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Testing Formal Predictions of Neuroscientific Theories of ADHD With a Cognitive Model–Based Approach

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Neuroscientific theories of attention-deficit/hyperactivity disorder (ADHD) alternately posit that cognitive aberrations in the disorder are due to *acute attentional lapses, slowed neural processing,* or *reduced signal-to-noise ratios.* However, they make similar predictions about behavioral summary statistics (response times [RTs] and accuracy), hindering the field's ability to produce strong and specific tests of these theories. The current study uses the linear ballistic accumulator (LBA; Brown & Heathcote, 2008), a mathematical model of choice RT tasks, to distinguish between competing theory predictions. Children with ADHD (n = 80) and age-matched controls (n = 32) completed a numerosity discrimination paradigm at 2 levels of difficulty, and RT data were fit to the LBA model to test theoretical predictions. Individuals with ADHD displayed slowed processing of evidence for correct responses (signal) relative to their peers but comparable processing of evidence for error responses (noise) and between-trial variability in processing (performance lapses). The findings are inconsistent with accounts that posit an increased incidence of attentional lapses in the disorder and provide partial support for those that posit slowed neural processing and lower signal-to-noise ratios. Results also highlight the utility of well-developed cognitive models for distinguishing between the predictions of etiological theories of psychopathology.

General Scientific Summary
Children with attention-deficit/hyperactivity disorder (ADHD) display slower response times and less accurate choices when completing choice response time tasks. This study demonstrates how mathematical models that describe cognitive processes underlying these tasks can be used to test theories about the causes of ADHD.

Keywords: ADHD, response times, LBA, model-based cognitive neuroscience

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The study of psychological dysfunction, like all sciences, advances through a process of conjectures and refutations. Scientific theories posit strong and specific predictions, and when they disagree with empirical data, a theory is discarded in favor of new theories that explain these data and make novel predictions of their own (Popper, 1963). However, the challenges of realizing such a progression continue to impede the growth of cumulative knowledge that is essential for public health. Attention-deficit/hyperactivity disorder (ADHD), one of the more prevalent mental health diagnoses in the United States (Fulton et al., 2009), is linked to a range of serious social and academic impairments (Loe & Feldman, 2007; Wehmeier, Schacht, & Barkley, 2010), but, due to the sparse knowledge of this disorder's etiology,[1] treatment is limited to interventions that work for a subset of children and fail to provide long-term gains (Molina et al., 2009). Coghill, Nigg, Rothenberger, Sonuga-Barke, and Tannock (2005) asserted that although multiple etiological theories of ADHD have been proposed, there are considerable challenges to testing them empirically, including causal heterogeneity, developmental change, and the need to integrate multiple levels of analysis (e.g., social and

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[1] Herein, we use the term *etiology* to mean any causal process that underlies the observed symptoms of attention-deficit/hyperactivity disorder, including distal factors, such as genes, as well as the neurocognitive processes that are the focus of this study.

genetic). In the decade since, important work has addressed these challenges (e.g., Fair, Bathula, Nikolas, & Nigg, 2012), but theories still abound, and many, including those present a decade ago, have yet to be conclusively refuted.

In the current study, we focus on another challenge to forming and testing causal theories of ADHD that we believe runs parallel to those identified by Coghill et al. (2005): the fact that controlled cognitive processes, which play a central role in neuroscientific theories of ADHD, are difficult to define and measure mechanistically. Aberrations in working memory, response inhibition, and decision-making are ubiquitous in psychiatric populations (Moritz et al., 2002; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005; Wright, Lipszyc, Dupuis, Thayapararajah, & Schachar, 2014); form the basis of many leading etiological theories of ADHD and related disorders (Nigg, 2000; Pennington & Ozonoff, 1996; Robbins, Gillan, Smith, de Wit, & Ersche, 2012); and serve as a crucial bridge between clinical research and current cognitive neuroscience. Yet, this line of work is currently limited by two interrelated factors.

The first challenge for this general paradigm is the fact that "executive functions," the broad category in which most cognitive processes relevant to psychopathology are placed (Halperin, 2016), are often difficult to define mechanistically. Critics have long pointed out that theories of these constructs tend to rely on a control "homunculus," or an intelligent agent who carries out complex operations (e.g., making decisions, managing memories) without an explanation of the specific mechanisms used to accomplish them (Monsell & Driver, 2000; Verbruggen, McLaren, & Chambers, 2014). Without a model of *how* computational and neural mechanisms complete cognitive tasks, findings of a cognitive deficit in a clinical condition may provide information about how the construct contributes to behavior (e.g., impulsive behavior due to poor inhibition) but are limited in their utility to test theories about the neurocognitive etiology of the condition.

Theoretical uncertainty about the mechanistic processes that underlie complex cognitive functions is compounded by the fact that dependent measures of cognitive performance are multidetermined. The integrity of a cognitive function is typically probed with summary statistics of behavioral performance on a task: response times (RTs) and accuracy rates. Yet, reductions in accuracy and/or increases in the mean or variance of RT may occur for a multitude of reasons: inefficiency in the core cognitive process, changes in caution (i.e., speed−accuracy trade-offs), momentary lapses of attention to the task, or difficulties initiating a motor response (McVay & Kane, 2012; Ratcliff & McKoon, 2008). This multidetermined nature is problematic because measurements of a process of interest may be contaminated by extraneous processes. Hence, competing etiological theories may posit that separate mechanisms are involved in a clinical condition, but the predictions they make about behavioral performance on a given task (e.g., slowed RT) may be identical.

Such is the case with several major neuroscientific theories of ADHD. Children and adults with ADHD display slower, less accurate, and more variable performance on a wide array of cognitive tasks (Castellanos et al., 2005; Epstein et al., 2011; Willcutt et al., 2005), and these findings have thus played a central role in many etiological theories. One causal account, endorsed by at least two major theories, posits that cognitive aberrations in ADHD are primarily due to intermittent attentional lapses, which temporarily hinder performance on affected trials but leave performance on other trials intact. The functional working memory model (Kofler et al., 2014; Rapport et al., 2008) holds that children with ADHD display incomplete neural maturation in the cortical regions that support the "central executive" component of working memory, causing increased mind-wandering and other variable processes that disrupt performance on an intermittent subset of trials. A similar proposal is made by a theory positing that spontaneous, low-frequency (<.1 Hz) fluctuations of the default mode network, a brain network that has been linked to resting and off-task states, produce periodic lapses of attention during task performance (Sonuga-Barke & Castellanos, 2007).

In contrast, a second causal account posits that cognitive deficits in ADHD are the result of metabolic constraints that limit the overall speed of neural computation. This account holds that children with ADHD exhibit insufficient production of lactate by astrocytes, which replenishes neural energy on repetitive tasks (Russell et al., 2006; Todd & Botteron, 2001). Insufficient production would be expected to delay the restoration of ionic gradients across neurons' cell membranes, which would slow neuronal firing rates and thus compromise the general speed with which individuals with ADHD would be able to complete repetitive cognitive operations. The behavioral neuroenergetics theory proposed by Killeen, Russell, and Sergeant (2013) refined this hypothesis by positing a model (detailed later) that describes the acute consequences of insufficient neuronal energy as slower and more variable RTs.

A third and distinct account is that cognitive deficits in ADHD are due to reductions in the ratio of task-relevant neural signal to task-irrelevant neural noise during cognitive processing. Karalunas, Geurts, Konrad, Bender, & Nigg, (2014) posited that state-regulation deficits in ADHD, mediated by impairments in phasic responses of, or top-down inputs to, the locus coeruleus−norepinephrine system (Aston-Jones & Cohen, 2005), result in reduced signal-to-noise ratios. Similarly, the moderate brain arousal (MBA) model (Sikström & Söderlund, 2007) references the concept of *stochastic resonance*, in which the addition of moderate amounts of noise to a system allows signals that would otherwise remain undetected to pass a detection threshold (Moss, Ward, & Sannita, 2004). The MBA model posits that individuals with ADHD require more noise than do typically developing individuals for stochastic resonance to occur, effectively reducing the signal-to-noise ratio.

Although these three general accounts—*acute attentional lapses, slowed neural computation,* and *reduced signal-to-noise ratios*—provide plausible explanations for behavioral dysfunction, the causal mechanisms that they highlight have similar effects on behavioral summary statistics: increases in the mean and variance of RTs (Karalunas, Nigg, et al., 2014; Killeen et al., 2013; Kofler et al., 2014). Because strong tests of theories require predictions to be unambiguous and distinct, analytic tools that extract additional information about the mechanisms that underlie task performance are essential to differentiate such theories.

Model-based cognitive neuroscience, an emerging field that integrates formal models of psychological processes with neuroscience methods and theory (Forstmann & Wagenmakers, 2015; Wiecki, Poland, & Frank, 2015), may provide a framework in which neuroscientific theories of ADHD, and other disorders, can be strongly distinguished. By formally describing how basic com-

putational mechanisms combine to execute complex tasks, mathematical models of cognition have the potential to move the field beyond verbal descriptions of cognitive dysfunction. In turn, the problem of multidetermined measures can be addressed by fitting these models to behavioral data, which allows researchers to observe how model parameters that represent specific mechanistic processes differ between clinically relevant conditions (White, Ratcliff, Vasey, & McKoon, 2010).

Sequential sampling models (Smith & Ratcliff, 2004), which explain decision-making as the gradual, noisy accumulation of sensory evidence in favor of each possible response, have already shown great promise in applications to clinical questions (White et al., 2010; Wiecki et al., 2015). Although there are multiple models in this class, they share the assumption that decisions are the result of a stochastic comparison process (Smith & Ratcliff, 2004), which can be understood with the metaphor of buckets that gather correct and erroneous drops of evidence. In a standard "numerosity discrimination" paradigm (Ratcliff & McKoon, 2008, p. 904), where participants decide whether a stimulus contains "many" (>50) or "few" (<50) asterisks (see Figure 1a), evidence from the stimulus would enter a noisy process in which it is compared to the *many* and *few* categories. Correct drops of evidence (i.e., drops in the *many* bucket after the presentation of a *many* stimulus) are bits of information that are attributed to the matching category, whereas erroneous drops are attributed to the other category. Typically, the bucket matching the stimulus fills first, but errors occur when noise causes excessive evidence to accumulate in the nonmatching bucket. "Accumulator" models (see Figure 1b) describe this process as a race between separate evidence accumulators toward a common threshold, whereas "diffusion" models (see Figure 1c) frame it as a single evidence total that moves between thresholds for each response (Smith & Ratcliff, 2004).

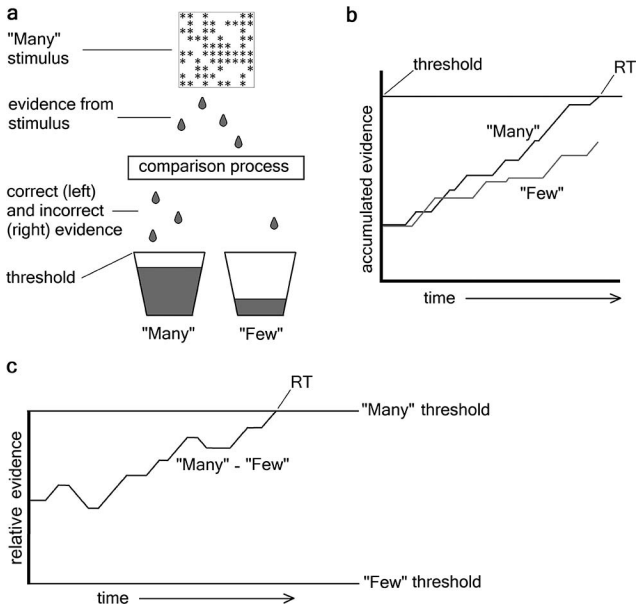
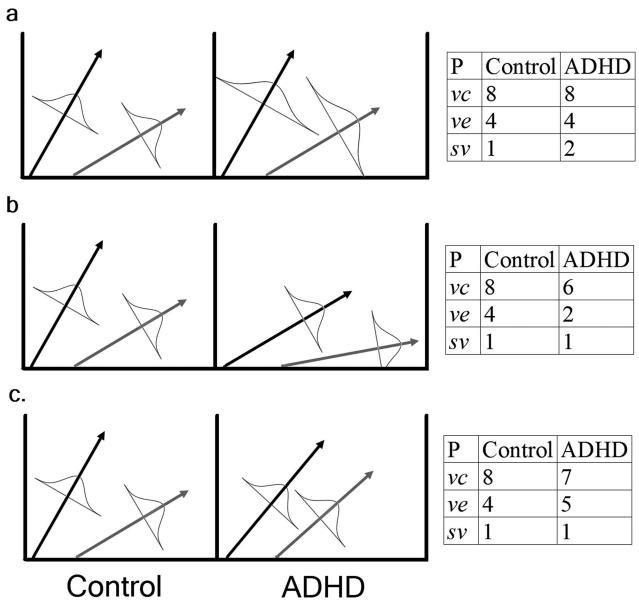


Figure 1. Representation of the stochastic comparison process assumed by sequential sampling models using the "bucket" metaphor (Panel a) and illustrations representing how "accumulator" (Panel b) and "diffusion" (Panel c) models each describe the decision process on a given correct trial. Information is gradually gathered from the stimulus, and then enters a stochastic process in which it is compared to the correct (many) and incorrect (few) stimulus categories. Many drops of information are routed to the correct bucket, but some are instead routed to the incorrect bucket due to noise. "Accumulator" models describe this process as a race between information accumulators over time that continues until one of the accumulators (in this case, the correct one) reaches an absolute threshold. "Diffusion" models describe this process as a relative evidence total (e.g., evidence for many vs. few) that drifts between two thresholds that represent each of the responses, until it terminates at one, and triggers the corresponding response. RT = response time

Initial work involving the Ratcliff diffusion decision model (DDM; Ratcliff, 1978), of the latter class, has already called the conventional interpretation of ADHD-related RT variability into question. Increased RT variability and positive skew are often assumed to reflect attentional lapses (Kofler et al., 2014). However, applications of the DDM to data from individuals with ADHD (Huang-Pollock, Karalunas, Tam, & Moore, 2012; Karalunas, Huang-Pollock, & Nigg, 2012; Metin et al., 2013) have consistently demonstrated that generally lower cognitive efficiency is sufficient to explain these RT features without reference to lapses. Despite these novel insights, no previous empirical studies have explicitly compared competing theories within the DDM framework. Furthermore, because the DDM does not estimate the accumulation speed of correct versus erroneous evidence separately (Smith & Ratcliff, 2004), the predictions of the *slowed neural computation* and *reduced signal-to-noise* accounts described earlier cannot be easily distinguished.

The linear ballistic accumulator model (LBA; Brown & Heathcote, 2008), which frames decisions as a race between two or more accumulators of smoothly and linearly increasing evidence for each response and provides a similar description of behavioral data to the DDM while simplifying several assumptions (Donkin, Brown, Heathcote, & Wagenmakers, 2011), allows the predictions of all three accounts to be made explicit. In the LBA, the rate of accumulation, or "drift rate," for an accumulator at a given trial is sampled from a normal distribution with a mean of v and a standard deviation of sv. Typically, the speed of evidence accumulation for the correct response (vc) competes with the speed of evidence accumulation for the error response (ve). The starting point of both accumulators is drawn from a uniform distribution bounded at 0 and the parameter A, and when one of the accumulators in the race reaches a response threshold (b), the corresponding response occurs. The model also has a nondecision time parameter (t_0), which indexes the amount of time in an RT that is taken up by processes that are not involved in the decision (e.g., encoding, motor response).

Relevant to the *acute attentional lapses* explanation, the LBA parameter indexing between-trial variability of drift rate (sv), which may intuitively be assumed to reflect variability in attentional state, has been both theoretically (Hawkins, Mittner, Boekel, Heathcote, & Forstmann, 2015) and empirically (McVay & Kane, 2012) linked to acute attentional lapses and mind-wandering. Therefore, attentional lapse theories predict that children with ADHD should show increases in sv relative to their typical peers (see Figure 2a). The explicit formalization of the behavioral neuroenergetics model (Killeen et al., 2013) describes the consequences of *slowed neural computation* by assuming that RTs in any task are the result of a single, general accumulation process toward a boundary. The drift rate of the process is assumed to be



P	Control	ADHD
vc	8	8
ve	4	4
sv	1	2

P	Control	ADHD
vc	8	6
ve	4	2
sv	1	1

P	Control	ADHD
vc	8	7
ve	4	5
sv	1	1

Figure 2. Demonstration of (left) and hypothetical parameter values for (right) three possible mechanistic causes for cognitive deficits in attention-deficit/hyperactivity disorder (ADHD): increased attentional lapses (Panel a), slowing in the general speed of neural computation (Panel b), and a reduced signal-to-noise ratio (Panel c). Black and gray arrows represent mean drift rates for correct (*vc*) and error (*ve*) information, respectively. Normal distributions centered about the arrows represent between-trial variance in drift. P = LBA model parameter.

determined by the amount of neuronal energy available and, for simplicity, to be the same for correct and error RTs. Therefore, as the available energy decreases, the rate of accumulation slows. In the LBA framework, such slowing should be reflected by reductions in both the *vc* and *ve* parameters in the disorder (see Figure 2b), because global slowing in firing rates would be expected to affect all neurons involved in the decision. Finally, the *reduced signal-to-noise* account posits that reductions in this ratio would lower an individual's ability to discriminate between stimuli, analogous to d' from signal detection theory (Karalunas, Geurts, et al., 2014). In the LBA, reduced d' can be reflected by reductions in the difference between the *vc* and *ve* parameters (Heathcote, Suraev, et al., 2015), as has been found in previous within-subject manipulations of stimulus discriminability (Ester, Ho, Brown, & Serences, 2014; van Maanen, Forstmann, Keuken, Wagenmakers, & Heathcote, 2016). Thus, reduced signal-to-noise should result in a lower ratio of *vc* relative to *ve* because of reductions in the former and increases in the latter (see Figure 2c), reflecting similar speed of neural computation but lower discriminability.

The current study seeks to explicitly test the predictions of the three causal accounts of ADHD-related performance deficits described earlier within the LBA model framework. We applied a hierarchical Bayesian implementation of the model (Turner, Sederberg, Brown, & Steyvers, 2013) to data from a perceptual decision task to produce accurate group-level estimates of the *vc, ve,* and *sv* parameters for children with ADHD and age-matched typically developing peers. It should be noted that these accounts do not provide an exhaustive list of etiological theories of ADHD and that findings of heterogeneity in the disorder (Fair et al., 2012; Karalunas, Fair, et al., 2014) suggest that hopes of identifying a single theory that explains impairment in all cases are likely unrealistic. However, we aimed to (a) provide initial steps toward using model-based analyses to narrow the field of proposed neuroscientific theories of ADHD and (b) demonstrate the utility of well-developed formal models from cognitive science for distinguishing the predictions of etiological theories of psychiatric disorders in general.

Method

Participants

Children ages 8−12 with (n = 80) and without (n = 32) ADHD (see Table 1) were recruited from a community sample as part of an ongoing study, which was approved by the Pennsylvania State University's Institutional Review Board (IRB No. 32126). Children with ADHD met criteria according to the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; American Psychiatric Association, 1994), including parent-reported age of onset, duration, cross-situational severity, and impairment based on the Diagnostic Interview Schedule for Children (4th ed.; DISC-IV; Shaffer, Fisher, & Lucas, 1997). In addition, at least one parent and one teacher report of behavior on the Attention, Hyperactivity, or ADHD subscales of the Behavioral Assessment Scale for Children (2nd ed.; BASC-2; Reynolds & Kamphaus, 2004) or the Conners' Rating Scales (Conners, 2001) was required to exceed the 85th percentile (T score > 61). Children in the ADHD group who were

Table 1
Descriptive Statistics of Diagnostic and Demographic Variables from Each Group

Variable	Control	ADHD
n (male:female)	32 (14:18)	80 (52:28)
No. of subtypes (H/I/C)		2/36/42
Age (in years)	9.03 (1.28)	9.43 (1.24)
Estimated full-scale IQ	105.31 (8.04)	102.54 (13.37)
Hyperactivity/impulsivity		
Total symptoms	.28 (.58)	5.66 (2.57)***
Parent BASC-2	40.97 (4.82)	67.38 (13.27)***
Parent Conners'	45.03 (3.24)	69.16 (14.42)***
Teacher BASC-2	42.97 (2.61)	58.82 (12.35)***
Teacher Conners'	45.03 (2.51)	58.26 (12.02)***
Inattention		
Total symptoms	.34 (.48)	7.91 (1.59)***
Parent BASC-2	42.72 (6.73)	66.51 (7.66)***
Parent Conners'	46.09 (3.91)	70.03 (10.54)***
Teacher BASC-2	43.16 (4.99)	60.90 (7.22)***
Teacher Conners'	46.59 (4.46)	59.14 (11.80)***
Comorbidity (DISC-IV; past year)		
MDD	0	4
GAD	0	6
ODD/CD	2/0	34/8

Note. ADHD = attention-deficit/hyperactivity disorder; H = Hyperactive/Impulsive type; I = Inattentive type; C = Combined type; BASC-2 = Behavioral Assessment Scale for Children (2nd ed.); Conners' = Conners' Rating Scales; DISC-IV = Diagnostic Interview Schedule for Children (4th ed.); MDD = major depressive disorder; GAD = generalized anxiety disorder; ODD = oppositional defiant disorder; CD = conduct disorder.
* $p < .05$. ** $p < .01$. *** $p < .001$.

prescribed a psychostimulant medication (n = 25; 31%) ceased taking their medication at least 24–48 hr in advance of the day of testing (Mdn = 56 hr). Controls had never previously been diagnosed or treated for ADHD and did not meet diagnostic criteria on the DISC-IV. In addition, they were required to fall below the 80th percentile (T score ≤ 58) on all of the previously listed rating scales. To equate IQ levels between groups, we excluded potential non-ADHD controls with an estimated IQ > 115, as well as children in both groups with IQ < 80.

The presence of common childhood psychopathology, such as anxiety, depression, oppositional defiant disorder, and conduct disorder, was assessed using the DISC-IV and standardized rating scales but was not exclusionary. Sample demographics, which reflected those of the larger region, were as follows: 71.4% Caucasian–non-Hispanic, 8.0% Caucasian–Hispanic, 1.8% other Hispanic, 10.7% African American, .9% Asian, 5.4% mixed, and 1.8% unknown–missing.

Experimental Procedure and Stimuli

The data described here were obtained during the second practice block of a task, which was designed to familiarize children with numerosity discrimination trials that would eventually be interleaved within a complex span working memory paradigm. Data from the first (simple spatial span) and third (complex spatial span, interleaved with numerosity trials) blocks are reported by Weigard and Huang-Pollock (2017). The majority of the control (n = 27; 84%) and ADHD groups (n = 71; 89%) in the current study were also part of the sample reported in this previous study. In the numerosity discrimination paradigm, children were asked to respond with a mouse click as to whether a randomly distributed array of black asterisks presented on an invisible 10 × 10 grid within a square box had "a lot" (i.e., >50, left mouse click) or "a little bit" (i.e., <50, right mouse click) of asterisks (called "candy"). One hundred trials were presented in random order; half were relatively easy to discriminate (Low Difficulty) and contained either 31–35 or 66–70 asterisks, whereas the other half were more difficult to discriminate (High Difficulty), containing either 41–45 or 56–60 asterisks. Stimuli remained onscreen until a response was made. A feedback cue (*Correct/Incorrect*) was then displayed for 500 ms above the stimulus, followed by a blank screen for 400 ms. Children were asked to complete the task as quickly and accurately as possible.

Model-Based Analyses

RT data were fit to a standard LBA model in which b was parameterized as the distance above the top of the start point distribution (A). An optimization model selection analysis (Donkin, Brown, & Heathcote, 2011), a procedure used in previous work to determine the best fitting sets of parameter constraints for the LBA (Heathcote, Loft, & Remington, 2015), was first used to select an optimal model to explain within-subject effects of stimuli and difficulty. Models with all possible sets of constraints were fit using maximum likelihood procedures, and the Akaike information criterion (AIC: Akaike, 2011) was used as an index of relative fit. This procedure suggested that an ideal model allowed (a) b to vary by response type (many−few) to account for response bias, (b) v to vary by accuracy of the information (correct/error, or *vc*/*ve*, as expected to account for above-chance performance) and difficulty (high−low), (c) sv to vary by accuracy (*svc*/*sve*) and stimulus type (many−few), and (d) common estimates of t_0 and A for all conditions and responses. To constrain the model (Donkin, Brown, & Heathcote, 2009), we fixed *sve* for "few" to 1 as a scaling parameter (the reasons for this constraint, and potential effects on group differences, are described in the online supplemental materials).

Following model selection, a hierarchical Bayesian version of the LBA (Turner et al., 2013) was fit to RT data from both groups to estimate posterior distributions over model parameter values. This method produces more stable parameter estimates than do traditional person-by-person analyses because it uses group-level posterior distributions of parameter values as prior distributions for individual parameter estimates (parameter "shrinkage"). The model assumed that individual-level parameters followed truncated normal distributions defined by two group-level hyperparameters, a mean (μ), and standard deviation (σ). Prior to estimation, RTs <200 ms and >3,000 ms were removed as fast guesses and outliers, respectively (these exclusion procedures eliminated <3.5% of the raw RT data), to prevent contaminant trials from affecting parameter estimates (Luce, 1986; Ratcliff & Tuerlinckx, 2002). To further reduce the influence of outliers and stabilize estimates, we employed a contaminant mixture (Ratcliff & Tuerlinckx, 2002) assumption in which 5% of trials, uniformly distributed between 200 ms and 3,000 ms and between correct and error responses, were assumed to be contaminants. Details of the priors, sampling procedure, and plots of model fit, which indicated that the model provided an adequate description of behavioral data, are available in the online supplemental materials.

Hypothesis Testing

Behavioral summary statistics were compared with traditional null-hypothesis significance tests (p values) and Bayes factors (BFs) using JASP (JASP Team, 2018).[2] BFs quantify the likelihood of the data under the research hypothesis (above 1) versus under the null hypothesis (below 1); a BF of 3, for example, indicates that the research hypothesis is 3 times more likely than the null hypothesis, given the observed data. Based on guidelines provided by Kass and Raftery (1995), BFs between 1 and 3 provide "anecdotal" or ambiguous evidence for the research hypothesis, BFs of 3−20 provide positive evidence, and BFs greater than 20:1 provide strong evidence.

For the estimated model parameters, we carried out inference at the group level using both a *BF* framework and a *parameter estimation* framework, which focuses on the location of model parameter posteriors. Each framework has individual limitations, and whether either provides an optimal method for Bayesian inference is the subject of current debate (Kruschke, 2013; Lee & Wagenmakers, 2014; Wagenmakers, Lodewyckx, Kuriyal, & Grasman, 2010). Inference in the parameter estimation framework was carried out by calculating Bayesian p values (Bp), which quantify the degree to which the posterior difference distribution is consistent with the hypothesis that a difference exists (Matzke,

[2] All Bayesian tests used standard JASP priors, including the Cauchy prior for effect size (width = .707) for t tests, prior effect size scale = .5 for fixed factors in analyses of variance, and prior effect size scale = 1 for random factors.

Hughes, Badcock, Michie, & Heathcote, 2017); B*p*s close to 0 indicate a greater likelihood of a difference. Inference using the BF framework was carried out using Savage-Dickey density ratios (*SDR*s), which approximate the BF by dividing the density of the posterior for a parameter at a value of interest from the density of the prior at the same value (Wagenmakers et al., 2010). Procedures for the calculation of both values are available in the online supplemental materials. We chose to use both methods of inference and, based on the Kass and Raftery (1995) guidelines,[3] adopted the following convention for interpreting results: If either $SDR < 3{:}1$ or B$p > .25$, evidence for the effect was considered ambiguous; if both $SDR > 3{:}1$ and B$p < .25$, evidence was considered moderate; and if both $SDR > 20{:}1$ and B$p < .05$, evidence was considered strong.

Results

Behavioral Summary Statistics

Mean RT. As expected, children with ADHD had longer RTs than did controls, $F(1, 110) = 12.63$, $\eta^2 = .10$, $p < .001$, BF = 23.65 (see Supplemental Table 1 in the online supplemental materials). There were also main effects of difficulty, $F(1, 110) = 95.25$, $\eta^2 = .46$, $p < .001$, BF > 10,000, and stimulus, $F(1, 110) = 31.15$, $\eta^2 = .22$, $p < .001$, BF > 10,000, such that RTs were generally faster for low-difficulty trials and stimuli in the *many* category. There were no significant interactions.

RT variability. Consistent with previous literature, children with ADHD had more variable RTs than did controls, $F(1, 110) = 21.54$, $\eta^2 = .16$, $p < .001$, BF = 1,015.67. Mirroring the mean RT effects, there were also main effects of difficulty, $F(1, 110) = 35.15$, $\eta^2 = .23$, $p < .001$, BF > 10,000, and stimulus, $F(1, 110) = 10.91$, $\eta^2 = .09$, $p = .001$, BF = 28.02, such that RTs were less variable for low-difficulty trials and *many* stimuli. There were no significant interactions.

Accuracy. Children with ADHD were less accurate than were controls, $F(1, 110) = 15.46$, $\eta^2 = .12$, $p < .001$, BF = 34.20, and accuracy was worse on high-difficulty trials, $F(1, 110) = 224.21$, $\eta^2 = .67$, $p < .001$, BF > 10,000. A small Group × Difficulty × Stimulus interaction was also detected by p values, but the BF indicated evidence against this interaction, $F(1, 110) = 4.00$, $\eta^2 = .03$, $p = .048$, BF = .02.

Model-Based Analysis

Posterior distributions for the group μ parameters are displayed as violin plots in Figures 3 and 4. Violin plots include both a standard boxplot that represents variability of the posterior samples and kernel density plots of the same samples. These plots represent uncertainty about the location of the μ parameter estimates but do not represent between-subjects variability. Between-subjects variability is, instead, captured by the σ hyperparameters and represented by population density plots that are reported in the online supplemental materials.

Correct drift rate. As expected, there was strong evidence for a main effect of difficulty (B$p = .01$, $SDR = 25.0{:}1$), such that *vc* was slower for more difficult trials (see Figure 3a). There was also strong evidence for a main effect of group (B$p = .01$, $SDR = 23.9{:}1$), in which children with ADHD displayed lower *vc* than did their peers. There was ambiguous evidence for a Group × Difficulty interaction (B$p = .66$, $SDR = 4.3{:}1$).

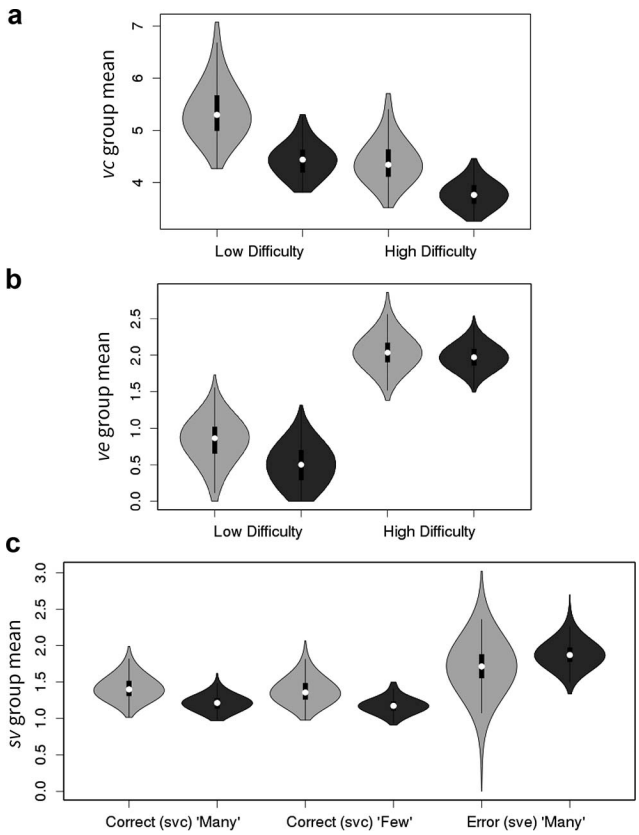


Figure 3. Violin plots representing the posterior density of group μ parameters for the average drift rate for correct evidence (*vc;* Panel a), average drift rate for error evidence (*ve;* Panel b), and between-trial drift variability for correct (*svc*) and error (*sve*) evidence (Panel c). Symbols within the violin density plots represent the median (white dot), interquartile range (black rectangle), and 1.5 times the interquartile range below the first quartile and above the third quartile (black lines) of the posterior samples. Light gray indicates control group; dark gray indicates the attention-deficit/hyperactivity disorder group.

Error drift rate. There was strong evidence for a main effect of difficulty (B$p < .01$, $SDR > 10{,}000{:}1$), such that *ve* was faster for the high-difficulty trials (see Figure 3b). This effect was expected because the reduced discriminability of high-difficulty stimuli should, theoretically, cause more evidence to be incorrectly routed to the error accumulator, consistent with previous research involving discriminability manipulations (Ester et al., 2014; van Maanen et al., 2016). In contrast, there was ambiguous evidence for group differences in *ve* (B$p = .20$, $SDR = .8{:}1$), and for an interaction (B$p = .29$, $SDR = .5{:}1$). Therefore, a reduction in *vc* and increase in *ve* explain the slower and more variable RTs, as well as higher error rates, observed in the high-difficulty condition,

[3] Conversions of BF criteria to Bp are based on the logic that if 3 to 1 odds in favor of the research hypothesis are considered "positive" evidence, a Bp of .25 (.25 = 1/4 = a 3 to 1 chance that the posterior is consistent with the hypothesis that a difference exists) should also be treated as positive evidence.

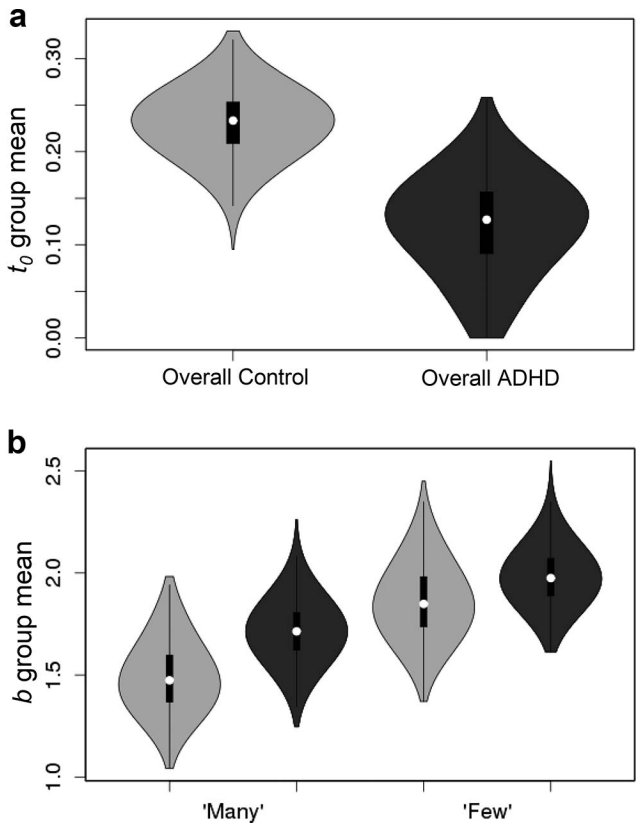


Figure 4. Violin plots representing the posterior density of group μ parameters for nondecision time (t_0; Panel a) and response boundaries (*b;* Panel b). Symbols within the violin density plots represent the median (white dot), interquartile range (black rectangle), and 1.5 times the interquartile range below the first quartile and above the third quartile (black lines) of the posterior samples. Light gray indicates control group; dark gray indicates the attention-deficit/hyperactivity disorder group.

reflecting decreased discriminability. However, slow and variable RTs, as well as higher error rates, among children with ADHD can only be attributed to slower *vc*.

Drift rate variability. Evidence for group differences in between-trial variability of drift rate was generally weak (see Figure 3c). Because *sv*s were estimated separately for *many* and *few* stimuli, based on the initial model selection analysis, and *sve* for *few* was fixed to 1, effects of group, stimulus, and interactions were probed for *svc,* whereas group effects in *sve* were probed for *many* stimuli. There was moderate evidence that *svc* was greater in controls (Bp = .05, *SDR* = 3.9:1), in the opposite of the hypothesized direction, and ambiguous evidence for an effect of stimulus (Bp = .38, *SDR* = 1.1:1) and an interaction (Bp = .49, *SDR* = 1.3:1). The *sve* of *many* stimuli (Bp = .28, *SDR* = .5:1) displayed ambiguous evidence for an effect of group. Thus, there was little evidence for group differences, and effects with the most evidence suggested greater *svc* in controls.

Nondecision time. There was moderate evidence (Bp = .02, *SDR* = 8.0:1) that nondecision time was shorter in ADHD (see Figure 4a). However, because shorter t_0 would be expected to decrease RT, and would not affect accuracy or RT variability, this difference does not explain the between-groups differences in summary statistics.

Start point variability and boundary. There was ambiguous evidence for group differences in *A* (Bp = .25, *SDR* = .7:1). Likewise, in *b* (see Figure 4b) there was ambiguous evidence for a main effect of group (Bp = .14, *SDR* = 2.1:1). Children displayed moderate evidence (Bp = .02, *SDR* = 10.4:1) for an effect in which boundaries for the *many* response were lower than those for the *few* response, and there was little evidence for an interaction (Bp = .37, *SDR* = 1.3:1). This pattern suggests that children were biased to respond *many* over *few* but that this bias does not contribute to group differences in RT and accuracy.

Between-subjects variability. ADHD is a heterogeneous disorder (Fair et al., 2012; Karalunas, Fair, et al., 2014), and aspects of the results just presented led us to speculate that the ADHD group may contain distinct subgroups of children with different impairments (see the Discussion section). We therefore sought to test the hypothesis that this group displays greater between-subjects variability in parameter estimates. To do so, we conducted a post hoc analysis in which we used Bayesian *p* values to assess whether group standard deviation parameters (σ) for *vc, ve, svc,* and *sve* (averaged between difficulty and *many/few* conditions) were greater in ADHD. We found rather weak evidence for greater between-subjects variability in *vc* in ADHD (Bp = .24) but moderate evidence for the same effect in *ve* (Bp = .07). In contrast, we found moderate evidence that *svc* estimates actually displayed greater between-subjects variability in the control group (Bp = .11) and little evidence for effects in *sve* (Bp = .35). Therefore, there is suggestive evidence that *ve,* and possibly *vc,* values are more heterogeneous in ADHD.

Discussion

The goal of the current study was to demonstrate the utility of formal cognitive models for better operationalizing and testing predictions from etiological theories of psychiatric disorders and to use this approach to provide a strong test of several neuroscientific accounts of cognitive deficits in ADHD. A hierarchical Bayesian implementation of the LBA model (Brown & Heathcote, 2008; Turner et al., 2013) was fit to behavioral data of children with ADHD and typically developing controls from a numerosity discrimination paradigm. Crucially, behavioral summary statistics demonstrated that children with ADHD were less accurate and had slower and more variable RTs than did their typically developing peers, suggesting that this simple task effectively captures the characteristic features of cognitive performance in ADHD. LBA parameters were then used to test three competing accounts of cognitive deficits.

Several results were highly relevant to these accounts. Both groups showed comparable within-subject effects of difficulty in which the rate of evidence accumulation for correct information (*vc*) decreased on high-difficulty trials but the rate of evidence accumulation for erroneous information (*ve*) increased. These effects reflect a decreased signal-to-noise ratio in the high-difficulty condition; high-difficulty stimuli are more similar to stimuli in the opposite category than are low-difficulty stimuli, which reduces their discriminability and causes more evidence to be accumulated in the incorrect accumulator, consistent with previous manipulations of stimulus discriminability (Ester et al., 2014; van Maanen

et al., 2016). However, group differences in mean drift rate parameters followed a distinct pattern. Although *vc* was lower in ADHD, there was little evidence that *ve* differed between groups, suggesting that slower, more variable and more erroneous responding in the ADHD group was driven only by slower accumulation of correct evidence. Further, between-trial variability in drift rate (*sv*) was unlikely to explain poorer performance in ADHD; evidence for group *sv* differences was relatively weak, and the most substantial evidence suggested that *svc* was actually greater in controls. Taken together, this pattern has implications for all three theoretical accounts.

The main prediction of the first general account, which posits that children with ADHD have a greater incidence of *acute attentional lapses,* was not supported by the model-based analysis. This account is proposed by at least two major theories, which hold that intermittent performance lapses occur either due to oscillatory activity within the default mode network during task engagement (Sonuga-Barke & Castellanos, 2007) or due to a core deficit in the central executive component of working memory (Kofler et al., 2014; Rapport et al., 2008). Evidence of group differences in the *sv* parameter, which has been empirically demonstrated to index such lapses (McVay & Kane, 2012), was weak and suggested that children with ADHD may actually have *less* between-trial variability in the speed of correct evidence accumulation (*svc*) than do controls.

Implications of the current results for the other two theoretical accounts are more complex. Because the *slowed neural computation* account (Killeen et al., 2013; Russell et al., 2006) predicts that the speed of evidence accumulation would be globally slowed in ADHD, it does not explain why only *vc* was reduced. Similarly, the *reduced signal-to-noise* account (Karalunas, Geurts, et al., 2014; Sikström & Söderlund, 2007) is partially supported by the decrease in *vc* relative to *ve*. However, it also predicts that ve would be greater in ADHD, similar to the effect produced by the within-subject discriminability manipulation. Although it appears that both of these accounts do not adequately describe the empirical data, there are two ways in which one or both may be compatible with the results.

First, it is possible that either may explain the selective effect in *vc* if specific assumptions are considered. Of the signal-to-noise theories, the MBA model, which posits that individuals with ADHD require more noise for stochastic resonance to occur, may provide a framework that most easily accounts for the data. If state-regulation processes increase signal-to-noise ratios through stochastic resonance, but this mechanism is less efficient in ADHD, controls would be expected to exhibit greater signal (*vc*) at comparable levels of noise (*ve*), as in the current study. However, the neuroenergetic theory (Killeen et al., 2013) may also explain the current data if it is assumed, for instance, that neurons that are most relevant for selecting correct responses exhaust their energy more quickly than do neurons that are less relevant to the task (and thus contribute to noise). To further explore the possibility that selective slowing in *vc* is congruent with one or both accounts, other predictions could be assessed in the LBA framework. The neuroenergetic theory predicts that neural speed (a) is reduced as time on task increases and interstimulus interval decreases and (b) is disproportionately reduced in ADHD (Killeen et al., 2013). Although the latter prediction has been confirmed with the DDM (Huang-Pollock et al., 2017), exploration of both predictions with the LBA could establish selective reductions in *vc* as a more specific marker of lower neuronal energy. Because the signal-to-noise accounts predict that arousal increases signal-to-noise ratios, experimental manipulations of reward or those that promote task engagement (e.g., gamelike features) would be expected to selectively increase *vc* but do so to a lesser extent in ADHD. Further, because phasic task-elicited pupil dilations may provide an index of arousal state (Gilzenrat, Nieuwenhuis, Jepma, & Cohen, 2010), a strong test of the signal-to-noise accounts could determine whether these pupillometric measures correlate with individual differences in, and mediate ADHD-related reductions in, *vc*.

Second, rather than explaining cognitive deficits across the broad population of individuals with ADHD, it is possible that one or both of these accounts may explain performance differences for distinct subsets of this population. Given recent evidence for heterogeneity in ADHD (Fair et al., 2012; Karalunas, Fair, et al., 2014), it is possible that a subgroup of children exhibits globally slowed processing, whereas a separate subgroup exhibits lower signal-to-noise ratios. Because both mechanisms would slow *vc,* but each would have opposite effects on *ve,* this combination could explain the group-level results. Indeed, our post hoc analysis provided tentative evidence that children with ADHD displayed greater between-subjects variability in mean drift, but not drift variability, parameters, and in *ve* in particular, providing initial support for this notion. The possibility that discrete subgroups exist could be further explored by entering individuals' LBA parameter estimates into clustering algorithms, such as community detection (Fair et al., 2012). Individual parameter estimates from the current study are inappropriate for these analyses because they were estimated with relatively few per-participant trials, and are therefore unreliable, and because individual parameters from hierarchical models are nonindependent. However, clustering analyses could be applied in the future to experimental data sets with larger numbers of trials per participant.

Several effects in LBA model parameters that were less relevant to explaining group differences in performance should also be noted, including the response bias effect in *b* and the finding that t_0 was shorter in ADHD. It is unclear why a bias toward *many* responses should exist in the numerosity task. However, because we called the asterisk stimuli "candy" in our instructions to children, this bias may relate to classic findings in which the subjective value of stimuli (e.g., many pieces of candy are better than few) affects perceptual estimation (Bruner & Goodman, 1947). The t_0 finding is consistent with findings in several previous DDM studies of ADHD (e.g., Karalunas & Huang-Pollock, 2013; Metin et al., 2013), although, because this parameter does not affect accuracy or RT variability, it does not explain the characteristic behavioral phenomena associated with ADHD. As has been previously noted (Karalunas, Geurts, et al., 2014), t_0 is likely multidetermined, and the implications of findings in this parameter are currently unclear.

Perhaps the most puzzling finding was the moderate evidence that *svc* was greater in controls, in the opposite direction of effects hypothesized by attentional lapse accounts. If *svc* is interpreted strictly as indexing lapses, this finding suggests the surprising conclusion that children with ADHD have fewer lapses than do their peers. However, several caveats of this finding, and our broader assertion that the *sv* results refute attention lapse accounts, are worth noting. First, the empirical evidence for *sv* as an index of

lapses is preliminary; a single study (McVay & Kane, 2012) found links between this parameter and reports of task-unrelated thoughts, as well as other constructs theoretically linked to lapses. A second limitation is that lapses may not cause drift variability that is perfectly Gaussian; because lapses would presumably cause trials with very slow drift rates, but not fast drift rates, there would be a disproportionate increase in variability at the low end of the drift distribution (rather than both ends, as in Figure 2a). Although a hypothetical model that accounts for negative skew in the drift distribution may better describe this phenomenon better than the LBA, with its assumption of Gaussian variability, McVay and Kane's (2012) work suggests that *sv* provides a reasonable approximation of variability caused by lapses. Finally, drift variability parameters are often more difficult to estimate than are other parameters in sequential sampling models (Voss, Nagler, & Lerche, 2013). Although our simulation−recovery study (see the online supplemental materials) suggested that the hierarchical model was able to obtain relatively reliable group estimates of *sv,* the reliability and construct validity of this parameter must be further explored in future research.

The current study has several additional limitations. First, because common psychiatric disorders that are often comorbid with ADHD were not exclusionary, it is possible that children with these conditions contributed to either the mean differences or the observed heterogeneity in the ADHD group. A second caveat is that data from a single task were used. Replication of the current results in different cognitive paradigms will be instrumental for both corroborating our findings and clarifying how task parameters may modulate them. A related limitation is that the current study did not explore how broader developmental, contextual, or biological factors, discussed by Coghill et al. (2005) as essential to testing theories of ADHD, were related to the model-based predictions. The combination of model-based techniques and prospective longitudinal studies that include measurements from multiple levels of analysis would arguably provide the most powerful tests of causal theories. We hope that the current study provides an initial step toward the regular inclusion of cognitive modeling in these larger studies.

In addition to these limitations of the data set, results also highlight some broader limitations with the model-based approach used. As stated in the introduction, the LBA was used in place of the DDM, another sequential sampling model commonly applied in the ADHD literature, because the race framework of the LBA allows separate estimates of drift for correct and incorrect evidence and thus allows the global slowing and lower signal-to-noise accounts to be explicitly distinguished. However, this raises a major conceptual issue with the approach of using well-developed models from cognitive science to test neuroscientific theories of clinical disorders; whether a model-based analysis provides evidence for a particular etiological theory is highly dependent on the assumptions of the specific model chosen. Furthermore, because the LBA analysis found partial support for both of these accounts, it could be argued that this approach was unable to parse them apart or address the possibility that both accounts could explain etiology for a subset of the heterogeneous clinical group. The LBA analysis advanced the field by identifying a unique mechanism of performance deficits in ADHD that both accounts must describe (selective slowing in *vc*) and by suggesting future tests of each account and methods for breaking down heterogeneity. Nonetheless, we acknowledge that as the nascent field of applying computational modeling methods to clinical questions continues to grow, it must wrestle with the major conceptual issues of the effects of model choice, how to interpret ambiguous results, and how to adequately describe heterogeneity in clinical populations.

Conclusions

The current study produced several key conclusions. First, the model-based analysis found that there was little evidence that children with ADHD exhibit greater between-trials variability in cognitive processing, casting doubt on etiological models that highlight intermittent performance lapses. Second, it demonstrated that children with ADHD display lower signal-to-noise ratios than do their peers in decision tasks but that this effect is distinct from those that occur in response to manipulations of stimulus discriminability; children with ADHD display slower accumulation of correct evidence (signal) but show similar accumulation rates of incorrect evidence (noise) compared to controls. Third, these results partially supported both the behavioral neuroenergetics theory (Killeen et al., 2013; Russell et al., 2006) and accounts highlighting lower neural signal-to-noise ratios (Karalunas, Geurts, et al., 2014; Sikström & Söderlund, 2007) and provide a roadmap for how these theories can be further tested, distinguished, and refined in a model-based framework. Specifically, they suggest that mechanisms through which signal detection is improved without concurrent reductions in noise should be further explored.

Overall, this work demonstrates how formal models of cognition can both make the mechanisms of etiological theories of psychopathology more explicit and provide strong tests of their predictions. Although the complexity of psychological phenomena may not allow all proposed mechanisms in such theories to be formalized, taking a model-based approach to those that can serves to advance the science as a whole by providing stronger conjectures and more definitive refutations.

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