, suggesting that altered speed–accuracy trade-off strategies are unlikely to be a primary mechanism underlying inhibitory control impairment [89]. The absence of elevated decision boundary in MDD, contrary to our prediction, may indicate that the heightened risk aversion often associated with depression [26] does not translate into more conservative response thresholds in inhibitory control contexts, or that such effects are more prominent in reward-related decision-making paradigms than in the cognitive control tasks used here.

Disorder-specific profiles in non-decision time and starting point bias

Beyond this shared impairment, the two disorders exhibited distinct profiles in other HDDM parameters. Prolonged non-decision time showed diagnostic specificity; specifically, SCZ was associated with significantly longer non-decision time than HC on both tasks, whereas MDD showed significant prolongation only on the Go/No-Go task, with no reliable effect on the Stroop task. This pattern suggests that non-decision time prolongation may serve as a more robust marker of SCZ. SCZ is characterized by widespread basic sensory processing deficits [90, 91] and psychomotor retardation [41]. Together, these perceptual and motor-level impairments constitute the neurobehavioral basis for prolonged non-decision time. In contrast, the neuropathology of MDD is more concentrated in prefrontal–limbic networks [92], with relatively limited impact on basic perceptual and motor functions. The task-specific prolongation of non-decision time observed in the MDD group on the Go/No-Go task may reflect the greater motor preparation demands of this paradigm, rather than a generalized perceptual–motor deficit.

On the Go/No-Go task, starting point bias was significantly reduced in both patient groups, indicating weakened prior preparation for Go responses. Rather than reflecting a more conservative decision strategy as we had hypothesized, this reduced starting point bias appears to reflect impaired sensitivity to environmental statistical regularities [93, 94]. On the Stroop task, starting point bias did not differ significantly among groups, likely because pre-decision bias plays a limited role when all trials require a response. Mediation analysis revealed that drift rate significantly mediated the effect of task condition on behavioral performance within each group, whereas starting point bias had a significant mediating effect in SCZ and a marginally significant effect in MDD. These findings suggest that patient impairments stem

555 from two computational sources, namely less efficient information processing and maladaptive
556 initial response preparation.
557 At the clinical level, computational parameters showed distinct symptom correlates across
558 the two disorders, partially supporting our third hypothesis. In SCZ, drift rate was significantly
559 negatively correlated with PANSS negative symptoms but not with positive symptoms.
560 Negative symptoms and cognitive impairment may both be rooted in prefrontal–striatal
561 dopamine hypofunction [70]. The positive correlation between non-decision time and negative
562 symptoms indicates that perceptual–motor slowing may reflect psychomotor dysfunction
563 associated with negative symptoms [91]. The significant correlations of drift rate and non-
564 decision time with illness duration are consistent with the progressive nature of cognitive
565 impairment in SCZ [95]. In MDD, drift rate was negatively correlated with HAMD-17 total
566 scores but unrelated to illness duration. These results suggest that impaired evidence
567 accumulation efficiency in this group is more state-dependent, consistent with findings of
568 partial cognitive recovery during remission [96].

569 ***Cross-task consistency and dedifferentiation in inhibitory control***

570 We examined cross-task parameter consistency to assess the internal integration of the
571 inhibitory control system. In HC, drift rates from the two tasks showed a moderate positive
572 correlation, suggesting that healthy individuals rely on domain-general evidence accumulation
573 mechanisms across both tasks. This correlation was significantly reduced in SCZ, whereas
574 MDD showed a trend toward reduction that did not reach statistical significance. Accordingly,
575 the inhibitory control system in SCZ appears to exhibit a more fragmented pattern. This
576 interpretation is consistent with the disconnection hypothesis of SCZ [97]. The core pathology
577 of this disorder may lie in the breakdown of functional integration among brain regions, rather
578 than in focal damage to any particular region [68, 98]. Cross-task correlations of non-decision
579 time showed a different pattern; specifically, SCZ exhibited the highest correlation, followed
580 by HC, whereas MDD showed the lowest correlation. In SCZ, perceptual–motor processing is
581 affected by both disease-related neuropathology and medication side effects. Because these
582 influences operate consistently across different tasks, they may account for the enhanced cross-
583 task correlation of non-decision time. The opposite patterns of cross-task correlation for drift
584 rate versus non-decision time suggest that the disconnection hypothesis may be more
585 applicable to higher-order cognitive processes than to basic perceptual–motor processes.

We also assessed task discriminability based on HDDM parameters. In HC, the accuracy of distinguishing between the two tasks was relatively high, with drift rate being the strongest contributor to discrimination. This finding indicates that healthy individuals employ distinguishable evidence accumulation patterns across the two task types. Task discriminability was significantly reduced in SCZ, with the discriminative contribution of drift rate weakened and the relative contribution of non-decision time increased, suggesting that computational differences between task types became blurred. MDD showed an intermediate pattern. Taken together, these results indicate that SCZ is characterized by reduced computational differentiation across inhibitory control tasks, with MDD occupying an intermediate position.

Limitations and future directions

Several limitations of the present study warrant consideration. First, the cross-sectional design limits our ability to draw causal inferences. Whether reduced drift rate is a consequence, cause, or concomitant of the illness requires clarification through longitudinal research. Future studies could track the trajectory of computational parameter changes in first-episode patients or individuals at high risk [99] to clarify their value as markers of disease progression and to determine whether these computational features precede clinical symptoms or fluctuate with symptom changes. Second, although sensitivity analyses indicated that group differences in drift rate remained stable after controlling for medication dose, the present study cannot completely rule out medication confounds, especially given that the effect size for non-decision time was somewhat attenuated after this adjustment. Including unmedicated patients would provide purer disease-related computational profiles; nevertheless, this approach poses ethical and feasibility challenges in clinical practice. Future research could compare medicated and unmedicated patients or employ longitudinal designs tracking parameter changes before and after medication adjustments to more clearly distinguish disease effects from medication effects. Third, the present study examined interference inhibition and response inhibition; however, inhibitory control is a multidimensional construct [11] that also includes other components such as cognitive inhibition. Future research could employ a broader set of task paradigms, such as the stop-signal task [100], directed forgetting task, and negative priming task, to obtain a more comprehensive computational characterization of inhibitory control and to test whether reduced drift rate is consistent across a wider range of inhibitory tasks. Finally, the present study did not include neuroimaging measures and, thus, could not directly examine

the correspondence between computational parameters and brain function. Previous research suggests that drift rate is associated with prefrontal–striatal circuit function and non-decision time with sensorimotor cortex activity [101]; however, whether these associations hold in psychiatric populations remains unclear. Future research could incorporate functional magnetic resonance imaging or electroencephalography techniques [102] to explore the neural basis of HDDM parameters and to test whether computational impairments mediate the relationship between neural abnormalities and behavioral deficits.

This study elucidated the computational mechanisms underlying inhibitory control (interference inhibition and response inhibition) impairments in patients with SCZ and MDD. Our findings revealed that reduced drift rate constitutes a shared transdiagnostic impairment, reflecting compromised efficiency in evidence accumulation across both disorders. In contrast, prolonged non-decision time appears to be a more disorder-specific feature characteristic of SCZ. The two disorders also differed in their clinical correlates; computational impairments in SCZ were associated with negative symptoms and illness duration, suggesting trait-like characteristics, whereas those in MDD were linked to current symptom severity, indicating more state-dependent features. These results suggest that computational parameters—particularly drift rate as a transdiagnostic index of processing efficiency and non-decision time as a disorder-differentiating marker—hold promise for informing differential diagnosis and guiding personalized intervention strategies.

Author Contributions: Tongyi Zhang: Conceptualization, Data curation, Formal analysis, Investigation, Methodology, Software, Visualization, Writing – original draft. Sirui Wang: Data curation, Investigation. Haoyang Shi: Data curation, Investigation. Jia Li: Methodology, Validation, Writing – review & editing. Xiaolong Yang: Conceptualization, Resources, Investigation. Jin Jiao: Resources, Investigation. Xiaoning Huo: Resources, Investigation. Xin Zhao: Conceptualization, Funding acquisition, Methodology, Project administration, Supervision, Validation, Writing – review & editing.

Competing Interests: The authors declare no conflicts of interest related to this work.

Acknowledgements: None.

Ethical standards: The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation and with the Declaration of Helsinki of 1975, as revised in 2008.

Funding Statement: This work was supported by the National Natural Science Foundation of China (No. 32260207 [to Xin Zhao]), the Lanzhou City Science and Technology Program Project (No. 2022-3-56 [to Xiaolong Yang] and No. 2022-3-55 [to Xiaoning Huo]), the Gansu Provincial Disease Prevention and Control Research Program (No. GSJKKY2025-19 [to Xiaolong Yang]), and the Gansu Provincial Health Industry Research Project (No. GSWSQN2025-25 [to Xiaolong Yang]). The funders had no role in study design, data collection and analysis, decision to publish, or preparation of the manuscript.

Data Availability: Data are available at https://github.com/tyzhang98/Inhibitory-Control-SCZ-MDD-Data. Scripts for the main analyses are available at https://github.com/tyzhang98/Inhibitory-Control-SCZ-MDD-Code.

Code Availability: Scripts for the main analyses are available at https://github.com/tyzhang98/Inhibitory-Control-SCZ-MDD-Code.

Preregistration: This study was preregistered with the Chinese Clinical Trial Registry prior to data collection (registration number: ChiCTR2500111117 ; https://www.chictr.org.cn/showproj.html?proj=290089).

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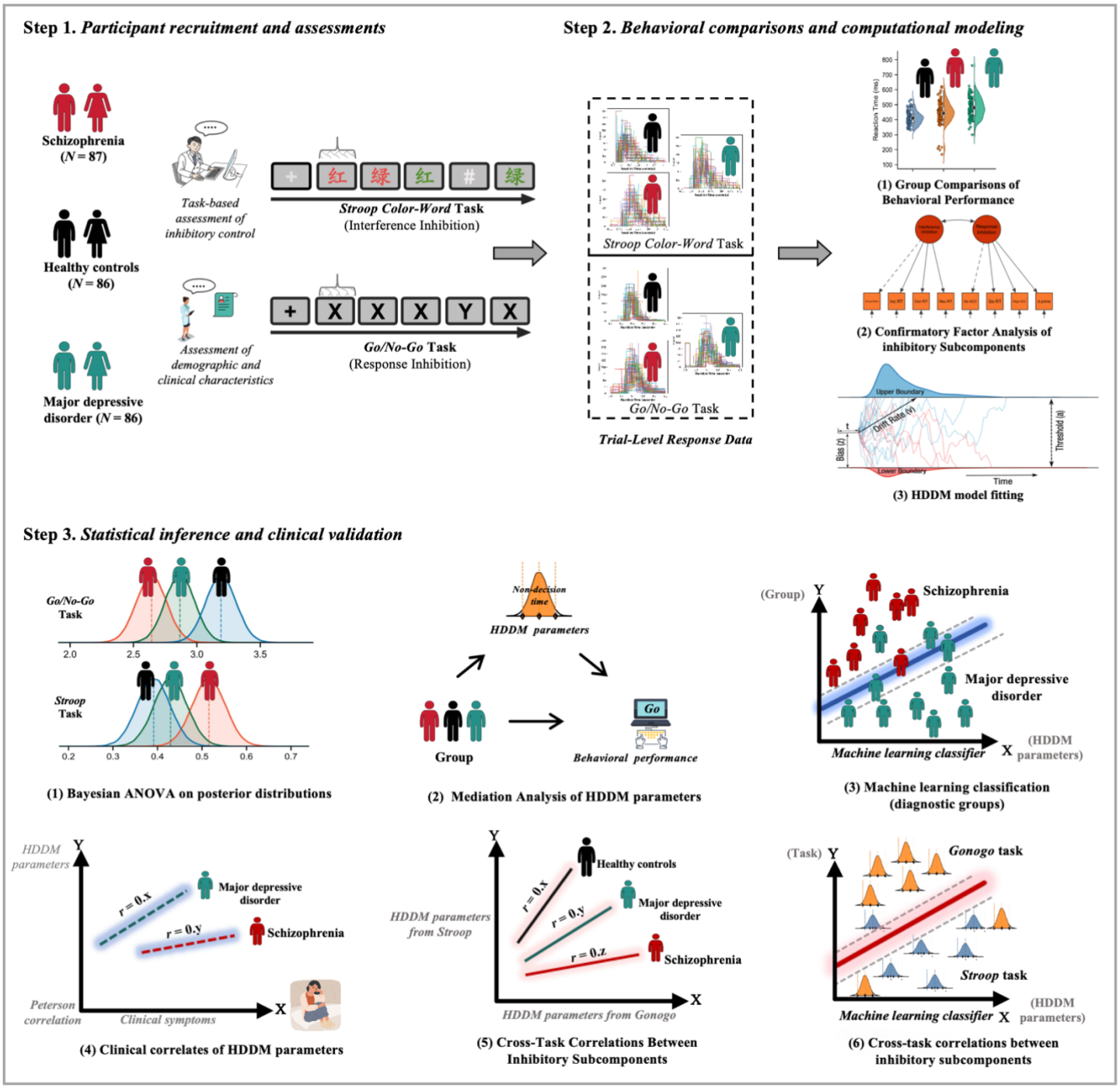
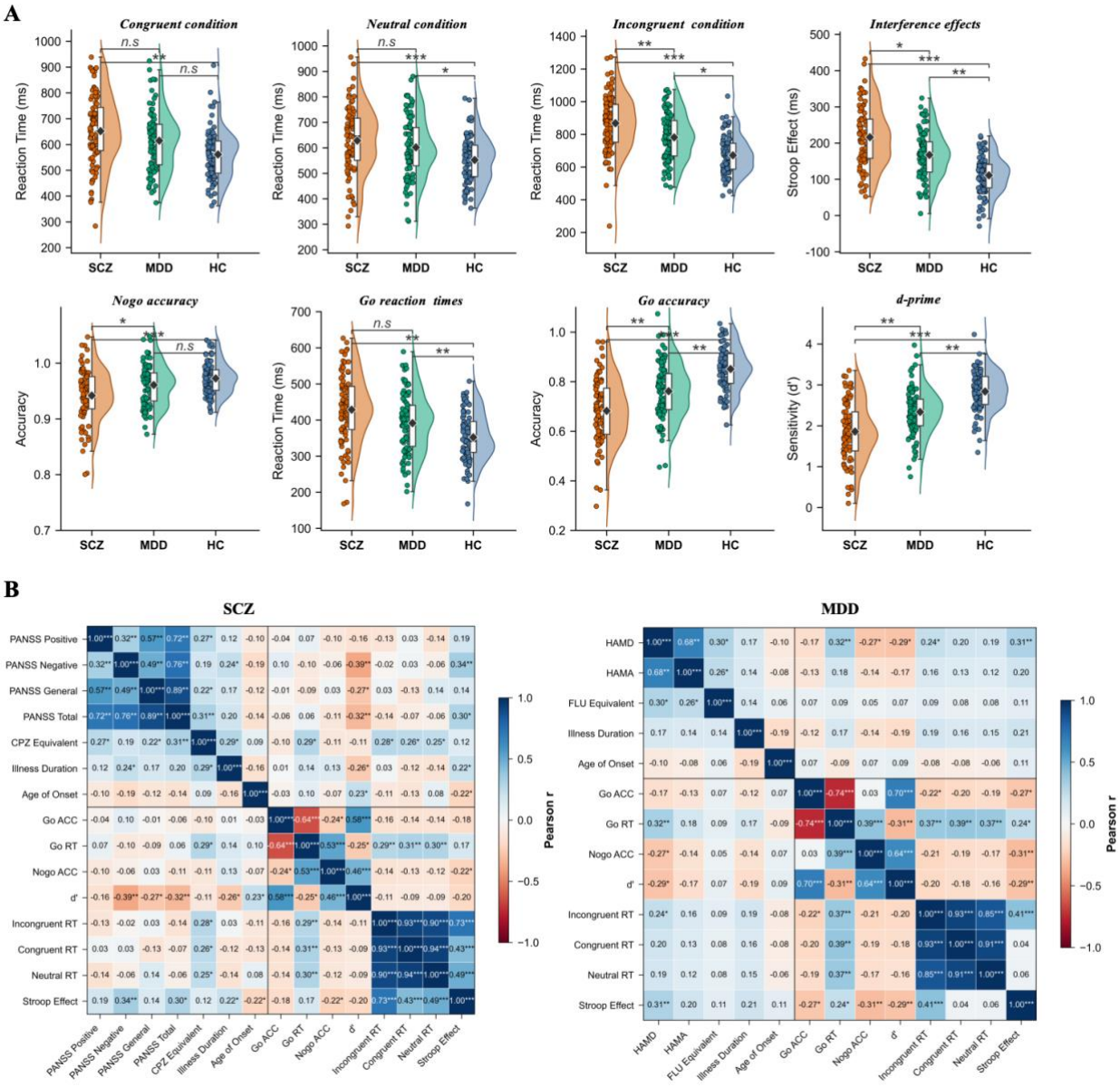


Fig 1. Study design and analytical framework.

The study comprised three phases. (**A**) Participants included patients with schizophrenia (SCZ), patients with major depressive disorder (MDD), and healthy controls (HC), matched for age, sex, and years of education. (**B**) Behavioral data analysis encompassed group comparisons of trial-level performance, multi-group confirmatory factor analysis examining the latent structure of inhibitory control, and hierarchical drift-diffusion model (HDDM) parameter estimation. (**C**) Computational mechanism analysis included Bayesian inference of group differences in HDDM parameters, mediation analyses examining the computational mechanisms underlying

behavioral impairments, support vector machine classification of diagnostic groups, correlation analyses between HDDM parameters and behavioral/clinical indices, cross-task parameter consistency analyses, and task-type classification.



936 **Fig 2. Behavioral performance and clinical correlates of inhibitory control.**

937 (**A**) Raincloud plots depicting group differences in Stroop interference effect (left) and the
938 discriminability index *d'* from the Go/No-Go task (right). Group comparisons were conducted
939 using analysis of covariance with age, sex, and years of education as covariates; *p*-values were
940 corrected for multiple comparisons using the Benjamini–Hochberg false discovery rate
941 procedure. (**B**) Partial correlations between behavioral performance indices and clinical
942 symptoms, controlling for age, sex, and years of education (Benjamini–Hochberg corrected).
943 ACC: accuracy; Con: Congruent; CPZ: Chlorpromazine; FDR: False discovery rate; FLU:
944 Fluoxetine; HAMA: Hamilton Anxiety Rating Scale; HAMD-17: 17-item Hamilton
945 Depression Rating Scale; HC: Healthy controls; Inc: Incongruent; MDD: Major depressive
946 disorder; Neu: Neutral; PANSS: Positive and Negative Syndrome Scale; RT: Reaction time;
947 SCZ: Schizophrenia.*p_{FDR}<0.05, **p_{FDR}<0.01, ***p_{FDR}<0.001.

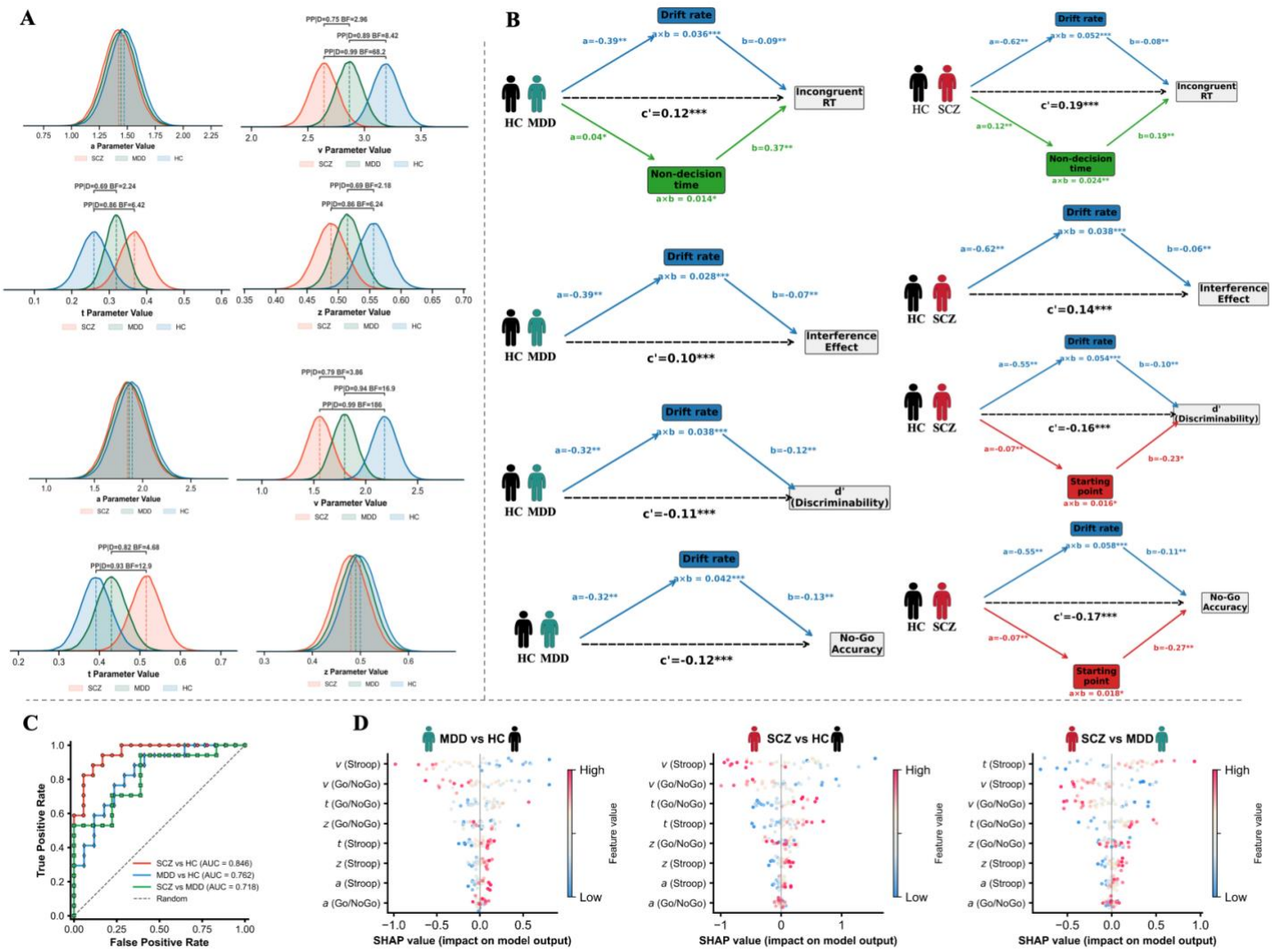


Fig 3. Hierarchical drift-diffusion model parameter estimates, mediation effects, and diagnostic classification.

(**A**) Posterior distributions of HDDM parameters across groups. Ridge plots display drift rate (*v*), non-decision time (*t*), starting point bias (*z*), and decision boundary (*a*) for each group and task. Vertical lines indicate posterior means; shaded regions represent 95% highest density intervals. (**B**) Mediation analyses examining HDDM parameters as mediators of group differences in behavioral performance. Path coefficients are standardized; only significant mediation pathways are displayed. (**C**) Receiver operating characteristic curves illustrating support vector machine classification performance using HDDM parameters as features. Each curve represents a pairwise diagnostic comparison; AUC values are displayed in the legend. (**D**) The SHapley Additive exPlanations (SHAP) summary plots illustrating feature importance for diagnostic classification. Features are ranked by mean absolute SHAP value (top to bottom); each point represents one participant from the test set; color indicates feature value (red and blue indicate high and low values, respectively); and horizontal position indicates the feature's contribution to classification. AUC: Area under the receiver operating characteristic curve; HC:

963 Healthy controls; HDDM: Hierarchical drift-diffusion modeling; MDD: Major depressive
964 disorder; SCZ: Schizophrenia. *p<0.05, **p<0.01, ***p<0.001.
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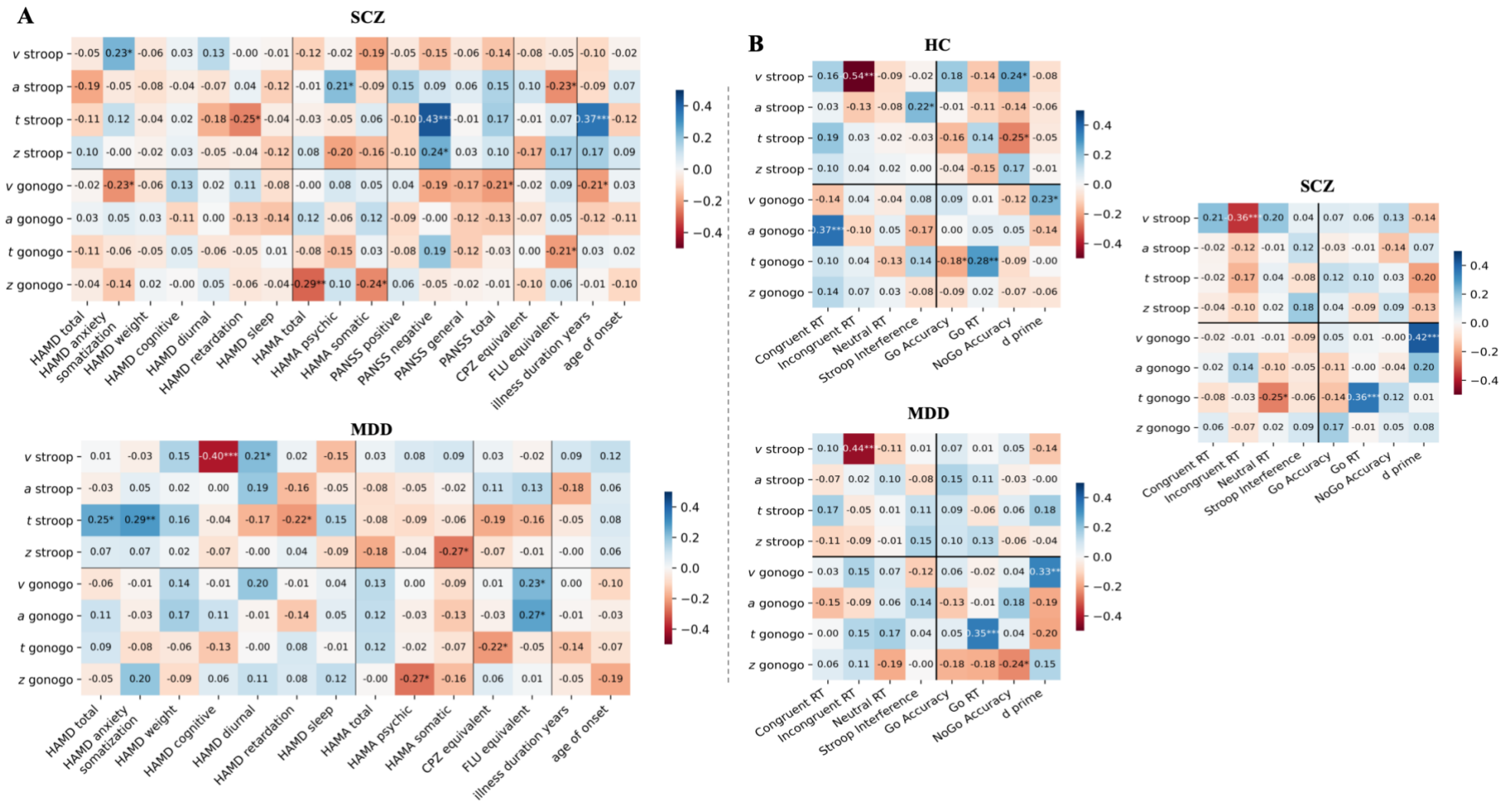

966 **Fig 4. Associations between HDDM parameters and clinical and behavioral indices.**

967 (**A**) Heatmaps displaying partial correlations between HDDM parameters and clinical symptom measures in the SCZ and MDD groups, controlling
968 for age, sex, and years of education. (**B**) Scatter plots with regression lines depicting correlations between HDDM parameters and behavioral task

indices across all three groups. All *p*-values were corrected using the Benjamini–Hochberg FDR procedure. *a*: Decision boundary; CPZ: Chlorpromazine; FDR: False discovery rate; FLU: Fluoxetine; HAMA: Hamilton Anxiety Rating Scale; HAMD: Hamilton Depression Rating Scale; HC: Healthy controls; MDD: Major depressive disorder; PANSS: Positive and Negative Syndrome Scale; RT: Reaction time; SCZ: Schizophrenia; *t*: Non-decision time; *v*: Drift rate; *z*: Starting point bias.

$*p_{FDR}<0.05$, $**p_{FDR}<0.01$, $***p_{FDR}<0.001$.

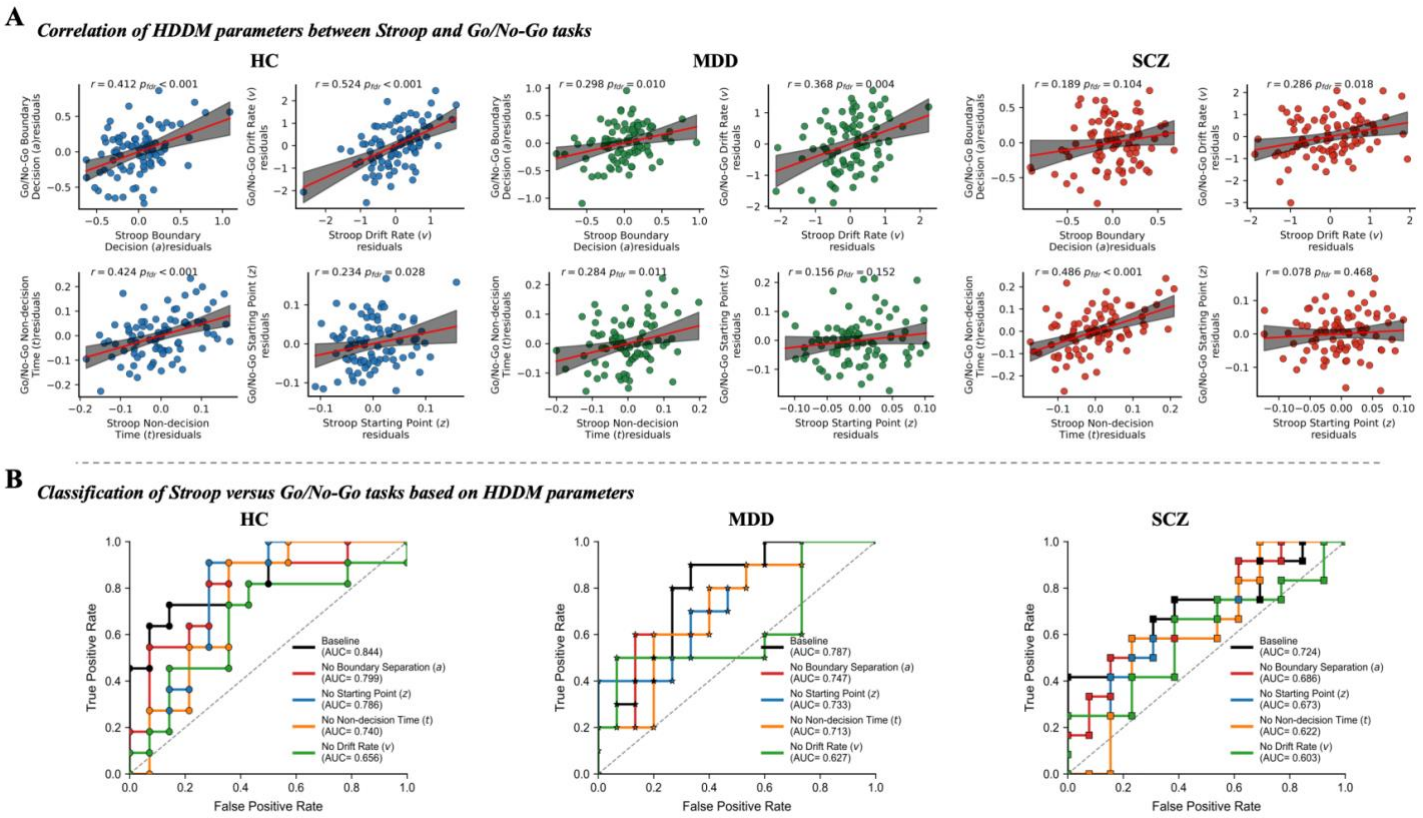

Fig 5. Cross-task parameter consistency and task discriminability.

(A) Scatter plots displaying within-group correlations of drift rate between the Stroop and Go/No-Go tasks. Each point represents one participant; regression lines with 95% confidence intervals are shown for each group. Pearson correlation coefficients are displayed; between-group differences in correlations were tested using Fisher's *z* transformation (Benjamini–Hochberg corrected). (B) Bar plots displaying AUC values for support vector machine classification distinguishing between Stroop and Go/No-Go task trials based on HDDM parameters, computed separately within each group. Error bars represent 95% confidence intervals derived from bootstrap resampling. Asterisks indicate significant differences from the HC group based on permutation tests. AUC: Area under the receiver operating characteristic curve; FDR: False discovery rate; HC: Healthy controls; HDDM: Hierarchical drift-diffusion modeling; MDD: Major depressive disorder; SCZ: Schizophrenia. *p_{FDR}<0.05, **p_{FDR}<0.01, ***p_{FDR}<0.001.

Table 1. Participant demographics and clinical characteristics

Variable	HC (*N*=86)	MDD (*N*=86)	SCZ (*N*=87)	Statistic	p_{FDR}-value
Demographic characteristics					
Age	35.86 (11.78)	38.34 (12.56)	36.72 (11.48)	F=1.14	0.322
Sex	M=43, F=43	M=41, F=45	M=47, F=40	χ^2=1.18	0.554
Ethnicity	Han=79, Other=7	Han=78, Other=8	Han=78, Other=9	χ^2=0.52	0.774
Education (years)	12.28 (3.54)	11.82 (3.68)	11.18 (3.52)	F=2.06	0.130
RPM	48.52 (8.68)	43.86 (9.42)	42.42 (10.76)	F=11.86	**<0.001** [a]
SES*	24.12 (6.18)	23.86 (6.72)	23.51 (6.35)	F=0.24	0.786
BMI	23.24 (3.58)	23.38 (3.82)	24.82 (4.18)	F=4.12	**0.042** [b]
Only child, yes, *n* (%)	14 (16.3%)	15 (17.4%)	12 (13.8%)	χ^2=0.48	0.786
Clinical characteristics					
Age of onset	–	30.86 (10.24)	27.00 (9.86)	t=−2.48	**0.014** [c]
Duration of illness (years)	–	7.48 (4.16)	9.72 (6.38)	t=−3.16	**0.002** [d]
Family history *n* (%)	–	19 (22.1%)	24 (27.6%)	χ^2=1.86	0.172
Antidepressant *n* (%)	–	78 (90.7%)	23 (26.4%)	χ^2=72.46	**<0.001**
Antipsychotics *n* (%)	–	18 (20.9%)	87 (100%)	χ^2 =113.82	**<0.001**
CPZ equivalent (mg/day)	–	152.36 (98.42) [e]	418.36 (228.74)	t=−5.86	**<0.001**
FLU equivalent (mg/day)	–	35.84 (14.62)	24.28 (12.36) [f]	t=3.24	**<0.001**
HAMD-17					
HAMD-17 total score	–	23.02 (4.58)	11.72 (5.18)	t=13.42	**<0.001**
HAMD anxiety/somatization	–	7.86 (2.14)	4.28 (1.92)	t=11.86	**<0.001**
HAMD weight	–	1.24 (0.68)	0.86 (0.72)	t=3.68	**<0.001**
HAMD cognitive disturbance	–	4.52 (1.86)	2.64 (1.48)	t=7.58	**<0.001**
HAMD diurnal variation	–	1.36 (0.82)	0.92 (0.76)	t=3.72	**<0.001**
HAMD retardation	–	4.18 (1.64)	2.42 (1.36)	t=7.82	**<0.001**
HAMD sleep disturbance	–	3.86 (1.42)	2.18 (1.28)	t=8.24	**<0.001**
HAMA-14					
HAMA total score	–	18.12 (5.36)	10.28 (4.72)	t=10.24	**<0.001**
HAMA psychic anxiety	–	10.24 (3.18)	5.86 (2.64)	t=10.12	**<0.001**
HAMA somatic anxiety	–	7.88 (2.86)	4.42 (2.38)	t=8.76	**<0.001**
PANSS					
PANSS positive	–	–	15.42 (5.36)	–	–
PANSS negative	–	–	19.84 (6.28)	–	–
PANSS general	–	–	35.16 (8.42)	–	–
PANSS total	–	–	70.42 (15.68)	–	–

Data are presented as the mean (SD) or n (%). Demographic variables were compared using one-way ANOVA (F) for continuous variables and chi-squared tests (χ^2) for categorical variables. Clinical variables were compared between the MDD and SCZ groups using

independent-samples *t*-tests for continuous variables and chi-squared tests for categorical variables. Bold values indicate statistical significance (p_{FDR}<0.05).

* SES was calculated using a composite index; detailed calculation methods are described in Supplementary Methods.

[a] *Post hoc* comparisons: HC>SCZ (p<0.001), HC>MDD (p=0.032); MDD vs. SCZ (p=0.186).

[b] *Post hoc* comparisons: SCZ>HC (p=0.048); SCZ vs. MDD (p=0.089) and MDD vs. HC (p=0.782) were not significant.

[c] SCZ<MDD (earlier age of onset in SCZ).

[d] SCZ>MDD.

[e] n=18 patients taking antipsychotics.

[f] n=23 patients taking antidepressants.

ANOVA: Analysis of variance; BMI: Body mass index; CPZ: Chlorpromazine; F: Female; FDR: False discovery rate; FLU: Fluoxetine; HAMA-14: 14-Item Hamilton Anxiety Rating Scale; HAMD-17: 17-Item Hamilton Depression Rating Scale; HC: Healthy controls; M: Male; MDD: Major depressive disorder; PANSS: Positive and Negative Syndrome Scale; RPM: Raven's Progressive Matrices; SCZ: Schizophrenia; SD: Standard deviation; SES: Socioeconomic status.

1011 **Table 2. Inhibitory control task performance across groups**

Measure	SCZ (*N*=87)	MDD (*N*=86)	HC (*N*=86)	*F*-value	Corrected *p*-value	Tukey's HSD *post hoc* comparison		
						SCZ vs. MDD	SCZ vs. HC	MDD vs. HC
Stroop task								
Congruent RT (ms)	652.18 ± 138.64	614.72 ± 116.48	561.34 ± 96.82	6.42	**0.002**	0.118	**0.001**	0.052
Incongruent RT (ms)	868.42 ± 172.36	782.16 ± 142.58	672.48 ± 114.26	24.86	**<0.001**	**0.004**	**<0.001**	**0.016**
Neutral RT (ms)	628.36 ± 132.48	601.84 ± 118.62	552.16 ± 92.74	9.86	**<0.001**	0.274	**<0.001**	**0.016**
Stroop interference (ms)	216.24 ± 82.46	167.44 ± 64.28	111.14 ± 52.36	32.48	**<0.001**	**0.018**	**<0.001**	**0.002**
Go/No-Go task								
Go accuracy	0.94 ± 0.05	0.96 ± 0.04	0.97 ± 0.03	10.24	**<0.001**	**0.042**	**<0.001**	0.062
Go RT (ms)	428.64 ± 96.82	391.28 ± 82.46	352.18 ± 66.74	14.86	**<0.001**	0.184	**<0.01**	**<0.01**
No-Go accuracy	0.68 ± 0.14	0.76 ± 0.11	0.85 ± 0.09	28.64	**<0.001**	**0.006**	**<0.001**	**0.008**
d′	1.86 ± 0.72	2.34 ± 0.58	2.84 ± 0.48	34.26	**<0.001**	**0.006**	**<0.001**	**0.008**

Data are presented as the mean±SD. Group differences were examined using ANCOVA with age, sex, and education as covariates. *Post hoc* pairwise comparisons were conducted using Tukey's HSD test. Bold values indicate statistical significance ($p<0.05$).

Stroop interference effect=incongruent RT − congruent RT.

$d'=z$ (hit rate) − z (false alarm rate), where hit rate=Go accuracy/100 and false alarm rate=(100 − No-Go accuracy)/100.

ANCOVA: Analysis of covariance; *d′*: Discriminability index; HC: Healthy controls; HSD: Honestly significant difference; MDD: Major depressive disorder; RT: Reaction time; SCZ: Schizophrenia; SD: Standard deviation.

1012