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Re-Examining the Stop-Signal Task to Test Competing Theories of AD/HD

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Abstract

The current study tested two competing models of Attention-Deficit/Hyperactivity Disorder (AD/HD), the inhibition and state regulation theories, by conducting fine-grained analyses of the Stop-Signal Task and another putative measure of behavioral inhibition, the Gordon Continuous Performance Test (G-CPT), in a large sample of children and adolescents. The inhibition theory posits that performance on these tasks reflects increased difficulties for AD/HD participants to inhibit prepotent responses. The model predicts that putative stop-signal reaction time (SSRT) group differences on the Stop-Signal Task will be primarily related to AD/HD participants requiring more warning than control participants to inhibit to the stop-signal and emphasizes the relative importance of commission errors, particularly "impulsive" type commissions, over other error types on the G-CPT. The state regulation theory, on the other hand, proposes response variability due to difficulties maintaining an optimal state of arousal as the primary deficit in AD/HD. This model predicts that SSRT differences will be more attributable to slower and/or more variable reaction time (RT) in the AD/HD group, as opposed to reflecting inhibitory deficits. State regulation assumptions also emphasize the relative importance of omission errors and "slow processing" type commissions over other error types on the G-CPT.

Overall, results of Stop-Signal Task analyses were more supportive of state regulation predictions and showed that greater response variability (i.e., SDRT) in the AD/HD group was not reducible to slow mean reaction time (MRT) and that response variability made a larger contribution to increased SSRT in the AD/HD group than inhibitory processes. Examined further, ex-Gaussian analyses of Stop-Signal Task go-trial RT distributions revealed that increased variability in the AD/HD group was not due solely to a few excessively long RTs in the tail of the AD/HD distribution (i.e., tau), but rather indicated the importance of response variability throughout AD/HD group performance on the Stop-Signal Task, as well as the notable sensitivity of ex-Gaussian analyses to variability in data screening procedures. Results of G-CPT analyses indicated some support for the inhibition model, although error type analyses failed to further differentiate the theories. Finally, inclusion of primary variables of interest in exploratory factor analysis with other neurocognitive predictors of AD/HD indicated response variability as a separable construct and further supported its role in Stop-Signal Task performance. Response variability did not, however, make a unique contribution to the prediction of AD/HD symptoms beyond measures of motor processing speed in multiple deficit regression analyses. Results have implications for the interpretation of the processes reflected in widely-used variables in the AD/HD literature, as well as for the theoretical understanding of AD/HD.

Third Advisor

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RE-EXAMINING THE STOP-SIGNAL TASK TO

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Disorder (AD/HD), the inhibition and state regulation theories, by conducting fine-

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inhibition, the Gordon Continuous Performance Test (G-CPT), in a large sample of

children and adolescents. The inhibition theory posits that performance on these tasks

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than control participants to inhibit to the stop-signal and emphasizes the relative

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primary deficit in AD/HD. This model predicts that SSRT differences will be more

attributable to slower and/or more variable reaction time (RT) in the AD/HD group, as

opposed to reflecting inhibitory deficits. State regulation assumptions also emphasize the

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Overall, results of Stop-Signal Task analyses were more supportive of state regulation predictions and showed that greater response variability (i.e., SDRT) in the AD/HD group was not reducible to slow mean reaction time (MRT) and that response variability made a larger contribution to increased SSRT in the AD/HD group than inhibitory processes. Examined further, ex-Gaussian analyses of Stop-Signal Task go-trial RT distributions revealed that increased variability in the AD/HD group was not due solely to a few excessively long RTs in the tail of the AD/HD distribution (i.e., tau), but rather indicated the importance of response variability throughout AD/HD group performance on the Stop-Signal Task, as well as the notable sensitivity of ex-Gaussian analyses to variability in data screening procedures. Results of G-CPT analyses indicated some support for the inhibition model, although error type analyses failed to further differentiate the theories. Finally, inclusion of primary variables of interest in exploratory factor analysis with other neurocognitive predictors of AD/HD indicated response variability as a separable construct and further supported its role in Stop-Signal Task performance. Response variability did not, however, make a unique contribution to the prediction of AD/HD symptoms beyond measures of motor processing speed in multiple deficit regression analyses. Results have implications for the interpretation of the processes reflected in widely-used variables in the AD/HD literature, as well as for the theoretical understanding of AD/HD.

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Introduction

Attention Deficit/Hyperactivity Disorder (AD/HD) is a heterogeneous

neurobehavioral disorder of childhood that affects 3 to 10 percent of children (APA,

1994; Barkley, 2006; Satcher, 1999). It is defined clinically by symptoms of inattention,

hyperactivity, and/or impulsivity that begin prior to age 7 and cause significant

impairment in academic, social, or occupational functioning (APA, 1994). Current major

theoretical models of AD/HD emphasize deficits in different domains, such as the state

regulation, executive inhibition, and delay aversion models, of which predictions from

the former two theories will be tested in the current study. While prior research indicates

that no single deficit in any of these domains is necessary or sufficient to explain all cases

(Nigg, 2005; Sonuga-Barke, 2005; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005),

it is suggested that these theoretical models may represent different causal pathways to

the disorder and are therefore important to further research.

Some findings that have been especially robust in the extant AD/HD literature,

and important to state regulation and executive inhibition theories in particular, are

differences in various reaction time (RT) variables. Specifically, research has shown that

children with AD/HD have slower mean reaction time (MRT) compared to controls on a

variety of tasks (Lijffijt, Kenemans, Verbaten, & van Engeland, 2005; Teicher, Ito, Glod,

& Barber, 1996; van der Meere & Sergeant, 1988). An even more common and

consistent finding is that children with AD/HD have greater response variability, or

standard deviation of reaction time (SDRT) in RT tasks, than controls (Castellanos et al., 2005; Klein, Wendling, Huettner, Ruder, & Peper, 2006; Kuntsi, Oosterlaan, & Stevenson, 2001; Rommelse et al., 2008; Zahn, Kruesi, & Rapoport, 1991). Several researchers have suggested that response variability is a hallmark of AD/HD (Buzy, Medoff, & Schweitzer, 2009; Castellanos & Tannock, 2002; Kuntsi & Stevenson, 2001), and state regulation theorists claim that these differences indicate poor regulation of arousal during tasks (Sergeant, 2000; Sergeant, Oosterlaan, & van der Meere, 1999).

Children with AD/HD are also commonly found to have longer stop-signal reaction time (SSRT) than controls (for reviews, see Nigg, 2001; Oosterlaan, Logan, & Sergeant, 1998; Willcutt, Doyle et al., 2005). SSRT is thought to provide a measure of how much warning an individual needs to stop a prepotent response, with longer SSRT suggesting that more warning is necessary. Executive inhibition theorists cite deficits in SSRT as evidence of poor behavioral response inhibition in AD/HD (Barkley, 1997; Nigg, 2000; Pennington & Ozonoff, 1996).

While these findings suggest different possible causal pathways to AD/HD, there is some suggestion that SDRT and SSRT may be reducible to other phenomena in children with AD/HD. More specifically, longer SDRT may be a by-product of a larger number of abnormally slow responses rather than overall RT slowing, and SSRT may be reducible to slower and/or more variable RT rather than a response inhibition deficit. Analyzing these variables with more fine-grained approaches is therefore pertinent to testing specific theories of AD/HD and contributing to a better understanding of the processes underlying commonly used measures in the AD/HD literature.

Major Theoretical Models of AD/HD

   State regulation. State regulation theories of AD/HD hold that the core deficit in AD/HD is difficulty maintaining an optimal state of arousal for task completion. The cognitive-energetic model is a specific approach to AD/HD that uses this framework (Sergeant, 2000; Sergeant et al., 1999). It involves different levels of processing, including a level of energetic "pools" corresponding to arousal, activation, and effort. Within this model, deficits in energetic pools are considered a primary feature of AD/HD, whereas other associated neuropsychological deficits are secondary. Specifically, slower and more variable reaction time in AD/HD (e.g., Klein et al., 2006) is postulated as evidence of difficulty maintaining appropriate cognitive activation. Research suggesting that manipulating event rate and reward can lessen inhibition deficits in AD/HD (Andreou et al., 2007; Scheres, Oosterlaan, & Sergeant, 2001) is also cited as evidence for poor state regulation; however, results of these studies are mixed (Buzy et al., 2009; Shanahan, Pennington, & Willcutt, 2008).

   An issue with this type of model is that slower and more variable reaction time is present in other disorders, such as intellectual disability (Nettelbeck & Wilson, 1997), high functioning autism (Verte, Geurts, Roeyers, Oosterlaan, & Sergeant, 2006), and traumatic brain injury (Segalowitz, Dywan, & Unsal, 1997), as well as in normal and pathological aging (MacDonald, Nyberg, & Backman, 2006). Furthermore, the cognitive-energetic model suggests that the energetic pools originate in subcortical structures such as the hippocampus, mesencephalic reticular formation, and basal ganglia (Sergeant, 2005), whereas structural and functional neuroimaging studies of RT and RT variability generally suggest more involvement of frontal cortical regions (for a review,

see MacDonald et al., 2006). While some issues regarding specific brain mechanisms are beyond the scope of the current study, a key question that will be addressed is whether greater RT variability in AD/HD is just a consequence of overall slower RT, or more consistent with state regulation models, is a distinct process resulting from increased variability in the fast and/or slow parts of the RT distribution in AD/HD.

Executive inhibition. Executive inhibition theories of AD/HD postulate a core deficit in behavioral inhibition (Barkley, 1997; Nigg, 2000; Pennington & Ozonoff, 1996). More specifically, models utilizing this approach suggest that voluntary response inhibition, mediated by the frontal lobes, is impaired in AD/HD (Aman, Roberts, & Pennington, 1998). Neuroimaging studies have supported involvement of frontal regions as neural correlates of response inhibition (Aron & Poldrack, 2005; Casey, 2000), and individuals with AD/HD have been shown to have poorer performance on putative response inhibition tasks (Willcutt, Doyle et al., 2005). This includes more errors of commission (i.e., responding to sequences other than the target sequence) on go/no-go tasks (Castellanos et al., 2000; Iaboni, Douglas, & Baker, 1995) and other continuous performance tests (for a review, see Losier, McGrath, & Klein, 1996), as well as poorer performance on measures of interference control (Jonkman et al., 1999; Reeve & Schandler, 2001), though results sometimes vary (Kuntsi, Oosterlaan et al., 2001; van Mourik, Oosterlaan, & Sergeant, 2005).

A more primary task postulated to provide evidence for a core behavioral inhibition deficit is the Stop-Signal Task (Logan, 1994), which is described in greater detail in the *Method* section of the current study. The well-replicated finding of longer SSRT in AD/HD is thought to reflect AD/HD participants needing more warning than

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controls to inhibit response to the stop-signal, and is thus believed to indicate poor voluntary motor response inhibition in AD/HD (for reviews, see Nigg, 2001; Oosterlaan et al., 1998; Willcutt, Doyle et al., 2005).

Similar to state regulation models, key findings used to support executive inhibition theories are not specific to AD/HD. For instance, poorer response inhibition, including slower SSRT, is found in several other disorders such as oppositional defiant disorder (ODD) and conduct disorder (CD; Oosterlaan et al., 1998; Sergeant, Geurts, & Oosterlaan, 2002), as well as adolescent substance use disorders independent of CD and AD/HD status (Nigg et al., 2006). Some research has also found slower SSRT in high functioning autism (HFA; Geurts, Verte, Oosterlaan, Roeyers, & Sergeant, 2004), although other studies have not (Ozonoff & Strayer, 1997). The underlying mechanisms of response inhibition are also unclear. Failure to inhibit responses could be due to weak top-down control, as suggested by executive inhibition theories, or to strong bottom-up impulses, or to both.

Recent research has also suggested that longer SSRT in AD/HD may not reflect poor response inhibition, but rather may be due to longer MRT in AD/HD (Alderson, Rapport, & Kofler, 2007; Alderson, Rapport, Sarver, & Kofler, 2008; Lijffijt et al., 2005). A central question to be addressed by the current study is therefore whether SSRT differences can be reduced to slower RT in AD/HD, which would be more sportive of state regulation than inhibition theory assumptions. If so, can other putative response inhibition measures also be reduced to RT related differences?

Delay aversion. Delay aversion is another theoretical model of AD/HD, which suggests that the core problem in AD/HD is preference for immediate, small rewards over

delayed, larger rewards (Sagvolden, Johansen, Aase, & Russell, 2005; Sonuga-Barke, 2002, 2003). Related constructs include delay of gratification (Mischel, Shoda, & Rodriguez, 1989) and delay discounting (Green & Myerson, 2004). Delay aversion theories are variants of motivational explanations of AD/HD, which propose that dysfunctional response to contingencies is the main deficit in AD/HD (Hartung, Milich, Lynam, & Martin, 2002; Newman & Wallace, 1993). Results of studies manipulating reward and punishment, however, have been mixed. Some find improved performance in AD/HD (Carlson, Mann, & Alexander, 2000; Konrad, Gauggel, Manz, & Scholl, 2000; Slusarek, Velling, Bunk, & Eggers, 2001), whereas others have not found an effect of response contingencies (Oosterlaan & Sergeant, 1998; Shanahan et al., 2008; Stevens, Quittner, Zuckerman, & Moore, 2002).

Aside from state regulation, delay aversion has generally had more support in the AD/HD literature than other motivational explanations, with several studies finding that individuals with AD/HD have a tendency to make choices that minimize delay (for reviews, see Luman, Oosterlaan, & Sergeant, 2005; Sonuga-Barke, Sergeant, Nigg, & Willcutt, 2008). It has been suggested, however, that delay aversion may be more related to comorbid conduct problems than AD/HD-related symptoms (Kuntsi, Oosterlaan et al., 2001). Recent research has also failed to find significant genetic effects for delay aversion, as compared to moderate heritability estimates for other constructs such as RT variability and response inhibition (Andreou et al., 2007; Kuntsi et al., 2006).

Differences in delay aversion have also not been replicated in the CLDRC sample to be utilized for the current study. Given the aforementioned issues, delay aversion will

not be further pursued in the current study, and as noted previously, the focus of analyses involving RT variables will be on state regulation and inhibition theories of AD/HD.

Multiple deficit. While core deficit models are useful for understanding possible causal pathways to the disorder, current evidence also argues against a single deficit model as necessary and sufficient to explain all cases of AD/HD (Nigg, 2005; Sonuga-Barke, 2005; Willcutt, Doyle et al., 2005). Instead, approaches that attempt to account for the heterogeneity of AD/HD are becoming more prevalent (Nigg, 2005; Sergeant, 2005; Sonuga-Barke, 2005). Some models propose independent pathways to AD/HD and emphasize potential single-deficit subtypes of the disorder (Nigg, Goldsmith, & Sachek, 2004; Sonuga-Barke, 2005), whereas others suggest that AD/HD results from additive or interactive effects of dysfunction in multiple domains (Pennington, 2006; Sergeant, Geurts, Huijbregts, Scheres, & Oosterlaan, 2003; Willcutt, Brodsky et al., 2005).

Various deficits are considered in these different models including those from executive function domains, such as response inhibition, vigilance, working memory, set-shifting, and interference control, as well as processing speed, response variability, and motivational processes such as delay aversion. Many of these constructs have been supported by factor analyses of various tasks (McGrath et al., 2011; Miyake et al., 2000; Shanahan et al., 2006; Willcutt et al., 2001; Willcutt, Pennington, Olson, Chhabildas, & Hulslander, 2005). A challenge of this approach is constructing a comprehensive theoretical model that considers all relevant deficits, yet that is not so broad in scope that it lacks specificity or appropriate theoretical rationale for the disorder. Following clarification of the RT variables claimed by state regulation and executive inhibition theories, a question to be addressed in the current study is how these variables fit into a

multiple deficit approach that incorporates various other constructs found to differentiate AD/HD and control groups.

Relationships among Primary Variables of Interest

SDRT and MRT. As noted previously, slower MRT and other processing speed deficits in AD/HD are common findings and have been replicated across various tasks, including slower MRT across RT tasks of varying demands (Klein et al., 2006), as well as slower naming speed (Shanahan et al., 2006; van Mourik et al., 2005) and poorer performance on processing speed tasks involving simple motor (Shanahan et al., 2006) and increased executive demands (Willcutt, Doyle et al., 2005). Even more common is greater RT variability in AD/HD (e.g., Castellanos et al., 2005; Klein et al., 2006; Rommelse et al., 2008); however, the specific relationship between slower and more variable RT can vary and has yet to be thoroughly evaluated in AD/HD.

An important issue to consider when examining the relationship between MRT and SDRT is that these summary statistics may mask important patterns in RT data (Heathcote, Popiel, & Mewhort, 1991; Spieler, Balota, & Faust, 2000). More specifically, the mean and standard deviation of RT provide information about possible group differences in these variables; however, these methods do not provide a detailed analysis of the distribution of the data. There is often a positive skew to RT data (Ratcliff, 1979), which depending on severity, may unduly influence the results and interpretation of summary statistics. For example, increased SDRT could be a consequence of slower MRT and therefore indicate more general processing speed deficits, as has been found in intellectual disability (Brewer & Smith, 1984). In contrast, MRT and SDRT could represent different processes, such that increased MRT and SDRT

are the result of more excessively long RT trials or "attentional lapses," as has been

suggested in AD/HD (Hervey et al., 2006; Leth-Steensen, Elbaz, & Douglas, 2000).

Since the specific distribution of RTs is condensed with summary statistics, this

relationship cannot be determined. Reliance on these statistics alone can therefore result

in a poor understanding of the extent and type of deficit that is present in a given group.

RT distribution analyses are a means of assessing this underlying relationship.

These types of analyses allow for the examination of patterns of performance by

analyzing trial-by-trial RT data (Heathcote, Brown, & Cousineau, 2004; Heathcote,

Brown, & Mewhort, 2002). Since RT distributions are often positively skewed (Ratcliff,

1979), it is important to use methods that account for the degree of skew and assess the

different patterns that may result. The ex-Gaussian curve is a particular type of

distribution that allows for this type of analysis. It takes into account the additive

combination of the independent Gaussian (normal) distribution, and exponential random

variables that make up the positive skew of the distribution (Burbeck & Luce, 1982). It

consists of three parameters that quantify these components: μ (mu), which is the mean

of the normal component; σ (sigma), which is the standard deviation of the normal

component; and τ (tau), which is the mean of the exponential component.

As can be seen in Figure 1, performance can be similar on the normal part of the

distribution (mean (mu) and standard deviation (sigma)), yet differ on the exponential

component (tau). When combined, this creates different shapes of the overall RT

distribution. If relying only on summary statistics, group (a) would have smaller MRT

and SDRT than group (b); however, important data regarding potential reasons for this

difference would be unaccounted for unless the shape of the distribution is also analyzed.

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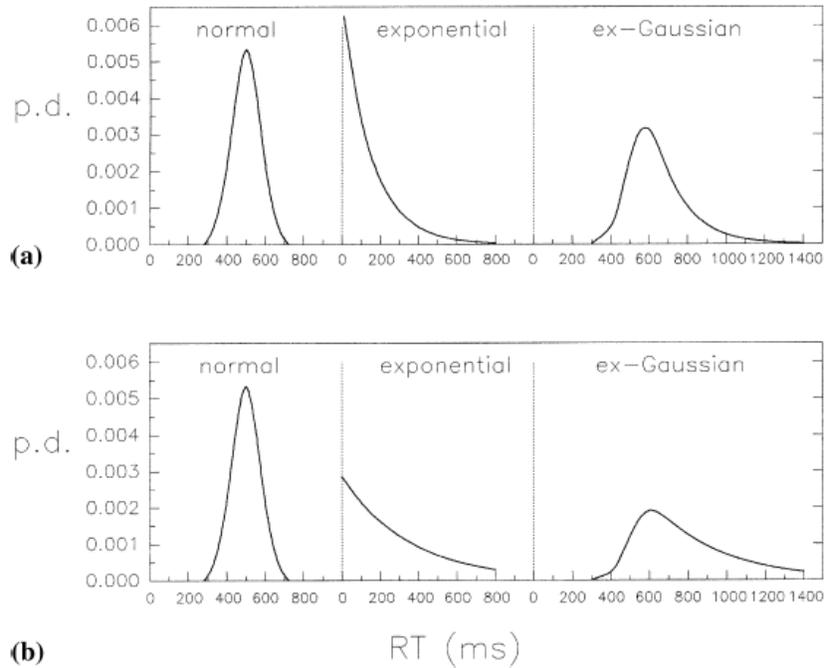
Figure 1. Probability density (p.d.) functions showing normal components of μ (mu) = 500ms and σ (sigma) = 75ms ((a),(b)), exponential components of τ (tau) = 150ms (a) and 350ms (b), and the resulting ex-Gaussian distributions (Leth-Steensen et al., 2000).

SDRT and MRT in AD/HD and other groups. RT distributions have shown different patterns in different groups. For instance, when examining the ex-Gaussian distribution in healthy aging (McAuley, Yap, Christ, & White, 2006), older adults were found to differ from young adults on all three components, indicating differences in each part of the RT distribution, with slower (mu), more variable (sigma), and more extreme (tau) response patterns in older adults. Children differed from young adults on sigma, but tau and mu were similar, indicating similar average RT and similar shapes of the tail of the distribution, but more variable responding in the normal part of the RT curve. In intellectual disability, while the ex-Gaussian curve was not specifically modeled, plotting the RT distribution revealed general RT slowing in individuals with intellectual disability compared to controls (Figure 2; Brewer & Smith, 1984).

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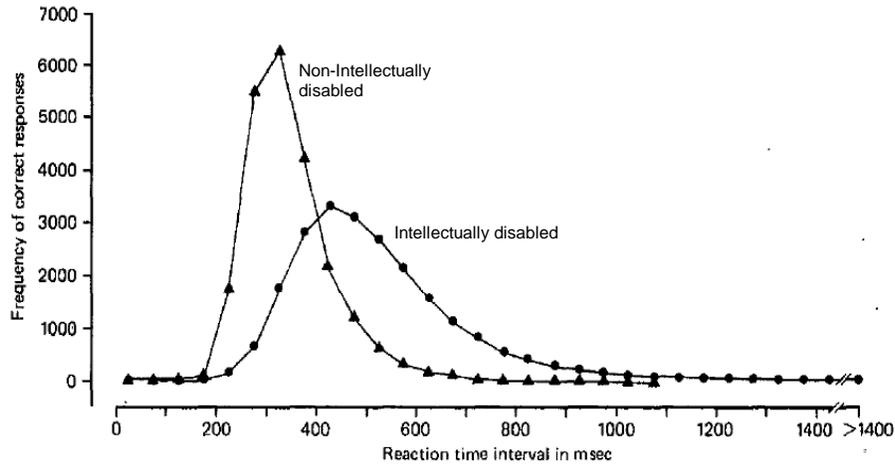
Figure 2. Distributions of reaction time frequencies for individuals with and without intellectual disability on a choice reaction time task (Brewer & Smith, 1984).

In contrast, it has been suggested that greater SDRT in AD/HD may not just be a consequence of slower MRT, as appears to be the case in intellectual disability. This is supported by research showing a larger effect size for SDRT as compared to MRT, suggesting that SDRT is not entirely accounted for by MRT (Klein et al., 2006; Lijffijt et al., 2005). Furthermore, factor analysis of various cognitive tasks indicates that measures of response variability cluster separately from other processing speed variables (Willcutt, Pennington et al., 2005).

Regarding RT distributions, some researchers have therefore hypothesized that differences in SDRT in AD/HD may primarily reflect differences in the exponential portion of the ex-Gaussian curve (Hervey et al., 2006; Leth-Steensen et al., 2000). Specifically, individuals with AD/HD may perform more similarly to controls on the mean (mu) and standard deviation (sigma) components of the normal Gaussian distribution, but vary from controls on the ex-Gaussian (tau) component, indicating

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similar performance with regard to the majority of RT trials but more excessively long RTs in the tail of the AD/HD distribution.

An initial study examining the ex-Gaussian distribution in AD/HD found that individuals with AD/HD had a larger tau component and similar mu and sigma compared to controls on a 4-choice RT task (Leth-Steensen et al., 2000). Another study utilizing a go/no-go task (Conners' Continuous Performance Test) also found the AD/HD group to have significantly larger tau than controls (Hervey et al., 2006), as well as larger sigma, indicating increased variability in the normal part of the distribution as well as in the tail. Additionally, mu was found to be faster in the AD/HD group compared to controls in this study, indicating faster responding in the normal part of the curve in the AD/HD group. Figure 3 depicts the different ex-Gaussian distributions for AD/HD and control groups from the Hervey et al. (2006) study.

RT DISTRIBUTION IN ADHD
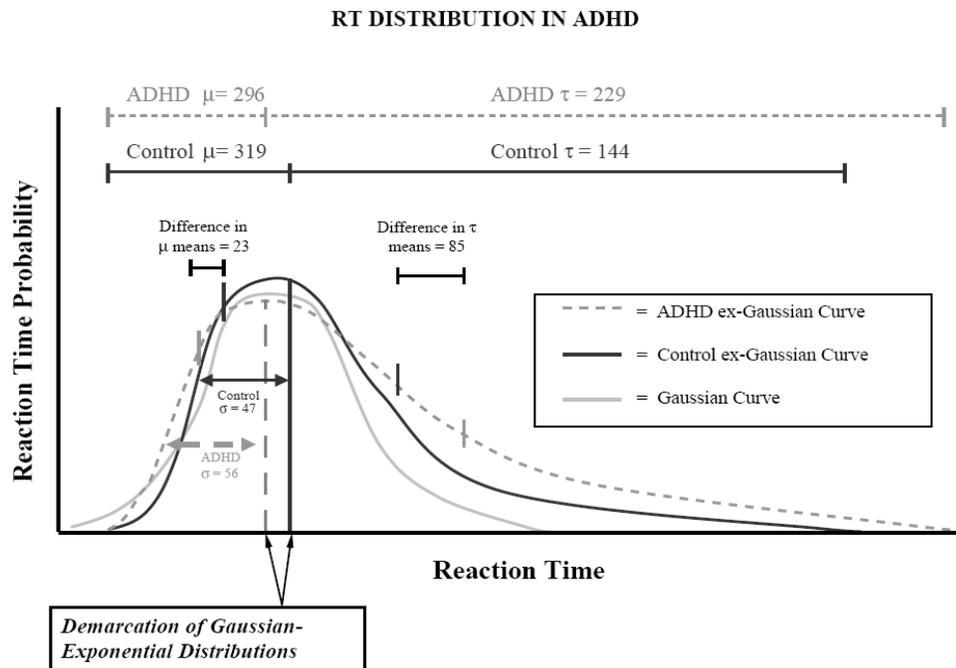
Figure 3. Reaction time differences (in milliseconds) between AD/HD and control groups on mu, sigma, and tau components of the ex-Gaussian distribution (Hervey et al., 2006).

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Given results of these studies, it has therefore been suggested that differences in MRT and SDRT in AD/HD may be primarily related to more excessively long RTs in the AD/HD distribution, potentially indicating more lapses in attention rather than general RT slowing seen in other groups. Results, however, have varied, with a recent study finding no significant differences between AD/HD and control groups in any ex-Gaussian components on a brief 2-choice RT task (Geurts et al., 2008). The ex-Gaussian distribution has also not been examined in the Stop-Signal Task, which differs from previous studies in the relative cognitive demands required for task completion. Further examination of the underlying relationship between MRT and SDRT in AD/HD with large samples and in another commonly used measure in the AD/HD literature (i.e., Stop-Signal Task) is therefore warranted.

SSRT and MRT. Stop-signal reaction time (SSRT) has been another important variable in AD/HD research. Individuals with AD/HD are often found to have longer SSRT than controls across a number of studies (for reviews, see Nigg, 2001; Oosterlaan et al., 1998; Willcutt, Doyle et al., 2005). Meta-analysis has also shown a larger effect for SSRT as compared to MRT in AD/HD (Oosterlaan et al., 1998). These results have been interpreted as reflecting a core deficit in behavioral inhibition. It is important, however, to understand how SSRT is derived and how it relates to MRT before drawing conclusions regarding its validity as a measure of behavioral inhibition. To do this, it is first necessary to describe the basic design of the Stop-Signal Task (Logan, 1994), which is often considered a primary measure for investigating behavioral inhibition in AD/HD.

The Stop-Signal Task involves a dual-task paradigm in which individuals are asked to respond to a primary 'go' stimulus and withhold responding to a secondary

'stop' stimulus. The traditional version of the task involves responding to different go-stimuli (e.g. the letters 'X' and 'O') using left and right response buttons, and withholding responses when the go-stimulus is paired with a stop-signal, generally an auditory tone. Stop-signals occur on a predetermined number of trials (e.g., 25%). On stop-signal trials, there is a delay between the presentation of the go and stop stimuli, referred to as the stop-signal delay (SSD). This metric can be somewhat confusing to interpret because *shorter* SSD indicates that the individual required *more* warning to inhibit a response to the stop-signal (i.e., a smaller interval between the go- and stop-signals). Conversely, *longer* SSD indicates that the individual did not need as much warning to inhibit a response (i.e., a larger interval between the go- and stop-signals). In other words, as SSD *increases*, warning time between go- and stop-signals *decreases*.

On contemporary versions of the task, SSD is varied based on task performance (e.g., Logan, Schachar, & Tannock, 1997). More specifically, if a response is successfully inhibited on a stop trial, SSD is increased (e.g., 50ms) for the following stop trial. If the response is not inhibited, SSD is decreased. This procedure is designed to approximate successful inhibition on 50% of stop-signal trials. In contrast, earlier versions of the task utilized a range of fixed SSDs relative to the participant's MRT rather than adjusting SSD based on trial-by-trial stop-signal performance (Logan, 1994). For both versions, RT to the go-signal is calculated as the time between the go stimulus and the individual's response. MRT is therefore the average of RTs to go-signal trials.

The Stop-Signal Task is based on the race model of behavioral inhibition (Logan, Cowan, & Davis, 1984), which posits that when go and stop processes are activated in close sequence, response inhibition depends on whether the stop process can overtake the

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go process. A slow reaction to the stop-signal results in a decreased likelihood that the

stop process will finish before the go process. Tracking versions of the task are therefore

designed to find the point at which the go and stop processes end at the same time, which

is defined as when the probability of inhibiting is at chance (i.e., 50%). Since SSRT is

not directly observable, it is inferred from the difference between the presentation of the

go- and stop-signals (i.e., SSD) and the average time required to process and produce a

go-signal response (i.e., MRT). For contemporary tracking versions of the task, SSRT is

therefore derived by subtracting mean SSD from MRT (SSRT = MRT - SSD; see Logan

et al., 1997). This relationship between SSRT, SSD, and MRT is depicted in Figure 4.
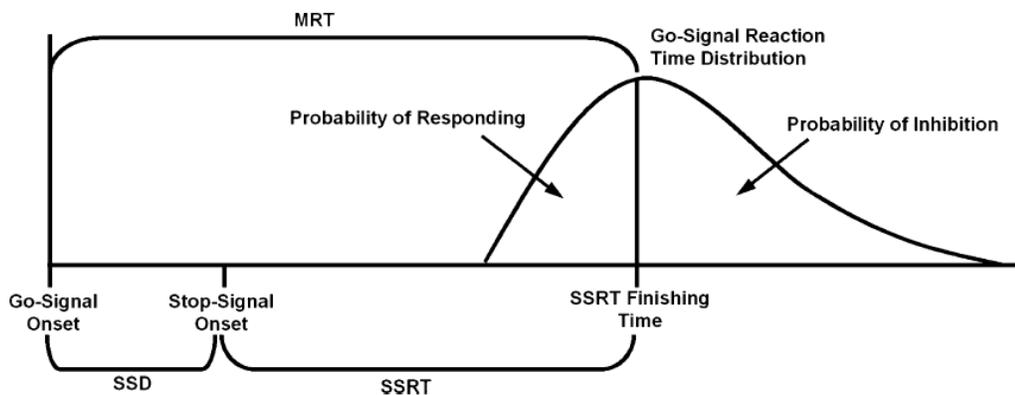
Figure 4. Relationship between mean reaction time (MRT), stop-signal delay
(SSD), and stop-signal reaction time (SSRT); SSRT = MRT - SSD (Alderson et
al., 2007).

SSRT and MRT in AD/HD and other groups. While calculating SSRT in this

manner provides a means of measuring an important process that cannot be directly

observed, there are also potential difficulties related to its interpretation. Group

differences in SSRT could be attributable to different combinations of outcomes for the

variables involved. For example, longer SSRT, which is thought to indicate poorer

response inhibition, could be due to similar MRT between groups and shorter SSD in one

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group. That is, each group required a similar amount of time to respond to the go-stimulus (MRT), but one of the groups required more warning to inhibit their response to the stop-signal (i.e., shorter SSD). This result would be consistent with behavioral inhibition model predictions, which posit that longer SSRT in AD/HD is more attributable to poor response inhibition. Chamberlain et al. (2006) found this type of relationship in individuals with obsessive-compulsive disorder (OCD) and trichotillomania compared to controls.
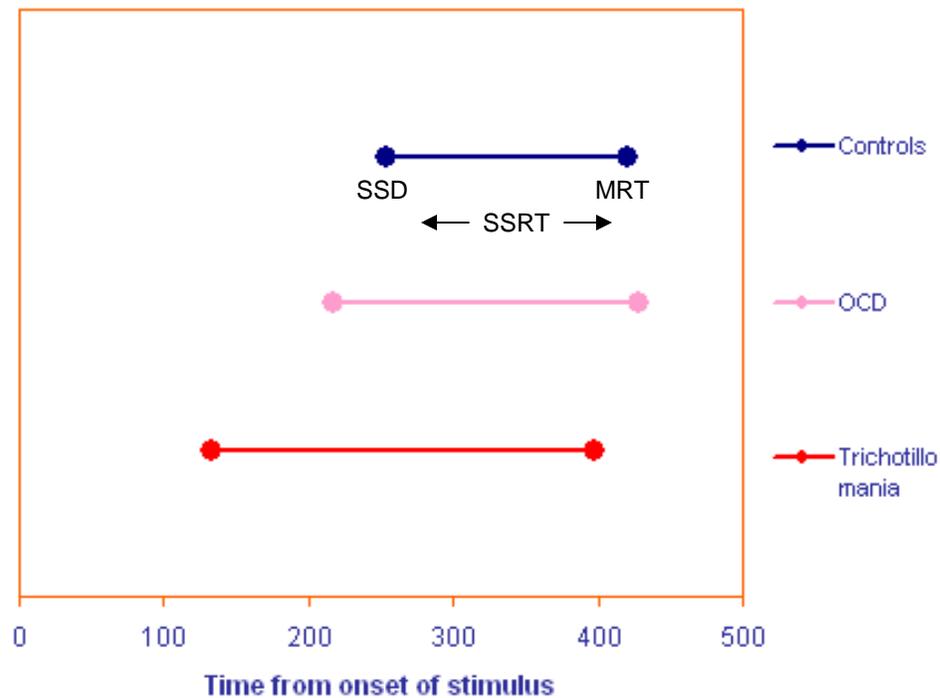
Figure 5. Stop-signal reaction time (SSRT) illustrated as the difference between stop-signal delay (SSD) and mean reaction time (MRT) in OCD, trichotillomania, and controls (Chamberlain et al., 2006).

As can be seen in Figure 5, longer SSRT in OCD and trichotillomania compared to controls is related to shorter SSD (i.e., requiring more warning to inhibit responses) for individuals with OCD and trichotillomania, whereas MRT is similar between groups. This pattern suggests that SSRT may index poor behavioral inhibition in these groups.

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Longer SSRT, however, may not always result from this specific relationship between SSD and MRT. Another possibility involves similar SSD between groups and slower MRT in one group. In this instance, each group requires a similar time interval to inhibit responses to the stop-signal (SSD), but one group requires more time to respond to the go-stimulus (MRT). This results in longer SSRT for the group with longer MRT; however, this pattern suggests that longer SSRT may not be due to poor behavioral inhibition, but rather to slower MRT, which is more consistent with state regulation predictions regarding longer SSRT in AD/HD.

Slower MRT as opposed to poor response inhibition has been suggested as a possible explanation for longer SSRT in children with AD/HD in two recent meta-analyses (Alderson et al., 2007; Lijffijt et al., 2005). Both reported significantly longer SSRT as well as slower MRT in children with AD/HD compared to controls; however, each study also reported nonsignificant group differences in estimated SSD in children. The group differences in SSRT were therefore attributed to attentional or cognitive processing deficits rather than deficient behavioral inhibition in children with AD/HD. It is also important to note, however, that Lijffijt and colleagues (2005) did find a significant group difference in estimated SSD in adults, suggesting that SSRT may represent different processes in different age groups. Alderson and colleagues (2007) did not include studies with adults in their review.

One major limitation to these meta-analyses is that none of the studies included in the analyses reported SSD. SSD was therefore estimated by pooling pooled standard deviations across both tracking and non-tracking versions of the Stop-Signal Task (Lijffijt et al., 2005) or algebraically calculating an estimated SSD metric for only 8

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studies using a tracking version of the task (Alderson et al., 2007). SSD has since been

directly examined in one study (Alderson et al., 2008), which reported a nonsignificant

group difference in SSD in a small sample (n = 23) of boys ages 8-12 with and without

AD/HD. Both meta-analyses and the recent study by Alderson et al. (2008) indicate the

need for reporting SSD in future Stop-Signal Task studies and for examining the

relationship between SSD, SSRT, and MRT in larger samples.

SSRT and other putative behavioral inhibition measures. While it has more

recently been suggested that longer SSRT may not represent poor inhibitory processes in

children with AD/HD, there are various other measures also thought to indicate inhibition

deficits in AD/HD. For instance, individuals with AD/HD have been found to show

poorer performance on measures of interference control, including more errors to

incongruent stimuli on flanker tasks (Carter, Krener, Chaderjian, Northcutt, & Wolfe,

1995; Jonkman et al., 1999). Poorer performance on the Stroop Interference task has also

been found (MacLeod & Prior, 1996; Reeve & Schandler, 2001), although results are

mixed (for a review, see van Mourik et al., 2005).

Aside from SSRT, a type of putative behavioral inhibition measure that more

consistently shows differences between AD/HD and control groups is continuous

performance tests (CPT) (for a review, see Losier et al., 1996). These tasks involve a

go/no-go procedure in which participants are instructed to respond to a specific target

sequence and withhold responding to non-target sequences over a period of several

minutes. CPTs vary with respect to factors such as the ratio of 'go' to 'no-go' stimuli,

the complexity and modality (i.e., visual versus auditory) of the target sequence, whether

the interstimulus interval is varied, and whether distractors are present during any of the

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task conditions. Some typical outcome measures include commission errors (i.e., responses to a sequence other than the target sequence), which are thought to reflect response inhibition and thus more consistent with inhibition theory assumptions, as well as omission errors (i.e., failing to respond to a target sequence), which are thought to provide a measure of vigilance and thus more consistent with state regulation assumptions. In a recent meta-analysis (Willcutt, Doyle et al., 2005), errors of commission on these tasks were shown to have an overall medium range effect size (d = .51), which is generally comparable to the effect size for SSRT (d = .61). Omission errors were also found to have a moderate effect size (d = .64).

Common versions of CPTs include the Conners' CPT (Conners, 1994), the Test of Variables of Attention (TOVA; Leark, Green, Kindschi, Dupuy, & Hughes, 2007), and CPT-AX tasks (Barch et al., 1997; Barch et al., 2001; Servan-Schreiber, Cohen, & Steingard, 1997). The current study examined a variant of a CPT-AX task, the Gordon CPT (G-CPT; Gordon, 1983). This is important because unlike other CPTs, which utilize a single target or non-target stimulus (e.g., respond when any letter besides 'X' appears, and do not respond when 'X' appears, as in the Conners' CPT), CPT-AX tasks have a multi-stimulus target sequence (e.g., respond only when the letter 'A' is followed by the letter 'X'; or when the number '1' is followed by the number '9,' as in the G-CPT). CPT-AX tasks such as the G-CPT thus incorporate a simple working memory component in which the stimulus must be maintained until the next stimulus appears.

As can be seen in Table 1, this type of task also allows for analysis of error *types* (Frank, Santamaria, O'Reilly, & Willcutt, 2007; Halperin, Wolf, Greenblatt, & Young, 1991). For instance, for the G-CPT in which the target sequence is a '1' followed by a

'9,' if an individual makes a response after the appearance of a '1' that is not followed by a '9' (X1X error; 'X' represents a number besides '1' or '9'), the error is thought to be attributed to difficulties inhibiting a prepotent go-response to the initial stimulus in the target sequence (Halperin, Wolf et al., 1991; Halperin et al., 1988). This type of "impulsive" error is most consistent with inhibition model assumptions regarding error performance in AD/HD. On the other hand, if an individual makes an error by responding to a number that comes after the correct sequence (i.e., a late response to the correct target sequence; 19X error), it is hypothesized that this error type represents a slow correct response rather than impulsivity (Halperin, Sharma, Greenblatt, & Schwartz, 1991). This type of "slow processing" error is therefore most consistent with state regulation assumptions of slower and more variable processing in AD/HD.

Table 1
G-CPT Error Types

Error Type	Description	Corresponding Theory
Overall Omissions	Inattentive	State Regulation
Overall Commissions	Poor inhibition	Executive Inhibition
X1X	Impulsive	Executive Inhibition
19X	Slow processing	State Regulation
Combined processes		
XX9	Failure to maintain context/impulsive	
XX1	Impulsive/random (depending on latency)	
X9X	Failure to maintain context/slow response	
XXX	Random (impulsive without relevant cues)	

Note. In the above sequences, 'X' represents any number that is not part of the target sequence (1 followed by 9).

Furthermore, some error types represent combinations of processes, which are less directly relatable to current theories of interest. More specifically, when an individual responds to a '9' that was not preceded by a '1' (XX9 error), it is reasoned that inattention (Halperin, Wolf et al., 1991), failure to maintain appropriate contextual

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information in working memory (Frank et al., 2007), or impulsive response to a stimulus cue contributed to the error. Other errors may involve failure to maintain context as well as slow response (e.g., '9' not preceded by a '1' and followed by another number; X9X error), "random" commission errors with no relevant stimulus cues (XXX error), or errors that may represent impulsivity or random errors depending on RT latency (i.e., '1' followed by an immediate response before another number is presented; XX1 errors) (Halperin, Wolf et al., 1991; Halperin et al., 1988). Despite presumed differences in underlying processes, all commission error types are generally included in a total commission error variable for CPT tasks, which may mask important patterns of performance between groups.

While commission error types have been examined in some studies with children (Halperin, Wolf et al., 1991; Halperin et al., 1988) and adults (Frank et al., 2007), studies have yet to incorporate analysis of commission error types with other putative measures of behavioral inhibition in the same study. Previous studies also have not examined the relationship between error types and specific theoretical approaches and whether the G-CPT is similarly reducible to state regulation versus inhibition processes.

Specific Aims and Hypotheses

The current study re-examined Stop-Signal Task RT variables and error types from the Gordon Continuous Performance Test (G-CPT) to test predictions of state regulation and executive inhibition theories of AD/HD, as well as to examine how these measures contribute to a multiple deficit model of the disorder, in a large sample of children and adolescents.

Specific aim 1.

Can increased SDRT be reduced to slow MRT in AD/HD? An in-depth examination of Stop-Signal Task go-trial response variability (i.e., SDRT) and its relationship to go-trial MRT, including ex-Gaussian distribution analyses, was conducted to test whether SDRT was reducible to overall slower RT or to more excessively slow RTs (i.e., "attentional lapses") in the tail of the AD/HD distribution. Increased variability as a distinct process that is separable from general RT slowing in AD/HD would provide support for state regulation assumptions emphasizing the importance of response variability in AD/HD. Based on extant research, it was hypothesized that increased SDRT in AD/HD would be related to a larger positive skew in the RT distribution of children with AD/HD (i.e., larger tau), rather than general RT slowing.

Specific aim 2.

Can Stop-Signal Task SSRT be reduced to slow MRT in AD/HD? The components of stop-signal reaction time (SSRT), including mean reaction time (MRT) and stop-signal delay (SSD; SSRT = MRT – SSD), were also thoroughly analyzed to assess whether SSRT represented a measure of response inhibition or was also reducible to MRT-related differences. A larger contribution of SSD to the putative SSRT group difference would suggest that SSRT was more related to poor response inhibition and thus more supportive of the executive inhibition theory. A larger contribution of MRT to SSRT, on the other hand, would suggest that SSRT was more attributable to state regulation processes rather than poor response inhibition in AD/HD. Similar to a recent study examining SSD in a small sample of boys (Alderson et al., 2008) and contrary to general assumptions regarding Stop-Signal Task SSRT, it was hypothesized that SSD

would not significantly differ between AD/HD and control groups, thus suggesting that longer SSRT in AD/HD was more reflective of state regulation rather than inhibition theory predictions.

Specific aim 3.

Can another putative inhibition measure also be reduced to slow processing? An additional presumed measure of response inhibition, the Gordon Continuous Performance Test (G-CPT), was also analyzed to further test competing inhibition and state regulation theory predictions. In contrast to anticipated results for SSRT components, it was hypothesized that G-CPT results would be more supportive of inhibition theory assumptions. More specifically, AD/HD participants would make proportionately more overall commission than omissions errors and more "impulsive" commissions than other commission error types.

Specific aim 4.

How do primary measures of interest contribute to a multiple deficit model of AD/HD? Overall, results of these analyses were therefore expected to provide support for both state regulation and executive inhibition theories of AD/HD depending on the specific task, and thus supportive of a multiple deficit approach to the general conceptualization of the disorder. Stop-Signal Task RT variables, as well as G-CPT commission error types, were therefore incorporated into multiple deficit analyses with various other tasks shown to differentiate AD/HD and control groups. It was hypothesized that these variables would represent distinct response variability and inhibition related constructs in exploratory factor analysis and contribute significantly to the prediction of AD/HD symptomatology in multiple regression analyses.

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Method

Participants

The present study is part of the ongoing Colorado Learning Disabilities Research Center (CLDRC) Twin Project (DeFries et al., 1997), in which 8 to 18 year-old twin pairs are recruited from school districts in the Front Range area to create population-based twin samples of children affected with AD/HD, reading disability, or both disorders, and children unaffected with either disorder. When possible, siblings of twin pairs who are 8 to 18 years of age and who meet inclusion criteria are also tested. Exclusionary criteria for participation in the CLDRC study include documented brain injury, significant hearing or visual impairment, or known genetic syndrome in either twin. Twin pairs are also excluded if one of the twins has received a diagnosis of an autism spectrum disorder, psychosis, bipolar disorder, or other pervasive developmental disorder. Participants with a Full Scale IQ score below 70 were also excluded from the current study.

For the current study, one twin or sibling was selected from each family to eliminate problems associated with observation independence. In order to maximize numbers of AD/HD participants for group comparisons in the current study, if only one twin or sibling in a family met diagnostic criteria for AD/HD (see *Measures* section below), that individual was included in the current sample. If more than one or no individuals met AD/HD criteria, one twin or sibling was randomly selected from that family. Additionally, individuals recruited into the clinical group at initial screening but

who did not meet AD/HD criteria during the study, as well as individuals with borderline AD/HD symptoms (defined as three to five symptoms of inattention or hyperactivity/ impulsivity), were excluded in group comparisons so as to approximate "clean" AD/HD and control groups. These individuals were included in analyses of continuous symptom dimensions in which the spectrum of AD/HD symptomatology, rather than dichotomous groups, was being examined.

Depending on the specific analysis, sample sizes varied due to the addition of different tasks at different points during the history of the CLDRC study. Specific sample sizes and characteristics are described in greater detail below.

Procedure

As part of the CLDRC study, twins complete a variety of neuropsychological and psychosocial measures at the University of Colorado at Boulder and the University of Denver. At the University of Denver, an e-prime tracking version of the Stop-Signal Task (referred to as the *Primary Stop-Signal Task* in the current study) is completed as part of the current battery. Participants completing testing since 2005 were administered this version, which allows for the collection of trial-by-trial RT data and is therefore suitable for fine-grained RT data screening procedures and ex-Gaussian distribution analyses. A sample of 189 participants (102 meeting AD/HD criteria and 87 controls) was included in analyses for the primary Stop-Signal Task version for the current study.

Individuals tested between 1998 and 2005 completed a tracking version with the same parameters as the primary version, but that only allowed for the collection of summary data statistics (referred to as the *Secondary Stop-Signal Task* in the current study). Examination of individual RT distributions and ex-Gaussian curve analyses were

therefore not possible with this version of the task. A sample of 298 participants (169 meeting AD/HD criteria and 129 controls) was included in analyses for the secondary Stop-Signal Task version for the current study.

The Gordon Continuous Performance Test (G-CPT) is also completed at the University of Denver and is part of the original task battery. It has thus been completed by a larger group of participants that represents a combination of individuals completing primary or secondary Stop-Signal Task versions (470 participants completing the G-CPT; 257 meeting AD/HD criteria and 213 controls).

Participants in the CLDRC study are also administered measures of general cognitive ability and other neuropsychological tasks relevant to AD/HD. In addition, parents and children complete a series of interviews and questionnaires regarding AD/HD and other psychosocial symptomatology. Parents also complete questionnaires regarding their children's medical and developmental histories, and teachers provide measures of school performance, attention, and behavior.

The battery is administered by doctoral students in psychology or advanced undergraduate students with experience working with children. Children receive $50 following each session for their participation and as incentive, receive additional rewards of up to $20 for completing computer-based tasks at the University of Denver. Parents receive $25 for completing questionnaires. The research protocols are approved by the Institutional Review Boards at each university.

Measures

　　AD/HD Rating Scale. The Attention-Deficit/Hyperactivity Disorder Rating Scale-IV (AD/HD-RS; DuPaul, Power, Anastopoulos, & Reid, 1998) is a questionnaire

that addresses DSM-IV criteria for AD/HD. It is administered to at least one parent and one teacher of each subject recruited for the study. In most instances, maternal reports are used in analyses, as more mothers are available to participate. Participants are diagnosed as having AD/HD if they are rated as having 6 or more inattentive symptoms, 6 or more hyperactive-impulsive symptoms, or 6 or more in both domains, rated by either a parent or teacher. Participants can be further classified as either AD/HD-Combined Type (AD/HD-C), AD/HD-Inattentive Type (AD/HD-I), or AD/HD-Hyperactive/ Impulsive Type (AD/HD-HI) in accordance with DSM diagnostic criteria based on symptom endorsements.

For primary analyses for the current project, participants meeting criteria for AD/HD-C and AD/HD-I subtypes were included, as the hyperactive/impulsive subtype has been shown to be less related to executive impairment (Bedard et al., 2003; Chhabildas, Pennington, & Willcutt, 2001; Schmitz et al., 2002). Data for included participants were analyzed without regard to subtype in order to maximize sample size and also because is has not been established that AD/HD-C and AD/HD-I subtypes differ in their performance on the Stop-Signal Task or other neuropsychological measures (Chhabildas et al., 2001; Nigg, Blaskey, Huang-Pollock, & Rappley, 2002; Nigg et al., 2005). Follow-up analyses included comparisons of AD/HD-C and AD/HD-I groups to examine whether results in the current study were qualified by these classifications.

Stop-Signal Task. The Stop-Signal Task (Logan, 1994) consists of go-stimuli (uppercase letters X and O) presented in the center of a computer screen for 1000ms each. Stimuli are preceded by a fixation point (i.e., dot) in the center of the screen for 500ms that serves as an indicator that a letter is about to appear. Participants are

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instructed to press keys corresponding to the X and O with either their right or left index finger as quickly as possible. Xs and Os appear in random order with equal frequency across task blocks.

The stop-signal consists of a brief auditory tone presented at different intervals following the go-stimuli. These intervals are called the stop-signal delay (SSD). Participants are instructed to inhibit pressing a key on trials in which a tone is presented. They are also instructed not to slow their responses to go-stimuli in anticipation of stop-signals. Stop-signals are presented on 25% of the experimental trials.

Tracking procedure. Stop-Signal Task measures for the current study incorporate a tracking procedure (e.g., Logan et al., 1997) in which the stop-signal delay (SSD) is initially set at 250ms. It is then adjusted based on the individual's performance on the previous stop-signal trial. If the participant successfully inhibits on a stop-signal trial, the SSD is increased by 50ms. If the participant fails to inhibit, the SSD is decreased by 50ms. This procedure is designed to approximate the SSD at which an individual's probability of inhibiting on a stop-signal trial is 50%.

Primary and secondary Stop-Signal Task versions. As noted previously, depending on when tested, participants for the current study either completed a more recent e-prime tracking version of the task that allows for the collection of trial-by-trial RT data (*Primary Stop-Signal Task*), or an earlier tracking version that did not incorporate trial-by-trial RT data (*Secondary Stop-Signal Task*). For both versions, participants initially completed one practice block consisting of 32 go-trials. Next, they were administered 1 practice and 6 experimental stop-signal blocks of 32 trials each (i.e.,

24 go-trials and 8 stop-signal trials per block). Across experimental blocks, a total of 144

go-trials and 48 stop-signal trials were presented for each version of the task.

Gordon Continuous Performance Test (G-CPT). The Gordon Continuous

Performance Test (G-CPT; Gordon, 1983) consists of single-digit numbers presented at

one-second intervals in the center of a computer screen. Participants are instructed to

watch the screen and press a button only when a particular sequence of numbers (1

followed by 9) appears. Two versions of the task are administered. One consists of a

single column of flashing numbers. The other has three columns of numbers flashing at

offset intervals. The two outer columns act as distractors, and participants are instructed

to only press the button when the specified sequence appears in the middle column. Each

version is 9 minutes long, and the target sequence appears 45 times out of a total of 540

numbers presented. Correct responses, omission errors (i.e., failing to detect the target

sequence), and commission errors (i.e., responding to a sequence other than the target

sequence), including specific types of commissions are recorded (see Table 1).

Additional measures. Participants are administered various additional

neuropsychological measures, which were incorporated into analyses assessing how

primary variables of interest fit into a multiple deficit approach to AD/HD. Detailed

descriptions of the task battery have been published previously (Shanahan et al., 2006;

Willcutt, Pennington et al., 2005); therefore, only a brief description is included here.

Measures are also grouped by associated constructs, which have also been defined in

previous studies (Shanahan et al., 2006; Willcutt, Pennington et al., 2005).

In addition to the Stop-Signal Task and G-CPT, which are comprised of variables

that putatively provide measures of the constructs of response inhibition (SSRT and G-

CPT commission errors), response variability (SDRT, ex-Gaussian tau), and vigilance (G-CPT omission errors), additional tasks include measures of motor processing speed, naming speed, and verbal working memory. Motor processing speed tasks involve nonverbal measures requiring speeded motor output and include Wechsler Intelligence Scale for Children, Third Edition (WISC-III) Coding and Symbol Search (Wechsler, 1991); Colorado Perceptual Speed (Decker, 1989); and the ETS Identical Pictures Test (French, Ekstrom, & Price, 1963). Naming speed tasks involve tests of verbal processing speed and include Rapid Automatized Naming of Colors, Numbers, Letters, and Pictures (Denckla & Rudel, 1974) and Stroop Test Word and Color naming, but not Interference, trials (Golden, 1978). Verbal working memory tasks involve repetition of simple verbal information with varying degrees of working memory load and include WISC-III Digit Span Backward (Wechsler, 1991); Nonword Repetition (Gathercole, Willis, Baddeley, & Emslie, 1994); Sentence Span (Kuntsi, Stevenson, Oosterlaan, & Sonuga-Barke, 2001; Siegel & Ryan, 1989); and Counting Span (Case, Kurland, & Goldberg, 1982; Kuntsi, Stevenson et al., 2001).

Primary Stop-Signal Task Data Preparation

      Group level for the primary Stop-Signal Task. As noted previously, 189

participants (102 meeting AD/HD criteria and 87 controls) were included in analyses for

the primary version of the Stop-Signal Task. Participants completing this measure were

first screened to determine whether they generally complied with the requirements of the

task. Similar to previous Stop-Signal Task studies (Rucklidge & Tannock, 2002;

Schachar, Mota, Logan, Tannock, & Klim, 2000), this included examining go-trial

accuracy, probability of inhibiting for stop-signal trials, and stop-signal reaction time

(SSRT) in each block. More specifically, if a participant's overall go-trial accuracy was

<66%, or if probability of inhibiting was <13% or >85%, the individual was excluded

from analyses because these values indicate poor adherence to task requirements and

would also yield questionable estimates of SSRT (Band, 1997). Participants' SSRT was

also examined, and those with SSRT <50ms in any block were investigated further. This

cutoff was chosen because SSRT <50ms is a magnitude that is not observed in normal

adults (Williams, Ponesse, Schachar, Logan, & Tannock, 1999), and is a common

screening criterion in other Stop-Signal Task studies (e.g., Rucklidge & Tannock, 2002;

Schachar et al., 2000). Similar to Schachar and colleagues (2000), participants with

SSRT <50ms in only one block and no additional data issues (i.e., accuracy and

probability of inhibiting within acceptable limits) were therefore included in analyses; however, the block with SSRT <50ms was excluded from the overall estimation of SSRT. Those with SSRT <50ms in multiple blocks were excluded altogether.

Based on the above criteria, a total of 12 participants (6.3%) were excluded from further analyses (7 from the AD/HD group and 5 from the control group). This is comparable to other studies using similar criteria (Rucklidge & Tannock, 2002; Schachar et al., 2000), in which approximately 5% of the total participants were excluded. Furthermore, of the 12 participants excluded for the current study, 6 (4 AD/HD, 2 control) participants were excluded for accuracy <66%; 3 (1 AD/HD, 2 control) were excluded for probability of inhibiting <13%; and 3 (2 AD/HD, 1 control) were excluded for SSRT <50ms in multiple blocks. The excluded participants did not differ from the remaining participants with respect to sex, age, reading ability, or inattentive or hyperactive/impulsive symptoms, but did differ on IQ, with excluded participants having lower Full Scale IQ (M = 95.38, SD = 10.71) than remaining participants (M = 108.78, SD = 12.97; t(187) = -3.63, p < .001).

Of the remaining 177 participants, 8 (4.5%) had only one block with SSRT <50ms and no additional data issues (3 AD/HD, 5 controls). These participants did not differ from the other participants in sex, age, reading performance, inattentive or hyperactive/impulsive symptoms, or IQ. Data were also examined for any extreme multivariate outliers (Tabachnick & Fidell, 2007), but it was not necessary to exclude any additional participants.

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Individual level for the primary Stop-Signal Task. After screening participants at the group level for general adherence to task requirements, all remaining participants' individual RT distributions were carefully visually examined to determine optimal cut-points for anticipatory and outlier trials.

Anticipatory trials. It is recommended that an absolute cut-off be used for rejecting fast RTs, which occur through anticipation (Ulrich & Miller, 1994). Luce (1986) has shown that for simple reaction time, the nondecision portion is at least 100ms in adults. Previous studies have therefore generally used absolute cut-offs ranging from 100ms to 200ms (e.g., Balota, Yap, Cortese, & Watson, 2008; Hervey et al., 2006; Schmiedek, Oberauer, Wilhelm, Suss, & Wittmann, 2007).

Visual inspection of distributions of correct go-trials for the current study revealed that 200ms was an appropriate cut-point for anticipatory trials for this sample. This threshold discarded RT trials that were clearly separated from the rest of the distribution without cutting into the distribution. An example of a distribution with anticipatory trials can be seen in Figure 6.

Using this criterion, an average of less than one RT per participant (0.35% of total go-trials) was excluded. The maximum number of discarded anticipatory go-trials, including correct and incorrect responses, was 8 (5.56% of total go-trials). While small for both groups, the percentage of discarded anticipatory trials was significantly larger for AD/HD participants ($M = 0.48\%$, $SD = 1.06\%$) than controls ($M = 0.19\%$, $SD = .60\%$; $t(150.7) = -2.24$, $p = .03$), with significantly more variance in the AD/HD than control group (Levene's test $F = 8.64$, $p = .004$). Reported t-statistics therefore reflect equal variances not assumed.
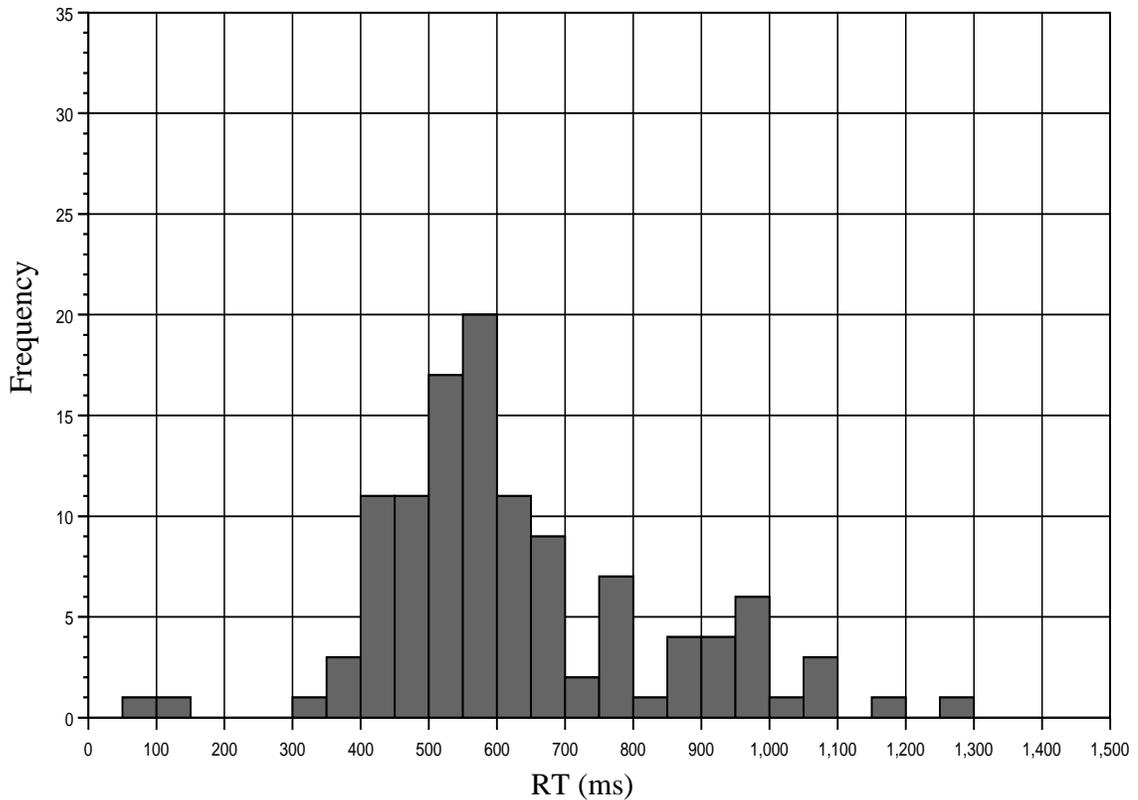
Figure 6. Example of an RT frequency distribution with anticipatory trials <200ms.

Outlier trials. RT distributions were also inspected for outliers in the tail. Visual

examination showed that several participants had trials that were clearly separated from

the rest of the distribution. Figure 7 provides an example of a subject with "too slow"

outlier trials that are clearly separated from the distribution.

Previous studies that have incorporated data trimming in the tail of the

distribution have generally discarded trials that were a certain standard deviation beyond

an individual's mean reaction time (MRT). This has ranged from 2.5SD to 4SD (e.g.,

Balota et al., 2008; Schmiedek et al., 2007). Using an absolute cut-off was also

considered; however, after inspecting numerous distributions, it was apparent that this

approach would only exclude outlier trials for some participants and would not exclude
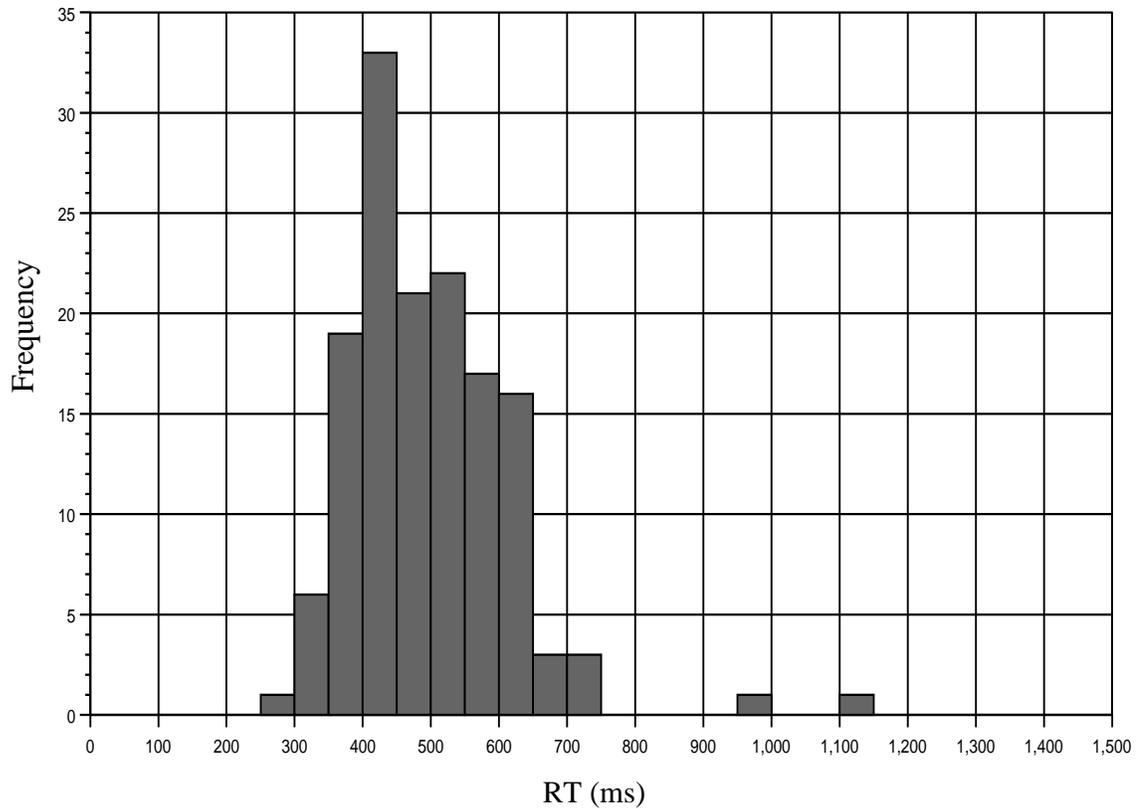
Figure 7. Example of an RT frequency distribution with outlier trials in the tail.

clear outlier trials for participants with a faster MRT. In other words, for a participant

with a relatively fast MRT, an RT of 1100ms may lie outside of the distribution (see

Figure 7 for an example); however, for an individual with a slower MRT, 1100ms may

clearly be a part of the distribution (Figure 8).

Based on these observations, a cut-off of 4SD beyond on individual's MRT was

used for this dataset. This provided the best compromise between excluding RTs that

were clearly separated from an individual's distribution, yet not cutting into the tail of the

distribution. Similar to anticipatory trials, an average of less than one RT trial per

participant (0.12% of total correct go-trials) was excluded. The maximum number of

excluded outlier trials was 2 (1.47% of total correct go-trials). In contrast to anticipatory

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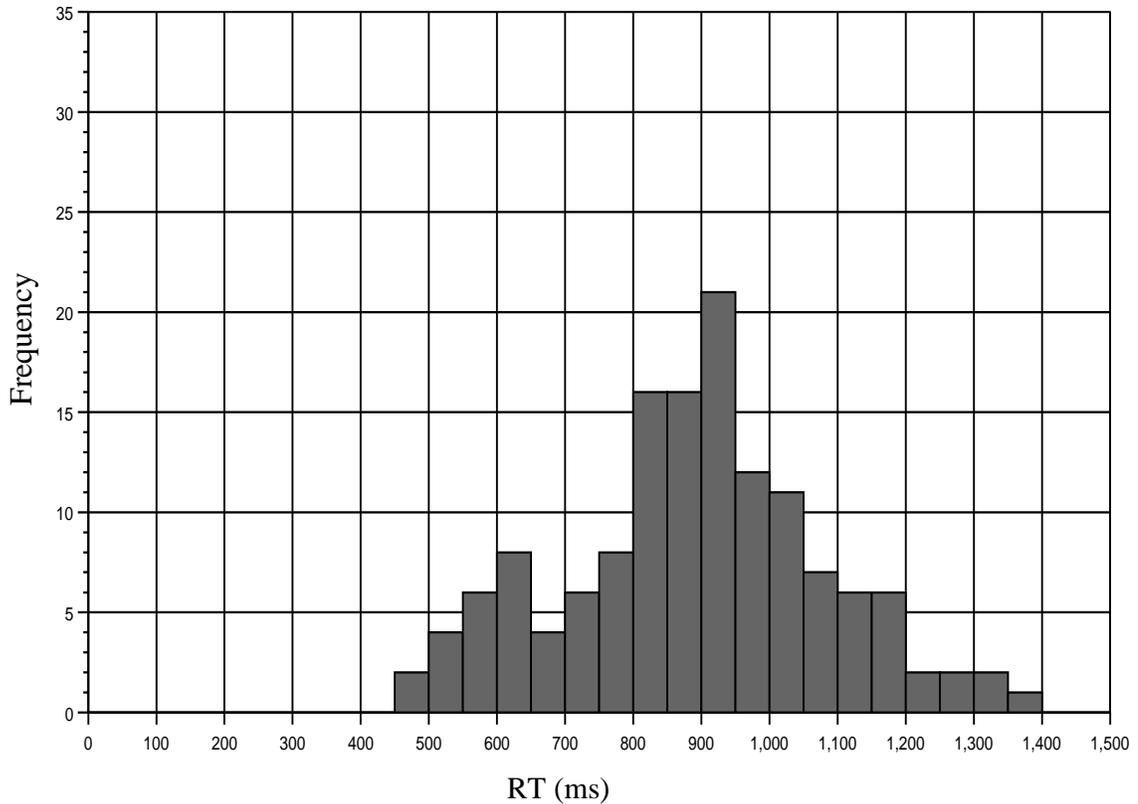
Figure 8. Example of an RT frequency distribution with slower MRT and no outlier trials.

trials, however, there was a higher percentage of excluded outlier trials for controls (M = 0.19%, SD = .38%) as compared to AD/HD participants (M = 0.06%, SD = .25%; t(137.0) = 2.58, p = .011), with significantly more variance in the control group than the AD/HD group (Levene's test F = 26.68, p < .001). Reported t-statistics therefore reflect equal variances not assumed.

After censoring anticipatory and outlier trials, there was an average of 133.8 (of 144 possible) correct go-trial RTs available per individual, with a minimum of 97 and a maximum of 144 trials.

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RT slowing across blocks for the primary Stop-Signal Task. An additional

issue to consider at the participant level is that an individual's RT may slow across trial

blocks, indicating waiting for the stop-signal when making go-trial responses. Including

participants who demonstrate this slowing pattern may influence results by artificially

inflating MRT estimates for a particular group and thus affecting the calculation of

SSRT. Individual RT distributions were therefore also examined for slowing.

Visual inspection of block-by-block RT distributions revealed that minor slowing

appears to occur for most individuals across the 6 test blocks, and only a couple of

individuals seemed to slow substantially across blocks. To more systematically

determine whether an individual should be excluded from analyses due to RT slowing, a

difference score between the MRTs for test blocks 6 and 1 was created for each subject.

This score was then standardized based on the overall group mean and SD of the

difference score. Any participants who were outliers on this variable (defined as $\geq$ 3SD

beyond the group mean) were to be further examined to determine their specific pattern

of slowing. If slowing primarily occurred during the final blocks, then RT variables for

that participant were to be based on the initial trial blocks in which significant slowing

did not occur. If slowing took place across the entire span of blocks, then the participant

would be excluded from further analyses.

One participant from the AD/HD group met this criterion. As can be seen in

Figure 9, this individual's MRT clearly slowed across all test blocks, indicating a change

in approach to the task. This individual was also an outlier when comparing test block 1

to earlier test blocks, further confirming that slowing occurred across all blocks rather

than only the final blocks. This participant was therefore excluded from further analyses.

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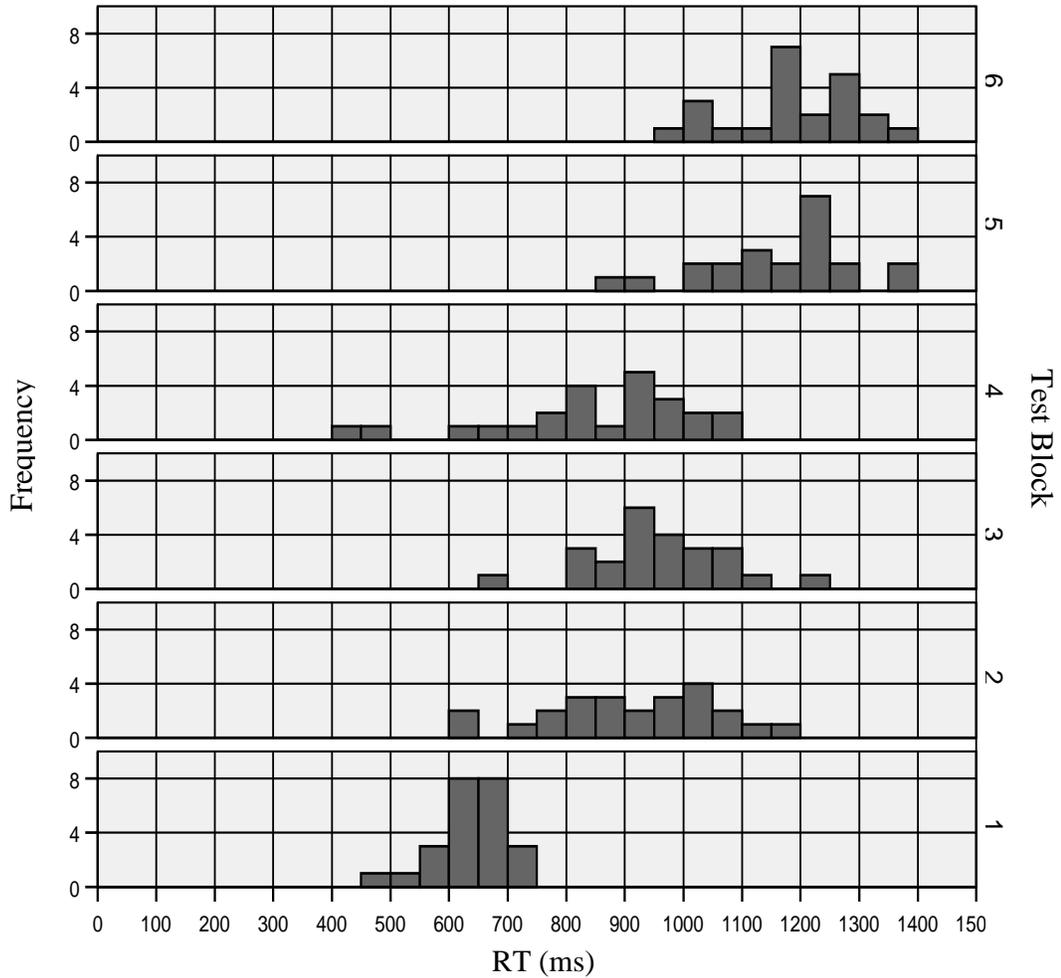
Figure 9. RT frequency distribution showing notable MRT slowing across test blocks.

In addition to comparing test blocks, difference scores were also created between practice block MRT (simple choice RT) and MRT from test blocks 1 and 6. This was done to examine whether any participants slowed more than expected when transitioning from a simple choice RT task to the Stop-Signal Task. Using the same criteria described above, no individuals were outliers on the practice MRT and test block 1 MRT difference score, indicating that there were not any participants who drastically slowed their MRT more than others when stop-signal trials were introduced. For the practice MRT and test

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block 6 MRT difference score, results were similar to those for test blocks 1 and 6 MRT

difference score and only indicated the same participant described above as an outlier.

Final sample characteristics for the primary Stop-Signal Task. After

completing the above data screening procedures, the final sample to be used in the

primary Stop-Signal Task analyses included 176 participants (13 total were excluded, 8

from the AD/HD group and 5 controls). Of the 176 participants, 94 were in the AD/HD

group and 82 were in the control group. Sample characteristics are reported in Table 2.

Table 2
Sample Characteristics for Primary Stop-Signal Task Analyses

	AD/HD Group (n=94)	Control Group (n=82)	Statistic
Age	12.08(2.76)	12.27(3.13)	$t(174)=0.43$, *n.s.*
Full Scale IQ	104.19(12.13)	114.04(11.93)	$t(174)=5.41$, ***p<.001***
AD/HD-I symptoms	7.07(2.15)	.33(.74)	$t(117.3)=-28.53$, ***p<.001******
AD/HD-H/I symptoms	3.98(2.90)	.29(.73)	$t(106.3)=-11.92$, ***p<.001******
Word Recognition[1]	-0.62(1.38)	0.24(1.10)	$t(172.8)=4.58$, ***p<.001******
Parental Education[2]	15.20(2.45)	16.28(1.71)	$t(157.6)=3.34$, ***p=.001******
Sex	69.1% male	46.3% male	$\chi^2(1, n=176)=9.39$, ***p=.002***
Ethnicity	77.8% Caucasian	86.6% Caucasian	$\chi^2(1, n=172)=2.25$, *n.s.*

Note. AD/HD-I = Attention-Deficit/Hyperactivity Disorder Inattentive symptoms; H/I = Hyperactive/Impulsive symptoms. [1]Standardized relative to overall sample mean and SD. [2]Mean of mother and father years of education. *Statistics represent equal variances not assumed due to significant Levene's test for equality of variances.

Similar to previous research (e.g., Barkley, 1997; Lahey et al., 1998), groups for

the current sample differed with respect to sex, IQ, AD/HD symptoms, and reading

ability, with the AD/HD group having significantly more boys, lower full scale IQ, more

inattentive and hyperactive/impulsive symptoms, and lower reading ability scores than

the control group. Average years of parental education were also lower in the AD/HD

group. Groups did not differ significantly with respect to age or ethnicity.

Secondary Stop-Signal Task Data Preparation

　　Group level for the secondary Stop-Signal Task. As described previously, 298

participants (169 meeting AD/HD criteria and 129 controls) were included in analyses for

the secondary version of the Stop-Signal Task, which includes the same task parameters

as the primary version but did not allow for collection of trial-by-trial RT data. Since the

secondary Stop-Signal Task does not include trial-by-trial RT data, participants could

only be screened for general compliance with Stop-Signal Task requirements at the group

level. Similar to the primary Stop-Signal Task data screening, this involved excluding

individuals with overall go-trial accuracy <66% and probability of inhibiting <13% or

>85%. Of the 298 participants who completed this version of the task, a total of 19

participants (6.4%) were excluded from further analyses (16 from the AD/HD group and

3 from the control group). Specifically, of these 19, 9 (8 AD/HD, 1 control) participants

were excluded for overall accuracy <66%; 6 (5 AD/HD, 1 control) were excluded for

probability of inhibiting <13%; and 4 (3 AD/HD, 1 control) were excluded for accuracy

<66% and probability of inhibiting <13%.

　　Since only summary variables were available, participants could not be screened

for SSRT <50ms in each block, as was done for the primary Stop-Signal Task. Data was

therefore screened for overall SSRT <50ms. No additional participants met this

exclusion criterion. Data were also similarly examined for any extreme multivariate

outliers (Tabachnick & Fidell, 2007), but it was not necessary to exclude any additional

participants.

　　The overall percentage of excluded participants for the secondary Stop-Signal

Task was virtually the same as that for the primary Stop-Signal Task (6.4% and 6.3%,

respectively). By group, 9.5% of AD/HD participants were excluded from the AD/HD group and 2.5% of controls were excluded from the control group for the secondary version of the task, compared with 7.8% of AD/HD participants and 5.8% of control participants for the primary version. Similar to the primary Stop-Signal Task, participants excluded from the secondary Stop-Signal Task did not differ from the remaining secondary version participants with respect to sex or reading ability, although did differ with respect to Full Scale IQ. Excluded participants had lower Full Scale IQ (M = 98.8, SD = 13.3) than remaining participants (M = 108.2, SD = 12.8; t(296) = 3.08, p = .002).

Additionally, in contrast to the primary Stop-Signal Task version, excluded participants from the secondary version also differed from remaining participants with respect to age. Excluded participants were younger on average (M = 9.7 years, SD = 1.8) than remaining participants (M = 11.6 years, SD = 2.6; t(23.7) = 4.35, p < .001). There was significantly more variance in the group of remaining participants (Levene's test F = 8.79, p = .003), so reported t-statistics are with equal variances not assumed. AD/HD symptomatology also differed, with the excluded participants having more inattentive (M = 7.0, SD = 3.1) and hyperactive/impulsive (M = 5.3, SD = 3.5) symptoms than the remaining participants inattentive (M = 4.0, SD = 3.7; t(21.8) = -4.1, p < .001) and hyperactive/impulsive (M = 2.3, SD = 2.8; t(296) = -4.4, p < .001) symptoms. Statistics for inattentive symptoms are for equal variances not assumed due to significant Levene's test (F = 23.4, p < .001).

Final sample characteristics for the secondary Stop-Signal Task. After completing the above data preparation, the final sample to be used in the secondary Stop-Signal Task analyses included 279 participants. Of those, 153 were in the AD/HD group

and 126 were in the control group. Similar to the sample used for the primary Stop-Signal Task analyses (see Table 2), the AD/HD and control groups differed with respect to sex, IQ, AD/HD symptoms, reading ability, and years of parental education, with the AD/HD group having significantly more boys, lower full scale IQ, more inattentive and hyperactive/impulsive symptoms, lower reading ability scores, and fewer years of parental education than the control group. Age and ethnicity again did not differ significantly between groups.

G-CPT Data Preparation

For the Gordon Continuous Performance Test (G-CPT), all participants with data for both the vigilance and distractibility conditions were included, and errors were combined across conditions for analyses. As noted previously, 470 participants were included in G-CPT analyses, with 257 participants in the AD/HD group and 213 in the control group. This sample also represents a combination of participants completing either primary or secondary versions of the Stop-Signal Task, as the G-CPT has remained the same during the ongoing history of the CLDRC study.

Participants completing the G-CPT were not excluded based on overall task performance for several reasons. Specifically, the G-CPT is a relatively simple task, and participants were only allowed to proceed to test conditions after demonstrating understanding of task parameters in practice sessions. G-CPT errors are also the variables of interest for this measure, and previous CPT studies have generally not excluded participants based on overall performance, with the exception of lack of task completion or failure to respond to any target sequences (Conners, Epstein, Angold, & Klaric, 2003; Mayes, Calhoun, & Crowell, 2001). All individuals who completed the G-

CPT for the current study made responses to target sequences. G-CPT data was therefore managed as described in the *Additional Data Preparation* section below.

As expected, demographics for the overall G-CPT sample were similar to those for the primary and secondary Stop-Signal Task versions (see Table 2).

Additional Data Preparation

Since it is expected that performance on RT and other variables will be correlated with age (Bedard et al., 2002; Dempster, 1981; Luna, Garver, Urban, Lazar, & Sweeney, 2004), age-adjusted scores were created for Stop-Signal Task and G-CPT variables, as well as other neuropsychological measures utilized in multiple deficit analyses (i.e., processing speed and working memory tasks). This was accomplished by regressing each variable onto age and age-squared and saving the standardized residual scores.

To account for outliers, scores for each measure falling more than three standard deviations beyond the mean of the overall sample were adjusted to this score. All variables of interest did not exceed greater than 2% outliers, and the majority of variables had less than 1% outliers. Following these adjustments, the distributions of each variable were examined for deviation from normality, and variables with excessive skewness or kurtosis (greater than ± 2 for either) were logarithmically transformed to approximate the normal distribution (Tabachnick & Fidell, 2007). The only variables requiring transformation to meet these criteria were G-CPT commission error types due to relatively low occurrence rates for specific error types. All commission error variables were within ± 2 for both skewness and kurtosis following logarithmic transformation.

Follow-up Analyses

   Effects of different RT data screening procedures. Since studies to date have employed a wide range of screening criteria, the effects of the different data screening procedures used in the current study were also examined. Specifically, the 13 participants excluded from the primary Stop-Signal Task and 19 participants excluded from the secondary task version due to poor adherence to task parameters were included in respective follow-up analyses. For the primary Stop-Signal Task, variations in individual RT trial data trimming criteria were also examined. This included screening of outlier trials in the tail of the distribution that were 2.5SD beyond an individual's MRT (2.53 discarded trials on average per individual (1.89% of total go-trails); no significant differences between AD/HD and control groups), which is the most conservative screening of outlier trials in the current literature (Balota et al., 2008). A condition that includes all outlier trials, as well as a condition without RT data trimming of any kind (i.e., includes anticipatory and outlier trials) were also examined.

   Group differences in Full Scale IQ. As was found in the current study, group differences in IQ are a common finding in the AD/HD literature (for a review, see Frazier, Demaree, & Youngstrom, 2004). Based on this association, some researchers suggest that IQ should be statistically controlled to make certain that neuropsychological deficits are not more parsimoniously explained by group differences in IQ (Lahey et al., 1998; Werry, Elkind, & Reeves, 1987). Others suggest, however, that AD/HD may cause an individual to perform poorly on measures of intelligence (Barkley, 1997), in which case controlling for IQ would remove variance associated with AD/HD. Some recent research also suggests that IQ should not be used as a covariate in any analyses

investigating underlying deficits in AD/HD or other populations (Dennis et al., 2009).

Given that these issues have not been entirely resolved, initial analyses were run without

controlling for IQ. Follow-up analyses included Full Scale IQ as a covariate for primary

findings. This was particularly relevant for Stop-Signal Task variables, since participants

excluded for poor task adherence were shown to have lower Full Scale IQ than remaining

participants on both versions of the task.

AD/HD subtypes. As noted previously, prior studies assessing

neuropsychological functioning in AD/HD subtypes have generally found few

distinctions between inattentive (AD/HD-I) and combined (AD/HD-C) subtypes

(Chhabildas et al., 2001; Nigg et al., 2002). A recent study examining response

variability in various RT tasks in AD/HD also did not find significant differences

between subtypes (Epstein et al., 2011). Participants meeting criteria for AD/HD were

therefore combined into a single AD/HD group for initial analyses. The AD/HD-I and

AD/HD-C subtypes were then compared to each other for primary findings to assess

whether these subtypes also performed similarly in the current study.

Age effects. It is well established that RT becomes faster throughout childhood

and adolescence across a range of tasks and that this relationship is nonlinear such that

there is a rapid decline in RT during childhood and slower change during adolescence

(Kail, 1991; Luna et al., 2004; Miller & Vernon, 1997; Weiler, Forbes, Kirkwood, &

Waber, 2003). Similar changes have been documented in the Stop-Signal Task

paradigm, with studies showing that RT to go-trials becomes faster from early childhood

through adolescence (Bedard et al., 2002; Williams et al., 1999). SSRT showed a similar

declining pattern across childhood and adolescence in these studies, although the

differences in age groups generally did not reach statistical significance. As noted

previously, age-related changes are also observed in other neuropsychological measures

(Dempster, 1981; Luna et al., 2004).

Given the wide age-range utilized in the current study (ages 8 to 18), it is

important to take potential age-related effects into account. Age-adjusted scores were

therefore created prior to analyses (see *Additional Data Preparation* section above), and

initial analyses were run with the overall sample. While age-adjusted scores account for

some age-related changes in performance on measures, it is also possible that age-related

patterns may be missed when combining across a wide age-range. Follow-up analyses

therefore addressed age as a continuous covariate in regression analyses with group

predicting primary variables of interest. Age groups based on the middle childhood (ages

8-12) and adolescence (ages 13-18) groups used in the Williams et al. (1999) and Bedard

et al. (2002) studies were also examined in 2 x 2 ANOVAs with group status as the other

independent variable and primary findings as dependent measures in order to assess

potential age-group related effects on results.

Sex-related differences. As noted previously, prior studies document that boys

are more likely to meet AD/HD criteria than girls (Costello, Mustillo, Erkanli, Keeler, &

Angold, 2003). While some studies of response inhibition and other executive function

tasks have not found significant differences related to sex (Houghton et al., 1999;

Rucklidge & Tannock, 2002), others suggest that performance may vary somewhat by

sex (Hartung et al., 2002; Nigg, 1999; Nigg et al., 2002). Additionally, regarding the

Stop-Signal Task, Nigg (1999) found that while a group by sex interaction was not

significant for SSRT, the effect size for SSRT was larger for girls than for boys

suggesting potential sex-related differences in Stop-Signal Task performance.
Furthermore, to date there has been only one direct study of Stop-Signal Task SSD
differences in AD/HD in a small sample of boys (Alderson et al., 2008). Given prior
research indicating potential sex-related differences in task performance in AD/HD, as
well as a lack of studies assessing SSD in both boys and girls, sex will be included in
follow-up analyses in a 2 x 2 ANOVA with group as the other independent variable and
primary findings as dependent measures.

        Comorbidity of AD/HD and other externalizing disorders. Another important
consideration is the potential effects of comorbidity of AD/HD and other disorders.
Specifically, disruptive behavior disorders including oppositional defiant disorder (ODD)
and conduct disorder (CD) have been found to frequently co-occur with AD/HD (Angold,
Costello, & Erkanli, 1999), and of particular relevance to the current study, poorer
performance on response inhibition tasks has also been found in ODD and CD (Milich,
Hartung, Martin, & Haigler, 1994; Oosterlaan et al., 1998).

        Given these findings, some researchers have statistically controlled for symptoms
of comorbid ODD and CD to examine whether deficits associated with AD/HD can be
explained by these associated disorders (Barkley, Edwards, Laneri, Fletcher, & Metevia,
2001; Nigg, Hinshaw, Carte, & Treuting, 1998; Oosterlaan, Scheres, & Sergeant, 2005).
In general, prior studies have found that group differences between AD/HD and control
participants remained significant after controlling for symptoms of ODD and CD, and a
meta-analysis of the Stop-Signal Task found that comorbid ODD and CD symptoms did
not significantly affect results (Lijffijt et al., 2005). Examination of G-CPT performance

in AD/HD and comorbid ODD and other symptoms including anxiety and depression revealed similar results (Mayes, Calhoun, Chase, Mink, & Stagg, 2009).

While these findings suggest that ODD and CD symptomatology are unlikely to affect results in the current study, given the association of ODD and CD with response inhibition measures in some studies and the inclusion of less commonly examined RT and G-CPT variables in the current study, follow-up analyses addressed whether primary results were qualified by co-occurring symptoms of ODD and CD.

For the current sample, parent ratings of ODD and CD symptoms were assessed with the DSM-III-R Diagnostic Interview for Children and Adolescents (DICA; Reich & Welner, 1988), and teacher ratings of ODD were assessed with the SNAP-IV (Swanson et al., 2001). ODD and CD symptoms were included as covariates in follow-up analyses with group predicting primary findings.

Results

Main Analyses

To recapitulate, the main hypotheses of this study involve testing competing theories of AD/HD by examining the Stop-Signal Task and another putative measure of behavioral inhibition, the Gordon Continuous Performance Test (G-CPT). The theories being tested are the inhibition and state regulation theories, as they make either explicit or implied competing predictions regarding results from these measures. A summary of the primary variables of interest and the corresponding hypothesized results for the inhibition and state regulation theories are presented in Table 3.

Table 3
Predicted Primary Results by Theory for AD/HD (A) Compared to Controls (C)

	Inhibition	State Regulation		Inhibition	State Regulation
Stop Task			G-CPT Errors		
SSRT	A>C	A>C	Omissions	--	**A>C**
MRT	**A=C**	**A>C**	Overall Commissions	**A>C**	--
SSD	**A<C**	**A=C**	X1X "Impulsive"	**A>C**	--
SDRT	--	A>C	19X "Slow Processing"	--	**A>C**
Mu	A=C	A=C			
Sigma	A=C	A>C			
Tau	A=C	A>C			

Note. Bolded items represent competing predictions and other primary comparisons of interest between theories. Abbreviations for Stop-Signal Task variables are as follows: SSRT = Stop-Signal Reaction Time; MRT = Mean Reaction Time; SSD = Stop-Signal Delay; SDRT = Standard Deviation of Reaction Time; Mu = ex-Gaussian mean of the normal component of the RT distribution; Tau = ex-Gaussian mean of the exponential (i.e., "tail") of the RT distribution. Abbreviations for Gordon Continuous Performance Test (G-CPT) variables are as follows: X1X error = initial target number (i.e., 1) followed by a non-target number; 19X error = correct target sequence (i.e., 1, 9) with response occurring after the presentation of another number.

As described previously, SSRT is calculated by the following formula: $SSRT = MRT - SSD$. As can be seen in Table 3, the inhibition theory predicts that participants with AD/HD will have longer SSRT compared to controls due primarily to shorter SSD in the AD/HD group (i.e., needing more warning to inhibit responses to the stop-signal than controls) and that MRT will be more similar between groups. In contrast, the state regulation theory posits that MRT-related differences will make a more notable contribution to SSRT group differences and that SSD will be more similar between groups (i.e., the AD/HD group will not need significantly more warning to inhibit responses to the stop-signal). Thus, while both SSD and MRT may show differences, the relative contributions of these variables to putative differences in SSRT will provide key comparisons of the competing theories. Namely, a larger SSD group difference that makes a more notable contribution to SSRT than MRT will be supportive of inhibition model predictions. In contrast, SSD being more similar between groups and making a relatively smaller contribution than MRT to SSRT differences will provide support for state regulation assumptions.

Additional predictions are also made regarding other RT-related variables. Specifically, the state regulation theory predicts greater response variability (i.e., SDRT) in AD/HD as a key finding and that greater SDRT is not just a by-product of general RT slowing. A further implication of this theory is that greater SDRT, and thus longer MRT, is due in part to more excessively long RTs in the tail of the distribution in the AD/HD group. If this implication is supported, ex-Gaussian distribution results are expected to show similar mu (i.e., ex-Gaussian mean of the normal part of the RT distribution) between groups and larger tau (i.e., ex-Gaussian mean of the "tail" of the RT distribution)

in the AD/HD versus control group. Larger sigma in the AD/HD group (i.e., ex-Gaussian standard deviation of the normal part of the RT distribution) is also implied by the state regulation theory as this would indicate increased variability across the normal part of the distribution in addition to variability in the tail. With respect to the inhibition theory, while it does not make specific predictions regarding response variability, in its simplest form it does not indicate differing shapes of RT distributions between groups and so assumes that ex-Gaussian parameters would be similar in AD/HD and control groups.

Regarding the G-CPT, the theories make competing predictions concerning the relative importance of particular error types on this task. Specifically, the inhibition theory predicts that AD/HD participants will make more commission errors compared to controls due to difficulties inhibiting responses. The state regulation theory, on the other hand, emphasizes the importance of greater omission errors in the AD/HD group due to poorer maintenance of optimal arousal during the G-CPT. Each theory also makes implied predictions regarding the importance of specific commission error types allowing for more in depth analysis of which theory is best supported by results form this measure. In particular, the inhibition model predicts more "impulsive" errors (i.e., X1X) due to problems inhibiting the prepotent "go" response to presentation of a 1 that is not followed by a 9. The state regulation theory emphasizes more "slow processing" errors (i.e., 19X), indicating slower and more variable responding to correct target sequences.

The following analyses test the aforementioned predictions and are organized into four sections: (1) primary Stop-Signal Task (includes trial-by-trial RT data) analyses, including ex-Gaussian distribution analysis; (2) secondary Stop-Signal Task (does not include trial-by-trial RT data) analyses; (3) analyses of G-CPT error types; (4) analyses

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examining the relationships between the Stop-Signal Task, G-CPT, and other

neuropsychological variables thought to be predictive of AD/HD symptomatology.

Primary Stop-Signal Task Results

Analysis of choice reaction time in the practice block. The initial practice

block, which does not include stop-signal trials, was analyzed first. This was to test

whether the current sample showed expected group differences in MRT and SDRT on a

brief measure of choice RT. As anticipated, groups differed with respect to MRT and

SDRT, with the AD/HD group having significantly slower MRT ($M = .06$, $SD = .95$; raw

mean = 502.5ms) compared to controls ($M = -.25$, $SD = .88$; raw mean = 481.0ms; $t(174)$

$= -2.25$, $p = .03$) and larger SDRT ($M = .08$, $SD = 1.01$; raw mean = 115.5ms) compared

to controls ($M = -.20$, $SD = .88$; raw mean = 105.8ms; $t(174) = -1.97$, $p = .05$). Small

effect sizes were observed for both, with Cohen's $d = .34$ for MRT and $d = .30$ for SDRT

(Cohen, 1988). Effect sizes for MRT and SDRT were more similar than observed in

some previous studies, which have shown a larger effect size for SDRT than MRT (Klein

et al., 2006; Leth-Steensen et al., 2000; Lijffijt et al., 2005). This is thought to be

attributable to the relatively small number of observations available in the practice block

for the current study ($M = 29.4$). In general, however, results are comparable to those

found in other studies of choice RT in AD/HD (Leth-Steensen et al., 2000; Teicher et al.,

1996) and indicate that the current sample is performing as expected on the initial portion

of the primary Stop-Signal Task.

Analyses of summary statistics in the primary Stop-Signal Task.

T-tests. To assess validity of the primary version of this task, groups were first

compared on probability of inhibiting to the stop-signal. Importantly, probability of

inhibiting was similar for the AD/HD and control groups (Table 4), indicating that the

stop-signal tracking procedure was successful and resulted in each group inhibiting close

to 50% of the time, on average. This finding indicates that the stop-signal tracking

algorithm performed as anticipated for the primary version of the task and also lends

support to the validity of the following primary Stop-Signal Task results.

Groups were then compared on summary statistics for correct go-trials. As can be

seen in Table 4, the AD/HD group was significantly more variable than the control group

and demonstrated a medium-range effect size for SDRT (d = .49). Furthermore, as for

most variables tested, Levene's test for equality of variances was nonsignificant for

SDRT (F = 1.22, p = .271). This suggests that the group difference in SDRT was not

primarily driven by more or less variance in the AD/HD compared to control group, such

as by a small group of AD/HD participants with extreme SDRT values. Visual

inspection of group distributions of SDRT was also supportive of this finding.

Table 4
Summary Statistics for the Primary Stop-Signal Task

	AD/HD (n=94) Mean(SD)[raw]	Control (n=82) Mean(SD)[raw]	t	p	Cohen's d
Prob. Of Inhib.	-.15(1.11)[52.1%]	.04(.96)[53.2%]	1.24	.22	.19
SDRT	.24(.89)[175.2ms]	-.23(1.04)[156.2ms]	-3.25	**.001**	.49
Mean RT (MRT)	.09(.90)[667.4ms]	-.17(1.0)[638.4ms]	-1.75	.08	.26
Median RT	.08(.94)[647.6ms]	-.14(.98)[620.9ms]	-1.51	.13	.23

Note. Values reflect means and standard deviations for age-standardized variables. Raw
RT means (ms) are in brackets for comparison, although were not used in calculating test
statistics or effect sizes.

Additionally, repeated measures analysis of SDRT across the six test blocks was

examined to assess whether the group difference in variability differed as a function of

test block. Results of the 2 x 6 mixed model ANOVA in which group was the between-

subjects factor and SDRT by block was the within-subjects factor did not indicate a

significant interaction between group and test block ($F(5,870) = 1.27$, $p = .285$). Thus, while variability differed between blocks ($F(5,870) = 5.61$, $p < .001$) and by groups overall ($F(1,174) = 10.23$, $p = .002$), the AD/HD group did not demonstrate more variable performance than the control group in either earlier or later blocks across the task.

For MRT, the AD/HD group's raw value was slower than the control group's; however, this difference did not reach statistical significance in this sample and was instead at a trend level ($p = .08$), with a small effect size ($d = .26$) consistent with meta-analysis of Stop-Signal Task MRT ($d = .29$; Lijffijt et al., 2005). Median RT was also calculated to examine whether AD/HD participants remained slower on a measure of central tendency that is less influenced by outlier trials. The group difference for median RT also did not reach significance ($p = .13$) and indicated a similarly small effect ($d = .23$).

The MRT and median RT results therefore indicate an overall small effect for RT slowing in the AD/HD group compared to controls and also suggest that MRT may not be unduly influenced by outlier RT trials, given similar effect sizes for MRT and median RT. More detailed examination of the tail of the RT distribution is described in the *Ex-Gaussian distribution analyses* section below.

Bivariate correlations and ANCOVAs. As described above, there was a medium-range effect size for SDRT ($d = .49$), whereas the effect size for MRT was small ($d = .26$), suggesting the relationship between SDRT and MRT should be further examined to more formally determine the extent to which larger SDRT in the AD/HD group may or may not be a consequence of slower MRT. As expected, bivariate correlations indicated a significant association between SDRT and MRT in the overall sample ($r = .50$, $p < .001$), as well as within AD/HD ($r = .40$, $p < .001$) and control ($r = .57$, $p < .001$) groups.

ANCOVA analyses were then conducted to assess whether group status continued to predict SDRT with MRT as the covariate, and vice versa. Group status remained a significant predictor of SDRT with MRT covaried ($F(1,173) = 7.41$, $p = .007$); however, MRT was no longer predicted at a trend level with SDRT covaried ($F(1,173) = 0.03$, $p = .86$). These results indicate that while SDRT and MRT are associated, larger SDRT in the AD/HD group is not just a consequence of slower MRT, but rather represents a more distinct process for the primary Stop-Signal Task in this sample.

Analyses with different data screening criteria. The above analyses were also completed with alternative RT data trimming criteria to examine how different screening procedures may influence results. This included trimming anticipatory trials <200ms and outlier trials >2.5SD beyond an individual's mean; trimming only anticipatory trials <200ms; and no data trimming at all. In general, results did not substantially vary when these different criteria were used. Of note, this also includes a similar group difference and effect size for SDRT with trimming of outliers at 2.5SD beyond an individual's mean ($t(174) = -3.54$, $p = .001$; $d = .46$). This suggests that RT variability in AD/HD on the primary Stop-Signal Task is not solely due to a few excessively long outlier trials in the tail of the AD/HD distribution.

Results were also generally the same when the $n = 13$ participants excluded for poor performance on the primary Stop-Signal Task were re-included in analyses.

Summary. Consistent with a key prediction for the state regulation theory, SDRT was found to be significantly larger in the AD/HD group. There was also a medium-range effect size for SDRT, which is considered to be at a level of practical significance (Sun, Pan, & Wang, 2010) and generally consistent with previous findings (Castellanos et

al., 2005; Klein et al., 2006; Rommelse et al., 2008). Results also revealed that larger SDRT in the AD/HD group was not just a consequence of general RT slowing, as is also predicted by the state regulation theory. Rather, MRT appeared more reducible to SDRT. Specifically, SDRT made a unique contribution to group differences in ANCOVA analyses, whereas MRT did not. Group differences in SDRT were also robust to RT trial trimming at 2.5SD beyond an individual's mean, suggesting that larger SDRT in AD/HD may be inherent to the overall performance of AD/HD participants on the primary Stop-Signal Task, rather than due solely to a few outlier trials. More detailed examination of the RT distribution is described in the *Ex-Gaussian distribution analyses* section below.

Since the inhibition theory does not make explicit predictions regarding variability, the SDRT results support the state regulation theory but do not reject the inhibition theory. Results of MRT analyses indicating a trend level group difference and small effect size are somewhat equivocal with respect to state regulation predictions that putative SSRT differences will be primarily related to MRT-related differences and inhibition assumptions that MRT will be more similar between groups and make less of a contribution to SSRT. Forthcoming analyses of a key competing prediction for both theories (i.e., results of SSD analyses, presented in the *SSRT analyses* section) will provide more definitive evidence as to which theory is best supported by SSRT findings.

Ex-Gaussian distribution analyses of the primary Stop-Signal Task. To further examine the RT distributions for AD/HD and control groups on the primary Stop-Signal Task, the ex-Gaussian distribution was modeled using the quantile maximum probability estimator (QMPE; Version 2.18), an open-source ANSI Fortran 90 statistical program used to estimate the parameters of RT distributions (Heathcote et al., 2004;

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Heathcote et al., 2002). The QMPE program utilizes quantile maximum likelihood estimations to calculate ex-Gaussian parameters and has been shown to perform effectively with as few as approximately 30 to 40 data points (Heathcote et al., 2002).

The ex-Gaussian distribution was also analyzed a second time by our consultant, Melvin J. Yap, Ph.D., from the National University of Singapore. Dr. Yap has expertise in RT distribution analyses and provided consultation regarding these analytical procedures. He also modeled the ex-Gaussian distribution using a script he designed for the R programming language to provide further confirmation of results. For all ex-Gaussian analyses, parameters were successfully estimated within acceptable limits for all participants (Heathcote et al., 2004; Heathcote et al., 2002).

T-tests. As can be seen in Table 5, while the raw values for mu (i.e., the mean of the normal component of the RT distribution) and sigma (i.e., the standard deviation of the normal component of the RT distribution) were slightly larger in the AD/HD group, they were not significantly different between groups, with effect sizes of only d = .12 and d = .17, respectively. In comparison to a small effect size for MRT (d = .26) and a medium-range effect size for SDRT (d = .49; see Table 4), results suggest that when RT trials from the tail of the distribution (tau) were excluded, AD/HD participants performed more similarly to controls. This is consistent with state regulation assumptions that AD/HD participants perform more similarly to controls with respect to MRT in the normal part of the distribution, and thus that generally slower MRT in AD/HD may be a consequence of a larger positive skew in the tail of the AD/HD distribution. While raw values for sigma were also slightly larger for AD/HD, results were not supportive of assumptions that variability is significantly increased in the normal part of the curve.

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Table 5
Ex-Gaussian Distribution Analyses for the Primary Stop-Signal Task

	AD/HD (n=94) Mean(SD)[raw]	Control (n=82) Mean(SD)[raw]	t	p	Cohen's d
Mu (μ)	.03(1.02)[534.2ms]	-.08(.93)[519.6ms]	-0.76	.45	.12
Sigma (σ)	.10(1.04)[113.3ms]	-.07(.95)[105.6ms]	-1.13	.26	.17
Tau (τ)	.10(1.14)[133.3ms]	-.14(.90)[118.8ms]	-1.53	.13	.23

Note. Values reflect means and standard deviations for age-standardized variables. Raw RT means (ms) are in brackets for comparison, although were not used in calculating test statistics or effect sizes.

The raw value for tau (i.e., the mean of the exponential component, or "tail," of the RT distribution) was also larger for the AD/HD group; however, the group difference in this parameter also did not reach statistical significance (p = .13). Results indicated a small effect size (d = .23). This is also inconsistent with state regulation predictions that increased variability may be due to more excessively long RTs in the tail of the AD/HD distribution. It is also of note, however, that no significant differences were found for any ex-Gaussian parameters in this sample. Taken together, results therefore suggest that small differences across all ex-Gaussian parameters contributed to the observed differences in overall SDRT and MRT, rather than a single component of the ex-Gaussian distribution being primarily responsible for these differences.

Vincentile plots. In addition to group comparisons of ex-Gaussian parameters, it is also recommended that ex-Gaussian analyses be supplemented with Vincentile plots (Balota et al., 2008), which provide a means of averaging the RT distributions across participants (Balota et al., 2008; Ratcliff, 1979; Rouder & Speckman, 2004). With the assistance of Dr. Melvin Yap, vincentile plots were created by ordering RTs from fastest to slowest for each participant and calculating the means of the first 10% of RTs, second 10%, etc. Figure 10 displays these ascending bins across participants in each group.
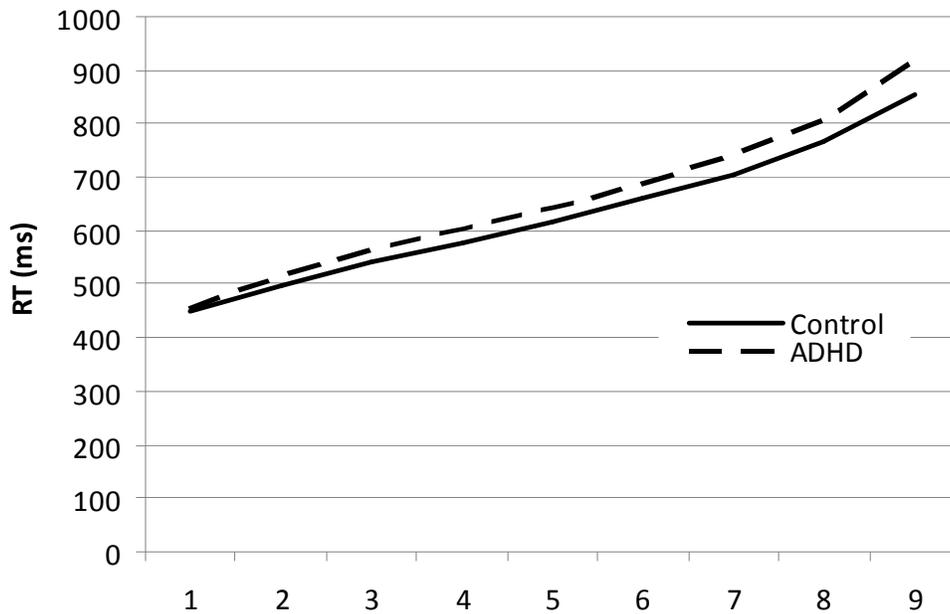
Figure 10. Vincentile plots showing means of the first 10% of RTs, second 10%, etc. for AD/HD and control groups.

While statistical tests were unable to be performed due to inadequate numbers of datapoints (i.e., 15 RTs or less per bin), visual inspection of the plots suggests findings consistent with the ex-Gaussian results. More specifically, plots show slightly slower raw mean RTs for the AD/HD group across the distribution that increase somewhat in the tail of the distribution, which is consistent with the modest raw value differences described above (Table 5). As demonstrated by the aforementioned statistical tests, however, these raw differences do not constitute statistically significant differences in the ex-Gaussian parameters within this sample.

Bivariate correlations and ANCOVAs. To further assess whether the ex-Gaussian parameters were estimated appropriately, bivariate correlations with corresponding summary measures were also performed. As anticipated, mu and MRT were highly positively correlated at $r = .91$ ($p < .001$). Correlations were also similar in

the AD/HD and control groups considered separately (r = .91, p < .001; r = .92, p < .001, respectively). Sigma and SDRT were also significantly associated in the whole sample (r = .40, p < .001), as well as within the AD/HD (r = .29, p = .004) and control (r = .50, p < .001) groups.

Tau's relationship with SDRT was also further explored given state regulation predictions that SDRT may be a consequence of tau. As expected, tau was also positively correlated with SDRT in the overall sample (r = .50, p < .001), as well as within the control (r = .57, p < .001) and AD/HD (r = .45, p < .001) groups. This suggests that for both groups, larger SDRT values were associated with larger tau values (i.e., longer tail of the distribution).

As described previously, however, the group difference in tau was found to be nonsignificant within this sample with a small effect size (Table 5), whereas SDRT was significantly larger in the AD/HD group with a medium effect size (Table 4). ANCOVA analyses in which group was used to predict SDRT with tau as the covariate, and vice versa, were therefore conducted to more explicitly examine the relationship between these two variables. Group status continued to significantly predict SDRT with tau covaried (F(1,173) = 8.20, p = .005), whereas group did not predict tau with SDRT covaried (F(1,173) = .01, p = .913). This indicates that while SDRT and tau are associated in each group, larger SDRT in the AD/HD group is also not just a consequence of tau (i.e., excessively slow RTs in the tail of the distribution).

Analyses with different data screening criteria. The ex-Gaussian analyses were also completed with the alternative RT data trimming criteria described above. The pattern of results was generally the same when trimming anticipatory trials <200ms and

outlier trials >2.5SD beyond an individual's mean, as well as when trimming only anticipatory trials <200ms. When no RT data trimming was used, however, results differed. The group difference in sigma approached statistical significance (t(174) = -1.94, p = .06) and showed a small effect size (d = .29 compared to d = .17 in the screened sample). Tau continued to be nonsignificant (t(174) = -0.97, p = .33), with a no longer small effect size (d = .15 compared to d = .23).

These differences appear appropriate when considering that no RT censoring includes anticipatory trials <200ms in the analyses, for which as noted previously, there are significantly more for the AD/HD group. These trials most notably influence the modeling of the normal part of the distribution, as more RT trials become included in the estimation of the normal component and fewer in the exponential component for some participants. Thus, sigma and tau are most affected, with variability in the distribution reflected more heavily in the normal part of the distribution (sigma) and less in the distribution's tail (tau).

The sensitivity of ex-Gaussian distribution modeling to variations in data screening procedures was also apparent when the n = 13 who were excluded for poor performance on the overall primary Stop-Signal Task were included back into analyses. As can be seen in Table 6, the mu and sigma parameters were very similar as compared to the cleaned sample with optimal data screening procedures (see Table 5). The group difference in tau, however, became significant, with the AD/HD group having a significantly larger value for tau than the control group.

These results indicate how the RT distributions of a relatively small number of participants (n = 13, 6.9% of the total sample) who did not adhere to task requirements

Table 6

*Ex-Gaussian Distribution Analyses for the Primary Stop-Signal Task in the Expanded
Sample (Includes n=13 Excluded for Poor Overall Performance on the Stop-Signal Task)*

	AD/HD (n=102) Mean(SD)[raw]	Control (n=87) Mean(SD)[raw]	t	p	Cohen's d
Mu (μ)	.01(1.05)[531.8ms]	-.07(.97)[521.0ms]	-0.57	.57	.08
Sigma (σ)	.12(1.05)[114.3ms]	-.04(.98)[107.1ms]	-1.10	.27	.16
Tau (τ)	.16(1.15)[137.1ms]	-.16(.91)[118.3ms]	-2.16	**.03**	.31

Note. Values reflect means and standard deviations for age-standardized variables. Raw RT means (ms) are in brackets for comparison, although were not used in calculating test statistics or effect sizes.

significantly affected the results of RT distribution analyses. Results therefore indicate the sensitivity of ex-Gaussian analyses to differences in data screening and emphasize the importance of utilizing careful data cleaning procedures when analyzing RT distributions.

Summary. While raw values for all ex-Gaussian components were larger in the AD/HD group, no ex-Gaussian parameters were significantly different between groups. This includes similar mu values, which is generally supportive of state regulation predictions that exclusion of RT trials in the tail of the distribution results in more similar performance between AD/HD and control groups, as well as similar sigma values, which is inconsistent with state regulation assumptions of significantly increased variability throughout AD/HD task performance.

Contrary to predictions in the current study, the positive skew (i.e., tau) of the AD/HD distribution, however, also was not significantly different from the control group. Additionally, group status was found to continue to significantly predict SDRT when tau was covaried, whereas group did not predict tau with SDRT covaried. Results therefore revealed that while a key state regulation prediction of larger SDRT in the AD/HD group was supported, an extension of this assumption suggesting that increased variability may be primarily attributable to more excessively slow RTs in the tail of the AD/HD

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distribution was not supported in this sample. Rather, results thus far indicate that increased variability in the AD/HD group appears prevalent across the RT distribution and intrinsic to the overall performance of AD/HD participants on the primary Stop-Signal Task, as opposed to being solely attributable to either slower MRT or a longer RT distribution tail in the AD/HD group.

In addition, results indicated that ex-Gaussian analyses were notably sensitive to data screening procedures. This emphasizes the importance of careful data cleaning prior to RT distribution modeling. It also suggests that variations in ex-Gaussian distribution findings within the current literature may be in part attributable to differences in RT data screening procedures.

SSRT analyses for the primary Stop-Signal Task. The final primary Stop-Signal Task analyses involved examining the chief Stop-Signal Task outcome measure, stop-signal reaction time (SSRT), and its components, MRT and stop-signal delay (SSD). These variables are associated in the following formula for calculating SSRT: SSRT = MRT – SSD (please see Figure 4 for additional description).

As described previously, these analyses are particularly important to the state regulation and inhibition theories, as both make competing predictions regarding the processes underlying supposed group differences in SSRT. Namely, the inhibition theory proposes that shorter SSD in the AD/HD group (i.e., the AD/HD group needing more warning to inhibit response to the stop-signal) is primarily responsible for SSRT group differences and that MRT makes a relatively small contribution to SSRT. The state regulation theory, on the other hand, proposes that AD/HD participants do not require significantly more warning than controls to inhibit the stop-signal response (i.e., similar

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SSD values between groups), and apparent SSRT differences are more attributable to MRT-related group differences.

T-tests. Groups were first compared on the calculated SSRT variable. As anticipated, there was a significant group difference in SSRT, with the AD/HD group having significantly larger SSRT than the control group (Table 7). The effect size for this difference was small to medium (d = .42). This is broadly consistent with previous SSRT findings, which generally indicate moderate effect sizes (Willcutt, Doyle et al., 2005).

The SSRT components were then examined separately. As noted previously and as can be seen in Table 7, there was a trend-level group difference in MRT (p = .08) with a small effect size (d = .26). Analysis of mean delay (SSD) showed the AD/HD group to have a shorter raw SSD than controls; however, the difference was nonsignificant (p = .72), with an effect size of only d = .06. This indicates that the AD/HD group did not require significantly more warning than the control group to inhibit responses to the stop-signal, which is consistent with state regulation theory predictions and contrary to inhibition theory predictions regarding SSD.

Table 7
Stop-Signal Reaction Time (SSRT) and Delay (SSD) for the Primary Stop-Signal Task

	AD/HD (n=94) Mean(SD)[raw]	Control (n=82) Mean(SD)[raw]	t	p	Cohen's d
SSRT	.24(1.11)[298.4ms]	-.20(.96)[260.7ms]	-2.80	**.006**	.42
Mean RT (MRT)	.09(.90)[667.4ms]	-.17(1.0)[638.4ms]	-1.75	.08	.26
Mean Delay (SSD)	-.09(1.05)[371.2ms]	-.03(1.01)[380.2ms]	0.36	.72	.06

Note. Values reflect means and standard deviations for age-standardized variables. Raw RT means (ms) are in brackets for comparison, although were not used in calculating test statistics or effect sizes.

Taken together, results therefore indicate that larger SSRT in the AD/HD group was more attributable to trend-level MRT-related differences than to SSD. This can be

further seen when comparing the relative contributions of MRT in each group (AD/HD = 667.4ms, control = 638.4ms; difference = 29ms) to SSD in each group (AD/HD = 371.2ms, control = 380.2ms; difference = -9ms). When combined, this produces the observed group difference in SSRT (AD/HD = 298.4ms, control = 260.7ms; difference = 37.7ms), with the MRT difference (29ms) making a larger contribution than the SSD difference (-9ms) to SSRT (i.e., 29ms - -9ms = 38ms).

Thus, while somewhat shorter although nonsignificant SSD and somewhat longer and trend-level MRT differences in the AD/HD group both contributed to the significant group difference in SSRT, MRT made a larger relative contribution to SSRT than SSD. This is more supportive of the state regulation theory, which predicts MRT-related differences as the primary reason for larger SSRT in the AD/HD group. It is contrary to inhibition theory assumptions, which posit that SSRT group differences are primarily attributable to shorter SSD in the AD/HD group.

***Bivariate correlations and regression analyses*.** Bivariate correlations were also conducted to further explore and confirm the relationship between SSRT and its components. As expected, results revealed a significant negative correlation between SSRT and SSD ($r = -.59$, $p < .001$), indicating that longer SSRT was associated with shorter SSD. Results were similar within AD/HD ($r = -.66$, $p < .001$) and control ($r = -.51$, $p < .001$) groups. MRT and SSD were also significantly related within the overall sample ($r = .79$, $p < .001$), as well as within AD/HD ($r = .76$, $p < .001$) and control ($r = .84$, $p < .001$) groups, with larger SSD being associated with larger MRT. Contrary to expectation, however, bivariate correlations between MRT and SSRT were not

significant in the overall sample (r = .03, p = .67), or in AD/HD (r = -.02, p = .860) or control (r = .03, p = .763) groups.

Given this seemingly counterintuitive result, the relationship between SSRT and its components was examined further with multiple regression analyses, which control for the associations between variables within the SSRT formula. When MRT, SSD, and their interaction were used to predict SSRT, the overall regression was significant (F(3, 172) = 2267.4, p < .001), with an adjusted R^2 of .975. As expected, all predictors were significant, including MRT (β = 1.280, SE = .022, p = <.001), SSD (β = -1.595, SE = .020, p < .001), and the MRT and SSD interaction (β = .032, SE = .012, p = .009). Regression results were similar for AD/HD and control groups analyzed separately.

This pattern of correlational and multiple regression analyses is an example of variable suppression (Kline, 2005). The specific relationship between the highly related SSRT variables, in which one had a negative (SSD) and the other a positive (MRT) association with SSRT, was masked in bivariate correlations that did not control for these associations. When corrections for relationships between variables were made in multiple regression analysis, however, the actual pattern was revealed and was consistent with expectations of significant associations between SSRT and each of its components.

Additional SSRT analyses. As previously described, results demonstrated that MRT made a relatively larger contribution to group differences in SSRT than SSD. Given the relationship between MRT and response variability indicating that SDRT is not reducible to MRT and in fact suggesting that the converse is true (i.e., SDRT showed robust group differences beyond MRT, whereas MRT did not predict group differences beyond SDRT), the contribution of response variability to SSRT was further explored.

To start, SSRT was calculated using different measures of central tendency, including median RT (SSRT = median RT – SSD) and ex-Gaussian mu (SSRT = mu – SSD) to assess whether the SSRT effect decreased as variability due to outliers in the tail of the RT distribution was reduced. When median RT was substituted for MRT in the SSRT formula, results were very similar to the original analysis. SSRT was significantly larger in the AD/HD group (t(174) = -2.79, p = .006), with an effect size of d = .42. Given similar effect sizes for median RT (d = .23) and MRT (d = .26), it is not surprising that substituting median RT in the SSRT formula produced analogous results. This reflects that typical measures of central tendency, and thus SSRT, were not unduly influenced by only a few outlier trials. RT outliers that were four standard deviations beyond an individual's mean were also screened prior to analyses, which reduced the likelihood of notable differences between mean and median RT due to a few extreme outlier trials.

When variability due to the exponential tail of the distribution (i.e., tau) was excluded by substituting mu (i.e., the mean of the normal part of the RT curve) in the SSRT formula, however, results differed. Using mu, the group difference in SSRT was no longer significant and instead was at a trend level (t(174) = -1.83, p = .07), with a small effect size (d = .28). This is compared to an effect size of d = .42 when using MRT in the standard equation (see Table 7). Since mu is less affected by RT variability occurring in the tail of the distribution than MRT, this result supports that observed group differences in SSRT are related to differences in RT variability. This result also further supports state regulation assumptions that response variability plays an important role in overall AD/HD Stop-Signal Task performance.

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Bivariate correlations and ANCOVAs. Bivariate correlations and ANCOVA

analyses were also conducted to further assess the relationships between SSRT, MRT,

and RT variability (i.e., SDRT and tau) and to further test whether SSRT differences

were reducible to response variability. As expected, SSRT and SDRT were positively

correlated within the overall sample ($r = .37$, $p < .001$), as well as within the AD/HD ($r =$

$.25$, $p = .01$) and control groups ($r = .43$, $p < .001$). SSRT and tau were also positively

associated in the overall ($r = .348$, $p < .001$), AD/HD ($r = .296$, $p = .004$), and control ($r =$

$.400$, $p < .001$) groups. As described previously, bivariate correlations between MRT and

SSRT were not significant ($r = .03$, $p = .67$), however, this was an artifact of variable

suppression (Kline, 2005), and multiple regression revealed MRT's positive relationship

with SSRT (see *Bivariate correlations and regression analyses* section above).

ANCOVA analyses were then conducted in which group was used to predict

SSRT with different covariates (i.e., SDRT, tau, and MRT), and vice versa. The goal of

these analyses was to further assess whether SSRT differences were better accounted for

by response variability versus MRT differences. When MRT was covaried, results

indicated that group status continued to significantly predicted SSRT ($F(1,173) = 7.59$, p

$= .006$) at a level very similar to SSRT alone (see Table 7). When SSRT was covaried,

group status predicted MRT at a nonsignificant trend level similar to MRT alone

($F(1,173) = 2.58$, $p = .09$; see Table 7), indicating that SSRT results were not

significantly affected by MRT and vice versa. Of note, when SSD was covaried, group

status also continued to significantly predict SSRT ($F(1, 173) = 10.19$, $p = .002$) and did

not predict SSD with SSRT covaried ($F(1,173) = 2.45$, $p = .119$). These results further

indicate that the group difference in SSRT was also not accounted for by SSD.

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Regarding measures of variability, when tau (i.e., variability in the tail of the distribution) was covaried, group also continued to significantly predict SSRT ($F(1,173) = 5.82$, $p = .017$), although the significance level was relatively less similar to the overall SSRT group difference than when MRT was covaried ($p = .017$, $p = .006$, respectively). Tau remained nonsignificant when predicted by group with SSRT covaried ($F(1,173) = .32$, $p = .558$). Thus, while tau and SSRT were related in each group, variability in the RT distribution tail alone did not fully account for the group difference in SSRT in ANCOVA analyses.

When SDRT (i.e., variability across the overall RT distribution) was the covariate, on the other hand, group status no longer significantly predicted SSRT and was instead at a trend level ($F(1,173) = 3.10$, $p = .08$). Group status continued to significantly predict SDRT with SSRT covaried ($F(1,173) = 5.78$, $p = .02$). Results therefore provide additional support that group differences in SSRT are more related to group differences in RT variability than to MRT or SSD differences alone. Results also further indicate the robustness of increased overall RT variability in the AD/HD group and support state regulation assumptions that RT variability is an important factor in AD/HD group performance on the primary Stop-Signal Task.

Analyses with different data screening criteria. Mean comparison analyses of SSRT and its components were also conducted with the alternative RT data trimming criteria. Results did not substantially differ when different criteria were used. Results were also generally the same when the $n = 13$ participants excluded for poor primary Stop-Signal Task performance were included in analyses.

***Summary*.** As anticipated, there was a significant group difference in SSRT in this sample, with the AD/HD group showing larger SSRT than the control group. Contrary to a key prediction of the inhibition theory, however, there was not a significant group difference in SSD, indicating that the AD/HD group did not need significantly more warning than the control group to inhibit the stop-signal response. SSD was also found to make less of a contribution to SSRT group differences than MRT. This is more supportive of the state regulation model, which proposes that group differences in SSRT are more attributable to MRT than to SSD differences, and it is less supportive of inhibition theory predictions that larger SSRT in the AD/HD group is more attributable to SSD than MRT.

Additionally, RT variability was shown to be important in SSRT group differences such that SSRT also appears reducible to increased response variability in the AD/HD group. Specifically, when the tail of the RT distribution was accounted for by substituting mu in the SSRT formula (SSRT = mu – SSD), the SSRT group difference was no longer significant and was instead at a trend level. Furthermore, group no longer significantly predicted SSRT when SDRT was covaried and was also at a trend level; however, SDRT was still significantly predicted when SSRT was covaried. The importance and robustness of increased RT variability in the AD/HD group was therefore further supported by SSRT analyses, providing additional support for state regulation assumptions that RT variability is a key component of AD/HD group performance.

Taken together, overall results of key primary Stop-Signal Task analyses were therefore more supportive of state regulation and less supportive of executive inhibition model assumptions.

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Secondary Stop-Signal Task Results

As described previously, the secondary Stop-Signal Task consists of the same task requirements as the primary Stop-Signal Task; however, it does not include trial-by-trial RT data. Thus, data screening at the individual RT trial level, as well as ex-Gaussian analyses could not be performed, which limits the ability to make detailed comparisons between the two task versions.

Additionally, analysis of the validity of the secondary version of the task revealed that the tracking algorithm performed differently within each group. Specifically, probability of inhibiting was significantly different between AD/HD (mean = -.19, SD = 1.0; raw mean = 48.0%) and control (mean = .31, SD = .82; raw mean = 52.4%) groups $(t(277) = 4.57, p < .001)$, with a medium effect size $(d = .54)$. This indicates that the tracking program did not produce the same average probability of inhibiting (i.e., 50%) for each group. Furthermore, statistics reported represent equal variances not assumed (significant Levene's test, $F = 6.69, p = .010$), with greater variability in probability of inhibiting within the AD/HD group compared to controls.

Reasons for these differences are not known, as the task is designed to approximate 50% probability of inhibiting across participants. Given the potential effects of task validity concerns on variables of interest, however, additional analyses of the secondary Stop-Signal Task were not performed.

Gordon Continuous Performance Test (G-CPT) Results

As described previously, the Gordon Continuous Performance Test (G-CPT) provides another putative measure of behavioral inhibition (i.e., commission errors). Analyses addressed overall commission and omission errors, as well as particular

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commission error types that are of theoretical interest in order to test inhibition and state regulation assumptions regarding G-CPT performance. Specifically, the inhibition theory suggests that the AD/HD group will make more overall commission errors and relatively more of the purely "impulsive" commission error type (X1X errors). The state regulation theory, on the other hand, emphasizes omission errors in the AD/HD group and implies the relative importance of "slow processing" commission errors (19X errors), which involve slower reaction to the correct sequence (please see Table 1 for additional description of these and other commission error types).

Furthermore, while overall commission and omission errors were analyzed to test whether results were similar to previous studies, primary G-CPT analyses for the current study involved testing error proportions. This allowed for the examination of the relative importance of particular error types between groups and thus more appropriately tested the aforementioned inhibition and state regulation assumptions.

Error proportions were therefore calculated by dividing the number of specific errors by the total number of commission and omission errors made throughout the task for each individual. Given this equation, only participants making at least one error were included in analyses so as not to have an undefined error proportion due to a zero denominator. This criterion excluded 12 of the 470 total participants (2.55%), of which there were 8 from the control group (3.76% of control participants) and 4 from the AD/HD group (1.56% of AD/HD participants). Error proportions and error means for all error types are presented in Table 8 for reference.

Table 8

Gordon Continuous Performance Test (G-CPT) Error Proportions and Raw Errors

	AD/HD (n=253)		Control (n=205)	
	Proportions	Errors	Proportions	Errors
Omissions	-.13(.94)[.52]	.18(1.07)[16.79]	.19(1.08)[.60]	-.24(.90)[11.09]
Commissions	.13(.94)[.48]	.17(.98)[21.10]	-.19(1.09)[.40]	-.28(.76)[9.13]
X1X (Impulsive)	.04(.94)[.20]	.12(.90)[5.82]	-.01(1.08)[.21]	-.16(1.03)[4.52]
19X (Slow Proc.)	-.01(.93)[.06]	.12(1.03)[2.34]	-.06(1.07)[.06]	-.22(.84)[1.31]
XX9	.12(1.02)[.04]	.17(1.06)[1.12]	-.29(.72)[.01]	-.33(.68)[.33]
XX1	.05(.96)[.08]	.11(.95)[4.07]	-.04(1.08)[.08]	-.21(.74)[1.63]
X9X	.11(.99)[.03]	.16(1.04)[1.41]	-.28(.85)[.02]	-.32(.76)[.45]
XXX	.21(1.09)[.07]	.15(.98)[6.33]	-.35(.72)[.02]	-.29(.58)[.90]

Note. Values represent normalized (i.e., age-standardized and transformed) means, with standard deviations in parentheses. Raw values are presented in brackets for comparison, although were not utilized in statistical analyses. For additional description of specific commission error types, please see Table 1.

Analyses of G-CPT overall commission and omission errors.

As anticipated, group comparison of overall commission and omission errors indicated results consistent with previous findings. Specifically, AD/HD participants made more overall commission errors on average than controls ($t(468) = -5.58$, $p < .001$), with a medium-range effect size ($d = .53$). AD/HD participants also made significantly more omissions than controls ($t(467.9) = -4.67$, $p < .001$), with a small to medium range effect size ($d = .43$).

Analyses of G-CPT error proportions.

Overall commission and omission error proportions. Error proportions for overall commission and omission errors were then included in a 2 x 2 mixed model ANOVA to assess the relative importance of commission and omission errors between groups. Group status was included as the between-subjects factor and commission and omission proportions as the within-subjects factor. As seen in Figure 11, results revealed a significant interaction between group and error type ($F(1,456) = 11.55$, $p = .001$).

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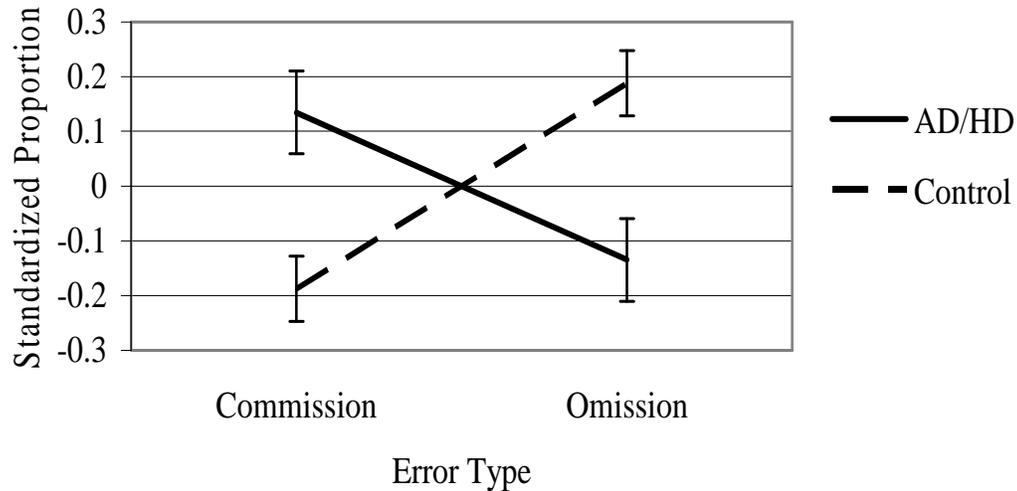
Figure 11. Error proportions for overall commission and omission errors for the AD/HD and control groups.

Tests of simple main effects showed that overall commission error proportion was significantly larger for the AD/HD group compared to controls ($F(1,456) = 11.55$, $p = .001$). In contrast, omission error proportion was significantly smaller for the AD/HD compared to control group ($F(1,456) = 11.55$, $p = .001$). The significant interaction therefore resulted from the differential importance of error type between groups and indicated that AD/HD participants made proportionately more overall commission errors and proportionately less omission errors than controls.

Proportions of commission error types. To address the relative importance of particular commission error types between groups, a 2 x 6 mixed model ANOVA was conducted. Group status was again the between subjects factor, with the six commission error types as the within-subjects factor. This included the two error types of primary interest to inhibition and state regulation theories (i.e., X1X "impulsive" and 19X "slow processing"), as well as the four other possible commission error types (see Table 1 for additional descriptions). Since the sphericity assumption was violated for this analysis

(Mauchly's Test of Sphericity, $\chi^2 = 88.99$, p < .001), results of within-subjects effects are reported with Greenhouse-Geisser correction.

As depicted in Figure 12, results revealed a significant interaction between group and error type ($F(4.62, 2105.5) = 6.06$, p < .001). Simple main effects of group at error type revealed that the AD/HD group did not make proportionately more X1X "impulsive" errors ($F(1,456) = .30$, p = .584) or 19X "slow processing" errors ($F(1,456) = .32$, p = .570) than controls, which were the primary errors of interest for inhibition and state regulation theories. Analysis of commission error types therefore did not provide further support for inhibition or state regulation predictions.
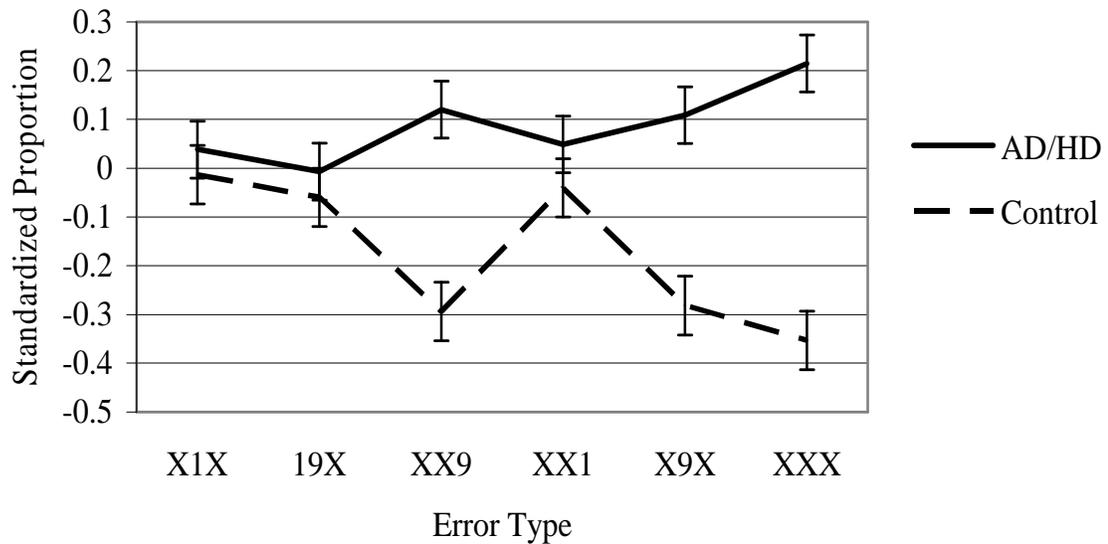
Figure 12. Error proportions for specific commission error types for the AD/HD and control groups.

Results for other error types less specifically related to theories of interest showed the following: There were no group differences for proportion of XX1 errors ($F(1,456) = .87$, p = .351), which may reflect impulsive responses to the go-signal or anticipatory reactions depending on response latency (Halperin, Wolf et al., 1991; Halperin et al., 1988). There were group differences, however, for proportions of other errors thought to

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reflect multiple processes such as inattention and/or poor working memory (Frank et al., 2007; Halperin, Wolf et al., 1991), with the control group making proportionately less XX9 ($F(1,456) = 23.90$, $p < .001$) and X9X ($F(1,456) = 20.05$, $p < .001$) errors compared to AD/HD participants. The control group also made proportionately less "random" XXX errors ($F(1,456) = 41.06$, $p < .001$) than the AD/HD group.

Examined further, results of simple effects of error type within each group indicated significant differences between error types within the control group ($F(5,452) = 5.12$, $p < .001$), with a lower proportion of XX9, X9X, and XXX errors compared to X1X, 19X, and XX1 errors ($p < .05$ for all comparisons). Within the AD/HD group, multivariate simple effects were not significant ($F(5,452) = 1.80$, $p = .111$). Specific error types within the AD/HD group were therefore not examined.

Taken together, primary errors of interest reflective of inhibition and state regulation predictions did not differentiate AD/HD and control groups. The significant interaction observed was therefore primarily due to the control group exhibiting proportionately less of several other error types compared to the AD/HD group and also in comparison to other errors within the control group, as opposed to the AD/HD group demonstrating a relatively higher proportion of specific error types.

Summary.

Consistent with inhibition theory prediction, analysis of overall commission and omission error proportions revealed commission errors to be of greater magnitude than omission errors within the AD/HD compared to control group. Specifically, the AD/HD group made proportionately more overall commission errors and proportionately less omission errors than controls.

Further examination of specific commission error types, however, did not provide additional differentiation regarding inhibition and state regulation predictions. Error types reflective of inhibition (X1X "impulsive") and state regulation (19X "slow processing") assumptions were not found to occur at significantly larger proportions in one group or the other. Other error types less directly related to inhibition or state regulation predictions and reflective of multiple processes (i.e., XX9, X9X) or "random" errors (i.e., XXX) did show group differences, with the control group showing a lower proportion of these errors compared to the AD/HD group and to other errors within the control group.

Commission error type results are therefore difficult to interpret with respect to a particular theoretical approach, however, suggest that groups may show differential performance for some error types. Additionally, it is of note that initial examination of particular commission error types indicated notable skewness and restricted variability due to low occurrence rates of the particular error types of interest (see *Data Preparation* section). While error types were able to be transformed to approximate normality, these considerations are believed to influence the robustness of these analyses and should be taken into account when interpreting commission error type results.

Multiple Deficit Results

Analyses thus far have assessed state regulation and inhibition related processes on two tasks widely-used in the AD/HD literature. Primary variables of interest were then incorporated into a multiple deficit model of AD/HD to examine how they contributed to predicting AD/HD symptom dimensions.

An exploratory factor analysis involving the combination of primary and secondary Stop-Signal Task variables was initially proposed; however, validity concerns for the secondary task precluded combining data across versions. A sample of participants with data for the primary Stop-Signal Task, G-CPT, and additional neurocognitive measures described previously (see *Measures* section) was therefore utilized for factor analysis. This sample included n = 240 participants and 20 total variables (12 participants/item). Based on recommendations suggesting at least 5 to 10 participants per item (Everitt, 1975; Gorsuch, 1983) and/or approximately 200-300 total participants (Comrey & Lee, 1992; MacCallum, Widaman, Zhang, & Hong, 1999) for an analysis of this nature, the current sample was deemed adequate.

Specific analyses therefore involved assessing the extent to which primary Stop-Signal Task RT variables (i.e., SSRT, SDRT, MRT, tau) and G-CPT variables (i.e., overall commissions, overall omissions; specific commission error types were excluded due to low occurrence rates and sample size) with demonstrated significant or trend-level group differences were related to each other and to measures of motor processing speed, naming speed, and verbal working memory identified in previous factor analyses (Shanahan et al., 2006; Willcutt et al., 2001; Willcutt, Pennington et al., 2005).

Current Stop-Signal and G-CPT variables loading on these factors would suggest that Stop-Signal and G-CPT measures do not represent response variability or inhibition related constructs that are separable from processing speed or working memory domains. Current variables of interest loading on separate factors, such as SSRT being associated with SDRT or with G-CPT commission errors, would support response variability or inhibition as more distinct neurocognitive factors, respectively. Since specific a priori

predictions were not made regarding the relationships among these variables, factor analyses and regressions are considered exploratory.

Exploratory factor analysis.

Principal axis extraction and direct oblimin rotation were used to extract factors with eigenvalues greater than one. Oblimin rotation was used because it has an oblique rotation permitting factors to correlate. It therefore requires fewer a priori assumptions about relationships among variables than orthogonal rotations. Additionally, the same number of factors and similar factor loadings were also attained when conducting principal components analysis with varimax rotation, indicating that results were robust across different extraction and rotation methods.

Results revealed six factors with eigenvalues greater than one, with an overall variance explained of 70.48%. These factors were labeled Naming Speed, Response Variability, Mean RT, Motor Processing Speed, Verbal Working Memory, and G-CPT Errors (Table 9). All six measures of verbal naming speed loaded on the first factor; Stop-Signal Task SSRT, SDRT and tau loaded on the second factor; Stop-Signal Task MRT was the only variable to load separately on the third factor; all five measures of motor processing speed loaded on the fourth factor; all three verbal working memory tasks loaded on the fifth factor; and commission and omission errors from the G-CPT loaded on the sixth factor.

Naming speed, motor processing speed, and verbal working memory factors were very similar to those extracted in recent factor and latent trait analyses with subsets of the present data (McGrath et al., 2011; Shanahan et al., 2006). This lends support to the overall validity of current factor analysis findings. G-CPT commission and omission

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errors have also been shown to load on the same factor when included together in factor

analysis (Willcutt, Pennington et al., 2005), suggesting a construct related to both

inhibition and execution related errors. The inability to include more theoretically-driven

commission error types due to data constraints may also contribute to commissions and

omissions loading on an overall error factor that potentially reflects method variance

rather than more distinct inhibition or vigilance related domains.

Table 9
Principal Axis Factor Analysis of Stop-Signal, G-CPT, and Other Neurocognitive Tasks

| | *Factor Loadings on the Six Extracted Factors* | | | | | |
Measure	Naming Speed	Response Variability	Mean RT	Motor Processing Speed	Verbal Working Memory	G-CPT Errors
RAN Color	**.68**	--	--	--	--	--
RAN Number	**.78**	--	--	--	--	--
RAN Letters	**.67**	--	--	--	--	--
RAN Pictures	**.71**	--	--	--	--	--
Stroop Word	**.69**	--	--	--	--	--
Stroop Color	**.50**	--	--	--	--	-.22
Stop-Task SSRT	--	**.34**	--	--	--	--
Stop-Task SDRT	--	.72	.61	--	--	--
Stop-Task Tau	--	**.84**	-.22	--	--	--
Stop-Task MRT	--	--	**.82**	--	--	--
WISC Symbol Search	--	--	--	**-.55**	--	--
WISC Coding	.25	--	--	**-.56**	--	--
CPS, Parts 1 & 2	--	--	--	**-.85**	--	--
CPS, Part 3	--	--	--	**-.75**	--	--
Identical Pictures	--	--	--	**-.83**	--	--
Sentence Span	--	--	--	--	**.67**	--
Counting Span	--	--	--	--	**.64**	--
WISC Digits Backward	--	--	--	--	**.60**	--
G-CPT Commissions	--	--	--	--	--	**.83**
G-CPT Omissions	--	--	--	--	--	**.68**
Eigenvalue	7.20	1.74	1.42	1.37	1.32	1.05
% variance explained	36.02	8.70	7.10	6.86	6.57	5.23

Note. Em dash (--) indicates factor loading less than an absolute value of .20. Loadings in bold type indicate primary factor loading. RAN = Rapid Automatic Naming test; SSRT = Stop-Signal Reaction Time; SDRT = Standard Deviation of Reaction Time; MRT = Mean Reaction Time; WISC = Wechsler Intelligence Scale for Children-Third Edition; CPS = Colorado Perceptual Speed test; G-CPT = Gordon Continuous Performance Test.

Additionally, contrary to inhibition theory assumptions, results also revealed that Stop-Signal Task SSRT did not load with the other putative measure of behavioral inhibition (i.e., G-CPT Commission errors), but rather with measures of responses variability (i.e., SDRT and tau). This may be in part related to lack of specific error types and other putative behavioral inhibition measures in the analysis; however, it is consistent with other findings in the current study suggesting that SSRT is more associated with response variability rather than reflecting inhibition-related processes. State regulation predictions regarding the importance of response variability as a distinct neurocognitive factor, as well as SSRT being less reflective of response inhibition than RT-related processes, is therefore further supported by these results.

It is also of note that neither Stop-Signal Task MRT nor SDRT loaded with other processing speed measures. This has been found in previous factor analyses that included Stop-Signal MRT (Shanahan et al., 2006) and SDRT (Willcutt, Pennington et al., 2005) with processing speed tasks. Results perhaps reflect the differences between RT data for individual trials and speeded measures that are quantified by overall performance (e.g., number correct in a certain amount of time). Motor processing speed measures in the current analysis also involve more visual attention (e.g., recognition of target items in item arrays) than is present in the Stop-Signal Task, which may also contribute to factor differences. Further, Stop-Signal MRT also did not load with other Stop-Signal RT measures, with the exception of SDRT demonstrating a secondary cross-loading with MRT. Consistent with other current findings, these results are supportive of a relationship between MRT and SDRT but that Stop-Signal MRT and response variability were generally separable constructs from processing speed and from each other.

Given that MRT was the only variable with a primary loading on the third factor, an additional principal axis factoring with oblimin rotation analysis was run with MRT excluded. Results were very similar to the initial analysis and indicated a five factor solution accounting for 66.88% of variance with no notable cross-loadings.

Regression analyses.

Based on factor analytic results, composite scores were created for each participant by calculating the mean of scores of measures loading on each component. Five factor composites, as well as Stop-Signal MRT, were included in analyses. As anticipated, the control group performed significantly better than the AD/HD group across all composites, with medium to large range effect sizes (Table 10). As noted previously, MRT showed a trend-level group difference, with a small effect size.

Table 10
Group Comparisons for Factor Composite Scores and Stop-Signal MRT

Factor/Measure	AD/HD (n=94)	Control (n=82)	t	p	Cohen's d
Naming Speed	-.27(1.06)	.48(.99)	4.87	**<.001**	.74
Response Variability	.20(.97)	-.27(.96)	-3.26	**<.001**	.49
Motor Proc. Speed	-.44(.97)	.64(1.01)	7.21	**<.001**	1.09
Verbal Working Memory	-.11(1.0)	.47(1.08)	3.73	**<.001**	.56
G-CPT Errors	.27(.99)	-.27(.79)	-3.95	**.001**	.60
Stop-Signal MRT	.09(.90)	-.17(1.0)	-1.75	.08	.26

Factors were then included in multiple regression analyses to determine which constructs best predicted AD/HD symptom dimensions. Continuous AD/HD severity ratings for overall, inattentive, and hyperactive/impulsive symptoms were used as dependent variables, and the above factors and Stop-Signal MRT were entered as independent variables (Table 11).

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Table 11
Multiple Regression Results for AD/HD Symptom Severity Ratings (n = 240)

	Overall			Inattentive			Hyperactive/Impulsive		
	β	SE	p	β	SE	p	β	SE	p
NS	-.005	.077	.948	-.053	.076	.520	.053	.081	.541
RV	.041	.066	.549	.080	.065	.236	-.015	.070	.834
PS	-.361	.074	**<.001**	-.350	.073	**<.001**	-.299	.078	**.001**
VWM	.008	.063	.909	.019	.062	.777	-.007	.066	.916
G-CPT	.090	.069	.184	.059	.068	.377	.108	.073	.125
MRT	.007	.061	.912	-.017	.060	.779	.034	.064	.593

Note. For overall AD/HD symptom severity: Multiple R = .423, Adjusted R^2 = .179; For Inattentive symptom severity: Multiple R = .440, Adjusted R^2 = .173; For Hyperactive/Impulsive symptom severity: Multiple R = .326, Adjusted R^2 = .083. NS = Naming Speed; RV = Response Variability; PS = Motor Processing Speed; VWM = Verbal Working Memory; G-CPT = Gordon Continuous Performance Test errors; MRT = Stop-Signal Mean Reaction Time.

Regression analyses were significant for overall $(F(6,233) = 8.44, p < .001)$, inattentive $(F(6,233) = 9.32, p < .001)$, and hyperactive/impulsive $(F(6,233) = 4.63, p < .001)$ AD/HD severity ratings. As can be seen, motor processing speed was the only significant predictor for each dimension. Response variability and other factors involving Stop-Signal or G-CPT measures were not found to predict variance above and beyond processing speed. State regulation assumptions that response variability, and inhibition model predictions that putative inhibition related factors (i.e., G-CPT errors), would represent unique predictors of AD/HD symptomatology were therefore not supported in the current sample.

Summary.

Exploratory factor analysis of Stop-Signal Task and G-CPT variables with other neurocognitive measures revealed motor processing speed, naming speed, and working memory factors similar to previous findings (McGrath et al., 2011; Shanahan et al., 2006). Consistent with state regulation predictions, response variability related measures

(i.e., Stop-Signal SDRT and tau) loaded on a distinct factor, indicating its separability from processing speed and working memory. Stop-Signal SSRT also loaded with SDRT and tau, as opposed to a putative inhibition measure (i.e., G-CPT commission errors), which was consistent with other findings in the current study and further supportive of state regulation predictions that SSRT was more associated with RT-related group differences rather than inhibition deficits in AD/HD.

Follow-up multiple regression analyses utilizing the extracted factors indicated motor processing speed as the only significant predictor of variance in overall, inattentive, and hyperactive/impulsive symptom severity dimensions. Additional support for response variability or G-CPT errors as unique predictors of AD/HD symptomatology within a multiple deficit model of AD/HD was therefore not found in the current sample. These factors were likely not as robust as other composites that contained several variables from multiple tasks, which may have contributed to reduced predictive power within the overall model. These limitations and suggestions for future research directions are further described in the *Discussion* section below.

Follow-up Analyses

Group differences in Full Scale IQ. As noted previously, results demonstrating significant group differences were re-run with Full Scale IQ as a covariate to assess whether findings could be accounted for by group differences in measures of general intelligence. When Full Scale IQ was covaried, results including group differences in Stop-Signal Task SDRT and SSRT and overall G-CPT commission errors remained significant. The only result that did not remain significant with Full Scale IQ covaried was overall G-CPT omission errors, which was instead at a trend level ($F(1,467) = 2.30$,

p = .07). Thus, primary findings for the current study were generally similar both with and without FSIQ covaried, indicating that the majority of group differences in key variables were generally not attributable to the commonly observed group differences in measures of general intelligence.

AD/HD subtypes. Results of analyses comparing AD/HD-Inattentive Type (AD/HD-I) and AD/HD-Combined Type (AD/HD-C) participants did not indicate any significant effects of AD/HD subtype. There were no significant differences between subtypes on primary measures of interest, including Stop-Signal Task SDRT, SSRT, MRT, SSD, and tau. Groups also did not differ significantly with respect to G-CPT commission or omission errors.

Age effects. Results showing significant differences in primary analyses were also re-run with age as a continuous covariate to assess whether findings were significantly influenced by age effects. When age was included as a covariate, results remained significant for all primary findings including Stop-Signal Task SDRT and SSRT, as well as G-CPT overall commission and omission errors.

When age was examined dichotomously by groups based on the middle childhood (ages 8-12) and adolescence (ages 13-18) groups used in studies by Williams et al. (1999) and Bedard et al. (2002), results of 2 x 2 ANOVAs did not reveal any significant interaction effects between age and AD/HD group status for any Stop-Signal Task variables, indicating that AD/HD and control group patterns of performance were similar across age groups for the Stop-Signal Task. G-CPT analyses, however, demonstrated significant interactions for both overall commission ($F(1,466) = 10.64$, $p = .001$) and omission ($F(1,466) = 5.21$, $p = .023$) errors. Examined further, results indicated that the

interactions for both error types were driven by significant group differences in the younger (ages 8-12) age group for commission ($p < .001$) and omission ($p = .002$) errors, and larger raw although nonsignificant differences for AD/HD versus control participants for commission ($p = .594$) and omission ($p = .980$) errors in the older age group (ages 13-18). While some studies have found group differences in G-CPT errors in child and adolescent populations (Mayes et al., 2001), others have not (Wherry et al., 1993), and studies of adults have indicated that the G-CPT has less discriminant validity in older age groups (Carlozzi & Horner, 2007).

Sex-related differences. To assess for potential sex-related effects on results, sex was included in a 2 x 2 ANOVA with group predicting primary variables of interest. No significant interaction effects were found for Stop-Signal Task or G-CPT variables. Thus, findings did not vary as a function of sex in the current sample.

Comorbidity of AD/HD and other externalizing disorders. Results could also not be accounted for by comorbid symptoms of oppositional defiant disorder (ODD) and conduct disorder (CD). When ODD and CD symptoms were included as covariates in analyses of primary results, Stop-Signal Task SDRT and SSRT, as well as G-CPT overall commission and omission errors remained significant.

Discussion

The current study tested two competing models of AD/HD, the inhibition and state regulation theories, by conducting fine-grained analyses of the Stop-Signal Task and another putative measure of behavioral inhibition, the Gordon Continuous Performance Test (G-CPT), in a large sample of children and adolescents. The inhibition theory predicts that performance on these tasks reflects increased difficulties for AD/HD participants to inhibit prepotent responses. The model posits that group differences in stop-signal reaction time (SSRT) on the Stop-Signal Task are primarily related to AD/HD participants requiring more warning than control participants to inhibit response to the stop-signal and emphasizes the relative importance of commissions, particularly the "impulsive" commission error type, over other error types on the G-CPT. The state regulation theory, on the other hand, proposes response variability due to difficulties maintaining an optimal state of arousal as the primary deficit in AD/HD. This model predicts that SSRT differences are more attributable to slower and/or more variable RT in the AD/HD group, rather than reflecting inhibitory deficits. State regulation assumptions also emphasize the relative importance of omission errors and the "slow processing" commission error type over other error types on the G-CPT.

Overall, results of Stop-Signal Task analyses were more supportive of state regulation predictions and showed that increased response variability in AD/HD was not reducible to slow mean reaction time (MRT) and also that response variability made a

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larger contribution to longer SSRT in the AD/HD group than inhibition-related processes.

Examined further, ex-Gaussian analyses of Stop-Signal Task go-trial RT distributions

revealed that increased variability in the AD/HD group was not solely due to a longer tail

in the AD/HD group RT distribution (i.e., tau), but rather indicated response variability

throughout AD/HD group performance on the Stop-Signal Task, as well as the significant

sensitivity of ex-Gaussian distribution analyses to variability in data screening

procedures. Results of G-CPT analyses indicated some support for the inhibition model,

although error type analyses failed to provide further support for either theory. Finally,

inclusion of primary variables of interest in exploratory factor analysis with other

neurocognitive predictors of AD/HD indicated response variability as a separable

construct and further supported its role in Stop-Signal Task performance and SSRT group

differences. Response variability did not, however, make a unique contribution to the

prediction of AD/HD symptomatology beyond motor processing speed in multiple deficit

regression analyses. Results have implications for the interpretation of the processes

reflected in widely-used measures in the AD/HD literature, as well as for the theoretical

understanding of AD/HD.

The current findings regarding Stop-Signal Task performance were generally

consistent with prior studies. Previous literature has shown increased response variability

to be a robust finding in AD/HD (Nigg, 2001; Oosterlaan et al., 1998; Willcutt, Doyle et

al., 2005). In the current study, SDRT was also found to be larger in the AD/HD group

with a medium-range effect size (d = .49). Analyses also showed that SDRT was not

reducible to slow MRT, and in fact, trend-level differences in MRT appeared reducible to

the significantly increased variability in the AD/HD group. Results were therefore

consistent with state regulation model assumptions that increased response variability is an important component of AD/HD group performance.

Previous results of Stop-Signal Task SSRT analyses have also typically shown longer SSRT in the AD/HD group (Nigg, 2001; Oosterlaan et al., 1998; Willcutt, Doyle et al., 2005), which was also found in the current study (d = .42). SSRT differences have generally been interpreted as reflecting an inhibition deficit; however, the few studies to date that have examined the components of SSRT (i.e., stop-signal delay (SSD) and mean RT (MRT)), suggest that SSRT may be more reflective of MRT-related differences as opposed to AD/HD participants requiring significantly more warning to inhibit the stop-signal response (Alderson et al., 2007; Alderson et al., 2008; Lijffijt et al., 2005).

Two of these studies are meta-analyses (Alderson et al., 2007; Lijffijt et al., 2005). A notable limitation to these investigations is that SSD had not been reported in studies included in the analyses and therefore was estimated. SSD has been directly examined in only one study to date (Alderson et al., 2008), with a relatively small sample of boys (n = 23). This study showed a nonsignificant group difference in SSD and attributed larger SSRT in the AD/HD group to slow MRT.

The current study expanded on this previous research by examining the components of SSRT in a much larger sample (n = 176) completing a tracking version of the Stop-Signal Task. Overall, general results of Alderson et al. (2008) were replicated, as the current study also found a nonsignificant group difference in SSD (d = .06). This was contrary to inhibition theory assumptions that the AD/HD group requires significantly more warning than the control group to inhibit the stop-signal response. Analyses for a secondary version of the Stop-Signal Task, which had the same task

parameters but did not include trial-by-trial RT data, were not completed due to the task tracking algorithm failing to converge on the intended probability of inhibition (50%) between groups.

The current study also further examined the relative contributions of SSD and MRT to the calculation of SSRT and found that the trend-level group difference in MRT made a larger contribution to SSRT than the smaller, although nonsignificant, SSD values in the AD/HD group. This result was more supportive of state regulation predictions that group differences in SSRT are more attributable to MRT than to SSD differences, and contrary to inhibition predictions that SSD makes a relatively larger contribution to SSRT. Additional analyses also showed larger SSRT in the AD/HD group to also be more reflective of increased RT variability than slower RT, which was further supportive of state regulation assumptions that RT variability is a key component of AD/HD group task performance. More specifically, group status no longer significantly predicted SSRT when SDRT was covaried; however, group continued to significantly predict SSRT when MRT was covaried. The SSRT group difference was also no longer significant when variability in the tail of the RT distribution was accounted for by substituting ex-Gaussian mu in the SSRT formula.

As noted previously, the present study also sought to extend examination of the Stop-Signal Task to more fine-grained analysis of the RT distribution by modeling the ex-Gaussian curve for go-trial RTs. When the current study was initially proposed, relatively few studies had examined the ex-Gaussian distribution in AD/HD, and no studies had analyzed the ex-Gaussian curve for Stop-Signal Task RT data. An initial prior study had shown similar performance between AD/HD and control groups on the

mean (i.e., mu) and standard deviation (i.e., sigma) of the normal part of the curve and a larger tail of the AD/HD distribution (i.e., tau) on a 4-choice RT task (Leth-Steensen et al., 2000). AD/HD group performance on the Conners' Continuous Performance Test demonstrated significantly faster mu, increased sigma, and a relatively larger effect for tau compared to controls (Hervey et al., 2006), further suggesting that increased response variability and slower MRT may be primarily attributable to more excessively long RTs (i.e., "attentional lapses") in the tail of the AD/HD distribution. Some variability in results had been shown, with one study that did not find significant group differences for any ex-Gaussian components on a 2-choice RT task (Geurts et al., 2008); however, given the overall existing literature at the time, it was hypothesized that the AD/HD group would show significantly larger tau than the control group in the current study.

Contrary to prediction, ex-Gaussian distribution analyses in the present study revealed larger raw values for mu, sigma, and tau in the AD/HD compared to control group; however, none of the parameters reached statistical significance. Results therefore indicated that increased variability in the AD/HD group was not primarily due to a larger RT distribution tail, but rather results suggested that small differences across the normal and exponential parts of the RT distribution contributed to overall increased variability in the AD/HD group.

Current results were consistent with Geurts et al. (2008) findings and also in line with a body of literature that emerged since the present study was initially proposed indicating notable variability in ex-Gaussian results across studies. Specifically, while previous studies have found significant differences in tau alone (Leth-Steensen et al., 2000), in tau as well as faster mu and increased sigma (Hervey et al., 2006), and no

differences in any components (Geurts et al., 2008), other more recent studies have shown increased sigma and tau with similar mu (Buzy et al., 2009; Vaurio, Simmonds, & Mostofsky, 2009), only increased tau (Epstein et al., 2011), or only increased sigma (Gooch, Snowling, & Hulme, submitted) in AD/HD compared to control groups. Similar to the current investigation, the latter two studies also examined the ex-Gaussian distribution in the Stop-Signal Task. While Gooch et al. (2008) also did not find significant group differences in tau, current results differ with respect to sigma, which was significantly larger in the AD/HD group in Gooch et al. (2008) and nonsignificant in the current study. In contrast, Epstein et al. (2011) found significantly larger tau in AD/HD and nonsignificant sigma and mu between groups for the Stop-Signal Task.

This variability in ex-Gaussian results may be due to different tasks with varying task demands used in previous studies (i.e., choice-RT (Geurts et al., 2008; Leth-Steensen et al., 2000), go/no-go (Hervey et al., 2006; Vaurio et al., 2009), working memory (Buzy et al., 2009), stop-signal (Gooch et al., submitted), multiple RT tasks (Epstein et al., 2011)). Previous studies have also differed with respect to task duration, with some utilizing very short tasks of around 3 minutes (Geurts et al., 2008) up to 21 minutes (Epstein et al., 2011). This may also account for disparate findings, with increased tau in the AD/HD group potentially related to longer tasks that allow for more opportunities for "attentional lapses" and thus greater variability (Johnson et al., 2007).

Additionally, prior studies examining ex-Gaussian distributions in AD/HD have varied with respect to data screening procedures. Some have screened anticipatory trials <100ms (Epstein et al., 2011; Gooch et al., submitted; Hervey et al., 2006), <150ms (Buzy et al., 2009; Geurts et al., 2008), or <200ms (Vaurio et al., 2009) without screening

for outlier trials. Another study screened outlier trials greater than four standard deviations beyond an individual's mean but did not screen anticipatory trials (Leth-Steensen et al., 2000). In general, studies utilizing anticipatory screening cite previous research such as Luce (1986), which describes the nondecision point for simple RT for adults as at least <100ms, as support for screening trials of at least this magnitude. Whether this cutpoint is optimal for children and adolescents across various types of RT tasks is less well-defined. To our knowledge, few if any previous ex-Gaussian studies have incorporated careful visual inspection of individual's RT distributions to determine whether particular screening criteria were most appropriate for the specific dataset being studied.

The current study therefore incorporated rigorous data preparation in order to optimize RT data for analysis and also to assess the effects of different screening procedures on results. Careful visual inspection of individual RT distributions for this sample of children and adolescents completing the Stop-Signal Task revealed that both anticipatory (<200ms) and outlier trials (greater than 4 standard deviations beyond an individual's mean) required censoring to produce cohesive distributions for analysis. Several participants were also excluded due to poor performance on the Stop-Signal Task, indicating lack of adherence to task parameters. As noted previously, based on this cleaned sample, ex-Gaussian distribution analyses revealed larger raw values for mu, sigma, and tau in the AD/HD compared to control group for Stop-Signal Task go-signal RT; however, none of the components reached statistical significance.

Of notable importance is that ex-Gaussian results in the current study were found to be highly sensitive to data screening procedures. Specifically, the group difference in

tau became statistically significant when the relatively few participants excluded for poor performance on the Stop-Signal Task (n = 13, 6.9% of the overall sample) were included back in analyses. This potentially explains differences between the current study and one recent study finding significantly larger tau in the AD/HD group for the Stop-Signal Task (Epstein et al., 2011). Epstein et al. (2011) excluded only participants with >50% omissions (number of participants excluded was not reported), whereas the current study excluded participants based on cutoffs for go-trial accuracy, probability of inhibiting, and SSRT indicative of poor task adherence suggested in previous Stop-Signal Task research (Rucklidge & Tannock, 2002; Schachar et al., 2000). Results may also vary with respect to differences in task parameters. Namely, the Stop-Signal Task utilized by Epstein et al. (2011) lasted for a duration of 21 minutes, whereas as the duration for the current Stop-Signal Task was approximately 6 to 7 minutes. As noted previously, this likely indicates the importance of task length on group differences in variability (Johnson et al., 2007).

Furthermore, including anticipatory and outlier RT trials back into current analyses (i.e., no RT data screening) resulted in a trend for larger sigma in the AD/HD group (p = .06). This result is more similar to the other recent study examining the ex-Gaussian distribution for the Stop-Signal Task (Gooch et al., submitted), which screened only anticipatory trials <100ms and found significantly larger sigma in the AD/HD group compared to controls. In addition, while task length was similar between Gooch et al. (submitted) and the current study, interstimulus interval varied (2000ms and 1500ms, respectively), which may also influence ex-Gaussian results.

To our knowledge, this is the first study of the ex-Gaussian distribution in AD/HD to incorporate rigorous preliminary data preparation procedures for determining

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appropriate RT cutpoints and also to examine the effects of these different screening criteria. Results indicate the importance of careful data examination prior to ex-Gaussian analyses and the sensitivity of distribution analyses to different screening criteria. Differences in these procedures, as well as variations in task parameters across studies, likely contribute to the notable variability in existing ex-Gaussian results.

In addition to fine-grained analyses of the Stop-Signal Task, the current study also sought to incorporate another putative behavioral inhibition measure in order to assess whether it was also reducible to inhibition or state regulation related processes. Consistent with prior results, current analyses demonstrated the expected group differences in increased overall commission and omission errors in the AD/HD group (Willcutt, Doyle et al., 2005). As an extension of previous literature, the current study also incorporated proportional error analyses to assess the relative importance of specific error types to one another. Results generally indicated support for the inhibition model, as AD/HD participants were found to make proportionately more commission errors and proportionately less omission errors than controls.

Analyses of specific commission error types were less definitive. AD/HD and control participants made similar proportions of errors thought to primarily reflect inhibition (X1X "impulsive") and state regulation (19X "slow processing") processes and showed variability with respect to other error types less related to theories of interest and believed to reflect multiple processes. Analyses of specific commission errors were also less robust than other findings due to low occurrence levels of particular error types and restricted variability requiring transformations to approximate normality.

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Finally, the current study also aimed to extend previous research by including primary variables of interest in exploratory factor analysis and multiple regressions with other neurocognitive predictors of AD/HD. Factor analytic results indicated additional support for state regulation predictions that response variability was a unique and robust factor in AD/HD group performance, as it loaded separately from processing speed, Stop-Signal Task MRT, and working memory factors. Results also further indicated that Stop-Signal Task SSRT was more reflective of response variability than inhibitory processes, with SSRT having the largest factor loading with SDRT and tau, as opposed to with G-CPT commission errors or MRT. SSRT also did not load with other measures of working memory, which is contrary to suggestions by Alderson et al. (2008, 2010) that working memory deficits may underlie SSRT group differences.

Present factor analyses were consistent with some and inconsistent with other previous studies utilizing subsets of the current data. Specifically, similar to Shanahan et al. (2006) and Willcutt et al. (2005), Stop-Signal Task MRT and SSRT did not load with measures of general motor processing speed in the current study. SSRT, however, also did not load with G-CPT commission errors as has been found in some previous investigations suggesting an inhibition factor (McGrath et al., 2011; Willcutt et al., 2001). Studies with other samples have also found SSRT to load with other putative inhibition measures, including Stroop interference and anti-saccade tasks (Miyake et al., 2000). Differences between these studies and the current investigation include that prior studies did not include Stop-Signal Task SDRT or G-CPT omission errors. In another investigation in which these variables were incorporated, a factor including SDRT, SSRT, and G-CPT commission and omission errors was extracted (Willcutt, Pennington

et al., 2005). While these measures were divided into a factor with SDRT and SSRT and another with G-CPT commission and omission errors in the present study, prior results indicate some support for the currently observed relationship between SSRT and SDRT.

Prior studies have also generally combined Stop-Signal Task variables from primary and secondary task versions. This was not possible in the current study due to validity concerns regarding the secondary version. Sample size was therefore smaller in the present study, which may contribute to some of the observed differences with current and previous research. Additionally, results also indicate the importance of including as many relevant predictors of AD/HD as possible in factor analyses, as the particular factors extracted vary with respect to the variables that are included.

Multiple regression analyses utilizing the extracted factors to predict AD/HD symptomatology did not provide additional support for response variability or other factors as unique predictors of AD/HD symptoms beyond variance explained by motor processing speed. This is consistent with previous studies indicating motor processing speed as a significant predictor of AD/HD (McGrath et al., 2011), although putative inhibition factors were also found to make a unique contribution in this prior research. Results also suggested that neither response variability nor inhibitory processes were solely or primarily responsible for variance in AD/HD in the current study. Thus, while the state regulation theory was best supported by current results and indicated response variability as an important and robust factor in AD/HD group performance, neither state regulation nor inhibition model processes were necessary or sufficient in explaining overall AD/HD symptoms on their own. Instead, in line with other previous literature (Pennington, 2006; Sergeant et al., 2003; Willcutt et al., 2010), a multiple deficit

approach to AD/HD appears most appropriate when developing a broad theoretical understanding of the disorder.

It is also of note that in the current study, there were measures from multiple tasks contributing to the motor processing speed factor, whereas the response variability factor was from a single measure, as was the G-CPT error factor. The overall variance explained was also low (adjusted $R^2 = .179$), indicating that AD/HD symptomatology was not well-accounted for by the constructs in the current model and further supports a multiple deficit approach to the disorder. It is possible that multiple measures contributing to response variability and/or other factors would increase their predictive power and contribute to additional variance explained. Other constructs such as delay aversion (Sagvolden et al., 2005; Sonuga-Barke, 2002, 2003), decision making (Garon, Moore, & Waschbusch, 2006), temporal processing (Radonovich & Mostofsky, 2004), and other putative behavioral inhibition measures (Jonkman et al., 1999; Miyake et al., 2000) shown to differentiate AD/HD and control groups may also contribute to explaining additional variance in AD/HD symptomatology.

Limitations

Limitations of the current study include inability to test for potentially convergent results between primary and secondary versions of the Stop-Signal Task. Analyses for the secondary version were unable to be completed due to the tracking procedure failing to converge at a similar percentage for probability of inhibiting to the stop-signal (i.e., approximately 50%) between groups. Reasons for this difference are unknown, however, are believed to affect the validity of results from this measure. While results of the primary version are believed to be reliable due to the availability of trial-by-trial RT data

and observed similar probability of inhibiting between groups, the inability to combine data across versions reduces the strength of the overall analyses, particularly for factor analysis and multiple regressions that require larger sample sizes. It also precluded analysis of potential convergent evidence for Stop-Signal Task results, as was initially proposed.

Lack of tasks involving simple RT and other less demanding RT processes also limits the generalizability of RT distribution analyses in the current study. The Stop-Signal Task involves additional demands compared to simple or simple-choice RT tasks, namely, the inclusion of an inhibitory signal amongst choice-RT trials. Previous studies have reported slowed go-RTs following stop-signal trials in both control and AD/HD groups (Alderson et al., 2008; Rommelse et al., 2008; Schachar et al., 2004). While prior investigations of less demanding RT tasks in AD/HD have also demonstrated RT and response variability group differences similar to the present study (Castellanos et al., 2005; Klein et al., 2006; Rommelse et al., 2008), current RT and ex-Gaussian distribution analyses for the Stop-Signal Task cannot be generalized to basic RT functioning in AD/HD due to the influence of stop-signal trials on go-RT.

The interpretation of factor analytic results is also limited by the lack of additional RT tasks in the current study. Specifically, since the Stop-Signal Task is the only measure with RT data, the response variability factor may be more related to method variance than a response variability construct in the current study. While not all Stop-Signal Task measures loaded on a single factor (i.e., MRT loaded separately) suggesting that response variability may not be entirely method related, inclusion of additional RT measures, particularly those with differing cognitive demands, would provide more

reliable support for a response variability construct. Similar limitations apply to the G-CPT error factor, which may also represent method variance.

The ability to generalize G-CPT findings to specific theory predictions is also limited in the current study. Previous investigations have shown slower and more variable RT in AD/HD on other go/no-go tasks (Hervey et al., 2006; Klein et al., 2006; Leth-Steensen et al., 2000); however, RT data was not available for the G-CPT for the current study. Examination of state regulation predictions regarding rate and variability of response could thus not be tested directly and were instead inferred from omission error rates and specific commissions thought to reflect "slow processing." While some support was found for inhibition theory related predictions, it is difficult to make more definitive assertions regarding the primary processes contributing to G-CPT performance due to these limitations. As noted previously, small occurrence rates and decreased variability of commission error types also limited the strength of these analyses.

Implications and Future Directions

Results from the current study have implications for the interpretation of commonly-used measures in the AD/HD literature, as well as for the theoretical understanding of AD/HD. Findings indicate that previous assumptions that Stop-Signal Task SSRT was primarily reflective of a motor inhibition deficit are incorrect. Rather, SSRT appears to reflect increased response variability in AD/HD, which is more supportive of the state regulation model. Implications of these results include the need for future studies to incorporate analysis of the components of SSRT. In particular, stop-signal delay (SSD) should routinely be reported in order to test whether the AD/HD group requires additional warning to inhibit stop-signal response. Inclusion of SSD will

also allow for examination of replication of current findings, as well as provide better data for future meta-analytic review of the components of SSRT. In-depth analysis of the components of other tasks thought to reflect inhibition or other processes is also implied in order to continue to increase understanding of the deficits that contribute to AD/HD.

Findings also indicate the importance of careful RT data preparation for future studies. As was shown with ex-Gaussian distribution analyses in particular, results were highly sensitive to data screening procedures. Inconsistencies within the previous literature examining ex-Gaussian distributions and other RT-related variables may therefore be in part attributable to differences in data preparation. Studies of RT measures should incorporate careful visual inspection of individual RT distributions, as well as examination of adherence to task demands, so that proper screening criteria can be applied. The incorporation of multiple RT tasks with varying demands within the same sample will also be important, so that the effects of increasing demands on RT in AD/HD can further understood. While one recent study included ex-Gaussian analyses of multiple RT tasks in the same sample (Epstein et al., 2011), results were limited by lack of rigorous data preparation.

Similar to other studies finding robust effects of increased intra-individual variability in AD/HD (Castellanos et al., 2005; Klein et al., 2006; Rommelse et al., 2008), current results demonstrated response variability to be an important factor in AD/HD group performance on the Stop-Signal Task. Results therefore supported the state regulation model as an important contributor to the theoretical understanding of AD/HD. Challenges arise, however, when considering the brain mechanisms associated with both state regulation and inhibition. The cognitive energetic model suggests involvement of

subcortical structures such as the hippocampus, mesencephalic reticular formation, and basal ganglia (Sergeant, 2005). Brain imaging studies of RT and RT variability, however, typically indicate involvement of frontal cortical regions (MacDonald et al., 2006), which have also been implicated in response inhibition (Boehler, Applebaum, Krebs, Hopf, & Woldorff, 2010). Furthermore, some previous studies of the effects of stimulant medication have shown decreases in RT and response variability with medication use (Epstein et al., 2006), as well as increased inhibitory control (Aron, Dowson, Sahakian, & Robbins, 2003; DeVito et al., 2009). It is therefore possible that state regulation and executive inhibition models are associated with similar neural substrates and thus less distinct than typically assumed. This would help to explain findings of both inhibition and response variability deficits in existing literature. Additional research is therefore necessary to clarify the neural substrates associated with the behavioral markers of response variability and inhibition.

Extending response variability findings to endophenotypic research is also implicated by the growing body of research indicating it as a robust factor in AD/HD. To date, several studies have suggested response variability as a candidate endophenotype for AD/HD (Castellanos & Tannock, 2002; Doyle et al., 2005). The particular measure of response variability that is most useful is less clear. While some previous literature indicates tau as a potential measure for differentiating AD/HD and control groups (Epstein et al., 2011; Hervey et al., 2006; Leth-Steensen et al., 2000), the notable variability across studies and high sensitivity of ex-Gaussian measures to RT screening procedures demonstrated in the current study suggest that tau may not be a robust measure suitable for endophenotypic research.

Difficulties also arise when attempting to elucidate the processes underlying tau. Some suggest excessively long RTs may represent "attentional lapses" (Epstein et al., 2011; Hervey et al., 2006; Leth-Steensen et al., 2000), although the particular nature of long RTs continues to be unclear. A recent RT simulation study examined the ex-Gaussian parameters and their potential relationship to cognitive processes (Matzke & Wagenmakers, 2009). The ex-Gaussian distribution was systematically varied and fit to data from a diffusion model with well-established cognitive interpretations (Ratcliff & McKoon, 2008; Wagenmakers, 2009). Results of Matzke and Wagenmakers (2009) indicated that ex-Gaussian components did not correspond to diffusion model parameters, and the authors recommended caution in interpreting ex-Gaussian changes in terms of cognitive processes. Thus, in addition to variability in existing literature and sensitivity of the ex-Gaussian distribution to varying task parameters, the processes related to ex-Gaussian components are also unclear. This further suggests that tau may not be an appropriate candidate endophenotype for AD/HD.

In addition to SDRT, other measures such as the coefficient of variation (Klein et al., 2006), intra-individual standard deviation (Williams, Strauss, Hultsch, Hunter, & Tannock, 2007) and spectral analysis of the RT distribution (Castellanos et al., 2005) have also demonstrated group differences and may provide additional measures for potential endophenotypic research. Clarifying how variability is defined and examining the behavioral correlates of variability will also be important in future research (Rapport, Kofler, Alderson, Timko, & DuPaul, 2009). Regarding inhibition, improved understanding of the specifics of this construct are also indicated, as research has

suggested similar concerns regarding the multifactorial nature of inhibition and potential overextension in its description (DeVito et al., 2009; Friedman & Miyake, 2004).

As noted previously, response variability has been found in other disorders (Segalowitz et al., 1997; Verte et al., 2006). The current study did not find that ODD and CD symptoms significantly affected results, and a recent study showed that group differences in response variability were not significantly influenced by comorbid depression or anxiety (Epstein et al., 2011). It has been suggested, however, that variability in AD/HD may be attributable to comorbid autism spectrum disorder (ASD) symptoms (Geurts et al., 2008). While participants in the current study were excluded for diagnosis of ASD, potential subclinical symptoms were not investigated due to lack of available data. Future research should therefore continue to examine the extent to which increased response variability is related to ASD symptoms or other comorbid disorders.

Additionally, while Stop-Signal Task follow-up analyses revealed that results did not differ as a function of age group, it is important to note that the current study only included children and adolescents. While current results indicated that longer SSRT in the AD/HD group was more attributable to differences in response variability than poor inhibition, there has been some suggestion that SSRT may reflect inhibitory processes in adult AD/HD populations (Lijffijt et al., 2005). To date, SSD has yet to be directly examined in adults with AD/HD. In-depth analysis of SSRT components in adults with AD/HD is therefore warranted to determine the extent to which developmental changes influence the processes underlying Stop-Signal Task performance.

Overall, contrary to longstanding assumptions that the Stop-Signal Task represents a measure of behavioral inhibition, the current study indicated that Stop-Signal

Task results were more supportive of state regulation predictions in a group of children and adolescents. Response variability appeared intrinsic to AD/HD group performance across the task and was more related to the primary outcome measure (SSRT) than inhibitory deficits. The current study also emphasized the importance of fine-grained analyses of task components in order to better understand the actual processes being measured. Some support remained for the executive inhibition account of AD/HD based on G-CPT results, and other neurocognitive constructs such as motor processing speed were predictive of AD/HD symptomatology. While response variability in particular warrants continued investigation as to related biological mechanisms and its potential as an endophenotype, results also indicated that a multiple deficit approach continues to be important toward improved understanding of the behavioral and biological manifestations of AD/HD.

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