Impaired Evidence Accumulation as a Transdiagnostic Vulnerability Factor in Psychopathology

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Abstract

There is substantial interest in identifying biobehavioral dimensions of individual variation that cut across heterogeneous disorder categories, and computational models can play a major role in advancing this goal. In this report, we focused on efficiency of evidence accumulation (EEA), a computationally characterized variable derived from sequential sampling models of choice tasks. We created an EEA factor from three behavioral tasks in the UCLA Phenomics dataset ($n=272$), which includes healthy participants ($n=130$) as well participants with schizophrenia ($n=50$), bipolar disorder ($n=49$), and attention-deficit/hyperactivity disorder ($n=43$). We found that the EEA factor was significantly reduced in all three disorders, and that it correlated with an overall severity score for psychopathology as well as self-report measures of impulsivity. Although EEA was significantly correlated with general intelligence, it remained associated with psychopathology and symptom scales even after controlling for intelligence scores. Taken together, these findings suggest EEA is a promising computationally-characterized dimension of neurocognitive variation, with diminished EEA conferring transdiagnostic vulnerability to psychopathology.

Introduction

The standard approach to psychiatric nosology, reflected in the widely used DSM¹ and ICD² systems, emphasizes discrete diagnostic categories, each assumed to have its own distinct pathophysiology. An alternative approach, central to the Research Domain Criteria (RDoC) initiative^{3–5}, understands mental disorders in terms of fundamental biobehavioral dimensions of variation that span disorders^{6,7}. In this second approach, there is a critical need to identify these fundamental dimensions of variation, which are assumed to operate at a latent mechanistic level and may not have easily appreciated, one-to-one relationships with observable symptoms³.

Computational psychiatry^{8–12} is a research field that aims to formally model complex behaviors, typically performance during carefully constructed experimental tasks, in order to better understand abnormal patterns of functioning in psychiatric disorders. Standard models of task performance used in this field typically feature a small number of parameters that reflect latent psychological functions, and which might represent candidate biobehavioral dimensions on which individuals vary in clinically-significant ways. Here we focus on sequential sampling models (SSMs)^{13–16}, a class of models that are widely-used in cognitive neuroscience and have a substantial track record of success in explaining behavioral and neural phenomena on choice tasks^{17–19}.

SSMs aim to capture performance on behavioral tasks in which subjects select from two or more response options—a class of tasks that subsumes a large portion of experimental cognitive paradigms. In the diffusion decision model (DDM)^{20,14,15}, a popular and well-validated SSM, task performance is described by a noisy decision process that drifts over time towards one of two decision boundaries. The average rate at which the process moves towards the

correct decision boundary is determined by the “drift rate” parameter v . The model also includes several additional parameters that determine other aspects of the decision process, which are discussed in Figure 1.

Figure 1: The Diffusion Decision Model (DDM). The model explains performance in a simple decision task in which participants must decide whether the stimulus presented was an “X” or “O”. Following stimulus presentation, the decision process drifts between the boundaries for each possible response. Gray traces represent the paths of the stochastic decision processes for individual trials. The black arrow represents the critical drift rate parameter, v , which determines the average rate at which the process drifts toward the correct response boundary (e.g., towards an “X” response on a trial in which an “X” stimulus was presented). The z parameter represents the starting point bias of the process (in the example, it is set midway between the two decision boundaries representing absence of bias). The a parameter determines where the upper boundary is set (with the lower boundary always set at 0) and can be used to index “response caution”, the quantity of evidence that is required before the decision boundary is reached. The model also includes a parameter for time spent on processes peripheral to the decision (**Ter**).

Although the DDM and related SSMs are used in experimental contexts to simulate and measure a wide variety of latent neurocognitive processes, the drift rate parameter is often of key interest. Individual differences in this parameter reflect the ability of the system to efficiently accumulate stimulus information that is relevant to selecting an appropriate response in the context of noisy information. Importantly, SSMs allow the drift rate to be disentangled from multiple other factors that affect task performance, which are indexed with the other model parameters, yielding much more precise estimates.

Efficiency of evidence accumulation (EEA), as indexed by the drift rate in SSMs, has several properties that make it attractive as a candidate transdiagnostic dimension. First, people exhibit stable individual differences in EEA^{21,22}, suggesting it has trait-like qualities. Second, EEA manifests across a wide variety of tasks, and individual differences in EEA are correlated across these tasks^{22,23}. This suggests EEA has some generality in influencing performance across multiple psychological domains. Third, EEA has been observed to be diminished in several psychiatric disorders^{16,24,25}, though most studies thus far have examined attention-deficit/hyperactivity disorder (ADHD)^{26–29}, and different tasks were used across these studies, making comparisons across disorders difficult.

The present study investigates the hypothesis that EEA is a transdiagnostic vulnerability factor in psychopathology, while addressing some of the limitations in previous research. We take advantage of the publicly-available UCLA Phenomics dataset, a large sample ($n=272$, with 142 having mental disorders) that was extensively characterized with structured clinical interviews, self-report scale measures of symptom dimensions, neurocognitive measures of cognition, and a number of behavioral tasks that can be analyzed in an SSM framework³⁰. We constructed an EEA factor from three behavioral tasks available in this sample, the Stroop task, Go/No Go task, and the Stop Signal task. We demonstrate that this EEA factor is substantially reduced across disorder categories, and it is significantly negatively associated with a global psychopathology severity score factor and trait impulsivity. We also assessed the relationship between EEA and general intelligence, and find that, although they are related constructs, EEA continues to explain unique variance in psychopathology even after accounting for individuals' intelligence.

Methods

1. Participants

Participants were recruited from the Los Angeles area as part of a larger study within the Consortium for Neuropsychiatric Phenomics at University of California, Los Angeles (www.phenomics.ucla.edu). All participants underwent structured clinical interviews, neuropsychological testing, and administration of a number of behavioral tasks. They in addition underwent two fMRI scanning sessions, but imaging data is not part of the present analysis. Comprehensive descriptions of the sample and measures collected are available elsewhere³⁰.

The present analysis relies on a sample of 272 subjects (Table 1). Psychiatric diagnosis was established by a Structured Clinical Interview for DSM-IV³¹. Primary diagnoses for the participants were: 130 healthy controls, 50 individuals with schizophrenia (21 paranoid type, 1 disorganized type, 11 undifferentiated type, 6 residual type, 11 schizoaffective), 49 individuals with bipolar I disorder (16 most recent episode manic, 24 most recent episode depressed, and 9

most recent episode mixed), and 43 individuals with ADHD (17 predominantly inattentive type, 7 predominantly hyperactive-impulsive type, 19 combined type).

	Healthy	Schizophrenia	Bipolar I	ADHD
<i>n</i>	130	50	49	43
<i>Age (mean, sd)</i>	31.3 +/- 8.7	36.5 +/- 8.9	35.3 +/- 9.0	33.1 +/- 10.8
<i>Gender (#male)</i>	68	38	28	21
<i>Years Schooling</i>	15.0 +/- 1.7	12.6 +/- 1.7	14.6 +/- 2.0	14.6 +/- 1.8

Table 1: Demographic Characteristics of the UCLA Phenomics Sample.

Some subjects were missing data for certain measures (Stroop Task $n=2$, Stop Signal Task $n=2$). As a result, the sample size available for most comparisons was 268 subjects.

Because there were differences in age, gender, and years of schooling across the patient groups, we re-ran all the analyses in Results sections 1 and 2 with age, gender, and years of schooling as covariates. Results from these analyses were highly similar, with all statistically significant results without these covariates remaining statistically significant with the covariates. The only exception is the comparison of EEA in the bipolar group with healthy individuals where the p value changes from 0.02 to 0.08.

Code files and data used for analyses in the current study are available on the Open Science Framework: <https://osf.io/cqksz/>. Raw data from the full original data set are available at: <https://openneuro.org/datasets/ds000030/versions/00016>.

2. Behavioral Tasks and Construction of an Evidence Accumulation (EEA) Factor

Three behavioral tasks in the UCLA Phenomics dataset met the basic assumptions of sequential sampling models. In particular, they were all choice tasks with discrete response alternatives, all afforded relatively rapid responses (under 1.5 seconds mean reaction time), and all had sufficient number of trials (ranging from 54 to 360 trials per task/condition). Full descriptions of all measures are available at the UCLA Phenomics Wiki page (<http://lcni-3.uoregon.edu/phenowiki/index.php/HTAC>). Brief descriptions follow:

Color/Word Stroop Task – This is a computerized version of the traditional Stroop task in which individuals are asked to respond as to the color (red, green or blue) of a word stimulus. The meaning of the word is either congruent with the color (e.g., “RED” in red coloring) or incongruent, matching a different possible color response (e.g., “BLUE” in red coloring). Participants were presented with 98 congruent and 54 incongruent trials in pseudorandom order. Each stimulus was presented for 150ms following a 250ms fixation cross and participants were allowed to respond during a subsequent 2000ms interval. Responses were immediately followed by an 1850-1950ms blank screen delay.

Go/No-Go Task – Participants were presented with a sequence of letters and were instructed to press a button as quickly as possible after the presentation of any letter except “X”, but to withhold their response after the presentation of “X”. The task contained 18 blocks presented in random order, with 6 blocks in each of three inter-trial interval (ITI) conditions: 1000ms, 2000ms, and 4000ms. Each stimulus was presented for 250ms and followed by a blank screen response interval of 750ms, 1750ms, or 3750ms depending on the ITI condition. Each block contained 20 trials (two of which were “X” trials) for a total of 360 trials across the entire task.

Stop Signal Task – Participants were presented with a series of “X” and “O” stimuli for 1000ms and were asked to press a button corresponding to the stimulus presented on that trial. Trials were preceded by a 500ms fixation cross and followed by a 100ms blank screen interval. On a subset of trials (25%, “stop” trials) an auditory tone which indicated that the participant should withhold their response to that trial (“stop signal”) was played following the stimulus presentation. The latency of the stop signal following the stimulus onset, or “stop signal delay” (SSD), was determined using a standard staircase tracking algorithm which dynamically adjusted the SSD on each “stop” trial in 50ms increments with the goal of obtaining an inhibition rate of approximately 50% for each individual. . Participants completed two blocks of 32 “stop” trials and 96 “go” (i.e., non-“stop”) trials each. The task’s primary dependent measure of response inhibition, stop-signal reaction time (SSRT), which is thought to index the latency of a top-down process that inhibits responses on “stop” trials³², was estimated using a quantile-based method outlined by³³. For the model-based analysis, we focused exclusively on the 192 “go” trials, following previous applications of sequential sampling models to the stop signal task^{26,34,35}.

The UCLA Phenomics dataset does not provide trial-level data for these tasks, but instead provides detailed per-subject summary statistics characterizing a wide variety of task dimensions. Thus, we used the “EZ-diffusion model” (EZDM:³⁶) approach. This method provides a closed-form analytic solution for estimating the main parameters of the DDM-type of sequential sampling model (see Introduction). Inputs to the EZDM procedure are three subject-level summary statistics: the proportion of correct decisions, the mean of correct response times, and the variance of correct response times. The EZDM procedure produces parameter estimates for individuals’ drift rate (ν), boundary separation (a : an index of response caution) and non-decision time (Ter : time taken up by perceptual and motor processes peripheral to processing of the choice).

As EZDM does not allow estimation of parameters that index response bias (which are likely relevant to the Go/No-Go task;³⁶) or additional parameters which describe features of performance in the incongruent condition of the Stroop task³⁷, it does not provide a comprehensive account of behavioral data. However, EZDM has been shown to produce parameter estimates and inferences that are highly similar to those drawn from more complex modeling methods^{38,39}, and some data suggests it recovers individual-differences—the main interest in this study—better than model-fitting methods that use trial-to-trial data⁴⁰. We also

note that, although the DDM is intended to describe two-choice tasks, it is possible to obtain comparable parameter estimates for three-choice paradigms, such as the current study's Stroop task, under the assumption that correct and error responses are similar across trials requiring different choices¹⁵. Hence, we adopted this simplifying assumption in order to allow drift rate estimates to be obtained from the Stroop task in this unique data set.

EZDM was applied separately to four task conditions: Stroop congruent trials, Stroop incongruent trials, Go/No-Go trials, and Stop Signal "go" trials. Parameters were estimated in R⁴¹ using the code from the original EZDM manuscript³⁶ with the scaling parameter (within-trial drift variability) set at 1. For the Go/No-Go task, proportion correct was computed by considering all trials (i.e., both "X" and non-"X"), and the response time mean and variance was set to the values for correct hits (trials on which individuals correctly inhibited their response do not have an observed response time), a method similar to that used in²⁷. For participants with perfect accuracy on any task, the edge correction recommended in the original article was used: We replaced the proportion correct value of 1 with a value that corresponds to the output of the following equation, where n is the number of trials in the task:

$$1 - \frac{1}{2n}$$

Next, factor analysis, implemented in SPSS 25 (IBM, Armonk, NY), was used to produce an EEA factor from the drift rate parameters derived from the four task conditions. This factor accounted for 50% variance of the variance in the drift rates. Factor loadings were, respectively for the four task conditions listed above: 0.82, 0.71, 0.73, 0.61. Cronbach's alpha was 0.69, and it did not increase by dropping any items.

3. Additional Neuropsychological and Symptom/Trait Factors

General Intelligence Factor

We produced a general intelligence factor from scores from nine cognitive tasks: WAIS-letter number sequencing, WAIS-vocabulary, WAIS-matrix reasoning, color trails test, WMS IV-symbol span, WMS IV-digit span, WMS IV-visual reproduction 1, WMS IV-visual reproduction 2, and California Verbal Reasoning Test. We used bifactor modeling, given prior results supporting the superiority of this type of model in this domain⁴². In particular, we utilized code from Dubois and colleagues⁴³, which in turn uses the omega function in the psych (v 1.8.4) package⁴⁴ in R (v3.4.4)⁴¹. The code performs maximum likelihood-estimated exploratory factor analysis (specifying a bifactor model), oblimin factor rotation, followed by a Schmid-Leiman transformation⁴⁵, yielding a general factor ("g") as well as specific factors.

Using this code, we found a bifactor model fit the data very well by conventional standards (CFI=0.991; RMSEA=0.030; SRMR=0.019; BIC=7.23). The solution, which included a general

factor and four group factors, is depicted in Figure 2. The general factor accounts for 72.2% of the variance (coefficient omega hierarchical ω^{46}), while the four specific factors accounted for 16.7% percent of the variance.

Figure 2: Bifactor Model of General Intelligence. We performed bifactor modeling of nine tasks in the UCLA dataset. The resulting model consisted of a general factor (“g”) and four group factors and exhibited excellent fit with the data. WMS = Wechsler Memory Scale; Repro. = Reproduction; WAIS = Wechsler Adult Intelligence Scale; Reason. = Reasoning.

Trait Impulsivity Factor

We used factor analysis to produce a trait impulsivity factor from scores on three self-report scales measuring impulsivity: Barratt Impulsivity Scale (total score) ^{47,48}, Dickman Impulsivity Scale (total dysfunctional) ⁴⁹, and Multidimensional Personality Questionnaire (self-control subscale) ⁵⁰. This factor accounted for 85% variance of the variance in the drift rates. Factor loadings for these scales, respectively, were: 0.91, 0.92, -0.94. Cronbach’s alpha was 0.77 and did not improve appreciably by removal of any of the scales.

General Severity Score for Psychopathology

Participants completed the Hopkins Symptom Checklist ⁵¹, a 56-item validated scale for symptoms associated with a broad range of psychiatric disorders. We used the global severity of psychopathology score from the Hopkins checklist, which is a summary score of total symptom load across all scale items.

Results

1. EEA is reduced across three mental disorders

As shown in Figure 3 and Table 2, the EEA factor was significantly reduced in all patients (collapsing across the three diagnoses) in comparison to healthy controls. It was also significantly reduced in each patient group separately in comparison to healthy controls. Effect sizes ranged from large (schizophrenia) to medium (all diagnoses and ADHD) to small-to-medium (bipolar disorder).

Figure 3: Impaired Evidence Accumulation Across Psychiatric Diagnoses. The evidence accumulation factor is significantly reduced in all patients, collapsing across the three diagnoses, in comparison to healthy controls. It is also reduced in each patient groups separately in comparison to healthy controls. HC= Healthy Controls; ALL DX=All three patient groups combined; SZ=Schizophrenia; BP=Bipolar Disorder; ADHD=Attention-deficit/hyperactivity Disorder; a.u.= arbitrary units. Error bars reflect represent standard error. *** = $p < 0.001$, ** = $p < 0.01$, * = $p < 0.05$.

	t	df	p value	Cohen's d
HC vs. ALL DX	5.48	266	<0.001	0.67
HC vs. SZ	6.57	174	<0.001	1.12
HC vs. BP	2.38	176	0.02	0.40
HC vs. ADHD	2.91	170	0.004	0.51

Table 2: Comparison of Efficiency of Evidence Accumulation (EEA) in Healthy Controls versus Patients with Psychiatric Disorders. EEA was significantly reduced across all disorder categories. HC= Healthy Controls; ALL DX=All three patient groups combined; SZ=Schizophrenia; BP=Bipolar Disorder; ADHD=Attention-deficit/hyperactivity Disorder.

2. EEA is inversely associated with the global psychopathology severity score and self-reported impulsivity

As shown in Figure 4, the EEA factor was inversely correlated with the global psychopathology severity score ($r=-0.197$, $p=0.001$). The EEA factor was also inversely correlated with the impulsivity factor ($r=-0.216$, $p<0.001$).

Figure 4: Evidence Accumulation Is Related to the P Factor and Impulsivity. The efficiency of evidence accumulation (EEA) factor was statistically significantly correlated with a global

psychopathology severity score derived from a self-report general symptom scale. The evidence accumulation factor was also significantly correlated with an impulsivity factor derived from three self-report scales of trait impulsivity. a.u.= arbitrary units.

3. EEA remains associated with psychopathology after controlling for general intelligence

The EEA factor was correlated with general intelligence ($r=0.432$, $p<0.001$), consistent with EEA playing a general role in diverse forms of cognitive processing. Given this correlation, we assessed whether after controlling for general intelligence, the EEA factor remained related to psychopathology, the global psychopathology severity score, and impulsivity. We found that even after these controls, EEA remained statistically significantly reduced in all patients (collapsing across the three diagnoses) in comparison to healthy controls ($F(1,267)=11.3$, $p=0.001$). It was also reduced in schizophrenia ($F(1,175)=8.5$, $p=0.004$) and ADHD ($F(1,171)=7.01$, $p=0.009$) but not bipolar disorder ($F(1,177)=2.1$, $p=0.15$). Additionally, the EEA factor remained statically significantly associated with the impulsivity factor (standardized beta $=-0.20$, $p=0.003$) and the global psychopathology severity score (standardized beta $=-0.12$, $p=0.03$).

4. EEA is more reliably linked to psychopathology than traditional dependent measures from the three behavioral tasks

We additionally assessed the relationship between more traditional dependent measures from the three behavioral tasks and measures of psychopathology. Table 3 shows five traditional dependent measures for these tasks (further clarification of these measures is found in the Table caption). We found that across 30 total comparisons, just five were statistically significant. It is also notable that in all five cases, the corresponding relationship between EEA and the respective measure of psychopathology was larger (compare Cohen's d values from Table 2 and Table 3).

	Stroop Reaction Time Effect	Stroop Accuracy Effect	Go/No-Go False Alarms	Go/No-Go d prime	Stop Signal SSRT
HC vs. ALL DX	$d=0.12$; $p=0.30$	$d=0.17$; $p=0.16$	$d=0.12$; $p=0.30$	$d=0.08$; $p=0.50$	$d=0.43$; $p=.001$
HC vs. SZ	$d=0.06$; $p=0.70$	$d=0.44$; $p=0.009$	$d=0.14$; $p=0.39$	$d=0.32$; $p=0.06$	$d=0.91$; $p<.001$
HC vs. BP	$d=0.07$; $p=0.66$	$d=0.11$; $p=0.49$	$d=0.04$; $p=0.81$	$d=0.11$; $p=0.50$	$d=0.21$; $p=0.20$
HC vs. ADHD	$d=0.23$; $p=0.18$	$d=0.17$; $p=0.33$	$d=0.21$; $p=0.24$	$d=0.09$; $p=0.59$	$d=0.17$; $p=0.32$

Table 3: Relationship Between Standard Dependent Measures In Three Behavioral Tasks And Measures Of Psychopathology. Of 30 comparisons, just five were statistically significant. Stroop

Reaction Time Effect = difference in reaction time between the incongruent and congruent condition; Stroop Accuracy Effect = difference in accuracy between the incongruent and congruent condition; d' prime = signal detection sensitivity parameter; SSRT = stop signal reaction time, i.e., the latency in initiating a stopping process after the presentation of the stop signal (this latency is inferred based on a model of task performance). d = Cohen's d . HC= Healthy Controls; ALL DX=All three patient groups combined; SZ=Schizophrenia; BP=Bipolar Disorder; ADHD=Attention-deficit/hyperactivity Disorder.

Discussion

Computational psychiatry opens up opportunities to rigorously identify and characterize clinically-significant biobehavioral dimensions of individual variation using precisely-specified mathematical models^{8,10–12}. This study appears to be the first to examine in a single group of subjects the relationships between evidence accumulation (EEA), a key computational parameter in sequential sampling computational models of choice tasks, and multiple measures of psychopathology diagnosis and symptoms. Our major findings are that: 1) EEA is significantly reduced in schizophrenia, bipolar disorder, and ADHD; 2) It is significantly negatively correlated with a global severity of psychopathology score as well as self-reported impulsivity; 3) EEA outperforms traditional metrics from behavioral tasks, such as reaction time and accuracy, in being more strongly associated with psychopathology and symptom scales. Taken together, these findings suggest diminished EEA is a promising computationally characterized transdiagnostic vulnerability factor in psychopathology.

Sequential sampling models (SSMs) are among the most widely used computational models in psychology and neuroscience^{13–16}. Key advantages of these models are that they capture complex and subtle features of response profiles across a broad range of psychological tasks⁵². Previous studies have also found that EEA values, as indexed by drift rate parameters in SSMs, are related across tasks^{21,22} and stable across sessions^{22,23}, suggesting they have trait-like properties. The current study extends these results by showing that EEA has broad, transdiagnostic relationships to psychopathology: It is reduced across all three disorder categories present in this sample, and it is inversely correlated with clinically relevant symptom scales.

Interestingly, while the EEA construct has been extensively invoked to explain task performance in hundreds of studies, the mechanistic basis of individual-differences in EEA, and how these differences relate to differences in other cognitive constructs, are much less explored (but see 29, 48). Several studies using perceptual decision tasks find drift rates are related to general intelligence^{54–57}, similar to the pattern we found in the present study. But, again similar to prior work, the correlations we found in this study between EEA and general intelligence were moderate in size, with individuals' general intelligence explaining less than 25% of the variance in EEA in this sample. Furthermore, there was evidence that EEA was distinct from these other constructs. For example, EEA continued to be related to multiple measures of psychopathology,

including schizophrenia, ADHD, trait impulsivity, and a global psychopathology severity score even after adjusting for individuals' general intelligence. Hence, although additional research is needed, current evidence suggests that EEA and intelligence are related, but psychometrically dissociable individual-difference dimensions, and that these dimensions each explain distinct portions of the variance in individuals' psychopathology symptoms.

In contrast to EEA, standard dependent measures from the Stroop, Go/No-Go, and Stop Signal tasks performed relatively poorly in terms of being associated with psychopathology measures, with just five significant effects observed in 30 comparisons (without applying multiple comparisons correction), and these effects were scattered across different comparisons (see also 59, 60). We propose two explanations for why EEA was more strongly related to psychopathology than traditional metrics.

First, a recent pair of influential studies, each examining multiple behavioral tasks, found that standard dependent measures from these tasks exhibit relatively poor test-retest reliability^{60,61}. These observations are further supported by theoretical work with simulations that shows that difference score metrics (for example, Stroop interference effects) fare particularly poorly^{62,63}. One reason is that subtracting scores from one condition against another will, given plausible assumptions, increase noise relative to signal, thus diminishing test-retest reliability⁶². Since the reliability of a measure sets a ceiling on how well it can correlate with another variable⁶⁴, the low reliability of standard metrics derived from these tasks could help to explain their weaker relationship with psychopathology. In contrast, EEA estimates from tasks with sufficient numbers of trials have generally been demonstrated to exhibit good reliability (e.g., $r_s > .70$)²¹, thus potentially enabling higher correlation with psychopathology.

A second possible interpretation for the lack of predictivity of standard metrics, one that is not mutually exclusive with the first, is that these metrics reflect complex interactions among multiple factors, some of which relate to psychopathology but others of which do not⁶². Computational modeling can play a key role in disentangling these interacting factors, allowing researchers to identify underlying parameters that are more directly associated with clinically-significant dimensions^{24–26,65}.

For example, standard dependent measures from behavioral tasks, such as task accuracy or reaction time, cannot distinguish whether lower values in an individual are due to lower intrinsic ability on the task or preferences to trade off lower accuracy for greater speed (or lower speed for greater accuracy)⁶⁶. SSMS, in contrast, provide an explicit model of this tradeoff^{14,52,65}, which allows the rate of evidence accumulation for the correct response option to be distinguished from the threshold of evidence at which a response is selected (where higher evidence thresholds correspond to greater preference for accuracy over speed). The results of this study provide support for the view that computational models enable better quantification of underlying dimensions of inter-individual variation and yield stronger relationships with measures of psychopathology^{16,25–27}.

We found that EEA is reduced in transdiagnostically across several disorder categories. It is notable that these disorders—schizophrenia, bipolar disorder, and ADHD—are quite different in their clinical presentation. Recent work on the hierarchical structure of psychopathology^{67–69} helps to make sense of how high-level transdiagnostic abnormalities can co-exist with fairly distinct disorder syndromes.

Hierarchical models of mental disorders posit that individual disorder syndromes are members of higher-order spectra^{67,69,70}, which standardly include an externalizing spectrum (which includes ADHD), thought disorder spectrum (which includes schizophrenia), and an internalizing spectrum (which includes bipolar disorder, although some models place bipolar at the intersection of internalizing and thought disorder spectra;⁷¹). Spectra are themselves highly inter-correlated, pointing to the presence of a super-ordinate spectrum, often called the P factor^{72–75}. Hierarchical models posit that abnormalities that confer vulnerability to mental disorders typically operate simultaneously at multiple levels of the hierarchy: abnormalities operating at the spectra or P factor-level confer transdiagnostic risk, while abnormalities operating at the syndrome level explain the distinctive symptom profiles of individual disorders.

Given a hierarchical view of the structure of psychopathology, our results are most consistent with EEA abnormalities operating at a relatively high level in the hierarchy. We observed statistically significant reductions in EEA in three disorder categories that span the three most commonly posited spectra (externalizing, internalizing, and thought disorder). Moreover, we found EEA was statistically significantly correlated with the global severity of psychopathology score derived from the Hopkin's Questionnaire. The view that EEA operates at a high level in the psychopathology hierarchy makes a second prediction: Reduced EEA predisposes individuals to later development of a wide variety of mental disorders if these individuals are exposed to other disorder-specific risk factors that interact with this predisposition. This prediction could be tested in future studies, for example developmental studies that measure EEA in youth prior to the onset mental disorders. In recent studies^{76,77}, we measured EEA in young adults in a Go/No Go task. We found EEA was significantly (inversely) associated externalizing symptoms⁷⁶ and, importantly, was prospectively related with later substance use severity⁷⁷, providing some initial evidence for this second prediction.

The nature of EEA itself lends further support to the idea that it is a transdiagnostic vulnerability factor for mental disorders. EEA is conceptualized as a basic ability to rapidly extract information from a stimulus to select contextually appropriate responses. This is a highly general ability and thus reductions in EEA would be expected to increase the probability that inappropriate responses are produced across diverse psychological domains (attention, threat detection, motivation). Consistent with a hierarchical model of psychopathology, other abnormalities might operate more locally in individual psychological domains (e.g., enhanced sensitivity of threat detection systems). Together, interactions between high-level abnormalities such as impaired EEA and more local domain-specific abnormalities could produce the actually observed clinical pattern involving both substantial covariance as well as specificity in psychiatric symptoms.

The current study has several limitations. First, we used an EEA factor derived from three tasks in the UCLA Phenomics sample that met basic assumptions of SSMs. Future studies should extend this work by examining a more diverse range of tasks. In addition, given the availability of detailed subject-level summary statistics, we used the EZDM approach for calculating SSM parameters, rather than performing trial-by-trial modeling. EZDM results, however, usually correlate highly with results from trial-by-trial approaches, and some studies suggest EZDM can be more effective in quantifying individual differences^{38–40}. Relatedly, newer specialized modeling approaches are emerging for modeling “conflict” tasks in which correct responding requires overriding a pre-potent response^{37,78,79}. This is a fast-evolving area of research and future studies could employ these newer approaches. Finally, this sample included three major mental disorders, schizophrenia, bipolar disorder, and ADHD. A number of others disorders—for example, major depression, obsessive compulsive disorder, autism—were not well-represented in this sample, and future studies should examine a wider range of clinical populations to assess the robustness of our results across disorder categories.

In sum, this study demonstrates that EEA, a key parameter in SSMs, is reduced across several psychiatric disorders and is correlated with a global psychopathology severity score and trait impulsivity. EEA may hold promise as a basic dimension of inter-individual neurocognitive variation, and impaired EEA may be a transdiagnostic vulnerability factor in psychopathology.

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References

1. APA, A. P. A. *Diagnostic and statistical manual of mental disorders : DSM-V*. (American Psychiatric Association, 2013).
2. World Health Organization. *International statistical classification of diseases and related health problems (11th Revision)*. (2018).
3. Cuthbert, B. N. & Insel, T. R. Toward the future of psychiatric diagnosis: the seven pillars of RDoC. *BMC Med.* **11**, 126 (2013).
4. Cuthbert, B. N. The RDoC framework: facilitating transition from ICD/DSM to dimensional approaches that integrate neuroscience and psychopathology. *World Psychiatry Off. J. World Psychiatr. Assoc. WPA* **13**, 28–35 (2014).
5. Morris, S. E. & Cuthbert, B. N. Research Domain Criteria: cognitive systems, neural circuits, and dimensions of behavior. *Dialogues Clin. Neurosci.* **14**, 29–37 (2012).
6. McTeague, L. M., Goodkind, M. S. & Etkin, A. Transdiagnostic impairment of cognitive control in mental illness. *J. Psychiatr. Res.* **83**, 37–46 (2016).
7. Goodkind, M. *et al.* Identification of a common neurobiological substrate for mental illness. *JAMA Psychiatry* **72**, 305–315 (2015).
8. Wang, X.-J. & Krystal, J. H. Computational psychiatry. *Neuron* **84**, 638–654 (2014).

9. Paulus, M. P., Huys, Q. J. & Maia, T. V. A roadmap for the development of applied computational psychiatry. *Biol. Psychiatry Cogn. Neurosci. Neuroimaging* **1**, 386–392 (2016).
10. Montague, P. R., Dolan, R. J., Friston, K. J. & Dayan, P. Computational psychiatry. *Trends Cogn. Sci.* **16**, 72–80 (2012).
11. Huys, Q. J., Maia, T. V. & Frank, M. J. Computational psychiatry as a bridge from neuroscience to clinical applications. *Nat. Neurosci.* **19**, 404 (2016).
12. Adams, R. A., Huys, Q. J. & Roiser, J. P. Computational psychiatry: towards a mathematically informed understanding of mental illness. *J Neurol Neurosurg Psychiatry* **87**, 53–63 (2016).
13. Forstmann, B. U., Ratcliff, R. & Wagenmakers, E.-J. Sequential sampling models in cognitive neuroscience: Advantages, applications, and extensions. *Annu. Rev. Psychol.* **67**, (2016).
14. Ratcliff, R. & McKoon, G. The Diffusion Decision Model: Theory and Data for Two-Choice Decision Tasks. *Neural Comput.* **20**, 873–922 (2007).
15. Voss, A., Nagler, M. & Lerche, V. Diffusion Models in Experimental Psychology. *Exp. Psychol.* **60**, 385–402 (2013).
16. White, C. N., Curl, R. A. & Sloane, J. F. Using decision models to enhance investigations of individual differences in cognitive neuroscience. *Front. Psychol.* **7**, 81 (2016).
17. Smith, P. L. & Ratcliff, R. Psychology and neurobiology of simple decisions. *Trends Neurosci.* **27**, 161–168 (2004).
18. Turner, B. M., Van, L. M. & Forstmann, B. U. Informing cognitive abstractions through neuroimaging: the neural drift diffusion model. *Psychol. Rev.* **122**, 312–336 (2015).
19. Cassey, P. J., Gaut, G., Steyvers, M. & Brown, S. D. A generative joint model for spike trains and saccades during perceptual decision-making. *Psychon. Bull. Rev.* **23**, 1757–1778 (2016).
20. Ratcliff, R. A theory of memory retrieval. *Psychol. Rev.* **85**, 59 (1978).
21. Lerche, V. & Voss, A. Retest reliability of the parameters of the Ratcliff diffusion model. *Psychol. Res.* **81**, 629–652 (2017).
22. Schubert, A.-L., Frischkorn, G., Hagemann, D. & Voss, A. Trait characteristics of diffusion model parameters. *J. Intell.* **4**, 7 (2016).
23. Ratcliff, R., Thapar, A. & McKoon, G. Individual differences, aging, and IQ in two-choice tasks. *Cognit. Psychol.* **60**, 127–157 (2010).
24. Heathcote, A. *et al.* Decision processes and the slowing of simple choices in schizophrenia. *J. Abnorm. Psychol.* **124**, 961 (2015).
25. White, C. N., Ratcliff, R., Vasey, M. W. & McKoon, G. Using diffusion models to understand clinical disorders. *J. Math. Psychol.* **54**, 39–52 (2010).
26. Karalunas, S. L., Huang-Pollock, C. L. & Nigg, J. T. Decomposing attention-deficit/hyperactivity disorder (ADHD)-related effects in response speed and variability. *Neuropsychology* **26**, 684 (2012).
27. Huang-Pollock, C. L., Karalunas, S. L., Tam, H. & Moore, A. N. Evaluating vigilance deficits in ADHD: A meta-analysis of CPT performance. *J. Abnorm. Psychol.* **121**, 360 (2012).
28. Weigard, A., Huang-Pollock, C. & Brown, S. Evaluating the consequences of impaired monitoring of learned behavior in attention-deficit/hyperactivity disorder using a Bayesian hierarchical model of choice response time. *Neuropsychology* **30**, 502 (2016).
29. Weigard, A. & Huang-Pollock, C. A diffusion modeling approach to understanding contextual cueing effects in children with ADHD. *J. Child Psychol. Psychiatry* **55**, 1336–1344 (2014).

30. Poldrack, R. A. *et al.* A phenome-wide examination of neural and cognitive function. *Sci. Data* **3**, 160110 (2016).
31. First, M. B., Spitzer, R. L., Gibbon, M. & Williams, J. B. *Structured Clinical Interview for DSM-IV Axis I Disorders, Research Version, Patient Edition (SCID-IP)*. (Biometrics Research, New York State Psychiatric Institute, 2004).
32. Logan, G. D., Cowan, W. B. & Davis, K. A. On the ability to inhibit simple and choice reaction time responses: A model and a method. *J. Exp. Psychol. Hum. Percept. Perform.* **10**, 276–291 (1984).
33. Band, G. P. H., van der Molen, M. W. & Logan, G. D. Horse-race model simulations of the stop-signal procedure. *Acta Psychol. (Amst.)* **112**, 105–142 (2003).
34. Karalunas, S. L. & Huang-Pollock, C. L. Integrating impairments in reaction time and executive function using a diffusion model framework. *J. Abnorm. Child Psychol.* **41**, 837–850 (2013).
35. White, C. N. *et al.* Decomposing decision components in the stop-signal task: a model-based approach to individual differences in inhibitory control. *J. Cogn. Neurosci.* **26**, 1601–1614 (2014).
36. Wagenmakers, E.-J., Van Der Maas, H. L. & Grasman, R. P. An EZ-diffusion model for response time and accuracy. *Psychon. Bull. Rev.* **14**, 3–22 (2007).
37. Ulrich, R., Schröter, H., Leuthold, H. & Birngruber, T. Automatic and controlled stimulus processing in conflict tasks: Superimposed diffusion processes and delta functions. *Cognit. Psychol.* **78**, 148–174 (2015).
38. Dutilh, G. *et al.* The quality of response time data inference: A blinded, collaborative assessment of the validity of cognitive models. *Psychon. Bull. Rev.* 1–19 (2018).
39. van Ravenzwaaij, D., Donkin, C. & Vandekerckhove, J. The EZ diffusion model provides a powerful test of simple empirical effects. *Psychon. Bull. Rev.* **24**, 547–556 (2017).
40. van Ravenzwaaij, D. & Oberauer, K. How to use the diffusion model: Parameter recovery of three methods: EZ, fast-dm, and DMAT. *J. Math. Psychol.* **53**, 463–473 (2009).
41. R Core Team. R: A language and environment for statistical computing. *Doc. Free. Available Internet Httpwww R-Proj. Org* (2015).
42. Cucina, J. & Byle, K. The bifactor model fits better than the higher-order model in more than 90% of comparisons for mental abilities test batteries. *J. Intell.* **5**, 27 (2017).
43. Dubois, J., Galdi, P., Paul, L. K. & Adolphs, R. A distributed brain network predicts general intelligence from resting-state human neuroimaging data. *Philos. Trans. R. Soc. B Biol. Sci.* **373**, (2018).
44. Revelle, W. *psych: Procedures for Psychological, Psychometric, and Personality Research*. (2016).
45. Schmid, J. & Leiman, J. M. The development of hierarchical factor solutions. *Psychometrika* **22**, 53–61 (1957).
46. Zinbarg, R. E., Revelle, W., Yovel, I. & Li, W. Cronbach's α , Revelle's β , and McDonald's ω H: Their relations with each other and two alternative conceptualizations of reliability. *psychometrika* **70**, 123–133 (2005).
47. Barratt, E. S. Anxiety and impulsiveness related to psychomotor efficiency. *Percept. Mot. Skills* **9**, 191–198 (1959).

48. Patton, J. H., Stanford, M. S. & Barratt, E. S. Factor structure of the Barratt impulsiveness scale. *J Clin Psychol* **51**, 768–74 (1995).
49. Dickman, S. J. Functional and dysfunctional impulsivity: personality and cognitive correlates. *J. Pers. Soc. Psychol.* **58**, 95 (1990).
50. Tellegen, A. & Waller, N. G. Exploring personality through test construction: Development of the Multidimensional Personality Questionnaire. *SAGE Handb. Personal. Theory Assess.* **2**, 261–292 (2008).
51. Derogatis, L. R., Lipman, R. S., Rickels, K., Uhlenhuth, E. H. & Covi, L. The Hopkins Symptom Checklist (HSCL): A self-report symptom inventory. *Behav. Sci.* **19**, 1–15 (1974).
52. Ratcliff, R. & Rouder, J. N. Modeling response times for two-choice decisions. *Psychol. Sci.* **9**, 347–356 (1998).
53. Shine, J. M. *et al.* The dynamics of functional brain networks: integrated network states during cognitive task performance. *Neuron* **92**, 544–554 (2016).
54. Schmiedek, F., Oberauer, K., Wilhelm, O., Süß, H.-M. & Wittmann, W. W. Individual differences in components of reaction time distributions and their relations to working memory and intelligence. *J. Exp. Psychol. Gen.* **136**, 414 (2007).
55. Ratcliff, R., Thapar, A. & McKoon, G. Effects of aging and IQ on item and associative memory. *J. Exp. Psychol. Gen.* **140**, 464 (2011).
56. Schulz-Zhecheva, Y., Voelke, M., Beauducel, A., Biscaldi, M. & Klein, C. Predicting fluid intelligence by components of reaction time distributions from simple choice reaction time tasks. *J. Intell.* **4**, 8 (2016).
57. Schubert, A.-L. & Frischkorn, G. T. Neurocognitive Psychometrics of Intelligence: How Measurement Advancements Unveiled the Role of Mental Speed in Intelligence Differences. *Curr. Dir. Psychol. Sci.* 0963721419896365 (2020).
58. Duckworth, A. L. & Kern, M. L. A meta-analysis of the convergent validity of self-control measures. *J. Res. Personal.* **45**, 259–268 (2011).
59. Toplak, M. E., West, R. F. & Stanovich, K. E. Practitioner review: Do performance-based measures and ratings of executive function assess the same construct? *J. Child Psychol. Psychiatry* **54**, 131–143 (2013).
60. Hedge, C., Powell, G. & Sumner, P. The reliability paradox: Why robust cognitive tasks do not produce reliable individual differences. *Behav. Res. Methods* **50**, 1166–1186 (2018).
61. Enkavi, A. Z. *et al.* Large-scale analysis of test–retest reliabilities of self-regulation measures. *Proc. Natl. Acad. Sci.* **116**, 5472–5477 (2019).
62. Miller, J. & Ulrich, R. Mental chronometry and individual differences: Modeling reliabilities and correlations of reaction time means and effect sizes. *Psychon. Bull. Rev.* **20**, 819–858 (2013).
63. Rouder, J. N. & Haaf, J. M. A psychometrics of individual differences in experimental tasks. *Psychon. Bull. Rev.* **26**, 452–467 (2019).
64. Nunnally Jr, J. C. Introduction to psychological measurement. (1970).
65. Ratcliff, R., Love, J., Thompson, C. A. & Opfer, J. E. Children are not like older adults: A diffusion model analysis of developmental changes in speeded responses. *Child Dev.* **83**, 367–381 (2012).
66. Wickelgren, W. A. Speed-accuracy tradeoff and information processing dynamics. *Acta Psychol. (Amst.)* **41**, 67–85 (1977).

67. Kotov, R. *et al.* The Hierarchical Taxonomy of Psychopathology (HiTOP): a dimensional alternative to traditional nosologies. *J. Abnorm. Psychol.* **126**, 454 (2017).
68. Markon, K. E., Krueger, R. F. & Watson, D. Delineating the structure of normal and abnormal personality: an integrative hierarchical approach. *J. Pers. Soc. Psychol.* **88**, 139 (2005).
69. Krueger, R. F. & Markon, K. E. Reinterpreting comorbidity: A model-based approach to understanding and classifying psychopathology. *Annu Rev Clin Psychol* **2**, 111–133 (2006).
70. Andrews, G. *et al.* Exploring the feasibility of a meta-structure for DSM-V and ICD-11: could it improve utility and validity?: Paper 1 of 7 of the thematic section: 'A proposal for a meta-structure for DSM-V and ICD-11'. *Psychol. Med.* **39**, 1993–2000 (2009).
71. Goldberg, D. P., Andrews, G. & Hobbs, M. J. Where should bipolar disorder appear in the meta-structure?: Paper 7 of 7 of the thematic section: 'A proposal for a meta-structure for DSM-V and ICD-11'. *Psychol. Med.* **39**, 2071–2081 (2009).
72. Lahey, B. B. *et al.* Criterion validity of the general factor of psychopathology in a prospective study of girls. *J. Child Psychol. Psychiatry* **56**, 415–422 (2015).
73. Lahey, B. B. *et al.* Is there a general factor of prevalent psychopathology during adulthood? *J. Abnorm. Psychol.* **121**, 971 (2012).
74. Caspi, A. *et al.* The p factor: one general psychopathology factor in the structure of psychiatric disorders? *Clin. Psychol. Sci.* **2**, 119–137 (2014).
75. Caspi, A. & Moffitt, T. E. All for one and one for all: Mental disorders in one dimension. *Am. J. Psychiatry* **175**, 831–844 (2018).
76. Weigard, A. *et al.* Cognitive modeling informs interpretation of go/no-go task-related neural activations and their links to externalizing psychopathology. *Biol. Psychiatry Cogn. Neurosci. Neuroimaging* (2019).
77. Weigard, A. S. *et al.* Evidence accumulation and associated error-related brain activity as computationally informed prospective predictors of substance use in emerging adulthood. *bioRxiv* (2020).
78. White, C. N., Ratcliff, R. & Starns, J. J. Diffusion models of the flanker task: Discrete versus gradual attentional selection. *Cognit. Psychol.* **63**, 210–238 (2011).
79. Hübner, R., Steinhauser, M. & Lehle, C. A dual-stage two-phase model of selective attention. *Psychol. Rev.* **117**, 759 (2010).