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Integrating impairments in reaction time and executive function using a diffusion model framework

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Abstract

Using Ratcliff's diffusion model and ex-Gaussian decomposition, we directly evaluate the role individual differences in reaction time (RT) distribution components play in the prediction of inhibitory control and working memory (WM) capacity in children with and without ADHD. Children with ($n=92$, $\bar{x}$ age= 10.2 years, 67% male) and without ADHD ($n=62$, $\bar{x}$ age=10.6 years, 46% male) completed four tasks of WM and a stop signal reaction time (SSRT) task. Children with ADHD had smaller WM capacities and less efficient inhibitory control. Diffusion model analyses revealed that children with ADHD had slower drift rates (v) and faster non-decision times (Ter), but there were no group differences in boundary separations (a). Similarly, using an ex-Gaussian approach, children with ADHD had larger τ values than non-ADHD controls, but did not differ in μ or σ distribution components. Drift rate mediated the association between ADHD status and performance on both inhibitory control and WM capacity. τ also mediated the ADHD-executive function impairment associations; however, models were a poorer fit to the data. Impaired performance on RT and executive functioning tasks has long been associated with childhood ADHD. Both are believed to be important cognitive mechanisms to the disorder. We demonstrate here that drift rate, or the speed at which information accumulates towards a decision, is able to explain both.

Keywords

ADHD; executive function; reaction time; ex-Gaussian; diffusion model

When speeded responses are required, children with ADHD often respond less accurately, are slower, and have more variable reaction times (RTs) compared to their same-aged peers (Castellanos et al., 2005; Douglas, 1999; Hervey et al., 2006; Kuntsi & Stevenson, 2001; Leth-Steensen, Elbaz, & Douglas, 2000). Like ADHD itself (Faraone et al., 2005), RT and RT variability are also highly heritable (Kuntsi et al., 2006; Kuntsi & Stevenson, 2001), leading some to suggest that these performance characteristics may be important cognitive mechanisms in the disorder that may elucidate underlying neural dysfunction (Castellanos & Tannock, 2002). In addition, slow RTs, as an index of information processing speed, have been hypothesized to explain ADHD-related deficits on a variety of executive tasks (Alderson, Rapport, & Kofler, 2007; Lijffijt, Kenemans, Verbaten, & van Engeland, 2005),

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which has implications for characterization of the ADHD cognitive phenotype and search for underlying mechanisms.

That being said, although slower RTs among children with ADHD is a common finding, it is certainly not ubiquitous, and there is significant between-study heterogeneity in the presence and size of group differences for mean RTs (MRT, Frazier, Demaree, & Youngstrom, 2004; Huang-Pollock, Karalunas, Tam, & Moore, 2012). Mechanistic interpretation of the presence or absence of group differences in RTs, either at the cognitive or neural levels, is also limited because RTs are influenced by multiple interacting processes. These include, but are not limited to, stimulus encoding, rate of information processing, motor preparation and output, speed-accuracy trade-off effects, and response bias (Ratcliff, 2002). All have been hypothesized to be involved in ADHD (Banaschewski et al., 2008; Halperin & Schulz, 2006; Hurks et al., 2005; Klimkeit, Mattingley, Sheppard, Lee, & Bradshaw, 2005; Lajoie et al., 2005; Sergeant, 2000; Sergeant & Scholten, 1985; Steger et al., 2001). Thus, modeling these processes and predicting how they ultimately produce a RT is critical not only to explaining the presence or absence of group differences in MRT, but also to understanding why speed of response for even simple judgments is so commonly associated with performance on higher level cognitive constructs (e.g. intellectual and executive functions [EF], Grudnik & Kranzler, 2001; Salthouse, McGuthry, & Hambrick, 1999; Schmiedek, Oberauer, Wilhelm, Süß, & Wittmann, 2007), and to developing a more comprehensive theory of why poor performance on tasks of EF and speeded processing are often observed in ADHD.

The importance of better understanding mechanisms contributing to group differences in RTs has recently lead to the use of alternative methods of characterizing RTs in ADHD, such as ex-Gaussian approaches (Buzy, Medoff, & Schweitzer, 2009; Epstein et al., 2011; Geurts et al., 2008; Leth-Steensen, et al., 2000). Ex-Gaussian analyses provide separate estimates of the mean (μ) and standard deviation (σ) of the Gaussian portion of the RT distribution, and the mean/standard deviation of the exponential portion of the distribution (τ). The most consistent significant ADHD-control group differences are reported in the exponential upper tail of the RT distribution (τ), suggesting that children with ADHD have a large number of excessively long RTs relative to non-ADHD controls (Buzy, et al., 2009; Epstein, et al., 2011; Leth-Steensen, et al., 2000). It may be that group differences in the upper-tail of the distribution account for group differences in mean or standard deviations of RTs, however, some studies continue to note group differences in both μ and σ in addition to groups differences in τ (Buzy, et al., 2009; Epstein, et al., 2011). Although the patterns of statistical significance are less consistent for μ and σ , a relatively small number of studies have applied ex-Gaussian analyses, leaving questions about which parameters best differentiate children with ADHD from non-ADHD controls.

Even if we accept that the largest group differences are seen in the exponential tail of the RT distribution, cognitive interpretations of ex-Gaussian parameters remain uncertain. τ has sometimes been interpreted as reflecting higher-order processing (e.g. attention lapses, Leth-Steensen, et al., 2000) while μ and σ relate more strongly to motor responding; however, the opposite interpretations have also been made (for review see Matzke & Wagenmakers, 2009). A significant limitation of the ex-Gaussian approach that may contribute to heterogeneity in findings and difficulty with interpretation of parameters is that they do not take into account response accuracy, which prevents direct assessment of speed-accuracy trade-off effects. Prioritizing speed over accuracy has often been noted as a characteristic of performance for children with ADHD (Mulder et al., 2010; Sergeant, Oosterlaan, & van der Meere, 1999), and the degree to which this occurs confounds the interpretation of RT data (Matzke & Wagenmakers, 2009).

Analyses based on a drift diffusion model of RTs (Ratcliff & Rouder, 1998) provide one way of statistically disentangling the multiple factors influencing RTs. Widely used in the cognitive psychology literature (Balota & Yap, 2011; Kühn et al., 2011; Ratcliff, Thapar, Gomez, & McKoon, 2004; Schmiedek, Lövdén, & Lindenberger, 2009; Spaniol & Bayen, 2005; Thapar, Ratcliff, & McKoon, 2003), diffusion models have been developed to explain decision-making in forced-choice paradigms for which relatively rapid (~1 second) response decisions are required (Ratcliff & Rouder, 1998). These models assume that information about a stimulus is accumulated via a noisy information accumulation process until a decision criterion is met, at which point a response is initiated. That is, when faced with a forced-choice decision, participants are assumed to set criteria for how much information they require to commit to either of the response options. Individuals who require less information will respond more quickly but will also make more errors than those who require more information. The process is described as “noisy” because random neural activity unrelated to the decision process impacts the speed and efficiency with which a person is able to accumulate information relevant to the decision itself. Finally, processes that are not directly related to the response decision, such as stimulus encoding and motor preparation, will also impact the final RT.

Practically, the diffusion model provides estimates of: 1) *drift rate* (v ; an index of how quickly and efficiently an individual can accumulate information to inform their response decision, which is theoretically linked to neural signal:noise ratio); 2) *boundary separation* (a ; how “sure” a person needs to be before committing to a response, or their speed-accuracy tradeoff setting); and 3) *non-decision time* (T_{er} ; the time it takes to complete all other information processes not involved in stimulus discrimination, e.g., encoding, memory access, motor preparation; Ratcliff & McKoon, 2008; see Figure 1 and Table 1).

Although not the only model for understanding RTs (for review see Bogacz, Brown, Moehlis, Holmes, & Cohen, 2006), diffusion models are well-validated and show strong convergent and divergent validity, such that manipulations of stimulus clarity uniquely impact drift rate and manipulations of the complexity of motor response required uniquely impact non-decision time (Voss, Rothermund, & Voss, 2004). Further, recent studies in both humans and non-human primates have demonstrated correspondence between diffusion model parameters and real-time functioning of neural networks (Beck et al., 2008; Philiastides, Ratcliff, & Sajda, 2006; Philiastides & Sajda, 2006; Ratcliff, Cherian, & Segraves, 2003; Ratcliff, Philiastides, & Sajda, 2009). Thus, application of diffusion models has particular promise for understanding the processes underlying RTs and for elucidating connections between underlying neurological dysfunction and performance on cognitive tasks.

In addition to group differences in RT distributions, there is robust evidence that children with ADHD perform more poorly on tasks of response inhibition and working memory (WM) relative to non-ADHD controls (Lijffijt, et al., 2005; Martinussen, Hayden, Hogg-Johnson, & Tannock, 2005; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005). However, at a mechanistic level how deficits in inhibition and WM may be substantively related to RT variables remains unclear. In the case of response inhibition, children with ADHD have slower stop signal reaction times (SSRTs) than non-ADHD controls but also tend to have slower MRTs (Lijffijt, et al., 2005). This lack of specificity has led to suggestions that group differences in SSRT are a result of a general cognitive deficit, such as slow or inefficient information processing, rather than a specific deficit in inhibition (Alderson, Rapport, Sarver, & Kofler, 2008; Lijffijt, et al., 2005). Such a finding suggests that of the RT components, drift rate (v) would be uniquely associated with SSRT. However, problems with motor preparation and output (Klimkeit, et al., 2005; Steger, et al., 2001) or strategic adjustments to response criterion that maximize both go-speed and stopping accuracy

(Verbruggen & Logan, 2009) could also result in similar effect sizes for group differences in MRT and SSRT.

Regarding WM, some of the strongest empirical evidence suggests that a large proportion of the developmental improvement in WM span can be attributed to increased processing speed (Fry & Hale, 2000; Kail, 2007), suggesting that drift rate may uniquely mediate WM capacity. Consistent with this possibility, Schmiedek et al. (2007) found that in young adults both speed-accuracy trade-offs (boundary separation) and information processing speed/efficiency (drift rate) were related to WM capacity, but that the strength of the relationship for drift rate was nearly twice that of boundary separation. RT decomposition approaches have not been applied to this question within the context of ADHD, however using a latent variable approach, Huang-Pollock, Karalunas, Tam, and Moore (2011) recently found that processing speed accounted for a significant proportion of variance in WM span among children with and without ADHD; however, which RT component(s) are most critically responsible for that relationship is not known.

The current study first examines group differences in the ex-Gaussian parameters that descriptively characterize the RT distribution of children with ADHD. We then apply diffusion model analyses to compare groups on parameters with well-validated cognitive interpretations. Finally, we determine which RT distributional components may help explain the relationship between ADHD status and poor EF.

Methods

Participants

Children between the ages of 8–12 with ADHD ($\bar{x}$ age=10.2 years, 67% male) and without ADHD ($\bar{x}$ age=10.6 years, 46% male) were community recruited from local schools, newspaper/radio ads, and distributed flyers in the Central, York, and Dauphin county areas of Pennsylvania as part of a study approved by the Pennsylvania State University Institutional Review Board (#32136). ~80% of both the ADHD and non-ADHD samples identified as Caucasian and non-Hispanic (82.8% and 79.1%, respectively). Both ADHD and non-ADHD groups reported a median annual household income of \$61,000–\$71,000. A full description of the final sample can be found in Table 2.

In an initial screening phase, children were screened as *potentially* in the ADHD group if both parent and teacher report exceed the 85th percentile (T-score >61) on either the (a) Behavioral Assessment Scale for Children (BASC-2: Reynolds & Kamphaus, 2004) Attention or Hyperactivity problem subscales or (b) Conners' Rating Scales—Revised (Conners: Conners, 2001) Inattention, Hyperactivity, or ADHD subscales. To be included in the final ADHD group, children were required to meet criteria for DSM-IV ADHD by parent report on the National Institute of Mental Health's Diagnostic Interview Schedule of Childhood for DSM-IV (DISC-4) including age of onset, impairment, and cross-situational severity. Using the “or” algorithm (Lahey et al., 1994) to integrate parent (DISC-IV) and teacher (ADHD-RS) report of specific symptoms, 40 children were identified as combined subtype, 48 with the primarily inattentive subtype, and 3 with the primarily hyperactive subtype. Thirty one children were prescribed a psychostimulant medication, and they were required to be medication free for at least 24 hours prior to the day of testing (mode > 48 hrs). Children taking non-stimulant medications were excluded from the study.

Non-ADHD controls did not meet criteria for ADHD on the DISC-IV. In addition, they were required to be below the 79th percentile (T-score ≤ 58) on all of the above listed rating scales and indices and must never have been diagnosed or treated for ADHD. Common childhood disorders (i.e., anxiety, depression, oppositional defiant disorder, and conduct

disorder) were identified using the DISC-4 (see Table 2), but the presence of these disorders was not exclusionary for either ADHD or controls. Children with estimated IQ <80 were excluded.

Procedures

Prior to participation, informed written consent from parents and verbal assent from children were obtained. Parents were given monetary compensation and informal feedback regarding relevant clinical findings. Children were given a small prize worth <\$2. All children completed the cognitive screening measures and the experimental paradigms in the same order as part of a larger test battery that took place in two, three-hour long testing sessions.

Cognitive Screening Measures—A 2-subtest short form (Vocabulary, Matrix Reasoning) of the Wechsler Intelligence Scale for Children—IV (WISC-IV; Wechsler, 2003) provided an estimated IQ for 94 (61%) participants. Another 59 (39%) participants completed a 4 subtest short form (Vocabulary, Matrix Reasoning, Arithmetic, Symbol Search). The correlation of the 2 and 4 subtest short forms with the full 12-subtest battery are 0.87 and 0.93, respectively (Sattler, 2008).

Working Memory—The Letter Span and Spatial Span tasks were based in part on the Eprime programs provided by Engle and colleagues (Conway et al., 2005). In the *Letter Span Task*, children sequentially viewed a letter of the alphabet at a pace of 1 per second. The number of to-be-remembered items increased from two to nine, and after all items were presented, children were asked to recall them in the order they were presented. Three items were presented per set size, and the task was discontinued if children failed all items of a set size. In the *Spatial Span Task*, children viewed a 4×4 grid in which one square randomly turned red. As with the Letter Span task, the number of to-be-remembered items increased from two to nine, and after all items were presented, children were asked to recall them in the order they were presented. Three items were presented per set size, and the task was discontinued if children failed all items of a set size.

We also administered Digits Backwards from the WISC-IV (WISC-IV: Wechsler, 2003), and a novel Finger Windows Backwards test that was adapted from the Finger Windows Forwards subtest of the WRAML-2 (Sheslow & Adams, 2003). In the *Digits Backwards* task, children listened to a research assistant read aloud a list of numbers at the pace of one per second. They were then asked to repeat the digits out loud in the correct backwards sequence. Two items were presented per set size and the task was discontinued if children failed both items of a set size. In the *Finger Windows Backwards* condition, children watched a research assistant place the tip of a pen through round holes or “windows” in an opaque plastic board one at a time, at the pace of one per second. Children were then asked to place their finger in the holes in the correct backwards sequence. The task was discontinued following three consecutive errors.

All WM tasks were scored using the “all or nothing” load scoring approach (Conway, et al., 2005), in which children received credit for the total number of to-be-recalled targets they recalled in the correct sequential order. Scores are reported as a proportion of the total number correct out of total possible correct. To compute reliability, three separate total scores were calculated for each child using the first, second, and third trials (or first and second trials if a third trial was not provided, as in Digit Span) at each span length. Chronbach’s α among those span scores was then calculated to determine whether performance was consistent across trials. α ’s were 0.85 for Letter Span, 0.77 for Spatial Span, 0.67 for Digits Backwards, and 0.77 for Finger Windows Backwards.

The amount of missing data was slight (2% for digits backwards, 3.3% for letter span, 3.9% for spatial span, and 5.2% for finger windows backwards) and missing completely at random, $\chi^2(38, N = 153) = 33.26, p = 0.69$; they were therefore replaced using single EM imputation (Widaman, 2006).

Stop Signal Reaction Time (SSRT) Task—Children were administered a 200-trial tracking version of the Logan Stopping Task. Children completed 20 practice trials to ensure understanding of the directions. The task was then administered in 5 blocks of 40 trials with an optional rest break between blocks. For each trial, a central fixation point appeared for 200 ms. On 75% of trials (i.e. “go” trials), an “X” or an “O” was presented in the center of the screen for 1000 ms, and children made a forced-choice response whether an “X” or an “O” had appeared. Children were given 2300 ms to respond, after which the next trial automatically commenced. On 25% of trials (i.e. 50 “stop” trials), an auditory tone was presented to indicate that they should not respond. An initial MRT was determined based on the practice trials and the auditory stop tone was initially set to occur 250 ms before the MRT. The MRT was then dynamically recalculated after each correct go trial and the delay at which the stop tone was presented was adjusted dynamically in 50 ms increments to maintain an overall ~50% accuracy rate. (In our sample, non-ADHD controls were able to maintain a slightly higher rate of successful inhibition than children with ADHD [57% vs. 52%, $p < 0.001$]). SSRT, the amount of time a child needs in order to successfully inhibit a response 50% of the time, was calculated by subtracting the mean delay from the child’s MRT. Reliability (α) across the five blocks of trials was 0.82 (number of hits), 0.95 (MRT), and 0.88 (SSRT).

Only children who were able to maintain > 70% accuracy rate on the “go” trials were included in analyses. This resulted in 9 children with ADHD being excluded. An additional 2 non-ADHD controls and 3 children with ADHD were excluded because they did not make any errors and so diffusion model error distributions could not be fitted to the data. Included children with ADHD did not differ from excluded children with ADHD in age, estimated IQ, number of attention symptoms, or number of hyperactive symptoms (all $p < .287$, all $\eta^2 < .01$). All results (including the sample description in Table 2) are presented with these 14 children excluded.

Although primarily used as a measure of response inhibition in the ADHD literature, the stop task has been conceptualized as consisting of two partially independent tasks—the “go” task, in which the child needs to decide whether an “X” or “O” was presented and respond appropriately, and the “stop” task, in which the child needs to inhibit the “go” process on the minority of trials. It is unclear how group differences in inhibitory control might affect the RT distribution (Schachar et al., 2004; Verbruggen, Logan, Liefvooghe, & Vandierendonck, 2008). In our sample, RTs to the go trials immediately preceded by a stop signal trial (referred to as stop + 1 trials) were slower than RTs to go trials not immediately preceded by a stop trial ($p = .001, \eta^2 = .07$), but stop+1 trial slowing did not differ between groups, $p = .198$). Although there was no group difference in the effect, to try to minimize the effect of the stop task context on estimation of diffusion and ex-Gaussian RT indices, stop+1 trials were removed from the RT series. Anticipation RTs < 150 ms, which accounted for less than 0.25% of the data, were also removed.

Ex-Gaussian parameters μ , σ , and τ were computed using the egfit tool in MATLAB (Lacouture & Cousineau, 2008). This function performs an iterative search process using a simplex method to compare the observed RT distribution to an ex-Gaussian probability density function (PDF) with known values of μ , σ , and τ . In each iteration, the parameter values of the PDF are adjusted until maximum fit to the observed data is achieved. Because ex-Gaussian models do not take into account response accuracy, these parameter estimates

were obtained from the distribution of correct RTs only (Epstein, et al., 2011; Hervey, et al., 2006; Leth-Steensen, et al., 2000). This resulted in a *minimum* of 72 trials available for analysis for each participant, consistent with the number of trials used in previous studies of ADHD (Buzy, et al., 2009; Geurts, et al., 2008; Leth-Steensen, et al., 2000).

Diffusion model parameters were estimated using the Fast-dm modeling technique (Voss & Voss, 2007). The Fast-dm program also uses an iterative distribution fitting approach with downhill-simplex method to compare the observed RT distribution to the distribution predicted to occur with specific values of v , a , and Ter . Thus, similar to the ex-Gaussian estimation process, parameters are derived via a multi-dimensional space search in which distributions with known diffusion parameters are compared to observed data until the maximum fit between the predicted and observed distributions is achieved. Full details of the estimation procedure are not described here due to space constraints but can be found in Voss et al., 2004 and Voss & Voss, 2007. Because both speed and accuracy are accounted for in the diffusion model, both the correct and error RTs were used, reflecting the upper and lower boundaries of the model, respectively. FastDM can reasonably recover parameter estimates with as few as 20 trials (Voss & Voss, 2007). Participants for whom the diffusion model was not able to adequately fit the data would have been excluded from analyses; however, no participants needed to be excluded for this reason. Groups did not differ in model fit with both groups' p values approaching 1 ($p = .96$ and $.98$ for the non-ADHD and ADHD groups, respectively). Smaller absolute values of v indicate slower drift rates. Smaller values of a indicate greater speed-accuracy trade off, and Ter is reported in seconds.

Results

Sample Characteristics

In general, no subtype differences were observed (the single exception is noted below) and so ADHD subtypes were collapsed for comparison with controls. Group differences on parent and teacher standardized ratings were consistent with the diagnostic groupings (all $\eta^2 > .42$, all $p < 0.001$; see Table 2). Children with ADHD did not differ from non-ADHD controls in their IQ or age (all $\eta^2 < .02$, all $p > .092$), but the ADHD group had a greater proportion of boys ($\chi^2[1] = 6.25$, $p = .012$). Results for the group differences reported below were all unchanged when gender, estimated IQ, and age were included in the model as covariates, and so results reported are without those covariates in the model.

Group Differences on Traditional Task Performance Measures

On the go-trials of the stop task, children with ADHD were less accurate and made more omissions than their non-ADHD peers (both $p < 0.001$). Groups did not differ in their MRTs for correct go trials ($F[1,151] = 0.11$, $p = .738$, $\eta^2 < .01$), but children with ADHD did have larger standard deviation of RTs than non-ADHD controls ($F[1,151] = 14.54$, $p = .001$, $\eta^2 = .09$). Children with ADHD also had slower SSRTs than non-ADHD controls ($F[1,151] = 40.40$, $p < .001$, $\eta^2 = .21$). There was a subtype difference for SSRT such that children with ADHD-C had significantly worse SSRTs than children with ADHD-I ($p = .01$). Finally, children with ADHD had worse performance on all of the WM tasks than controls (all $p < 0.05$; see Table 2 for full description of task performance).

Ex-Gaussian Analyses and Diffusion Model Analyses

When groups were compared on ex-Gaussian parameters, children with ADHD did not differ from non-ADHD controls in either μ ($F[1,151] = 1.62$, $p = .205$, $\eta^2 = .01$) or σ ($F[1,151] = 1.85$, $p = .175$, $\eta^2 = .01$). Children with ADHD did have larger values for τ than non-ADHD controls ($F[1,151] = 9.24$, $p = .003$, $\eta^2 = .06$), indicating that increased SDRT was a result of a larger number of RTs in the exponential upper tail of the distribution.

To clarify the specific cognitive processes that underlie group differences in RT distributions, diffusion model analyses were also applied. Children with ADHD had slower drift rates than non-ADHD controls ($F[1,151]=23.86$, $p<.001$, $\eta^2=.14$) and faster non-decision times ($F[1,151]=5.17$, $p=.024$, $\eta^2=.03$). However, groups did not differ in their speed-accuracy tradeoffs ($F[1,151]=2.70$, $p=.102$, $\eta^2=.02$; see Table 2 for group means and standard deviations). Correlations between the stop task, WM, ex-Gaussian, and diffusion model parameters can be found in Table 3.

Mediation Analyses

To determine which diffusion model RT components were associated with and might account for ADHD-related deficits in inhibitory control, we conducted a multiple mediator regression analysis (Preacher & Hayes, 2008). Model paths reflecting group differences (which are described above) are not discussed again, but can be found in Figures 2 and 3. Slower drift rates were associated with worse inhibitory control ($p = 0.005$); this was not the case with either a or Ter (both $p > 0.46$). The Sobel test of the indirect effect of ADHD status on inhibitory control through drift rate was significant (total unstandardized indirect effect = 35.61, SE = 10.86, $p = 0.001$), indicating that drift rate mediated the relationship between ADHD status and inhibitory control, accounting for 24% of the variance in the path from ADHD status to SSRT. The indirect effects for a (total unstandardized indirect effect = 1.30, SE = 2.57, $p = 0.61$) and Ter (total unstandardized indirect effect = 3.02, SE = 4.20, $p = 0.47$) were not significant. A significant direct path from ADHD status to SSRT was also observed ($p < 0.001$; for full model see Figure 2).

To determine which diffusion model RT components were associated with and might account for WM deficits in ADHD, we conducted an SEM analysis with the three primary diffusion model variables as mediators. The error residuals for those parameters were allowed to correlate (Preacher & Hayes, 2008). The four WM tasks loaded onto a single domain general WM construct. When this latent WM factor was set as the dependent variable, the fit of the model was very good, $\chi^2[14] = 17.69$, $p = 0.22$, CFI = 0.98, RMSEA = 0.04. Only the parameter estimate for v was significantly associated with WM capacity ($p=0.02$), in which faster drift rates predicted greater WM capacity. The Sobel test of the indirect effect of ADHD status on WM capacity through drift rate was significant (total unstandardized indirect effect = -0.01 , SE = 0.01, $p = 0.02$) indicating that drift rate mediated the relationship between ADHD status and WM capacity, accounting for 21% of the variance in the path from ADHD status to WM capacity. The indirect effects for a (total unstandardized indirect effect = 0.01, $p = 0.36$) and Ter (total unstandardized indirect effect < 0.001 , $p = 0.48$) were not significant and neither could be considered mediators. As with findings for SSRT, a significant direct path from ADHD to WM was also observed ($p<0.001$; for full model see Figure 3), and the standardized direct effect of ADHD status on WM prior to the addition of the mediators was also significant ($\beta = -0.43$, $p<0.001$).

In mediation analyses using the ex-Gaussian indicators, the same general pattern of results was found. That is, the Sobel test of the indirect effect of ADHD status on inhibitory control through τ was significant (total unstandardized indirect effect = 22.88, SE = 9.15, $p = 0.006$), indicating that τ mediated the relationship between ADHD status and inhibitory control, accounting for 19% of the variance in the path from ADHD status to SSRT. The WM model with ex-Gaussian terms as mediators was a poor fit to the data ($\chi^2[14] = 25.89$, $p = 0.03$, CFI = 0.96, RMSEA = 0.08) but τ predicted WM capacity in the expected direction (standardized regression = -0.26 , $p = 0.007$). Neither μ nor σ were mediators for either SSRT or WM variables.

Discussion

There is robust evidence that relative to non-ADHD controls, children with ADHD perform more poorly on tasks of inhibitory control and WM, and that when speeded responses are required, their performance is often characterized by slower and more variable RTs. We demonstrate that these phenomena can be integrated and understood within a diffusion model approach that isolates RTs into separable components. Specifically, children with ADHD have slower drift rates than non-ADHD controls, reflecting slower and less efficient information processing. In turn, slower drift rates partially mediate the relationship between ADHD and poor EF performance (as shown in Figures 2 and 3). Although data is not longitudinal and causality cannot be firmly established, results lend support to recent theories that implicate a general cognitive deficit in processing efficiency that contributes to poor performance on a variety of EF tasks among children with ADHD (Alderson, et al., 2007; Lijffijt, et al., 2005). However, this general deficit partially mediates performance, leaving room for the possibility of specific deficits in higher-order cognitive functions or other factors to explain remaining variance.

These results also help explain large between-study heterogeneity for group differences in RTs. Though slow RTs are a prominent feature in the cognitive landscape of ADHD, like EF deficits, which are found in only 30–50% of children with ADHD (Nigg, Willcutt, Doyle, & Sonuga-Barke, 2005), slow RTs are by no means ubiquitous (Frazier, et al., 2004; Huang-Pollock, et al., 2012; Lijffijt et al., 2004). Using the traditional analysis approach, we found no group differences in MRT, which could have been interpreted as evidence that in processing speed for children with ADHD was equivalent to non-ADHD controls. Using the diffusion model approach, however, it became clear that information processing speed/efficiency, as indexed by drift rate, is in fact slower for children with ADHD than controls.

Slower and less efficient information processing in the ADHD sample was offset by faster non-decision times (means and effect sizes for each are summarized in Table 2 under *Diffusion Modeling Components*), which indicates that children with ADHD spent less time on tasks such as stimulus encoding, motor preparation, or both. Thus, group differences in cognitive processing speed are obscured using traditional MRT analyses because children with ADHD differ from non-ADHD controls on two processes with opposing effects on the final RT. Although the effect for drift rate was stronger than that for non-decision time, unless a method of decomposing RTs is utilized, true group differences in processing speed/efficiency may not always be observed.

Similarly, when ex-Gaussian decompositions of the RT distribution were conducted, children with ADHD differed from non-ADHD controls in τ , the exponential tail of the distribution, but not in μ or σ , which characterize the normal portion of the distribution. This is consistent with previous studies demonstrating that group differences in mean and standard deviation of RT are attributable to children with ADHD having a large number of very long RTs (Buzy, et al., 2009; Hervey, et al., 2006; Leth-Steensen, et al., 2000). Group difference in τ are consistent with the those found in drift rate because small changes in drift rates have more effect on RTs in the upper tail of the distribution than on those in the leading edge (for more detail see Wagenmakers, Grasman, & Molenaar, 2005). Also consistent with the fact that slow drift rates and τ have similar effects on the RT distribution, τ mediated ADHD-related deficits in SSRT and WM similarly to drift. However, the effect size for group difference in τ (as reported in Table 2) was only half of that for drift rate and the meditational model relating τ to WM was a poor fit to the data. Taken together, results indicate that in the current sample τ was a less sensitive indicator of cognitive dysfunction than drift rate. One reason for the smaller effects and weaker mediation may be that, similar to MRT, τ is impacted by multiple cognitive processes only some of which differ between

groups (Matzke & Wagenmakers, 2009); however, additional studies comparing models will be critical for the field in determining how best to interpret parameters and capture the cognitive processes that are impaired in ADHD.

The finding that children with ADHD have slow drift rates is consistent with results from two other independent samples (Karlunas, Huang-Pollock, & Nigg, 2012), as well as with evidence from a meta-analysis of continuous performance tasks that found slower drift rates for children with ADHD than non-ADHD controls (Huang-Pollock, et al., 2012). Findings are also consistent with recent evidence that children with ADHD are less able to suppress the neurologic activation of the resting state (i.e. default-mode network activation) during active or effortful cognition (Fair et al., 2010; Fassbender et al., 2009). Single-cell recordings in non-human primates (Beck, et al., 2008; Ratcliff, et al., 2003) and imaging studies in humans (Heekeren, Marrett, & Ungerleider, 2008; Ratcliff, et al., 2009) both suggest that drift rate reflects the signal:noise ratio in neural circuits responsible for the decision process. When the signal:noise ratio is higher, drift rates are faster. As the signal:noise ratio decreases, drift rates become slower. Thus, difficulty suppressing resting state activation would also be expected to result in lower signal:noise ratios and slower drift rates.

Resting-state activation in ADHD has most often been described as contributing to “attention lapses,” in which the child is temporarily distracted or disengaged from the task (Sonuga-Barke & Castellanos, 2007; Weissman, Roberts, Visscher, & Woldorff, 2006). In contrast, we describe how resting-state activation may directly impact the efficiency of decision processes via decreasing signal:noise ratios (Broyd et al., 2009; Deco, Jirsa, McIntosh, Sporns, & Kotter, 2009; MacDonald, Li, & Bäckman, 2009). Theories of resting-state activation hypothesize that (a) stable differences in functional connectivity and white matter integrity (Castellanos, Kelly, & Milham, 2009), and/or (b) motivational factors that impact the shift between resting and active states (Sonuga-Barke & Castellanos, 2007), may both influence signal:noise ratios in information processing. The exact relationship between measures of attention, motivation, drift rate, and neural activation in resting-state networks is not clear. However, as drift rates appear to be a highly sensitive indicator of cognitive dysfunction in ADHD, future research directly addressing these relationships will be important.

As noted above, children with ADHD demonstrated *faster* non-decision times than non-ADHD controls (see group means reported in Table 2). On one hand, this could be interpreted as children with ADHD having better encoding or motor preparation than non-ADHD controls; however, this interpretation would contradict research demonstrating impaired motor function in children ADHD (Rommelse et al., 2008; Suskauer et al., 2008), and so it is important to remember that faster is not always synonymous with better. Instead, it may be that children with ADHD are inefficient in their approach and do not spend enough time on these non-decisional processes, which may contribute to poorer overall performance. Further, although the diffusion model provides parameters with more specific cognitive interpretations than other RT models, non-decision time continues to reflect the impact of multiple cognitive processes. It is not currently possible to say which of these contributes to the group differences in this sample. Additional studies directly manipulating the complexity of motor response or correlating non-decision times to physiological indices of encoding and motor preparation will be necessary to fully understand group differences in non-decision time.

Though statistically significant, the effect size for non-decision time was small. Two previous studies, one also using a stop task, found no group differences in non-decision time (Huang-Pollock, et al., 2012; Karlunas, et al., 2012). Given the well-described

heterogeneity in ADHD, it is possible that the effect in the current sample is driven by a small but meaningful subgroup of children with large deficits in one of the processes contributing to non-decision time. This would increase between-study heterogeneity in effects detected. Future studies that better characterize subgroups using methods such as latent class or graph theoretical approaches (Fair, Bathula, Nikolas, & Nigg, 2012; Newman, 2006) will be critical for understanding within-group heterogeneity. Similarly, our study does not address whether slow drift rates are specific to ADHD or also characterize other psychiatric disorders. Given that neither RT variability generally, τ specifically (Geurts, et al., 2008), nor difficulty suppressing resting state activation (Buckholz & Meyer-Lindenberg, 2012) are specific to ADHD, it is unlikely that drift rate will prove to be so. This may allow drift rate to be used as a marker of trans-diagnostic risk factors, which are of particular interest in efforts to improve definitions of psychiatric disorders (Insel et al., 2010), but future research is needed to fully confirm drift rate's sensitivity and specificity to ADHD.

In the current study, children were asked to place equal emphases on the speed and accuracy of the go process, and in this context children with ADHD did not trade speed for accuracy to a greater degree than controls (i.e. they did not differ in boundary separation), a finding which was replicated in two additional independent samples (Karalunas, et al., 2012). Results suggest that choice impulsivity (e.g. prioritizing speed over accuracy due to poor task engagement or heightened desire to avoid challenging tasks, Winstanley, Eagle, & Robbins, 2006) is not a primary determinant of task performance in ADHD. However, previous work found that when specifically asked to emphasize accuracy over speed, children with ADHD did trade speed for accuracy to a greater degree than non-ADHD controls (Mulder, et al., 2010). Together, these findings suggest that children with ADHD may have difficulty flexibly modulating their speed-accuracy trade-off settings to meet specific task demands. In future studies, it will be important to fully explore the degree to which children with ADHD can flexibly adjust their response style to meet task demands.

Although the current data suggest that children with and without ADHD do not differ in their speed-accuracy trade-offs in neutral instructional contexts, it is important to note how the context of the stop signal paradigm may have impacted results. Previous work in adults suggests that individuals strategically slow their responding on go trials, to minimize failures of inhibition on stop trials. This strategy results in the diffusion model detecting wider boundary separations than would be seen in a non-stop task context (Verbruggen & Logan, 2009). In our sample, non-ADHD controls successfully inhibited more frequently than children with ADHD, which could suggest they adopted this strategy. The concern is that if non-ADHD controls adopt this strategy, it may be reflected in their having larger boundary separations on the stop task than they would on a non-inhibition task and could result in finding a group difference in boundary separation that resulted only from the contextual task demands. However, even in the presence of this potential confound we found no group differences in boundary separation. The demands of the stop task may also change how motor preparatory processes are carried out, impacting non-decision times. Despite concerns, the diffusion model has been applied successfully to a wide range of tasks including the stop task, with consistently good fit to the data (Ratcliff & McKoon, 2008), and all participant data in our sample was well fit to the model. To mitigate the effects of stop trials on parameter estimates, we removed all stop+1 trials but it will be critical for future studies to apply the diffusion model in other choice RT paradigms to fully explore these potential confounds.

Limitations & Future Directions

The current results help elucidate mechanisms contributing to cognitive impairments in ADHD. Impairments in EF have been linked directly to poor academic achievement and

problematic skill acquisition in ADHD (Biederman et al., 2004; Huang-Pollock & Karlunas, 2010). Better understanding how molar EF deficits arise is therefore critically important to the development of better psychoeducational intervention strategies. Current results suggest that a common deficit in rapid, efficient decision-making accounts for a substantial proportion of variance in EF ability among children with and without ADHD. Ultimately, finding interventions to directly target slow, inefficient processing may be a more successful than targeting individual EF weaknesses. That being said, it is important to note that significant direct paths from ADHD to inhibitory control and to WM were also found (as shown in Figures 2 and 3, respectively). This either indicates the presence of additional mediators that were not accounted for in the model, or, because EFs are not unitary (Miyake et al., 2000), that the remaining variance reflects residual ADHD-related deficits specific to response inhibition and WM. Future studies will be required to explore these possibilities.

Several other areas will also be critical for future research to address. First, in the absence of longitudinal data or direct task manipulations of WM and inhibitory demands, it is not possible to firmly conclude that slow drift rates cause impairments in EF. The correlations between mediator and outcome could be reversed, such that poor WM and inhibition actually cause slow drift rate. Although our data cannot rule out this possibility, our interpretation of directionality rests on strong empirical evidence demonstrating that developmental improvements in WM can be attributed to development of processing speed (rather than the other way around, Fry & Hale, 2000; Kail, 2007) and with previous work suggesting that slow processing speed would result in poor inhibition (Alderson, et al., 2007; Lijffijt, et al., 2005). However, longitudinal studies with cross-lag designs that can address causality between processes that are developing in tandem (e.g. Blandon, Calkins, Grimm, Keane, & O'Brien, 2010) or direct task manipulations will be important for fully understanding the causal relationships between these processes.

Second, diffusion model parameters were estimated from the same task that was used to estimate inhibitory control. Although the “go” and “stop” tasks that are embedded in the stop signal paradigm have been conceptualized as reflecting independent processes, estimation from the same task potentially increases the correlations between diffusion model and inhibitory control measures. However, of the diffusion model parameters, only drift rate mediated the relationship between ADHD status and inhibitory control. If mediation effects were an artifact of having estimated the diffusion parameters from the go trials of the task, then we might expect both drift rate and non-decision time to be mediators, or for all three diffusion parameters to show similar effect sizes and correlations with the SSRT outcome, which they do not. Further, drift rate also mediated the relationship between ADHD status and WM deficits. The specificity of mediation effects to a single diffusion model parameter and the replication of results across two prominent EFs suggests that results cannot simply be attributed to procedures used to estimate diffusion parameters. Nonetheless, future studies using two distinct tasks for measurement of inhibitory control and diffusion model parameters will be required.

Finally, our community recruitment procedures resulted in a sample of children with ADHD who are highly representative of the population of children with ADHD in terms of subtype composition, gender ratios, and proportion of children taking stimulant medications (Froehlich et al., 2007). Nonetheless, this resulted in the control and ADHD samples having different gender ratios. The inclusion of gender as a covariate did not affect group differences on any measure; however, samples size was not large enough to test mediational models separately for boys and girls. In future studies, it will be important to determine whether mediation effects differ by gender.

Conclusion

Overall, we present an integrated account based on a diffusion modeling technique that explains why children with ADHD often (but not always) present with slow and variable RTs, and why slower and more variable RTs regularly co-occur with impaired EF in ADHD. Such work has direct implication for prominent etiologic theories of ADHD and the search for cognitive endophenotypes, and it provides one method by which to better understand the mechanisms underlying cognitive deficits in childhood ADHD. Future work examining individual differences in the flexibility of these parameters to context, as well as their reliability across time, and relationship to neural correlates will be important next steps to assist in the elucidation of cognitive mechanisms characterizing ADHD.

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$$\text{Reaction Time} = \text{Non-decision time} + \text{Decision Time}$$

Figure 1.

Diffusion model parameters depicted for a hypothetical single trial (adapted from Ratcliff & Rouder, 1998). Drift rate (v) is the rate at which information accumulates towards a decision boundary, determined by speed of information processing and “noise” unrelated to the decision processes; boundary separation (a) indicates the conservativeness of the response criterion with wider separations indicating more conservative responding; and non-decision time (Ter) includes all non-decision processes, such as stimulus encoding and motor preparation.

Figure 2. Multiple mediation model demonstrating the relationships between ADHD, RT components, and stop signal reaction time (SSRT). Standardized parameter estimates displayed. *** $p < 0.001$, * $p < 0.05$.

Figure 3. SEM demonstrating the relationships between ADHD, RT components, and WM capacity. Standardized parameter estimates displayed. *** $p < 0.001$, * $p < 0.05$.

Table 1

Description of Diffusion Model Parameters

<i>Parameter</i>	<i>Interpretation</i>	<i>Additional Explanation</i>
Drift Rate (v)	Speed/efficiency of information processing	The average slope of the line reflects the rate of information processing. Deviations from the average slope indicate the effects of random neural noise on the decision process. Larger values of v indicate faster and more efficient information processing.
Boundary Separation (a)	Speed-accuracy trade-off setting	The more conservative the response criterion, the farther apart the response boundaries are. Thus, larger values of a indicate greater emphasis on accuracy than speed in the decision processes
Non-decision Time (Ter)	Time for all non-decisional processes	Ter processes prior to the start of the decision process (e.g. stimulus encoding) and following the response decision (e.g. motor response preparation and execution). Larger values of Ter indicate longer non-decisional processing times

Table 2**Sample Characteristics and Task Performance Measures**

	<i>Control</i>	<i>ADHD</i>	<i>Group Comparison</i>
<i>Basic Demographics</i>			
N	62	91	
(Boys:Girls)	29:33	61:30	$\chi^2(1) = 6.25, p = .012$
Subtype (C,I,H)		40,48,3	
Full Scale IQ	106.94 (10.40)	104.42 (11.73)	$F(1,151) = 1.86, p = 0.175, \eta^2 = 0.01$
% Caucasian & non-Hispanic	79.1	82.8	$\chi^2(1) = 1.71, p = .191$
Median Annual Household Income	\$61,000–\$70,000	\$61,000–\$70,000	$Z = -1.24, p = .216$
Age (years)	10.56 (1.21)	10.19 (1.39)	$F(1,151) = 2.87, p = 0.092, \eta^2 = 0.02$
Total #Att Symptoms	0.79 (1.01)	8.05 (1.21)	$F(1,151) = 1522.61, p < 0.001, \eta^2 = 0.91$
Total #HI Symptoms	0.29 (0.52)	4.99 (3.01)	$F(1,151) = 147.09, p < 0.001, \eta^2 = 0.49$
ODD (<i>n</i>)	1	30	$\chi^2(1) = 23.07, p < .001$
CD (<i>n</i>)	0	6	$\chi^2(1) = 4.35, p = .037$
GAD (<i>n</i>)	1	6	$\chi^2(1) = 2.17, p = .140$
MDD/Dysthymia (<i>n</i>)	0	1	$\chi^2(1) = 0.73, p = .391$
<i>Working Memory</i>			
Letter Span	0.29 (0.14)	0.20 (0.08)	$F(1,146) = 20.44, p < 0.001, \eta^2 = 0.12$
Spatial Span	0.26 (0.10)	0.21 (0.09)	$F(1,145) = 9.76, p = 0.002, \eta^2 = 0.06$
Digits Backwards	0.27 (0.10)	0.24 (0.10)	$F(1,148) = 4.01, p = 0.05, \eta^2 = 0.03$
Finger Windows Backwards	0.36 (0.14)	0.28 (0.13)	$F(1,143) = 12.62, p = 0.001, \eta^2 = 0.08$
<i>Stop Task</i>			
% Choice Accuracy	94.1 (7.96)	86.1 (10.43)	$F(1,151) = 26.07, p < 0.001, \eta^2 = 0.15$
# Omissions	3.10 (4.04)	6.33 (5.41)	$F(1,151) = 16.00, p = 0.001, \eta^2 = 0.10$
Mean RT	749.97 (174.66)	758.04 (123.71)	$F(1,151) = 0.11, p = 0.738, \eta^2 < 0.01$
SD RT	196.50 (61.82)	231.88 (52.33)	$F(1,151) = 14.54, p = 0.001, \eta^2 = 0.09$
Stop Signal RT	285.73 (90.02)	422.51 (152.18)	$F(1,151) = 40.40, p < 0.001, \eta^2 = 0.21$
<i>Ex-Gaussian Components</i>			
Mu (μ)	596.60 (182.64)	564.55 (128.59)	$F(1,151) = 1.62, p = .205, \eta^2 = 0.01$
Sigma (σ)	113.92 (59.12)	126.86 (56.74)	$F(1,151) = 1.85, p = .175, \eta^2 = 0.01$
Tau (τ)	148.36 (75.63)	185.42 (72.95)	$F(1,151) = 9.24, p = .003, \eta^2 = 0.06$
<i>Diffusion Modeling Components</i>			
Drift rate (v)	-3.14 (0.85)	-2.53 (0.67)	$F(1,151) = 23.86, p < 0.001, \eta^2 = 0.14$
Boundary Separation (a)	1.72 (0.33)	1.65 (0.34)	$F(1,151) = 2.05, p = 0.154, \eta^2 = 0.01$
Non-Decision Time (Ter)	0.46 (0.13)	0.42 (0.09)	$F(1,151) = 5.17, p = 0.024, \eta^2 = 0.03$

Note: Att = Attention, HI = Hyperactive/Impulsive, ODD = oppositional defiant disorder, CD = conduct disorder, GAD = Generalized Anxiety Disorder, MDD = Major Depressive Disorder, RT = Reaction time, SD = Standard Deviation. Smaller absolute values of v indicate slower drift rates. Smaller values of a indicate greater speed-accuracy trade off, and Ter is reported in seconds.

Table 3

Correlation Matrix for ex-Gaussian, Diffusion Model, Working Memory, and Stop Task Outcomes

	1	2	3	4	5	6	7	8	9	10	11
	μ	σ	τ	ν	a	Ter	SSRT	LSpan	DSB	Sspan	FWB
1	1										
2	.73***	1									
3	-.36***	-.21**	1								
4	-.28***	.03	.64***	1							
5	.37***	.25***	.25**	-.22**	1						
6	.85***	.38***	-.40***	-.38***	.13	1					
7	-.16*	.09	.42***	.44***	-.15	-.23**	1				
8	.20**	.04	-.24**	-.32***	.10	.17*	-.21**	1			
9	-.03	-.10	-.15	-.07	-.13	-.03	-.12	.30***	1		
10	-.04	-.15	-.26**	-.32***	-.01	.07	-.27***	.37***	.25**	1	
11	-.10	-.07	-.24**	-.31***	.03	0.04	-.39***	.48***	.26***	.51***	1

Note: SSRT = Stop Signal Reaction Time Task, LSpan = Letter Span, DSB = Digit Span Backwards, Sspan = Spatial Span, FWB = Finger Windows Backwards,

***
p 0.001,

**
p 0.01,

*
p<0.05