# Modeling The Effects Of Methylphenidate On Interference And Evidence Accumulation Processes Using The Conflict Linear Ballistic Accumulator

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## Abstract

*Rationale*: Although methylphenidate and other stimulants have been demonstrated to improve task performance across a variety of domains, a computationally rigorous account of how these drugs alter cognitive processing remains elusive. Recent applications of mathematical models of cognitive processing and electrophysiological methods to this question have suggested that stimulants improve the integrity of evidence accumulation processes for relevant choices, potentially through catecholaminergic modulation of neural signal-to-noise ratios. However, this nascent line of work has thus far been limited to simple perceptual tasks and has largely omitted more complex "conflict" paradigms that contain experimental manipulations of specific top-down interference resolution processes. *Objectives and Methods*: To address this gap, this study applied the Conflict Linear Ballistic Accumulator (LBA), a newly proposed model designed for conflict tasks, to data from healthy adults who performed the Multi-Source Interference Task (MSIT) after acute methylphenidate or placebo challenge. *Results*: Model-based analyses revealed that methylphenidate improved performance by reducing individuals' response thresholds and by enhancing evidence accumulation processes across all task conditions, either by improving the quality of evidence or by reducing variability in accumulation processes. In contrast, the drug did not reduce bottom-up interference or selectively facilitate top-down interference resolution processes probed by the experimental conflict manipulation. *Conclusions*: Enhancement of evidence accumulation is a biologically plausible and task-general mechanism of stimulant effects on cognition. Moreover, the assumption that methylphenidate's effects on behavior are only visible with complex "executive" tasks may be misguided.



*Keywords*: methylphenidate, stimulants, evidence accumulation, conflict tasks, executive functions, cognitive modeling, computational psychiatry, Bayesian

Methylphenidate and other stimulants are among the most effective pharmacological interventions for symptoms of attention-deficit/hyperactivity disorder (ADHD: Pliszka & AACAP Work Group on Quality Issues, 2007; Stuhec, Munda, Svab, & Locatelli, 2015). The pharmacological action of stimulants at the cellular level, which involves blocking dopamine and norepinephrine reuptake from the synapse, has been well-characterized for decades (Solanto, 1998). However, the precise mechanisms through which these biophysical processes alter complex behavioral outcomes are still only partially understood.

A wealth of previous research demonstrates that acute stimulant administration increases accuracy and response speed on choice reaction time tasks and improves performance across a variety of paradigms thought to index complex cognitive functions implicated in ADHD, including attention, working memory and response inhibition (e.g., Coghill et al., 2014; Pietrzak, Mollica, Maruff, & Snyder, 2006; Reid & Borkowski, 1984; Rosch et al., 2016; Strand et al., 2010). Recently, Hawk and colleagues (2018) provided the first direct empirical evidence that methylphenidate-related improvements in cognitive performance partially mediate drug effects on clinical outcome measures in ADHD. Although this line of work supports the hypothesis that stimulants' effects on cognition are an important component of their clinical utility, an explanatory gap remains; a biologically-plausible account of how stimulant effects on catecholamine action improve performance across a variety of higher-order cognitive domains has yet to be elucidated.

One candidate account has been suggested by recent work within the framework of formal evidence accumulation models, which explain response time and accuracy on choice tasks as the product of a race between accumulators that gather noisy evidence for each possible choice over time until one choice "wins" by reaching a critical threshold of evidence (Smith & Ratcliff, 2004). Multiple models in this class have demonstrated considerable success in explaining patterns of behavioral data across a wide

variety of cognitive tasks (e.g., Brown & Heathcote, 2008; Usher & McClelland, 2001; Ratcliff & McKoon, 2008). Their biological plausibility is strongly supported by single-cell recordings in primates (Gold & Shadlen, 2007; Hanes & Schall, 1996; Smith & Ratcliff, 2004), which suggest that spiking activity within certain neural subpopulations during choice tasks displays properties consistent with an evidence accumulation process.

Given the importance of evidence accumulation for cognition, it is possible that methylphenidate and other stimulants improve cognitive task performance by enhancing evidence accumulation through their influence on catecholamine systems that modulate neural signal-to-noise ratios. This idea found support in a recent study by Fosco, White and Hawk (2017) in a model-based analysis of cognitive performance in children with ADHD, a condition associated with less efficient evidence accumulation overall (Ziegler, Pedersen, Mowinckel, & Biele, 2016; Weigard, Huang-Pollock, Brown & Heathcote, 2018). They found that the drug dramatically increased children's efficiency of evidence accumulation toward correct choices, reduced their threshold for responding (i.e., causing a faster, less cautious response style), and lengthened time spent on non-decision processes (e.g., motor speed). In addition, using electrophysiological methods, Loughnane et al. (2019) found that stimulant administration prior to oddball task performance in healthy adults reduces the latency and increases the build-up rate of the P3b potential, which is thought to correspond to evidence accumulation (Kelly & O'Connell, 2013).

These observations support the promising hypothesis that methylphenidate and other stimulants enhance evidence accumulation by selectively targeting relevant evidence (increasing signal) and/or by reducing variability in accumulation processes (decreasing noise). However, a key gap in existing work is that it has been limited to investigation of relatively simple perceptual tasks, for which most evidence accumulation models were designed. In contrast, most tasks in the literature on stimulants and ADHD involve more complex paradigms that are designed to explicitly measure specific top-down interference

resolution processes thought to be aberrant in ADHD (Coghill et al., 2014; Willcutt et al., 2005). For example, in "conflict" tasks, where irrelevant stimulus attributes, such as location in the Simon task (Hedge & Marsh, 1975) or nearby visual stimuli in the Flanker task (Eriksen & Eriksen, 1974), conflict with relevant attributes, a top-down interference resolution process is theorized to overcome the influence of irrelevant information. Hence, an important question concerns the selectivity of the drug's effects: does methylphenidate improve performance generally across both conflict and non-conflict tasks (or conflict and non-conflict conditions within a task), or are its effects at least partially selective to the top-down interference resolution process that is postulated to be operative under conditions of conflict? Evidence accumulation models could be highly useful in addressing this question, but extending these models to conflict tasks has proven challenging. There have been several important attempts to formulate evidence accumulation models for such tasks (Hübner, Steinhauser, & Lehle, 2010; Ulrich, Schröter, Leuthold, & Birngruber, 2015; White, Ratcliff & Starns, 2011), but their parameter recovery has thus far been found to be relatively poor (White, Servant & Logan, 2018), which significantly limits their use as measurement tools (Heathcote, Brown & Wagenmakers, 2015).

Recent work by Heathcote, Hannah and Matzke (under review) instead used existing parameters from the linear ballistic accumulator (LBA: Brown & Heathcote, 2008), a well-established evidence accumulation model, to explain behavioral effects on standard conflict tasks. The LBA (Figure 1a) frames decisions as a race between two or more accumulators (one for each possible choice) that gather evidence at a constant rate until one reaches a response threshold, denoted by a *b* parameter. On a given trial, the rate of accumulation for accumulators is drawn from a normal distribution with a mean of *v* and standard deviation of *sv*, which are both typically estimated separately for accumulators that *match* the stimulus (correct) and that *mismatch* the stimulus (incorrect). The model also contains parameters for uniformly-distributed start point noise (*A*) and time taken up by non-decision processes (*t0*).

Importantly, to account for conflict effects, Conflict LBA theory (Heathcote et al., under review) proposes that early interference from irrelevant stimulus attributes can be described as a priming effect that gives the accumulator for the choice favored by these attributes a head start in the race. Since increases in LBA start points are identical to threshold reductions, this effect is reflected by reductions in *b* for the matching (correct) accumulator on congruent trials and for the mismatching (error) accumulator on incongruent trials (Figure 1a). The theory further assumes that a top-down process attempts to correct for irrelevant information during the trial, but that this correction is often imperfect and varies substantially from trial to trial. This variability results in greater *sv* on incongruent trials while miscalibration in the top-down process is reflected by altered (typically reduced) *v* on incongruent trials. Heathcote et al. (under review) demonstrated that different combinations of these mechanisms (priming, plus calibration and variability in interference resolution processes) accounted for hallmark phenomena (e.g., delta function shapes: de Jong, Liang, & Lauber, 1994) across a variety of conflict tasks.

The current study applies the Conflict LBA to data from healthy adults who completed the Multi-Source Interference Task (MSIT: Bush & Shin, 2006) in a double-blind, placebo-controlled experimental trial of methylphenidate. We aimed to test whether methylphenidate effects on evidence accumulation seen in prior studies can be extended to conflict tasks. In addition, we were interested in whether methylphenidate affects the integrity of evidence accumulation in both congruent and incongruent task conditions or, alternatively, selectively facilitates processes in the incongruent condition, such as the top-down interference resolution process thought to operate in this condition.

## Methods

### Sample and Experimental Procedures

48 healthy participants were recruited from the community through ads on University of Michigan websites and flyers placed on campus and in other Ann Arbor locations. Of this initial sample,

two participants were excluded for failing to complete one of the experimental sessions and an additional participant was excluded due to apparent misunderstanding of the task (<50% accuracy in the incongruent condition at one session), leaving 45 participants (age 21.9 +/- 3.6 years, range 18-33; 24 females) for analysis.

All experimental procedures were approved by a local institutional review board and were in accordance with the ethical standards of the Declaration of Helsinki. In a double-blind, randomized, cross-over design, participants received either 40mg of methylphenidate (MPD) or a placebo (PBO) 80 minutes prior to performing an event-related MSIT task during fMRI scanning. Herein, we focus exclusively on modeling the behavioral data because we were concerned that fMRI results would be beyond the scope of the study and make the current report overly complex. Order of drug administration (MPD vs. PBO) was counter-balanced across subjects. The dose of MPD was chosen to optimize predicted effects on task performance in an acute dosing context, consistent with recent studies (Clatworthy, et al. 2009, Schlösser, et al. 2009). In particular, dosing was adjusted upwards relative to clinical dosing schedules to account for the fact that clinical dosing takes advantage of chronic administration to achieve higher steady-state blood levels.

The MSIT was modeled after the version of the task used by Bush et al. (2008). Participants were presented with white three-digit stimuli on a black background and were instructed to press a response button (1, 2 or 3) to indicate which digit was unique (e.g., respond “2” for the stimulus 020). In the congruent condition, the unique digit was always in the same serial position as the correct response and the other digits were 0s (e.g., 100, 003), while in the incongruent condition the position of the unique digit was mismatched with the that of the correct response and other digits represented competing responses (e.g., 233, 212). Participants completed 100 trials in each congruency condition, which were pseudo-randomly interspersed, at each experimental session. Subjects were told to respond

to stimuli rapidly and accurately. During each trial, stimuli appeared for 500ms, which was followed by a jittered ITI of 1-8.5s.

**Specification of Models**

The MSIT paradigm contains two possible forms of interference: a Simon-like effect, which primes the choice matching the serial position of the target in both congruent and incongruent conditions, and a Flanker-like effect, which produces interference in the incongruent condition only, as the flanking elements in the congruent condition (0s) do not correspond to a possible response. The Conflict LBA was previously found to describe behavioral data from both the Simon and Flanker paradigms well (Heathcote et al., under review), suggesting that this framework could jointly account for both types of effects on the MSIT. However, the LBA, like many cognitive and neuroscience models, displays highly-correlated parameters that can be difficult to estimate when individual-level data are sparse (Kolossa & Kopp, 2018; Gutenkunst et al., 2007). As a result, inclusion of all Conflict LBA parameters in situations where the number of trials in each condition is small and/or when errors are rare may result in parameter tradeoffs that compromise interpretability (e.g., Heathcote, Loft & Remington, 2015), making simplified models more useful (Heathcote et al., under review). A key concern for the current analysis is that *sv* parameters are particularly difficult to estimate in evidence accumulation models, in part because they trade off with *v* parameters, leading to inaccurate or biased estimates of both (Boehm et al., 2018).

As the MSIT had relatively few trials in each cell and low error rates, we sought to fit simplified versions of the Conflict LBA. On the basis of results from a targeted model selection analysis and several practical considerations, all of which are reported in detail in Supplemental Materials, we selected two models for use in the primary analyses: 1) a “mean drift rate” (MDR) model, in which *v* was allowed to vary by match/mismatch, congruency condition and drug condition, while *sv* was only

allowed to vary by match/mismatch, and 2) a "drift rate variability" (DRV) model in which *sv* was allowed to vary by match/mismatch, congruency condition and drug condition, while *v* was only allowed to vary by match/mismatch. Results from both models are reported below because parameter tradeoffs would likely prevent effects in *v* and *sv* from being easily distinguished from one another in models of this data set. Hence, we were able to assess whether evidence accumulation processes were enhanced by the drug or were enhanced in the congruent, relative to the incongruent, condition, but we were limited in our ability to make specific inferences about whether these improvements were due to increases in average rates of evidence accumulation or reductions in the variability of evidence accumulation.

We fixed *sv* for the mismatching accumulator in the MDR model and *sv* for the congruent condition mismatching accumulators in the DRV model to 1 in order to identify parameters (see Donkin, Brown & Heathcote, 2009). Due to the previous findings of stimulant effects on response thresholds and non-decision time (Fosco et al., 2017), both models also allowed *b* and *t0* to also vary by drug condition (MPD/PBO). Both models allowed *b* to explain priming from the Simon-like effect only (Figure 1b) due to practical challenges (Supplemental Materials) with allowing *b* to simultaneously explain the Flanker-like effect; nine *b* parameters were estimated in each drug condition, with different thresholds for each accumulator when the target was located at each of the three possible positions. This allowed for both priming (i.e., lower *b* for the accumulator corresponding to the target position) as well as response bias (i.e., different thresholds for each accumulator, allowing a lower *b* for those corresponding to favored responses). A single *A* parameter was estimated for each model across all conditions.

**Parameter Estimation and Hypothesis Testing**

Prior to model-fitting, RTs <300ms were removed as likely fast guesses (<1% of data). Due the task's low error rate, we used hierarchical Bayesian methods in Dynamic Models of Choice (DMC: Heathcote et al., 2018) to fit the Conflict LBA, as such methods allow estimation of group-level

parameters that can be used for inference and inform individual-level estimates, even when parameters are not well-identified at the individual level alone. In this model, group distributions of all LBA parameter values were assumed to follow truncated positive normal distributions described by location ($\mu$) and scale ($\sigma$) hyper-parameters, for which broad and non-informative priors were posited (all priors reported in Supplemental Materials). Differential evolution Markov chain Monte Carlo simulations (Turner, Sederberg, Brown & Steyvers, 2013), with start points determined by earlier simulations at the individual level, were used to sample from posterior distributions of all group- and individual-level parameter values. Sixty-five separate chains were used in the simulations, and thinning (retaining only every $10^{th}$ sample) was implemented to save file space. After an initial burn-in period that lasted until convergence was indicated by both visual inspection of chains (to ensure they were overlapping and stable) and the Gelman-Rubin statistic (G-R<1.10: Gelman & Rubin, 1992), 300 iterations of the simulation were retained, leaving 19,500 posterior samples per-parameter for inference.

Model fit was assessed with posterior predictive plots (Gelman, Meng & Stern, 1996), which allow visual inspection of how well the model accounts for key effects in behavioral data. Effects in parameter values were assessed using 95% credible intervals (CIs) of average posterior difference distributions of individual-level parameters. We considered effects to be reliable if the 95% CI did not contain 0. Procedures for calculating posterior difference distributions are described in Supplemental Materials. All code and data are available at: osf.io/t3cn5.

## Results

### Model Fit

Analyses of behavioral summary statistics (Supplemental Materials) indicated that RTs were longer and accuracy was lower in the incongruent, relative to congruent conditions, and that RTs were shorter in the MPD, relative to PBO, conditions. Plots of posterior predictive data compared with

empirical data for group average correct[1] RT quantiles (.1, .5 and 9) and group average accuracy rates are displayed in Figure 2 for both models. Black points and lines represent empirical data while gray violin density plots represent the range of values predicted by 500 samples drawn from posterior distributions of model parameters. Therefore, the density and spread of the violin plots represent uncertainty in predictions of the model due to uncertainty about the values of its parameter estimates. Both models provide an excellent description of correct RT quantiles and capture the RT main effects present in empirical data: shorter RT in the MPD condition and longer RT in the incongruent condition. Although both models captured the general pattern of lower accuracy rates in the incongruent condition well, predicted absolute values of accuracy rates in this condition were lower than those in the empirical data. However, the difference was of a small magnitude (2-3%), suggesting that this misfit is relatively minor.

Figure 3 displays group-averaged delta functions, which plot the difference between congruent and incongruent correct RT quantiles (.1, .3, .5, .7 and .9) on the y-axis against their mean on the x-axis, and conditional accuracy functions (CAFs), which display mean accuracy rates as a function of RT quantile bins. Delta functions showed a linear increasing pattern in both drug conditions, which is atypical for most variants of the Simon task and more common in Flanker paradigms (Heathcote et al., under review). Nonetheless, both models captured this pattern well, suggesting that they provided an excellent description of interference effects in RT. CAFs of the PBO condition indicated that fast (<.25 quantile) and slow (>.75 quantile) incongruent condition responses were slightly less accurate than responses in the middle of the range. CAFs of the MPD condition showed a clearer trend of reduced accuracy for fast responses, suggesting fast errors due to a possible speed/accuracy trade-off in this condition. The DRV model appeared to provide a better description of these CAF trends than the MDR

[1] Predictive plots of error RTs would be difficult to interpret due to low error rates, and are therefore not reported.

model.

**Model Parameter Estimates**

We focused on drug and congruency condition effects in several main parameters of interest, which are displayed in Figures 4 and 5 for the MDR and DRV models, respectively. For the MDR model, we assessed effects in both the mean *quantity* of evidence individuals are able to accumulate (the average of *v.match* and *v.mismatch*) and the mean *quality* of that evidence (*v.match* minus *v.mismatch*). Increases in evidence *quantity* mostly reduce response times, while increases in evidence *quality* mostly lead to more accurate responding. For the DRV model, we separately assessed effects in drift variability for the matching (*sv.match*) and mismatching (*sv.mismatch*) accumulators. For both MDR and DRV models, drift rate effects were also assessed using a *sensitivity* metric (Heathcote el al., 2015; Winkel et al., 2016), which is similar to d' from signal detection theory in that it indexes the difference between mean rates of the matching and mismatching accumulators (signal) relative to their variability (noise):

$$sensitivity = (v.match - v.mismatch)/\sqrt{(sv.match^2 + sv.mismatch^2)/2}$$

This summary metric provides an index of discrimination between choices in an accumulator model that can be compared across the MDR and DRV models despite the fact that, as noted above, congruency or drug effects in sensitivity cannot be specifically attributed to either *v* or *sv* differences given limitations of the current data set. For ease of interpretation, we averaged *b* parameter estimates for accumulators that were primed and separately averaged those that were not primed, with the goal of investigating effects of priming and drug condition on individuals' response thresholds in both MDR and DRV models. Finally, we assessed drug effects on non-decision time (*t0*) in both MDR and DRV models. For inference, medians (Δ) and 95% CIs of posterior difference distributions for all effects are reported below. Posterior difference distributions of interactions indicate the main effects of priming (primed – non-primed) or congruency condition (congruent - incongruent) in the PBO condition subtracted from

the same effects in the MPD condition.

*MDR model*. Evidence *quantity* was greater overall in the congruent condition, Δ =.36, CI = [.32, .41], and greater in the MPD condition, Δ =.19, CI = [.14, .24]. There was also evidence for a congruency x drug interaction, Δ =.14, CI = [.05, .23], in which the congruency-related differences in evidence *quantity* were of greater magnitude in the MPD condition, Δ =.43, CI = [.36, .50], than in PBO, Δ =.29, CI = [.24, .35]. Similarly, evidence *quality* was greater overall in the congruent condition, Δ =.41, CI = [.32, .51] and greater overall in the MPD condition, Δ =.13, CI = [.04, .22]. A congruency x drug interaction in which congruency-related effects on evidence *quality* were lower in MPD than in PBO was not reliably different from 0, Δ =-.15, CI = [-.33, .03]. *Sensitivity* was greater overall in the congruent condition, Δ =.47, CI = [.36, .57], and greater in the MPD condition, Δ =.15, CI = [.04, .25], likely because of the increased evidence *quality* present in both conditions. A congruency x drug interaction effect in *sensitivity* was not reliably different from 0, Δ =-.17, CI = [-.37, .03]. Response thresholds were lower for primed accumulators, Δ =-.63, CI = [-65, -.61], and lower overall in the MPD condition, Δ =-.18, CI = [-20, -.16]. A priming x drug interaction, Δ =-.06, CI = [-.10, -.02], indicated that the priming effect was larger in the MPD, Δ =-.66, CI = [-.69, -.64], than PBO condition, Δ =-.60, CI = [-.63, -.58]. Non-decision times were longer in MPD, Δ =.037 seconds, CI = [.030, .043].

*DRV model*. For *sv.match*, rates were less variable in the congruent condition, Δ = -.05, CI = [-.06, -.03], and less variable in the MPD condition, Δ = -.07, CI = [-.09, -.06]. There was evidence for a congruency x drug interaction, Δ = -.04, CI = [-.07, -.02], in which worsening of *sv.match* in the incongruent condition was of greater magnitude in the MPD condition, Δ = -.07, CI = [-.09, -.05], than in the PBO condition, Δ = -.03, CI = [-.04, -.01]. For *sv.mismatch*, rates were more variable in the congruent condition, Δ = .27, CI = [.25, .29], while the drug effect was not credibly different from 0, Δ = -.02, CI = [-.04, .00]. As *sv.mismatch* was fixed to 1 in the congruent condition, interactions in this

parameter were not assessed. *Sensitivity* was greater overall in the MPD condition, Δ =.28, CI = [.18, .38], consistent with findings from the MDR model, although increased sensitivity in the DRV model was due to reduced drift rate variability rather than to improved mean quality of evidence. In contrast to MDR model findings, *sensitivity* was lower in the congruent condition, Δ = -.88, CI = [-.99, -.78], and there was evidence for a congruency x drug interaction, Δ = -.28, CI = [-.48, -.08] in which congruency-related reductions in sensitivity were greater for the MPD, Δ =-1.02, CI = [-1.20, -.86], than for the PBO condition, Δ = -.74, CI = [-.87, -.62]. However, this interaction should be interpreted with caution due to the paradoxical direction of the congruency main effect (which implies better performance in the incongruent condition), and the fact that *sv.mismatch* was fixed to 1 across congruent trials, which may have led to reduced estimates of MPD-related sensitivity effects on these trials. Thresholds were lower for primed accumulators, Δ =-.70, CI = [-71, -.69], and lower in the MPD condition, Δ =-.14, CI = [-15, -.12]. A priming x drug interaction effect was not credibly different from 0, Δ =.00, CI = [-.02, .02]. The drug effect on non-decision time was not credibly different from 0, Δ =.001 seconds, CI = [-.006, .007].

## Discussion

The current study examined MSIT task data from healthy adults who received acute methylphenidate challenge in a randomized, placebo-controlled, double-blind, cross-over study. We applied two variants of the Conflict LBA (Heathcote et al., under review), a framework that extends the established LBA model (Brown & Heathcote, 2008) to describe interference-related processes in conflict paradigms. Consistent with previous research on simple perceptual tasks (Fosco et al., 2017; Loughnane et al., 2019), we found that methylphenidate improved performance by reducing response thresholds and by enhancing the sensitivity (signal-to-noise ratio) of evidence accumulation processes, although we were unable to pinpoint whether evidence accumulation was improved due to increases in the average quality of evidence (signal) or to reductions in evidence accumulation variability (noise).

Our results suggest that enhancement of evidence accumulation, in combination with associated reductions in response threshold, is a biologically-plausible, task-general mechanism through which stimulants improve cognition.

In contrast, methylphenidate did not appear to selectively influence processes that were directly probed by the experimental conflict manipulation. Differences between congruent and incongruent trials on the MSIT were accounted for both by a priming effect in thresholds, which biased early evidence toward choices indicated by irrelevant information, and by differences in drift rate processes between congruent and incongruent trials. Alternate models suggested that either 1) evidence quality and quantity were reduced in the incongruent condition (MDR model) or 2) rates for the matching accumulator become more variable, and those for the mismatching accumulator became less variable, in the incongruent condition (DRV model). The former effects may reflect miscalibration or insufficiency of interference resolution processes in the incongruent condition, while the latter may reflect variability in these processes (Heathcote et al., under review). Although inferences varied somewhat between the two models, there was agreement across models that methylphenidate did not reduce either the priming effect caused by irrelevant information or differences in evidence accumulation between congruent and incongruent trials. Indeed, when drug-related interaction effects were found, there was evidence that priming effects and congruency-related differences in drift rate variables were actually *increased* by the drug.

Notably, the presence of fast errors and reductions in response thresholds under stimulant challenge suggests that participants implemented speed/accuracy trade-offs in conditions where their evidence accumulation was improved, which are considered adaptive because they often further reduce RT without a significant decrement to accuracy rates (Dutilh, Vandekerckhove, Tuerlinckx, & Wagenmakers, 2009). However, it is unclear whether such adaptive threshold reductions are a direct

effect of the drug, or simply a secondary response to drug-related enhancements in evidence accumulation. It is also notable that, despite the general consistency of our findings with those of Fosco et al. (2017), only the MDR, and not the DRV, model replicated their finding of increased non-decision times under stimulant challenge. The fact that this effect, which is difficult to interpret (Fosco et al., 2017) and mirrors paradoxical findings of shorter non-decision times in ADHD (Karalunas, Geurts, Konrad, Bender, & Nigg, 2014; Weigard et al., 2018), was not robust across models suggests that it may result from parameter trade-offs, although additional work is needed to confirm this possibility.

Taken together, this pattern of results suggests that methylphenidate facilitates conflict task performance by enhancing evidence accumulation processes across all task conditions, rather than by selectively influencing specific processes, such as top-down interference resolution processes, that are proposed to be specifically activated in incongruent conditions. Such an account explains why methylphenidate-related facilitation effects are present across a wide variety of cognitive paradigms (Coghill et al., 2014; Pietrzak et al., 2006), including simple choice tasks (Reid & Borkowski, 1984). Moreover, it is consistent with an emerging set of findings from tasks that do not have overt manipulations aimed at taxing specific top-down processes (Fosco et al., 2017, Loughnane et al., 2019). When considered within the emerging body of work on methylphenidate and evidence accumulation processes, the current findings also support a biologically-plausible account of stimulant effects on behavior. Stimulants may increase the overall integrity of evidence accumulation across a wide variety of tasks and experimental conditions by facilitating catecholamine systems that optimize neural signal-to-noise ratios through modulation of arousal and/or neural gain, such as the locus coeruleus norepinephrine (LC-NE) system (Aston-Jones and Cohen, 2005). Such an account would be consistent with theories that implicate poor signal-to-noise ratios across a wide variety of tasks in ADHD due to deficits in catecholamine-mediated state-regulation processes (Karalunas et al., 2014; Weigard et al.,

2018; Sikström & Söderlund, 2007). It would also be consistent with related theories that implicate metabolic limits on evidence accumulation efficiency in ADHD, which are hypothesized to be related to norepinephrine action (Killeen, Russell, & Sergeant, 2013).

Findings from this study should be considered within the context of several limitations. First, the analysis involved simplified versions of the Conflict LBA that assumed priming could account for interference caused by the Simon-like manipulation, only, and that were not able to distinguish effects in mean drift rates from those in drift rate variability. Although these choices were informed by model selection analyses and practical constraints that are detailed in Supplemental Materials, it is unclear whether they affected our substantive inferences. Second, our MSIT task had a very low error rate, which both made parameter estimation difficult and may prevent our findings from generalizing to tasks with higher error rates. Both limitations underscore the need to experimentally validate a measurement model of the MSIT task, which would likely involve changes to the task that both increase the number of trials per cell and generate errors for the purpose of improving parameter estimation. Finally, although this study focuses on modeling behavioral data, our key conclusions should also be tested at the neural level of analysis. Specifically, future work should determine whether methylphenidate's effects on neuroimaging indices are also similar between conflict and non-conflict conditions.

In sum, we applied the Conflict LBA (Heathcote et al., under review), a novel mathematical model of conflict task performance, to MSIT data from healthy adults receiving acute methylphenidate or placebo challenge. We found stimulants improve task performance by enhancing evidence accumulation processes across both congruent and incongruent conditions, suggesting absence of selective facilitation of processes specific to the incongruent condition (e.g., top-down interference resolution processes). Our findings help clarify methylphenidate's mechanism of action and invite further computational and neurobiological investigation.

Figure Captions

**Figure 1.** Schematic of the model. **a)** LBA model of a standard Simon task, where shape (square vs. triangle) is the relevant information, including the priming process proposed by the Conflict LBA: *A* = start point variability, *b.pr* = threshold for primed accumulator, *b.non-pr* = threshold for non-primed accumulator, *v* = mean drift rate, *sv* = drift rate variability, *mat* = accumulator matching the stimulus, *mis* = accumulator mismatching the stimulus. **b)** Conflict LBA model used to explain behavior on the MSIT, which includes a similar (Simon-like) priming effect to that displayed above and also allows drift rate parameters to vary by congruency condition. Note that, in our primary analyses, we only allowed *v* to vary by congruency condition in the MDR model and only allowed *sv* to vary by congruency condition in the DRV model. For drift rate parameter labels: *c* = congruent, *i* = incongruent.

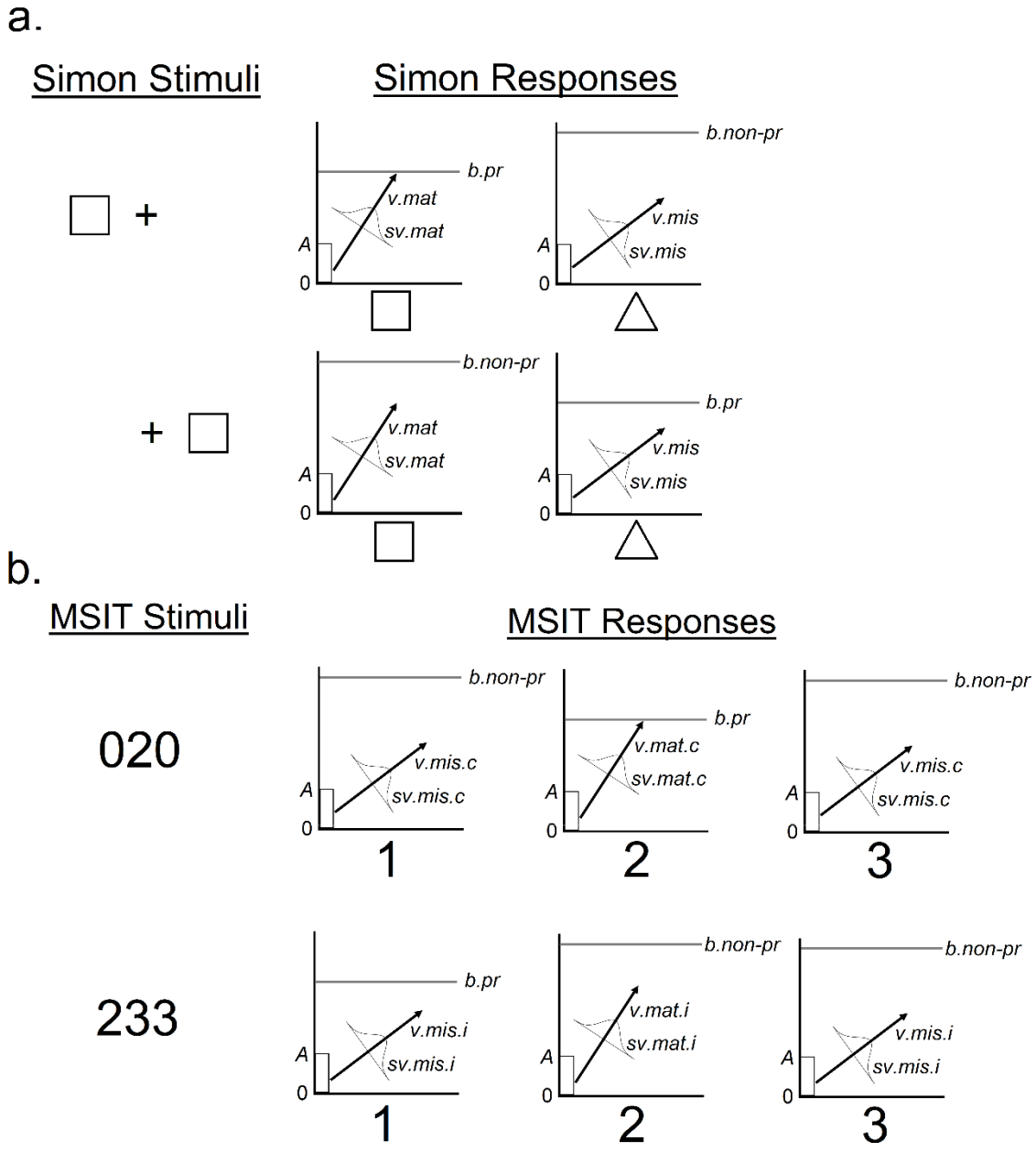


**Figure 2.** Posterior predictive plots of group average correct RT quantile and accuracy rate summary statistics from the empirical data (black points and lines) plotted with those predicted by 500 posterior samples (represented by gray violin density plots) from the MDR (left) and DRV (right) models.

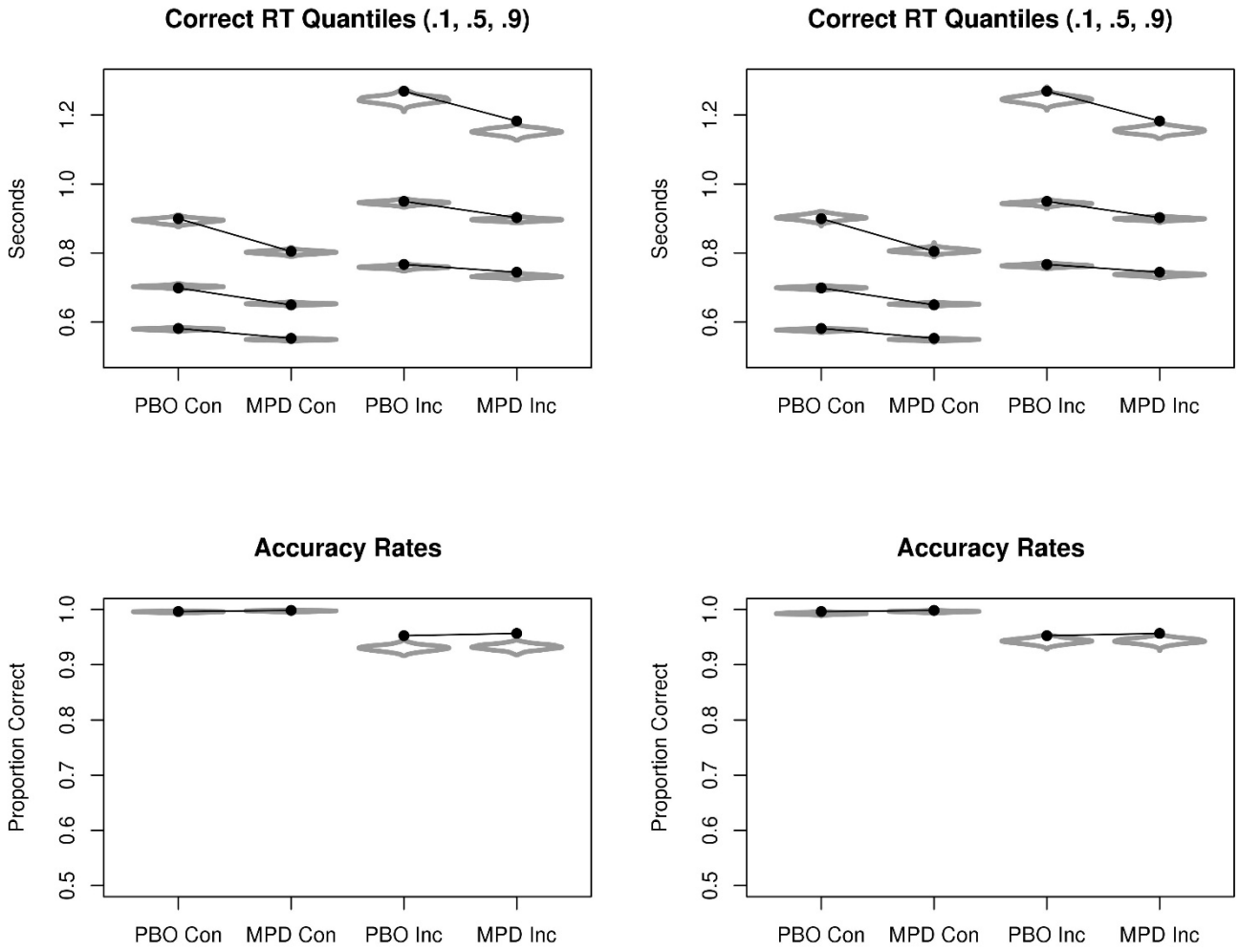


**Figure 3.** Group average delta functions and conditional accuracy functions (CAFs) for the empirical data and for values predicted by the MDR (left) and DRV (right) models. Delta functions represent the difference between congruent and incongruent correct RT quantiles (.1, .3, .5, .7 and .9) on the y-axis against the congruent/incongruent mean on the x-axis, for empirical data (black points and lines) and data predicted by 500 posterior samples from the model (small gray points = data predicted by each individual sample, gray lines = average of data predicted by all posterior samples). CAFs display accuracy rates for different RT quantile bins in the congruent (dotted lines) and incongruent (solid lines) conditions. Black lines and points represent accuracy rates for each bin in the empirical data while gray violin plots represent accuracy rates predicted by 500 posterior samples from the model. For all plots: Con = congruent, Inc = incongruent, MPD = methylphenidate condition, PBO = placebo condition.

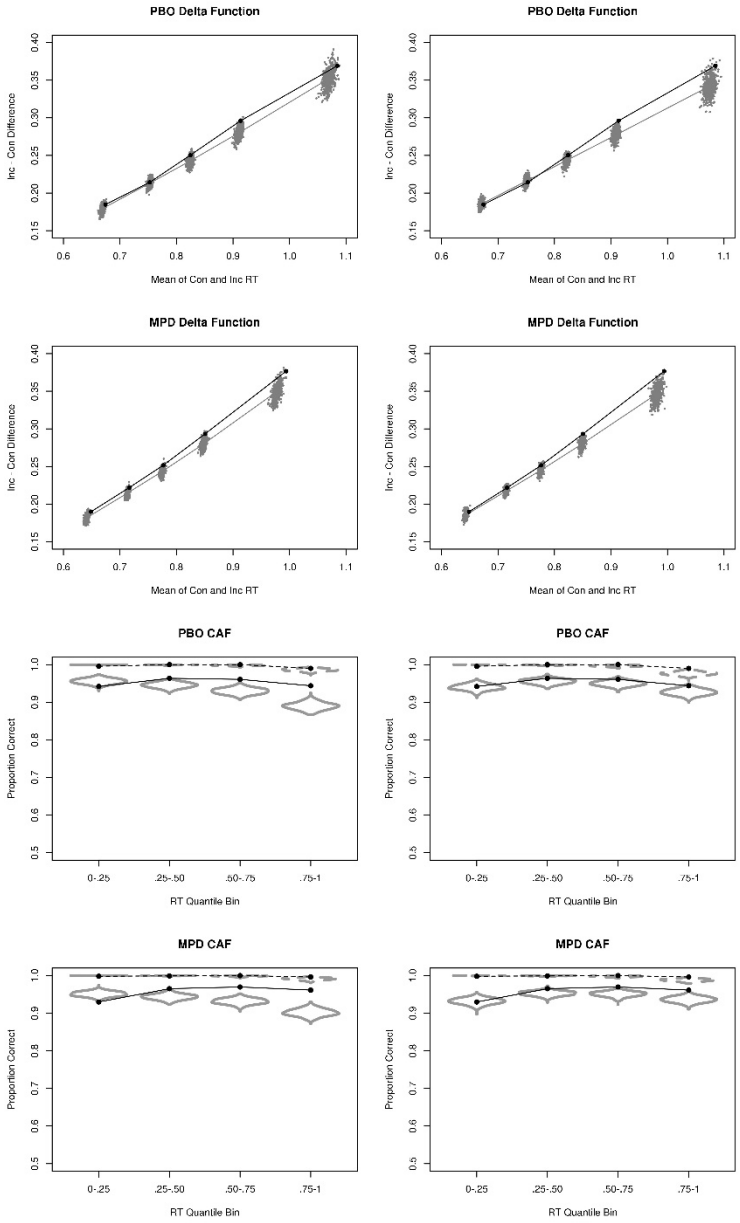


**Figure 4.** Medians (gray bars) and 95% credible intervals (black error bars) for average posterior distributions of MDR model parameters of interest in the placebo (PBO) and methylphenidate (MPD) conditions. Parameters including overall evidence quantity (average of *v.match* and *v.mismatch*), the congruency effect in evidence quantity, overall evidence quality (*v.match* minus *v.mismatch*), the congruency effect in evidence quality, overall perceptual sensitivity, the congruency effect in perceptual sensitivity, overall response threshold (*b*), priming effects in response threshold, and non-decision time (*t0*). CIs were obtained by averaging over samples from posterior distributions of individual-level parameter values.

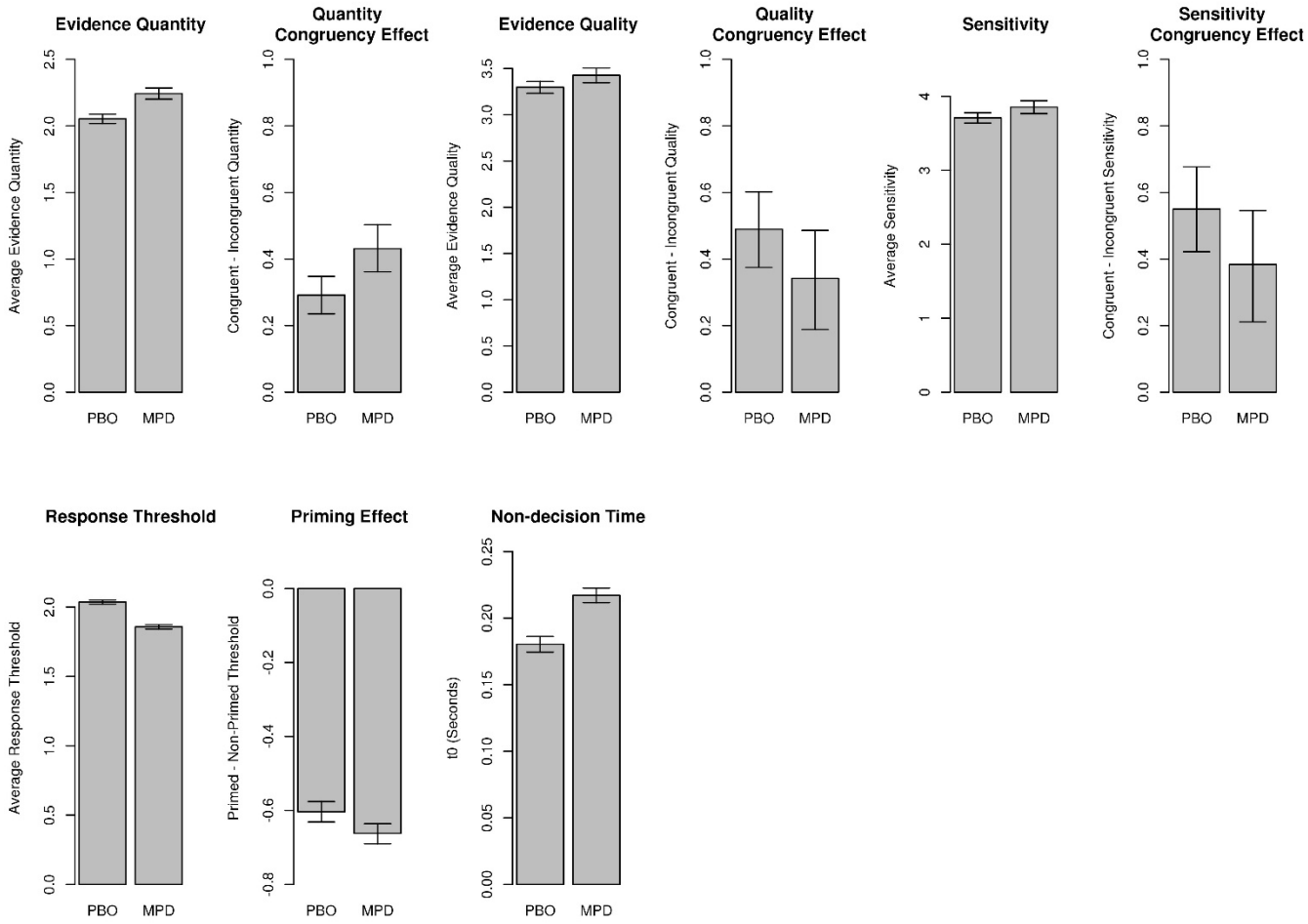
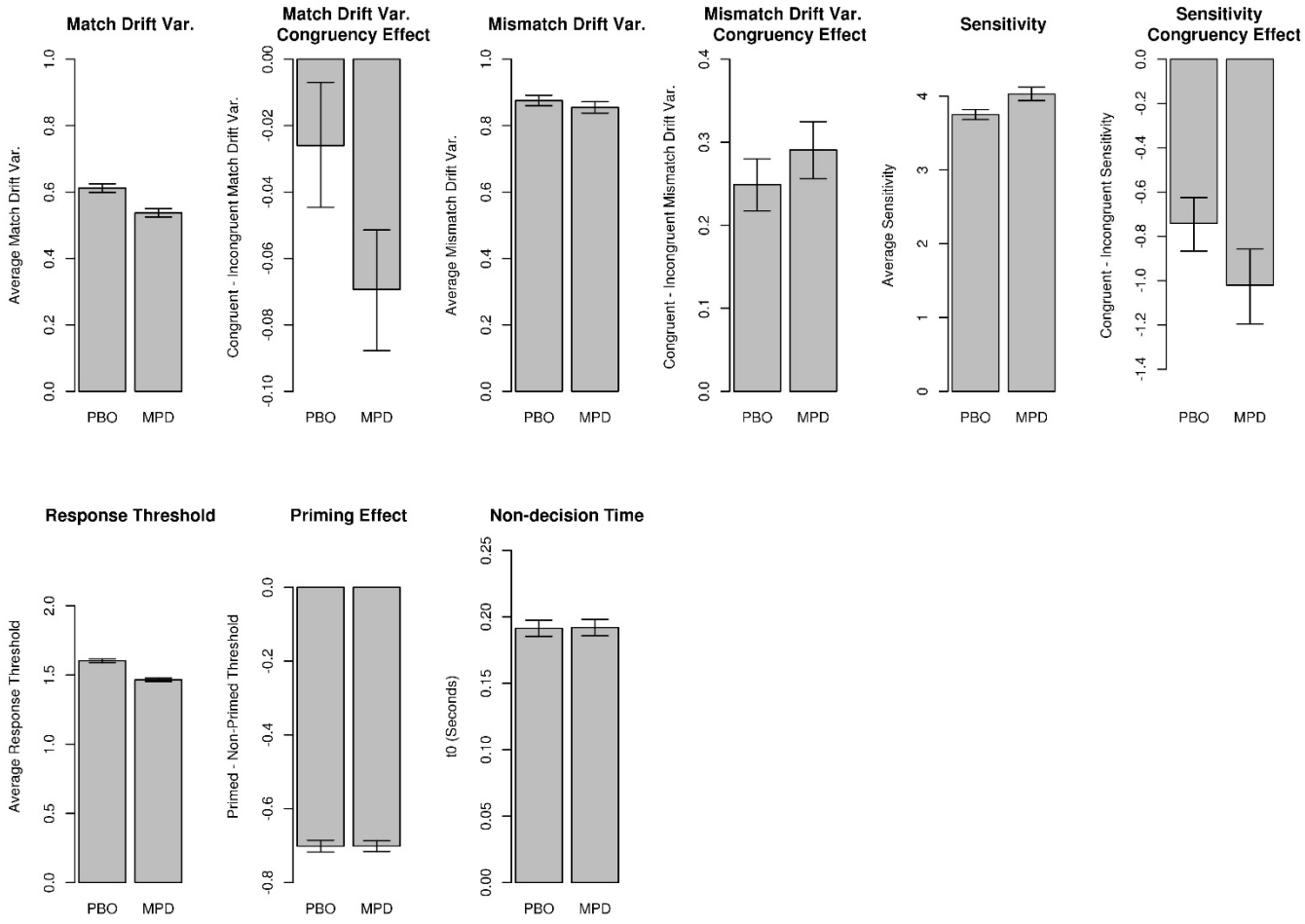


**Figure 5.** Medians (gray bars) and 95% credible intervals (black error bars) for average posterior distributions of DRV model parameters of interest in the placebo (PBO) and methylphenidate (MPD) conditions. Parameters including overall rate variability of the matching accumulator (*sv.match*), the congruency effect in *sv.match*, overall rate variability of the mismatching accumulator (*sv.mismatch*), the congruency effect in *sv.mismatch*, overall perceptual sensitivity, the congruency effect in perceptual sensitivity, overall response threshold (*b*), priming effects in response threshold, and non-decision time (*t0*). CIs were obtained by averaging over samples from posterior distributions of individual-level parameter values.



## Conflict of Interest Statement

On behalf of all authors, the corresponding author states that there is no conflict of interest.

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**Supplemental Materials for**:

# Modeling The Effects Of Methylphenidate On Interference And Evidence Accumulation Processes Using The Conflict Linear Ballistic Accumulator

Alexander Weigard, Andrew Heathcote and Chandra Sripada

**Model Selection and Specification Details**

As noted in the text, the Conflict LBA, like many cognitive and neuroscience models, displays highly-correlated parameters that can be difficult to estimate when individual-level data are sparse (Kolossa & Kopp, 2018; Gutenkunst et al., 2007), which often makes simplified models more useful for measurement (Heathcote, Hannah & Matzke, under review). Therefore, we sought to use LBA model variants in the current analysis that were simple enough for us to obtain interpretable parameter estimates, while also providing the best possible description of the MSIT data. To do so, we took the results of the previous model selection analysis conducted by Heathcote et al. (under review), as well as several practical considerations, into account before conducting a focused model selection analysis with the MSIT data set.

Results from the Heathcote et al. (under review) model selection, in which multiple archival data sets from the Stroop, Simon and Flanker paradigms were fit to different variants of the Conflict LBA, are displayed in Supplemental Table 1. Akaike information criterion (AIC: Akaike, 1973) suggested that Stroop and Simon paradigms were generally well-described by models that included both a priming process in the *b* parameter and differences in *sv* between congruent and incongruent trials. Flanker paradigms, in contrast, tended to be best described by models that included differences in both *v* and *sv* between congruent and incongruent trials, and did not include priming effects in *b*.

The MSIT is assumed to contain two distinct types of interference effect: a Simon-like effect, which primes the choice matching the serial position of the target in both congruent and incongruent conditions, and a Flanker-like effect, which produces interference in the incongruent condition only, as the flanking elements in the congruent condition (0s) do not correspond to a possible response. Although a comprehensive model selection analysis, such as the one conducted by Heathcote et al. (under review), would be an ideal procedure for determining which combination of Conflict LBA parameters bests

describes one, or both, of these potential interference effects, we were unable to conduct such an analysis due to several practical constraints.

First, as noted in the text, *sv* parameters tend to trade off with *v* parameters in evidence accumulation models, which often limits researchers' ability to index effects in *v* separately from those in *sv* (Boehm et al., 2018). Such trade-offs often occur when there is little information about the shape of the error RT distribution (i.e., few error RTs are present). Participants in the current study had very low error rates (<5% on average for incongruent trials, <1% for congruent trials), and pilot analyses we conducted with the MSIT task suggested that models which allowed both *v* and *sv* to vary by drug and congruency conditions displayed evidence of parameter trade-offs. Therefore, we assumed that, given the trial number and error rates in the MSIT data set, it would be difficult to determine whether experimental effects could be uniquely attributed to *v* or *sv*. Instead, we opted to fit one model which allowed only *v* to explain relevant experimental effects (mean drift rate model: MDR) and a second model which allowed only *sv* to explain these effects (drift rate variability model: DRV). Such a procedure allowed us to explore whether drift rate processes differed by congruency and drug conditions in one or both of these plausible models, and to determine whether effects in other parameter estimates were robust between the models. We also used a *sensitivity* index, which, as described in the text, takes both mean drift rates and drift rate variability into account, to test whether experimental effects in overall efficiency of evidence accumulation (i.e., signal-to-noise ratios) were similar between models.

Second, although it is plausible that both the Simon-like and Flanker-like interference effects caused priming in *b*, modeling priming from the Flanker-like effect as separate from the Simon-like effect would likely be difficult for several reasons. The Simon-like effect is present in both congruent and incongruent conditions, allowing *b* parameters for Simon-primed accumulators to be identified from correct responses in the congruent condition and incorrect responses in the incongruent condition and *b*

parameters for non-primed accumulators to be identified from incorrect responses in the congruent condition and correct responses in the incongruent condition. However, the Flanker-like effect is only present in the incongruent condition, meaning that *b* parameters for the Flanker-primed accumulators can only be identified from incorrect responses in incongruent condition, which are very rare. Furthermore, on half of the trials in the incongruent condition, the Simon-like and Flanker-like effect favored different responses (e.g., 131, 233), while on the other half of trials the response favored by the Simon-like effect was the same as that favored by the Flanker-like effect (e.g., 211, 332), making the two effects even more difficult to disentangle. Hence, we did not attempt to explain the Flanker-like effect with priming in *b* parameters. Although this constraint may have prevented the models in the current study from providing a comprehensive description of the data, the model selection analysis from Heathcote et al. (under review) indicated that priming effects in *b* were rarely needed to account for interference effects in Flanker tasks, suggesting that this constraint was reasonable.

Finally, because of the large number of parameters and subjects in the hierarchical MSIT models, estimating these models required a high degree of computational resources, including system memory and computation time. Because of this practical constraint, a systematic model selection analysis, in which all possible combinations of parameters would be allowed to vary to explain both the congruency and drug effects, would be challenging due to the resources needed to estimate the large number of models involved.

Working within these constraints, we conducted a focused model selection analysis with the goal of identifying best-fitting MDR and DRV models that could both be used for inference in the current study. Models considered in the MDR class allowed *v*, *b* and *t0* to vary by drug condition. Models considered in the DRV class models allowed *sv*, *b* and *t0* to vary by drug condition. All models in both classes allowed *v* and *sv* to vary by match/mismatch, *b* to vary by choice accumulator (one, two, three),

and had single estimates of start point variability (*A*). In each class, four different models were estimated and compared 1) a "null" model, which did not allow either drift rate parameters or priming effects in *b* to explain congruency effects in the MSIT, 2) a "*b* only" model which only allowed Simon-like priming effects in *b* to explain congruency effects, 3) a "rate only" model which only allowed rate parameters (*v* for the MDR class and *sv* for the DRV class) to explain congruency effects, and 4) a "rate + *b*" model which allowed both the rate parameter (*v* for the MDR class and *sv* for the DRV class) and Simon-like priming effects in *b* to explain congruency effects. Results of the model comparison analysis are reported in Supplemental Table 2. In both classes, the "rate + *b*" model was clearly indicated as the best fitting model by both the deviance information criterion (DIC: Spiegelhalter, Best, Carlin, & Van Der Linde, 2002) and the Watanabe-Akaike information criterion (WAIC: Watanabe, 2010). The difference in WAIC between this model to the next-best-fitting model, when compared to the standard error (SE) of the difference using the "paired estimate" method (Vehtari, Gelman, & Gabry, 2016), was large for both the MDR (WAIC difference = 235.5 , SE = 50.2) and DRV (WAIC difference = 376.6, SE = 62.9) class models. The results of this model selection analysis suggested that, regardless of whether drift rate effects are attributed to changes in *v* or *sv*, both drift rate parameter differences between congruent and incongruent trials and Simon-like priming effects in *b* are needed to explain congruency-related performance differences. Therefore, the "rate + *b*" models were selected for use in further analyses in the main text.

**Priors for Hierarchical LBA Model Parameters**

As strong priors for parameters of the LBA have not yet been established, we used relatively broad and mildly informative priors, following Turner et al. (2013). In the hierarchical model, priors for individual-level parameters are not explicitly set because group-level parameters act as priors for individual-level parameters. Priors for all group-level scale (σ) parameters were exponential

distributions with a scale of 1. Priors for group-level location (μ) parameters, which were the same regardless of the factors these parameters varied by (e.g., congruency and drug conditions), were truncated normal (*TN*) distributions with the following locations, scales, lower bounds, and upper bounds:

$$A_{\mu} \sim TN(\mu=1, \sigma=0.5, 0, \infty)$$
$$b_{\mu} \sim TN(\mu=1, \sigma=0.5, 0, \infty)$$
$$v_{\mu} \sim TN(\mu=2, \sigma=1, 0, \infty)$$
$$sv_{\mu} \sim TN(\mu=1, \sigma=1, 0, \infty)$$
$$t0_{\mu} \sim TN(\mu=1, \sigma=0.5, .1, \infty)$$

**Calculation and Interpretation of Credible Intervals for Effects**

Credible intervals (CIs) for inference were obtained from posterior difference distributions of individual-level parameter values. To test main effects, samples from all distributions in each of the two conditions were averaged within-condition and within-subject, and the resulting average posterior distributions for one condition were subtracted from those of the other condition. For example, to test for drug main effects on evidence quality, parameter estimates for evidence quality on congruent PBO and incongruent PBO trials were averaged within each individual and the resulting distribution was subtracted from each individual's distribution of evidence quality averaged across congruent MPD and incongruent MPD trials. To test interactions, difference distributions for one effect were created at each level of the other effect and one of these distributions was then subtracted from the other to create a single difference distribution for the interaction effect. For example, to test for congruency x drug interactions in evidence quality, parameter estimates from the incongruent MPD condition were subtracted from those in the congruent MPD condition, parameter estimates from the incongruent PBO condition were subtracted from those in the congruent PBO condition, and the latter distribution was then subtracted from the former. Following the calculation of posterior difference distributions at the

level of each individual, samples were averaged across all individuals to create an averaged posterior difference distribution, which was then used for inference. CIs were obtained by calculating the .025 and .975 quantiles of each averaged posterior difference distribution.

**Effects in Behavioral Summary Statistics**

Evidence for effects in behavioral summary statistics (median correct RT, accuracy rate) was assessed using Bayesian repeated-measures ANOVAs in the JASP statistics program (JASP Team, 2016; Wagenmakers et al., 2018)[2]. Results from Bayesian repeated-measures ANOVAs and descriptive statistics for RT and accuracy are displayed in Supplemental Table 3. For median correct RT, the model with the most substantial evidence included both congruency and drug main effects ($BF_{10}$ = 2.399e+45). This model was more than 1000 times more likely than models which contained the congruency effect only ($BF_{10}$ = 1.988e+42) and the drug effect only ($BF_{10}$ = 1.148) and was 3.5 times more likely than the model which contained both main effects as well as a congruency x drug interaction ($BF_{10}$ = 6.857e+44). Therefore, the most-likely model suggests that RTs were longer overall in the incongruent condition and shorter in the MPD condition, but that congruency effects did not differ by drug condition. For accuracy rates, the model with the most substantial evidence included a congruency main effect only ($BF_{10}$ = 8.719e+9). This model was 5.3 times more likely than the second most-likely model, which contained both congruency and drug main effects ($BF_{10}$ = 1.643e+9) and was 21.4 times more likely than one which contained both main effects and an interaction ($BF_{10}$ = 4.072e+8). A drug effect only model was not supported ($BF_{10}$ = 0.185). Therefore, the evidence suggests that accuracy was worse in the incongruent condition, but that there were no main effects or interactions involving methylphenidate.

[2] Bayesian repeated measures ANOVAs compare models assuming different combinations of factor effects using Bayes Factors (BFs), which are intuitively interpreted as an odds ratio, with values >1 providing evidence that observing the data makes the model more likely and <1 indicating the model becomes less likely. Standard JASP priors (r scale = .5 for fixed effects, r scale = 1 for random effects) were used for all analyses.

**Supplemental Table 1.** Results from the model selection analysis of Heathcote et al. (under review), in which versions of the Conflict LBA that allowed all possible combinations of parameters to explain behavioral interference effects were fit to archival conflict task data sets from Pratte, Rouder, Morey, and Feng (2010), Heathcote and Hannah (2013), and White, Ratcliff and Starns (2011). The "Experiment" column at left indicates the study, experiment number, and type of task used in the experiment. The remaining columns to the right list AIC values, summed over individual participants' fits and reported relative to the best model (which has a 0 entry), for models in which the parameters in the column label were allowed to explain interference effects. This table reproduces information from a similar table present in Heathcote et al. (under review) with permission from the authors of the original manuscript.

| Experiment | *b*+*v*+*sv* | *b*+*sv* | *b*+*v* | *v*+*sv* | *b* | *sv* | *v* |
|---|---|---|---|---|---|---|---|
| Pratte et al., (2010), Exp. 1 (Stroop) | 115 | 37 | 2 | 2 | 63 | 521 | 0 |
| Pratte et al., Exp. 3 (Stroop and Simon) | 130 | 0 | 31 | 83 | 17 | 295 | 61 |
| Pratte et al., Exp. 5 (Stroop and Simon) | 190 | 0 | 34 | 122 | 25 | 481 | 63 |
| Heathcote & Hannah (2013) (Simon) | 118 | 0 | 2 | 23 | 216 | 657 | 320 |
| White et al., (2011), Exp. 1 (Flanker) | 41 | 46 | 110 | 0 | 486 | 3612 | 1040 |
| White et al., Exp. 2 (Flanker) | 0 | 265 | 521 | 45 | 968 | 5868 | 1560 |
| White et al., Exp. 3 (Flanker) | 46 | 528 | 794 | 0 | 1837 | 5342 | 2098 |
| White et al., Exp. 4 (Flanker) | 189 | 163 | 761 | 0 | 2629 | 5764 | 1699 |
| White et al., Exp. 5 (Flanker) | 445 | 803 | 1437 | 0 | 1263 | 3168 | 230 |

**Supplemental Table 2.** Results from the focused model selection analysis of mean drift rate (MDR) and drift rate variability (DRV) class models, including values of the deviance information criterion (DIC: Spiegelhalter, Best, Carlin, & Van Der Linde, 2002) and the mean of the Watanabe-Akaike information criterion (WAIC: Watanabe, 2010).

| Model Class | Interference Effect Parameters | WAIC | DIC |
|---|---|---|---|
| MDR | rate + *b* | -16227.1 | -16244.7 |
| | rate only | -15686.1 | -15621.2 |
| | *b* only | -15991.6 | -16018.3 |
| | null | -4475.04 | -4358.02 |
| DRV | rate + *b* | -16475.9 | -16568.8 |
| | rate only | -10927.2 | -11186.4 |
| | *b* only | -16099.4 | -16142.5 |
| | null | -4529.21 | -4422.17 |

**Supplemental Table 3**. Model comparison results from Bayesian repeated-measures ANOVAs conducted with median correct RT (in seconds) and accuracy rate data in JASP. P(M) shows the uniform distribution of probabilities across all candidate models, P(M|data) shows posterior model probabilities, $BF_{10}$ shows the Bayes Factor for each model compared to the null, and $BF_M$ shows the change from prior model odds to posterior model odds (Wagenmakers et al., 2018). Odds in favor of one model relative to another model can be quantified by dividing the $BF_{10}$ of the first model by the $BF_{10}$ of the second.

**Median Correct RT Model Comparison**

| Models | P(M) | P(M\|data) | $BF_M$ | $BF_{10}$ |
|---|---|---|---|---|
| Null model (incl. subject) | 0.200 | 3.240e-46 | 1.296e-45 | 1.000 |
| congruency | 0.200 | 6.440e -4 | 0.003 | 1.988e+42 |
| drug | 0.200 | 3.721e-46 | 1.489e-45 | 1.148 |
| congruency + drug | 0.200 | 0.777 | 13.952 | 2.399e+45 |
| congruency + drug + congruency ✻ drug | 0.200 | 0.222 | 1.143 | 6.857e+44 |

**Median Correct RT Descriptive Statistics**

| Congruency | Drug | Mean | SD |
|---|---|---|---|
| Con | PBO | 0.699 | 0.115 |
| | MPD | 0.649 | 0.092 |
| Inc | PBO | 0.950 | 0.147 |
| | MPD | 0.901 | 0.129 |

**Accuracy Rate Model Comparison**

| Models | P(M) | P(M\|data) | $BF_M$ | $BF_{10}$ |
|---|---|---|---|---|
| Null model (incl. subject) | 0.200 | 9.285e-11 | 3.714e-10 | 1.000 |
| congruency | 0.200 | 0.810 | 17.009 | 8.719e+9 |
| drug | 0.200 | 1.715e-11 | 6.860e-11 | 0.185 |
| congruency + drug | 0.200 | 0.153 | 0.720 | 1.643e+9 |
| congruency + drug + congruency ✻ drug | 0.200 | 0.038 | 0.157 | 4.072e+8 |

**Accuracy Rate Descriptive Statistics**

| Congruency | Drug | Mean | SD |
|---|---|---|---|
| Con | PBO | 0.996 | 0.007 |
| | MPD | 0.998 | 0.005 |
| Inc | PBO | 0.952 | 0.058 |
| | MPD | 0.957 | 0.063 |

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